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World Congress of Regional Anesthesia and Pain Therapy

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JUNE 2003

ASRA News

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

ASRA NEWS

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



Terese T. Horlocker, MD
President

President's Message

"Regional anesthesia has come to stay. Its development and progress have been slow, principally because the anesthetist must have an accurate knowledge of anatomy and a high degree of technical skill in order that the anesthesia may be safe and satisfactory, and that the operation not delayed." These words by surgeon William J. Mayo opened the foreword to Gaston Labat's *Regional Anesthesia, Its Technic and Application*. Published in 1922, Labat's text popularized regional anesthesia in the United States by describing techniques already familiar to European surgeons and anesthesiologists. The ongoing relevance and success of the subspecialty was documented on August 2, 1923 with the founding of the American Society of Regional Anesthesia.

The art and science of regional anesthesia have progressed significantly over the last century, resulting in improved safety and increased success rates. The frequency of serious complications related to regional anesthesia continues to decrease and is similar, if not superior, to that of general anesthesia. Improved methods of neural localization and imaging such as fluoroscopy, high-resolution ultrasound and stimulating catheters have facilitated accurate needle/catheter

placement. Although technical skill is still required, most clinicians agree identification of the psoas compartment block is more readily achieved with a nerve stimulator than a loss of resistance. Most importantly, prospective randomized clinical investigations have demonstrated improved outcomes for patients undergoing major surgical procedures when regional anesthesia and analgesia is utilized. Thus, issues regarding safety, success rate, and efficacy have been addressed.

However, it is noteworthy that several of the early concerns have changed little. For example, an understanding of anatomic relationships, neural innervation, and physiology remain paramount in the application of regional anesthetic and analgesic techniques. Many clinicians do not have ready access to an anatomy laboratory and classic anatomical atlases were constructed by anatomists, not regional anesthesiologists, resulting in illustrations that depict neural anatomy with the "wrong" limb orientation and/or cross-sectional view. Ongoing pressures to increase operating room efficiency result in confrontations regarding block time and time to hospital dismissal. These issues must be addressed, either with data that refute operating room inefficiency following regional techniques, or with a recognized benefit/outcome that overcomes shortsighted beliefs.

The American Society of Regional Anesthesia and Pain Medicine, initially ordained the Labat Society, is well suited to the challenge. The Society's mission is to associate physicians and scientists who are engaged in regional anesthesia for surgery, obstetrics and pain medicine; to encourage education and research in these areas for the benefits of physicians and the public; and to publish the highest quality scientific information on these subjects. How will we meet the needs of our clinician constituents and patients? In what ways can we establish an ongoing tradition for future residents and fellows?

The Society will continue to sponsor both a Regional Anesthesia (Spring) and Pain (Fall) Meeting. Both annual meetings will emphasize evidence-based analysis to support innovative as well as historical treatment modalities. Dr. Marc Huntoon, Program Chair of the 2003 Fall Program, has devised a program that is cutting edge and clinically relevant. Nationally and internationally recognized speakers in pain management will provide lectures both in basic science and clinical care. A **Comprehensive Treatment of Chronic Back Pain Workshop** is tentatively planned for the 2004 Fall Annual Meeting.

Dr. Joseph Neal, Chair 2004 Spring Program, has continued to evolve the regional anesthesia meeting. Following the precedent of previous Consensus Conferences on Neuraxial Anesthesia and Anticoagulation, and the Conference on Local Anesthetic Toxicity, the 2004 Spring Annual Meeting will include a **Consensus Conference on Infectious Risks of Regional Anesthesia**. The success of the Comprehensive Upper and Lower Extremity Workshops will be applied to an extended **Current Regional Anesthetic Concepts and Workshop**, scheduled for the final general session of the 2004 Spring Annual Meeting.

The Society website (asra.com) will undergo extensive revision. Inclusion of text descriptions and video demonstrations of regional/pain techniques, as well as improved references and links, will improve the utility and functionality of the site.

Pain and Regional Anesthesia Fellow involvement will be encouraged by scholarships awarded competitively to residents and fellows who submit an abstract to the Spring or Fall Annual Meetings (*see Resident Activities on page 3*). The Cracker Barrel session at the 2004 Spring Meeting will focus on the use of innovative techniques, including virtual anatomy, to teach and master regional anesthesia.

