Utilizing Problem-Based Learning in Anesthesiology Careers

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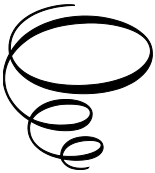
Edited by

Stacey Watt and Patricia Fogarty Mack

Cambridge Scholars Publishing



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CHAPTER ONE

INTRODUCTION TO PROBLEM-BASED LEARNING

THE IMPORTANCE OF PROBLEM-BASED LEARNING: HOW AND WHY IT MATTERS

PATRICIA FOGARTY MACK, MD
STACEY WATT, MD, MBA, FASA

The use of problem-based learning began as a method of teaching in the mid-1960s at McMaster University. This method of instruction spread quickly throughout the medical community and soon became a staple of medical education. In contrast to traditional instruction, problem-based learning is learner-centered in the context of a case or problem that requires resolution. In challenging the learners to solve open-ended and sometimes difficult questions, the participants must engage in active learning in pursuit of the resolution. Discussions are facilitated by a faculty member (or more advanced learner) who guides the discussion while ensuring all voices are heard.

The essential elements of a problem-based learning activity are small groups and guidance by a faculty member or advanced learner skilled at facilitating coactive learning discussions. Groups of between five and ten often provide enough learners to ensure active communication, but not so many as to inhibit some participants from engaging in the discussion. The faculty member should have experience in moderating discussions. Since problem-based learning is not a teacher-centered platform of learning, in which the faculty member lectures on a topic and the learners are often passive

recipients, the faculty member must learn to guide discussions through questioning.

To set up a successful problem-based learning discussion (PBLD) session, the writer must pay close attention to the objectives of the PBLD activity in addition to the scenario that will be described to the learners. What do you want your learners to take away from the session? This step should be the first of writing a PBLD and should take the most time. Objectives that start with active words such as "list, compare, differentiate, demonstrate, explain, identify, or locate" are great examples of successful objectives. Objectives that convey a vague knowledge of the topic, such as "know" or "understand," should be avoided.

The next step, after writing objectives, is to design the scenario of the PBLD. How will the problem the learners need to solve emerge through the scene you describe? The scenario should be interesting and provide multiple avenues for conversation. Try to avoid obvious or easy scenarios. The goal is to challenge your learners to think through the case with which they are presented. Remember to include some guiding questions within your case discussion. Choose questions that will stimulate your learners to ask more questions or clarify the ideas and plans they discuss during the PBLD. Guiding questions can be included or withheld from the PBLD scenario that is shared with the learners prior to the activity. Some moderators like to have the learners think about questions before the session, while others choose to leave the guiding questions for the discussion itself to help move the discussion forward. After you have written the objectives and case scenario, do some further research regarding the topic to ensure you are able to successfully guide a discussion through the stages of discovery as the case evolves. Once you and the learners are prepared for an active discussion, the PBLD activity can commence.

As you sit your group down for the discussion, it is important to revisit the learning objectives and unofficial rules of the PBLD. The unofficial rules are: to be respectful to all learners involved within the activity; to allow everyone to join in the conversation; and to encourage the sharing of ideas and asking of questions. Once you have set forth the ground rules you can introduce the case. The best way to warm up the PBLD group is to start with a simple question. From that point on, the moderator's job is to keep the conversation moving and not to lecture the learners about the topic material. It is important for the moderator to not monopolize the conversation, but serve as a catalyst for conversations between the learners.

PBLD activities should take approximately forty-five minutes to one hour to complete. After the activity concludes, take a minute to ask how the learners feel about the activity. You can also ask them to provide feedback on how to improve the activity, or ask what questions they would include in the discussion the next time the PBLD is given. Feedback is valuable for not only the other learners but also the moderator.

We hope that by reading this book you will be inspired to include problem-based learning in your educational tool belt. Use these cases as an introduction to problem-based learning, but be encouraged to go forth and create your own PBLDs, and even join the ranks of PBLD moderators at national and international meetings!

