ASRA NEWS

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

NOVEMBER 2017

In This Issue

ASRA PAIN MEDICINE EDUCATION RESEARCH

Advancing the science and practice of regional anesthesiology and pain medicine to improve patient outcomes through research, education, and advocacy

Table of Contents

American Society of Regional Anesthesia and Pain Medicine

Four Penn Center West, Suite 401 • Pittsburgh PA 15276
phone toll free 855-795-ASRA • fax 412-471-7503 • www.asra.com

ASRA News is published quarterly in February, May, August, and November. ISSN: 2474-2864

President's Message

Taking Stock of Education, Research, and Advocacy

As we approach the end of the year, it is natural to look ahead to the coming year and think about renewal. A new year, new resolutions, maybe a new perspective on an old problem—this is a perfect time to assess our progress and focus on where we are headed and what we need to do to reach our goals. Although we still have a couple of months left in 2017, we are quite enthusiastic about all that 2018 has to offer.

This is the perfect time to think about renewing your commitment to ASRA. In 2018, we have several new and expanded benefits that make your membership more valuable than ever, although our membership prices will remain the same as 2017. ASRA membership runs on a calendar basis from January through December, so be sure to renew early so that you don't miss out on even a moment of your benefits. If you are a fellow, be sure to select the "Young Professional" membership, which provides you with 3 or 4 years of membership for one low cost. This is part of ASRA's commitment to support those new to the profession. Once you become part of the ASRA family, we hope you will continue your loyalty and pass those messages to physicians who follow in your footsteps. You'll receive notification of your membership renewal soon, so watch your mail!

You'll also want to renew your membership in order to receive your subscription to our highly cited peer-reviewed journal, *Regional Anesthesia and Pain Medicine* (*RAPM*). We are thrilled to announce that we will be increasing the frequency of publication to eight issues in 2018, with a goal to eventually reach 12 times per year. A steadily increasing number of submissions to *RAPM*, which reflects the growing prestige that publication in the journal carries, has resulted in an overflow of articles. Publishing more issues will help to ensure that the articles are released in a timely fashion so that you can put the evidence into practice as soon as possible. As the journal's Impact Factor continues to increase, we are excited about even more growth for this valued member benefit.

This September, the ASRA Board had a retreat that included some time for strategic planning ideas for the coming 5 years. As a result of that process of taking stock, we have developed a new list of exciting projects that we plan to tackle in 2018. As the year progresses, I'll be sharing those new projects with you in this space.

Our ongoing goal is always to provide a wealth of practical and evidence-based resources to our members as we all work together to advance the science and practice of regional anesthesia to improve patient outcomes through research, education, and advocacy. If you read my column in August, you know that we have implemented a simpler, streamlined process for our research grants that enables potential researchers to submit a one-page letter of intent for review and approval before preparing a full-length research proposal. I also talked about the Faculty Development project that will, among other things, improve the resources available to members to become effective faculty members. This group's work is well under way with a variety of new offerings in the works.

Asokumar Buvanendran, MD
ASRA President

In addition to these initiatives, ASRA is also growing our continuing medical education (CME) meetings, including offering the Introduction to Perioperative Point-of-Care Ultrasound Course twice in 2018 (February 24–25 in San Diego, California, and December 1–2, in Chicago, Illinois). The CME Committee Meeting works diligently to create a collection of offerings based on the latest information, evidence-based practice, and world-renowned faculty. We recently met and developed a vision and goals for this group to ensure continued success in meeting the needs of our members (see sidebar).

Probably the most exciting event we have planned for 2018 is our World Congress on Regional Anesthesia and Pain Medicine being held April 19–21 at the Marriott Marquis in New York City. You can read more about the event in Meeting Chair Vincent Chan's article on page 10 and in an article by Narinder Rawal on page 13 that features the history of this event. We bill it as "five societies and four years in the making," because it brings together the five sister societies in regional anesthesia and pain medicine across the globe every 4 years, with nearly double the normal number of sessions and activities and programming that will appeal to the global audience. Our goal is to learn from and share with one another as we acknowledge that ASRA does not represent the entire world in terms of regional anesthesia and pain medicine. Providers in North America have much to learn from our colleagues in Asia, Africa, South America, and Europe, and we look forward to learning what our counterparts are doing to improve patient care, wherever they practice. To that end, we will offer a discounted registration tier for

Vision for ASRA CME:

- Be the number-one rated educational society for acute and chronic pain globally.
- Enhance ASRA's educational offerings by involving patients (in person or via electronic avenues) in the development of materials.

attendees coming from low-resource countries, as defined by the World Bank. We will also offer live streaming of content for those who are unable to attend in person. These additional offerings are possible only through the support of our industry partners, so we sincerely thank them for their generosity.

"Our ongoing goal is always to provide a wealth of practical and evidence-based resources to our members."

With regard to our advocacy pillar, ASRA continues to advocate for our members by working closely with the Centers for Medicare and Medicaid Services (CMS). We recently submitted a letter to CMS regarding the 2018 Quality Payment Program Year 2 Proposed Rule with recommendations to offer flexibility and reduce burden of the program. The Practice Management Committee has developed materials to support, inform, and educate our members. ASRA has also collaborated with the American Society of Anesthesiologists to develop six pain quality measures that reflect the appropriate care and measurements that physician anesthesiologists and pain medicine practitioners provide as part of their services. These measures will be developed through a Technical Expert Committee of ASRA-ASA and will be submitted to CMS this month, and we will provide a copy of the recommendations on our website. Finally, as I close, I read with keen interest a recent article on radiofrequency denervation of the lumbar region in the *Journal of the American Medical Association*. The ASRA Board of Directors is reviewing this publication to formulate an appropriate response.

What else can ASRA be doing to help you provide the best care for your patients? If you have suggestions, please email me at ASRAPresident@asra.com. And thank you for your support of ASRA!

Thinking Outside the Box

At the end of August, I headed to Schaumburg, Illinois, as part of a 12-member technical expert panel. The panel is composed of members of the American Society of Anesthesiologists (ASA) and ASRA and is working on the development of outcome metrics related to regional anesthesia and pain medicine (RAPM). I was happy to see ASA and ASRA leaders working together to give the RAPM community a voice in suggesting which metrics we should use to measure performance in these subspecialties. I expect more collaboration in the future between the two societies.

This was one of many advocacy efforts ASRA does on your behalf. This first in-person meeting concluded with six measures to be developed by this committee and later posted for public comment. These measures include use of multimodal analgesia, use of regional anesthesia for total knee arthroplasty, and implementing safeguards to ensure safe opioid use by pain practitioners.

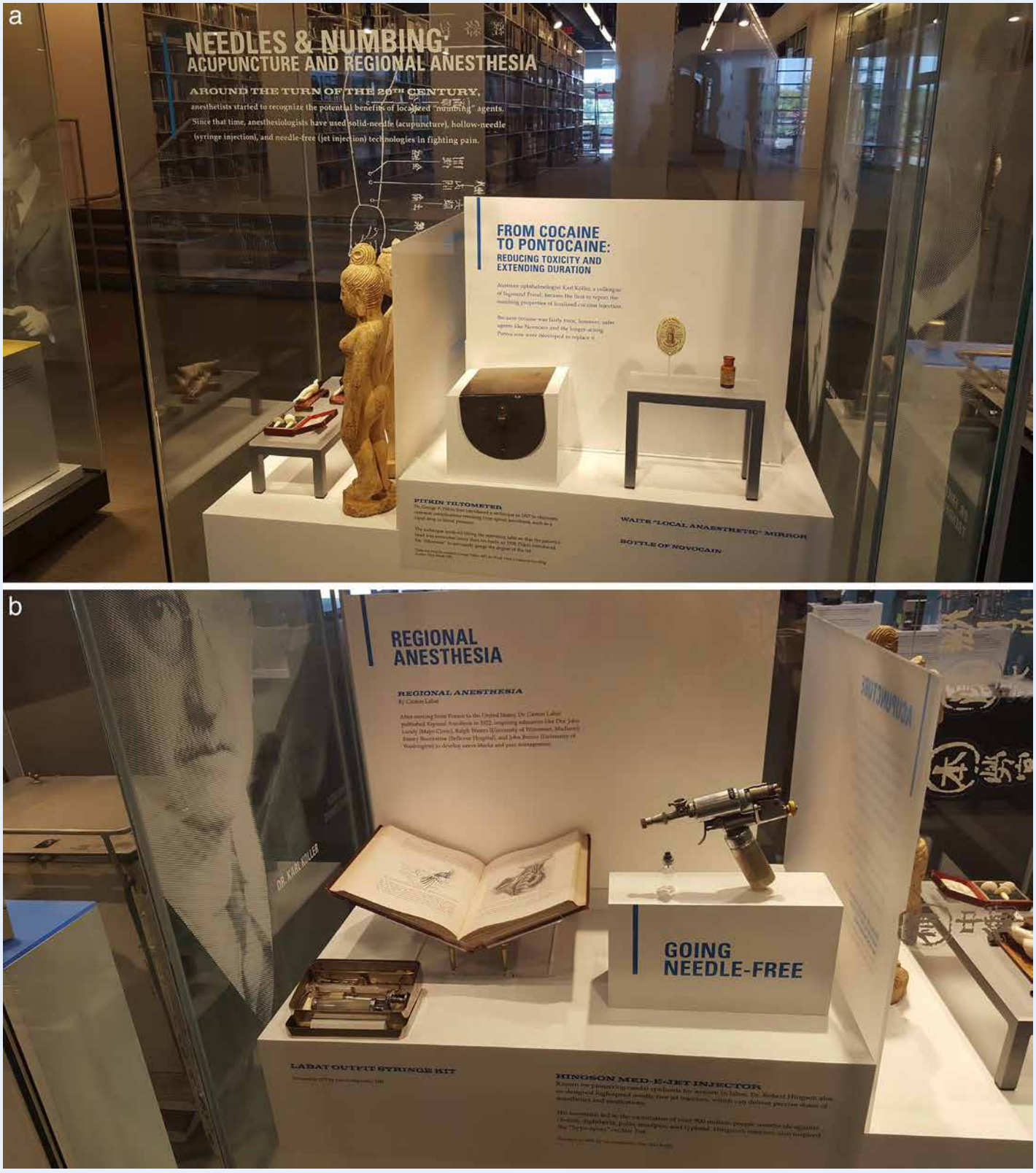
As a side note, this was my first visit ever to the ASA headquarters in Schaumburg, Illinois. ASA has done a very nice job in putting together a spectacular mural timeline exhibit (from darkness to light) representing the history of anesthesiology (Figure 1). I was particularly interested in some of the artifacts that represent the evolution of RAPM (Figure 2). If you are in the area, I encourage you to arrange a visit to ASA headquarters and spend some time in the Wood Library-Museum of Anesthesiology. I was glad I had this opportunity. The more I get involved with the RAPM community, the more I realize how creative these folks are. They always have a knack for innovation and thinking outside the box.

Nabil Elkassabany, MD, MSCE
ASRA News Editor

This issue of the *ASRA News* features some cutting-edge uses of regional anesthesia in critically ill patients and cardiac surgery. In addition, we feature an article about use of nerve blocks for patients having head and neck, ear, nose, and throat surgery. We also present the third installment of the problem-based learning discussion (PBLD) series. This article puts social media to work in an innovative and unprecedented way. As we have done in the prior two PBLDs, we solicit responses to different "twists" in the case scenario from experts in the field, compile these responses, and present to you the summary of these experts' opinions. We also post the stem

Figure 1: *"From Darkness to Light": Anesthesia History Exhibit at the Wood Library-Museum of Anesthesiology.*

Figure 2: *Exhibits from the Wood Library-Museum of Anesthesiology in Schaumburg, Illinois.*



case on the ASRA website, design a Twitter poll with questions about the case, and open it to all RAPM tweeps from around the globe to participate. It is impressive to see such a large number of responses representing a wide range of practice from different parts of the world. If you want to be part of this *ASRA News* feature, send an e-mail to asranewseditor@asra.com. You can contribute an interesting case scenario you experienced or be part of the expert panel that answers the discussion questions. You can also tweet about it using the hashtag #ASRAPBLD.

"The more I get involved with the RAPM community, the more I realize how creative these folks are."

In the May 2017 issue of the *ASRA News*, Dr Steve Hanling and colleagues presented an article about using stellate ganglion block for treatment of depression and posttraumatic stress disorder. This article stirred up a lot of discussion at that time, especially since a similar article was published concurrently in the *Wall Street Journal*. Dr Lipov, the medical director of the pain clinic at Illinois Masonic Hospital in Chicago, sent us a comment regarding Dr Hanling's article. We have included the comment and response from Dr Hanling in our Letters to the Editor section.

The 16th Annual Pain Medicine Meeting being held November 16–18 is almost here, and I am excited to read about all the educational offerings that ASRA has planned. I am also excited about the upcoming 2018 World Congress on Regional Anesthesia and Pain Medicine being held April 19–21, 2018, in the city that never sleeps: New York City. In this issue, you can learn about the history of the World Congress and preview some of what we have in store for you during the meeting. See you in New York!

All of this is included in this special issue of *ASRA News*, but there's even more. You have to read it all to learn it all.

16th Annual Pain Medicine Meeting at Disney's Yacht and Beach Club Resorts: Leading With Quality

It is my pleasure to invite you to the 16th Annual Pain Medicine Meeting: Leading With Quality, taking place November 16–18, 2017, at the Disney Yacht & Beach Club Resorts at Walt Disney World in Lake Buena Vista, Florida.

Building on successes from previous meetings, the scientific/education planning committee considered past-attendee feedback, input from several ASRA special interest groups, and consultation with world-renowned leaders in pain medicine to create an exciting and informative meeting that you won't want to miss.

INNOVATIVE PROGRAM CONTENT

Thursday's refresher courses feature international experts in the field of pain medicine, with basic science and clinical updates on complex regional pain syndrome, opioids, and spinal cord stimulation. A highlighted session will focus on spinal pain, from diagnosis to best practices, for optimizing patient outcomes. This outstanding panel will be moderated by Carlos Pino, MD, and includes leading experts David Kennedy, MD, James Rathmell, MD, Steven P. Cohen, MD, and Chad Brummett, MD.

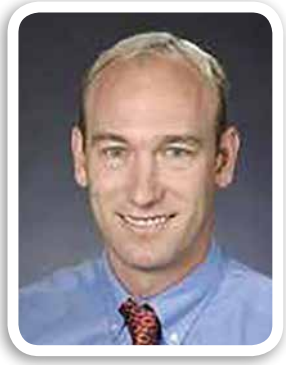
Take advantage of lunch with an expert with one of 25 problem-based learning discussions where you can interact directly with nationally recognized faculty on topics ranging from basic sciences to percutaneous image-guided lumbar decompression, neuromodulation, challenging clinical scenarios, coding, and practice management. This is truly one of the special aspects of the ASRA meeting, bringing faculty and participants together for rich discussion and networking.

Friday's parallel sessions include trigeminal neuralgia, palliative medicine, regenerative medicine, and treating patients with challenging conditions, including fibromyalgia, facial pain, urogenital pain, and terminal cancer. Moderated by Andrea Nicol, MD, this session features international experts Daniel Clauw, MD, Ursula Wesselmann, MD, Leonardo Kapural, MD, and Afton Hassett, PsyD. The afternoon concludes with two focused plenary panels on opioid management "from initiation to termination" and advancements in the field of neurostimulation.

Saturday's plenary sessions feature intrathecal drug delivery, mechanisms, and best practice guidelines, as well as sessions on physician burnout and musculoskeletal diagnosis and treatment.

We are especially proud to present a must-attend session, "Prospering in the New Healthcare Environment," featuring Joseph Perz, DrPH, MA, from the Centers for Disease Control and Prevention, and David W. Baker, MD, MPH, FACP, from the Joint Commission. They will discuss infection control and the Joint Commission's newly revised Pain Assessment and Management Standards slated to take effect in January 2018. This session can be attended individually or as part of the full-day ASRA-ASA Practice Management Portfolio, offered at no additional charge. The program additionally offers a session titled "Reporting Measures and Payment Models," featuring Matthew Popovich, PhD, from the American Society of Anesthesiologists and Richard Rosenquist, MD, from the Cleveland Clinic, as well as "Maintaining Premier Status Through Continual Improvement," covering topics such as key metrics, contracts, hiring, and practice efficiency. This innovative program is designed for interactive discussion on key aspects of coding/compliance, managing a pain practice, and key aspects of MACRA in 2017 and in the years to come. These sessions are truly a must for both physician leaders and practice management specialists, designed to help your practice reach its full potential.

"The outstanding scientific program and networking opportunities will make this meeting a unique and informative experience not to be missed."



Kevin Vorenkamp, MD
Chair, Scientific/Education Planning Committee
Director, Pain Medicine Fellowship
Department of Anesthesiology and Pain Medicine
Virginia Mason Medical Center
Seattle, Washington

WORKSHOPS AND INTERACTIVE SESSIONS

In addition to the main meeting sessions, you will have the opportunity to attend many hands-on workshops in areas of ultrasound and fluoroscopy, including regenerative medicine, radiofrequency ablation techniques, and surgical practicum. The highly popular ultrasound workshops, first demonstrated at ASRA and continuously evolving to meet learners' needs, now will offer a multistaged curriculum that will prepare individuals interested in pursuing the Pain and MSK Interventional Ultrasound Certificate.

PA/NURSE PRACTITIONER/NURSING PROGRAM

We realize the importance of nonphysician providers in our busy health care system and continue to be excited about the growth of this aspect in our program. In addition to the dedicated PA/NP/Nursing program, participants also have the opportunity to attend

targeted interactive sessions on intrathecal therapy, diagnostic imaging, and the hands-on physical exam workshop. Plus, don't miss the PA/NP/Nursing Meet and Greet on Friday at 10 a.m. in the Exhibit Hall!

RESIDENT AND FELLOW PROGRAM

Our Resident and Fellow Educational Program reflects input from the Resident Section Committee, requesting not only relevant clinical information but also pertinent information from leading clinical and practice management experts on how to transition successfully into practice. The procedural workshops on fluoroscopy and ultrasound-guided procedures fill up early, so register soon! Residents and fellows also have the opportunity to enjoy a beverage while networking with fellow trainees and members of the Association of Pain Program Directors at the Resident/Fellow and Pain Program Directors Meet and Greet being held on Friday at 5:30 p.m.

SPECIAL EVENTS

In addition to the moderated ePosters and abstracts, several other activities are occurring alongside the main meeting. Learn about emerging therapies and innovative solutions in the exhibit hall, or attend the non–continuing medical education breakfast and luncheon events. The popular Wine and Bubbly Networking Reception and Exhibit Hall Grand Opening will be held on Thursday evening at 5:15 p.m. Plus, don't miss our popular Excellence in ASRA Awards Luncheon on Saturday, featuring past president Michael Stanton-Hicks, MD, of the Cleveland Clinic presenting the always thought-provoking John Bonica Lecture (Figure 1).

Figure 1: *Dr Michael Stanton-Hicks, MD, 2017 John Bonica Lectureship Award recipient.*

Finally, be sure to plan to stay for the Saturday Annual Meeting Celebration on Shipwreck Beach where faculty and participants can relax together and have some fun after the meeting (Figure 2). This year's event is a kid-friendly, casual beach party with something for everyone: BBQ, cocktails, games, music, and a crazy Hawaiian shirt contest, as well as some fun Disney surprises. The party is on a beach, so wear your flip flops and bring your family and friends.

WELCOME TO DISNEY

In addition to the exceptional Disney's Yacht & Beach Club facilities, hospitality, and service, we are confident that the outstanding scientific program and networking opportunities will make this meeting a unique and informative experience not to be missed. The world of pain medicine is an exciting area in which to work, and we'll continue to meet and bring inspired people together in forums like this to ensure our organization and discipline remains at the cutting edge. Register at www.asra.com/pain.

Figure 2: *Shipwreck Beach is the site of the Saturday Celebration at Disney's Yacht & Beach Club Resorts in Lake Buena Vista, Florida.*

2018 World Congress of Regional Anesthesia and Pain Medicine

What an honor and exciting opportunity for ASRA to host the 2018 World Congress on Regional Anesthesia and Pain Medicine on April 19–21, 2018, at the Marriott Marquis Hotel in Times Square, New York City! This is the first time in ASRA's history that we've had the privilege of planning and organizing such a prestigious international event. In keeping with the spirit and vision of the World Congress, ASRA will convene one of the largest gatherings of international leaders, content experts, and delegates from around the world who will share global perspectives on regional anesthesia, both acute and chronic pain management, along with global educational and clinical care issues. Together with our four sister societies (African, Asian and Oceanic, European and Latin America Societies of Regional Anesthesia), we are committed to bringing this world-class education event to all physician, nursing, and physician assistant colleagues who are dedicated to this specialized segment of patient care. We expect to welcome more than 2,000 delegates and will provide unique networking and learning opportunities for colleagues at all career levels and from all geographic locations, irrespective of their practice setting, public or private.

For our colleagues living and working in low-income countries (as classified by the World Bank), ASRA is committed to offering discounted registration fees for both the overall meeting and individual workshops. And for those who cannot attend in person, ASRA will provide complimentary live streaming for the most popular sessions.

Here are some of the program highlights to pique your interest and attention.

Vincent W.S. Chan, MD, FRCPC, FRCA
Chair, Scientific/Education Planning Committee
Professor of Anesthesia
University of Toronto
Toronto, Ontario, Canada

FACULTY HIGHLIGHTS

During the congress, you will have the opportunity to meet with more than 160 national and international faculty, many of whom are innovators and champions of the latest regional anesthesia and pain medicine initiatives. To embrace the spirit of the World Congress, ASRA has invited officials and distinguished speakers from all five of the sister societies. You will no doubt gain insightful knowledge, clinical pearls, and

international perspectives from these experts, helping you further refine well-established techniques of regional anesthesia and pain management and define the role and efficacy of new block approaches and emerging clinical practice trends.

PROGRAM AND CONTENT HIGHLIGHTS

The 2018 World Congress scientific program will feature many learning formats with rich content that is suitable for learners of all levels. There will be 28 regional anesthesia and acute pain parallel sessions and 31 hands-on workshops. For chronic pain topics, there will be 19 parallel sessions and 12 hands-on workshops. Also included are 24 problem-based learning discussions, a full-day Physician Assistant/Nurse Practitioner/Nurse program, special Resident/Fellow workshops, and plenty of ePoster opportunities throughout the meeting. There will be no shortage of interesting sessions for you to attend.

Here are some of the highlights of the regional anesthesia program.

- 360° roundtable discussion on enhanced recovery after surgery with a patient-surgeon-anesthesiologist dialogue
- Mini symposium on how to set up a fast-track joint replacement surgery program
- Several innovations in acute pain medicine panels
- Best evidence updates on the use of local anesthetic adjuncts, intravenous analgesics, and neuraxial anesthesia and analgesia
- Interactive "tips for experts by experts" sessions to discuss challenging clinical cases through procedure video presentations, medico-legal case discussions with lawyer participation, and a number of pro-con debates to address contemporary concepts and controversies

The chronic pain program comprises:

- Newer topics feature regenerative medicine, marijuana and cannabinoids in chronic pain management, relevant anatomy and imaging to improve success in pain diagnosis and treatment, complications of chronic pain management, and medico-legal case discussion with lawyer participation.
- The Ultrasonography in Pain Medicine special interest group (SIG)–initiated panel will feature advances in musculoskeletal imaging and interventions.
- The Neuromodulation SIG session will discuss best practices and safety of neuromodulation therapy.
- The Headache SIG panel will present modern headache care involving neurology and interventional pain medicine.
- A member-submitted panel on challenging centralized pain syndromes will round out the member-submitted topics and expert faculty.

Some global issues included in the program are:

- Opioid epidemics: global perspective on the appropriate use of chronic opioids
- An International Neuromodulation Society (INS) panel focusing on advances in neuromodulation
- An international panel discussing outcomes that matter to regional anesthesia
- International perspectives on education in regional anesthesia and acute pain medicine
- Innovative pain medicine initiatives around the world

WORKSHOP HIGHLIGHTS BEYOND THE REGULAR OFFERINGS

New Regional Anesthesia Topics.

- Half-day, preconference workshop titled "Introduction to Perioperative Point-of-Care Ultrasound" on airway, lung, diaphragm, stomach, and eFAST
- Half-day, preconference workshop titled "Introduction to Focused Cardiac Ultrasound" covering transthoracic echocardiography
- Interactive demonstration workshops focusing on newer block techniques such as PECS, serratus plane, iPACK, quadratus lumborum, erector spinae, and retrolaminar blocks
- Half-day cadaver needling and catheter insertion hands-on practice
- Ultrasound for neuraxial blockade for difficult spine anatomy
- Half-day special cadaver dissection and anatomy workshop
- Mini boutique New York School of Regional Anesthesia 3D anatomy and live scanning workshop

New Chronic Pain Topics.

- Ultrasound for regenerative medicine
- Half-day advanced neuromodulation techniques (DRG/HF, peripheral, trigeminal, lamitrode)
- Half-day musculoskeletal ultrasound for joint examination and injection
- Half-day interventional cancer pain management involving neurolytic blocks, intrathecal pump, and vertebroplasty

Other exciting offerings include high-fidelity simulator workshops for thoracic blocks and crisis management for the regional anesthesiologist. Again, a registration discount is provided as an incentive for delegates from low-income countries to attend. Traditional landmark and nerve stimulator–based nerve block techniques will be taught in addition to ultrasound. New interactive sessions will teach prevention of physician burnout and stress management and a new concept of green anesthesia: the leading role of regional anesthesia.

"Together with our four sister societies (African, Asian and Oceanic, European, and Latin American Societies of Regional Anesthesia), we are committed to catering this world-class education event."

