agreement can soon be found, 1974 might well see an even greater disintegration of the mental health services than occurred after the implementation of the Local Authority Social Services Act of 1970 whereby medical and social services have become further separated than ever before. It would be a grave mistake to reduce psychiatric hospital accommodation before an adequate and well integrated community psychiatric service has been established.

The views expressed are my own and do not necessarily reflect those of my employing authorities.

—I am, etc.,

U. P. SEIDEL

London N.W.6

¹ *The Mental Health Services after Unification* London, B.M.A. Publication, 1972.

Cold Drink and Syncope

SIR,—A case is reported of a man who had a syncopal attack on drinking a glass of cold beer.

A 41-year-old Royal Air Force N.C.O., serving in the sultanate of Oman, was drinking a glass of cold beer after a warm afternoon's work. On taking an initial large mouthful, he swallowed, experienced a very severe pain of a "bursting" nature in the lower retrosternal region, and collapsed pulseless to the floor. This was witnessed by a trained nurse. Almost immediately he recovered and felt perfectly fit and well. On further questioning it appeared that he had suffered similar episodes on three occasions since the age of 17 years and always when drinking cold liquids quickly.

On examination after five minutes his colour was good, and no abnormality was detected. His temperature was 98.4°F (36.9°C); pulse 80/minute, regular, and good volume; blood pressure 125/80; no cardiovascular abnormalities detected and the fundi were normal. Routine investigations were all normal.

Usually syncope is due to an extreme fall in blood pressure, or a slowing or standstill of the heart. The precipitating causes are protean. It is suggested that in this case the mechanism of collapse was as follows. The drinking of the cold fluid led to an acute spasm of the cardia and dilatation of the oesophagus. This caused an increase in vagal tone, thus slowing the heart and producing a sharp fall in cardiac output, sufficient to cause syncope.

The history of drinking cold fluids, followed immediately by a "bursting" lower retrosternal pain, suggests that spasm of the cardia leading to oesophageal distension initiated vagal stimulation. Weiss and Ferris¹ described a case in which a patient with an oesophageal traction diverticulum experienced fainting and cardiac standstill when swallowing. This was reproduced by oesophageal distention and prevented by atropine. In this case there was no evidence of oesophageal diverticulum on barium studies. The history and circumstances make a diagnosis of intermittent heart block unlikely, particularly as the symptoms had only occurred three times in the previous years, and only on rapid ingestion of cold fluids. Also the electrocardiogram was normal, with no QT prolongation as in the cases reported by Gale.²

Although ingestion of cold fluids has

been shown to delay repolarization of the posterior heart wall as demonstrated by E.C.G. changes in normal and abnormal hearts,³ there is no evidence that this is deleterious to the subject. In that series maximal electrocardiographic changes occurred within five minutes of drinking iced water, and subsided completely within minutes. In the case described here there were no T wave changes to suggest a direct action of the cold beer on the myocardium.—I am, etc.,

DAVID J. RAINFORD

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¹ Weiss, S., and Ferris, E. B., *Archives of Internal Medicine*, 1934, 54, 931.

² Gale, G. E., Bosman, C. K., Tucker, R. B. R., and Barlow, J. B., *British Heart Journal*, 1970, 32, 505.

³ Dowling, C. V., and Hellerstein, H. K., *American Heart Journal*, 1951, 41, 58.

St. Stephen's Hospital Group

SIR,—The many tributes paid to Dr. A. G. Signy in your obituary columns (1 July, p. 53; 8 July, p. 118) were richly deserved, both professionally and personally. General practitioners in the Kensington area have a particular debt to him for the pathology service that he developed at St. Stephen's Hospital, and this department still serves us magnificently. Let not my comments now detract from the praise justly due to Gordon Signy and to the department.

But this superb department is in the wrong place: it has contributed greatly to the killing of St. Mary Abbot's Hospital and is within the next few years in danger of being left high and dry as St. Stephen's Hospital is smothered by the new Charing Cross Hospital. St. Stephen's Hospital is on a site of only five acres; it will have no well-defined catchment area, its rebuilding programme has halted in pathetic disillusion and confusion about its status and role for the future, and this in turn has had that same effect upon its staff.

