

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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### 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.<sup>1</sup> 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,<sup>3</sup> 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,<sup>1</sup> 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.<sup>1,2</sup> 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 metastases<sup>3</sup> and by

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

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### 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.<sup>1,2</sup>

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 prolinaemia—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 prolinaemia (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 prolinaemia 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 out<sup>3</sup> 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 citruillinaemia 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, dietician, 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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### 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.,

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### 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