The Wednesday premeeting sessions will provide opportunities for attendees to obtain two important certifications:

- Written and practical exam for the ASRA Pain and MSK Interventional Ultrasound Certificate
- European Diploma in Regional Anesthesia & Acute Pain Management (EDRA) Part 1 written exam

RESIDENT AND FELLOW WORKSHOP HIGHLIGHTS

A full-day preconference Advanced Neuromodulation Comprehensive Hands-On Workshop with Practical Case Management for Future Implanters will provide didactic and hands-on training led by 17 world-class experts geared exclusively to pain fellows. Scholarships for this non–continuing medical education event will be provided as a result of a detailed application process. In addition, three special Resident and Fellow workshops are planned: one for regional anesthesia and perioperative point-of-care ultrasound, one for chronic pain fluoroscopy-guided injections, and one for ultrasound-guided injections.

NURSING AND PHYSICIAN ASSISTANT PROGRAM HIGHLIGHTS

Physician assistants, nurse practitioners, and nurses are an integral part of the meeting. A full-day, dedicated program will provide an update on opioids, low-back pain physical assessment and interventions, and chronic pain management in special challenging patient populations. We extend a warm welcome to our nursing and physician assistant colleagues to attend not only the dedicated program but the entire World Congress.

SOCIAL EVENTS

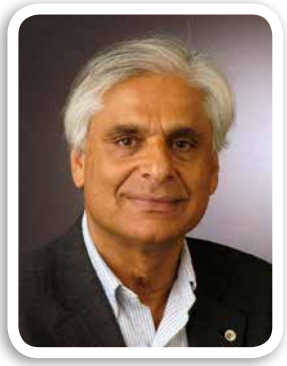
After a day of intensive learning, we invite you to enjoy a time of relaxation and social gathering with your colleagues. Come to the World Congress Opening Ceremony on Thursday to greet the presidents of the five regional anesthesia and pain medicine societies and enjoy a New York–style entertainment show performance (exact nature to be kept secret at this time) before heading to the Wine and Bubbly reception. On Saturday, all are welcome to the Saturday Celebration overlooking Times Square.

Are you coming to New York, the city that never sleeps? Are you coming to the first ever ASRA-hosted World Congress of RAPM? I hope you are! Come and experience this international gathering where education, scholarly exchange, and networking opportunities are all bundled together in the typical ASRA family style. We look forward to welcoming you to the World Congress next April. See you in New York!

A Brief History of the World Congress of Regional Anaesthesia

The concept of having a World Congress of Regional Anesthesia was an evolutionary process that started in the 1980s with joint ASRA and European Society of Regional Anesthesia (ESRA) meetings in Vienna (Austria) and Williamsburg, Virginia (United States) and in the 1990s in Brussels (Belgium), Auckland (New Zealand), and Quebec (Canada), the latter two under the auspices of the International Society of Regional Anaesthesia (ISRA).

The idea of arranging a World Congress of Regional Anesthesia to promote regional anesthesia and analgesia techniques with an international perspective and supported by all the major regional anesthesia societies was floated in ESRA under the leadership of Narinder Rawal (secretary general) and André van Zundert (president), with the ESRA Board approving the event. Instead of the regular annual meeting in 2002 in Barcelona, it was decided to have a truly international scientific program by including faculty from all four major regional anesthesia societies: ASRA, ESRA, the Latin America Society of Regional Anesthesia (LASRA), and the Asian and Oceanic Society of Regional Anesthesia (AOSRA), including presidents and secretaries from each society. More than 110 international speakers from all four societies participated. The extensive scientific program included topics of interest to delegates from low-resource countries, and a large number of workshops included cadaver and anaesthetized pigs. The Barcelona World Congress on Regional Anesthesia was very successful, with the highest number of attendees ever in a regional anesthesia congress (about 1,800). Michael Cousins from Australia was the Carl Koller award recipient that year (Figure 1).



Narinder Rawal, MD, PhD, FRCA, EDRA
Professor
Örebro University
Örebro, Sweden

The success of the first World Congress of Regional Anesthesia encouraged the presidents and secretaries of all four regional anesthesia societies to have such meetings at 4-year intervals on a rotating basis. It was agreed that all the administrative and financial arrangements would be the responsibility of the organizing society. The second World Congress took place in Rio de Janeiro (Brazil) in 2006 under the auspices of LASRA (Figure 2). Although there were several administrative challenges, approximately 700 delegates attended the congress.

Figure 1: *The first joint World Congress on Regional Anaesthesia and Pain Therapy was held in Barcelona, Spain.*

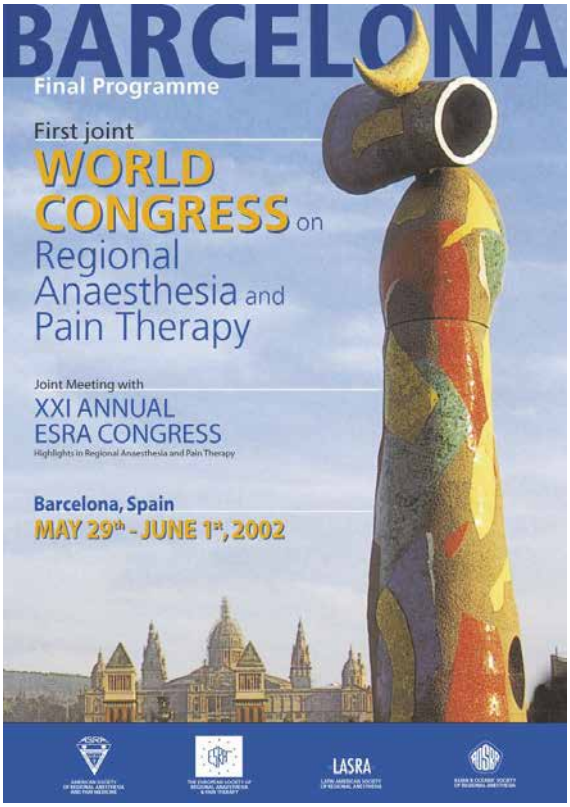


Figure 2: *Rio de Janeiro was the site of the 2nd World Congress in 2006.*

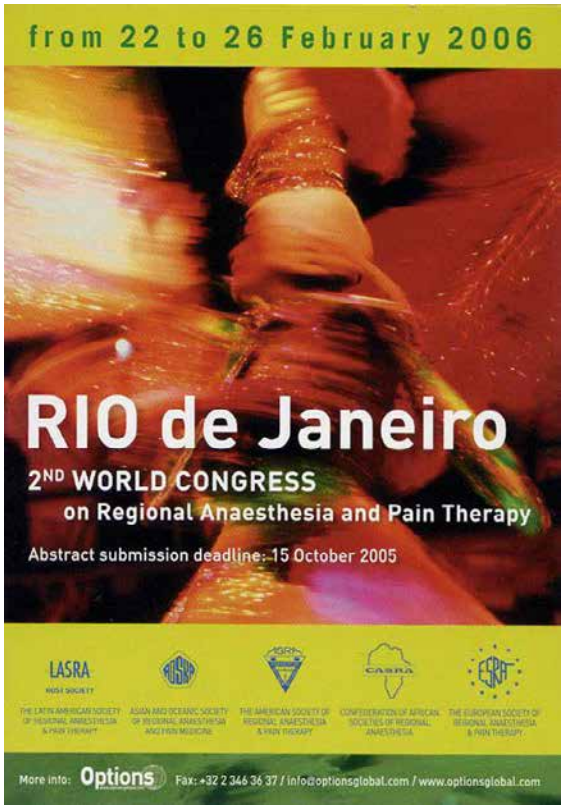


Figure 3: *The 3rd World Congress of Regional Anaesthesia and Pain Therapy was held in Sydney, Australia.*

Susilo Chandra (Indonesia) and Stephen Gatt (Australia) were formally awarded the third World Congress meeting held in Sydney, Australia, in 2010 (Figure 3). Again, because of administrative problems, the congress was postponed until 2013. Chandra Kumar (Singapore), Ezzat Aziz (Egypt), Susilo Chandra (Indonesia), and André van Zundert (the Netherlands) provided important support in reviving the project. Despite the many challenges including funding problems, the congress president Stephen Gatt managed to put together the congress in 2013 with more than 80 international speakers and approximately 700 delegates. The scientific program included several workshops with phantoms, models, cadavers, and anesthetized pigs as well as ultrasound-guided regional techniques. The 4th World Congress was awarded to Ezzat Aziz (Egypt) and Milton Raff (South Africa) under the banner of the newly formed African Society of Regional Anesthesia. The congress was scheduled to take place in Cairo, but owing to political turmoil in Egypt, it was agreed to move the venue to Cape Town, South Africa (Figure 4). The congress took place just 1 year later in 2014 under the presidency of Ezzat Aziz with Chandra Kumar (Singapore) as scientific chairman and Manoj Karmakar (Hong Kong) as workshop chairman. Narinder Rawal (Sweden) supervised both scientific committees supported by two representatives from each zonal society. Approximately 880 delegates participated, including 110 international speakers. Model, cadaver, and anesthetized pigs as well as ultrasound-guided regional techniques were included in the workshop program. The scientific program was adapted to the international ethos and participation of the congress. In addition to the usual scientific program, there were sessions relevant to

Figure 4: *Final program of the 4th World Congress of Regional Anaesthesia & Pain Therapy.*

delegates from Africa, Asia, and Latin America.

After Barcelona, Rio de Janeiro, Sydney, and Cape Town, the World Congress now moves to North America under the leadership of Asokumar Buvanendran, MD, ASRA president, and Vincent Chan, past-president ASRA and chair of the 2018 World Congress Scientific/Educational Planning Committee. The choice of New York as the venue for the 5th World Congress of Regional Anaesthesia is in keeping with the tradition of selecting truly extraordinary locations. We look forward to another world-class congress in one of the greatest cities in the world.

"The success of the first world congress encouraged the presidents and secretaries of all four zonal regional anesthesia societies to have such meetings at 4-year intervals on a rotating basis."

ACKNOWLEDGMENTS

The author would like to acknowledge the important contributions of the following persons in preparing this article: André van Zundert (Australia), Chandra Kumar (Singapore), Ezzat Aziz (Egypt), and Steven Gatt (Australia).

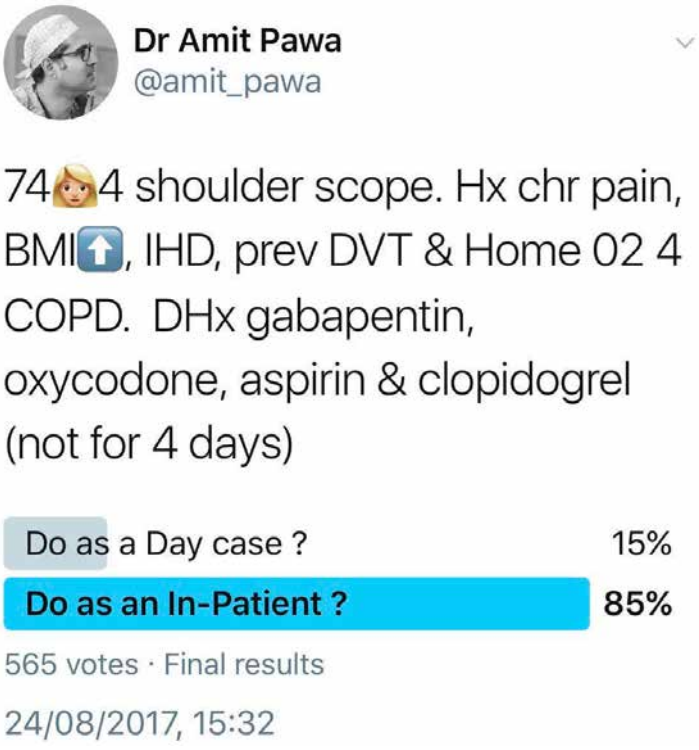
Problem-Based Learning Discussion (PBLD): Postoperative Pain Management in Patients Undergoing Shoulder Arthroscopy

Editor's note: We hope you enjoy this third installment of the problem-based learning discussion feature for the *ASRA News*. We contacted some of the readership to provide responses to the case selected for this feature. **To keep this feature going, though, *we need your help!***

1. Please send deidentified cases you would like to see discussed in this format to the *ASRA News* at asranewseditor@asra.com. We will collectively choose the most suitable cases for discussion.
2. Please let us know if we can count on you as a contact to reply to cases and provide your opinion on how you would manage said case. Please send your name, practice setting, and contact information to asranewseditor@asra.com.

Thanks, and enjoy!

A 74-year-old woman presents for left shoulder arthroscopy. She suffers from chronic shoulder pain, obesity (body mass index [BMI] of 45), coronary artery disease (drug-eluting stent placed 18 months ago), and previous deep venous thrombosis (DVT). She is also using 2 L of oxygen continuously because of chronic obstructive pulmonary disease (COPD). Medications include gabapentin 600 mg every 8 hours, oxycodone 20 mg every 4 hours as needed, metoprolol, simvastatin, aspirin, and clopidogrel, which has been held for 4 days. Her cardiologist deemed her to be at a low risk from a cardiac standpoint and stated that no further cardiac testing is needed before surgery.



Melanie Donnelly, MD
Associate Professor
University of Colorado
Aurora, Colorado

Kristopher Schroeder, MD
Associate Professor
University of Wisconsin
Madison, Wisconsin

Dr Schroeder provided the case, and Dr Donnelly compiled the responses.

Would you prescribe any oral premedications (eg, gabapentin, opioids) prior to the surgical procedure?

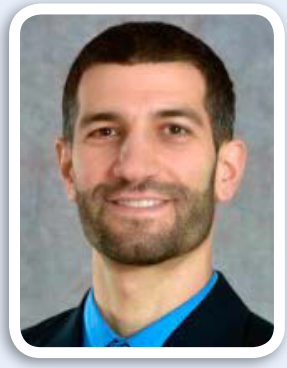
Dr Auyong: Multimodal analgesics are an important part of managing perioperative pain. For most outpatient shoulder surgeries, I like to administer acetaminophen and nonsteroidal anti-inflammatory drugs (NSAIDs) prior to surgery. Typically, I do not give gabapentin for outpatient surgeries because of the risk of postoperative sedation. However, in a patient who is already on gabapentin and oxycodone, I would not hesitate to ensure she took those medications preoperatively as well.

Dr Maniker: I would ensure that she has taken her gabapentin on the morning of surgery and would prescribe preoperative 1 g oral acetaminophen and 200 mg celecoxib.

Dr Harrington: This patient would be given 1,000 mg oral acetaminophen prior to surgery. She would also be instructed to take her usual 600 mg gabapentin preoperatively.

Contributors:

David Auyong, MD
Staff Anesthesiologist
Virginia Mason Medical Center
Seattle, Washington



Robert B. Maniker, MD
Assistant Professor of
Anesthesiology
Columbia University
New York, New York

Brian E. Harrington, MD
Staff Anesthesiologist
Billings Clinic Hospital
Billings, Montana

Amit Pawa, BSc, MBBS, FRCA, EDRA
Consultant Anesthetist
Department of Anaesthesia
Guy's & St Thomas' NHS Foundation Trust
London, United Kingdom

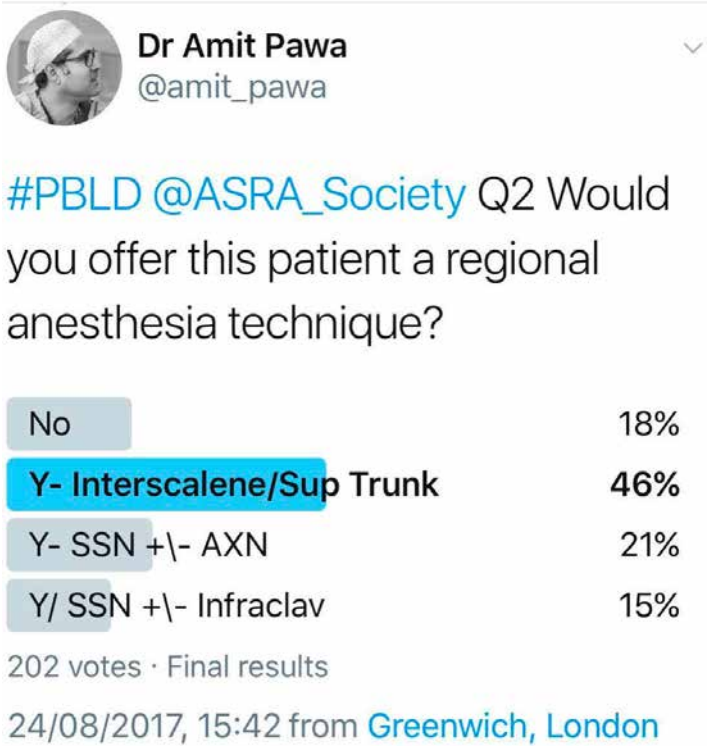
An NSAID (oral celecoxib or intravenous ketorolac) would be used on a case-by-case basis in consultation with the surgeon.

Dr Pawa: Under normal circumstances for this type of surgery, I would not routinely prescribe oral premedication. In this particular instance, in the context of preexisting chronic pain, preoperative opioid and gabapentin use, and significant respiratory disease, I would prescribe at least her usual drugs prior to the surgical procedure so at least I would be starting at her baseline.

Would you offer a regional anesthesia technique? Which block? Would you use a catheter?

Dr Auyong: I would absolutely offer a regional anesthesia technique for postoperative analgesia. My approach would be a continuous suprascapular catheter via an anterior approach. My research team and I have recently completed two studies evaluating the anterior approach to the suprascapular nerve, one of which was published this year. We showed that continuous suprascapular catheters had equivalent analgesic efficacy as interscalene catheters but preserved at least 82% of vital capacity after 24 hours of continuous infusion compared with only 62% for interscalene catheters. Therefore, in this patient with restrictive lung disease secondary to obesity and preexisting COPD, I would prefer an anterior approach suprascapular nerve catheter for analgesia while maximizing our chances at preserving her lung function.

Although regional anesthesia could be used for surgical anesthesia, the patient's baseline oxygen requirement, her obesity, and the sitting position used for shoulder arthroscopy would make any additional airway support during the case difficult. Therefore, I would prefer to secure her airway from the beginning of the case and use general anesthesia (GA).



Dr Maniker: Yes, I would recommend long-acting, single-shot, combined infraclavicular and suprascapular nerve blocks. I would avoid interscalene or supraclavicular block because the patient has a low pulmonary reserve (given her continuous oxygen requirement) and would likely not tolerate ipsilateral phrenic block. Infraclavicular block would cover the axillary nerve, as well as lateral pectoral and upper and lower subscapular nerves. When combined with a suprascapular nerve block, this should provide good postoperative analgesia and only spares the supraclavicular nerves from the superficial cervical plexus. I would consider catheters but would need to further discuss issues, including coagulation status, plan for timing of hospital discharge, and interference with the surgical field.

Dr Harrington: I am concerned about respiratory reserve as well as platelet function (in the face of clopidogrel plus aspirin). A regional technique of single-shot suprascapular block plus surgical wound infiltration would be encouraged.

Dr Pawa: There are clearly several options for anesthesia and analgesia here with a number of potential risks and complications. My concerns with a classic interscalene block as the sole mode of anesthesia here relate to the impact of phrenic nerve palsy on her COPD and her being able to tolerate this perioperatively. One option is to perform a single-shot interscalene or superior trunk block and use a continuous positive airway pressure (CPAP) mask perioperatively to support respiration. I would also be aware that performing a plexus block with clopidogrel use within 7 days also makes the risk of hematoma a concern and would dissuade me from using a catheter.

I could do the interscalene and combine it with GA, or my backup plan would be to perform ultrasound-guided suprascapular and axillary nerve blocks supported by sedation or a GA. These techniques may be challenging in someone of her size, but a clear risk-versus-benefit discussion with the patient would help me reach a decision.

How does the presence of clopidogrel, elevated BMI, and history of chronic pain influence your decision?

Dr Auyong: Whenever confronted with a difficult decision, I try to look at this from the patient's perspective. First, in regard to the clopidogrel, there is always a balancing act between anticoagulation, risk of recurrent clots (DVT or pulmonary embolism), risk of stent thrombosis, and risk of procedural bleeding. For a compressible nerve block that has significant analgesic or outcome benefit for the patient, I would proceed with the nerve block despite not having been off clopidogrel for 5 to 7 days.

Second, this patient has several comorbidities (obesity and chronic pain) that, if combined with poor postoperative analgesia, could place her at significantly higher risk for postoperative complications. The alternative primary analgesic is using opioids, which comes with obvious unwanted side effects. Obesity is the most common cause of restrictive lung disease, and patients with restrictive lung disease are most reliant on diaphragmatic movement for ventilation. Therefore, obese patients are most affected by hemidiaphragmatic paralysis seen in brachial plexus regional anesthesia.

To better assess the effect of possible hemidiaphragmatic paralysis on this patient, I would place a suprascapular catheter using a short-acting local anesthetic (lidocaine or chloroprocaine). I would then monitor the patient and evaluate the effect of the nerve block for 20 minutes. If the patient has clinical dyspnea, it indicates that she is unable to tolerate any decrease in diaphragm function. In this scenario, I would not initiate the continuous infusion through the catheter because of the poor clinical outcome of phrenic nerve paralysis in this patient. If the patient does well and does not have side effects from the bolus of local anesthetic, I would start the continuous infusion via the suprascapular nerve catheter.

Finally, in regard to her chronic pain, I know her preoperative reliance on opioids also increases her postoperative risk for complications, especially in the setting of obesity. Her history of chronic pain makes me all the more apt to offer regional anesthesia via a continuous catheter.

Dr Maniker: Because clopidogrel has not been held for 7 days, an increased, albeit low, risk of bleeding remains because of platelet inhibition. Additionally, nerve blocks in this case are at relatively peripheral and compressible locations. Furthermore, these decisions require the weighing of overall risk and benefit. Avoiding peripheral nerve blocks would result in administration of more opioids in the intraoperative and immediate postoperative periods and risk significant respiratory depression in this patient with morbid and extreme obesity (class III). Given these considerations, I would still proceed with peripheral nerve blocks.

Dr Harrington: I would be reluctant to perform any brachial plexus block if clopidogrel was discontinued fewer than 5 days prior without first documenting a normal platelet function assay.

Because of her elevated BMI, decreased functional residual capacity and respiratory reserve make any brachial plexus block above the clavicle (interscalene or supraclavicular block) hazardous.

This patient has chronic pain with significant opioid tolerance, making regional techniques attractive. I would definitely administer ketamine intraoperatively.

Dr Pawa: The chronic pain history emphasizes the importance of maintaining her usual drug therapy in the perioperative period and

would steer me toward using a regional anesthesia technique in some way if only to minimize her additional opiate requirement. The use of dual antiplatelet therapy within 7 days does induce a mild amount of anxiety, and the potential for a deep plexus in view of her BMI may steer me more toward the peripheral techniques (suprascapular nerve and axillary nerve).

The patient discusses with you her fear that her pain has been incredibly poorly controlled with previous surgical interventions and that this frightens her more than other potential complications. The surgeon approaches you and would very much prefer a catheter technique.

Would this impact your willingness to perform this technique, and if so, how? Why?

Dr Auyong: The focus should be on what is best for the patient, not the surgeon. I would plan on a continuous catheter technique after discussing risks, benefits, and options with the patient.

Dr Maniker: Infraclavicular and suprascapular catheters are reasonable to consider, given the patient's chronic pain and opioid tolerance as well as her pulmonary disease, which would render the negative respiratory effects of opioids particularly deleterious for her in the postoperative period. This would require further discussion with the patient as well as with the surgeon regarding any interference of the catheters on the surgical field.

Dr Harrington: In my hands, the only effective catheter technique under these circumstances would be a continuous interscalene block. Because of the pulmonary risks involved, if an interscalene catheter was considered necessary, I would insist that the procedure be performed as an inpatient.

Dr Pawa: Clearly, my aim would always be to deal with the patient's concerns and deliver the safest and most appropriate anesthetic. I would establish which techniques had been used before and why they had been ineffective. If I was sure that she had understood the risks involved and this was clearly documented, I would carefully perform a catheter technique. The only additional advantage of a catheter technique in this context is that there has been at least one case report where postoperative compromising phrenic nerve palsy was reversed by administration of saline via the interscalene catheter.

Following placement of an interscalene catheter, negative test dose, and catheter dosing with 10 mL 0.5% bupivacaine, the patient is brought to the operating theater. The patient assumes a fully supine position while transferring to the operating room table and describes significant chest heaviness.

What is in your differential diagnosis?

Dr Auyong: The differential diagnosis is wide ranging and includes cardiac, pulmonary, and neurologic issues. Top on the differential is hemidiaphragm paralysis from phrenic nerve impairment. As previously indicated, an interscalene catheter was placed and dosed with a long-acting local anesthetic. Based on the time frame to the onset of symptoms and the patient's position, I would be most concerned about diaphragm paralysis.

Dr Maniker: Highest on the differential would be symptomatic phrenic block. Other possibilities include myocardial infarction, pulmonary embolism, anxiety, pneumothorax, and gastroesophageal reflux disease.

Dr Harrington: The differential would include unilateral phrenic nerve paresis, symptomatic coronary artery disease, and pneumothorax.

