this difference in dietetic control may account for the altered insulin-sensitivity exhibited in coma by the two groups of patients.—We are, etc.,

D. M. DUNLOP.
J. B. DONALD.

Edinburgh.

SIR,—Professor R. H. Micks (July 24, p. 200) stated, "As far as I know there is no record of a patient ever having passed from diabetic coma into a fatal hypoglycaemic attack." The implication is that this does not happen, but such a conclusion is hazardous. I would suggest that hypoglycaemia in the energetic treatment of diabetic coma, without facilities for frequent blood-sugar estimations or utilization of such, or without the concomitant use of glucose, is common and is occasionally fatal. I have certainly met with it when emergency treatment, particularly over week-ends, was in the hands of relatively inexperienced hospital residents. Publications of such cases may be advisable, but too much significance should not be attached to their absence.

At a 1947 endocrine conference, when I spoke of the dangers of overdosage with desoxycortone, three doctors informed me of their experience of acute adrenal insufficiency being transferred into fatal hypopotassemia, and expressed their appreciation that the danger had been aired. Existing publications hardly reflected such experiences. Hormones are indeed powerful agents, and overdosage no less a danger than timid inadequate therapy.—I am, etc.,

London, W.1.

S. L. SIMPSON.

SIR,—The following case illustrates two points in Professor R. H. Micks's article (July 24, p. 200).

The patient, a girl of 21 years, was admitted to hospital with a 7 months' history of great lassitude and amenorrhoea; she had had a boil on her buttock for 10 days. Retrosternal oppression and increased laboured respiration had been present for 12 hours. Temperature was 101.5° F. (38.6° C.), pulse 144, respirations 28. Her face was flushed, mouth parched, tongue red and dry; there were no chest signs, the heart was normal, the abdomen resistant. The patient was not comatose, and diagnosis of hyperglycaemia was only made after the urine had been examined and was found to be loaded with albumin, sugar, and acetone. Further questioning disclosed thirst and polyuria for seven months, blurring of vision for some weeks, and pruritus vulvae for two days. Blood sugar was 400 mg. per 100 ml., and the B.S.R. was 62.

The orthodox treatment of insulin and glucose was commenced with 50 units of soluble insulin and 50 g. of glucose by mouth at 9 a.m. and repeated at half these doses two-hourly. The patient grew worse throughout the day, vomited 18 oz. of dark-coloured fluid at 9.30 p.m., and became comatose. An intravenous dextrose saline was then given. Her rapidly worsening condition gave rise to anxiety, and the dose of insulin and glucose was stepped up to 25 units and 20 g. hourly, and then half-hourly, until a total of 750 units of insulin had been given. Meanwhile catheter specimens of urine were examined with each dose for fear of hypoglycaemia. The large doses of insulin were administered as a desperate and final measure and were considered a gamble.

With a total of 750 units by 7 a.m. the following morning the patient showed marked improvement. Blood sugar had fallen to 85 mg. per 100 ml., though the urine still showed sugar +++ (3%). Three days later she had a fasting blood sugar of 296 mg. per 100 ml., which rose to 405 mg. one hour after glucose and was 367 mg. after 4 hours. Sugar +++ and acetone ++ were found in each specimen of urine during sugar-tolerance test. Further progress was uneventful.

This case is of interest as demonstrating Professor Micks's points of the dangers of too conservative an administration of insulin and of gastric dilatation. Large insulin dosage at an early stage would have been safer. Glucose was given both by mouth and intravenously in liberal amounts in accordance with current usage. The withholding of glucose in diabetic coma is a modern departure and an interesting one.—I am, etc.,

Stirchley, Birmingham.

ROBERT J. D. BROWNE.

SIR,—To me it appears that Professor R. H. Micks (July 24, p. 200) implies that injection of insulin into the tissues of an "inaccessible" diabetic patient is devoid of danger. What else can be meant by advocating that "the doctor called to a case of coma should always give at least 100 units before even ordering the ambulance"? Perhaps the context makes it sufficiently

clear that his advice to general practitioners supposes competence to differentiate between diabetic coma and insulin reaction. Without presuming to give an opinion on the reasonableness of this supposition, I venture to record the impression gained as a hospital resident that many doctors are unwilling to attempt clinical diagnosis of the unconscious diabetic patient, and to suggest that the advice quoted above is not devoid of danger.

I have in mind an instance which may be without parallel but not without moral. A doctor summoned to the patient of a colleague (who was away) found a diabetic man unconscious. The doctor gave insulin and reassurance. Some four hours later the doctor prescribed more insulin and a hot-water bottle. Four hours after that the patient arrived in hospital, where consciousness was shortly restored with intravenous glucose. The patient recovered from his hot-water bottle burns in a fortnight, and from the empyema which complicated his pneumonia in a further six months. When the doctor who had prescribed the insulin was told the history of the case, the only comment was, "At least my insulin did him no harm."

May I submit that Professor Micks's counsel to the general practitioner should be reworded: "The doctor called to a case of coma should always order the ambulance before giving any insulin"?—I am, etc.,

Droitwich Spa.

