Rectal and Vaginal Temperatures

SIR,—The recent interesting correspondence on vaginal and other temperatures in the *Journal* calls for further data. As I have some, I submit the following résumé.

In 1934 I read a paper at the British Association in Aberdeen, dealing with the significance of diurnal variations of vaginal temperature. At that time we could find no previous records. Dr. Havelock Ellis wrote to me about my then current research: "You raise an interesting question about the temperature of the os uteri, which I have never thought about, and I am sorry to say know nothing about. I cannot recall having come across it among the books I have by me."

The making of observations originated with my testing the efficacy of greasy soluble suppositories. At my Mothers' Clinic I had had recorded the daytime temperatures of the cervical region in over 100 normal women and found that they were higher than anticipated. This led me to make a more searching examination of some selected cases, such as women doctors and midwife nurses, who could manipulate a thermometer in their own vaginas with sufficient care. Thereafter I had simultaneous readings made of all the three loci—mouth, rectum, and the cervical end of the vagina. Each set of the three thermometers used to take readings were of identical make, carefully checked in air temperatures, and all were left in place for ten minutes for each reading. Though they were "half-minute" thermometers, I found that the readings were not entirely reliable until each had been in for ten minutes. A number of records were made per woman at 6 a.m., 12 noon, and 10 p.m. An average chart of the kind is shown in Fig. 1 (....=oral, —=rectal, ----=vaginal temperature).

FIG. 1

It will be seen that the general relation of the rectal temperature to that of the vagina is different from what is now currently supposed, and that, though the rectal temperature is higher than the vaginal early in the day before exercise has been taken, it is markedly lower at midday, when the cervical temperature reached its peak. In addition to these, I made very special readings for a few days, in one case, of six sets of three simultaneous readings in each 24 hours, waking the subject by alarm clock, so that the readings were at 2 a.m., 4 a.m., 6 a.m., 12 noon, 6 p.m., and 10 p.m. The burden on the experimental subject was so great that this chart (Fig. 2) is the only one, and

FIG. 2

is of special interest. In it the oral temperature is of course always well below all the others, but both the rectal and vaginal temperatures show curves more complicated than where the readings were three in 24 hours.

Though some charts were shown in Aberdeen in 1934, these original records have never been published, and so have not hitherto been available to those now interesting themselves in the questions of rectal and other temperatures.—I am, etc.,

London, W.1.

MARIE C. STOPES.

Harrison's Grooves

SIR,—Drs. J. Naish and H. R. E. Wallis (March 20, p. 541) deserve congratulations for their lead in debunking the long-entertained ideas about the causation of Harrison's grooves. As a teacher I was always dissatisfied with the aetiological explanation given in the textbooks about Harrison's grooves and was collecting clinical evidence to disprove the "rickety" hypothesis of the grooves when it was a pleasure to come across the article by the above doctors in the *Journal*.

One thing definitely emerges from my observations, and it is that Harrison's grooves are not limited only to the condition of rickets. They appear in a number of heterogeneous conditions which produce debility and muscular asthenia in the child—as in prolonged illness, protracted convalescence after acute diseases, chronic malnutrition, chronic respiratory infections, chronic anoxic conditions, etc. The essential factor in the production of the grooves is muscular weakness. This in turn causes the kyphotic posture in the sitting child and the "pot belly" or protuberant abdomen. I fully agree with Dr. R. Good (April 10, p. 707) that distension of the abdomen is also an important contributing factor, but I do not believe with him that frequent distension of the abdomen after hearty feeding is one of the causative factors. If it were as Dr. Good writes, then the incidence of Harrison's grooves would be much greater, and would be taken as physiological rather than pathological. For, if we remember right, all of us have taken frequent and hearty feeds in our days.

I believe that the laxity of the abdominal musculature and kyphotic sitting posture, with or without enlargement of liver and spleen, are the important causes of abdominal distension. In the kyphotic sitting posture with abdominal distension the lower ribs can be actually seen splaying out with the costal border everted, producing horizontal grooves above on both sides. As days pass by, the ribs become fixed in the new position, thus perpetuating the grooves. In rickets the grooves are particularly common and pathognomonic of the condition because all the causative factors are present.

It will be of interest in this connexion to note that in India in persons of a particular community who often have huge obese abdomens due to affluence, and asthenic musculature due to lack of exercise and sedentary habits, horizontal grooves on the lower part of the costal cage are visible which closely simulate the Harrison's grooves in the child. Thus, I strongly believe that the aetiology of Harrison's grooves lies not in rickets as such but in the lax and asthenic musculature, kyphotic sitting posture, and pot belly or protuberant abdomen.—I am, etc.,

Poona, India.

P. L. DESHMUKH.

Pain in Phantom Limbs

SIR,—I have been much interested in the recent correspondence in the *Journal* on "Pain in Phantom Limbs" and in the various views that have been expressed, for when in charge of amputation cases in the Indian Army from 1944 to 1946 I had the opportunity of observing many patients with phantom pain. With Dr. J. Donaldson Craig's views (May 8, p. 904) I am in almost complete agreement. I would, however, point out that operations on amputation stumps, such as neurectomy or reamputation, do not always fail to relieve phantom pain. In some cases, especially those with obvious stump defects, the result of such operations can be most gratifying.

Like Dr. Craig and others¹ I have found that satisfactory relief of phantom pain (and of stump tenderness) can be obtained in many cases by interruption of the sympathetic chain, though I have found operative sympathectomy to be more satisfactory in most cases than repeated procaine injections. The effect of a procaine injection should of course always be observed first. In a series of 16 cases of post-amputation pain and tenderness I found that interrupting the sympathetic chain produced complete or very satisfactory relief in 9 (56%).

I have found, as has Dr. Craig, that cases that are not benefited by interruption of the sympathetic chain can sometimes be relieved for prolonged periods by repeated procaine injections into nerve trunks, and occasionally even by a single injection. Such a result, however, is not usual, and I agree with Mr. Leon Gillis (June 5, p. 1108) that in the majority of such cases only very temporary relief of pain is produced, even by repeated injections. A method I have sometimes employed with occasional success for lower-limb

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.

Sudan Medical Service, Malakal.

Province Medical Inspector.