they come to apply for life insurance. This course of action is certain to affect adversely our relations with our patients. We should tell our patients instead that they can at any time request that the details of highly confidential problems are not to be entered into the NHS record envelope. There is, however, the problem of the patient who moves away and whose practitioner is asked, at a later date, to provide a report from the records for a third party. As the patient's present practitioner one has no control over exactly what might be extracted from the records for such a report, although only the information relevant to the purpose of the report should be included. Many doctors keep highly confidential information about patients between their ears. Others keep confidential notes separate from the record envelope, but available to partners, and do not send them on with the envelope should the patient move. A useful technique which is used in this practice is to make an entry "confidential information" and merely to add the name or names of the doctor or doctors who are aware of the information, which is not recorded at all.

We should be ready to discuss our patients' most sensitive problems with them, but we should also be prepared to discuss with them exactly what detail is entered into the record envelope. We may thus save them from anxiety if they are later asked to give permission for medical reports to be supplied to a third party.

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Appeasement 1977 style

SIR,—Dr J Tudor Hart (2 January, p 171) is very misguided in his criticism of the BMA and this journal.

I remember the heady days at the birth of the NHS when we were full of juvenile enthusiasm and ready to storm the citadel. We really believed that the millennium had arrived and we could look forward to an endless vista of comprehensive care when everyone could have unlimited free access to the best that money could buy. Alas, this was a pipe dream; our professional leaders at the time were a little wiser than us and at least could see that this could never come to pass.

But Dr Hart must wake up. Has he remained in a trance for the last quarter of a century? Let him open up his eyes and see what is going on.

We have never been unsympathetic to the needs of other health workers, but I have had my own problems, as I'm sure that he has too. We have throughout this time been ever frustrated by the dead hand of bureaucracy, but this is now getting worse with the proliferation of administrative bodies and interference by politicians. We now see the cooks and porters demanding a say in the admission and treatment of patients in hospitals and threatening to shut them down if they don't get their way. I can't really believe that Dr Hart can be happy about this.

The scene is again changing as a result of recent Acts of Parliament. The Association has had to become a trade union, but it may not have escaped Dr Hart's notice that at the last Annual Representative Meeting the members recoiled in horror at the mention of a closed shop. At a recent local divisional

meeting our members clearly indicated by an overwhelming majority that they would never again consider withdrawal of medical services as part of industrial action. We at least have learnt something by experience.

I was horrified recently to see the size of the membership of our local community health council. God help us if it ever develops enough teeth to cause serious damage, as some of its members clearly want it to. I shudder whenever I hear the words "team" or "teamwork" used. This often leads to "medicine by committee" and is a certain recipe for getting nothing done.

So let Dr Hart come down from the clouds, re-enter the world, and listen to his colleagues. He too might learn something.

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Thyroid disease, asthma, and prostaglandins

SIR,—The association between the worsening of established asthma and the onset of hyperthyroidism^{1,2} was discussed in a recent leading article (5 November, p 1173) and was confirmed in the preliminary retrospective survey reported by Jean Fedrick and Dr J A Baldwin (10 December, p 1539). Treatment with anti-thyroid drugs ameliorates the symptoms of asthma. It was suggested that excess thyroid hormone(s) might exacerbate asthma either by altering cellular levels of cyclic nucleotides and sensitivity to catecholamines or by increasing the rate of metabolism of corticosteroids, but as yet there is little satisfactory experimental evidence for these theories. We wish to suggest an alternative working hypothesis—namely, that the worsening of asthma may be caused by potentiation of the direct actions of prostaglandins (PGs) on the airways as a result of decreased pulmonary PG metabolism consequent to the hyperthyroid state.