Finally, the durability of a specialty is based on its ability to foster creativity and

President's Message Continued

inventiveness. Although sponsorship of the previously described activities is helpful, it is not enough. ASRA must aggressively sponsor research to advance the technical, basic science and clinical aspects of the practice. In return, residents, clinicians, and basic scientists should feel compelled to submit original work for presentation at our annual meetings and publication in our journal, *Regional Anesthesia and Pain Medicine*.

The specialty continues to advance, perpetually balancing scientific achievements, medicolegal issues, and economic constraints. Labat, a surgeon himself, understood the surgeon's point of view regarding block failures and surgical delays. Yet he remained dedicated to the application of regional anesthetic and analgesic techniques. Labat concluded his book, "Regional anesthesia is an art. Remembering that even experts may fail, we should try often and again, observing scrupulously its principles, until we succeed."

Terese T. Horlocker, MD
President, ASRA

Journal Update: Editor-in-Chief's Report

In conjunction with ASRA's educational mission, *Regional Anesthesia and Pain Medicine* continues to publish articles of interest to our readership. Further, the journal actively encourages the submission of research related to our subspecialty. Besides original articles and reviews, the journal has recently published updated Guidelines on Anticoagulation and Neuraxial Anesthesia (May / June 2003 issue). Other articles of special interest have included selections from the 2001 Conference on Neurotoxicity of Local Anesthetics and the July 2002 review article on Brachial Plexus Anesthesia, which summarizes the scholarly research from the ASRA Intensive Upper Extremity workshop initiative. The companion review article stemming from the Lower Extremity Intensive workshop should be published this fall.

Satisfaction with the journal appears to be remarkably high. A readership survey conducted by the journal's publisher (Elsevier) found that subscribers to *Regional Anesthesia and Pain Medicine* exhibit the highest ratings for reading frequency and reading thoroughness as compared with the other three major anesthesiology journals. As a benefit of subscribing, readers are reminded that by visiting www.rapm.org they now have access to full text articles archived back to January 2000.

On behalf of the Editorial Board, I wish to express our highest gratitude to Steve Abram, MD and André Van Zundert, MD, who retired from the editor's board after many years of service to the journal. Their excellent reviews, editorial insight, and dedication to *Regional Anesthesia and Pain Medicine* will be missed.

Joseph M. Neal, MD
Editor-in-Chief

Op Ed - Opinion Editorial

Everybody has an opinion about a topic that relates to her/his professional activities. As the new editor of YOUR newsletter, I invite you to submit a short, concise Op Ed for consideration for publication in the newsletter.

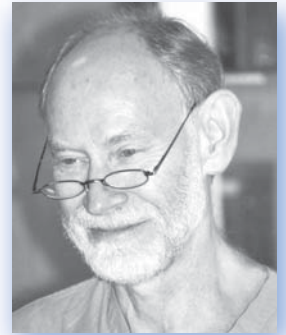
Highest priority will be given to Op Eds that offer constructive, provocative, thoughtful views about what might be done to improve the quality of the work-product our daily activities generate.

Have some thoughts about how to improve the Society's meetings, journal, service to its members; how to improve regional anesthesia utilization, techniques, safety? If yes, share the thoughts with your colleagues by writing an Op Ed.

Communication is something about which I have a clear opinion. Most of us could do a better job of communicating. Failure of parties (individuals, organizations) to communicate freely, openly and honestly is a root cause of considerable stress, anxiety, and conflict. Not long ago, I participated in a strategic planning activity that included a goal of improving communication. The task force charged with addressing this had to be reformed as the original task force never met. That made a statement to me about the communication problem!

My goal is for the newsletter to be an instrument that effectively communicates information that will improve the quality of all of our professional activities AND I WANT YOU TO HELP ME ACHIEVE THIS GOAL!

James E. Heavner, DVM, PhD
Editor



NEWS is a publication of the
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Bier Blocks: New Tricks for an Old Dog

Case presentation: A healthy, 76 kg, 42 year-old male was scheduled for right thumb ligament repair and percutaneous pinning of the proximal interphalangeal joint.

Surgery was estimated to take one hour. The patient requested Intravenous Regional Anesthesia (IVRA). Midazolam 2 mg and fentanyl 50 mcg were given as premedicants. IVRA was administered with 40 ml 0.5% lidocaine, ketorolac 10 mg, clonidine 100 mcg, and ketamine 10 mg. An upper arm single tourniquet was used. Tourniquet time for the surgery was 52 minutes at 300 mm Hg. The patient denied any surgical or tourniquet pain.

