The Ischemic Limb and Regional Anesthesia

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INTRODUCTION

There are two basic clinical scenarios;

1. The patient who develops unexpected limb arterial ischemia subsequent to undergoing a neuraxial or peripheral nerve block. There are three subgroups of unexpected limb ischemia;
   - The limb ischemia is random event unrelated to the nerve block intervention or the surgical invention.
   - The limb ischemia is a direct consequence of the nerve block.
   - The limb ischemia is a direct consequence of the surgery or the surgical pathology.

   It may not initially be apparent into which subgroup a patients falls.

2. The patient who has a known risk of compartment syndrome, and who undergoes a nerve block for analgesia of that limb, for a bone fracture and surgery.

   Clinically the differentiation of arterial limb ischemia and compartment syndrome may not be clear, and the syndromes may be concurrent and overlapping.

   The big late problem for the anesthesiologist is that the nerve block can be incriminated in obscuring the diagnosis of limb ischemia and delaying the timely recognition of the ischemia of whatever cause. Limb ischemia can result in death, amputation of the limb, or loss of limb function. This problem is amplified by the fact that most surgeons are fairly ignorant of the exact effects of nerve blocks and when limb ischemia develops in the presence of neuronal block the anesthesiologist who has detailed knowledge and experience of nerve block effects can play a major role in the diagnosis of limb ischemia being made being made on time and the limb being saved.

   It is important for any anesthesia provider to have basic knowledge of this subject in order to act as consultant about these problems, even if they do not do perform regional anesthesia. An anesthesia provider’s good advice on managing a patient who has had regional anesthesia and has a possible ischemic limb can greatly help prevent serious patient morbidity and mortality.

   It is also important to know when to use or not use regional anesthesia when limb ischemia is already present or potentially able to develop. Regional anesthesia is not always absolutely contraindicated in potential limb ischemia but patient care has to be specifically focused after the nerve block.
CASES FOR DISCUSSION:

CASE #1: The ischemic leg and the epidural:

A fourteen-year old girl undergoes a twelve hour anesthetic for an intestinal procedure. A routine general anesthetic is given, with an epidural block for post-operative analgesia. The patient is operated in the supine position with the legs in lithotomy. The surgeons observe in the evening that one leg is moving slightly and the other not at all. They attribute this to the continuous epidural block.

The next day an anesthesiologist routinely checks the girl’s epidural block and analgesia. It is noted her left foot is immobile. This was considered very abnormal because the epidural infusion is a non-paralyzing concentration of local anesthetic. There is evidence of reduced foot perfusion but not absent perfusion. The legs are not painful. The anesthesiologist communicates concerns to the surgical team.

On the second day after surgery the anesthesiologist sees the patient and again feels great concern for the persistent motor paralysis in the feet. It was considered absolutely not due to the epidural analgesia. After much activity and communication driven by the anesthesiologist bilateral compartment syndromes are diagnosed and fasciotomies are performed. Ultimately a below knee amputation is done on the worst affected leg.

The surgeon’s argue strongly that the epidural obscured the diagnosis of the bilateral calf compartment syndromes. The anesthesiologists strongly counter argue the point. The compartment syndromes are accepted to have resulted from the continuous 12 hours the patient spent in lithotomy with the primary surgery.

Key observations:

- Critical signs of the compartment syndrome were present on the first evening after surgery, even though fasciotomies were only done 36 hours later.
- The epidural did not prevent the diagnosis of compartment syndrome being made, once the diagnosis was actually considered.
- The paretic foot was initially, in ignorance, wrongly attributed to the epidural block.
- Earlier diagnosis of compartment syndrome would likely have prevented the later need for a leg amputation.

CASE #2: The ischemic foot after vascular repair to the leg.

