Anaesthesia for Reconstructive Free Flap Surgery

Jane Quinlan

Reconstructive free flap surgery is a complex method of wound closure for large wounds not amenable to linear (primary) closure. It involves the transfer of free tissue (skin, muscle, bone, bowel or a combination) to a site of tissue loss where its circulation is restored via microvascular anastomoses. A muscle flap produces a more even contour and better aesthetic appearance than that achieved by a simple skin graft and provides a better defence against infection. The defect may be caused by trauma, infection or extensive surgery (e.g. mastectomy, head and neck cancer). The site and size of the defect determines which flap is used. The most commonly used flaps are the gracilis muscle for lower leg trauma; latissimus dorsi and rectus abdominis for breast reconstruction; and pectoralis major and radial forearm flap for head and neck reconstruction.

In patients with lower third tibulofibular defects, free tissue transfer is typically required. The bony injury should be repaired and adequate debridement achieved before skin and muscle coverage begins. This should occur within the first 6 days after injury before colonization of the wound and the risk of complications increases. In patients with multiple trauma, any life-threatening injuries must be addressed first and the patient's haemodynamic status stabilized before reconstructive surgery is contemplated.

Flap transfer

The free flap is transferred with its accompanying artery and vein, which are then reattached to vessels at the donor site using microvascular techniques. The stages of flap transfer are:

- flap elevation and clamping of vessels
- primary ischaemia as blood flow ceases and intracellular metabolism becomes anaerobic (this is dependent on surgical time and lasts 60–90 minutes)
- reperfusion as the arterial and venous anastomoses are completed and the clamps released
- secondary ischaemia is subsequent to hypoperfusion of the flap (minimized by appropriate anaesthetic management).

Primary ischaemia

With cessation of blood flow, the flap becomes anoxic. In the presence of anaerobic metabolism, lactate accumulates, intracellular pH drops, ATP decreases, calcium levels rise and pro-

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inflammatory mediators accumulate. The severity of the damage caused by primary ischaemia is proportional to the duration of ischaemia. Tissues with a high metabolic rate are more susceptible to ischaemia, therefore skeletal muscle in a flap is more sensitive to ischaemic injury than skin. At the conclusion of primary ischaemia, the changes in the flap tissue include:

- narrowed capillaries due to endothelial swelling, vasoconstriction and oedema
- sequestration of leucocytes ready to release proteolytic enzymes and reactive oxygen intermediates
- diminished ability of endothelial cells to release vasodilators and degrade ambient vasoconstrictors
- end-organ cell membrane dysfunction and accumulation of intracellular and extracellular toxins
- up-regulation of enzyme systems to produce inflammatory mediators.

Reperfusion begins with the release of the vascular clamps. Normally, the re-establishment of blood flow reverses the transient physiological derangement produced by primary ischaemia. The flap recovers with minimal injury and normal cellular metabolism is restored. However, an ischaemia/reperfusion injury may result if factors in the flap are unfavourable. Prolonged ischaemia time or poor perfusion pressure make this more likely. Reperfusion injury occurs when the restored blood flow allows the influx of inflammatory substrates that may ultimately destroy the flap.

Secondary ischaemia occurs after a free flap has been transplanted and reperfused. This period of ischaemia is more damaging to the flap than primary ischaemia. Flaps affected by secondary ischaemia have massive intravascular thrombosis and significant interstitial oedema. Fibrinogen and platelet concentrations are increased in the venous effluent. Although skin flaps can tolerate 10–12 hours of ischaemia, irreversible histopathological changes in muscle can be seen after 4 hours.

Causes of flap failure: the general causes of poor flap perfusion may be classified as arterial, venous or resulting from oedema. The arterial anastomosis may be inadequate, in spasm or thrombosed. The venous anastomosis may similarly be defective, in spasm or compressed (e.g. by tight dressings or poor positioning). Oedema reduces flow to the flap and may be a result of excessive crystalloids, extreme haemodilution, trauma from handling or a prolonged ischaemia time. Flap tissue has no lymphatic drainage and is therefore susceptible to oedema.

Microcirculation: the blood flow through the microcirculation is crucial to the viability of a free flap. The microcirculation is a series of successive branchings of arterioles and venules from the central vessels. Regulation of blood flow and oxygen delivery is accomplished by three functionally distinct portions of the microcirculation: the resistance vessels, the exchange vessels and the capacitance vessels.

