ASRA NEWS

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In This Issue

Advancing the Science and Practice of Regional Anesthesia and Pain Medicine

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American Society of Regional Anesthesia and Pain Medicine

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President's Message – Getting Involved with ASRA

When members of the American Society of Regional Anesthesia and Pain Medicine (ASRA) were surveyed recently as part of our strategic planning process, two messages clearly came forth. First, many members asked how to become more involved in the Society; and second, some members expressed concern that decisions were not always transparent with regard to election to the Board of Directors, naming of awardees, selection of committee members, and choosing of faculty for the annual meetings. The members were absolutely correct in their expressions of concern and frank criticism. In reality, we as a Society have not done a stellar job of living up to these expectations, particularly over the past several years. The reasons for these lapses are myriad and not always excusable, but they are not sinister. One can point to recent change of ASRA's management team, a new executive director, and re-ordering of ASRA's senior leadership – all periodic adjustments that professional societies go through from time-to-time. But the administrative distractions of the past few years have also caused the Board of Directors to lose focus on critical tasks such as appropriate updating of our Bylaws and our Administrative Procedures, ensuring compliance within the rapidly changing world of continuing medical education (CME) accreditation, and timely appointment and rotation of ASRA committee chairs and members. I am therefore providing you with an update on two intrinsically related issues – adherence to and improvement of ASRA's policies, and how members can become more involved in the Society if they so choose.

ASRA is incorporated in the Commonwealth of Virginia – a reflection of our 1975 founding and decision to partner with Ruggles, a management company based in Richmond. The Society's governance emanates from the legal document that is our Bylaws, which have been amended over the years as the Society has grown and matured. During 2012, our Bylaws were completely reassessed and updated and, after review by our association attorney, approved by the Board on January 4th of this year. The Bylaws set the structure for how ASRA is managed - from the description of the officers, to how the directors-at-large are elected, to what are the standing committees, including the qualifications and term limits for chairs and members. Since its founding, ASRA has been structured based on a top down management paradigm; that is, the Board elects the officers, the Board elects the directors-at-large, and the President appoints most of the committee chairs and members, with the Board of Directors having final approval over these appointments. The Society's Administrative Procedures spell out the day-to-day details that range from officer job descriptions, down to and including how large our checking account balance can become before the excess is moved to short-term investments.

Joseph M. Neal, M.D.
ASRA President

The past several years, culminating with the recent updating exercise, have resulted in several changes to the ASRA Bylaws that have specifically targeted the goal of increasing member opportunity to serve on the Board of Directors and to play a greater role in the nomination process for directors and committee members. In the August 2012 issue of the ASRA Newsletter, I described a major change to the Bylaws that involved reconfiguring the Board of Directors structure. This amendment was intended to increase (by approximately 25%) the opportunity for any given member to serve on the Board of Directors and possibly to become an officer of the Society. In brief, eliminating the position of Associate Treasurer and replacing that person with a seventh Director-at-Large opened more chances to serve on the Board and decreased the length of time officers serve. A parallel reduction of director terms from a maximum of three two-year terms to a maximum of five years reduces the time directors serve and increases competition for election to the office of Treasurer, from which all higher officer positions ascend sequentially. In November 2012, the Board approved further changes to the standing committee structure. With the goal of providing a direct line of communication from the Board to the committees in order to foster consistent implementation of strategic goals, the Bylaws were amended such that the chairs of the Research, Membership, and Communications Committees would be directors of the Society appointed for a two-year term. The Communications Chair now oversees the work of several sub-committees (Newsletter, E-News, Website, and Social Media), each of which is chaired by a non-board member. Because of its critical importance to the Society's mission and continued accreditation as a CME provider, the chair of the CME Committee continues to be a past officer or current member of the Board. To ensure balanced expertise on two crucial committees, the Bylaws were amended to mandate that the CME and the Research Committees each have a vice-chair of opposite sub-specialty than the chair.

So how do these changes impact member participation in ASRA? The Board reconfiguration now requires the solicitation of member-generated nominations for open director-at-large positions. While election of directors remains the purview of the Board itself, as specified in the Bylaws, the Nominations Committee now gives serious consideration to member input. The existing practice of soliciting nominations from the membership continues with regard

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Editorial

Edward R. Mariano, M.D., M.A.S.
Editor

In this issue of ASRA News, we feature some really amazing content. Dr. Philip Peng completes his two-part series on ultrasound-guided injections for shoulder pain, Dr. Peter Cheng brings us the first part of his two-part series on rotator cuff sonopathology, Dr. Daniel Borman reviews epidural steroid injections, and Dr. Michael Barrington updates us on the groundbreaking AURORA project.

However, I do want to highlight a special Pro-Con feature dedicated to the controversial topic of regional anesthesia and analgesia in the patient at risk for acute compartment syndrome. I want to personally thank our surgical colleagues from the University of Alberta who were willing to write a thoughtful "Con" article for our newsletter. Before jumping to debate each of their points, we need to give them careful consideration. With the paucity of evidence-based recommendations on this topic, it is crucial to have an open honest dialogue between all members of the healthcare team. This Pro-Con is not meant to provide answers but to provide talking points for an ongoing conversation.

In my previous position at UCSD, we had a Level 1 trauma center where we would keep one operating room (OR) set up and warm at all times for the occasional direct-to-OR resuscitation. We saw all types of acute and subacute orthopedic trauma, and no two cases were approached the same way. Did I consider regional analgesia for each of these patients? Yes. Did I perform regional analgesia for all of them? No.

In order to have a meaningful discussion on this topic with our surgical colleagues, we must first be part of the conversation. In the specialty of Regional Anesthesia and Acute Pain Medicine, this means emphasizing more the "Acute Pain Medicine" part than the "Regional Anesthesia" part. The value that we bring to perioperative patient care must be more than just a set of interventional peripheral nerve and neuraxial block techniques. We have to know when these techniques are and are not indicated and have other modalities for analgesia at our disposal when providing consultation on complicated trauma patients. In addition, the service we provide cannot be time-limited. How can we say that superior pain control is only available from 7 am to 5 pm, not including weekends and holidays?

When it comes down to it, managing patients at risk for compartment syndrome is tough. The benefits of analgesia have to be weighed with the potential for neurovascular compromise. Sometimes you will perform regional analgesic techniques for them; other times you won't. Sometimes, you will place catheters that you can dose later when the risk profile improves; other times you may be consulted for help later in the hospital stay. Sometimes you will convince the surgeon to preemptively perform fasciotomies in a patient in whom you anticipate a difficult postoperative course. The context for this decision-making will vary from institution to institution, but ongoing communication with the surgical team is indispensible. Be a consultant; be available; and continue to be part of the conversation.

Making Epidural Steroid Injections Safer for Patients

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In light of recent events regarding epidural injections and fungal meningitis outbreaks, it is an ideal time to review and reflect on the necessary steps to ensure safe epidural steroid injection (ESI) practice. These steps can be divided into 3 categories: pre-procedural, procedural, and post-procedural. Essential pre-procedural steps include a detailed history and physical, diagnostic imaging, documentation of risks and benefits, and the determination of whether more appropriate alternatives to ESI exist for the treatment of the patient's pain. It is also important to include all physicians involved in the care of the patient to ensure optimal care management.[1] There are many procedural steps necessary for performing safe ESI including, but not limited to, sterile technique, proper monitoring, time-outs, and the use of fluoroscopy. Post-procedural steps include adequate nursing care to monitor vital signs and post-ESI pain scores, patient education regarding possible complications, proper follow-up, and, of course, documentation of these steps.

The first step for any patient encounter is to perform a detailed history and physical examination. The most common indication for ESI is radicular pain due to a herniated disc. The straight-leg raise (SLR) test and crossed SLR test are the most effective physical exam maneuvers at a pain physician's disposal; both of these

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Regional Anesthesia in the Patient at Risk for Acute Compartment Syndrome

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The Use of Regional Anesthesia When the Risk of Compartment Syndrome Exists: Yes!