A middle aged man underwent a four-hour right-sided repair and graft of the femoral artery to the tibial artery in the lower leg. The patient received a general anesthetic and a psoas compartment block plus parasacral sciatic nerve block. After regaining full consciousness after surgery and anesthesia the patient complained of pain in the right foot. The anesthesiologist immediately returned to the patient and examined him with the concern that the sciatic nerve block had somehow failed.
Upon examination of the patient the foot was Pulseless, Pale, Paresthetic, and Paralyzed. The surgeon was informed and without question had the patient immediately returned to the operating room for immediate reoperation. The surgeon had been silently harboring a technical concern about the one graft anastomosis all the time. Under general anesthesia the suspect graft anastomosis was revised. The foot regained its pulses and pink color, and the patient was pain free upon awakening from the second anesthetic.

**Key observation;**
- Ischemic pain can “break-through” an otherwise functional nerve block. The mechanism for this will be discussed.

**CASE #3. Knee arthroplasty after general anesthesia and nerve blocks with an unsuspected arterial injury.**

A seventy-plus year old lady underwent total knee arthroplasty. A general anesthetic was done, and femoral and sciatic nerve block performed for post-operative analgesia. The nerve blocks persisted to the next morning after surgery and the leg was still anesthetic and paralytic. This foot paralysis was of longer duration than usual after a single shot sciatic nerve block. The surgeon asked the anesthesiologist to evaluate the leg and remove the femoral nerve catheter. The surgeon did not see the patient himself. The lower leg sensation partially recovered by later in that day. Pulses were present all this time.

By the second day after surgery following day the calf was significantly painful and the surgical knee minimally painful. There was conflicting opinion on the presence of ankle pulses at this time. A popliteal fossa ultrasound examination excluded DVT, but did not assess arterial blood flow. An arterial injury to the knee arthroplasty was only recognized on the third day and despite vascular repair a partial foot amputation was finally needed. The surgeon blamed the anesthesiologist for not (1) “alerting him sufficiently” about the problem and (2) that the presence of the nerve blocks prevented the correct diagnosis being made timeously. The anesthesiologist refuted the surgical accusations.

**Key observation;**
- Surgical caused vascular injuries may not result in immediate complete arterial occlusion and the clinical picture may evolve in slow and confusing ways.
- The first sign of vascular compromise may be a slow recovery of a nerve block in the same leg.
- A surgeon is primarily responsible for his own patient’s post-surgical care at all times, particularly of mutterers that are absolutely surgical.

**CASE #4. Re-paralysis sets in after nerve block partially recovers.**

Patient undergoes trans-arterial axilla block for awake hand surgery. The surgery is successfully completed. The motor function of the arm returns after 8 hours and very slight pain starts after 12 hours but the patient is
content. At 18 hours the hands becomes weak again and the patient remains otherwise comfortable. At 24 hours after surgery the elbow becomes weak and the surgeon consultants the anesthesiologist about when the block will resolve. The patient is otherwise comfortable. The anesthesiologist sees the patient and is slightly perplexed and advises further observation. At thirty six hours after surgery the shoulder becomes a bit weak and pain starts to present in the forearm, proximal to the surgical site. There is growing concern that something is medically wrong. An axilla swelling is noted when the axilla is examined. Investigations reveal a hematoma of about 500 ml volume surrounding the neurovascular from the axilla extending into the infraclavicular zone. The hematoma is surgically explored and washed out and no specific bleeding point needs repair. The arm makes a slow recovery over 6 months but with some persistent residual weakness. It is finally realized that there was a compartment syndrome of just the neurovascular bundle in the axilla-infraclavicular region probably due to arterial bleeding after the trans-arterial axilla block technique.

Key observation;

- **Re-paralysis** after nerve block starts recovering is a very serious and urgent sign to investigate.
- **Paralysis that progressively climbs up a limb over time** is always a very ominous sign. (“climbing paralysis”)
- The need to operate, and explore an acute compressed nerve is as urgent as that for needing to operate an epidural hematoma. If surgery is delayed to later that about 8 hours after the first symptoms permanent lesions neurological are like to result.

Case #5. Nerve block fails to relieve post-surgical pain = first sign of compartment syndrome.