CHAPTER TWO

ACUTE AND CHRONIC PAIN

TOPIC: THE STELLATE GANGLION BLOCK IN COMPLEX REGIONAL PAIN SYNDROMES

SAPAN SHAH, MD
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Objectives

(1) Identify complex regional pain syndromes (CRPS) based on clinical signs and symptoms
(2) Diagnose CRPS based on established criteria and diagnostic tools
(3) Understand treatment strategies for CRPS
(4) Understand the utility of the stellate ganglion block (SGB) in CRPS

Background

A forty-two-year-old retired veteran presents with arm pain and swelling after being struck by a small caliber firearm. He has comorbid depression and generalized anxiety. His gunshot wound was superficial and was successfully repaired with skin and fascial sutures. In the six months since the injury, he has continued to experience increasing pain which extends from the left biceps to the left hand. There is no clear dermatomal distribution. He also notes intermittent numbness in the area of pain and the range of motion at the elbow and wrist are decreased. The whole arm is extremely sensitive, so he does not allow anyone to touch his arm. He denies fevers, chills, loss of weight, or appetite. There is no rash or redness. He has no neurologic deficit. There are no issues with ambulation. On physical examination there is a well-healed, superficial laceration on his left biceps. He has tactile allodynia throughout the left arm. There is no sensory deficit.

There is a mild contracture at his left elbow, and mild swelling noted around the wrist. The left forearm is hairless and feels damp to the touch, with skin mottling. He has effort limited weakness in the wrist and elbow as well as decreased hand-grip strength. The remainder of his physical examination is unremarkable. He is unable to participate in physical therapy because of pain. He has been unable to tolerate the sedative effects of several medications that he has trialed, including pregablin, baclofen, and oxycodone. He came in because he wanted to consider some nerve blocks that could help him tolerate the pain.

Past medical history – anxiety, depression

Past surgical history – none

Allergies – none

Social history – nonsmoker, no illicit drug use, social alcohol use

Medications – Celebrex 200 mg po bid prn, Tramadol 50 mg po twice a day, Gabapentin 300 mg po tid

Guiding Questions

(1) What diagnoses would you consider in the differential and what is your working diagnosis?
(2) What is the difference between CRPS 1 and 2?
(3) How would you diagnose CRPS?

Discussion

If a patient has pain that is disproportionate to the inciting event and healing time frame, complex regional pain syndrome (CRPS) should be considered in the differential diagnosis. CRPS is a complex chronic pain condition usually involving the extremities. There are two types of CRPS based on the etiology of inciting injury:

- CRPS 1 – patient may have no history of inciting trauma or minor unnoticeable trauma
- CRPS 2 – patient may have obvious or major injury

Mechanism

The exact mechanism of CRPS is not fully understood, but inflammation, pain amplification, and central sensitization are involved in its evolution

Diagnosis

CRPS is a clinical diagnosis of exclusion. The Budapest Criteria standardize the diagnosis of CRPS

- three of four symptoms of dysfunction in sensory (pain), vasomotor (temperature, color), pseudomotor (edema, sweating), or trophic (weakness, stiffness, hair/nail changes) categories
- three of four signs of dysfunction in the sensory (allodynia), vasomotor (temperature and color changes or symmetry), pseudomotor (edema and sweating changes or asymmetry), or trophic (motor weakness, decreased range of motion, tremor, dystonia, hair and nail changes or asymmetry) categories

Differential Diagnosis

- Infection – lack of systemic symptoms, normal WBC and normal inflammatory markers rule out infection.
- Compartment syndrome – a surgical emergency. Symptoms are pain, swelling, weakness, numbness, color changes, and weak pulse. Normal intra-compartmental pressure = 0 to 8 mmHg.
- Tissue injury – pain, swelling, and erythema should prompt investigation into tissue injury. X-ray, CT, or MRI testing may identify fracture or tears in the muscles, ligaments, and tendons.
- Peripheral vascular disease – deep venous thrombosis and peripheral vascular ischemia can present with pain, swelling, and erythema. An arterial and venous doppler ultrasound typically diagnoses peripheral vascular disease.
- Neuropathy – pain and weakness are attributed to central or peripheral neuropathy, and secondary to direct or indirect nerve injury. Electromyography (EMG) and nerve-conduction velocity studies (NCV) may be of use.
- Thoracic outlet syndrome – compression of nerves or blood vessel at the thoracic outlet by trauma, tumor, or a cervical rib can cause swelling and erythema.

