Wetting and Soiling

SIR,—We are concerned that there may be some misunderstanding of Dr. A. C. Woodmansey's advice on the treatment of disorders of elimination in children (15 July, p. 161). Child psychiatrists tend to see a very biased sample of enuretic children and to a lesser extent of those with encopresis; this sample tends to be biased in favour of those children and families who show undue anxiety about the problem. To this extent and in the need to relieve the anxiety rather than resorting immediately to symptomatic treatment, we would fully agree with Dr. Woodmansey. We would also agree that in many cases it is unrealistic to anticipate biological maturity.

However, there are many children in the community, and particularly those who attend their general practitioners and school or local authority enuretic clinics, who are not seriously disturbed but are merely worried, fed up, and self-conscious about the problem, which particularly handicaps them when they have to sleep away from home—for example, when on holiday. We find that these children gain great confidence and esteem from successful symptomatic treatment—for example, by conditioning which may have a success rate of 92%.¹

There are families where the disorder of elimination has come to assume disproportionate significance, and the disturbed family situation which may result requires careful investigation and management. Yet other cases require more detailed physical investigation to exclude cases of faecal impaction and urinary abnormalities.

It might be possible to argue with Dr. Woodmansey's conception of the theory of the enuresis alarm as being aversive; in our practice few children see it as punitive and in fact find its use most rewarding. We feel that due weight should be given to this form of help rather than mere reassurance and a "wait and see" policy, which is hardly likely to appeal to mothers who have to wash and dry night wear and at least two sheets every day, year after year.—We are, etc.,

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SYLVIA DISCHE

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School Health Service,
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¹ Young, G. C., *British Journal of Hospital Medicine*, 1969, 2, 628.

SIR,—It would be unfortunate if Dr. A. C. Woodmansey's views on enuresis expressed in his review article (15 July, p. 161) were to be regarded as other than a very personal account of the problem. By labelling as infantile incontinence all enuresis that is not due to fear, he implies that developmental factors are of little importance in aetiology beyond the age of infancy. Such a view is not held by many child psychiatrists or paediatricians, nor is it supported by the facts. By subtly condemning the use of the electric enuresis alarm he suggests depriving children and families of one of the few treatments of demonstrated effectiveness. By failing even to mention the use of amitriptyline and imipramine he achieves the doubtful distinction of having written a review article about a condition while completely ignoring what are probably the most com-

mon remedies for the condition applied by general practitioners.

Of course, nocturnal enuresis sometimes arises through fear and is perpetuated by habit. Discussion with parents and child aimed at removing the tension surrounding it will sometimes (perhaps often) relieve the symptom without the necessity for specific treatment. But acceptance of the importance of psychological factors should not preclude the use of physical treatments where these have proved value.—I am, etc.,

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SIR,—I read with interest the article by Dr. A. C. Woodmansey (15 July, p. 161) but I was somewhat surprised he rejects completely all symptomatic treatments of nocturnal enuresis, including the alarm and drugs.

Over 20 years I have found that much of the emotional distress is secondary to the symptom, and this distress arises even with the most sympathetic parents and where there is no evidence that there has ever been coercive attempts at "training." The maturational defect which delays cortical control persists for some time and often creates the emotional distress rather than the reverse.—I am, etc.,

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Supersonic Radiation Risks

SIR,—I have read with interest your leading article (15 July, p. 130) on "Supersonic Radiation Risks."

I feel I should point out an error. It states that "A mass miniature radiography chest x-ray examination on an old set may give 10 mrems. . . ." Mass radiography of the chest with old equipment could give an exposure of 1,000 mrems. With modern equipment this is frequently between 100 and 200 mrems. Of course, this is only to a portion of the body and if the gonads are not in the beam they only receive a few mrems or fractions of a mrem.—I am, etc.,

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SIR,—Your leading article (15 July, p. 130) on "Supersonic Radiation Risks" might create the impression that persons sitting close to colour television sets are actually subjected to x rays at an exposure rate of 0.5 mrems per hour. Such an exposure rate, at 50 mm from the outer surface, is in fact the upper limit of x-ray emission from television sets.¹

In view of possible public concern, it is important to point out that recent studies² on British receivers show that the exposure rate at the surface of any set leaving a manufacturer is unlikely to exceed 0.02—that is, one twenty-fifth of the limit. The probability of excessive emission is slight except in deliberately applied severe test conditions.

