

ASRA NEWS

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American Society of Regional Anesthesia and Pain Medicine

520 N. Northwest Highway • Park Ridge, IL • 60068-2573
 phone: (847) 825-5586 • fax: (847) 825-5658 • www.asra.com

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President's Message

I would like to send a note of genuine appreciation to all of the ASRA members who took the time to complete the electronic membership survey this past year. The survey results are essential as we begin strategic planning to determine the direction of the society for the next several years. We obtained critical information about member demographics, specific topics of interest, areas for improvement, and how the members would like to be more involved with the society. Here is a summary of the survey results.

Demographics

Of the approximately 4,500 ASRA members, 1,122 completed the survey for a response rate of about 25 percent. According to our consultants, this is considered an incredibly high response rate. The members of our society are 77 percent male and 23 percent female, with two peaks in age groups 31-45 years old (42 percent) and 46-65 years old (46 percent). The largest areas of subspecialty interest are regional anesthesia (83 percent), acute pain management (72 percent), chronic pain management (39 percent), obstetric anesthesia (37 percent), cancer pain management (25 percent) and pediatric anesthesia (23 percent). The majority of members are in a hospital-based academic/teaching practice (52 percent) with the next largest group of members (32 percent) in private practice.

In General

Overall summary responses were very positive; however, there were some specific areas noted for potential improvement. Members responded that they belong to ASRA to improve their knowledge and skills as an anesthesiologist, and the majority (88 percent) were very satisfied with their membership in ASRA. The following benefits were noted to be very important to ASRA members and areas in which they were satisfied with the current products — Regional Anesthesia and Pain Medicine journal (94 percent), promotion of research (86 percent), ASRA Practice guidelines (84 percent) and the Annual Meetings (68 percent).

Areas for Improvement

Despite the fact that overall survey results were very positive, there were two specific areas noted for improvement — one concerned educational products and benefits that members would like to receive, and the second was that members (55 percent) would like the opportunity to become more involved in the society. Asking members to complete a survey is only the first step for the society during an improvement process. The second step involves what actions will follow in response to the survey results. Therefore, I would like to address each of these two areas for potential improvement.

Educational Products and Benefits

Members suggested that they would like more information on practice management programs (44 percent) and information about reimbursement issues (42 percent). The primary mission of ASRA is that of an education and research society and not an advocacy society. Although we do not intend to change the primary mission of the society from education and research to advocacy, we are committed to providing information that members believe to be relevant to their practice. So even though you will not see billing and reimbursement refresher courses or workshops at any upcoming meeting, you can expect to hear those topics discussed in a panel presentation. The changing landscape of anesthesia reimbursement is particularly challenging and may vary from state to state. We will try to present these topics in an up-to-date and thoughtful manner. ASRA is particularly fortunate in that we have several members of the society who are involved and very knowledgeable about reimbursement issues for both pain management and regional anesthesia at the national and legislative level.



Julia E. Pollock, M.D.

Involvement in the Society

Only 40 percent of members believe that ASRA had offered them an opportunity to participate in the society's activities, and 55 percent would like to be more involved with the society. Sixty percent would like to participate in teaching activities, and 55 percent would like to participate in an ASRA committee. Because the annual meetings are the biggest educational component of what ASRA offers its members, we are committed to guaranteeing only the highest-quality instruction. Because of this, we will continue to insist that instructors and lecturers come highly recommended and have been previously observed in a teaching situation. Thus the most obvious way for members who would like to become more involved in ASRA is for them to join one of ASRA's committees. There currently are six ASRA committees—Communications, Continuing Medical Education, Membership, Newsletter, Research and Industry Relations. Each of these committees is anxious for new members who are committed, thoughtful and willing to work hard. ASRA is currently initiating a process similar to the one utilized

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Editorial

ASRA: Going International



Colin J.L. McCartney, M.B., Ch.B.,
F.R.C.P.C.
Editor, ASRA News
Department of Anesthesia
Sunybrook Health Sciences Centre
University of Toronto

particularly well placed to answer the question, “How Do Local Anesthetics Make Us Numb?” Finally, Drs. Flagg and Narouze maintain the recent quality of articles on chronic pain with their contributions on “Intrathecal Pumps for Chronic Pain” and “MILD.” Thanks to all authors for their contributions to this issue, but in particular to the newsletter committee who maintains incredible energy in providing ideas and authors for the articles that fill each issue.

I am always interested in meeting new people in my travels and discovering a little about the background of colleagues from other continents. At the recent NWAC conference in Rome, I was sitting next to such a colleague on a bus travelling back to the conference hotel.

This issue of the newsletter features several articles by leading clinicians and scientists in the specialty of regional anesthesia and pain medicine. Brendan Finucane, M.B., B.Ch., from the University of Alberta, has long had an interest in the origins of our specialty and provides a fascinating article detailing “Regional Anesthesia: How It All Began” Gary Strichartz, Ph.D., from Harvard Medical School, a leading scientist in the investigation of many aspects of pain, has long been an expert in the field of local anesthetic mechanisms. He is therefore par-

“Professor Ezzat Aziz is a complex and impressive individual. Both a consultant anesthetist in England and a professor of anesthesia in Cairo, Egypt, he now has the distinction of being the Founding President of the African Society of Regional Anesthesia (AFSRA).”

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Prof. Aziz recently helped to organize the inaugural conference for the society, which took place in Cairo. While AFSRA has only recently been established, there were over 900 anesthesiologists present at this conference, including Vincent W. S. Chan, M.D., ASRA Immediate Past President. This marked one of the first conferences on regional anesthesia in both Egypt and the African continent, introducing doctors to regional anesthetic techniques that may benefit their patients and practice. Presentations on the use of ultrasound, complications in regional anesthesia and the use of continuous peripheral nerve blocks were some of the many presentations that focused on encouraging and improving the use of regional anesthesia.

Before the establishment of AFSRA (www.afsra.org), Africa was the only continent without a regional anesthesia society. Professor Aziz states that, “[AFSRA] was started to promote regional anesthesia as a simple and safe anesthetic option in African hospitals.” With over 50 members mainly from Egypt, South Africa, Kenya and abroad, the society hopes to expand the practice and promote regional anesthesia through regular workshops held throughout the continent.

In 2012, the ASFRA annual conference will be held on July 30-31 in Nairobi, Kenya and, ultimately, ASFRA hopes to be able to host the World Congress of Regional Anesthesia.

We wish Professor Aziz and his colleagues every success with their vision to promote the use of regional anesthesia in Africa. Enjoy the August issue and the rest of your summer.

President's Message

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the American Society of Anesthesiologists, which allows members to self-nominate to committees. You can expect to see this on the ASRA website within the next couple of months. I encourage everyone who is anxious to be more involved with ASRA to select the committee that they are most interested in and self-nominate!

In conclusion — thanks again to all of the members who took the time to complete ASRA’s online survey. It would be impossible for the society to improve without your thoughtful input. Additionally — for those of you who would like to be more involved with ASRA—we welcome your energy and ideas and encourage you to become involved.

ASRA E-News Bulletin: News You Can Use Wherever You Are

The ASRA E-News Bulletin is the brainchild of Immediate Past President Vincent W.S. Chan, M.D., and has been bringing updated news and events bimonthly to ASRA members since February 2010. In its concise all-electronic format, E-News provides members with the latest information on upcoming conferences and workshops as well as a detailed calendar of events, commentaries on the top abstracts in regional anesthesia and pain medicine as selected by the E-News committee members, and brief evidence-based review articles on emerging practice trends related to the specialties of regional anesthesia and pain medicine. Previously available only online, the E-News Bulletin is now delivered directly to members via email in an easy-to-read layout (even on your smartphone) for busy members on the go, in the operating room or waiting in line at the DMV.

If you haven't checked out E-News yet, do you want to know what you've been missing? In addition to expert commentary on the top need-to-know articles of the preceding two months, E-News has also featured one article per issue on radiation safety, opioid pharmacology, emerging technology, updates on billing and coding for regional anesthesia, controversies related to certain pain procedures, and strategies to prevent surgical site infection for interventional pain physicians.

