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Correspondence

Evaluation of a developmental screening system for use by child health nurses

Sir,

Dr Eu has carried out an interesting and useful evaluation of the Woodside developmental screening system,[1] but there seems to be an important error in her interpretation of the results.

The figure given in Table 3 for children passing the screening test is based not on the total number of children who were passed as normal in the study population (397) but on the sample of 77 (approximately 20%) who were tested with the Griffiths scales. If we assume that this sample was a fair reflection of the total population of 'normal' children, then of the 397 assessed as normal on the screening test, the Griffiths would be expected to identify 26 as abnormal and 371 as normal. When these figures are entered in Table 3 the sensitivity looks less impressive. In this study the screening system in fact identifies only half of the children who would be defined as having developmental problems by the Griffiths scales.

Table *Table 3 (Eu[1]) with modification ('doubtful' cases omitted as in original)*

Woodside	*Griffiths*		*Total*
	Abnormal	*Normal*	
Abnormal	22	5	27
Normal	26	371	397
Total	48	376	424

Sensitivity 22/48=0·46. Specificity 371/376=0·99.

The problem of low sensitivity might well be intrinsic to the whole concept of developmental screening 'and does not necessarily reflect any particular inadequacy of the Woodside system. It will always be difficult to achieve high sensitivity in screening for disorders that are by their nature difficult to define and have an unpredictable natural history.

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Dr Eu comments:

I am grateful for the opportunity to respond to Dr Hall's letter on my evaluation of the Woodside system as used by child health nurses.[1] His assumption that 'the Griffiths would be expected to identify 26 as abnormal and 371 as normal' is quite reasonable on the details given in the paper, but incorrect in point of fact. The 77 children who had been assessed as 'normals' on the screening assessment and were subsequently retested on the diagnostic Griffiths test represented a validation sample and not a norming or standardising sample. They were selected, in retrospect, either from child care centres for convenience or because parents were worried about their 'normal' child: a large proportion was from the lowest socioeconomic group where the prevalence of 'doubtfuls' and 'abnormals' was more than twice that for the other socioeconomic groupings (Table 4[1]). The value of 0·81 presented for sensitivity would thus have been appreciably higher if the validation sample of 77 had in fact been 'a fair reflection of the total population of 'normal' children, and probably only marginally lower if the figures had been adjusted proportionally as suggested by Dr Hall.

Interestingly enough, of the five children who were assessed as 'normal' on the Woodside and abnormal on the Griffiths, two were listed to be further followed up because they failed the hearing or vision assessment, a third child was recorded by the child health nurse as being 'uncooperative', and a fourth as being 'difficult to assess'. Such children should in fact have remained uncategorised—that is, neither as 'normal' or 'abnormal', nor even as 'doubtful'—if the nurse was unable to obtain a satisfactory developmental assessment.

Therefore, though I agree that it would be ideal for the *whole* population sample to undergo both the screening assessment and the diagnostic test, I would differ with Dr Hall's view that 'it will always be difficult to achieve high sensitivity' in developmental screening. Furthermore, I do not believe that developmental disorders are 'difficult to define' or that they have an 'unpredictable natural history'. In three useful longitudinal studies Werner[2] showed a good correlation between early assessment and reassessment after 20 years, Griffiths[3] in the validation of her psychological test found that testing 270 children over periods of between three and 62 months showed that the correlation coefficient for obtaining 'repeatable results' was r=0·77, and most recently Ross *et al*[4] have shown that in 94 children with a birth weight of <1501 g there was 'a significant correspondence between classification of children on the Bayley MD1 at 12 months and on IQ at 3 years (χ^2=40·9, p<0·001)'.

The two major forces in child development are biological and environmental. In my view the biological forces tend to be relatively stable and therefore predictable, and any difficulties that might arise in predicting final outcome tend to result from variations in the environmental forces.

References

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[3] Griffiths RG. *The abilities of young children*. High Wycombe: The Test Agency, 1970.
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Gonadal function after testicular radiation for acute lymphoblastic leukaemia

Sir,

We were interested to read the recent paper by Leiper *et al* about the effect of direct testicular irradiation on Leydig cell function in boys treated for acute lymphoblastic leukaemia.[1] The questions they attempted to answer are important ones. We need to know if Leydig cell vulnerability to radiation damage is age related and if such damage is reversible with time.

