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# American Society for Peripheral Nerve

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## Future ASPN Meetings

**2014 Annual Meeting**

January 10-12, 2014  
Grand Hyatt Kauai Resort & Spa  
Kauai, Hawaii

**2015 Annual Meeting**

January 23-25, 2015  
Atlantis Resort & Casino  
Paradise Island, Bahamas

**2016 Annual Meeting**

January 15-17, 2016  
The Westin Kierlan Resort & Spa  
Phoenix-Scottsdale, Arizona



## Message From the President

Dear Colleagues,

The leaves have turned to brilliant colors and now begin to fall. I can tell you that our society continues

to evolve, and with each passing season, it becomes stronger and more entrenched. The ASPN serves as major educator of medical professionals concerning the pathophysiology and management of peripheral nerve disorders. It brings together students and teachers, scientists and practitioners, old and new friends. It is a society that bridges many disciplines and as such, provides a platform to foster collaboration and enhance constructive feedback. I am indeed proud to be an active member of the ASPN and strongly encourage others to do the same.

The society will again hold our annual meeting in conjunction with the AAHS and ASRM. This relationship and collaboration requires attention, commitment, and adjustments. There have been some financial concerns, but the various leaderships have moved beyond selfish interests to embrace the concept that the whole is greater than the sum of the parts. The meeting in Hawaii will reflect the ability of three program chairs to cooperate on the highest of levels and formulate a truly outstanding educational and scientific event. In her column, our program chair Chris Novak, has highlighted some of the sessions. Our invited speaker, Jaishri Blakeley, has had a meteoric rise in

the neuro-oncology world. She now directs a rather massive effort aimed at understanding and treating peripheral nerve tumors. We will be having exciting morning sessions designed to bring into focus cutting edge research and its application to patient care. The shared Saturday morning will have a session looking at the future of limb transplant and bionic replacement; what are the pros and cons of each approach, where should the research dollars be going, what the ethical concerns. We will end our meeting with a novel session. I have asked our past presidents to serve on two back to back panels. Some of our newest members are being charged with presenting to the panels their most challenging clinical cases. We will explore how the panelists would have managed the cases in the past and what they are currently doing. Where has our discipline been and where are we going.

The ASPN is not only a scientific society, but is also a group of people with many diverse interests, hobbies, cultural backgrounds, and extra-curricular passions. Lorinda, my sons Micah and Adam, and I look forward to hosting each and every one of you in Hawaii. Bring your tennis racket, yoga matt, surfboard, hula-hoop, grass skirt, and dancing shoes. Don't forget to bring the novel you wanted to finish reading and of course any family members who may simply want to watch a spectacular sunrise or sunset over the Pacific.

Allan J. Belzberg, MD  
ASPN President



## From the Editor's Desk

This edition of the ASPN Newsletter is dedicated to our presidents, present and past.

Our society has been so lucky as to have such dedicated, knowledgeable, skilled and top notch clinicians and researchers as our presidents. In this edition you will see a special section containing several articles by some of our presidents. The articles reflect the wide scope of their interests and wisdom. Some of the articles are scientific, demonstrating the depth of their knowledge and the numerous contributions they have made to our profession, and the others are reflective in nature showing their wisdom that they have acquired through years of training and experience. You will see also how one of our past presidents, Susan Mackinnon, MD, has been honored with the prestigious Jacobson Innovation award from the American College of Surgeons.

In this edition you will also see the program for our forthcoming annual meeting in Kauai, Hawaii in January 2014. Drs. Belzberg and Novak have arranged for an exciting program that will tantalize our scientific and social curiosities. I echo our president and program chair's invitation to all of our members to attend this meeting. It promises to be a fantastic meeting especially in view of the beautiful location.

Again, our deep thanks and gratitude are extended to all our presidents for what they have offered to our society; their commitment, their time, their expertise and their devotion. My personal thanks are also extended to Mrs. Martha Espronceda, for her help in all the stages of development of this newsletter. I really appreciate all your hard work, Martha. See you all in beautiful Kauai

Nash Naam, MD  
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## Interested in being more involved with ASPN? - Join one of the ASPN committees.

The ASPN committees are volunteer driven. These committees benefit the entire membership as their work is important to the society. Each committee is unique and equally important. Nominate yourself or others for the 2014-2015 committees by emailing [marthaespronceda@isms.org](mailto:marthaespronceda@isms.org) or [drnaam@handdocs.com](mailto:drnaam@handdocs.com).

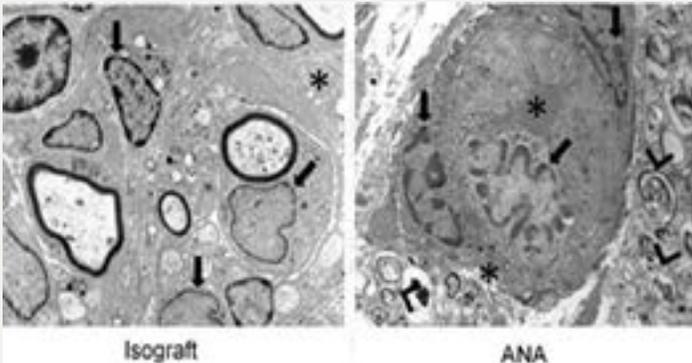
### Available Committees:

Bylaws Committee  
Coding and Reimbursement Committee  
Education Committee  
Finance Committee  
Grant Generation Committee  
Membership Committee  
Newsletter Committee  
Nominating Committee  
Scientific Program Committee  
Technical Exhibits/Website Committee  
Time and Place Committee

# What's New in Peripheral Nerve

## Molecular Imaging: The Future of Peripheral Nerve Injury Diagnosis

*Matthew D. Wood, PhD, Mikhail Berezin, PhD,  
Walter Akers, DVM, PhD and Susan E. Mackinnon, MD*



A dilemma with all nerve injuries (closed, open, gunshot wounds) is the difficulty in distinguishing recoverable (Sunderland I to III) from non-recoverable (Sunderland IV and V) nerve injuries in a timely manner. Additionally, predicting the longitudinal extent of the nerve damage for Sunderland fourth and fifth degree injuries can be challenging and is essential, so that the repair can be performed outside the zone of injuries. Current diagnostic techniques focus primarily on neurological and electrodiagnostic examinations requiring significant clinical experience and lengthy observance periods (possibly 3 months) to identify the severity of axonal injury. Delayed treatment for non-recoverable injuries leads to atrophy of muscle and chronic denervation, both of which are correlated with poor regenerative outcomes. Furthermore, improved diagnosis of nerve injury would also avoid unnecessary surgery for recoverable injuries (Sunderland II and III). Therefore, additional methods to diagnosis injury severity in a timely manner would be a powerful tool for the clinic.

Diagnosis of nerve injury could be enhanced by imaging technology to increase diagnostic accuracy and decrease the observance period needed to gauge injury severity (recoverable versus non-recoverable injuries). Non-invasive imaging has begun to emerge as a complimentary diagnostic aid. Biomedical modalities including computed tomography (CT), ultrasound (US), and magnetic resonance (MR), primarily provide gross morphological information about the state of the peripheral nerve and surrounding tissue (swelling and edema; tissue discontinuity: Sunderland IV or V injuries). They provide limited information regarding the severity of injury or the process of axonal regeneration (Sunderland I-III injuries).

