The pathophysiology of the arterial tourniquet: a review

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Arterial tourniquets are widely used in limb surgery to reduce intra-operative bleeding, thereby providing for better operative conditions. There are, however, a number of consequences, both localised and systemic, related to tourniquet use. While these may be relatively benign in the healthy patient, they may be devastating in the patient with, for example, poor cardiac reserve [1, 2, 3].

LOCALISED CONSEQUENCES

NERVE INJURY

The incidence of nerve injury is reported to be 1:3000 – 1:11000 for the upper limb and 1:13000 – 1:250000 for the lower limb [1,2,3,4], and may range from parasthesiae to paralysis. The radial nerve is most commonly involved, followed by the ulnar and median nerves. This is because of the anatomy of the radial nerve – it gets compressed against the humerus. Kurihara and Goto [4] reported two cases of radial nerve paralysis after a tourniquet pressure of 250mmHg for 45 – 65 minutes, in patients with previous humeral fractures. Presumably greater pressure was applied to the radial nerve in the presence of a bony deformity. In the leg, the common peroneal nerve is most at risk [3].

The aetiology of nerve injury is mechanical pressure rather than ischaemia. The greater the pressure, the greater the injury [6]. There is more potential for injury with an Esmarch bandage (pressures of 1000mmHg may be generated) than with a pneumatic tourniquet [1,2]. For this reason, the Esmarch bandage is not recommended for exsanguination [1]. The arm may be exsanguinated by elevation at 90° for 5 minutes [7] and the leg by elevation at 45° for 5 minutes [8].

The nerve injury is greatest where the shear stress is greatest, i.e. at the proximal and distal edges of the cuff [1,2,3,5]. The pressure causes intussusception of the nerve under the cuff into the adjacent part, seriously distorting the microstructure [3]. Compression also causes intraneural microvascular abnormalities and oedema, thereby compromising local tissue nutrition. Axonal degeneration may occur. Damage may also occur at the nodes of Ranvier [1]. Intermittent reperfusion does not prevent the compressive neuropathy [5]. This compressive neuropathy may result in secondary features of denervation in distal tissues, e.g. delayed recovery of blood flow, increased vessel spasm, haemorrhage and oedema [6].

CONSEQUENCES OF AN ARTERIAL TOURNIQUET	
LOCALISED	**SYSTEMIC EFFECTS**
· Nerve injury · Muscle injury · Vascular injury · Skin injury · Haematoma/ bleeding · Tourniquet failure	· Cardiovascular · Respiratory · Cerebral circulatory · Haematological · Temperature changes · Metabolic

The prognosis of tourniquet induced nerve injuries is good – permanent deficits are rare and most injuries heal spontaneously within 6 months [1,2,3].

Not all post – tourniquet neurologic deficits are due to the tourniquet. The differential diagnosis includes pre-existing neurologic disease, nerve damage secondary to the surgery or trauma, injury due to malpositioning, injury due to haematoma or oedema, nerve damage either by needle trauma during the performance of a regional block or by chemicals (local anaesthetic solutions), spontaneous e.g. herniation of a cervical disc or other causes. It is imperative that any patient experiencing post-tourniquet neurologic dysfunction be thoroughly examined and treatable causes ruled out. Neurological consultation and electrophysiological studies may be required, as well as physiotherapy, occupational therapy etc. The offending tourniquet should be checked, e.g. for gauge malfunction [2].

MUSCLE INJURY

Muscle injury is caused by ischaemia beneath and distal to the cuff. The injury is greatest beneath the cuff because of the combination of ischaemia and mechanical deformation of the tissue. The extent of the damage is related to the duration of the ischaemia [3]. With time the intracellular concentrations of creatine phosphate, glycogen, ATP and oxygen decrease [1,2]. Mitochondrial PO_2 reaches zero within 6 minutes [9]. Creatine phosphate is depleted by 2 hours and the ATP supply is exhausted

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by 3 hours [1]. Beyond 3 hours the metabolic recovery rate of muscles is greatly prolonged. Lactate and potassium concentrations and the P_aCO_2 increase with increasing duration of ischaemia. Toxic oxygen radicals e.g. hydrogen peroxide form in the muscle. Adhesion molecules and cytokines are activated [1]. Intracellular pH decreases – a pH of 6.0 is reached by 4 hours. Intravenous pH in the limb decreases and a pH of 6.9 corresponds to the fatigue point of muscle. Further ischaemia may produce irreversible muscle damage [2].