A patient underwent distal humerus fracture repair under general anesthesia. An infraclavicular block catheter was placed but not injected until after surgery. The patient was initially comfortable. At fourteen hours after surgery the patient developed severe pain in the forearm despite the running nerve block infusion. A perineural catheter top up of 20 ml 0.5% ropivacaine was injected that did not relieve the pain. Typically post-surgical blocks relieve pain dramatically and well before paresis develops. Compartment syndrome was immediately suspected, confirmed with pressure studies and the patient was swiftly operated with reasonable outcome.

Key observations;

- Ischemic pain of a compartment syndrome broke through a functioning nerve block and resisted treatment by nerve block top up with full strength drugs.
MAIN DISCUSSION

There are three sections for discussion;

1. Limb ischemia secondary to a nerve block.
2. Unexpected limb ischemia secondary to the surgical pathology or surgery, in the presence of a nerve block
3. Anticipated potential limb ischemia from compartment block, after limb trauma or surgery, in the presence of a nerve block.

1. LIMB ISCHEMIA SECONDARY TO A NERVE BLOCK INTERVENTION

This had been described secondary to Bier's block, also called Intravenous Regional Anesthesia (IVRA)¹. One case described ischemia resulted from unintentional intra-arterial injection of the nerve block drugs as all the hand arteries were thrombosed and ischemia was immediately apparent after release of tourniquet of short duration. It has also resulted from a recognized nerve block drug mixture errors. In one such case a large volume of hypertonic saline was used instead of normal saline, to dilute the local anesthetic.

2. UNEXPECTED LIMB ISCHEMIA SECONDARY TO SURGERY OR LIMB TRAUMA, WITH AN ASSOCIATED NERVE BLOCK.

These cases are uncommon, but often catastrophic due to poor understanding of the effects of regional anesthesia and the fact that limb ischemia is not considered initially. This leads to late diagnoses being made with high patient morbidity. Case #3 presented above is an example of this.

Unexpected limb ischemia can occur with any major orthopedic surgery associated with nerve blocks for post-surgical analgesia. Although such events are rare overall, they have been observed mostly following knee arthroplasty.

There is often loss of tissue and permanent loss of function primarily due to corrective vascular surgery and or fasciotomies being performed too late.

The reasons that the full and correct diagnosis being made late stems from three factors;

- The rarity of the condition contributes to ignorance and inexperience of the medical practitioners. Typically one only diagnoses conditions one thinks about. The remedy is specific education on to close this knowledge gap.
- The ischemic condition typically progresses slowly. Signs and symptoms are few initially, and then slowly accumulate. However
the very first signs and symptoms are fully compatible with the full and final diagnosis.

- **An accompanying nerve block may confuse the practitioners** initially but the nerve block does not obscure nor prevent the correct diagnosis being made. Almost always in descriptions of these cases it is the anesthesiologist who contributes significantly to the final and correct vascular diagnosis being made while the surgeon follows a very hands-off approach. It could be argued the presence of nerve block brought an anesthesiologist into the patient's post-operative care to the patients benefit.
  - Modern over-busy surgeons too often relegate critical patient care to physician extender PAs (Professional assistants) with limited medical training, to inexperienced surgical trainees, and to ward nurses. This contributes to the failure to recognize the earliest signs of ischemia for what they are.

The **Calligaro** vascular surgical perspective review showed acute arterial injury occurs in 0.17% of knee total arthroplasties and 0.08% of hip total arthroplasties. In 50% of cases the diagnosis was delayed until the day after surgery. Concomitant regional anesthesia was not identified as a risk factor in delaying the vascular injury diagnosis.

The **Pal** orthopedic series case review of 9 cases found 80% of arterial injuries following knee arthroplasty had serious morbidity. Pal also observed that delay in full diagnosis of the arterial injury was the main risk factor for morbidity. Pal did not attribute delays to associated nerve blocks and suggested best prevention lay in education and closing the knowledge gap.

The **Walker and Bosenberg** anesthesiology perspective of compartment syndromes stated that it could be argued ANY form of analgesia, such as PCA morphine, could equally or more so conceal, “abnormal pain” than a nerve block. It would also be unethical to withhold opiate medication from a patient with susceptibility for compartment syndrome and the argument could be extended that it thus not inappropriate to utilize nerve blocks in such patients.

