concentrations. In our experience patients receiving 100 mega units of penicillin daily in 1.5 litres of infusion have daily urine volumes of about 2 litres. It may be that other alterations in renal function, as mentioned by Brunner and Frick, contributed to hypokalaemia in their patients.

Having tried a number of methods we now use a continuous infusion with three half-litres of 0.18% saline, to which is added penicillin G in quantities of 30, 40, 30 mega units, dividing each ration approximately equally between the sodium and potassium salt. This gives an infusion solution of approximately 280 mOs/litre. We have no difficulty in obtaining potassium penicillin, and indeed alterations to the plasma sodium and potassium levels can be induced by varying the amount of the appropriate penicillin salt. Using this method, we have treated patients for up to three months with daily infusions of 100 mega units of penicillin without electrolyte imbalance, neurotoxicity, marked changes in serum osmolality, or indeed haemolysis.[2] "Massive" intravenous penicillin therapy is useful and safe, provided it is realized that as well as a large dose of penicillin the patient will be receiving a load of water, electrolyte, and possibly other solute such as glucose.—We are, etc.,

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Training of Surgeons

SIR,—Mr. Neville Stidolph proposes a training system for surgeons which is tied to the present requirement of about 100 new consultant surgeons per year (9 November, p. 379). Of all British candidates setting out on a surgical career only 140, which is 12% of the original field, will attain an English Fellowship each year. Thus after six years of training even those relatively few who have successfully gained a Fellowship are not assured of a consultancy. This situation, he says, "is manifestly unjust." It is therefore proposed not to increase the number of consultants required each year to 140 but to cut down the number of candidates who will successfully achieve a Fellowship to 100.

The proposals outlined by Mr. Stidolph are presumably of a long-term character. As such it is strange to realize that his entire proposal rests upon the assumption that Britain should, would, and could employ overseas graduates to fill 75% of the registrar posts in surgery indefinitely. Is it really feasible to plan the long-term reorganization of surgical training and staffing upon such an assumption?

At the second Commonwealth Medical Conference held last September the Indian delegation, in answer to a questionary, stated that,

(*a*) Provision of undergraduate medical education abroad for the various categories of professional staff in health services is not necessary. (*b*) For postgraduate or postbasic level also it is in general not necessary except in specialized branches.[1]

The Indian delegation also stated that "there are places for 6,000 postgraduates a year in medical institutions in India."[2] Considering that the number of medical graduates was about 7,600 (1967) this would, therefore, indicate no shortage of postgraduate places in India. In addition, the Pakistani delegation[3] stated that, "Their own needs [in regard to training facilities] . . . were now limited, to a large extent, to certain more sophisticated specialties."

The statements cited above are in contradiction to the assertion that "the developing countries need and will need for many years the facilities for postgraduate and specialist training which exist . . . in the U.K." If India and Pakistan do not require the postgraduate facilities available in Britain, why then should their graduates continue to come here? The advantages to an individual doctor from India or Pakistan who achieves an English Fellowship are obvious. However, from Mr. Stidolph's figures we can see that only 6% of the overseas doctors who make the attempt do manage to achieve a Fellowship in the Royal College of Surgeons. The situation is then that large numbers of overseas doctors come to Britain to train for higher qualifications which few will achieve. Mr. Stidolph does not produce any evidence that the men coming here are "the outstanding products of their universities" who were "selected to be the future consultants" and teachers in their own countries. The truth is that we know very little about what happens to overseas doctors after having trained in Britain.

Mr. Stidolph warns that Russia, Germany, and the United States of America provide funds and scholarships to draw overseas doctors to their countries. So that if we in Britain "do not recognize our obligation to cater for their needs the graduates will go elsewhere." At one and the same time Britain can fulfil her obligations to the Commonwealth, and keep her hospitals staffed, and the Royal College of Surgeons will be able to control more carefully than ever those who are permitted entry.

There is no avoiding the fact that Britain needs more *British* doctors, which in turn means that a training and hospital staffing structure is required which will be based upon the work needs of hospitals being filled by British doctors. This is possible only if all doctors in training can reasonably expect to become recognized specialists (with appropriate rewards) as proposed by the Royal Commission on Medical Education.

Such changes are also likely to act as a major deterrent to emigrating doctors, the major part of whom leave this country because of the frustrations they experience in trying to find permanent positions in the hospital service. If Mr. Stidolph's proposals are acted upon they will add to these frustrations, and thus to the volume of emigration; but then if 75% of the junior hospital staff are to be drawn from abroad perhaps it will not matter.—I am, etc.,

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Alcohol and Drugs

SIR,—General practitioners received last month a circular from the Committee on Safety of Drugs, accompanied by a letter from the Medical Assessor. This latter concerns the interaction between alcohol and drugs, and what advice should be given to patients on this matter. Though I agree with most of what is said, I feel I must challenge the statement embodied in the last sentence, "a commonsense rule, when you prescribe any drug affecting the central nervous system, is to warn the patient not to take alcohol whilst under treatment."

