exact nature remains uncertain. The suggestion of Mr. Slapak *et al.* that it may have an immunological basis does not seem to us to be either premature or unreasonable.—We are, etc.,

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Antiserum in Gas Gangrene

SIR,—Gas gangrene occurs sporadically in civilian surgical practice, commonly after amputation for peripheral vascular disease,^{1,2} or after operations breaching the gastrointestinal tract, or in association with contaminated traumatic wounds.

The present therapeutic approach is empirical, and may be summarized as follows. Gas gangrene is a highly lethal disease. Therefore if its presence is merely suspected it is justifiable to discharge a battery of therapeutic measures, whose efficacy, singly or in combination, will never be put to the acid test of controlled clinical trial, given due regard for medical ethics.

Therefore the use of any of the four mainstays of treatment—namely, amputation, penicillin, gas gangrene antiserum, and hyperbaric oxygen—is based on hope rather than expectation of success. My only purpose in writing is to examine the place of antiserum in the treatment of gas gangrene. Recourse to the literature for guidance is disappointing. The use of antiserum seems to be based on extensive animal experimental work of questionable relevance to the human condition. However, in war wounds it has been found³ that the mortality in 20 patients who received no antiserum was 90%, but this was reduced to 57% in 100 patients who received antiserum. The authors confess that this series probably comprises only 50–60% of the actual incidence of the condition in their theatre of war. This was a retrospective survey, with all the inherent pitfalls of this method of measurement.

There is therefore very little evidence in favour of the therapeutic efficacy of antiserum. But there is indirect evidence that it is ineffective—namely, in a separate series of 9 of the 12 fatal cases treated with antiserum had therapeutically adequate blood levels of antiserum at the time of death.⁴ Furthermore, antiserum was widely used in the first world war, inconsistently in the second world war, and rarely in the Korean war. The incidence of gas gangrene in three representative series is as follows:

	First World War (France) Pettit (1919) ⁵	Second World War (India) North (1947) ⁶	Korean War Howard and Inui (1954) ⁷
No. of wounds ..	4,377	4,600	4,900
Gas gangrene ..	221	32	4
% incidence ..	5%	0.7%	0.08%
Case fatality rate	27%	31.3%	0

In addition, North⁶ reported that he treated Chinese and American soldiers, but that antiserum was available only for the latter. On

analysis there was no significant difference in the mortality from gas gangrene between the two groups, so its use was discontinued.

One might conclude from these figures that antiserum caused positive harm. In fact the decreased incidence has been attributed to more rapid clearance of casualties with speedy debridement of wounds, and may have been influenced by other factors such as increased use of penicillin; but I submit that it is unlikely to have been due to antiserum and that the use of this substance should be discontinued.—I am, etc.,

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Rehabilitation after Head Injury

SIR,—Mr. Walpole Lewin (24 February, p. 465) is to be congratulated on emphasizing the need for proper facilities for rehabilitation after head injury. The problem has of course increased markedly—for example, in Edinburgh our admissions from head injury have doubled in the last five years. There, as in Cambridge, these patients are under the care of the surgical neurologist, but in Edinburgh they are admitted to a head and spinal traumatic unit, part of the accident/emergency service and quite separate from elective surgical neurology. Our admissions now total between 1,200 and 1,300 a year, but the number of severely injured (post-traumatic amnesia of more than 24 hours) is higher—13%, as opposed to Mr. Lewin's figure of 7%.

As he so rightly emphasizes, our concept of recovery from brain injury is outmoded. Long-term improvement appears to be increasingly possible even after severe head injury, but more often in the young, who have an inherent capacity to learn. Thus planned rehabilitation of a progressive and imaginative kind designed for each patient's particular needs is throwing a new light on the understanding of recovery by the use of alternative cerebral pathways.

In the south-eastern region of Scotland, the injured from the last war were cared for in the brain injuries unit of Bangor Hospital under the care of Professor Norman Dott. As in Oxford with the late Sir Hugh Cairns, rehabilitation was already advanced in 1941, and it has continued since in a more elaborate form. Even the mildly concussed are seen in a follow-up clinic after one week so that their return to work can be planned successfully, and this step would seem to have reduced the incidence of the post-concussional syndrome. The more severely injured are rehabilitated at the Astley Ainslie Hospital (rehabilitation hospital of 230 beds in a large park 1½ miles from the centre of the city), on an inpatient basis in the first instance, by a skilled team of physiotherapists, occupational therapists, speech therapists, social workers, and nursing and medical staff. They are allowed home as soon as this important step

would seem wise, and sometimes it is an important one in furthering the progress of the patient. However, if the adult members of the family are out at work then these patients are treated at the "day unit" of the hospital by the same staff. Thus there is complete continuity of management, with the general practitioner being brought in increasingly at this stage.

Those patients who remain unconscious to all intents and purposes for many months or even years may well be required to be cared for in a special part of the rehabilitation unit. Such a special unit is now being planned.

I warmly support Mr. Lewin's recommendation that an organization for co-ordinating services for head injuries be set up and that some special rehabilitation centres should be developed to deal with brain-injured patients.—I am, etc.,

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Problems Associated with L.S.D.

SIR,—Many cannabis smokers are aware that prolonged coma occurs with an excessive intake of this drug. I saw the patient reported by Dr. R. Clayton (20 January, p. 163) shortly before his admission to hospital, as he was under my care for psychotherapy at University College Hospital. He had begged me to prescribe him tincture of cannabis, as his anxiety was intolerable and he wanted to be "stoned out of his mind all the time for the rest of his life." He refused to consider admission as an inpatient, and agreed to try diazepam. I have seen him since his episode with Dr. Clayton and had his account of what happened.

It was not clear to anybody exactly how much of anything this patient had taken, but it was clear to me that after a period in which the police had closed up the supply of cannabis temporarily the patient was only too anxious to get as good a "high" as possible as soon as he laid hands on the drug again. During the 24 hours before admission he had smoked cannabis in large quantities, as well as taking lysergic acid diethylamide (L.S.D.), diazepam, and probably paracetamol.

I have seen two previous episodes where a patient became semicomatose for a long period when taking cannabis. On one occasion the patient, aged 14, combined his first reefer with two or three "purple hearts" and two or three double whiskies. On the other a cannabis habitué smoked several cannabis cigarettes to ward off the symptoms of impending influenza—he may have taken paracetamol as well, but, like the patient shared with Dr. Clayton, he "couldn't rightly remember."

The literature on cannabis reports potentiation of barbiturates by cannabis,^{1,2} and Miras' reports prolongation of the barbiturate sleeping time in rats which had been given sublethal doses of cannabis, which lasted for 30 days after the last dose. The same sensitivity to barbiturates after cannabis had been put in the food for six months was present for the same length of time but to a lesser degree. It is more likely to occur