The downgrading of St. Mary Abbot's Hospital to just three principal departments (E.N.T., geriatrics, and psychiatry) is a waste of the greater potential of the site and a tragic waste of a flourishing, friendly hospital which used to serve its area comprehensively and effectively. If only the Group Pathology Laboratory, which was the first building purpose-designed for the group, had been sited at St. Mary Abbot's Hospital, none of this might ever have happened. Come to that, is it really too late to cut the losses and start again?—I am, etc.,

ROBERT LEFEVER

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Payment by Colour

SIR,—I would like to make certain points in regard to advertisements in the *B.M.J.* for medical posts in the Republic of South Africa. An advertisement for a professional post at a teaching hospital or a governmental public health post may sound straightforward. The successful applicant will not, however, necessarily be the best qualified or the most hardworking. The prime consideration is that he should be white.

A successful applicant—for example, for a physician's post at an African hospital—would find he is working alongside African

doctors doing the same work for just over half his salary and coloured doctors doing the same work for two-thirds of his salary. This would be, to say the least, embarrassing for the newly-enrolled British doctor. Perhaps there should be a warning in the "Important Notice" column for him.

Further facts should be taken into account. In the liberal and internationally known medical schools where white, coloured, and African students are admitted only whites are allowed into white wards for teaching. Coloured and Africans are not allowed to mix socially with white students. Even if he came top of his year a non-white would not get a post at his teaching hospital. In the unlikely event of a non-white doctor being in a non-white ward of any hospital with white nurses, he would not be permitted to issue orders to them. Possibly the British medical profession would prefer that a certificate of respectability was not given to such a system by allowing advertisements to appear.—I am, etc.,

M. S. KING

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Facial Palsy in Tetanus

SIR,—Palsy of skeletal muscles in tetanus is unusual. An unusual case of tetanus presenting with facial palsy is reported here.

A male aged 42 years fell from a bicycle and abraded the skin. Eight days later he developed lock-jaw and was admitted to Darbhanga Medical College Hospital where a diagnosis of tetanus was made. The next day he developed stiffness of the neck and the recti abdominis, showed some opisthotonus, and a risus sardonicus was evident on the left side of the face. Apart from the frontal belly of the occipito-frontalis the right side of his face was paralysed. The patient could not close the right eye. No other neurological abnormality was detected. The pulse rate was 120/min, but no other abnormalities were found.

For about a week there was no change in the patient's condition, and he had no muscle spasms. Gradually he began to improve and three weeks after the accident the rigidity of the neck and recti abdominis had disappeared, and the heart rate had returned to normal. Ten days later the facial palsy had recovered.

Local tetanus is a well-recognized entity but facial palsy as one of its manifestations is certainly unusual. The condition is mentioned by Patel¹ but not by Kloetzel² in his study of 84 cases of severe tetanus. The explanation of this condition is difficult. Wright *et al.*³ believe that the toxin is absorbed by the motor end plates and spreads up in the spaces between the nerve fibres. The toxin then excites the anterior horn cells and later diffuses to involve the whole of the central nervous system. This explanation is adequate to explain risus sardonicus but not the facial palsy of the present case. If the toxin acted on the facial nucleus (the cranial homologue of anterior horn cells) the frontal belly of the occipitofrontalis should have been involved.

If it is assumed that the toxin acts on the motor end plates the present case should be easy to explain. It is possible that the toxin ascended up the nerve fibres to reach the C.N.S. and excite various muscles. In the

motor end plates of the lower portion of the face where the injury was situated the concentration of the toxin could have been high enough to produce palsy. The other possibility would be for the toxin to ascend up the nerve fibres in high enough concentration to paralyse the motor end plates while some toxin could be absorbed via the blood stream to reach the C.N.S., and give rise to generalized excitatory features.