After 2 hours at 200 – 300 mmHg, histological changes e.g. inflammatory cells, focal necrosis, regional necrosis and hyaline degeneration may be seen in the muscle beneath the cuff. It takes 4 hours for these changes to develop in the distal muscle [1].

Tourniquet induced ischaemia and reperfusion not only generates hydrogen peroxide - it also causes significant increases in xanthine oxidase activity in both local and systemic blood [10]. Xanthine oxidase contributes to injury of skeletal muscle, myocardium, kidneys and lungs after ischaemia and reperfusion. Hydrogen peroxide may also be generated by activated neutrophils. Antioxidant mechanisms provide protection e.g. intact red cells scavenge hydrogen peroxide. The increase of hydrogen peroxide at the start of reperfusion may be due to pooling and release in the absence of red cells to scavenge it [10]. Propofol has been shown to attenuate ischaemia – reperfusion induced lipid peroxidation [11]. Ischaemic preconditioning of skeletal muscle, on the other hand, was not shown to be beneficial [12].

After tourniquet release, microvascular permeability increases and oedema may form. Rarely, a compartment syndrome may develop. It may take up to 1 month for the oedema to resolve. Creatine kinase and myoglobin levels also increase post tourniquet release, but not significantly so unless the tourniquet time has been longer than 2 hours [2].

Should it be necessary to use a tourniquet for longer than 2 hours, it is recommended that the limb be reperfused periodically to allow for metabolic recovery of the muscle and maintenance of ATP levels. Recommendations vary from 10 minutes hourly [1] to 15 – 20 minutes every 2 hours [2].

The "post – tourniquet syndrome" is thought to be caused mainly by oedema. The affected limb is stiff, pale, weak but not paralysed, and subjectively numb without objective anaesthesia. It typically resolves over 1 – 6 weeks [1,2].

Routine tourniquet use results in weakness and delayed post –operative recovery. Force production is markedly decreased both beneath and distal to the tourniquet [6], with the directly compressed muscle being more severely affected. Greater pressures produce greater functional impairment. Fast twitch fibres are affected more than slow twitch fibres. Wide, properly fitting cuffs require lower inflation pressures, which may reduce muscle injury [6].

VASCULAR INJURY

Vascular injuries are fortunately rare [1,2,13]. They are usually associated with peripheral vascular disease and fractures of atheromatous plaques by pressure [1,13], or plaque dislodgement [2], or thrombosis due to lack of blood flow [1,13].

SKIN INJURIES

Skin injuries are uncommon. Esmarch bandages twist and stretch the skin [2], while pressure necrosis and shearing have been described with pneumatic tourniquets because of inadequate padding or bad application. Chemical burns have been reported with alcohol based cleansing solutions held against the skin under pressure [15]. Choudhary et al [14] reported friction burns from the movement of a fully inflated tourniquet over bare skin.

HAEMATOMAS/ BLEEDING

Because of tourniquet inflation, bleeders may not be identified intraoperatively. Once the tourniquet is released, a haematoma may develop or there may be a potential for acute blood loss superimposed on the haemodynamic changes of tourniquet release [2]. However, tourniquet release for haemostasis has actually been shown to increase bleeding [16]. Haematomas, arterial injuries and a compartment syndrome may all result in a delayed return of blood flow after tourniquet release [2].

TOURNIQUET FAILURE

Bleeding may occur despite a properly applied and inflated tourniquet, in a patient with noncalcified vessels. This is the phenomenon of tourniquet ooze. Blood bypasses the tourniquet through the medulla of the humerus or femur. Oozing typically starts about 30 minutes after tourniquet inflation [3]. Increasing the tourniquet pressure does not help [2].

