

Arterial tourniquets

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Key points

Complications of tourniquet use are rare, but can be catastrophic.

Local effects of arterial tourniquets result from tissue compression beneath the cuff and ischaemia distal to it.

Tissue compression predominantly affects nerve tissue, whereas muscle is more susceptible to ischaemia.

Widespread systemic effects of arterial tourniquets usually result from cuff inflation and deflation.

Limits for inflation pressures and times should be tailored to individual patients.

A tourniquet is a device which is used to control the flow of blood to and/or from an extremity. The word tourniquet itself derives from the French verb *tourner* (to turn) and was first used by the eighteenth-century French surgeon Louis Petit describing the screw-like device he strapped to the thighs of patients undergoing leg amputations, to reduce blood loss. This article focuses on the use of arterial tourniquets. The arterial tourniquet is usually a pneumatic device consisting of an inflatable cuff connected to a compressed gas supply. The measurable and high pressures that such tourniquets can generate allow controlled arterial compression and distal circulatory stasis.

The most common use for such tourniquets is in surgical procedures on the extremities, where the tourniquet is of great value in optimizing operating conditions by creating a bloodless surgical field. Examples include joint replacements and the repair of tendons, nerves, and blood vessels.

Physiological effects of tourniquet application

Tourniquets are used to facilitate surgery which aims to restore or improve the function of a patient's upper or lower limb. However, incorrect use of such a device may cause permanent damage and loss of function in the limb. It is therefore important that operating department personnel understands the physiological effects of tourniquets in order to use them safely.

Local effects

Local effects are the result of tissue ischaemia distal to the inflated tourniquet and a combination of ischaemia and compression of the tissues beneath it.

Muscle

Following inflation of the tourniquet, there is a progressive decrease in P_{O_2} and an increase in P_{CO_2} within muscle cells. Energy stores steadily decline with time and intracellular stores of

ATP and creatine phosphate are exhausted after 2 and 3 h, respectively. Lactate concentration increases with the switch to anaerobic metabolism and, with the increasing P_{CO_2} , contributes to the development of an intracellular acidosis. Changes to the histological appearances of muscle fibres occur after tourniquet inflation. Marked changes in mitochondrial morphology are visible after 1 h of ischaemia. Muscle underlying the tourniquet subjected to both ischaemia and compression is prone to developing changes such as local fibre necrosis after inflation times as short as 2 h.

Microvascular injury occurs in muscle after ischaemia of greater than ~2 h duration. After tourniquet release, increased vascular permeability results in interstitial and intracellular oedema which frequently leads to the 'post-tourniquet syndrome', in which the patient has a swollen, pale, stiff limb with weakness but no paralysis. This post-tourniquet syndrome typically lasts 1–6 weeks.

Nerve

A physiological conduction block develops between 15 and 45 min after inflation of a cuff around the arm to a supra-systolic pressure. The conduction block affects both motor and sensory modalities and is reversible after deflation of the cuff. The rate of development of this conduction block is the same with cuff pressures of 150 and 300 mm Hg which suggests that ischaemia rather than direct compression is the cause.¹

Direct mechanical compression of nerves is responsible for a second, longer lasting nerve conduction block called 'tourniquet paralysis'. Higher cuff pressures (e.g. 1000 mm Hg maintained for >1 h) can cause morphological changes within the larger myelinated nerves which is most marked at the sites where the pressure gradient between compressed and uncompressed nerve is greatest: at the proximal and distal edges of the tourniquet. The pressure gradient results in displacement of the nodes of Ranvier, stretching and degeneration of

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paranodal myelin and impaired nerve conduction lasting up to 6 months.

Systemic effects

Several systemic effects occur with inflation and deflation of a limb tourniquet.

