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also does not help in this respect and a pressure flow study of micturition is mandatory.

The urethral pressure profile is helpful in its indication of the maximum pressure exerted by the urethral sphincter; however, the results vary widely in both males and females and the two sexes should not be compared as the functional anatomy is completely different.[1]

The comment in relation to table V (p 1103, para 4) that four of the unstable bladders demonstrated a pressure rise to below 13 cm H_2O appears to ignore the basic standards of the International College of Surgeons, which accepts a 15 cm H_2O pressure rise as being necessary prior to a diagnosis of instability.

The definition of bladder atonicity also seems rather all-encompassing. Bladders with large capacities may empty effectively. Large residuals certainly occur with both high pressure and low pressure outflow obstruction and also with decompensation of the detrusor in the absence of obstruction.

It is unfortunate that a study of this sort, which requires much hard work, did not provide guidelines for the prediction of specific features which relate to incontinence in the elderly. A short précis of the approach to the treatment of this difficult clinical problem would have been welcomed.

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[1] Farrar DJ, Whiteside CG, Osborne JL, Turner-Warwick RT. *Surg Gynaecol Obstet* 1975;**141**: 875-81.
[2] Plate P, Susset J. *J Urol* 1980;**123**: 64-74.
[3] Abrams PH. *Urol Clin North Am* 1979;**6**: 103-10.

**We sent these two letters to the authors, who reply below.—Ed, *BMJ*.

Sir,—While we agree with Drs Shah and Whiteside that the history suggests an urodynamic diagnosis in some elderly incontinent patients, in more we find that this is not the case. All patients referred to our clinic had been seen by doctors and most had been treated prior to referral without effect.

We can only accept their criticism and that of Dr H D H Eastwood that some of our male patients might have had outflow obstruction. We did not do pressure flow studies of micturition except in patients in whom we thought there was a clinical possibility of outflow obstruction. In this respect we perhaps underinvestigated our patients. Outflow obstruction was not thought to be present in any of the 22 patients with unstable detrusor contractions, and one reason might be that the patients were selected in that they were all referred by other doctors. This may also explain the lack of patients with genuine stress incontinence, which we always look for.

Mr Hinton and Mr Stanton have still not answered why urodynamic assessment is "necessary for accurate diagnosis" of incontinence in young patients while in the older the algorithmic method is advocated. Could not this be applied at any age? We concede that we have overstated our case, and agree with their present suggestion that the algorithmic approach is intended for assessment at the primary care level, where we find it practised, and not for specialist urodynamic clinics. We also agree that faecal impaction and urinary tract infection can be diagnosed without cystometry, and would not now do this examination before successful treatment of these conditions.

Mr Hilton and Mr Stanton have slightly misread our urodynamic data. No patient in our normal group had a capacity of less than 100 ml, even though some elderly women are very small in stature. (For example, one woman of 79 years was only 122 cm in height and weighed 26·3 kg.) As we stated in our paper, it is the overall appearance of the cystometric trace which enabled us to categorise patients and not individual volumes and pressures. This explains why four patients were put into the unstable group despite not their meeting the criteria of the International Continence Society. It could be argued that these four should have been in a separate group but there was nothing apart from the pressure of the contraction at incontinence to distinguish them from others in the unstable group. It is also possible that three of our patients in the "normal" group were miscategorised since they had pressure rises at capacity of from 20 to 39 cm H_2O. However, these patients were not incontinent, had normal bladder sensation, and did not show any unstable contractions. We entirely agree that the "normal" group cannot strictly be classed as normal since they were not selected from individuals with no recent urinary complaint. We agree too that the irritable and atonic groups are both clinical and urodynamic diagnoses. The cystometric trace is usually characteristic in the irritable group, and most patients with atonic bladders have large capacities with only small rises in pressure with filling. The latter group were almost all referred by the local urologists and so had had outflow obstruction excluded. Therapy is based on, and yet different for, each diagnostic category, which must therefore be found as accurately as possible. These two groups are particularly responsive to drug treatment, details of which will be published later.