Other causes of inadequate haemostasis include arterial and venous leakage due to inadequate pressure, calcified, incompressible vessels and inadequate exsanguination [2].

SYSTEMIC CONSEQUENCES

CARDIOVASCULAR FEATURES

Cardiovascular features are related to all stages of tourniquet use, from exsanguination to inflation, maintenance and deflation. Patients with poor cardiac reserve may tolerate these changes poorly, and cardiac arrests related to acute volume overload have been reported [2,3].

Limb exsanguination and subsequent tourniquet inflation increase blood volume and systemic vascular resistance. Central venous pressure increases by up to 14 – 15 cmH_2O and blood volume by up to 800 ml following exsanguination of both legs [1,2]. The changes in central venous pressure and blood pressure may be transient or may be maintained until tourniquet release.

Patients with poor cardiac reserve may not tolerate the sudden increase in blood volume. Relative contraindications to exsanguination include poor cardiac reserve, serious limb infection (potential for systemic spread), and severe trauma to the limb (the circulation to a limb that has suffered a severe crush injury may be precarious, and tourniquet use may allow the necrosis of potentially viable tissue)[2].

Approximately 30 – 60 minutes after tourniquet inflation, heart rate and blood pressure increase – this is "tourniquet pain". An awake patient will complain of a vague, dull pain that becomes so severe as to be unbearable. It will occur despite an adequate sensory level. The incidence increases with increasing age and duration of surgery, and with lower limb surgery [1]. The exact aetiology is unclear, but it is thought to be due to a cutaneous neural mechanism. The pain is probably mediated by the unmyelinated, slow conducting C fibres that are usually inhibited by the earlier arriving fast impulses conducted by the myelinated A-delta fibres. The A-delta fibres are blocked by mechanical compression after about 30 minutes, while the C fibres continue to function [1,2,3,17,18].

Many techniques have been tried in order to decrease the incidence or severity of this pain, but the only thing that works reliably is tourniquet deflation. If the patient is under spinal or epidural anaesthesia, the loss of sensation to touch, not just pinprick, should extend above tourniquet level, as touch is consistently detected several dermatomes below those that are insensitive to pinprick or cold [22], and where there is touch sensation, pain will occur. Other methods used to try to decrease the incidence of pain include the addition of adrenaline to the local anaesthetic, the type of local anaesthetic (less pain with bupivacaine than with tetracaine [20, 21], less pain with isobaric than with hyperbaric bupivacaine [21]), addition of clonidine or morphine [23] and alteration of the dose of local anaesthetic [20]. The onset of "tourniquet pain" has been delayed by the application of EMLA cream to the tourniquet site and by subcutaneous infiltration of local anaesthetic [19]. Estebe et al [18] looked at the effects of changes in cuff width and pressure on tourniquet pain and found that a wider cuff is less painful than a narrower one at lower pressures, and that the lower pressures are effective at occluding blood flow.

With tourniquet deflation, central venous pressure and mean arterial pressure decrease, reaching a maximum at 3 minutes and taking approximately 15 minutes to return to normal. The decrease may be profound and cardiac arrests have been reported [2, 3]. The decrease is a result of the combination of a shift of the blood volume back into the limb, a post – ischaemic reactive hyperaemia, bleeding from nonligated vessels [24] and washout of metabolites from the ischaemic areas into the systemic circulation [25]. The cardiac index increases to compensate, mainly by an increase in the myocardial inotropic state [25]. The mean decrease in systolic blood pressure is 14 – 19 mmHg and the mean increase in heart rate is 6 – 12 beats/minute [2].

RESPIRATORY EFFECTS

As the tourniquet is deflated and the limb reperfuses, CO_2 and metabolites, e.g. lactate, are returned to the systemic circulation. The end tidal CO_2 ($ETCO_2$) increases by 0.75 – 18 mmHg – it is greater with the lower limb than the upper [1], and greater in men than in women, because of a man's greater muscle bulk [27]. Bourke et al [26] found that the increase in $ETCO_2$ was related to ischaemic time. The $ETCO_2$ peaks at 1 – 3 minutes, returning to baseline at 10 – 13 minutes in a spontaneously breathing patient [1,26,27]. The increase in $ETCO_2$ will be prolonged in mechanically ventilated patients unless the minute volume is increased. The mixed venous saturation decreases transiently, but a drop in the arterial saturation is unusual [2].

