conflicting results obtained in these controlled trials on the efficacy of omeprazole in gastric body ulcers may conceivably depend on the fact that this drug acts only partially through correcting the pathophysiological alterations that seem to predominate in this disease.

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Dopa responsive dystonia

SIR,—The onset after birth and the progressive course of the illness in the patients reported by Drs Kathryn Boyd and Victor Patterson¹ do not necessarily preclude a diagnosis of cerebral palsy.

Motor patterns may occasionally change with time in patients with cerebral palsy.² Several authors have emphasised that, although cerebral palsy is usually due to a non-progressive cerebral insult, the clinical expression is not inevitably unchanging, or non-progressive.^{2,4} The evolution from hypotonia in early infancy to spasticity in later life has been well documented.⁵ Progressive dystonia or choreoathetosis, or both, may develop within the first two decades in children who appear initially to have a static encephalopathy. In the patient reported by Montagna *et al* abnormal movements developed in the fifth year of life after birth anoxia; the movements progressively worsened and were resistant to drug treatment, including levodopa.⁴

In contrast to the lesson given by Drs Boyd and Patterson that dopa responsive dystonia may be misdiagnosed as cerebral palsy, other authors^{1,4} have emphasised the converse—for example, that some patients diagnosed as having dystonia musculorum deformans (and presumably also dopa responsive dystonia) may have cerebral palsy. Clearly, the differential diagnosis of early onset and progressive dystonia must include both disorders.

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SIR,—We have recently come across another case of dopa responsive dystonia,¹ which had this time been labelled as psychogenic.

A 24 year old woman developed inversion posturing of the right foot at the age of 12 such that her shoe was rapidly worn down along the outstep. By 17 her right leg had stiffened and she walked

with the ankle plantar flexed. Over the next three years the left leg became similarly affected and she appeared to be walking on tiptoe. A notable feature was the considerable variability in the symptoms. She was seen by several consultants in various specialties, and at the age of 19 a psychogenic disorder was diagnosed. In the five years before presenting to us her gait became even more bizarre with opisthotonic like arching of the spine, such that she required support to walk to prevent her falling over backwards. The symptoms remained variable. There was no family history of any movement disorder. Examination confirmed abnormal posturing in the sitting position, but careful assessment of tone did not reveal any extrapyramidal increase. There was no bradykinesia.

The patient started taking Sinemet 110 (levodopa 100 mg, carbidopa 10 mg), one tablet three times a day, and after two weeks was walking almost normally. Withdrawing the Sinemet caused symptoms to recur after four days and its reintroduction rapidly led to their resolution. The patient has remained well and in full time employment with this treatment.

The misdiagnosis of dystonia as a psychiatric disorder is well recognised.² Progressive dystonia with diurnal fluctuation is also very uncommon (three out of 932 patients with dystonia in one series³). In our experience dystonia is rarely misdiagnosed as cerebral palsy and is much more commonly thought to be psychogenic, particularly in the early stages. We agree with Drs Boyd and Patterson that a greater awareness of dopa responsive dystonia is needed and would include psychiatrists as another group requiring some knowledge of this condition. However, as it is so uncommon, referral to a neurologist should be essential to allow greater treatment experience to emerge.

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Practice annual report

SIR,—The advice given by Dr Stefan Cembrowicz¹ is timely in view of the government's proposals for making the production of an annual report part of every general practitioner's terms and conditions of service.²

We thoroughly endorse his observation that producing an annual report acts as "a painless introduction to simple performance review." We were, however, rather disappointed to find only a passing mention of short or long term objectives for his practice.

In the Oxford general practice outcomes project a framework is being developed whereby an annual report is centred on a set of mutually agreed objectives. The objectives might be long term and overarching—for example, to improve access to services offered by the practice—or short term and specific—for example, to establish a diabetic register in 1989.

The objectives are related to the skills that the practice staff should possess, and they should encompass the three aspects of the service that contribute to good quality care—the structure or resources of the practice, the process or activity of the practice, and the outcome or results. The last of the three is the most difficult to measure but is in our opinion the most crucial aspect of quality.

As it would not be possible or even desirable to audit every objective every year establishing a set of "tracer" objectives that encompass structure, process, and outcome and relate to different skills in different areas of the service is advisable. These objectives might be changed every few years to prevent boredom. Setting objectives, making them central to the annual report, and reviewing progress towards them subsequently helps to establish a clear framework in which all those who work in the practice can strive to improve quality. We are interested to hear about other work on objective led annual reports.

