Case Series

Journal of Veterinary Emergency and Critical Care **22**(3) 2012, pp 355–360
doi: 10.1111/j.1476-4431.2012.00747.x

Autologous blood transfusion following red blood cell salvage for the management of blood loss in 3 dogs with hemoperitoneum

Caroline Hirst, BVetMed, MRCVS and Sophie Adamantos, BVSc, DACVECC, MRCVS

Abstract

Objective – To describe the use of autologous transfusion using a red blood cell salvage device for the management of large volume hemorrhage in 3 dogs with hemoperitoneum.
Case Series Summary – Three dogs were managed for large volume hemorrhage by autologous transfusion of red blood cells after cell salvage. In all cases, blood was salvaged from the abdominal cavity during surgery. The causes of hemorrhage included testicular arterial hemorrhage after castration, hepatic parenchymal hemorrhage following hepatic dissection for intrahepatic portosystemic shunt ligation, and intra-abdominal serosal hemorrhage associated with *Angiostrongylus vasorum* infection. In all cases, autologous transfusion was not associated with any identified complications and contributed to improved cardiovascular stability and packed cell volume.
New or Unique Information Provided – This case series is the first to describe the use of a semiautomated red blood cell salvage system for the clinical management of acute hemorrhage in dogs. This case series provides evidence that this procedure can be used safely and effectively for the management of clinical hemorrhage. On this basis, further veterinary evaluation can be justified.

(J Vet Emerg Crit Care 2012; 22(3): 355–360) doi: 10.1111/j.1476-4431.2012.00747.x

Keywords: autotransfusion, hemoperitoneum, intraoperative bleeding, transfusion medicine

Introduction

Transfusion of blood products can be life saving[1] and its practice in veterinary medicine is increasing worldwide with the growing availability of banked blood supplies. Transfusion reactions, while relatively uncommon, are well recognized and documented in the veterinary literature.[2–5] In people a large number of serious complications are recognized including inadvertent administration of mismatched transfusions,[6] transmission of infectious diseases,[7,8] hemolytic transfusion reactions,[6] immunosuppression,[9–11] transfusion-related acute lung injury,[12] transfusion-related sepsis,[8] and upregulation of systemic inflammation.[13,14] Recent and ongoing research increasingly suggests that there is a correlation between homologous blood transfusion and mortality in human patients.[15–17] A number of recognized biochemical, biomechanical, and oxidative storage lesions such as increased lactate, decreased 2,3-diphosphoglycerate, decreased membrane area, decreased deformability, hemoglobin oxidation, hemoglobin denaturation, and lipid peroxidation[18,19] detected in stored blood products are known to adversely affect red cell lifespan and function and are postulated to contribute significantly to the undesirable side effects of homologous transfusion.[20–22] In order to reduce or avoid homologous transfusions, cell salvage is frequently used in human practice during situations of large volume blood loss.[23]

Cell salvage is a technique by which blood shed intra- or postoperatively is collected, washed, and returned

Abbreviations

aPTT	activated partial thromboplastin time
FFP	fresh frozen plasma
pRBC	packed red blood cell
PT	prothrombin time
TTP	total plasma protein

From the Department of Veterinary Clinical Sciences, The Royal Veterinary College, North Mymms, Hatfield, United Kingdom.

No offprints will be provided by the authors.

The authors declare no conflict of interest.

Address correspondence and reprint requests to
Ms. Sophie Adamantos, Department of Veterinary Clinical Sciences, The Royal Veterinary College, Hawkshead Lane, North Mymms, Hatfield, AL9 7TA, United Kingdom.
Email: sadamantos@rvc.ac.uk
Submitted September 5, 2011; Accepted March 24, 2011.

to the patient as an autologous transfusion. Despite a wealth of information on this method as applied to a number of different clinical situations in the human literature,[24–26] little information exists on its use in veterinary species. This case series describes the use of cell salvage as a technique to reduce homologous transfusion in 3 dogs. To the authors' knowledge, this is the first case series to describe this application of cell salvage in dogs.