I want to thank the members of the E-News Committee, Drs. Ellen King, Colin McCartney, Steven Orebaugh, David Provenzano, and Chris Wu, as well as Julie Kahlfeldt and Jim McGreal, for their hard work and creative contributions to this ongoing project.

If you have ideas for new content or suggested article topics, please send them to me at emariano@stanford.edu.



Edward R. Mariano, M.D., M.A.S.
Associate Professor of Anesthesia
Stanford University
School of Medicine
Chief, Anesthesiology and
Perioperative Care
VA Palo Alto Health Care System



American Society of Regional Anesthesia and Pain Medicine

E-News - May, 2011

In This Issue

- 36th Annual Regional Anesthesia Meeting and Workshops
- 2011 Workshops
- E-News Picks the Top Abstracts in Regional Anesthesia and Pain Medicine
- Prevention of Surgical Site Infection

Calendar

May 2-4
ASA Legislative Conference
American Society of Anesthesiologists

May 21, 2011
Controversies and Fundamentals in Regional Anesthesia" - 15th Annual Symposium

36th Annual Regional Anesthesia Meeting and Workshops



Join us for the 36th Annual Regional Anesthesia Meeting and Workshops in beautiful Las Vegas, Nevada.

- Meeting Site
- Preliminary Program Brochure (PDF - 1.7 MB)
- Abstract Submission

Regional Anesthesia: How It All Began



**Brendan T. Finucane, M.B., B.Ch.,
B.A.O., F.R.C.A., F.R.C.P.C.**
Professor Emeritus
Department of Anesthesiology and
Pain Medicine
University of Alberta
Edmonton, Alberta, Canada

Section Editor:
Derek Dillane, M.D.

"In the field of observation chance favours only the prepared mind."

- Louis Pasteur

It would be very difficult to write a comprehensive history of regional anesthesia (RA) in a few thousand words; therefore, I will highlight the most important event that led to the development of regional anesthesia, and that of course was the discovery of local anesthesia. Although the origin of local anesthesia can be traced back to the time of the Incas (Figure 1), the real pursuit began in 1855 when Gaedcke¹ prepared an amorphous substance from the coca plant and named it erythroxyline.

Almost 30 years later a pivotal event took place in Vienna in the summer of 1884 that was the breakthrough to the discovery of local anesthesia.

Few will argue that RA began with Koller's brilliant observations of the effects of cocaine on the surface of the eye. It was the final piece of the puzzle required to solve the mystery that many toyed with decades before. Koller was only 27 when he made this discovery. However, it wasn't just happenstance that led Koller to make this breakthrough. Koller was a scientist in his own right having worked in Professor Stricker's laboratory for a number of years before his landmark discovery. Stricker was an experimental pathologist and supervised Koller's research on embryology. Koller published an important paper on the mesoderm² while under Stricker's supervision; therefore, he was fully familiar with research methodology. But Koller was also studying to be an ophthalmologist, and he was very concerned about the anesthesia issues surrounding eye surgery.

"The unsuitability of general narcosis for eye operations; for not only is the co-operation of the patient greatly desirable in these operations, but the sequelae of general narcosis-vomiting, retching and general restlessness-are frequently such as to constitute a grave danger to the operated eye; and this was

*especially the case at the time when narcosis was not skilfully administered as it is now, by trained experts. Eye operations were formerly being done without any anesthesia whatsoever"*³

It is clear from Koller's notes that he was driven to find a solution to this problem and he had tried many different medications, including chloral hydrate, bromide, morphine and other substances without success. Cocaine was the new chemical that had been discovered in the late 19th century, and doctors in Europe and North America were experimenting with it enthusiastically for all types of complaints. Freud and Koller were both assigned to the Vienna General Hospital in the 1880s and knew each other quite well (Figure 2). Freud published a 25-page treatise on the properties of cocaine titled "Über Cocaine" in 1884⁴ and encouraged Koller to help him with some of his clinical research.

Sigmund Freud was very interested in the pharmacologic effects of cocaine and especially the central effects. In this same article Freud wrote: *"to many doctors cocaine seems fated to fill the gap in medical psychiatric treatment, which provides enough means of lowering the heightened excitement of nerve centers, but knows no means of raising the lowered functioning of these. According to them coca is recommended for the most varying kinds of psychic weakness. His paper ended with the following paragraph: "The attribute of cocaine and its salt, the numbing of the skin and the mucous membrane with which it comes in contact in concentrated solutions may lead to other uses especially in diseases of the mucous membrane. Following Collin, Charles Fauvel praises cocaine in the treatment of the pharynx and describes it as le tenseur par excellence des cordes vocales. More uses that stem from the anesthetic effect of cocaine might very well develop."*

It is worth repeating the circumstances that led to Koller's "eureka" moment. In the summer of 1884, Freud had taken time off to visit his fiancée in Hamburg, Germany and asked Koller to continue his research on cocaine. Koller allowed one of his

colleagues, Dr. Engel, to sample the powdered cocaine. Engel immediately remarked "how that numbs the tongue," and Koller responded by saying "Yes that has been noticed by everyone that has eaten it... and in the moment it flashed upon me that I was carrying in my pocket the local anesthetic for which I had searched some years earlier." Koller went straight to his laboratory and asked his assistant for a guinea pig for the experiment. This moment was observed by a Dr. Gaertner,⁵ an assistant in Stricker's laboratory who said the following: *"A few grains of cocaine were dissolved in a small quantity of distilled water. A large lively frog was selected from the aquarium and held immobile in a cloth, and now a drop of the solution was trickled into one of the protruding eyes. At intervals of a few*

"Koller went straight to his laboratory and asked his assistant for a guinea pig for the experiment."

seconds the reflex of the cornea was tested by touching the eye with a needle.... After about a minute came the great historic moment, I do not hesitate to designate it as such. The frog permitted his cornea to be touched and even injured without a trace of reflex action or attempt to protect himself whereas the other eye responded with the usual reflex action to the slightest touch. 'Now it was necessary to go one step further and to repeat the experiment upon a human being. We trickled the solution under the upraised lids of each other's eyes. Then we put a mirror before us, took a pin in hand and tried to touch the cornea with its head. Almost simultaneously we could joyously assure ourselves, "I can't feel a thing." This information was obtained from Koller's daughter who went through his papers after his death and found notes her father had left about the actual discovery. This information is also published in the Psychoanalytic Quarterly in 1963 some 20 years after Koller's death in 1944.

Although we do not know the date of this discovery, we know that it occurred in the summer of 1884. Koller was unable to present his findings at the pending meeting of the prestigious Heidelberg Ophthalmological Society, which was scheduled for September 15, 1884. He asked one of his colleagues, a Dr. Josef Brettauer of Trieste, to present on his behalf. Dr. Brettauer demonstrated Koller's observations at the meeting. Within weeks, news of the recent discovery had spread like wildfire all over Europe, and on October 11, an ophthalmologist from New York, Dr. Noyes, published a report about this great discovery in the Medical Record in New York.⁶

Clearly ophthalmologists all over the world were very excited about this breakthrough, and Dr. Noyes went so far as to say, "The momentous value of the discovery seems likely to be in the eye practice, of more significance than has been the discovery of anesthesia by chloroform or ether in general surgery and medicine." Koller finally got a chance to present his brilliant discovery to the Viennese Medical Association on October 17, 1884. As with many great discoveries, Koller's observations were not without controversy. Dr. Königstein, who was a more senior ophthalmologist assigned to the Vienna General Hospital, was asked by Freud to study the effects of cocaine on the eye but did not have the same success as Koller. In fact, Königstein questioned Koller's success because he was unable to demonstrate the beneficial effects of cocaine on the eye. The reason for Königstein's failure was most likely because he tried cocaine in patients who had inflammation of the eye and had pre-treated many of them with topical alcohol, preventing cocaine from exerting its beneficial effect.

Königstein also read a paper at the October 17 meeting of the Viennese Medical Association describing the

beneficial effects of cocaine in the eye but did not mention Koller's work. Freud was alarmed with Königstein's aberrant behavior and wrote the following letter to Koller:

Dear Friend:

I am aghast at the fact that in K's published paper there is no mention of your name; and I don't know how to explain it in view of my knowledge of him in other respects; but I hope you will postpone taking any steps until I have talked to him, and that you will, after that create a situation in which he can retract.

