of them unresponsive to pain; grade 4) who had abscesses which were both massive and multilocular. (The accompanying illustration shows EMI scans of one of these cases; the brain oedema and mass effect are obvious.) The response of these patients to immediate excision, with simultaneous reduction of tentorial hernia, was rapid, with complete recovery.

EMI scans of massive and multilocular right frontal abscess before and after excision.

Deep abscesses in the thalamus, pons, or medulla cannot be excised, but are very rare; multiple abscesses certainly can be excised, as in one of the cases reported in our original article.

Finally, we are not alone in our views about primary excision of cerebral abscess. French neurosurgeons at the Bristol meeting of the Society of British Neurological Surgeons in April 1977 pointed out that they had reached this conclusion some years ago.¹

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¹ Taylor, J C, and Choudhury, A R, *Journal of Neurology, Neurosurgery and Psychiatry*, 1977, **40**, 1026.

Radiation of the young brain

SIR,—No mention was made in your leading article entitled "Does radiation of the young brain affect growth hormone?" (27 August, p 536) of the important observations regarding the moderate impairment of growth and development which occurred in persons in Hiroshima who were exposed during intra-uterine life or early childhood to a single dose of mixed neutron-gamma radiation from the atomic bomb.¹⁻³ In Nagasaki, where the

atomic bomb radiation was almost pure gamma in type, growth alterations in exposed infants and children were not significantly different from those in the non-exposed. It was observed in Hiroshima that the earlier the age at time of exposure, the greater the impairment of growth. Air dose radiation measurements for those with growth reduction were mostly in the range of 50-150 rads for the children exposed in utero and 100-300 rads for those who were exposed during the first 5-10 years of life. Pituitary gland and hypothalamus dose estimates for the bomb survivors would have been only a fraction of those for the same tissues in children who receive therapeutic craniospinal radiation. It is unlikely that the radiation doses to the pituitary glands and hypothalamic areas of the Hiroshima exposed were large enough to have produced enough direct tissue damage to have resulted in permanent impairment of glandular function. The possibility of increased glandular tissue sensitivity during early development, such as occurs with the thyroid, resulting in a growth defect due to reduction in growth hormone or primary or secondary hypothyroidism cannot be excluded however.⁴

Growth impairment following radiation exposure during infancy and childhood is probably related to many factors, of which the quality, intensity, duration, and location of the radiation, age at time of exposure, and a great many other host factors play impairment roles. Structural changes may be due to primary or secondary reduction in the production of growth or thyroid hormones, direct damage to bone and other structural tissues, nutritional inadequacies, or various combinations of these factors.

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- ² Belsky, J L, and Blot, W J, *American Journal of Public Health*, 1975, **65**, 489.
- ³ Blot, W J, *Journal of Radiation Research*, 1975, **16**, suppl, p 97.
- ⁴ Conard, R A, *Brookhaven National Laboratory—Report 50424*, 1975.

Thyroid disease and asthma

SIR,—A recent leading article (5 November, p 1173) described an association between severity of asthma and hyperthyroidism such that the asthma appeared to improve with adjustment to the euthyroid state. Similarly asthma was said to improve if the patient became hypothyroid. These findings prompted us to analyse the data of the Oxford Record Linkage Study to ascertain whether patients who were hyperthyroid were at increased risk of developing asthma and conversely whether hypothyroid patients were at reduced risk of doing so.

The data analysed for this purpose consisted of details of all hospital discharges and deaths occurring to a defined population over a five-year period. The data were linked together into person-orientated longitudinal records^{1 2} and analysed using our standard package,³ which computes the expected number of persons to have any association between diseases after taking account, inter alia, of the variation of each disease with sex, age, and time period. Corrections are introduced for loss of the cohort through death and migration out of the area.

Of 890 persons on the files with a diagnosis of hyperthyroidism but no record at that time of asthma, only one was subsequently admitted with asthma. The expected number of persons to have this association was 0.9. In contrast, there were 316 persons with a diagnosis of hypothyroidism, two of whom were subsequently admitted with asthma (both after an interval of four months). The expected number of persons with this combination was 0.27 ($P=0.03$).

Analysing the files from the opposite viewpoint, there were 1360 persons with admissions for asthma but no record of thyroid disorders. Of these, one subsequently developed hypothyroidism (expected = 0.29, $P=0.25$) and four developed hyperthyroidism (expected = 0.87, $P=0.01$).

