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CME Needs Assessment

2002 Annual Fall Pain Meeting, Pointe Hilton at Squaw Peak

Carl Koller Memorial Research Grant

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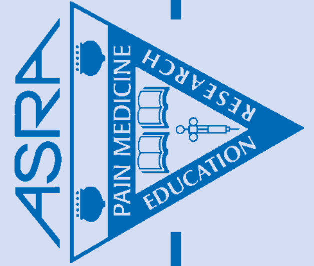

MAY 2002

ASRA News

A PUBLICATION OF THE AMERICAN SOCIETY OF REGIONAL ANESTHESIA AND PAIN MEDICINE

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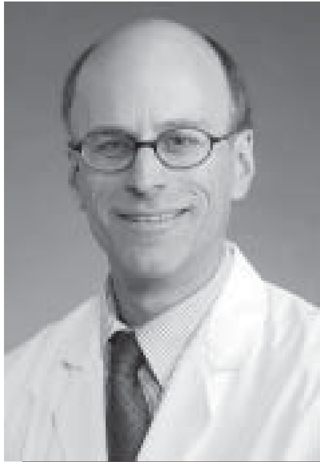

James C. Eisenach, MD
President, ASRA

President's Message

Big Change in Annual Meeting (Actually, MeetingS)

The exciting news is that regional anesthesia is expanding in many areas – besides its birthplace in the operating room and labor suite, application of regional anesthesia to acute and chronic pain management has blossomed in the last decade. Particularly in the area of chronic pain, this has attracted a very different practitioner than the traditional regional anesthetist in the operating room. In fact, many individuals devote full or nearly full time to chronic pain treatment, and have little to do with anesthesia for surgery or analgesia for obstetrics or postoperative patients.

Given this expansion and diversity in applications of regional anesthesia, I felt, and the ASRAPM Board of Directors agreed, that it would be best to acknowledge these differences in the structure of our annual meeting. As a result, starting this year there will be TWO annual meetings. The spring meeting, just held in Chicago, will be devoted to regional anesthesia as it is applied during surgery for anesthesia and after surgery for analgesia, as well as for obstetrics. A new meeting, to be held for the first time in Phoenix, November 7-10, 2002, will focus on application of regional anesthesia to the treatment of chronic pain. Reflecting this change, the two honorary lectures given at our meetings, the Labatt and Bonica lectures, will be separated – the Labatt lecture at the spring meeting related to surgical / postoperative / obstetric regional anesthesia and the Bonica lecture at the fall meeting related to chronic pain.

I recognize that this will be a disappointment to those with interests in both areas and who wish to attend sessions on both areas in one meeting. Others will question, "Do we really need another pain meeting?" There are several compelling reasons to make this change. For one, there was a sense of frustration with one combined meeting, which could not adequately cover all the topics appropriate to both general areas of regional anesthesia. For another, anesthesiologists often voice the opinion that we need a pain meeting and organization to call our own, and where our unique abilities and methods are discussed in a positive light. Several individuals on the current and past Board of Directors of ASRAPM believe that our society should represent the home for anesthesiologists using regional techniques in the practice of pain medicine. That clearly drove the name change of the society from ASRA to ASRAP(ain) M(edicine).

Yet another advantage of splitting into two annual meetings is the opportunity to enlarge the amount of original work presented at the meeting in abstract form. The traditional spring meeting has been dominated by intraoperative and postoperative regional anesthesia abstracts, with relatively few addressing chronic pain. We feel an independent meeting will foster more abstract submissions. In addition, the fall meeting will occur immediately after the Society for Neuroscience annual meeting, and we will actively encourage basic scientists studying chronic pain mechanisms to present their work at both venues. This also allows us to bring in international experts at reasonable cost, since they are already in the US for the Neuroscience meeting. Please note the Call for Abstracts in this Newsletter and at the ASRA website (www.asra.com) for the fall meeting, and encourage your colleagues to submit original work.