With kind regards, Dr. Sigm. Freud

Königstein subsequently published papers on the 19th and 26th of October 1884 on the local anesthetic effects of cocaine (Wien Med Presse)⁷ and again did not mention anything about Koller's work but instead added a footnote at the end of the paper:

It remains to precise my relations to the communication by Dr. Koller on September 15 at the Congress in Heidelberg and with regard to the fact that the complete anesthesia of the cornea and conjunctiva from cocaine were then for the first time pronounced. I started my investigations independently of Mr. Koller and had however at the time of his communication not arrived at a definite judgement regarding my own reservations of anesthesia from coca. My further experiments soon gave me confirmation of Koller's statements, though other observations mentioned here were absolutely independent.

One may speculate that Königstein being the more senior ophthalmologist in the Vienna General Hospital was not happy at being upstaged by his junior colleague and was somewhat disingenuous in his reaction to the matter. It is also interesting to note that Koller did not ask Königstein to participate with him when he was carrying out those final, illuminating experiments on animals and man.

Another individual objected to Koller's priority claim. Professor MJ Rossbach⁸ of the Pharmacological Institute at Würzburg was familiar with von Anrep's work on cocaine and claims that Koller did not give von Anrep appropriate recognition for his published work on the subject of cocaine in 1880. Von Anrep's paper⁹ described a number of experiments with cocaine on mammals, birds and some self-experimentation. At the conclusion of his report, von Anrep wrote the following:

I have had the intention after the study of the physiological effects of cocaine on animals also to experiment on man. Other engagements until now have prevented me from doing so, and

"In fact, Königstein questioned Koller's success because he was unable to demonstrate the beneficial effects of cocaine on the eye."

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Regional Anesthesia: How It All Began

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the animal experiments do not permit practical conclusions. In spite of this I would like to recommend cocaine as a local anesthetic and in melancholiacs.

In truth, von Anrep's work on cocaine was referred to by Koller in his original paper.¹⁰

Morton's public demonstration of etherization on October 16, 1846 was also very controversial. We all agree that many others before him had successfully anesthetized patients but nobody can argue that Morton's demonstration on that fateful day was the lightning rod that sparked the use of general anesthesia all over North America and Europe within a matter of weeks. The same is true for Koller's discovery. He took that final step that was required to convince the world of ophthalmology that cocaine was a highly effective local anesthetic when applied to the eye allowing major eye operations to be painlessly performed. In the words of Clark¹¹: "There is however a world of difference between a bright idea and a successful achievement, and the main credit must always go to the man who possesses the energy, determination and skill to demonstrate the possibility of progress along some new path."

Freud, on the other hand, behaved in a very magnanimous way to Koller's discovery. If anyone was entitled to share authorship with Koller on this discovery it should have been Freud because, after all, he had already published information on the effects of cocaine on mucous membranes in his paper and may have influenced Koller's thought process. It is likely that if Freud was in Vienna, Koller would have invited him to witness the experiments. On his return to Vienna, Freud was surprised that Koller had made this great discovery and not Königstein because he had asked Königstein to carry out these experiments on the eye. His actual words are as follows:

When I returned from my holiday I found that not he but another of my friends Carl Koller (now in New York), whom I had also spoken to about cocaine, had made the decisive experiments upon animals' eyes and had demonstrated them at the Ophthalmological Congress at Heidelberg. Koller is therefore rightfully regarded as the discoverer of local anesthesia by cocaine, which has become so important in minor surgery; (but I bore my fiancée no grudge for her interruption of my work).¹²

It is very clear in Freud's mind that he did not deserve authorship on this sentinel paper, and this event is a reminder to all of us about the whole question of authorship and plagiarism in those days and the unwritten

rules that applied then. Koller did share authorship with Freud on some of his papers on cocaine. In one of these publications, Freud sent an autographed copy of their paper to Koller upon which he wrote, "Seinem lieben Freunde Coca Koller."

In most situations, one would imagine that Koller would have been a celebrity in his own city, but that was far from the truth. Even though Koller was revered around the medical world for his great discovery, he could not secure a job in his own institution because of rampant anti-semitism in Austria and Germany at that time. Furthermore, Koller was quite outspoken and diplomacy was not one of

"An anti-semitic insult was directed towards Koller, who responded by punching the doctor in the ear. The other doctor (Friedrich Zinner) was also in the military reserves and he had no choice but to challenge Koller to a duel."

his strongest suits. Any chance Koller may have had to secure a position in Vienna was negated by an incident that occurred in the emergency room of Vienna General Hospital on January 7, 1885. This information was obtained from Koller's papers found by his daughter long after his death.

Freud like all young men in Austria at that time was required to serve in the military. His rank was Oberarzt (First Lieutenant) in the Army reserve. Among Koller's papers was a summons from the police and a newspaper article in the *Neues Wiener Abendblatt* January 7, 1885. The newspaper article described a duel that took place in Vienna. Following is a summary of events that lead to this duel: Koller was on duty in the emergency room of the hospital. Koller examined a patient who had injured his finger, and a tourniquet had been applied to the finger. Koller asked that the tourniquet be removed because it was interfering with the circulation. One of the other doctors objected to the removal of the tourniquet and then Koller cut the ring bandage from the patient. An anti-semitic insult was directed towards Koller, who responded by punching the doctor in the ear. The other doctor (Friedrich Zinner) was also in the military reserves and he had no choice but to challenge Koller to a duel. The challenge was accepted. The duel took place on January 6, 1885 at the Cavalry Barracks at Josefstadt. Koller had no former experience with foils but was given a quick lesson before the event. Koller prevailed in the third round by seriously wounding his opponent in the head and arm. The duel was stopped and Dr. Zinner was taken to the General Hospital. Even though Koller received many laudatory letters for his bravery on this occasion, it did nothing to help secure a position for him in Vienna. The police eventually dropped all charges against him. Koller worked in both Holland and London in the intervening period and eventually set sail for the New World in May 1888 on S.S. Saale, a sailing ship bound for New York.

Koller spent the rest of his life in America in the private

practice of ophthalmology in New York. He received every award imaginable from many countries in the world for his outstanding discovery. Eventually the University of Heidelberg presented him with the Kussmaul Medal in 1928 in commemoration of the discovery, which was first announced there on September 15th 1884.

In his obituary published in the *Archives of Ophthalmology*, Dr. S. Bloom wrote:¹³

He was not a calm person, nor had he ever any hesitation about expressing criticism of himself or others if he discovered error. Like all scientifically minded people he despised insincerity in medical practice and often jibed at it. To all with whom he came in contact he was a stimulating personality, always speculating about the unknown and the unsolved problems in all lines of endeavour. Friends, colleagues, and patients sensed in him a real person, true, reliable, fearless....

There are many similarities in the two greatest discoveries in the history of anesthesia. Eradication of pain was the driving force in both discoveries. In Morton's case, the removal of dental roots to create a better fit for his dental prostheses drove him to find a solution. In Koller's case, the enormous pain that patients suffered in the process of safely restoring vision motivated him to find an answer. They were both quite young men, aged 27, when they made their discoveries. Both men suffered enormous disappointment in their lives and did not receive appropriate recognition until much later in life; and in Morton's case only after his premature death. Neither received any major financial gain for their discoveries. It was not serendipity that led them to their discoveries but rather an inner drive to find the answer. In both cases, the potential for these medications to induce anesthesia had already been observed. These two men found a way to put the final piece in place and we certainly recognize them for their great achievements today, and they will be remembered in perpetuity for what they did for mankind.

