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

B H RAMSAHOYE
S V DAVIES
H DASANI
Haemophilia Centre, University Hospital of Wales, Heath Park, Cardiff CF4 4XW

J F PEARSON
Department of Obstetrics and Gynaecology, University Hospital of Wales, Heath Park, Cardiff CF4 4XW

1 Walker ID, Walker JJ, Colvin BT, Letsky EA, Rivers R, Stevens R. Investigation and management of haemorrhagic disorders in pregnancy. *J Clin Pathol* 1994;47:100–8.
2 Ramsahoye BH, Davies SV, Dasani H. Management of pregnancy and delivery in von Willebrand's disease. *Blood* 1993; 82(Suppl 1):1509.
3 Ramsahoye 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).
4 Adashi EY. Lack of Improvement in von Willebrand's disease during pregnancy. *N Engl J Med* 1980;**303**:1178.
5 Conti M, Mori D, Conti E, Muggiasca ML, Mannucci PM. Pregnancy in women with different types of von Willebrand's disease. *Obstet Gynaecol* 1986;**68**:282–5.

Haemorrhagic disorders in pregnancy

The guidelines produced by the Haemostasis and Thrombosis Task Force mention the management of congenital platelet disorders.[1] For those patients who do require platelet transfusions, single donor platelets or platelet type specific donor platelets are advised.

I have recently treated a 16 year old girl with storage pool deficiency, during labour, by using a single infusion of ABO compatible but otherwise unmatched leucocyte depleted single donor platelets (Cobe Spectra Blood Processor) containing a platelet content of $2{\cdot}8 \times 10^9$/l and a leucocyte content of $0{\cdot}5 \times 10^6$/l.

There was little bleeding after giving birth.

I suggest that such depleted platelets are a practical alternative to infusing single leucocyte undepleted donor or type specific platelets when the aim is to reduce the incidence of platelet antigen specific and HLA alloimmunisation.[1] An added advantage is the reduced risk of transmission of cytomegalovirus.[2]

Although DDAVP has been used without complication during labour, cases of maternal water retention precipitating grand mal seizure (after repeated treatment) have been reported.[3]

J JIP
Department of Haematology, Bolton General Hospital, Minerva Road, Bolton BL4 0JR

1 Walker ID, Walker JJ, Colvin BT, Letsky EA, Rivers R, Stevens R. Investigation and management of haemorrhagic disorders in pregnancy. *J Clin Pathol* 1994;47:100-8.
2 Klapper EB, Goldfinger D. Leukocyte depleted blood components in Transfusion Medicine. *Clin Lab Med* 1992;12:711–21.
3 Chediak JR, Alban GM, Maxey B. von Willebrand's disease and pregnancy: Management during delivery and outcome of offspring. *Am J Obstet Gynecol* 1986; 155:618–24.

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 triat.[1] We have been using ZPP assays (protofluorimeter, 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 presume 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 β 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 + $HbA_2 \geqslant 3{\cdot}6\%$) our mean (SD) ZPP is 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 received a 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.[2]

B PAUL
D BRUMMITT
Department of Haematology, Rotherham General Hospital, Moorgate Road, Oakwood, Rotherham, South Yorks S60 2UD

1 Tillyer ML, Tillyer CR. Use of red cell distribution width and erythrocyte zinc protoporphyrin in differential diagnosis of alpha and beta thalassaemia and iron deficiency. *J Clin Pathol* 1994;47:205–8.
2 Hastka J, Lasserre JJ, Schwarzbeck A, Strauch M, Hehlmann R. Washing erythrocytes to remove interferents in measurements of ZPP by front face haematofluorometry. *Clin Chem* 1992;38: 2184–9.

Drs 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*[1] lowers all ZPP values by about 15 μmol/mol 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 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*[2], using the ProtoFluor 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 Brumfitt's 15 μmol/mol haem is lower. It is difficult, however, to compare reference ranges without knowing about the number and manner of selection of subjects or the nature of the frequency distributions, and Paul and Brumfitt give us no information on theirs. If iron stores fall below 50 μg/l of ferritin then the ZPP starts to rise.[3] If the proportion of subjects with ferritin concentrations below this differs in the populations studied, the ranges, and particularly the upper references ranges, 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 European and 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-calculating our reference ranges using only 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 ($p < 0{\cdot}0005$; Mann-Whitney U test) and is not explained by differences in ferritin concentrations. The pooled estimate of the reference range from this group, which consisted of equal numbers of men and women, was 38–102 μmol/mol haem (n = 258), so an uneven mix of the sexes could not account for any significant bias in our reference range.

The original purpose of our study was to establish the extent of iron deficiency and prevalence of haemoglobinopathies in our population: It was therefore important to 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 HbA_2 quantitation in its presence. Rapid results are clearly important when screening an antenatal population where the genetic risk is high.

1 Hastka J, Lasserre JJ, Schwarzbeck A, Strauch M, Hehlmann R. Washing erythrocytes to remove interferents in measurements of zinc protoporphyrin by front-face hematofluorometry. *Chem Chem* 1992;38:2184–9.
2 Labbe RF, Rettmer Rl. Measurement of zinc protoporphyrin with the ProtoFluor-Z system. *Clin Chem* 1990;36:702–3.
3 Labbe RF, Rettmer RL. Zinc protoporphyrin: a product of iron-deficient erythropoiesis. *Semin Hematol* 1989;36:40–6.
4 Tillyer ML, Varawalla NY, Tillyer CR, Sandhu P, Modell B. Thalassaemia, abnormal haemoglobins and iron deficiency in a British Asian population. *Clin Lab Haematol* 1993;15:157–64.