liaison with a drug counselling service, this regimen could be easily undertaken in a primary care setting with the support of a local drug counselling service.

While it is recognized that interpretation of the results may be limited by the small sample size, this lofexidine based regimen may provide the practitioner with an effective alternative method of patient detoxification from opiates which minimizes problems potentially encountered with other regimens in common use.

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Reference

- Gold MS, Potash AC, Sweeney DR, et al. Opiate detoxification with lofexidine. Drug Alcohol Depend 1981; 8: 307-31
- Washton AM, Resnick BR. Lofexidine in abrupt methadone withdrawal. *Psychopharmacol Bull* 1982; 4: 220-221.
- Washton AM, Resnick BR, Gayer G. Opiate withdrawal using lofexidine, a clonidine analogue with fewer side effects. J Clin Psychiatry 1983; 44:

Pertussis in a four-week old baby

Sir,

Baby H was a full-term, normal delivery weighing 3.9 kg. Aged four weeks she developed a dry, 'sticky' cough and increasingly severe cyanotic attacks. Aged five weeks she was admitted to hospital. On admission baby H was apyrexial, but was noted to have a paroxysmal hacking cough. A clinical diagnosis of pertussis was made by a paediatrician familiar with neonatal pertussis in the Middle East (E M). Despite a first pernasal swab being negative, a second pernasal swab yielded a heavy growth of Bordetella pertussis; a throat swab gave a light growth.

Baby H became bradycardic during the bouts of coughing, the heart rate falling from 120 beats per minute to as low as 44 beats per minute, taking a few minutes to return to normal. This was accompanied by cyanosis, and the oxygen saturation fell to 64% on a pulse oximeter reading. She was given oxygen as required and prescribed a two-week course of oral erythromycin 50 mg twice daily. Her cyanotic attacks gradually became shorter and less severe, so that she was able to be discharged home after nine days.

Baby H's mother had had a severe cough with bouts of whooping which had begun three weeks before baby H's admission to hospital. The mother had never had pertussis infection as a child, and had received only diphtheria and tetanus immunization at a time when the side effects and sequelae of pertussis vaccine were under public debate. A pernasal swab was negative. The mother was given a two-week course of erythromycin 250 mg six hourly.

Baby H was the index case in a small epidemic of six cases so far: three siblings living in the same street aged between 14 months and eight years nine months presented to their general practitioner with a three-week history of severe cough when baby H was still in hospital. Two further unconnected cases living in different areas were notified at the same time. These cases were given a two-week course of erythromycin to reduce the carriage rate of the bacterium. Three secondary cases occurred (after the incubation period of pertussis). In the Isle of Wight the immunization target rate for pertussis of 90% has been surpassed.

Pertussis is a dangerous infectious disease that is well controlled by widespread immunization in industrialized countries. Outside industrialized countries and where immunization rates fall below 70% in developed countries epidemics occur every four to five years, and can be controlled only by widespread immunization; other measures, such as antimicrobial chemotherapy, offer only negligible benefit. In the developing world it remains a source of high morbidity, with a mortality of up to 50% in infants under six months presenting with symptoms.² In babies aged less than three months paroxysms of cough may begin or end with apnoea; fatal asphyxia can occur.³ Complications include central nervous system sequelae of anoxic brain damage, and lung damage.

This case is reported to emphasize the fact that pertussis may occur as early as four weeks of age, or even in the newborn, as there appears to be no inborn immunity.3 Isolation of the causative agent remains the gold standard for the early diagnosis of pertussis. For this purpose a pernasal calcium alginate swab is preferred to a nasopharyngeal swab, with direct inoculation of a charcoal horse blood agar plate.4 Longstanding cough of unknown aetiology is the main indication for pertussis serology (enzyme linked immunoabsorbent assay). Control of pertussis is now dependent on the use of acellular pertussis vaccine as the fourth and fifth doses of diphtheria, pertussis and tetanus immunization, given at age 15 months and prior to school entry, as licensed in the United States of America from late 1991.1 Treatment of pertussis should be limited to the use of one of the three antibiotics erythromycin, ampicillin and co-amoxiclay, to which this pathogen has been shown to be most sensitive.5 These antibiotics have no protective effect against the development of bronchopulmonary complications defined by the secondary bacterial flora, and therefore have no value in prophylaxis.