CEREBRAL CIRCULATORY EFFECTS

Middle cerebral artery flow increases after tourniquet deflation, related to the increased $ETCO_2$ [28]. This increase is larger with lower limb surgery than with upper [28]. Patients with reduced intracranial compliance may be at higher risk for adverse effects related to the increase in cerebral blood flow [28]. Maintenance of normocapnia prevents the increase in cerebral blood flow [28,29].

HAEMATOLOGICAL EFFECTS

The tourniquet causes changes in both coagulability and fibrinolysis. Despite the fact that tourniquets cause venous stasis (and would therefore be presumed to cause thrombosis), tourniquets do not cause deep vein thrombosis [30,31,32]. Tissue damage induces coagulation factors and activates platelets. Pain (surgical and tourniquet) provokes catecholamine release, exacerbating the state of hypercoagulability [33]. Tissue ischaemia causes tissue plasminogen activator release, activating the antithrombin III and thrombomodulin – protein C anticoagulant system in the affected limb [1,34]. This enhanced fibrinolysis may play a protective role in deep vein thrombosis development [34]. Thrombolytic activity in the peripheral blood increases after tourniquet release, reaching a peak at 15 minutes and returning to baseline by 30 minutes. It then falls below baseline for the next 48 hours [32].

While the tourniquet does not cause deep vein thrombosis, fatal pulmonary emboli have been reported at all stages of tourniquet use, from exsanguination to deflation [1,35,38]. Patients at high risk for deep vein thrombosis (DVT) and pulmonary embolism include those with lower limb trauma, prolonged immobilisation (greater than 3 days [2]) or a history of DVT's [1].

Venous embolism is common after tourniquet deflation, though it is not always clinically apparent [38] The maximum amount of thrombi appear 50 seconds after tourniquet deflation and emboli are not detected by transoesophageal echo 2 minutes after cuff deflation [36]. The embolus may consist of air, marrow contents, clot [38] or cement [37]. Venous embolism is more likely to occur when the marrow cavity is instrumented [35], but avoidance of such manoeuvres does not prevent it [37]. The type of surgery may also influence the incidence of pulmonary embolism – Hirota et al [35] found an increased incidence of pulmonary emboli in total knee arthroplasties compared with anterior cruciate ligament reconstructions. Parmet et al [37] compared the incidence of emboli in patients undergoing total knee arthroplasty with or without tourniquets. They found that those having the tourniquet had a 5.3 fold increased risk of large emboli.

Sickle cell Haemoglobinopathy

Sickling is predisposed to by circulatory stasis, acidosis and hypoxaemia, all of which happen with the use of a tourniquet. Systemic release of anaerobic metabolic products with cuff deflation may also induce sickling. Intravascular sickling may therefore theoretically occur with tourniquet use in susceptible patients, and this has indeed been reported [1]. Others have described the use of tourniquets, with no harmful effects [48,1,2].

TEMPERATURE CHANGES

In both adults and children, core temperature increases during tourniquet use [39,40]. Tourniquet inflation decreases heat transfer from the central to peripheral compartment, decreases the surface area available for heat loss and decreases the heat loss from the distal skin, allowing the temperature to rise. The increase in temperature may sometimes be larger than predicted, and it is postulated that a slow release of ischaemic metabolites, which raise the temperature, may occur via the bone [40]. In children the temperature may rise by as much as 1 – 1.7° C [39]. After cuff deflation, a "redistribution hypothermia" may occur as the cold extremity is reperfused [40].

METABOLIC CHANGES

With reperfusion of the affected limb, potassium, lactate, CO_2 and other ischaemic metabolites are washed into the systemic circulation. Potassium and lactate concentrations increase for approximately 30 minutes and pH decreases transiently [1,3,24].

