627), all earlier workers failed to demonstrate specific antibodies in the serum of patients.[1-7] Even Hermans' group,[8 9] as you mention, failed to detect antibodies to cartilage or chondrocytes using very elegant methods, but the increased lymphocyte transformation detected by them occurred in all of three cases of relapsing polychondritis and also in nine out of 12 cases of rheumatoid arthritis and in one of two cases of gouty arthritis among the control subjects, in the presence of the same antigens. Increased lymphocyte transformation by itself is not accepted as an indisputable index of cell-mediated immunity because a humoral or a cell-mediated mechanism, or both in combination, or a "non-specific" mitogenic effect could each transform lymphocytes.[10] The possibility of the occurrence of a "non-specific" mitogen in the antigenic material used was not excluded. Further, as the increased lymphocyte transformation occurred in many cases of rheumatoid arthritis and one of gouty arthritis, the operation of a factor common to all these arthritides (including relapsing polychondritis) seems likely.

Hughes *et al.*[11] have recently demonstrated, by the immunofluorescent technique, positive human fetal cartilage staining in two out of three cases of relapsing polychondritis and in all of 12 cases of rheumatoid arthritis but no staining in 32 control subjects. Again, like Herman's findings, their results seem to indicate the occurrence of an antibody in the serum of patients with these chronic arthritides. We[12] have shown the presence of cell-mediated immunity to human laryngeal cartilage proteoglycan (kindly supplied by Dr. Helen Muir of the Kennedy Institute) by two in vitro methods—macrophage migration inhibition[13] and lymphocyte transformation—in two patients with relapsing polychondritis, whereas nine control subjects suffering from a variety of arthritides gave negative results. Further, we were unable to show the presence of humoral antibodies to cartilage in the serum of these two patients by immunofluorescent staining (kindly done by Dr. G. Loewi, Dr. G. Johnson, and Mr. D. Kingston of this unit separately) or to proteoglycan by gel immunodiffusion. Subsequently we were in a position to study three other cases of relapsing polychondritis (to be published). The following results were obtained in them when the same four tests were applied. The first case showed no cell-mediated immunity and in the two humoral tests showed positive immunofluorescent staining of fetal cartilage but negative immunodiffusion with proteoglycan. The second case showed positive cell-mediated immunity and weakly positive immunofluorescent staining of cartilage but negative immunodiffusion with proteoglycan. The third case showed positive cell-mediated immunity but negative humoral tests like the two reported cases.[12] These findings are difficult to interpret. We feel that humoral immunity could be an early manifestation of the disease, while cell-mediated immunity could be a late manifestation. The interesting feature is the development of cell-mediated immunity against a normal biochemical constituent of the tissues affected in the disease process.

Histochemical studies indicate that the chrondromucoprotein of the cartilage ground substance is affected in the pathological process of the disease. There is some evidence[14 15] to suggest that the arterial walls may contain a similar substance and this could perhaps explain the involvement of the arteries in the disease. One of the two patients under our care referred to above[12] (in fact, the first) presented as a case of Takayasu's syndrome, and over the years she developed both arthritis and finally chondritis. She showed a marked degree of cell-mediated immunity to human laryngeal cartilage proteoglycan. Another patient referred for our opinion had angiographically established Takayasu's disease but had no evidence of arthritis or chondritis clinically or radiologically. She showed a mild degree of cell-mediated immunity to the same proteoglycan antigen. It could well be that the arteritis of Takayasu's type and polychondritis of relapsing type are variants of the same disease or disease process.—We are, etc.,

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The Blind in Hospital

SIR,—Some years ago I spent three months in a surgical ward. During that time the sister spoke to me exactly once. Doubtless she was often in the ward, but I never knew because I am totally blind. It would have been a help because the nurses were continually changing. Over a period of two or three weeks I was asking nurses to ask Dr. X. to see me, without success. Eventually I phoned him from my bed and asked him if he had had a holiday. He was furious and said that he had been in the ward every day. The staff often forget that you are blind and completely fail to communicate. The situation was desperate for me and I would prefer to die in the gutter rather than go back.

If I am blind I never know who you are and rarely if you are there at all, because of background noise. People think I do not want to speak because I cannot speak first. Would it not be possible to hang up a little label saying, perhaps, "Blind but not deaf," or "Tell me you are here"?—I am, etc.,

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Clinical Experience with the Dalkon Shield

SIR,—We were interested to read Dr. J. S. Templeton's letter (8 September, p. 542) concerning our preliminary report on our clinical experience with the Dalkon Shield (21 July, p. 143) and would like to comment on some of the points that he has raised.

One of the prime objectives of our paper was to point out that in our hands the Dalkon Shield results were not as good as those reported by Davis[1] and his colleagues who had developed the intrauterine device (I.U.D.). We therefore presented our data in a similar way to that which they had done, using the Pearl index and the life table method.[2] It is possible that their results might be related to the use of a spermicide, which Davis recommends in the first few months of use. Furthermore, in the multicentre analysis reported by the family planning unit at the University of Exeter, there is no information on spermicide usage either.[3] We quoted the use of spermicide in 5% of our cases. Variation in spermicide usage may be one of the factors related to the widely differing pregnancy rate in these 10-12 clinics, of which, incidentally, ours is one (the pregnancy rate in these clinics varies from 0·8 to 8·4%). We, too, have little doubt that valuable complementary data are derived from studies such as the multicentre trial quoted as well as experience in an individual clinic.

We have used the accepted definition of parity as a pregnancy which has gone beyond 28 weeks. Therefore our nulliparous patients were those who had been pregnant and had had a pregnancy terminated; thus they were able to accommodate the larger Dalkon Shield without any difficulty. It was in the two patients in whom the uterine cavity was small that we used the smaller Dalkon Shield.

As far as the training of doctors in the insertion of I.U.D.s is concerned, we accept that this may well contribute to a higher pregnancy rate, bearing in mind that if an I.U.D. is to be inserted in large numbers, it is to be expected that the skill of individual operators will vary.

We have since had our data analysed according to the currently recommended cumulative life table method[4] and our pregnancy rate findings with this method of analysis were not dissimilar from those reported.—We are, etc.,

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One Thousand Vasectomies

SIR,—With reference to the report by the staff of the Margaret Pyke Centre (27 October, p. 216) I was interested to note that it was considered unreasonable to insist on sperm-free specimens before taking the responsibility of pronouncing sterility.

I have performed some 5,000 vasectomies