The resistance vessels are the muscular arterioles that control regional blood flow. Arterioles range in diameter from 20 μm to 50 μm and contain a relatively large amount of vascular smooth muscle in their walls. Alterations in vascular smooth muscle tone are responsible for active constriction and dilation in arterioles and thus control resistance to blood flow.

The capillaries constitute the network of vessels primarily responsible for the exchange function in the circulation. Small bands of vascular smooth muscle, the precapillary sphincters, are located at the arterial end of many capillaries and are responsible for the control of blood flow within the capillaries.

The venules act as the capacitance vessels, which collect blood from the capillary network and function as a reservoir for blood in the circulation.

The vascular bed of skeletal muscle has rich adrenergic innervation and therefore has a marked vasoconstrictor response to neural stimulation, primarily through the resistance vessels. Precapillary sphincters also constrict in response to sympathetic stimulation, but are sensitive to local factors such as hypoxia, hypercapnia, and increases in potassium, osmolality and magnesium, which may cause relaxation. Other vasoactive hormones (e.g. renin, vasopressin, prostaglandins, kinins) also have a role in microvascular control.

Transplanted vessels in a free flap have no sympathetic innervation but are still able to respond to local and humoral factors, including circulating catecholamines.

The flow behaviour (rheology) of blood in the microcirculation is determined by the red cell concentration, plasma viscosity, red cell aggregation and red cell deformability. Following all surgery under general anaesthesia, the changes in blood rheology include:

- increased platelet aggregation and adhesion
- an impairment of red cell deformability
- an increase in whole blood viscosity
- increased clotting factors
- increased plasma fibrinogen and red cell aggregation
- disturbance of fibrinolysis.

Normal levels of 2,3-diphosphoglycerate (2,3-DPG) are required for optimal red cell deformability. After blood transfusion, this deformability is impaired owing to the negligible amount of 2,3-DPG in stored blood.

Physiology

The physiological status of the patient has a major influence on the viability of the transferred tissues, so the conduct of anaesthesia and postoperative management have a direct effect on outcome. Surgery is long (often 6–8 hours) with multiple sites for tissue trauma, resulting in extensive blood and fluid losses as well as heat loss. The resulting hypovolaemic vasoconstriction and hypothermia, if not corrected, compromise blood flow to the flap and result in flap failure.

Even with good fluid management, blood flow to a flap may decrease by 50% for 6–12 hours postoperatively. The guiding principle of anaesthesia for free flap surgery is the maintenance of optimum blood flow. The determinants of flow are summarized by the Hagen–Poiseuille equation:

$$\text{Laminar flow} = \frac{\Delta P \times r^4 \times \pi}{8 \times \eta \times l}$$

where: ΔP is the pressure difference across the tube, r is the radius of the vessel, η is viscosity and l is the length of the tube.

From this we may deduce that the goals of anaesthesia for free flap surgery are vasodilatation, good perfusion pressure and low viscosity.

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Vasodilatation

Vessel radius is the most important determinant of flow, for the vessels supplying the flap as well as those in the flap.

Temperature – the patient should be kept warm in theatre, the recovery room and the ward for the first 24–48 hours. This is best achieved by raising the ambient temperature in theatre and by using a warm air blanket. Active warming should begin before the start of anaesthesia because patient cooling occurs rapidly after induction of anaesthesia. In an awake patient, the central core temperature is higher than that of the peripheral tissue and skin temperature. After the induction of anaesthesia, vasodilatation modifies the thermal balance between compartments. The volume of the central compartment enlarges leading to a decrease in its mean temperature, while the temperature of the peripheral and skin compartments increases. At thermoregulation, the size of the central compartment becomes smaller owing to vasoconstriction, which leads to an increase in the mean temperature, although the peripheral and skin temperatures fall.

In addition to vasoconstriction, hypothermia also produces a rise in haematocrit and plasma viscosity, the aggregation of red blood cells into rouleaux, and platelet aggregation. These effects may reduce the microcirculatory blood flow in the flap.

Fluid – peripheral vasoconstriction due to an underestimation of fluid losses is common. There are two operating sites in free flap transfer: the donor site and the recipient site. Both have insensible fluid losses and both may have blood losses. A warm theatre environment also increases fluid loss. Modest hypervolaemia reduces sympathetic vascular tone and dilates the supply vessels to the flap. An increase in central venous pressure of 2 cm H_2O above the control measurement can double the cardiac output and produce skin and muscle vasodilatation. Figure 1 gives a guide to fluid management.