The numbers of cases in these analyses are small, but nevertheless a significant association with asthma after treatment for hypothyroidism may indicate that treatment of this state increases the likelihood of development of asthma. Conversely, the association between asthma and subsequent hyperthyroidism might suggest that the treatment of the asthma contributed to the development of thyrotoxicosis. We hope to extend the analysis to a large cohort covering a longer time period in the near future.

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¹ Acheson, E D, *Medical Record Linkage*. London, Oxford University Press, 1967.

² Baldwin, J A, *Proceedings of the Royal Society, Series B*, 1973, **184**, 403.

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Oral contraceptives, smoking, and venous thromboembolism

SIR,—It may be premature to conclude from the data presented by Professor D H Lawson and others (17 September, p 729) that differences in fibrinolytic activity between smokers and non-smokers play no part in venous thrombosis in women on oral contraceptives. Their study arose from our suggestion¹ that the increase in "coagulability" in women on oral contraceptives may be compensated for by an increase in fibrinolytic activity in non-smokers but only to a limited extent in smokers.

Our reservations partly arise from the design of their study, which precludes satisfactory consideration of the role of modifying as well as initiating factors; both of these should be considered when assessing a process such as venous thrombosis in which changes occurring after the first development of a thrombus are obviously crucial in terms of clinical outcome. The women in their study had (a) survived and (b) been admitted to hospital and thus cannot be said to represent the full clinical spectrum of thromboembolism. It is possible that those women with only minor episodes and who may not be admitted to hospital are those with particularly active fibrinolytic responses and that this activity is partly due to the fact that they do not smoke. Conversely, women who die before they reach hospital may be those with poor fibrinolytic responses determined in part by the fact that they do smoke. If the balance between coagulation and fibrinolytic activity does not play much part in determining the onset of a venous thrombus it may nevertheless be con-

cerned with the subsequent propagation and eventual size of the clot,² and smoking could then be of considerable relevance. These possibilities can really be satisfactorily explored only by using data on both smoking and oral contraceptive practice in studies which include fatal and non-fatal events. Two prospective studies^{3,4} may further clarify these points. Meanwhile there is already some evidence⁵ that enhancement of fibrinolytic activity prevents recurrent episodes.

Professor Lawson and his colleagues do not say how many of the women with thromboembolism had had previous episodes, nor whether any of the controls may have had such episodes (prior to their admission for some other reason). If, where venous thrombosis is concerned, those with a probable cause for their disease fare better, once that cause is removed, than patients whose condition has no such removable cause⁶ the data presented could underestimate the effect of smoking. Finally, there must be some reservations about a study drawing conclusions on haemostatic mechanisms in which no direct studies of these mechanisms were carried out.

We agree that if there is a balance between coagulation and fibrinolytic activity which is disturbed by smoking it is more likely to be of aetiological significance in myocardial infarction, where the epidemiological picture is one of apparent synergism between the effects of smoking and oral contraceptive usage,⁷ and it was for this reason that we discussed our original findings in terms of myocardial infarction¹ and not venous thromboembolism. It is certainly the case that smoking is not associated with death from venous thrombosis in men,⁸ and other findings⁹ in women indicate the same for non-fatal events. For reasons we have already given, however, it would be valuable to have simultaneous data on fatal and non-fatal events where information on smoking and oral contraceptive use was also available. It would be a pity if the study by Professor Lawson and his colleagues was accepted as general evidence against the possibility of a balance between coagulation and fibrinolysis which could be disturbed by influences such as oral contraceptives and smoking. This hypothesis is supported by our own¹ and other¹⁰ findings and if further substantiated could have implications for preventing thrombosis.

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¹ Meade, T W, et al, *British Journal of Haematology*, 1976, **34**, 353.

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³ Royal College of General Practitioners, *Lancet*, 1977, **2**, 727.

⁴ Vessey, M P, et al, *Lancet*, 1977, **2**, 731.

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⁶ Doll, R, *British Medical Journal*, 1974, **3**, 466.

⁷ Mann, J I, et al, *British Journal of Preventive and Social Medicine*, 1976, **30**, 94.

⁸ Doll, R, and Peto, R, *British Medical Journal*, 1976, **2**, 1525.

⁹ Vessey, M P, and Doll, R, *British Medical Journal*, 1969, **2**, 651.

¹⁰ Kernoff, P B A, and McNichol, G P, *British Medical Bulletin*, 1977, **33**, 239.

"Curing" minor illness in general practice

SIR,—Dr G N Marsh's article (12 November, p 1267) deserves the widest possible attention and acceptance by the medical profession.

