available and these were cleared up within the hour. I have more trouble with obscure pot plants with esoteric names which have been sampled by children than I do with chemicals.—I am, etc.,

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Poisoning by Tricyclic Drugs

SIR,—The treatment of overdose with tricyclic antidepressants has recently been reviewed in *Prescribers' Journal*.¹ To summarize, the article stresses the importance of treatment of any respiratory or circulatory failure. It emphasizes that sympathomimetics must be given with great caution because of the pre-existing danger of cardiac irregularity, and indicates the importance of E.C.G. monitoring until well after apparent recovery. The treatment of convulsions is also considered.

I feel that this article is incomplete insofar as an important, though little appreciated, aspect of the syndrome has been omitted. Severe cases may show a profound metabolic acidosis often with considerable hypokalaemia, both of which further predispose to major cardiac dysrhythmias. Treatment, as expected, should be with alkali and intravenous potassium, often in massive quantities.² Thus it is strongly recommended that frequent electrolyte and blood gas analyses are made in all such cases, so that supportive therapy may be given where indicated.

I write this letter because the aforementioned journal does not publish correspondence.—I am, etc.,

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¹ Matthew, H., *Prescribers' Journal*, 1971, 11, 120.
² Dannert, F., and Rosen, A., *Läkartidningen*, 1967, 64, suppl. 4, p. 80.

Transrectal Prostatic Biopsy

SIR,—We read with interest the article on transrectal prostatic biopsy by Mr. W. F. Hendry and Mr. J. P. Williams (4 December 1971, p. 595). We have been using the Silverman needle for taking prostatic tissue for biopsy by the transrectal route. This we do with the patient in the lithotomy position after giving droperidol 20-30 mg intravenously. The operation can be done as an outpatient procedure and causes minimal discomfort to the patient.

The needle is fixed to the palmar aspect of the gloved right index finger by means of two rubber bands, the end of the needle stopping $\frac{1}{2}$ in (12.7 mm) short of the finger tip. A split P.V.C. tubing covers the needle and finger as they are inserted into the rectum so as to prevent damage to the rectal mucosa. Suspicious areas of the prostate can be felt with the finger tip, the needle is inserted into the parenchyma of the gland, and only then is the harpoon advanced. By this technique tissue for biopsy can be taken from specific points of the prostate under direct finger control.—We are, etc.,

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Psittacosis

SIR,—The statement in your leading article (1 January, p. 1) that "British budgerigars seem rarely to cause human infections" may be true, but it rather distracts attention from the leading role of these common household pets as causes of clinically significant human psittacosis. Among 48 cases diagnosed over 20 years in this laboratory whose probable sources of infection were traced budgerigars were incriminated in 28, pigeons in 10, parrots in two, and miscellaneous animals in eight.¹ Budgerigars also led the field in the analyses of cases in the United Kingdom and in the Republic of Ireland during 1970² and (under the synonym "parakeets") in the U.S.A. during the same year.³

The budgerigars associated with the cases diagnosed here had often been recently purchased and usually, when traceable, were bred in this country. One family outbreak of four cases, one of them severe, was caused by a budgerigar which sickened after being purchased and brought to Glasgow from Newcastle upon Tyne.⁴ The bird originated in a well-managed breeding establishment in the south of England. Occasional deaths had occurred among the breeding stock but no necropsy examinations had been made.

It is apparent that psittacosis is endemic in at least some breeding stocks in Britain, as elsewhere, but causing little disease under well-managed conditions. The latent infection is likely to be activated by the stress of transportation to the dealer. A "spot of antibiotic" in the drinking water is recognized to reduce mortality from infections in recently received consignments of birds, but is unlikely to succeed in eradicating (as opposed to suppressing) activated psittacosis, which may recrudescence after sale of the bird. Attempts to make friends with a recently purchased pet, which may be too unwell to avoid the new owner's close advances, lead easily to mouth-to-beak intimacies well calculated to transmit psittacosis, or other infections for that matter. Common-sense hygienic practices and particular caution in dealing with recently-purchased birds are necessary to minimize the risks of acquiring serious infection from these attractive domestic pets.—I am, etc.,