It should be added that the average view-

ing distance is 2.5 m, where the exposure rate is one-fiftieth of the rate at 50 mm.—I am, etc.,

M. C. O'RIORDAN

National Radiological Protection Board,
Surrey

¹ British Standards Institution, *Specification for safety requirements for mains-operated household sound and vision equipment*. London, B.S. 415, 1972.

² O'Riordan, M. C., and Casbolt, P. N., *Nature*, 1970, 228, 420.

Neurotic Dyspnoea

SIR,—I was specially interested in the feature "Second Opinion please; Problems with Asthma" (1 July, p. 37).

We all from time to time meet patients whose confidence is severely shaken—in themselves, in their physicians, or in the treatment they are having. Three cases illustrate my point. A 72-year-old man had suffered from a coronary occlusion and was discharged on the usual treatment, including anticoagulants. A few days after leaving hospital he complained of feeling very short of breath, but examination did not reveal any special finding to account for the dyspnoea, and to begin with he was reassured and told that the cause of the symptoms was undue anxiety concerning his condition. However, the dyspnoea continued and worsened. A few days later he had to be readmitted to hospital, where unfortunately after about twelve hours he died.

A woman in early middle age of good mental stability and intellectual competence was short of breath. She had been particularly short of breath for some weeks and could not explain why. Her case had been investigated by a number of doctors and no adequate cause for her shortness of breath had been discovered. She said to me "I suppose I must be getting neurotic." There was nothing significant to find on physical examination and chest x-ray had been passed as normal. I was, therefore, not able to make a confident diagnosis, although I made a suggestion as to the possible cause of the symptoms, and about three quarters of an hour later an incident occurred suggesting that my supposition could well be right.

About a year ago I was concerned in the attempted resuscitation of a young woman of 19, who had been brought to the hospital unconscious. We were not able to revive her, and it is probable that death had occurred in the ambulance on the way to the hospital. It seems that the patient had been taken to her doctor by her parents because she had become extremely short of breath and after diagnosing asthma he sent her to the local chest clinic for x-ray. About one minute after the chest x-ray had been exposed the patient collapsed and had to be rushed to hospital by ambulance.

In the first case necropsy showed that the patient had a pulmonary embolus. In the second case, not long after I had left the patient an attack of chest pain and a small haemoptysis occurred. She was put on anticoagulants, her symptoms improved, and later she was able to return to work. In the last case there was of course a coroner's inquiry and it was found that the cause of death had been pulmonary embolism.

Certainly small, and many large, pulmonary emboli do not cause pleural pain, and do not cause haemoptysis. It is possible that quite a large number are in fact

symptomless, and probably very frequent in the population. The body has a thrombolytic system. I believe therefore that clots in the vessels are fairly common and are dissolved again without always causing symptoms. I think that we now know that pulmonary emboli can occur without there being any good clinical evidence of a donor site, such as a thrombosed vein (13 December 1969, p. 676).

It is extremely difficult to know when the diagnosis of a pulmonary embolism should be made, and it may be that some of us who are aware of its frequency have a tendency to over-diagnose the condition. However, I believe that many cases of apparent neurotic dyspnoea—for which no cause can be found—are in fact due to multiple pulmonary embolism.¹ Fortunately, the majority of cases do not prove to be fatal, but I have reached the stage that I become immediately extremely worried whenever I hear of an individual who is short of breath with apparently no cause. I am sure that patients with genuine dyspnoea from pulmonary embolism are very likely to lose confidence in themselves, in their physicians, and in their treatment. I know of nothing so likely to relieve the breathlessness as anticoagulants, as I think that there can be a genuine physical basis for dyspnoea in pulmonary embolism which is difficult to diagnose.—I am, etc.,

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Edgware General Hospital,
Edgware, Middx

¹ Greenberg, H. G., *American Review of Respiratory Diseases*, 1965, **92**, 215.

