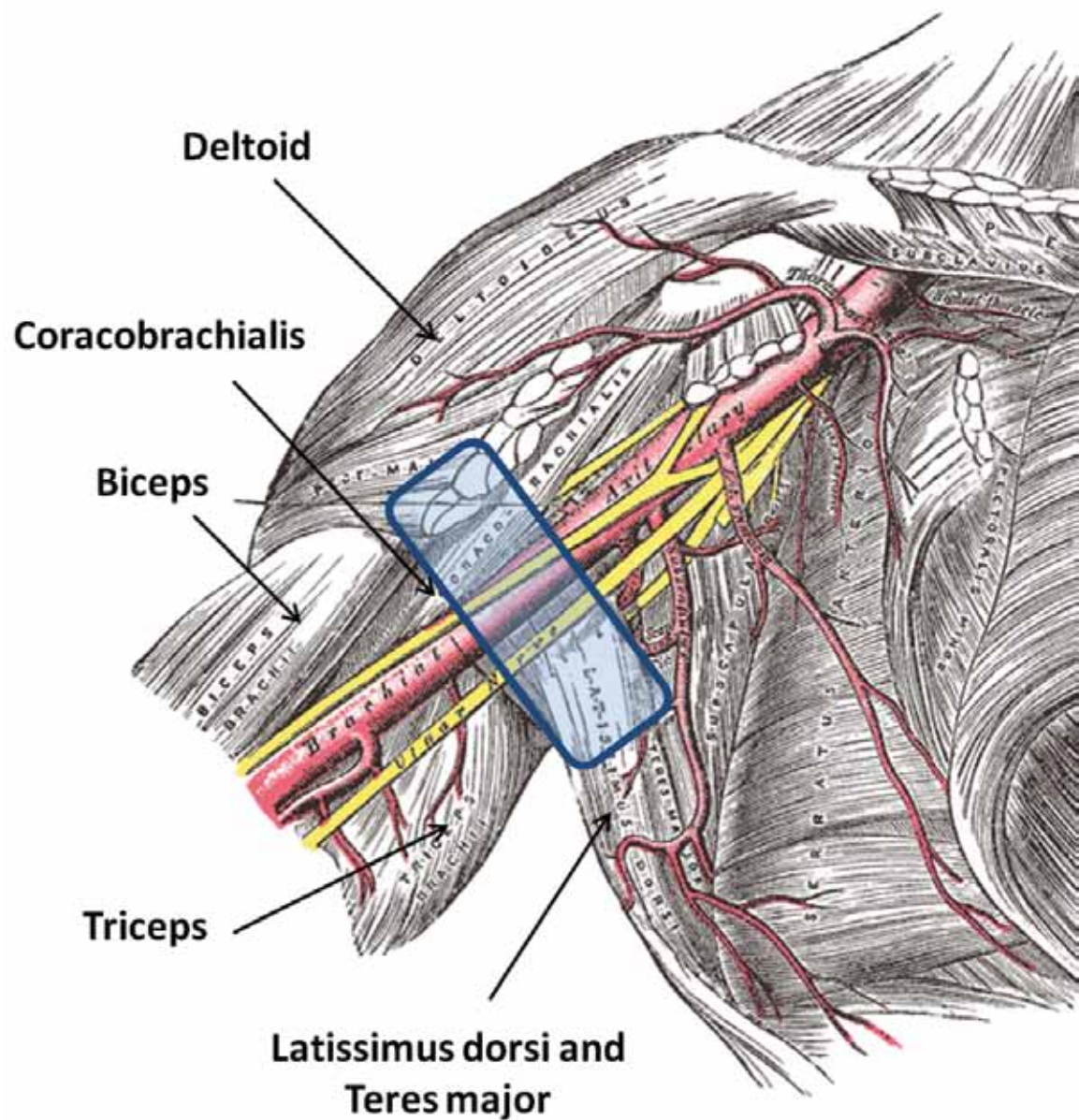


ASRA NEWS

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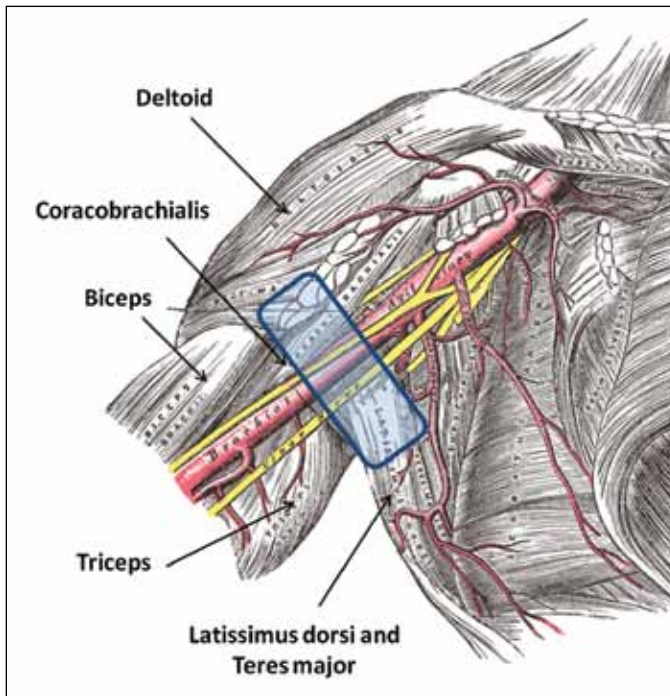
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President's Message

We are in the final stages of planning for the 10th Annual ASRA Pain Medicine Meeting and Workshops to take place at the Hilton New Orleans Riverside in New Orleans, Louisiana. Program Chair Gary Brenner and the Program Committee have done an amazing job preparing for this meeting. Some notable highlights include

the Thursday November 17 combined ASRA/FAER lecture by Dr. Timothy Brennan M.D., Ph.D., on Closing the Gap Between Pain Research and Pain Management, the Bonica Lecture on Friday presented by Bonica Award Recipient James Rathmell M.D., the paired refresher course lectures covering both basic sciences and clinical application, as well as ultrasound and imaging workshops, and problem based breakout sessions. There is a two-day Physician Assistant Education Program covering topics such as opioid management and therapies for neuropathic and spine related pain. Additionally there is a two and one-half day program exclusively for residents and fellows. This will include a discussion of healthcare reform and the future of pain medicine, three general sessions, problem based learning discussions and a four and one hour-half hour hands-on workshop with fluoroscopy, ultrasound and basic surgical skills.

As Dr. Brenner and his committee were working in preparation for the upcoming pain meeting, in May the Society concluded one of our most well attended and successful meetings ever --the 36th annual Regional Anesthesia Meeting and Workshops at Caesars Palace in Las Vegas, Nevada. This meeting boasted over 1000 attendees and featured the presentation of the distinguished service award to Guy Weinburg M.D., the Gaston Labat lecture by awardee Terese Horlocker M.D., and separate programs for residents/fellows and acute pain nurses, as well as workshop, parallel sessions and refresher courses.

The hard work that goes into the planning of these meetings and the successful conduction of these meetings is really the primary focus of the American Society of Regional Anesthesia and Pain Medicine. Members occasionally ask why ASRA is not more involved in political advocacy and practice management. Although we recognize that advocacy and practice management are very important, the primary mission of ASRA always has been and will continue to be education and research. That is why within the last year the society has undertaken several new projects. The first of these projects is the new pain research grant. This grant will serve to complement the already existing Carl Koller Grant. The Koller grant is given bi-annually in the sum of \$75,000 to fund projects focusing on local anesthetics. I am happy to report that

the Koller fund is self-sustaining at the current funding level. This is why it is an ideal time to introduce a second grant focusing on pain medicine. I encourage all members to make a donation no matter how small, to this grant on the ASRA website.

A second new project for ASRA this year involves collaboration with the American Society of Anesthesiology on some educational material. One of these collaborative projects will be the MOCA or maintenance of certification for pain practioners. The second project will be the development of an ultrasound portfolio to help clinicians document continuing medical education or additional training in ultrasound guided regional anesthesia. Both of these projects should ultimately help anesthesiologist verify their training, experience and fund of knowledge.

