Finally, your attempts to discredit our report and to ignore many of our arguments and your reference to "allegations" in the face of substantial evidence against South African psychiatry resemble the utterances of South Africa's apologists when they defend apartheid. Your "third option" (persuasion and encouragment) is an unsuccessful attempt to sit on a non-existent fence: you are either on the side of the dispossessed black majority who call for South Africa's isolation or are on the side of the apartheid regime which calls for the maintaining of links. At a time when the debate about South Africa in international health circles is intensifying it is disturbing to find *The Lancet* defending the practitioners of apartheid health care.

Anti-Apartheid Health Committee,
Anti-Apartheid Movement,
13 Mandela Street,
London NW1 0DW

J. FLUXMAN

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SIR,—Your editorial on apartheid and psychiatry replied to emotion with sense and pragmatism. There is in southern Africa a tribal conflict—a number of black tribes being successfully suppressed by a group of whites. To some, all blacks look the same, and to others all whites do. This may be a prevailing attitude in South Africa, but do we also have to adopt it? In much of Africa (Zimbabwe, Uganda, Nigeria, Burundi) many bloodier tribal conflicts are—or were—in progress. There, one black tribe is suppressing or even trying to eliminate, usually savagely, another black tribe. Happily blacks in South Africa are at much less risk of death by gun, starvation, or disease than the Ndebele or the underdogs in the other countries cited. Tribal prejudice and conflict is to be deplored, but are we to ostracise physicians from Zimbabwe because they happen to belong to and support the ruling tribe, or would the secretary for health of the British Anti-Apartheid movement have us say patronisingly that this doesn't count because they don't know any better? We must aim to eliminate tribalism and chauvinism on all fronts, both by education and by example, and not by ill-conceived boycotts.

Clinical Sciences Building,
University of Alberta,
Edmonton, Alberta, T6G 2G3, Canada

A. S. RUSSELL

DISEASE IN SOUTH AFRICA

SIR,—Dr Walker (Nov 17, p 1158) challenges Dr Seedat's view that in apartheid South Africa "the system makes effective medical care for the Black populations unattainable". Walker argues that the persistence of ill-health among blacks does not indicate indifference by government and that apartheid has little to do with it.

Britain, Canada, and Australia have so far failed to eliminate inequities in the health of their non-white minority populations but the relevance of this observation to the inequities that afflict the 84% black majority in South Africa is obscure to me. Walker seems to imply that the South African situation is as bad as it is because there the impoverished majority is also non-white, and that to alleviate the health problems of such people is especially difficult.

No epidemiologist will deny that the control of disease is complex but this does not lead to the conclusion that the ills of South African blacks relate solely to the health problems that these people have in common with the socioeconomically destitute elsewhere in the world and not to apartheid. In the more urban parts of this wealthy country, the risk of death for blacks compared with whites is 6-fold greater in infancy, and 14-fold at ages 1–4 years. The chance of death from gastroenteritis is 20, from tuberculosis 27, and from nutritional diseases 62 times greater.[1-3] In the rural areas—dumping grounds for the unemployed, the disabled, dependent women and their children, and the aged—there are no systematic data, but in certain areas infant mortality has been estimated at about 30% and at least 20% overall. Malaria is resurgent, cholera has appeared for the first time, and poliomyelitis is still epidemic. While it is indeed difficult to disentangle the effects of class, culture, race, and historically determined poverty, these disparities are not simply the result of those factors. They are in good part the result of a constitution that legalises racial discrimation.[3,4] South Africa's system permits political, social, residential, and occupational segregation, prohibits free movement in the search for work, enforces resettlement in desolate places, and blocks access to education and other services that could relieve the miseries of the people discriminated against.

Gertrude H. Sergievsky Center,
Columbia University,
New York, NY 10032, USA

MERVYN SUSSER

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EMBRYO QUALITY AND PREGNANCY RATES IN IN-VITRO FERTILISATION

SIR,—Dr O'Neill and Dr Saunders (Nov 3, p 1035) describe the use of embryo-derived platelet-activating factor as an indicator of embryo viability before transfer, during treatment by in-vitro fertilisation (IVF). They confirm our estimate[1] that only 40% of embryos have the capacity to implant. Further analysis of our data reveals where current IVF technology is limited in its potential to achieve pregnancies.

