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Certification of Cot Deaths

SIR,—We wish to correct an impression which may have been given in the article on the "Welfare of Families of Children Found Unexpectedly Dead" (4 March, p. 612) that in these cases the diagnosis of "cot death" or "sudden unexpected death in infancy" should not be given on the death certificate and that the most likely cause should be stated instead. This is not our intent or wish.

We recommend that the information "sudden death" *should* be put on a death certificate but that it be entered as secondary information to that cause of death which the pathologist considers the most likely after all aspects of the case have been considered. Such as:

Example 1—1(*a*) Acute cardiopulmonary failure due to 1(*b*) acute infection of respiratory tract (*Haemophilus influenzae*) (sudden unexpected death in infancy). Such a case was one in which there was pulmonary oedema, but no pneumonia or bronchitis was found on histological examination. There were minimal inflammatory changes in the nasopharynx and *H. influenzae* was grown from the respiratory tract.

Example 2—1(*a*) Oedema of the lung and acute cardiac failure due to 1(*b*) gastroenteritis (organism not identified) (sudden unexpected death in infancy). Such a child was one who had had, perhaps, two loose stools prior to death, no organisms were grown from the gut or respiratory tract, and there were no signs of inflammation in the gut, minimal hypoxic and fatty change in the liver, and some oedema of the lung.

Example 3—1(*a*) Oedema of the brain with convulsions due to 1(*b*) gastroenteritis (sudden unexpected death in infancy). This child had had a possible convulsion and vomited once the day before death. The brain was swollen and the only other finding, apart from some oedema of the lung, was a small amount of fatty change in the liver.

Example 4—1(*a*) Acute laryngotracheitis due to 1(*b*) acute infection of the respiratory tract (organism not identified) (sudden unexpected death in infancy). This child had some symptoms of a cold and histological examination showed a small amount of infiltration of the mucosa of the trachea and a small amount of oedema of the lung but no more than is likely to have been found or have occurred in a child who could well have recovered from the conditions. There was respiratory tract infection in the family.

If this registration practice is used, and some have been using it in recent years, the deaths will be primarily allotted to the likely underlying disease group (acute infection, bronchiolitis, gastroenteritis, etc.) but it will be possible to extract and analyse this whole group of unexpected child deaths in their own right. In this way it will be possible for the Office of Population Censuses and Surveys to study the incidence and the different underlying causes of this distressing syndrome.—We are, etc.,

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Dangers of Diazoxide

SIR,—With reference to the comments of Dr. M. S. Knapp and his colleagues (28 October, p. 229) concerning the dangers of diazoxide, we have now been using diazoxide as a hypotensive agent in labour for two years. Initially the drug was compared with intravenous bethanidine[1] and found to be more rapid and more predictable in its hypotensive action. Bethanidine, like other adrenergic neurone blocking agents, can cause a paradoxical rise in the blood pressure, whereas this is not a feature of diazoxide.

Hyperglycaemia in the neonate was not a problem. Dr. Knapp refers to the paper by Milner and Chouksey,[2] who describe the complication of alopecia in the neonate when diazoxide was used in pregnancy. In the four patients reported the drug was administered orally for 19-69 days and in doses up to 800 mg/day. The dose delivered to the fetus under these conditions can therefore be high. Two of these patients were diabetics. I must again stress that diazoxide in our series was used only in labour and not for long-term therapy. We have now used the drug intravenously 68 times in 53 patients with severe hypertension in labour. In only two cases was a total of three injections (900 mg) necessary and during a period of less than 24 hours. There was no evidence of congenital abnormalities in the infant, nor was there any perinatal morbidity that could be related to the use of the drug. It is worthy of note that since diazoxide has been in use at this hospital there has not been a case of eclampsia in a booked patient. The paper relating our experience is awaiting publication.

We have not used the drug by the oral route because of the reluctance of the distributors in Australia to release it for the purpose of treatment of hypertension. They have been aware of the potential hazards of prolonged oral treatment with respect to carbohydrate metabolism and fetal complications.

In the report of our initial investigation we have suggested that the use of diazoxide in pregnancy should be avoided in diabetic patients and that it should not be used on a long-term basis, but only in labour, nor combined with other hypotensive agents. In this way the side effects are minimal and there does not appear to be any adverse effect on the fetus in our experience. With these guide lines the dose of the drug used by Milner and Chouksey[2] and Boulos *et al.*[3] will not be reached in labour.

It is contended that provided caution is exercised (as with any effective hypotensive agent used in labour) intravenous diazoxide is a valuable drug in the treatment of hypertensive crises in labour.—I am, etc.,

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Nutritional Rickets in Immigrants

SIR,—In their criticisms of our short report on the biochemical response of Pakistani immigrants with late rickets and osteomalacia to a chupatty-free diet (19 August, p. 446), Drs. S. P. S. Teotia and M. Teotia (14 October, p. 111) make no reference to the detailed studies which we have previously carried out on the pathogenesis of these conditions.[1-3] These specify the biochemical, radiological, and clinical criteria which were used to establish the diagnosis of rickets and osteomalacia. In view of our recently published account of late rickets and osteomalacia in the Glasgow Pakistani community (17 June, p. 677) it was not thought necessary to reiterate these criteria in detail. Previous studies have included investigations to exclude renal disease and malabsorption; no evidence for these conditions has been found.

Serum proteins are estimated routinely with estimations of serum calcium, inorganic phosphorus, and alkaline phosphatase in our laboratory and have invariably been within normal limits. Because of this no correction was applied to serum calcium values. We have previously published an account of serum alkaline phosphatase levels in white Glasgow school children.[4] The levels found in rachitic Pakistani children are in most cases greatly in excess of these or of any published data on normal serum alkaline phosphatase values during adolescence. Drs. Teotia and Teotia imply that dietary deficiency of calcium and vitamin D may play a part in the aetiology of late rickets and osteomalacia in the Glasgow Asiatic community. Detailed dietary studies[2] have shown no evidence for this hypothesis and the nutritional status of Pakistani and Indian immigrants in Glasgow is good. Other workers have confirmed our findings that the vitamin D intakes of Asiatic immigrants are usually similar to those of the indigenous United Kingdom population.[5-7] The suggestion that soft water may be involved in the aetiology of Asiatic rickets and osteomalacia is equally untenable. These conditions have been reported from most major centres of immigrant population in the U.K. and not simply from a soft water area such as Glasgow

We find it difficult to understand Drs. Teotia and Teotia's dismissal of the role of dietary phytate in the aetiology of Asiatic rickets in the U.K. in view of the striking biochemical responses noted in our subjects. Since the only variable in our experiment was that of dietary phytate this would seem to establish a prima facie case for considering this substance as aetiologically significant. The absence of more detailed balance studies does not invalidate the results obtained so far, which confirm those of M. R. Wills and his colleagues.[6] Our data have the additional merit of being obtained without alteration in other environmental or dietary factors of possible aetiological importance. This situation would not obtain if the subjects were transferred to a metabolic unit.

We cannot, of course, speak authoritatively of rickets and osteomalacia occurring in the Indian subcontinent. The case for dietary phytate being involved in the aetiology of these conditions in communities in which unleavened bread is consumed as a main source of cereal is at present unproven, though we find the arguments advanced by J. G. Reinhold[8 9] and by Wills *et al.*[6] persuasive. Clearly, dietary phytate is not the only possible cause of rickets and osteomalacia,