Whatever the explanation, facial palsy as a complication of tetanus is a rare occurrence. An intriguing aspect of the case was that the forehead had escaped while the lower portion of the face (including the orbicularis oculi) was affected.—We are, etc.,

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- 1 Patel, J. C., in *Text Book of Medicine*, ed. R. J. Vakil, p. 73, Bombay, Association of Physicians of India, 1969.
- 2 Kloetzel, K., *Journal of the American Medical Association*, 1963, 185, 559.
- 3 Wright, E. A., Morgan, R. S., and Wright, G. P., *Journal of Pathology and Bacteriology*, 1950, 62, 569.

Cryo-immunotherapy

SIR,—The findings of Drs. Geoffrey Taylor and J. L. I. Odili (22 April, p. 183) prompt me to report the treatment of five patients with metastatic adenocarcinoma of the prostate by multiple in situ freezing of their primary prostatic tumour.¹ The clinical responses of these five patients (Cases 1-5) as well as that of one patient (Case 6) receiving only a single freeze are summarized in the Table.

Case	Age	Site of Metastases*	Clinical Response
1	68	4th/5th Cervical Vertebrae	Remission
2	69	Lung	"
3	51	Cervical Lymph Nodes	"
4	67	Left Pelvis/Symphysis	Healing
5	51	Lumbar Spine/Pelvis	Remission
6	66	Lung	"

* Determined by histology or by x-ray.

In addition to the localized destruction of the primary tumour and alleviation of local urinary tract obstruction, subsequent immunological studies of these patients (24 June, p. 767) have suggested, primarily on the basis of indirect evidence, that freezing constitutes an antigenic stimulus of sufficient magnitude that it is able to induce tumour rejection. Perhaps the strongest evidence in support of this concept comes from studies of Neel and others,² who have demonstrated that tumour specific transplantation immunity is consistently greater following in situ tumour eradication by freezing than after complete excision. Of importance here, as we have emphasized,¹ has been the suggestion that immunity is not uniquely due to cryonecrosis but rather results from the prolonged exposure of the host to tumour specific transplantation antigen(s) in situ.^{1,2} The feasibility of this hypothesis is strengthened by observations in patients in which the presence of necrotic malignant tissue, present as a result of incomplete coagulation of tumour of the bowel, resulted in not only disappearance of the tumour, but in many cases of distant metastases³ and by

the more recent findings of Takeda *et al.*⁴ following tumour ligation.—I am, etc.,

R. J. ABLIN

Immunology Section, Renal Unit,
Memorial Hospital and Department of Medicine,
Southern Illinois University School of Medicine,
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- 1 Ablin, R. J., Soanes, W. A., and Gonder, M. J., *Cryobiology*, 1971, 8, 271.
- 2 Neel, H. B., Ketcham, A. S., and Hammond, W. G., *Surgical Forum*, 1970, 21, 120.
- 3 Strauss, A. A., Saphir, O., and Appel, M., *Schweizer medizinische für Wochenschrift*, 1956, 86, 606.
- 4 Takeda, K., Aizawa, M., and Kikuchi, Y., *Gann*, 1966, 57, 221.

Screening for Inherited Metabolic Disease

SIR,—We were interested to read the "Screening for Inherited Metabolic Disease by Plasma Chromatography (Scriber) in a Large City" by Dr. D. N. Raine and others (1 July, p. 7), as we have had considerable experience of this screening technique for the past six years.^{1,2}

Although the details of the Birmingham procedure are very similar to our own, there are two main differences. We are screening up to 75,000 babies a year from an area of between 3,500 and 4,000 square miles. For these two reasons we have to accept specimens by post as well as local transport and very few of our bloods are tested on the day they are taken. This has demanded a pragmatic appraisal of the technique and we have decided to look for six aminoacid disorders—phenylketonuria, histidinaemia, homocystinuria, maple syrup urine disease, tyrosinaemia, and prolineaemia—although we would notice a marked increase of basic aminoacids, alanine, and tryptophan. We do not think we could detect 22 aminoacidopathies using this technique in a comprehensive screening programme.

