

has any relevant symptoms. If asthma is diagnosed this is fully discussed with parent and child and types of treatment outlined. If the diagnosis is new or if treatment administered so far is ineffective such information is passed on (*most politely*) to the family doctor. There is rarely a response. When the child is next seen at school, one is told that: (i) the cough/wheezing is better or; (ii) that it persists with or without treatment, or; (iii) 'my doctor says this isn't asthma'. Thus identification of asthma in school is easy; improving treatment is more difficult.

With regard to the three questions asked by Drs Hill, Britten, and Tattersfield:

1 How well are schools informed about children with asthma? This varies greatly. The question then is who should tell whom? School medical officers do not see children in this area until the term after school admission, furthermore, they are well advised on confidentiality of medical information. It would be confusing and unhelpful if the school doctor were to tell the school that a child has asthma, especially if parent or family doctor is unwilling to accept this diagnosis. Such a problem is not insoluble but takes time and tact.

2 How much access should children have to inhalers? The answer should surely be an individual one depending on many factors such as age, reliability, capability etc. Unfortunately, there have been schools where head-teachers disallow any treatment in school or insist on mothers coming to school to administer it. It is to be hoped there is now a more considerate view.

3 Should teachers be given instructions in managing childhood illness? For asthma this would be generally useful but such advice appears to attract interest mainly from those personally involved with asthma and those teaching the very young.

In conclusion, there is undoubtedly a need for much more widespread education on asthma and your Journal is to be congratulated on having four articles on this subject in the April issue.

Meanwhile, one continues to try to spread the word as 'that school doctor.'

#### References

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## Autosomal dominant polycystic kidney disease in children

Sir,

Taitz *et al* refer to autosomal dominant polycystic kidney disease (ADPKD) in children and the value of the family history in its diagnosis.<sup>1</sup> Their observation is in accordance

with our experience in the cooperative study on cystic kidneys of the *Arbeitsgemeinschaft für Pädiatrische Nephrologie*. We know about 49 children with ADPKD, including cases with 'Potter sequence', as well as children with only slight ultrasonographic changes without any renal impairment. The authors' conclusion, drawn from the bimodal distribution of the age at detection of previously published cases, which postulates two distinctive groups is not without problems. As the authors admit themselves, childhood cases may be underreported. Complete ascertainment of a great number of children at risk is necessary; and rare cases with enlarged kidneys should also be detected. There are at least six possible explanations for early onset, of which Taitz *et al* mention only two (1 and 2):

- 1 Homozygosity for ADPKD is unlikely because, as the authors mention, there are no reports of children with early signs both of whose parents have ADPKD.
- 2 Cases of early manifestation might be due to a specific genotype. The authors favour the hypothesis that children with grossly enlarged kidneys are either compounds for the gene of the autosomal recessive (ARPKD) as well as of the autosomal dominant variety, or that they are heterozygous for both mutations at different loci.
- 3 Early clinical signs could be part of a spectrum of severity, ranging from cases with grossly enlarged kidneys in newborn infants to children who don't show any symptoms until adulthood. Age at onset might then show a unimodal distribution due to a polygenic background, as is known from Huntington's chorea.<sup>2</sup>
- 4 One possibility could be the existence of 'modifying alleles' at the same gene locus<sup>3</sup> to explain rare pedigrees of spinal muscular atrophy.<sup>4</sup> The critical study would be to screen systematically a large number of children at risk for ADPKD, starting at birth, or even prenatally. A bimodal distribution of age at onset would strongly point to the existence of specific genotypes, either due to compounds or double heterozygosity, where the frequency of the second mode would even permit an estimate of the prevalence of the responsible gene(s).
- 5 Although unlikely because of transmission through both affected fathers as well as affected mothers, sex related modifying influences should also be considered as occurs in early onset cases of myotonic dystrophy.
- 6 Rare exogenous factors (infections, nutrition?) should also be considered.

Despite the so far unknown nature of early manifestation of ADPKD in children the observation of familial cases should be taken into account in genetic counselling.

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<sup>4</sup> Becker PE. Atrophia musculorum spinalis pseudomyopathica. Hereditäre neurogene proximale Amyotrophie von Kugelberg und Welander. *Z Mensch Vererb-Konstit Lehre* 1964;37:193-220.

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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*<sup>1</sup> 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.<sup>2</sup>

In 1978 I already recommended that 24 hour urinary iodine excretion should be measured in cases of transient hypothyroidism of the newborn.<sup>3</sup> 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.<sup>2</sup>

### References

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