He shows quite clearly that refusing to provide household remedies for minor illness leads to a reduction in prescribing and an increase in health education; that is good for patients. He shows that medical time is thereby saved with a reduction in medical irritation; that is good for doctors. He shows that the extra medical time can be devoted to providing more care for more deserving patients; that is good for patients. By implication, some of that extra care might be devoted to items of public policy which are separately remunerated; that would be good for doctors.

With all these benefits for patients and doctors coupled with savings on the national drugs bill it should not be beyond the wit of our medicopoliticians to devise a scheme whereby reduced prescribing costs by the individual doctor were related to increased income for the individual doctor.

I for one shall work to have Dr Marsh's scheme adopted in my practice and I hope many other GPs will follow suit. It should lead to improved quality of care for patients and improved quality of life for doctors.

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SIR,—I was interested to read Dr G N Marsh's article (12 November, p 1267), especially as I heard him speak on the same subject a few months ago. He has at least done something about "curing" minor illness in general practice instead of talking or, more often, complaining about it as the rest of us tend to do.

However, I would take issue with him on one point. I have found that the time for health education is often not during a consultation. The patient (or parent if a child is the patient) is often not receptive if you make him feel that he has been bothering you unnecessarily. He may resort to such comments as, "It's the first time that I have been to the doctor for five years" or "I've never called the doctor out at night before." The attempt at health education therefore may backfire and destroy a budding doctor-patient relationship which may have implications for a future occasion, when the patient's illness is more serious.

I feel the best time to give health education is when the patient is well. This is best done with groups—for example, school classes, young mothers' groups, etc—or even using the mass media. In such a group any feeling of guilt or inadequacy on the part of the patient is easily submerged. This allows the doctor to be more forthcoming and pointed in his comments without giving offence to any individual person. It also allows the patient to be more receptive and therefore the health education to be more effective.

W F WALLACE

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SIR,—Having just completed a morning's work in my surgery, seeing 23 patients and not once prescribing chemotherapy, not to mention cough medicines, tonics, and other simple remedies, I was at once irritated by the article by Dr G N Marsh (12 November, p 1267).

What makes this good doctor think that his own rather parochial experience has to have

universal application? He admits to previous overuse of inappropriate powerful medication, especially antibiotics. Why does he assume that no GP has ever done what he has now decided to do? Why does he state that something in the order of £10m can be saved in one month by GPs if the implementation of his policy is universal when GPs have, in many cases, already implemented it?

I get a little tired of university professors telling us what to do. I get more upset when one of our own colleagues strikes the same note.

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First morning urine culture

SIR,—It is well recognised that in patients with bacteriuria a high fluid intake with frequent micturition may result in a marked reduction in the concentration of organisms recovered on culture of clean-catch midstream specimens of urine.¹ Since patients with recurrent frequency or dysuria or both quickly learn the value of a high fluid intake doubt may develop regarding the validity of quantitative culture carried out on specimens of urine obtained several hours after the patient has woken and has drunk liberally. In such patients culture of urine samples obtained on first waking might demonstrate significant bacteriuria no longer evident later in the day. To investigate this we arranged for symptomatic patients to obtain urine cultures using the Leigh and Williams paper strip technique² on first waking on each of three mornings following a clinic attendance. None of these patients had had antibiotics within the previous three weeks, nor were they given during the study period.

There were 71 patients who were symptomatic but abacteriuric at the time of attending the clinic. Cultures carried out on the three subsequent mornings were all negative in 66 cases. In three of the remaining five the first morning urine cultures were equivocal, but on obtaining further samples bladder bacteriuria was excluded. In only two of the 71 patients was significant bacteriuria found, both in young women known to take large volumes of fluid when symptomatic.

There were a further nine symptomatic patients in whom culture of a clean-catch midstream specimen taken in the clinic yielded equivocal results (more than 10^3 but less than 10^5 enterobacteria/ml or a mixed growth). First morning urine culture in four showed significant bacteriuria, but the remaining five patients yielded negative urine cultures.

These findings confirm that spurious false-negative urine cultures can occasionally be obtained in symptomatic patients, but this is uncommon. We conclude that early morning urine culture has a limited cost-effectiveness when culture of midstream specimens of urine collected in the clinic is unequivocally negative. It is, however, of value when the initial urine culture is equivocal.

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¹ Cattell, W R, et al, in *Urinary Tract Infection*, ed F O'Grady and W Brumfit, London, Oxford University Press, 1968.

² Leigh, D A, and Williams, J D, *Journal of Clinical Pathology*, 1964, **17**, 498.