BRIAN WEBBER.

Pain in Phantom Limbs

SIR,—In their letter (July 31, p. 267) Dr. R. E. M. Bowden and Dr. J. R. Napier put forward a point of view that I think should not be allowed to pass unchallenged. They state that cases of phantom limb pain should be divided into two separate and distinct types according to whether pain is (1) burning in character or (2) stabbing, shooting, or tingling. (They do not mention that in addition to these various degrees of cutaneous pain patients with phantom pain also experience deep muscle and joint pain.) Their view is, I realize, a commonly held one, but several reasons can be given for disagreeing with it.

1. *Experimental Observations.*—Among the many interesting observations on pain sensation made by Sir Thomas Lewis and still not sufficiently well known, one was that exactly the same type of cutaneous stimulus could produce either burning pain or a stabbing, pricking sensation, the result depending entirely on the intensity and duration of the stimulus. A brief stimulus gives rise to "pricking" pain, a more prolonged stimulus to "burning" pain; and this is always so in whatever way cutaneous pain is provoked. In other words, the difference between burning pain and a stabbing, pricking, or tingling sensation is merely a difference in intensity and does not necessarily involve any difference in the nature of the causative lesion.

2. *Clinical Features.*—In addition to patients who have constant burning pain in a phantom limb and those who have constant stabbing, pricking, or tingling sensation, there are many who, while they usually have a pricking or tingling sensation, experience burning pain during periods of exacerbation of pain such as commonly occur in these cases. There are also patients who generally do not suffer from any type of phantom pain but at intervals have attacks of pricking pain or of burning pain. And in many patients who have severe burning pain for some weeks or months after amputation the severity of the pain gradually lessens, so that later they have a stabbing, pricking type of pain, and later again experience even this type of pain only at intervals. It seems impossible to regard these different types of phantom pain as indicating different conditions, for that would imply that one condition could change for a period into another. The more reasonable view would seem to be that the nature of the condition remains the same, the only change being variation in intensity of pain. Moreover, tender neuromata may be present in patients with burning pain as well as in patients with less severe pain, and in both groups pressure on tender neuromata causes exacerbation of pain. Such associated features as cutaneous tenderness, cyanosis, and hyperhidrosis are as a rule prominent only in severe examples of the condition, with burning cutaneous pain and usually also severe muscle and joint pain.

3. *Response to Sympathectomy.*—Dr. Bowden and Dr. Napier state that patients with the less severe type of pain are unlikely to respond to sympathectomy. I carried out sympathetic procaine blocks in several patients of this group and found that in them, just as in patients with severe burning pain, this produced immediate relief in some and not in others. Subsequent operative sympathectomy was, however, practically never carried out in patients without burning pain, as in these patients the phantom sensation was seldom unpleasant enough to make them anxious to undergo a major operation for its relief.

For these reasons, then, I regard all cases of phantom pain as being similar in origin and mechanism of production, whether they have burning pain or not. It is, however, possibly not altogether desirable for the word "causalgia" to be used to describe all these cases, with their varying painful sensations, or even for the term "minor causalgia" to be used for the less severe examples. As pointed out by Dr. Bowden and Dr. Napier, Weir Mitchell originally coined the word "causalgia," meaning literally "fire pain," to describe only the single symptom of burning pain; and it might still be desirable to use it only in this sense, or at least only for cases with this type of pain, without of course implying that there is any difference in pathogenesis between this "causalgic" pain and lesser degrees of pain following nerve trauma.—I am, etc.,

Belfast.

J. A. W. BINGHAM.

Fibroma of the Labium Majus Complicating Pregnancy

SIR.—It must be very rare for tumours of the vulva to reach such a size during pregnancy that they constitute a possible difficulty in labour. The following report describes the occurrence of such a tumour.

CASE REPORT

A Muganda woman came to the hospital in October, 1947, to await her confinement. She had had two previous normal deliveries and was then about eight months' pregnant. It was a vertex presentation, and there was no disproportion. From the vulva hung an elephantoid mass the size of which can be gauged from the accompanying photograph.

This mass had first appeared in January, 1947, and the patient must have become pregnant very shortly afterwards. As the tumour was likely to cause obstruction to delivery it was decided to perform caesarean section at term, and then remove the mass later. Accordingly on Nov. 19, 1947, when labour had just begun, a classical caesarean section was carried out.

Three weeks later, on Dec. 12, 1947, the tumour was removed. It was found to be arising from the right labium majus, with a broad pedicle extending up to the right of the symphysis pubis. The mass weighed approximately 5½ lb. (2.4 kg.). Recovery was uneventful, but the patient insisted on leaving hospital before the perineal wound had completely healed.

Dr. R. B. Baird, of the Medical Laboratory, Kampala, reported on the tumour as follows: "Consists of a mass of fibro-fatty tissue in which there are fairly numerous dilated lymph vessels. I can find no evidence of lymphogranuloma or of filariasis, but cannot definitely exclude the latter. Probably a fibroma."