Given ASRA's focus on education and research, it is important for members to recognize that the program committee and board of directors do take very seriously the suggestions of the membership for educational projects. To that end the program committees and Program Chairs of upcoming meetings review all suggested program topics and strive to incorporate them when appropriate into the meeting content. Thus at future annual meetings you will see topics related to reimbursement and practice management. Although these topics will never take the place of basic science or translational research in the program (nor should they) we do recognize that these topics are important to our members. We also recognize that it is important to provide education not only on the latest gene therapy but also on topics that directly impact the practices of all or our members.



Julia E. Pollock, M.D.

“Although we recognize that advocacy and practice management are very important, the primary mission of ASRA always has been and will continue to be education and research.”

How I Do It

Ultrasound-Guided Axillary Block



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The ultrasound-guided axillary block is an excellent technique for providing surgical anesthesia at and below the elbow. It is at least as effective as the supraclavicular and infraclavicular blocks, if not more so because of the ability to individually target the four main terminal nerves of the arm.¹ It is also a safe approach, due in part to the shallow depth of the brachial plexus in this location, which allows excellent visualization of both nerves and needle. Inadvertent intraneural and intravascular injections are the only significant risks, and both

are easily avoided. Finally, it is an easy block to learn and perform, as this article will illustrate.

Patient Positioning and Equipment Selection

The patient is placed supine with the arm abducted 90 degrees at the shoulder. Inability to abduct the arm represents one of the few absolute contraindications to this block. A high-frequency (>10 MHz) linear array transducer and 22-gauge 50 mm block needle are recommended. A peripheral nerve stimulator may be

used to confirm the identity of the individual nerves, however as practitioners gain experience, they will find that it is by no means essential.

The Scanning Phase

1. Obtain the Conjoint Tendon Zone (CTZ) view:

The transducer is oriented perpendicular to the axis of the brachial plexus and axillary artery to image the neurovascular bundle in cross-section. It is essential that the transducer is placed as proximal in the axilla as possible. At this level, the conjoint tendon of the latissimus dorsi and teres major (and not the triceps) lies posterior to the neurovascular bundle. The conjoint tendon appears as a sloping hyperechoic line on ultrasound (Figure 1). The conjoint tendon is the key landmark in the axillary block as the entire neurovascular bundle lies superficial (anterior) to it.²

2. "Bounce" the transducer to identify the axillary veins:

The veins in the axillary neurovascular bundle are revealed by varying the downward pressure of the transducer in a "bouncing" motion to alternately expand and compress the veins. This serves two purposes.

- 1) **To identify the location of the veins and minimize the risk of inadvertent puncture.** There are usually at least two veins, one in the posterolateral quadrant of the neurovascular bundle, and one in the anteromedial quadrant (Figure 2b).
- 2) **To better delineate the nerves.** With compression, the median, ulnar and radial nerves come together and may appear as a single hyperechoic mass (Figure 2a). The expanded veins will separate the nerves into distinct structures (Figure 2b). The ability to compress and expand the veins also distinguishes them from nerves with large hypoechoic elements (see example in Figure 3), which instead of being compressed, will "roll" into different positions around the artery.

3. Use a "traceback" approach to locate and confirm the identity of the individual nerves:

The typical locations of the nerves with respect to the axillary artery, have been described by Retzl³ and Christophe⁴. Their location and identity can be confirmed by scanning distally along the arm and observing the characteristic course that each nerve takes.

- a. **Median and ulnar nerves.** The median and ulnar nerves both lie very close to the axillary artery in the axilla. More distally, the ulnar nerve diverges in a medial direction away from the artery, while the median nerve remains adjacent (usually lateral) to the artery (Figure 4). Because of its subcutaneous location, the ulnar nerve can be difficult to distinguish from pockets of adipose tissue on a

Figure 1



In the conjoint tendon zone view of the axillary brachial plexus, the entire neurovascular bundle is located anterior to the conjoint tendon. (A=artery, MCN = musculocutaneous nerve, MN = median nerve, RN = radial nerve, UN = ulnar nerve, V = vein). (Used with permission of www.usra.ca).

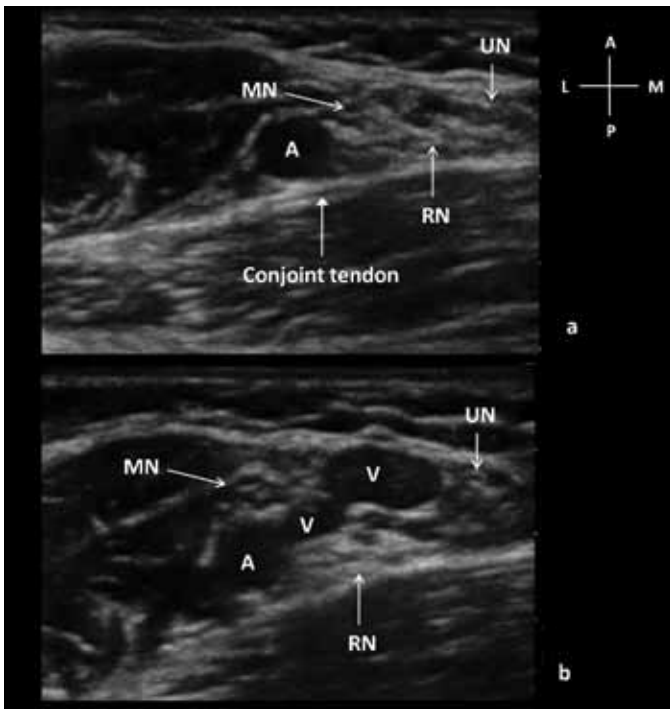
Figure 2a and 2b

Figure 2a. With the veins compressed, the nerves appear as a single hyperechoic structure. Figure 2b. With the veins (V) expanded, the individual nerves are now apparent. (A= artery, MN = median nerve, RN = radial nerve, UN = ulnar nerve). (Used with permission of www.usra.ca).

Figure 3

An example of hypoechoic (dark) median and ulnar nerves, which may be mistaken for blood vessels. However, they do not pulsate (unlike arteries) and are non-compressible (unlike veins). The radial nerve, on the other hand, is almost always hyperechoic with indistinct margins. (A= artery, MN = median nerve, RN = radial nerve, UN = ulnar nerve). (Used with permission of www.usra.ca).

static image, but is easily recognizable on dynamic scanning.

- b. **The radial nerve.** This has traditionally been regarded as the most difficult nerve to visualize.⁵ However in the CTZ view it is almost always found sandwiched between the axillary artery and the conjoint tendon. Its appearance in this location, as a hyperechoic structure with indistinct margins, resembles that of a “cotton-wool ball” (Figures 2 and 3). Once again, its identity may be confirmed by scanning distally and observing the nerve descend in a fascial plane between the long and medial heads of the triceps muscle toward the posteromedial aspect of the humerus (Figure 5). The profunda brachii artery accompanies the radial nerve and can be identified as a hypoechoic pulsatile round structure.
- c. **The musculocutaneous nerve.** The take-off of the musculocutaneous nerve from the lateral cord is highly variable. In general, the nerve lies lateral to the axillary artery, and by scanning in a proximal-distal direction can be observed to “slide” in a lateral-medial direction in the fascial plane between the biceps and coracobrachialis muscles. Its cross-sectional shape varies from triangular to elliptical (Figure 6).

The Needling Phase

Different methods of performing the axillary block have been described, including using out-of-plane (OOP) needle guidance⁶ instead of an in-plane (IP) approach, and a perivascular technique^{7,8} in which the aim is to deposit local anesthetic in a circumferential pattern around the artery rather than targeting individual nerves (perineural technique). It has been suggested that anesthesiologists who are familiar with the neurostimulation-guided axillary blockade may find the OOP technique easier to learn because of the similar approach to the nerves.⁶ However, many people find that visualizing and tracking the needle tip is more difficult with an OOP approach compared to an IP approach, and this may increase the risk of inadvertent vascular or neural trauma. In the hands of trainees, the perivascular technique is associated with a shorter block performance time compared to the perineural technique; however, its efficacy has not been confirmed for local anesthetic (LA) volumes <40 mL.⁷ My preferred method is to use an in-plane needle approach and a perineural technique, as I believe this affords more precision and thereby greater efficacy and safety.