Case Summaries

Dog 1

A 1-year-old male neutered Bernese mountain dog weighing 39 kg was referred following diagnosis of hemoperitoneum after routine castration. On presentation, physical examination was consistent with moderate-to-severe hypovolemic shock with a pulse rate 180/min, pale mucous membranes with prolonged capillary refill time, and poor pulse quality with notable pulse deficits. Noninvasive blood pressure measured by the oscillometric method demonstrated a systolic pressure of 119 mm Hg and diastolic pressure of 70 mm Hg. Electrocardiogram confirmed sinus tachycardia. Initial blood work revealed a PCV of 46% (reference interval 38–55%) and refractometric total plasma protein (TPP) of 62 g/L (6.2 g/dL; reference interval 49–71 g/L). Lactate was increased; 4.4 mmol/L (reference interval 0.6–2.5 mmol/L). Coagulation times were within reference intervals: prothrombin time (PT) 16 seconds (reference interval 12–17 s) and activated partial thromboplastin time (aPTT) 48 seconds (reference interval 71–102 s). Ultrasound-guided abdominocentesis yielded a sanguinous fluid with a PCV of 36% and TPP of 56 g/L (5.6 g/dL). An in-house blood smear examination revealed clumps of platelets at the feathered edge, however a buccal mucosal bleeding time was not performed. The dog was stabilized prior to anesthesia with 2 consecutive 12.5 mL/kg crystalloid[a] fluid boluses, totaling 1 L, which resulted in improved pulse quality and blood pressure; systolic 135 mm Hg measured by Doppler sphygmomanometry and a reduction in heart rate to 127 beats/min. The dog was anesthetized for surgery using methadone[b] 0.25 mg/kg IV as a premedicant and IV fentanyl[c] 7.5 μg/kg and midazolam[d] 0.4 mg/kg for anesthesia induction. General anesthesia was maintained with isoflurane[e] in oxygen administered via a circle circuit. During exploratory laparotomy, hemorrhage from both testicular arteries was identified. Ligation successfully resolved the ongoing hemorrhage; 1.2 L of blood was suctioned from the dog's abdominal cavity and was processed through the autotransfusion red cell separator[f] to yield approximately 650 mL of salvaged packed red blood cells (pRBC). The dog was blood typed intraoperatively as DEA 1.1 positive prior to receiving 1 homologous unit; total volume approximately 325 mL of pRBC administered over 1 hour. Three hours following conclusion of the homologous transfusion hemoglobin concentration was 90 g/L (9 g/dL), PCV was 24% and TPP was 38 g/L (3.8 g/dL). Based on these results, the salvaged blood was administered while the dog was recovering from anesthesia over 5.5 hours, resulting in a postautotransfusion hemoglobin concentration of 128 g/L (12.8 g/dL), PCV of 37%, and TPP of 44 g/L (4.4 g/dL). Coagulation times 24 hours after presentation were within reference interval. Additional fluid therapy consisting of 5 mL/kg/h isotonic crystalloid and an additional 5 mL/kg isotonic crystalloid bolus intraoperatively and 2 mL/kg/h postoperatively was used to correct the dog's volume deficit and maintain euhydration due to a poor appetite during the recovery period. A total of 9.7 L isotonic crystalloid[b] was administered over 96 hours during the dog's hospitalization period. The dog developed short runs of ventricular premature contractions during the postoperative period requiring ECG monitoring but made a full recovery and was discharged after 5 days. PCV at the time of discharge was 44% and TPP was 52 g/L (5.2 g/dL), hemoglobin concentration was 132 g/L (13.2 g/dL). No transfusion reactions were recorded either with the homologous or autologous transfusion.

Dog 2

A 2-year-old male entire Samoyed weighing 20 kg was admitted for surgical management of a hepatic arteriovenous malformation and intrahepatic portosystemic shunt. Preoperatively the dog was cardiovascularly stable. Blood work revealed a PCV of 46% (reference interval 38–55%) and TPP of 61 g/L (6.1 g/dL; reference interval 49–71 g/L). Coagulation times (PT and aPTT) were within reference intervals: PT 16 seconds (reference interval 12–17 s) and aPTT 100 seconds (reference interval 72–102 s). The dog was pre-emptively blood typed and several units of type-matched blood were available in anticipation of intraoperative blood loss. The dog received pethidine[g] 5 mg/kg IM as a premedicant and anesthesia was induced with alfaxalone[h] 1.5 mg/kg IV. General anesthesia was maintained with isoflurane in oxygen administered via a circle system in combination with a continuous rate infusion of fentanyl[d] 0.2 μg/kg/min. Partial attenuation of the intrahepatic shunting vessel was achieved by ligation with 3–0 prolene in addition to attenuation of the major arterial supply to the hepatic arteriovenous malformation by means of an encircling 2–0 prolene ligature. The dog became markedly tachycardic (200/min) in the immediate postoperative period. Supplemental pain relief with methadone[c] 0.2 mg/kg did not result in any improvement in heart