Acute compartment syndrome (ACS) can occur in any closed compartment but is most commonly seen in the osteofascial compartments. It is caused by reduced tissue perfusion secondary to elevated pressure. ACS is principally of concern during emergency surgery, primarily of leg[1] and forearm fractures.[2] The incidence is 4.3% in patients with tibial shaft fracture and 3.1% with diaphyseal forearm fracture.[3] A number of case reports describe the occurrence of ACS following elective joint arthroplasty.[4-5] Regional anesthesia is commonly used in trauma patients, soft tissue injuries, and elective joint surgery. The benefits are proven and significant. From the patient's perspective, it seems obvious that the risk of ACS does not necessarily have to contraindicate the use of regional anesthesia. However, practice seems to be based on the opinion that sensory blockade and the relief of postoperative pain precludes any use of regional anesthesia.[6] Unfortunately, this perspective is not based on randomized-controlled trials that compare outcomes in patients at risk of ACS who have received a regional anesthesia technique versus those who have not. Rather, clinical practice is based on case reports and retrospective case series. Physicians opposed to the use of regional anesthesia report that the technique masks the pain related to ACS. However, it is well demonstrated in the early stages of compartment syndrome that the sensitivity of pain and passive stretch pain as diagnostic signs is only 19% while the positive predictive value for both signs is 14%.[7] On the other hand, it has been seriously postulated that

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CON

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Acute Compartment Syndrome: Caution Ahead

Acute compartment syndrome (ACS) is a potentially devastating feature of extremity injury, characterized by acute and rapidly-progressive neurological and muscular dysfunction associated with an increase in intra-compartmental pressure in an injured extremity. ACS can occur in any closed fascial compartment in the upper or lower extremity. Estimates of incidence following tibial fracture are approximately 7% (ranging from 2.7% to 11%)[1] while forearm and elbow injuries account for 8.5% of ACS.[2] The best possible outcome follows prompt diagnosis and immediate fasciotomy of the compartments involved to relieve the pressure and re-establish circulation. Given the reported incidence and the potentially devastating sequelae of ACS, the use of regional anesthesia needs to be carefully scrutinized before being considered as an option for surgical fixation of extremity injuries.

Pathophysiology of ACS

An initial insult causes tissue ischemia in the affected fascial compartment. This insult can be an initial increase in pressure (either an extra- or intra-compartmental source) or from a direct transient ischemic injury leading to an inflammatory process whereby local edema causes an increase in swelling. Eventually, the intra-compartmental pressure is sufficiently high that it prevents blood flow through the low-pressure capillary system in the affected tissues, and a local shunt develops. Tissue perfusion is decreased further which worsens the local ischemia. This creates a positive feedback loop that propagates the ischemic insult to the local tissues resulting in ACS (Figure 1). This is complicated by a direct ischemic injury to the basement

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compartment ischemia activates nociceptors via bradykinin, serotonin, adenosine and hydrogen ion excitation that can render ineffective the sensory blockade of regional anesthesia.[7,8] The most important aspect in the management of ACS is early diagnosis with treatment onset in less than 6 hours.[9]

Neuraxial Blockade And Lower Limb Compartment Syndrome Risk

Neuraxial blockade can be used for anesthesia and/or postoperative analgesia in trauma and burns patients. Epidural anesthesia demonstrates several benefits compared with general anesthesia in reducing postoperative thromboembolic disease and respiratory complications.[10-12] When the risk of ACS exists, the challenge for the anesthesiologist is to obtain motor and sensory blockade duration more or less identical to the duration of surgery. The use of short acting local anesthetics seems to be the solution. Epidural administration of lidocaine 1.5% or 2-chloroprocaïne 3% results in a duration of sensory blockade less than 220 minutes and 160 minutes, respectively.[13] For postoperative analgesia with an epidural catheter, many studies show that the use of local anesthetic (LA) of low concentration allows excellent analgesia. The full regression of sensory block occurs within 2 to 4 hours after stopping the infusion.[14-15] Several cases of ACS involving epidural analgesia have been reported.[16] However, in most cases the infusion drug and concentration were not specified, and subsequently it is difficult to attribute the delay of ACS diagnosis to the regional analgesic technique alone. For spinal anesthesia, an alternative is either the use of low doses of hypobaric bupivacaine or the use of continuous spinal anesthesia (which has never been associated with ACS). A randomized, double-blinded study of 150 patients undergoing elective unilateral orthopedic surgery with either hypobaric bupivacaine 0.15% 4.5 mg (3 mL; Group 1), 6.0 mg (4 mL; Group 2), or 7.5 mg (5 mL; Group 3) reported a dose-dependent increase in the duration of the block from 1:55 +/- 00:20 hours (Group 1) to 2:15 +/- 00:22 hours (Group 2), and 3:15 +/- 00:31 hours (Group 3).[17]

Peripheral Nerve Blockade (PNB) And Limb Compartment Syndrome Risk

This is clearly the most contentious area. In all operating theaters and emergency units, patients have not benefited from PNBs due to the purported risk of ACS. It will be helpful at this juncture to appraise the published case reports to date (Table 1).

In all described patients, the association between single-injection or continuous PNB and delayed diagnosis of compartment syndrome is questionable. Most importantly, ischemic pain was present in all cases which was not relieved by a single-injection or continuous infusion of LA administered at anesthetic or analgesic doses that had been effective earlier in the postoperative period. Again we wish to stress that the choice of LA for surgical anesthesia and LA concentration for postoperative analgesia are the most important factors to avoid prolonged motor and sensory blockade. In the case of procedures not normally associated with severe post-operative pain, it seems to be appropriate to use short acting LA as lidocaine, 2-chloroprocaine, and mepivacaine for surgical anesthesia.[22] Plain lidocaine 1.5% for axillary block has a duration of anesthesia of up to 175 minutes; whereas 2-chloroprocaine 3% results in sensory blockade of up to 112 minutes and 101 minutes for sciatic and femoral block, respectively.[23,24] For surgical procedures normally associated with significant post-operative pain, if a risk for ACS exists, a delay in starting the continuous infusion should be considered, or the application of a very low concentration of local anesthetic to prevent profound sensory and motor blockade may be appropriate.[7]

Conclusion

The pernicious influence of tales and legends can be difficult to eradicate from our clinical practice. With regard to the role of regional anesthesia, primarily PNB, in masking the occurrence of ACS after trauma surgery, it appears that one is presumed guilty until proven innocent.[6] The risk of compartment syndrome should not contraindicate the use of regional anesthesia. However, its use remains controversial among orthopedic/trauma surgeons and anesthesiologists worldwide. It is very important to really understand the pathophysiology of this syndrome, the characteristics of ischemic pain, and the importance of rapid surgical management. The most important thing is the establishment of vigilant clinical monitoring for compartment syndrome after surgery. Until stronger evidence is available, clinical practice should not be based on unfounded fears that may be denying patients the benefits of regional anesthesia and analgesia.

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Table 1

Reference	Surgery	Technique used	Comments
Hyder[18]	Intramedullary nail for a tibial fracture	Femoral "3-in-1" block; bupivacaine 0.5%	**Pain** in the calf and anterior part of the leg; anterior tibial compartment necrosis (peroneal nerve territory). **3-in-1 block held responsible for delayed diagnosis.**
Noorpuri[19]	Forefoot revision arthroplasty	Ankle block; bupivacaine 0.25%	**Severe pain** unrelieved by pain killers within 12 hours. Fascitomy without sequelae.
Uzel and Steinmann[20]	Intramedullary nail for femoral shaft fracture	Femoral nerve block; 20 mL ropivacaine 0.75%	**Severe thigh pain** and elevated anterior thigh compartment pressure. Fasciotomy without sequelae.
Cometa[8]	Distal femoral and proximal tibial osteotomy	**Continuous** sciatic and femoral nerve blocks with ropivacaine 0.2%	**Severe leg pain** without other symptoms on second post-operative day. Tense calf on the operative side. Fasciotomy with sequelae.
Walker[21]	Left calcaneal osteotomy and Achilles tendon lengthening	**Continuous** popliteal block; initial bolus of 15 mL of bupivacaine 0.5% with epinephrine then ropivacaine 0.2% at 8 mL/hr	**Increasing pain** in the foot on day one. Cast removal and infusion stopped.
Aguirre[7]	Complex distal humerus fracture	**Continuous** infraclavicular block; bolus 30 mL ropivacaine 0.5% then ropivacaine 0.3% at 6 mL/h plus an additional bolus of 5 mL.	**Severe forearm pain** 14 hours after surgery; compartment pressure measured at 40 mmHg; Fasciotomy without sequelae.

