trained. I shared his opinion then, and still do.

It is common knowledge that many patients, who, on doctors' orders, have received months of heat, traction, massage, and exercises from a physiotherapist for a painful neck or back, eventually go off on their own and are put right by a treatment or two from a lay manipulator. Is there any good reason why these simple manoeuvres should not have been carried out at a doctor's request by the physiotherapist?

However, the other aspect of the question must be considered. The effectiveness of the physiotherapist also depends on doctors not sending along unsuitable cases. For example, they should no longer send patients along with a "frozen shoulder" for vain physiotherapy, when a few intra-articular injections of hydrocortisone provide the effective treatment.

The coin has two sides. There would, in my opinion, be plenty enough physiotherapists to go round if on the one hand they were sent only such cases as were likely to respond, and on the other they were taught as students a direct manual approach to all suitable lesions.—I am, etc.,

JAMES CYRIAX.

London W.1.

REFERENCE

1 Watson-Jones, Sir R., *The Times*, 10 July, 1962, p. 11.

Seebohm Sequel

SIR,—I have much sympathy for Dr. G. W. Roberts's concern (28 November, p. 561) over the implementation of the Local Authority Social Service Act. Your leading article should now read "Seebohm Sequelae" for the proposed Misuse of Drugs Bill would replace the Advisory Committee on Drug Addiction with an advisory council which will include one medical practitioner, one dental surgeon, one veterinary surgeon, and one pharmacist as well as somebody else identified with the drug industry, and 14 people with recent and extensive experience of the social aspects of drug addiction. The Bill is primarily concerned with prescribing of drugs of addiction and the treatment and rehabilitation of addicts, which would appear to me to be medical matters.

Once upon a time there were few social workers, but now they have proliferated to such an extent that they are usurping the role of the doctor at all levels. If it were shown that they had the necessary skills and were effective in their allotted tasks one would have to concede that for the sake of progress these changes must be. But there is not a shred of evidence that this is so. Our universities and colleges have been churning out social workers for many years now, and departments of social science compare in size with those of science, engineering, and medicine, but apart from spearheading the attack on our culture and its values they have made precious little impact on the problems of our society; indeed we are faced with a rapid increase rather than a reduction in crime, divorce, illegitimacy, abortion, drug addiction, battered babies, parental and child neglect, and whatever else you may care to mention.

Most social workers are intelligent and educated, but these qualities do not in themselves guarantee that they are effective in their work—which is not entirely surprising for neither have those who teach them shown such competence. Their roles may be defined, but they have not yet been established, and one would have thought before increasing the size of our social services, providing career structures, and giving social workers authority over doctors, one would have investigated what they are capable of doing and what it costs and compared cost and efficiency with alternative methods.

A year ago I decided to experiment and took on two trained nurses who were health visitors in a neighbouring borough. After three months' attendance at our inpatient and day hospital units with a planned course of instruction, they were more competent in dealing with material degrees of psychiatric disability in the community than a recently qualified psychiatric social worker, even though their educational attainments were less. Of even greater importance, they could effectively carry a larger case load.

Seebohm has reinforced a trend which is most questionable, especially as social services now come under one Ministry and their cost will encroach on the financing of the Health Service. It occurs to me that the time is appropriate for a bit of cost accounting. I realize that we in psychiatry are also vulnerable and it may need the Auditor-general to put our own house in order. On the debit side should go the prominent part some psychiatrists have played in encouraging the present trend.—I am, etc.,

MYRE SIM.

Queen Elizabeth Hospital, Birmingham.

Endocrine Function in Homosexuals

SIR,—We have read with interest the communication from Dr. J. A. Loraine and others (14 November, p. 406), but we would question the interpretation of the data on which they base their conclusion that there is a significant endocrine abnormality in homosexuals.

The testosterone and epitestosterone excretion in the three male subjects is compared with that found in a group of normal heterosexual males. Reference to the paper by Cooper *et al.*[1] shows that this normal group consisted of 14 subjects in whom the testosterone values ranged from 35·4-147·4 μg./24 hours and the epitestosterone values from 4·7–52·4 μg./24 hours. The mean values for the individual male homosexuals (testosterone, 39·6, 48·2, and 56·7 μg./24 hours; epitestosterone, 13·6, 16·1, and 24·7 μg./24 hours) all fell within these ranges. It is not clear from either paper how the mean values of 73± 34·3 μg./24 hours for testosterone and 35·4± 19·7 μg./24 hours for epitestosterone were calculated for the normal group, and it is also therefore difficult to understand how the highly significant differences of each individual from the group mean were derived and thereafter to assess the validity of these findings. A similar argument can be advanced about the data presented on the female subjects.

We should also have appreciated rather more extensive clinical data on the female homosexuals, in view of the menstrual irregularity in three of these women in one of whom a tentative diagnosis of the Stein-Leventhal syndrome had been made. Since it has been reported[2] that the plasma testosterone is high in this syndrome, the finding of a high urinary testosterone excretion in Subject 4 may well be a reflection of this clinical situation.