Dr Pawa: In this scenario, the differentials would be cardiac chest pain, phrenic nerve palsy, pneumothorax, and intrathecal spread of local anesthetic.

a


Dr Amit Pawa
@amit_pawa
#ASRAPBLD Q4 After ISB cath,-ve test dose & 10 ml 0.5% bupi, pt describes significant chest heaviness. Differential Diagnosis? Free txt ans
26/08/2017, 01:24 from Greenwich, London
View Tweet activity
3 Retweets 3 Likes
James Stimpson @j... · 27/08/2017
Replying to @amit_pawa
Catheter malposition and too much volume / concn. ? IT / epid/

b


James Stimpson @j... · 27/08/2017
Replying to @amit_pawa
Catheter malposition and too much volume / concn. ? IT / epid/ anterior (Pn) / post (LTn). Intra or post-op? ?PTx. ? Surg emph? Surg Fluid?
Dr Amit Pawa @ami... · 27/08/2017
Thx J. This was pre-op asra.com/news/173/probl... #ASRAPBLD
Husni Alakkad @Hu... · 27/08/2017
Replying to @amit_pawa
Have you considered doing diaphragmatic ultrasound?
Dr Amit Pawa @ami... · 27/08/2017
Certainly could do, but the question is designed to generate discussion as to all potential

c


Dr Amit Pawa @ami... · 27/08/2017
Certainly could do, but the question is designed to generate discussion as to all potential causes. US could help differentiate tho
Galal Gargodhi, MD · 26/08/2017
Replying to @amit_pawa
you are looking for trouble lately amit
Galal Gargodhi, MD · 26/08/2017
Replying to @amit_pawa
1- IT /epi injection(don't trust TD 2- phrenic nerve block 3- LAST 4- allergic react 5-pnemo 6- if u r creative vert art inject or RLN block

d


risk based on her Hx:
PE
Acute MI
Bronchospasm
Henry Hammerbeck · 26/08/2017
Good PBL. Thanks.
Dr. Guy Weinberg ... · 26/08/2017
Replying to @amit_pawa
Chest heaviness is v scary here. Phrenic palsey usually = air starvation or dyspnea. I'm concerned about IV injection of the epi in the LA.
chris smith @smith... · 26/08/2017
Replying to @amit_pawa
#ASRAPBLD...hemidiaphragm paralysis

Is there anything you could have changed regarding this patient's care that may have reduced the probability of this outcome?

Dr Auyong: These are the things I would have done rather than placing an interscalene catheter with bupivacaine: (1) anterior approach suprascapular catheter, (2) dosing of the catheter with short-acting local anesthetic (lidocaine or chloroprocaine), (3) small-volume, intermittent dosing of catheter (<5 mL).

Dr Maniker: Interscalene block could have been avoided to prevent phrenic nerve blockade.

Dr Harrington: Although it may not have made any difference, I would not use a high concentration of local anesthetic (0.25% bupivacaine would probably be as effective as 0.5%). Furthermore, I would use a shorter-acting local anesthetic agent, such as mepivacaine or lidocaine, so that if severe pulmonary compromise ensues, it will be shorter lived.

Dr Pawa: Potentially, I could have used a slow, incremental loading of the catheter with a lower concentration of local anesthetic (assuming regional anaesthesia was being used as the sole mode of anesthesia). If the interscalene catheter was being used for analgesia only, I would avoid a bolus dose and start the local anesthetic infusion alone without bolus.

An electrocardiogram and chest x-ray fail to demonstrate any significant abnormalities other than an elevated left hemidiaphragm. The patient is more comfortable with the head of the bed elevated and with the provision of supplemental oxygen.

Would you proceed and induce general anesthesia?

Dr Auyong: Yes, I would proceed with induction of a general anesthetic. Hemidiaphragmatic paralysis is a known side effect of brachial plexus regional anesthesia. If the patient was clinically unstable, I would consider an infusion or bolus of saline through the catheter to dilute the local anesthetic already delivered to help decrease the duration and severity of the side effects related to the block.

Dr Maniker: If the patient was hemodynamically stable and oxygenating appropriately, I would proceed.

Dr Harrington: Yes. Although I generally do these cases with a laryngeal mask airway, I would intubate this patient.

Dr Pawa: If the patient was expecting awake surgery, and assuming the block is effective, I would attempt the use of a CPAP mask or of high-flow, humidified nasal oxygen, and proceed. If the patient was expecting a general anesthetic, I would induce anesthesia.

The surgical procedure is uncomplicated, and no additional intraoperative opioids are required. At the conclusion of the case, the patient is extubated and transferred to the postanesthesia care unit, where her oxygen saturation is noted to be 88% on 2 L nasal cannula. The patient is asymptomatic, but her oxygen saturation fails to improve over the course of 3 hours.

The procedure was planned to be performed on an ambulatory basis. With the removal of supplemental oxygen, the patient's oxygen saturation falls to 86%.

Would you be comfortable discharging the patient home?

Dr Auyong: First, I would check the patient's preoperative oxygenation. Next, if these oxygen saturation values are significantly lower than her preoperative baseline, I would give the patient some time, incentive spirometry, and better positioning (sitting upright or standing) to improve her oxygenation. However, in the setting of this patient's multiple comorbidities and lack of improvement in her postoperative course with time, I would recommend the patient be admitted overnight.

Dr Maniker: This depends on the patient's baseline oxygen saturation. If it is close to baseline, I would recommend temporarily increasing the supplemental oxygen and discharge with close watch by a family member or caretaker. If this is a significant change from the patient's baseline and does not improve with increased supplemental oxygen, I would have the patient admitted for observation overnight.

Dr Harrington: No. As previously stated, I would not be comfortable doing this case at an ambulatory center if an interscalene block was planned.

Dr Pawa: No.

Would you be comfortable initiating an infusion of local anesthetic through the interscalene catheter?

Dr Auyong: No, because bupivacaine was already dosed and no additional opioids have been required, I would not elect to initiate a continuous infusion of local anesthetic via the interscalene catheter at this time. Because the patient has hemidiaphragmatic paralysis, additional dosing may delay her improvement in pulmonary function and postpone her discharge further. Continuous infusion of local anesthetic is similarly associated with phrenic nerve impairment as a single-injection bolus at the interscalene level. I would, however, leave the catheter intact for future dosing.

Dr Maniker: No. Interscalene catheters have been associated with phrenic block and respiratory events, even if the initial bolus doses did not result in symptomatic pulmonary compromise.

Dr Harrington: At this point, the catheter appears to be functioning well. Although I would usually initiate a low-volume infusion for a case like this, in this patient I would not start a baseline infusion but would prefer to first try a patient-controlled intermittent bolus technique (4 mL 0.2% ropivacaine with a 60-minute lockout).

Dr Pawa: I would leave the catheter in situ and only cautiously commence an infusion, or administer a low-volume bolus if pain became an issue overnight. This patient would have continued administration of low-flow oxygen and vital signs measurement monitoring throughout.

If yes, what would your infusion strategy be?

Dr Auyong: Options for infusion would be (1) intermittent, low-volume bolusing of the interscalene catheter as needed (no continuous rate), (2) infusion of chloroprocaine so any clinical symptoms of phrenic paralysis would be short lived, or (3) replacing the interscalene catheter with a more distal brachial plexus approach such as a suprascapular catheter.

Dr Maniker: If the catheter was used, the infusion should be initiated with very low volume and in a well-monitored setting.

Dr Harrington: As before: no baseline infusion with a patient-controlled intermittent bolus (4 mL 0.2% ropivacaine with a 60-minute lockout). Continue multimodal therapy (acetaminophen plus gabapentin) on a scheduled basis, with oxycodone available PRN. If this approach was inadequate, I would begin a low-volume infusion of ropivacaine (4 mL/hr) on top of the patient-controlled intermittent bolus.

Dr Pawa: I would use infusion of a low-volume, low-concentration solution such as 0.125% bupivacaine or 0.2% ropivacaine at 4–5 mL/hr.

A low-volume infusion of 0.2% ropivacaine at 4 mL/hr is initiated, and the patient is transferred to the floor for observation and supplemental oxygen administration. The following day, the patient's oxygen saturation has now normalized and she is prepared for discharge. The surgeon, after further discussions with cardiology, would like to restart clopidogrel therapy immediately.

Do you have any concerns sending a patient home with an interscalene catheter while on clopidogrel? Warfarin? Low molecular-weight heparin (LMWH)? If you have treated these differently, why?

Dr Auyong: In general, if patients are on anticoagulation, I recommend removal of continuous nerve blocks upon discharge home. However, this patient is now asymptomatic and likely receiving significant analgesic benefit from the continuous nerve block. It appears that discharging this functioning continuous block is in the best interest of the patient. I would discuss the risks, benefits, and options for analgesia with the patient and if she understood, would allow discharge home with anticoagulation. Because this is an interscalene block at a compressible area, I am less concerned about a small hematoma from the catheter remaining in place and eventually being removed. It is important that the patient and her caregiver understand the risks of going home with the nerve block while anticoagulated. If the patient had a follow-up appointment with the surgeon within the next few days, I would recommend removal of the catheter while in the surgeon's office.

Dr Maniker: I would be hesitant to send this patient home with an interscalene catheter given the risk of symptomatic phrenic paresis and the impact on pulmonary function. In addition, perineural bleeding from catheter or its removal would not be recognized and therefore I would not send the patient home with a catheter if anticoagulated with clopidogrel or warfarin.

Dr Harrington: I would like to hold clopidogrel until the catheter is removed. Prophylactic dose LMWH (40 mg/d) would be preferable and recommended. Although warfarin would be acceptable for a few days (because of its delayed effect), it doesn't appear to be indicated in this case.

Dr Pawa: I have major concerns with clopidogrel and warfarin and indwelling catheters. Once those therapies are reinstituted, intentional or unintentional catheter removal could be problematic.

Prophylactic LMWH is a once-a-day therapy, and at least planned catheter removal can be carefully planned 12 hours after last dose.

Following a discussion with the surgeon and cardiologist, the decision is made to send the patient home with aspirin and LMWH therapy until the interscalene catheter is removed. The patient lives three blocks from the hospital, and the patient's daughter is an internist who vows to monitor the catheter site closely for any signs of bleeding. The patient is sent home with an indwelling interscalene catheter. Following a successful 3-day ambulatory infusion, it is now time for the interscalene catheter to be removed.

What steps or precautions do you normally take at the time of peripheral nerve catheter removal (eg, pausing infusion, family member to assist with removal, coached on phone)?

Dr Auyong: Normally, we give instructions for catheter removal preoperatively and in the recovery room. Additionally, we call the

patient daily as long as the continuous nerve catheter is in place. After the continuous infusion is complete, we typically have the patient's caregiver pull the catheter at home. If, however, the patient is uncomfortable performing catheter removal at home, we offer to talk the patient through the procedure on the phone or have the catheter removed in the surgeon's office during the follow-up visit.

Dr Maniker: Patients are instructed prior to hospital discharge and over the phone about catheter removal as well as monitoring after removal. The patient is instructed to contact the service if any evidence of bleeding, swelling, significant erythema, or paresthesia develops or if the denseness of numbness increases over time.

Dr Harrington: Normally, patients can remove the catheter themselves after the home infusion is complete. It would not be uncommon to have a patient who lives this close return to the hospital for catheter removal. In this particular case, the patient's daughter would ideally remove the catheter, if she's comfortable.

Dr Pawa: I do not send patients home with ambulatory catheters in my current practice, and so my answers to this would not be based on my experience. I would be more comfortable with coached removal over the phone that was assisted by a family member (assuming adequate preoperative training).

Does the presence of LMWH therapy alter your planning?

Dr Auyong: I would recommend pulling the continuous catheter during the trough, prior to the next dose of LMWH. I would instruct the patient to apply pressure if bleeding starts or persists at the catheter insertion site. If the patient is uncomfortable with pulling the catheter at home, I would suggest having the catheter pulled in the surgeon's office at the follow-up visit.

Dr Maniker: In this case, given that the daughter is a physician, I would feel comfortable with the daughter assisting in catheter removal and monitoring the site afterward.

Dr Harrington: Removal of the catheter should be timed to be no sooner than 10–12 hours after the last dose of LMWH.

Dr Pawa: Yes, assuming a once-daily dosing, I would want the catheter removal to be at least 12 hours after last dose.

Resistance is encountered with attempted catheter removal.

What steps do you take when resistance is encountered with attempted catheter removal, and how do you plan to remove it?

Dr Auyong: If resistance is encountered, we have patients change position and try pulling the catheter again. Often a simple position change assists in helping the catheter slide out. If the catheter continues to have resistance, we offer to have the patient present to us for catheter removal or to have the catheter removed during their follow-up appointment in the surgery clinic. If the catheter was indeed stuck, I would ensure a member of the peripheral nerve catheter team was present at the surgical appointment to assist in the catheter removal.

Dr Maniker: Gentle continuous traction is first applied. Next, arm movements such as abduction as well as neck flection and extension can be attempted while providing gentle catheter traction. Next, a small amount (3–5 mL) of preservative-free sterile normal saline can be injected through the catheter, which has been reported to aid in removal of peripheral nerve catheters. This can also be performed under ultrasound to visualize the catheter trajectory. Unfortunately, catheters can become knotted and in rare cases may require surgical removal.

Dr Harrington: Although catheters can usually be removed by patients at home, if any resistance is encountered, the catheter should be removed only by anesthesia personnel. Steady tension on the catheter is advised.

Dr Pawa: In this instance, I would advise the patient to return to the hospital and aim to use ultrasound or x-ray to determine whether the catheter had kinked or knotted. If no knotting or kinking were found, I would apply continuous steady traction.

How would this be altered if the patient complained of paresthesias with attempted catheter removal?

Dr Auyong: If paresthesias were encountered during catheter removal, I would not allow further traction or pulling on the continuous catheter. I would have the patient present in person for evaluation by myself and the peripheral nerve catheter team. My evaluation would entail a physical exam and ultrasound exam of the brachial plexus (with and without traction on the catheter). If paresthesias persisted without the ability to remove the catheter, I would consult my neurosurgical colleagues for further evaluation and possible surgical removal.

Dr Maniker: I would stop traction on the catheter. The catheter could be knotted and either wrapped around a nerve or positioned in a way that contacts or compresses a nerve. Patient positioning could be changed with very gentle traction, which is again stopped with any patient report of paresthesia. As previously mentioned, ultrasound could be used to define the course of the catheter, but surgical consultation may be needed to remove the catheter.

Dr Harrington: In that case, I would be concerned about knotting. I would try to visualize the catheter by injecting a

small volume of contrast material under fluoroscopic guidance. Imaging would provide guidance as to the safest means of removing the catheter.

Dr Pawa: I would cease pulling and ask for a surgical opinion.

How would you proceed if you attempted to remove the catheter and you think you left a portion behind in the patient?

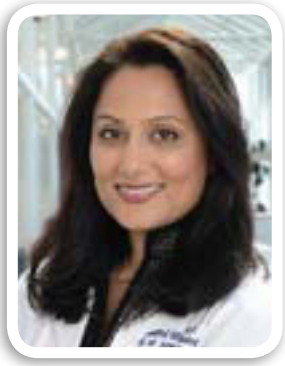
Dr Auyong: If a portion of the catheter was left behind in the patient, I would first and foremost inform the patient. If asymptomatic, I would reassure the patient that retained foreign bodies are generally not removed surgically. I would examine the patient with ultrasound to see if I could locate any part of the catheter and document in the patient's medical record. Unless the retained catheter caused symptoms, I would not push forward with any further evaluation.

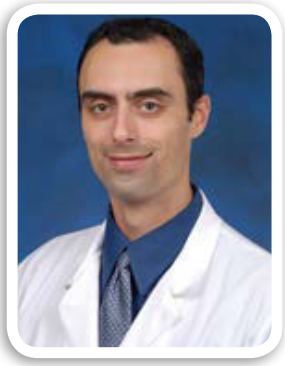
Dr Maniker: I would obtain imaging (bedside ultrasound and possible computed tomography) and consider obtaining a surgery consult for exploration and removal.

Dr Harrington: A surgical consult would be obtained for open removal of the broken fragment.

Dr Pawa: I would organize imaging and a consultation with my surgical colleagues, with a view to facilitate surgical removal if required.

2016 Winner of the ASRA Chronic Pain Grant: Effectiveness of OnabotulinumtoxinA (Botox®) in Pediatric Patients Experiencing Migraines: A Randomized, Double-Blinded, Placebo Crossover Study in the Pediatric Pain Population





Principle Investigator:
Shalini Shah, MD
Assistant Clinical Professor

Faculty Mentor:
Joseph Rinehart, MD
Associate Clinical Professor

Department of Anesthesiology and Perioperative Care
University of California
Irvine, California

While many chronic pain conditions are manageable, migraine pain can be devastating. Migraine episodes are unpredictable in onset and duration and profoundly debilitating for sufferers. Recently, onabotulinumtoxinA (OBTA) was approved for the prophylaxis of adult migraine symptoms by the United States Food and Drug Administration (FDA), which has dramatically altered the way pain physicians approach migraine pain.

Most adults who suffer with migraines have their first headache during childhood or adolescence.[1] Although many preventative agents appear to be safe for use in children, none are currently FDA approved for that age group (apart from topiramate, which achieved on-label status in 2014). As a result, despite experiencing significant disability, the vast majority of children who present to their physician with migraine headaches do not receive prophylactic therapy.[2] A 2003 study published in *JAMA* found that health care costs, work-related disability for parents, and lost educational opportunities for children lead to an annual economic impact in the United States of approximately $36 billion, because of both direct medical costs and lost productivity into adulthood.[3]

Thus, treatment for pediatric head pain is an extrapolation from all that we have learned about adult headaches combined with what we have learned from working with children in pain. Pediatric pain medicine historically has its own challenges, largely suffering from underassessment and treatment paradigms extrapolated from adult literature that may not work as well in the pediatric population. Current clinical studies, when able to demonstrate efficacy, may not demonstrate safety, or vice versa. In the case of pediatric migraines, the best current treatment recommends involves nonsteroidal anti-inflammatory drugs, acetaminophen, and antiemetics.

"Treatment for pediatric head pain is an extrapolation from all that we have learned about adult headaches combined with what we have learned from working with children in pain."

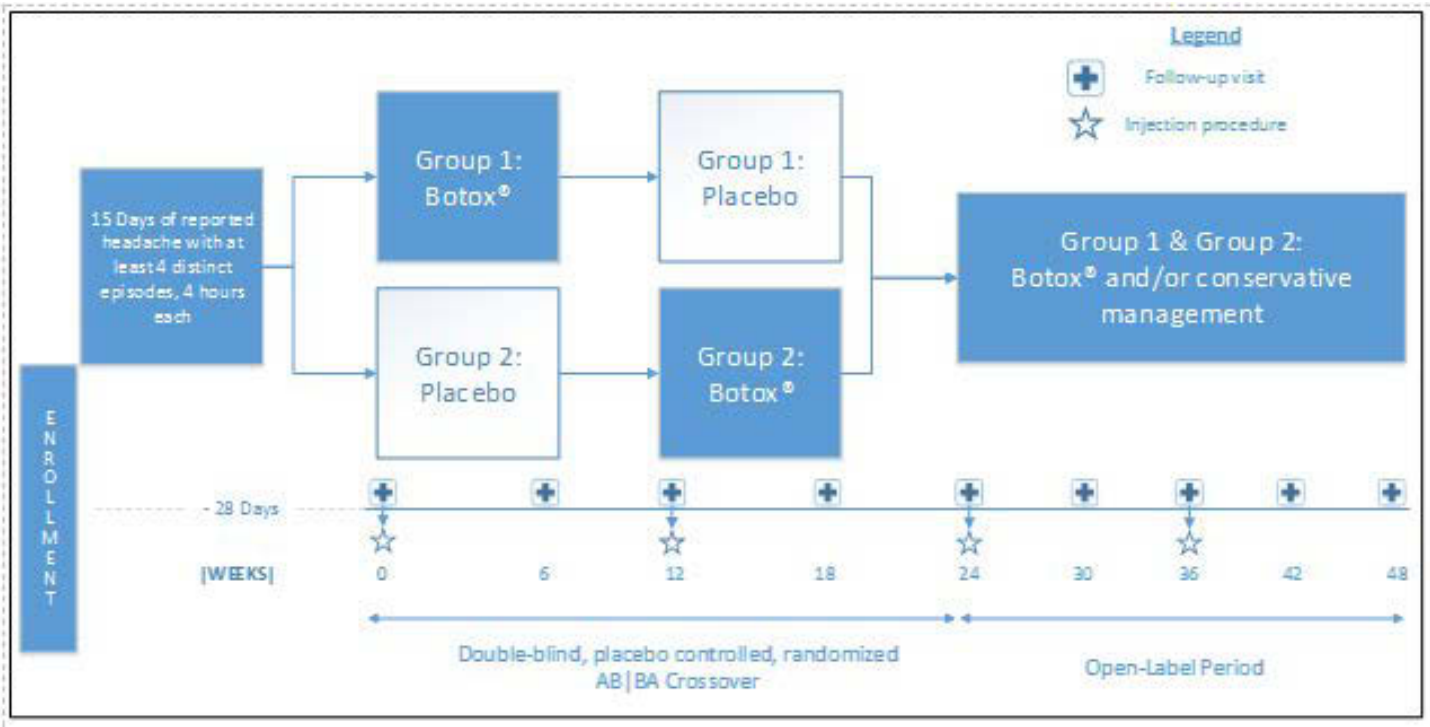
The significance of this study is to evaluate the efficacy of OBTA (sold commercially as Botox®) for the treatment and prophylaxis of pediatric migraine in a randomized, double-blinded, placebo crossover study. No trials currently exist in literature studying OBTA for efficacy and/or safety for indication of pediatric migraine, although significant contributions have been made by retrospective case series over the past 10 years.[2,7–10] Additional historical and longitudinal interest for the design of this study comes from the principal investigator's (PI) extensive use of off-label OBTA to treat refractory pediatric migraine over the past five years.

We had impressive anecdotal evidence: Patients who presented with refractory migraines who were treated with OBTA in the PI's clinical practice saw a substantial reduction in migraine days and migraine duration. In some cases, this meant a significant improvement in school attendance and daily functional status, and the effect seemed to persist over longitudinal treatments. In preparation for proposal of the prospective study, we went back and more formally assessed the treatment effect of OBTA for refractory pediatric migraine and found strong evidence in support of its use; the manuscript based on this work is currently under review for publication.

Looking at our historical off-label data, we realized we had an ideal candidate to investigate for ASRA's goal of identification of novel applications of existing therapeutics. Moreover, the findings from this trial may benefit an understudied pain population with the long-term aim of obtaining a new FDA indication to "on-label" status. The proposal also carries policy and legislative impact by fulfilling the federal initiative to design and conduct trials in the pediatric pain population within the confines of the Best Pharmaceuticals for Children Act (BPCA) of 2002. The goal of the BPCA program is to improve pediatric therapeutics through preclinical and clinical drug trials that lead to drug labeling changes.

Because we intended to formally study an off-label use of OBTA, we had to submit an Investigational New Drug (IND) application to the FDA prior to institutional review board (IRB) approval. The FDA was very responsive to our application and, after some minor revisions, approved our application. However, we then discovered

Figure 1: *Study design.*



that botulinum toxin is classified as an agent of chemical warfare in the United States and labeled as a bioterrorism agent. Because of this, the National Institutes of Health confirmed that our study falls under the United States Government Policy for Institutional Oversight of Life Sciences Dual Use Research of Concern (DURC), and we were subjected to additional review to confirm that our study is not a threat to national security. Finally, on March 1, 2017, our IRB allowed us to proceed under protocol HS 2016-3108.

Applications of botulinum toxin A have been shown to be generally safe in the pediatric population for indications—such as localized or segmental spasticity disorders, bladder hypertonicity, and vestibular migraine in patients as young as 2 years of age, although the majority of the class I and II studies included abobotulinumtoxinA (trade name Dysport®).[4] With the encouraging data presented at the 14th Congress of the International Headache Society (held in 2009 in Philadelphia, PA) for adults with chronic migraines, the PREEMPT Data,[5,6] and the experiences of several retrospective case series, further exploration is reasonable for the potential role for OBTA in the management of chronic migraine in the pediatric population.

The study itself has three specific aims:

Aim 1: To test whether OBTA is superior to placebo in reducing headache frequency, intensity, and pediatric migraine-related disability (efficacy).

Aim 2: To evaluate the incidence of adverse events of OBTA administration in children ages 8–17 (safety, tolerability).

Aim 3: To evaluate whether OBTA can contribute to reduction in preventive and rescue medication, emergency room and hospital admissions, and health care costs (hospital and pharmacy resource utilization).

The study's goal is to provide an overall framework so that primary and secondary outcomes are easily defined, measurable, and validated as an acceptable means to gauge clinical success and longitudinally assess response.

Of note, given the existing evidence of efficacy in available scientific data (adult and retrospective pediatric) and personal experience in the pediatric population, the PI did not consider it reasonable to prolong withholding of OBTA for the purpose of study. Thus, a desirable study design minimized the placebo control period while still allowing for comparison of OBTA to a control group. After consideration of the alternatives, an AB/BA crossover design was selected as the best option. The influence of confounding covariates was reduced because each crossover patient served as his or her own control. A 4-week baseline prior to treatment would act as a no-treatment control in comparison to the treatment and placebo. In an attempt to demonstrate superiority of the study drug over conservative medical management, we did not exclude patients on preventive or abortive

migraine medication. Following the crossover period, all patients will proceed to an open-label treatment phase with OBTA.

FUTURE DIRECTIONS

We have begun our journey to deliver better relief to children in pain, and we, as investigators and physicians, have gathered new insight into the success of nontraditional thinking for sustainable pain relief in this vulnerable population. More specifically, we discovered that the general pediatric community as well as patients and families welcomed this experimental and investigational option more warmly than traditional methods of treating migraine pain—reminding us why we physicians continually pursue research so enthusiastically. We learned (through a very arduous process) that performing clinical trials in the pediatric population—even more so in the pediatric *pain* population—is trenched with bureaucratic protocols and processes; however, based on our early data, it was well worth the struggle. Ultimately, our overriding rationale is to demonstrate efficacy, tolerability, and safety of OBTA for pediatric migraine, thereby potentially hastening the lengthy process to evaluate OBTA for approval in the pediatric population. The most urgent goals for pharmaceutical innovation are the development of pathomechanism-based antimigraine drugs and personalized therapy tailored to children and adolescents experiencing migraines.