The key points to raise concern for a limb that can point to a vascular injury are;
- Unexpected pain in the limb, similar to the pain of failed a failed nerve block.
- Earlier onset of severe pain after surgery than that expected from the typical duration of the nerve block.
- Pain in the presence of muscle palsy. More typically pain starts only when muscle strength returns or a modest while after that, but NEVER before that.
- **Anatomically Inappropriate pain** - Pain in the limb form an area not operated upon.
- Persistent weakness in a limb at a time the nerve block is expected to be resolved.
Persistent coldness in a leg after surgery under tourniquet. The surgical limb is commonly colder than the non-operated limb immediately after surgery but they should be both equally warm within single hours.

3. ANTICIPATED POTENTIAL LIMB ISCHEMIA SECONDARY from COMPARTMENT BLOCK TO SURGERY OR LIMB TRAUMA, WITH AN ASSOCIATED NERVE BLOCK.

This concerns the use of a nerve block where the injury or surgery has an association with the development of compartment syndrome. There is no good scientific evidence to rule that nerve blocks should be performed or not be performed in this scenario. The ASRA May 2013 Pro-Con debate on the subject illustrates this.

There are many highly significant benefits to the use of regional anesthesia in limb and orthopedic surgery. In the PRO argument for using regional anesthesia it is pointed out that there are at least six case reports of ischemic pain breaking through a nerve block. This author has had two personal cases of acute arterial occlusion present as pain in the presence of established working nerve blocks. The acute arterial occlusion was easily diagnosed while examining the limb while assessing a seeming failed nerve block after surgery in a patient with unexpected pain upon awakening. The patients had each undergone femoral popliteal arterial grafts under general anesthesia with psoas compartment blocks and sciatic nerve blocks for post-operative analgesia. The big argument against nerve blocks is that it will obscure signs of pain pointing towards a compartment syndrome. This is clearly untrue. Furthermore compartment syndrome can occur in the absence of pain without any nerve block present. The presence of both rest pain and calf-stretch-pain in a calf only, furthermore, only has a 14% sensitivity for the diagnosis of compartment syndrome in the calf.

In Europe and much of the world it is common to perform single limb orthopedic surgery under regional anesthesia only, e.g. humerus and forearm fractures.

The ASRA-PRO nerve block debaters argue that concern for a compartment syndrome indicates the need to monitor the patient more closely regardless, but does not justify omission of the benefits of regional anesthesia.

The ASRA-CON nerve block debaters however emphasize that the most important clinical signs of compartment syndrome require a conscious patient. The important early clinical signs are rest pain, increased pain on muscle stretch, and paresthesia. They thus argue anything that confuses that assessment should be avoided, namely nerve blocks. They could thus also argue against treating the pain with opiates, which would be universally seen as unethical. This is defensive medicine argument that does not consider the benefits of nerve blocks to patients.
This CON argument also does not consider the fact that there are no case reports of compartment syndrome in the presence of a nerve block where there were not suspicious case features recognized very early on and the prime problem was the physician’s lack of examining the patient sufficiently and lack of contemplating a vascular diagnosis. This reflects less a problem with nerve block and more an education gap of surgeons (and anesthesiologists too) to be closed.

Also, there is no controlled study of ischemic injuries in orthopedic limbs with a group receiving nerve blocks and a matching group who did not receive nerve blocks. Thus all evidence does not show nerve blocks delay the actual diagnosis of ischemia, although the cases reported do show much clinician failure to consider limb ischemia timeously. There are many case reports actually showing it was anesthesiological care of the nerve block that primarily lead to the surgeons to the correct diagnosis.

Arteries deliver blood to the limb and veins drain that blood away after it has passed through the capillary system. Capillary patency requires that capillary luminal pressure exceed the extrinsic tissue pressure about the capillary. Blood flows across the capillary if the arteriolar to venule pressure gradient is positive.

