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ASSESSING AND REPLENISHING LOST VOLUME

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INTRODUCTION

Classically, shock is defined as a state of inadequate tissue perfusion for the metabolic needs of the patient. This state of inadequate blood flow may manifest clinically as tachycardia, pallor, oliguria, the development of lactic acidosis and altered mental status.

Shock is either hypovolemic, cardiogenic, anaphylactic or cytotoxic. Hypovolemic shock classically associated with postpartum hemorrhage is due to loss of circulating blood volume. Hypotension is often present in severe cases, but is a late sign and is a poor guide to the volume of blood lost, as pregnancy is accompanied by an alteration of cardiovascular physiology and the response to blood loss and its management may differ to the non-pregnant situation. Maternal blood volume increases, total red cell mass also increases but to a lesser extent, systemic vascular resistance is reduced, and cardiac output becomes more dependent on body position.

Massive postpartum hemorrhage accounts for 35% of obstetric admissions to intensive care in the UK[1,2]. These patients demand rapid assessment and judicious replenishment of lost circulating volume, albeit within the context of the compensatory effects of hypovolemic shock and the physiological changes seen in late pregnancy.

PHYSIOLOGY

The normal circulating blood volume for a healthy non-pregnant adult is 70 ml/kg, or 7.5% of body weight. Cardiac output is 4–6 l/min, and the non-pregnant adult systemic vascular resistance is 10–15 mmHg/l/min (900–1200 dyne.s/cm[5]). Maternal blood volume increases during pregnancy to 40% above baseline by the 30th week, with an accompanying but smaller (20–30%) increase in red cell volume. Cardiac output increases to 50% above pre-pregnancy levels by the 24th week. Systemic blood pressure is more variable in healthy uncomplicated pregnancy, with a small fall in the first and second trimesters, but a return to pre-pregnancy levels by the third. Resting heart rate increases progressively in the first and second trimesters to 15–20 beats per minute above pre-pregnant levels. In addition to these changes, other changes also take place in the autoregulation of intravascular volume and the circulation, both of which affects the body's response to blood loss. Examples include a blunted response to angiotensin II, which may in part be due to an increased production of nitric oxide[3], a decreased tolerance to postural changes and an increased cardiac noradrenaline turnover[4,5].

Circulating volume, clinical signs of hypovolemia and the body's ability to compensate for volume loss are also all affected by pregnancy-related diseases and their treatment, the effects of which continue on into the early postpartum period. Pre-eclampsia, for example, causes a contracted effective arterial blood volume compared with the normal peripartum state. Vascular reactivity is increased, and widely used drugs such as hydralazine and magnesium compromise the body's ability to produce compensatory vasoconstriction in the face of hemorrhage. Indeed, it appears that there is a failure to increase plasma volume and reduce systemic vascular resistance in pre-eclampsia, due to inadequate trophoblastic invasion into the spiral arteries of the uterus[5]. Pre-eclamptic patients thus have an increased tendency to develop pulmonary edema during volume replacement due to many factors, including increased capillary permeability, hypoalbuminemia and left ventricular dysfunction[6].

Normal delivery results in predictable losses of 300–500 ml blood volume for vaginal deliveries and 750–1000 ml for Cesarean section births (see Chapter 4). However, in addition to blood lost from the body, a substantial amount of blood is also redirected into the systemic circulation, often referred to as the autotransfusion effect. This results in an increase in cardiac output by as much as 80%. The effect persists in uncomplicated patients, gradually returning to non-pregnant levels at 2–3 weeks[5].

ASSESSMENT OF CIRCULATING BLOOD VOLUME

Young healthy adults can compensate for the loss of large volumes from the circulation with few obvious external signs. Accurate assessment of blood loss can be difficult for the experienced as well as the inexperienced examiner, as described in Chapter 4.