We have shown elsewhere^{3,4} that breakdown of PGE₂ and PGF_{2α} in vitro by the lungs and kidneys of rats made hyperthyroid by an 18-day course of L(-)thyroxine injections (200 µg subcutaneously) is reduced by a mean of 49.1 ± 5.6% (n = 15) compared with euthyroid controls (P < 0.01), most probably because of decreased levels of the enzyme PG 15-hydroxydehydrogenase which catalyses the first step in the pathway of biological inactivation of the "classical" PGs. A deficiency of this magnitude in PG metabolism might be sufficient in vivo to potentiate the actions of PGs released endogenously in the lung. Normally the intensity and duration of action of PGs are restricted by rapid breakdown close to the site of release.

It is known that PGE₂ and PGF_{2α} (as well as the more potent but evanescent PG endoperoxides and thromboxanes) are synthesised and released from the lung,⁵ and enhanced output follows several types of physical insult⁶ or immunological challenge.⁷ PGF_{2α} is always bronchoconstrictor, either on isolated tracheal smooth muscle or by aerosol in vivo; PGE₂ is generally bronchodilator, although constriction is seen after intravenous administration; so far as is known the unstable intermediates are constrictor.^{8,9} Furthermore, asthmatic subjects are much more sensitive than controls to the bronchoconstrictor effects of PGs,¹⁰ and levels of the principal

blood-borne PG metabolites are elevated several-fold after challenge.¹¹ Taken together, this evidence strongly implicates PGs as important humoral mediators of asthma, although certainly not necessarily the sole mediators. To strike a proper note of caution, it should be noted that aspirin-like PG synthetase inhibitors at best afford only feeble protection against or poor relief from asthma and that PGs may themselves have anti-inflammatory, anti-allergic properties¹² as well as their better-publicised pro-inflammatory actions.^{13,14}

Our hypothesis for the worsening of asthma in the hyperthyroid state due to defective PG metabolism could be tested (albeit indirectly) in a number of simple ways. For example, it is conceivable that these rare patients might benefit from therapy with aspirin-like drugs more than the "usual" asthmatic subject (provided that their asthma is not precipitated by aspirin hypersensitivity) and that they may demonstrate exaggerated responsiveness to other effects of PGs when administered exogenously.

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¹ Settipane, G A, Schoenfeld, E, and Harmolsky, M W, *Journal of Allergy and Clinical Immunology*, 1972, **49**, 348.

² Bush, R K, Ehrlich, E N, and Reed, C E, *Journal of Allergy and Clinical Immunology*, 1977, **59**, 398.

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⁵ Mathe, A A, *Acta Physiologica Scandinavica*, 1976, suppl 441.

⁶ Palmer, M A, Piper, P J, and Vane, J R, *British Journal of Pharmacology*, 1973, **149**, 226.

⁷ Piper, P J, and Vane, J R, *Nature*, 1969, **223**, 29.

⁸ Mathe, A A, et al, *New England Journal of Medicine*, 1977, **296**, 850 and 910.

⁹ Smith, A P, in *The Prostaglandins*, vol 1, ed P W Ramwell, p 203. New York, Plenum Press, 1973.

¹⁰ Mathe, A A, et al, *British Medical Journal*, 1973, **1**, 193.

¹¹ Green, K, Hedqvist, P, and Svanborg, N, *Lancet*, 1974, **2**, 1419.

¹² Morley, J, in *Rheumatoid Arthritis*, ed J L Gordon and B L Hazleman, p 43. Amsterdam, Elsevier/North-Holland, 1977.

¹³ Vane, J R, in *Advances in Prostaglandin and Thromboxane Research*, vol 2, ed B Samuelsson and R Paoletti, p 791. New York, Raven Press, 1976.

¹⁴ *Prostaglandin Synthetase Inhibitors*, ed H J Robinson and J R Vane. New York, Raven Press, 1974.

Respiratory crisis in Parkinson's disease

SIR,—Respiratory infections are common among elderly patients with Parkinson's disease and pose a real problem to clinicians engaged in their care. The mortality from this cause can be reduced to a minimum if this medical emergency in an elderly ill patient is recognised early and the proper therapeutic regimen with amantadine and antimicrobial agents instituted promptly.