rate and an increase in lactate from 2.2 mmol/L (reference interval 0.6–2.5 mmol/L) to 4.5 mmol/L suggested hypoperfusion. An unexpectedly large volume peritoneal effusion was identified ultrasonographically. Hemoperitoneum was confirmed by ultrasound-guided abdominocentesis, which yielded sanguinous fluid with a PCV of 28% and TPP of 25 g/L (2.5 g/dL) compared to peripheral blood with a PCV of 33% and TPP 18 g/L (1.8 g/dL). A 10 mL/kg crystalloid fluid bolus was administered prior to reintroduction of anesthesia. General anesthesia was induced with intravenous fentanyl 5 μg/kg and midazolam 0.5 mg/kg and maintained with isoflurane[f] in oxygen administered via a circle system in combination with a continuous rate infusion of fentanyl. Repeat exploratory laparotomy identified hemorrhage at the site of parenchymal dissection for management of the intrahepatic portosystemic shunt. Control of this hemorrhage proved extremely challenging and was eventually achieved with placement of a pledgeted mattress suture across the site; 1.3 L sanguinous fluid was suctioned from the abdominal cavity. While the salvaged blood was processed through the red cell salvage device, the dog received 2 rapidly bolused homologous pRBC units before reinfusion of the salvaged cells. Additional boluses of 10 mL/kg colloid[i] and 20 mL/kg crystalloid fluid therapy concurrent to an ongoing rate of 20 mL/kg/h crystalloid fluid contributed to an increase in blood pressure measured by means of an arterial catheter placed in the metatarsal artery increased from 50 mm Hg systolic at initiation of the first homologous pRBC transfusion to 120 mm Hg systolic at the start of the autologous transfusion. Systolic blood pressure remained stable above 120 mm Hg throughout the rest of the surgery and recovery period. An approximate volume of 750 mL of autologous blood was autotransfused. Autologous transfusion increased the PCV from 22%, TPP 20 g/L (2.0 g/dL) after 2 homologous units of pRBC to 31%, TPP 34 g/L (3.4 g/dL). The dog received 1 further unit of type-matched pRBC during the second surgery giving a final postoperative PCV of 42%, TPP 38 g/L (3.8 g/dL), and 1 unit the following day due to an 11% drop in PCV from 35% to 24%. A coagulopathy was identified during the second surgery, presumed dilutional in origin, and was managed with 5 units of fresh frozen plasma (FFP) (approximating 30 mL/kg). Coagulation times were confirmed to be acceptable following this: PT 20 seconds, aPTT 120 seconds. No transfusion reactions were identified following transfusion of the homologous or autologous red blood cells. The dog was euthanized 3 days after surgery due hepatic encephalopathy, collapse, hypotension, coagulopathy, suspected portal hypertension, refractory hypoglycemia, and the guarded long-term prognosis.