In cases of hemorrhage symptoms often precede signs. These include unexplained anxiety and restlessness, the feeling of breathlessness (with or without an increased respiratory rate), and a sensation of being cold or generally unwell. For healthy, non-pregnant adults, hypovolemia and associated signs can be divided into four stages (Table 1). These range from the largely undetectable stage 1 with less than 15% loss of volume, to the severe life-threatening stage when more than 40% has been lost. Unfortunately, comparable tables for early and late pregnancy and the immediate postpartum period have not been compiled, but the signs follow a similar pattern.

The most important principle in the treatment of postpartum hemorrhage is early recognition and prompt correction of lost circulating volume, together with simultaneous medical and/or surgical intervention to prevent further loss. Early recognition of life-threatening physiological derangements can be improved by the use of early-warning scoring systems.

Recording physiological observations at regular intervals has long been routine practice in hospitals. Early-warning scores derived from simple routine physiological recordings can identify patients with greater risk of critical illness and mortality. Such scores can be used to flag the early but sometimes subtle signs of concealed but largely compensated hemorrhage in the early postpartum patient and have been recently recommended for use by the Confidential Enquiry into Maternal and Child Health report of 2004[7]. These scores use the physiological parameters most likely to detect impending life-threatening compromise. These usually comprise respiratory rate, heart rate, systolic blood pressure, temperature and mental awareness. Each variable is assigned a weighted score and the total score is the sum of these. This allows a trigger value for ward staff to call for assistance from intensive care or other senior staff. Such systems have been shown to be reproducible and effective at predicting the likelihood of progressing on to critical illness. They are well suited to the early detection of the

Table 1 Stages of shock

Classification	Class 1	Class 2	Class 3	Class 4
Blood loss (% volume lost)	10–15%	15–30%	30–40%	> 40%
Conscious state	Alert, mild thirst	anxious and restless	agitated or confused	drowsy, confused or unconscious
Respiratory rate	normal	mildly elevated	raised	raised
Complexion	normal	pale	pale	marked pallor or gray
Extremities	normal	cool	pale and cool	cold
Capillary refill	normal	slow (> 2 s)	slow (> 2 s)	minimal or absent
Pulse rate	normal	normal	elevated	fast but thready
Systolic blood pressure	normal	normal	normal or slightly low	hypotensive
Urine output	normal	reduced	reduced	oligoanuric

Modified from Baskett PJF. ABC of major trauma. Management of hypovolaemic shock. *BMJ* 1990;300: 1453–7

often subtle signs of unappreciated blood loss and can be easily introduced. Altered normal physiology in late pregnancy and the early postpartum period demands that these scores, usually derived from general surgical or medical patients, be modified for this population as shown in Table 2.

Once the possibility of intravascular depletion has been raised, a prompt clinical assessment is urgent, as the clinical condition of the patient can change rapidly. Clinical assessment, in association with non-invasive and invasive monitoring where appropriate, must be made by senior clinicians (if available), with special attention to repeated assessment at frequent intervals to detect the problem as early as possible. If senior clinicians are not available, they should be notified as described in the protocols in Chapters 22 and 50.

Clinical examination is performed simultaneously with incident-related history taking. This history may elicit the more obvious features of shock such as overt blood loss and pain, but may also elicit the more subtle features such as general malaise, anxiety and restlessness, a poorly defined sense of doom and breathlessness. Physical examination is directed to the fundamental areas of vital function, the conscious state and airway protection, the adequacy of respiratory function, oxygenation and circulation. In particular, the following should be assessed and documented:

(1) Early stages of shock are associated with restlessness and agitation, sometimes with a heightened sense of thirst, but these progress to drowsiness when around 30% of blood volume is lost. Loss of consciousness is a very late sign, with significant risk of imminent death.

(2) Tachypnea is an early sign, partly driven initially by the anxiety, but is an independent sign, and the respiratory rate increases with progressive blood loss and will usually exceed 20 breaths/min when 30% of blood volume is lost.

(3) Oxygenation becomes harder to assess clinically as peripheral pallor becomes more marked, and the pulse oximeter becomes less reliable as peripheral perfusion becomes weaker.