Imaging at the molecular level would be invaluable, as this would provide information regarding axonal injury and regeneration. Molecular probes functionalized to bind specific biomarkers could provide timely identification and visualization of these processes for diagnostic purposes. Molecular imaging could also augment existing imaging modalities, as current non-invasive imaging could be paired with targeted imaging probes for detection of biological processes. However, molecular imaging, which has facilitated major advances in many clinical areas, has yet to be developed in the management of major peripheral nerve injuries.

Peripheral nerve injury and subsequent regeneration is a segmented process characterized by temporal and spatial changes in protein expression in nerves relative to the lesion site. Injured axons retract a variable distance and undergo a brief dormant phase during which injury-induced molecular signaling cascades are initiated. The regenerating axons express guidance-related proteins on the axonal growth cone following injury. Additionally, loss of axonal contact in the distal nerve initiates an inflammatory cascade that activates and recruits Schwann cells and macrophages that express distinct molecular markers as the degrading neural elements are cleared. Schwann cells change expression of growth factor and their receptors, cytoskeletal proteins and myelination proteins. During axonal regeneration, axons induce further changes to support cells, as myelin is produced to insulate the axons. Overall, peripheral nerve injury and axonal regeneration has plentiful biomarker targets available for detection in molecular imaging applications.

Other clinical areas have already taken advantage of targeted contrast agents for imaging, as these techniques have enabled deep tissue imaging for assessing arterial disease, brain function and cancer. The development of molecular imaging to peripheral nerve markers will have enormous potential for further basic science and translational studies to understand nerve injury and for diagnostic aid in the clinic.

## A Piece of My Mind

Loree K. Kalliainen, MD, MA, FACS

Program Director, Hand Surgery Fellowship  
Department of Plastic & Hand Surgery Regions Hospital  
St. Paul, MN

After a 12 day vacation in New Zealand in 2012, my husband and I decided that we wanted to spend more time there, meeting people and traveling. Vacation time being short, and cash being finite, I sought out working opportunities. A full year long sabbatical was not of interest to me, but, working with a head hunter, I found a three-month locum tenens position near Wellington, the capitol. I sent in my CV, two letters of recommendation, and photocopies of all of my certificates; the headhunter negotiated my salary and perks (including round trip airfare!). Once I had a formal job offer, I was able to get a national medical license and a work visa. All-in-all, it was very straightforward. We found a furnished apartment on-line (<http://www.bookabach.co.nz/>); it was on the hillside with spectacular views of Wellington Harbor. Though not plush, it had the basics, and the owners loaned us one of their cars for our stay which made everything easier. Ten minutes from the hospital driving on the “other” side of the road along curving mountain roads into the valley, I was happy that the car radio only had AM as it was important to focus, focus, focus on the driving. Only once did I end up on the wrong side of the road, and that was quickly corrected when I saw an oncoming car.

I hadn't realized that the hospital had a plastic surgery training program, and I was most fortunate to inherit one of the best residents (registrars) with whom I've ever worked from the Consultant whose job I was assuming. Organized, meticulous, knowledgeable, Dr. Amelia Murray oriented me; and I guided her, American style, through a variety of interesting cases. We did a lot of skin cancer resection and grafting, and the department chair, Mr. Chris Adams, kindly “collected” complex hand and nerve cases for me prior to my arrival. I was able to take the registrars through several nerve and tendon transfers and non-carpal tunnel nerve decompressions. I was able to do teaching sessions and finished off my stay with a QI study.

New Zealand has both public and private systems, and, much as we ask if you are academic or private here in the states, they were curious as to whether I was “public or private” at home. In Minnesota, I am technically in private practice but work in an independent academic trauma center, which has the philosophy of provision of care for all regardless of ability to pay, and perform clinical research. They didn't quite get it; to be fair, people here don't always get it either. Given the massive degree of anxiety related to current changes in our health care system, it was a fascinating opportunity to see how socialized medicine actually works as I was entirely in their public system. With exceedingly rare exceptions, patients appreciate their health care and are willing to wait their turns as they know that access is at least fair. The on-call surgeon was responsible for triaging elective patients into clinic; given their large prevalence of skin cancer,

if the lesion wasn't fairly large and symptomatic, your wait was long. It's totally pragmatic. A small basal cell carcinoma won't kill you, and if they addressed every small skin cancer, other more pressing issues would not be cared for. There are about 70 plastic surgeons in the entire country of 5 million. As a nation, they have made societal choices that we have, thus far, been unwilling to, but, again, it is more equitable.

Working in a different system was also an amazing opportunity to be “stupid.” At the end of my first case, the nurse asked what I would like to use to dress the wound.

Me: “A roll of Kerlix and an ace, please”

Her: “What?”

Me: “What?”

Most products had different names, and the ace wrap did not exist in my hospital. Lab values were different (glucose of 7?) Expectations by patients, physicians, staff, and trainees were different. Doing more under local was different (taking split thickness skin grafts, resection of 1/3 of the lower lip, Dupuytren's releases). I do a lot under local here, but this pushed my experience farther and would have pushed many U.S. patients over the edge. Not knowing everything off of the top of one's head does a lot to combat mental rigidity, and I had to quickly become relaxed about it. Thankfully, my coworkers were very collegial about my knowledge gaps regarding their system. There were many similarities between my system in the Hutt Valley and here at home: strong focus on the Triple Aim; heavy importance based on collegiality, culture, communication, quality, and service; knowledgeable and skilled partners; dedicated and friendly trainees, nurses, and therapists.

Though it was a disruption to my practice, my partners here were very accommodating. Fortunately, I am a member of a large group and so I could have my patients cared for while I was gone. I front-loaded all of my call from the three months to the preceding three months, performed the first stage of multi-stage operations before leaving town, and communicated with my patients before leaving. The dogs went to my father's. The neighbors watched the house and collected the mail. My husband was able to continue writing for an ad agency, and he met a lot of green energy people in New Zealand. We made great friends from the Wellington Vintage Car Club, did a scooter rally across the Northern Plateau, hiked the Queen Charlotte Track, sea kayaked, visited vineyards, went to the theater, museums, and concerts, and were invited to several people's houses for dinner and fun conversations. In all, it was a wonderful experience for us, and, thankfully, the group that I joined enjoyed meeting us also!



**A. Lee Dellon, MD, PhD**  
**ASPN President - 1992-1993**

## The Pudendal Nerve

The pudendal nerve is one that few members of the American Society for Peripheral Nerve Surgery have ever thought about either in terms of anatomy, patient symptoms, or surgical approaches.