The patient was discharged home 11 minutes after surgery. The patient's pain score remained 0 (0-10 scale) for 16 hours after surgery. Acetaminophen 650 mg and codeine 60 mg (two Tylenol #3s) were taken for pain in the first 24 hours postoperatively.

This article will review safety considerations, and limitations of traditional IVRA. Next, advances in IVRA pharmaceuticals and technique will be discussed. The article will conclude with a pharmaco-economic discussion, and recommendations for future investigations.

Safety considerations: Although the technique is simple, safety considerations remain paramount. At least seven deaths, two cardiopulmonary arrests, and many seizures have been reported with IVRA¹. Unintentional leakage of the local anesthetic solution under the inflated tourniquet seems to have been causative in many of these cases. Radiocontrast studies indicate that anesthetic solution can leak under a correctly inflated tourniquet. Such leakage occurs almost completely via the venous system, and demonstrates that venous pressures during injection can exceed tissue pressures under the tourniquet.

Placement of a distal IV catheter, esmarch bandage limb exsanguinations, injection over at least 90 seconds, and double tourniquet inflation pressures of at least 300 mm Hg minimize the risk of anesthetic solution escaping the limb under an inflated tourniquet.

Traditional limitations of the technique:

1. **Onset time.** With the use of approximately 200 mg of 0.5% lidocaine or prilocaine, the time from local anesthetic injection until surgical anesthesia is present is typically 10-15 minutes^{2,3}.

2. **Tourniquet pain.** Nerve ischemia and compression are the main nociceptive stimuli with tourniquet pain. Unmyelinated C fibers appear to be the major neural pathway for transmission of tourniquet pain signals⁴. Significant tourniquet pain is usually present by 20-25 minutes after tourniquet inflation. Even with double tourniquets, significant pain is typically present by 40 minutes after initial tourniquet inflation.

3. **Post-operative analgesia.** Because of the rapid reperfusion of the limb after tourniquet deflation, IVRA has typically provided minimal analgesia after surgery. Post-operative analgesia has traditionally been a major advantage of brachial plexus analgesia compared to IVRA.

New pharmacologic adjuvants and techniques:

Ketamine. Ketamine profoundly enhances the efficacy of bupivacaine wound infiltration^{4,5}. Because tourniquet pain is transmitted via unmyelinated C-fibers, the ability of ketamine to block nociception by antagonism of NMDA receptors has been investigated in the context of IVRA. Ketamine 0.1 mg/kg added to IVRA dramatically reduces tourniquet pain, and decreases the need for intraoperative opioid supplementation. The degree of postoperative analgesia with this use of ketamine remains to be studied.

Clonidine. Like ketamine, clonidine synergistically interacts with lidocaine to inhibit C-fiber action potentials. Clonidine may also facilitate peripheral mobilization of endogenous opiates. The addition of clonidine (0.1-0.15 mcg/kg) to IVRA significantly reduces tourniquet pain compared to local anesthetic alone^{4,6,7}. Additionally, profound postoperative analgesia was evident for nearly six hours after tourniquet deflation. In comparison with ketamine 0.1 mg/kg, clonidine 1 mcg/kg was more effective than placebo and less effective than ketamine at controlling tourniquet pain. No studies have evaluated the combination of ketamine and clonidine as adjuvants to IVRA.

Ketorolac. Ketorolac 60 mg added to IVRA resulted in reduced tourniquet pain and 12-16 hours of postoperative analgesia⁸. These authors later performed a dose-ranging study of ketorolac in IVRA using an upper arm tourniquet, and concluded that the benefits of ketorolac incrementally increased up to 20 mg; no further benefits were evident with larger doses⁹. When comparing ketorolac in IVRA using a forearm versus upper arm tourniquet, these authors also demonstrated that 10 mg of ketorolac with a forearm tourniquet IVRA was more effective at providing postoperative analgesia compared to 20 mg of ketorolac with an upper arm tourniquet IVRA³.

A concern with the use of ketorolac is localized platelet inhibition, which could result in wound hematomas. No published study has examined this potential complication.