Causes of loss of tissue perfusion are (1) significant decrease in arterial pressure, (2) significant in venous pressure on the drainage side of the limb, and (3) significant increase in extrinsic tissue pressure in the limb. There is furthermore a time factor. Limb perfusion can be marginal and tissue viability can be sustained for certain period only. The viability period will depend on the severity of, for example, drop in proximal arterial pressure. Also different tissues have different critical ischemic periods after which necrosis can develop, which depend on temperature. Cooled tissues may survive longer. Approximate absolute warm ischemic periods that tissues can survive are as follows

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Safe survival in hours</th>
<th>Intermediate period- and potential consequences</th>
<th>Absolute irreversible necrosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin</td>
<td>2 hours</td>
<td>2 to 12 hours – Bed sores and variable dermis loss,</td>
<td>12-24</td>
</tr>
<tr>
<td>Bone</td>
<td>8 hours</td>
<td>8 to 12 hours</td>
<td>12-24</td>
</tr>
<tr>
<td>Muscle</td>
<td>2 hours</td>
<td>2 – 4 hours – significant muscle loss</td>
<td>4-6</td>
</tr>
<tr>
<td>Nerves</td>
<td>2 hours</td>
<td>2- 3 hours – Neuropraxia- from axon death – full recovery in months</td>
<td>8 hours - Permanent insensate limb. Schwann cells do not survive, there axons cannot regrow.</td>
</tr>
</tbody>
</table>

Intra-compartmental pressure; this can be measured in numerous ways including using the Stryker pressure monitor with insertion of a needle into the tissues. Studies and clinical experience suggest that with normal blood pressure and a Stryker pressure under 30 mm Hg no tissue damage will occur.
CONCLUSION

Important points to retain are that in patients who have had elective or emergency surgery to a limb and who have a nerve block;

- Always be alert to post-operative arterial ischemia or a compartment syndrome, because the limb is shared with the surgeon.
  o To think of one of these two conditions requires one to consider both conditions
- There are always early signs and symptoms of arterial ischemia and compartment syndrome.
  o The early signs and symptoms will be unusual in some way.
  o The early signs and symptoms may not permit a full and correct diagnosis to be made, but should red-flag the patient for re-assessment under 6 hours again.
- Ominous signs and symptoms are
  ❖ Pain that returns before motor function.
  ❖ Patients whose block recovers and then relapses.
  ❖ Climbing new paralysis or re-paralysis.
  ❖ Unusual pain; more severe than expected, sooner than expected, more analgesia used than expected
  ❖ Unusual paralysis, that last longer than expected.
  ❖ Note, the absence of pain does not exclude arterial ischemia nor compartment syndrome, even in a limb without a nerve black.

The anesthesiologist may be a very good person to advise the less than focused or informed surgeon to about the potential for ischemia, even if the diagnosis is not the anesthesiologist primary responsibility.

**Question.** Is it inappropriate to place a nerve block in a patient with forearm fracture, a tibia fracture or any other fracture?

**A** = NO. The nerve block does not alter the need to monitor the patient for arterial ischemia or compartment syndrome, and the nerve block does not prevent the diagnosis of arterial ischemia or compartment syndrome being made correctly and timeously. The nerve block may hold many advantages for the patient undergoing surgery as well as analgesia or other benefits after surgery.
ADDENDUM

Clinical causes of ACUTE limb ischemia:
1. Atherosclerotic arterial disease – diseased arteries can be injured by surgical handling and tourniquets and undergo acute occlusion.
2. Bone fractures - fracture bleeding into closed osseo-fascial compartments can increase the compartment pressure.
3. Limb compression during unconsciousness or sedation with patients lying too long in poor positions, e.g. lithotomy.
4. Anticoagulants - induce spontaneous bleeding into compartments without any recognized trauma to the tissues.
5. Intravenous fluid extravasation – this results from misdirected fluid therapy.
6. Infection causes limb swelling
7. Compartment syndrome - many causes

COMPARTMENT SYNDROME

This discussion considers only the acute form of compartment syndrome. Compartment syndrome occurs in any relatively closed fascial limb compartment containing of muscle and bone, that has blood vessels and nerves that past to more distal and where the fascial boundaries that restrict expansion. The syndrome develops from anything causing swelling of any of the tissues within the compartment. When the compartments capacity to buffer or absorb swelling is exceeded intra-compartmental pressures increases and the micro-vessels undergo compression. The extrinsic compressive pressures exceed the intrinsic intraluminal blood pressure the blood vessels cease to transmit blood. Venules occlude first due to having the lowest intraluminal pressure and this leads to added edema within the compartment due to fluid leakage. Next capillaries become occluded and tissue ischemia results. Different tissues will cease to function, according to their specific sensitivity to ischemia and different tissues will also undergo death and necrosis according to their specific ability to tolerate a period of anoxia. Dead muscle may need to be excised after blood flow is restored, nerves may be injured and take months to recover, and sometimes never recover.