My interest in the pudendal nerve followed from my attempts to restore sensation to the feet of diabetics. One day, one of my patients who had regained sensation in his feet, asked me if I could do the same thing by surgery for his penis. What was I to say? Actually I was not aware then that about 50% of diabetic men with neuropathy have some degree of erectile dysfunction. My anatomy dissections were at first focused upon the sites where parasympathetic and sympathetic nerves within the pelvis could be come entrapped as they exited the pelvis to enter the corpora cavernosa. Those early anatomy dissections led to a surgical approach to divide the urogenital diaphragm. During this phase of my anatomical and clinical investigation, I began to question the traditional anatomic pathway for the dorsal branch of the pudendal nerve. This led to our description of what I now call the inferior pubic ramus canal, a location along the course of the dorsal branch that is narrowed anatomically,<sup>1</sup> and could be the source of compression in, for example, bicycle riders with numbness in the penis and erectile dysfunction. In conjunction with one of former fellows, Oskar Aszmann, MD, who is now Professor of Plastic Surgery and Director of the Center of Extremity Reconstruction and Rehabilitation, Medical University of Vienna, a series of diabetic men with neuropathy and erectile dysfunction had a surgical decompression of the dorsal branch of the pudendal nerve, and were documented with neurosensory testing with the Pressure-Specified Sensory Device™, to have improved sensibility.<sup>2</sup> During a cadaver dissection with a Gynecologist from our Johns Hopkins faculty, Richard Marvel, MD, I realized that his

use of the term “Alcock’s Canal” and mine were a little different, leading to a historical paper that gives insight into this transition zone of the pudendal nerve across the pelvis.<sup>3</sup>

In 2010, Dr. Marvel invited me to give the first C. Paul Perry Lecture, a founders’ lecture, for the International Pelvic Pain Society. This was an eye opening meeting for me, introducing me to the concepts that were 28 million women in the U.S.A. with chronic pelvic pain. Now it is important to recognize that this society, the IPPS, defines pelvic pain as extending from the umbilicus to the mid thigh, and their meeting schedule is heavily involved with subjects such as intersitital cystitis, endometriosis, irritable bowel syndrome, vulvodynia, and chronic prostatitis. However, when pelvic pain was felt to involve the pudendal nerve, a clinical problem they termed “pudendal neuralgia,” all patients who came to surgery had a transgluteal approach to divide the sacrotuberous and/or sacrospinous ligaments. This surgery is done in the U.S.A. by a very few surgeons: two Gynecologists, one Urologist, one Orthopedic Surgeon, and one Neurosurgeon. Of interest, this surgery was first done in Egypt for chronic rectal pain by Dr. Safik, in 1995, now deceased. The work subsequently became centered in Nantes, France, with Dr. Robert, and is continued there and elsewhere in France and Belgium by his “disciples.”

**Table 1: Results of Pudendal Nerve Decompression**

Author/Year	Surgical Approach	Structure Divided	# of Patients	Excellent	Good	Failure	Months to Recovery
Safik/1995	Lithotomy IschioRectal	A	10	80*		20	N/A
Safik/1996	Lithotomy IschioRectal	A	21	60**		40	N/A
Mauillon/1993	Prone Transgluteal	SS, ST, FP, Ap	12	25**	9	66	3-12
Bautrant/2003	Lithotomy IschioRectal	Ap	104	62	25	13	0-12
Beco/2004	Lithotomy IschioRectal	Ap	14	68		32	6-12
Robert/2005	Prone TG (M)	ST, SS & Ap	16	70% > 30% relief			3-12
Ansell/2007	Prone Transgluteal	SS, S, FP, Ap	58	44	23	33	N/A
Robert/2007	Prone TG (M)	ST, SS, Ap	?	40	30	30	4-18
Filler/2009	Prone TG (H)	P, ST***	147 (185 ops)	20	67	13	4-12
Anatolak/2009	Lithotomy IschioRectal	SS, AP					
Dellon/2009	Lithotomy Pubic Ramus	Aa	5	100			2-6
Hibner/2010	Prone TG (M)	ST**** Ap	?	33	33	34	18-24

## Featured This Fall - ASPN Past Presidents: Pearls and Insights

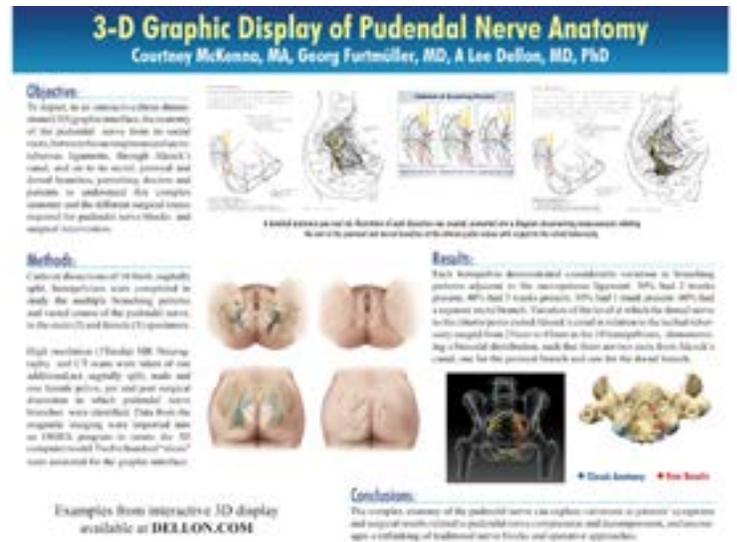
ST, sacrotuberous ligament; SS, sacrospinous ligament; FP, falciform process of ST ligament; P, pyriformis muscle division; A, Alcock's Canal, either Aa, anteriorly at exit of canal, or Ap, posteriorly at entrance to canal; NA, data not available,

TG, transgluteal either (H) high and lateral or (M) medial, oblique over tuberosity.

- \* Symptoms were erectile dysfunction with perineal hypesthesia
- \*\* Symptoms were fecal incontinence with perianal hypesthesia
- \*\*\* Partial sacrotuberous ligament division was done in just 12% of surgeries
- \*\*\*\* After ST division, pudendal nerve is wrapped in collagen tube, which is then filled with platelet rich plasma

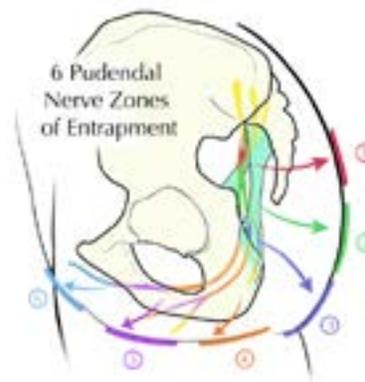
Outcomes reportedly are frequently poor, and not often excellent, with improvements described as taking 18 to 24 months. This seemed, therefore, like an exciting area for a Peripheral Nerve Surgeon to become involved. I have summarized the world experience in this area in Table 1. This may be found in Chapter 12 on Pelvic Pain, in my book, *PAIN SOLUTIONS*, available for free download on the home page of Dellon.com, which you can link to directly at <http://www.dellon.com/ps/chapter12.pdf>. The references for Table 1. are given in this book chapter.

For me, the anatomy and of the pudendal nerve is challenging in the cadaver, and difficult in the operating room. To better understand it, and to provide a method for patient education and physician education, a Master's Thesis project was begun in 2011 with Courtney A. McKenna, in the Johns Hopkins School of Art as Applied to Medicine. This has resulted in a 3D model, with traditional anatomy on the right side of the pelvis and the anatomy as understood from our new dissections on the left side of the pelvis. This is available to view as it rotates at my website, Dellon.com. It can be linked to directly from this article. It is on the bottom of the home page. The poster presented to the 2012 meeting of the IPPS is given in Figure 1., below. This was achieved by scanning a cadaver in a 3T MRI, then subtracting all but the bone signals, and then "sculpting" in the observed nerves from the cadaver dissections. Then the illustration was made to rotate not only 360 degrees, but also into the prone and lithotomy positions to make it relevant to clinical problems. This anatomy research has been accepted for publication.<sup>4</sup>



**Figure 1.** Poster from the 2012 International Pelvic Pain Society meeting.

As a Peripheral Nerve Surgeon, it was clear that not all pudendal neuralgia patients had a compression beneath the sacrotuberous ligament. Therefore, the approach that I have suggested follows our approach to pain in the upper or lower extremity. What was the injury? Is the pain or symptoms likely to be from a nerve injury, with a neuroma being present, or likely to be from a nerve compression, with a site of chronic compression being present. The first sensory branch of the pudendal nerve is to the rectum. Therefore, if rectal symptoms are present, a posterior transgluteal approach is indicated. If there no rectal symptoms, but the symptoms in the man include the penis and/or posterior scrotum & perineum, or in the women include the clitoris and/or labial & perineum, then an anterior inferior pubic ramus approach is indicated. Potential sites of entrapment and surgical approaches are given in Figure 2., below.