Will this be just another pain meeting? Absolutely not! Meeting chairs Jim Rathmell and Dan Carr have assembled national and international leaders in chronic pain management, both in basic science and clinical care. The meeting will begin with a day of Translational Sessions, in which a basic scientist is paired with a clinician to discuss how new understanding and new pharmacologic developments are being applied to the care of patients. Although the focus will be on regional anesthesia techniques, a broad range of methods used in the treatment of chronic pain will be covered. As much as possible, the strength of clinical evidence to support 'cutting-edge' as well as older therapies will be discussed. In addition, Richard Rauck, the current President of the Association of Pain Fellowship Directors, will give a keynote lecture regarding changes in education of fellows in pain medicine.

The first Bonica lecturer for the new fall meeting will be Maria Fitzgerald, a former student of Patrick Wall and leading investigator in developmental aspects of pain. She will discuss how nociceptive systems develop and how injury or inflammation in early life alters sensory perception and perhaps sets the stage for chronic pain disorders later in life.So, this is a year of big change, as we respond to the successful expansion of regional anesthesia. I hope to welcome several hundred attendees to our first fall chronic pain meeting in Phoenix, just as I have my fellow obstetric and postoperative analgesiologists in Chicago!

James C. Eisenach, MD
President

Research Update: *In Vitro* Assessment of Local Anesthetic Neurotoxicity

In vitro models uniformly indicate that clinically available concentrations of local anesthetics (LAs), particularly lidocaine, can be neurotoxic, consistent with the rare occurrence of persistent lumbosacral neuropathy following spinal anesthesia. The translation of *in vitro* findings to clinical practice requires caution and further research: "…it is important to consider the [cultured neuronal cell] line as a starting point for investigations and that findings made with it ultimately must be verified in normal tissues."(Greene et al., 1991) This Research Report will provide a summary of current *in vitro* evidence of LA neurotoxicity, and a brief overview of possible mechanisms being investigated using *in vitro* models.

Strengths and limitations of *in vitro* models for investigation of LA neurotoxicity. Isolated neurons *in vitro* allow exclusion of all systemic, non-neuronal factors (e.g., vascular, immune system), so that observed toxicities can be considered an indication of intrinsic, direct neurotoxicity. *In vitro* systems allow exposure of neurons to known concentrations of LA, so that dose is concentration, and other factors such as pH and temperature can easily be held constant. This offers a better defined peri-neuronal LA concentration than whole animal systems, where subarachnoid administration of LA involves variable dilution with CSF and ongoing diffusion out of the CSF, and dose and concentration are not equivalent, but also limits the translation of *in vitro* results to the clinical situation.

Major questions to be asked in interpreting *in vitro* studies are:

1. How does the dose tested correspond to CSF concentrations in clinical spinal anesthesia?
2. How closely does the *in vitro* system correspond to the intact spinal cord?
3. How does the exposure time tested correlate with clinical exposures?
4. Does the assay of neurotoxicity utilized indicate irreversible injury and eventual neuronal death, or reversible injury with full recovery possible?

The former has the strongest implication for clinical practice, as the end-point of death or irreversible injury is straightforward and clearly to be avoided. The latter may be more important for determining the mechanism by which LAs are neurotoxic, and devising eventual treatments or new drugs, but may be difficult to translate into implications for clinical practice.

Questions 2-4 are best considered in the context of specific experiments (below). General guidelines for question 1 can be estimated. Well-mixed administration of 70 mg lidocaine (0.5-5%) yielded CSF concentrations of ~0.2-0.4% for the first 10 min, and ~0.1-0.2% 10-30 min after administration.(Van Zundert et al., 1996) CSF concentrations in cases of neural injury after spinal anesthesia are not known, but often involved 5% lidocaine administered with poor mixing and maldistribution in the CSF or excessive doses, presumably yielding higher peri-neuronal concentrations. Hence, the range most relevant to irreversible neural injury is probably ³ 1% lidocaine.

