doubtful whether treatment after eight years is worth attempting. This was why I was disappointed in your leading article (10 January, p. 62). No mention was made of the urgent need for early diagnosis and treatment. As an aid to early diagnosis the use of a check-list is valuable.² But this of course should merely serve to alert the doctor or health nurse as to the possibility of autism.

The need for early treatment is apparent when it is realized that the autistic child is cut off, by the very nature of his condition, from all normal developmental and learning processes. Thus he becomes progressively more and more isolated. A vicious circle has been set up which he cannot break. But with very early treatment he can be helped to live a nearly normal life.³ As Dr. Sim states this is admittedly a small group, but the stakes are very high, as without treatment the child will almost certainly end as a permanent inmate of a mental hospital.

With Dr. Sim's second point I entirely agree. I too am besieged by pressure groups, being currently involved with no less than thirteen. Each of these trumpets the cause of some childhood illness. They all want better facilities for their child—and who can blame them. Some societies are immensely rich. Some, just as worthy, are very poor. Some handicapped children and their parents have no special association or pressure group to barrack for them. This is a phenomenon of the age. But I don't think it can be stopped by disbanding the various societies. The more you suppress these dedicated parents the more actively they will lobby for their "rights." And already much of their fund-raising activity is helping to provide needed research money. Almost all these societies are concerned with long-term handicaps. What therefore is required is specially trained paediatricians who can assess and treat handicapped children and co-ordinate the many disciplines and organizations who have to deal with them. The multidisciplinary approach is the answer to the care of the handicapped child, but without co-ordination this can result in the frustrated mother pushing her child aimlessly from office to office.—I am, etc.,

J. RENDLE-SHORT.

Royal Children's Hospital,
University of Queensland,
Brisbane, Australia.

REFERENCES

- 1 Rendle-Short, J., *Medical Journal of Australia*, 1969, 2, 245.
- 2 Clancy, H., Dugdale, A., and Rendle-Short, J., *Developmental Medicine and Child Neurology*, 1969, 11, 432.
- 3 *Infantile Autism*, 16 mm. colour film, Sandoz Ltd., 1968.

Value of Necropsy

SIR,—May I support Dr. A. J. Ferris (3 January, p. 46) in his anxiety over the lack of communication between the coroner and his practice? While I have not experienced a precisely similar situation to the one he describes I have been increasingly disturbed at this communication gap, particularly manifest after sudden-death post mortems when information as to the results is not communicated routinely to us.

No doubt conditions vary in different parts of the country, and while we are particularly fortunate in this area to enjoy a long tradition of harmonious relations with

our coroners the communication problem remains, and seems to worsen with time.

I would therefore suggest that consequent on a doctor's statutory obligation to the coroner a similar obligation should be placed on him to at least fulfil the following points:

(1) To receive from the practitioner a written report on all cases reported under the Coroners' Acts 1887-1926.

(2) To inform the practitioner concerned of the time and venue of the post mortem.

(3) To send a copy of the post-mortem report on all cases to the reporting practitioner.

The forensic implications of sudden death cannot in my opinion be satisfactorily met unless these conditions are fulfilled, quite apart from the medical instruction involved and perhaps to a slight extent even the simple matter of courtesy may have relevance.

If these "Coroners Duties in relation to Medical Practitioners" were to be nationally adopted I have little doubt the benefits accrued would greatly exceed the costs and effort involved.—I am, etc.,

G. C. MATHERS.

Gloucester.

Immunotherapy in Acute Leukaemia

SIR,—Active immunotherapy in the treatment of acute leukaemia may well prove valuable in the management of patients with this disorder. Its possible role in the elimination of a residuum of leukaemic cells following chemotherapy appears reasonable enough to warrant its trial. The best means of utilization of such treatment is as yet unknown. The results obtained in a small number of patients by Drs. R. J. Guyer and D. Crowther (15 November, p. 406) using *Bordetella pertussis* vaccine suggest that this form of immunotherapy, superficially at least, may not be as effective as that described by Mathé *et al.*¹ using B.C.G. vaccine. Although the choice of *B. pertussis* vaccine appears to be prompted by its minimal side-effects and its lymphocytic action in animals, there are perhaps some reasons why this drug may not be the most suitable for promoting cell-mediated immunotherapy in acute leukaemia.