When a patient with undiagnosed or untreated Parkinson's disease develops a chest infection his respiration is impaired by the rigidity of the intercostal muscles so that he is unable to cough up the infected sputum. In treating a chest infection in such a patient, therefore, antimicrobial agents, physiotherapy to the chest, and oxygen are not enough and an anti-Parkinsonian drug is indicated in addition, consideration being given to rapidity of action. When used alone in doses of 100 mg twice a day the therapeutic effects of amantadine are evident within 48-72 h. More effective action can be achieved when amantadine is added to an existing regimen of treatment consisting of anticholinergic agents or levodopa or both,

but if the patient is not already taking anti-Parkinsonian drugs levodopa is not a very good agent to use in these circumstances, as it takes a longer time, usually about three weeks, to show its therapeutic effect. Sacks *et al*¹ reported that 12 of 25 patients treated with levodopa for the first time developed "florid respiratory crises," while an additional eight patients developed respiratory and phonatory "tics."

The mortality in Parkinsonian patients with respiratory infection is much higher than in other groups of ill elderly patients. In the past this type of incident, which should be termed a "respiratory crisis," has not attracted sufficient attention from practising physicians. If geriatricians were more alert to the danger and to the need to give additional treatment for their patients' Parkinson's disease when respiratory infection occurs a large number of deaths in the elderly could be prevented, particularly in the winter.

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¹ Sacks, O W, Kohl, W, and Messeloff, C, *Lancet*, 1970, **1**, 1006.

Necrotising enterocolitis

SIR,—Your leading article on necrotising enterocolitis of the newborn (21 January, p 132), although commendable in its discussion of epidemiology and possible aetiology, gives little useful guidance on the indications for surgery. Failure to improve on conservative management is a prime indication, but you make no mention of the commonest indications—peritonitis and signs of perforation with free gas seen on a plain radiograph. A review of 12 cases coming to surgery in one hospital in Bristol showed the indications to be peritonitis (6 cases), perforation (4 cases), and obstruction (2 cases) and in this series 75% recovered. In addition, in other published series I have studied¹⁻³ "persistent bleeding from the gut" has not been an indication for surgery in any case.

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¹ Stevenson, J K, *et al*, *American Journal of Surgery*, 1969, **118**, 260.

² Dudgeon, L D, *et al*, *Journal of Pediatric Surgery*, 1973, **8**, 607.

³ Stein, H, Kalvin, I, and Faerber, E N, *Journal of Pediatric Surgery*, 1971, **10**, 943.

Treatment of hereditary angioneurotic oedema with methandienone

SIR,—The usual treatment of hereditary angioneurotic oedema in the past few years has been with epsilon-aminocaproic acid and possibly with fresh frozen plasma transfusions on rare occasions if a severe attack with laryngeal obstruction threatens. Androgens have also been used and Spaulding¹ showed that methyltestosterone had a beneficial action. Later less masculinising androgens, fluoxymesterone and oxymetholone,² and also danazol³ have been shown to be effective.

We report here the excellent results of treating two patients with methandienone (Dianabol). They were non-related men aged 31 and 27 and had had attacks of angioneurotic oedema and abdominal pain every few weeks for many years. In both cases the

C1 esterase inhibitor was absent or, in one case, was on one occasion only 10% of the mean normal adult value. Both patients had been helped by epsilon-aminocaproic acid but attacks still occurred every few weeks and plasma transfusions had been necessary at times.

Both patients have now been on methandienone 5 mg twice daily for 10 and 8 months, respectively, and during these periods have remained completely clear of attacks. In addition, in both cases the level of C1 esterase inhibitor has returned to normal values. No adverse effects have been noted.

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¹ Spaulding, W B, *Annals of Internal Medicine*, 1960, **53**, 739.

² Davis, P J, Davis, F B, and Charache, P, *Johns Hopkins Medical Journal*, 1974, **135**, 391.

³ Gelfand, J A, *et al*, *New England Journal of Medicine*, 1976, **295**, 1444.