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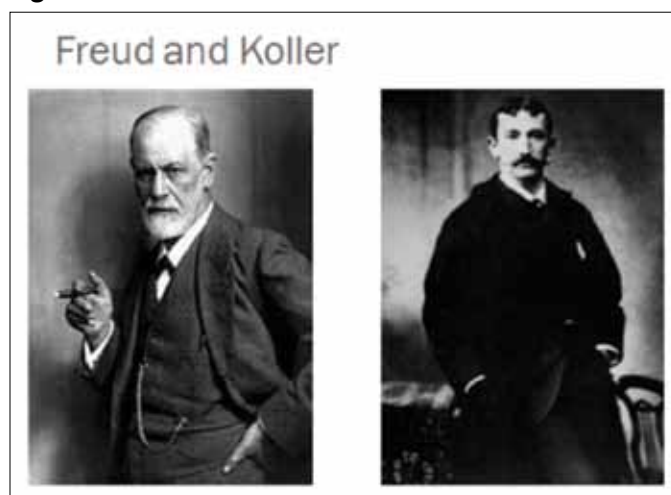
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Figure 1



The Incas are reported to have used cocaine for trepanation procedures centuries before the local anesthetic properties were described. (Picture of Machu Picchu obtained from <http://thebesttraveldestinations.com/machu-picchu-peru/>)

Figure 2



Freud and Koller worked together in the Vienna General Hospital in the 1880s

Intrathecal Pumps for the Treatment of Chronic Pain: A Closer Look



Artemus Flagg II, M.D., M.P.H.
Chief Anesthesiology Resident
Johns Hopkins University
Chair, Resident Section Committee

economic toll of chronic pain may be estimated at \$100 billion a year in the United States.⁵ The mainstay in the management of chronic pain has focused on conservative therapies and systemic analgesics.⁶ Unfortunately, many physicians have equated conservative care as a continuing escalation of opioids despite unchanging pain scores, side effects, and risks of diversion and abuse. For unresponsive cases of intractable chronic pain, continuous intrathecal therapy has become widely used since the approval by the Food and Drug Administration in the 1980s.⁷

An intrathecal pump is an implantable medical device that allows the patient to receive concentrated doses of medication delivered directly to the intrathecal region via a catheter. The first clinical use of an implantable intrathecal opioid delivery device was demonstrated in 1981 for use in chronic pain of malignancy.⁸ Since that time, numerous studies have validated the implantation of an intrathecal pump as an advanced therapy for patients with chronic malignant pain, however this form of therapy is a topic of debate amongst physicians when applied to patients with chronic non-malignant pain.⁹⁻¹⁴ The debate for not using these agents include the risks involving the potential development of tolerance and complications arising from the implantation of the intrathecal pump itself, which can be grouped into surgical, mechanical, pharmacological, and medical (i.e. endocrine, edema, granuloma formation, and infection).¹ Proponents for the implantation of intrathecal pumps in patients suffering from chronic non-

Approximately 16 to 23 percent (50 to 70 million) of the population suffers from pain, which is under-treated or not treated at all.¹ Chronic pain, which is commonly defined as pain persisting longer than six months or pain persisting past the expected time of healing, is a tragically overlooked public health problem.² The financial and social burden of chronic pain is greater than that of diabetes, heart disease, and cancer combined.^{3, 4} A 1998 National Institutes of Health (NIH) report concluded that just the

“Patients needing intrathecal drug delivery are often divided into those with chronic potentially life ending illnesses such as cancer, and those with chronic non-malignant pain most commonly related to diseases of the spine.”

malignant pain often suggest a potential improvement in psychosocial function in patients that had failed to respond to standard multimodal therapy.¹⁵ Recent data obtained from a retrospective chart review cohort study observed a reduction of visual analog pain scale scores and a decrease in oral opioid consumption in patients receiving intrathecal morphine infusions.¹⁶

Patients needing intrathecal drug delivery are often divided into those with chronic potentially life ending illnesses such as cancer, and those with chronic non-malignant pain most commonly related to diseases of the spine. For those with chronic malignant illnesses, this type of therapy is generally recommended for individuals with a life expectancy of greater than three months, although there has been an increased trend towards survival in those with intrathecal agents as compared to comprehensive medical management. This trend may blur the lines as to patient selection.¹⁷ The use of intrathecal pumps in patients with chronic non-malignant pain is an accepted therapy within the standard of care, although some questions remain unanswered. Many patients with chronic non-malignant pain benefit from oral analgesics medications, physiotherapy or biofeedback, spinal cord stimulation, nerve blocks and neurodestructive procedures. Some clinical studies have demonstrated that oral opioid therapy results in significant pain reduction

in only about one half of chronic pain patients and that this number may decline over time. Functional improvement is also poorly supported by long-term opioid therapy in the chronic non-cancer pain patient. Spinal cord stimulation is a preferred

method of treatment when appropriate, particularly in the setting of neuropathic pain.¹⁸

As residents rotating through the pain division at our respective institutions, our goals are to learn and to help facilitate and improve clinic operations. When given the opportunity to interview, exam, and present a potential candidate to our attending for an intrathecal pump we must have a general understanding of which patients are potential candidates for an intrathecal drug trial. Intrathecal drug pumps are implanted to chronic pain patients when conservative therapies have failed, the patient is not a candidate for additional surgery or finds this option unacceptable, the patient has had side effects or inadequate relief from oral or transdermal medications, the patient has no active or untreated addiction, and the patient does not have any psychological problems that would warrant against the procedure. The procedure is contraindicated in patients with a coagulopathy or systemic infection. Additional contraindications to

placement include: 1) those individuals with significant skin breakdown anywhere along the course of the proposed position of the tunneled catheter, 2) patient's whose body size is not sufficient to accept pump bulk and weight, and 3) when the patient's body habitus would limit accessibility to the pump for required refills.¹⁹

Before a permanent pump can be implanted, the patient will undergo a trial to see if the device will improve their symptoms. During the trial the planned drug is infused through an indwelling catheter that could be placed intrathecally or epidurally. Intrathecal catheter placement may be a more accurate representation of the patient's potential response, but no study has shown any superior predictability as compared to epidural infusion. A 50 percent improvement in pain score and improvement in function for the trial is considered successful, in the setting of acceptable side effects.²⁰

At our institution the implantation of the intrathecal pump is a fellow-level procedure with supervision by the attending. In general, there are two parts to the procedure: 1) the placement of the catheter in the intrathecal space surrounding the spinal cord, and 2) the placement of the pump reservoir in the abdomen. As a result of this procedure chronic pain patients should require a reduced dosage of oral pain medications, may experience a significant reduction in pain, and could potentially benefit from an overall improvement in their psychosocial function.¹⁴ By achieving a higher level of psychosocial function, these individuals could once again have the opportunity to become active and productive members of society.

So are we helping or hurting our patients? The selection of the patient who will receive an implantable intrathecal pump is as important as each step in the procedural process itself (just like any elective procedure). Through a carefully designed selection process, which screens for initial candidates, a thorough and formal patient education format (with emphasis on the patient's responsibilities, risks of the procedure, and complication rate), and a multimodal approach to the patient's overall psychosocial model, with an ultimate emphasis on improving functional outcome, we are helping our patients. Needless to say, the selection process for such an intervention is indeed one of utmost importance and one that we (the residents) play a vital role in.

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Fall Pain Medicine Meeting in New Orleans



Gary J Brenner, M.D., Ph.D.
Program Chair

It is with great pleasure that I invite you to join us in this little corner of America's south, New Orleans, where European traditions blend with Caribbean influences, the history is as colorful as the local architecture and the food is the stuff of legends. This is a place where Haitian and African Creoles were instrumental in creating Jazz and Cajun zydeco...a place like no other where a way of life that began three centuries ago remains influential today.

ASRA prides itself on providing excellent, cutting-edge education for our members and attendees. By attending this meeting, pain medicine physicians will have access to a wide variety of lectures and workshops addressing multiple central topics in pain medicine.

On the opening day, we will continue in the tradition of ASRA meetings where the Refresher Courses address basic science and clinical applications of a number of key topics, including complex regional pain syndrome, opioid-induced hyperalgesia and radiofrequency lesioning.

This year, problem-based learning discussions will be held on Thursday and Saturday afternoons. These small, roundtable discussions will be limited to nine participants and be led by an expert in pain medicine. Attendees will have the opportunity to interact with their colleagues, share interesting cases and learn in a very unique setting.

