certificate and accepts a fee from his N.H.S. patient may also be challenged.¹

Lastly, I fail to understand Dr. Hughes's analogy between abortion for inconvenient pregnancies and operations for acute appendicitis. Even a psychiatrist like myself knows that the latter is a serious risk to life or health while the former very, very rarely causes material disability. But Dr. Hughes declares his attitude to abortion in his last words: they should be dealt with "... without question." One may ask: "Where do we go from here?"—but more pertinently: "How did we get here?"—I am, etc.,

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REFERENCE

- ¹ *Sunday Mirror*, 16 February 1969.

SIR,—I think we must all be a little tired of the diatribes from some members of the medical profession in the press and on television against the Abortion Act. There are quite a number who find it is satisfactory and do not need to strike the attitudes of the "unco' guid." We see these patients at clinics, and we take them into National Health Service hospitals, either maternity units or gynaecological units, and whenever possible do the operation personally. There is no question of fees being paid. I am a little amazed at the howls of protest that it is interfering with the ordinary work of units and outpatient clinics. It is unlikely that Ayrshire has better morals than any other county in Great Britain. The patients in the district can be dealt with expeditiously, being admitted and discharged within two to three days. I have not, as yet, found that it is making my waiting-list longer or interfering with the intake of patients into the maternity units.

All told, the number that have been performed during the past 12 months since the Act would be approximately 120. This number has been done by three obstetricians and gynaecologists, who are very grateful for the co-operation from the practitioners in the district, who take great care to send only those patients whom they consider justify therapeutic abortion. With this co-operation there is no time wasted, and it does not take a genius to weed out the odd one in which this is not a justifiable procedure. Naturally, if some colleagues do not co-operate, then more cases have to be done by the others. The only way I have found that a waiting-list in gynaecology increases above the average in any particular district is when the hospital beds are not being fully utilized.—I am, etc.,

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Premenarchal Pregnancy

SIR,—Mr. S. Bender (22 March, p. 760) confirms once more the tendency for these patients to be free from obstetrical anxieties.

In a case described by James and Davies¹ the patient was aged 11 years and 3 months at the time of referral. She was then about

24 weeks pregnant, and the pregnancy started when she was approximately 10 years and 9 months. Her pregnancy was normal, labour ended in a spontaneous delivery, and the puerperium was uneventful. Where birth registration is compulsory this patient must be among the youngest.

Greulich and Thomas² maintained that there is a "prepuberal" spurt of growth in the female pelvis. Harris³ in a study of 500 cases between 12 and 16 years arrives at the conclusion that "the white girl of 13 to 16 has as large a pelvis as that of her older sister." Fairfield⁴ in her survey states that advance psychological effects have been exaggerated. Mr. Bender briefly mentions the medico-legal aspect of this kind of problem. Douglas Miller⁵ was one of the first to discuss the importance of this. The James and Davies case record contains a medico-legal commentary written by Mr. H. Edmund Davies, Q.C. (now Lord Justice Edmund Davies). In this commentary there is a carefully reasoned assessment of the legal position which might also apply to Mr. Bender's case.—I am, etc.,

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- ² Greulich, W. W., and Thoms, H., *Yale Journal of Biology and Medicine*, 1944, **17**, 91.
- ³ Harris, J. W., *Johns Hopkins Hospital Bulletin*, 1922, **33**, 12.
- ⁴ Fairfield, L., *Lancet*, 1940, **2**, 61.
- ⁵ Miller, D., *Transactions of the Edinburgh Obstetrical Society*, 1931, **52**, 161.

Specific Antibodies and Renal Transplantation

SIR,—It is always a stimulating pleasure to study the views of Mr. P. J. Morris and his colleagues (22 March, p. 758). If I may say so, the central theme of their article—specific antibodies appearing in the serum before and after removing a rejected kidney allograft—is of some general theoretical interest in transplantation immunology. If it is alleged that antibody detectable in the serum after a rejected kidney has been removed was in actual fact in the serum all along, but was undetectable because it was being constantly absorbed by the kidney, one can at least set up experiments to prove or disprove this point. One can remove the organ, as we did,¹ before rejection takes place, and then follow the appearance of antibody in the serum.