It seems to me this is a statement typical of someone who is not in active medical practice. It ought to be obvious that a complete ban on consumption of alcohol while taking such drugs is most impractical. Does the Medical Assessor not realize that there are very many people who are on regular doses of drugs affecting the central nervous system, and very many of these naturally enough wish to drink alcohol. To tell them not to do so is anything but common sense and is the sort of advice that will be simply ignored to the detriment of both patient and doctor–patient relationship.—I am, etc.,

Armley, Leeds. G. E. PHILIP.

Frusemide for Cardiac Failure in Infancy

SIR,—Dr. K. A. Harrison (12 October, p. 84) has reported the use of ethacrynic acid for blood transfusions in 30 cases of severe anaemia of pregnancy. I would like to report the use of the diuretic frusemide in a premature infant with cardiac failure due to coarctation of the aorta who required a blood transfusion for severe anaemia.

A twin, of 32 weeks' gestation, birth-weight 2 lb. 12 oz. (1.2 kg.) was noted to have a systolic murmur and absent femoral pulses on initial examination; blood pressure in the right arm was 65 mm., right leg 45 mm. systolic by the flush method. He developed cardiac failure at the age of 6 weeks, which responded to treatment with digoxin. By the age of 9 weeks his haemoglobin had fallen to 49%, pulse 200 per minute, respiratory rate 80 per minute, and his liver was enlarged 1½ fingerbreadths. In order to avoid an exchange transfusion and to save the saphenous veins for subsequent cardiac catheterization he was treated with frusemide 5 mg. intravenously followed by 60 ml. of packed cells over the next 20 hours. During this period the pulse rate fell progressively to 120 per minute, and the respiratory rate fell to 70 per minute. A month later he had gained 28 oz. (0.8 kg.) in weight, and had no signs of cardiac failure. His haemoglobin was 101%.

I suggest that a powerful diuretic such as frusemide or ethacrynic acid is useful in the management of cardiac failure due to anaemia in the neonatal period.—I am, etc.,

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Insanity and Tumours

SIR,—We read with interest the report by Dr. R. Hunter and his colleagues (6 July, p. 9) of three cases of frontal meningiomas presenting psychiatrically. We report below the features of one such patient who came under our care recently.

A 44-year-old English woman was referred to the neurosurgical unit of the University College

Hospital (U.C.H.), Ibadan, on 23 April 1968, from the psychiatric unit on account of increasing drowsiness and the finding on physical examination of bilateral papilloedema and left hemiparesis. She had been under treatment for paranoid schizophrenia since September 1967.

She was first seen in this hospital in April 1961 after miscarrying a 10-week pregnancy, her second within two years. Her outside doctor attributed these to inadequate rest in the early months of pregnancy, "due to her high sense of duty." Her first child was delivered normally at home in 1959. There was no record of her postnatal state. She delivered her second child normally in April 1964 five weeks premature. Six weeks later she reluctantly resumed duty, and for the first time in her life suffered "a terrible headache."

Since then she has had recurrent attacks of frontal headaches weekly. She was treated for migraine and obtained minimal relief for four weeks. In January 1965, on account of the increasing severity of headaches and the occasional association of vomiting, she attended the medical outpatient department of U.C.H., Ibadan. She was found to be bradykinetic (due to repeated sedation), otherwise there was no abnormality on physical examination. A diagnosis of depression was made. Two days later she asked for her discharge and she was sent home on meprobamate.

In September 1967 she was admitted into the psychiatric unit on account of headaches, loss of memory, and talkativeness. Her usual frontal headaches became obstinate, and at their severest she vomited easily. Early in the year she had lost her job as secretary when the decay in her memory became manifest, a feature which later became progressive. She slept a lot, and, when not sleeping, talked a lot. She was admitted, and treatment was instituted for paranoid schizophrenia. There were frequent reports of her suddenly and spontaneously becoming stuporose and dribbling saliva.

It became obvious that an organic lesion was probably responsible for her strange behaviour. Radiography of skull showed some erosion of her dorsum sella. On the carotid angiograms the right middle cerebral artery was displaced medially and upwards by a mass in the middle-third of the sphenoidal wing. The vascular flush suggested a meningioma. A right temporal osteoplastic craniotomy was performed and a large discrete meningioma, about 5 cm. by 4 cm. by 6 cm., was removed from the sphenoidal wing. It was highly cellular and relatively vascular and histological sections later showed it to be angioblastic in nature. The patient made a satisfactory recovery and was discharged home from hospital 18 days after her operation. Her headaches ceased, she became mentally clear, and talked freely without lack of wit. Her memory improved significantly, and an intelligence test performed on her while on holiday in England placed her Wechsler scale at 130.