Friday's session will feature a morning general session devoted to "Controversies in Interventional Pain Medicine." This session includes presentations on key areas such as whether medial branch blocks should be performed prior to radiofrequency lesioning, the utility of vertebral augmentation, and the value of discography as a diagnostic technique.

Many of the sessions and topics in the 2011 meeting were suggested by last year's attendees. ASRA is dedicated to meeting and exceeding the expectations of our participants. One afternoon session, titled "Discussion of Joint ASRA/ESRA Recommendations for USPM Education and Training," will explore likely changes in ultrasound training requirements and how this will impact the field of pain medicine.

ASRA is proud of its ability to provide first-class hands on workshops in interventional pain medicine techniques. In addition to a large number of two-hour hands-on sessions, each day we offer a "special workshop" that is designed to focus on a major area of interventional pain medicine combining didactics and hands-on training. The areas covered in these special workshops will be:

- Ultrasound for interventional pain medicine (full day)
- Spinal cord stimulation (1/2 day)
- Vertebral augmentation (1/2 day)

Our resident programming continues to provide an exceptional chance for trainees to learn from internationally renowned pain medicine practitioners. The opening session will address "Pain and Healthcare Reform: A Discussion on Healthcare Reform and the Future of Pain Medicine," sure to be full of valuable information and insight. Saturday's session includes the ever-popular Resident Jeopardy! followed by an afternoon of hands-on workshops. The development of the resident program was guided by ASRA's resident leadership to ensure that the focus is optimally tailored to the needs of physicians at that level of training.

In summary, the Program Committee and I warmly welcome you to participate in ASRA's 2011 Fall Pain Meeting. Our outstanding faculty will provide both didactics and hands-on training offering learning opportunities in pain medicine second to none. So please join us in one of the nation's most historic and exciting cities for a fantastic educational and cultural experience.



Minimally Invasive Lumbar Decompression (MILD®)

'To Do or Not to Do'

For decades, lumbar spinal stenosis has been treated surgically via open decompression. However, the traditional surgical treatment of lumbar spinal stenosis (LSS) is often associated with significant postoperative pain, disability, and dysfunction. With the recent emphasis on minimally invasive approaches in spine surgery to facilitate recovery, a number of new techniques has been introduced.¹ The most recent one is the MILD®, Minimally Invasive Lumbar Decompression.² The procedure is performed under fluoroscopy and involves limited percutaneous laminotomy and debulking of the hypertrophic ligamentum flavum to restore space in the stenosed spinal canal with minimal disruption of surrounding muscular and skeletal structures. (Figures 1 and 2) Accordingly, the indication for the procedure is symptomatic lumbar spinal stenosis with radiographic evidence of hypertrophic ligamentum flavum as the main contributing factor.

The safety of the procedure was reported in a retrospective study of 90 procedures.³ There were no major adverse events or complications related to the devices or procedure. No occurrence of dural puncture or tear, blood transfusion, nerve injury, epidural bleeding, or hematoma were observed. Early functional outcomes (six-week follow-up) were assessed in a multi-center prospective observational study (N= 72 patients).⁴ It showed statistically and clinically significant reduction of pain as well as improvement in physical function and mobility.

Only recently, the safety and efficacy profiles at one year post-procedure were evaluated in a prospective multicenter study and results were reported in an abstract at the last American Academy of Pain Medicine Meeting (AAPM) and later posted in Pain Medicine News.⁵ Improved outcomes in this study (N= 170 procedures) included a significant reduction in pain (defined as ≥ 40 percent) in 79 percent of patients, significant improvements in function as measured by the Oswestry Disability Index in 71 percent of patients; and statistically significant improvements in patient satisfaction as measured by the 12-Item Short Form survey.

There has been a rapidly growing interest in this new procedure over the last few months as evidenced by the plethora of abstracts in all national pain and spine meetings. Although the MILD® procedure seems to be very safe and efficacious in selected patients, it is premature to recommend its widespread use in patients suffering from lumbar spinal stenosis without well controlled studies comparing its outcomes with those of the more tra-

ditional open approaches. My own opinion is that the MILD® procedure should be offered to those selected patients with symptomatic lumbar spinal stenosis where the MRI clearly demonstrates that the hypertrophic ligamentum flavum is the "main" or "only" reason behind the reduction of the dural sac cross sectional area (and this is quite rare)...or in those patients with contraindications to an open surgical approach. Many clinicians look at the mild procedure as something to fill the "treatment gap" between failed conservative management (e.g. physiotherapy, pharmacological treatments, acupuncture, epidural steroids) and invasive open surgery.

However, we need to remember that it is very common to have multifactorial causes for lumbar spinal stenosis (e.g., disc protrusion, facet hypertrophy, and osteophytes) and one cannot accurately estimate which factor contributes the most to the stenosis. While open surgical approaches can take care of more than one pain generator at the same time, the MILD® procedure will help only those patients who have a predominately hypertrophic ligamentum flavum. Patients should be made aware of this when offering the MILD procedure. We really need well designed controlled studies comparing this approach to other surgical approaches to validate early encouraging results. If positive it would support the use of this promising minimally invasive procedure that can be performed as an outpatient under local anesthetic and moderate sedation.

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Samer Narouze, M.D., M.S.
Clinical Professor of Anesthesiology and Pain Medicine, OUCOM
Clinical Professor of Neurological Surgery, OSU
Associate Professor of Surgery, NEOUCOM
Chairman, Center For Pain Medicine
Summa Western Reserve Hospital

"There has been a rapidly growing interest in this new procedure over the last few months as evidenced by the plethora of abstracts in all national pain and spine meetings."

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Hydrodynamics of the Spinal Epidural Space and Evidence for a Direct Connection With the Venous Circulation



Charles W. Buffington, M.D.
Professor of Anesthesiology
University of Pittsburgh
School of Medicine
Staff Anesthesiologist
UPMC-Montefiore Hospital
Pittsburgh, Pennsylvania



Ulla Elisabet Blix, M.D.
Visiting Assistant Professor
of Anesthesiology
University of Pittsburgh
School of Medicine
Staff Anesthesiologist
UPMC-McKeesport Hospital
McKeesport, Pennsylvania

Section Editor:
Steven Orebaugh, M.D.

The epidural space is a potential space, filled with fat and vessels, which surrounds the dura throughout its length in the spinal canal. Its importance to anesthesiologists is obvious, as a repository of local anesthetic solutions to produce anesthesia, and as a potential source of complications when neuraxial blocks are provided. We sought to elucidate the pressure and fluid dynamics that govern the physiology of the epidural space in several animal and human cadaver models.

In the 1990s the spectre of an epidural hematoma hung over operative epidurals in which inadvertent venous puncture occurred. This was a major concern in patients who would receive heparin during the course of surgery. We reasoned that the pressure difference between blood in the vein and the spinal epidural space (“transmural pressure”) would be important in hematoma formation. In anesthetized pigs, we intentionally punctured an epidural vein and left the catheter inside. We measured epidural pressure through a second catheter at an adjacent interspace. We found that transmural pressure of the vein was low, on the order of 1-2 mmHg and did not change with maneuvers that increased venous pressure (such as a simulated Valsalva manoeuvre) because epidural space pressure increased at the same time (Figure 1). This finding indicates that the pressure gradient driving blood out of epidural veins is very small and suggests, by exclusion, that hematomas arise when arteries rather than veins are damaged.¹

Next we tested the hypothesis that tachyphylaxis to local anesthetic agents during epidural anesthesia is due to increasing loss of local anesthetic from the epidural space. We injected saline into the spinal epidural space (SES) at different flow rates with a constant flow pump and measured SES pressure through a second epidural needle at an adjacent interspace. SES pressure gradually increased and then, to our amazement, reached a stable plateau after several minutes of fluid infusion (Figure 2). Pressure declined to baseline when the pump was stopped. The plateau confirms that fluid was leaving the SES at the same rate it was entering, and, further, that the fluid was flowing into another space that is large and compliant.²

We found that a plot of plateau pressures versus the corresponding flow rates yielded a straight line in individual animals (Figure 3). This line was not altered by subsequent infusions meaning that increased loss of local anesthetic is not the basis of tachyphylaxis. The straight line had a zero-flow intercept at a pressure slightly above central venous pressure. We found no evidence of a critical opening pressure, and plateau pressure did not bend off at higher flow rates, indicating that there is no recruitment of additional exit pathways with increased flow and SES pressure.