Milgrom *et al.*² reported that antibodies could not be demonstrated in the sera of recipients until after the removal of the kidneys, and suggested that this was because the antibodies were mopped up by the kidneys. I,³ too, have been guilty of alleging that a supersaturation of antibodies was mopped up by the renal vasculature in an effort to explain the sudden onset of afferent vascular spasm—a phenomenon which at long last was confirmed⁴ and its importance in rejection conceded. I at least admitted that I was borrowing the mopping up idea from my predecessors in experimental nephritis. I can now attempt to explain afferent vascular spasm as the result of increased venular resistance, but I withdrew my previous speculation many years ago because I came to realize that I was discussing only "alleged" antibodies. Milgrom *et al.*² tested for renal cell agglutinins, whereas Mr. P. J. Morris and his colleagues have used the lymphocytotoxicity test. I understand that

these tests detect different things. Manzler⁵ could detect serum leucocytotoxins during the height of rejection in about 30% of humans, but Yamada and Kay⁶ found them in all dogs rejecting kidneys. So we are not even discussing the same antibody part of the time, and when we are the reports are quite contradictory. Recently the Kyoto group⁷ have even failed to confirm the report of Milgrom *et al.*² that agglutinins rise after the removal of the first rejected kidney, and this would fit more accurately the natural history of second-set kidneys.

How easy it is to toss around! To prove their role in rejection one must demonstrate their site of union and the cytopathological effects, and be quite guarded about the results of elution tests. After the very balanced and mature analysis of Lindquist *et al.*,⁸ with which I am in general sympathy, one has to mind one's step. It is surely time to pause and learn not to suspect an antibody lurking under every tubule cell, and to refrain from attributing miraculous strike capability powers to the humble interstitial lymphocyte. The second-set reaction,⁹ under strict conditions of assessment, can reasonably be attributed to the effects of high affinity antibodies because one can demonstrate the site of union and the cytopathological effects.

The literature dealing with the period after transplantation when sensitization occurs is quite chaotic. Some authors claim that even one hour is sufficient. I am relieved that Mr. P. J. Morris and his colleagues consider that some considerable time must elapse before sensitization occurs. My own experimental evidence⁹ indicated that sensitization occurs after rejection, and this could account for the appearance of antibodies in the serum after rejection and removal of the transplanted kidney and also for the morphological differences between first and second set kidneys. On the whole I am in agreement with those who view rejection as due to a humoral mechanism, but I do not see that a sufficiently strong case has been made out for antibody involvement in first-set rejection. Even the evidence of glomerular damage in long-surviving immunosuppressed human kidneys is becoming more and more difficult to assess confidently.

I see no experimental evidence that should make immunologists change their conventional views¹⁰ about an antibody response being a blunderbuss of antibodies of all shapes and sizes, only some of which exactly fit the specific antigen. Thus, most antibody reactions after the early phase must be poly-specific and become more so as time goes by. This is one possible anatomical explanation of cross-sensitization. There are reports¹¹ in the literature which indicate that even when leucoagglutinins and leucocytotoxins are demonstrable in the serum a second-set reaction need not ensue; rejection can also occur without this type of antibody appearing in the serum. The failure to cause damage to the contralateral kidney of the donor by the passive transfer of serum from the recipient of a rejected kidney, and the fact that some hours are required for the destruction of a second set kidney in a sensitized recipient, can reasonably be explained by high affinity antibodies¹² the concentration of which in the serum would not be expected ever to reach high titres. Other antibodies which may or may not appear after transplanting an organ, such as heterophile antibodies, leucocytotoxins, and leucoagglutinins, may be harmless by-products of