Dog 3

A 9-year-old male entire Labrador retriever weighing 40 kg was referred following diagnosis of hemoperitoneum and exploratory laparotomy during which the source of hemorrhage could not be identified. At presentation, the dog was showing signs of profound decompensated hypovolemic shock with ongoing hemorrhage from the surgical incision site; the dog was obtunded and had a sinus tachycardia (confirmed by ECG) of 200/min with poor peripheral pulse quality and pale to white mucous membranes with indeterminate capillary refill time. Noninvasive blood pressure measured by Doppler sphygmomanometry was 105 mm Hg. Venous lactate was 3.5 mmol/L (reference interval 0.6–2.5 mmol/L). Initial blood work revealed a PCV of 34% (reference interval 38–55%) and TPP of 68 g/L (6.8 g/dL; reference interval 49–71 g/L). PT and aPTT were slightly prolonged; PT 19 seconds (reference interval 11–17 s) and aPTT 108 seconds (reference interval 72–102 s). Estimated platelet count from blood smear examination was 44×10^9/L (reference interval 150–900 $\times$ 10^9/L). Thoracic radiographs revealed a peripheral alveolar lung pattern consistent with *Angiostrongylus vasorum* infestation. The dog was stabilized with crystalloid fluid boluses totaling 100 mL/kg and colloid boluses totaling 25 mL/kg. One unit of FFP was also administered. During repeat laparotomy, a large volume, approximately 700 mL, of blood was suctioned from the abdominal cavity, which was processed through the autotransfusion red cell separator to provide 320 mL red blood cell suspension which was autotransfused. PCV measured at induction of anesthesia was 14%, TPP 24 g/L (2.4 g/dL). Two units of type-matched autologous pRBC increased this to 25%, TPP 22 g/L (2.2 g/dL) during the surgery. Subsequent postoperative reinfusion of the homologous cells resulted in a small increase in PCV to 27%, TPP 32 g/L (3.2 g/dL), likely due to ongoing blood loss from the surgical wound and into the peritoneal cavity and urinary bladder. The dog received an additional unit of type-matched pRBC 2 days later due to a fall in PCV to 12%, TPP 40 g/L (4.0 g/dL). FFP was administered as needed to attempt to correct a coagulopathy identified postoperatively (PT 45 s, aPTT 169 s). The dog received 5 units of FFP over 12 hours immediately postoperatively. PT was within reference interval (16 s), following this; however, aPTT was still prolonged (134 s), and remained so despite a further 3 units of FFP administered over the following 2 days (aPTT 166 s). Generalized serosal oozing was observed but no definitive source of bleeding was identified during laparotomy. The dog was definitively diagnosed with *A. vasorum* infection several days later with a positive Baermann fecal examination. *A. vosorum* infection has been documented to cause alterations in both primary

and secondary hemostasis but the exact mechanism by which this occurs is, as yet, undetermined.[26–28] The dog made a rapid and full recovery following appropriate anthelmintic treatment with imidocloprid[j] and was discharged after 6 days.

Discusssion

Cell salvage is a technique by which blood shed intra- or postoperatively is collected, the RBCs are washed and then returned to the patient. Less dense elements such as plasma, activated clotting factors, anticoagulants, systemic medications, and complement are all removed as effluent in the centrifugation and washing process. The cases described in this report underwent cell salvage using a semiautomated autotransfusion cell separator system. Using this system, shed blood is suctioned directly from the patient into a reservoir. A dual lumen tube may be used to deliver anticoagulant directly to the site to be mixed immediately with shed blood as it is suctioned or the reservoir may be primed with anticoagulant solution. Both heparin and citrate are acceptable anticoagulants with this technique. In these cases, heparin was used at a dilution of 30,000 units per L of saline and mixed with the salvaged blood via a dual lumen tip suction line. As the majority of the anticoagulant is removed during centrifugation, relative overanticoagulation is tolerated.[29] Sequential controlled aliquot volumes of 125 mL blood are removed from the reservoir and processed into the washing bowl where centrifugation with isotonic saline for approximately 5 minutes removes the unwanted components including plasma, free hemoglobin, cellular fragments, and bacteria[29,30] before the cells are resuspended in normal saline in an infusion bag. Typically in people, the reinfusion solution has a PCV of approximately 60% and this has been shown to be equal or superior to banked blood in terms of red cell osmotic resistance, morphology, pH, and levels of 2,3-diphosphoglycerate.[31,32] There are no current veterinary studies comparing salvaged autologous units of blood to typical stored homolgous units. The washed cells must be reinfused back to the patient within 6 hours as there is no preservative or glucose added to the suspension. A leukoreduction filter incorporated into the readministration line reduces autologous transfusion of activated white cells to the patient.[33,34]

No studies have been performed to establish any benefit or detriment of cell salvage to dogs using this system. In people, a number of advantages have been demonstrated including little to no risk of administration of contaminated or mismatched blood products to the patient,[33] reduced risk of transfusion complications and reduced hospital stay.[33] In addition to this, cost calculations within the human field have confirmed that set up for cell salvage is comparable to the cost of 2 crossmatching procedures making this a financially viable alternative to homologous transfusions.[35] Given the lack of control subjects and the complex nature of the cases reported here, the authors have refrained from commenting on the potential benefits of autologous transfusion to these patients, although it can be assumed that the total demand for homologous transfusions was reduced. All the dogs described here also received homologous red cell transfusion and thus although the requirement was reduced it was not completely removed. The differing etiologies and nonstandardized way in which the need for blood was assessed by clinical impression limits the ability to compare outcome and response to cell salvage in the cases reported here.

