The part of the lower urinary tract in the female that does relate to vaginal development is the distal urethra. Both these structures derive a common origin from the Müllerian tubercle and this is reflected in their structure, and in particular the stratified squamous epithelium that covers both organs. It is the stratified squamous epithelium of the urethra that is the oestrogen-sensitive component of the urinary tract. The trigone develops quite independently from the vagina. With age there is increasing spread of squamous epithelium along the urethra and this may extend up on to the trigone in certain patients. However, the precise relationship of this squamous spread to urinary tract symptoms is not completely understood. Certainly it is optimistic in the extreme to claim that urinary stress incontinence can be cured by oestrogen alone. Oestrogen will undoubtedly help those patients with atrophic urethritis and a urethral syndrome, particularly when the latter is associated with urge incontinence. Similarly the oestrogens will help in the preoperative preparation for patients undergoing vaginal surgery.

Finally, I think it should be said that a prostatectomy can cure the overflow incontinence associated with chronic retention. The improvement in catheter materials has certainly made catheter drainage more acceptable and there will always remain patients who are not suitable for any form of treatment by surgery. However, as you make clear, long-term catheter drainage is the final degradation for the urinary tract. An aggressive approach to the understanding and management of urinary incontinence remains a priority for surgical development in Britain.

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Uncovering physical illness in elderly patients with dementia

SIR,—We were interested to read your recent leading article (10 December, p 1499) on this subject, as we recently undertook physical examinations and selected laboratory investigations on patients in a long-stay psycho-geriatric ward in what you so quaintly refer to as a "residential psychiatric unit."

Twenty-one patients who had been admitted more than 12 months (mostly 1-15 years) previously were examined. They were aged between 65 and 86; one was male. The most striking finding was that so few patients were without any abnormality. Indeed, there were only two patients without a significant anomaly and one of these refused venepuncture.

Physical examination revealed only a small number of abnormalities, though constipation was common. On the other hand laboratory investigation, following a standard protocol with additional tests when indicated, yielded a mass of abnormal results. Serum folate levels were commonly in the low or low normal range and this was associated with mild macrocytic anaemia in six patients; the explanation for this folate deficiency was not clear but may have been dietary. Two patients had mild iron deficiency anaemia with occult blood in the stools. Three patients had a raised serum alkaline phosphatase value; two of these had a raised erythrocyte sedimentation rate (ESR) and one had known pelvic masses.

There were, perhaps surprisingly, no abnormalities of thyroid function or of plasma calcium or electrolyte levels. The blood urea concentration was raised in one patient. Urinary infection occurred, asymptotically apart from the almost invariable urinary incontinence, in six patients.

Your leading article argues for a rational approach to ensuring the physical wellbeing of the elderly demented patient. Our experience suggests that the traditional "annual physical" is not rational but, rather, time-consuming and ineffective. On the other hand a combination of careful examination when the clinical state changes and, say, biennial laboratory screening using some modification of the above protocol might be more helpful to the patient. Such problems as folate deficiency and constipation are surely preventable and should be prevented. A difficulty arises with the interpretation and investigation of such "abnormalities" as a raised ESR¹ or mild degrees of iron deficiency; we feel that over-zealous investigation may not be acceptable for many of these patients, but of course a balance must, as always, be drawn between the discomfort of investigation and the distress of occult disease rendered still more occult by the patients' mental clouding.

We should like to thank the nursing staff of Ward 24, Friern Hospital, for their unstinting assistance in this project, and the laboratory staff of Friern and the Royal Free hospitals who carried out the investigations.

TERENCE CUBITT
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London N11¹ Boyd, R V, and Hoffbrand, B I, *British Medical Journal*, 1966, 1, 901.

Boston Collaborative Drug Surveillance Program

SIR,—In the book review by Professor C T Dollery (17 December, p 1592) he reported that the Boston Collaborative Drug Surveillance Program is "now discontinued." We have no idea where Professor Dollery got this impression, but we wish to correct the error. We have continued to carry out intensive in-hospital monitoring, currently in five countries. In addition, we have expanded the monitoring programme to include surgical patients. Last year we published 18 original papers and currently have some 12 in press. We look forward to an interesting and productive future.

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The NHS 5-ml spoon

SIR,—A J Cronin's Drs Finlay and Cameron investigated the quantities of medicine administered when ordinary teaspoons and tablespoons were used. It seems that there has been very little improvement since their day.

A little investigation, asking patients to help, indicates that the 5-ml NHS issue measuring spoon (BS 3221/4) delivers between 2.5 ml and 4 ml when used in the home. The most likely quantity is 3 ml. At 4 ml the spillage between bottle and mouth is much and messy. Under laboratory conditions the

5-ml spoon does indeed measure 5 ml if the meniscus is observed correctly. However, in this state it is nearly impossible to hold it in the hand without spillage. Careful ordinary mortals align the edges of the spoon with the highest part of the convex meniscus, so have a volume of about 4 ml.

The NHS spoon offends against most rules governing accurate measurement. It is shallow, with a very wide "neck" (meniscus surface). I suggest that its use be discontinued and that a new 5-ml measure with suitable handle should replace it. The new measure should have a flat bottom, vertical sides, and a depth equal to the radius of the bottom. This shape should give a reasonably convenient and acceptable measure for ordinary use. It need not cost more or involve more manufacturing problems than the present inadequate spoon.

S W V DAVIES

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Confidentiality and life insurance

SIR,—The letter from Dr T M Pickard (10 December, p 1544) and the comment from Mr V M Kendall (14 January, p 109) raise wider issues concerning the information contained in the NHS medical record envelope. On the one hand we must maintain accurate records containing those personal details about patients which are essential for good general practice care. On the other hand we must encourage our patients to talk to us about deeply personal issues and be able to assure them that whatever they say is truly in confidence and will not be passed on to any third party.

Mr Kendall points out that loose terminology may cause confusion in assessing reports from doctors. It may also cause confusion among doctors sharing the patients' care if imprecise diagnostic labelling is used in records. Thus the unhappy patient should be described as unhappy and not depressed; a single marginally elevated blood pressure reading should not be labelled as hypertension.

There appears to be an increasing tendency for patients to be asked to give permission for their medical histories to be supplied not only to life insurance offices but also to other third parties. One would accept Mr Kendall's comment that life offices are providing a service in the interests of the patient, but many employers are now also asking prospective employees to sign a declaration permitting the general practitioner to release his or her medical history to the company. It is made clear to the person that he or she will not be employed unless this is done. Many members of the public realise that this practice is growing. A most undesirable trend is that the patient may now refrain from discussing with his or her GP those confidential and personal issues which are so important in the management of patients in general practice, knowing that information about these problems may subsequently be extracted from the medical record and passed to a prospective employer. For example, in this practice a patient with a sexual deviation who clearly needed help procrastinated for several years before finally asking if he could discuss the problem without the details being recorded in the medical record envelope.

Dr Pickard suggests that we tell our patients that anything they say to us may be taken down and used in evidence against them when

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,