Neostigmine. In IVRA, Turan and colleagues² demonstrated that neostigmine 0.5 mg added to prilocaine IVRA decreased block onset time by 60%, increased motor block during surgery, decreased the need for intraoperative opioids, and increased the quality of anesthesia. Neostigmine also prolonged the time until first request for postoperative opioids by 20 minutes. Patients receiving neostigmine had an intraoperative heart rate of approximately 10 bpm less than the control group. Whether this lower heart rate was consequent to muscarinic effects of neostigmine or better pain control was uncertain.

Sodium Bicarbonate. Alkalinization of 0.5% prilocaine very slightly diminished onset time¹⁰. A similar study with 0.75% prilocaine found no change in onset time, but less pain on injection and less intraoperative opioid needs with pH adjusted local anesthetic. Unfortunately, studies involving lidocaine IVRA have been unable to demonstrate any advantages to alkalinization.

Opioids. Fentanyl, sufentanil, meperidene, and morphine have all been studied as adjuvants for IVRA¹⁰. Overall, the results have been conflicting as far as the ability of opioids used in IVRA shortening block onset time or potentiating intraoperative analgesia. After tourniquet deflation, nausea, vomiting, and dizziness have all been reported when opioids are added to IVRA.

Muscle relaxants. Most studies of adding paralytics (pancuronium, atracurium, mivacurium) to IVRA¹⁰ were supportive, with improved operating conditions in situations where motor block is advantageous, such as fracture reduction or tendon repair. Patients consistently have delayed return of extremity muscle strength after release of the tourniquet. Atracurium 0.03 mg/kg (approximately 2 mg in a 70-kg adult) has been recommended when motor blockade is deemed essential.

Long-acting local anesthetics. Several groups have investigated performing IVRA with levo-bupivacaine or ropivacaine¹¹. Results have consistently demonstrated slower onset times compared with lidocaine or prilocaine. Duration of postoperative analgesia has been increased with l-bupivacaine or ropivacaine, but to a much lesser degree compared to clonidine or ketorolac.

In this author's opinion, the modest post-operative analgesia provided by these long acting local anesthetics does not compensate for the slow onset times or higher toxicity risks incurred.

Forearm tourniquet (versus upper arm). Use of a forearm tourniquet may be an option for some distal upper extremity procedures. Several advantages are evident³. Forearm tourniquet pain is delayed in onset and of lesser severity compared to upper arm tourniquets. Approximately 50% less local anesthetic solution can be used when a forearm tourniquet is chosen, which should lower toxicity risks. 10 mg of ketorolac used in forearm tourniquet IVRA provided longer postoperative analgesia compared to ketorolac 20 mg used with an upper arm tourniquet IVRA. Finally, concerns regarding increased risk of nerve injury or poor tourniquet function with forearm tourniquets has not been substantiated in any study.

Pharmaco-economic studies. IVRA and axillary block both reduced postoperative pain and decreased antiemetic medication usage compared to general anesthesia. IVRA was the most time and expense efficient, with shorter operating room times, faster discharge, lower pharmaceutical costs, and least postoperative nursing care requirements.¹²

Table 1. Useful IVRA Pharmacologic Adjuncts

Ketamine 5-10 mg	Clonidine 50-100 mcg
Ketorolac 10-20 mg	Neostigmine 0.5 mg
Atracurium 2 mg	

Conclusions: IVRA is a safe, technically easy, and highly time-efficient anesthetic. Traditional limitations of the technique included slow block onset, tourniquet pain, limited postoperative analgesia, and local anesthetic toxicity concerns. Each of these concerns has been mitigated with pharmacologic adjuncts and improved technique. Neostigmine greatly speeds block onset time. Clonidine, ketamine, ketorolac, and neostigmine all substantially reduce tourniquet pain. Clonidine and ketorolac especially provide prolonged postoperative analgesia.

Procedural modifications have also improved IVRA safety and efficiency. Use of a distal IV catheter, local anesthetic solution injection over >90 seconds, esmarch bandage wrapping, and use of tourniquet pressures of at least 300 mm Hg with double lumen tourniquets have all been shown to prevent sub-tourniquet venous leakage of anesthetic solution.

Future areas of research include dose response studies to define the optimal dose of these adjuvants. Also, synergy likely exists when these agents are co-administered, so clinical studies defining optimal drug combinations would be clinically beneficial.

