amputation pain is repeated spinal analgesia, this being simpler to carry out than an effective block of the sciatic nerve. I do not consider that injection of local analgesic drugs often produces permanent relief, but relief for several months, which occasionally occurs, is better than no relief at all, and the injections can be repeated.

Chordotomy I found to be disappointing in the two patients on whom I employed it, though in one of them considerable improvement was produced; and before chordotomy is considered in patients in whom sympathetic block has failed I would suggest that procaine injections into nerve trunks or spinal analgesia should always be given a trial.

I agree with Dr. Craig and disagree with Mr. Gillis about the relationship of phantom pain to causalgia following peripheral nerve injuries. The character of the pain and its frequent aggravation by emotional disturbances and temperature changes, and in the lower limb by defaecation and micturition, are strikingly similar in both conditions. With complete nerve section, too, the pain that sometimes develops is identical with causalgic pain following incomplete nerve lesions, though the peripheral changes, such as increased sweating, cyanosis, and hyperalgesia, do not of course occur. These patients are in exactly the same condition as patients with amputations except that the hand or foot has not been amputated. And, moreover, if amputation is performed in patients with causalgia the pain often continues as before, though now felt as phantom pain.^{3,4}

The hyperalgesia found in the hand and foot in patients with classical causalgia is, of course, absent in patients with phantom pain after amputation, but I would like to point out that the hyperalgesia sometimes found in amputation stumps presents exactly the same features and to suggest that the mechanism of production is essentially the same. It is customary to regard tender hyperalgesic stumps as a condition quite separate and distinct from phantom pain, but in my experience almost all patients with hyperalgesic stumps have phantom pain, though, as pointed out by both Mr. Gillis and Dr. Craig, patients are often so afraid of their sanity being doubted if they talk of their phantom pain that they frequently complain only of the tender stump. It is not very uncommon for a patient with a tender stump to mention that he also feels pain in the absent hand or foot only after he has been under one's care for a considerable time and after he has learned that his complaints will not be scoffed at. Mr. Gillis's statement that following finger amputations causalgic pains are frequent but phantom pains rare is, I believe, to be explained in this way: I have had some patients who, in addition to pain and tenderness about the amputation scar, experienced pain that was felt to be in the amputated finger, and it is probable that many others have similar pain but do not mention it.

A further point of similarity between the two conditions is their response to sympathectomy. I have found that, as with phantom pain, sympathectomy relieves causalgic pain after nerve injuries in many cases, but not in all. This would seem to be the experience of most British surgeons who have dealt with these cases.

Not the least interesting aspect of this subject is the number and variety of the views that have been put forward in an effort to explain the many puzzling observations that have been made. Most of these views, including those put forward by Mr. Gillis and Dr. Hugh. H. Carleton (May 29, p. 1052), have not been supported by any convincing evidence. Investigations that I carried out on a number of patients provided evidence in favour of quite a different view.⁵ Incidentally, the findings in patients with phantom pain and in patients with causalgic states after peripheral nerve injuries were exactly comparable, and thus support the view that these conditions have a similar aetiological basis.—I am, etc.,

Belfast.

J. A. W. BINGHAM.

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Diagnosis of Gonorrhoea

SIR,—Dr. F. J. T. Bowie (May 29, p. 1030) does not say what he regards as "reasonably adequate measures" of identifying the gonococcus. Is he satisfied with the demonstration of the germ in a Gram-stained smear, or does he require proof by its isolation in pure culture and confirmation by the appropriate sugar tests?

My practice in the Army is to treat for gonorrhoea after I have taken an exhaustive history and found a typical discharge in which I have identified presumptively the gonococci by their staining reactions and their shape in or around the leucocytes.

If the history, the discharge, or the smear is not typical, I send the man for culture of his discharge before starting treatment. In marital cases I make it clear to the man that success in culturing the gonococcus from his discharge will be more cogent evidence against him in a court of law, and that he has the right to refuse this test if he does not wish scientifically to incriminate himself.

I disagree that a "delay of a day or so while awaiting a laboratory report on a sample of pus is of little significance." Although I have not yet classified my results, I am forming the impression that acute gonorrhoea treated within 48 hours of the appearance of the discharge by 250,000 units of penicillin is far more likely to settle down without the continuing of the discharge than when treatment is started on the third day. These cases seem more apt to develop a persistent posterior urethritis or a prostatic urethritis. I have repeatedly found that men coming for treatment after the third or fourth day of the clap, when there is an early secondary infection, do not pass the final test of cure at the end of the third month and on some occasions have been returned to the S.T.C. long before this by their M.O. for further treatment.—I am, etc.,

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Symmetrical Gangrene in an African

SIR,—The following case of symmetrical gangrene in an African, though it does not add anything to the aetiology of the disease, is of interest in that it presents many similarities to those described by Dr. Michael Gelfand (June 14, 1947, p. 847) and Dr. J. G. Salter (July 19, 1947, p. 108).

A northern Sudanese aged about 35, a sailor on one of the Nile steamers, was admitted to Malakal hospital on April 16, 1948, complaining of fever. His general condition was good; he was not unduly undernourished. Blood slides for the first four days were negative for malaria, but as his spleen was just palpable, and malaria is very common among these sailors, he was given three tablets of paludrine in a single dose on admission. At the time we were trying out this single-dose therapy for malaria. He had no other antimalarial drugs at all.

The fever did not react to this treatment and continued to vary from 100° F. (37.8° C.) to 103° F. (39.4° C.) for over a week, after which it settled slowly. A white cell count taken on April 20 showed 5,150 cells per c.mm. The Widal and blood-culture tests were negative for typhoid, paratyphoid, and mellitensis infections. There was no oedema of the feet at any time. Heart and lungs were normal. No helminthic infections were found in faeces or urine. On admission the urine was acid and clear, but on April 19 it was alkaline and had a trace of albumin. No sugar was found in the urine at any time. B.P. 130/90.

On April 24, eight days after admission and ten days from the first symptoms, the patient complained of inability to use his right leg. The limb was cold and resembled a spastic paralysis with loss of pain below the knee, but no reflexes could be elicited at all. On April 25 there was evidence of gangrene with a line of demarcation about one-third up the leg. On April 26 the line of demarcation had reached the heads of the tibia and fibula. It proceeded no further. Postural treatment with warmth was given and the condition eased somewhat. There was a return of warmth and feeling for a short period to the upper part of the leg, but this again went off after 24 hours. On April 28 a total blood count was as follows. Red cells 3,090,000; white cells 2,200 per c.mm. and haemoglobin 67%.

By May 1 the condition was stationary, the temperature slightly above normal, but the general condition of the patient worsened. He lost weight, lost interest in his surroundings. On May 3 gangrene developed suddenly in the left leg, went through the same stages as in the first except that the process was much more rapid and complete within 24 hours. The line of demarcation in the left leg was almost exactly identical with that in the right. The patient died quite suddenly on May 4, 1948.

The condition would appear to resemble that described by Gelfand except that there was no oedema. The patient being a Moslem, a necropsy was denied us. He had no obvious signs of deficiency disease such as pellagra.

I am indebted to Dr. E. P. Pratt, Director S.M.S., for permission to publish this case report, and to Dr. R. Kirk, F.R.S.E., for advice and interest.

—I am, etc.,

J. F. E. BLOSS.

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Province Medical Inspector.