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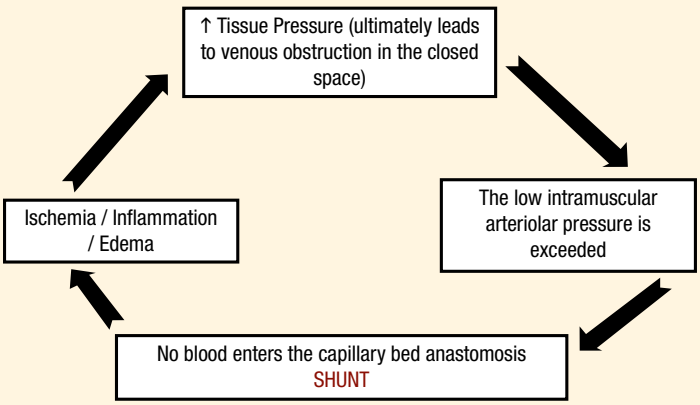
membrane of the vessels supplying the compartment, resulting in a local transudate into the interstitial space once flow is re-established.[2] Once the cycle of progressively-worsening ischemia has begun, there is a limited amount of time available for diagnosis and treatment before irreversible damage to nerves and muscles in the affected compartment occurs. A time from diagnosis to fasciotomy of as little as seventy minutes has been associated with poor outcomes.[3]

Diagnosis of ACS

Diagnosis is made by the interpretation of clinical signs and symptoms by an experienced clinician in an alert, cooperative patient who is at risk for ACS. The initial symptom is acute, severe, unremitting pain to the extremity that should prompt an immediate clinical assessment of the sensory and motor function of the nerves traversing the relevant compartment. Other clinical signs include progressive paresthesia to the skin and paralysis of muscles supplied by the nerves that run through the affected compartment (Table 1). A new progressive deficit in this setting indicates a localized shunting of blood through the affected compartment and tissue ischemia necessitating immediate fasciotomy.[2]

Figure 1

The pathophysiological process of acute compartment syndrome is illustrated as a positive feedback loop that continues



It is clear that clinical findings are of paramount importance in the diagnosis of ACS (Table 1).[1,4-6] In the unconscious or otherwise uncooperative patient, the diagnosis is more difficult to make, and the syndrome is less clearly defined. Attempts to diagnose ACS in this complex group of patients have been very unsatisfactory, with experts highlighting the lack of diagnostic tools at our disposal beyond clinical examination.[4] Pressure monitoring is not the definitive answer as will be further discussed. In practice, we likely over-diagnose ACS in the unconscious patient due to fear of a missed syndrome. However, fasciotomy has documented adverse effects and unnecessary surgery should be avoided.[7]

Table 1

Number of articles reporting various clinical findings related to acute compartment syndrome (adapted from: Ulmer T. Journal of Orthopaedic Trauma 2002;16(8):572-7)

Conclusion	Pain	Paresthesia	Pain with Passive Stretch	Paresis
Most important/ reliable sign	3	5	5	2
Subjective/ unreliable sign	4	3	3	3
Earliest sign	8	4	2	0
Latest sign	0	5	0	5

Regional anesthesia blocks the initial pain response and precludes recognition of the early, reversible stages of ACS. Likewise, once concern regarding ACS has been raised, neural blockade renders the detailed examination of the patient for progressive sensory and motor deficits unreliable, if not impossible. Given the often rapid progression of the disease, the inability to promptly diagnose ACS can lead to permanent deficits. Regional anesthesia in these at-risk patients compromises the chances for a prompt diagnosis and its associated improved outcome. Ulmer eloquently showed that clinical examination has a high specificity (97%) and negative predictive value (98%) indicating that absence of clinical signs correlates well with absence of ACS.[6] Furthermore, the presence of 3 signs (pain, pain with passive stretch, and paresis) has 93% odds of being associated with ACS.[8] This specificity will be compromised with the use of regional anesthesia. Other clinical signs not affected by nerve blockade have been shown

"Once the cycle of progressively-worsening ischemia has begun, there is a limited amount of time available for diagnosis and treatment before irreversible damage to nerves and muscles in the affected compartment occurs."

to be unreliable. Palpation of the limb to estimate compartment hypertension has been shown to have poor inter-observer reproducibility.[9] Vascular findings of pulselessness and pallor should not be present due to ACS but instead indicate an acute arterial lesion that requires a completely different approach and treatment.

Prolonged motor and sensory blockade due to long-acting local anesthetic agents (e.g., bupivacaine 0.5%) may last up to 20 hours while even intermediate-acting agents such as lidocaine can result in blocks lasting up to 5 hours. It is obvious that a block lasting up to 5 hours can greatly harm our ability to recognize, diagnose, and treat an ACS within a 1-2 hour time frame, the current standard of care.

Pressure Monitoring

Although compartment pressure can be accurately measured with the use of a simple hand-held device, the utility of this measurement is wanting. Since ACS is a progressive phenomenon, single measurements of compartment pressure are not useful in the absence of clinical findings suggestive of ACS. Transiently-increased compartment pressures alone have been found to be non-predictive for the development of ACS in patients undergoing tibial nailing.[10] Some centers have successfully implemented a system of continuous compartment pressure surveillance for patients at high risk of ACS.[11] However, the use of continuous pressure monitoring as a screening tool for ACS has not obviated the need for a detailed neurological examination, and its transferability to other centers has not been universally successful.[5]

Conclusion

The current practice of orthopaedic trauma surgeons reflects that, "Despite its drawbacks, clinical assessment is still the diagnostic cornerstone of ACS."[4] There is little evidence to suggest that deviation from this philosophy is compatible with safe practice at this time. Thus, any technique that impairs the clinician's ability to perform a reliable clinical examination in a cooperative, alert patient following surgery on the leg or arm at risk for ACS should be carefully considered.

For the full reference list, please see the online version at:
http://www.asra.com/publications-newsletters.php

President's Message – Getting Involved with ASRA *continued...*

to the Labat, Bonica, and Distinguished Service Awards. What about members who wish to become more involved with the Society over and above offering nominations? Here, there have been two major process improvements for how members make known their desire to serve on committees or meeting faculty.

Beginning this past February, members were offered the opportunity to volunteer for membership on certain ASRA committees by using an on-line process that involves self-nomination and an accompanying letter of recommendation. Unfortunately, despite moderately high interest, not every volunteer could be matched with a committee appointment because the number of volunteers exceeded the available positions identified in the Bylaws. The President, in consultation with the appropriate committee chairs, attempted to take into consideration the necessary balance between pain and regional anesthesia specialists, age groups within the Society, and particular expertise of the volunteer. In addition, the President and Board consciously managed appointments so as to minimize the same individuals serving on multiple committees. The Bylaws stipulate that member-at-large appointment is for a three-year term and that terms be staggered to ensure continuity within the committee structure. The exception is the annual meeting program committees, which are one-year terms. Because few appointments were made over the past two years, some members-at-large were appointed recently for terms less than three years to re-establish staggered terms. After a member has served one term on a committee, they can be re-appointed for a second and final three-year term after an absence of at least one year from membership on that committee.

Members frequently ask how to become part of the annual meetings faculty. This is an issue with which ASRA leadership has struggled mightily over the years – trying to balance providing opportunity for young faculty members against the clear and consistent feedback from meeting attendees that they expect refresher course lecturers, practice advisory panels, and parallel topic discussants to be selected from the best experts. For nearly a decade the Society has experimented with the 'associate faculty' concept, in which younger colleagues have been invited to share workshop or resident session teaching duties with a senior colleague. While generally successful, some associate faculty mistook this as a guaranteed entry into more visible roles; conversely the Society occasionally felt discomfort when an 'associate faculty' member had been in that role for five or six years. Consequently, the CME Committee has thoughtfully deliberated and will present for the Board's expected approval a well-defined policy for those who wish to become associate faculty. First, potential associate faculty members will state their intent via a formal application that is accompanied by recommendation from someone familiar with their teaching abilities. The appropriate program committee will then attempt to place as many volunteers as possible as co-faculty for workshops and some resident programs. Importantly, an individual will be limited to the associate faculty role for no more than three meetings. After this initial opportunity, future appointment will be as full faculty in accordance with the needs of the program committee for that particular meeting. Selection of major lecturers will continue to be based on their reputation and acknowledged expertise, which typically is exemplified by their record of publication or demonstrated leadership on the topic.

How I Do It: Ultrasound-Guided Injection for the Shoulder (Part II)

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This article summarizes the anatomy, sonoanatomy, efficacy, accuracy, and the injection techniques for the glenohumeral joint (GHJ) and subacromial subdeltoid bursa (SASDB). Patient selection for those injections is also reviewed. A detailed review can be found in two recent articles.[1,2]

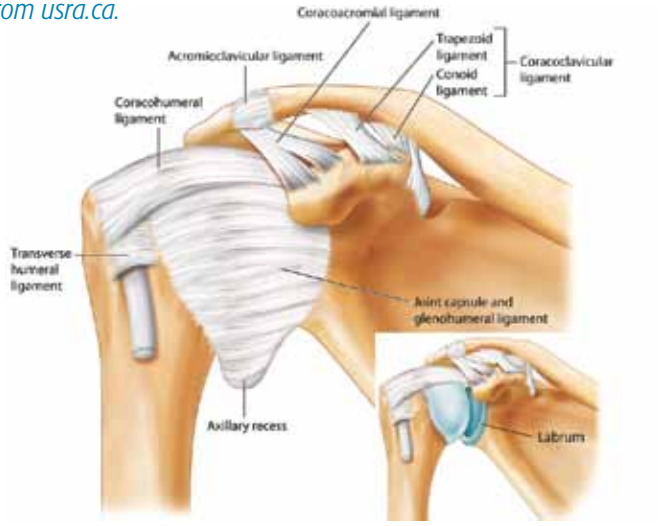
Glenohumeral Joint

Anatomy and Sonoanatomy

The GHJ is a ball-and-socket joint composed of a round humeral head and a relatively small, flat pear-shaped glenoid fossa. The glenoid cavity is deepened by the presence of a fibrocartiginous rim, the *glenoid labrum* (Figure 1). The joint capsule is attached medially to the margin of the glenoid cavity extending to the base of the coracoid process and laterally to the anatomical neck of the humerus (Figure 1).[3,4] The synovial membrane lines the capsule on its deep surface and overlies the long head of biceps (LHB) tendon. From there, three recesses are formed: the biceps tendon sheath anteriorly, the subscapularis recess medially, and the axillary pouch inferiorly (Figure 2).[3] The presence of the LHB recess allows a potential portal for GHJ entry.[1,3]