Adrenocortical suppression in workers manufacturing synthetic glucocorticoids

SIR,—I have read the paper by Dr R W Newton and others (14 January, p 73) three times but found no mention of the name of the offending synthetic glucocorticoid. Such reticence may be rather touching for the drug company concerned, but is surely scientifically unacceptable. With the missing name supplied their paper may well become of great interest, while without it it seems pointless and even (for those who have guessed wrongly) misleading.

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*.*The authors preferred not to name the drug on the grounds that the manufacturers had been so helpful in arranging the study.—Ed, *BMJ*.

Otosclerosis and the operating microscope

SIR,—As a retired otologist I greatly appreciate the short leading articles in the *BMJ* which so often give some idea of what aspects of medical science are attracting attention at the present time. It distressed me, therefore, in the recent article on otosclerosis (14 January, p 63) to find some serious inaccuracy in historical fact. To some this may seem of small importance, but as these articles are well documented they may be accepted and quoted and error then become fact. I refer to the information regarding Julius Lempert which is not in accordance with fact.

Julius Lempert never used the operating microscope; instead he used Cameron magnifying spectacles. The article referred to¹ makes no mention of a microscope but refers on p 62 to the necessity for "powerful magnifying glasses" which are the Cameron glasses referred to above.

The credit for the introduction of the operating microscope must go to Sweden, where Professor Gunnar Holmgren pioneered the fenestration operation with great pertinacity and courage in face of failure. He stimulated workers in many countries, including Julius Lempert in America. Lempert's great contribution was the description of the

one-stage operation, which was made possible by the introduction of chemotherapy. In conclusion I would add that I have seen Julius Lempert operate many times in New York and counted him as a close friend for whose surgical achievements I have great admiration.

There are other matters in this article with which one might well disagree, but these are matters of opinion. At least let us get the matters of fact correct.

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¹ Lempert, J, *Archives of Otolaryngology*, 1938, **28**, 42.

Threadworms in homosexual males

SIR,—Threadworm infestation is common in childhood, when it is acquired by eating contaminated food or having contact with infected clothing or other fomites. In recent years infections have been described in homosexual males,¹ but the mode of transmission in such cases has not been clearly defined. The following is a report of a young homosexual male who apparently acquired the infection during oroanal sexual intercourse.

An 18-year-old mechanic attended a department of sexually transmitted diseases complaining that he had had perianal irritation, particularly at night, for two weeks and had noted "worms" on his stool on one occasion. Threadworms in the anal canal were noted on proctoscopy. Rectal cultures for *Neisseria gonorrhoeae* and for *Chlamydia trachomatis* were negative, as were serological tests for syphilis.

He had had one sexual relationship during the preceding nine months and on that occasion, two months before attending the clinic, had had oroanal and orogenital contact only. His contact, a 20-year-old clerk, attended the clinic several days later at the request of the patient. He was asymptomatic, but a Scotch tape swab from the perianal region² showed five eggs typical of those of *Enterobius vermicularis*. This young man had had several homosexual relationships, all casual, within the preceding six months, but none could be contacted to be examined.

Oroanal contact is a common practice among homosexual males, at least 70% of those attending this clinic having performed this at some time. While it is impossible to be absolutely certain that the patient described above acquired his infection in this way and not, for example, from contaminated fomites, the history of contact with an infected person followed by a prepatent period is strongly suggestive that this was the mode of transmission.

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¹ Markell, E K, and Voge, M, *Medical Parasitology*, 4th edn, p 357. Philadelphia, Saunders, 1976.

² Waugh, M A, *Transactions of the St John's Hospital Dermatological Society*, 1972, **58**, 224.

Central dopamine blockade in anorexia nervosa

SIR,—Dr M R Trimble (10 December, p 1541) reported a rapid weight gain in a female patient with anorexia nervosa treated by weekly injections of fluspirilene. The patient also "felt very much better" and "lost her