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.,

MYRE SIM.

Edgbaston,
Birmingham.

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.,

RICHARD DE SOLDENHOFF.

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Ayr, Scotland.

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 Davies[1] 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 Thomas[2] maintained that there is a "prepuberal" spurt of growth in the female pelvis. Harris[3] 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." Fairfield[4] 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 Miller[5] 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.,

J. R. E. JAMES.

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Wales

REFERENCES

1 James, J. R. E., and Davies, H. Edmund, *Journal of Obstetrics and Gynaecology of the British Empire*, 1952, **59**, 363.
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 allotransplant—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 confirmed[4] 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. Manzler[5] could detect serum leucocytotoxins during the height of rejection in about 30% of humans, but Yamada and Kay[6] 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 group[7] 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,[3] 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 evidence[9] 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 views[10] 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 polyspecific and become more so as time goes by. This is one possible anatomical explanation of cross-sensitization. There are reports[11] 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 antibodies[12] 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

some other significant antigen-antibody reaction. Let us move on to typing basement membrane antigens.—I am, etc.,

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REFERENCES

1 Dempster, W. J., Calnan, J. S., and Kulatilake, A. E., *British Medical Journal*, 1963, **1**, 23.
2 Milgrom, F., Litvak, B. I., Kano, K., and Witebsky, E., *Journal of the American Medical Association*, 1966, **198**, 226.
3 Dempster, W. J., *British Journal of Surgery*, 1953, **40**, 447.
4 Rosen, S. M., Truniger, B. P., Kriek, H. R., Murray, J. E., and Merrill, J. P., *Journal of Clinical Investigation*, 1967, **46**, 1239.
5 Manzler, A. D., *Transplantation*, 1968, **6**, 787.
6 Yamada, T., and Kay, J. H., *Surgery*, 1968, **63**, 637.
7 Shikata, T., personal communication, 1968.
8 Lindquist, R. R., Guttmann, R. D., Merrill, J. P., and Dammin, G. J., *American Journal of Pathology*, 1968, **53**, 851.
9 Dempster, W. J., *British Journal of Urology*, 1955, **27**, 66.
10 Humphrey, J. H., and White, R. G., *Immunology for Students of Medicine*, 1964, 2nd ed. Oxford, Blackwell.
11 Doniach, I., Crookston, J. H., and Cope, T. I., *Journal of Obstetrics and Gynaecology of the British Empire*, 1958, **65**, 553.
12 Karush, F., and Eisen, H. N., *Science*, 1962, **136**, 1032.

Absence from Work Attributed to Sickness

SIR,—Your leading article (15 March, p. 657) impels me to recall the incontrovertible fact that we doctors are not trained to recognize or diagnose normal workaday health. It is not in our curriculum: no questions are asked about it in our final exams.

And so there may arise the ludicrous situation in which the cautious doctor waits for the patient to say that he now feels perfectly well, while the anxious patient is awaiting the doctor's verdict that he is completely cured. Result: a draw, with a replay in a week's time. Meanwhile, another sickness certificate and some more face-saving pills. I hope I am exaggerating.—I am, etc.,

GEORGE DAY.
Mundesley,
Norwich.

SIR,—The Ministry's figures for days lost due to sickness absence, mentioned in your leading article (15 March, p. 657), refer to all insured persons; it is the experience of some of those practising in industry that the figures for those actually employed are considerably lower.

It is fair to say that the services available in Britain for the prevention and treatment of illness are extensive and elaborate, so that any major reduction of illness must await the education of the population, improvement of the environment, and medical research. In the meantime, under the circumstances of generous social benefits, the scope for improvement lies in trimming the edges of sickness absence. While this depends partly on the judgement of general practitioners, it is very greatly influenced by the patient's attitude to his work—job satisfaction—which is the province of management.

Doctors should be careful, therefore, to avoid fostering the opinion, widely held in the lay mind, that sickness absence is a medical matter remediable exclusively by medical means.

It is time for the profession to tell industry clearly that further medical improvement, beyond the standard now commonplace in those undertakings which employ doctors, will be slow, laborious, and costly, but that a substantial improvement is attainable by enlightened management and leadership. Medicine is no substitute for management.—I am, etc.,

G. R. KERSHAW.
Rugby, Warwicks.

Mortality Among Widowers

SIR,—Even the quality English press is romantic, and the title of this paper (22 March, p. 740) ensured publicity. It is thus unfortunate that some of the rules of population studies were broken and that there is no precise account of how the "expected" number of deaths was calculated. According to one paragraph: "The average age of widowers in any age group is about one year older than the average of married men within the same age group. This might explain up to a quarter of the excess overall mortality among widowers." My interpretation of this unpublished calculation is that the expected number of deaths (153) has been multiplied by 1·1, the factor by which male mortality rates at ages over 55 rises for each additional year of age. The expected published figure of 153 is raised to 168; and the excess mortality of actual deaths over expected falls from 213−153=60 to 213−168=45—that is, a reduction of a quarter. The excess mortality is then not 140% of expectation but 127%, and one wonders why a different figure is given in the summary.