**Figure 2.** In zone one, the usual cause is a tumor of the sacral plexus that can be approached through the pelvis. Zone 2 would be a cause related to a fall posteriorly and would be a transgluteal approach to get to the sacrotuberous ligament. Zone 3 would be the entrance to

Alcock's canal, and can be approached the same as in Zone 2. Rectal surgery can injure the rectal branch in Zone 3. A pelvic fracture could injure the pudendal nerve in Zone 3 and 4, as well as placement of mesh for prolapse of the rectum or bladder

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into the vagina. Zone 5 and Zone 6 are the sites respectively for entrapment of the perineal and the dorsal branches of the pudendal nerve, and can be injured in cycling or childbirth, or transvaginal surgery.

As most readers have never seen these nerves, I thought it would be instructive to see them, through the anterior approach (see **Figure 3**).

A.



B.



**Figure 3.** Lithotomy position, left incision over inferior pubic ramus alongside labia. A) orienting view. Upper blue loop on dorsal pudendal br. to clitoris. Lower blue loop on perineal br. pudendal nerve to labia. B) After neurolysis, note red, narrowed, inflamed area where dorsal br. was compressed, and thicker whitish area more proximally.

My current research is related to outcomes related to these surgical approaches for pelvic pain. Patients are selected only after they have had a positive response to a pudendal nerve block and only after they have had a full trial of medical management and pelvic floor therapy. Working with Gynecologists from the New York University faculty, we have been using validated outcome instruments such as the VQ (vulvar pain functional index) and the FSFI (female sexual function index), and in men the CPSI (chronic prostatitis symptom index), and comparing these to the standard Numerical Analog Scale. The early results of analyzing the confounding affect of comorbidity on pelvic

pain surgery demonstrates that the most significant predictor of failure is untreated anxiety/depression. This has importance to all peripheral nerve surgery, as it is critical to get the patient's underlying central nervous system into the most peaceful resting state we can prior to operating on the peripheral nervous system. Involving someone trained in psychotropic medication, like a Psychiatrist, is most helpful. The subject of central sensitization would be a good one for a future ASPN Newsletter to consider.

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**Susan E. Mackinnon, MD**  
ASPN President - 1995-1996

### **Schwann Cell Senescence: A New Consideration in Nerve Repair and Grafting**

*Gwendolyn Hoben MD, PhD, Amy M Moore MD, Matthew Wood PhD, and Susan E Mackinnon MD*

*Washington University School of Medicine, Department of Surgery,  
Division of Plastic Surgery, St. Louis, MO*

When approaching a complete motor nerve injury, there are several factors critical to the outcome: time from injury, distance to the target muscle, technique of the repair, and patient characteristics. Timing of the repair is critical. Delays in repair and associated prolonged periods of denervation lead to loss of muscle mass and integrated motor function. Attempting to re-innervate denervated muscle after 12-18 months is futile due to changes at the muscle level. While timing of treatment is crucial, the distance to the target muscle is dependently related. Injury to the ulnar nerve in the axilla is much less likely to have a positive outcome in the hand than an injury to the nerve at the wrist because the rate of innervation, approximately 1mm/day or 1in/month, is inadequate to reach the muscle before muscle loss. A long regeneration distance makes it unlikely that axons will reach the target muscles in time. Besides timing and distance, the repair technique and patient age play a role. Increased surgical experience and younger patients are associated with improved outcomes. Despite taking all of these factors into account, there are still other variables at play. New evidence suggests that changes in the regenerative microenvironment may also be important in the clinical outcome, most notably is the phenomenon referred to as Schwann Cell (SC) senescence. The goal of this review is to introduce SC senescence, summarize recent data relating to its effect on nerve repairs, and to offer clinical nerve grafting techniques to improve regeneration.

The regenerative microenvironment of the injured nerve is composed predominantly of SCs, fibroblasts, macrophages, and a milieu of growth factors, extracellular matrix components, cell adhesion molecules and matrix metalloproteinases that contribute to regeneration. One of the most studied components is SCs. In the uninjured nerve, SCs provide growth and structural support to the axons. They are responsible for myelination, creating extracellular matrix, and secreting neurotrophic factors. After an injury, their gene expression and function change dramatically. They change to a regenerative phenotype, begin proliferating, increase migratory activity toward the injury site, and secrete

growth factors beneficial to nerve regeneration. SCs are also responsible for mechanical cues to regeneration: clearing axonal debris and laying down laminin to guide growing axons. The benefit of SCs is clear when comparing nerve autografts (isografts in animal models), which contain native SCs, to acellularized nerve allografts (ANAs). In animal models, isografts regenerate 50-100% more nerve fibers, likely attributable to the presence and function of viable SCs.

Beyond the simple presence of SCs, the SC must be in the regenerative phenotype. However, new evidence indicates that in certain nerve injuries, the SCs undergo senescence. Senescence is permanent growth arrest associated with a change in the secretory phenotype of the cell. These changes are typically induced by replicative exhaustion and chronic cellular stress, both of which are characteristics of the nerve injury environment. Senescence also increases with age and may be an explanation why we see slower, less effective regeneration in older animals. Senescent SCs have reduced proliferation, decreased growth factor production, and result in poor myelination. In parallel to the aging SC, a likely reason for these changes in the nerve injury microenvironment is chronic cellular stress; sources of which include inadequate growth conditions secondary to ischemia and prolonged signalling for proliferation. As the nerve gap lengthens and there is a greater regenerative distance, the factors leading to chronic cellular stress increase. Greater distances mean increased proliferative burden on the SCs to fill the gap. Further, the longer gap involves a greater distance from the vascularized nerve ends, resulting in an ischemic environment with reduced nutrient support.

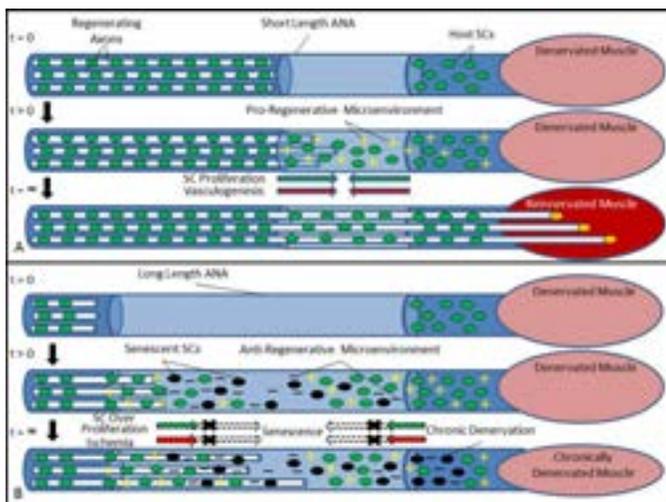
Evaluating the results from long nerve gap (>30mm) models and the difficulty with regeneration across ANAs at this distance was the impetus to look for senescence. Long nerve gaps are a clinical challenge and at a cellular level are also highly conducive to cellular stress. In contrast, there are numerous successful strategies to repair short gap (<30mm) injuries to small diameter nerves with the use of autografts, conduits and ANAs. However, in moving to the long gap injuries (>30mm) and especially larger diameter nerves, only autografts have shown consistent successful outcomes. Autografts, however, do come at a price: donor site morbidity and increased operative time compared to using an “off-the-shelf” product.