1. The radial nerve:

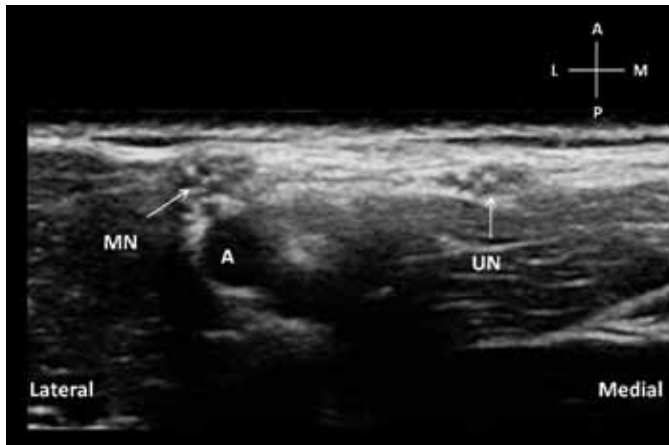
The key to targeting the radial nerve is to use the conjoint tendon to direct the spread of LA between the tendon and the nerve, as described by Gray.² The needle

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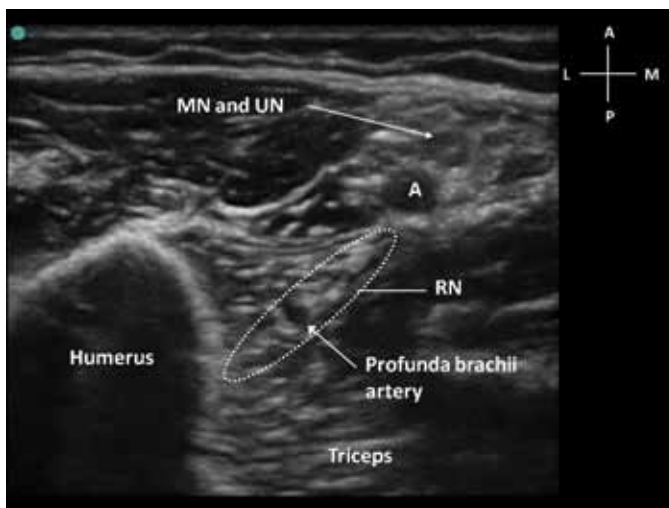
Figure 4



Mid-humeral view of the median nerve (MN), which lies immediately adjacent to the brachial artery (A); and the ulnar nerve (UN), which is subcutaneous and medial to the artery. (Used with permission of www.usra.ca).

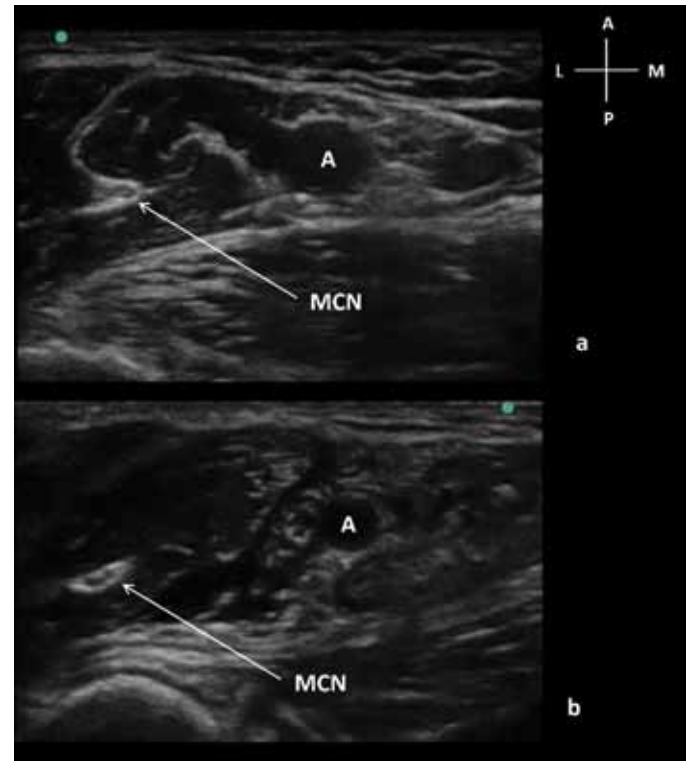
is inserted from the lateral side of the transducer onto the conjoint tendon, just where it meets the axillary artery (Figure 7a). If there is a vein in this location, it should be compressed and the junction between the vein and the tendon targeted instead. With the needle tip lying on the surface of the conjoint tendon, 0.5-1 mL boluses of LA are injected, and this will hydrodissect the plane between the tendon and the neurovascular bundle (Figure 7b). This also has the effect of lifting the whole plexus to lie more superficially. If necessary, the needle may be advanced

Figure 5



Distal to the conjoint tendon zone, the radial nerve runs posterolaterally in a fascial plane between the long and medial heads of the triceps toward the humerus. It is accompanied by the profunda brachii artery. (A= artery, MN = median nerve, RN = radial nerve, UN = ulnar nerve). (Used with permission of www.usra.ca).

Figure 6



The musculocutaneous nerve (MCN) is located lateral to the axillary artery (A) in a fascial plane between the biceps and coracobrachialis muscles. It has a cross-sectional appearance that varies from triangular (a) to elliptical (b). (Used with permission of www.usra.ca).

under the artery to promote LA spread under and around the radial nerve. A total of 5-8mL of LA is usually injected in this location. Bruhn et al.⁹ have described an alternative medial-to-lateral needle approach to the radial nerve in order to avoid the vein commonly found in the posterolateral quadrant, but the principle of using the conjoint tendon to direct LA spread under the nerve remains the same.

2. The median nerve:

The needle is now withdrawn until the tip lies in the subcutaneous layer. It is then re-advanced in a shallow trajectory toward the median nerve, aiming to pierce its investing fascia at a tangent to the surface of the nerve so as to avoid accidental transfixion of the nerve (see section on "Clinical Pearls" on the next page). Penetration of the fascia is signalled by a tactile "pop," whereupon LA is injected in 0.5-1 mL boluses to surround the median nerve and to hydrodissect a safe passage toward the ulnar nerve (Figure 8). A total of 5-8 mL of LA is usually injected here, the endpoint being circumferential spread of LA around the nerve.

3. The ulnar nerve:

The needle is advanced further into the anteromedial quadrant of the neurovascular bundle and adjacent to the ulnar nerve (Figure 9). Injection of 0.5-1 mL boluses of LA, up to a total of 5-8 mL, in this area will surround the

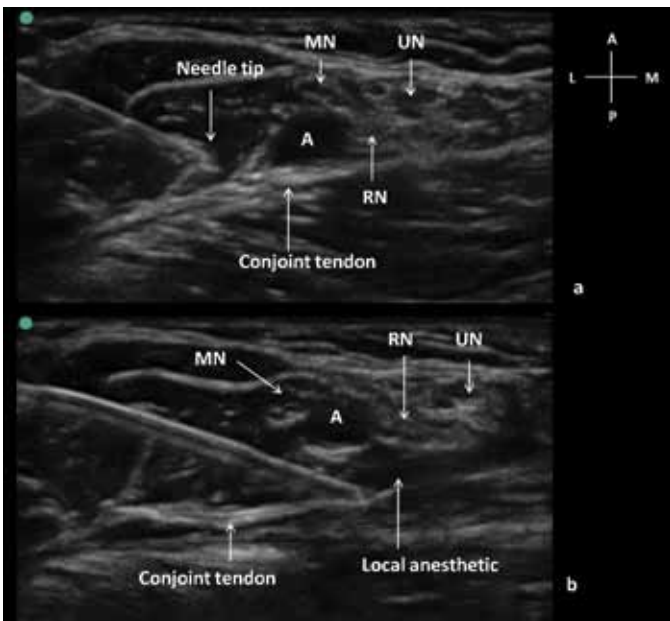
Figure 7a and 7b

Figure 7a. The needle tip is advanced toward the posterolateral junction of the axillary artery (A) and the conjoint tendon. Figure 7b. Injection at this point causes local anesthetic to spread anterior to the conjoint tendon and under (posterior to) the radial nerve, lifting it up. (A=artery, MN = median nerve, RN = radial nerve, UN = ulnar nerve). (Used with permission of www.usra.ca).

ulnar nerve and usually delineate it clearly. Once again, the endpoint for injection is circumferential spread of LA around the nerve.