Figure 1

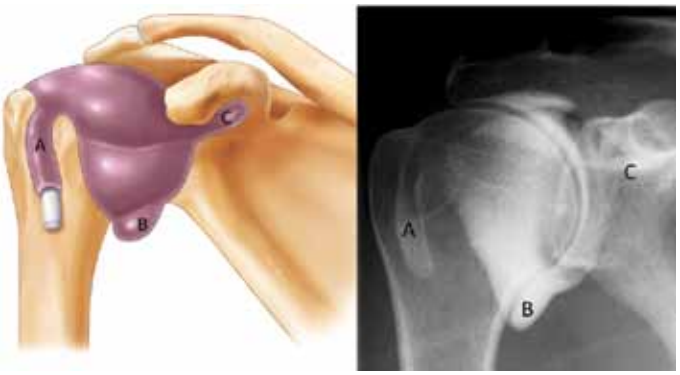
Glenohumeral joint showing various ligaments and the joint capsule. The anterior capsule is reinforced by the superior, middle and inferior glenohumeral ligament. The insert shows the articular surface, glenoid process, and labrum. Reprint with permission from usra.ca.



The GHJ can be accessed anteriorly, posteriorly, and from the rotator cuff interval.[1] The techniques for the anterior and posterior approaches will be described. For the posterior approach, the patient is placed in either the sitting or lateral decubitus position with the ipsilateral hand placed on the contralateral shoulder. A linear probe of frequency 6-13 MHz is usually used unless the patient is of very high body mass index or strong muscular build. The ultrasound probe is placed just caudal to the acromion over the infraspinatus tendon (Figure 3). The key structures to identify are humeral head, labrum, infraspinatus tendon, and joint capsule. For the anterior rotator cuff interval approach, the patient is placed in supine position and a high frequency linear probe 6-13 MHz is used. The first scan is similar to the scanning of the long head of the biceps (LHB) tendon at the bicipital groove. Following this scan, the LHB is traced in the cephalad and medial direction until it is seen in between the supraspinatus and subscapularis muscle underneath the coraco-humeral ligament (Video 1; http://www.asra.com/publications-newsletters.php).

Figure 2

The drawing of three main recesses of the joint (left): A=the biceps tendon sheath; B=the axillary pouch; C=the subscapular recess; and the corresponding radiographic (arthrogram) appearance (right). Reprint with permission from usra.ca.

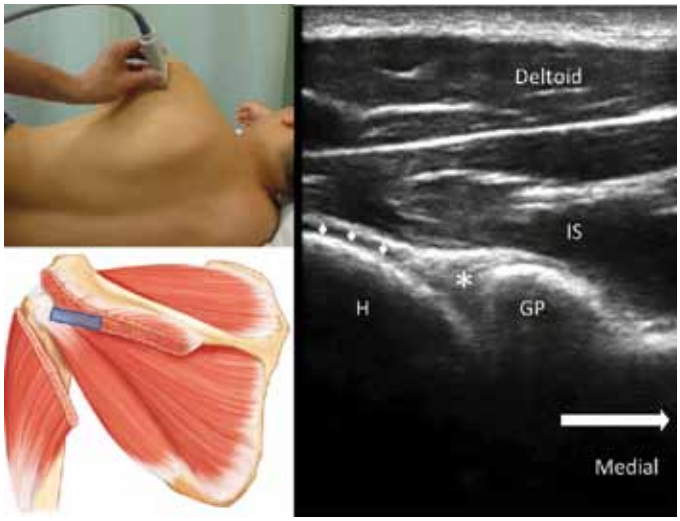


Injection Techniques

The target for the posterior approach is between the free edge of labrum and the cartilage of humeral head underneath the capsule (Video 2; http://www.asra.com/publications-newsletters.php).[1,2] Once the target is obtained, a 22 gauge 3.5-inch spinal needle is inserted from lateral to medial direction with in-plane technique. The injectate is 4 mL of local anesthetic with steroid (e.g., 2% lidocaine and 40 mg methylprednisolone acetate). If one encounters resistance during injection with the posterior approach, two maneuvers can be used to adjust the needle: turn the bevel 90 degrees or withdraw the needle slightly. Correct placement of injectate in the posterior approach will result in the spread of medication beneath the joint capsule.

Figure 3

*Ultrasound image of the posterior glenohumeral joint. The glenoid process and humeral head both appear as hyperechoic structures with anechoic shadow. The insert on the top shows the position of the patient and the ultrasound probe while one below shows the probe position and the structures underneath; IS=-infraspinatus muscle; H=humeral head; GP=glenoid process; *=glenoid labrum; ◆=the articular cartilage of the humeral head. Reprint with permission from usra.ca.*



The target for the rotator cuff interval approach is the space on either side of the LHB deep to the coracohumeral ligament (Figure 4). Once the target image is obtained, a 25 gauge 1.5-inch needle is inserted either out-of-plane (caudal to cephalad direction) or in-plane (lateral to medial direction). The injectate is local anesthetic with steroid as previously described.[1,2]

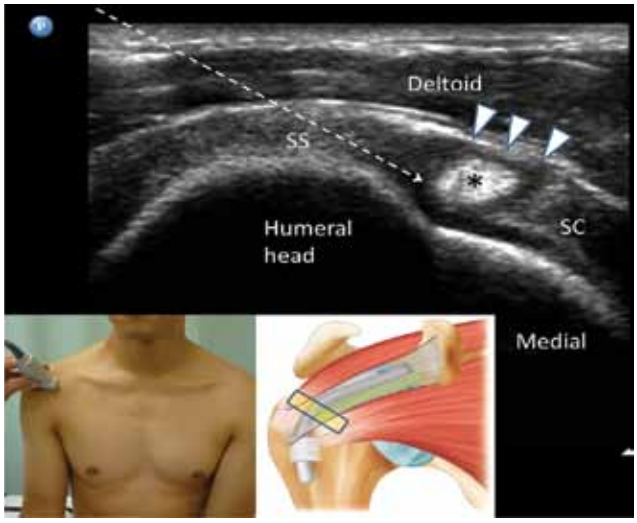
Literature Review

The main indications for GHJ injection are adhesive capsulitis and glenohumeral arthrosis. The efficacy for adhesive capsulitis had been reviewed.[5] Compared with control and physiotherapy, the review showed that multiple injections were beneficial until 16 weeks from the date of the first injection. In terms of multiple injections, this review supported that up to 3 injections were beneficial, but there was limited evidence that 4 to 6 injections were beneficial. The role of GHJ injection as part of the conservative therapies for adhesive capsulitis needs to be emphasized. Intra-articular steroid injection has been shown to produce significant reduction in pain and disability after treatment when combined with exercise versus exercise alone.[51]

The role of GHJ injection in the management of arthrosis is unclear. There are no studies specifically addressing the efficacy in GHJ arthrosis. A recent practice guideline from the American Academy of Orthopedic Surgery found no evidence to support

Figure 4

Ultrasound image of the rotator cuff interval and the needle insertion trajectory. The insert shows the position of the probe and corresponding anatomical structures underneath. The LHB tendon (*) is always hyperechoic at this level and sandwiched between the supraspinatus tendon (SS) laterally and subscapularis tendon (SC) medially. The coracohumeral ligament (arrow heads) forms the roof of the interval. The needle (indicated by the dashed arrow) is inserted from lateral to medial into the rotator cuff interval. Reprint with permission from usra.ca.



or refute the use of intra-articular corticosteroid injection for the treatment of GHJ arthrosis.[7] There is some evidence to support the use of viscosupplement in GHJ osteoarthritis, an off-label use for viscosupplement, based on an industry-sponsored study.[8]

The overall success rates of the landmark-based technique range from 27% to 100%.[1] In contrast, the success rates of the ultrasound-guided studies are consistently higher. A recent review examining the accuracy of GHJ injection demonstrated that the pooled success rates of the landmark-based and image-guided were 79% and 95% respectively.[9] Of note, an interesting comparison study was conducted examining the ultrasound-guided approach and landmark-based (conventional) approach in various joint interventions. The ultrasound-guided injections were all performed by a rheumatology trainee (9 months into a rheumatology program with 8 sessions of MSK ultrasound training), and the conventional approach was all performed by 9 rheumatology consultants with a median of 9 years of experience.[10] The accuracy rates for shoulder injections were 63% and 40% for the ultrasound and conventional groups, respectively. Despite the contrast in experience, the trainee achieved better accuracy, which was associated with better pain relief at the sixth week.