To better understand regenerative failure associated with long gap nerve repairs, our group examined acellular nerve allografts ranging in length from 20-60mm in a rat sciatic nerve transection model. After 10 weeks, nerve regeneration in the 60mm ANA grafts was inferior to that in the 20mm grafts. Additionally, the length of the axon regeneration front was directly affected by the length of the graft. In a

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## Featured This Fall - ASPN Past Presidents: Pearls and Insights

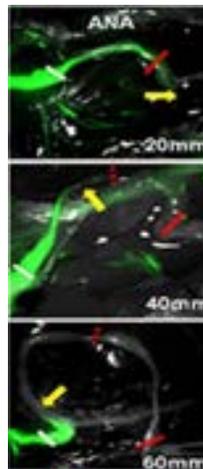
60mm ANA, the axonal front was only 5mm while that in the 40mm ANA was nearly 20mm. In contrast, the axonal front completely cleared the 20mm gap. These surprising data indicated that the regenerative microenvironment worsened as the length of the ANA increased, so we hypothesized that senescence may be a contributing factor. SCs from the distal nerve graft were evaluated for protein and genotypic markers of senescence: senescent associated  $\beta$ -galactosidase, p16, interleukin-6, and interleukin-8. In long ANAs, the expression of these senescence-associated markers increased with increasing distance from the proximal edge of the injury



(Figure 1).

Additionally, electron microscopy (EM) showed changes associated with senescent cells: altered chromatin and abnormal extracellular matrix.

The proteins secreted by SCs are of the utmost importance for the regenerative microenvironment provided to axons. Given the reduced nerve growth found in the long ANAs, especially at the distal end, it would be expected that the quantities of neurotrophic growth factors produced by senescent SCs were altered. Gene expression analysis in an in vitro model of senescent SCs showed altered expression for glial-derived neurotrophic factor (GDNF), nerve growth factor (NGF), and brain-derived neurotrophic factor (BDNF) compared to normal SCs. GDNF has been found to have a direct trophic effect on dorsal root ganglion cells, motor neurons, and autonomic neurons. NGF is important in sensory and sympathetic nerve fiber growth. It guides axon growth, and it has been found to have increased secretion in SCs near the distal end of injured nerves. NGF also increases SC secretion of BDNF, a neurotrophic factor that targets motor neurons and increases both myelination and the diameter of regenerated nerves. Senescence-related decreases in the local concentration of these growth factors could strongly impair



axon growth (Figure 2).

Another clinical scenario conducive to chronic cellular stress is “banking” a nerve graft for future reconstruction. “Banking” a nerve graft involves performing a proximal nerve coaptation to a donor motor nerve and allowing the nerve to regenerate over time through the graft. Traditionally, banked nerve grafts did not include a distal connection of the graft. For example, in the case of a panbrachial plexus injury where a free functional muscle transfer is anticipated for elbow flexion, a long nerve graft may be connected proximally to a donor

nerve (i.e. intercostal nerves) and banked in the subcutaneous tissue. This banked nerve is then monitored with a Tinel’s sign to determine when sufficient axons have regenerated to power the transferred muscle. A study from our group in 2002 looked at long isografts (8cm) connected end to side to the sciatic nerve and connected distally end to end to the tibial nerve in a rat model. One of the experimental groups was similar to a banked nerve in that the distal end was not connected to the posterior tibial nerve but instead left free. Compared to the group that had a distal coaptation, there were 80% fewer regenerated nerve fibers. In retrospective analysis of those nerve samples, the group without a distal coaptation showed senescent changes on EM while the distally connected nerve graft did not. Similar results were also found on retrospective analysis of nerve graft samples taken from a primate study: the proximal trunk of the facial nerve was transected and grafted with a 10cm sural autograft and either connected distally or not. Again, the lack of a distal connection resulted in senescent changes in the SCs. In the situation of a banked nerve there is no distal nerve end to provide healthy SCs or increased vasculature to supply appropriate blood and nutrients to cells within the graft, thus contributing to chronic cellular stress even in the case of an autograft that has SCs.

Senescence due to chronic cellular stress fits into the current paradigms of nerve repair. Shorter gaps result in less cellular stress and have better prognosis, while the worsened prognosis of nerve repairs in long gap injuries and older patients fits well with senescent changes. As we learn more about this process and what variables lead to senescence, these lessons can be applied to clinical practice. Future work is needed to examine strategies to prevent senescence in long gap injuries and better engineer ANAs to be more effective. Moreover, senescent changes in the other cells involved in nerve regeneration, fibroblasts and endothelial cells, also need to be examined. The evidence of senescence in banked grafts

is highly suggestive that connecting a banked nerve graft distally could be beneficial in reducing the chronic cellular stress environment and thus, better maintaining the axons for regeneration. There are many questions yet to be answered regarding SCs and senescence, but the evidence is mounting that senescent SCs are another critical variable in nerve repair.

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### Figure Legends

**Figure 1:** ANAs were placed in Thy 1-GFP expressing rats such that the growing axonal front shows green fluorescence, the most distal axonal front at 10 wks is indicated by the yellow arrow, the solid white line is the proximal coaptation and the solid red line is the distal coaptation. The hash marked red line indicates the graft suture line. These images show that the axonal growth front is inversely related to the length of the graft.

**Figure 2:** ANAs lack SCs and thus, require host SCs to populate the graft and support axonal regeneration. A) In small volume grafts, normal levels of host SC proliferation are sufficient to populate the graft. These SCs create a regenerative microenvironment sufficient to successfully reinnervate the target muscle. B) In large volume ANAs, the volume to be filled and the length for revascularization is so great as to create significant chronic cellular stress, resulting in SC senescence. The lack of a regenerative microenvironment prevents successful reinnervation.

### Mackinnon receives Jacobson Innovation Award

**Honor recognizes pioneering surgeon's innovative use of nerve-transfer procedures**

Renowned surgeon Susan E. Mackinnon, MD, of Washington University School of Medicine in St. Louis and Barnes-Jewish Hospital, received the 2013 Jacobson Innovation Award of the American College of Surgeons (ACS) at a dinner in her honor Friday, June 7, in Chicago.

Mackinnon, the Sydney M. Shoenberg Jr. and Robert H. Shoenberg Professor and chief of the Division of Plastic and Reconstructive Surgery, received the international surgical award for her leadership in developing innovative nerve-transfer procedures to treat patients with devastating peripheral nerve injuries.



AMERICAN COLLEGE OF SURGEONS

*A. Brent Eastman, MD, president of the American College of Surgeons, presents the College's 2013 Jacobson Innovation Award to Susan E. Mackinnon, MD, for her leadership in developing nerve-transfer operations.*

The Jacobson Innovation Award honors living surgeons who have been innovators of a new development or technique. The award stems from a gift from Julius H. Jacobson II, MD, and his wife, Joan. Jacobson is a general vascular surgeon known for his pioneering work in the development of microsurgery.

Before Mackinnon's surgical approach came to light, the traditional treatment of peripheral nerve injuries involved repairing the nerve at the site of the injury with microsutures and expendable sensory nerves from elsewhere in the body. However, with this method, nerve regeneration was slow and return of muscle function was often poor. In 1991, Mackinnon began zeroing in as closely as possible on the motor endplates of the muscle that had been denervated.