Another potential manifestation of LA neurotoxicity may be the clinically defined syndrome of Transient Neurologic Symptoms (TNS), manifest by buttock or leg pain or dysesthesia after spinal anesthesia, and far more common after lidocaine than bupivacaine. If it does represent lidocaine neurotoxicity, it is reversible, and not concentration dependent above a clinically administered concentration of 0.5%.(Pollock et al., 1999) Hence, *in vitro* study of lidocaine concentrations of ~0.1-0.5%, using assays sensitive to reversible injury, is probably most relevant to TNS. *In vitro* models offer a possible advantage in studying early, reversible LA neurotoxicity, because they bypass a limitation of animal and clinical studies: the neurologic exam is usually an exquisitely sensitive indicator of even minor injury; but cannot easily be assessed in the early stages of LA administration because the desired pharmacologic effect of LAs is to block nerve conduction.

Differences in neurotoxicity among LA agents. The majority of data from *in vitro* studies involves lidocaine, presumably because lidocaine has been the LA most implicated in clinical manifestations of LA neurotoxicity. Unfortunately, there has not been a systematic comparison of clinically available LAs in all the *in vitro* models reported, and generally the comparative toxicity in one model cannot be confidently extrapolated to another model without doing those experiments. Comparisons with other LAs should be interpreted based on clinically equipotent concentrations (e.g., 0.25% bupivacaine compared with 1.0% lidocaine). Previous clinical practice tended to use non-equipotent concentrations of these agents in spinal anesthesia (0.75% bupivacaine, 5% lidocaine). This by itself would create an apparent increased toxicity of lidocaine in clinical cases of maldistribution, where concentration approximates peri-neuronal dose. *In vitro* studies at neutral pH in

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ASRA News
is a publication of the American Society of Regional Anesthesia & Pain Medicine.

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fact cannot study concentrations of bupivacaine much greater than 0.6%, because of its insolubility at pH 7.4, while >5% lidocaine is easily soluble at pH 7.4. (Clinical preparations of 0.75% bupivacaine are acidic, pH ~4, to allow solution.) Hence, although there is good evidence from *in vitro* studies that equipotent bupivacaine is less toxic than lidocaine (below), some of the difference in apparent clinical toxicity may simply reflect the physicochemical solubility limits of the different LAs and resultant concentrations in which they are clinically supplied.

***In vitro* evidences of LA neurotoxicity.**

Irreversible blockade of nerve conduction is an indicator of toxicity specific to neurons, although it uses acutely isolated nerve preparations that are not necessarily viable long-term. Desheathed bullfrog sciatic nerves were exposed to LA for 15 min, then washed extensively. Lidocaine 5% or tetracaine 0.5% caused irreversible total conduction blockade. This was true for lidocaine 5% even with only a 5 min exposure. Lidocaine 1.5% or bupivacaine 0.75% caused 25-50% residual block, while 0.06% tetracaine had minimal effect, similar to 7.5% glucose alone.(Lambert et al., 1994) In a subsequent report using a similar model to investigate concentration dependence, lidocaine caused a detectable irreversible block after washout beginning at 40 mM (~1%), and increasing with increasing concentrations to complete ablation of the compound action potential at 80 mM (~2%).(Bainton and Strichartz, 1994) In a similar frog nerve model, irreversible conduction block was tested with 12 hr exposure to LA concentrations 80 times their EC_{50} for reversibly blocking compound action potentials (so that equipotent concentrations as defined by the model were compared), followed by 24 hr wash.(Hampl et al., 1999) Lidocaine (~2%) caused the greatest degree of irreversible conduction block (95%) > bupivacaine (82%) ~ procaine (78%) >> tetracaine (13%). In single crayfish giant axons, lidocaine 40 mM (~1%) for 30 min, or 80 mM for 15 min, caused irreversible loss of action potential, despite return to a normal membrane potential; 40 mM for 15 min had no effect after washing.(Kanai et al., 1998)

Neuronal death is an unequivocal indicator of neurotoxicity. Acute, high dose toxins generally cause necrosis, manifest by a loss of plasma membrane integrity. Lower toxin doses may cause either necrosis or apoptosis, induction of programmed cell death with internal destruction of the neuron while the plasma membrane stays intact until an advanced stage.