(4) A fall in the jugular venous pressure occurs reasonably early, but is partly compensated

Table 2 Modified early obstetric warning system. Reproduced with permission by Dr R Jones, Consultant Anaesthetist, Royal Berkshire Hospital, UK, from unpublished work in progress

				Score			
	3	*2*	*1*	*0*	*1*	*2*	*3*
Respiratory rate (bpm)		< 8		9–18	19–25	26–30	> 30
Pulse rate (bpm)		< 40	40–50	51–100	101–110	111–129	> 129
Systolic blood pressure (mmHg)	< 70	71–80	81–100	101–164	165–200	> 200	
Diastolic blood pressure (mmHg)				< 95	95–104	> 105	
Conscious level	unresponsive	responds to pain	responds to voice	alert	irritated		
Urine hourly (ml/h) or in 24 h	0	< 30 (< 720 ml)	< 45 (< 1000 ml)	> 45 (> 1000 ml)			

Final score = sum of individual scores at any one time
Action:
Score 0 or 1   Repeat observations when appropriate for clinical scenario
Score 2   Inform midwife in charge, repeat in 15 min
Score 3   Inform midwife in charge, obstetric registrar and duty anesthetist
Score ≥ 4   As above but the consultant obstetrician should be informed
Consider informing duty consultant anesthetist and intensive care team

for by a reduction on venous capacitance. However, the jugular veins can be hard to visualize reliably in postpartum women.

(5) A more reliable indication of hypovolemia from the central venous pressure is the poor increase observed following volume administration.

(6) The pulse rate increases after around 15–20% of blood volume has been lost, but this sign can be unreliable as a sinus tachycardia is physiological in late pregnancy and in the early postpartum period.

(7) Capillary refill is slowed after 15% of blood volume is lost and is almost completely absent when 40% of volume is lost.

(8) Blood pressure is well maintained, despite a falling cardiac output and tissue perfusion, until over 30–40% of circulating volume is lost.

MANAGEMENT

When the compensating mechanisms maintaining the blood pressure have been exhausted, the blood pressure can fall dramatically. At this point, shock is advanced and the risk of imminent death is significant. Once significant blood loss has been recognized, volume replacement has begun via large-bore peripheral access, medical therapies have been used and found ineffective, and surgical intervention has been organized, other methods to more accurately assess volume status and adequacy of the circulation should be used to aid clinical assessment.

The first and simplest of these is invasive measurement of central venous pressure. A central venous catheter can be placed in any central vein, but it should be remembered that, in hypovolemia, identification of a central vein may be difficult without the use of ultrasound. The internal jugular vein is the preferred site in this situation, as the femoral vein is relatively inaccessible, and the subclavian route may have a higher risk of complications in late pregnancy and the early postpartum period, especially if inserted under urgent conditions.

The National Institute for Clinical Excellence (NICE) has recently issued guidance stating that cannulation of central veins using two-dimensional ultrasound imaging should be the preferred method, with the evidence strongest for the internal jugular route[8]. In the healthy, non-pregnant adult, the systemic venous capacity is 3–4 liters, or 75% of circulating volume. If the tone of the venous capacitance vessels did not change as volume was lost from the circulation, the central venous pressure would fall quickly and early, with early compromise of the cardiac output. However, as blood volume is lost, the tone in these venous capacitance vessels increases, moving blood centrally, and maintaining central venous pressure.