Cardiovascular effects

After limb exsanguination and tourniquet inflation, there is an increase in systemic vascular resistance and an effective increase in circulating blood volume. This leads to an increase in central venous pressure and in most instances an accompanying increase in systolic arterial pressure, both of which are usually transient. Application of bilateral thigh tourniquets can increase the effective circulating blood volume by up to 15% (~750 ml in an adult). Such large increases in circulating blood volume may cause large and sustained increases in central venous pressure and circulatory overload. Cardiac failure and cardiac arrest have been reported after the application of bilateral thigh tourniquets. Following the initial transient increase in arterial pressure, it is common to see a second, gradual increase in arterial pressure. This is thought to accompany the development of tourniquet pain and develops a variable period of time after inflation. The rise in arterial pressure can be attenuated by the addition of ketamine (0.25 mg kg⁻¹),² this may also help with tourniquet pain.³

On tourniquet deflation, post-ischaemic reactive hyperaemia is seen; this causes a transient increase in the volume of blood in the limb compared with baseline levels. Simultaneously, metabolites from the ischaemic limb are released into the systemic circulation. In combination with the redistribution of blood flow, this often causes a decrease in both central venous and systolic pressures, which is temporary but can be dramatic.

Respiratory effects

Deflation of the tourniquet is followed almost immediately by an increase in end-tidal carbon dioxide concentration ($F_{E'CO_2}$) which usually peaks within 1 min. The increase in $F_{E'CO_2}$ occurs for two reasons: mixed venous P_{CO_2} increases (after release of hypercapnic blood from the ischaemic area distal to the tourniquet into the systemic circulation) and also cardiac output increases after tourniquet deflation (in response to the decrease in arterial pressure described above). The peak increase in end-tidal CO_2 concentration is greater with deflation of lower limb tourniquets (0.7–2.4 kPa) than with upper limb tourniquets (0.1–1.6 kPa).⁴ The duration of the increase in $F_{E'CO_2}$ depends on the ventilatory characteristics of the patient. In spontaneously breathing patients, minute ventilation increases rapidly and so $F_{E'CO_2}$ returns to baseline values within 3–5 min. In patients undergoing controlled ventilation, the $F_{E'CO_2}$ will typically remain raised for >6 min unless minute ventilation is deliberately increased.

Central nervous system effects

The increase in P_{aCO_2} which accompanies deflation of the tourniquet causes an increase in cerebral blood flow. Measurements of middle cerebral artery blood flow velocity show an increase of up to 50%. In patients with head injuries, the increase in cerebral blood flow can cause an increase in intracranial pressure and worsen the degree of secondary brain injury. Hyperventilation after tourniquet deflation can prevent deleterious increases in intracranial pressure by maintaining normocapnia.

Haematological effects

The haematological effects of tourniquets are complicated. Tourniquet inflation during surgery is associated with a global hypercoagulable state. This is attributable to increased platelet aggregation caused by catecholamines released in response to pain from surgery and the tourniquet itself. However, there is no difference in the incidence of deep vein thrombosis in surgery on lower limbs performed with and without a tourniquet. After deflation of the tourniquet, there is a brief period of increased fibrinolytic activity. This increase is maximal at 15 min and returns to preoperative levels within 30 min of tourniquet release, but may nevertheless cause increased bleeding. The increase in fibrinolysis is caused by release of tissue plasminogen activator, which is thought to be produced by the vasa vasorum in the affected limb in response to the acidosis and hypoxaemia associated with tourniquet application.

Temperature effects

Inflation of arterial tourniquets is associated with a gradual increase in core body temperature caused by reduced heat transfer to and heat loss from the ischaemic limb. The magnitude of this increase is small, ~0.5°C after 2 h of inflation. Tourniquet deflation causes a transient decrease in core temperature, primarily caused by redistribution of body heat. In addition, it is associated with the return to the systemic circulation, of a small amount of hypothermic blood from the ischaemic limb.