Our statement that incontinence in most elderly patients is not associated with immobility and cerebral disease was based on the results of the mobility assessment, mental test scores, and clinical examination—the point being that previously incontinence in older patients was thought to be largely due to immobility and cerebral disease, whereas we feel that this reflected the selection of patients for investigation. Since a large number of our patients were from geriatric medical wards, it would be surprising if these diagnoses were not still relatively common. The incidence would drop even more, we suspect, in unselected elderly incontinent patients living in the community. Immobility itself should not cause incontinence at any age unless the patient is helpless and without help. Is the incontinence following an unstable contraction in a patient who has become less mobile due to his immobility or to his bladder instability?

Similarly, there are many elderly patients with cerebral disease who are not incontinent, and many who are but have no cerebral disease. We feel that the association between incontinence and cerebral disease in elderly patients is not inevitable, as previously suggested. In some it is unquestionably connected, nevertheless, the urodynamic findings of those in whom it is connected are no different from those in whom it is not.

There were a great many data on these patients that could not be included because of space. We hope to rectify this in the near future, and in particular to report on the effects of management. We cannot show from our present data, however, that elderly patients who have had cystometry do better than those who have not. We believe that they do; certainly none had had this investigation before referral and only 16 (the "normal" group) had achieved continence.

Finally, incontinence, like the examining finger on the irregular pulse, indicates that something is wrong. Given that the clinical history in an elderly incontinent patient is "of limited value in establishing the cause of urinary incontinence," as Mr Hilton and Mr Stanton said in their paper, if the patient has no demonstrable stress incontinence and no palpable bladder, faecal impaction, or urinary tract infection, and a trial of therapy has been given by a competent doctor, is urodynamic investigation not indicated? Only 22 out of our patients could have avoided such investigation on these clinical grounds. The procedure is quick, easy, and attended by minimal discomfort; has a very low complication rate; and gives almost 100% accuracy of diagnosis and therefore a clear indication of how to manage the patient. How many other invasive hospital investigations can claim this? We remain therefore unrepentent if a little chastened.

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Asbestos fibres and the environment

Sir,—I found the two articles by Daphne Gloag (14 and 21 February, pp 551 and 623) very interesting. But I was surprised that she made no reference to dust from brake linings of motor vehicles as a source of atmospheric contamination, not at the point of manufacture but from vehicles in motion. There must be enormous quantities of dust loaded with fibres being released daily and this would, I expect—with carbon monoxide and lead—be a major pollutant in traffic jams.

Furthermore, what about the risk to the do-it-yourself motorist when he renews his brake linings and is intimately exposed to the fine dust which has to be cleaned from the drums?

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**Daphne Gloag writes: "Raised air concentrations of asbestos, roughly proportionate to the number of vehicles passing, were found at toll stations where sampling was done in an American survey.[1] But although brake linings contain a substantial proportion of chrysotile most is changed morphologically by the friction and the remaining chrysotile fibres are mainly short[2]—and so possibly less hazardous. Nevertheless, even a small proportion of unchanged fibres could be harmful. There is some evidence of asbestosis in vehicle maintenance workers; and some mesotheliomas have been found in garage workers,[2] which may have been caused by their occupation, though in a recent case-control study no increased risk was evident.[3] Thus there seems to be a distinct but small occupational hazard which suggests that do-it-yourself car maintenance would have negligible risks."—Ed, *BMJ*.

[1] Bruckman L, Rubino RA. *Journal of the Air Pollution Control Association* 1978;**28**: 1221-6.
[2] Lorimer WV, Rohl AN, Miller A, Nicholson W, Selikoff IJ. *Mt Sinai J Med NY* 1976;**43**: 207-11.
[3] McDonald AD, McDonald JC. *Cancer* 1980;**46**: 1650-6.

Plasma protein binding of drugs

Sir,—I would agree with Drs W E Lindup and M C L'E Orme (17 January, p 212) that it is difficult to define what is a "highly protein-bound drug. The concept of protein binding in antibiotic therapy is important as is only the unbound fraction of the drug which can act against bacteria.

As high protein binding may affect the distribution of drugs in "the tissues" where the majority of infections occur I was particularly interested to study the interrelationship of binding and tissue penetration. By using a Cantharides blister technique[1] which induces a mild inflammatory response one can sample the blister fluid and determine the protein free fraction of drug. Seven β-lactams were studied (fig)—Bay k 4099 is an experimental penicillin