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¹ Grist, N. R., in *Annual Report on the Work of the Infectious Diseases Hospitals and their Associated Laboratory Services*, ed. T. Anderson. Glasgow Western Regional Hospital Board, 1969.
² *British Medical Journal*, 1971, 2, 539.
³ *Morbidity and Mortality, Weekly Report*, 1971, 20, 295.
⁴ Pinkerton, I. W., in *Annual Report on the Work of the Infectious Diseases Hospitals and their Associated Laboratory Services*, ed. T. Anderson. Glasgow Western Regional Hospital Board, 1965.

SIR,—In your leading article (1 January, p. 1) on psittacosis you drew attention to the risks of exposure to diseased birds and the inhalation of dust from their excreta. A more direct method of human infection is from the bite of a bird.

A woman aged 40 purchased two yellow-backed lorykeets (originating from Malaya), from a pet shop on 30 September 1971. Shortly after purchase the birds appeared to be sick, and they died on 2 and 3 October respectively. One of them bit her on the right forearm on 1 October. Two days later she be-

came ill with fever, shivering, headache, cough, and chest pain. She improved on tetracycline. On 6 October a chest radiograph showed a segmental pneumonia in the left upper lobe. Serial complement fixation tests showed rising titres for psittacosis: 11 October, positive 1/20; 18 October, positive 1/40; 2 December, positive 1/320. She was not seriously ill at any stage and received no further antibiotic treatment after a week. The pneumonia cleared gradually, and by 2 December resolution was complete. The incubation period after infection by inhalation is usually regarded as 7-14 days. The shorter incubation period of two days in this case may be explained by the infected beak of the diseased bird having transferred bedsoniae directly into the blood stream.

Though reports of cases of psittacosis in Britain are relatively uncommon the disease is probably more prevalent than is realized. In a study carried out in one chest clinic in the South Bedfordshire and North Hertfordshire areas during 1954-65 100 cases of bedsonial infection were diagnosed, most occurring in a single outbreak in 1956. The commonest source of infection was the budgerigar.¹ There are some six million budgerigars in Britain and probably one household in every six has one. Prophylactic antibiotic treatment suppresses bedsonial infection but may not completely eradicate it, and shortly after the purchase of the bird the antibiotic effect passes off and the disease flares up.² Though these birds are attractive they may be highly dangerous, especially when recently purchased, and this needs to be more widely recognized by the public. Currently there is no adequate control of the importation and supply of birds to pet shops in Britain, and I consider that a more positive preventative approach is needed.—I am, etc.,

ALEX SAKULA

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¹ Erooga, M. A., in *Some Diseases of Animals Communicable to Man in Britain*, ed. O. Graham-Jones, p. 143. Oxford, Pergamon Press, 1968.
² Bisseru, B., *Diseases of Man Acquired from his Pets*, p. 121. London, Heinemann, 1967.

Leukaemia on Myeloma

SIR,—Dr. I. C. Quirt and others (22 January, p. 248), in their first and last sentences, state that acute leukaemia arose following melphalan therapy. It also followed extensive radiotherapy and an unstated amount of cyclophosphamide.

Surely the implication that the leukaemia resulted from melphalan is unwarranted?—I am, etc.,

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Head Injuries in Children

SIR,—I was disappointed when reading your otherwise excellent leading article on "Head Injuries in Children" (22 January, p. 196) to find no mention of the part played by skull x-rays in the management of this very common but, happily, often trivial injury. Every child who comes to hospital after a head injury has a skull x-ray and when asked about the purpose of this procedure the doctor who ordered it invariably replies "for medicolegal reasons."

In my view routine skull x-rays are not

only unnecessary but may be positively dangerous, and should be carried out only for two reasons. One indication is to provide evidence where none other exists of a head injury having been sustained (the child who has been found unexplainably unconscious or where battering is suspected). The other is to confirm a clinical diagnosis of depressed fracture of more than trivial degree.