These results did not correlate with the traditional notion that the SES is a potential space that should open up as fluid is infused. Further, we expected SES pressure to increase continuously as infused fluid exhausted the capacitance of the space and its extensions into the nerve roots. So the question became: “where is the fluid going?”

To answer this question, we took a number of animals to the Radiology department after euthanasia. There, we used a cine X-ray device with digital subtraction capabilities to photograph the movement of contrast media in the epidural space. An injection of contrast in the mid-thoracic space outlined the space and nerve roots in the lumbar region. A second injection after the camera was repositioned, showed contrast leaving the epidural space in the high thoracic and cervical region. The contrast revealed a network of small channels around the spine that coalesced into veins deep in the neck (Figure 4). Contrast was observed in the heart in several animals.

These observations prompted a closer look at what appeared to be a direct connection between the high thoracic and cervical SES and the venous circulation.³ A macromolecular tracer (Evans Blue dye permanently attached to albumin) was injected into the SES of another set of animals, and blood samples were collected sequentially after plain normal saline was infused into the SES. We found that 30-70% of the EB dye reached the blood stream. Since the EB-albumin complex cannot cross vessel walls by diffusion, this indicates a connection between the SES and venous circulation that is capable of handling large molecules and bulk flow. After euthanasia and

exsanguination, we injected microfil, a liquid containing tiny yellow latex particles plus a catalyst, into the SES and allowed the microfil to set up. Careful dissection revealed yellow microfil in veins leaving the spine, but microscopic inspection of nerve roots has so far failed to identify the actual anatomical connection between the SES and epidural veins.

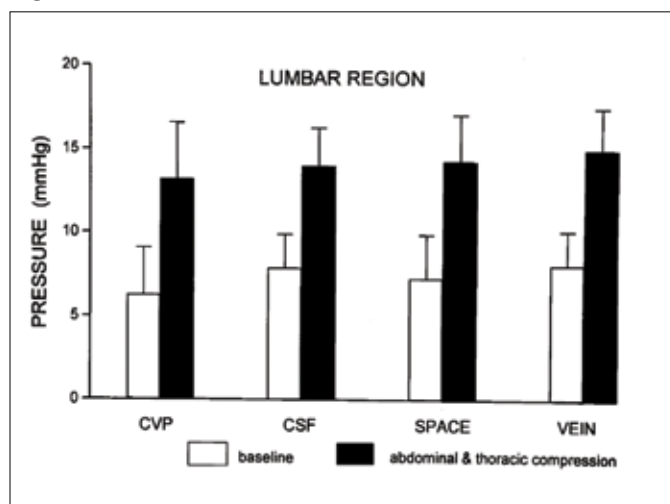
In another investigation of epidural space physiology, we studied recently-deceased cadavers.⁴ Contrast was injected into the low thoracic epidural space and a 64-slice CT scan from the top of the head to the coccyx was performed. Contrast was found in veins surrounding the spine in all subjects, primarily in the cervical area and at the level of the brachial plexus (Table 1). Contrast could be traced to the internal jugular veins in a number of these bodies.

The emerging concept is that fluid leaves the spinal epidural space through direct connections with the venous circulation that are located primarily in the area of the brachial plexus. These connections are capable of handling bulk flow and form the major site of resistance when fluid is injected into the SES.^{2,5} Such connections may explain systemic local anesthetic toxicity in the absence of inadvertent intravascular injection.⁶ They likely explain why air injected into the epidural space quickly reaches the heart.⁷ They may also explain the early peak in systemic local anesthetic concentration that follows epidural injection.^{8,9} Further, loss of local anesthetic via these connections in the C-6 to T-2 region may explain why "very high" epidurals are uncommon. Finally, the results help explain the curious finding that CSF pressure does not rise dramatically when large volumes of saline are infused into the epidural space during spinal cord cooling and epiduroscopy.^{10,11}

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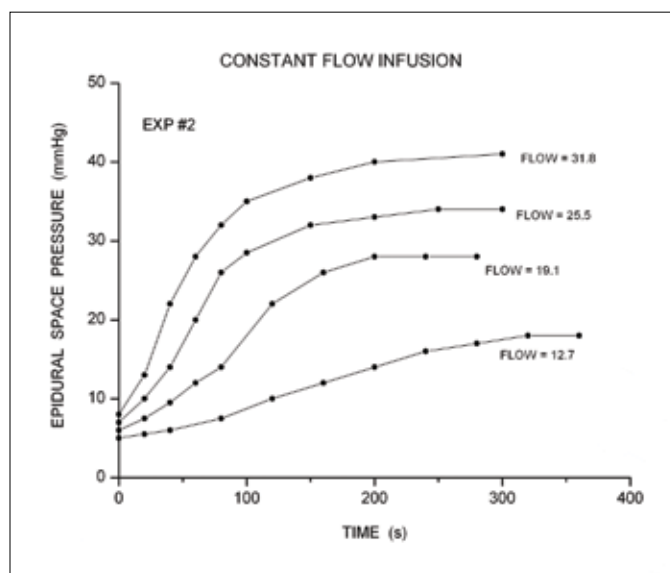
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Figure 1



Pressures measured simultaneously in an epidural vein, the spinal epidural space, the subarachnoid space and the right atrium are shown at baseline and during external manual compression of the animal's chest and abdomen to simulate a Valsalva maneuver. The difference between the pressure inside the epidural vein and that in the surrounding epidural space was on the order of 1-2 mmHg and did not increase during external compression because epidural vein pressure and the surrounding epidural space pressure both increased. Data are from 8 pigs.

Figure 2



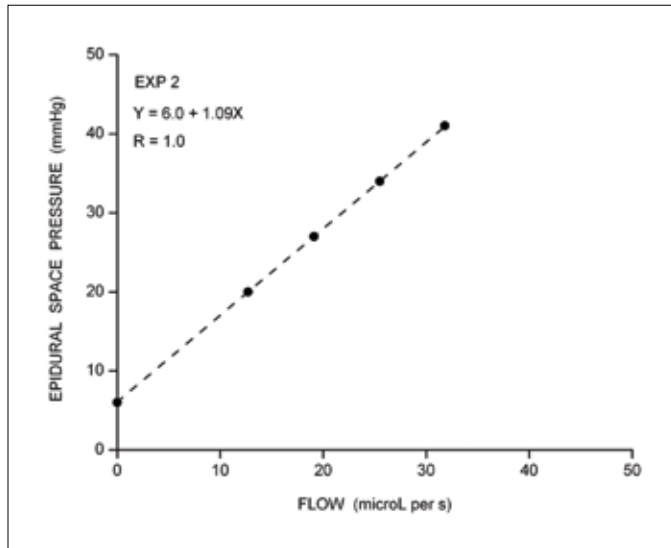
During constant flow infusion of saline into the low thoracic spinal epidural space of one pig, epidural pressure increased to a steady plateau. Faster infusion rates resulted in higher plateau pressures.

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Hydrodynamics of the Spinal Epidural Space and Evidence for a Direct Connection With the Venous Circulation

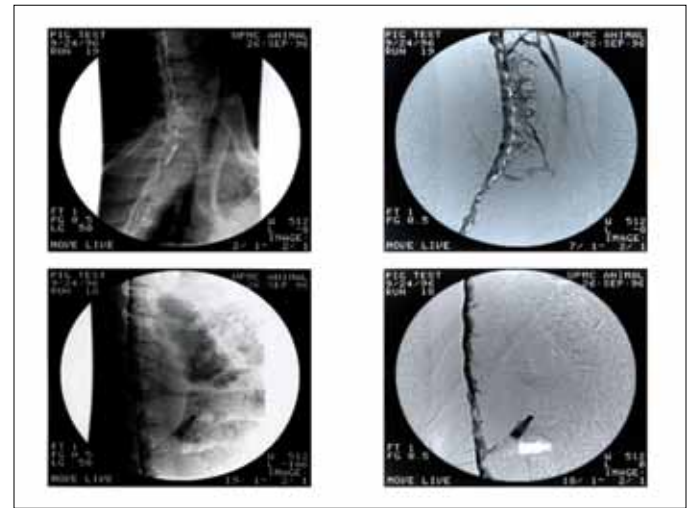
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Figure 3



Steady-state Plateau epidural space pressure during saline infusion at different rates in the same animal is shown in Fig. 2. The line is straight and has a Y axis intercept slightly above central venous pressure. No evidence of a critical opening pressure or recruitment was observed.

