This deplorable state of affairs, which is a cause of much wasted time and many unnecessary x-ray examinations, can be directly related to the very severe reduction in the amount of time which our medical schools allocate to the teaching and study of anatomy. In practical terms, it means that students become involved with contemplation of the abnormal before they have acquired a sound knowledge of the normal.

Perhaps the tide is now beginning to turn, for, during the past year or so, there have been at least three publications affirming the importance of teaching radiological anatomy from the outset of the preclinical course. Can this really be just a coincidence?

KENNETH SWINBURNE

Wharfedale General Hospital,
Otley, W Yorks LS21 2LY

¹ Keats, T E, *Atlas of Normal Roentgen Variants*. Chicago, Year Book Medical Publishers, 1973.

² Swinburne, K, *Lancet*, 1978, 1, 433.

³ Goldberg, B, *British Medical Journal*, 1978, 1, 1017.

⁴ Reidy, J, *et al*, *Clinical Radiology*, 1978, 29, 591.

SIR,—The 28 specialties listed in the survey by Professor V Wright and others (24 March, p 805) would obviously relate very much to what medical practitioners had themselves learned in medical school. To us it was most striking, but not surprising, that no mention was made of nutrition at all, except to suggest metaphorically that medical students know what they enjoy academically, but not necessarily what is good for them: "The student knows what is palatable, but not always what is nutritious." Ironically, this is literally the situation in practice for those graduating with the curricular emphases suggested in this present study.

The absurd paradox of not including such a universal subject as nutrition in the curriculum is highlighted most clearly by current correspondence in the same issue (p 818) concerning "Prescription for a better British diet." How, one may ask, can the medical profession make the slightest informed input into these decisions if they have no knowledge because of an almost complete lacuna in their training? More importantly still, how can they advise their patients appropriately?

D B JELLIFFE

E F PATRICE JELLIFFE

Division of Population, Family, and
International Health,
University School of Public Health,
Los Angeles,
California 90024, USA

Breast cancer in young women

SIR,—I was interested in your leading article on age and death in breast cancer (27 January, p 211). I would like to present further data supporting the thesis that breast cancer in young women is less aggressive than is currently believed.

Twenty-seven women (27-45 years old) were treated by modified radical mastectomy and those with large tumours and extra-mammary spread also received radiotherapy with or without chemotherapy and oophorectomy. The lymph nodes, all containing macrometastases, were not grouped by levels, the value of which is now in doubt.¹ Of these 27 patients, 20 had moderately well-differentiated carcinomas, two infiltrating comedocarcinomas, two invasive lobular carcinomas, and one a medullary carcinoma.

I have no similar data on my older patients

and for comparison I have used figures given in the literature for short-term treatment failure and five-year survival rate. It is clear that, despite adverse prognostic factors,¹⁻³ these patients have done surprisingly well. The quoted incidence rate of short-term treatment failure at 12, 18, and 24 months is 12.2%, 21.2%, and 41.2% respectively²; of my 27 patients, only one died 11 months after surgery. The overall five-year survival rate ranges from 46% to 65%³; only one of my 15 patients followed up for over five years has so far died of breast cancer (93.3% five-year survival rate).

These figures, though based on a small series, justify optimism and I feel that physicians should judiciously inform the public of this "good news." It may help to break through the fear barrier and lead young women to examine their breasts more regularly and report early any abnormal findings. Earlier diagnosis can only improve their chances of survival.

H HONORÉ

Memorial University of Newfoundland,
Grace General Hospital,
St John's,
Newfoundland, Canada

¹ Fisher, E R, *et al*, *Cancer*, 1978, 42, 2032.

² Fisher, E R, Gregorio, R M, and Fisher, B, *Cancer*, 1975, 36, 1.

³ Diekamp, U, Bitran, J, and Ferguson, D J, *Journal of Reproductive Medicine*, 1976, 5, 255.

Hypnosis

SIR,—I was most interested in your correspondents' reactions to my submission that the hypnosis lobby has not yet conclusively proved its case.

Regarding Dr H K Gooding's comments (14 April, p 1017), of course Dr D Waxman (19 August, 1978, p 571) uses the method responsibly; but will all the others attracted to this still esoteric therapeutic technique? Again, Dr Gooding's rapport with his smoking patient is obviously of excellent placebo value at least. Is there any more to it? He himself is, of course, not obliged to conduct controlled trials with individual patients (though the simple mirror-image trial, using the patient as his own control, is always open to him); but he should always remember that spectacular "results" may not be due to the treatment given.

