

Growth hormone for children with Prader–Willi

I have always maintained that paediatricians are very nice people, but we can be autocratic in the way we make decisions. Because of lack of data, many times there is uncertainty about the correct length of therapy or diagnostic test to perform. For example, I have been involved in numerous discussions over the years about the likelihood of herpes encephalitis in young acutely ill, febrile infants. The history and physical examination are consistent with, but do not confirm herpes and for various reasons the diagnostic tests are equivocal. Our consultants almost always recommend 21 days of acyclovir. I have often thought, and at times asked, why not talk with the parents about the various therapeutic options: treat or don't treat; 14 days versus 21 days. The general response from my colleagues is that we should decide what is best, and with some brief explanation, emphasise the serious consequences of the disease, and make a recommendation to the parents to treat for 21 days. Our recommendations, regardless of the condition and diagnostic or therapeutic uncertainty, are always quite similar—we select the most conservative course of action. This is contrast to many conditions in adult medicine in which patient-centred care is championed. Most women with breast cancer and men with prostatic cancer are offered various therapeutic options. Rarely would a physician presume to know what is best for their patient. So how does this discussion relate to growth hormone for children with Prader–Willi syndrome? Clearly there are risks and benefits. Some data suggest that growth hormone can lead to sudden death because of worsening of obstructive sleep apnoea, it may improve or exacerbate scoliosis, and it may improve cognitive functioning. Finally, there may be unspecified, and to date unknown, long-term adverse affects of growth hormone. How do we present these data, with all

their uncertainty, to parents so that they can make a truly informed decision? Or do we just say, in our experience this is what we think is best.

What do we learn from epidemiological studies?

Two recent reports have sparked my interest. In this issue, Sommerfield and colleagues from Scotland tell us that the incidence of infantile hypertrophic pyloric stenosis (IHPS) is declining, and this decrease occurred before the decline in the rates of sudden infant death syndrome following the back-to-sleep campaign. They suggest that the decline in IHPS rates occurred 5–10 years prior to the change in sudden infant death syndrome (SIDS) rates – indicating that the sleep position of infants is not the reason. They dismiss a change in diagnostic coding, arguing it has been unchanged in Scotland over the past two decades. This article is similar to a recent report that indicates a dramatic increase in the number of children diagnosed with coeliac disease in Denmark.¹ Once again, the authors of this paper argue that diagnostic coding is not the principal reason for this increase. If the racial/ethnic mix of a population is stable, it is hard to imagine that genetic shift is the cause for these changes—genetic shift takes centuries to occur. Are these studies important and what do they tell us? Well it depends on the problem and if it is getting better or worse. If it is serious disease, and appears to be increasing, like autism, then the public, scientists and policymakers worry a great deal. Studies are launched and explanations sought. If it is a less serious disorder, and the problem is improving, it gets little attention, like IHPS. Regardless, these studies remind me of the mysteries of medicine. The prevalence of specific diseases often change for reasons that are frequently unclear. In addition, studies of this type can confirm the findings from previous research. This is best exemplified

by a recent paper that suggests breast cancer rates have declined in the US following the precipitous drop in the use of hormone replacement therapy.² My conclusion – following the rates of certain diseases, particularly when we are uncertain about their aetiology, is fascinating science. *See pages 1003 and 1007*

TB in England and Wales

Dr Abubakar and colleagues present a remarkably comprehensive overview of tuberculosis (TB) in England and Wales between 1999 and 2006. Over 3500 cases are detailed, including basic demographic, clinical and microbiological characteristics of the patients. Rates for children born outside the UK were 10 fold greater than those born in the UK (37 per 100 000 versus 2.5 per 100 000). Only about 1 in 4 cases were confirmed by culture. Rates of multi-drug resistant isolates remain low—2.3%. *See page 1017*

And in the tradition of the *BMJ*

We present a whimsical view of life from some of our Aussie friends: “The seven ages of man and woman.” Unfortunately, I am familiar with geriatric orthopaedic surgeons, as I contemplate my third orthopaedic procedure in the past 3 years since I still believe I can engage in sport. *See page 1075*

Season's greetings

So this brings to close my 5th year as Editor-in-Chief. It remains a privilege and honour to serve in this position. As always I wish you and your family good health in the year to come.

References

1. **Olsson C**, Hernell O, Hornell A, *et al*. Difference in celiac disease risk between Swedish birth cohorts suggests an opportunity for primary prevention. *Pediatrics* 2008;**122**:528–34.
2. **Ravdin PM**, Cronin KA, Howlader N, *et al*. The decrease in breast-cancer incidence in 2003 in the United States. *N Eng J Med* 2007;**356**:1670–4.