Following a successful pilot study[2] a service programme was established in collaboration with the PIVET Laboratory, Perth. Our techniques[3] are similar to those used in the UK and at other Australian centres.[4,5] So far we have achieved 70 clinical pregnancies, and 31 healthy infants have been delivered. Despite experience with more than 500 laparoscopic attempts the pregnancy rate is still below 20% (14% of laparoscopies or 16% of embryo transfers in 1984). The fluctuation of pregnancy rates throughout the year seems to be a universal problem, and to reduce the effects of technical deficiencies we chose three periods with pregnancy rates of more than 20% per transfer for data analysis. 165 transfers were studied—44 in December to March, 1983, 59 in January to March, 1984, and 62 in April to May, 1984. Embryos were graded by stereomicroscopy before transfer, for morphological appearance. A score of 3 points was allocated for embryos with clear regular blastomeres and no fragmentation, and ½ or 1 point was removed for moderate or severe fragmentation and for gross irregularity of or granularity/darkness within blastomeres.

37 pregnancies resulted from the 165 embryo transfers (22·4%). The total number of embryos derived was 419 and 49 gestational sacs implanted successfully (11·7%) (table).

73 (17·4%) of embryos were "poor" (scoring zero, ½, or 1), 230 (54·9%) were "fair" (1½ or 2), and 116 (27·7%) were "good" (2½ or 3). No pregnancies resulted when poor embryos had been the only ones transferred. Also, if one assumes that the higher grade embryos were the ones which successfully implanted in cases of multiple embryo transfers, no pregnancies were dependent on the successful implantation of an embryo graded as poor. When poor embryos were excluded from the analysis the implantation rate rose to 14·9% (49/328). Where at least one embryo implanted, the 49 gestational sacs were achieved from the transfer of 106 embryos (46·2%).

Where pregnancy ensued it is reasonable to assume that both maternal factors and technical factors were entirely favourable for implantation. The finding of 46% viable embryos is consistent with our earlier estimate[1] and that of O'Neill and Saunders. However, of all the embryos transferred only 15% implanted. Since the results analysed were those achieved in the most successful sessions, when technical factors were probably ideal for both the conceiving and non-conceiving groups of patients, it would seem that in 31% of cases (46% minus 15%) maternal factors prevented nidation.

IMPLANTATION RESULTS

Embryos transferred	No of gestational sacs					
	Embryos	0	1	2	3	4
All embryos						
1	38	35	3	—	—	—
2	106	42	8	3	—	—
3	72	18	4	1	1	—
4	140	27	6	1	1	—
5	45	4	1	3	1	—
6	18	2	—	1	—	—
Total	419*	128	22	9	3	—
"Morphologically reasonable" embryos transferred						
1	24	21	3	—	—	—
2	92	35	8	3	—	—
3	51	11	4	1	1	—
4	98	19	6	1	1	—
5	45	4	1	3	1	—
6	18	2	—	1	—	—
Total	328	92	22	9	3	—

*49 of 419 embryos implanted (11·7%).
†49 of 328 embryos implanted (14·9%).

Possible adverse maternal factors are an abnormal hormonal milieu,[6] poor luteal function resulting from the flushing of granulosa cells during oocyte recovery,[7] and the unusual stresses of IVF.

17% of embryos could be graded as non-viable, on the basis of stereomicroscopy. With exclusion of embryos with severe fragmentation and grossly irregular and dark, granular blastomeres, a proportion of embryos (55% of those graded "fair" in this series) will still contain minor morphological anomalies, though they will still be capable of generating a successful pregnancy. An alternative test to morphology, such as measurement of platelet-activating factor, is a promising development. The selection of embryos for transfer (or for cryopreservation) might thus be put on a more rational and efficient basis rather than the transfer of all embryos, which is wasteful and leads to false hopes in patients, or of those graded fair or good on morphological criteria, 23% of which are non-viable.