Oxygen consumption (VO_2) increases by 55% and CO_2 production (VCO_2) by 80% 2 minutes post release, normalizing by 8 – 10 minutes [2,25,27]. This increase in VO_2 provides the energy needed to replenish both the high-energy phosphate and oxygen stores depleted during ischaemia (fast component), and the energy needed to convert lactate to glycogen (slow component) [25]. These changes may be smaller in females because of their smaller muscle mass [27]. Others have found that the changes may be proportional to the duration of ischaemia [25] or body surface area [27]. The metabolic changes are not related to occlusion pressure [1].

DRUG KINETICS

Tourniquet inflation isolates the limb from the rest of the body, altering the volume of distribution, sequestering drugs in the limb (if given before inflation), or preventing them from reaching the limb (if given after inflation) [1]. In order to prevent postoperative infection, prophylactic antibiotics need to reach the tissues in adequate concentrations before tourniquet inflation – at least 5 minutes is required [41,45]. Fentanyl and midazolam sequestered in the limb are released into the systemic circulation after cuff deflation. These increased levels may be clinically significant, especially in the elderly, and prolonged postoperative observation (up to 4 hours) is necessary [42]. Vecuronium, on the other hand, does not show clinically significant increases after tourniquet release, and the reversibility of the block is not affected [43]. When nondepolarising muscle relaxants are given prior to tourniquet inflation, the onset of the blockade in the isolated limb is altered with rocuronium but not with vecuronium [44].

RECOMMENDATIONS FOR SAFE TOURNIQUET USE

Tourniquets should undergo regular maintenance. Prior to use, they should be inspected for cleanliness, integrity and function. The appropriate gas for compression should be selected (never oxygen or nitrous oxide because of the fire risk). An appropriate cuff must be selected – the widest one possible as a wider cuff occludes at lower pressures. Contoured cuffs are appropriate for tapered limbs. The cuff should overlap by 7 – 15 cm. The cuff should be positioned around the widest circumference of the limb, with soft, wrinkle-free padding beneath it. Once inflated, the cuff should not be readjusted. The cuff and padding should be kept dry [46].

It must be remembered that nerves are susceptible to mechanical pressure and muscles are susceptible to prolonged ischaemia. Safe limits for time and pressure are controversial, but the shortest possible time at the lowest pressure is safest. An upper limit of 1.5 – 2 hours in healthy patients has been described, with shorter times, if it is used at all, in patients with peripheral vascular disease/ the elderly/ trauma patients [1]. If it is absolutely necessary to use a tourniquet for longer, it should be deflated every 1 – 2 hours for 10 – 15 minutes to allow for reperfusion of the limb [1,2].

Documentation should include the following:

- Cuff location
- Skin protection
- Pressure
- Tourniquet time
- Skin and tissue integrity before and after use
- Evaluation of the entire extremity
- Identification/ serial number and model of the tourniquet
- Identity of the person who applied it[46]

Recommended pressures are 50 (arm) – 150 (leg) mmHg above systolic blood pressure [1]. The tourniquet should be inflated and deflated as quickly as possible to prevent blood from being trapped in the limb when arterial blood pressure is greater than tourniquet pressure, and the veins are occluded [2].

Reusable tourniquets (of the type used for taking blood/ inserting drips) have been shown to have the potential for disease transmission [47]. Kam et al [1] questioned whether the same might apply to pneumatic tourniquets.

From the above discussion it is clear that there are many possible problems associated with tourniquet use. Contraindications to tourniquet use include the following:

- Peripheral vascular disease
- Severe trauma to the limb
- Head injury/ CNS disorder, peripheral neuropathy
- Severe infection of the limb
- DVT in the limb
- Severe arthritic changes/ bony spurs/ previous fracture of the limb
- Poor skin condition of the limb
- Arteriovenous (AV) fistula
- Lack of appropriate equipment
- Sickle cell haemoglobinopathy[2]

CONCLUSION

Tourniquets are widely used and help to provide optimal operating conditions. However there are many potential problems associated with their use. Thus, one should weigh up the risks versus the benefits for each patient.

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