ACKNOWLEDGMENTS

We wholeheartedly thank ASRA and the Committee on Research for its generous support, guidance, and enthusiasm in supporting the study of migraine pain in children. We thank our team for its endless commitment and late-night meetings, as well as our patients for taking the journey with us towards discovery. We hope our work will be a springboard for future physicians to develop a contribution to pediatric migraine, and we welcome all opportunities for collaboration.

REFERENCES

1. Jacobs H, Gladstein J. Pediatric headache: a clinical review. Headache 2012;52(2):333–339. doi: 10.1111/j.1526-4610.2011.02086.x
2. Hershey AD, Powers SW, Coffey CS, Eklund DD, Chamberlin LA, Korbee LL. Childhood and Adolescent Migraine Prevention (CHAMP) study: a double-blinded, placebo-controlled, comparative effectiveness study of amitriptyline, topiramate, and placebo in the prevention of childhood and adolescent migraine. Headache 2013;53(5):799–816. doi: 10.1111/head.12105
3. Stewart WF, Ricci JA, Chee E, Morganstein D, Lipton R. Lost productive time and cost due to common pain conditions in the US workforce. JAMA 2003;290:2443–2454. doi: 10.1001/jama.290.18.2443
4. Delgado MR, Hertz D, Aisne M, et al. Practice parameter: pharmacologic treatment of spasticity in children and adolescents with cerebral palsy (an evidence-based review): report of the Quality Standards Subcommittee of the American Academy of Neurology and the Practice Committee of the Child Neurology Society. Neurology 2010;74:336–343. doi: 10.1212/WNL.0b013e3181cbcd2f
5. Aurora SK, Winner P, Freeman MC, et al. OnabotulinumtoxinA for treatment of chronic migraine: pooled analyses of the 56-week PREEMPT clinical program. Headache 2011;51(9):1358–1373. doi: 10.1111/j.1526-4610.2011.01990.x
6. Dodick DW, Turkel CC, DeGryse RE, et al. OnabotulinumtoxinA for treatment of chronic migraine: pooled results from the double-blind, randomized, placebo-controlled phases of the PREEMPT clinical program. Headache 2010;50(6):921–936. doi: 10.1111/j.1526-4610.2010.01678.x
7. Ahmed K, Oas KH, Mack KJ, Garza I. Experience with botulinumtoxin type A in medically intractable pediatric chronic daily headache. Pediatr Neurol 2010;43:316–319. doi: 10.1016/j.pediatrneurol.2010.06.001
8. Chan V, McCabe EJ, MacGregor, DL. Botox treatment for migraine and chronic daily headache in adolescents. J Neurosci Nurs 2009;41(5):235–243.
9. Kabbouche M, O'Brien H, Hershey, AD. OnabotulinumtoxinA in pediatric chronic daily headache. Curr Neurol Neurosci Rep 2012;12(2):114–117. doi: 10.1007/s11910-012-0251-1
10. Schroeder AS, Huss K, Blaschek A, et al. Ten-year follow-up in a case series of integrative botulinum toxin intervention in adolescents with chronic daily headache and associated muscle pain. Neuropediatrics 2012;43(6):339–345. doi: 10.1055/s-0032-1329612

Urine Drug Screens for Opioid Maintenance: Is It That Simple?

Geeta Nagpal, MD
Program Director, Multidisciplinary Pain Medicine Fellowship
Assistant Professor, Department of Anesthesiology
Northwestern University, Feinberg School of Medicine
Evanston, Illinois

Section Editor: Lynn Kohan

CASE PRESENTATION

A 54-year-old male presented to the pain medicine center for evaluation of chronic neck pain and transfer of medication management. He was taking oxymorphone extended-release (ER) tablets 10 mg twice a day and one to two tablets of oxycodone 10 mg per day. The state prescription monitoring program confirmed his prescriptions of 60 tablets of oxymorphone ER 10 mg and 150 tablets of oxycodone 10 mg, filled monthly. His last prescription was filled 25 days prior. When asked about how much remaining oxycodone he has, he replied that he only had a few tabs left. He added that he had taken one tab of oxymorphone and one tab of oxycodone in the morning prior to his arrival at the clinic. A urine sample was taken, and the results are reported in the Table.

He is MOST likely taking which of the following?

A. He is taking oxymorphone and oxycodone.
B. He is taking oxymorphone, oxycodone, and morphine.
C. He is taking oxymorphone, oxycodone, and heroin.
D. He is taking oxymorphone.

> "A great man once said that the true symbol of the United States is not the bald eagle. It is the pendulum. And when the pendulum swings too far in one direction, it will go back."
>
> – Ruth Bader Ginsberg

Nearly one-third of the American population has experienced or is living in chronic pain, defined as pain that is persistent and lasts more than 3 to 6 months. Over the past several decades, the development of opioid medications for the treatment of pain has increased dramatically. With this increase, we have seen yearly prescriptions of opioids catapult from 76 million to greater than 250 million over a 20-year period,[1] which, unfortunately, is directly correlated with an increase in opioid abuse.[2] In response to this increase in opioid maintenance, dependence, and addiction, the Centers for Disease Control and Prevention (CDC) published guidelines for the prescribing of opioids for chronic pain in March 2016.[3]

With the growing epidemic, providers must effectively monitor the use of prescription opioids to identify misuse, addiction, and diversion. Some examples of the tools available include state prescription drug monitoring programs and urine drug testing. There are two fundamental questions that lead a clinician to order a urine drug screen (UDS): (1) Is the patient taking the prescribed medication, and (2) is the patient abstaining from the use of nonprescribed controlled and illicit substances? The CDC suggests obtaining a UDS before the initiation of opioid treatment and to consider screening at least annually. However, the interpretation of drug testing is far less straightforward than expected, yet the ramifications can be significant. Occasionally, it can be difficult to interpret a result as normal or abnormal based on opioid compounds found in the urine. Misinterpreting results can lead to false reassurance or incorrect conclusions about medication use and abuse.

Table 1: *Lab results.*

Opiate	Qualitative lab result	Lab result (ng/mL)	Assay cutoff (ng/mL)
Opiates	**Positive**	458	50
Codeine	Negative		100
Morphine	**Positive**	252	100
Hydrocodone	Negative		100
Hydromorphone	Negative		100
Norhydrocodone	Negative		100
Oxycodone	Negative		100
Oxymorphone	**Positive**	1423	100
Noroxycodone	Negative		100

Accurate interpretation of a UDS requires knowledge of urine metabolites, specificities and sensitivities of the assay, and detection times. Some opioids produce metabolites chemically identical to another opioid, which may complicate the interpretation of the UDS. A common example is codeine, a prodrug that metabolizes to morphine in approximately 90% of Caucasian patients.[4] Interestingly, in a 2007 survey of physicians who routinely order UDSs, only 29% knew that morphine is a metabolite of codeine and should be expected on UDSs in patients taking codeine.[5] In a more recent study of knowledge and confidence in UDS interpretation of internal medicine residents, less than 30% correctly answered what the expected metabolites would be in a patient prescribed acetaminophen/codeine.[6] Unfortunately, many were confident in their incorrect response.

Incorrect conclusions may also be drawn from ordering inappropriate tests. UDSs are most commonly performed using immunoassays or mass-spectrometry. Opiate immunoassays are

relatively inexpensive (Medicare midpoint of $20.22–$107.85),[7] whereas confirmatory mass-spectrometry–based methods have a higher analytical sensitivity and specificity, incurring much higher costs (Medicare midpoint of $158.98–$343.07).[7] Clinicians must know which drugs are tested in the particular panel ordered.

In fact, the term "drug screen" is a misnomer because it suggests that it detects all drugs in a given class. For example, the common immunoassay for the detection of opiates uses an antiopioid antibody that detects morphine and will show positive if a patient is taking morphine, codeine, or heroin. The test may or may not detect semisynthetic opioids (such as hydrocodone and oxycodone) and will not detect synthetic opioids (such as buprenorphine and fentanyl). Hydrocodone is the most commonly prescribed opioid in the United States, yet the opiate screen may be considerably less sensitive for this drug. In a review of urine specimens with unexpected negative opiate immunoassay results in hydrocodone users, 72.3% were found to be positive for hydrocodone or its metabolite using confirmatory testing.[8]

"With the growing epidemic, providers must effectively monitor the use of prescription opioids to identify misuse, addiction, and diversion."

For the patient presented, the mass-spectrometry screen was positive for oxymorphone and negative for oxycodone. Oxymorphone is a metabolite of oxycodone and is expected in the UDS of a patient taking oxycodone, although the opposite is not true; that is, this patient is taking oxymorphone and not taking the oxycodone as he stated. He was prescribed 150 tabs per month, states he takes one to two per day, and has only a few tabs left. The numbers and the UDS do not add up, raising the suspicion for misuse or diversion. In addition, the morphine screen is positive. The common reflex is to assume he is taking nonprescribed or illicit substances; the level of morphine detected is consistent with the use of morphine, heroin, or ingestion of poppy seeds. In this case, accusing the patient of using nonprescribed or illicit substances may be wrong—unfortunately, there is no way to differentiate. Because of the oxycodone discrepancy, the clinical decision was to wean opioids.

For the same patient, if a basic opiate immunoassay screen were done, the interpretation of the results may have resulted in a very different outcome. The benefit of the opiate immunoassay is that it is rapid, sensitive, widely available, and relatively inexpensive. A major disadvantage is that semisynthetic opioids may not be detected, making the interpretation of compliance nearly impossible. The opiate immunoassays perform very well when compared to confirmatory screens in evaluating morphine; however, the cross-reactivity varies among manufacturers for oxycodone and oxymorphone.[9]

In this case, the opiate may have been positive because of the presence of morphine or because of the minor cross-reactivity with the semisynthetic medications. The test would have come back as positive, giving no insight that the patient was not taking oxycodone. There are immunoassays designed to detect specific semisynthetic opioids, but those are not typically included in the basic screens. The immunoassay specifically for oxycodone and its metabolite, oxymorphone, has a sensitivity and specificity of approximately 99%.[9]

In the United States, we are enveloped in a crisis where overdose from opioids is the leading cause of accidental death.[10] Although we hope to see a decrease in prescribing opioids, it is important that we do not let the pendulum swing back to the practice of reserving opioids for end-of-life care. It is essential for physicians who prescribe opioids to monitor their patients closely and identify signs and symptoms of misuse and abuse. UDSs give unbiased and reproducible objective data and are an important tool in the setting of addiction and pain management. Although numerous guidelines recommend UDSs for pain management patients as a tool to monitor compliance, there is a lack of specific recommendations for which to order and at what frequency. Therefore, clinicians must understand the capabilities and limitations of assays performed to prevent incorrect interpretation.

Answer: D

REFERENCES

1. Volkow ND. America's addiction to opioids: heroin and prescription drug abuse. The National Institute on Drug Abuse: Senate Caucus on International Narcotics Control. Available at: https://www.drugabuse.gov/about-nida/legislative-activities/testimony-to-congress/2016/americas-addiction-to-opioids-heroin-prescription-drug-abuse. Published May 14, 2014. Accessed September 8, 2017.
2. Chen KY, Chen L, Mao J. Buprenorphine-naloxone therapy in pain management. Anesthesiology 2014;120(5):1262–1274. doi: 10.1097/ALN.0000000000000170
3. Dowell D, Haegerich TM, Chou R. CDC guideline for prescribing opioids for chronic pain—United States, 2016. JAMA 2016;315(15):1624–1645. doi:10.1001/jama.2016.1464
4. Tenore P. Advanced urine toxicology testing. Journal of Addictive Diseases 2010;29(4):436–448. doi: 10.1080/10550887.2010.509277
5. Reisfield G, Bertholf R, Barkin R, Webb F, Wilson G. Urine drug test interpretation: what do physicians know? J Opioid Manag 2007;3(2):80–86.
6. Starrels JL, Fox AD, Kunins HV, Cunningham CO. They don't know what they don't know: internal medicine residents' knowledge and confidence in urine drug test interpretation for patients with chronic pain. J Gen Intern Med 2012;27(11):1521–1527. doi: 10.1007/s11606-012-2165-7
7. Centers for Medicare and Medicaid Services. Clinical laboratory fee schedule. Available at: https://www.cms.gov/Medicare/Medicare-Fee-for-Service-Payment/ClinicalLabFeeSched/index.html. Accessed June 15, 2017.

8. Bertholf R, Johannsen L, Reisfield G. Sensitivity of an opiate immunoassay for detecting hydrocodone and hydromorphone in urine from a clinical population: analysis of subthreshold results. J Anal Toxicol 2015;39(1):24–28. doi: 10.1093/jat/bku109
9. National Academy of Clinical Biochemistry. Laboratory Medicine Practice Guidelines. Using clinical laboratory tests to monitor drug therapy in pain management patients. Available at: https://www.aacc.org/~/media/practice-guidelines/pain-management/rough-draft-pain-management-lmpg-v6aacc.pdf. Accessed June 7, 2017.
10. Centers for Disease Control and Prevention, National Center for Health Statistics. Multiple cause of death 1999–2015. Available at: https://wonder.cdc.gov/wonder/help/mcd.html. Accessed September 8, 2017.

A Review of Pain Management in the Intensive Care Unit

Jagan Ramamoorthy, MD
Assistant Professor
Medical Director of
Cardiothoracic ICU
Department of Anesthesiology
University of Wisconsin, Madison
Madison, Wisconsin

Noreen E. Murphy, MD
Fellow in Critical Care Medicine
University of Wisconsin, Madison
Madison, Wisconsin

Section Editor: Kristopher Schroeder

Pain in critically ill patients is often underdiagnosed and undertreated. In this population, there are many potential barriers to pain recognition and management. Untreated and undertreated pain is distressing for patients, family members, and caregivers; in addition, neglected pain may contribute to increased morbidity and mortality.

Assessment of pain in the intensive care unit (ICU) can be difficult; many critically ill patients cannot communicate their discomfort because of intubation, sedation, or cognitive impairment. However, in its "Clinical Practice Guidelines for the Management of Pain, Agitation and Delirium in Adult Patients in the Intensive Care Unit," the Society of Critical Care Medicine (SCCM) recommends that pain be routinely monitored in all adult ICU patients.

Unfortunately, it is difficult to estimate the incidence of pain in critically ill patients because pain assessment tools and protocols for the management of pain are rarely applied. A Canadian study of 51 ICUs found that less than 20% of ICUs used pain assessment tools and only 25% of ICUs used pain protocols. A separate multicenter observational study found that 90% of patients in the ICU were being actively treated with opioids whereas only 42% had undergone a pain assessment. Similarly, Payen et al reported that pain was not assessed in 53% of patients who were receiving analgesia, and when pain was assessed, specific pain tools were used only 28% of the time.[1–3]

"It is difficult to estimate the incidence of pain in critically ill patients because pain assessment tools and protocols for the management of pain are rarely applied."

However, studies have aimed to quantify the incidence of pain in critically ill patients. We know from prospective descriptive studies that the presence of an endotracheal tube has been reported as a constant source of discomfort at rest and that routine procedures—such as tracheal suctioning, position changes, and line removal—cause pain.[4] One study suggested that pain is frequent with an incidence of 50% in medical and surgical patients at rest and 80% during common care procedures.[5] Another study showed similar results when patients recently discharged from the ICU were interviewed about their pain during hospitalization. Nearly 50% of patients reported recall of pain during their ICU stay. Fifteen percent of ICU patients reported extremely severe pain or moderately severe pain occurring at least half the time. Not surprisingly, nearly 15% of patients were dissatisfied with pain control during their ICU stay.[6] Another study showed that 63% of patients received no analgesics before or during painful procedures.[7]

WHY SHOULD WE CARE?

In the article "Pain Management: A Fundamental Human Right," Brenan et al wrote, "Unreasonable failure to treat pain is viewed worldwide as poor medicine, unethical practice, and an abrogation of a fundamental human right."[8] Faber-Langendoen et al wrote, "Many believe the obligation of clinicians to tend to patients' suffering is the essence of the medical profession." In addition to the ethics of pain management, medical outcomes are improved when pain is optimally managed.[9]

Pain assessment in patients on mechanical ventilation has been independently associated with a decrease in hypnotic drug dosing, duration of mechanical ventilation, and duration of ICU stay.[10] Pain contributes to hypoventilation and reduced cough, which increases atelectasis and sputum retention. These mechanisms are thought to be responsible for the increased rate of ventilator-associated pneumonia (VAP) in patients who are not routinely assessed for pain. Payen et al demonstrated decreased risk of VAP in patients routinely assessed and treated for pain.[10] Chanques et al validated those findings when they reported significantly decreased risk of VAP and duration of mechanical ventilation when pain was routinely assessed and treated.[11] Without using validated pain assessment tools and protocols, patients in the ICU are often managed inappropriately with sedation medications. Continuous sedation, titrated to a light level and with daily sedation interruptions, has been associated with an increased duration of mechanical

Table 1: *Critical care pain observation tool.*

Behavior	Patient response	Score
Compliance with ventilator	Tolerating ventilator	0
	Coughing but tolerating	+1
	Fighting ventilator	+2
Facial expression	Relaxed, neutral	0
	Tense	+1
	Grimacing	+2
Body movements	No movements	0
	Protection	+1
	Restlessness	+2
Muscle tension	Relaxed	0
	Tense/rigid	+1
	Very tense/rigid	+2

Table 2: *Behavioral pain score.*

Behavior	Patient response	Score
Compliance with ventilator	Tolerating ventilator	+1
	Coughing but tolerating most of the time	+2
	Fighting ventilator	+3
	Unable to control ventilation	+4
Facial expression	Relaxed, neutral	+1
	Partially tightened	+2
	Fully tightened	+3
	Grimacing	+4
Upper limb movements	No movement	+1
	Partially bent	+2
	Fully bent with finger flexion	+3
	Permanently retracted	+4

ventilation and ICU length of stay when compared to sedation-free protocols.

Less sedative medication allows for early mobilization, which in turn results in improved outcomes. In studies designed to compare standard of care versus early mobilization of ventilated patients, patients who were randomized to early mobilization had shorter ICU and hospital lengths of stay and were less likely to die or be rehospitalized in the year following their critical illness.[12,13]

Other potential acute negative effects of untreated pain include delirium, self-harm from accidental removal of lines or tubes, sympathetic activation with increased catecholamine release leading to tachycardia and increased systemic vascular resistance, increased cardiac workload leading to oxygen supply demand mismatch, and myocardial ischemia.[14,15] In addition to acute negative health effects, untreated and undertreated pain has been associated with the development of chronic medical issues, including chronic pain, long-term psychological illness, and lower quality of life.[16,17]

HOW SHOULD WE ASSESS PAIN IN THE ICU?

Two sensitive and validated measures are used to assess pain in patients unable to communicate their pain: the Critical Care Pain Observation Tool (CPOT) and the Behavioral Pain Score (BPS).

CPOT evaluates four behaviors—facial expressions, body movements, muscle tension, and compliance with the ventilator for mechanically ventilated patients or vocalization for nonintubated patients—rated on a scale of 0–2 with a total score ranging from 0–8 (see Table 1).[18]

BPS evaluates three behaviors—facial expressions, upper limb movements, and ventilator compliance—rated on a scale of 1–4 with a total score ranging from 3–12 (see Table 2).[19]

The SCCM makes numerous recommendations about treating pain in the ICU. They acknowledge the presence of pain at rest and in routine care, and they make recommendations that pain be routinely monitored using BPS or CPOT for patients who are unable to self-report.[20] They emphasize that validated scoring systems should be used to assess pain and state that changes in blood pressure and tachycardia should not be routinely used as measures for assessment of pain.[21] For more information regarding the SCCM recommendations, refer to the SCCM pain, agitation, and delirium guidelines as well as the ABCDEF bundle for prevention of postintensive care syndrome.

Table 3: *Analgesic selection and dosing in the presence of renal or hepatic impairment.*

Drug	Renal failure	Hepatic failure
Opioids	Avoid meperidine,* dextropropoxyphene Likely should avoid morphine,** hydromorphone, codeine*** Dose adjust tramadol, methadone No adjustment needed for fentanyl, oxycodone, bupernorphine	Avoid mepiridine Likely should avoid methadone Dose adjust tramadol, dextropropoxyphene No adjustment needed for fentanyl, morphine
Local anesthetics	No adjustment needed	May need to adjust dose if prolonged use
NSAIDs	Avoid in severe renal impairment	Reduce dose
Acetaminophen	No adjustment needed	Avoid or reduce dose
TCAs	Metabolite accumulation may increase risk of side effects	Not enough data
Anticonvulsants	Gabapentin should be dose adjusted based on creatinine clearance	Avoid carbamazepine, valproate
Ketamine	No adjustment needed	Not enough data

* Active metabolite normepiridine can lead to neurotoxicity.
** Active metabolites morphine-6-glucuronide (M6G) and morphine-3-glucuronide (M3G) may cause myoclonus, seizure, hyperalgesia, allodynia.
*** Prodrug of morphine can lead to M6G and M3G accumulation.

Aside from difficult assessment of pain in the critically ill patient, there are many other obstacles to pain management in the ICU patient.

1. ***Impaired Renal and/or Hepatic Clearance***
Critically ill patients often have organ failure with associated decreases in renal or hepatic clearance; thus, drug choice and dosing should be carefully considered. Table 3 reviews considerations of analgesic selection and dosing in the presence of renal or hepatic impairment.

2. ***Hemodynamic Instability***
Patients in ICUs are often hemodynamically unstable. Hypotension after the use of opioids is generally due to blunting of sympathetic responses and may unmask hypotension. For this reason, bolus doses should be administered slowly, and short-acting opioids are preferred.

3. ***Obstacles to Regional Anesthesia***
Regional anesthesia may be considered as an adjunct to decrease opioid consumption in the critically ill surgical patient. However, coagulopathy of the critically ill and anticoagulant medications should be considered carefully prior to the implementation of regional anesthesia.[22] In addition, systemic infection and positioning challenges (eg, fractures and an inability to cooperate) may preclude safe neuraxial or peripheral nerve blockade. The SCCM makes no recommendation for neuraxial/regional analgesia over systemic analgesia in medical ICU patients due to lack of evidence, but they do acknowledge thoracic epidural superiority over parenteral opioids for abdominal aortic surgery.[20]

4. ***Pharmacologic Side Effects***
Drug side effects may slow recovery, worsen patient outcomes, or create new issues. Opioids can contribute to ileus, delirium, and respiratory depression. It is generally accepted that patients with long-term exposure to high-dose opiates may develop physiologic dependence.

Intravenous opioids are first-line therapy for non-neuropathic pain. Opioids may be administered by the patient's RN on a scheduled or as-needed basis, but they may also be administered using patient-controlled analgesia (PCA), in which the patient is given the ability to self-administer pain medication. Any opioid can be administered by PCA pumps; however, meperidine is not recommended for repeat dosing because it lowers the seizure threshold and has a dysphoric effect.[23] In general, basal infusions are not recommended, but they may be appropriate for opioid-tolerant patients and select patients in the ICU. PCA may not be appropriate for a substantial portion

Figure 1: *Risk factors for the development of chronic post-ICU pain.*[a]

Severe sepsis
Acute respiratory distress syndrome (ARDS)
Surgery
Presence of preoperative pain
Prolonged ICU stay
Prolonged hospitalization
Prolonged mechanical ventilation
Post ICU depression or anxiety
Posttraumatic stress disorder (PTSD)
Use of corticosteroids
Use of nondepolarizing neuromuscular blockers

[a] Source: U.S. Pharmacist, 2016. https://www.uspharmacist.com/article/chronic-posticu-pain-and-postintensive-care-syndrome

of the intensive care population because of underlying disease processes and the systemic effects it may have on cognition.[24]

It is now recognized that long-term survivors of medical and ICUs are at high risk for developing chronic pain syndromes.[25] Risk factors for development of chronic post-ICU pain are described in Figure 1.[26]

Patients at high risk for neuropathic pain—for example, Guillain-Barre syndrome, burns, amputations, and spinal cord injury—should be considered for early administration of gabapentin and carbamazepine; however, these medications have not been shown to be consistently effective.[20,27] Burn patients receiving dressing changes should be treated with fast-acting opioids, anxiolytics, and ketamine to decrease anticipatory anxiety and development of post-traumatic stress disorder and chronic pain.[28]

Although opioids are considered first-line therapy for non-neuropathic pain, a multimodal approach to pain management may help decrease opioid requirements and thus the side effects of opioid use. Nonsteroidal anti-inflammatory drugs are effective but may be contraindicated because of the risks of gastric ulcers, bleeding, and renal dysfunction. Nonpharmacologic interventions for pain management (music therapy, relaxation) should be considered because they are generally low risk, low cost, and safe; unfortunately, there is a lack of evidence to make a strong recommendation for use.

Palliative and end-of-life pain management is also an important concern for physicians in intensive care units because 20% of patients who die in the hospital report pain and 50% of hospice patients report daily pain. Alleviation of dyspnea and pain should be the goal of drug therapies.[11]

In summary, critically ill patients routinely experience pain and are often not able to communicate this to their healthcare team. Undertreated pain can contribute negatively to both short- and long-term outcomes. Pain should be routinely assessed and treated in critically ill patients using validated assessment tools such as CPOT and BPS.