Regardless of the reason for the discrepancy between potential and actual embryo implantation rates, current techniques of oocyte recovery with embryo transfer in the same cycle are likely to continue to produce a limited pregnancy rate. We should be encouraging the development of alternative methods of oocyte recovery (eg, ultrasound-guided aspiration, avoiding general anaesthesia and laparoscopy) and the transfer of embryos in hormonally optimal cycles, and that means encouraging work on embryo cryopreservation with a view to transfer at a time when maternal factors are more favourable.[8]

Clinical assistance was provided by Dr S. R. Turner and Dr A. J. Murphy. Embryo cultures were done by J. M. Yovich, J. D. Stanger, S. C. McColm, B. D. Newman, and S. M. Junk at PIVET Laboratory, Wembley, Western Australia.

Department of Obstetrics and Gynaecology,
University of Western Australia,
King Edward Memorial Hospital,
Perth, Western Australia 6008

JOHN L. YOVICH

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IVF VERSUS NATURE

SIR,—Father Fleming and Ms Iglesias (Jan 19, p. 168) cite an isolated finding by Whittaker et al,[1] who suggested that human embryo wastage in nature is only 8%. They ignore all other data (see review by Biggers[2]) which show natural wastage to be much higher than this. The paper by Whittaker and co-workers contains a basic flaw. Whittaker et al gave as evidence of an ovulatory cycle a single-shot estimation of plasma progesterone greater than 1 ng/ml. This is unacceptable, and fertility specialists are unhappy about regarding single-shot levels even ten times higher than this as definite evidence of a fertile cycle. It is highly probable that Whittaker et al were not studying fully fertile cycles, which would explain why they found such a low incidence of pregnancy and why their data do not agree with more detailed studies by others. Moreover, a single negative human chorionic gonadotropin value during the luteal phase does not prove that conception did not occur in the cycles they studied.

Fleming and Iglesias state that in vitro fertilisation (IVF) and embryo transfer (ET) "are experimental procedures that are enormously wasteful of human life". The fact is that IVF and ET are established treatments, practised successfully in over twenty countries. We doubt if the once-desperate mothers of over 800 babies would agree with your correspondents. On the contrary, human life in these cases would have been impossible without IVF. Of course, results will improve with more research. Had their argument been applied, for example, to renal transplantation in its early stages when this procedure carried a high mortality, the most successful means of preserving human life for patients with renal failure would never have been established.

Institute of Obstetrics and Gynaecology,
Hammersmith Hospital,
London W12 0HS

ROBERT WINSTON
STEPHEN HILLIER

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DESIGN OF THERAPEUTIC STUDIES IN HERPES SIMPLEX ENCEPHALITIS

SIR,—The Swedish trial of acyclovir versus vidarabine in herpes simplex encephalitis (HSE)[1] brings into focus the diagnostic difficulties associated with this disease. Three problems are apparent: (1) the interpretation of diagnostic tests; (2) the grouping, for analytical purposes, of patients diagnosed by different means; and (3) the design of a proper clinical trial.

Skoldenberg and colleagues fail to draw attention to problems inherent in serological techniques to diagnose HSE. The largest series of patients with brain-biopsy proof of diagnosis[2] demonstrated a 90% sensitivity and an 80% specificity of serological assays, rates no worse than those in most of the much smaller series cited by Skoldenberg et al. The Swedish workers imply that the sensitivity and specificity of antigen detection in the brain tissue is 100%; this is incorrect. Why did they differ from all other workers in being unable to detect virus by isolation techniques in patients whose antigen tests were defined as "positive" on brain tissue? The recognition that several patients also elicited antibody response to varicella-zoster virus was not explained.

The main sites of herpes simplex virus involvement in the brain in patients other than the newborn are the temporal lobes. Involvement of the frontal or other lobes is much less common. Skoldenberg and colleagues' paper does not contain important data on site yet from correspondence with the Swedish group we learn that more "positive" results were obtained from frontal lobe than temporal lobe biopsy material (11 versus 9). Although virus was isolated from 7 of the 9 positive cases from the temporal lobe, in only 3 of 11 cases was virus isolated from the frontal lobe. Thus, "antigen" detection and/or positive serology provided "confirmation" of the diagnosis of HSE in 8 of 11 biopsy-investigated patients from a brain site which is infrequently affected by herpes simplex virus.