these were mild, in eight moderate, and in three severe. Two patients with severe symptoms were pale and sweating, with almost unrecordable diastolic pressures, and the clinical picture suggested peripheral blood pooling with low cardiac output. The mean maximum erect pressure reduction was 63/48 mm Hg (from 165/112 mm Hg) at a mean of 90 min after the dose. Mean pulse rate rose 10/min. Postural hypotension lasted up to four hours in 11 patients and up to seven hours in three. Two severe reactors had had no previous drug therapy. Subsequently 22 additional patients were studied in the same manner with a single dose of 0.5 mg, 11 of them in a double-blind within-patient crossover study with placebo. There were no significant postural symptoms in this group and there was a mean maximum fall in erect diastolic pressure of approximately 25 mm Hg.

These observations indeed confirm that initial symptoms of postural hypotension after the first dose of prazosin are dose-dependent and that the initial dose should not exceed one 0.5-mg tablet. Many of these patients were subsequently treated with prazosin. Doses of thiazides and beta-blocking agents were not increased and in some cases were reduced. Our preliminary observations suggest that the therapeutic response may be better and the dose of prazosin required may be lower in those patients in whom the hypotensive response to the first dose is abrupt. We believe that a significant initial postural fall in blood pressure may be indicative of a subsequent satisfactory response to this very useful drug.

A S TURNER

Cardiology Department,
Napier Hospital,
Napier, New Zealand

Possible association of Madelung's deformity with Huntington's chorea

SIR,—Recently correspondents have discussed in your columns (3 July, p 46; 14 August, p 420) the desirability of publicity directed at the lay public concerning Huntington's chorea. As a direct result of this publicity a member of a Huntington's chorea family has approached me for assistance in clarifying their family history. A diagnosis of Parkinson's disease had been made in a member of the family who would have had to have Huntington's chorea to substantiate the subsequent diagnoses. On studying this family it became apparent that a wrist deformity (possibly Madelung's deformity) was also inherited as a dominant condition. It also appeared that the wrist deformity was inherited only by those members who went on to develop Huntington's chorea.

Although the members of this family have not been seen in person, considerable correspondence has taken place with them and their medical practitioners and it was felt that the family needed to be investigated more thoroughly as there was a possibility of a linkage between the wrist deformity and Huntington's chorea. To this end a request was inserted in the latest newsletter of the Association to Combat Huntington's chorea for news of any people in choreic families who also had a wrist deformity. To date there has been one reply from a young woman in such a family who states that she has Madelung's deformity which was successfully operated upon some years ago. Although Madelung's

deformity is very unlikely as a genetic link in all cases of Huntington's chorea, there appears to be a strong possibility of it being linked in the family originally investigated. The next step must therefore be to trace the family of the young woman with Madelung's deformity to see if she is related to the original family, and a continuing search must be made for other patients in whom these dominant diseases coexist. Madelung's deformity sometimes shows up only radiographically, so it is hoped that x-ray films of the wrists of all members of these families will be undertaken.

In the meantime we must again look at the question of ethics with regard to counselling such a family. Should this link prove to be predictive, could it in all fairness be used when we have nothing in terms of treatment to offer the unfortunate carriers of the gene? This was the problem the levodopa provocation test produced some years ago, as discussed in the *BMJ*.¹ The majority solved this problem by not using levodopa. An obvious wrist deformity is much less easy to ignore.

ADRIAN CARO

Huntington's Chorea Research
Project (East Anglia),
Dereham Hospital,
East Dereham,
Norfolk

¹ *British Medical Journal*, 1972, 3, 540.

Immunofluorescent sperm antibodies in virgins

SIR,—Your recent leading article on antibodies to spermatozoa (2 October, p 774) appeared to recommend the use of immunofluorescent techniques and staining patterns to distinguish between normal and pathological autoantibodies.

From previous studies on infertile patients¹⁻³ I cannot share this enthusiasm, as a large number of positives were not only found in infertile couples but also in control groups with no particular pattern or titre appearing significant. There are, however, potential errors in the use of serum from pregnant or previously pregnant patients as control. To allay any such criticisms serum from virgins was tested for sperm antibodies by the indirect immunofluorescent technique on the rationale that females who have never had contact with spermatozoa should not have circulating sperm antibodies.

Serum from 66 informed virgin volunteers aged between 18 and 45, obtained from random sources, was tested for the presence of antibodies against sperm at a titre of 1 in 25 by the indirect immunofluorescent technique, with and without methanol fixation.¹ From table I it can be seen that positives were

TABLE I—Immunofluorescent sperm antibodies: virgin serum

Total No	No positive
66	20 (30%)

TABLE II—Positive immunofluorescence patterns: virgin serum

Equatorial	Tail	Postnuclear cap	Mid-piece	Achrosomal	More than one pattern
21%	29%	8%	0	41.6%	20%

obtained from 20 (30%) of these virgins, which surely is indictment enough of any method. From table II it would also appear that examination of the staining pattern is of little help, for all patterns were found to be present, except mid-piece, with achrosomal the most common. Indeed, more than one pattern was present in 20%.

This high number of positives in a group of subjects who should be negative reflects the intrinsic weakness of the method. False-positives may occur due to cross-reaction with bacterial antibodies,⁴ non-specific binding of immunoglobulins,⁵ or involvement of antibodies from beneath the cell membrane of the sperm.⁶

It would therefore appear that the indirect immunofluorescent test is of little value as a measure of sperm antibody presence, and, although it may be a more acceptable immunological test than some of the others available, the question of the significance of sperm antibodies as a cause of infertility cannot be answered by its use.

R F HARRISON

Department of Obstetrics
and Gynaecology,
Trinity College at the Rotunda
Hospital,
Dublin

¹ Wall, J R, et al, *American Journal of Obstetrics and Gynecology*, 1975, 121, 198.

² Wall, J, et al, *Fertility and Sterility*, 1975, 26, 1035.

³ Harrison, R F, et al, *Reproduction*, 1976, 2, 338.

⁴ Hansen, K B, and Hjort, T, *Clinical and Experimental Immunology*, 1971, 8, 9.

⁵ Petrunia, D, Taylor, P, and Watson, J, *Fertility and Sterility*, 1976, 27, 655.

⁶ Hansen, K B, *Acta Obstetrica et Gynecologica Scandinavica*, 1974, 53, 69.

Teaching of anatomy

SIR,—Having read with keen interest your leading article on this subject (11 September, p 603), I wonder whether I could give my views as an anatomist teaching in Germany.

I have just returned from a most stimulating meeting on "computerised axial tomography" in London (11-15 October). This meeting and the enormous progress made by British inventiveness offer a very stimulating challenge to the anatomist who teaches clinical or topographical anatomy. Axial tomography has enlarged the scope of anatomy teaching enormously, a subject which can only be taught in its clinical implications by medically trained lecturers. Clinical students, clinicians, and radiologists have now to know more anatomy, not less, and have to adapt their teaching to the new demands of axial tomography, demanding more anatomy from a clinical diagnostic point of view. But your leading article clearly points out that anatomy is taught on a very much reduced scale in Britain. In Germany standards have been very significantly and drastically cut—for example, dissection of the body has been reduced to one winter term only. Altogether the preclinical curriculum has been reduced from five terms to four—two summer terms and two winter terms. Two terms account for about 5½ months of one year. While term work has thus been reduced, new subjects