ARTERIAL TOURNIQUETS
ANAESTHESIA TUTORIAL OF THE WEEK 200

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Arterial tourniquets are widely used in surgery on the extremities but may have significant systemic as well as local effects. This tutorial examines the adverse effects of tourniquets, their mechanisms of action and how to minimise these in order to achieve maximum effect and ensure patient safety.

SELF ASSESSMENT (MULTIPLE CHOICE QUESTIONS)

Before continuing, try to answer the following questions. The answers can be found at the end of the article, together with an explanation.

1. Pathophysiological effects of an arterial tourniquet are
   a. After tourniquet inflation, there is a continuous rise in systolic blood pressure and central venous pressure.
   b. After deflation, the rise in end-tidal carbon dioxide tension is greater in lower limb tourniquets than upper limb tourniquets.
   c. The increase in circulatory blood volume after deflation can cause secondary brain injury in patients with normal intracranial pressure.
   d. They increase the incidence of post-operative deep vein thrombosis.
   e. The rate of increase in carbon dioxide is three times greater than the rate of increase in oxygen consumption.

2. Contraindications for tourniquet use are
   a. Sickle cell disease
   b. Severe crush injuries
   c. Deep vein thrombosis
   d. Severe limb infections
   e. Diabetes

3. Regarding local effects due to arterial tourniquets, following are true
   a. Marked changes in mitochondrial morphology are visible after 1 hour of ischaemia.
   b. Post-tourniquet syndrome typically lasts 1-6 months.
   c. After inflation, ischaemic damage to nerves causes tourniquet paralysis.
   d. Intracellular acidosis is purely due to anaerobic metabolism.
   e. The initial physiological conduction block is due to mechanical compression of the nerves.

4. Complications of tourniquets are
   a. Acute vascular insufficiency
   b. Compartment syndrome
   c. Chemical burns are the most common type of skin injury
   d. Intra-operative bleeding
   e. Pulmonary embolism
INTRODUCTION

Jean Louis Petit coined the word “Tourniquet” from the French word tourner (to turn) in 1718 when he used them for lower limb amputations to reduce blood loss. Arterial tourniquets are usually pneumatic devices consisting of an inflatable cuff connected to a compressed gas supply. The high pressures generated can be measured and allow controlled arterial compression.

The most common indication for tourniquet usage is in surgery on the extremities (such as joint replacements and neurovascular bundle repairs) and provides a bloodless operative field and decreases the peri-operative blood loss. They are also used in intravenous regional anaesthesia (Bier’s block), management of complex regional pain syndromes and in the management of localised malignancy for isolated limb perfusion. Its use in the pre-hospital setting and in battle fields is controversial. However, there is growing evidence suggesting correct usage of tourniquet saves lives by improving haemorrhage control.

Tourniquet usage is associated with several risks and benefits. Therefore, personnel in the operating department including the anaesthetist should be aware of the changes that occur during and after its use. This article addresses certain important issues associated with the use of arterial tourniquets which can have important implications for anaesthetists and allow them to use tourniquets safely and effectively.

PATHOPHYSIOLOGY

Both systemic and local effects occur during tourniquet usage. Systemic effects are usually related to the inflation and deflation of tourniquets whereas local effects are usually due to ischaemia or direct mechanical pressure.

Systemic effects

Cardiovascular system

Although cardiovascular changes are clinically insignificant in a healthy patient, tourniquets can cause detrimental effects in patients with cardiac dysfunction. Cardiac arrest and circulatory overload have been reported after the usage of bilateral thigh tourniquets in the medical literature. Tourniquet inflation causes a shift of blood volume into the central circulation and increases the systemic vascular resistance. This is seen as a transient increase in systolic blood pressure and central venous pressure. After 30 – 60 minutes, there is a second, gradual rise in arterial blood pressure and heart rate probably related to ‘tourniquet pain’.

Tourniquet deflation leads to the redistribution of the circulating volume back into the limb and post ischaemic reactive hyperaemia. Metabolites accumulated in the limb are released into the systemic circulation. All these will lead to a transient fall in arterial blood pressure and central venous pressure. Cardiac dysrhythmias rarely occur. The literature suggests that a propofol based anaesthetic may attenuate the reperfusion injury as it has been shown to limit superoxide generation.