Neuronal death—acute exposure. In the ND7 cell line (derived from rat DRG) with a 60 min exposure protocol and determining cell death by loss of a cytoplasmic fluorophore, 5% lidocaine caused widespread necrosis, with 50% cell death at 40 min. Lidocaine 2.5% caused observable cell death, but not significantly different from control. Bupivacaine 0.625%, lidocaine 1%, and lower concentrations did not cause any neuronal death within 60 min.(Johnson and Uhl, 1996; Johnson et al., 2002b) In primary cultures of adult rat DRG neurons assayed by trypan blue staining, 15 min exposure to 30 mM (~0.8%) or 100 mM lidocaine followed by 1 hr recovery caused cell death greater than control, 20-30%.(Gold et al., 1998) Quantitation of this data was complicated by lifting of neurons from the plating substrate used (itself an indication of injury) and consequent loss to analysis. Electrophysiologic study of individual neurons in the same report gave an EC_{50} of 14 mM (0.4%) for depolarization, with all neurons completely depolarized at 100 mM, consistent with loss of plasma membrane integrity and cell death.

In the crayfish single axon model, lidocaine 80 mM (~2%) for 30 min caused complete irreversible loss of membrane potential as well as action potential, consistent with cell death.(Kanai et al., 1998) In isolated rat sciatic nerve exposed to lidocaine 80 mM for 15-120 min, exposures of 60 and 120 min caused LDH release, consistent with cell death. Exposures of 15 and 30 min did not cause LDH release.(Kanai et al., 2000)

Neuronal death—chronic exposure. In the mouse DRG culture model, cell death was assessed by loss of bright appearance under phase microscopy, which may be a less sensitive and specific method than methods using membrane impermeant dyes. Lidocaine 30 mM (0.8%) for 18 hr caused 100% neuronal death; 0.6 mM (0.02%) lidocaine for 18-42 hr caused 20-40% death, and 0.06 mM lidocaine caused no death.(Hiruma et al., 1999) In the ND7 model, toxicity has been assessed by flow cytometry of large numbers of cells stained with propidium iodide, which detects necrotic and late apoptotic cells. Neurotoxicity was both time- and concentration-dependent, with longer exposures causing toxicity at much lower concentrations than exposures of an hour or less. After 2 hr exposure, >75% neurons were dead with 0.5% lidocaine or 0.125% bupivacaine, although 0.25% lidocaine or 0.0625% bupivacaine were not toxic. After 4 hr, >50% neurons were dead with 0.25% lidocaine or 0.0625% bupivacaine. In contrast to 60 min exposures, there was no difference between equipotent lidocaine and bupivacaine for 2 or 4 hr exposures.(Johnson and Uhl, 2000) After 24 hr, >75% neurons were dead with 0.125% lidocaine.(Johnson, 2000) In the human neuroblastoma line SK-N-MC, LAs caused cell death after 24 hr exposure (assayed by trypan blue staining). IC_{50} for lidocaine was 0.7 mM (~0.02%). When assessed by equipotent concentrations, toxicity of lidocaine ~ procaine > tetracaine ~ dibucaine. (Kim et al., 1997) Parallel scoring of stained nuclei demonstrated that most neuronal death was apoptotic.