REFERENCES

1. Payen J-F, Chanques G, Mantz J, et al. Current practices in sedation and analgesia for mechanically ventilated critically ill patients: a prospective multicenter patient-based study. Anesthesiology 2007;106(4):687–695. doi: 10.1097/01.anes.0000264747.09017.da
2. Burry LD, Williamson DR, Perreault MM, et al. Analgesic, sedative, antipsychotic, and neuromuscular blocker use in Canadian intensive care units: a prospective, multicentre, observational study. Canadian Journal of Anesthesia 2014;61(7):619–630. doi: 10.1007/s12630-014-0174-1
3. Kumar AB, Brennan TJ. Pain assessment, sedation, and analgesic administration in the intensive care unit. Anesthesiology 2009;111(6):1187–1188. doi: 10.1097/ALN.0b013e3181c0d1b5
4. Siffleet J, Young J, Nikoletti S, Shaw T. Patients' self-report of procedural pain in the intensive care unit. J Clin Nurs 2007;16(11):2142–2148. doi: 10.1111/j.1365-2702.2006.01840.x
5. Chanques G, Sebbane M, Barbotte E, Viel E, Eledjam JJ, Jaber S. A prospective study of pain at rest: incidence and characteristics of an unrecognized symptom in surgical and trauma versus medical intensive care unit patients. Anesthesiology 2007;107:858–860. doi: 10.1097/01.anes.0000287211.98642.51
6. Desbiens NA, Wu AW, Broste SK, et al. Study to Understand Prognoses and Preferences for Outcomes and Risks of Treatment. Pain and satisfaction with pain control in seriously ill hospitalized adults: findings from the SUPPORT research investigations. Crit Care Med 1996;24:1953–1961.
7. Puntillo KA, Wild LR, Morris AB, et al. Practices and predictors of analgesic interventions for adults undergoing painful procedures. Am J Crit Care 2002;11(5):415–429.
8. Brennan F, Carr DB, Cousins M. Pain management: a fundamental human right. Anesth Analg 2007;105:205–221. doi: 10.1213/01.ane.0000268145.52345.55
9. Faber-Langendoen K, Chao T. Pain control. In: 20 Common Problems: Ethics in Primary Care. Sugarman J (ed.). New York, New York: McGraw-Hill; 2000:199–210.
10. Payen JF, Bosson JL, Chanques G, Mantz J, Labarere J. Pain assessment is associated with decreased duration of mechanical ventilation in the intensive care unit: a post hoc analysis of the DOLOREA study. Anesthesiology 2009;111(6):1308–1316. doi: 10.1097/ALN.0b013e3181c0d4f0
11. Chanques G, Jaber S, Barbotte E, et al. Impact of systematic evaluation of pain and agitation in an intensive care unit. Crit Care Med 2006;34(6):1691–1699. doi: 10.1097/01.CCM.0000218416.62457.56
12. Morris PE, Griffin L, Berry M, et al. Receiving early mobility during an intensive care unit admission is a predictor of improved outcomes in

acute respiratory failure. Am J Med Sci 2011;341:373–377. doi: 10.1097/MAJ.0b013e31820ab4f6

13. Schweickert WD, Pohlman MC, Pohlman AS, et al. Early physical and occupational therapy in mechanically ventilated, critically ill patients: a randomised controlled trial. Lancet 2009;373:1874–1882. doi: 10.1016/S0140-6736(09)60658-9
14. Reade M, Phil D, Finfer S. Sedation and delirium in the intensive care unit. N Engl J Med 2014;370:444–454. doi: 10.1056/NEJMra1208705
15. Page GG, Blakely WP, Ben-E liyahu S. Evidence that postoperative pain is a mediator of the tumor-promoting effects of surgery in rats. Pain 2001;90:191–199.
16. Schelling G, Stoll C, Haller M, et al. Health-related quality of life and posttraumatic stress disorder in survivors of the acute respiratory distress syndrome. Crit Care Med 1998;26:651–659.
17. Myhren H, Ekeberg O, Toien K, Karlsson S, Stokland O. Posttraumatic stress, anxiety and depression symptoms in patients during the first year post intensive care unit discharge. Crit Care 2010;14:R14–R14. doi: 10.1186/cc8870
18. Gélinas C, Fortier M, Viens C, Fillion L, Puntillo KA. Pain assessment and management in critically ill intubated patients: a retrospective study. Am J Crit Care 2004;13:126–135.
19. Payen JF, Bru O, Bosson JL, et al. Assessing pain in critically ill sedated patients by using a behavioral pain scale. Crit Care Med 2001;29(12):2258–2263.
20. Barr J, Fraser GL, Puntillo K, et al. Clinical practice guidelines for the management of pain, agitation, and delirium in adult patients in the intensive care unit. Crit Care Med 2013;41(1):263–306. doi: 10.1097/CCM.0b013e3182783b72
21. Young J, Siffleet J, Nikoletti S, et al. Use of a behavioural pain scale to assess pain in ventilated, unconscious and/or sedated patients. Intensive Crit Care Nurs 2006;22:32–39. doi: 10.1016/j.iccn.2005.04.004
22. Horlocker TT, Wedel DJ, Rowlingson JC, et al. Regional anesthesia in the patient receiving antithrombotic or thrombolytic therapy: American Society of Regional Anesthesia and Pain Medicine evidence-based guidelines (3rd ed.). Reg Anesth Pain Med 2010;35:64–101.
23. Latta KS, Ginsberg B, Barkin RL. Meperidine: a critical review. Am J Ther 2002;9(1):53–68.
24. Grass JA. Patient-controlled analgesia. Anesth Analg 2005;101:S44–S61.
25. Baumbach P, Gotz T, Gunther A. Prevalence and characteristics of chronic intensive care–related pain: the role of severe sepsis and septic shock. Crit Care Med 2016;44(6):1129–1137. doi: 10.1097/CCM.0000000000001635
26. Johnson J, Al-Dahir S. Chronic post-ICU pain and post-intensive care syndrome. US Pharm 2016;41(3):HS11–HS15. https://www.uspharmacist.com/article/chronic-posticu-pain-and-postintensive-care-syndrome. Accessed September 9, 2017.
27. Pena L, Moreno CB, Gutierrez-Alvarez AM. Pain management in Guillain-Barre syndrome: a systematic review. Neurologia 2015;30(7):433–438. doi: 10.1016/j.nrl.2014.04.009
28. Upton D, Andrews A. The impact of stress at dressing change in patients with burns: a review of the literature on pain and itching. Wounds 2014;26:77–82.

A Novel, Clinic-Based Approach to Address Patients with Complex Back Pain in a Veterans Administration Hospital: The Back Pain Home

Over the past several decades, we have witnessed the care of patients with spine-related pain growing in complexity. There is a tremendous expansion in the modalities available to the interventional pain, neurosurgical, physical medicine, and rehabilitation (PM&R), as well as psychological specialties for the treatment of chronic painful conditions of the spine. Although the additional resources are welcome developments, they have a significant downside. When commonly employed treatment plans are spread out concurrently over several specialties, we can see a wasteful overlap of resources. The multiple processing of initial intakes, imaging, electromyography (EMG), physical therapy (PT), and surgical or interventional pain procedures consume significant clinic capital. This problem becomes more pronounced in patients with complicated clinical pictures as they become disproportionate utilizers of care.

To address this problem and streamline care, involved providers at the Veterans Administration (VA) Hospital in Palo Alto, California, created a new clinical environment: the Spine Clinic. We staff our clinic with attending-level physicians from the pain medicine, neurosurgery, and PM&R specialties. We also have providers from PT and psychological specialties. During an evaluation at the Spine Clinic, the patient presents with all providers simultaneously. Prior to seeing the patient, clinicians review the patient's history, prior imaging, EMGs, physical exams, and psychological demeanor from prior documentation, when available.

In selecting patients for the Spine Clinic, we have centered on treating patients whose high degree of spinal pathology requires frequent provider input. We chose those patients for several reasons, one of which was to preserve the standard tiered system of managing spine-related pain. This system relies on primary care providers to spearhead the delivery of care through the consult process. We recognize the effectiveness of this system in managing most patient complaints and therefore chose not to alter it. Rather, Spine Clinic patients are selected by providers themselves from a cadre of pre-existing clinic patients. Participation in the Spine Clinic requires no litmus test, although typical Spine Clinic patients have had significant previous interactions with one of our involved services and failed to make meaningful progress with their condition. While there are no direct consultations available to outside providers, the Spine Clinic staff identify appropriate patients based on their knowledge of them and of other specialties. By avoiding compartmentalization of specialties, we enjoy a broader understanding of other involved professionals and gain an appreciation for the effectiveness and appropriateness of various plans of care. We feel that this cross-training has become invaluable in directing all our clinic patients into appropriate care.

The Spine Clinic uses the well-developed concept of a multidisciplinary care model. Significant data has shown that addressing the physical as well as the biopsychosocial pathologies of patients leads to better outcomes. Additionally, some patients who have completed all reasonable, validated conservative and interventional care have been deemed to be nonsurgical candidates and yet still suffer from chronic daily nonmalignant pain of spinal origin. The ubiquitous emotional pathology of such patients often remains unaddressed in solitary clinics. We have found the collaborative, multidisciplinary setting to be helpful because it allows us to address the patient's outstanding questions and emotional state in a comprehensive manner. It also sets up a unique support system for physicians, allowing us to collaboratively address a patient's consideration of more high-risk, expensive, and often-unproven treatment modalities. As such, the Spine Clinic practitioners are occasionally in the situation of having nothing else reasonable to offer. We believe that having this discussion with patients is important. In a traditional clinical construct, patients may leave a clinic or become *lost to follow-up*, only to matriculate to another clinical provider and repeat already disproven modalities. The Spine Clinic's cooperative construct allows providers the unique capability of telling a patient that there is likely nothing else to be done. This allows us to address outstanding questions and emotional issues that can assist the patient in adhering to a reasonably conservative plan of care.

Peter Barelka, MD
Assistant Clinical Professor
Department of Anesthesia, Pain and Perioperative Medicine
Stanford University Hospital
Stanford, California

Section Editor: Magdalena Anitescu, MD

"During an evaluation at the Spine Clinic, the patient presents with all providers simultaneously."

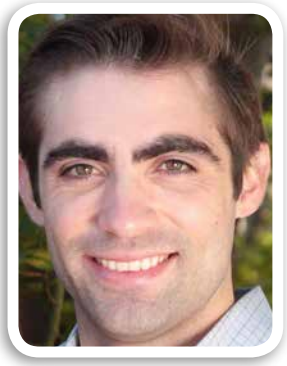
Our Spine Clinic approach allows for a streamlined clinic experience for the patient. No longer are patients asked to complete up to five or more separate consultations and imaging appointments to determine a plan of care. Rather, we offer timely input from all the providers: a one-stop shop for patients that brings to bear all available resources of various specialties. The time a patient saves—although perhaps difficult to objectively assess—is subjectively clear to see. The typically larger catchment area of patients in the VA system may amplify this characteristic and result in more significant time savings. However, even patients

from smaller regional hospitals and clinics may notice a significant time savings.

Another benefit to our Spine Clinic system is that it provides patients and caregivers with a cleaner line of communication. The traditional system of individual specialist consultation, although usually appropriate, can result in loss of valuable information. This is most often seen in complicated chronic pain patients. For instance, in the traditional consult system, a neurosurgical patient may be asked to return to the Pain Clinic to be evaluated for a series of selective nerve blocks to assist in determining pain location. However, even this seemingly innocuous plan has countless opportunities for failure: The patient could convey to the pain physician a different location or type of pain, or voice his or her desire to change the ultimate goals. The patient may simply fail to follow up on the visit. Either way, the result is the same: confusion on the part of the patient and provider alike. Patients and providers can quickly find themselves operating from very different starting points, seeking different outcomes. In the Spine Clinic, all providers who will render care discuss the plan of care with the patient. We significantly curtail possible confusion by simply having all providers in the same room discussing treatment options with the patient.

Initial feedback from our patients indicates the Spine Clinic has been well received. Patients enjoy the single visit construct in which they can get what they see as instantaneous feedback to their questions and concerns. Whether clinicians feel it is reasonable to consider what could be called *customer satisfaction*, its prominence in current and future medical care is certain. When we consider the current volatile political situation surrounding medical reform in this country, it will be necessary to implement novel, efficient delivery systems for care. All recently adopted and currently proposed legislation prominently features payment systems that emphasize efficiency and standardization of care. Future changes will likely see a continuation of the policies laid out in Medicare Access, the CHIP Reauthorization Act (MACRA), and other bundled payment plans with an overall trajectory that addresses redundant and inefficient processes. It seems reasonable that some coordination of care is necessary at the clinic level to efficiently address the further growth requirements of the field. At the VA Hospital in Palo Alto, California, involved providers have found that seeing certain patients simultaneously in a multidisciplinary setting helps address complex, spine-related pain in a way that improves patients' clinical experience and clinic efficiency.

Head and Neck Regional Anesthesia Techniques



Brandon Kandarian, MD, MS
Resident Physician, PGY-4
Department of Anesthesiology, Perioperative & Pain Medicine
The Mount Sinai Hospital
New York, New York

Adam Levine, MD
Professor
Department of Anesthesiology, Perioperative & Pain Medicine, Pharmacological Sciences, and Otolaryngology
The Mount Sinai Hospital
New York, New York

Samuel DeMaria Jr., MD
Associate Professor
Department of Anesthesiology, Perioperative & Pain Medicine
The Mount Sinai Hospital
New York, New York

Section Editor: Kristopher Schroeder, MD

The head and neck have rich nerve supplies with reliably identifiable surface and bony landmarks. This makes administering regional anesthesia for head and neck surgical procedures practical and effective. Although novel for otolaryngologic surgical procedures, the use of regional anesthesia is commonplace for dental and oral-maxillofacial procedures. Head and neck regional anesthesia is typically employed for postoperative analgesia and placed safely after the induction of anesthesia, but it can also be used effectively as the primary anesthetic (eg, superficial cervical block for carotid endarterectomy). Additionally, patients presenting for head and neck pathology may require awake intubation techniques, and regional nerve blocks can be used to augment local anesthesia topicalization. Finally, regional blocks placed intraoperatively have been used effectively as the sole anesthetic for exploration and hemostasis in the rare and catastrophic event of postoperative bleeding. As with all regional anesthetic techniques, it is important to discuss the planned block with the surgeon preoperatively on a patient-by-patient basis as some blocks are contraindicated based on the patient's pathology, covered below. In addition to providing intraoperative and postoperative analgesia, certain blocks can be combined with epinephrine to improve the operative conditions for the surgeon. In the following discussion, we will describe the head and neck blocks typically used in our practice.

"Although novel for otolaryngologic surgical procedures, the use of regional anesthesia is commonplace for dental and oral-maxillofacial procedures."

FRONTAL NERVE BLOCK (V1)

The trigeminal nerve, the largest of the cranial nerves, provides sensory and motor innervation to the face. The nerve roots originate in the pons, and the sensory fibers are sent to the trigeminal ganglion. From the trigeminal ganglion, the nerve splits into three major branches: The ophthalmic (V1) nerve, the maxillary (V2) nerve, and the mandibular (V3) nerve.

The ophthalmic division of the trigeminal nerve provides pure sensory innervation to the top third of the face (Figure 1). The frontal nerve is the largest branch of the ophthalmic nerve, and it divides into terminal branches called the supraorbital and supratrochlear nerves (Figure 2). The frontal nerve block can be used to block both nerves.[1] These single-shot nerve blocks can be used in ophthalmologic, craniotomies, frontal sinus surgery, and cosmetic nasal surgery. It is performed by palpating the supraorbital notch (located at the middle portion of the superior orbit) and injecting 1–2 ml of 0.5% bupivacaine (blocking the supraorbital nerve) and directing the needle to the medial brow (Figure 3). It is important to palpate the orbit with the free hand and leave a finger on the orbit throughout the block to minimize the risk of injection of local anesthesia into the globe of the eye.[2] The area of anesthesia includes the upper eyelid, forehead, bridge of nose, and scalp (Figure 4).

INFRAORBITAL (V2)

The infraorbital nerve provides sensation to the cheek, upper lip, eyelid, and lateral aspect of the nose (Figure 5). We routinely use bilateral single shot infraorbital blocks combined with bilateral single-shot sphenopalatine blocks for functional endoscopic sinus surgery (FESS) procedures. A randomized study at our institution showed that this block, when combined with a sphenopalatine block, decreases opiate requirements and postanesthesia care unit time.[3] An infraorbital block may also be used for procedures

Figure 1: *Sensory innervation of the head and neck.*

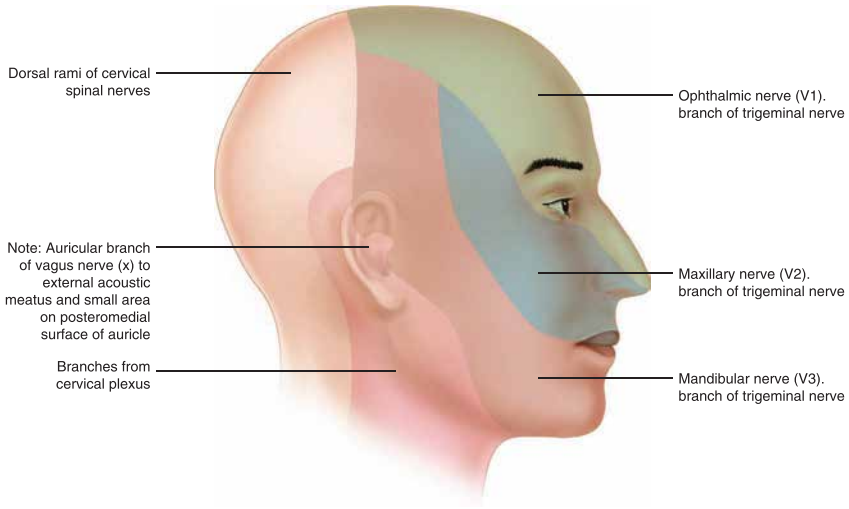
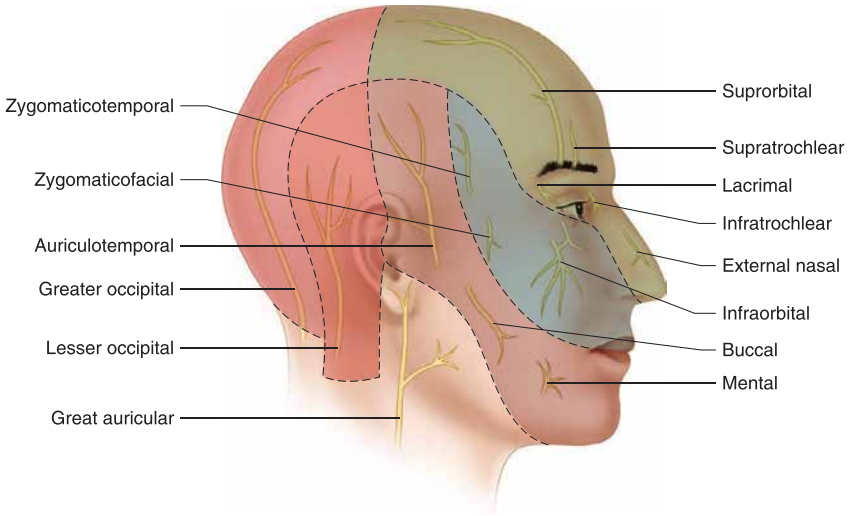


Figure 2: *Supraorbital and supratrochlear nerves.*



involving the upper lip, nose, and maxillary teeth. Although there are three approaches to block the infraorbital nerve (sublabial, direct, or transnasal), we routinely use a transnasal approach, which reduces the chance of a noticeable facial skin puncture postoperatively.

First, the infraorbital foramen, where the infraorbital nerve is located, is palpated by finding the groove located inferior to the orbit rim, 3 cm from the midline of the face.[4] This finger is left in this notch both as a marker for the needle trajectory and also to prevent the needle from entering the orbit. A 1.5-inch 25-gauge needle is inserted through the nares and tunneled superficially under the skin toward the infraorbital foramen, stopping at the halfway point between the edge of the nose and the infraorbital foramen (Figure 6). The needle is then aspirated, and 2 ml of 0.5% bupivacaine is injected.[5] The block is repeated contralaterally for bilateral surgical procedures.

Contraindications to this procedure include neoplasms or arteriovenous malformations involving the nasal cavity. If such contraindications exist, we perform this block using a sublabial approach. In this approach, the free finger is again left on the infraorbital notch for reasons previously mentioned, and the needle is passed starting between the gums and lips until the needle is again positioned midway between the edge of the nose and the infraorbital groove, where the needle is aspirated and 2 ml

Figure 3: *Supraorbital and supratrochlear nerve blocks.*

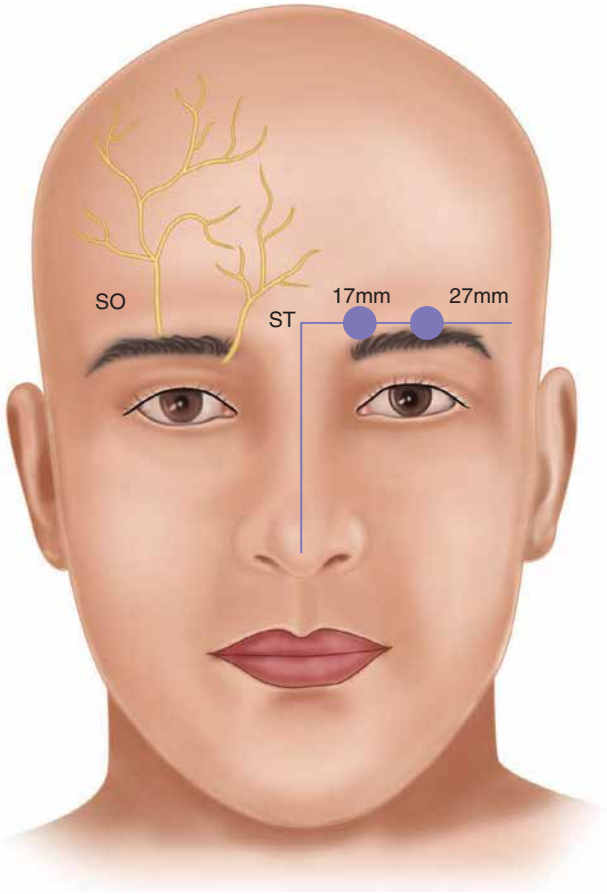


Figure 4: *The area of anesthesia covered by supraorbital and supratrochlear nerve blocks.*

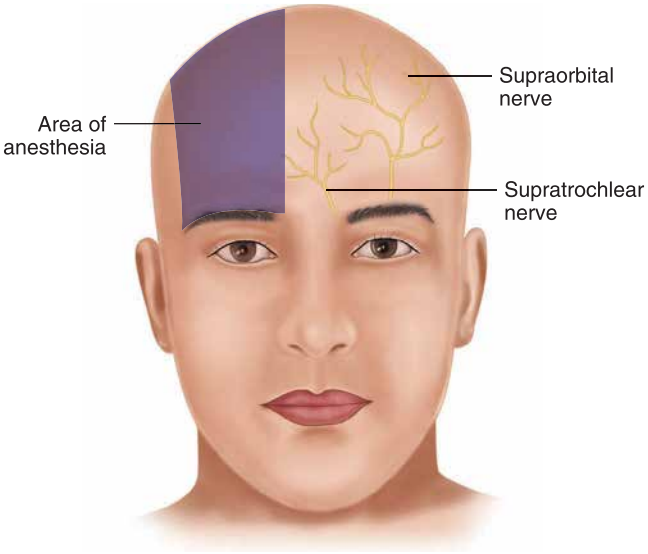


Figure 5: *Infraorbital nerve and the area of anesthesia covered by blocking it.*

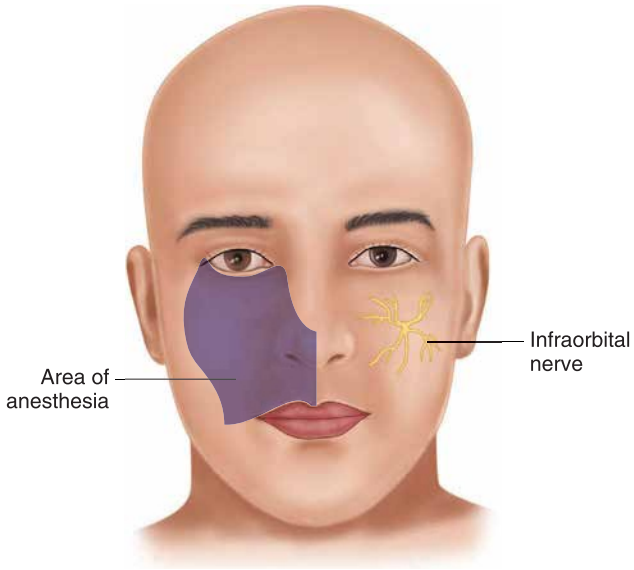


Figure 6: *Trans-nasal approach to the infraorbital nerve block.*

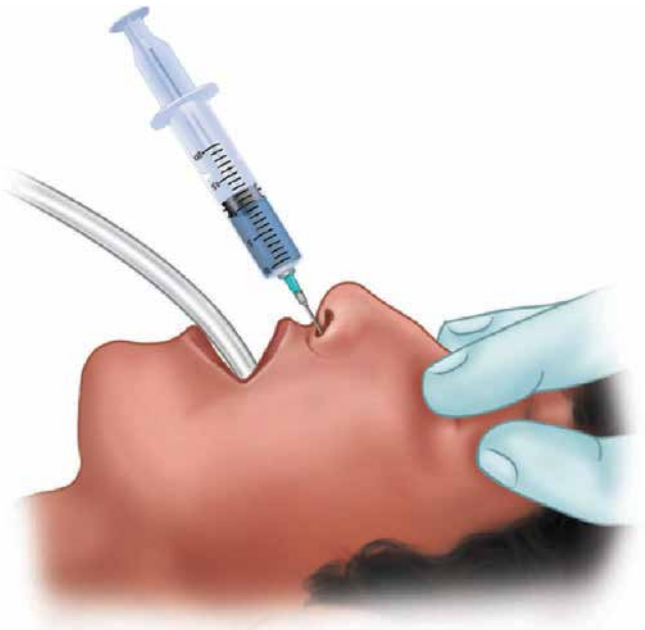
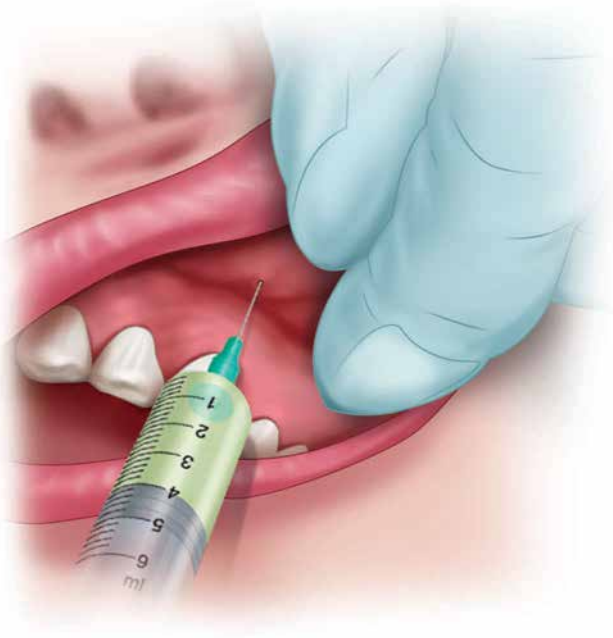


Figure 7: *Sublabial approach to the infraorbital nerve block.*



of 0.5% bupivacaine is injected (Figure 7).[6] If an intraoperative computerized tomography image guidance for FESS is used, this block should be performed after registration because the system uses skin depth, and topography that can be altered by this block.