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References

- Hodder SL, Mortimer EA. Epidemiology of pertussis and reactions to pertussis vaccine. Epidemiol Rev 1992; 14: 243-267.
- Robinson MJ (ed). Practical paediatrics
- Edinburgh: Churchill Livingstone, 1986: 251-252. Rendle-Short J, Gray OP, Dodge JA. Synopsis of children's diseases. 6th edition. Bristol: Wright, 1985: 100.
- Hopper JE. Keuchusten: diagnostik, klinik and
- therapie. Monatsschr Kinder 1990; 138: 244-248. Petrova MS, Shakirova RG, Antonova NA, Feoktistova GN. Antibiotikoterapiia pri kokliushe u detei. Antibiot Khimioter 1992; 37: 40-43.

Alternative contracts in the NHS

I have followed with interest the correspondence¹⁻³ concerning the 1986 British Medical Journal articles⁴⁻⁶ which I wrote with Denis Pereira Gray and Alan Maynard. Julian Tudor Hart describes 'two diametrically opposed paradigms [which] compete for the minds of young doctors':1 these are characterized as a competitive market and a cooperative public service. He asserts that the former model 'by definition... entails winners and losers', and contrasts this with the latter in which 'bad practice could be resourced rather than punished.'1

Hart suggests that our interpretation of the 1986 proposal for a good practice allowance was incompatible with his vision of a cooperative public service. I cannot agree. A careful reading of our articles⁴⁻⁶ will reveal that we expressly warned against the dangers of creating winners and losers; we urged that the good practice allowance be designed to achieve comparable standards of quality in all general practices; and we argued that poor practice should be corrected, not by financial punishment but by additional resourc-

Hart writes 'when paradigms collide, some mutual incomprehension is probably inevitable'.3 I want to suggest that such mutual incomprehension is far from inevitable. Indeed, Popper demonstrates that in science competing paradigms may continue side by side for long periods of time, and that new paradigms do not obliterate old ones, but rather incorporate them into more successful models.7 Although the variously constructed models for the health service are political rather than scientific, the principle of adaptation and improvement rather than of conflict, victory and defeat, holds.

Our papers were intended to offer the best available way out of what seemed to us to be the impasse of National Health Service general practice in the mid-1980s.4-6 Hart describes '...the old ramshackle independent professional paradigm', and chronicles the sad consequences of 30 years of professional burnout in the Upper Afan Valley since 1961. It may be timely to remember that prior to 1990 there were a number of perceived crises in the NHS. To suggest that those of us who sought to vary the arrangements for the NHS that were then extant, were in some way wilfully damaging a sound working model of public service on the basis of a speculative model of market economics, is to misremember history with a vengeance.

The successful future of the NHS will demand not the collision of paradigms, but what Handy describes as the management of paradoxes.8 General practice has in the past been fairly successful in managing three quite different and mutually contradictory models of medical care - the biotechnical, the biographical and the proactive. Now the challenge is to reconcile the new contractual accountabilities of a managed care model with the moral obligations of a professional public service.

I realize that in prolonging this correspondence I risk the danger of this exchange of letters being seen as no more than a spat between grumpy old men. But I share with both Hart and Gray a sense of the importance of clarifying the history of events and ideas. One of the consequences of this correspondence has been to provoke me to re-read what I wrote and published a decade ago. Such reading is rarely an unmixed pleasure. However, I take courage from the writer Saul Bellow. In the preface to a recent collection of his own past essays he writes about the relative discomfort of re-reading ones own past pronouncements.9 He concludes drily that it gives great satisfaction '...to have rid oneself of tenacious old errors. To enter an era of improved errors.'9

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References

- Hart J. Burnout or in to battle? [letter]. Br J Gen Pract 1994: 44: 96
- Gray DP. Alternative contracts in the NHS [letter]. Br Í Gen Pract 1994; **44:** 479.
- Hart JT. Alternative contracts in the NHS [letter]. Br J Gen Pract 1994; 44: 593.