In the past, it has been stated that endocrine factors play no part in the causation of homosexuality,[3] but recent developments have reopened this field of inquiry. More elegant and sensitive assay techniques in endocrinology are now available, in part owing to the important contribution of Dr. Loraine and his group. Recently, too, there has been a series of reports based mainly on animal work of the importance of endocrine factors particularly at critical phases of development in the moulding of sexual behaviour and orientation.[4-6] It is to be welcomed that the Edinburgh group are working in this field, but we cannot accept that the results so far presented show that there is any important endocrine difference between homosexuals and normal subjects.—We are, etc.,

CHRISTINE G. RYRIE.
J. CHRISTIE BROWN.

Bethlem Royal and Maudsley Hospital, London S.E.5.

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Living it up with Concorde

SIR,—Your correspondents R. T. W. L. Conroy (14 November, p. 434) and others (3 October, p. 53) would find a great deal of evidence available to substantiate Squadron Leader A. N. Nicholson and Dr. F. S. Preston's remarks (24 October, p. 242) if they went along to any aviation medicine library and looked at the many recent papers written on the subject and the results of symposia held on flight crew rest activity cycles.

Large scale experiments have been going on for the past few years to study the subject extensively, and Dr. Nicholson and Dr. Preston have been involved very deeply in such work. A lot is already known on the extent and importance of the disturbances encountered.—I am, etc.,

I. C. PERRY.

Department of Aviation Medicine, H.Q. Army Aviation, Stockbridge, Hants.

Continuous Ventilation and Oedema

SIR,—A girl aged 4 years with a history of haematemasis and melaena was admitted to the paediatric intensive care unit because she had aspirated blood and gastric contents. After a short interval she developed cyanosis, dyspnoea, bronchospasm, and tachycardia. Ronchi and rales were heard

and irregular mottled opacities showed in the chest *x*-ray.

Treatment was started with oxygen and bronchotracheal lavage, followed immediately by intermittent positive pressure ventilation with oxygen-enriched air via an oral cuffed Flotex tube. This was followed by antibiotics, bronchodilator drugs, and steroids; the patient was controlled by relaxants and sedatives. Intravenous Intralipid and Aminosol-Fructose-Ethanol was given. On the ninth day of continuous intermittent positive pressure ventilation she developed pitting oedema of the face and extremities. Ascites was present with typical signs of everted umbilicus, shifting dullness, and fluid thrill. Biochemical findings revealed that her serum protein, serum electrolytes, and serum osmolarity were within normal limits. She had a normal electrocardiograph.

The peripheral oedema and ascites were thought to be due to continuous intermittent positive pressure ventilation, as in the case of Dr. J. T. Styles and others (29 August, p. 522) and she was, therefore, subsequently ventilated with a subatmospheric expiratory phase down to -5 cm. H_2O. The treatment was combined with intravenous frusemide. Steroid therapy was stopped as it causes sodium retention. This treatment led to a disappearance of peripheral oedema and ascites. A gross diuresis of two litres of urine was achieved in 24 hours.

The interesting feature of this case lies in the fact that peripheral oedema and ascites developed so rapidly while the child was having intermittent positive pressure ventilation. This treatment has been employed in adult patients with poliomyelitis over much longer periods of time in our special care unit. Peripheral oedema and ascites have not been seen there.—I am, etc.,

K. T. TCHEN.

Department of Anaesthetics,
Saint Mary's General Hospital,
Portsmouth.

Nocturia during Steroid Therapy

SIR,—Your recent leading article entitled Nocturia during Steroid Therapy (24 October, p. 193) prompts me to record some findings in three recent cases of hypertrophic osteoarthropathy associated with lung cancer. All three noted nocturia. None was receiving any therapy. There were no restrictions in diet. All were up and about for 14 to 15 hours each day.

Case 1.—Male aged 57 years with squamous cell carcinoma of right lung. Plasma sodium 135 mEq/1., and plasma potassium 4·2 mEq/1.

Case 2.—Man aged 63 with inoperable squamous cell carcinoma of right lung.

	8a.m. to 8p.m.	8p.m. to 8a.m.
Urine volume (ml.) av. over 6 days ..	580 (545)	1120 (580)
Total Sodium (mEq.)	61 (58)	139 (84)
Total Potassium (mEq.)	31 (24)	34 (27)
S.G.8	1·017 (1·015)	1·012 (1·016)

The figures in parentheses are the values found five weeks after lobectomy for removal of the tumour. Further assessment will be carried out six months after operation.

Plasma sodium 135 mEq/1., and plasma potassium 4·3 mEq/1.