In studies involving the effect of vaccines on peripheral blood lymphocytes in short-term culture,² transformation, and by inference response to antigenic stimulus, did not occur under the influence of *B. pertussis* vaccine on cells from subjects who had presumably been sensitized to this antigen. Indeed, the cultured lymphocytes showed considerable morphological damage in this situation. Additionally a series of experiments by Levine and Wenk^{3,4} on the production of acute allergic encephalomyelitis in rats have indicated that *B. pertussis* vaccine produces an accelerated form of this disorder, which appears to be due to the adjuvant effect of this vaccine on immunological mechanisms only when given at or near to the time of antigenic stimulus. Fujii⁵ has also reported the effect of anti-*B. pertussis* vaccine on lymphocytes in vitro, where their survival seemed to be significantly prolonged, and confirmed the anti-"blastogenic" effect of *B. pertussis* vaccine on phytohaemagglutinin-stimulated lymphocytes.

In vivo infection with pertussis is stated to cause anergy in humans⁶ and the vaccine is known to inhibit the tuberculin reaction in B.C.G.-sensitized guinea-pigs.⁷ Therefore although *B. pertussis* vaccine appears to have the advantage of minimal side-effects in normal humans and in subjects with acute leukaemia, if large-scale trials of immunotherapy are to be undertaken then the somewhat ambiguous role of the vaccine on cell-mediated immunological responses should be considered when the chance of an active non-specific immunotherapeutic agent is made.—I am, etc.,

STUART ROTH.

Faculty of Medicine,
The University,
Southampton.

REFERENCES

- 1 Mathé, G. *et al.*, *Lancet*, 1969, 1, 697.
- 2 Elves, M. W., Roath, S., and Israëls, M. C. G., *Lancet*, 1963, 1, 806.
- 3 Levine, S., and Wenk, E. J., *Proceedings of the Society for Experimental Biology and Medicine*, 1966, 122, 115.
- 4 Levine, S., and Wenk, E. J., *American Journal of Pathology*, 1967, 50, 465.
- 5 Fujii, K., *Hiroshima Journal of Medical Sciences*, 1968, 17, 261.
- 6 Fanconi, G., and Wallgren, A., *Lehrbuch der Pädiatrie* 8th. ed., Basel, Schwabe, 1967.
- 7 Floersheim, G. L., *International Archives of allergy and Applied Immunology*, 1965, 26, 340.

Survival After 195 Defibrillations

SIR,—Mr. M. Cross (27 December, p.808) questions in his letter the possibility of the effect of hypercapnia on electric excitability of the myocardium in our patient. As a matter of fact, we were considering the same problem, and the patient was put on a Bird respirator when the PCO₂ reached 107 mm. Hg on 16 May. However, the patient's blood pressure reacted by dropping from 120/70 to less than 80/50 in forty-five minutes, and ventilating was therefore discontinued, with resulting increase of blood pressure to 120/70 within 30 minutes.

In the afternoon of the same day—that is, about five hours later—PCO₂ reached its peak, 115 mm. Hg, and we again started ventilating the patient, but blood pressure reacted by dropping to 70/? within 30 minutes and later to 50/0. After two hours it was decided to abandon ventilation and blood pressure returned to 100/60 in 90 minutes and was 120/80 six hours later. PCO₂ then decreased to 89 mm. Hg on 20 May and was under 75 mm. Hg three days later, reaching 50 mm. Hg on 30 May. During the hypercapnia all heavy sedation was stopped and the patient encouraged to breathe deeply. Oxygen was given by a nasal catheter at the rate of 2 litres per minute.

It is, of course, possible that the high PCO₂ had some relation to the repeated attacks of ventricular fibrillation although it would be difficult to prove it. It was, however, interesting that only very occasional ventricular ectopic beats were monitored between the attacks of ventricular fibrillation.—I am, etc.,

M. MICHAEL KUBIK.

Burton Road Hospital,
Dudley, Worcs.

Campbell de Morgan Spots

SIR,—As acting medical officer of health for Lancaster and area during the outbreak of the lesions described by Dr. R. H. Seville and his colleagues (14 February, p. 408) I