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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- ¹ Wood, J W, et al, *American Journal of Public Health*, 1967, **57**, 1374.
- 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.

³ Baldwin, J A, Simmonds, H, and Fedrick, J, submitted for publication.

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-