hospital admission in order to determine which patients may wish and be suitable for I.U.D. insertion or tubal ligation at the time of pregnancy termination.—I am, etc.,

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Schistosomal Involvement of the Bowel

SIR,—I was most interested in the paper by Dr. S. M. Sherif (14 March, p. 671) on malabsorption and schistosomal involvement of the jejunum.

I agree with Dr. Sherif about the rarity of schistosomal infestation of the small bowel, but my experience does not accord with the statement "Rarely the appendix and the caecum are affected." Here, at least, massive infiltration of the appendix with ova of *Schistosoma mansoni* is very common in appendices submitted to histological examination, whether acutely inflamed or not. We admit an average of 75 cases per annum of acute appendicitis to this hospital of 1,000 predominantly acute beds.—I am, etc.,

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E.E.G. and Survival

SIR,—I should like to draw the profession's attention to the fact that the E.E.G. Society has a subcommittee to deal with the problem of the study of resuscitation and survival. This subcommittee has made some provisional recommendations regarding the recording from subjects whose survival is in doubt. These are provisional because it is felt that further evidence is needed before more dogmatic statements can be made, and the main recommendations, which are summarized below, are largely concerned with the technique of recording to demonstrate the absence of E.E.G. activity.

Electrodes of the "stick-on" type seem preferable to pad electrodes; needle electrodes may sometimes have to be used. Placement of electrodes should allow bilateral assessment of all major brain areas, and the minimum number of recording electrodes should be eight. Electrode resistances should not exceed 5K Ω per electrode (or 10K Ω per pair—6K Ω impedance). Where resistances appear less than 1K Ω per pair, the electrodes should be specially checked. It is desirable to include E.C.G. and other extra-cephalic leads.

Equipment with at least eight channels is recommended to permit simultaneous recording from as many areas of the scalp as possible, with a minimum of six channels of E.E.G. There should be the usual range of high frequency filters and time constants; and the noise level should be such that cerebral potentials* of 2 μ V peak to peak could reasonably be recognized in the write-out.

It is advisable to use a time constant of at least 0.3 sec. for most of the recording, and a paper speed of 1.5 cm./sec. for at least part of the recording. The amplification should be such that 2 μ V signals could be readily identified; this may require increased gain to 3.5 or 2.5 μ V/mm. level. Montages should include long interelectrode distances (> 10 cm.) and preferably some monopolar/average reference derivations. The apparatus and connections should be tested by systematic and deliberate touching of electrodes to produce extraneous potentials. It is desirable to demonstrate the absence of E.E.G. response to stimuli (e.g. pain, sound, light).

There should be 20 minutes actual recording though 30 minutes would be preferable. Attention should be paid to recording conditions to identify and eliminate extraneous signals. The record should be interpreted by a medically-qualified neurophysiologist / electroencephalographer, who has been present during at least part of the recording. Where satisfactory recording conditions cannot be obtained it may be impossible to provide a reliable assessment.¹

It remains certain that the E.E.G. on its own cannot enable one to give a definite prognosis on survival. The whole clinical situation has to be taken into account, especially since certain factors (e.g., intoxications, hypothermia) can appear to abolish temporarily much of the electrical activity of the brain. Nevertheless, the E.E.G. is one of the useful tools in assessing the chances of survival of an individual.

One important point on which evidence is still lacking is the minimum time for which the persistent absence of E.E.G. potentials in scalp recording can be regarded as indicating irreversible inactivity of the brain. It seems likely that many hours are necessary, and that such an interval may be needed between "inactive" records before a definitely bad prognosis may be given.—I am, etc.,

ELMAN POOLE,

Secretary,

E.E.G. Society Subcommittee for
Resuscitation and Survival.

Churchill Hospital,
Oxford.

REFERENCE

- 1 E.E.G. Society Subcommittee for Resuscitation and Survival, *Recommendations*, April, 1970.

Buruli Ulcer

SIR,—I was very interested to read your leading article entitled "Buruli Ulcer" (16 May, p. 378).

Last year I spent two months in Peru, and during my stay I spent some time in the humid jungle regions of the country in search of the Buruli ulcer. The climate of the Buruli district is similar to that of the Amazon basin in Northern Peru, and it seemed reasonable to suspect the existence of *Mycobacterium ulcerans* in this area. In Iquitos, situated on the Amazon and the largest town of Peru's jungle regions, I came across an interesting case which I would like to report here.

The patient was a boy aged 4 years living in the centre of Iquitos. He was brought to the dermatologist by his father who gave details of the history. The boy developed a small nodule above the right knee. The nodule grew until it reached the size of a pea when a small hole appeared in the centre and began exuding fluid. The ulcer grew in size as dead skin from the periphery sloughed off. No pain referable to the lesion had ever been experienced. After two months the boy was seen at the hospital. The ulcer was then 2 cm. in diameter, the skin was separated from the subcutaneous tissues, and a probe could be passed under the undermined edges of the ulcer. The surrounding skin was a pink-red colour and was not warmer than elsewhere. There was no pain or tenderness and no inguinal lymphadenopathy.

The ulcer was curetted and a considerable amount of abnormal adipose tissue evacuated. The destructive process extended deeply, involving the fascia of the quadriceps.

Unfortunately no pathological or bacteriological examinations were performed on the specimens because the necessary equipment was not available. The boy was Mantoux positive, and it was assumed that the ulcer was caused by *Mycobacterium tuberculosis*. The treatment for six months with P.A.S., isoniazid and streptomycin resulted in a complete cure leaving a well-formed scar.

In view of the history given and the nature of the ulcer it seems very likely that it may have been a result of infection with *Mycobacterium ulcerans*.—I am, etc.,

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Home from Hospital

SIR,—I refer to your leading article (18 July, p. 119), discussing the home care of patients discharged from hospital, and how far their needs had been met.

The final paragraph contains the statement "Hospitals know little of their patients' home circumstances, doctors do not hear of their patients' hospital treatment. . . ." This is a generalization which is quite untrue of many hospitals. It is the practice in my own group to make every possible inquiry into home circumstances before the discharge of a patient, and every general practitioner receives two discharge reports—a brief interim one at the time of discharge, and a full copy of the summary within a week or two at the most.

Time spent on ward rounds at the bedside of patients whose hospital investigation and treatment are nearing completion and are shortly to be discharged is more concerned with the home circumstances and care available than with the clinical details. Consultants, residents, sisters, welfare officers, etc., are all involved, and repeated interviews with relatives are arranged, or attempted, in some cases without success. Obviously the results are not always satisfactory, but to imply that hospitals in general make no effort to this end is surely quite unjustifiable.—I am, etc.,

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Oral Contraceptives and Hypertension

SIR,—I refer to your leading article (16 May, p. 378), just to hand.

It may be of interest to note that I first drew attention to this in 1963 and since then I have had three published references there-to.¹⁻³ May I draw attention to my comments at the World Congress,² and to the *Medical Journal of Australia*,³ where I wrote "The table suggests . . . rise of blood pressure [is] more likely to occur in those who have had toxæmia of pregnancy."

In conclusion, may I state that the blood pressure levels may be very high. In two of my patients the readings were 220/120 and 210/110, and since I have seen two patients on the pill for up to 10 years with a diastolic pressure of 150. It would seem that there