Inevitably we have more specimen casualties than Dr. Raine, but our overall repeat rate is very similar at 5.5-6.0% over the years. We believe this will be lowered now all the health visitors in the region have experience of the method, and we have made some minor alterations in our laboratory procedures.

We have found a high incidence of prolineaemia (1:2,000) in our initial testing which in the main settles to normal within 8 weeks, but in 18 babies the levels remained high for 6 to 7 months when all settled except for two. We have never seen a persistent pronounced iminoglycinuria. One of these babies, who comes from a poor home background, is a little slow in his development. We wonder whether prolineaemia has been a significant factor in the severely handicapped adult cases where it has been detected, but if it has then this would seem to be a different group from our own cases.

We have pointed out³ that transiently raised levels of the aminoacids were mainly associated with high protein intakes and dropped when these were reduced. Our other cases followed a rise in tyrosine and settled within a few months with evidence suggesting a temporary hepatic upset. One child with homocystinuria has been detected subsequently, but here the methionine level was appreciably higher than in the other cases.

Histidinaemia has been diagnosed on six occasions, though we have found raised levels of histidine more frequently. As we do not carry out secondary staining with Ehrlich's,

because citrullinaemia does not come into our terms of reference and proline is detected after ninhydrin staining, we can stain the entire paper with Pauly's reagent when we suspect a baby to have a raised plasma histidine. This allows us to detect urocanic acid as well as measure histidine and helps us to decide whether to bring the child to hospital or ask for a further blood sample in four weeks time.

Our costing of 10p a test includes postage, stationery, clinical assistance, and telephone costs which are appreciable. Our laboratory charges are very similar to the 5p quoted by Dr. Raine. Our results very briefly are as follows.

Number of bloods tested from October 1965 to June 30 1972, 160,843

Phenylketonuria	=	25
Histidinaemia	=	6
Homocystinuria	=	1
Prolinaemia	=	2
Hyperlipidaemia	=	1

Finally, we fully agree with Dr. Raine and his colleagues that there must be a close link between the clinician, dietitian, and biochemist, who should have full supporting facilities, but equally important is the development of a good and harmonious working arrangement with all the medical officers of health and their staffs.—We are, etc.,

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Willink Biochemical Genetics Laboratory,

I. B. SARDHARWALLA

Royal Manchester Children's Hospital,
Pendlebury, Manchester

- 1 Komrower, G. M., Sardharwalla, I. B., and Bridge, C., *13th International Congress of Paediatrics*, 1971, 1, p. 447.
- 2 Sardharwalla, I. B., Komrower, G. M., Gordon, D. B., and Bridge, C., *Annals of Clinical Biochemistry*, 1972, 9, in press.
- 3 Komrower, G. M., and Robins, A. J., *Archives of Diseases in Childhood*, 1969, 44, 418.

General Practice Records

SIR,—Dr. K. S. Dawes's survey of general practice records (22 July, p. 219) prompts me to ask if a correlation exists between quality of records and quality of care. I would expect such a correlation to be strong. As students and residents we are taught the importance of comprehensive notes. Hospital doctors maintain the practice, but many of us outside hospitals clearly do not, and the reason is clear—few of our colleagues are likely to see and to criticize.

It is relevant to recall the almost hysterical opposition of a few years ago to the notion of merit awards in general practice, an opposition due fundamentally to a disinclination to be compared one with another. I have always believed that a man, his equipment, and his records can produce an accurate assessment of his competence. Judgement by one's peers is largely absent in general practice and merit awards would have provided a strong stimulus to improvement. It is sad that we were not big enough to accept the challenge.—I am, etc.,

DEREK WILKINS

Clanfield, Hants

Streptococcal Sore Throat

SIR,—May I point out to Dr. M. H. Hughes (5 August, p. 349), that my comments on Exogen swabs are contained not only in my thesis but also in a paper entitled "Throat