Christopher Viscomi, MD

Complete references at www.asra.com

Resident Activities at the 2003 Annual Spring Meeting

The ASRA Resident Section participated in several activities at the 2003 Annual Meeting in San Diego, California. The 9th Annual Resident Forum Cracker Barrel was held on Thursday, April 3, 2003. This year's topic was "Designing and Evaluating Clinical Trials". The speakers were Drs. Chris Wu and Michael Ferrante; Dr. Eric Wellmeyer, the Chair of the ASRA Resident Section, moderated the panel. The Cracker Barrel was well attended, despite the limited number of residents attending the ASRA Annual Meeting due to a conflict with the Western Anesthesia Residents Conference.

The ASRA Resident Section Committee (RSC) held its biannual meeting (the minutes are on the website at www.asra.com). Some of the topics highlighted were membership recruitment and retention, resident participation in the Annual Fall Pain Meeting, and the availability of scholarships to help with the cost of attending ASRA spring or fall meetings. It was an honor to have Dr. Eisenach (2002 ASRA President) and Dr. Horlocker (2003 ASRA President) attend our meeting. It is clear that they strongly encourage resident participation and are willing to continue supporting the efforts of the RSC.

The Committee thanked Dr. Eric Wellmeyer (UCLA), for his dedication and hard work as the exiting Chair; and Dr. Susan McDonald for continuing to serve as the faculty advisor. The next meeting of the RSC will be at the ASA Annual Meeting in San Francisco in October.

The ASRA Board of Directors feels strongly that resident and fellow participation is crucial to the success of the spring and fall ASRA meetings. In order to help residents and fellows attend, the Board of Directors will award ten, \$1000 scholarships per year.

Information on abstract submission, as well as scholarship applications will appear in the ASRA Newsletter and/or on the ASRA website.

Planning for the 2004 Annual Meeting in Orlando, FL is already underway and will include the 10th Annual Resident Forum Cracker Barrel. The topic will deal with interesting and innovative ways to teach regional anesthesia. Please visit the RSC web page at www.asra.com for details regarding resident events, future meetings and how to contact ASRA RSC members.

Sandra Kopp, MD

Chair, Resident Section

Resident Membership Grows

For the tenth year, the Society will be providing a one-year complimentary membership to all CA2's (**graduation 2005**) that have not already enjoyed this complimentary year.

In addition, Pain Fellows (2003-2004 academic year) will be provided an opportunity to affiliate.

Benefits of membership begin with the new academic year and include a subscription to *Regional Anesthesia and Pain Medicine*, the Newsletter, and reduced fees at all meetings, eligibility for research starter grants and access to the web site member only section.

Research Update: Pain Centralization and the Transition from Acute to Chronic Pain

Some good ideas die for lack of a champion, while others catch on like wildfire. The difference may have less to do with data than with the way the idea resonates with preconceptions, misconceptions, and the sheer will of people that it turn out to be right. Thus with the idea of “preemptive analgesia” and its conceptual antecedent “pain centralization”. Persistent pain, so it is thought, leaves a trace in the brain, changing from a transient, tractable form to a chronic form that is hard to treat. But is this really so? The logic and data behind preemptive analgesia in the setting of acute and subacute postoperative pain has been laid out in a number of recent reviews (Moiniche, 2002; Niv, 1998). Here I will focus on centralization as a factor in chronic pain.

Does pain in fact centralize?

It is widely held that chronic pain is like a cerebral mountain gorge, carved into the landscape of the brain by torrents of pain that have flowed through it. This idea probably originated in the rich literature on amputation phantoms. Pain felt in the limb prior to amputation is sometimes reported to persist in the phantom. The pain, originally of clear peripheral tissue origin, is said to “centralize”, i.e. to become “burned into the brain”, where it is therapeutically inaccessible. The typical story is of a painful lesion or cramp that the amputee swears can still be felt vividly in his phantom as a “pain memory” (Katz and Melzack 1990). Interestingly, reports of preamputation sensations preserved in phantom limbs extend to limb postures and even to accessories such as rings and bracelets worn before the amputation. Apparently centralization is not restricted to pain. Indeed, it may harken to the process whereby our entire body-image is established and maintained.