Confusion surrounding the concept of venous return can be dispelled if it is thought of in terms of right atrial pressure rather than an increased flow of blood to the right atrium. As blood is lost, the volume in the venous capacitance vessels is reduced and the tone in these vessels increases. The central venous pressure falls progressively, but to a lesser degree due to the compensatory increase in this venous tone. Figure 1 shows the relationship between venous capacitance and central venous pressure during acute blood loss and immediate replacement. As blood is lost from the circulation, the patient follows the line A to B (Figure 1). The central venous pressure falls slowly at first, then more steeply as the extent of blood loss increases. As volume is returned to the circulation, the patient will follow first the line B to C and then
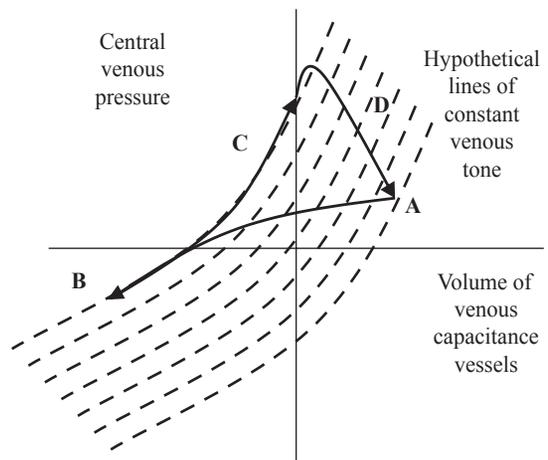

Figure 1 Central venous pressure and venous capacitance during blood loss and replacement. Modified from Bradley RD. *Studies in Acute Heart Failure*. London: Edward Arnold, 1977:11

C to D, rather than simply returning from B to A. This is due to a number of factors and is addressed in a later section in this chapter.

The ability to palpate a peripheral pulse is a good sign, but correlates poorly to any specific arterial pressure. Loss of peripheral pulses is a late preterminal sign of hypovolemic shock. Arterial pressure is most simply measured using a sphygmomanometer. This is familiar to all clinicians and uses a Riva Rocci cuff placed around the upper arm. The correct size of cuff must be chosen, because, if the arm is too large for the chosen cuff, the estimated reading will be falsely high.

Frequently, arterial blood pressure is measured non-invasively by means of an automated version of this technique. Unfortunately, such methods become inaccurate and unreliable when the blood pressure deviates significantly from normal, especially in times of poor peripheral perfusion and hypotension[9], which are the very conditions seen in marked hypovolemia due to postpartum hemorrhage. Automated non-invasive blood pressure devices tend to over-estimate hypotension and under-estimate hypertension. They can repeatedly cycle in an attempt to measure when marked hypotension is present, which can delay its early recognition. Finally, they can sometimes give a totally erroneous reading suggesting an adequate blood pressure when, in reality, the blood pressure is absent or unmeasurable in the face of marked hypovolemic shock, thereby giving a false sense of security[9].

The most reliable method of arterial blood pressure measurement under abnormal or rapidly changing conditions is by means of an intra-arterial cannula and direct measurement via a transducer. This method has the added advantage of providing easy access for blood samples for arterial blood gas analysis and other blood tests. Although such systems commonly used heparinized saline, the use of heparin is unnecessary[10], and the use of plain saline allows samples to be taken for coagulation tests without fear of contamination and erroneous results.

Hemoglobin estimates indicate the concentration of hemoglobin in the sample. The simplicity of this statement only reinforces the point that the concentration of hemoglobin following acute blood loss reflects either medical intervention or the patient's compensation to blood loss. Acute blood loss alone will not change the concentration of hemoglobin in the blood left in the system. The concentration is reduced only when the lost volume is replaced by internal fluid shifts or external fluids are added to the system that are low in hemoglobin. If left untreated, however, acute blood loss results in a fall in hemoglobin concentration after about 4–6 h due to internal compensatory fluid shifts. In contrast, intravenous administration of fluid will dilute the hemoglobin more quickly.

Further information regarding the circulation status can be gained by more invasive or complex techniques. These include the pulmonary artery catheter, pulse contour analysis, and the esophageal Doppler technique. The measurement of cardiac output by means of the thermodilution technique using a pulmonary artery catheter was first described by Bradley and Branthwaite in 1968[11] and subsequently popularized by Swann, Ganz and co-workers[12]. Despite being the gold standard for invasive assessment of cardiac output and left atrial pressure for several decades, the technique has become less favored of late amid gathering evidence that the risks may outweigh the benefits in many circumstances[13]. The benefit of the pulmonary artery catheter is that it provides reliable measurements even in the face of changing body position and is equally effective in the awake conscious patient and the sedated or anesthetized patient receiving artificial ventilation. The risks include those associated with the insertion of a large-bore cannula into a central vein, which is higher risk in the volume-deplete patient suffering massive blood loss, even with the aid of the two-dimensional ultrasound technique. The risks also relate to risks of infection, cardiac arrhythmias, pulmonary artery damage and lung injury. Review of the use of the pulmonary artery catheter in the obstetric population[14] shows that most of the experience has been in patients with pre-eclampsia and eclampsia rather than massive postpartum hemorrhage.

