cigarette manufacturers to carry out the necessary studies since it is impossible to patent any modification that might be suggested by such research. The massive investment in the development of artificial tobacco substitutes appears not to lead to any substantial improvement in safety, since the carbon monoxide yield remains unchanged and the tar : nicotine ratio is increased.

The reason for the introduction of tobacco substitutes is not to benefit the consumer but to increase the producer's profit. Since the tobacco industry lacks the financial inducement to try and introduce a safer cigarette, perhaps the introduction of a variable rate of duty on cigarettes, taxing safer cigarettes less harshly than those brands considered harmful, would be to the good of both manufacturers and consumers. Since the Government is involved in a campaign to lessen the hazards due to cigarette smoking, it would perhaps be appropriate for it to support research into the role of oxidants and other factors in smoking-related disease. The necessary money to finance this research could be found if the suggested levy on less safe cigarettes was used solely for this research.

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Classification of cigarettes

SIR,—In his recent article (12 June, p 1430) Dr M A H Russell discusses the desirability of smokers changing their brand of cigarette to gain a satisfying nicotine intake for the least intake of tar.

While agreeing with this aim, I wonder if a greater success would not be achieved if the figures for tar and nicotine yields were actually printed on the cigarette packets. Unless a smoker had access to the Health Departments' tables of tar and nicotine yields,[1] which are not publicised as widely as they might be, the present system of five categories stamped on the packets could be rather unhelpful to smokers attempting gradually to reduce their tar intake. This largely stems from the range of tar yields represented by each category. For example, in the "low tar" category the range is from 1·25 to 9·58 mg/cigarette and in the "low to middle tar" category from 11·16 to 15·89 mg/cigarette. Ranges similar in extent to that in the latter category are found in the other three groups. Without access to the tables it would be impossible to determine how much less tar a smoker would be exposed to by changing brands, especially within any one category.

At a time when increasing quantification of values is being sought the present system of classifying tar and nicotine yields is surely outmoded and if the Department of Health is serious in its desire for smokers to reduce their tar intake there can be little excuse for not printing actual values on cigarette packets. During a recent visit to Austria I was interested to note that this was already standard practice on all cigarettes offered for sale, including British brands.

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[1] Health Departments of the United Kingdom, *Tar and Nicotine Yields of Cigarettes*. London, DHSS, 1976.

Orf in Britain

SIR,—While admitting that a British doctor in the centre of a large city might see only an occasional case of orf (Dr Rupert Mason, 26 June, p 1587), the contrary is more likely to be the case for the rural practitioner. I recently reported a small epidemic in a rural area which occurred in the spring of 1975.[1]

There is little doubt that the occurrence of this disease is associated with outbreaks of animal vesicular disease in sheep. This is common in farming areas and is due to a pox virus infection affecting the udders of lactating ewes, causing vesicular eruptions. The infection is so severe, and presumably painful, to the lamb that it may die through an inability to suckle. Infection of humans by the virus causes an eruption not unlike an infected wart which is given the name "contagious pustular dermatitis." The lesion is classically about 1 cm in diameter and has a firm, red base surmounted by a reddish blue capsule, which after a few days may turn into a greyish yellow vesicle with the appearance of a pustule. This may enlarge and become flat-topped, though the bulla often becomes encrusted and its centre may be umbilicated. If the lesion occurs adjacent to the nail bed it can be mistaken for paronychia. If such a mistaken diagnosis is made and the lesion incised the absence of pus or exudate should in itself provide a clue to the correct diagnosis.

It appears likely that the virus requires certain favourable climatic conditions for an epidemic to develop, and I suspect that these conditions coincide with lambing only in occasional seasons. When there is a serious outbreak among sheep the country doctor will almost inevitably see cases among his farming patients. Infection is most commonly by direct contact of infected animal to man and consequently the most frequently affected parts are the fingers and hands. However, it seems also likely that occasional infection is due to contact with contaminated fencing or hedgerows, and undoubtedly some of the cases I have seen have fallen into this category.

There is no effective curative treatment. The lesion is best left and, if possible, kept dry. The condition is always self-limiting and complete resolution within three to five weeks is the rule. Occasionally secondary bacterial infection complicates lesions of orf, but this usually responds to a course of an antibiotic, allowing natural resolution of the underlying virus lesion to take place.

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[1] Hall, M S, *Journal of the Royal College of General Practitioners*, 1976, **26**, 203.

Genetic counselling in Huntington's chorea

SIR,—Drs Gillian M and N W Glendinning (3 July, p 46) are, of course, right that the family doctor is the ideal person to inform and support families in which a member has Huntington's chorea. First, however, the family doctor has to learn that a person on his list belongs to such a family, either from the individual himself or from a colleague who has made the diagnosis in the affected member. Perhaps in rural Somerset one may rely on this invariably taking place in one way or another. It is not so over most of the country. When the diagnosis is made, say, in a 50-year-old patient neither the neurologist nor the family doctor will always see that this diagnosis is made known to all the patients' children, who may be scattered around Britain or even abroad, or to the children's family doctors. For example, we know of one family in which the mother died after 17 years in hospital; two years after her death her six adult daughters did not know of, or did not realise the significance of, the diagnosis.