 Gray DP. Marinker M, Maynard A. The doctor, the patient, and their contract. I. The general ractitioner's contract: why change it? BMJ 1986; **292:** 1313-1315.
- Marinker M, Gray DP, Maynard A. The doctor, the patient, and their contract. II. A good practice allowance: is it feasible? *BMJ* 1986; **292**: 1374-1376.
- Maynard A, Marinker M, Gray DP. The doctor, the patient, and their contract: III. Alternative contracts: are they viable? *BMJ* 1986; **292:** 1438-1440.
- Popper K. The myth of the framework. London:
- Routledge, 1994. Handy C. *The empty raincoat*. London: Hutchinson, 1994
- Bellow S. It all adds up. London: Secker and Warburg, 1994.

Prevention of end stage renal failure

Sir.

Cairns and Woolfson have written an important editorial on the current potential for slowing the rate of progression of renal failure (November Journal p.486). However, while they discuss early detection of at-risk adult patients with hypertension, diabetes, proteinuria or haematuria in primary care, they omit to mention detection of the at-risk child.

Thirteen per cent of adults in Europe accepted onto renal replacement programmes in 1992 had pyelonephritis (European dialysis and transplant association renal replacement register, unpublished data). A considerable proportion of these cases will have arisen from processes in childhood. These processes include coarse renal scarring associated with infection in the presence of urinary reflux, almost certainly beginning in the first two years of life.1

Clinical and experimental evidence strongly suggest that the rapid introduction of antibacterial treatments can limit or prevent development of renal scarring.2-8 In other European countries such as Sweden considerably more attention has been paid to swift detection and treatment of infant urinary tract infections than in the United Kingdom, and although ascertainment bias may be a problem, it appears that there is a lower prevalence of renal failure associated with urinary infection in childhood in Sweden than in the UK.9 Urinary infection is common in early childhood, but nonspecific in its presentation. Further epidemiological studies are required to define the antecedents of chronic pyelonephritis in adult life, and the exact incidence and proportion of children with infection at risk of renal damage. Meanwhile, failure to investigate promptly urinary tract infections in children and to arrange follow up and prophylactic antibiotics appears to be contributing to avoidable renal damage in the UK.10

Before appropriate action can be taken the diagnosis must be considered, and inclusion of this cause of avoidable end stage renal failure in editorials and review articles will help to keep it in mind.

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References

- 1. Jacobson SH, Eklof O, Eriksson CG, et al. Development of hypertension and uraemia after pyelonephritis in childhood; 27 year follow up. BMJ 1989; 299: 703-705.
- Ransley PG, Risdon RA. Reflux and renal scarring. Br J Radiol 1978; 51 suppl 14: 1-35.
 Shah KJ, Robins DG, White RHR. Renal scarring
- and vesico-ureteric reflux. Arch Dis Child 1978; 53: 210-217
- 4. Ransley PG, Risdon RA. Reflux nephropathy: effects of antimicrobial therapy on evolution of the early pyelonephritic scar. *Kidney Int* 1981; **20**: 1193-1196.
- Winberg J, Bollgren I, Kallenius G, et al. Clinical pyelonephritis and focal renal scarring. Pediatr Clin North Am 1982; 29: 801-814.
- Smellie JM, Normand JCS, Ransley PG, Prescod N. The development of renal scars: a colloborative study. BMJ 1985; 290: 1957-1960. Wikstad I, Hannerz L, Karlsson A, et al. DMSA
- scintigraphy in the diagnosis of acute pyelonephritis in rats. *Pediatr Nephrol* 1990; **4:** 331-334. Smellie JM, Poulton A, Prescod NP. Retrospective
- study of children with renal scarring associate with reflux and urinary infection. BMJ 1994; 308:
- 9. Esbjörner E, Aronsow F, Berg U, et al. Children with chronic renal failure in Sweden 1978-1985. Pediatr Nephrol 1990; 4: 249-252.
- South Bedfordshire practitioners' group.

 Development of renal scars in children: missed opportunities in management. *BMJ* 1990; **301:** 1082-1084.

Chlamydia trachomatis

Thompson and Wallace report finding a 3% prevalence of positive monoclonal antibody tests for C trachomatis in 145 asymptomatic women aged between 15 and 29 years presenting to their general practitioners for routine cervical cytology (letter, December Journal, p.590). They do not quote test sensitivity or specificity and it is therefore impossible to estimate positive and negative predictive values. In simple terms, we do not know how many infected or uninfected women were wrongly identified by false negative or false positive tests.

The recommendation that general practitioners screen for C trachomatis in 'a selected population on the basis of age