Anaesthesia – isoflurane has the advantage over other volatile anaesthetics and propofol that it causes vasodilatation with minimal myocardial depression. Propofol inhibits platelet aggregation which could reduce the risk of thrombosis. This may be due to an effect of intralipid on the platelet–erythrocyte interaction, and by the increased synthesis of nitric oxide by leucocytes.

Vasospasm of the transplanted vessels may occur after surgical handling or after damage to the intima of the vessels, and can occur during surgery or postoperatively. The surgeons may use topical vasodilators such as papaverine, lidocaine (lignocaine) or verapamil during the operation to relieve the vasospasm.

Sympathetic blockade – epidural, brachial plexus or interpleural local anaesthetic infusions, used intraoperatively and postoperatively, provide sympathetic blockade to further dilate vessels. Concerns have been raised that the sympathetically-denervated transplanted vessels would be unable to dilate after lumbar epidural blockade, resulting in a 'steal' effect reducing flap blood flow. In fact, provided any hypotension due to the sympathetic block is treated appropriately, blood flow to the flap improves as a result of the increased flow through the feeding recipient artery. Other advantages of epidural analgesia include a reduction in intraoperative and postoperative blood loss and vessel spasm; a lower incidence of deep venous thrombosis; improved diaphragmatic function and more rapid postoperative recovery. Good analgesia reduces the level of circulating catecholamines and avoids the vasoconstrictor response to pain.

Guide to fluid management

Crystalloids
- 10–20 ml/kg to replace preoperative deficit
- 4–8 ml/kg/hour to replace insensible losses

Colloids
- 10–15 ml/kg for haemodilution
- To replace blood loss

Blood
- To maintain haematocrit at 30%

Dextran
- Often given postoperatively

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Perfusion pressure

The preservation of a good perfusion pressure with wide pulse pressure is essential to flap survival. Appropriate anaesthetic depth and aggressive fluid management are usually all that is needed. Most inotropes are contraindicated owing to their vasoconstrictive effects, but if required, dobutamine and low-dose dopamine could be used.

Viscosity

Isovolaemic haemodilution to a haematocrit of 30% improves flow by reducing viscosity, reducing reperfusion injury in muscle and increasing the number of patent capillaries, which decrease tissue necrosis. Further reductions in haematocrit do not provide much more advantage because the curve of viscosity versus haematocrit flattens off markedly (Figure 2). If the haematocrit falls further, the marginally improved flow characteristics from a lower viscosity may then be offset by a reduction in oxygen delivery:

$$DO_2 = CO \times [(Hb \times sat \times 1.34) + (PaO_2 \times 0.003)]$$

A low haematocrit also increases myocardial work, therefore care should be taken in patients with poor cardiac reserve.

Practical conduct of anaesthesia

Monitoring

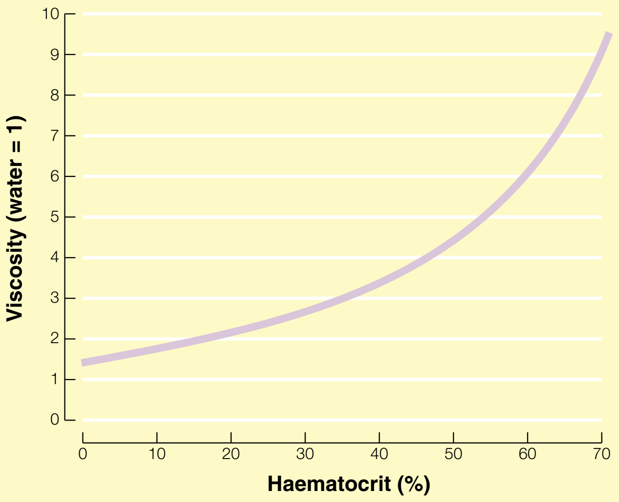
In addition to basic monitoring, these patients require invasive blood pressure monitoring to enable safe manipulation of the perfusion pressure. Direct measurement of arterial pressure gives a continuous record of the pressure and is more accurate than non-invasive indirect techniques. An arterial cannula also provides access to blood gas analysis and haematocrit estimations.

Central venous pressure reflects cardiac filling pressures and can be manipulated to increase cardiac output.