Metastatic Calcification and Dialysis

SIR,—I read with great interest your leading article on "Metastatic Calcification and Dialysis" (25 March, p. 762). I would like to comment on the statement that inhibitors, such as those described by Yendt *et al.*,¹ are removed by haemodialysis. In a previous study² my colleagues and I confirmed Yendt's earlier observation that uraemic serum contains a factor that inhibits the calcification of ricketic rat cartilage in vitro. However, when we compared blood specimens obtained from patients on chronic treatment before and after haemodialysis and peritoneal dialysis, we found that, at the end of the procedure, the haemodialysis specimens still contained the inhibitor, whereas those obtained at the end of peritoneal dialysis did not. We concluded that peritoneal dialysis removes this inhibitor but haemodialysis does not.

The fact that pyrophosphate is removed by haemodialysis³ suggests that Yendt's inhibitor is not pyrophosphate, but rather a large molecule or a molecule bound to protein. It remains to be demonstrated whether there is a causal relationship between this finding (removal of the inhibitor by peritoneal dialysis but not by haemodialysis) and our observation that osteomalacia of renal osteodystrophy progresses in patients on chronic haemodialysis but not in those on chronic peritoneal dialysis.⁴ On the other hand, the observed persistence of Yendt's inhibitor in the blood of haemodialysed patients does not necessarily contradict the observation that soft-tissue calcification progresses in some haemodialysis patients.⁵ These two observations can be reconciled when one recalls that calcification is the end

result of a process involving, among others, calcifying solutions (CaXP), pH, and inhibitor(s) of calcification, some of which may be dialysable and some not.—I am, etc.,

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- 1 Yendt, E. R., Connor, T. B., and Howard, J. E., *Bulletin of the Johns Hopkins Hospital*, 1955, **96**, 1.
- 2 Oreopoulos, D. G., Pitel, S., Husdan, H., deVeber, G. A., and Rapoport, A.; presented at the American Society of Nephrology, November, 1971.
- 3 Russell, R. G. G., Bisaz, S., and Fleisch, H., *Archives of Internal Medicine*, 1969, **124**, 571.
- 4 Oreopoulos, D. G., *et al.*, presented at the Henry Ford Hospital, International Symposium on Clinical Aspects of Metabolic Bone Disease, June, 1972.
- 5 Boner, G., Jacob, E. T., Pevzner, S., and Jungmann, A., *Israel Journal of Medical Sciences*, 1971, **7**, 1182.

Carcinoma of the Cervix and Herpesvirus

SIR,—Some of the avenues of research recommended in your leading article (3 June, p. 548) concerning herpes simplex virus 2 (HSV-2) and cervical carcinoma are impractical. To monitor women who have been exposed to HSV-2 for many years would be difficult. The clinical diagnosis of herpes genitalis in high risk groups for development of cervical carcinoma is uncommon. Not one case has been confirmed during an on-going colposcopic and histological study of 700 such women confined to a penal institution, even though high prevalence rates of major cervical epithelial abnormalities and venereal disease exist.¹ These women are notoriously difficult to follow up because of their social environment and would respond poorly to epidemiological investigation. If women are included in such a study on serological rather than clinical evidence of infection then the high prevalence rate of HSV-2 antibody present in the general population would further complicate selection.²

Your suggestion that the recent studies recognizing incorporated viral DNA in tumour nuclei could profitably be extended to determine whether "malignancy arises from this integration of viral and cellular DNA," is not novel. Reid³ demonstrated histologically the uptake by cervical cells of fragments of human sperm (DNA) and their subsequent incorporation into the cells' genome. By chemical analysis he showed that the admixture of the nucleic acids of these genomes produced a possible mutation.⁴ If incorporated DNA be mutagenic as you imply, and remembering that cervical carcinoma is a coitally transmitted disease, the amount of DNA of spermatozoal origin normally in contact with the cervix far exceeds that derived from a viral source. Spermatozoal DNA would seem to be a more likely mutagen than HSV-2, as proposed by Reid and Coppleson.⁵

