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Dr Taitz comments:

Zerres and Propping suggest a number of alternatives to those we put forward to attempt to explain the number of cases of adult polycystic kidney disease presenting in the newborn period with renal masses, and the apparent paucity of such reported cases in older children. We agree that it is essential for larger groups of 'at risk' children to be followed up from the newborn period into adolescence before a clear explanation will emerge for this phenomenon. Unfortunately, the current literature contains several isolated reports of cystic disease in older children, but few family or population studies which throw much light on this question.

Although we have our own favourite hypothesis at present, we would not wish to give the impression that we think that the various alternatives, including those suggested by the writers can be discounted, quite the contrary. We did consider the proposition that there might be a specific allele, but then one would have expected that the affected parent would have had renal masses from birth. We have now increased the number of offspring of children who have one parent with APKD to over 40 but have still not found an older child with kidney masses. It may be that only by pooling resources from a number of centres will an answer to this intriguing question emerge.

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Transient congenital hypothyroidism

Sir,

Danziger, *et al*[1] report a case of a transient hypothyroidism (seven months) in a girl whose mother used povidone iodine preparation during pregnancy. They emphasise that the newborn infant had hypothyroidism as a result of exposure to iodine used topically by her mother. They base their hypothesis on the fact that the iodine crosses the maternal skin and loads the immature thyroid gland which becomes saturated. Hormonogenesis is suppressed (Wolff Chaikoff process). After the age of 7 months the iodine loading probably stops and the thyroid hormonogenesis returns to normal. The authors did not prove the iodine overload, however, and they do not report the results of measurements of maternal or neonatal total blood iodine concentrations or ioduria. The permeability of skin and the effect of iodine loading vary.[2]

In 1978 I already recommended that 24 hour urinary iodine excretion should be measured in cases of transient hypothyroidism of the newborn.[3] I emphasised that a common and apparently harmless practice—that is, disinfecting the skin with iodine—results in a profound hormonal disorder, which is dangerous for a neonate. Although this is reversible, thyroid disorder induced in the first days or weeks of life is not necessarily without long term effects.[2]

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