4. The musculocutaneous nerve:

Depending on how far lateral the musculocutaneous nerve lies from the axillary artery, the needle may need to be withdrawn and re-inserted through a second skin puncture. The nerve lies in the fascial plane between the biceps and coracobrachialis muscles. Once again, the needle should be advanced to contact the nerve at a tangent to its surface and pierce this fascial plane (Figure 10a, page 16). Three to five mL of LA injected in this plane should encircle the nerve (Figure 10b, page 16).

Figure 8

The needle is advanced at a tangent to the median nerve in order to pierce its enveloping fascia without transfixing the nerve. Local anesthetic (LA) is injected here to surround the nerve and to hydrodissect a safe passage toward the ulnar nerve. (A=artery, MN = median nerve, RN = radial nerve, UN = ulnar nerve). (Used with permission of www.usra.ca).

Clinical Pearls

Multiple fascial septae are present within the neurovascular bundle, such that each nerve is contained within its own fascial envelope. In order to pierce this investing fascia without also piercing the epineurium, the needle should always approach the nerve at a tangent to its surface. Safety is suggested by the nerve “rolling” away from the advancing needle tip as it tents the investing fascia. Entry into the fascial envelope is signalled by a tactile “pop.”

A similar principle applies to avoiding vascular puncture. It is extremely difficult to inadvertently pierce a vein or artery with a B-bevelled block needle if the needle contacts the vessel wall at a tangent.

Intraneural injection and intravenous injection are the two most significant risks of this block. Both, however, can be recognized early and easily with ultrasound; intraneural injection by nerve expansion and intravascular injection by the lack of visible LA spread. The initial injection of LA should therefore only be made with the needle tip in view, and in boluses of less than 1 mL. Additional signs of possible intraneural injection include pain and resistance to injection; if these are elicited, injection should be stopped immediately and the needle tip repositioned. Intravascular injection may also be signalled by an increase in heart rate if epinephrine-containing LA solutions are used. Practitioners should continue to be vigilant for all these signs throughout the entire injection process.

Deliberate intraneural injection is not recommended. Instead, the aim is to deposit LA within the investing fascia surrounding each nerve but outside the epineurium. Block onset can be expected within 15 minutes of injection, if not sooner.

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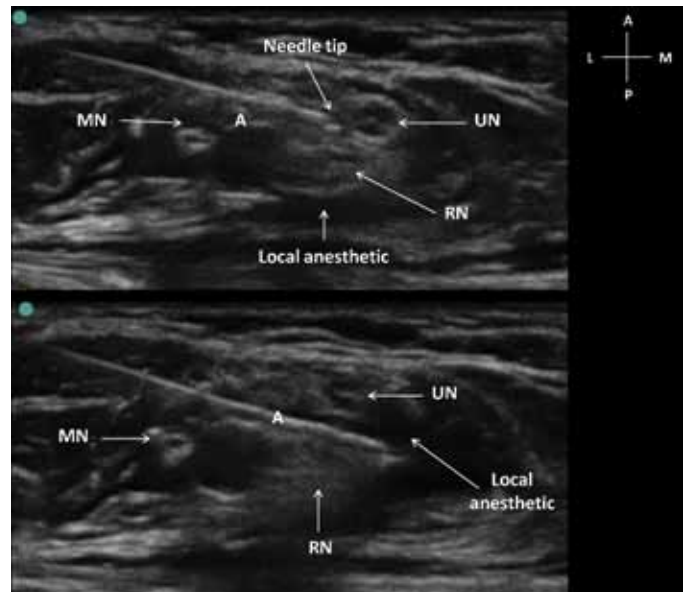
Figure 9a and 9b

Figure 9a. The needle is advanced over the axillary artery (A) toward the ulnar nerve (UN) at a tangent to its surface. Figure 9b. Injection here surrounds the ulnar nerve (UN) and also often spreads superficially to the radial nerve (RN). (MN = median nerve). (Used with permission of www.usra.ca).

Novice Behaviors Associated with Learning Ultrasound-Guided Regional Anesthesia



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When our academic practice started “teaching” ultrasound-guided regional anesthesia (UGRA) in 2002, we were unaware of the optimal learning environment for nurturing these novel skills. This prompted us to try to objectively establish common quality-compromising behaviors associated with UGRA. Our fundamental objective was to create training interventions that were specifically designed to target these negative behaviors. To this end, we comprehensively videotaped 520 peripheral nerve blocks performed by novice anesthesiology residents.¹ Both gross procedural steps and ultrasound images were recorded for the entirety of each block. The video data were then systematically reviewed off-line.

We tracked the occurrence of departmentally recognized errors. There were 398 errors consisting of failure to visualize the needle while it was being advanced (43 percent), unintentional probe movement (26.9 percent), inadequate equipment preparation (11.6 percent), poor ergonomics (7.8 percent) and the neural target malpositioned (4.7 percent). The fact that 43 percent of the recognized errors consisted of the needle being advanced without its visualization is striking given that one of the key theoretical advantages of using ultrasound guidance is the ability to image the needle as it is directed toward the target! The most interesting aspect of our video study is that we discovered multiple quality-compromising behaviors that we had not previously recognized. These included: 1) failure to recognize maldistribution of local anesthetic, 2) fatigue, 3) screen left-right confusion, 4) failure to recognize direct muscle stimulation and 5) bizarre original needle insertion sites.

Based on the above findings, we created simulator-based training modules that highly encouraged or

even forced the residents to commit the various errors and quality compromising behaviors. Our approach to simulation is to use a combination of industry-produced simulators as well as biological simulators. The commercial products included a head, neck and upper-torso simulator for upper-extremity blocks. We also utilized an ultrasound training gel block for practicing needle insertion techniques. The biological models included the use of a raw turkey breast with olives imbedded as targets. Examples of the specific simulation scenarios included: 1) *Torso simulator with bad ergonomics*: In this situation, we lowered the procedure table and placed the ultrasound machine behind the operator. This forced an awkward and uncomfortable scanning and needling position. 2) *Limitations of the needle insertion techniques*: In this scenario, we would use the block simulator to insert the needle with the out-of-plane technique and toward an arbitrary blood vessel imaged in short-axis. We did this at progressive depths and were inevitably able to demonstrate how the needle tip can easily pass through the target; this interesting phenomenon was “proved” by transitioning the imaging to the long-axis view of the blood vessel and needle. Here the trainee could easily see the needle penetrating through the posterior wall of the simulated blood vessel.

3) *Screen left-right confusion*: Simulating this error was rather fun, as we would secretly flip the screen image so that left was now right and right was now left. This can be accomplished on most ultrasound machines by pressing a simple toggle button. When the trainees would insert his or her needle, it would eventually appear on the opposite side of the ultrasound screen than anticipated. The needle would usually transgress multiple unintended structures.

This forced error would highlight the reason to definitively, prior to needle insertion, correlate the orientation of the ultrasound machine with anatomic sidedness of the patient.

When quality-compromising behaviors are experienced during the simulator training (whether forced or unforced), the trainees are given direct feedback and an opportunity to resolve the negative behavior. If the novice is unable to eliminate the error, he or she is asked to cycle back through the exercise until resolution is confirmed.