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Burns from a tumble drier

SIR,—With respect to the article by Messrs K Herbert and S K Varma¹ I would like to report on a 3 year old girl, who presented at this hospital's casualty department in September 1988.

Apparently, she had been playing that evening and her clothes had been wet. She placed these in the tumble drier and then climbed in herself, managing to close the door behind her. Her father found her and brought her to the hospital, a delay of about 10 minutes, where I was the senior house officer on duty. On arrival she was diagnosed as having a cardiorespiratory arrest. She was resuscitated and intubated, but although she developed a cardiac output, she never showed spontaneous respiration. There was evidence that she had vomited and aspirated and there were full thickness burns to her hands and buttocks. She remained hypotensive despite vigorous fluid replacement with plasma protein fraction and crystalloid. She was then transferred to the regional paediatric unit, the Royal Belfast Hospital for Sick Children, where she was found to have developed a disseminated intravascular coagulopathy, and she subsequently died that night. I wholly agree, therefore, with the recommendations of Messrs Herbert and Varma for greater safety precautions with these domestic appliances.

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Tetrahydroaminoacridine in Alzheimer's disease

SIR,—Dr Jane Byrne and Professor Tom Arie's editorial¹ prompts us to report the effect of physostigmine in another poorly understood syndrome, that of Cheyne-Stokes respiration. We suggest that this might be an expression of a similar deficit in the cholinergic system.

After a mild left hemiparesis a frail 92 year old normotensive woman who was taking digitalis for atrial fibrillation developed ischaemic gangrene of her right foot and leg. The leg was amputated through the knee without preoperative medication under spinal analgesia. She was alert and cheerful throughout, vital signs were unaffected, and blood loss was minimal.

Two hours after the operation, after the effects of the analgesia had worn off, she became stuporous with Cheyne-Stokes respiration (Glasgow coma scale score >8²). She was cold and clammy (axillary temperature 35.6°C), and she had pinpoint pupils. Her pulse rate and volume and

systemic blood pressure were normal. She was given oxygen. Her blood glucose concentration was 3 mmol/l, and it did not improve after injection of 20 ml 50% dextrose solution intravenously. Naloxone 0.4 mg by slow intravenous injection evoked restlessness and movement of the arms and legs and her pupils dilated and reacted sluggishly to light. The pattern of her breathing did not change; stupor rapidly returned, and the pupils constricted. Twenty minutes later physostigmine 2 mg intravenously in divided doses was given empirically. Her pupils rapidly dilated and became reactive with a consensual reflex, her temperature rose, and normal breathing was restored. Although confused, she could converse. Thereafter post-operative recovery was uneventful.

Cheyne-Stokes respiration is commonly a harbinger of death. Various ascribed to poor systemic circulation, raised intracranial pressure, impaired lung dynamics, and reduced sensitivity of the brain stem to hypoxia,³ the condition lacks a truly scientific rationale. General anaesthesia has been incriminated but not spinal analgesia,⁴ and the cause of Cheyne-Stokes respiration in our patient remains obscure.

Although Cheyne-Stokes respiration can revert to normal breathing without treatment, we conclude in this case that reversion was synchronous with and related to the empirical use of physostigmine. If that is so Cheyne-Stokes respiration in our patient may have been the result of a deficit in her cholinergic system. Dr Byrne and Professor Arie stated that "there are good reasons why manipulating the cholinergic system might be effective in treating Alzheimer's disease."¹ One swallow does not make a summer, but little harm and possible good may come from manipulating the cholinergic system in patients with Cheyne-Stokes respiration of obscure aetiology.

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Does constitutional hypotension exist?

SIR,—Dr John Pemberton's recent article highlights the interesting problem of constitutional hypotension¹ and whether this should be recognised as a disease and treatment given to increase blood pressure.