We believe that individuals at risk have a right to know the truth of their situation, including the genetic risks, so that they themselves can make informed choices about family planning. We believe that suitable publicity will help to achieve this and alert families to the need to go to their family doctors for help. Methods of publicity must be constantly reviewed and adapted to current needs. We look forward to the day when all Huntington's chorea families are helped with "gentleness and care" by sympathetic family doctors like the Glendinnings.

After the recent film on Yorkshire TV and other publicity, Combat received 296 letters, mostly from Huntington's chorea families, only one of which was in any way critical. The common refrain in these letters was "we knew that Huntington's chorea was in our family but not what it meant for us and our children."

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Urinary retention in women

SIR,—Your leading article on this subject (26 June, p 1554) draws timely attention to the frequently underdiagnosed (and therefore often maltreated) condition of urinary retention. One of the difficulties is an adequate definition of acute and chronic retention. Doran and Roberts[1] state that acute retention of urine should be painful and acute in onset and that less than 1 l of urine should be obtained on catheterisation. Unfortunately this definition is too narrow and precludes some genuine cases in which retention is painless and where amounts of urine greater than this can be removed at the first episode of retention. Chronic retention was not defined by Fox *et al*,[2] but it may be considered to be the result of a single or repeated episode of acute urinary retention producing painless bladder distension, voiding taking place by overflow.

I would like to emphasize two points. Firstly, the role of drugs in causation of retention and difficulty in voiding, and secondly, the prodromal state of straining to void and a slow stream. In a series of 440 women seen over the last five years with voiding disorders who have been referred to urodynamic clinics, 27 (6%) had either acute or chronic retention or a noticeably slow stream. Of these five patients had a relevant drug

history—namely, Bellergal (belladonna alkaloids with ergotamine and phenobarbitone), amitriptyline, Stelabid (isopropamide and trifluoperazine), orphenadrine, and phenytoin. Two patients had been on a combination of Bellergal and amitriptyline. All of these drugs have an anticholinergic effect and will produce a decrease in bladder contractility. There were seven patients with a slow stream. Their aetiology is shown in the table; one of them has gone into retention but subsequently has been relieved. Of the total cases, 15 were found to have a neurological component—namely, multiple sclerosis, prolapsed intervertebral disc, laminectomy, tethered cord, and ependymoma of the spinal cord. I am unable to support your contention that hysteria is not a common cause as six patients were found to be suffering from this.

Aetiology*	Acute retention (8)	Chronic retention (12)	Slow stream (7)
Drug	2	2	1
Neurological	6	6	3
Hysteria	2	4	
Other		1 pelvic exenteration 1 diabetes mellitus	1 distal urethral stenosis 1 following vaginal surgery 1 No abnormality found

*Some patients had a multiple aetiology

I would like to place greater emphasis on the early diagnosis of these conditions by the use of voiding pressure and flow rate studies aided by electromyography of the external urethral sphincter and the use of pharmacological agents (for example, carbachol and alpha-blocking drugs such as phenoxybenzamine) to determine subsequent treatment.

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[1] Doran, J, and Roberts, M, *British Journal of Urology*, 1976, **47**, 793.
[2] Fox, M, Jarvis, G J, and Henry, L, *British Journal of Urology*, 1976, **47**, 797.

SIR,—I was interested to read your leading article on this subject (26 June, p 1554), the references of which are misquoted.

For many years[1-4] I have stressed how common urinary troubles, including obstructions, are in women. The female bladder and its mechanisms are heir to all the disorders affecting the male. This is not surprising as embryologically both have the same origin. Every structure present in the one is represented in the other, either fully matured or rudimentary according to the genetic sex. Both in lectures and articles I have tried to spread the gospel of the truth of the significant incidence of urinary obstruction in the female and discussed the causes, differential diagnosis, and treatment.

Many of the obstructions occur at the bladder neck and simulate the common prostatic obstructions in the male. All the various male syndromes, including acute and chronic retention of urine, chronic retention with overflow, secondary infection, haematuria, latent uraemia, etc, also occur in the female. Recently[5] I reviewed 84 such cases, mostly treated by perurethral resection of the obstructing tissue. Some years ago in one family I removed the grandfather's, his son's, and his daughter-in-law's prostate with equally satisfactory results.

I would hope the profession is now much more aware of the frequency of retention of urine in women than your leading article would suggest.

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[1] Moore, T, *Proceedings of the Royal Society of Medicine*, 1953, **46**, 558.
[2] Moore, T, *Lancet*, 1960, **1**, 1305.
[3] Moore, T, *Congrès de la Société Internationale d'Urologie*, 1961, vol 2, p 363.
[4] Corrin, B, Mayor, D, and Moore, T, *Journal of Urology*, 1963, **90**, 434.
[5] Moore, T, *European Urology*, 1975, **1**, 32.

**We regret the errors in our references to which Mr Moore refers. In each case the date of publication should read 1975, not 1976 as printed.—ED, *BMJ*.