Beyond simulating the procedural steps associated with an ultrasound-guided block, the novice needs to study ultrasound images and video to learn how to interpret the various patterns of gray that are presented in real-time. Is the spread of local anesthetic correct? Is the injection extravascular? Does the patient have an

“It is my hope that at some point our community will have an online, open-access, comprehensive and searchable database to facilitate learning the interpretive skills associated with UGRA.”

anatomical anomaly? Where is the pleura? Is the targeted structure actually a tendon instead of the nerve? These are just a few of the clinically pertinent questions that should be answerable. Further, these diagnostic skills are not effectively taught through manikin simulation. This is why we have created a comprehensive video library that is designed to allow the novice to repeatedly review actual ultrasound images representing the various interpretative skills defined by the American Society of Regional Anesthesia and Pain Medicine.² Our video library is searchable and contains rare events such as the ultrasound appearance of a likely intrafascicular injection. The novice can learn to compare the ultrasound patterns that are associated with high-quality vs. morbid (rare) events without waiting for long-term clinical experience to provide the exposure.

At our institution, we are fortunate to have access to human cadavers for dissection. The cadaver is available for both residents and staff to advance their appreciation of human anatomy. In my opinion, the availability of such a resource is the most effective strategy for understanding the three-dimensional relationships of key anatomical structures associated with regional anesthesia. As an example (Figure 1), I have noticed that after the residents and fellows dissect out the relationship of the fascia iliaca to the femoral nerve and artery, they truly understand two key principles: 1) The femoral artery and nerve are NOT in the same anatomical compartment. 2) This block will not work unless the local anesthetic is deposited posterior to fascia iliaca. What is really exciting is when they take this gross anatomical knowledge and couple it to information embedded in the image library searched under "fascia iliaca" or "femoral nerve." They will then see through video and still images that the block needle does not need to contact the femoral nerve. When the needle is inserted posterior and lateral to the fascia iliaca, the local anesthetic will spread unimpeded toward and around the femoral nerve.

Although we believe our training interventions have helped resident performance in terms of quality, safety and efficacy, we do not have definitive data to "prove" this. We would need a randomized trial in which we compare the training intervention with our traditional approach of what amounted to the infamous "see one, do one, teach one" model. Unfortunately, such a methodology is not a realistic possibility in our practice. What I know for sure following the institution of our training interventions is that everyone is speaking the same technical language as well as sharing the same technical objective of generating a safe and effective perineural spread of local anesthetic. In essence, this statement to the novice now has real meaning: "Please image the femoral nerve in short-axis,

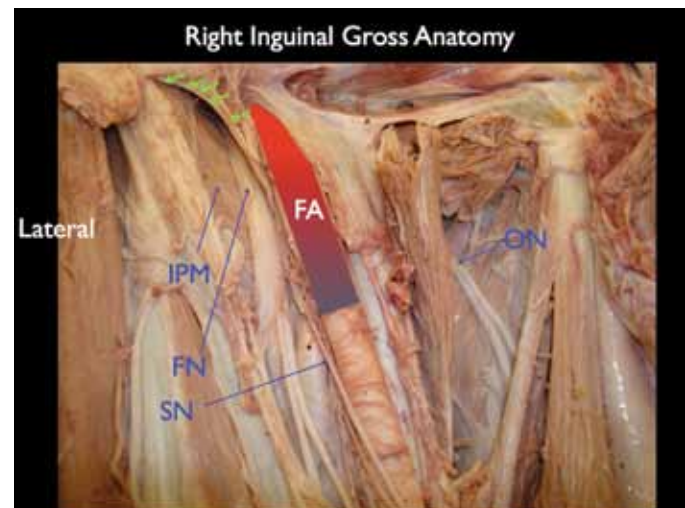
use the in-plane needle insertion technique, puncture the fascia iliaca 1 cm lateral to the femoral nerve, and confirm medial spread of local anesthetic toward the femoral nerve."

I recognize that all institutions are under immense financial pressures, and the ability to conduct supervised training processes may become extremely limited. In fact, this author's ability to run the aforementioned training products is currently challenged. It is my hope that at some point our community will have an online, open-access, comprehensive and searchable database to facilitate learning the interpretive skills associated with UGRA. This resource would lessen the burden of the local-level programs and allow them to focus on the simulated procedural component training.

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Figure 1:



Right inguinal dissection. FA = femoral artery, IPM = iliopsoas muscle, FN = femoral nerve, SN = saphenous nerve, ON = obturator nerve. Green arrows indicate the fascia iliaca.

Development of Lipid Emulsion Treatment for Local Anesthetic Systemic Toxicity



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Local anesthetic systemic toxicity (LAST) is one of the most serious complications of regional anesthesia and has been widely documented since cocaine was first introduced as a local anesthetic.¹

A new era in LAST began in 1979 when Albright reported on a cardiac arrest following inadvertent injection of bupivacaine intravenously. He noted that local anesthetic agents may result in almost simultaneous seizures and cardiovascular collapse without preceding hypoxia from clinical doses of local anesthetic administered intravascularly.² More recently, Di Gregorio et al. reviewed the published cases of LAST from

the past 30 years,³ a period of time when the mechanisms behind systemic toxicity and appropriate treatment remained unclear.

Theoretically, intracellular fatty acid derivatives accumulate during myocardial ischemia, contributing to ischemia-induced arrhythmias. Professor Guy Weinberg and his research team speculated that bupivacaine induced arrhythmias by inhibiting carnitine-mediated mitochondrial fatty-acid uptake; therefore, a pretreatment infusion of lipids might exacerbate the arrhythmias. In their study, rats were pretreated with either a lipid infusion or saline, and arrhythmias were induced by an intravascular injection of bupivacaine. They discovered that arrhythmias were *reduced* in rats pretreated with a lipid infusion but not in untreated rats.⁴ This chance experimental finding over time led to current treatments for LAST. In 1998, Weinberg et al. reported that pretreating rats with a lipid emulsion infusion increased the bupivacaine dose and serum concentration required to produce asystole, and animals that were resuscitated from bupivacaine overdose with a lipid infusion had greater survival rates than controls.^{5,6} In 2003, he reproduced his findings in dogs. Circulatory collapse was induced by intravascular injection of bupivacaine in 12 dogs. Ten minutes after cardiac arrest, half received a lipid infusion and half received saline only.

Dogs receiving a rescue lipid infusion returned to sinus rhythm in less than five minutes with concurrent cardiac massage, and an adequate blood pressure was obtained within 10 minutes, while the control group failed to develop a cardiac rhythm or adequate blood pressure after circulatory collapse.⁶ Weinberg proposed a mechanism of action of lipid emulsion known as the “lipid sink” theory. According to his “lipid sink” theory, the emulsion creates a lipid plasma state, which extracts the lipid soluble local anesthetic molecules from the water-soluble plasma state and compartmentalizes them. Sequestering the local anesthetic prevents it from acting on the cardiac tissue, hence reducing its toxic properties. Weinberg’s lipid sink theory was supported by his published findings, which revealed increased elimination of radiolabeled bupivacaine from cardiac tissue after administration of a lipid emulsion infusion.⁷

Rosenblatt reported the first use of a 20 percent lipid infusion to resuscitate a patient from prolonged cardiac arrest in 2005.⁸ Following placement of an interscalene block, the patient became incoherent and developed a tonic-clonic seizure. The ECG showed asystole without an identifiable pulse, and resuscitation by advanced cardiac life support (ACLS) guidelines was instituted. In preparation for cardiopulmonary bypass, a code team member suggested the use of lipids, and 100 mL of 20 percent lipid emulsion was given intravenously. After the patient’s cardiac rhythm returned to sinus, he was placed on a continuous infusion of lipid emulsion. When he was extubated 2.5 hours later, he was awake and responding appropriately.⁸

“A new era in LAST began in 1979 when Albright reported on a cardiac arrest following inadvertent injection of bupivacaine intravenously.”