My thanks are due to Dr. R. B. Baird for his pathological report, and to Dr. Hebe Welbourn for the photograph.

—I am, etc.,

Kampala, Uganda.

W. R. BILLINGTON.

Acute Intussusception in Childhood

SIR.—The interesting and thorough paper by Drs. Brenda Morrison and Donald Court (April 24, p. 776) brought to my mind an experimental and clinical study on the subject made by my former chief, Dr. W. Obadalek, which appeared in 1929 in *Brun's Beiträge zur klinischen Chirurgie* (146, 668). Its clinical part is based on 53 cases of intussusceptions in infants and children ranging in age from 3 months to 13 years, 28 of them under 2 years old. They were treated in the surgical wards of the Children's Hospital in Brno (Czechoslovakia) between 1910 and 1928. Since 1921 the number of infants among the material increased steadily, apparently because the condition was more frequently diagnosed by the general practitioner.

Obadalek's diagnostic observations concur with those of Morrison and Court and so do the results of treatment, which in 50 cases was surgical. A diagnostic "pointer" which Obadalek stresses is dullness on lumbar percussion of the supine patient which disappears after he has been turned on the opposite flank, thus indicating free fluid in the abdominal cavity. He found it in all cases of his material, and its great value in his opinion lies in the fact that it helps to differentiate non-surgical diseases—e.g., dysentery, purpura, constipation, alimentary intoxication—from abdominal conditions requiring surgical attention. In doubtful cases a diagnostic enema (without x ray) often produced blood where none had been observed before.

The high proportion of intussusceptions in childhood—53 out of a series of 101 cases of ileus treated altogether in that period—Obadalek explains by (1) stronger intestinal peristalsis and more irritable intestinal mucosa in children; (2) the unequal growth of ileum and colon, resulting in a difference in diameter between ileum and colon, which is most marked in infants of 4–6 months; and (3) a mobile caecum frequently found in infancy and early childhood. The intussusception, he believes, is initiated by a localized intestinal spasm. In 15 out of his 50 operated cases congenital or inflammatory changes were found in the intestine concerned, or in its vicinity: 4 polypi, 3 Meckel's diverticula, 1 tuberculous ulcer, and 7 cases of inflammation of the appendix ranging from chronic disease involving the regional lymphatics to gangrene.—I am, etc.,

Derna, Cyrenaica.

F. BARBER.

Filariasis

SIR.—The initial clinical manifestation of *Loa loa* may only appear several years after infestation with the parasite, and infected individuals may not develop symptoms until after taking up residence in this country. Furthermore, as these symptoms are thought to be due to an allergic reaction to toxic products of the worm resulting in urticarial eruptions and painless swellings in the subcutaneous tissues, either localized (Calabar swellings) or diffuse, the condition may be diagnosed as angioneurotic oedema, and the underlying cause passed unrecognized. The following case report may be of interest.

A male, aged 26, was referred to hospital on April 7, 1948, because of a painless swelling of the left arm of 5 days' duration, and itching of the left shoulder. There was no history of relevant illness in childhood or adolescence, and the family history showed no proneness to allergic disorders. He had served in Southern Nigeria from 1943 to 1946, with only a few months' interval in 1944, and had had eight attacks of malaria. Repatriated in November, 1946, he had been fit and had worked up to the present illness.

On general examination he was well nourished; all the internal organs were normal. Slight conjunctival injection and palpebral swelling in the left eye was noticed. There was uniform swelling with no tenderness and with only slight pitting on pressure of the left axilla, arm, forearm, and hand. A mild papular eruption could be felt over the pruriginous area. There were no nodules. He was recommended to take "anthisan," 200 mg. 3 times a day, and to report in one week's time. The swelling did not subside with this treatment, and he was therefore admitted on April 15.

The history of prolonged residence in Southern Nigeria led us to suspect African filariasis, more commonly known as *Loa loa*, but no microfilariae were seen in the blood films taken on numerous occasions over a period of several days. A concentration test was also negative. Blood counts, however, showed on repeated occasions a high total leucocyte count, 21,000–32,000, with a marked eosinophilia of 51.5%–65%. The laboratory of the London School of Hygiene and Tropical Medicine kindly performed a complement fixation test with dirofilarial extract, and this was found to be positive.

The swelling slowly subsided and had practically disappeared by April 24, there remaining only a palpable gland in the left axilla. The patient was discharged on April 26. On April 30 the painless, diffuse swelling reappeared in the left supraclavicular region and shoulder. It disappeared on May 4. The patient was later transferred to another hospital for chemotherapy.

The diagnosis of *Loa loa* was based in this case upon the following significant points: (1) the occurrence of angioneurotic symptoms in an individual who had returned from a filarial region in West Africa; (2) the finding of leucocytosis with very high eosinophilia; and (3) the finding of a positive complement fixation reaction with dirofilarial extract. A skin test with the extract, often positive in the disease, was not carried out in this patient, under the mistaken impression that it might not be