The really crucial part of the management of head injuries consists of careful observation in order to detect the onset of complications, haemorrhage, and meningitis. This is just as necessary for patients without a fracture as it is for those who have one, and this is the reason why I regard the taking of a skull x-ray as potentially dangerous. For if on the strength of a negative skull x-ray the patient is sent home and no warning given to the parents, they may have a false impression that all must be well and so be reluctant to associate any untoward symptoms that come on within the next 48 hours with the head injury.

What is needed is a period of careful observation by someone (the parents may be excellently suitable for this) who will notice the onset of untoward symptoms and, having been warned by the doctor to take them seriously in the unlikely event of their occurring, will not hesitate to bring the child back to the family doctor or the casualty department for re-appraisal. The decision whether to admit a child to hospital for observation following a head injury depends not on the presence or absence of a skull fracture but on the quality of the observation which is available in the home, the time of day, and, perhaps, the degree of disturbance of consciousness with which the injury is accompanied.

The lawyers ask for a skull x-ray simply because they have been wrongly led by the medical profession to believe that it is an essential part of the proper management of all head injuries. They would as readily ask for the burning of a candle at the bedside or the letting of a pint of blood if the profession had led them to suppose these things to be equally necessary.

Does the medical profession really believe that this wasteful and often frightening ritual non-investigation is always necessary? If so, let it say so and give convincing reasons for saying so. If not, let it speak out against it so that casualty officers and hospital junior staff can come off this absurd medicolegal hook.—I am, etc.,

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Dahlak Blindness

SIR,—In your correspondence columns you published recently (25 December 1971, p. 811) a description of a corneal disease termed Dahlak blindness. This disease is said to be similar to Labrador keratopathy and Bietti's dystrophy. While surveying eye diseases among the Nama people of South-west Africa during July 1971, I found a similar corneal dystrophy to be present in these people. Fifty per cent of the Nama over 35 years of age show some form of corneal dystrophy. Like Dahlak dystrophy, and like Freedman's and Bietti's dystrophy, climate seems to play a part.

This dystrophy will be reported on in detail.—I am, etc.,

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Childhood Hypoglycaemia

SIR,—Your leading article on "Childhood Hypoglycaemia" (1 January, p. 5) drew attention to the importance of this condition as a possible cause of brain damage. Recently two groups of workers^{1,4} have reported combined physiological, biochemical, and neuropathological studies of profound hypoglycaemia in primates. All animals developing brain damage had experienced blood glucose levels below 20 mg/100 ml for 2-10 hours. However, hypoglycaemia of this severity could last 3-5 hours without producing brain damage. Initially, the brain damage takes the form of "ischaemic cell change" affecting neurones in the neocortex, hippocampus, and cerebellum.⁵ Thus the lesions cytologically and in their pattern of selective vulnerability resemble those seen after cerebral hypoxia or oligoemia. The effects of arterial hypotension or cerebral hypoxia may be additive to those of hypoglycaemia in producing such damage.^{3,4}

These experiments were performed in adult animals and all cases subsequently showing brain damage had experienced periods of profound C.N.S. depression (usually with an isoelectric E.E.G.). Further experiments in newborn animals will be required to resolve the problem of brain damage occurring after symptomless episodes of hypoglycaemia.

Your article referred to the importance of convulsions as a symptom of hypoglycaemia. Less well recognized is the possibility of hypoglycaemia as a secondary feature of prolonged seizures. In children with status epilepticus blood glucose may be normal or elevated at the time of admission to hospital, but may subsequently fall to critical levels. Experimental studies in adolescent primates^{6,7} indicate that hypoglycaemia in status epilepticus may be severe enough to be a contributory cause to the permanent neurological disability commonly seen after prolonged seizures.⁸ It is advisable that children with prolonged seizures, if they are not receiving intravenous glucose, should have serial blood glucose determinations continuing well beyond the end of clinical seizure activity.—I am, etc.,