Case 3.—Male aged 63 with squamous cell carcinoma of right lower lobe. Lobectomy performed, 6 November 1970. Plasma sodium 138 mEq/1., and plasma potassium 3·3 mEq/1.

	8a.m. to 8p.m.	8p.m. to 8a.m.
Urine Volume (ml.) av. over 6 days ..	425	1260
Total Sodium (mEq.)	24	117
Total Potassium (mEq.)	22	33
S.G.	1·017	1·009

	7a.m. to 3p.m.	3p.m. to 11p.m.	11p.m. to 7a.m.
Urine Volume (ml.) av. over 5 days ..	340	520	900
Total Sodium (mEq.)	16·5	35	65
Total Potassium (mEq.)	10	15	17
S.G.	1·013	1·013	1·010

It has been suggested that the oedema and increase in blood flow in hypertrophic osteoarthropathy might be the result of inappropriate stimulation of extra-renal volume receptors (7 March, p. 630). Thus, by day when the patients are up and about, this inappropriate stimulation results in an increase in extracellular fluid volume, with the passing of urine low in volume and low in total sodium. At night a counter to this daytime imbalance might be the naturesis and increased water excretion which follows the overdistension of the atria when excess fluid shifts to the thorax in the supine position.

The cause of new periosteal bone formation in osteoarthropathy is obscure. An increased exchangeable calcium pool with high bone accretion rate has been found in a case of osteoarthropathy.[1] Bone contains a significant amount of exchangeable sodium. Could an increased amount of exchangeable sodium in bone be a prime factor in the calcium disturbance?—I am, etc.,

LESLIE DOYLE.

Baguley Hospital,
Manchester.

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1 Joplin, G. F., and Fraser, T. R., *Proceedings of the Royal Society of Medicine*, 1965, **58**, 576.

Phenytoin, Folate, Vitamin B_{12}, and Cyanide

SIR,—There is evidence that folates lower phenytoin serum levels and enhance its metabolism.[1-3] Thus the improvement in mental state and the increase in fit frequency produced by folic acid might be partly mediated through a decrease in tissue levels of phenytoin.[4]

The part played by vitamin B_{12} seems to be similar. Cytochrome P450 has a crucial role in the oxidative detoxication of a drug, but it is competitively inhibited by other drugs and powerfully by cyanide. There is a small metabolically active pool of cyanide present normally, and this is probably formed from the action of thiocyanate oxidase present in red cells on thiocyanate in plasma. There is a reciprocal relationship between the concentration of plasma cyanide and vitamin B_{12}.[5] Hydroxocobalamin is a powerful cyanide antagonist, with the formation of cyanocobalamin. Consequently phenytoin can impair the detoxication of cyanide and vice versa. It is possible that plasma cyanide concentration may tend to rise excessively in vitamin B_{12}-deficient subjects, and some of the neuropsychiatric complications may result from the neuropathic effects of cyanide.[5] However, with deficiency of vitamin B_{12}, N^5 methyl tetrahydrofolic acid cannot be converted back to tetrahydrofolic acid via the vitamin B_{12}-dependent pathway and therefore accumulates in the plasma. This reduces the amounts of folate available for other metabolic pathways and so results in a functional folate deficiency.

It would also be expected that the rate of accumulation of cyanide is a function of thiocyanate oxidase concentration (and therefore red cell mass).[5] The hypothesis fits in with clinical observations. Patients with uncomplicated pernicious anaemia have a lower serum folate concentration than patients with subacute combined degeneration. Severe anaemia conferred "protection" from neurological complications, and it is usually accepted that the adverse neurological effects of treating pernicious anaemia with folic acid occur after the haematological remission.[5] Administration of folic acid lowered serum-vitamin-B_{12} levels in patients on anticonvulsants.[6] Leber's hereditary optic atrophy may result from a defect in the detoxication of cyanide as well as retrobulbar neuritis of vitamin-B_{12} deficiency and tobacco amblyopia.[7] Hydroxocobalamin has been superior to cyanocobalamin in the treatment of these cases[7 8] Remarkably, toxic amblyopia has also been reported from phenytoin.[9] Phenytoin-induced neuropathy has been unresponsive to folic acid supplements. Thus an interference with the cyanide metabolism can be postulated to play a part in the neuropsychiatric side effects of phenytoin in some patients. It is important that treatment with hydroxocobalamin rather than cyanocobalamin is given with folic acid, since cyanocobalamin may be potentially harmful.[8]

Phenytoin has also a direct neurotoxic action. Since the toxic signs of phenytoin include drowsiness, ataxia, giddiness, blurred vision, nystagmus, confusion, and fits—quite like those of bromide, digitalis, and lithium—it is conceivable that they are due to their interference with the action of adenosine triphosphatase-sodium pump and ion fluxes in nerve cells.[4]—I am, etc.,

N. M. A. VIUKARI.

Department of Psychiatry,
University Hospital,
Helsinki 18, Finland.

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