Fortunately, pulse contour analysis is a realistic alternative to the pulmonary artery catheter[15]. The system requires only standard peripheral arterial and central venous cannulae.

A common example of this technique uses cardiac output estimated initially and periodically thereafter by the lithium dilution technique (LiDCO Ltd., Cambridge, UK[16]). The pressure waveform is analyzed using this static cardiac output measurement as a reference to estimate a stroke volume. Changes to the pulse waveform and heart rate from this point are used to estimate stroke volume and cardiac output on a continuous display. Calibration is performed by periodic re-assessment of the cardiac output using the lithium dilution technique. Limitations relate to issues of non-linearity or aortic compliance, how closely the radial arterial pulse waveform resonance relates to the proximal aortic waveform and, therefore, stroke volume, the common problems of damped arterial waveforms, drift between static measurements of cardiac output, and problems associated with poor transmission of pulse waves in severe arrhythmias[17]. Despite these concerns, this technique can provide a continuous idea of cardiac output, systemic blood pressure and vascular resistance in any patient with central venous and peripheral arterial access and is well suited for monitoring the postpartum patient who has undergone massive hemorrhage and is undergoing resuscitation. Other pulse contour analysis systems are also commercially available that use alternative methods for measurement of the reference cardiac output, such as the PiCCO system (Pulsion Medical Systems, Munich, Germany) which employs a transpulmonary thermodilution measurement from an axillary or femoral artery.

REPLENISHING LOST VOLUME

Replacing lost circulating volume should commence as soon as significant bleeding is recognized and, ideally, before the signs of significant hypovolemia have developed. Important initial measures are to simultaneously provide supplemental oxygen, ensure multiple large-bore peripheral intravenous access, undertake an initial rapid clinical assessment and summon senior members of assistance from anesthesia, intensive care, surgical and hematology departments when these individuals are available. Each institution should have a rapid response protocol in place for the management of massive hemorrhage and postpartum hemorrhage in particular. This protocol should be familiar to all, easily accessible and followed (see Chapters 13 and 22).

The principal underlying aim of volume replacement during and following massive postpartum hemorrhage is restoration and maintenance of tissue perfusion to all body organs in order to maintain cellular function and viability. Although the initial focus is on restoration of the common clinical indicators of shock, the clinician must proceed further. Even if all conventionally used criteria resolve, shock may still be present on a cellular, tissue or organ basis[18]. Compensated shock is the term often used to describe the state where conventional hemodynamic parameters have been returned to normal, despite persisting occult tissue hypoperfusion, typically in the splanchnic bed. In spite of adequate volume replacement, patients may develop multiple organ dysfunction with its associated morbidity and mortality.

Replenishing the lost volume must take place simultaneously with control of the bleeding. Medical and surgical attempts to control bleeding must not be delayed by prolonged volume resuscitation in the face of ongoing blood loss. Volume resuscitation should be aimed at restoration of circulating blood volume as well as returning the oxygen-carrying capacity and hemostatic functions to an effective, albeit subnormal, level.