Of particular interest was the onset of the patient's symptoms six weeks after the delivery of her second child. Examples have been described of meningioma which showed accelerated development during pregnancy, and in each case the meningioma has been suprasellar, parasellar, or at the sphenoidal wing in location. Sensitivity to hormonal effects and increase in physical size of the tumour as part of the generalized water retention are reasons adduced for changes in pregnancy. What bearing the histological nature of the meningioma has on its biological behaviour during pregnancy remains a subject for speculation. Any increase in size in the angioblastic meningioma of our patient during her pregnancy was probably due to engorgement of the vascular elements within the neoplasm. One of us (E. L. O.) has witnessed enlargement of intracranial aneurysms during pregnancy.—We are, etc.,

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Bilateral Parietal Thinning in Bronze Age Skull

SIR,—Bilateral parietal thinning or biparietal resorption was noted on a female cranium recovered from Harappa (in West Punjab), which is the type-site of the Indus civilization belonging to the Bronze Age cultures and dated 2300 B.C. The age at death appeared to be about 45 years. This is probably the only reported palaeo-pathological case from the Indo-Pakistan subcontinent.

The cranium, which was unearthed from a regularly disposed burial at cemetery "R 37," is otherwise normal and well preserved. It has two irregular large holes in each parietal bone. Examination has revealed that these holes are artificial and post-mortem breaks occurring at the sites of lesions causing depressions. These depressions are situated bilaterally and almost symmetrically. The thickness of the vault bones at different places beyond the margin of the lesions is on an average 6 mm. From the outer margin of the lesions, where the normal bone thickness is retained, to the margin of the breakages, there is a decreasing gradient in thickness of the bone. This thinning was due to the migration of the outer table at the area of involvement, and the gradient was formed by gradual disappearance of the diploic surface before finally exposing the inner table just near the margin of the holes. It thus involved a resorption of bone and a deposition of compact bony tissue along the surface. This condition of thinning is by itself indicative that the woman had suffered from a lesion of a severe nature. This evidence and the specific localization suggest that it was a typical case of thinning of the parietals, which is not to be confused with other congenital anomalies located very near to the parietal foraminae.[1]

This condition was recognized on some ancient Egyptian crania by Smith.[2] He found those skulls having "strange, large symmetrical depressions of the parietal bones."[3] Rowling[4] also noted this in the mummies of Thutmosis III and Meritamon of the New Empire period, and also in Khety from the middle kingdom. This abnormality has interested pathologists as a contemporary disease involving an atrophy of the parietals, usually accompanying old age. It has been concluded that it is a congenital dysplasia of the diploë of non-progressive type and a static nature of abnormality.[5–7] Camp and Nash[7] reported 119 cases (80 males and 39 females), of which 10 were 30 years or less, one was a 4-year-old child, and another one was a 9-week-old infant. Examining 26 cases (19 females ranging from 54 to 86 years), Epstein[8] concluded that this change is associated with post-menopausal and senile osteoporosis. A further attempt was made to reveal the cause of the disease, its peculiarity of localized susceptibility, and the role of osteoporosis and ageing in the thinning.[9] Investigating only two patients, one a female of 85 years and another a male of 28 years, it was postulated that the thinning may be an acquired and progressive disease (progression was diagnosed in the female), and the localized thinning is explained in terms of decreased osteoblastic activity resulted from gonadal insufficiency, senility, or other causes of osteoporosis in a region where there is a little stress or strain.[9]

It is apparent that this pathological change, the origin of which has been traced back to ancient civilizations, is not dependent on age or sex, but the exact cause is as yet not understood.—I am, etc.,

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Dr. Thomas Percival and Jane Austen

SIR,—When living in Southampton in 1808 Jane Austen sent a letter to her sister Cassandra in which she mentioned the arrival in the town of a new doctor—"We have got a new physician, a Dr. Percival, the son of a famous Dr. Percival of Manchester, who wrote moral tales for Edward to give to me."[1]

This reference is of particular interest. Dr. Edward Percival was the eldest surviving son of Dr. Thomas Percival, whose book on medical ethics is a classic known to every doctor. Jane Austen was, however, referring to another of Dr. Percival's writings intended for young children. He married in 1767 and as his children were growing up he wrote a series of short tales, each illustrative of one of the moral virtues, so that the series demonstrated the harm produced by selfishness, untruthfulness, and the like. The first part was published in 1775, and later a second and third part were added to it. The *Dictionary of National Biography* states that this book "achieved great popularity," and we gather from Jane Austen's statement that her brother Edward gave her a copy when she was a little girl. The full title of the book was *A Father's Instruction to his Children.* Though we have no doubt that as a child Jane enjoyed reading the tales, we believe that the preface may have had more influence on her precocious mind, for in it are given the reasons why Dr. Percival wrote the book. They were three in number. Firstly, to inspire the young with a love of moral excellence; secondly, to awaken curiosity and to convey in a lively manner knowledge of the works of God; and, thirdly, *to promote more early acquaintance with the use of words and ideas.*