A natural corollary of the idea of pain centralization is the belief that one should be able to prevent the transition of pain from acute (and treatable) to chronic (and intractable) by applying analgesic modalities before “burning-in” has occurred. “If you wait, it will be too late!” Unaccountably, this dictum has attached itself to certain specific chronic pain conditions, notably phantom limb pain and RSD (reflex sympathetic dystrophy, now termed chronic regional pain syndrome (CRPS1), but not to others, such as headache or arthritis. Early, and not too well controlled attempts to capitalize therapeutically on the idea of centralization raised hopes. Bach et al. (1988) for example, reported that dense pre-surgical spinal block would prevent the later appearance of phantom limb pain, a result reported in several additional studies as well. Unfortunately, larger and better designed studies failed to obtain this outcome (Nikolajsen et al. 1998).

With respect to “pain memories” in amputees, it is difficult to verify tales of “burning in” objectively. A vague, tingling paresthesia, due to ectopic firing originating in a nerve end neuroma or axotomized dorsal root ganglion (DRG) for example, can easily become a ring in the mind of a suggestible patient. And a neuropathic paroxysm felt in the phantom limb can evoke vivid memories of a preexisting pain, especially when there is prior belief in centralization, and encouragement from the attending physician. Note that I do not question the reality of phantom pain; only that it is the same pain felt prior to the amputation. With respect to RSD, the “evidence” is completely circular. If pain evaporated early, either due to treatment or coincidentally, then it is gone and will not become chronic. All chronic pain was once acute pain that did not stop. The fact that it did not stop with therapy, or on its own, implies that it was of a persistent nature from the beginning. Who’s to say that it could have been stopped with early treatment but no longer can

be? But if pain is in fact a cause of plastic change in neural structures, if pain does “centralize”, the matter is not just of theoretical interest. There is urgency. Exploiting a brief window of opportunity could spell the difference between cure and chronic suffering.

Is pain *per se* risk-factor ?

Consistent with the concept of pain centralization is the observation that patients who end up with chronic pain are more often than not the ones who had particularly severe pain at the beginning, e.g. acutely after a surgical procedure. A relationship between intense acute pain, and subsequent chronic pain has been documented in a number of conditions. For example, particularly intense pain during the time of the shingles rash is a clear risk factor for the subsequent development of postherpetic neuralgia (PHN), and acute pain after thoracic surgery predicts long-term post-thoracotomy pain (Kalso, 1997; Katz, 1997).

But this sort of statistical correlation does not necessarily reveal mechanism or prove causation. Consider two alternatives, one focusing on the painful disorder and the other on the pain sufferer. Particularly intense early pain is likely to be a result of a particularly severe pain-provoking disease or trauma. If the precipitating disorder is more severe, it is not unexpected that the resulting pain will be more intense and longer-lasting. Likewise when we consider the patient. It is a fact that some people appear to suffer more from noxious stimuli than others. Perhaps this is due to early learning and other psychosocial and existential factors, or perhaps there is an inherited predisposition to pain. Whatever the reason, a pain-prone individual is likely to have more severe pain at all time points, early and late. Both of these alternative explanations see the late pain as deriving from basically the same process as the early pain. There is no “transition” from acute to chronic in the sense of a change in pain mechanism. The occurrence of A, and then B, is a simple acknowledgement of the arrow of time. While early treatment of pain is no doubt desirable in its own right, there is no urgent time window that makes immediate action a priority.

Like other treatment ideas, the efficacy of modalities that are presumed to prevent pain centralization can only be documented in prospective, placebo controlled clinical trials, difficult as such trials might be from a practical and ethical point of view. In the meanwhile, it is worth noting that pain centralization is not evident in a universe of clinical pain problems that, unlike RSD and phantom limbs, have clear causes and definitive treatments. For example, large numbers of patients suffer for years from osteoarthritic pain, but when hip replacement surgery is carried out, the hip pain is nearly always eliminated. Likewise, the pain of childbirth does not persist for long after delivery, and the pain of a kidney stone does not persist once the stone has passed. Indeed, if pain centralization were in fact a robust and common phenomenon, every pain we ever felt would still be felt.

These considerations, in the absence of solid evidence to the contrary, undermine the concept of pain centralization as a biological reality. There may in fact be no “transition” of pain from acute to chronic. However, this radical conclusion must be tempered by two essential distinctions.