A sensitive assay for toxicity *in vitro* is to determine the effect of a putative toxin on cell division and growth. This approach has significant limitations for neurons, because in the adult CNS neurons are terminally differentiated cells which do not generally replicate. At the least, however, it indicates a perturbation of neuronal homeostasis with the potential for toxicity in mature neurons, and may be a more sensitive indicator of toxicity than overt neuronal death. Control experiments to establish parameters for an *in vitro* model using the rat pheochromocytoma neuronal line PC-12 to investigate lidocaine tachyphylaxis found that lidocaine 2 mM (0.05%) completely inhibited cell division in the absence of nerve growth factor, and completely inhibited neurite outgrowth in the presence of nerve growth factor (i.e., under neuronal differentiation conditions).(Wilder et al., 1993) Lidocaine 0.5 mM did not affect growth in that model. Other reports have used neurite growth as a toxicity assay of LAs. Hiruma et al. showed that 6 μM (0.0002%), 0.6 mM (0.02%), and 30 mM (0.8%) lidocaine for 2-42 hr inhibited neurite growth in primary cultures of mouse dorsal root ganglion (DRG) cells, with both time- and dose-dependent effects.(Hiruma et al., 1999), see also (Kanai et al., 2001) Saito et al. reported that both tetracaine and bupivacaine caused growth cone collapse and neurite destruction in three different embryonic chick neuronal culture models (DRG, retinal ganglion cell layer, and sympathetic ganglion chain).(Saito et al., 2001) Effects were both time (1 hr vs. 24 hr) and concentration dependent (0.01-1.0 mM). ED_{50} at 60 min was 0.1-2.3 mM, and at 24 hr was 0.03-0.8 mM. Tetracaine was significantly more toxic than bupivacaine (lower ED_{50}) in both the retinal and sympathetic ganglion models, although not in the DRG model.

More subtle, potentially reversible neurotoxicity can be indicated by morphologic alterations of neurons in culture. Rounding and blebbing are unequivocal, nonspecific indicators of neuronal injury, caused by alterations in the cytoskeleton, plasma membrane, and extracellular adhesion molecules, and are most easily seen in cell culture where neurons are not so tightly constrained in space by surrounding cells. Rounding is evident in a loss of asymmetry and collapse to a spherically symmetric neuron, with loss of neurite fine structure. It is a sensitive and early indicator of injury, and is reversible. A more severe insult is followed by blebbing, an out-pouching of the cell surface, caused by Ca^{2+}-activated proteolysis of cytoskeletal anchoring points for the plasma membrane. It can be reversible in its early stages, or can progress to cell death if prolonged. Rounding and blebbing have been observed in both necrotic and apoptotic cell death from multiple causes. While they are not specific for mechanism and severity of injury, they are highly sensitive for detecting neuronal injury.(Schwartz et al., 1993; Zahrebelski et al., 1995)

Both lidocaine and bupivacaine caused striking morphologic alterations in the ND7 neuronal line, with lidocaine having a greater effect than bupivacaine. Bupivacaine 0.125 - 0.625% caused a marked, concentration increase in blebbing after 60 min exposure, from 20% to 60% neurons blebbed.(Johnson and Uhl, 1998) Lidocaine ³1% caused >90% neurons blebbed at 60 min, while 0.5% lidocaine caused 40% blebbing.(Dadarkar et al., 2001; Johnson et al., 2001; Johnson et al., 2002a) (Photomicrographs of blebbing after LA are published with the cited abstracts.) Blebbing increased during the second hour for bupivacaine treated neurons and 0.5% lidocaine. Blebbing by lidocaine was significantly greater than that by bupivacaine.

The more sensitive indicator, rounding, was also assayed in ND7 neurons exposed to 0.25% lidocaine for 45 min, then recovered in buffer for 120 min. The majority (>70%) of neurons were rounded after 45 min 0.25% lidocaine, although rounding occurred within 5 min for most neurons. Most neurons recovered to a normal neuronal morphology, requiring >15 min. At the end of the experiment, essentially all neurons were still viable by assay of plasma membrane integrity (propidium iodide), but 20% were committed to apoptotic neuronal death.(Johnson and Uhl, 2001) (Photomicrographs from this study are available at http://www.asa-abstracts.com/ for the cited abstract.)