Amoxycillin, talampicillin, and ampicillin

SIR,—I hope you will permit me to reply to the various points raised by Dr D A Leigh (24 July, p 232) in response to the views I expressed (10 July, p 106) on the propriety of prescribing new derivatives of ampicillin in preference to the original drug.

(1) Dr Leigh gives the impression that the *British National Formulary* (p 104) restricts its preference for amoxycillin over ampicillin to circumstances in which high blood levels are important. In fact, the relevant passage reads, "It [amoxycillin] should therefore replace ampicillin, especially when high blood levels are important."

(2) Evidence that amoxycillin penetrates bronchial secretions more readily than ampicillin, and that this is not dependent on the level of purulence, in no way proves that it is more effective than ampicillin in the treatment of bacterial infections of the respiratory tract. Similarly, although higher peak serum levels can be obtained with amoxycillin and talampicillin than with ampicillin, this cannot and should not be regarded as a valid reason for prescribing the more expensive ampicillin derivatives unless there is incontrovertible evidence that they are superior in therapeutic effect to ampicillin itself. I would challenge Dr Leigh to cite any such evidence.

(3) With regard to relative costs, I note that Dr Leigh equates *500 mg* 4 times daily of ampicillin with 250 mg 3 times daily of amoxycillin and talampicillin. In fact, it was shown in one of the articles[1] he quoted in his letter (admittedly reporting an uncontrolled clinical trial) that "the overall success rate with ampicillin (*250 mg* 4 times daily) was 96% and with talampicillin (250 mg 3 times daily) was 94·8%. If he accepts the validity of these figures, the cost comparison he should have made was with *250 mg* 4 times daily of ampicillin. On that basis one day's treatment with amoxycillin would cost three times as much, and talampicillin more than twice as much, as one day's treatment with ampicillin.

(4) Dr Leigh chided me for not referring to the articles[1 2] he quoted in support of his contention that the clinical use of talampicillin "should be associated" with a lower incidence of diarrhoea than ampicillin. Both articles, however, reported "open" uncontrolled clinical trials, the scientific reliability of which must always be open to question. There is, as far as I know, no reported evidence of a significantly lower incidence of diarrhoea with amoxycillin than with ampicillin.

To be frank, my contretemps with Dr Leigh is something of a side issue. The main purpose of my last letter was to draw attention to the irresponsible attitude of the *British National Formulary* in stating that amoxycillin "should replace oral ampicillin" when there is no evidence that ampicillin is not equally effective in those clinical circumstances where amoxycillin is now being prescribed. In 1975 the National Health Service was spending over £10 million on ampicillin. The highly successful advertising campaign now being promoted to popularise amoxycillin and talampicillin could at a very conservative estimate increase the bill for this group of antibiotics by £5 million. For all we know a similar situation may exist in respect of a large number of other drugs, and the squandering of money on expensive new derivatives of drugs which are not demonstrably superior in therapeutic effect to the original product may well be depriving the National Health Service of resources it could use much more profitably for other purposes.

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[1] Knudson, E T, and Harding, J W, *British Journal of Clinical Practice*, 1975, **29**, 255.
[2] Jaffe, G, *et al*, *Practitioner*, 1976, **216**, 455.

Effect of salicylates on creatinine clearance

SIR,—In their interesting article (3 July, p 16) Drs H C Burry and P A Dieppe have shown that in some patients with rheumatoid arthritis and in some normal subjects salicylates raise the serum creatinine level and reduce the creatinine clearance, but they incline to reject the idea that a change in glomerular filtration rate is responsible. Earlier this year we reported this change in serum creatinine in five subjects (four with systemic lupus erythematosus and one normal) in a study protocol of the hepatic effects of aspirin.[1] In our patients, however, the blood urea nitrogen was affected as well as the creatinine. We have now extended our observations to a substantial number of patients with lupus or rheumatoid arthritis and found similar effects. Furthermore, we have established that inulin clearance is reduced in parallel with creatinine clearance, strongly suggesting that glomerular filtration is indeed altered.

In view of the known effects of salicylates on prostaglandin synthesis and the role of prostaglandins in regulating renal blood flow the mechanism for these effects on glomerular filtration probably involves prostaglandins. In accord with that possibility we have observed similar effects on creatinine clearance by two other non-steroidal anti-inflammatory drugs, indomethacin and naproxen, which also inhibit prostaglandin synthesis, and studies on fenoprofen[2] suggest that it too may cause a similar change. It is not clear from Drs Burry and Dieppe's study or from ours whether or not there is attenuation of the changes with time.

Since the observed changes in serum creatinine and creatinine clearance do appear to be caused by changes in renal function we believe that the statement that "clearly the creatinine clearance test is not suitable for assessing renal function in patients suffering from rheumatoid arthritis" might be better phrased to suggest that renal function ought to be assessed with caution in patients taking drugs that may alter it, such as salicylates or other non-steroidal anti-inflammatory drugs.

Although now and then there are new things under the sun, both Drs Burry and Dieppe and we appear to have rediscovered renal effects