Subacromial Subdeltoid Bursa

Anatomy and Sonnanatomy

There are four rotator cuff muscles: subscapularis (SSC), supraspinatus (SS), infraspinatus (IS) and teres minor (TMi) muscles. The rotator cuff is a tight layer of tendons around the GHJ on the anterior (SSC), superior (SS), and posterior (IS and TMi) aspects of the shoulder.[1,3,11] It plays an important role to stabilize the humeral head in the shallow glenoid fossa during movement of the arm. The subacromial subdeltoid bursa (SASDB), the largest bursa in the body, is located inferior to the acromion, the coraco-acromial ligament (CAL), and the deltoid muscle. It overlies the superior aspect of the SS tendon.[3,11] It also extends anteriorly to cover the bicipital groove and medially to the coracoid process (subcoracoid bursa). The lateral border may reach approximately 3 cm below the greater tuberosity.[3] The main role of the SASDB is to minimize attrition of the cuff against the coracoacromial arch (acromion and CAL) and deltoid muscle during movements of the arm.

To reveal the SASDB, the patient is placed either supine or sitting in the reclining position with the back supported. The patient is advised to put the hand in modified Crass position (gesture of "put the money back in the back pocket"). The ultrasound probe used is high frequency 6-13 MHz linear, and the SASDB can be viewed in either long- or short-axis (Video 3 and 4; http://www.asra.com/publications-newsletters.php).

Injection Technique

The target is the bursa outlined by the peribursal fat. A 25 gauge 1.5-inch needle or 22 gauge 3.5-inch spinal needle is inserted in-plane. If the needle tip is in the bursa, a small volume (0.5 mL) of injectate will be seen spreading across the bursa plane (Video 5; http://www.asra.com/publications-newsletters.php). Once the position of the needle is satisfactory, 4 mL of local anesthetic with steroid (0.25% bupivacaine and 40 mg methylprednisolone acetate) can be injected.

Literature Review

The indication for SASBD injection is subacromial impingement syndrome, which covers a constellation of conditions: partial- and full-thickness rotator cuff tear and rotator cuff tendinopathy.[12] The common presentations of patients with rotator cuff disease are pain and stiffness. Pain is the predominant symptom, often most troubling at night and with overhead activities. The rotator cuff tear can be articular-sided, bursal-sided, or intratendinous, and the incidence increases with age. The tear can be partial- or full-thickness, but partial tendon lesions are often much more painful than full-thickness tears.[13]

Physical examination reveals pain with active range of motion between 60 and 100 degrees of abduction (painful arc), as well as loss of active range of motion but relatively-preserved passive range of motion. Both are suggestive of a rotator cuff pathology. Provocative tests for rotator cuff pathology are Hawkins' impingement test, Neer's test, and "empty can" supraspinatus test.[2]

The efficacy of SASDB injections for rotator cuff disease has been the subject of multiple reviews.[1] One of the more recent systematic review included 9 randomized controlled trials specifically appraising the use of subacromial steroid injections in rotator cuff disease.[14] The authors concluded that subacromial steroid injection was not efficacious in the treatment of rotator cuff disease. It is important to note that the injection techniques included in those 9 studies were all "blind" injection, with the exception of 1 study in which injections were performed with X-ray guidance. Previous investigation has suggested that the X-ray-guided technique is not reliable or dependable.[15]

In a practical clinical setting, subacromial injection is usually performed in a multimodal approach with physiotherapy or a rehabilitation protocol. A recent large, pragmatic, randomized controlled trial[16] showed that the subacromial steroid injection decreased pain and improved functional outcome at 1 and 6 weeks, but there was no difference compared with exercise alone at 3 and 6 months. The absence of long-term efficacy is not uncommon for interventions of common MSK problems. In examining results from recent high-quality randomized controlled trials for common MSK disorders, Foster et al found no or very small differences in the effectiveness of different approaches when based on long-term outcomes (6-12 months).[17] This has been exemplified by the various shoulder injection techniques described in the previous sections.

The SASDB is a very small target, and precise injection should be performed with image guidance. With the exception of one study (which showed 100% success rate[18]), the literature demonstrates success rates for landmark-based approaches ranging from 29% to 70% in clinical studies and 70% to 91% in cadaver studies.[1] The success rates were similar irrespective of different approaches (posterior, anterolateral, and lateral) for the blind injection and experience and confidence of the practitioners. In studies in which the blind procedures were performed by very experienced orthopedic surgeons and shoulder specialists, the confidence correlation (the accuracy rate when the practitioners were very confident that they were accurate) ranged from 42% to 66%.[19,20]

Among those commonly-used imaging modalities, Mathews and Glousman[15] found that X-ray was an unreliable method in confirming the location of contrast in the subacromial space. Ultrasound-guided injection was validated with MRI in one study, and the accuracy was 100%.[33] The use of the ultrasound-imaging technique in the diagnosis of rotator cuff disease has been extensively investigated, and the reliability is comparable with that of MRI.[21-23]

Conclusion

Application of ultrasound for shoulder injection is increasingly popular. Ultrasonography allows accurate localization of the various target structures for shoulder injections and real-time guidance of needle insertion. An in-depth understanding of the anatomy and sonoanatomy is of paramount importance in performing these ultrasound-guided injections.

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Calcific Tendinosis of the Rotator Cuff: Sonographic Evaluation

Introduction

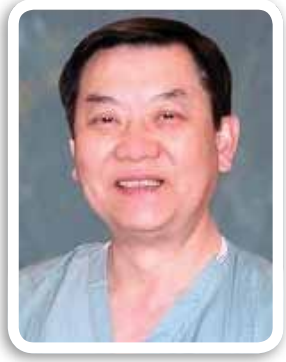
Application of ultrasound in pain medicine is emerging as a popular technique because of the following multiple advantages: absence of ionizing radiation, and the ability to image soft tissue in high resolution, which provides physicians an ideal imaging modality to diagnose and guide intervention. However, unlike our colleagues in radiology, physiatry, rheumatology, and sports medicine, anesthesiologists have yet to embrace ultrasonography for the diagnosis of musculoskeletal diseases due to obstacles such as medical liability, training, and credentialing. Nevertheless, anesthesiologists have readily accepted ultrasound-guidance for interventions such as joint and tendon sheath injections.

Calcific tendinosis is a common source of shoulder pain that is easily diagnosed by radiography and sonography. Interventional treatment options include arthroscopic excision and debridement, ultrasound-guided fenestration (tenotomy), aspiration, lavage, and steroid injection.[1,2]

This article will review the clinical presentation of calcific tendinosis of the rotator cuff and demonstrate the salient sonographic features of this disease that will help guide treatment. The following issue will present my approach for treatment using fenestration, lavage, and steroid injection.

Etiology and Pathophysiology

Calcific tendinosis refers to the deposition calcium crystals within the rotator cuff (Fig. 1A).[2] Although the calcium deposit may remain silent, it can cause pain in about one-half of the population with this condition.[3] Potentially lasting a few years, the calcium deposit will eventually undergo spontaneous resorption with resolution of pain. Although the calcium plaques may appear in any of the four tendons of the rotator cuff, the plaques are located mostly within the supraspinatus tendon and rarely in the subscapularis tendon. The presenting symptoms are highly variable and depend on the stages of calcification.



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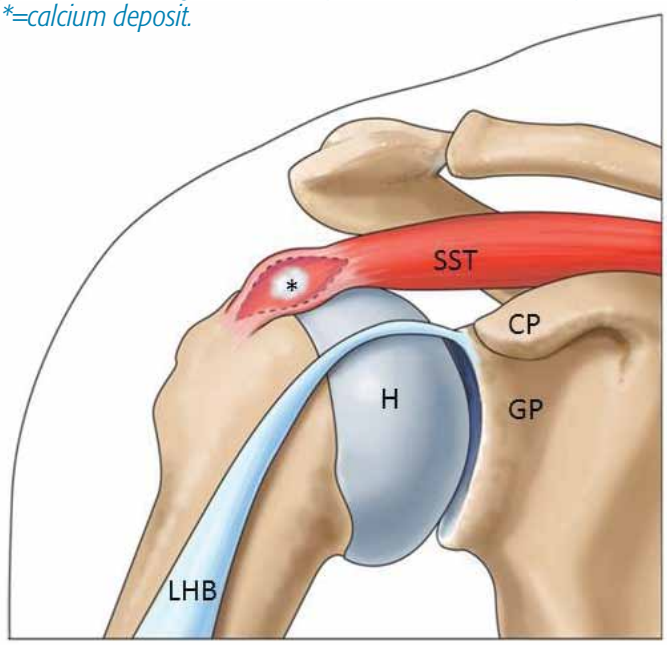
Uhthoff et al[2] have described three stages of calcification:
1. Precalcific stage (fibrocartilaginous metaplasia of the matrix)

Figure 1A

*Calcium deposit embedded within the supraspinatus tendon; H=humeral head; SST=supraspinatus tendon; GP=glenoid process; LHB=long head of biceps brachii; CP=coracoid process; *=calcium deposit.*



2. Calcific stage:
    a. formative phase (calcium deposition within fibrocartilaginous matrix)
    b. resorptive phase (cell-mediated resorption)
3. Postcalcific stage (tendon reconstitution of the matrix)

In the formative phase, the discomfort is usually minimal followed by moderate to severe pain with decreased range of motion in the subsequent resorptive phase. Finally, granulation replaces the calcification, and the tendon undergoes fibrosis and eventual repair.[2]

Calcific tendinosis must be distinguished from dystrophic calcification. The former is cell-mediated, occurs in healthy tissue, and is self-limiting; the latter occurs in degenerative tissue, and does not spontaneously heal.[2]

Since many patients with shoulder pain may have incidental calcific tendinosis, this confounding finding may easily lead the clinician to treat the calcification, resulting in poor outcome. Hence knowledge of the natural history of calcific tendinosis and the differential diagnosis of shoulder pain is paramount for ascertaining the correct diagnosis and its subsequent treatment.