Guiding Questions

(4) What are the treatment options for CRPS?
(5) What is the utility of SGBade in this setting?
(6) What are the side effects and complications associated with SGBs?

Discussion

The multimodal treatment strategy of CRPS is individualized with a goal to increase functionality and sensitization

– physical and occupational therapy. First-line treatment for CRPS physical and occupational therapies focuses on general exercise, functional training, transcutaneous electrical nerve stimulation (TENS), and graded motor imagery.

- Psychosocial and behavioral therapy – adding psychosocial and behavioral rehabilitation is beneficial with physical therapy [7]. In addition to cognitive behavioral therapy, developing coping mechanisms, distraction techniques, and participating in biofeedback are essential in CRPS.
- Analgesics – finding combinations that will allow patients to continue with PT and OT is essential.
  - Topical medications – lidocaine and capsaicin are useful and have negligible side-effect profiles
  - NSAIDs – useful in neurogenic inflammation in early CRPS. Unlikely to benefit late stage CRPS
  - Gabapentinoids – gabapentin and pregablin may be useful for neuropathic pain
  - Antidepressants – SNRI, SSRI, and tricyclic antidepressants (TCA) decrease pain and treat depression
  - Ketamine – useful due to its anti-NMDA receptor properties theorized to “reset” pain channels
  - Bisphosphonates – decreases bone resorption
  - High-dose vitamin C
- Stellate ganglion blocks (SGB) identify the source of pain, decrease pain, and improve function
- Spinal cord stimulator – electrical stimulation of the dorsal column may benefit neuropathic chronic pain

Case continued

An uncomplicated left SGB with USG was performed in the office. His pain significantly decreased for a week. Sequential blocks were done every two weeks with an increasing duration of relief. After the fourth SGB he performed better with PT and function improved with less pain.

Guiding Questions

(7) What is the anatomy of the stellate ganglion and how would you block it?
(8) What is the utility of a SGB in CRPS?
(9) What are the signs of a successful SGB
(10) What are the side effects and complications of SGBs?

Discussion

Anatomy

- Provides sympathetic efferents to the upper extremities, head, neck, and heart
- A large star shaped 1–3 cm long and 3–10 mm wide ganglion formed by the inferior cervical and the first thoracic ganglion at the anterior aspect of C7 - T1 vertebrae
- Borders
  - Lateral – scalene muscles, phrenic nerve and the carotid sheath
  - Medial – longus colli muscle
  - Posterior – head of the first rib, prevertebral fascia, and C7 transverse process
  - Anterior – vertebral artery and posterior fascia of the carotid sheath
  - Inferior – lung pleura