INFRA-ALVEOLAR (INFERIOR ALVEOLAR NERVE BLOCK, V3)

The mandibular (V3) nerve is the largest branch of the trigeminal nerve. It exits the skull through the oval foramen and then divides into the posterior trunk, which becomes the inferior alveolar nerve that innervates the molar and premolar teeth. This branch travels posterior and lateral to become the lingual nerve along with the inferior alveolar artery and vein, and then exits the mental foramen to become the mental nerve, which provides sensation to the lower lip and chin. The lingual nerve provides sensory innervation to the anterior two-thirds of the tongue, mucosa of the floor of the mouth, and lingual gingiva. Both the inferior alveolar nerve and lingual nerve are blocked concurrently with this block. We use this block for procedures involving the mandible, all mandibular teeth, the floor of the mouth, the anterior two-thirds of the tongue, and the mucosa and skin of the lower lip and chin.

The target for the block is the mandibular nerve as it travels on the medial aspect of the ramus of the mandible, prior to its entry into the mandibular foramen.[7] We use a 25-gauge needle 1.5 inches in length for the single shot. The patient is supine, the mouth is opened, and the cheek is retracted. The coronoid notch and the pterygomandibular raphe are identified.[8] The coronoid notch is the most concave area on the anterior border of the ramus, shown in Figure 8a as the innermost solid blue lines. If not obvious on exam,

Figure 8: *The inferior alveolar and lingual nerve blocks.* **A:** *The coronoid notch represented as innermost solid blue lines.* **B:** *Needle approach to the inferior alveolar nerve block.*

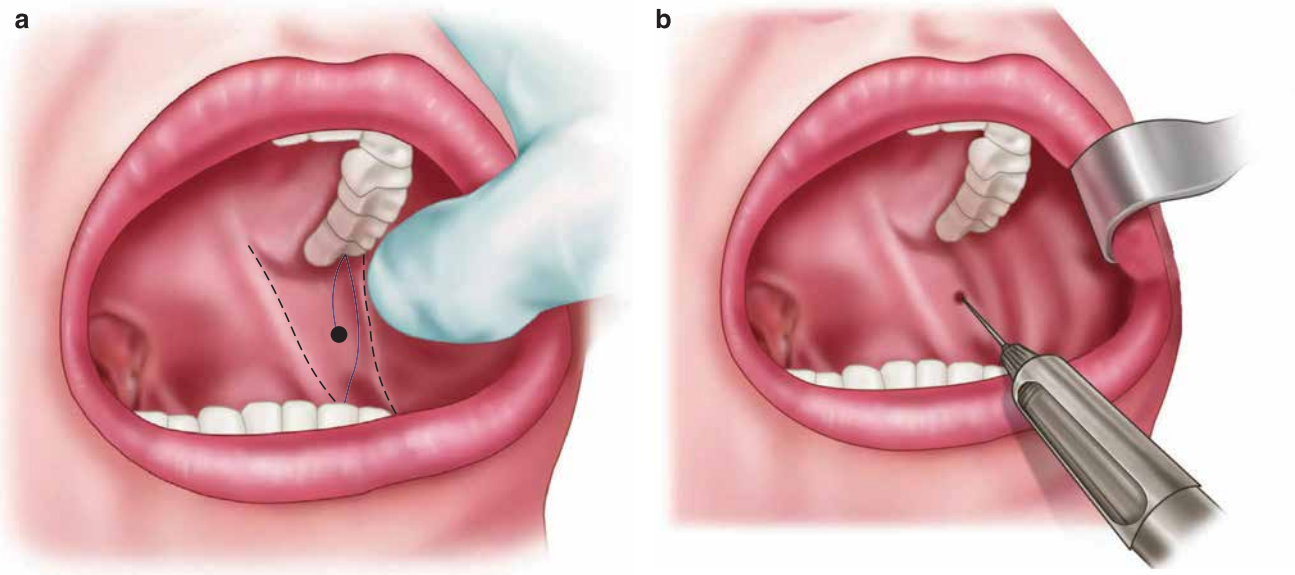
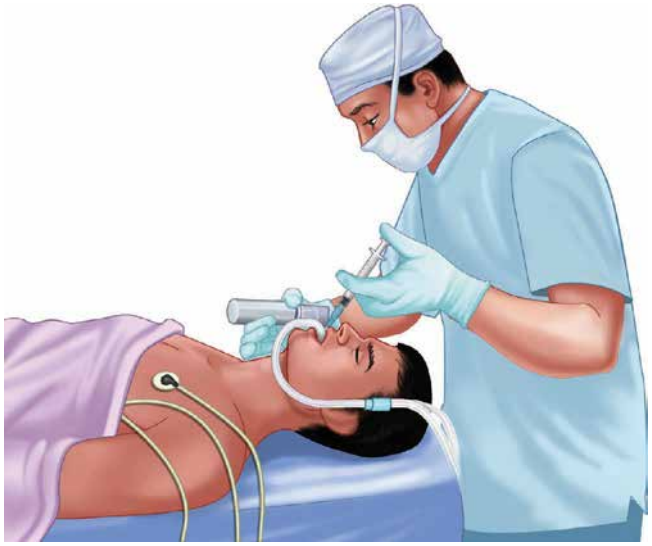


Figure 9: *Transoral approach to the sphenopalatine nerve block.*



the notch can be palpated with a finger. The pterygomandibular raphe is indicated by black dashed lines in the same figure.[2] The needle is then placed at the injection site from the contralateral premolar region indicated by the black dot in Figure 8b. The needle is advanced until the mandible is contacted (typically 25–35 mm deep). Once the mandible is contacted, withdraw the needle one millimeter and redirect the needle slightly posterior and inject 2–5 ml of 0.5% bupivacaine. Two to four ml of 0.5% bupivacaine should be injected continuously while the needle is withdrawn to block the lingual nerve.

SPHENOPALATINE

The sphenopalatine ganglion also originates from the maxillary branch of the trigeminal nerve. It provides sensation to the hard palate, soft palate, tonsils, nasal and pharynx mucosa, posterior portion of the nasal septum, and paranasal sinuses. At our institution, we routinely perform a transoral approach to place the sphenopalatine nerve blocks. We perform the block post induction and endotracheal intubation.

After extending the neck, we use a Macintosh 3 blade to sweep the tongue and the endotracheal tube out of the field while illuminating the palate (Figure 9). We identify the greater palatine foramen by locating a groove that is medial to the space between the first and second upper molars (approximately 0.5–1 cm medial). We then use a 1.5-inch 25-gauge needle for injection. The needle is bent at a 90-degree angle at 1.5 cm from the tip and then inserted up to that bend in this groove into the pterygopalatine fossa where the sphenopalatine ganglion is located (Figure 10). The needle is aspirated, and 1.5 ml of 1–2% lidocaine with 1:100,000 epinephrine is injected on each side for bilateral surgical procedures.[5] Localized blanching of the hard palate confirms the correct location when injecting. The blanching is secondary to vasoconstriction of the internal maxillary artery, which is located in the pterygopalatine fossa.[4] Epinephrine added to the local anesthetic helps both decrease absorption in the area as well as improve the surgical field.

Possible complications include intravascular injection, infraorbital nerve injury, and transient diplopia. The sphenopalatine nerve block should be avoided in cases where pathology may involve the pterygopalatine fossa. If the surgeon will be using computed tomography guidance for FESS, this block should be placed before registration because the positioning required for the block (head extension) can disrupt the imaging band on the forehead.

SUPERFICIAL CERVICAL BLOCK

The superficial cervical plexus provides sensation from the mandible to the clavicle and can be used for central (thyroid, parathyroid, or thyroglossal cyst) or lateral (neck dissections, lymph node biopsy) surgical procedures. For central procedures, the block is placed bilaterally. If the block will be used for postoperative pain, the landmarks are identified and marked preinduction, and the block is placed postinduction.

While supine, patients are asked to contract their sternocleidomastoid muscle (lift their head against your hand placed on their forehead) to identify the posterior border of the sternocleidomastoid.[2] The flexed muscle seen in the neck contralateral to the direction the patient is pushing is the sternocleidomastoid muscle. The posterior border of this muscle is demarcated with a marker. The midline between the mastoid

Figure 10: *Sphenopalatine nerve blocks.*

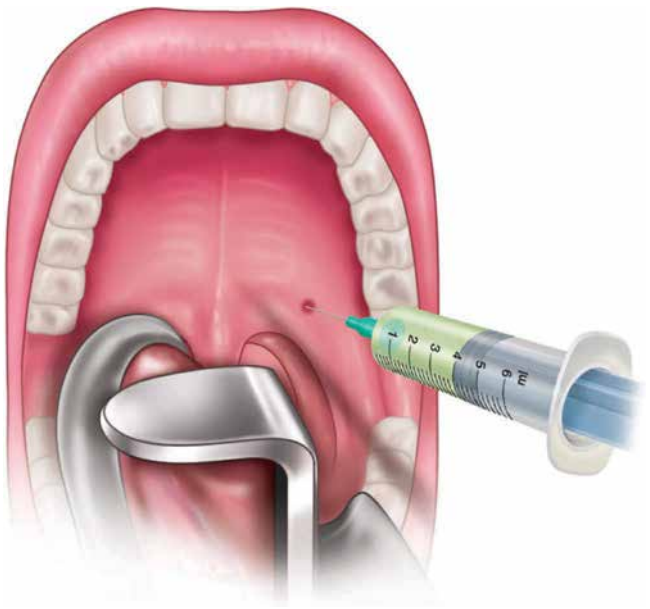
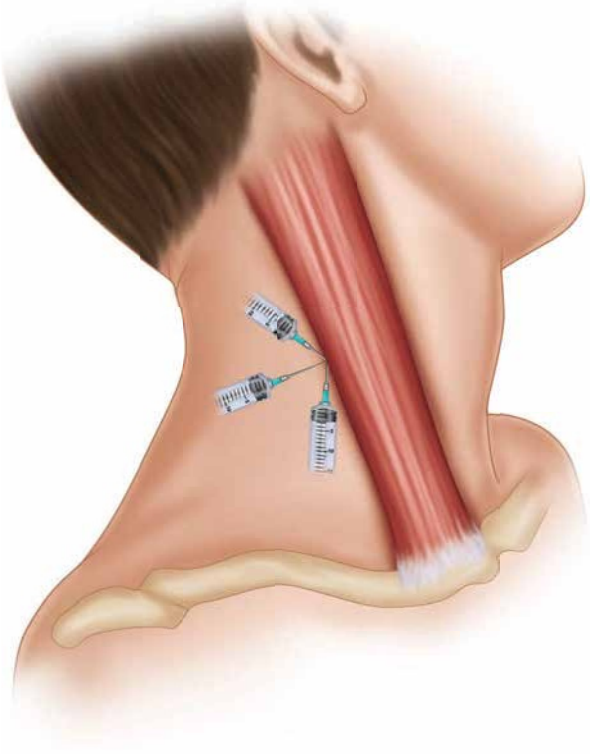


Figure 11: *Superficial cervical plexus block.*



process and the prominent tubercle of C6 or Chassaignac tubercle at the level of the cricoid cartilage are then demarcated on this border. This midway point is the estimated location of where the cervical plexus emerges.[5,6] The block can be performed either awake or asleep, depending on the procedure and patient preference. Next, a 1.5-inch, 25-gauge needle is used to inject 10 cc of 0.5% bupivacaine superficially along this demarcated border (Figure 11). It is important to remain superficial throughout the injection and to aspirate multiple times during injection to ensure a vessel has not been entered. Risks are minimal for this procedure because the injection is superficial along the border of the muscle. Newer techniques with ultrasound have been described, and several of our faculty use this technique effectively.[9]

RECURRENT (TRANSTRACHEAL) AND SUPERIOR LARYNGEAL BLOCKS

For awake intubations, we routinely perform a transtracheal block to anesthetize the recurrent laryngeal nerves and a direct bilateral superior laryngeal nerve block. We use these blocks to supplement our topicalization of the oropharynx before performing an awake intubation technique, but we also use them for laryngoscopy, bronchoscopy, and transesophageal echocardiography. The superior laryngeal nerve can be blocked at the thyrohyoid membrane (between the superior cornu of the thyroid and the hyoid bone). The superior laryngeal nerve innervates the base of the tongue, the posterior surface of the epiglottis, and the arytenoids.

To perform the block, the patient lies supine and the head is turned away from the side to be blocked. The free hand is used to palpate the hyoid bone or the thyroid cartilage, which can be reliably identified in the majority of patients, and hold it between the index finger and thumb. The superior laryngeal nerve runs slightly lateral to the tubercle of the greater horn of the hyoid bone, and this is the target for the single shot block.[5] The index finger is left on the opposite side of the hyoid, pushing down for hyoid stabilization and identification. A 25-gauge needle is inserted until resistance is felt as it hits the greater horn of the hyoid bone or thyroid cartilage. The needle is then withdrawn 1 mm and checked for negative aspiration, and then 2 ml of 2% lidocaine are injected. This block is done bilaterally.

The recurrent laryngeal or transtracheal block is performed to anesthetize the recurrent laryngeal nerve. The recurrent laryngeal nerve innervates the glottis and the trachea. For the transtracheal block, the patient is positioned supine and the cricothyroid membrane is palpated. A 20-gauge peripheral venous catheter with local anesthesia is inserted into the space while aspirating with a 5-cc catheter until a pop is felt and air bubbles return, confirming position within the trachea. The needle is then removed, leaving the catheter in place to provide immediate access to the airway. A 5-ml syringe filled with 4 ml of 4% lidocaine is reattached and aspirated again to confirm correct position (air bubbles seen on aspiration, negative for blood). The patient is then asked to take a deep breath as the local anesthesia is injected. The patient will typically cough as the local anesthesia coats the vocal cords, so we inform the patient to anticipate this event during consent and again right before we inject.

In addition to being an effective method for anesthetizing the recurrent laryngeal nerve, this technique simultaneously can be used in learning how to perform an emergency needle cricothyrotomy. If this block cannot be performed because of pathology in the area or difficulty in confirming the location, the recurrent laryngeal can also be blocked by inserting an epidural catheter into the fiberoptic scope and injecting 4 ml of 4% lidocaine under direct visualization of the vocal cords.

CONCLUSION

Despite having described several regional blocks commonly used in our day-to-day practice, this is not an exhaustive list of the nerve blocks for head and neck surgical procedures. By forming strong relationships with the hospital's otolaryngologists over the years, we were able to collaborate and build our current repertoire of regional block techniques. The combination of anatomic and ultrasound-based regional techniques helps us to educate our residents in different techniques for regional blocks. In addition, because our hospital has adopted enhanced recovery after surgery (ERAS) protocols for different surgical procedures, we have

incorporated these regional techniques into the head and neck surgery ERAS protocols to attempt to speed recovery and decrease perioperative opioid use.

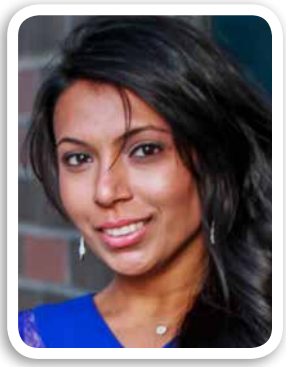
ACKNOWLEDGMENT

All images are taken with permission from Springer Nature Publishing Rights and Permissions group.

REFERENCES

1. Kanakaraj M, Shanmugasundaram N, Chandramohan M, Kannan R, Perumal SM, Nagendran J. Regional anesthesia in faciomaxillary and oral surgery. J Pharm Bioallied Sci 2012;4:S264–S269. doi: 10.4103/0975-7406.100247
2. Levine A.I , DeMaria Jr S. Regional anesthesia. In: Ferraro's Fundamentals of Maxillofacial Surgery. Taub PJ, Patel PK, Buchman SR, Cohen M (eds.). New York, New York: Springer 2015:77–90.
3. DeMaria Jr S, Govindaraj S, Chinosorvatana N, Kang S, Levine AI. Bilateral sphenopalatine ganglion blockade improves postoperative analgesia after endoscopic sinus surgery. Am J Rhinol Allergy 2012;26(1):e23–e27. doi: 10.2500/ajra.2012.26.3709
4. Levine AI, Govindaraj S, DeMaria S, eds. Anesthesiology and Otolaryngology. Dordrecht, Netherlands: Springer;2013.
5. Richardson J. Atlas of interventional pain management. Br J Anaesth 2004;92(4):604–605. doi: 10.1093/bja/aeh544
6. Barash PG, Cullen BF, Stoelting RK, Cahalan M, Stock MC. Clinical Anesthesia. 6th ed. Philadelphia, Pennsylvania: Lippincott Williams & Wilkins;2009.
7. Khalil H. A basic review on the inferior alveolar nerve block techniques. Anesth Essays Res 2014;8(1):3–8. doi:10.4103/0259-1162.128891
8. Takasugi Y, Furuya H, Moriya K, Okamoto Y. Clinical evaluation of inferior alveolar nerve block by injection into the pterygomandibular space anterior to the mandibular foramen. Anesth Prog 2000;47:125–129. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2149033/ Accessed September 9, 2017.
9. Hadzic A, Carrera A, Clark TB, et al. Hadzic's Peripheral Nerve Blocks and Anatomy for Ultrasound-Guided Regional Anesthesia. 2nd ed. New York, New York: McGraw-Hill;2012.

Ketamine Infusion on Regular Wards: A Myth or Reality?



Sarah Choxi, MD
Clinical Associate
Department of Anesthesia and Critical Care
University of Chicago
Chicago, Illinois

Xiaoying Zhu, MD, PhD
Assistant Professor
University of Virginia Health System,
Department of Anesthesiology, Pain Management Center
Charlottesville, Virginia

Section Editor: Lynn Kohan, MD

The clinical use of ketamine for sedation, catalepsy, somatic analgesia, bronchodilation, and sympathetic nervous system stimulation began in 1970; however, its use has been limited mostly to pediatric and trauma anesthesia because it can cause side effects, especially the psychotropic effects.[1] Because of its strong analgesic effect, ketamine has recently emerged as a promising adjunct for pain management as an alternative to narcotic medications to end the dreaded opioid epidemic. Ketamine works mainly as an N-methyl-D-aspartate (NMDA)-receptor antagonist but also enhances descending inhibition and has anti-inflammatory properties.[1–5] The NMDA antagonism helps to attenuate central sensitization and palliate neuropathic pain, which are believed to play significant roles in the development and propagation of chronic pain states.[6–8]

In recent years, a relatively large body of evidence has accumulated showing the beneficial effects of intravenous ketamine infusion in patients with chronic refractory pain states, including fibromyalgia, neuropathic pain, phantom limb pain, postherpetic neuralgia, complex regional pain syndromes (CRPS), diabetic neuropathy, sickle cell pain during acute crises, and central pain related to stroke or spinal cord injuries.[9–13] However, to date, no guideline has been developed for its use or a protocol to standardize doses and duration because of the lack of quality studies and sufficient evidence.

INFUSION PROTOCOLS

Because of the potential side effects of tachyarrhythmias, hypertension, and psychomimetic effects, ketamine continuous infusion was, historically, mostly limited to intensive or intermediate care settings and thus associated with high costs. Ketamine, however, can be administered safely on a nonacute, inpatient ward. Following are the infusion protocols that have been implemented at the University of Chicago Medical Center and University of Virginia Health System. When used in subanesthetic doses, ketamine is considered safe and side effects are generally well tolerated[1] or readily treatable. No major complications have occurred in our patients so far.

"To date, no protocol has been developed to standardize doses and duration of ketamine infusion because of the lack of quality studies and sufficient evidence."

University of Chicago. Patients are admitted to the inpatient ward, where low-dose ketamine infusions outside of the intensive care unit are managed by the acute pain service staffed by faculty physicians trained in pain medicine and anesthesiology. Conditions commonly treated with intravenous ketamine infusions include neuropathic pain, CRPS, refractory headache or back pain in patients with Chiari malformations, refractory abdominal pain from inflammatory bowel disease or celiac artery compression syndrome, and pain related to vaso-occlusive sickle cell crisis. Infusions are dosed based on ideal body weight and started at 1 mcg/kg/min and titrated to effect or best tolerated dose without significant cardiovascular or central nervous system side effects up to a maximum dose of 5 mcg/kg/min for a course of 1–5 days. During this time, patients are closely monitored with routine vital signs of blood pressure, pulse, respiratory rate, pain score, and sedation level assessed 1 hour post dose following the first dose or dose increase and then every 4 hours and continuous pulse oximetry throughout infusion. The acute pain service is alerted for systolic blood pressure greater than 160 mm Hg, respiratory rate less than 10 breaths/min, any acute change in mental status (eg, blunted affect, emotional withdrawal, thought disorder, delirium), or any difficulty in arousal despite continuous stimulation. Laboratory test results and electrocardiograms are checked periodically upon the discretion of the acute pain service attending. Oxygen therapy via nasal cannula is available to maintain oxygen saturation above 92% with a bag valve mask available at the bedside in case of severe hypoxia. Supportive medications such as naloxone, lorazepam, and prochlorperazine are readily available. At time of initiation, strong consideration is given to decreasing or modifying opioid and nonopioid analgesics with concomitant use of ketamine intravenous infusions.

University of Virginia. At University of Virginia Health System, ketamine infusions are also performed on an inpatient ward and

are also managed by the acute pain service by trained faculty. Conditions commonly treated with intravenous ketamine infusions include CRPS and refractory neuropathic pain of various causes. The infusion is typically started at 0.1 mg/kg/hr, then increased slowly as tolerated to 0.5–0.75 mg/kg/hr, with the entire course lasting for 5–7 days depending on a patient's response. During this time, patients are monitored closely, Laboratory test results and electrocardiogram checked periodically, and side effects treated in a timely fashion. Benzodiazepines have been used to minimize its psychotropic side effects. When used in subanesthetic doses, ketamine is considered safe, and side effects are generally well tolerated[1] or readily treatable. No major complication has occurred in our patients so far. Similar to the University of Chicago, strong consideration is given to decreasing opioid analgesics.

ACUTE PAIN

Ketamine given preoperatively, intraoperatively, or postoperatively has been shown to decrease postoperative pain and reduce perioperative opioid consumption in opioid-dependent patients.[14–16] Ketamine infusions should be considered in the treatment of refractory acute pain after surgery or from trauma, in the intensive or intermediate care setting, as well as on the regular floor, especially for patients who are opioid tolerant. Including ketamine in the enhanced recovery after surgery protocols is likely beneficial.

Many questions still remain regarding ketamine. The incidence and degree of side effects from ketamine depend on dosage. The existing evidence also suggests that the analgesic effect of ketamine is both dose[9] and duration[1,2] dependent. However, no consensus exists on ketamine infusion protocols regarding dose, titration, infusion duration, and frequency of repeated infusions. Randomized controlled trials are needed to answer these questions. A consensus or guideline on ketamine infusions is needed, as well. A ketamine registry may be helpful to report complications.

CONCLUSION

Current evidence has shown that ketamine infusion is effective in treating chronic and acute refractory pain. It appears ketamine infusion treatment can be administered on the inpatient ward. A consensus or guideline on ketamine infusion is needed.