In our own study, published early last year, the results showed that six of the seven boys irradiated during prepubertal life had an absent testosterone response to human chorionic gonadotrophin stimulation.[2] Two of the four boys irradiated during puberty had an appropriate basal testosterone concentration, but the testosterone response to human chorionic gonadotrophin stimulation was subnormal in three of the four. Thus we suspect that the Leydig cells of the pubertal testis may be less vulnerable to radiation damage than those of the prepubertal testis. We do not have sufficient data to indicate if age, as distinct from pubertal state, might influence the degree of testicular damage.

We found evidence that severe Leydig cell damage was present irrespective of whether the boys were studied within one year or between three and five years after irradiation, suggesting that recovery is unlikely. We note that in the seven boys studied sequentially by Leiper *et al*,[1] five showed no improvement in Leydig cell function, while two had subnormal peak testosterone concentrations of 4·6 and 6·8 nmol/l (1·33 and 1·96 ng/ml), respectively, after human chorionic gonadotrophin stimulation. The basal testosterone concentration may well have been adequate for pubertal development to occur in these boys, but unless there is a further improvement in Leydig cell function they will both probably require androgen replacement therapy once puberty is completed to allow normal libido and sexual performance and to avoid osteoporosis.

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Drs Leiper, Grant, and Chessells comment:

We welcome the comments of Shalet and Morris-Jones in response to our paper.[1] We agree that it is important to know if Leydig cell vulnerability to radiation is age related and if such damage is reversible with time.

In their paper Shalet *et al* documented severe Leydig cell damage after testicular irradiation. This was irrespective of whether subjects were studied within one year or between three and five years from the time of radiotherapy, suggesting that recovery of function is unlikely.[2] Our findings were similar to this in the majority of boys studied. In two of the seven cases studied sequentially using the human chorionic gonadotrophin test, however, at least partial recovery was clearly shown. The two subjects mentioned were studied two and three years after irradiation, when they were aged 10·9 years (Tanner Stage I) and 10·6 years (Tanner Stage II), respectively. The first boy had a basal testosterone concentration of 1·1 nmol/l (0·31 ng/ml) rising to 4·6 nmol/l after a three day human chorionic gonadotrophin test, while the second had a basal concentration of 0·8 nmol/l (0·23 ng/ml) rising to 6·8 nmol/l. The testosterone responses after human chorionic gonadotrophin stimulation in these two children only six months after irradiation were grossly inadequate.[3] Plasma testosterone rose from a basal concentration of 0·5 nmol/l (0·14 ng/ml) to a peak of 1·7 nmol/l (0·49 ng/ml) in the first boy and from 0·4 to 1·2 nmol/l (0·12 to 0·35 ng/ml) in the second. We acknowledge that there is a possibility that these children may need hormone supplementation in adult life for the reasons stated by Shalet and Morris-Jones in their letter, and vigilant follow up is mandatory.

With our present data on small numbers of subjects it is difficult to be sure that the degree of testicular damage sustained is age related rather than related to pubertal state at the time of radiotherapy. All our subjects were, however, prepubertal at the time of radiation. We are currently attempting to clarify this with further prospective studies.

References

[1] Leiper AD, Grant DB, Chessells JM. Gonadal function after testicular radiation for acute lymphoblastic leukaemia. *Arch Dis Child* 1986;**61**:53–6.
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[3] Leiper AD, Grant DB, Chessells JM. The effect of testicular irradiation on Leydig cell function in prepubertal boys with acute lymphoblastic leukaemia. *Arch Dis Child* 1983;**58**:906–10.

Prophylaxis of febrile convulsions: searching for the best

Sir,

As confirmed by Knudsen in *Archives*[1] and elsewhere,[2] the short term prophylaxis with diazepam seems sufficiently effective, feasible, and advantageous. His considerable effort[1] in codifying the risk of recurrence (age being the most predictive factor, on the basis of natural history, for the longer time span in which the central nervous system is evolving) must be considered as another step toward personalising the prophylaxis.

The temperature of 38·5°C, however, can be a near uncontrollable level of fever, adding an unnecessary extra