During the precalcific stage, tenocytes becomes chondrocytes. In the early calcific stage (formative phase), the calcium deposit, appearing like chalk, develops around the chondrocytes. After a variable period of inactivity (resting phase), the disease progresses to the resorptive phase wherein vascular channels develop at the periphery of the calcification. Migration of macrophages and multinucleated giant cells soon removes the calcium deposit by phagocytosis. Finally, in the postcalcific stage, granulation tissue with fibroblasts and vascular channels grows into the location previously occupied by the calcification.

Diagnosis and Clinical Presentation

During the resorptive phase, patients present with limited range-of-motion and pain in the shoulder. Intratendinous calcification is evident on radiographs and sonograms. Contrary to the popular belief that pain starts with the onset of the disease, most patients are relatively asymptomatic, as the intratendinous tissue tension is minimal. However, during the resorptive phase, the intratendinous tissue tension increases due to cell exudate and vascular channel growth that elicit pain. Tendon hypertrophy impinges on the subacromial space, restricting shoulder movement.[2]

During the acute phase, severe pain restricts mobilization of the glenohumeral joint, and patients hold their arms next to their bodies in internal rotation. However, during the subacute and chronic phases, patients may complain of variable pain and tenderness that radiates to the humeral head.

Radiographic and Sonographic Analysis

The positions of the shoulder and of the transducer for insonation of the supraspinatus tendon are demonstrated in Fig. 1B. For an excellent review of musculoskeletal sonography that describes technique and illustrates cross-sectional images, please refer to the education section of www.essr.org. Plain film radiography (Fig. 2) and sonography (Fig. 3) demonstrate intratendinous calcification. In the resportive (acute) phase, the sonographic image of calcium deposit may appear faint and cloudy. However, in the formative phase, the deposit appears well-defined, dense and homogenous. Calcification is difficult to identify on MRI, and tendon edema or enlargement may incorrectly suggest a rotator cuff tear. T1-weighted images shows the area of decreased signal intensity, whereas T2-weighted images demonstrate bands of increased intensity suggestive of edema.[2]

In a study of 94 patients with calcific tendinosis diagnosed on radiography and variable degrees of symptoms, Chiou et al[4] classified the plaque, based on its sonographic morphological appearance, into four groups:

1. Type 1 (Fig. 3): arc-shaped has an intense echogenic arc with acoustic shadowing .
2. Type 2 (Fig. 4): fragmented or punctate, demonstrate at least two echogenic area penetrable to the US beam and devoid of shadowing.
3. Type 3 (Fig. 5): nodular, one echogenic nodule also without shadowing.
4. Type 4 (Fig. 6): cystic, an anechoic area within bright echogenic walls and having weak internal echoes.

Figure 1B

Shoulder and transducer positions for insonation of the supraspinatus tendon. With the arm externally rotated and elbow flexed and rotated posteriorly, the palm of the hand is placed on the superior part of the wing of the iliac crest. The transducer is placed over the tendon.

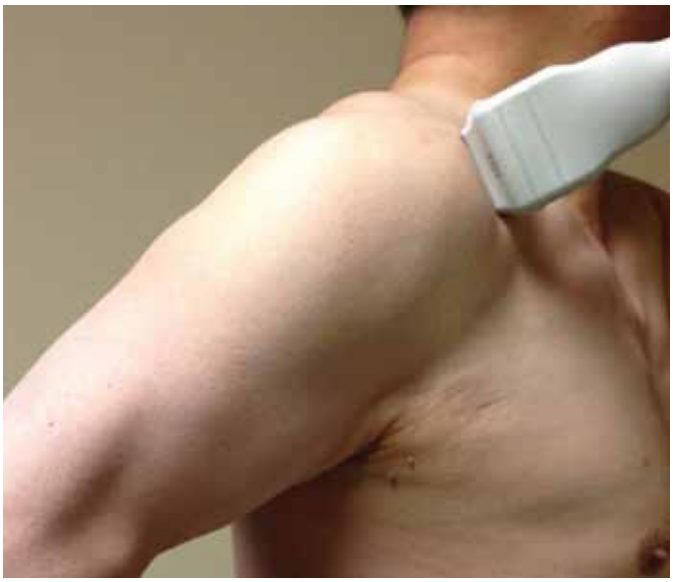
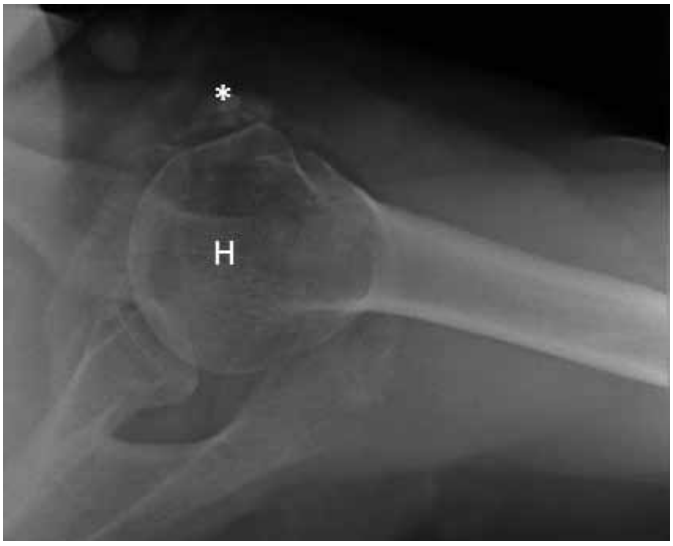


Figure 2A & 2B

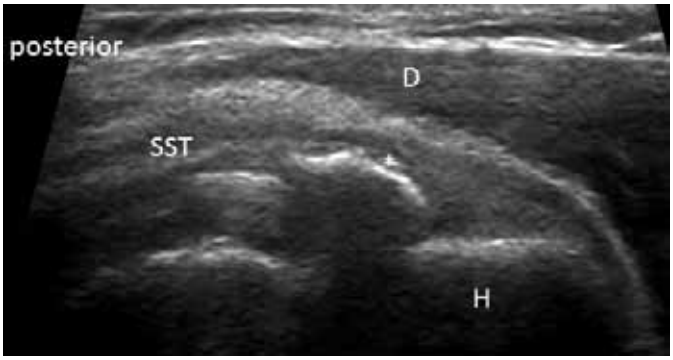
*2A: Shoulder radiograph (inferio-superior axial projection). Calcium deposit located within the supraspinatus tendon; H=humeral head; *=calcium deposit. 2B: Shoulder radiograph (anterio-posterior projection). Calcium deposit embedded within the supraspinatus tendon near its insertion on the greater tuberosity of the humeral head; H=humerus; *=calcium deposit.*



Using color Doppler sonography (Fig. 7) to identify the vascular channels, Chiou discovered an association between the degree of symptoms and the color Doppler signals. These channels correspond

Figure 3

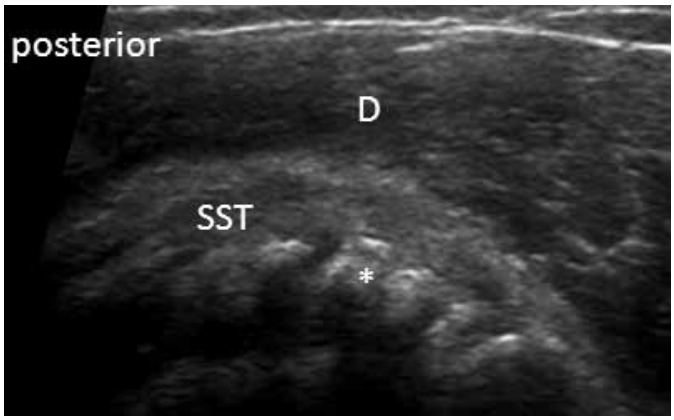
*Shoulder sonogram, supraspinatus tendon, longitudinal view. Arc-shape (Type 1) calcium deposit (*). Note the acoustic shadow beneath the superior surface of the calcium deposit; D=deltoid muscle; H=humeral head; SST=supraspinatus tendon; *=calcium deposit.*



to the increased vascularity described by Uhthoff et al.[5] Although the presence of color Doppler signals in the tendon may be a sign of increase vascularity in the resorptive phase, it may also be a twinkling artifact. Interested readers may wish to contact the author to learn more about this important distinction.