This groundbreaking case ushered in the clinical application of lipid emulsions as a viable treatment for LAST. Further evidence supporting the use of lipid infusions include the successful resuscita-

tion of a patient with ropivacaine-induced asystole after axillary plexus block, another patient with local anesthetic-induced cardiovascular collapse after supraclavicular brachial plexus block, and a child with ropivacaine- and lidocaine-induced ventricular arrhythmia following posterior lumbar plexus block.^{9,10,11}

These findings have significantly changed how we treat LAST. In 2007, the ASRA Board of Directors formed a panel of experts to develop treatment recommendations (“Practice Advisory on Treatment of Local Anesthetic Systemic Toxicity” at www.asra.com).¹ Care with administration of local anesthetics and vigilance to the signs and symptoms of LAST are the most important elements of prevention. Recommendations include initially stabilizing the airway, suppressing seizure-like activity with a benzodiazepine, and starting BLS/ACLS

protocols. Lipid rescue therapy should then be instituted with an initial bolus of 1.5 mL/kg intravenously over one minute followed by a continuous infusion of 0.25 mL/kg/min.¹ If cardiovascular collapse is still present, the recommendation is to repeat the bolus and double the infusion rate to 0.5 mL/kg/min if blood pressure remains low. After cardiovascular stability is obtained, the infusion should be continued for at least 10 minutes. If lipid rescue is unsuccessful, cardiopulmonary bypass may be necessary. As soon as LAST is suspected, the cardiothoracic surgeon, anesthesia team and perfusionist should be alerted to the possible need of cardiopulmonary bypass. The panel recommends avoiding vasopressin, calcium channel blockers, beta-blockers or additional local anesthetics during resuscitation, which may alter the effectiveness of the lipid infusion. Although propofol is constituted as a lipid emulsion, it should only be used in low dose for seizure suppression and not as a substitute for lipid therapy. Because of its standard formulation (1 percent propofol in 10 percent lipid emulsion), delivering enough lipid emulsion to treat LAST would result in a large, potentially lethal dose of propofol. Lastly, the panel recommends posting all events at www.lipidrescue.org and/or www.lipidregistry.org.¹

Local anesthetic systemic toxicity has been a problem for as long as local anesthetics have been used in medical practice. Individuals who provide regional anesthesia should have an adequate understanding of LAST and the recent ASRA guidelines. The use of lipid emulsion for the treatment of LAST has been a positive step in the treatment of this serious problem.

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Table 1: ASRA Recommended Algorithm

The image shows the cover and first page of the ASRA Practice Advisory on Treatment of Local Anesthetic Systemic Toxicity (LAST). The cover features the ASRA logo and the title 'Practice Advisory on Treatment of Local Anesthetic Systemic Toxicity'. The first page is titled 'APPENDIX 3' and contains the following text:

AMERICAN SOCIETY OF
REGIONAL ANESTHESIA AND PAIN MEDICINE

Practice Advisory on Treatment
of Local Anesthetic Systemic Toxicity

For Patients Experiencing Signs or Symptoms of
Local Anesthetic Systemic Toxicity (LAST)

- Get Help
- Initial Focus
 - Airway management: ventilate with 100% oxygen
 - Seizure suppression: benzodiazepines are preferred
 - Basic and Advanced Cardiac Life Support (BLS/ACLS) may require prolonged effort
- Infuse 20% Lipid Emulsion (values in parenthesis are for a 70 kg patient)
 - Bolus 1.5 mL/kg (lean body mass) intravenously over 1 min (~100 mL)
 - Continuous infusion at 0.25 mL/kg/min (~18 mL/min; adjust by roller clamp)
 - Repeat bolus once or twice for persistent cardiovascular collapse
 - Double the infusion rate to 0.5 mL/kg per minute if blood pressure remains low
 - Continue infusion for at least 10 mins after attaining circulatory stability
 - Recommended upper limit: approximately 10 mL/kg lipid emulsion over the first 30 mins
- Avoid vasopressin, calcium channel blockers, β -blockers, or local anesthetic
- Alert the nearest facility having cardiopulmonary bypass capability
- Avoid propofol in patients having signs of cardiovascular instability
- Post LAST events at www.lipidrescue.org and report use of lipid to www.lipidregistry.org

<http://www.asra.com/publications-local-anesthetic-systemic-toxicity-2010.php>

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Acupuncture for Chronic Pain



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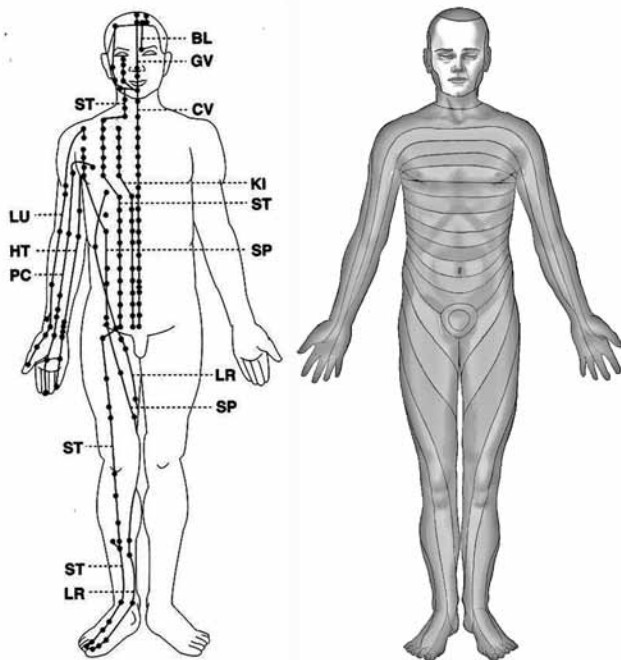
Acupuncture is a part of the practice of traditional Chinese medicine (TCM) that has been used to treat health conditions, including pain, for over 3,000 years. Though acupuncture is probably best known for its use in treating pain conditions in the West, only about 30 percent of acupuncture literature concerns its use for treating pain. All but two classical acupoints have at least one described pain indication, however.

Acupuncture use in the United States has grown in the last three decades, with 1 percent (~2.1 million) of individuals reporting recent acupuncture treatment, and an estimated \$5 billion were spent out of

pocket on this modality in 2006.

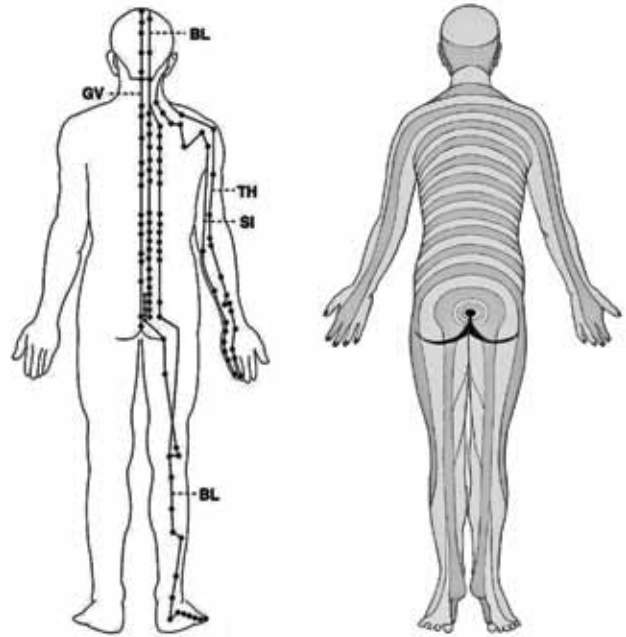
The term acupuncture describes a group of procedures that stimulate precise anatomical locations (acupoints) by a variety of techniques (pressure, needling, heat, etc.) to produce clinical effects. There are 361 classical acupoints, 95 percent of which were described by 200 AD.

Figure 1a



Acupuncture meridians and dermatomes- anterior view.

Figure 1b



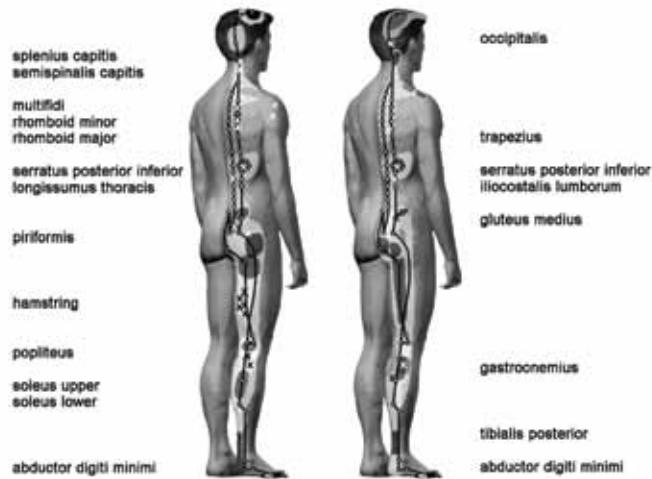
Acupuncture meridians and dermatomes- posterior view.