Possibly unjustly, I suspect a second serious fallacy. The expected figure needed to be carefully adjusted for the year and month in which each actual death occurred. In 1957, the year in which the wives died, the first quarter had a crude male death rate of 12·9 per 1,000, one of the lowest rates, if not the lowest, recorded for a winter quarter. As always there was a rebound. Mild weather and the absence of epidemic respiratory infection merely postpones many deaths in the aged to the next winter. The mortality of widowers dying within six months of their wives will thus have occurred mainly in the third and fourth quarters of 1957 and the first quarter of 1958, when rates were high. Unless the authors had access to unpublished information and were able to allow for this difficulty, their "expected" figure is too low and needs to be multiplied, probably by a factor in the region of 1·1. This would raise their expected figure to 185 and reduces the excess mortality to 115%.

It is also wise in any statistical paper to calculate an alternative expected figure based on assumptions less favourable to one's hypothesis. We should be told the expected number of deaths based on the experience of all males rather than on all married males. The reason for this is that estimates of a total population are more reliable than estimates of its component sub-groups. A proportion of the population is living in sin on census night and untruthfully describes itself as married. The married population is thus exaggerated in comparison with the death certificates, on which marital status is more likely to be correctly entered. When the Registrar General does his divisions death rates in married men are minimized, and conversely they are exaggerated in unmarried men.

Finally, which is the cart and which the horse? Both members of a married couple die within months of each other. But which one fell ill first? The problem of nursing and extra care is too much for the other. The sick one is transferred to hospital and the tired and worried one has the further strain of journeys to hospital. Only too often it is the tired one who collapses and dies first. The heart hardens in age, spiritually as well as physically. Aged people surprise their friends by their philosophical acceptance of bereavement. They stand grief better than worry or unaccustomed physical exercise. We should have far more facts and more arguments before accepting the conclusions of this stimulating paper.—I am, etc.,

DENYS JENNINGS.
Budleigh Salterton,
Devon.

SIR,—The report of "broken heart" by Dr. C. Murray Parkes and his co-authors (22 March, p. 740) and the previous studies cited prompt me to suggest a probable connexion with giant cell arteritis, at least in some instances. Hughes and I,[1] in a series of 76 patients, found that a depressive state, often concealed, preceded the somatic manifestations of the disease by a few weeks or months, and that it commonly followed the death of a near or dear relative. Deaths of a cat or dog were provocative on two occasions in the series, while separation other than by death, such as by marriage, emigration, or admission to mental hospital, had affected a number of patients.

Nine patients presented with angina pectoris or cardiac infarction, and cardiac infarction and left-heart failure featured largely in those who were dead at follow-up. Since that report nine years ago, when the suggestion was first advanced, I have been made even more aware of the frequency with which the classical manifestations of giant cell arteritis precede or follow, by months rather than years, an episode of cardiac infarction, and this has led me to watch for cardiac infarction as a manifestation of underlying giant cell arteritis—a sequence of events for which there is ample support in a pathology of the disease. Resident staff continue to fear for my reason when I ask for a history of head and face pain, or "rheumatism," in a patient with coronary infarction whom I hear has been recently bereaved. At such times a persistently raised sedimentation rate, for weeks or months after infarction for no obvious cause, prompts a trial of corticosteroids to the frequent relief of angina, fever, or cerebral confusion. Only last week I saw a woman who developed psoriasis in 1954 a few months after the death of her husband. She was surprised when the first symptoms of polymyalgia rheumatica in the autumn of 1968 coincided with the first notable regression of psoriasis. Cured in twelve hours of her polymyalgia by corticosteroids, I thought it prudent to inquire what stress had promoted it. Characteristically, she said she hadn't a trouble in the world. However, prompted by anecdotes from previous patients, she suddenly said, "That's it," and told me how she had assisted the vet. to put her beloved old dog down in the spring. She was haunted for months in her dreams by its last screams. Her age and love of dogs had decided her not to take another; she was silently suffering from