Metabolic effects

Deflation of the tourniquet after 1–2 h of ischaemia is associated with small increases in plasma concentrations of potassium and lactate. Peak increases of 0.3 and 2 mmol litre⁻¹, respectively, occur 3 min after deflation.⁵ Lactate and carbon dioxide returning to the systemic circulation from the ischaemic limb cause a reduction in arterial pH. Reperfusion of the ischaemic limb and the other haemodynamic changes associated with tourniquet deflation can cause brief increases in oxygen consumption and carbon dioxide production. The magnitude of these changes correlates with the duration of ischaemia. All of these changes are fully reversed within 30 min of tourniquet deflation.

Complications associated with the use of arterial tourniquets

Ischaemia and compression can lead to damage to any of the tissues in the limb. However, when used carefully, the tourniquet is a safe device and complications are fortunately rare. Consequently, it is difficult to make precise estimates of the incidence of complications following the use of tourniquets. One of the largest studies of tourniquet use and its complications was performed in Norway by Odinson and Finsen⁶ during a 2 yr period. Out of 63 484 operations performed under a tourniquet, only 26 complications that might have been caused by the tourniquet were reported (an incidence 0.04%).

Nerve injury

Neurological injuries after tourniquet use are probably the most common complication and can be the most devastating. In Odinson and Finsen's large series, 15 neurological complications were reported (incidence 0.024%); of which, two were permanent and the remainder resolved within 6 months.⁶ Lower limb tourniquets were more likely to produce neurological complications than upper limb tourniquets. The nerves most commonly affected are the sciatic nerve in the lower limb and the radial nerve in the upper limb.

Although longer tourniquet times are independently associated with increased risk of neurological injury,⁷ mechanical pressure probably plays a more important role than ischaemia in nerve injury. Large diameter nerve fibres are more susceptible to pressure, so there is a relative sparing of sensation compared with motor function. The role of mechanical pressure in nerve injury probably explains why the Esmarch bandage (which can generate pressures >1000 mm Hg) is associated with a higher incidence of nerve injury. The effects of nerve compression at the site of tourniquet application may make injury at a more distal site (caused by ischaemia or surgical trauma) more likely due to the neural 'double crush'.

Muscle injury

The post-tourniquet syndrome results in a swollen, stiff, weak limb. Very rarely the post-ischaemic swelling and oedema, in combination with reperfusion hyperaemia, may lead to the development of a compartment syndrome. Rhabdomyolysis directly related to the tourniquet use has been reported, but is extremely rare. Most cases have been associated with unusually long ischaemic times or high cuff inflation pressures.

Skin injury

Chemical burns are the most common form of skin injury and occur when alcohol-based solutions used for skin preparation seep beneath the tourniquet and are then held against the skin under

pressure. Friction burns resulting from movement of poorly applied tourniquets have also been reported.

Vascular injury

Arterial injury after tourniquet use is uncommon, but can be catastrophic. In one series, only seven arterial injuries were reported in >5000 knee arthroplasties, but three of these resulted in amputation.⁸ Acute vascular insufficiency is thought to occur when mechanical pressure from the tourniquet damages atheromatous vessels, causing plaque rupture. Consequently, many experts recommend avoiding the use of arterial tourniquets in patients with peripheral vascular disease.

Intraoperative bleeding

Common causes of intraoperative bleeding include incomplete exsanguination of the limb and a poorly fitting or under-pressurized cuff. Intraoperative bleeding may also be caused by blood entering through the intramedullary vessels of long bones.