Common symptoms include giddiness, blackouts, and fainting, all of which occur with changes in posture and resultant cerebral hypoperfusion.² To investigate the relation of the above symptoms to blood pressure we recently asked 71 elderly patients (aged 65-90 years) attending a day hospital to fill in a questionnaire regarding symptoms of giddiness, blackouts, and fainting. With the results unknown to the examiner blood pressure was recorded supine and again erect after two minutes' standing using a Hawksley random zero sphygmomanometer. Seventeen (24%) admitted to one or more of the above symptoms. There was no relation between supine systolic blood pressure and symptoms (eight patients had a systolic blood pressure >11 mm Hg, of whom only two had symptoms and both had a postural fall in systolic blood pressure >20 mm Hg). Moreover, 12 of the 17 symptomatic versus 11 of the 54 asymptomatic patients showed a postural fall in systolic blood pressure greater than 20 mm Hg ($p < 0.001$, $\chi^2 = 12.7$). These results suggest that the postural

fall in blood pressure rather than constitutional hypotension was the main cause of symptoms. Therefore, we strongly support Dr Pemberton's conclusions that at the present time there are insufficient data to support the view of constitutional hypotension as a separate disease. We believe that treatment in patients with low blood pressure should be reserved only for those who have symptoms of postural cerebral hypoperfusion after lying and standing blood pressures have been recorded and should be given only after other medical causes have been excluded.

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Percutaneous balloon valve dilatation

SIR,—In their editorial Drs Marc J Levine and Raymond G McKay stated that advanced age is a sufficient risk factor for mitral valve replacement to warrant balloon valvuloplasty as the preferred first line treatment for mitral valve stenosis.¹ Assuming that by advanced age they were referring to patients over 70, I would like to clarify the position of mitral valve replacement in this population.

Most elderly patients are referred for mitral valve replacement after prolonged medical treatment and preoperatively are classified as New York Heart Association functional class IV. Kay *et al* have noticed that "much of the great mortality associated with mitral valve replacement in the elderly was due to the practice of withholding valve replacement from patients until they were end stage IV cardiac cripples;" their series of 30 functional class III patients gave an early mortality of about 3%.² This compares closely with mortality of 3% for the under 65 population.³ There is also great variation among surgical centres, mortality in some varying between 20% and 57%^{4,5} and in others between 5% and 12%,^{6,8} many of these patients being in functional class IV at surgery. In the elderly population most deaths and failure to improve after a mitral valve replacement are a consequence of intrinsic myocardial disease rather than valve related problems, which tend to be the main contributors in those under 65.⁹ Elderly patients surviving the operation achieve functional improvement similar to the under 65 population, 90% improving by one to two functional classes.^{10,11} Therefore, the evidence to date suggests that mitral valve replacement will benefit the elderly and should be offered while the patient still has reasonable myocardial function, ideally not worse than that in class III. Age should not be grouped with severe pulmonary hypertension and biventricular failure as a high risk factor for mitral valve replacement.

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India in AD 2001

SIR,—Professor Sunil K Pandya referred to the formation of a Science Advisory Council to the Prime Minister of India to advise the government on planning for AD 2001.¹ Among other activities the council has been requested to help the country meet the basic needs of every person: food, housing, health, employment, and education.

Social and health indicators for Kerala and India (latest figures available)

Indicator	Kerala	India
Birth rate	22.4	33.6
Crude death rate:		
Urban	6.7	9.0
Rural	7.1	15.1
Infant mortality rate	27.0	96.0
Life expectancy at birth:		
Men	67.0	55.0
Women	70.0	54.0
Adult literacy rate:		
Men	75.26	46.89
Women	65.73	24.82

Sources: *Health Profile Kerala*, Department of Health and Family Welfare (1983), and *Statistics for Planning*, Department of Economics and Statistics (1986), Trivandrum, Kerala.

In this context I wish to point out that the state of Kerala in India has already achieved remarkable success in improving the health of its people.² Kerala is poor even by Indian standards with an income per head substantially lower than the all India average.³ The table gives some of the basic indicators for the state of Kerala and for all India which illustrate the success achieved. The experience of Kerala would certainly be of use to Indian planners.

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Merit awards—£100m management tool

SIR,—About a third of the annual £72m for merit awards is spent each on C, B, and A plus A+ awards. I have been puzzled by the white paper's failure to grasp this opportunity to reform merit awards more effectively as a carrot for NHS work (including, of course, teaching and research). This is one area where the government could and should have been more radical and imaginative. One of the supposed changes proposed—to restrict progression to the remaining three levels to C award holders—is indeed not a change at all.

At present the system ostensibly tries to reward excellence in clinical, administrative, and academic work. It is not considered to be relevant how much