Coagulopathy during intraoperative cell salvage in a patient with major obstetric haemorrhage

Editor—Rudra and Basak\(^1\) are to be congratulated on their successful management of major obstetric haemorrhage in a patient with an atonic uterus. The latest CEMACH Report\(^2\) has highlighted atonic uterus as the commonest cause of fatal maternal haemorrhage.

However, we would take issue with the suggestion that intraoperative cell salvage contributed in any way to the coagulopathy. Although the authors do not clearly state the timing of the coagulation results relative to the various transfusions, it would appear that the decrease in haemoglobin to 4.7 g dl\(^{-1}\) with prolonged activated partial thromboplastin time and prothrombin time occurred during brisk haemorrhage before infusion of cell-saved blood, and after transfusion of allogeneic blood. Fresh frozen plasma (FFP) and cryoprecipitate were appropriately given to correct the clotting defect. The total blood loss was 10 litre, with 2200 ml of the total red cell replacement being cell-salvaged blood, and the remaining 9 units being allogeneic blood, accompanied by further FFP, cryoprecipitate, and platelets. The degree of coagulopathy seems entirely in keeping with a 10 litre blood loss, and within the common experience of most obstetric anaesthetists. The authors state that ‘Coagulopathy is a potential complication associated with cell salvage, especially in large volume retransfusion, as the washing process removes platelets and clotting factors, leaving only the red cells suspended in normal saline’. If you replace massive blood loss with cell-saved blood without giving appropriate clotting products, you will indeed get a coagulopathy, exactly as you would if you replaced massive blood loss with just allogeneic red cells without appropriate clotting products, as neither transfusion contains any platelets or clotting factors. This would not be a ‘complication associated with cell salvage’ it would be a complication associated with inappropriate management of haemorrhage—fortunately not a feature in this case. The requirement for clotting factor replacement is entirely dependent on the volume of clotting factors lost and is the same whether allogeneic or salvaged blood is transfused. Older techniques and machines may have allowed some heparin/citrate contamination of the final product, but recent work shows total removal of heparin\(^3\) and no measurable increase in plasma heparin after cell salvage infusion;\(^4\) this is not a current concern. Similarly, older studies on the possible activation of platelets and leucocytes\(^5\) are no longer clinically relevant, as more recent studies have specifically measured cell salvage wash-out of cytokines including: leucotriene B\(_4\); tumour necrosis factor-\(\alpha\); granulocyte colony-stimulating factor; interleukin-6; neutrophil elastase; thrombin–antithrombin complex; prothrombin activation peptide; d-dimer; and tissue plasminogen activator—all of which have been shown to be effectively removed with good final product quality.\(^4 6 7\)

Thankfully, amniotic fluid embolus remains entirely theoretical and has never been demonstrated clinically. The ‘single case’ to which the authors refer\(^8\) was in a letter from Holland in 2000, describing the death of a severely anaemic Jehovah’s Witness with pre-eclampsia and HELLP, who died of a hypoxic cardiac arrest shortly after extubation after emergency Caesarean section, and whose management included the infusion of up to 200 ml of cell-saved blood.\(^9\) This case is not accepted by most authorities as being related to cell salvage.

We fully agree with the authors that ‘prompt detection and early intervention’ remains the key to successful management of coagulopathy associated with major obstetric haemorrhage.

Conflict of interest

None declared.

S. Catling\(^1\)*
S. L. Haynes\(^2\)
\(^1\)Swansea, UK
\(^2\)Manchester, UK
*E-mail: sue.catling@btopenworld.com


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