Dr Gooding refers to the studies of Dr G P Maher-Loughnan as proving the efficacy of the hypnotic approach. However, the latter's first paper¹ describing a controlled trial of hypnosis in the symptomatic treatment of asthma admits that his finding of its value "is based purely on a subjective assessment, and there remain many questions to be solved concerning satisfactory objective methods of assessment." There do indeed. When objective methods—that is, lung function tests—were included in a later, much larger, controlled trial,² the results of these were the same in the experimental and control groups, suggesting that hypnosis does not exert a physiological effect, that any improvement is a subjective, placebo one. Even with regard to the various subjective assessments used, while independent clinical assessors did find the asthma much better in more of the hypnosis group (59% as against 43% in the controls, not a huge advantage), there was no difference (for males) in the scores for wheezing or bronchodilator use. Females differed, for some obscure reason. If, then, this is the definitive trial of the value

of hypnosis in a classically psychosomatic (so-called) condition such as asthma, to my mind the point is not proved beyond doubt. Moreover, the very first controlled study³ showed that hypnotic suggestion failed to produce any improvement in 25 asthmatic children.

Dr E W Rees (14 April, p 1018) in his first paragraph appears to discount the value of scientific evidence, yet goes on to emphasise the value of the "evidence" in favour of hypnosis. Again, he (and also Dr Waxman—19 May, p 1354) seems to link hypnosis with psychotherapy. This is surely unfortunate in view of the grave doubts of many about the scientific status of psychotherapy, not to mention its potential negative effects.^{4 5}

The comments of Dr P G F Nixon and Dr G D K Flint (5 May, p 1212) are helpful and interesting, but miss my main points. Of course, any doctor or dentist may practise hypnosis if he finds it useful, just as he may employ acupuncture, transcendental meditation, or any other paramedical technique; but to suggest that resources should be poured into their widespread application before their value has been firmly ascertained is, in my opinion, unjustified. The controlled trials that have been done do not show a convincing superiority for hypnosis over waking suggestion or other simple placebo techniques such as breathing exercises.

Finally, with regard to the importance of suggestion in this area, may I refer your correspondents to the current analogous controversy in your columns about the value of the "fringe" technique of homoeopathy, in particular Dr R S Walker's opinion (28 April, p 1147) that its practitioners appear to have surprisingly little insight into the powers of suggestion?

H G KINNELL

University Department of Psychological
Medicine,
Newcastle General Hospital,
Newcastle upon Tyne NE4 6BE

¹ Maher-Loughnan, G P, *et al*, *British Medical Journal*, 1962, 2, 371.

² British Tuberculosis Association, *British Medical Journal*, 1968, 4, 71.

³ Burns, C L C, and Morrison Smith, J, *British Journal of Diseases of the Chest*, 1960, 54, 78.

⁴ Rachman, S, *The Effects of Psychotherapy*. Oxford, Pergamon Press, 1971.

⁵ Kinnell, H G, *Lancet*, 1978, 1, 882.

Oestrogen-induced hypocalcaemia in hypoparathyroidism

SIR,—I read with great interest the fascinating observation of a case of hypoparathyroidism whose symptoms and biological features were aggravated by the administration of oestrogens, as reported by Drs D Verbeelen and M Fuss (24 February, p 522).

This is reminiscent of the observations reported more than three decades ago by Klotz and Barbier,¹ who demonstrated this phenomenon up to four times in one of their hypoparathyroid patients. One decade therefore before the introduction of sodium EDTA as a calcium-lowering agent a test of sudden hypocalcaemia was devised, using oestradiol benzoate 20 mg intramuscularly or subcutaneously^{2 3} to evaluate parathyroid reserve. The degree and the duration of the hypocalcaemia were proportional to the severity of the hypoparathyroid state. Experimental support for this oestrogen-induced hypocalcaemia had been provided by the work performed in Liège by Mathieu and coworkers⁴ in parathyroidectomised dogs.

These observations do not detract from the interesting points made by Verbeelen and Fuss with more modern methods, especially the induction of hypercalcaemia on oestrogen withdrawal, as well as of hypocalcaemia on oestrogen administration. They merely indicate that the latter phenomenon is a perennial one.