Possible mechanisms of neurotoxicity. The mechanism responsible for LA neurotoxicity is not fully elucidated. Current *in vitro* mechanistic data can be noted only briefly here because of space limitations. Na^+ channel block is not a likely toxic mechanism. The ED_{50} for Na^+ channel block, ~100 μM, is orders of magnitude less than the ED_{50} for neurotoxicity in most *in vitro* systems. The Na^+ channel blocker tetrodoxin, structurally unrelated to clinical LAs, was not neurotoxic *in vivo*(Sakura et al., 1995) or *in vitro*.(Wilder et al., 1993; Gold et al., 1998; Johnson and Uhl, 2001)

In vitro models have shown LAs to cause derangements in three vital areas of neuronal homeostasis at clinically relevant concentrations: (1) inhibition of rapid axoplasmic transport of organelles and chemicals between cell body and synapse;(Fink et al., 1972; Byers et al., 1973; Lavoie et al., 1989; Kanai et al., 2001) (2) prolonged elevation of cytoplasmic calcium beyond the transient peaks seen during normal neuronal firing;(Johnson and Uhl, 1996; Kim et al., 1997; Gold et al., 1998; Johnson et al., 1998a; Johnson et al., 2002b) and (3) inhibition of mitochondrial function. (Haschke and Fink, 1975; Johnson et al., 1998b) Each of these areas interacts with the other: axoplasmic transport requires energy and Ca^{2+}-dependent proteins, Ca^{2+} homeostasis is energy-dependent, and the mitochondria not only provide energy but also store large amounts of Ca^{2+} which can be released into the cytoplasm. Hence, an initial insult to any one of these areas could be manifest as subsequent derangements in the other areas, and the initial site of LA injury in the neuron remains to be determined.

In conclusion, there is abundant evidence from *in vitro* models for LA neurotoxicity at concentrations relevant to CSF concentrations after spinal anesthesia. Comparison between *in vitro* studies is limited by differences in models and LAs tested, and extrapolation in clinical use requires caution and further research. However, the available *in vitro* studies do suggest that lidocaine is generally more neurotoxic than most other LAs, that higher concentrations of LA (³ 1% lidocaine) can cause acute necrotic neuronal death, and that lower concentrations of LAs can cause reversible injury which may progress to delayed apoptotic death. The mechanism of LA neurotoxicity is unlikely to be Na^+ channel block. There is clear evidence that LAs disrupt axoplasmic transport, intracellular Ca^{2+} homeostasis, and mitochondrial energetics.

Acknowledgment: Supported by NIH GM59271.

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Complete References are at www.asra.com

How Do I Do Suprascapular & Intrabursal Blocks

Nerve blocks, once within the sole domain of our specialty, but now provided by numerous practitioners, are a common therapy utilized in the contemporary management of patients with acute and chronic pain. They are most effectively used within the framework of a comprehensive program of treatment modalities because the nerve block may relieve the pain but will not affect the psychosocial consequences of pain. Local anesthetics can provide prompt, but often only, temporary relief of pain. A number of adjunctive medications are now used to 'extend' the duration of benefit.

The advantages of such interventional therapy include not only (the expected) reduction in pain, but also less reliance on analgesic medications, increased cooperation (by the motivated patient) with the physical rehabilitation program, improved functional ability, enhanced quality of life, and less need to consider surgical approaches. Careful selection of patients for nerve block therapy will foster favorable outcome data. In general, the benefits of nerve block therapy are greater than the possible side effects which generically include neuritis, creation of a neuropathic pain focus, drug reactions and toxicity, lack of effect contributing to disappointment and frustation in the patient, or (perhaps a more problematic issue in patients with embedded chronic pain), perceived complications.

Shoulder pain is a common and disabling complaint of patients, and we have valuable therapy to apply in their care. While acute shoulder pain has not often been observed by anesthesiologists, the advent of acute pain services and the attendant follow-up of patients in the days immediately after surgery has revealed a number of patients with new onset pain after procedures performed with the patient in an awkward position. The acute, dull, aching pain results in decreased range of motion (ROM) and discomfort.