Respiratory system

Tourniquet inflation has minimal effects on the respiratory system where as deflation leads to an immediate increase in end-tidal concentration of carbon dioxide ($F_{CO2}$) which peaks at 1 minute. It usually returns to baseline within 10minutes. The increase is greater with release of lower limb tourniquets than upper limb tourniquets. Deflation leads to an increase in cardiac output and the hypercapnic venous blood in the ischaemic limb causes the increase in $F_{CO2}$. This is seen as increase in minute ventilation in spontaneously breathing patients. In controlled ventilation, increasing the minute ventilation for 5 minutes prior to deflation keeps the increase in carbon dioxide levels to a minimum.
**Central nervous system**

The rapid increase in $F_{\text{E}CO_2}$ after tourniquet deflation increases the cerebral blood flow which peaks at around 2 minutes and returns to baseline within 10 minutes. There is published evidence to suggest there is a 50% increase in middle cerebral artery velocity during this period. This may prove detrimental by worsening the secondary brain injury in patients with increased intracranial pressure such as that seen in head injuries. Maintaining normocapnia can prevent this increase in cerebral blood flow during deflation.

**Haematological changes**

The effects on the haematological system are complex. Tourniquet inflation is associated initially with an overall state of hypercoagulability whereas towards the later stage of inflation and after deflation an increased thrombolytic effect is seen. Pain due to surgery and the tourniquet itself release catecholamines which may cause platelet aggregation. Tissue compression from the application of Esmarch bandages contributes to this increased platelet aggregation. These effects manifest themselves as a systemic hypercoagulability. Despite this hypercoagulable state, tourniquets do not increase the incidence of deep vein thrombosis. A brief period of reduced coagulation is produced after deflation of the tourniquet. Tissue plasminogen activator is released due to the ischaemic effects after inflation which leads to hypoxemia and tissue acidosis. These cause systemic fibrinolysis after the tourniquet is deflated. This transient fibrinolysis is one of the contributors in post tourniquet bleeding.

**Temperature regulation**

Core body temperature is gradually increased after tourniquet inflation due to the decreased heat loss from the affected limb and the reduced metabolic heat transfer from the central compartment to the peripheral compartment. Deflation leads to a transient fall in core temperature due to the redistribution of body heat. Hypothermic blood from the ischaemic limb increases the fall in core temperature.

**Changes in Metabolism**

Metabolic changes almost always follow tourniquet deflation and these changes are fully reversed within 30 minutes. Tourniquet release after 1-2 hours of limb ischaemia increases the plasma potassium and lactate concentrations. Arterial pH of the systemic circulation falls due to the increased lactate and carbon dioxide from the ischaemic limb. Depending on the duration of tourniquet inflation, oxygen consumption and carbon dioxide release increase after deflation, the rate of the latter exceeding the former by a ratio of 3:1.

**Local effects**

These occur as a result of ischaemia and mechanical compression of the tissues underneath and distal to the tourniquet.

**Nerve**

Supra-systolic pressure leads to development of a reversible physiological conduction block in both motor and sensory nerves approximately 15 – 45 minutes after cuff inflation. Published evidence suggests ischaemia to be the cause of such a conduction block as its rate of development is same with cuff pressures of 150 or 300 mmHg. ‘Tourniquet Paralysis’ accounts for the second, long lasting nerve conduction block. Mechanical compression seems to be more responsible for this than ischaemia. Morphological changes are seen in the large myelinated nerves especially underneath the proximal and distal edges of the tourniquet after 2 hours of inflation. This can last as long as 6 months, but permanent changes are rare.
**Muscle**

Muscle cells experience progressive tissue hypoxia and hypercapnia following cuff inflation. Energy stores are progressively consumed and finally exhausted. Hypercapnia along with anaerobic metabolism is implicated in the development of intracellular acidosis and even inflation times as short as 60 minutes can produce morphological changes in mitochondria and local fibre necrosis. Ischaemia lasting longer than 2 hours can cause microvascular injury in the muscle. After cuff deflation, the patient can develop a pale, swollen and a stiff limb with weakness but no paralysis. This condition is termed ‘Post-tourniquet syndrome’ and is due to an increased vascular permeability leading to interstitial and intracellular oedema.

**TOURNIQUET PAIN and TOURNIQUET INDUCED HYPERTENSION**

In awake patients, this is experienced as a vague, dull aching pain in the affected limb associated with an increased blood pressure. Patients under general anaesthesia manifest it as an increased heart rate and arterial pressure. It is extremely difficult to prevent or treat the tourniquet pain. Several theories exist explaining the mechanism of this pain. It is suggested that unmyelinated, slow C-fibres that are normally inhibited by large, fast, myelinated A fibres mediate this pain. Approximately 30 minutes after cuff inflation, A-delta fibres are thought to be blocked before the C-fibres by mechanical compression and this seems to take away the post-synaptic inhibition of C-fibres by the large fibres. The skin under the inflated tourniquet provides the continuous stimulation of these C-fibres. Associated increases in arterial pressure and heart rate are humoral responses to the pain which, again, are a challenge to treat. Tourniquet pain is a problem even in patients with an effective central neuraxial blockade. This is supported by an in-vitro finding that smaller C-fibres are more resistant to local anaesthetics. C-fibres recover from intra-thecal block before A-fibres and start conducting dull tourniquet pain. There is no convincing evidence in the literature to suggest that there is an ideal way to treat this tourniquet pain. Numerous studies recommend various techniques such as the addition of adjuncts to the local anaesthetic solution (clonidine, morphine, epinephrine, alkalisation of local anaesthetic), low dose intravenous ketamine, pre-operative gabapentin, but none has attained complete success in tourniquet pain relief.