Figure 4



Plain films of the thoracic (top panels) and lumbar (lower panels) spine of one pig are shown in the left column. Digital subtraction images following injection of contrast into the epidural space are shown in the right column. Contrast was confined to the epidural space and nerve roots in the lumbar region but filled a plexus of small veins surrounding the cervical and high thoracic spinal column. These veins coalesced into deep cervical veins.

Table 1

Body #	1	2	3	4	5	6	7	8	9	10
Age	78	67	31	49	49	73	82	52	55	71
Gender	M	M	M	F	M	M	F	M	M	M
Anti-coagulated?	No	No	No	Yes	No	Yes	Yes	Yes	No	Yes
High Cervical Veins										
C1 – C5	+		++	+++	++	++		+++		+++
Brachial Plexus										
C6 – T2	++	++		++		+++	++	++		++
Azygous System			+	+		+		+	+	
Lumbar Veins				+	+			+	+	

Contrast injected into the epidural space was found in veins draining the spinal column in all 10 bodies studied. A qualitative 3-point scale was used to estimate the significance of the venous connections that were observed with “+” indicating one or more small veins and “+++” indicating bilateral veins originating at multiple levels and providing enough contrast to delineate collecting veins far downstream.

How Do Local Anesthetics Make Us Numb?

In the years since Carl Koller first demonstrated local anesthesia we have learned much about the physiology and the molecular pharmacology of that action, based on the blockade of nerve impulses, to the point where we can identify the very amino acids on the voltage-gated sodium channels where local anesthetics bind to produce this effect. Yet the realities of conduction block that underlie the loss of sensation during local anesthesia are complex, subtle and still incompletely understood. My objective in this article is to stimulate you to consider what's actually happening when you make a patient numb from local anesthesia, and perhaps to advance our understanding of this process through your own questions and investigations.

Conduction block is itself a complex process. How is the impulse propagated along an axon under normal conditions, and what changes when a local anesthetic is present? Let's use an example from conduction in a myelinated nerve fiber, but the same explanation applies to unmyelinated fibers, i.e., C-fibers. Imagine that an action potential's rapid depolarizing phase is occurring at one node of Ranvier, the result of a large flow of Na^+ ions across the axonal membrane through voltage-sensitive "Na⁺ channels". The current from this flow has to return to its source, and a major pathway is along the inside of the axon, via the conducting axoplasm, and then out through the adjacent node, with some of the remaining current spreading further, to the next node, and even to several nodes beyond. Delivery of the current to the nearest, adjacent node rapidly depolarizes it to a potential where many of the voltage-sensitive Na^+ channels will open, allowing Na^+ current to flow in and depolarize the membrane even further, generating another action potential. The current that flows from one node to another as a result of one impulse is always much larger than the current needed to bring the second node to threshold (this ratio is "the margin of safety" for conduction). During conduction this process occurs in temporal sequence as the impulse propagates along the axon. In fact, action potentials spread so far so quickly in myelinated nerves that impulses are occurring at 5-6 nodes simultaneously. The impulses in such saltatory conduction are more accurately thought of as dancers linked together in a Balkan line dance than as a solitary couple tripping across the ballroom floor.

Imagine a model in which local anesthetic is applied uniformly (a totally hypothetical situation) along a stretch of the axon, and is able to block, at rest, about half of the Na^+ channels in each of the 15 anesthetized nodes¹ (Figure 1). At the first local anesthetic exposed node there is about a 30% drop in the impulse amplitude, but as the impulse travels along successive nodes exposed to the drug the impulse continues to decrease. This "decremental conduction" reflects the step-wise loss of impulse depolarization as each successive node is less able to generate an inward current.² Since not all the channels are blocked by the

drug, impulse blockade is only partial, and local circuit currents are still large enough to stimulate the next, compromised node. In fact, there's enough current from the last exposed nodes to raise the downstream, un-anesthetized nerve to threshold and stimulate a normal, full-blown impulse, as shown by the resurrected amplitudes at the right-hand end of Figure 1. Even if all the channels in every node were totally blocked, the impulse would still spread passively down the axon and, if the exposed region were short enough, would be able to provide current adequate to bring the axon beyond the anesthetic-exposed region to threshold. The impulse would be slowed, but not blocked. So there is a critical length of exposure to effect impulse blockade, characteristic for each type of nerve fiber, which is shorter as the fibers get smaller.³

Local anesthetics show a use-dependent action in impulse blockade⁴. The occurrence of action potentials predisposes the nerve to greater blockade, because the Na^+ channel conformations that occur from depolarization have higher affinities for these drugs than do the resting channel. (Both open and inactivated channel conformations bind local anesthetics more tightly than the resting closed conformation, but the binding to the inactivated channels occurs too slowly to be of much importance during the very brief (1-2 msec) depolarization of an impulse.⁵) Immediately after an impulse, more channels are drug-bound and, importantly, local anesthetic dissociation from the closed channels that are present during the interval between impulses is slow enough that a subsequent impulse, as might occur in a "physiological train of impulses", will propagate into a membrane that is still in this higher drug-bound state. Drug binding to the channels will increase as impulses repetitively depolarize the exposed nerve, and this will cause the impulses to decline increasingly over time, the phenomenon of use-dependence. The higher the frequency of impulses, the greater the resulting use-dependent action⁴. For a propagated impulse, use-dependence enhances decremental block, such that the increased



Gary Strichartz, Ph.D.
Pain Research Center
Department of Anesthesiology,
Perioperative and Pain Medicine
Brigham & Women's Hospital and
Harvard Medical School
Boston, Massachusetts

Section Editor:
Steven Orebaugh, M.D.

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How Do Local Anesthetics Make Us Numb?

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binding of local anesthetics results in a steepening decline of the impulse amplitude along subsequent nodes. This is modeled incisively in Figure 1, where the second impulse in the train of action potentials, applied at 50 sec^{-1} , is unable to span the length of anesthetized nerve successfully, and conduction along the fiber is eventually blocked. By the time the tenth impulse arrives, decremental block is even more developed and the impulse cannot extend beyond about the 8th node.⁶

Does any of this occur clinically? The answer is yes; every peripheral nerve block has a marginal zone where, even if the local anesthetic concentration is high enough to effect a complete block of resting channels, the impulse would still fall decrementally until it failed. In this zone, the drug concentration, diluted by diffusion from the injection locus, is low enough that use-dependent actions still occur. These processes are only clinically important, however, during the onset and regression of nerve block, where fibers are only weakly anesthetized. During a clinical nerve block, the dose of drug delivered to the axon reaches a high enough concentration that all the Na^+ channels are blocked in their resting conformation, and so no additional, use-dependent block can occur.⁷

What is that critical concentration for block and how is it related to the delivered dose? You might inject 1-2% lidocaine for peripheral nerve blockade, for example, equal to a concentration of $\sim 38\text{-}75 \text{ mmoles/L}$. Investigations of isolated axons, without major tissue barriers, however, shows that only about 1 mmole/L is adequate to block impulses in all classes of nerve fibers⁸. But if less than $\sim 20 \text{ mmoles/L}$ (0.5%) lidocaine is injected in vivo, anesthesia is incomplete⁹. This stark contrast in concentrations injected versus needed for blockade of a fiber is a result of the inefficient transfer of the extraneurally delivered drug into the nerve compartment containing the fibers. The limited diffusion of local anesthetic across the perineurium, in particular, coupled with its active removal by the local vasculature results in a net delivery of less than 0.05 of the injected drug¹⁰ accounting for the 20:1 ratio between the minimally effective concentration in vivo and the critical blocking concentration of an exposed axon.