Her surgical approach involves working with expendable branches within major nerves that are nearer the compromised muscle. Essentially, the nerve-transfer technique changes a high-level proximal injury (such as at the neck) to a more distal injury (such as at the axilla, arm, forearm or hand) and avoids the detrimental impact of prolonged muscle denervation.

Mackinnon performed the first nerve transplant in 1988, using nerves from a cadaver to restore feeling and movement to a boy's injured leg. That landmark operation began a quarter century of novel work in nerve transplantation and led to many other surgical firsts.

In 2012, Mackinnon and her surgical team at Barnes-Jewish received worldwide attention for a nerve-transfer procedure that successfully enabled a quadriplegic patient to regain some use of his hand.



AMERICAN COLLEGE OF SURGEONS

*Pictured (from left) are Samuel A. Wells Jr., MD, former head of the Department of Surgery at the School of Medicine; Susan E. Mackinnon, MD; Timothy Eberlein, MD, current chairman of the Department of Surgery; and*

*Bernard Langer, MD, former chairman of the Department of Surgery at the University of Toronto. The three men — the only chairmen of surgery Mackinnon has worked for — attended the American College of Surgeons dinner Friday, June 7, in Chicago, at which Mackinnon received the 2013 Jacobson Innovation Award.*

Mackinnon's groundbreaking work has produced a paradigm shift in the treatment of peripheral nerve injuries. Today, surgeons introduce new nerve transfers on a regular basis. By contrast, nerve grafts — which previously added a year or two of nerve regeneration — are avoided altogether. Legions of patients who have regained function in their injured arms and legs have benefited from Mackinnon's insightful approach to developing nerve-transfer operations.

Mackinnon, a fellow of the Royal College of Surgeons of her native Canada and of the American College of Surgeons of the United States and Canada, has been responsible for the interdisciplinary training of a generation of specialists interested in the surgical treatment of peripheral nerve injuries, including neurosurgeons, orthopedists and plastic surgeons.

She credits her success in the field to three decades of research funding support that she has received from the Medical Research Council in Canada and the National Institutes of Health (NIH) in the United States. She also credits her success to having worked at two outstanding academic institutions — the University of Toronto and Washington University — and with a talented research team for three decades.



**Allen Van Beek, MD, PhD**  
ASPN President - 1997 - 1998

### Some Things That Haven't Changed

Dr. Naam asked if I would contribute something noteworthy to the newsletter. When Nash asks one to work it is difficult to decline. I was once told that "saying no makes your yes more significant," but not when Nash asks. There would likely be a high definition photo of saying no that would show up. Many would probably admitted toward the end of one's career it is much easier to decline request and it would probably also be granted that professing successes, failures and experience are broader compared to when one is starting a career.

One observation and a salute to AAHS, ASRM and ASPN are the foresight of past leaders and flexibility of present leadership to keep the three organization's meeting together. It is an efficient, creative, and economical way to gain access to the acknowledge leaders in reconstructive surgery from throughout the world. It's truly amazing, and we need to encourage leadership to keep this unique affiliation intact.

Our practices have changed as technology advanced but there are some principles regarding the peripheral nerve that remain. I'll list some of those caveats, previously called pearls. Now they are called my preferred technique in texts.

1. When dissecting in areas where nerves exist use loupe magnification. There aren't many places where small nerves aren't present. Severe even a very small nerve and there may be significant consequences.
2. When injecting local anesthetic in areas where nerves exist use #27 gauge or smaller needles. The diameter of a 27 needle is about the same size as a fascicle. of the digital nerve. If injecting around large nerves use ultrasound to guide the injections. Sticking a needle directly into an intact peripheral nerve should be avoided if possible and injecting nerves with pharmacological material may compound the problem. Using small caliber needles also requires using smaller volume syringes to allow easier injection control.
3. When coagulation is necessary adjacent to peripheral nerves use a micro bipolar coagulation system and identify the bleeder point more accurately using the previously mentioned loupes. When over 48 years of age you need the loupes even more. I prefer 4X power loupes.

4. When repairing small nerves use small sutures, when repairing large nerves use small sutures for repairs. Remember the sutures materials will be embedded within the neuroma.
5. When fascicles are dangling outside your completed repair site trim them back so that can coapt and align more readily.
6. If you don't have a variable nerve stimulator available don't attempt advanced nerve reconstruction or transfers.
7. Learn how to use intraoperative nerve stimulation and recording techniques and have the equipment available. Use ICD-9 code 95920 for reimbursement.
8. The repair of a peripheral nerve in adults never achieves normal status and explaining that to patients prior to repair makes impact management after repair easier.
9. A Hand Therapist in your office will add to your practice effectiveness and personal happiness.

These are all Level V pearls, but I hope you find them interesting.



**David T.W. Chiu, MD**  
ASPN President - 1999 - 2001

### Strategic Application of the Autogenous Venous Nerve Conduit (AVNC)

## Technique for Management of Defect of the Superficial Radial Nerve

Utilizing a vessel segment as a bridge graft to restore the continuity of a damaged peripheral nerve has been a recurrent aspiration for over a century, however none of the attempts prior to 1980 proved to be fruitful. Such dismal history led to the concluding summation of its unqualified failure in the second edition of the monumental treatise *Nerve and Nerve Injury* by the late Sir Sydney Sunderland. However, along with the escalating success of the technique of autogenous nerve grafting for peripheral nerve reconstruction, the need for source of nerve graft has equally escalated. Extensive review of literature led to the conclusion that prior experimentation lacked the sophistication of microsurgery and most likely resorted to use allo-graft for interposition vessel bridge grafting. Such revelation prompted a young resident to pursue a comparative study utilizing autogenous interposition reversed vein graft as a bridge graft to reconstruct a sciatic nerve with a 1 cm nerve gap in rodents.<sup>1</sup> The success of this experimentation led to a single blind prospective comparative study utilizing autogenous segments of saphenous brevis as reversed bridge graft reconstructing a series of distal cutaneous sensory nerves. Such clinical trial validated the experimental observations.<sup>2</sup> The term “nerve conduit” was coined and the technique of AVNC was subsequently adopted as a commonly used clinical procedure; a CPT code 64911 was assigned in 2007.

Since this epical success, a global effort has been made to develop alternative synthetic nerve conduits and alternatives to nerve conduits. Much progress has been made, however, for nerve defect of 3 cm or less the most applicable biological nerve conduit remains the autogenous venous nerve conduit.

Herewith presented is a very specific situation whereby the superficial radial nerve is injured causing a segmental defect. Owing to its proximity to a large tributary of the cephalic vein from the injured superficial radial nerve, utilizing reversed cephalic vein graft as a nerve conduit proved to be most efficacious. A series of three patients who have an average of twenty five years of follow-up has ascertained its efficacy. The case with thirteen years of follow-up is presented as to the following. The patient was a thirty three year old left hand dominant amateur hockey player who reportedly sustained a laceration when another hockey player skated over his

outstretched left forearm. This resulted in a crush laceration of his superficial radial nerve and the cephalic vein at the distal forearm level. Exploration revealed transaction of all but one fascicle of the superficial radial nerve. There was a segmental defect of 3 cm. A 4 cm segment of the distal and proximal stump of the cephalic vein was harvested and was utilized as a reverse interposition venous nerve conduit (see diagram). Ten years following the reconstruction, examination revealed 10/10 sensory indexing and no Tinel or parathesia. This case example confirms the applicability of autogenous venous nerve conduit (AVNC) for overcoming a nerve defect of about 3 cm in the superficial radial nerve.