REFERENCES

1. Niesters M, Martini C, Dahan A. Ketamine for chronic pain: risks and benefits. Br J Clin Pharmacol 2014;77:357–367. doi: 10.1111/bcp.12094.
2. Noppers I, Niesters M, Aarts L, Smith T, Sarton E, Dahan A. Ketamine for the treatment of chronic non-cancer pain. Expert Opin Pharmacother 2010;11:2417–2429. doi: 10.1517/14656566.2010.515978.
3. Hirota K, Lambert DG. Ketamine: new uses for an old drug? Br J Anaesth 2011;107:123–126. doi: 10.1093/bja/aer221.
4. Niesters M, Khalili-Mahani N, Martini C, et al. Effect of subanesthetic ketamine on intrinsic functional brain connectivity: a placebo-controlled functional magnetic resonance imaging study in healthy male volunteers. Anesthesiology 2012;117:868–877. doi: 10.1097/ALN.0b013e31826a0db3.
5. Niesters M, Aarts L, Sarton E, Dahan A. Influence of ketamine and morphine on descending pain modulation in chronic pain patients: a randomized placebo-controlled cross-over proof-of-concept study. Br J Anaesth 2013;110:1010–1016. doi: 10.1093/bja/aes578.
6. Woolf CJ. Central sensitization: implications for the diagnosis and treatment of pain. Pain 2011;152(3 suppl):S2–S15. doi: 10.1016/j.pain.2010.09.030.
7. Coderre TJ, Katz J, Vaccarino AL, Melzack R. Contribution of central neuroplasticity to pathological pain: review of clinical and experimental evidence. Pain 1993;52:259–285.
8. Okon T. Ketamine: an introduction for the pain and palliative medicine physician. Pain Physician 2007;10:493–500.
9. Sigtermans MJ, van Hilten JJ, Bauer MC, et al. Ketamine produces effective and long-term pain relief in patients with Complex Regional Pain Syndrome Type 1. Pain 2009;145:304–311. doi: 10.1016/j.pain.2009.06.023.
10. Schwartzman RJ, Alexander GM, Grothusen JR, Paylor T, Reichenberger E, Perreault M. Outpatient intravenous ketamine for the treatment of complex regional pain syndrome: a double-blind placebo controlled study. Pain 2009;147:107–115. doi: 10.1016/j.pain.2009.08.015.
11. Kiefer RT, Rohr P, Ploppa A, et al. Efficacy of ketamine in anesthetic dosage for the treatment of refractory complex regional pain syndrome: an open-label phase II study. Pain Med 2008;9:1173–1201. doi: 10.1111/j.1526-4637.2007.00402.x.
12. Eide PK, Jorum E, Stubhaug A, Bremnes J, Breivik H. Relief of post-herpetic neuralgia with the N-methyl-D-aspartic acid receptor antagonist ketamine: a double-blind, cross-over comparison with morphine and placebo. Pain 1994;58:347–354.
13. Nikolajsen L, Hansen CL, Nielsen J, Keller J, Arendt-Nielsen L, Jensen TS. The effect of ketamine on phantom pain: a central neuropathic disorder maintained by peripheral input. Pain 1996;67:69–77.
14. Safavi M, Honarmand A, Nematollahy Z. Pre-incisional analgesia with intravenous or subcutaneous infiltration of ketamine reduces postoperative pain in patients after open cholecystectomy: a randomized, double-blind, placebo-controlled study. Pain Med 2011;12:1418–1426. doi: 10.1111/j.1526-4637.2011.01205.x.
15. Loftus RW, Yeager MP, Clark JA, et al. Intraoperative ketamine reduces perioperative opiate consumption in opiate-dependent patients with chronic back pain undergoing back surgery. Anesthesiology 2010;113:639–646. doi: 10.1097/ALN.0b013e3181e90914.
16. Barreveld AM, Correll DJ, Liu X, et al. Ketamine decreases postoperative pain scores in patients taking opioids for chronic pain: results of a prospective, randomized, double-blind study. Pain Med 2013;14:925–934. doi: 10.1111/pme.12086.

Neuraxial Analgesia for Cardiac Surgery

Benjamin A. Abrams, MD
Cardiothoracic Fellow

Nathaen S. Weitzel, MD
Associate Professor

Department of Anesthesiology
University of Colorado Anschutz Medical Campus
Aurora, Colorado

Section Editor: Kristopher Schroeder, MD

Anesthesiologists have long debated the utility of neuraxial techniques in cardiac surgery. This approach continues to be explored as the field of cardiac anesthesia trends away from high-dose opioid regimens and toward balanced techniques that allow for expedited recoveries.[1] Increasing data support the efficacy of thoracic epidural analgesia (TEA) and intrathecal analgesia as beneficial components of these fast-track approaches. However, unique considerations in cardiac surgery patients, such as the requirement for full anticoagulation to facilitate cardiopulmonary bypass, continue to limit their application in this area. The risks associated with neuraxial techniques in this setting have potentially devastating consequences, but at the same time, they occur infrequently enough that an accurate incidence remains difficult to establish. As discussed in this review, such uncertainty is a defining feature in the debate about the application of neuraxial analgesia in cardiac surgery.

The pathophysiologies encountered in cardiac surgery offer a variety of potential mechanisms for neuraxial analgesia to exert beneficial effects. Multifactorial inflammatory responses have been correlated with a variety of adverse outcomes, including cardiovascular, pulmonary, renal, hematologic, and neurologic dysfunction, and can be attenuated in cardiac surgery patients with neuraxial analgesia.[2–4] High spinal analgesia (45-mg hyperbaric bupivacaine), as a supplement to general anesthesia (GA) in patients undergoing coronary artery bypass graft (CABG) and/or aortic valve replacement, favorably alters the net inflammatory response based on measurements of serum biomarkers.[5] High spinal analgesia (37.5-mg hyperbaric bupivacaine) also decreases serum concentrations of epinephrine, norepinephrine, and cortisol in the post–cardiopulmonary bypass (CPB) period for CABG surgery.[6]

In addition to attenuating the inflammatory response, neuraxial analgesia provides reliable sympatholysis. Neuraxial blockade for many cardiac procedures requires at least a T1 dermatome level, which results in complete sympathectomy. Blockade of cardiac accelerator fibers (T1–T4) combined with dense analgesia reduces the sympathetic response to surgical stimulation and limits myocardial oxygen demand. Additionally, TEA has been shown to reduce the incidence of supraventricular tachyarrhythmias (SVTs) in cardiac surgery when compared to GA alone, further reducing myocardial oxygen demand.[7] With respect to myocardial oxygen supply, coronary vasodilation secondary to sympathectomy can improve myocardial perfusion, particularly in the setting of coronary artery disease (CAD). In patients with multivessel CAD, TEA improves myocardial perfusion in response to sympathetic activation as demonstrated by nuclear medicine imaging.[8] Conversely, sympathectomy can potentially impair myocardial perfusion if systemic vasodilation reduces coronary perfusion pressure.

"Despite growing evidence for the efficacy of neuraxial analgesia, many anesthesiologists remain hesitant to use these approaches based on some unique considerations in the cardiac surgery population."

The improvements in myocardial oxygen balance may extend beyond sympathectomy and may also result from the excellent pain control afforded by neuraxial analgesia. Improvements in postoperative pain scores and opioid requirements have been demonstrated with TEA over GA alone.[9,10] Improved analgesia likely contributes to reductions in mechanical ventilation time and respiratory complications as well.[7,10,11]

The extreme application of neuraxial analgesia in this context is to perform cardiac surgery without general anesthesia.[12,13] Until recently, most of the reported cases were for single-vessel, off-pump CABG surgeries because of the limited options for graft harvesting sites under neuraxial anesthesia. However, Watanabe et al[14] published data from a large series of awake patients undergoing off-pump CABG for which multiple vessels could be grafted through advancements in surgical technique and regional anesthesia. For example, radial artery grafts were harvested by either extending neuraxial blockade to a C5 level or through brachial plexus nerve blocks. Additionally,

Table 1: *Recent meta-analyses of neuraxial analgesia in cardiac surgery.*[7,10,11]

Author, year	No. of studies/patients	Intervention	Significant findings	No effect
Bignami et al, 2010	33/2,366	GA versus GA+TEA	↓ARF ↓Mechanical ventilation time ↓ *Composite* mortality/MI	↔Mortality ↔MI
Svircevic et al, 2011	28/2,731	GA versus GA+TEA	↓SVT ↓Respiratory complications	↔Mortality ↔MI ↔Stroke
Zhang et al, 2015	25/3,062	GA versus GA+TEA	↓SVT ↓Respiratory complications ↓Intubation time ↓ICU time	↔Mortality ↔MI ↔Stroke

Abbreviations: ARF, acute renal failure; GA, general anesthesia; ICU, intensive care unit; MI, myocardial infarction; SVT, supraventricular tachyarrhythmia; TEA, thoracic epidural analgesia

epidural anesthesia could be extended inferiorly to cover incisions for gastroepiploic artery harvesting. The avoidance of general anesthesia certainly has the potential to enhance recovery and improve outcomes after cardiac surgery.

In recent years, multiple meta-analyses assessing the efficacy of TEA in cardiac surgery have been published (Table 1), as well as a large Cochrane review.[7,10,11,15] No clear mortality benefit has been shown, but other endpoints demonstrate advantages associated with the application of TEA for cardiac surgery. For example, a meta-analysis by Bignami et al[11] found that TEA with GA compared favorably with GA alone and resulted in a reduced incidence of acute renal failure and duration of mechanical ventilation. Notably, the composite endpoint of mortality and myocardial infarction (MI) was reduced, but the study was underpowered to detect a benefit when these endpoints were considered individually. More recently, Zhang et al[10] published a meta-analysis showing that the addition of TEA over GA alone decreased the risk of respiratory complications, SVT, time to extubation, and length of stay in the intensive care unit (ICU).

With respect to spinal analgesia for cardiac surgery, a meta-analysis by Zangrillo et al[16] found no difference in outcomes, including mortality, perioperative MI, and length of hospital stay. However, only 1 of the 25 studies used local anesthetic in the intrathecal dosing regimen. The other studies administered intrathecal opioid alone or in combination with clonidine.

Despite growing evidence for the efficacy of neuraxial analgesia, many anesthesiologists remain hesitant to use these approaches based on some unique considerations in the cardiac surgery population. In particular, full heparinization generates increased concerns about epidural hematoma and the devastating possibility of paraplegia. Additional complicating factors include the risk of post-CPB coagulopathy and the fact that medical management for many of those patients includes antiplatelet therapy. In fact, concurrent aspirin use with systemic heparinization is a known risk factor for epidural hematoma after neuraxial instrumentation.[17]

The actual risk of epidural hematoma in this setting is difficult to assess because it is a relatively rare event. From 1966–2012, 3 of 16,477 patients who received TEA for cardiac surgery suffered catheter-related epidural hematomas.[18] Based on these data, the estimated risk of epidural hematoma is 1 in 5,493 cases. A different approach applied mathematical models to estimate the frequency of a rare event that has never occurred, estimating the risk of epidural hematoma in this context to be roughly 1/1,528 and 1/3,610 for epidural and spinal techniques, respectively.[19]

Given the paucity of data to estimate risk of hematoma formation, many anesthesiologists who currently place epidural catheters for cardiac surgery remain abundantly cautious. Typical recommendation for initiating systemic heparin regimens after neuraxial instrumentation is to delay at least 1 hour after the procedure.[17] In contrast, considering the full anticoagulation

Table 2: *Recommendations for neuraxial techniques in the setting of full anticoagulation for cardiopulmonary bypass.*[17,20]

Coagulopathy	Neuraxial blocks should be avoided in patients with preexisting coagulopathies, regardless of the etiology.
Traumatic tap	Surgery should be delayed 24 hr in the event of bloody neuraxial instrumentation.
Heparin timing	Systemic heparinization should be delayed at least 60 min after neuraxial instrumentation.
Heparin dosing and reversal	Administer the smallest dose of heparin for the shortest duration possible, as compatible with therapeutic requirements for cardiopulmonary bypass.
Epidural catheter removal	Catheters should be removed only after normal coagulation has been confirmed, and close monitoring for epidural hematoma formation should be continued.

required for cardiopulmonary bypass, many anesthesiologists will place epidural catheters only on the day prior to cardiac surgery and will insist that surgery be delayed at least 24 hours in the event of bloody attempts.[20] This approach to traumatic insertion was published by the American Society of Regional Anesthesia and Pain Medicine in the most recent guidelines for regional anesthesia in patients receiving antithrombotic therapy (Table 2).[17] Unfortunately, these practice patterns are prohibitive for institutions that use same-day admit surgery. With respect to resource management, delaying surgery by 24 hours in the event of traumatic neuraxial attempts is inefficient for operating room use and adds costs secondary to prolonged hospitalizations. This reality has likely played a key role in limiting the application of neuraxial analgesia to cardiac surgery, particularly in the United States.

In addition to bleeding risks and delayed surgery, hypotension is another potential deterrent for anesthesiologists considering neuraxial techniques in this population. During the pre-CPB period, the hemodynamics associated with many of the pathologies that will, by definition, be encountered in cardiac surgery might not tolerate the sympathectomy associated with neuraxial blockade. Additionally, hypotension during the post-CPB period is relatively common and potentially quite profound, and certain etiologies, such as vasoplegia syndrome and myocardial stunning, might be exacerbated by any degree of sympathectomy. Overall, this aspect of neuraxial analgesia in cardiac surgery has received a limited amount of investigation.[21]

Interestingly, from the high spinal data from Lee et al,[5] no statistically significant difference in phenylephrine usage occurred during the pre-CPB, on-CPB, or post-CPB periods. However, a statistically significant increase in post-CPB inotrope requirements occurred in the high spinal group, which has been shown elsewhere in the literature.[22] Conversely, separate work from Lee et al[6] showed that high spinal analgesia results in less β-adrenergic receptor dysfunction, along with a statistically significant higher cardiac index in the post-CPB period.

Overall, growing evidence supports the benefits of neuraxial analgesia in cardiac surgery, including the attenuation of stress responses, improvements in myocardial perfusion, and superior pain control with reduced opioid requirements. Potential clinical benefits include reduced pulmonary complications, renal injury, and arrhythmias, as well as earlier extubation and shorter ICU stays. However, no clear mortality data support the use of neuraxial techniques in this setting.

Meanwhile, the risk of spinal cord injury from hematoma remains somewhat unclear, and evidence is limited and conflicting for the hemodynamic impacts of neuraxial blockade in these patients. As such, the application of neuraxial analgesia to cardiac surgery remains controversial until more definitive evidence becomes available.

Therefore, optimal patient selection is recommended when considering neuraxial techniques, and the risk factors for epidural hematoma and hemodynamic instability must be considered for each patient before pursuing any type of neuraxial blockade. It is also important for anesthesiologists to continue to optimize perioperative analgesic regimens through the application of thoughtful multimodal approaches (eg, acetaminophen, sternal blocks).

REFERENCES

1. Davy C. Fast track cardiac surgery pathways: early extubation, process of care, and cost containment. Anesthesiology 1998;88(6):1429–1433.
2. Levy JH, Tanaka KA. Inflammatory response to cardiopulmonary bypass. Ann Thorac Surg 2003;75(2):715–720.
3. Chaney MA. Intrathecal and epidural anesthesia and analgesia for cardiac surgery. Anesth Analg 2006;102(1):45–64. doi: 10.1213/01.ane.0000183650.16038.f6.
4. Kowaleski S, Seal D, Tang T, et al. Neuraxial anesthesia for cardiac surgery: thoracic epidural and high spinal anesthesia—why is it different? HSR Proc Intensive Care Cardiovasc Anesth 2011;3(1):25–28.
5. Lee TW, Kowalski S, Falk K, et al. High spinal anesthesia enhances anti-inflammatory responses in patients undergoing coronary artery bypass graft surgery and aortic valve replacement: randomized pilot study. PLoS One 2016;11(3):e0149942.

6. Lee TW, Grocott HP, Schwinn D, et al. High spinal anesthesia for cardiac surgery: effects on β-adrenergic receptor function, stress response, and hemodynamics. Anesthesiology 2003;98:499–510.
7. Svircevic V, Dijk DV, Nierich AP, et al. Meta-analysis of thoracic epidural anesthesia versus general anesthesia for cardiac surgery. Anesthesiology 2011;114(2):271–282. doi: 10.1097/ALN.0b013e318201d300.
8. Nygrd E, Kofoed KF, Freiberg J, et al. Effects of high thoracic epidural analgesia on myocardial blood flow in patients with ischemic heart disease. Circulation 2005;111:2165–2170. doi: 10.1161/01.CIR.0000163551.33812.1A.
9. Porizka M, Stritesky M, Semrad M, et al. Postoperative outcome in awake, on-pump, cardiac surgery patients. J Anesth 2011;25:500–508. doi: 10.1007/s00540-011-1159-7.
10. Zhang S, Wu X, Guo H, et al. Thoracic epidural anesthesia improves outcomes in patients undergoing cardiac surgery: meta-analysis of randomized controlled trials. Eur J Med Res 2015;20(1):25. doi: 10.1186/s40001-015-0091-y.
11. Bignami E, Landoni G, Biondi-Zoccai GG, et al. Epidural analgesia improves outcome in cardiac surgery: a meta-analysis of randomized controlled trials. J Cardiothorac Vasc Anesth 2010;24(4):586–597. doi: 10.1053/j.jvca.2009.09.015.
12. Watanabe G, Yamaguchi S, Tomiya S, et al. Awake subxyphoid minimally invasive direct coronary artery bypass grafting yielded minimum invasive cardiac surgery for high risk patients. Interact Cardiovasc Thorac Surg 2008;7(5):910–912. doi: 10.1510/icvts.2007.173377.
13. Karagoz HY, Sönmez B, Bakkalaglu B, et al. Coronary artery bypass grafting in the conscious patient without endotracheal general anesthesia. Ann Thorac Surg 2000;70(1):91–96.
14. Watanabe G, Tomita S, Yamaguchi S, et al. Awake coronary artery bypass grafting under thoracic epidural anesthesia: great impact on off-pump coronary revascularization and fast-track recovery. Eur J Cardiothorac Surg 2011;40:788–793. doi: 10.1016/j.ejcts.2011.01.034.
15. Svircevic V, Passier MM, Nierich AP, et al. Epidural analgesia for cardiac surgery. Cochrane Database Syst Rev 2013;(6):CD006715. doi: 10.1002/14651858.CD006715.pub2.
16. Zangrillo A, Bignami E, Biondi-Zoccai GG, et al. Spinal analgesia in cardiac surgery: a meta-analysis of randomized controlled trials. J Cardiothorac Vasc Anesth 2009;23(6):813–821. doi: 10.1053/j.jvca.2009.07.002.
17. Horlocker TT, Wedel DJ, Rowlingson JC, et al. Regional anesthesia in the patient receiving antithrombotic or thrombolytic therapy: American Society of Regional Anesthesia and Pain Medicine evidence-based guidelines (third edition). Reg Anesth Pain Med 2010;35(1):64–101.
18. Hemmerling TM, Cyr S, Terrasini N. Epidural catheterization in cardiac surgery: the 2012 risk assessment. Ann Card Anaesth 2013;16:169–177. doi: 10.4103/0971-9784.114237.
19. Ho AM, Chung DC, Joynt GM. Neuraxial blockade and hematoma in cardiac surgery: estimating the risk of a rare adverse event that has not (yet) occurred. Chest 2000;117:551–555.
20. Chaney MA. Intrathecal and epidural anesthesia and analgesia for cardiac surgery. Anesth Analg 1997;84(6):1211–1221. doi: 10.1213/01.ane.0000183650.16038.f6.
21. Casalino S, Mangia F, Stelian E, et al. High thoracic epidural anesthesia in cardiac surgery: risk factors for arterial hypotension. Tex Heart Inst J 2006;33:148–153.
22. Caputo M, Alwair H, Rogers C, et al. Thoracic epidural anesthesia improves early outcomes in patients undergoing off-pump coronary artery bypass surgery. Anesthesiology 2011;114(2):380–390. doi: 10.1097/ALN.0b013e318201f571.

Examining Advanced Modalities for Thoracic Epidural Catheter Placement

Laura Kirk, MD
Resident
Department of Anesthesiology
University of Denver, Colorado

Nathalie Lunden, MD
Assistant Professor

Ryan Ivie, MD
Assistant Professor

Department of Anesthesiology
Oregon Health and Science University
Portland, Oregon

Section Editor: Kristopher Schroder, MD

Epidural analgesia is a popular method of postoperative pain control and is particularly useful when used as part of a multimodal pain medication regimen. However, epidural catheter failure is a frequent problem. Lack of a uniform outcome measure results in heterogeneous estimates of thoracic epidural failure rates, but reported rates range from 34% for overall failure to 13% for technical failure alone.[1,2]

Technical failure is a particular challenge for the teaching physician who must facilitate a trainee's practice-based learning of a procedure for which tactile feedback is relied on to confirm correct epidural placement.[3] The role of additional modalities to confirm or facilitate epidural placement may be of notable benefit in this setting. This requires broadening our current skill set in an attempt to improve the success of thoracic epidural placement and thereby provide more reliable pain control.

Traditional methods of ensuring entry into the epidural space include loss of resistance (LOR) to air/fluid-filled syringe, hanging drop technique, and fluid column drop (drip method).[4] Of these traditional bedside techniques, loss of resistance to air and/or saline was overwhelmingly the most popular method used by those surveyed.[5] This is likely secondary to the fact that it is generally easily taught, gives decent tactile and visual feedback, and can be done efficiently in a cost-effective manner. Despite these advantages, it is still affected by significant false-positive rates (17%).[6,7]

In an attempt to improve the failure rates for epidural placement, more advanced methods of analyzing epidural catheter placement have been described (Table 1). These approaches can demonstrate not only entry into the epidural space, but also other properties of the newly placed catheter that may help predict its effectiveness in providing analgesia. These include epidural waveform analysis, nerve stimulation, ultrasonography, and fluoroscopy.

Epidural pressure waveform analysis has become a topic of study following the observation that a drop of saline hanging from the hub of a needle pulsated synchronously with the heart beat once the needle was in the epidural space.[8] It is theorized that this pulsation is a dampened waveform originating in the pulsating spinal cord and conducted through the cerebrospinal fluid and the dura mater to the epidural space.[9,10] Epidural waveform analysis can provide a simple and low cost confirmation of LOR by connecting a sterile pressure transducer to our standard operating room monitors. Leurcharusmee et al[11] conducted a blinded observational study of patients undergoing thoracic epidural placement and found a 91.1% sensitivity and 83.8% specificity with this technique. An earlier study conducted by de Medicis et al[12] in patients undergoing lumbar and thoracic epidurals had a lower sensitivity of 81%;

Table 1: *Examples of single studies comparing loss of resistance to alternative localization/confirmation techniques.*

Technique	Results versus LOR	Metric
Nerve stimulation[23]	99% versus 57%	Midline placement into epidural space at goal spinal segment confirmed by radiograph
Epidural waveform analysis[24]	98% versus 76%	Block to ice in at least two dermatomes bilaterally
Ultrasonography[17]	93.9% versus 66.7%	Successful LOR within two or fewer needle punctures
Fluoroscopy[18]	98% versus 74%	Catheter placement in the epidural space as seen on epidurogram following live fluoroscopically guided placement.

Abbreviation: LOR, loss of resistance

however, they studied the pressure waveform through the catheter, which may be less accurate. This technique can be performed quickly with equipment that is already readily available in operating rooms at low cost and minimal time.

Nerve stimulation via the epidural catheter has proven to be beneficial in confirmation of catheter placement into the epidural space. Tsui et al[13] demonstrated improvement in catheter placement confirmation and predicted function. Since that time, the Tsui Test has been described for use in postoperative analgesia, pediatric setting, chronic pain, and obstetric anesthesia. Subsequent studies have verified its high rate of sensitivity and specificity since its description.[7] Advantages include the ability to determine the spinal level of the epidural tip as well as intrathecal, subdural, and intravascular detection. One drawback may be that a specialized catheter is necessary when using bipolar electrical stimulation. However, the technique described by Tsui et al[14] uses monopolar stimulation, which can be performed with commonly available epidural catheters. Additionally, patient discomfort should be considered before performing this technique.

Regional anesthesiologists are increasingly adept at the use of ultrasonography. Therefore, using ultrasonography to assist with neuraxial techniques is a natural progression for many regionalists. Preprocedural ultrasound scanning provides reliable and accurate information on several critical aspects needed for successful epidural placement, such as the interspace level, the midline of the spine, the window between spinous processes/laminae, and depth to ligamentum flavum/dura.[15] In 2002, Grau et al[16] observed that women who received labor epidurals with ultrasound assistance had fewer attempts, more complete analgesia, and improved pain scores as compared to the LOR-only group.

"The role of additional modalities to confirm or facilitate epidural placement may be of notable benefit."

Although most of the published literature thus far has focused on the obstetric population, there has been an increased use of ultrasound guidance for thoracic epidural placement. A recent study evaluating thoracic epidural placement demonstrated no significant decrease in procedure time, but did report a reduction in pain scores in the postanesthesia care unit (PACU) and number of needle puncture sites.[17] It is important to note that all patients studied had a mean age of 58 years and body mass index of 27 kg/m^2. Considering the current thoracic data, there may be less benefit to those with low predicted difficulty. The additional time and skill required for ultrasound-assisted placement may be warranted in patients with known or anticipated difficult epidural placement because of body habitus or spinal abnormalities. Future advancements making real-time ultrasound visualization of Tuohy needle advancement more logistically feasible may dramatically improve the utility of ultrasonography in varied patient populations.

Finally, the use of fluoroscopy for catheter placement and confirmation of catheter tip position has demonstrated not only decreased failure rates but also improved patient outcomes.[18] Real-time fluoroscopic guidance allows visualization of the predicted spread of infusate by examining the pattern of dye spread on epidurogram. The improvement in catheter tip location with this technique has been associated with reduced PACU and hospital length of stay and improved pain scores.[18,19] The downside to this technique is the equipment, financial, and personnel resources required to use fluoroscopy as well as the risk of radiation exposure. These limitations have prevented fluoroscopy from becoming standard practice in the perioperative setting. However, this modality can be of great benefit in patients for whom epidural placement is known or anticipated to be difficult.

Additional, novel techniques and devices are being described that may have potential clinical applications in the future. A real-time, 3D ultrasound rendering technique with needle guide has been developed and is undergoing preliminary tests in humans.[20] A mobile optical probe mounted inside a standard epidural needle has also been developed that alarms once the tissue within the epidural space is detected. This device has thus far been tested only in animals.[21] A small ultrasound transducer, inserted into a Tuohy needle, has been used in a porcine model to detect dura mater and the epidural space.[22]

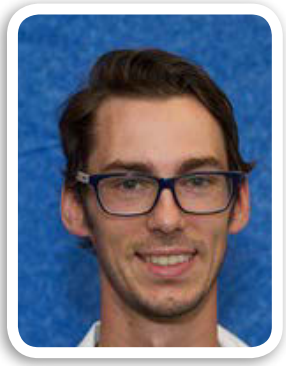
Although traditional methods of thoracic epidural catheter placement are generally simple and easily taught, the above modalities can be useful adjuncts. Financial and time constraints may dictate that some modalities are reserved for especially difficult cases, but their use should still be considered on a case-by-case basis. As evidence grows for these techniques in varied clinical circumstances, certain ones may be adopted as a standard modality, especially if they are low cost and simple. In the meantime, having the knowledge and skills to use these techniques provides the clinician with additional tools when traditional methods fail, potentially improving patients' analgesia and outcomes.

