products in the puerperium is not necessary, patients should receive careful instructions to report excessive vaginal bleeding, so that measure to increase the vWF:Ag and VIII:C can be instituted without delay.

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3 Ramshohe BH, Dasani H, Pearson JF, Davies SV. Obstetric management in von Willebrand's disease: a report of 23 cases and a review of the literature. (In press.)

Haemorrhagic disorders in pregnancy

The guidelines produced by the Haemostasis and Thrombosis Task Force mention the management of congenital platelet disorders. For those patients who do not require platelet transfusions, single donor platelet transfusions are advised.

I have recently treated a 16 year old girl with storage pool deficiency, during labour, by using a single unit of ABO compatible but otherwise unmatched leucocyte depleted single donor platelets (Cobe Spectra Blood Processor) containing a platelet content of 2.8 × 10^11 and a leucocyte content of 0.5 × 10^9.

There was little bleeding after giving birth.

I suggest that such depleted platelets are a practical alternative to infusing single leucocyte depleted donor or type specific platelets when the aim is to reduce the incidence of platelet antigen specific and HLA alloimmunisation. An added advantage is the reduced risk of transmission of cytomegalovirus.

Although DDAVP has been used without complication during labour, cases of maternal water retention precipitating grand mal seizure (after repeated treatment) have been reported.

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Zinc assays in patients with α and β thalassaemia trait

We read with interest the investigations by Tillyer and Tillyer on the interpretation of zinc protoporphyrin (ZPP) assays in patients with alpha and beta thalassaemia trait. We have been using ZPP assays (protoporphyrin, Helena laboratories) as a screen for iron deficiency since July 1991.

The normal range quoted by Tillyer and Tillyer is much wider than we would expect from our own experience, and assume that some of their "normals" with high ZPP values were indeed iron deficient, especially as 25% of subjects were pregnant. Other reasons for an increase in ZPP must also be considered, such as interfering substances and, rarely, lead poisoning. Our 95% confidence range is 30-65, rather than 38-104 μmol/mol haem.

We agree that in thalassaemia trait the ZPP is wider and merges into those ranges found in iron deficiency. It may indicate some impaired iron utilisation in thalassaemia trait. In 58 consecutive patients with β thalassaemia trait (microcytosis + HbA2 > 0.30 SD) ZPP was 63-7 (19.7) μmol/mol haem. A ZPP of > 120 is likely to indicate iron deficiency in thalassaemia trait and we usually request postponing haemoglobinopathy studies until the patient has finished that trial of iron treatment. If the ZPP value is raised out of clinical context, it may be useful to make repeat measurements with washed red cells to remove interfering substances.

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D M L and C R Tillyer comment: The "normals" we studied were not clinically iron deficient: the MCV and MCH of this group corresponded exactly to established reference ranges and all had plasma ferritin values well within the accepted normal range. We found, as stated in the paper, no evidence that ZPP in pregnant women was higher than in non-pregnant women, and exclusion of pregnant women from the group gives exactly the same reference ranges. Pre-washing the red cells, as proposed by Hastka et al lowers all ZPP values by about 15 μmol/mol and so this procedure could not be used without establishing new reference ranges. Furthermore, not only were the drugs causing substantial interference, which is extremely unlikely in the outpatient and general practice population group we studied, but the washing procedure failed to remove completely the interference from some of these drugs. Lead poisoning would also be rare in this adult population.

Labbe et al., using the ProtocFluor system quote a reference range of 30-80 μmol/mol haem. Our upper reference limit is 24 μmol/mol haem higher than this, and Paul and Brumfit's 15 μmol/mol haem is lower. It is difficult, however, to compare reference intervals and manner of selection of subjects or the nature of the frequency distributions, and Paul and Brumfit give us no information on theirs. If iron stores fall below 50 μg/l of ferritin then the ZPP starts to rise. If the proportion of subjects with ferritin concentrations below this differs in the populations studied, the ranges, and particularly the upper reference range, could differ. The populations may not be functionally iron deficient, but can vary in the extent of their iron stores as measured by ferritin. Populations from South Asia, like the one we studied, suffer from a greater prevalence of iron deficiency than those from other more developed American populations, largely for nutritional reasons, and women in general have a greater prevalence of iron deficiency than men. Their storage iron may be within the "normal" range but in many cases is only just above this.

The ZPP value of women was about 13 μmol/mol haem higher than the men, on average, in our study. If separate reference ranges are calculated for men and women, the 95% non-parametric ranges are 37-75 μmol/mol haem (n = 196) and 41-115 μmol/mol haem (n = 381), respectively. On re-examining the data, as we only had male-female pairs matched for ferritin (to within ±2.5%), we found that the 95% non-parametric ranges were 37-79 μmol/mol haem for men and 43-110 μmol/mol haem for non-pregnant women (n = 129). This sex difference is highly significant (p < 0.0005; Mann-Whitney U test) and is not explained by differences in ferritin concentrations. The pooled estimate of the reference range, which consisted of equal numbers of men and women, was 38-102 μmol/mol haem (n = 250), so an uneven mix of the sexes could not account for any significant bias in our reference ranges.

The original purpose of our study was to establish the extent of iron deficiency and prevalence of haemoglobinopathies in our population: It is thought that this study an unselected group of subjects as seen in clinical practice and to assign them to diagnostic groups with reliable criteria which we regularly use. Using these criteria, over 40% of our subjects were classified as iron deficient. Haemoglobinopathy screening must take this into account. We found that the diagnosis of β thalassaemia trait was unaffected by coincident iron deficiency, and we therefore do not delay HbA2 quantitation in its presence. Rapid results are clearly important when screening an antenatal population where the genetic risk is high.

Zinc assays in patients with alpha and beta thalassaemia trait.

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