The juxtaposition of the cephalic vein and the superficial radial nerve is an anatomical serendipity. The excellence in result warrants the consideration of such application, should similar defect present itself. While the author and many others consistently achieved excellent results utilizing AVNC as a means of peripheral nerve reconstruction for nerve gap less than 3 cm,<sup>3</sup> there are distractions such as the speculation that the vein graft might collapse. However, previous study in my laboratory determined that after the interposition vein graft is set in place, despite meticulous hemostasis, oozing of blood will occur at the nerve end. The blood will fill and distend the lumen of the interposition vein graft. Exploration of the vein graft within the first week revealed a “blood-sausage-like” appearance.<sup>4</sup> In conclusion, autogenous venous nerve conduit is an applicable technique for peripheral nerve reconstruction in distal sensory nerve with a nerve gap of 3 cm or less. The proximity of the superficial radial nerve to the cephalic vein particularly constitutes a strategic situation whereby its AVNC application is inviting!

#### Diagram



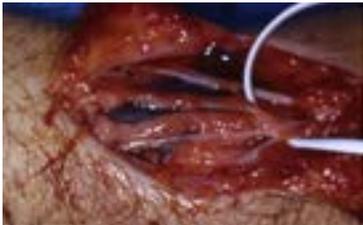
**A. Laceration caused by a skate**



**B. Laceration through the superficial radial nerve (SRN) and the cephalic vein**



**C. All but one fascicle of the SRN was transected**



**D. A 4 cm segment of both the distal and proximal stumps of the cephalic vein was harvested. The transected fascicle of the SRN was separated into two groups and bridged with the cephalic AVNC**

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**William Kuzon, MD**  
ASPAN President - 2002 - 2003

## Ramblings of an Old Dwarf

"We are like dwarfs sitting on the shoulders of giants. We see more, and things that are more distant, than they did, not because our sight is superior or because we are taller than they, but because they raise us up, and by their great stature add to ours."

—John of Salisbury, 1159 AD

When Nash asked me to write a short piece for the ASPN newsletter, I had one thought: despite a lot of opinion to the contrary, getting old, or at least getting older, is great. To me, the biggest advantage of aging is that you can say pretty much what ever you want. If you say smart stuff, you get credit for being wise. If, despite being somewhat less stupid than your younger self, you say stupid stuff, it get's written off as the ramblings of an old guy with no repercussions. Here we go.

I was a chief resident at the University of Toronto when I attended ASPN's first scientific meeting in 1991, I've attended every single meeting since, and I had the honor of serving as ASPN president for 2002-2003. That's surprising to me because I definitely don't consider myself a nerve surgeon or a nerve researcher. In fact, I'm actually not all that interested in nerves. Rather, I have had a career-long, abiding interest in the structure and function of the most awesome organ system we possess, skeletal muscle. The nervous system does control our muscles, of course, so I haven't been able to avoid work on peripheral nerves entirely, and as I've already mentioned, I've certainly hung out with and observed you nerve-geeks and your work over a long period of time. From this detached, minimal-skin-in-the-game vantage point, take what follows as either wisdom or as demented ramblings.

In 1906, Ramon Cajal was awarded, along with Camillo Golgi, the Nobel Prize in Physiology and Medicine "in recognition of their work on the structure of the nervous system." In the field of peripheral nerve research, Cajal is best known for his experiments on axonal regeneration, as it had been previously thought that injured nerves could not re-grow. In the early 1950's, Rita Levi-Montalcini discovered and characterized nerve growth factor. NGF was one of the first growth factors discovered and led to her sharing the Nobel Prize in Physiology and Medicine in 1986 with Stanley Cohen, who described EGF. Cajal and Levi-Montalcini's work spawned an enormous body of peripheral nerve research on the use of growth factors or other means to speed up or improve the vigor of axonal regeneration. The solution to the problem

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## Featured This Fall - ASPN Past Presidents: Pearls and Insights

of peripheral nerve injuries seemed obvious: make axons regenerate better. However, this huge amount of work ignored two things: 1) left alone, axons regenerate really well and 2) the problem with recovery after nerve injuries isn't because axons don't regenerate well, it's because peripheral nerves, unlike skeletal muscle, are really stupid. Transected peripheral axons regenerate like crazy but, unfortunately, they have no idea where they are going. Experimental work showing that motor axons preferentially grow towards motor targets and sensory nerves prefer sensory targets and that Schwann cells have a spectrum of phenotypes to support axons with differing responsibilities has not translated into anything with clinical utility, most certainly because the magnitude of these effects is far too small to have any hope of organizing the robust regeneration of tens of thousands of axons in a mixed human peripheral nerve. Because nerves are so stupid, the only hope of getting the circuitry hooked up right is to pre-select axons with homogenous purpose and then to give them no choice about where they can go. That is why the recent, wide application of nerve transfers to peripheral nerve surgery has been the biggest advance in this field during my career. Of course, the idea of nerve transfers has been around for more than 100 years; the obvious solution was sitting in front of us all along.

In the early 1990's, Fausto Viterbo revived interest in "termino-lateral-neurorraphy." Again, this was not a new idea, having been reported by Balance as early as 1903. Regardless, TLN enjoyed a significant revival with dozens of presentations at ASPN and many publications on this topic in the few years after Viterbo's first paper. A central question in much of this work was could axonal sprouting and regeneration occur in the absence of axotomy? Did we need to make an "epineural window," a "perineural window," or no window? Did we need to cut axons? After a lot of effort, we concluded that what was already known was still true - axons can sprout without being cut first. However, while peripheral axotomy results in robust regeneration, nodal sprouting from intact peripheral axons, along the course of an otherwise intact peripheral nerve is not robust enough to be of any use clinically. Why did we perseverate on this blind alley? We knew we could partially transect any peripheral nerve without compromising distal function. We knew that axotomy results in robust axonal regeneration. Terzis, May, and others had already described the utility of partial axotomy with an end-to-side coaptation for XII-VII transfer. Aha! "TLN" with axotomy is a nerve transfer! The obvious, clinically relevant answer was in front of us all along.

Another issue that has consumed nerve-geeks over time is "denervation atrophy." Based on long-standing clinical observations that peripheral nerve repairs resulted in the recovery of function if the motor axons reached the muscles quickly but that motor nerve repairs were futile if the period of denervation was prolonged, it became dogma that after some period of time, a year approximately, changes in the

muscle fibers precluded synaptogenesis. Changes in the expression of myocyte membrane receptors essential to synaptogenesis became a focus of multiple research groups. Ditto for the "senescence" of satellite cells. Interventions to prevent these changes proliferated. Electrical stimulation, exogenous growth factors, physiotherapy, sensory protection, babysitting, etc. were targeted at maintaining the muscle fibers in a state receptive to reinnervation. Then, in a series of elegant experiments, Tess Gordon demonstrated conclusively (to me at least) that the primary reason long-term denervated muscles fail to recover after peripheral nerve repair is not related to changes in the myocytes, but to the collapse and fibrosis of the intramuscular endoneurial conduits necessary for intramuscular axonal regeneration. Sensory protection and babysitting may help the myocytes, but their primary benefit is maintenance of the endoneurial conduits. Once again, aha! The solution to long-term, denervation atrophy is to prevent the conduits from collapsing. What do we do that when a distal muscle is denervated from a proximal nerve injury? The obvious answer sitting in front of us: reinnervate that muscle quickly using a nerve transfer.