Figure 4

*Shoulder sonogram, supraspinatus tendon, longitudinal view. Fragmented or punctate (Type 2) calcium deposit; D=deltoid muscle; SST=supraspinatus tendon; *=calcium deposit.*



Chiou et al[4] noted that patients with calcium in the formative phase were mostly asymptomatic; however, those with fragmented or punctate, nodular and cystic types of calcium deposit demonstrated moderate to severe pain consistent with the report of Uhthoff et al.[5] Hence, they may serve as markers for the resorptive phase.

Le Goff et al[6] identified a few sonographic features associated with pain: large calcium deposits, presence of a power Doppler signal within the calcium deposit, and widening (effusion or hyperplasia) of

Figure 5
*Shoulder sonogram, longitudinal view. Nodular (Type 3) calcium deposit. Note the absence of acoustic shadowing and heterogeneous calcium deposit; D=deltoid muscle; SST=supraspinatus tendon; *=calcium deposit.*

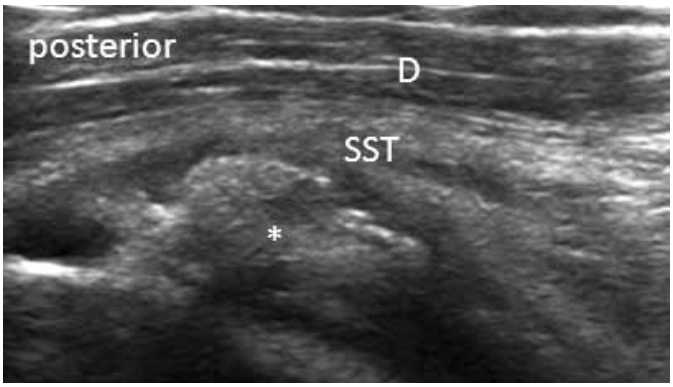


Figure 6
*Shoulder sonogram. Cystic shape (Type 4) calcium deposit; N=needle; *=cyst; C=calcium deposit; SST=supraspinatus tendon.*

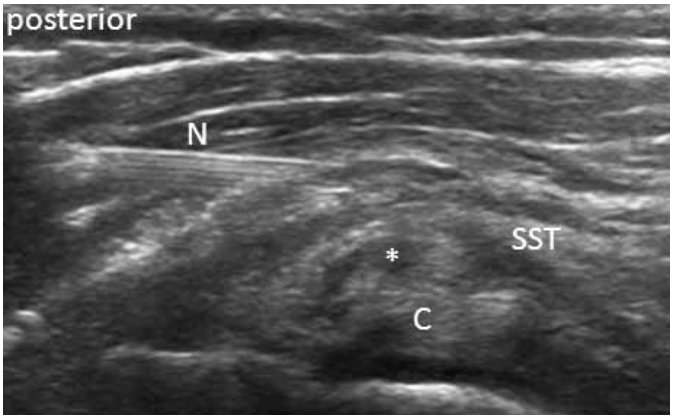
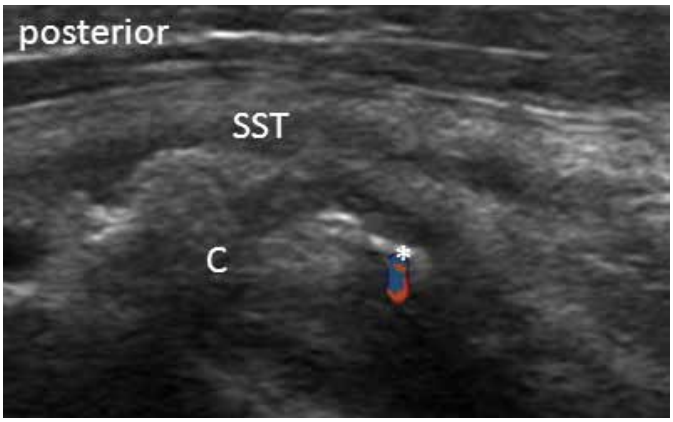


Figure 7
*Color Doppler signal within the calcium deposit and supraspinatus tendon suggesting an increase in vascularity; C=calcium deposit; SST=supraspinatus tendon; *=color Doppler signal.*



the subacrominal bursa. His study confirmed the findings of Chiou et al[4] in that patients with a fragmented appearance have a greater incidence of pain which occurs at the beginning of the resorptive stage.

Summary and Conclusions

Given the many causes of shoulder pain, diagnosing patients may be challenging. For patients with calcific tendinosis of the rotator cuff, the presence of calcium deposit alone may be an incidental finding, as many patients are asymptomatic. Sonographic tendon evaluation supplements the clinical examination and radiograph to determine the stage of presentation. For patients who have failed treatment, this evaluation can demonstrate residual calcium deposit within the resorptive phase that needs additional therapy as well as other causes of shoulder pain, including rotator cuff tear and tenosynovitis of the long head of the biceps brachii.

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AURORA – A Clinical Registry

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The concept of a registry is simple: a place where records are kept. Clinical Registries systematically and uniformly collect information from people who undergo a procedure, are diagnosed with a disease, or use a health care resource.[1] The American Heart Association defines a clinical registry as a prospective observational database of a clinical condition, procedure, therapy or population, in which there are no registry-mandated approaches to therapy and relatively few inclusion and exclusion criteria.[2] This is very different to the conduct of a controlled-clinical trial where rigid filters in the form of inclusion and exclusion criteria are often applied before sampling can occur. This process of exclusion generates internal validity but may limit application of results to a broader population. Despite these limitations, the randomized controlled trial (RCT) is the gold standard for determining if a therapy is efficacious. The focus of clinical registries is to capture real-world clinical practice (i.e., native hospital behavior) in large patient populations independent of the environment of a controlled clinical trial. Clinical registries are important for monitoring and benchmarking the quality of clinical care and are critical to clinical practice improvement. Clinical registries can serve multiple functions such as public health surveillance and performance assessment. They can be used as vehicles for quality improvement, to evaluate trends in clinical practice, and to monitor the safety and efficacy of a drug or device in Phase IV studies.[2,3] Determining if best practice and evidence-based guidelines are being adhered to, or if the results of RCTs apply to routine practice (effectiveness study) are further valid uses. There are many examples of successful clinical registries or databases from surgery,[4] intensive care,[5] and internal medicine.[6]

The Australian and New Zealand Registry (AURORA) is an example of a clinical registry established to determine the quality and safety of our contemporary practice of peripheral regional anesthesia. The project commenced in 2006 during a period in which regional anesthesia was evolving because of increased utilization of peripheral regional anesthesia and ultrasound-guided techniques. In previous studies peripheral nerve blocks (PNB) were performed using nerve stimulator technology, and therefore their results did not completely apply to this new clinical technique. Existing literature included studies using self-reporting methodologies that were considered inadequate to guide risk disclosure.[7] Monitoring the quality and safety of regional anesthesia is important for informed patient consent and clinical decision-making because regional anesthesia is often considered the alternative anesthetic technique by many patients and anesthetists. The public's perception of the risks associated with anesthesia is primarily related to the extremely rare risk of death due to general anesthesia. Overall, the risks of general anesthesia tend to be more easily understood and thus accepted by patients. An anesthetist may recommend regional anesthesia but patients' preconceptions may influence how receptive they are to an alternative technique. An additional burden is therefore placed on the clinician wishing to perform a regional anesthetic technique when a new set of potential benefits and complications are provided to patients.

AURORA Results to Date

Detailed methodology, outcome definitions, follow-up pathway and preliminary results of this project were published in 2009.[8] During 2006-2012 approximately 35,000 PNBs were captured by the registry. Ultrasound-technology was used in 81% of PNB during this period. PNB was an effective technique for enhancing early postoperative recovery with median pain scores in Post Anaesthesia Care Unit (PACU) being zero in all PNB categories except for trunk. Overall, 65% of patients required no additional analgesia, 23% intravenous opioid analgesia, and 8% oral analgesia, respectively. A total of 48% of patients were designated as being ready to depart the PACU within 30 minutes. Patient-rated outcomes indicate that patients were satisfied with the information provided to them and interactions with their anesthetist. However there is room for improvement because a significant proportion of patients reported moderate or severe pain following resolution of peripheral regional anesthesia.