Tourniquet pain

Inflation of a tourniquet is followed by the development of a dull, aching pain. Such pain can accompany otherwise adequate regional anaesthesia and can be severe enough to necessitate conversion to general anaesthesia. Various theories exist about how such pain arises. It is thought that tourniquet pain is predominantly mediated by unmyelinated, slowly conducting C-fibres which, as stated earlier, are less affected by the compressive effects of tourniquet inflation than larger fibres. One theory suggests that tourniquet pain arises from selective transmission by cutaneous C-fibres; these fibres are continuously stimulated by skin compression from the tourniquet, and their post-synaptic effect at the dorsal horn is no longer inhibited by input from larger nerve fibres whose transmission has been blocked. This theory is supported by the finding that during i.v. regional anaesthesia, tourniquet analgesia is prolonged by application of EMLA cream beneath the tourniquet.⁹

However, tourniquet pain can complicate spinal or epidural anaesthesia, despite apparently adequate anaesthesia of the sensory dermatome underlying the tourniquet. One explanation of this is offered by the *in vitro* finding that smaller unmyelinated C-fibres are more resistant to local anaesthetic-induced conduction block than larger, myelinated A-fibres.¹⁰ After intrathecal administration of an adequate dose of local anaesthetic, conduction in both A- and C-fibres is blocked. However, as the concentration of local anaesthetic in the cerebrospinal fluid decreases, C-fibres start to conduct impulses before the A-fibres, resulting in a dull tourniquet pain in the presence of an anaesthetic which, assessed by pinprick, appears adequate.

Just as there is no completely satisfactory explanation for the phenomenon of tourniquet pain, there is no completely satisfactory solution. Various techniques have been used, including increasing the density of central neuraxial block by using adjuncts to bupivacaine such as epinephrine, morphine, and clonidine. Alternatively,

some success has also been reported with the administration of preoperative gabapentin and the use of low-dose (0.1 mg kg⁻¹) i.v. ketamine.³

Tourniquet-induced hypertension

A gradual increase in arterial pressure is frequently observed a variable time after tourniquet inflation. The exact mechanism of this tourniquet-induced hypertension is not known, but it has been suggested that it represents activation of the sympathetic nervous system in response to the development of tourniquet pain. Plasma norepinephrine concentrations increase in parallel with arterial pressure during tourniquet inflation. In awake volunteers, the increasing arterial pressure runs parallel to development of pain, supporting this theory.¹¹ Clonidine attenuates sympathetic activity and, if given preoperatively, blunts the hypertensive response.

Tourniquet pressures and safe inflation times

The issues regarding safe inflation pressures and times for tourniquets are controversial. No strict guidelines exist, in part because there is no general consensus but also because each patient is different and what is safe for one limb is not necessarily safe for another.

Pressures

Surveys of orthopaedic surgeons reveal two common practices regarding inflation pressures: (i) to inflate the tourniquets to fixed pressures (typically 250 mm Hg for upper arm and 300 mm Hg for the thigh); and (ii) to inflate to pressures which are a fixed amount above systolic arterial pressure (typically +100 mm Hg for upper arm and +100–150 mm Hg for the thigh).

Inflating cuffs to a fixed pressure have been criticized because it takes no account of the patient's usual arterial pressure. It has been shown that younger age is an independent predictor of neurological injury after prolonged tourniquet times. The authors of this study hypothesized that this was the result of younger patients having lower systolic pressures, meaning there was a larger difference between tourniquet inflation pressure and arterial pressure leading to excessive compression.⁷

The Association of peri-operative Registered Nurses (AORN) in the USA recommends inflating tourniquets to pressures based on the limb occlusion pressure (LOP).¹² This value is determined by gradually increasing the pressure in the tourniquet while assessing distal blood flow with a Doppler probe held over a distal artery. The LOP is the pressure in the cuff at which the arterial pulse disappears. The LOP is usually higher than systolic pressure because the pressure from the tourniquet which is transmitted to deep underlying soft tissues is frequently less than that in the tourniquet itself. The percentage of transmitted tourniquet pressure varies inversely with limb circumference (hence the practice of using higher pressures on the thigh than on the upper arm). The

AORN guidelines recommend that the tourniquet is inflated intraoperatively to a pressure higher than the LOP; a safety margin is added to cover intraoperative fluctuations in arterial pressure. If LOP is <130 mm Hg then 40 mm Hg is added, 60 mm Hg added if LOP is 131–190 mm Hg, and 80 mm Hg added if LOP is >190 mm Hg. So a young, slim, normotensive adult may require a cuff pressure <200 mm Hg.