**TOURNIQUET PRESSURES and DURATION OF INFLATION**

Prolonged ischaemia generally affects muscles and mechanical pressure damages nerves. No strict guidelines are found on safe limits of pressures and time of inflation. It is logical to use the lowest pressure that causes arterial occlusion for the shortest time possible. Common practice is to either use fixed pressures (250mmHg for upper limbs and 300mmHg for lower limbs) or inflate by a certain fixed pressure above systolic blood pressure (SBP) (100mmHg above SBP for upper limbs and 100-150mmHg for lower limbs). The former practice has been criticized as it ignores individual patient’s arterial pressure. This can lead to over-inflation, especially in younger patients as their SBP can be much lower. The American Association of peri-operative Registered Nurses (AORN) recommends inflation pressures to be based on limb occlusion pressures (LOP). LOP is the pressure in the cuff at which the arterial pulse distal to the tourniquet disappears and the distal blood flow which denotes the arterial pulse is assessed using a Doppler probe. LOP is usually higher than the pressure that is actually transmitted to the extremities and is also dependent on the circumference of the limbs. This explains why LOPs for a thigh is higher than that for an arm. The recommendations add a safety margin to the LOPs to compensate for the intra-operative blood pressure fluctuations (table1).

**Table 1: AORN recommendations**

<table>
<thead>
<tr>
<th>LOP (mmHg)</th>
<th>Safety margin (mmHg)</th>
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<tbody>
<tr>
<td>&lt;130</td>
<td>40</td>
</tr>
<tr>
<td>131-190</td>
<td>60</td>
</tr>
<tr>
<td>&gt;190</td>
<td>80</td>
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Most recommendations in the literature suggest the duration of inflation should not exceed 90 – 120 minutes in healthy adults. There is, approximately, a threefold increase in the risk of neurological complications for each 30-minute increase in inflation time. General opinion is that the tourniquet should be left deflated for at least 10 minutes once the tourniquet time has exceeded the safe limit. This is to establish a period of reperfusion and it corresponds to the restoration of ATP in the muscle cells. Safe practice should include exsanguinations of the limb prior to tourniquet re-inflation.

**DRUG KINETICS**

Drugs administered before tourniquet inflation can accumulate in the isolated limb and there is a possibility of redistribution after deflation, whereas drugs administered after inflation may have altered pharmacokinetics due to the change in the blood volume. The clinical significance of such variation in drug pharmacokinetics is limited. However, clinical evidence suggests that prophylactic antibiotics need to be given at least 5 minutes before tourniquet inflation to allow good tissue penetration. (The Isolated Forearm Technique is still occasionally used to assess the depth of anaesthesia.)

**CONTRAINDICATIONS**

There are no absolute contraindications to tourniquet application but adequate care should be taken in the following group of patients while using them.

- Severe peripheral vascular disease
- Sickle cell disease
- Severe crush injury
- Diabetic neuropathic patients
- Patients with history of deep vein thrombosis and pulmonary embolism

**COMPLICATIONS**

Complications are rare when the tourniquets are used carefully. Odinsson and Finsen carried out a large study on the use of tourniquets in Norway over a 2 year period and showed the incidence of complications to be only 0.04%.

**Muscle injury**

Rhabdomyolysis directly related to tourniquet use has been reported but, fortunately, is extremely rare. A swollen and oedematous ischaemic limb in conjunction with a reperfusion hyperaemia may also lead to a compartment syndrome but, again, this is very rare. The combined effects of muscle ischaemia, oedema and microvascular congestion lead to the “post-tourniquet syndrome”, which is characterised by subjective numbness of the limb without objective anaesthesia. It is probably the one morbidity associated with tourniquets which least is appreciated.