Commonest sites of compartment syndrome are the lower leg, followed by the forearm. Compartment syndrome within the upper arm, hand, foot and thigh compartment syndromes do occur but are rarer.

Causes of compartment syndrome are;
1. Deep Venous Thrombosis (DVT).
2. IV fluid extravasation.
4. Pressure applied to outside of the limb;
   - Over tight bandages and wound dressings.
   - Over tight plaster casts.
   - Legs in lithotomy too long.
• The patient lying too long in one position; alcohol intoxication, long duration surgery, especially in lithotomy.

5. Crush injuries
6. Excessively long duration severe arterial hypotension. Mild persistent arterial hypotension can aggravate any other different risk factor for compartment syndrome

The diagnosis of compartment syndrome is based upon a triad of;

1. Recognizing the risks for compartment syndrome beforehand and having a heightened alertness.
2. Patient Clinical signs and symptoms
3. Special tests, such as measuring compartment pressures with devices like the Stryker pressure meter. There are numerous other ways to measure intra-compartmental pressures. It has been suggested an intra-compartmental pressure exceeding 30 mm Hg indicates a compartment syndrome. It has also been suggested that pressure difference between the diastolic pressure and the compartment exceeding 30 mmHg is critical to exceed lest tissues become ischemic.

CLINICAL SIGNS AND SYMPTOMS OF COMPARTMENT SYNDROME
The classics signs are the six “P”s;

I. Pain – out of proportion to that expected, or an increasing consumption of analgesia medications.
   ◊ Note however, that silent compartment syndrome can occur even in the absence of a nerve block7.
   Although pain is very typical of a compartment syndrome it is not a diagnostic requirement.

II. Paresthesia – numbness and tingling, even in a nerve blocked limb

III. Pallor - the distal tissues (hand or foot) may show evidence of reduced blood perfusion or slow capillary refill signs.

IV. Paralysis – a late sign

V. Pulselessness – it is important to note (a) this is very late sign, and (b) if there is one compartment in the limb not experiencing compartment syndrome but that transmits an artery to distal a distal pulse may be present in the presence of severe compartment syndrome in an adjacent limb compartment. Typically an advanced forearm compartment syndrome always ends with loss of all wrist pulses, but calf a severe posterior compartment syndrome may be accompanied by some still palpable ankle pulses.

VI. Pressure within the compartment is high. This can be palpated, or measured with device, as the Stryker intra-compartment portable monitor. The compartment (e.g. calf) will also appear swollen and shiny, and the skin can be
mottled or bruised and abnormal. A Stryker pressure over 30 mm Hg is considered a positive for compartment syndrome. It is noteworthy that compartment pressure does not have to exceed arterial pressure in order to cause irreversible muscle and nerve damage. That is, the presence of distal pulses do not exclude the fact that nerve and muscle necrosis is occurring.

The ONLY treatment of acute compartment syndrome is fasciotomy with possible treatment of the cause, such as artery repair or thrombolytic therapy for a DVT etcetera. In orthopedic surgery open fixation of the fracture includes an effective fasciotomy and control of bleeding. The risk of compartment syndrome is highest with closed treatment of fractures, and much lower after open fixation surgery. It is unusual to perform nerve blocks for patients with closed treated fractures as simple immobilization greatly reduces the injury pain. It is true that increasing pain in an un-operated immobilized fracture is a highly significant sign of compartment syndrome.

References

1 BOOK; Finucane’s Complications of Regional Anesthesia. 1999 Churchill Livingston. Page 216