The application of cell salvage is most common and most useful when large volume blood loss can be anticipated. In people, such situations include cardiac, orthopedic, obstetric, urologic, vascular, hepatic, and neurological surgeries. In veterinary patients of appropriate size, where hemorrhage of a large enough volume is suspected, this system can be used successfully to manage cases of hemoperitoneum despite differing etiologies. In such cases, preoperative abdominocentesis confirming the diagnosis allows preparation of the device in anticipation of the start of surgery. The authors have also successfully used this system for the retrieval and reinfusion of blood lost into the pleural cavity following cardiac bypass surgery for correction of pulmonic stenosis. This patient had received a number of homologous transfusions prior to the cell salvage procedure. In light of this, it could not be included in this case series as it was not considered a true homologous transfusion due to the presence of previously transfused autologous cells within the salvaged product.

Absolute contraindications to the use of cell salvage include contamination with solutions that would result in hemolysis if transfused, such as hypotonic fluids, hydrogen peroxide, or alcohol.[36] A large number of relative contraindications are stated in the human literature where readministration may harm the patient. In these situations the risk of autologous transfusion is weighed up against the risks of homologous transfusion or alternative volume support[37] on a case-by-case basis. Among these relative contraindications are salvage from bacterially contaminated sites, obstetric procedures, and surgeries involving malignant neoplastic tissue. Current data from the human literature suggest that cell processing followed by passing the autologous blood through a leukoreduction filter prior to transfusion reduces bacterial load to a tolerable level[38,39] and virtually eliminates white blood cells and malignant cells.[40,41] Amniotic fluid[42] and other undesirable contaminants such as fat and bone chips are reliably removed during the

centrifugation process. No veterinary studies have yet been carried out to establish whether the cell salvage technique adequately removes unwanted components such as bacterial contaminants, aberrant endoparasitic larvae, and neoplastic cells from harvested blood although it would seem likely. These relative contraindications were not encountered in the cases reported here; however, in order to further our applications of these systems prospective studies are warranted.

Risks associated with autologous transfusion are mostly related to technique and machinery failure. Suctioning of the blood results in pronounced activation of inflammation.[43] Although washing and centrifugation removes the majority of inflammatory markers, some remain and these may contribute to the postoperative systemic inflammatory response in patients receiving autologous transfusions.[44–46] Inadequate washing resulting in free hemoglobin can lead to renal injury,[47] which has been documented in dogs,[48] although not in association with autologous transfusion methods. The data available for veterinary species are too limited at this time to establish whether these risks also apply to our patients. These complications directly associated with the cell salvage procedure were not identified in the cases reported here; however, it is prudent to expect that similar complications may occur in dogs and further studies are warranted.

Conclusion

The 3 cases reported here demonstrate that an autotransfusion cell separator device can be used successfully in dogs in a number of situations where large volume hemorrhage has occurred. The facility to autotransfuse reduces demand for homologous transfusion products and, in our limited experience, is not associated with significant complications. Veterinary data on the use, complications, contraindications, and risks associated with cell salvage are lacking and further investigations are warranted.

Acknowledgments

The authors would like to acknowledge Professor Daniel Brockman for securing the Electa Autotransfusion Cell Separator as part of the Cardiopulmonary Bypass Programme at the Royal Veterinary College.

Footnotes

[a] Vetivex 11, Dechra, Shropshire, UK.
[b] Physeptone, Martindale, Romford UK.
[c] Sublimaze, Janssen-Cilag, High Wycombe, Bucks, UK.
[d] Hypnovel, Roche, Welwyn Garden City, Herts, UK.
[e] Isoflo, Abbott Animal Health, Maidenhead, Berks, UK.
[f] Electa Autotransfusion Cell Separator, Dideco, Mirandola, Italy.
[g] Pethidine, Dechra, Shropshire, UK.
[h] Alphaxan, Vetoquinol, Buckingham, UK.
[i] Voluven, Fresenius-Kabi, Germany.
[j] Advantage, Bayer plc, Newbury, Berks, UK.

