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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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,