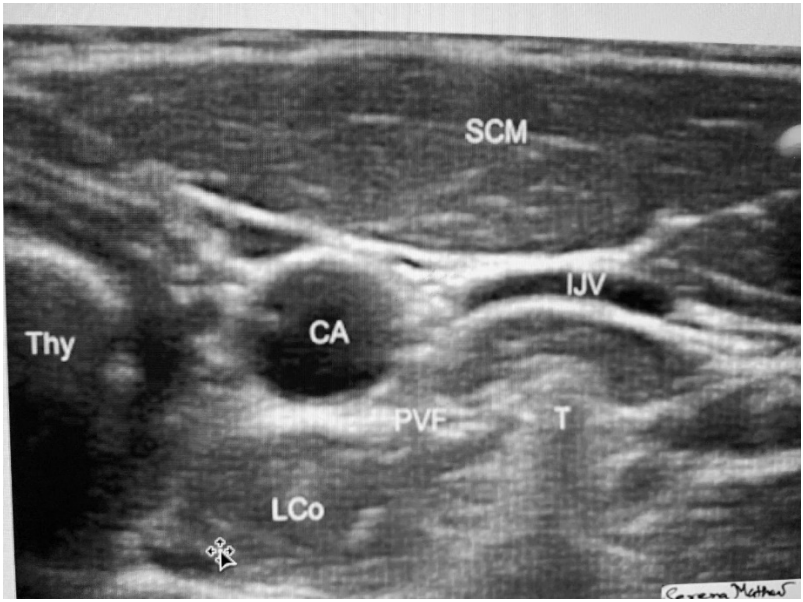
SGB

Intravenous access should be established. Standard monitoring includes pulse oximetry and noninvasive blood-pressure monitoring. Emergency airway equipment, oxygen, and vasoactive medications should be available. Skin temperature is monitored using skin thermistors. Patient is positioned supine with neck in mild extension. Sterile precautions are used throughout. Image-guided SGBs increase safety and accuracy compared to blind injections.

Ultrasound Guidance

- Transducer is placed perpendicular to the tracheal axis at the cricoid and moved inferiorly until the superior aspect of the thyroid gland is visualized. The transducer is relocated laterally to visualize the anterior aspect of the C6 tubercle.
- Using the in-plane approach, the needle is directed to the prevertebral fascia of the Longus-colli muscle (between the posterior aspect of the carotid artery and the tip of C6 anterior tubercle).
- After negative aspiration, 0.5 cc of 1% lidocaine is injected as a test dose and the injectate is seen to spread in real time. The fluid spreads along the paravertebral fascia to the stellate ganglion.
- 5–10 cc of a mix of local anesthetic with or without steroids is injected in 1–2 cc increments with intermittent aspiration (1% Lidocaine or 0.25% Bupivacaine or Ropivacaine with or without 10–20mg of Triamcinolone).

Fig. 2.1. Ultrasound Image for SBG placement



SCM: Sternocleidomastoid muscle
IJv: Internal Jugular Vein
LCo: Longus Colli Muscle
CA: Carotid Artery
PVF: Paravertebral Fascia
Thy: Thyroid gland
T: Transverse Process

Fig. 1. USG image for SGB

Fluoroscopy guided SGB

The C6–C7 paratracheal approach is most commonly used under fluoroscopic guidance.

- The target is the junction of the transvers process with the body at C6 or C7 level on the ipsilateral side.
- Local anesthesia is injected at the target site using 1–2 cc of 1% lidocaine using a 25G1.5 inch needle.
- A 25 G needle is advanced in a gun barrel orientation until bony contact is made where the tubercle of the transverse process meets the lateral edge of the vertebral body at C6 or C7.
- After negative aspiration for blood or CSF, inject 1–2 cc of contrast under continuous fluoroscopy.
- Confirm absence of intravascular uptake and contrast along the longus colli muscle.
- If there is no cephalo-caudad spread between tissue planes it's an IM injection in the longus colli.
- 0.5 cc of 1% lidocaine is the test dose that confirms the absence of intravascular uptake.
- 5–10 cc of a mixture of local anesthetic with or without steroids is injected in increments with intermittent aspiration (1% Lidocaine or 0.25% Bupivacaine or Ropivacaine, with or without 10–20mg of Triamcinolone).

Fig. 2.2. Fluoroscopic guided SGB showing contrast along the longus colli

Marker of successful blockade

- Ipsilateral rise in temperature by at least 1ºC is the golden sign of a successful block.

Common side effects

Horner syndrome – ptosis, miosis, anhydrosis, enophthalmos, lacrimal and nasal congestion, flushing of the face.

Brachial plexus block – weakness of ipsilateral arm.

Phrenic nerve block – ipsilateral diaphragmatic palsy.

Recurrent laryngeal nerve palsy – hoarseness of voice.