Central changes after peripheral nerve injury

First, up until now I have been discussing whether pain, *per se*, centralizes, permanently changing its biological mechanism and its response to therapy. This scenario must be distinguished from a discussion of nerve pathology that may be painful. I have *not*

called into question the evidence that conditions such as trauma to peripheral nerves, or viral infection, can begin in the periphery and in time have pathological consequences also for the central nervous system (CNS). The evidence that peripheral pathology may precipitate (in the causal sense) CNS changes is strong (Devor, 1988; Melzack, 2001). although the question of which particular secondary CNS changes are important for chronic pain is far less clear (Devor 2001). Thus, the well-documented correlation between severe early pain and PHN in herpes zoster infection could reflect a more severe viral infection of the sensory ganglia (DRGs) in some patients, or less immune defense. A more severe infection is expected to result in more ectopic impulse discharge as infected DRG neurons wither and die, and hence more acute-phase pain. In addition, a more severe infection is expected to cause the death of a larger proportion of the neurons in the affected DRG(s) and hence more severe deafferentation and deafferentation pain (Dworkin et al. 1998).

Correspondingly, reducing the extent of nerve damage caused by the pathological process *is* an urgent priority with only a limited window of opportunity available. But the treatment must attack the cause of the neural injury. Treating the pain alone, using analgesics, may be ineffective. Treatment with antiviral medication in order to minimize DRG cell loss, on the other hand, has a different and much more compelling logic, and is likely to have very different consequences for the transition from acute pain to chronic pain (Dworkin et al. 1998). Of course, to achieve preemptive protection from chronic PHN pain using this approach, the antiviral treatment must in fact be effective in reducing viral burden and saving DRG neurons from degenerative changes. Otherwise, the anticipated benefit is not likely to be observed.

Psychological consequences of persistent pain

A second sense in which the transition from acute pain to chronic appears to be undeniable is the effect that protracted pain may have on the psyche of the patient, and on his/her family, carers and immediate society. There is solid evidence for a sad downward spiral from mature independence to frustration, disability, withdrawal, and depression, all due (in a causative sense) to unrelieved pain *per se*. Up to a point this downward spiral can be aborted and reversed by adequate pain relief. But in some individuals, at least, learned pain behavior and invalid status might become "burned into the soul" and become irreversible, even if definitive pain relief were eventually provided. Psychosocial deterioration due to chronic pain may indeed reflect neuroplastic psychobiological processes in the CNS, but this is a very different realm of discourse than normally comes under the heading of "pain centralization" (Niv and Devor 1998).

Conclusions

The idea of pain centralization has a great deal of intuitive appeal. It is also a tale with a moral: "Don't delay till tomorrow what you should do today". Unfortunately, the evidence upon which this belief rests is, for the most part, evanescent. Only a handful of reports in humans (e.g. Obata et al. 1999), and a few more in animal models (Melzack et al. 2001), are not easily dismissed. Nonetheless, wisdom demands that we keep an open mind. The absence of evidence is different from evidence of absence. The concepts of pain centralization, and of the inevitable transition of pain from acute to chronic forms, need to be viewed as interesting speculations. They should not, however, be taken as established fact.

Marshall Devor, PhD

Complete references at www.asra.com



**American Society of
Regional Anesthesia
& Pain Medicine**

Breakfast Panel at the ASA 2003 Annual Meeting

**LOWER EXTREMITY
PERIPHERAL NERVE BLOCK:
EVERYTHING YOU NEED TO
KNOW FROM THE EXPERTS**

**Wednesday, October 15
7:30 am to 8:45 am**

Moderator: Joseph M. Neal, MD
*Staff Anesthesiologist
Virginia Mason Medical Center
Seattle, Washington*

CLINICAL APPLICATIONS AND OUTCOME

Terese T. Horlocker, MD
*Professor of Anesthesiology
Mayo Clinic
Rochester, MN*

EQUIPMENT AND COMPLICATIONS

F. Kayser Enneking, MD
*Associate Professor of Anesthesiology
University of Florida
Gainesville, FL*

VIDEO PRESENTATION OF TECHNIQUES

Vincent W. S. Chan, MD, FRCPC
*Professor of Anesthesiology
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Annual Fall Pain Meeting & Workshops

November 13-16, 2003

Sheraton San Diego Hotel & Marina San Diego, CA

Refresher Courses / Workshops / Master Classes / Poster Discussions / PBLDs / Bonica Lecture

Scientific Chair: Marc A. Huntoon, MD, Mayo Clinic, Rochester, MN