B. S. MELDRUM

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- 1 Khan, K. J., and Myers, R. E., in *Brain Hypoxia (Clinics in Developmental Medicine, 39/40)*, p. 185. London. Heinemann, 1971.
- 2 Myers, R. E., and Khan, K. J., in *Brain Hypoxia (Clinics in Developmental Medicine, 39/40)*, p. 195. London. Heinemann, 1971.
- 3 Meldrum, B. S., Horton, R. W., and Brierley, J. B., in *Brain Hypoxia (Clinics in Developmental Medicine, 39/40)*, p. 207. London. Heinemann, 1971.
- 4 Brierley, J. B., Brown, A. W., and Meldrum, B. S., in *Brain Hypoxia (Clinics in Developmental Medicine, 39/40)*, p. 225. London. Heinemann, 1971.
- 5 Brierley, J. B., Horton, R. W., and Meldrum, B. S., *Brain Research*, 1971, **25**, 483.
- 6 Brierley, J. B., Horton, R. W., and Meldrum, B. S., *Journal of Physiology*, in press.
- 7 Meldrum, B. S., Horton, R. W., and Brierley, J. B., *Electroencephalography and Clinical Neurophysiology*, in press.
- 8 Aicardi, J., and Chevrie, J. J., *Epilepsia*, 1970, **11**, 187.

Creatinine Clearance and Glomerular Filtration Rate

SIR,—Drs. W. M. Bennett and G. A. Porter (9 October 1971, p. 84) reported that the endogenous creatinine clearance is an accurate measurement of glomerular filtration rate and that the ratio of creatinine clearance to inulin clearance is almost unity. They postulated that the discrepancy between their results and our findings¹ was attributable to the normal values of inulin clearance reported by us being low and to the possibility that our patients were not in a steady state after preclearance water loading.

In 1966² we reported that normal values of inulin clearance were between 105 and 135 ml/min in our renal laboratory. These are similar to the values reported by Homer Smith.³ The values of creatinine clearance and inulin clearance in 37 normal men and women reported by us in 1969¹ were lower than those reported by us in 1966² and by Homer Smith³ because the age range of subjects was extended to between 20 and 68 years. The lower mean value for glomerular filtration rate reflects the lower values known to occur in normal older subjects.^{4,5} Our patients were in steady states after preclearance water loading as demonstrated by three 20-minute consecutive inulin and endogenous creatine clearances, which were within 8%.

The fact that endogenous creatinine clearance overestimates actual glomerular filtration rate is reported not only by us but also by numerous other investigators, including Berlyne and colleagues.⁶ Reubi,⁷ in reviewing the literature, also concluded that in large groups of normal subjects the average creatinine/inulin clearance ratio does not differ significantly from unity, but in a given case the ratio is unpredictable and the creatinine clearance cannot be considered a precise measure of the glomerular filtration rate. There is great variability of the creatinine/inulin clearance ratio in individual cases. Our data¹ showed that the creatinine/inulin clearance ratio rises with decline of inulin clearance. These results are in agreement with those obtained by Reubi,⁷ who compared endogenous creatinine clearance with glomerular filtration rate measured by the clearance of thiosulphate.

A careful analysis of Bennett and Porter's data reveals a wide range of endogenous creatinine clearance at a given value of inulin clearance despite a linear correlation between inulin clearance and creatinine clearance. According to the authors' Figure 1, for an inulin clearance of about 100 ml/min endogenous creatinine clearances ranged from 75 to 130 ml/min, and at an inulin clearance of about 40 ml/min endogenous creatinine clearance ranged from 15 to 50 ml/min. Therefore we conclude, as we did in 1969,¹ that a single determination of endogenous creatinine clearance is unpredictable and cannot be considered a precise measure of the glomerular filtration rate, because of a wide range of endogenous creatinine clearance at a given value of inulin clearance. However, in serial determinations of endogenous creatinine clearance an excellent correlation was found between the rate of decline or increase in inulin and creatinine clearance. Therefore, endogenous creatinine clearance is very useful in following the direction of change of glomerular filtration rate during the course of renal disease.