During the study period of January 2007 through May 2012, there were 22 episodes of local anesthetic systemic toxicity (LAST; 13 minor; 8 major and one cardiac arrest) from 25,336 PNBs. Overall, the incidence of LAST was 0.87 per 1000 PNB. AURORA has demonstrated that ultrasound guidance may be protective for LAST. When PNB was performed with ultrasound technology, the incidence of LAST was reduced compared to techniques not utilizing ultrasound. This finding was consistent using multiple analytical techniques and may represent the first statistical evidence that ultrasound guidance improves safety during PNB.[9]

The incidences of late and long-term PNB-related nerve injury were 0.6 and 0.30 per 1000 PNB regardless of technology used. These incidences were calculated from a denominator comprising the total number of brachial plexus, femoral, and sciatic nerve blocks. Fortunately, in the majority of cases the long term outcomes for these patients were favorable. In this study, there was no significant difference in the incidence of late or long-term PNB-related nerve injury when PNB performed with ultrasound was compared with no ultrasound. Many observers would consider it plausible that PNB-

related nerve injury would be reduced when ultrasound-guided techniques were compared with techniques not employing ultrasound technology. The major value and utility of ultrasound-guided PNB includes dynamic visualization of needle placement and avoidance of nerve trauma. However, in addition to physical trauma there are other potential mechanisms including direct local anesthetic toxicity. A RCT comparing ultrasound-guidance with techniques not employing ultrasound would require a prohibitively large number of patients if permanent nerve injury were the primary outcome.[10,11] The AURORA results indicate that if a difference in nerve injury truly exists when ultrasound guidance is compared with non-ultrasound techniques, a very large sample, at least 30,000 per group would be required. It is now accepted that ultrasound guidance has not reduced the incidence of nerve injury caused by PNB.[10] Even if a larger registry had an appropriate sample size, it would be very unlikely these days to generate a large cohort of PNB performed without the use of ultrasound technology. Furthermore, the clinical presentation of nerve injury, its investigation and ascertainment of its etiology are complex. In many clinical presentations of perioperative nerve injury, it is impossible to be absolutely confident of the cause. Distinguishing anesthetic, patient and surgical causes of nerve injury are notoriously difficult.

Some consider that regional anesthesia introduces a non-essential procedural risk into the already complex perioperative environment. The AURORA results clearly demonstrate that the incidence of serious complications attributable to PNB is extremely low. When the infrequency of serious complications documented in this study is combined with the proven efficacy of ultrasound-guided PNB,[11] it is difficult not to promote this imaging modality for routine use for PNB. The evidence for the efficacy of ultrasound-guided PNB is robust for commonly performed upper and lower extremity PNB, but less definitive for truncal blocks to date.

The Future

The rarity of complications associated with regional anesthesia presents us with a challenge – the requirement for large sample sizes. A second challenge is to distinguish outcomes directly attributable to PNB from other causes. The development of a clinical registry with well-defined outcomes provided a framework for assessment of quality and safety of contemporary PNB. This registry and its collaborative infrastructure provides an opportunity to develop what this author has described as a "virtual department of anesthesiology."[12] If large enough, this entity would comprise centers with distinctly different practice patterns, providing the basis for a pseudo-randomized clinical trial. Ideally these practice patterns would include some of the continuous stream of newly described techniques. As creative and innovative as these new techniques often are, they should be tested for clinical effectiveness and safety before their widespread use is promulgated. Registries provide an important mechanism to do what other industries routinely do to stay competitive – continually monitor products for quality and take steps to improve when indicated. Some experts have called for a registry for every medical condition and invasive procedure.[13] AURORA is a registry for PNB.

In anesthesia we should be looking for efficient mechanisms to add value to our daily professional activities. Recording and reporting outcomes from routine care is one method of doing so. Competition from alternative anesthesia providers and modes of local anesthetic delivery[14] should prompt us to examine ways of adding value to what we do. A significant proportion of our population requires anesthesia services every year; therefore we should treat anesthesia as a public health issue. Collecting data from routine care and collaborating in multi-center registries as virtual departments of anesthesiology provide us with an opportunity to extend our repertoire and become public health physicians. Registries provide an infrastructure for measuring and reporting key outcomes crucial to any improvement. This registry provides both a template for such a research infrastructure and demonstrates a low incidence of serious morbidity that others can benchmark their practice against in virtual departments of anaesthesiology. Because this project now collects outcome data from Australia, New Zealand, Malaysia, and the United States, it has been renamed the International Registry of Regional Anaesthesia. The collaborators and hospitals that have contributed to this project are located at www.regionalanaesthesia.wordpress.com/collaborators.

References

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testing modalities should be used in the evaluation of radicular-type pain. A joint clinical practice guideline has been published by the American College of Physicians and the American Pain Society that covers the diagnosis and treatment of low back pain,[2] and joint practice guidelines for chronic pain management have been published by the ASA and ASRA.[3] Another key pre-procedural step is to document neurologic defects and any signs or symptoms of cauda equina syndrome, including bladder dysfunction, saddle anesthesia, and limb weakness, prior to performing an ESI. Clinicians should obtain a thorough medication history, especially antiplatelet and anticoagulant medications, and follow the updated ASRA evidence-based guidelines accordingly.[4] These guidelines include discontinuation of the following medications before any neuraxial procedure: clopidogrel (7 days prior), warfarin (5 days prior), intravenous unfractionated heparin (4 hours prior), and low molecular weight heparin (24 hours prior).4 It is also important to look for bruising and any signs of infection as other disease processes may interfere with proper care of the patient.[5]

Diagnostic imaging is essential to review prior to performing an ESI. MRI and CT scans have similar sensitivity and specificity in making a diagnosis of a herniated disc, but an MRI is preferred due to the lack of radiation exposure in comparison to CT scan, as well as more detailed visualization of soft tissue structures such as ligaments and cartilage. All findings from the history, physical examination, and diagnostic imaging should be well documented, allowing for a detailed assessment and treatment plan. All patients' treatment plans should include a comprehensive, multimodal, interdisciplinary approach as this has been shown to be the most effective for subacute and chronic low back pain management.[2,3,6,7] Finally, the risks, benefits, and alternatives should be discussed with the patient prior to performing ESI. It should be noted at this time that ESI treatment alone offers no significant improvement beyond three months of pain relief or functional benefit.[5]

The next step, of course, is to perform the procedure itself. Most importantly, the clinician must ensure basic hygiene and sterility according to the ASRA practice advisories for preventing infectious complications for regional anesthesia and pain procedures.[8-10] ASA standard monitoring should be employed for all procedures. A timeout should always be performed prior to any invasive procedure to confirm correct patient, procedure, and site, and medications should be reviewed along with their expiration dates. It is advisable to limit steroid dosing due to potential side effects.[11] Further, results of a randomized clinical trial has shown no difference in outcomes between low-dose methyprednisolone (40 mg) and high-dose methylprednisolone (80 mg); therefore, ESI should be limited to less than 80 mg methylprednisolone or equivalent.[12]

Fluoroscopic-guided ESI with the use of contrast is recommended to ensure safe needle placement into the epidural space.[1] Different techniques can be utilized to maximize the efficacy of treatment. ESI, using a transforaminal or parasagittal technique, can target the specific side that is causing the most pain. However, when using the transforaminal technique, it is recommended to limit injections to the lumbar region of the spine when possible,[13,14] although thoracic and cervical injections may be performed when clinical indications outweigh the risks involved with this procedure. Very serious complications, including paraplegia and death, have been reported with transforaminal ESI.[15] Another technique available to the pain management physician is the caudal ESI, which may be indicated for the treatment of lumbar radiculitis and pain related to disc herniation.[16] Repeat ESI may be considered if these partial goals (e.g., improved function and/or decrease usage of analgesic medications) are achieved, but clinicians are advised to wait two weeks prior to considering further injections.[17]

After the procedure is performed, vital sign and pain score assessments should continue until the patient is eligible for discharge. Discharge instructions should be explained to the patient, and any questions the patient may have should be answered. It is advisable that patients do not drive for 24 hours following ESI. Patients should also be made aware of potential post-procedural complications such as headache, fever, chills, nausea, vomiting, weakness, numbness, paralysis, and incontinence.[17] Patients should be instructed to notify the physician immediately if they suffer from any of these complications. It is important to schedule follow-up appointments within one month to assess the efficacy of the ESI treatment.

In summary, ESI offers potential therapeutic benefits to patients but is not an entirely benign procedure; therefore each ESI must be performed safely and for appropriate indications. The ESI process should always begin with a detailed and targeted history and physical exam. Documentation should indicate medical necessity, and guidance regarding what "medical necessity" entails can be found at the Center for Medicare & Medicaid Services' website: http://www.cms.gov/.18 Imaging should always be reviewed and taken into account prior to performing ESI. Proper procedural steps and sterile technique should be employed. Finally, adequate assessment should be performed after the procedure and timely follow-up scheduled. These steps cannot prevent all complications, but following them is something we can do to ensure the highest quality of care for our patients.

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