Recent evidence suggests that certain males are more likely than others to induce carcinoma in their female partners. In Puerto Rico cervical carcinoma developed in eight wives whose husbands had penile cancer.⁶ None arose in a matched control group. The evidence that one man had cohabited with a number of women who subsequently developed cervical premalignancy and malignancy in an Atlanta housing estate⁷ suggests that a high risk male as well as female exists in relation to the aetiology of this malignancy.

The increasingly liberal sexual behaviour of certain Jewish males could also account for the alteration in the traditional immunity of the Jewess to cervical cancer.⁸ Martin⁹ showed the increased incidence of venereal disease in husbands of American Jewesses with this malignancy. Ravich¹⁰ suggests that the increase in prostatic cancer in Jewish males, in whom it is still uncommon, may result from high rates of venereal infections and increased cohabitation inter-ethnically among these men. Israeli Jewish women with cervical cancer, interviewed by H. Pridham (personal communication), were more likely to have had coitus with a non-Jewish male and to have husbands who were more promiscuous than the ordinary population controls.

Although these data suggest that a venereally-transmitted mutagenic agent like HSV-2 is involved no direct evidence of this exists. Indeed, no elevation of viral antibody levels in husbands of women with cervical cancer is reported. However, an alternative explanation could be that some alteration has occurred in spermatozoal DNA which makes it highly mutagenic when incorporated into the cervical cell. B. L. Reid (personal communication) has recently demonstrated the complicated structure of DNA existing in the mucoid coat surrounding the sperm head. Changes in this structure, possibly as a result of promiscuity, may hold the key to an increased "neoplastic ability" of certain males. It is hoped that further research on this malignancy will tend to concentrate as much on the male as on the female.—We are, etc.,

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MARGARITA STEVENSON

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- 1 Singer, A., and Stevenson, M., *Acta Cytologica*, 1972, in press.
- 2 Singer, A., *British Medical Journal*, 1971, **1**, 458.
- 3 Reid, B. L., *Lancet*, 1964, **1**, 21.
- 4 Reid, B. L., and Blackwell, P. M., *Australian Journal of Experimental Biology and Medical Science*, 1967, **45**, 323.
- 5 Reid, B. L., and Coppleson, M., in *Scientific Basis of Obstetrics and Gynaecology*, ed. R. M. McDonald. Oxford, Churchill, 1971.
- 6 Martinez, I., *Cancer*, 1969, **24**, 777.
- 7 Smith, D., and Jenkins, R. S., *American Journal of Obstetrics and Gynaecology*, 1969, **104**, 603.
- 8 Coppleson, M., and Reid, B. L., *Pre-clinical Carcinoma of the Cervix Uteri*, Oxford, Pergamon Press, 1967.
- 9 Martin, C. E., *American Journal of Public Health*, 1967, **57**, 803.
- 10 Ravich, A., *Cancer*, 1971, **27**, 1493.

Wine and Migrainous Neuralgia

SIR,—Migraine is probably related to ingestion of food. Fothergill blamed malt liquors and other food in sensitive individuals as causal.¹ Wilks recognized migraine, as did many before him, as a spasm of vessels from vasomotor phenomena, sympathetic stimulation, and vasodilatation.² Other work^{3,4} confirms the food relationship. Hanington⁵ related the vasomotor phenomena to tyramine in the food.

We have assayed the histamine, tyramine, bradykinin, and "other" amine content of red and white Australian wines, the former of which precipitate migrainous neuralgia in a migraine subject and the latter of which do not. This subject suffered severe attacks at least once every four weeks for many years, but was symptom free for 18 months after avoiding cheese, chocolate, strong beer, red