tinue to use cephaloridine as a local application in the prophylaxis of potentially contaminated wounds. We are indebted to Glaxo Research Ltd. for the supply of cephaloridine (Ceporin) and Aspro-Nicholas Ltd. for the gentamicin (Genticin).—We are, etc.,

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Referees and Rejects

SIR,—Dr. Samuel Johnson was only once worsted in debate, and even then by a woman. "Money," he declaimed, "there is no happiness that money lets in." "Very true," replied Mrs. Thrale, "but how much unhappiness does it keep out?" Mutatis mutandis, Mrs. Thrale—a very sensible person—would have said the same about medical referees.

I am greatly surprised to learn that nowadays authors argue with referees. My most earth-shaking papers were always returned, but no editor ever gave his reasons. In fact the last one you sent back to me did not even report your personal regret. In my declining years as a referee it never for an instant struck me that I should explain myself. In both cases Martial's all-sufficient reason was self-evident—"I do not love thee, Dr. Fell. . . ." Remember the Lord Chief Justice's advice to the young judge; "Give your verdict; it will probably be correct. Never give your reasons; they will usually be wrong."

Is it possible that we have been watching too much professional soccer, where referee baiting is half the fun? For your own sake, dear Editor, watch your step. All too soon we may see you carrying your leader writers shoulder high in a lap of honour round Tavistock Square and successful authors will be kissed on at least two cheeks.

Virgin authors should not be put off. There is no paper so brilliant that it will never be accepted by anyone; and none so poor that some good Samaritan will not give it room. Indeed some journals never publish anything else. And remember they all sell reprints, and the least-regarded journals of uncontrolled experiments and plagiarized clinical reports have usually the glossiest covers.—I am, etc.,

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Epidemic Neuromyasthenia

SIR,—We would like to thank Drs. G. G. Wallis and F. S. Perry (23 March, pp. 574 and 575) and Dr. R. A. Thompson (6 April, p. 60) for their interest in our paper on epidemic neuromyasthenia (23 February, p. 301).

In answer to Dr. Wallis the considerations which forced us to limit investigations, by the same token prevented us from involving psychiatrists in the evaluation of these patients. However, we did not feel there was sufficient psychiatric symptomatology to justify psychiatric referral. Of those affected, 50% had been nursing for less than 18 months. Morale was good and there was no "belle indifférence." We feel that disturbed consciousness is not a necessary feature of infective illness. Imipramine and amitriptyline were used in doses of up to 150 mg/day for up to four weeks.

Dr. Perry draws attention to the fact that the hospital patients were not affected and seeks an explanation. We felt that symptomatic illness may be age-related as the hospital patients were children. Since there are reports of children affected[1 2] Parish[3] suggested that the limited physical activity of hospital patients may protect them. We agree that nurses were predominantly involved, possibly due to their close proximity in the nurses' home. We also agree that we did not find an infective agent, but there was suggestive indirect evidence of an infective process. Toxic agents have been sought unsuccessfully in previous epidemics.[1 2 4 5] In fact one of us (A.J.S.) previously investigated a similar outbreak[6] for possible toxins with negative results. We felt that though this was not a good reason for not looking again, in our outbreak the evidence for a poison was slim and we were endeavouring to limit investigations.

We agree with Dr. Thompson that we overstated the relationship between anti-complementary activity and immune complexes in serum. We would add that sera from our patients were not bacterially contaminated, were tested within days or weeks of collection, and were consistently anti-complementary with higher titres in the acute stage of the illness.

One purpose of our paper, having taken investigations to a certain point, was the hope that others may have a platform from which to embark on further attempts to discover the cause of this odd disease.—We are, etc.,

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1 Sigurdsson, B., *et al.*, *American Journal of Hygiene*, 1950, **52**, 222.
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Primary Responses to Transplantation Antigens

SIR,—The techniques of undirectional mixed lymphocyte culture (M.L.C.) and cell mediated lympholysis (C.M.L.) have recently allowed the in-vitro study of the induction and effector phases of primary responses to transplantation antigens in man. In reciprocal M.L.C. the cells of mothers and newborns had given poor proliferative responses.[1]

We studied 13 families and unrelated controls using lymphoid cells (adult and cord blood) in the M.L.C. and C.M.L. technique previously described.[2] Results[3] showed again[1] that the newborns' cells underwent more cellular proliferation than controls when stimulated by the unrelated cells (110·7 ± SE 16·9 *v* 89·6 ± 15·6 × 10^3 c.p.m. ^{3}H-thymidine incorporation). However, in the same cultures fewer cytotoxic lymphocytes were generated than in the control cultures, as judged by the percentages of ^{51}Cr-release in target specific C.M.L. (28·4 ± SE 5·3% *v* 39·4 ± 6·0%). This suggests that at least two different subsets of lymphoid cells respond to an allogeneic stimulus by cellular proliferation with and without the development of cytotoxicity. The proliferative non-cytotoxic subset would make a higher proportion of the newborns' cells than of the adults' cells, thus suggesting an earlier function during ontogeny and phylogeny. The newborns responded to their mothers by low cellular proliferation (48·5 ± 13·4 × 10^3 c.p.m.; newborns *v* fathers 72·3 ± 14·1 × 10^3 c.p.m.) and extremely low cytotoxicity (9·8 ± 3·0%; newborns *v* fathers 20·6 ± 5·9%. These M.L.C. were across one major histocompatibility difference only).

The mothers were either primiparae or secundiparae, which did not seem to affect the results. The mothers' cells at delivery had practically a normal ability to develop cytotoxicity towards allogeneic cells (of unrelated 34·6 ± 9·1%; of father 36·2 ± 7·8%) and also developed cytotoxic lymphocytes towards cells of the newborns (23·5 ± 9·3%, as compared to 31·4 ± 9·3% in the fathers *v* newborns' reactions. These C.M.L. tests were across one major histocompatibility difference only). Low cellular proliferative and high cytotoxic responses were particularly found in the mothers *v* newborns reactions. Further study is needed to see whether this pattern could be due to in-vivo acquired immunity to paternal alloantigens. The overall results of C.M.L. alone did not reveal transplantation immunity in the mothers at delivery. On the other hand they clearly indicated that tolerance is not the mechanism which explains the successful allograft of pregnancy.

This work was supported by grant No. 3.695.71 of the Swiss Foundation for Scientific Research. G.D.B. is currently at the Cellular and Tumor Immunology Section, Laboratory of Cell Biology, National Cancer Institute, Bethesda, U.S.A.—We are, etc.,

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Sudden Death in Infancy

SIR,—We would support the view expressed by Dr. R. H. Anderson and others (20 April, p. 135) that further study is needed of the conducting tissues of the heart in the sudden infant death syndrome. There are, however, several aspects of their paper which we feel require comment.