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Chemotherapy related pulmonary embolus recognised

Dr Brian M J Cantwell and colleagues (16 July, p 179) reported the association between induction chemotherapy of non-Hodgkin's lymphoma and the development of pulmonary embolus.

Two days after we had read their article one of our patients with a high grade lymphoma who had recently begun chemotherapy was admitted with a history of gradually increasing shortness of breath on exercise which had developed over the preceding three days. There was no associated haemoptysis, chest pain, or calf swelling. His chest radiograph showed bilateral hilar lymphadenopathy due to lymphoma and an electrocardiogram showed no abnormal results. On clinical examination his pulse rate was 84 beats per minute and regular and his respiratory rate 18 breaths per minute. The jugular venous pressure was not raised and heart sounds were normal. He showed no evidence of cardiac failure. Investigations showed that he had a haemoglobin concentration of 102 g/l and a white cell count of $2{\cdot}0\times10^9$/l. Apart from the history of breathlessness, no laboratory or clinical findings supported a diagnosis of pulmonary embolus. Indeed, previous tests of respiratory function had shown a reduced transfer factor of 17, thought to be related to his treatment with bleomycin, which together with the low haemoglobin concentration might have accounted for his shortness of breath. Bearing in mind Dr Cantwell's and colleagues' short report, however, we performed a ventilationperfusion scan urgently. This showed multiple ventilation-perfusion mismatches that were considered to be diagnostic of multiple pulmonary emboli. He was given heparin and subsequently warfarin. He improved noticeably and was able to continue with his chemotherapy. Interestingly, plasma concentrations of protein C and protein S, both important vitamin K dependent anticoagulant and profibrinolytic agents, have been shown to decrease in patients receiving chemotherapy for breast cancer,[1] and this may also be relevant to lymphoma.

We believe that this case exemplifies the value of the clinically oriented short report, the merits of which are to increase awareness of hitherto unrecognised or unappreciated clinical scenarios. Its message can be quickly put into clinical practice, as witnessed by our recent experience.

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Renal transplantation from HBsAg positive donors to HBsAg negative recipients

We would like to add our experience of transplanting kidneys from donors positive for hepatitis B surface antigen to recipients negative for the antigen to the results of Drs M K Chan and W K Chang (20-27 August, p 522). (Hepatitis B surface antigen was determined by enzyme linked immunosorbent assay (ELISA).) We performed three such transplant operations with the protection of hyperimmune gammaglobulin. Two kidneys were from cadaveric donors and one from a living donor. Like Drs Chan and Chang, we encountered no adverse effects. All three patients had functioning grafts and normal liver function and were negative for hepatitis B surface antigen after three years, one year, and four months. We would like, however, to make three points.

Firstly, in areas endemic for hepatitis B the prevalence of naturally occurring immunity is high—for example, in Saudi Arabia the rate of carriers of hepatitis B surface antigen is 8·8% and that of subjects positive for hepatitis B surface antibody is 40-50%.[1] Immune patients are therefore easy to find, and we offer cadaveric kidneys from donors who are positive for hepatitis B surface antigen only to such immune patients. In the event of a patient negative for hepatitis B surface antigen having a living donor who is positive for hepatitis B surface antigen we wait for the immunity to develop in the patient by vaccination before a transplant operation is carried out. In any case the recipient always receives hyperimmune gammaglobulin at the time of the operation, which is similar to what Drs Chan and Chang suggested.

Secondly, to boost the pre-existing immunity of the patients we give a booster dose of vaccine together with the hyperimmune gammaglobulin at the time of the operation.

Thirdly, none of our donors were positive for hepatitis B e antigen, but it is probably safe to use such donors under the circumstances described above because it has been shown that giving hyperimmune gammaglobulin and vaccination to newborn infants born to mothers who were positive for hepatitis B e antigen protected them from developing the disease.[2] The virus load for such infants is probably far greater than that expected from an affected kidney transplant.

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Dyslexia

Sue Fowler and I would be among the first to agree with Professor William Yule (20-27 August, p 501) that dyslexia may have many causes. We do not, however, agree that language disorders are more important than visuomotor deficits in causing reading difficulties.

Bryant and Bradley found in their milestone study that linguistic ability (rhyming and alliteration) at the age of 4 only predicted between 6 and 10% of the variance in reading ability of the same children three years later.[1] Moreover, the children who improved most from their treatment were those who received both training in segmenting spoken words into their constituent sounds and exposure to the shapes of the letters that represent them. These results suggest other factors, such as visuospatial perceptual ability, may be as important as linguistic skill in determining progress in reading.

We have consistently found that about two thirds of children whose reading ability falls more than two standard deviations behind that expected from non-verbal intelligence quotient scores ("dyslexics") have impaired binocular control and a poor visual sense of direction,[2] which may explain their visual reading errors. Contrary to Professor Yule's view we find that few normal readers face this visuomotor disability, even when we choose as controls younger normal children with the same intelligence quotient scores who are already reading as well as the children with dyslexia.[1] The results of this match of reading age suggest that good binocular control is an essential prerequisite for learning to read normally—a not unexpected conclusion when you consider that seeing letters stably and clearly must be as important in reading as matching them with the separate sounds of the spoken word.

We have also shown that children with poor binocular control are significantly worse readers than their peers with good control[1]; we also have results suggesting that impaired binocular control in preschool children predicts their subsequent failure in reading. Bishop *et al*, using the same test as us, also found that children with poor binocular control tended to be poor readers, but they attributed both their visuomotor and reading disabilities to their having a lower intelligence quotient.[3] This argument was circular, however, because they used an intelligence quotient test that itself penalised poor readers.

Finally, we have shown that treatments that improve the binocular control of children with dyslexia, such as monocular occlusion or vergence exercises, often help them to learn to read. Dorothy Bishop has re-analysed some of our results and questions whether they show an effect of treatment. But she employed a measure of reading ability that is much less sensitive than the one we used, particularly over the lower range where children with dyslexia lie. Hence she missed the real reading progress made by children whose binocular control improved. We also have reanalysed these results and confirmed the effectiveness of treatment in improving binocular control, and subsequently reading, even when performance intelligence quotient and initial reading ability have been allowed for.

In conclusion, we believe that there is currently good evidence that impaired binocular control may contribute to many of the reading problems of children with dyslexia. An important part of the management of such children is therefore to identify those who suffer from this disability and to attempt to rectify it. Successful treatment can often greatly alleviate their problems, although it by no means removes their need for linguistic training and good remedial teaching.

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Salt saga continued

Professor J D Swales's editorial (30 July, p 307) represents the Intersalt data as evidence against the salt-hypertension hypothesis. But linear responses in biological end points usually require a logarithmic change in stimulus, as when doses of drugs are doubled or halved. With salt a series of cultures would need to be ranked by sodium excretion on a log scale such as 1-25, 26-50, 51-100, 101-200, and 201-400 mmol/day, with similar numbers in each cell. The Intersalt study treated four cultures with low salt intake as outliers and analysed a clump of others in the range 101-200 mmol/24 h or close to