More commonly, patients are referred to pain centers with more chronic complaints of shoulder pain, stiffness and decreased ROM, and some degree of frozen shoulder/adhesive capsulitis. Acute bursitis (subacromial, subdeltoid)results in pain complaints around the shoulder or the cap of the upper lateral arm with the patient saying they can't find a comfortable position in which to hold the shoulder. Pain is increased with shoulder movement. If chronic, there may be calcium deposits found on x-ray in the bursae. If bicipital tendinitis is present, pain is localized to the antero-lateral shoulder in the region of the bicipital groove, increased with flexion of the arm, and may radiate down the arm. Additional diagnoses to consider include acromioclavicular joint arthritis and rotator cuff tendinopathy.

SUPRASCAPULAR BLOCK

Indications: In ACUTE pain circumstances of shoulder pain after positioning for a surgical procedure (i.e., post-thoracotomy, post-nephrectomy), for relief of postoperative pain (i.e., post-arthroscopy), and following shoulder dislocation.

In CHRONIC pain patients with adhesive capsulitis/frozen shoulder or chronic shoulder pain from arthritis or other degenerative conditions.

Contraindications: Infection at the needle insertion site, patient refusal, and perhaps the patient who is fully anticoagulated (since the needle will pass through muscle tissue)

Anatomic considerations: The suprascapular nerve is a derivative of C 5-6 roots of the brachial plexus. It comes off the plexus above the clavicle, courses through the posterior triangle of the neck under the border of the trapezius muscle. It runs through the supra-scapular notch and under the transverse scapular ligament.

It provides motor branches to the supraspinatus muscle (a primary abductor of the shoulder) while running laterally along the supraspinatus fossa to wrap around the lateral margin of the scapular spine and descend in the infraspinatus fossa. It provides motor branches to the infraspinatus muscle (a primary external rotator of the shoulder), as well as sensory branches to the posterior and superior portions of the shoulder joint capsule, the acromioclavicular joint, and the tendinous portion of the rotator cuff

Technique: The patient assumes a seated position with their arms in their lap. The medial and acromial borders of the scapular spine are palpated and the midpoint along a line connecting them is marked. One cm above this midpoint mark will be the point of needle insertion. The skin area is prepped and draped in routine fashion. 1% plain xylocaine is used to create a skin wheal and infiltrate along the proposed path of needle placement. An 8cm, 22 gauge needle has a marker threaded up its shaft. The needle with the stylet in place is directed through the skin wheal, in a plane that is perpendicular to the skin. It is advanced through the supraspinatus muscle to the back of the scapula. This defines the depth of the bone. The marker on the needle shaft is moved up the shaft 1.5cm. The needle is withdrawn to the subcutaneous level and re-directed 15 degrees laterally and 15 degrees cephalad, and inserted to the depth of the marker. The needle tip is now in the vicinity of the suprascapular notch.

After aspiration is negative for blood (there are vessels running with the nerves), 7-10 mL of 0.25% bupivacaine are injected. There is very little/no cutaneous anesthesia consequences of this block, so decreased pain (from analgesia of the shoulder joint capsule) and improved ROM (from decreased muscle spasm of the supra- and infra-spinatus muscles) are signs of success. (figure 1)

Complications: Infection, bleeding, and pain at the injection site (you ARE going through skin, muscle, and bone, at least twice, depending upon how satisfied you are with your needle placement) are possible. If you go over the top of the scapula or through the notch, you can inject drug into the brachial plexus or the vessels associated with it, or create a pneumothorax.

INTRABURSAL BLOCK

Indications: ACUTE bursitis presents as new onset anterior-lateral shoulder pain. The subacromial and subdeltoid bursae are contiguous structures, so it is not necessary to define which bursa is causing more trouble. The pain can be moderate to severe and markedly limits ROM. This arises after many kinds of trauma, including abnormal positioning and repetitive motion activities.