Complications

Faulty needle placement	Local anesthetic related	Infection
Hematoma	Intravascular injection	Soft infection tissue
Nerve injury	Seizures	Osteitis
Brachial plexopathy	Ipsilateral arm weakness due to brachial plexus block	Meningitis
Pulmonary injury	Total spinal	
Pneumothorax		
Chylothorax/ hemothorax		
Esophageal perforation		

Utility of SGB

SGB decreases sympathetic activity causing vasodilation, improved blood flow, increased temperature, decreased sweating, and decreased pain sensitivity of the ipsilateral arm. Resultant analgesia helps to tolerate PT and OT. It is a diagnostic tool that identifies sympathetic mediated pain. It can be repeated sequentially for therapeutic purposes. A positive response (> 50% analgesia) predicates a positive response to neurolysis. According to traditional thinking, SGB response was diagnostic for CRPS; it is now well understood that SGB does not impact patients in whom pain is not sympathetically mediated i.e. pain of CRPS may be independent of sympathetic innervation. SGB can be repeated sequentially. Subsequently, ablation of the stellate ganglion may be done with RFA or chemical neurolysis.

Take-home points

(1) CRPS is a chronic neuropathic pain condition that occurs with and without trauma but presents with pain, swelling, weakness, and skin changes.
(2) CRPS is a diagnosis of exclusion but has criteria that suggest diagnosis.
(3) Physical and occupational therapy is the mainstay of treatment. Behavioral therapies, medications, and interventions are important analgesic modalities
(4) SGBs may be useful as a diagnostic or therapeutic maneuver in CRPS.

(5) Imaging guidance is essential for safe execution of the block.

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TOPIC: RFA OF GENICULAR NERVES FOR CHRONIC KNEE PAIN

MICHAEL KHALILI, MD
PHILIP LIN, MD
LEENA MATHEW, MD

Objectives

(1) Understand the prevalence and diagnosis of Knee Osteoarthritis (OA)
(2) Understand conservative therapy for chronic pain in OA
(3) Understand relevant neuro-anatomy of the knee joint
(4) Understand the utility of diagnostic blocks of the genicular nerves
(5) Understand application and mechanism of Radiofrequency Ablation (RFA)

Background

A fifty-five-year-old man presents with progressive knee pain refractory to usual treatments. He had multiple intra-articular steroid injections with transient relief. Platelet rich plasma injection and viscous supplementation did not provide an acceptable analgesia. He is "weirded out" by stem-cell injections and "doesn't want to talk about it." He is averse to surgery because of his comorbidities. At presentation, he is on long-term opioid therapy. He has no systemic symptoms. His mood and appetite are normal. He is not a smoker and only uses alcohol socially. He denies any aberrant drug use. He was told to come to the pain center for some "nerve blocks."

Past medical history – fatty liver, morbid obesity, obstructive sleep apnea on CPAP, gastroesophageal reflux disease, hypertension, hyperlipidemia, OA, and erectile dysfunction.

Past surgical history – tonsillectomy as a child.

Allergies – none.

Medications – oxycodone, diclofenac, hydrochlorothiazide, aspirin and omeprazole.

Physical Exam

120 kg, Pulse 76/min, BP 125/70mmHg, RR 19, T 37˚C. He was a wheelchair-bound man with antalgic gait. Notable genu varum, poor posture, and stance. He had no leg edema, rash, or erythema. His range of motion (ROM) was limited in all planes at both knee joints with pain and crepitation throughout ROM. No joint redness, swelling, or effusion. He had exquisite medial joint line tenderness. There was no meniscal or ligamentous injury or knee instability. Muscle tone and bulk were normal. The neurological exam was normal.

Imaging Studies

X-ray bilateral knees – joint space narrowing (JSN), osteophytes, and sub-chondral sclerosis (Grade 3)

Guiding questions

(1) At what stage of OA is surgical therapy warranted?
(2) What are opioid-sparing alternatives of pain management in this patient?
(3) What nerve blocks can be offered in this setting?

Case continued

The diagnosis of Grade 3 OA was confirmed. Opioid taper schedule was discussed with the patient in the context of incorporating lifestyle modification and invasive interventions. He was considering diagnostic genicular nerve blocks under fluoroscopy to elucidate the source of pain, provide temporizing relief, and predicate a response to genicular nerves' RFA (RFA).

Guiding questions

(4) What is the innervation of the knee joint?
(5) How are diagnostic genicular nerve blocks done and what is the utility of performing them?