Initial volume replacement enhances right atrial filling and improves cardiac output. As shock develops with blood loss, venous tone increases as described above. Volume administration should be rapid, but titrated to the right atrial filling pressure. Initially, right atrial filling pressure may be restored by a smaller volume than that lost due to the reduced capacity in the venous capacitance vessels. Indeed, immediate rapid re-infusion of the entire volume lost may provoke fluid overload if the tone in the capacitance vessels did not decrease as rapidly. This can be appreciated from Figure 1; here, rapid volume replacement occurs along the line B to C. Central venous pressure rises despite the intravascular compartment remaining depleted. As the venous tone relaxes following resuscitation, further volume administration can occur with a central venous pressure falling toward

normal as the volume in the venous capacitance becomes replete (line C to D). Thus, volume administration should be rapid but infused in discrete volume challenges, with the effect on the right atrial filling pressure, systemic blood pressure and other hemodynamic variables being monitored. Commonly, 250–500 ml of either a crystalloid or a colloid is administered over a period of 10–20 min as the urgency dictates (a patient with life-threatening class 4 shock will receive 2–3 liters more quickly, but, even then, the principles of monitoring the hemodynamic variables during the infusion of fluid remain). Simple measures of tissue under-perfusion, which may persist after apparent restoration of global hemodynamics, include the base deficit and serum lactate. Efforts to measure and enhance tissue perfusion should continue until all such parameters return to normal. More specific measures to monitor tissue perfusion, including tissue oxygen tension devices[19] and gastric tonometry[20], are not widely used.

The best fluid to use for volume expansion in hemorrhagic shock remains a matter of debate. Both crystalloid and colloid are effective, but each has advantages and disadvantages[21] (Table 3). One recent large study showed no difference in mortality in intensive care patients requiring volume expansion whether this expansion was made with saline or albumin[22]. Colloids expand the intravascular space preferentially, whereas crystalloids quickly become distributed throughout the extracellular space. Saline has the disadvantage of hyperchloremia, which causes a dilutional or hyperchloremic acidosis[23,24]. The use of crystalloids is not associated with anaphylaxis, whereas colloids such as the gelatins can produce severe life-threatening reactions, although this is less common with hydroxyethyl starch[25]. Crystalloids have minimal effect on coagulation other than a dilutional effect, although saline infusions may have a procoagulant effect[26]. Overall, crystalloids have a lower cost and lower incidence of side-effects, but the colloids have several theoretical advantages regarding tissue edema and oxygen delivery to the tissues. Despite intense debate and research interest, neither crystalloids nor colloids have been shown to be superior to one another regarding survival outcome from hemorrhagic shock.

It is essential that a protocol be available for the use of blood products in instances of massive bleeding. In the UK, the responsibility for maintaining such a protocol lies with the

Table 3 Intravenous fluids

Type of fluid	Advantages	Disadvantages
Crystalloids		
Saline	cheap; easily available; long history of use	produces a hyperchloremic acidosis; small procoagulant effect
Hartmann's	no risk of anaphylaxis; minimal direct effect on the base deficit; easily available	mildly hypotonic
5% dextrose	no place in acute expansion of the intravascular space	hypotonic; no significant expansion of the vascular space; rapid distribution to intracellular and extracellular spaces
Hypertonic saline	rapid expansion of the intravascular space in excess of the volume infused; possible beneficial effects on red cell and endothelial edema and capillary blood flow	insufficient data; uncertainty regarding possible adverse effects such as on the immune system
Colloids		
Gelatins	largely remains in the intravascular space for 2–4 h	risk of anaphylaxis; no clear survival advantage over crystalloids
4% human albumin	more physiological than gelatins; remains predominantly in the intravascular space for 12 h	expensive; no clear survival advantage over crystalloids
Hydroxyethyl starch	remains in the intravascular space for 12–24 h	risk of coagulopathy, renal injury and reticulo-endothelial accumulation

Hospital Blood Transfusion Committee, a multidisciplinary committee that all hospitals must by law ensure is in place and answerable to the hospital executive. It is unacceptable to have situations where the laboratory insists on blood samples being sent for blood count and coagulation studies before any blood products are issued; the on-call hematology consultant should be actively involved and aid with the use of blood, fresh frozen plasma, platelets, cryoprecipitate and the use of the new recombinant activated Factor VII.