Classical acupuncture points with similar therapeutic properties are arranged on meridians (Figure 1). There are 12 principal meridians symmetrically arranged around two midline meridians (one on the ventral surface of the body and another on the dorsal aspect of the body). Each of the 12 principal meridians is named for the organ it is associated with (influences), and blood and *qi* (~energy) are perceived to flow cyclically through these meridians.

In the extremities, acupuncture meridians follow dermatomal/ myotomal distributions. As examples, in the lower extremities, the spleen meridian follows an L4 nerve root distribution, the gallbladder an L5 distribution, the bladder meridian an S1 distribution, and the kidney meridian an S2 distribution.

In TCM, pain results when the normal cyclic flow of blood and/or *qi* in a meridian is interrupted. The pain may be felt locally or along the traversing meridian as well (e.g., back pain extending down the posterior leg in a bladder meridian distribution analogous to S1 sciatica). The acupuncturist needles not only the local site of pain where tenderness is present, but also distal limb points on the involved meridian (one crossing the local pain site) to attempt to restore normal circulation of blood and *qi* in the meridian. The *deqi* sensation sought during acupoint needling may be described as numbness (A-beta fiber activation) or as an aching, dull, sore, heavy and/or warm sensation (A-delta and C fiber activation).

Contemporary myofascial pain data serve to support acupuncture theory. Over 93 percent of common trigger points described by Travell and Simons are anatomically proximate to classical acupoints and have similar pain and somatovisceral indications. The clinical phenomenon of

Figure 2

Myofascial-referred pain patterns sum to reproduce the course of the Bladder meridian. The thin black line is the bladder meridian with classical acupoints shown, concentrated referred pain shaded grey within lesser referred pain shaded white.

the spread of qi with acupoint stimulation is conceptually similar to that of inducing referred pain with trigger point stimulation. Thus, the referred-pain data from Travell and Simons serves to confirm the physiologic presence of the acupuncture meridians as demonstrated in Figure 2.

Pomeranz's work in the 1970s demonstrated the key role of endogenous opioids in acupuncture's pain relieving effects. Opiate antagonists reliably prevent acupuncture analgesia, but may not fully reverse acupuncture analgesia (perhaps due to dynorphin activation in acupuncture analgesia).

Contemporary neural imaging studies confirm the importance of the endogenous opioid system and the central nervous system in acupuncture analgesia. PET scans demonstrate that acupuncture produces short- and long-term increases in limbic system mu-opioid binding potential and reduction in clinical pain that is not seen in sham acupuncture. fMRI studies demonstrate that acupuncture produces deactivation of limbic structures (including the amygdala, hippocampus, and cingulate gyrus) via a mechanism that is distinct from painful or sham stimulation.

The peripheral nervous system is important in transduction of acupuncture's clinical effects such as pain relief. Transection or anesthetic block of a peripheral nerve in the distribution of a given acupuncture point will eliminate (or nearly completely eliminate) that point's clinical effects.

Clinical studies in the last 20 years confirm acupuncture's efficacy in treating pain conditions. The randomized, controlled study by Witt et al.¹ of 3,600 subjects with neck pain compared acupuncture to usual care (medications and physical therapy) and found subjects receiving acupuncture had statistically significant improvements in neck pain and disability scores compared to usual care

(56.5 percent vs. 21.6 percent, $p < 0.001$). The GERAC study of 1,162 patients with lumbar pain randomized subjects to acupuncture, sham acupuncture or conventional treatment (drugs, physical therapy and exercise). Subjects receiving acupuncture had clinically and statistically significant improvements in pain and back-related disability scores (55 percent responders) compared to those receiving conventional treatment (33 percent responders). Cherkin et al.² compared acupuncture, simulated acupuncture, and usual care in 638 individuals with chronic lumbar pain and found acupuncture interventions produced statistically significant improvements ($p \leq .003$) in Roland Morris scores and dysfunction scale ($p < 0.001$) compared to usual care both at short- and long-term (52 week) follow-ups. Large, placebo-controlled trials have also demonstrated acupuncture's efficacy in treating knee osteoarthritis and migraine headaches. The World Health Organization in 2003³ concluded that there is evidence that acupuncture may be helpful in treating a variety of pain conditions, including dental pain, tennis elbow, sciatica, low-back pain, rheumatoid arthritis, headache, migraine, trigeminal neuralgia, intercostal neuralgia and peripheral neuropathy.

A major challenge for controlled acupuncture trials has been to find adequate sham acupuncture interventions, since fMRI and clinical studies have demonstrated that minimal needling or pressure of skin over acupuncture and non-acupuncture point locations produces physiologic responses that can reduce pain. Thus, Park or Streitberger non-penetrating sham needles are not physiologically inert devices, which may explain why studies performing "sham" acupuncture interventions with these devices often have produced clinical benefits similar to verum acupuncture and that are superior to no intervention or standard care.

Beyond its demonstrated efficacy in treating chronic pain, acupuncture has an excellent safety record and is cost-effective. Health economists generally consider a health intervention cost effective if it costs less than \$50,000 per quality adjusted life year (QALY). Acupuncture has been shown to be cost effective for treating headache (\$15,100 USD per QALY gained) and low-back pain (\$7,000 USD per QALY gained). These costs then compare favorably to use of long-term medication for low-back pain (not cost effective), epidural spinal injections for sciatica (\$73,400 USD per QALY gained) or surgery (\$77,600-\$115,600 per QALY gained) for treating chronic back pain from stenosis. With aging populations in developed countries and rising health care expenditures, acupuncture may thus warrant an expanding role in future care of persons with pain conditions.

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Spinal Cord Stimulation Complications: An Example of the Personal Anecdote in the Lay Media



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Is the case report or a physician's personal experience the new accepted evidence-based medicine? Or is this simply the evidence base for pain medicine? The danger of the anecdotal story is the influence that it carries on decision-making for patients and their network of friends and families, either swaying people for or against any particular medication or therapy. Pain medicine physicians often develop their practice methods based on the teachings and experience of their fellowship training, colleagues, extended educational network and personal experience. A challenge with the practice of medicine, and pain medicine particularly, is the lack of knowledge of peer-reviewed evidence or the lack of such evidence for efficacy and safety of therapies. In the vacuum that is left, the case report, the professional anecdote and the urban myth can intercalate and become dogma. This can be especially challenging in the field of neuromodulation, which is highly competitive not only from the perspective of the makers of the medical devices but also the physicians who employ these therapies.

The March 7, 2011 issue of *TIME* magazine was titled "Understanding Pain." The topical coverage of the series of articles ranged from the basic science to alternative and complementary medical therapies. One article focused on the story of a patient with complex regional pain syndrome (CRPS) of the upper extremity. *TIME*'s summary sentence for the story "Living with Pain" was "the hunt for relief can lead to extreme - and dangerous -

places." That is a frightening statement and can be true in some cases. However, the question is whether it applies to this patient's story or the pain therapies that were being discussed. What the article goes on to "expose" is not an experimental or extreme therapy that left a patient in a debilitated state. Instead, it tells the story of a patient who has a family history of chronic pain who rejects treatment mainstays for the "safer" route of opioid therapy. Her pain specialist recommended physical therapy and pain psychology therapy, but these were rejected by the patient as being too labor- and cost-intensive. These therapies represent the foundation of functional recovery with CRPS. One of the troubling aspects of the article is that it describes neuromodulation (spinal cord stimulation, or SCS) with a highly unbalanced perspective on the therapy. The author primarily cites the risks and limitations while downplaying the potential benefits associated with the therapy. The article references a pain physician who has heard of three cases of paralysis following spinal cord stimulator implantation and that no surgeon will implant the stimulator because of the risk of paralysis. The cliffhanger of the story leaves the reader with the impression that paralysis is an imminent risk that the patient is weighing heavily in her decision-making. While this is not the belief of the general medical community, it has a substantial influence on our patients, their acquaintances and their family, and could restrict access to the pain care that is sorely needed in patients such as these.