Case continued

Genicular nerve blocks were done with fluoroscopy guidance resulting in > 80% relief with improved functionality for two weeks. A subsequent genicular RFA was done with excellent response. Six months later he continued to have pain relief. With increasing activity, he lost twenty pounds and discontinued opioids.

Guiding questions

(6) What is RFA?
(7) What are some long-term benefits of RFA?
(8) When and how would you proceed with an RFA?

Discussion

OA (OA), a degenerative disease, is the most common cause for knee pain. It has a lifetime prevalence of ~45% and is a leading cause of disability worldwide. It develops gradually over one to two decades with increasing disability. OA may affect one of or all three compartments (patellofemoral, medial, and lateral tibiofemoral) of the knee.

The pathophysiology of knee OA

The pathophysiology of OA involves the breakdown of the cartilaginous matrix leading to subchondral sclerosis and osteophyte formation. Inflammatory and biomechanical processes are involved in the presence of increasing age, obesity, diabetes, systemic inflammatory disease, limb malalignment like genu valgus, genu varum, or dysplasia, trauma, and metabolic syndromes. Modifiable and non-modifiable risk factors impact the development of OA.

- Non-modifiable risk factors – age, genetic mutations, congenital factors.
- Modifiable – the most common modifiable factor is obesity. Mechanisms like excess joint loading; inflammatory signaling and behavioral factors like diminished activity and loss of muscle strength are implicated. 1 lb. weight gain adds 2—4 lbs. of extra pressure on the knees.

Symptoms of knee OA

- Knee pain – mild to severe, dull, sharp, constant or intermittent, flares with activity.
- Decrease in range of motion over time.
- Grinding sounds or crepitus during ROM.
- Swelling, locking, and giving way of the knee.
- Pain related disability – difficulty walking, climbing stairs, performing chores.
- Joint pain/stiffness after prolonged sitting which loosens up in thirty minutes (gelling).

Diagnosis

The diagnosis of knee OA relies on the history and a physical exam. It may be confirmed with X-rays that show JSN. The American College of Rheumatology allows for diagnosis of knee OA without imaging or labs.

Grades of OA – the traditional Kellgren and Lawrence system classifies knee OA using radiography:

- grade 0 – no radiographic features of OA are present
- grade 1 – doubtful joint space narrowing (JSN) and possible osteophytic lipping
- grade 2 – definite osteophytes and possible JSN on antero-posterior weight-bearing radiograph
- grade 3 – multiple osteophytes, definite JSN, sclerosis, possible bony deformity
- grade 4 – large osteophytes, marked JSN, severe sclerosis, and definite bony deformity

Treatment of OA

Therapies for OA focus on analgesia, and improving the function and quality of life.

Non-surgical Treatment

- Heat and cold treatments.
- Lifestyle factors – modify activity, weight loss.

- Physical therapy – muscle strengthening exercises, orthotics, braces, gait training, posture mechanics.
- Pharmaceuticals – NSAIDs, acetaminophen, muscle relaxants, topical agents.
- Injections
  - Local anesthetic and corticosteroid injections.
  - Viscous-supplementation with hyaluronic acid based injections.
  - Platelet-rich plasma (PRP).
  - Stem-cell therapies.
  - Genicular blocks – diagnostic blocks followed by knee denervation with RFA.
- Surgery – a definitive solution in advanced knee OA; 90% patients after TKA have great results > 20 years.

Indications for Genicular Nerve blocks

- Symptomatic knee OA (OA) grades 1–4 recalcitrant to conservative modalities.
- Patients after failed TKR with no identifiable cause of the pain or prosthetic failure.
- Poor surgical candidates because of medical comorbidities and/or high body mass index (BMI).
- Patients who do not want to have surgery.
- Patients who had a prior successful genicular nerve RFA (GNRFA).