The Trendelenburg position is often used in the management of the hypotensive patient, but its benefit has been questioned. The concept is to displace blood from the lower limbs centrally, to increase preload and enhance cardiac output as a temporary measure until adequate blood volume can be restored. However, there is little proof that this theoretical benefit transpires in practice. Sibbald and colleagues in 1979[27] showed that, in hypotensive patients, the Trendelenburg position did not significantly increase preload, but did increase afterload and blood pressure at the expense of cardiac output. A recent review of available data concludes that the Trendelenburg position 'is probably not a good position for resuscitation of patients who are hypotensive'[28].

The conventional approach to severe hemorrhage, where the endpoint is euvolemia with restoration of a normal blood pressure, heart rate and cardiac output, has been questioned in the out-of-hospital trauma setting[29]. Although not based on evidence from the obstetric population, the physiological rationale may still be applicable. Falling blood pressure and cardiac output, together with increased sympathetic tone and release of endogenous catecholamines, reduce the rate of blood loss. Restoration of these parameters without control of the bleeding will increase the total volume of blood loss, increasing the degree of coagulopathy, reducing oxygen-carrying capacity and ensuing multiple organ dysfunction. Low-volume fluid resuscitation for hemorrhagic shock may be a possibility[30] and the evidence suggests that volume resuscitation should be deliberately limited to the minimum required to sustain vital organ function until the bleeding has been arrested, such as by surgery[29,31].

Recently, small-volume hypertonic resuscitation has been advocated for hemorrhagic shock. The concept is that a relatively small infused volume will cause much larger expansion of the circulation by drawing water into the intravascular compartment. There is evidence that there may be beneficial effects of endothelial and red cell edema and capillary flow, but there are concerns regarding other potentially adverse effects such as that on the immune system[32]. This latter concern has not been shown to be a problem in clinical practice[33].

Maintenance of the hemoglobin concentration is essential to maintain oxygen-carrying capacity and delivery to the tissues. Titration of fluid and blood products to an exact hemoglobin level in a rapidly bleeding patient is difficult. A hemoglobin level of 7–8 g/dl appears an appropriate threshold for transfusion in the intensive-care population, with possible benefit for a higher level of 9 g/dl for those with ischemic heart disease[34]. It is logical to aim at the high end of the target range when resuscitating from hemorrhagic shock as there is a tendency to drift down. A target of 10 g/dl has been suggested as a reasonable goal in the actively bleeding patient[35].

Coagulation disorders are both predisposing factors for, and consequences of, massive postpartum hemorrhage. A bleeding diathesis from a coagulopathy, thrombocytopenia or platelet dysfunction may result from pre-existing disease, a pregnancy-acquired disorder, such as eclampsia, or treatment, such as aspirin. Massive blood loss also creates both a coagulopathy and thrombocytopenia through dilution and consumption. These issues and their management are discussed in detail in Chapters 25 and 26.

SUMMARY

Rapid assessment of the presence of occult bleeding or intravascular volume depletion is essential. The body can compensate for blood loss such that, by the time obvious clinical signs are present, a significant volume can already be lost and tissues already in a state of hypoperfusion. Normal physiological adaptations in late pregnancy that persist into the postpartum period can make recognition and quantification

of intravascular loss difficult, and can render the body less capable of withstanding massive blood loss. This can be further complicated by pregnancy-related disease such as pre-eclampsia and its treatment, and modalities such as hydralazine and magnesium.

Assessment of both the degree of loss and the response to volume replacement require clinical skills, invasive hemodynamic monitoring and the early involvement of senior clinicians. There is no one correct fluid to use. It is usual to use a combination of crystalloids or colloids and blood products to maintain a hemoglobin concentration of near 10 g/dl during the actively bleeding period (7–9 g/dl is probably safe once the active bleeding has been stopped). Coagulopathies and thrombocytopenia also need to be corrected with appropriate transfusion products and with active involvement of the hematologists. There may be a place for limited volume expansion before the bleeding has been stopped surgically to reduce the volume lost, but this must not be at the cost of demonstrable organ ischemia.

Prompt recognition, close monitoring of volume status, rapid arrest of the bleeding and adequate volume resuscitation are all required but when used together can reduce mortality from postpartum hemorrhage.

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