**Nerve injury**

Neurological injuries are probably the most common complications associated with tourniquets and they range from paraesthesia to paralysis. Mechanical pressure and faulty aneroid gauges have been implicated in these nerve injuries. The radial nerve, followed by the ulnar and median nerves in the upper limb and the sciatic nerve in the lower limb are most commonly involved and it would appear that large diameter nerve fibres are more commonly affected. Using an Esmarch bandage increases the likelihood of causing nerve injuries and this may explain the fact that nerves are more susceptible to mechanical pressure. The effects of nerve compression at the tourniquet site may make injury caused by ischaemia or surgical trauma at a more distal site more likely. Most of them resolve spontaneously within 6 months, however, and only on rare occasions is the damage permanent.
Skin injury

This uncommon complication is due to pressure necrosis or friction burns caused by movement of badly applied tourniquets. Chemical burns have been reported especially in extreme age groups and are caused by cleaning solutions used for skin preparation. These solutions seep beneath the tourniquets causing chronic exposure under pressure to the skin.

Vascular injury

These rare catastrophic complications are thought to occur when mechanical pressure from the tourniquet damages the atheromatous plaques in the blood vessels leading to plaque rupture. Ultimately, they can lead to the requirement for amputation of a limb. Therefore, caution should be exercised before using the tourniquet in patients with severe peripheral vascular disease.

Intra-operative bleeding

A poorly fitting or an inadequately inflated cuff as well as incomplete exsanguinations of the limb can increase intra-operative bleeding. Furthermore, increased intravascular coagulation and fibrinolysis can contribute to excessive bleeding.

Pulmonary embolism (PE)

Rare episodes of PE have been reported during leg exsanguination, both after inflation and following deflation. These can occur more commonly in patients with a history of deep vein thrombosis and atherosclerotic vessels.

TOURNIQUET SAFETY

Most complications associated with tourniquet usage are due to human errors or equipment failure. Hence good clinical practice is paramount in prevention. There are several recommendations which help to ensure the safer use of tourniquets. All appropriate staff should be properly trained in their use.

Before applying a tourniquet:

- Regular maintenance checks of tourniquets are essential to ensure their reliability.
- Aneroid pressure gauges should be checked against calibration devices.
- The whole system should be leak proof.
- There should be no fluctuations in pressure with time.
- Thorough visual inspection should be carried out prior to use.
- Cuffs should exceed the circumference of the limb by 7 – 15 cm.
- Cuff width should be appropriate to the size of the patient.
- Certain group of patients are at high risk for tourniquet application – eg. morbidly obese patients, those who have had a prolonged immobilisation prior to surgery, patients with deep vein thrombosis, isolated limb malignancies and infections, patients with left ventricular dysfunction. Appropriate care should be taken before exsanguination in these patients.

During use:

- Pressure gauges should be continually monitored to detect pressure variations.
- Cuffs should be placed at a point of maximum circumference of the limb.
- Adequate padding, avoiding folds, should be placed underneath the tourniquet.
- Once applied, the cuff should not be rotated into a new position.
- Skin cleaning solutions should not be allowed to seep or accumulate under the cuff.
- Tourniquet inflation time and pressure should be kept to a minimum and should relate to the patient’s age and comorbidities.
- Surgeons should be kept informed of the inflation times.
- Appropriate documentation of tourniquet usage should be recorded in the patient’s notes.

After the use:

- Reusable cuffs should be thoroughly cleaned to prevent cross infection.
- The affected limb should be thoroughly inspected.
SUMMARY

• It is important to be aware of the physiological effects of tourniquets and the implications of this in anaesthesia as they may affect any system in the body.
• There are no absolute contraindications to tourniquet use. However, care should be taken in high risk patients.
• Tourniquet pain and tourniquet induced hypertension are very difficult to prevent and treat.
• Pharmacokinetics of drugs can be altered during tourniquet use and antibiotic timing is very important in these patients.
• Complications are rare, but if they occur, they can be devastating to patients.
• Tourniquet inflation pressures and duration should be carefully monitored and it is the responsibility of everyone involved in patient care to ensure to be within recommendations.
• Tourniquet safety checks should be carried out at regular intervals

ANSWERS TO QUESTIONS

1. F T F F T
   There is a transient increase in SBP and CVP. Secondary brain injury may happen in patients with increased intra-cranial pressure such as Head injury. There is no evidence to suggest that tourniquets increase the incidence of DVT.

2. T T T T F
   There are no absolute contraindications to tourniquet use. Care should be taken in patients with diabetic neuropathy.

3. T F F F F
   Post tourniquet syndrome typically lasts for 1 – 6 weeks. Normally, nerves are susceptible to mechanical pressure and muscles to ischaemia. Intracellular acidosis is due to both anaerobic metabolism and increased carbon dioxide production. The initial physiological conduction block is due to ischaemia and the second, long lasting block is due to mechanical pressure.

4. T T T T T

REFERENCES AND FURTHER READING