REFERENCES

1. Hermanides J, Hollmann MW, Stevens MF, Lirk P. Failed epidural: causes and management. Surv Anesthesiol 2013;57:43. doi: 10.1097/01.sa.0000424242.67406.ab.

2. McLeod G, Davies H, Munnoch N, Bannister J, MacRae W. Postoperative pain relief using thoracic epidural analgesia: outstanding success and disappointing failures. Anaesthesia 2001;56:75–81.
3. Johnson T. Counterbalancing clinical supervision and independent practice: case studies in learning thoracic epidural catheter insertion. Br J Anaesth 2010;105:772–776. doi: 10.1093/bja/aeq233.
4. Michel MZ, Lawes EG. Identification of epidural space by drip method. Reg Anesth Pain Med 1991;16(4):236–239.
5. Wantman A, Hancox N, Howell PR. Techniques for identifying the epidural space: a survey of practice amongst anaesthetists in the UK. Anaesthesia 2006;61:370–375. doi: 10.1111/j.1365-2044.2006.04534.x.
6. Sharrock NE. Recordings of, and an anatomical explanation for, false positive loss of resistance during lumbar extradural analgesia. Br J Anaesth 1979;51(3):253–258.
7. Tran DQ, Gonzalez AP, Bernucci F, Finlayson RJ. Confirmation of loss-of-resistance for epidural analgesia. Reg Anesth Pain Med 2015;40:166–173. doi: 10.1097/AAP.0000000000000217.
8. Aldrete J, Auad O, Gutierrez V, Wright A. Alberto Gutierrez and the hanging drop. Reg Anesth Pain Med 2005;30:397–404.
9. Ghia J. Confirmation of location of epidural catheters by epidural pressure waveform and computed tomography cathetergram. Reg Anesth Pain Med 2001;26:337–341.
10. Lennox PH, Umedaly HS, Grant RP, et al. A pulsatile pressure waveform is a sensitive marker for confirming the location of the thoracic epidural space. J Cardiothorac Vasc Anesth 2006;20:659–663. doi: 10.1053/j.jvca.2006.02.022.
11. Leurcharusmee P, Arnuntasupakul V, Chora De La Garza D, et al. Reliability of waveform analysis as an adjunct to loss of resistance for thoracic epidural blocks. Reg Anesth Pain Med 2015;40:694–697. doi: 10.1097/AAP.0000000000000313.
12. de Medicis E, Tetrault JP, Martin R, Robichaud R, Laroche L. A prospective comparative study of two indirect methods for confirming the localization of an epidural catheter for postoperative analgesia. Anesth Analg 2005;101:1830–1833. doi: 10.1213/01.ANE.0000184130.73634.BE.
13. Tsui B, Gupta S, Finucane B. Confirmation of epidural catheter placement using nerve stimulation in obstetric patients: the tsui test. Reg Anesth Pain Med 1998;24:17–23.
14. Tsui B, Finucane B. Verifying accurate placement of an epidural catheter tip using electrical stimulation. Anesth Analg 2002;94:1670. doi: 10.1213/00000539-200206000-00063.
15. Balki M. Locating the epidural space in obstetric patients-ultrasound a useful tool: continuing professional development. Can J Anaesth 2010;57:1111–1126. doi: 10.1007/s12630-010-9397-y.
16. Grau T, Leipold RW, Conradi R, Martin E, Motsch J. Efficacy of ultrasound imaging in obstetric epidural anesthesia. J Clin Anesth 2002;14:169–175.
17. Auyong DB, Hostetter L, Yuan SC, Slee AE, Hanson NA. Evaluation of ultrasound-assisted thoracic epidural placement in patients undergoing upper abdominal and thoracic surgery: a randomized, double-blind study. Reg Anesth Pain Med 2017;42:204–209. doi: 10.1097/AAP.0000000000000540.
18. Parra MC, Washburn K, Brown JR, et al. Fluoroscopic guidance increases the incidence of thoracic epidural catheter placement within the epidural space: a randomized trial. Reg Anesth Pain Med 2017;42:17–24. doi: 10.1097/AAP.0000000000000519.
19. Yeager MP, Bae EE, Parra MC, et al. Fluoroscopy-assisted epidural catheter placement: an exploratory analysis of 303 pre-operative epidurograms. Acta Anaesthesiol Scand 2016;60:513–519. doi: 10.1111/aas.12649.
20. Beigi P, Malenfant P, Rasoulian A, et al. Three-dimensional ultrasound-guided real-time midline epidural needle placement with epiguide: a prospective feasibility study. Ultrasound Med Biol 2017;43:375–379. doi: 10.1016/j.ultrasmedbio.2016.08.033.
21. Gong CS, Lin SP, Mandell MS, et al. Portable optical epidural needle—a CMOS-based system solution and its circuit design. PLoS One 2014;9:e106055. doi: 10.1371/journal.pone.0106055.
22. Chiang HK, Zhou Q, Mandell MS, et al. Eyes in the needle: novel epidural needle with embedded high-frequency ultrasound transducer—epidural access in porcine model. Anesthesiology 2011;114:1320–1324. doi: 10.1097/ALN.0b013e31821b5746.
23. Hayatsu K, Tomita M, Fujihara H, et al. The placement of the epidural catheter at the predicted site by electrical stimulation test. Anesth Analg 2001;93:1035–1039.
24. Arnuntasupakul V, Van Zundert TC, Vijitpavan A, et al. A randomized comparison between conventional and waveform-confirmed loss of resistance for thoracic epidural blocks. Reg Anesth Pain Med 2016;41:368–373. doi: 10.1097/AAP.0000000000000369.

Pediatric Regional Anesthesia and Chronic Postoperative Pain



Jacob Bray, MD
Resident

Lynda Wells, MD
Associate Professor

Department of Anesthesiology
University of Virginia Health System
Charlottesville, Virginia

Section Editor: Lynn Kohan, MD

Recent literature has established that regional anesthesia in the pediatric population is safe and effective for acute postoperative pain control. Publications from the Pediatric Regional Anesthesia Network (PRAN) have largely established the safety profile of peripheral nerve blocks and neuraxial procedures in the pediatric population, performed while under general anesthesia and while awake.[1,2] Although the techniques offer advantages in helping to control acute postoperative pain, the question of whether regional anesthesia offers advantages in decreasing the development of chronic postsurgical pain (CPSP) is still unresolved.

The incidence and prevalence of CPSP in the pediatric population have previously been poorly described.[3] Typically, CPSP is defined as pain affecting the surgical area for more than 3 months postoperatively. Unfortunately, the literature largely varies when describing other aspects of CPSP such as pain severity, frequency, and social, physical, and functional limitations. In recent studies, follow-up data also largely omit important social, mental, and physical limitation as a result of CPSP. Clinically relevant outcomes other than the presence of pain include physical limitations, school days missed, social isolation, and pain-related anxiety. These data points have been largely absent from the limited retrospective and prospective studies available.

The prevalence of CPSP in the adult population can range from 20–80%, depending on the type of surgery.[4] In a recent publication, Batoz et al[5] sought to prospectively evaluate the incidence of CPSP in the pediatric population aged 6–18 years. After observing 258 children, they found a 10.9% prevalence of CPSP. Previous reports by Pagé et al[6] and Aasvang et al[7] found similar results when they looked at mixed surgical procedures with a 22% prevalence and inguinal hernia repairs with a 13.5% prevalence, respectively.

"The question of whether regional anesthesia offers advantages in decreasing the development of chronic postsurgical pain is still unresolved."

Evidence suggests that preemptively preventing peripheral and central sensitization to noxious stimulation by a multimodal analgesic approach can help limit the development of chronic pain. Regional and neuraxial anesthesia have been a key component to various multimodal approaches in various enhanced recovery protocols. Paravertebral blocks and epidural anesthesia have been found to be effective in reducing the occurrence of CPSP in the adult population, although mixed results have been published regarding other types of regional blocks.[8,9] Unfortunately, no high-quality study to date has evaluated the effect of regional or neuraxial anesthesia on the development of CPSP in pediatric population. Batoz et al[5] reported that 163 of 258 patients underwent some type of regional nerve block. Of those 163 patients, 19 (11.7%) went on to develop CPSP.[5] They reported that regional anesthesia was not found to be a risk factor for developing CPSP, although their study was not designed to determine the effects that regional anesthesia may have on the development of CPSP. Thus, no inferences can be made regarding the potential positive or null benefit this population would possibly obtain.

Common types of pediatric surgical procedures that seem to have a propensity for the development of CPSP are major orthopedic procedures, thoracotomies, and inguinal hernia repairs. These surgeries often lend themselves readily to peripheral or neuraxial regional anesthesia techniques as part of multimodal pain regimens. Previously mentioned studies have identified acute pain after surgery as a risk factor for developing CPSP in adults.[4]

Recent advancements in the understanding and implementation of multimodal analgesia in adults in enhanced recovery protocols have led to reduced postoperative pain scores, earlier hospital discharges, and reduced opioid consumption.[10] The development of enhanced recovery programs and use of regional and neuraxial techniques could help reduce the development of CPSP in the pediatric population. Currently, evidence is insufficient to support or oppose regional anesthesia in the pediatric population as a potential adjunct to limit CPSP. Although PRAN has clearly demonstrated the safety of pediatric regional anesthesia, continued work is needed to demonstrate how pediatric regional and neuraxial anesthesia may affect acute postoperative pain and CPSP.

REFERENCES

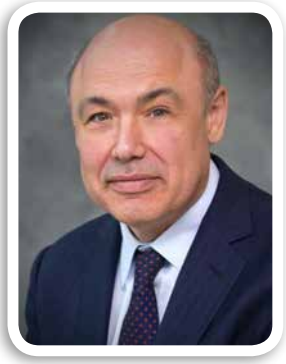
1. Taenzer AH, Walker BJ, Bosenberg AT, et al. Asleep versus awake: does it matter—pediatric regional block complications by patient state: a report from the Pediatric Regional Anesthesia Network. Reg AnesthPain Med 2014;39:279–283. doi: 10.1097/AAP.0000000000000102.
2. Suresh S, Long J, Birmingham P, De Oliveira GS. Are caudal blocks for pain control safe in children: an analysis of 18,650 caudal blocks from the pediatric regional anesthesia network (PRAN) database. Anesth Analg 2015;120(1):151–156. doi: 10.1213/ANE.0000000000000446.
3. Nikolajsen L, Brix LD. Chronic pain after surgery in children. Curr Opin Anesthesiol 2014;27(5):507–512. doi: 10.1097/ACO.0000000000000110.
4. Katz J, Seltzer Z. Transition from acute to chronic postsurgical pain: risk factors and protective factors. Expert Rev Neurother 2009;9(5):723–744. doi: 10.1586/ern.09.20.
5. Batoz H, Semjen F, Bordes-Demolis M, Bernard A, Nouette-Gaulain K. Chronic postsurgical pain in children: prevalence and risk factors: a prospective observational study. Br J Anaesth 2016; 117(4):489–496. doi: 10.1093/bja/aew260.
6. Pagé MG, Stinson J, Campbell F, et al. Identification of pain-related psychological risk factors for the development and maintenance of pediatric chronic postsurgical pain. J Pain Res 2013; 6:167–180. doi: 10.2147/JPR.S40846.
7. Aasvang EK, Kehlet H. Chronic pain after childhood groin hernia repair. J Pediatr Surg 2007;42:1403–1408. doi: 10.1016/j.jpedsurg.2007.03.042.
8. Andreae MH, Andreae DA. Regional anaesthesia to prevent chronic pain after surgery: a Cochrane systematic review and meta-analysis. Br J Anaesth 2013;111(5):711–720. doi: 10.1093/bja/aet213.
9. Bong CL, Samuel M, Ng JM, Ip-Yam C. Effects of preemptive epidural analgesia on post-thoracotomy pain. J Cardiothorac Vasc Anesth 2005;19:786–793. doi: 10.1053/j.jvca.2005.08.012.
10. Shah PM, Johnston L, Sarosiek B, et al. Reducing readmissions while shortening length of stay: the positive impact of an enhanced recovery protocol in colorectal surgery. Dis Colon Rectum 2017;60(2):219–227. doi: 10.1097/DCR.0000000000000748.

Stellate Ganglion Block for Posttraumatic Stress Disorder: A Call for the Complete Story, and Continued Research

I read with interest the report of Hanling et al[1] in the May issue of the *ASRA News* "Stellate Ganglion Block for Posttraumatic Stress Disorder: A Call for Clinical Caution and Continued Research." The authors suggested in the report that stellate ganglion block (SGB) lacks efficacy for the treatment of posttraumatic stress disorder (PTSD). Essentially, the *ASRA News* article was a summary of a 2016 publication in *Regional Anesthesia and Pain Medicine,* where the first randomized, blinded, sham-controlled study was performed at the Naval Medical Center San Diego.[2] The purpose of the current communication is to clarify certain comments as to SGB use for PTSD treatment.

From a historical perspective, we were the first to report the use of SGB for treating PTSD in 2008.[3] The following should be noted:

1. SGB should be performed on the right side to address PTSD: "This is likely because initial case series happened to be performed on the right side in the patients with pain conditions."[1] Actually, our 2010 article discussed SGB as being performed by using right-sided preference and that this was necessary because of the known right-sided amygdala activation noted in PTSD[4] as well as subsequent descriptions by Alkire et al[5] in 2015. Furthermore, SGB was used to treat PTSD because of a rational prediction of the effect, based on the work of Telaranta[6] with endoscopic thoracic sympathectomy used to treat anxiety and PTSD. The history of the evolution of SGB as a viable treatment for PTSD was documented in a 2012 publication by our team in the peer-reviewed *Journal of Affective Disorder*.[7]
2. "Correlation with current functional MRI (fMRI) studies has not provided a convincing model to date."[1] That is true; however, no fMRI studies have been conducted to assess this issue. Yet, neuroimaging studies have been done and were published by Alkire et al[5] in 2015. They used two separate fluorodeoxyglucose positron-emission tomography (PET) brain scans, the first prior to SGB and the second post SGB. PET is considered by some as being more specific than fMRI for the diagnosis of PTSD. The patients in the PET study were drawn from the VA Long Beach Healthcare System in Long Beach, California. The authors' conclusions were as follows: "SGB had efficacy for significantly reducing PTSD symptoms in a rapid and sustained manner."[5] The right amygdala and hippocampal areas appear to be relatively overactive when PTSD symptoms are prominent and become deactivated following SGB.[5] Alkire et al[5] used the Clinically Administered PTSD Scale (CAPS) as the diagnostic and follow-up tool for PTSD (as did Hanling et al[1] in their study). On follow-up, Alkire et al[5] were able to demonstrate CAPS reduction that mirrored right-sided amygdala deactivation. The 2016 report by Hanling et al[x] has recently been evaluated by a Veterans Administration evidence-based synthesis program.[8] The determinations of this synthesis are summarized below.



Eugene Lipov, MD
Medical Director
Pain Clinic
Department of Anesthesiology
Illinois Masonic Medical Center
Chicago, Illinois

The study essentially compared ultrasound-guided SGB with 5 mL of 0.5% ropivacaine to an inactive sham SGB procedure performed with normal saline solution in 42 male military participants with both combat and noncombat PTSD. SGB was administered on the right side of the neck, generally at the C6 level.[8]

Although in previous case series the most commonly used anesthetic type and dosage have been 7 mL of ropivacaine or a bupivacaine 0.5% solution, this trial used 0.5% ropivacaine, a 28% lower dose, and provided no rationale for doing so. Although the stellate ganglion is typically located anatomically between C6 and C7, the level of target needle placement was C5 to C6 in this study.[8] Although the study authors confirmed that the injection was typically at C6, some could have been at C5 and may have missed the stellate ganglion.[8] The use of saline instead of an active control that mimicked the side effects of SGB was potentially inadequate and may have reduced the effectiveness of the blinding, as patients may have been able to easily tell if they received local anesthetic SGB or the sham block, based on the occurrence of the Horner's syndrome–expected ptosis. Effectiveness of the blinding was not formally assessed.[8] Finally, there were more active-duty participants in the SGB group (96% vs 73%), attrition was high overall (57%)—primarily because they were "lost to follow-up at 3 month post treatment or completed outside of 3 month posttreatment window"—and was higher in the SGB group (67% vs 40%), and the study did not report on or account for potential between-group differences in concurrent PTSD treatments.[8] "Because these findings come from a single study with imprecise findings, moderate methodological limitations, and did not directly focus on clinically relevant outcomes or use the most commonly administration techniques and anesthetics, they provide an insufficient basis upon which to draw conclusions about SGB for the treatment of PTSD in Veterans."[8] At the conclusion of the *ASRA News* article by Hanling et al,[1] a question was raised as to why the results are so different between the retrospective study by Mulvaney et al[9] in which 166 patients had marked improvement and the study conducted at

the Naval Medical Center San Diego that did not do better than placebo. One large difference is the population group that Hanling et al[2,8] studied. The patients undergoing SGBs may have been inappropriate for the study because most were in the process of undergoing a disability evaluation and may have had secondary financial incentives to resist treatment.[2,8] Marked improvement in PTSD symptoms has been shown at five independent medical institutions: Mulvaney et al[9] at Walter Reed Medical Center; Alino et al[10] at Tripler Army Hospital; Alkire et al[5] at Long Beach, California, Veterans Administration; Hicky et al[11] at the Naval Medical Center San Diego; and Lipov et al[3,4] at the Advanced Pain Centers. To date, more than 2,500 military personnel have been treated with SGB with good to very good success (unpublished).

Hanling et al[1] went on to discuss potential rare but "catastrophic risk" of SGB as one of the reasons SGB should not be used to treat PTSD. Wulf and Maier[12] conducted a single, large study of SGB risks in 1992 (pre ultrasound or fluoroscopic-guidance era) in which 45,000 SGBs were performed. The incidence of severe complications was 1.7 in 1,000 blockades. No fatalities were reported. Most complications were related to the central nervous system toxicity from rapid local anesthetic absorption (ie, convulsions).[12] A high subarachnoid block was reported in six cases, high epidural blockade in three, pneumothorax in nine, and allergic reactions in two patients.[12] It is likely that in the current ultrasound and/or fluoroscopic guidance era, where imaging is widely accepted as being a standard of care, further reductions will occur in reported complications. Given the known suicide risk associated with PTSD of 22 per day,[13] a possible complication rate of 1.7 out of 1,000 pales by comparison (0.17%). Furthermore, PTSD symptoms are positively correlated with suicide risk.[14] Finally, SGB has been reported to impact suicidal ideation.[10,15]

In summary, I believe that the randomized controlled trial by Hanling et al[2] should not prevent practitioners from offering SGB as a valued and safe treatment for PTSD. A well-powered study is being conducted in three sites at Womack Army Medical Center, with disability evaluation patients excluded for reasons associated with secondary gain issues. One of the limitations of the current study is the lack of fMRI response monitoring. Veterans Administration evidence-based synthesis program recommends an fMRI evaluation in a study where SGB is used to treat PTSD symptoms.[8]

REFERENCES

1. Hanling S, Fowler I, Hackworth R. Stellate ganglion block for posttraumatic stress disorder: a call for clinical caution and continued research. ASRA News. May 2017;10–14.
2. Hanling SR, Hickey A, Lesnik I, et al. Stellate ganglion block for the treatment of posttraumatic stress disorder: a randomized, double-blind, controlled trial. Reg Anesth Pain Med 2016;41(4):494–500. doi: 10.1097/AAP.0000000000000402.
3. Lipov EG, Joshi JR, Lipov, Sanders SE, Siroko MK. Cervical sympathetic blockade in a patient with post-traumatic stress disorder: a case report. Ann Clin Psychiatry 2008;20(4):227–228. doi: 10.1080/10401230802435518.
4. Lipov E. Successful use of stellate ganglion block and pulsed radiofrequency in the treatment of posttraumatic stress disorder: a case report. Pain Res Treat 2010;2010:963948. doi: 10.1155/2010/963948.
5. Alkire MT, Hollifield M, Khoshsar R, Nguyen L, Alley S, Reis, C. Neuroimaging suggests that stellate ganglion block improves post-traumatic stress disorder (PTSD) through an amygdala mediated mechanism. Paper presented at: Anesthesiology 2015 Annual Meeting; October 24, 2015; San Diego, CA. Available at: http://www.asaabstracts.com/strands/asaabstracts/abstract.htm;jsessionid=0CAC170518A556F620F2245A983B8314?year=2015&index=5&absnum=3003. Accessed September 3, 2017.
6. Telaranta T. Treatment of social phobia by endoscopic thoracic sympathectomy. Eur J Surg 1998;164(S1):27–32.
7. Lipov E, Kelzenberg B. Sympathetic system modulation to treat post-traumatic stress disorder (PTSD): a review of clinical evidence and neurobiology. J Affect Disord 2012;142(1–3):1–5. doi: 10.1016/j.jad.2012.04.011.
8. Center C, Peterson K, Bourne D, Anderson J, Mackey K, Helfan, M. Evidence Brief: Effectiveness of Stellate Ganglion Block for Treatment of Posttraumatic Stress Disorder (PTSD). Washington, DC: Department of Veterans Affairs; 2017.
9. Mulvaney SW, Lynch JH, et al. Stellate ganglion block used to treat symptoms associated with combat-related post-traumatic stress disorder: a case series of 166 patients. Mil Med 2014;179(10):1133–1140. doi: 10.7205/MILMED-D-14-00151.
10. Alino J, Kosatka D, McLean B, Hirsch K. Efficacy of stellate ganglion block in the treatment of anxiety symptoms from combat-related post-traumatic stress disorder: a case series. Mil Med 2013;178(4):e473–e476. doi: 10.7205/MILMED-D-12-00386.
11. Hicky A, Hanling, S, Pevney E, Allen R, McLay RN. Stellate ganglion block for PTSD. Am J Psychiatry 2012;169(7):760. doi: 10.1176/appi.ajp.2012.1111172.
12. Wulf H, Maier C. Complications and side effects of stellate ganglion blockade: results of a questionnaire survey. Anaesthesist 1992;41(3):146–151.
13. Kemp J, Bossarte R. Suicide Data Report: 2012. Washington, DC: Department of Veterans Affairs, Mental Health Services, Suicide Prevention Program; 2013.
14. McKinney JM, Hirsch JK, Britton PC. PTSD symptoms and suicide risk in veterans: serial indirect effects via depression and anger. J Affect Disord 2017;214:100–107. doi: 10.1016/j.jad.2017.03.008.
15. Lipov E. Marked impact on suicidal ideation and suicidology in a military populations following a cervical sympathetic blockade. In: Guide to the 2015 Annual Meeting: Integrating Body and Mind, Heart and Soul; May 14, 2015; Toronto, Canada. Abstract 802.

The Authors Respond

Steven Hanling, MD, CDR, MC, USN
Navy Pain Medicine Specialty Leader
Staff Pain Medicine Physician and Anesthesiologist

Ian Fowler, MD, CDR, MC, USN
Director Pain Medicine Center & Fellowship Program
Staff Pain Medicine Physician and Anesthesiologist

Robert Hackworth, MD
Staff Pain Medicine Physician and Anesthesiologist

Naval Medical Center San Diego
San Diego, California

We would like to thank Dr Lipov for his interest in our work. The goal of the *ASRA News* article was to review (1) the status quo of research on the use of stellate ganglion block (SGB) for the treatment of posttraumatic stress disorder (PTSD), (2) its implication on current clinical practice, and (3) to *encourage continued research* while taking reasonable steps to ensure patient safety.

Without addressing each of Dr Lipov's statements point by point, we will leave it to the reader to study all sources of information and come to reasonable conclusions based on available evidence. The *ASRA News* article can serve as a guide to the literature even if your assessment of the literature differs from ours.

We would, however, like to respond to a few specific items. Dr Lipov commented on how the characteristics of the study population may have affected the results of our study.

The potential impact of the study population and the methodology has been consistently discussed by each of the authors at live meetings, within our previously published article, and in the recent *ASRA News* article. The conclusion of our study and the abstract presented at the American Academy of Pain Medicine 2015 annual meeting[1] was as follows:

- "We cannot demonstrate any advantage of SGB over sham injection for the treatment of PTSD.
- It is possible that SGB was underdosed, or that there are subpopulations that benefit.
- SGB for PTSD is supported by evidence from case series, but this RCT did not support those findings."

The letter also mentioned the previous positive trials. These positive trials were noted in the *ASRA News* article and include our own previous publication of a small case series showing success of SGB for PTSD at Naval Medical Center San Diego noted in the letter as reference 13. It was this very success that led us to want to perform further research.

As for the comment concerning SGB-related complications, we agree that catastrophic or severe complications are rare but do occur as noted by Dr Wulf's article (reference 12 in the letter), which reported on a survey of 76 departments in West Germany with a response rate of 51%. Therefore, the results are subject to responder bias and potential underreporting of catastrophic outcomes because of legal concerns or market forces. Regardless of whether such bias exists, our point on risk was meant to alert all clinicians who perform the procedure to study the literature and available information for side effects and harm that can result from this intervention. As mentioned in our letter, although rare, catastrophic events can and do happen and performing this intervention should not be taken lightly. Care and caution should always be used with SGBs.

Dr Lipov also wrote, "To date, more than 2,500 military personnel have been treated with SGB with good to very good success (unpublished data)."

The authors are unable to comment on the safety and efficacy results of unpublished data.

REFERENCES

1. Medina-Torne S, Hanling S, Lesnik I, et al. US Navy's First Functional Restoration Pain Program: improving readiness, restoring function, and relieving pain. Paper presented at: 2015 American Academy of Pain Medicine Annual Meeting; March 19–22, 2015; Washington, DC. Available at: http://www.painmed.org/2015posters/abstract-172/. October 5, 2017.