CHRONIC complaints of shoulder pain will likely include chronic bursitis as well as some degree of bicipital tendinitis. An intrabursal block is combined with the suprascapular block when the patient is going to their first physical therapy session to maximize the benefit of the therapy.

Contraindications: Infection at the skin site of needle insertion, patient refusal, and previous injection of steroids within the biceps tendon in the last six to twelve months (yes, this IS an arbitrary statement)

Anatomic considerations: The bicipital groove serves as a notable landmark through which access to the subacromial (and subdeltoid) bursae can be gained. The bursae serve as cushions between the muscles inserting or orginating from the nearby bones. When the bursae distend, as with inflammation, pain with motion becomes manifested.

Technique: The patient sits comfortably with their arms flexed and across their lap. To accentuate identifying the icipital groove, the ipsilateral hand is supinated to externally rotate the shoulder. The skin in the area is prepped and draped in routine fashion. A skin wheal is created with 1% plain xylocaine at the level of the bicipital groove. The acromion and the joint space below it are defined. An 8cm, 22 gauge needle (or a 25 ga needle in smaller patients, but beware of the great possibility of deflection of the needle given its small gauge and the density of the tissues being approached) is advanced through the wheal (at approximately a 45 degree angle to the skin) to contact the bottom of the acromion. The needle is withdrawn to a near-subcutaneous level, and the angle of approach lessened so the needle will slide into the bursa below the acromion (usually about 1.5cm farther than when bone was contacted) (figure 2). The stylet is removed and placed over the skin (parallel to the needle) to demonstrate the location of the tip (hopefully under the acromion).

A syringe containing 2mL 0.25 % bupivacaine and 40mg (1mL)-80mg (2mL) methylprednisolone is attached and 75% of the solution volume injected. The needle is withdrawn at least 50% of its depth and the remainder of the solution injected to treat the bicipital tendinitis.

Complications: Infection, bleeding, and pain at the injection site are possible. The patient should be cautioned that the local anesthetic effect might dissipate before the steroid action is present so recurrent pain may be an issue in the first 24 hours after injection. Repeated injections of steroids into the biceps tendon can lead to rupture, thus the cautionary comments about knowing how many previous injections the patient has received.

NEAT THINGS TO (ALSO) KNOW ABOUT THESE BLOCKS

Suprascapular:

1) if the patient places the ipsilateral hand on the opposite shoulder, the scapula is moved away from the chest wall and the likelihood of pneumothorax decreased
2) with patients in the sitting position, and with the potential pain from needle passage through skin and muscle to bone, vagal reactions are not uncommon. So, have your resuscitation equipment/drugs handy.
3) deposteroids are injected if there is clinical evidence that the suprascapular nerve is entrapped by the transverse scapular ligament. This is manifested by atrophy of the supraspinatus muscle, noticed upon inspection of the back and comparing one side to the other.
4) this block can be repeated every day for a week, before the patient goes to PT. However, there are reports of myo-necrosis with repeated injections with high concentration of local anesthetics
5) coordinate the patient receiving block therapy with the PT appointments. This way, you get a third party observer's input about the patient's progress AND you can more accurately assess how much of the patient's problem is reversible with PT (as opposed to their needing manipulation under anesthesia to restore ROM).
6) the patient can use walk-the-fingers-up-the-wall therapy at home to supplement the formal PT. The patient must stand perpendicular to the wall to gain true abduction of the arm.

Intrabursal:

1) expanding a painful bursa with the injectate will cause more discomfort. Warn the patient that they may have pain the night of the injection, as the depo-steroid may take 24-36 hours to create benefit.
2) the anterior portion of the deltoid muscle may cover the bicipital groove, so it may be hard to delineate bicipital tendinitis from muscle pain
3) biceps tendon rupture is a possibility if too many steroid injections are given through the tendon. Set a limit on how many you will do.

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