are becoming resistant to proguanil or else an already proguanil-resistant strain has been introduced to Manus from the mainland of New Guinea, for several patients were seen towards the end of my stay there who contracted slide-positive benign tertian malaria despite (their veracity was not in question) the above-mentioned suppressive routine. The administration's solution to this problem is the advocacy of a double daily suppressive dose. The question may well be asked, When will this process end? Presumably, when a strain similar to that reported in *Nature* (1951, **168**, 332) is encountered, we shall have to take 55 tablets of proguanil daily.—I am, etc.,

West Ryde, N.S.W., Australia. ROLAND G. CHAMBERS.

Disappearing Naevi

SIR,—In his interesting communication Dr. F. Parkes Weber (October 27, p. 992) refers to the spontaneous disappearance, or partial disappearance, of certain types of naevi in children. The incidence of naevi in the newborn, particularly on the forehead, in relation to the eyelids, the root of the nose, and the nape of the neck is much higher than many realize. Most of these disappear during the first year of life. The fact is not so widely known that in women there is a tendency for naevi to appear and sometimes to grow rapidly during pregnancy, only to regress equally rapidly in the puerperium. This tendency is particularly marked with naevi of the face, lips, tongue, and gums. Their growth is sometimes so rapid during the later weeks of pregnancy that they are mistaken for malignant tumours. It is interesting that the sites in which they are found so closely resemble those in which naevi of the newborn commonly occur. It would seem reasonable to suggest that the same maternal factors (probably hormonal) are responsible for these similar manifestations in both mother and infant.—I am, etc.,

Oxford. JOHN STALLWORTHY.

Treatment of Migraine

SIR,—The excellent annotation on this subject (September 1, p. 538) has been followed by correspondence (September 15, p. 673), and as one who suffered severely from migraine from boyhood until the late fifties I think my personal experience will be of interest.

My attacks came on quite suddenly and occurred from once to several times a week, interfering with schooling during specially bad weeks. The worst attacks occurred during the 1914–18 war, and on one occasion the attack was so severe that it became necessary to hold on to a railing with my right hand while my left arm and leg were in constant tremor. During that particular attack my mind was so confused that I could not have given my name. It was followed by a dazed feeling in my head for some hours before finally passing off. In one instance just before entering the examination hall for the paper on medicine I experienced so severe an attack that vision became difficult and I could not read one word of the paper for nearly an hour.

The actual attack always presented in the same sequence: an ache suddenly occurred in the right supraorbital nerve immediately above the eye, and was quickly followed by a feeling of tightness just inside the skull about an inch higher up than the ache. It always seemed to me to be a constriction of an artery. Soon afterwards there was marked throbbing slightly to the outer side of the apparent constriction, and while the throbbing lasted the headache continued, and the worse the throbbing the more intense the headache. The duration was usually for some hours only, but in bad attacks it lasted for 24 hours or more.

Although during the attacks I experienced some nausea I never vomited. Usually a neuralgic pain over the right forehead followed the attack and gradually disappeared. The sight of the right eye always became blurred until the migraine passed away. My eyesight had been fully corrected since my youth.

Various drugs were tried for relief of the pain, but usually without much, if any, success. I found the most useful to be tablets of phenacetin, caffeine, and aspirin. Much more relief was obtained by cold-sponging the right temple and the top of the head. No cause for the migraine was discovered.

Fifteen years ago I was treated successfully for an affection of the right antrum and ethmoid, sphenoid, and frontal sinuses, and there is no doubt whatever that since that time I have hardly ever been troubled with migraine. This may be important in other cases, but probably there is more than one affection capable of causing arterial spasm.

I might mention that as a child, and until the age of 20, I suffered from white (dead) fingers in cold weather, evidently owing to spasm of the artery, and a brother of mine who also suffered from migraine was similarly affected.

—I am, etc.,

London, N.W.8. BERNARD MYERS.

Acute Tuberculous Septicaemia with Leucopenia

SIR,—The cases described by Drs. K. Ball, H. Joules, and W. Pagel (October 13, p. 869) prompt us to publish the following case.

A business man, aged 62, was admitted on August 1. He complained of vague malaise, anorexia, occasional diarrhoea, and loss of weight for three months. For several weeks he had had night sweats and more recently rigors with fever up to 101°–104° F. (38.3°–40° C.). On examination he was a wasted, ill man. Temperature 102° F. (38.9° C.), pulse 96. His tongue was moderately coated. The liver was smoothly enlarged by three fingerbreadths, and the spleen was enlarged by two. There was no palpable lymphadenopathy. The heart, lungs, and C.N.S. were normal. He had old bilateral iridectomies.

His white cells numbered 2,400 per c.mm. (neutrophils 78%, lymphocytes 18%, Türk 4%), Hb 51%, red cells 3,730,000 per c.mm., colour index 0.68; Van den Bergh reaction was delayed; serum bilirubin 2.25 mg.%; thymol turbidity 2 units; serum colloidal gold 000000. Prothrombin index (Quick) 83%. Blood culture was sterile, and the urine showed only a trace of albumin. Chest skiagram normal and barium meal and follow-through showed no abnormality of the gastro-intestinal tract.

The patient went steadily downhill with fever up to about 102° F. daily. Needle biopsy of liver was contemplated, but in view of his condition was felt to be unjustified. No final diagnosis was reached and he died 18 days after admission, approximately six weeks after the onset of fever and three to four months from his first vague symptoms.

At necropsy the relevant positive findings were: scarring at the apices of both lungs with a few adhesions low on the left side: oedema and bronchopneumonia at the bases; enlargement sometimes with caseation of deep cervical, mediastinal, and retroperitoneal lymph nodes and less marked enlargement and lymph nodes throughout the mesentery; enlargement of the spleen with whitish, necrotic nodules up to 0.5 cm. diameter; enlargement and softening of the liver, which contained numerous smaller whitish areas; one small whitish nodule in the left kidney; apparent regeneration of the bone marrow of the right femur down to the junction of its lower and middle thirds.

Histologically the mediastinal and retroperitoneal lymph nodes were largely replaced by caseation with moderate lymphocytic and endothelioid cell reaction and an occasional Langerhans giant cell. Most of the splenic and hepatic lesions showed necrosis with an occasional giant cell but little or no round cell infiltration. The left kidney showed a small area of round cell infiltration. The apex of the right lung showed pleural thickening but no marked change in the lung tissue. The lesions in the lymph nodes, spleen, and liver contained a moderate number of acid-fast bacilli. This case would appear to resemble the anatomical form (*a*) described in the article. The lack of round cell infiltration and the abrupt change from normal to necrotic tissue were the predominant histological findings in the liver and spleen suggesting an inadequate reaction to the infection.

If his condition had permitted, liver biopsy would have seemed a logical method of arriving at the diagnosis. Our thanks are due to Dr. R. B. Terry, physician, and Dr. G. L. Robinson, pathologist, for facilities to publish this case.—We are, etc.,

Greenwich, S.E.10. S. R. GLOYNE. R. KING-BROWN.

Serum Potassium Deficiency

SIR,—Dr. J. A. Strong (October 27, p. 998) has fallen into the common error of considering threshold voltage measurements a reliable indication of neuromuscular excitability.

The level of threshold stimulation depends on the two factors skin resistance and tissue excitability. Environment, skin preparation, and technique have been shown to have great influence on skin resistance, and hence on threshold measurements.[1 2 3] For