References

1. Hebert PC, Wells G, Tweeddale M. Does transfusion practice affect mortality in critically ill patients? Transfusion Requirements in Critical Care (TRICC) Investigators and the Canadian Critical Care Trials Group. Am J Respir Crit Care Med 1997; 155(5):1618–1623.
2. Prittie JE. Controversies related to red blood cell transfusions in critically ill patients. J Vet Emeg Crit Care 2010; 20(2):167–176.
3. Owens SD, Oakley DA. Transmission of visceral leishmaniasis through blood transfusions from infected English foxhounds to anaemic dogs. J Am Vet Med Assoc 2001; 219(8):1076–1083.
4. Harrell KA, Kristensen AT. Canine transfusion reactions and their management. Vet Clin North Am Small Anim Pract 1995; 25(6):1333–1364.
5. Ralphs C, Jessen C, Lipowitz A. Risk factors for leakage following intestinal anastomosis in dogs and cats: 115 cases (1991–2000). J Am Vet Med Assoc 2003; 223(1):73–77.
6. Eder AF, Chambers LA. Non-infectious complications of blood transfusion. Arch Pathol Lab Med 2007; 131(5):708–718.
7. Kleinman S, Chan P, Robillard P. Risks associated with transfusion of cellular blood components in Canada. Transfus Med Rev 2003; 17(2):120–162.
8. Sandler SG, Yu H, Rassai N. Risks of blood transfusion and their prevention. Clin Adv Hematol Oncol 2003; 1(5):307–313.
9. Bernard A, Meier C, Ward M, et al. Packed red blood cells suppress T-cell proliferation through a process involving cell-cell contact. J Trauma 2010; 69(2): 320–329.
10. Vamvakas E, Blajchman M. Transfusion-related immunomodulation (TRIM): an update. Blood Rev 2007; 21(6):327–348.
11. Vamvakas E. Pneumonia as a complication of blood product transfusion in the critically ill: transfusion-related immunomodulation (TRIM). Crit Care Med 2006; 34(suppl 5):S151–S159.
12. Sokolovic M, Pastores SM. Transfusion therapy and acute lung injury. Expert Rev Respir Med 2010; 4(3): 387–393.
13. Bilgin Y, Brand A. Transfusion-related immunomodulation: a second hit in an inflammatory cascade? Vox Sang 2008; 95(4):261–271.
14. Beale E, Zhu J, Chan L, et al. Blood transfusion in critically injured patients: a prospective study. Injury 2006; 37(5): 455–465.
15. Hebert P, Wells G, Blajchman A, et al. A multicenter, randomized, controlled clinical trial of transfusion requirements in critical care. N Engl J Med 1999; 340(6):409–417.
16. Corwin H, Gettinger A, Pearl R, et al. The CRIT study: anemia and blood transfusion in the critically ill – current practice in the United States. Crit Care Med 2004; 32(1):39–52.
17. Vincent J, Baron J, Reinhart K, et al. Anaemia and blood transfusion in critically ill patients. JAMA 2002; 288(12):1499–1507.
18. Ho J, Sibbald W, Chin-Yee I. Effects of storage on efficacy of red cell transfusion: when is it not safe? Crit Care Med 2003; 31(suppl 12):S687–S697.
19. Chin-Yee I, Arya N, d'Almeida M. The red cell storage lesion and its implication for transfusion. Transfus Sci 1997; 18(3):447–458.
20. Raat N, Ince C. Oxygenating the microcirculation: the perspective from blood transfusion and blood storage. Vox Sang 2007; 93(1):12–18.
21. Gauvin F, Spinella P, Lacroix J. Association between length of storage of red blood cells and multiple organ dysfunction syndrome in pediatric intensive care patients. Transfusion 2010; 50(9): 1902–1913.
22. Tinmouth A, Ferfusson D, Chin Yee I, et al. Clinical consequences of red cell storage in the critically ill. Transfusion 2006; 46(11):2014–2027.
23. The use of autologous blood. The National Blood Resource Education Program Expert Panel. JAMA 1990; 263(3):414–417.

