LETTERS TO THE EDITOR

The place of computed tomography and lumbar puncture in suspected bacterial meningitis

Editor,—We should like to respond to the recently published annotation on the place of computed tomography and lumbar puncture in suspected bacterial meningitis.1

The table of contraindications to lumbar puncture in the child with suspected acute bacterial meningitis is welcome and we would agree that lumbar puncture should be avoided in these clinical situations. However, we would not accept that a contraindication to lumbar puncture amounts to a specific indication for computed tomography, which seems to be the inference. We would agree that computed tomography is indicated if the differential diagnosis of bacterial meningitis is in any doubt but this applies irrespective of whether lumbar puncture is contraindicated. We think one should endeavour to separate the contraindications to lumbar puncture from the indications for computed tomography.

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Dr Mellor comments:

I have to disagree with Drs Davidson and Carty as I believe that computed tomography is indicated in these children. With the exception of septic shock, all the contraindications to lumbar puncture given in my annotation are clinical features suggestive of raised intracranial pressure in a child with suspected bacterial meningitis. In this situation the diagnosis of bacterial meningitis cannot be confirmed because lumbar puncture is contraindicated. Antibiotic treatment must be given promptly but some doubts have to be entertained about the diagnosis even by the experienced paediatrician. Some of the conditions that may mimic the presentation of bacterial meningitis with raised intracranial pressure (posterior fossa tumours, acute hydrocephalus, cerebral abscess, intracranial bleeding) require early diagnosis for appropriate management. This can be achieved safely by computed tomography.

RAPID DIAGNOSIS OF MALIGNANCY USING FLOW CYTOMETRY

Editor,—The paper by Williamson et al on flow cytometric diagnosis of malignancy illustrated some of the well known values of this aid to diagnosis.1 However, although they did not state directly that their approach provided proof of malignancy, it is worth reminding clinicians that it is usually inappropriate to make the diagnosis of malignancy by immunophenotyping alone.

Others have shown large numbers of CD10, CD19, terminal transferase (Tdt), and HLA-DR positive lymphoid cells in the bone marrows of children with non-malignant disorders such as transient red cell aplasia and thrombocytopenic purpura as well as a range of non-haemopoietic tumours2 which could lead the unwary into making spurious diagnoses of leukaemia. Until large numbers of reactive nodes have been studied it may remain difficult to distinguish them from a greater variety of lymphomas than T cell lymphoma and Hodgkin’s disease. In cases where the diagnosis of non-Hodgkin’s lymphoma (NHL) has been made by other methods, this approach does enable subclassification to be carried out. Demonstration of monoclonal surface immunoglobulin (the diagnostic hallmark of B-NHL) is virtual proof of malignancy, provided light chain restriction is found. This is one of the few circumstances in which immunophenotyping can by itself reveal malignancy in childhood.

Similarly, within the non-haemopoietic tumours this approach has benefits and limitations. UJ13A, so useful in detecting neuroectodermal cells, will also react with Ewing’s sarcoma3 and rhabdomyosarcoma4 as will antibodies to vimentin which also react with lymphoid tumours. If carefully constructed panels are used the results may still help the pathologist/haematologist assign lineage.

These caveats should be appreciated by clinicians who should resist the temptation to rely too heavily on surface marker studies, at the expense of histological and cytological appearances, as an indication of malignancy. I fully support the addition of immunophenotyping of fresh cell suspensions to the battery of currently available diagnostic techniques.

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Prepubertal height velocity references over a wide age range

Editor,—The construction of new, prepubertal height velocity reference values to take account of early and late developers is welcome5 but does not overcome the fundamental problem inherent in using any velocity standards to describe short term growth in an individual child. We showed that even in experienced hands, a 5 year old child estimated to be growing at the 25th centile for velocity, may in fact lie anywhere from the 10th to the 50th centile.

The reference to our work is inaccurate. We did not state that ‘velocity is unstable over time’. We stated that we had found no significant correlation between two consecutive 12 month velocity values, which is not surprising in view of the unavoidable imprecision of height measurement.6 We would like to stress again the serious limitations of using short term velocity to access the growth of a child.

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Drs Rikken and Wit comment:

We fully agree with Dr Voss that the variability of subsequent height velocities seriously limits the diagnostic power of one whole year height velocity in a clinical setting. When stating that height velocity is not very useful, we think that we have accurately, though indeed not literally and somewhat understated, referred to the work of Dr Voss and collaborators. The main purpose of our velocity standards was not to label short term growth in an individual child as good or poor, but rather to supply a tool for analysing growth velocity both before and during treatment in groups of prepubertal children of different ages. We think that in such growth studies height velocity SD score is a useful parameter of the growth response, despite its obvious limitations in terms of accuracy.

Secondary thrombocytosis

Editor,—We read with interest the paper by Vora and Lilleyman on secondary thrombocytosis in children.7 As stated by the authors in their article, several recent studies have focused on the role of interleukin-6 (IL-6) in stimulating platelet production: in particular, thrombocytosis was observed in IL-6 transgenic mice,8 and administration of IL-6 in primates induced bone marrow thrombocytopenia and increased platelet counts.9

One of the disease conditions associated with marked increase in platelet count is systemic onset juvenile chronic arthritis (JCA). We have recently analysed IL-6 concentrations in patients with systemic onset JCA, using a hybridoma growth assay with the murine hybridoma B9, and found significantly increased serum and synovial fluid IL-6 concentrations in patients with active disease.10 Serum IL-6 concentrations were significantly correlated with platelet counts (r = 0.554, p = 0.001). The determination coefficient (r²) for the association of platelet counts with serum IL-6 concentrations implied that approximately 31% of the variability of platelet counts could be explained by serum IL-6 concentrations. Therefore, our data support the hypothesis that increased IL-6 production plays a part in the thrombocytosis observed in patients with systemic onset JCA, and possibly in other inflammatory diseases.

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