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 oligaemia. 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 imprtance 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.,

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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 findings[1] 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 1966[2] 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 1969[1] were lower than those reported by us in 1966[2] and by Homer Smith[3] 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 data[1] 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.