24. Wang G, Bainbridge D, Martin J, et al. The efficacy of an intraoperative cell saver during cardiac surgery: a meta-analysis of randomized trials. Anesth Analg 2009; 109(2):320–330.
25. Wylie S, Stockton E. The use of the cell saver in obstetrics. Br J Hosp Med (Lond) 2005; 66(11):652.
26. Ashworth A, Klein AA. Cell salvage as part of a blood conservation strategy in anaesthesia. Br J Anaesth 2010; 105(4): 401–416.
27. Chapman P, Boag A, Guitian J, Boswood A. *Angiostrongylus vasorum* infection in 23 dogs (1999–2002). J Small Anim Pract 2004; 45(9):435–440.
28. Helm J, Morgan E, Jackson M. Canine *angiostrongylus*: an emerging disease in Europe. J Vet Emerg Crit Care 2010; 20(1):98–109.
29. King D, Borner U, Von Bormann B. Heparin elimination and free haemoglobin following cell separation and washing of autologous blood with Cell Saver 4. Anasth Intensiv Notfallmed 1988; 23(2): 88–90.
30. Bland L, Villarina M, Arduino M. Bacteriologic and endotoxin analysis of salvaged blood used in autologous transfusion during cardiac operations. J Thorac Cardiovasc Surg 1992; 103(3): 582–288.
31. Serrick CJ, Scholz M, Melo A, et al. Quality of red blood cells using autotransfusion devices: a comparative analysis. J Extra Corpor Technol 2003; 35(1):28–34.
32. Kirkpatrick UJ, Adams RA, Lardi A, et al. Rheological properties and function of blood cells in stored bank blood and salvaged blood. Br J Haematol 1998; 101(2): 364–368.
33. Waters JH. Indications and contraindications of cell salvage. Transfusion 2004; 44(suppl 12):S40–S44.
34. Esper S, Waters J. Intra-operative cell salvag: a fresh look at the indications and contra-indications. Blood Transf 2011; 9(2):139–147
35. Savvidou C, Chatziioannou SN, Pilichou A, et al. Efficacy and cost-effectiveness of cell saving blood autotransfusion in adult lumbar fusion. Transfus Med 2009; 19(4):202–206.
36. Waters JH, Sprung J. Errors during intraoperative cell salvage because of inappropriate wash solutions. Anesth Analg 2001; 93(6):1483–1485.
37. Waters JH, Tuohy MJ, Hobson DF, et al. Bacterial reduction by cell salvage washing and leukocyte depletion filtration. Anesthesiology 2003; 99(3):652–655.
38. Timberlake GA, McSwain NE. Autotransfusion of blood contaminated by enteric contents: a potentially life-saving measure in the massively hemorrhaging trauma patient. J Trauma 1988; 28(6):855–857.
39. Waters J, Tuohy M, Hobson D. Bacterial reduction by cell salvage washing and leukocyte depletion filtration. Anaesthesiol 2003; 99(3):652–655.
40. Edelman MJ, Potter P, Mahaffey KG, et al. The potential for reintroduction of tumor cells during intraoperative blood salvage: reduction of risk with use of the RC-400 leukocyte depletion filter. Urology 1996; 47(2):179–181.
41. Perseghin P, Vigano M, Rocco G, et al. Effectiveness of leukocyte filters in reducing tumor cell contamination after intraoperative blood salvage in lung cancer patients. Vox Sang 1997; 72(4):221–224.
42. Potter PS, Waters JH, Burger GA, et al. Application of cell-salvage during cesarean section. Anesthesiology 1999; 90(2):619–621.
43. Vermeijden WJ, Hagennars A, Oeveren WV, et al. Do repeated runs of a cell saver device increase the pro-inflammatory properties of washed blood? Eur J Cardiothorac Electrophysiol 2009; 20:280–283.
44. Gharehbaghian A, Haque KM, Truman C, et al. Effect of autologous salvaged blood on postoperative natural killer cell precursor frequency. Lancet 2004; 363(9414):1025–1030.
45. Lakshminarasimhan K, Wee M. Perioperative cell salvage. Cont Ed Anaes Critical Care & Pain 2010; 10(4):104–108.
46. Sandoval S, Alrawi S, Samee M. A cytokine analysis of the effect of cell saver on blood in coronary bypass surgery. Heart Surg Forum 2001; 4(2):113–117.
47. Klodell CT, Richardson JD, Bergamini TM, et al. Does cell-saver blood administration and free hemoglobin load cause renal dysfunction? Am Surg 2001; 67(1):44–47.
48. Harrison H, Bunting H, Ordway N, et al. The pathogenesis of the renal injury produced in the dog by haemoglobin or methhemoglobin. J Exp Med 1947; 86(4):339–356