C NAGANT DE DEUXCHAISNES

Arthritis Unit, Department of Medicine,
Louvain University in Brussels,
St-Luc University Hospital,
B-1200 Brussels

¹ Klotz, H P, and Barbier, P, *Annales de Biologie Clinique*, 1947, 1, 13.

² Klotz, H P, and Klotz-Valensi, R, *Bulletins et Mémoires de la Société Médicale des Hôpitaux de Paris*, 1947, 28-9, 857.

³ Justin-Besançon, L, et al, *Bulletins et Mémoires de la Société Médicale des Hôpitaux de Paris*, 1949, 1112, 451.

⁴ Mathieu, F, *Archives Internationales de Physiologie*, 1934, 38, 365.

A Copper 7 IUCD with no copper

SIR,—I was interested to read Drs Elspeth M Williamson and R Jane Berry's letter "Defects in copper winding of intrauterine devices" (5 May, p 1215) as the previous day I had removed one of these devices which had no copper on it at all. There was a small black speck attached to the lower end of the stem where the copper winding usually begins and a few more in the mucus surrounding it, but these were washed away by the nurse and are not available for analysis. This IUCD had been fitted by a colleague in the same clinic on 24 April 1974 and the patient, who had been abroad, had not returned for examination since six weeks after the device was fitted. She had no complaints but thought that it was time that she had a check-up. She was refitted with a new Copper 7.

It seems to me that this loss of copper was not due to any defect in winding but that it had been dissolved away during the length of time that it had remained in the uterus. The Gravigard is a small plastic device with 32 cm of copper wire 0.2 mm in diameter wound round its stem giving a surface area of 200 sq mm, its original weight being 95 mg.¹ This makes it 7-10 times as effective as a similar plastic device without copper.^{2,3} The increased contraceptive effect appears to be due to the presence of dissolved copper in the uterus. The initial rate of copper elution is 60 µg/day, which decreases to 12 µg by the end of the first year and to less than 10 µg daily during the second and third year.⁴ The amount of copper diffusion may be affected by the deposit of calcium on the device.⁵ Contraceptive effectiveness may therefore be diminished by loss of copper or by its being covered with calcium. Even so, the Copper 7 appears to remain effective for longer than the two years recommended by the manufacturer as the pregnancy rates continue to fall from 1.6 per 100 women in the first year of use to 0.8 in the fourth year (Searle Laboratories, personal communication); Zipper *et al*¹ found a similar fall from 2.3 to 0.9. Rates for expulsion and removal of the device for bleeding or pain were diminished after the first year.

Although changing of the Copper 7 is recommended after two years, it has been my experience that many of the problems following the first fitting recur after reinsertion of a device and it does not seem good clinical practice to remove and reinsert an IUCD as a routine when a patient has no complaints. However, in view of the findings of Sir

Williamson and Berry and my experience it would seem prudent to renew Copper 7s after about three years of use until more is known about the factors governing the length of their effectiveness in individual patients.

MARY POLLOCK

Family Planning Clinic,
Raymede Health Centre,
London W10

¹ Zipper, J, *et al*, *Contraception*, 1976, 13, 8.

² Medel, M, *et al*, in *Proceedings of Family Planning Research Conference: A Multidisciplinary Approach*, ed A Goldsmith and R Snowdon, p 49. Amsterdam, Excerpta Medica, International Congress Series No 260, 1972.

³ Tauber, P F, *et al*, *IRCS Medical Science*, 1978, 6, 527.

⁴ Stewart, W C, *et al*, in *Analysis of Intrauterine Contraception*, ed F Hefnawi and S J Sigal, p 152. Amsterdam, North Holland, 1975.

⁵ Gosden, C, Ross, A, and Loudon, N, *British Medical Journal*, 1977, 1, 202.

Diagnostic sand

SIR,—The detailed study made of the accuracy of the diagnostic content of Hospital Activity Analysis by Drs A M George and Dr G B Maddocks (19 May, p 1322) confirms what many of us have thought for years—that is, that HAA is an impressive edifice built on diagnostic sand.

The fault lies with us, however, for the responsibility for accurate disease indexing must be a medical one. I do this personally on all discharges, but feel that perhaps the registrar is the doctor who might be held accountable for this exercise—surely this sort of responsibility is the origin of the title "registrar."