One of the challenges of pain medicine is that our patients comprise a vulnerable population who can go to extreme lengths in pursuit of pain relief. The challenge for the pain provider is that with most pain disease states, we simply do not have enough evidence to recommend these treatments conclusively. But to truly acquire this evidence, we would need to perform lengthy and expensive randomized, double blind controlled trials examining interventional pain therapies. These types of studies are unlikely to be funded to the necessary extent (or at all) in the current economic and research climate. Given this construct, it behooves the pain physician to discuss with and educate the patient on what is known and what is not with our patients regarding the efficacy and safety of the potential therapies. This is not to say that there is no role for personal (or physician) experience with a therapy. The three major companies all have patient ambassador programs that allow prospective patients to speak with uncompensated patients who have received benefit from neuromodulation therapy.

Reports of SCS complications often accompany efficacy data in the research trials of efficacy. The most common complications associated with SCS are factors associated with failure of the equipment or SCS programming, including lead migration, lead fracture, lead disconnection, intermittent stimulation secondary

to hardware failure, over- or understimulation due to programming errors or battery failure.¹⁻³ Complications resulting in patient harm include infection, pain at the site of the battery, hematoma, CSF leak, seroma and skin erosion.¹⁻³ Although none of the above complications are innocuous, there is limited systematic evidence reporting serious neurological complications resulting directly from implantation of spinal cord stimulator systems. There have been a series of case reports of neurologic compromise from SCS implantation. The first was a case of paralysis following SCS lead implantation mistakenly placed into the spinal cord parenchyma⁴. More recently, a physical medicine and rehabilitation group reported a series of four cases of paresis following spinal cord stimulator permanent lead implantation; interestingly, all patients became symptomatic days following their procedure.⁵ One case was related to direct cord injury presumably during the lead implantation, and the remaining three cases were due to cord compression (two from epidural hematomas and one from thoracic spinal stenosis worsened by lead placement). These reports are likely to be underestimating the true incidence of these types of injuries; however, the lack of reports of these injuries in the controlled studies and meta-analysis of SCS therapies lend credence to the presumption of rarity. The complications related to SCS implantation, such as epidural hematoma, result from patient factors, including bleeding disorders, anti-coagulation, anti-platelet therapies and technical surgical factors such as implantation technique, experience of the implantation surgeon, and possibly the size of the SCS implant. Complications related to cord compression can result from spinal stenosis secondary to patient factors such as thoracic or cervical disc herniation, ligamentous hypertrophy, facet hypertrophy as well as surgical factors including the size of the SCS implant and whether the site was surgically decompressed prior to implantation. In order to reduce the risks of these serious complications, patients need to be optimized for surgery by examining the factors that anesthesiologists are used to screening for, such as suspected bleeding disorders and anti-coagulant or anti-platelet therapy, but also the factors that some are less commonly examined, including preoperative MRI of the areas of the spine where the leads will be located. In the cases where moderate to severe spinal stenosis are present, a neurosurgical consult would be warranted to determine if decompression prior to implantation is appropriate. In cases where mild stenosis is present, the physician might consider using a cylindrical lead(s) instead of bulkier paddle-type leads. Although, the absolute incidence of spinal stenosis in the thoracic region

“Will this article negatively influence patient’s willingness to undergo spinal cord stimulation? The answer is that it already has.”

is very low,⁶ its presence should alter the approach to the implantation of SCS.

The *TIME* article neglects to have a fact-based discussion of the surgical risks, but the greatest limitation is that it neglects to discuss the dissonance between the benefits of the SCS therapy and the patient’s expectations. The lack of complete resolution of the patient’s symptoms is portrayed as a failure of the therapy. From the physician perspective, successful neuromodulation trials are categorized based on pain relief of at least 50 percent with coverage of the painful areas as well as improved sleep, function, mood and ability to wean medications. One of the conflicts of the treatment of many painful conditions is that the progress these patients make is more often based upon the effort they put into their therapy and working through their pain to improve function. The therapy for CRPS is not a zero sum game as presented in the article; comprehensive

therapy, including physical therapy, rational medication therapy and cognitive behavioral therapy, is the best route to increased function. The patient’s refusal to involve his/herself in intensive physical therapy and psychological therapy will most likely lead to progressive loss of function regardless of the choice of treatment: opioid or spinal cord stimulation. A more relevant question for this article might have been “is this patient an appropriate candidate for spinal cord stimulation?” However, that topic would have been less sensational than the approach the author employed.

Will this article negatively influence patient’s willingness to undergo spinal cord stimulation? The answer is that it already has. This can, however, be made into an opportunity to have more detailed discussions with our patients about SCS therapy. It also shines a light on the need for a database of complications (and efficacy) of neuromodulation techniques such as spinal cord stimulation.

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How I Do It - Ultrasound-Guided Axillary Block

Continued from page 7

Figure 10a and 10b

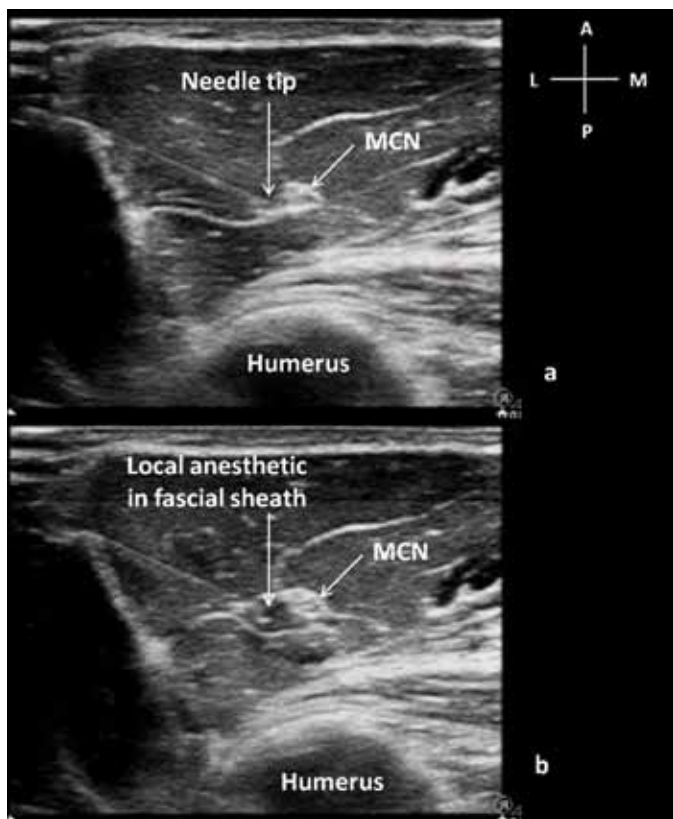


Figure 10a. The musculocutaneous nerve (MCN) is approached at a tangent, the aim being to enter the enveloping fascia without piercing the nerve. Figure 10b. Injection with the needle tip within the fascial sheath will surround the nerve with local anesthetic. (Used with permission of www.usra.ca).

A peripheral nerve stimulator can be used to confirm the identity of the nerves, but we do not recommend seeking a minimum current threshold (e.g. $\leq 0.5\text{mA}$) as the endpoint of needle positioning since this has been shown to correlate poorly with needle-nerve contact.¹⁰ Instead, one should seek an appropriate pattern of LA spread on injection. With experience, the individual nerves can be recognized by their sonoanatomy alone, and neurostimulation becomes unnecessary.

Summary

The axillary block is an extremely reliable and effective method of providing surgical anesthesia of the upper limb at and below the elbow. It is particularly suited for relatively inexperienced practitioners because of its low risk and the potential for safe practice of the in-plane needling technique.

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