Neuro-anatomy of Knee Joint

Innervation of the knee joint is complex

Genicular nerves (GN):	Nerve to the Vasti	Others
Superior-lateral GN (SLGN)	Nerve to vastus medialis (NVM)	Infra-patellar branch of Saphenous nerve (IPBSN)
Inferior-lateral GN (ILGN)	Nerve to vastus lateralis (NVL)	Common fibular nerve (CFN)
Superior-medial GN (SMGN)	Nerve to vastus intermedius (NVI)	Recurrent fibular nerve (RFN)
Inferior medial GN (IMGN)		Obturator nerve (ON)

Anterior knee is innervated by: SLGN, ILGN, SMGN, IMGN, NVM, NVL, NVI, and CFN.

It helps to think of the knee in four quadrants:

- Supero-lateral quadrant innervated by NVL, NVI, SLGN, and CFN.
- Inferolateral quadrant innervated by ILGN and RFN.
- Superomedial quadrant innervated by SMGN, NVI, and NVM.
- Inferomedial quadrant innervated by IMGN and IPBSN.

The SMGN, SLGN, and IMGN are the main targets for RFA. These articular branches from the sciatic and femoral nerves course along the periosteum and terminate in the knee capsule.

SLGN – main innervation for the supero-lateral quadrant; it rises from either the sciatic nerve or the CFN.

SMGN – main innervation of the supero-medial quadrant; it rises from the femoral nerve.

IMGN – main innervation of the infero-medial quadrant; it rises from the tibial nerve.

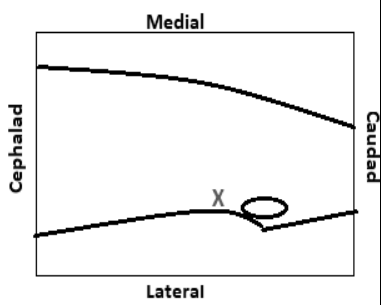
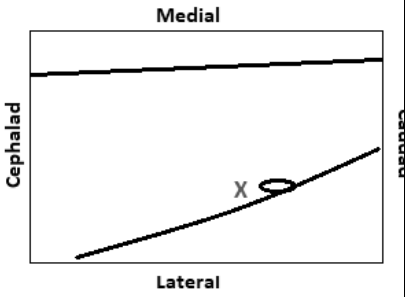
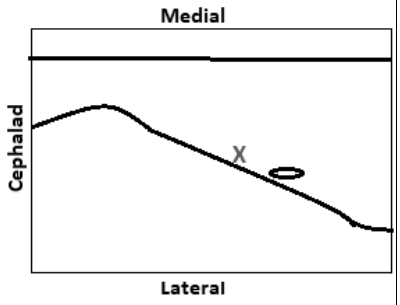
Rationale for diagnostic blocks

Initial SMGN, SLGN, and IMGN blocks with local anesthetic +/- steroids are diagnostic to assess for analgesic response. Diagnostic blocks predicate potential relief from a therapeutic RFA. The criteria for a successful genicular nerve block is reduction in pain score by > 50% for a time frame concordant with at least the duration of action of the local anesthetic. Patients with excellent response to diagnostic genicular nerve blocks are candidates for RFA.

Landmarks for blockade

Imaging allows accuracy. The blocks are performed with fluoroscopy using bony targets or with ultrasound visualization. The SLGN and SMGN lie on the periosteum at the lateral and medial confluence of the distal femoral shaft and epicondyles. MGN lies on periosteum at the medial junction of the tibial plateau and shaft.

Fig. 2.3. Ultrasound visualization of SMGN, SLGN, and IMGN

A)	B)	C)
		
SMGN: External rotation of ipsilateral hip and transducer aligned transversely over medial femoral epicondyle and then moved up to adductor tubercle. The target is proximal and anterior to the adductor tubercle.	**SLGN**: Internal rotation of ipsilateral hip and the lateral aspect of knee joint facing superiorly. The transducer is aligned transversely over femur at junction of lateral epicondyle and shaft of femur where SLGN and vessels are identified.	**IMGN**: External rotation of hip with flexed knee and USG transducer along the short axis of tibia and medial collateral ligament (MCL) where the IMGN lies with the IMGA and IMGV blood vessels underneath the MCL.