JOHN ANDERSON
Chairman,
Medical Records Advisory
Group

Taunton and Somerset Hospital,
Taunton, Somerset TA1 5DA

Airways insufficiency and pituitary failure

SIR,—The letter by Dr P d'A Semple and others, "Pituitary suppression in chronic airways disease?" (19 May, p 1356), contains the interesting suggestion that raised intracranial pressure may account for their findings of depressed testosterone and raised prolactin levels. Dr R A Stockley and I have looked at a series of 16 patients with severe chronic bronchitis.¹

Eight patients, whose pituitary, thyroid, and adrenal hormone levels were measured either at rest, after exercise, or during sleep, all showed apparent abnormalities in various parameters. Two of these patients, together with a further eight patients also with severe bronchitis, had a combined pituitary function test.² We found that only one patient in this second group had an inadequate response. Although hypothalamohypophyseal damage does occur in these patients, we felt it unethical to measure cerebrospinal fluid pressure levels. Without these measurements it would seem premature to suggest that intracranial hypertension is the cause. Our data suggest that static testing is inadequate in assessing endocrine activity in patients with respiratory insufficiency and that dynamic testing is essential. Dr Semple's tests might have overestimated the problem in sick patients if sex-hormone-binding globulin has not been estimated. Underventilation of short duration did not appear to be a cause of pituitary dysfunction

when 50 patients maintained on artificial ventilation were assessed.³

Corroborative evidence that cerebrospinal fluid pressures are more important than generally recognised is provided by separate studies, which show that hypothalamohypophyseal damage may be associated with what is called "benign intracranial hypertension."⁴ Undoubtedly intracranial hypertension does occur with carbon dioxide retention, and papilloedema is well recognised,⁵ but its exact relationship to the endocrine disturbances remains hypothetical.

STEPHEN G BARBER

General Hospital,
Birmingham B4 6NH

¹ Barber, S G, and Stockley, R A, submitted for publication.

² Harsoulis, P, *et al*, *British Medical Journal*, 1973, 4, 326.

³ Barber, S G, *Acta endocrinologica*, 1979, 90, 211.

⁴ Barber, S G, and Garvan, N, submitted for publication.

⁵ Crofton, J, and Douglas, A, *Respiratory Diseases*, 2nd edn, p 371. Oxford, Blackwell Scientific Publications, 1975.

Contaminated hospital water supplies

SIR,—The reports (3 February, p 350) that hospital water supplies have been shown to be contaminated by a variety of bacteria, larvae, and other forms of microbial life should awaken control of infection teams to the possibility that the domestic water supply in hospitals may be a source of potentially pathogenic micro-organisms.

Examination of the water supply of our hospital was undertaken prior to the opening of a new sterile production unit in the pharmacy department, and revealed intermittent contamination by small or large numbers of Gram-negative bacilli, often between 10³ and 10⁵ colony forming units per ml. The predominant organisms were two types of *Pseudomonas* species, one of which was identified by the computer trials laboratory at the National Collection of Type Cultures as an atypical strain of *Pseudomonas pickettii*, while the other strain resisted precise identification, although it was confirmed as a pseudomonad able to grow at 42°C. Growth at 37°C required 48 hours' incubation.

Not only were the organisms present in the cold water, but the hot water system was also extensively colonised. At this time the hot water was maintained at only 42-3°C, owing to misinterpretation of the DHSS guidance¹ recommending that domestic hot water in most patient areas of general hospitals should be supplied at 50-60°C, whereas in the areas for geriatric, mentally ill, mentally handicapped, and paediatric patients it should be at only 43°C. This had been implemented some six months previously.

The source of the organisms in the water in our hospital can only be postulated. The local water authority produced figures to show that when we first detected the problem the mains supply to the hospital was wholesome, containing less than 10 organisms per ml after 24 hours' incubation at 37°C, and no coliforms or *Escherichia coli*. However, these incubation conditions would not have detected our strains, which required 48 hours for growth on nutrient agar at 37°C. Small numbers of pseudomonads may therefore have reached the hospital through the mains water, and have colonised the hot water system because the temperature was insufficient to kill bacteria, especially if thermophilic. We also postulated that the cold water may have been recontaminated from the hot water through mixer taps, thus continuing the cycle of events, although contamination may equally well have occurred in the opposite direction. Once the hot water temperature had been raised to 60°C, colonisation with *Pseudomonas* species disappeared within a matter of hours from cold and hot water systems alike. Samples examined