How much impulse blockade is actually necessary to produce numbness, an insensate condition? The answer may surprise you, but sometimes patients report no sensation from stimulation when impulse blockade is incomplete. Studies of evoked potentials recorded from the scalp of subjects receiving local anesthetic show that electrical potentials can be detected in the brain, evoked by peripheral stimulation, in some patients who are numb to that stimulation^{11,12}. Afferent activity reaching the central nervous system is sometimes imperceptible. We also know, from microneurography studies, in which impulses stimulated and recorded in the periphery from superficial cutaneous nerves of human subjects can be compared to

perceived sensations, that a minimal impulse frequency is often required before sensation is experienced¹³.

What are the clinical ramifications of these observations? They might be quite important when pre-operative assessments impact post-operative outcomes. Imagine that you have administered a local anesthetic and are determining the success of the block, examining the patient by stimulating the skin with a minimally sharp instrument, e.g. the classic, plastic "cocktail sword" or a non-penetrating hypodermic needle, and asking about sensation. Just because the patient feels nothing doesn't mean that all afferent input to the CNS is absent. More critically for care, the types of nerve fibers you are stimulating by this weakly noxious procedure are small diameter myelinated A-delta nociceptors¹⁴, fibers that are significantly more sensitive to block by local anesthetic than the non-myelinated C-fibers that carry signals from more intense pain⁷. Numbness to A-delta fiber stimulation may be accompanied by afferent activity in the C-fibers, activity that is known to be critical for the sensitization of the central nervous system^{15,16} that underlies the prolonged, perhaps the chronic form of post-operative pain¹⁷. Thus, the patient may be under-anesthetized for the degree of impulse blockade that is necessary for prevention of post-operative pain, yet effective testing to assess this degree of block requires a strong noxious stimulus, which conscious patients should not, and ethically cannot, be subjected to.

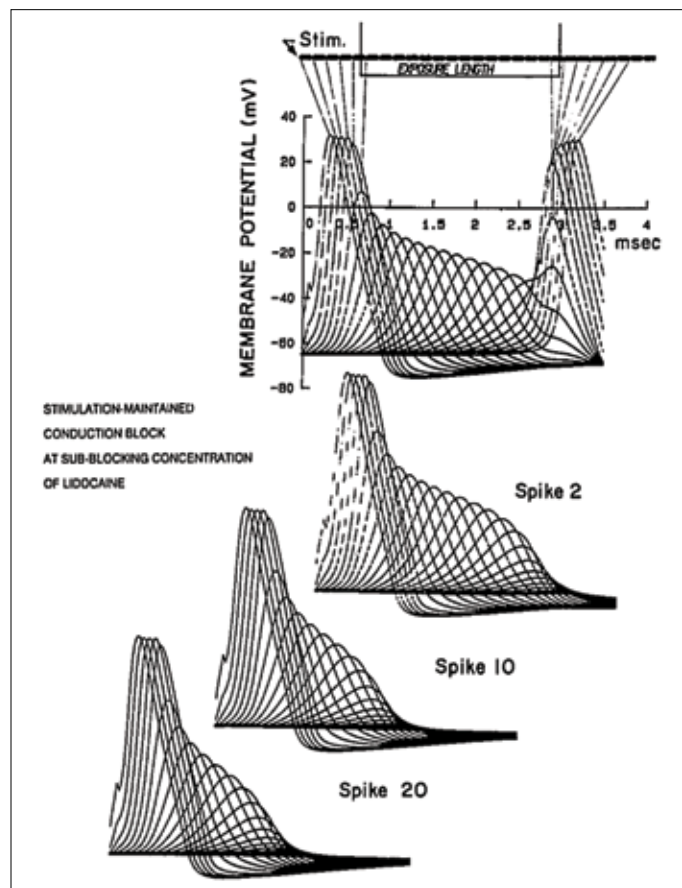
Motor function deficits are not useful assays in this regard. The major reason for muscle weakness after peripheral nerve block is probably the blockade of impulses in the small myelinated A-gamma fibers that set the tone of the intrafusal fibers containing the muscle spindles. An absence of impulses in these A-gamma fibers immediately reduces the tone in the spindles, leading to a fall in the output of the Ia sensory afferents that carry impulses from the spindle's stretch receptor to the large motor neurons in the spinal cord's ventral horn. In the absence of this Ia input, the tonic output of these large motor neurons, which drive the power-producing extrafusal fibers, falls to near zero, effecting a flaccid paralysis. None of this informs us about the response of nociceptive C-fibers, however.

It may be possible to counter some of this problem by increasing the administered concentration of the anesthetic, but the specter of toxicity, both local and systemic, looms large and we would be foolhardy to ignore it¹⁸. This puts us in a bind. Perhaps you will be able to figure a way out of this conundrum, which would be a boon to patients and practitioners alike. Not only would acute postoperative pain be reduced, but the incidence of chronic postoperative pain, that occurs with alarmingly high frequency after many surgical procedures, e.g., thoracotomy, herniorrhaphy, would also be lowered. It is, I believe, worth thinking about.

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Figure 1



A computer simulation of impulse conduction in a myelinated nerve fiber and its extinction by a combination of local anesthesia and high-frequency stimulation. A train of 20 impulses is initiated by a supramaximal electrical stimulus at the left-most node of Ranvier in this fiber, which contains 15 nodes (not drawn to anatomical scale) exposed to local anesthetic. Impulses propagate left-to-right, and their time-course at each successive node is shown by the membrane potential vs time traces. In this simulation a local anesthetic dose was chosen to cause decremental decline of the impulse without total blockade for the first impulse in the train (0.29 mmol/L of lidocaine was chosen, based on known potencies for blocking Na⁺ and K⁺ channels in nerve⁵). Propagation of the second impulse, shown as "Spike 2," is unsuccessful as the impulse decrements even more steeply, due to use-dependent effects from the first impulse, and fails to generate enough current to excite the last node. The use-dependent effect on decremental conduction along the anesthetized axon is even more obvious for spikes 10 and 20. (Reprinted from Ref. 6).



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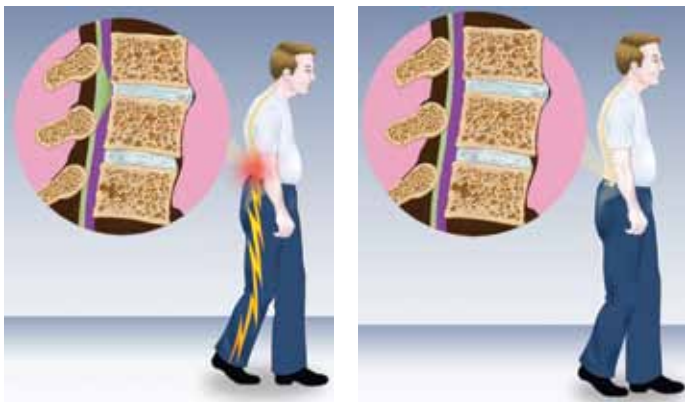
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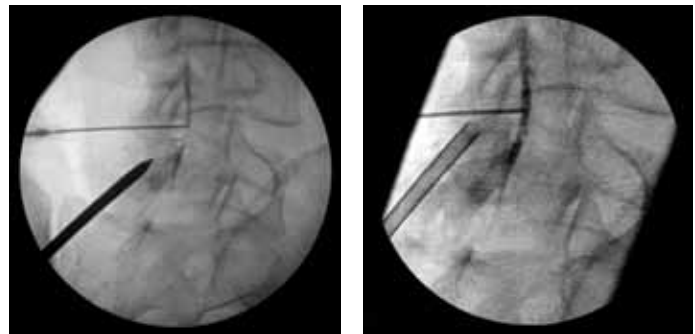
Figure 1



Left: Lumbar spinal stenosis secondary to hypertrophic ligamentum flavum (green).

Right: Normal spinal canal. Notice the patent epidural space (purple).

Figure 2



Left: Pre-procedure X-ray. Epidurogram showing a filling defect (arrows) secondary to hypertrophic ligamentum flavum.

Right: Post-procedure X-ray. Epidurogram showing resolution of the filling defect after debulking of the hypertrophic ligamentum flavum.