Duration

All tourniquets should be kept inflated for the minimum length of time possible. The maximum duration that a tourniquet can be inflated continuously before a period of limb reperfusion is necessary has not been precisely established. In practice, safe inflation time will be determined by the patient's age, physical health, and integrity of the vascular supply to the limb. Most recommendations in the literature suggest a period of 1.5–2 h in a healthy adult, which corresponds to the point at which muscle ATP stores are depleted. Although ischaemia has typically been associated with muscular rather than neurological injury, there is an approximate three-fold increase in the risk of neurological complications for each 30 min increase in tourniquet time.⁷

If the duration of surgery exceeds the maximum safe inflation time, the tourniquet should be deflated for a short period. This allows metabolic waste products to be removed and oxygenated blood to perfuse the limb. It is generally considered that the tourniquet should be left deflated for 10–15 min before re-inflation and this seems to correspond to restoration of muscle ATP levels. It is important to re-exsanguinate the limb before inflating the cuff again after reperfusion.

Conclusions

Arterial tourniquets are widely used in orthopaedic, plastic, and reconstructive surgery where they are invaluable in providing excellent operating conditions and reducing blood loss. However, the process of exsanguination followed by ischaemia provides a physiological insult with both local and systemic consequences of which the anaesthetist should be aware. Complications in young, fit patients are fortunately very rare, but the risks of tourniquet-related injury are increased in the elderly and those with co-morbidities such as peripheral vascular disease. Increased complication rates are also seen with higher inflation pressures and longer ischaemic times and so anaesthetists should be aware of recommended operating practice.

References

1. Lundborg G. *Nerve Injury and Repair*. Edinburgh: Churchill Livingstone, 1988
2. Satsumae T, Yamaguchi H, Sakaguchi M et al. Preoperative small-dose ketamine prevented tourniquet-induced arterial pressure increase in orthopedic patients under general anesthesia. *Anesth Analg* 2001; **92**: 1286–9

3. Segerdahl M, Ekblom A, Sollevi A. The influence of adenosine, ketamine, and morphine on experimentally induced ischemic pain in healthy volunteers. *Anesth Analg* 1994; **79**: 787–91
4. Dickson M, White H, Kinney W, Kambam JR. Extremity tourniquet deflation increases end-tidal pCO₂. *Anesth Analg* 1990; **70**: 457–8
5. Modig J, Kolstad K, Wigrew A. Systemic reactions to tourniquet ischaemia. *Acta Anaesthesiol Scand* 1978; **22**: 609–14
6. Odinson A, Finsen V. Tourniquet use and its complications in Norway. *J Bone Joint Surg* 2006; **88B**: 1090–2
7. Horlocker TT, Hebl JR, Gali B et al. Anesthetic, patient and surgical risk factors for neurologic complications after prolonged total tourniquet time during total knee arthroplasty. *Anesth Analg* 2006; **102**: 950–5
8. Insall JN, Windsor RE. *Surgery of the Knee*, 2nd Edn. New York: Churchill Livingstone, 1993
9. Lowrie A, Jones MJ, Eastley RJ. Effect of a eutectic mixture of local anaesthetic agents (EMLA) on tourniquet pain in volunteers. *Br J Anaesth* 1989; **63**: 751–3
10. Gissen AJ, Covino BG, Gregus J. Differential sensitivities of mammalian nerve fibres to local anesthetic agents. *Anesthesiology* 1980; **53**: 467–74
11. Hagenouw RR, Bridenbaugh PO, van Egmond J, Stuebing R. Tourniquet pain: a volunteer study. *Anesth Analg* 1986; **65**: 1175–80
12. Recommended practices for the use of the pneumatic tourniquet in the perioperative practice setting. *AORN J* 2007; **86**: 640–55

Please see multiple choice questions 16–18