Core temperature measurement is essential when active warming is instituted. A nasopharyngeal or rectal probe is used intraoperatively for a continuous reading, while intermittent tympanic or axillary measurements are used in the recovery and ward areas.

Peripheral temperature is also measured because a fall in skin temperature can reflect hypovolaemia and vasoconstriction. A difference of less than 2°C between core and peripheral temperatures indicates a warm, well-filled patient. The temperature of

Viscosity versus haematocrit



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the skin flap is sometimes monitored postoperatively because a drop in temperature may herald flap failure. However, this is not a sensitive test, because by the time the temperature has fallen the flap will have suffered sufficient vascular damage to render it virtually unsalvageable.

Urine output is another indicator of volume status. A urine output of 1–2 ml/kg/hour should be maintained intraoperatively and postoperatively with appropriate fluid management. Diuretics are contraindicated in these operations because volume depletion compromises flap survival.

Induction

Active warming starts before the patient is asleep. The ambient temperature in theatre is raised to about 22–24°C, a level high enough to reduce patient heat loss, but not too hot to be uncomfortable for the theatre staff. The patient is covered in a hot air blanket before induction of anaesthesia and this remains in place while the patient is prepared for theatre. Once in theatre, the blanket is moved to enable surgical access, but with as much surface area coverage as possible.

If appropriate, a regional block is inserted, preferably to cover the free flap recipient site (rather than the donor site) for the full benefit of the sympathetic block. The patient is intubated and ventilated; and a large gauge peripheral line, central line, arterial line, urinary catheter and core temperature and skin temperature probes are positioned.

Nitrous oxide diffusion into air in the stomach combined with gastric stasis results in gastric distension, with associated postoperative nausea and vomiting. A nasogastric tube is therefore sited at intubation, left on free drainage, then aspirated and removed at the end of the operation.

Fluid, administered through a fluid warmer, is started in the anaesthetic room to compensate for preoperative dehydration.

Principles of perioperative and postoperative care

- Maintain high cardiac output
- Normal arterial blood pressure (systolic >100 mm Hg)
- Low systemic vascular resistance
- Normothermia
- High urine output (> 1 ml/kg/hour)
- Effective analgesia
- Haematocrit 30–35%
- Monitoring of blood flow in flap (Doppler postoperatively)

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Maintenance

Careful positioning of the patient is imperative for such a long operation. Limbs are positioned and supported to avoid neurological damage or vascular compression. Eyes are taped and lightly padded to reduce the incidence of corneal abrasion and prevent drying of the cornea.

Prophylaxis against deep venous thrombosis is necessary for all patients. Subcutaneous heparin or low-molecular-weight heparin is given preoperatively, while anti-embolism (TED) stockings and compression boots are used intraoperatively.

The patient is ventilated to normocapnia. Hypocapnia increases peripheral vascular resistance and reduces cardiac output, while hypercapnia causes sympathetic stimulation. If the surgeon uses the microscope for vessel preparation or anastomosis on the chest or abdomen, the tidal volume is reduced to minimize movement in and out of the surgeon's field of vision. The respiratory rate is then increased to maintain minute ventilation.

Controlled hypotension is useful during the initial dissection and is most easily achieved using epidural local anaesthetic and/or isoflurane. An infusion of glyceryl trinitrate may be added if needed.

Crystalloids are used to replace the preoperative fluid deficit from starvation and to cover intraoperative insensible losses. The latter are high because the warm theatre increases evaporative losses from the two operating sites. Excessive use of crystalloid may precipitate oedema in the flap.

Hypervolaemic haemodilution is achieved using colloids.

Blood gas analysis and haematocrit measurement should be carried out at the start of the operation and repeated every 2 hours.

By the time the flap is reperfused, the patient should be warm, well-filled and sympathetically blocked with a high cardiac output.

Emergence and recovery

The patient should wake up pain-free. Analgesia is maintained postoperatively with local anaesthetic infusions for regional blocks, intravenous patient-controlled analgesia, or both. Coughing and vomiting increase venous pressure and reduce flap flow, so smooth emergence and extubation are needed. The principles of perioperative and postoperative care are listed in Figure 3. ◆

FURTHER READING

MacDonald D J F. Anaesthesia for Microvascular Surgery. *Br J Anaesth* 1985; **57:** 904–21.

Sigurdsson G H, Thomson D. Anaesthesia and Microvascular Surgery: Clinical Practice and Research. *Eur J Anaesthesiol* 1995; **12:** 101–22.