So, the biggest transformative "innovation" in clinical nerve surgery during my career was not an innovation. The idea was not new, the signals pointing to its utility were there and were obvious, but it still took 100 years to figure it out. Is this an isolated example? Well, what is the other huge advance in Plastic Surgery in the past 20 years? My answer would be Ian Taylor and other's re-discovery of the blood supply to the skin via myocutaneous and fasciocutaneous perforators. This "innovation" is based on anatomical knowledge that has been present since at least the mid-19th century and the paramedian forehead flap, first reported in 600 AD, can be considered a perforator flap. Muscle, myocutaneous, and perforator flaps were performed in the 1800's.

Why does it take hundreds of years to rediscover good ideas?

I think it's because as researchers, we are fascinated by what's new, novel, first, and unique (all words that are banned in my research group, by the way). We are also technocrats, fascinated by high tech, by molecular mechanisms, and the like. And, unfortunately, the lack of a comprehensive, scholarly approach to research is often apparent in our journals and at our meetings (don't get me started on the body-count, 4 minute talk, minimal-reportable-data-unit approach to science today). So, rant or wisdom, the stories of nerve transfers and perforator flaps demonstrate that we would all do well to spend more time in the library discovering and understanding low-tech, old "innovations." Let's not forget or ignore the vantage we can get from the shoulders of giants.

Or, put another way:

"Some people feel the rain. Others just get wet."

- Bob Dylan



**Steven McCabe, MD**  
ASPN President - 2004 - 2005

### The Cause of Carpal Tunnel Syndrome?

#### Introduction

Understanding the causation of Carpal Tunnel Syndrome (CTS) has been elusive despite the ubiquitous nature of this disorder. To date there has been no comprehensive theory to coordinate multiple seemingly disparate factors into a unifying theory of causation.

#### Causation

In an interesting historic review written by Evans, an observation is made that the concept of causation has changed with our ever improving concepts of complex disease.<sup>1</sup> Rothman also describes a sufficient cause and a complex causal mechanism.<sup>2</sup> A sufficient cause will always produce the effect in short order. A complex causal mechanism is a series of events that will ultimately produce the effect. A casual mechanism need not be exclusive in that there can be more than one chain of events that will produce the outcome. This is useful from an intervention standpoint and it also describes a framework upon which to try to understand causation in CTS.

A lot of evidence has accumulated over many years that compression of a peripheral nerve results in biochemical, ultrastructural, microscopic, and macroscopic changes in the nerve. There is also evidence that links nerve compression of the median nerve in the carpal tunnel with the symptoms of CTS.

Further to this, MRI studies confirm that wrist flexion can cause a reduction in the cross section of the carpal tunnel.<sup>3,4</sup> Multiple studies show that wrist flexion or extension increase the pressure in the carpal tunnel, and we believe increased pressure will cause changes in the median nerve that can result in the clinical syndrome we know as CTS.

#### The Potential Role of Sleep Position

When the median nerve is compressed, patients will develop symptoms in the hand. Night waking is one of the classic symptoms of CTS and is one of the key features in its diagnosis. When patients wake with numbness in their hand it is reasonable to believe the median nerve is being compressed. One potential cause of increased pressure at night is fluid redistribution that occurs in a recumbent

position. A second possible reason is that when we sleep our wrists can assume a posture of flexion or extension, compressing the median nerve as noted above. We were motivated by the possibility that the compression caused by wrist position was an initial step in ongoing nerve compression that could ultimately lead to the clinical syndrome.

We discovered there were demographic similarities between CTS and insomnia. Insomnia is about ten times more prevalent than CTS so we did not believe the sleep disturbance caused by CTS could be the cause of this similarity with insomnia.

Beyond age and gender we found the medical and personal characteristics that were associated with CTS were associated with sleep disturbances. We created a hypothesis that the associations of CTS acted through an increased preference for sleeping on the side that in turn increased the probability of wrist deviation, compressing the median nerve, and subsequently causing CTS.<sup>5</sup>

In our second study, we found an association between stated preference for sleeping on the side, gender, and increasing age in women.<sup>6</sup> In our third study we found that men and young women with CTS stated a preference for sleeping on their side significantly more than control patients.<sup>7</sup> In our fourth study, we found CTS patients had varied and multiple sleep disturbances.<sup>8</sup>

#### Summary

Considering sleep position on the side as a critical step in a complex causal mechanism we can put together a comprehensive theory for causation in CTS. The critical but simple causal pathway is that the associations of CTS increase the likelihood of sleeping on the side, increasing the probability of wrist flexion or extension, which increases the pressure in the carpal tunnel during sleep, resulting in CTS. Any medical problem that makes the nerve more susceptible to pressure or increases the volume of the contents of the carpal tunnel will similarly increase the risk of compression or increase the probability that a given amount of compression will cause symptoms. It seems possible that this population of patients is prone to sleep disturbance and the presence of CTS may be one of many that bring the patient to the attention of the physician. Careful evaluation of sleep disturbance may be important in the management of CTS in the future.

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**Robert C. Russell, MD**  
ASPN President - 2008 - 2009

### Moscow Microsurgery

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I helped organize the first microsurgical symposium in the former Soviet Union sponsored by Sharppoint Inc in 1983, which took place at the Petorski Institute in Moscow. Jim Steichen, my hand and microsurgery mentor and I spent three days lecturing, rounding, and exchanging knowledge with the few Soviet surgeons doing microsurgery at the time. The iron curtain was still down and there was very little, if any, information exchanged between East and West. There was a lot of uncertainty and suspicion on both sides.

The head of Soviet microsurgery was Professor Krylov, who asked me if I wanted to do a case with his residents while I was there. I was a young attending surgeon at SIU and was certain I was the best microsurgeon the world had ever seen, so of course, wanting to make my country proud, I said yes before finding out what kind of case they were doing. I asked if I should be there at 7 a.m. but was told, "No, around 10 a.m. would be fine". When I arrived at 9:30, Professor Krylov showed me into his office next to the surgical suite and asked if I wanted coffee and a cookie. It was then that I asked about the case. "Oh", he said, "It's an eight-year-old boy with an iatrogenic brachial plexus injury following removal of a supraclavicular mass at another hospital somewhere else in the Soviet Union". Now, at home, Allan VanBeek and I had done perhaps 5-6 plexus cases but I had very little experience and was definitely not an expert. So here I am in Moscow, about to enter an operating room, with residents I had only recently met, to perform surgery on a pediatric patient with a challenging nerve injury, in a scarred bed, without doing any pre-operative evaluation or case preparation before surgery. No books, no English, no knowledge about the case, definitely now the dumbest microsurgeon in the world.

I entered the operating room with a rapid heart rate, surrounded by personnel who didn't speak English; about to explore a major nerve injury on a child I had never met. As I walked to the table with all sorts of ideas for exposure, dissection techniques, and repair solutions running through my mind, the residents informed me, through an interpreter, that they had actually started the case at 7:30 a.m. and had already dissected out the divided upper trunk of the brachial plexus, which now had both freshly cut ends sitting out on a piece of background ready for the "professor" to sew the ends together under the microscope. So my heart rate immediately returned to normal but I learned a number of valuable lessons, like asking how cold and deep the water is before jumping in the lake, but most importantly, I got a big



Ivica Ducic, MD, PhD  
ASPN President - 2011 - 2012

### Nerve Surgery: Privilege, Entitlement or Ignorance?

**Patient I.** It is late afternoon on a busy clinic day. I am entering the exam room to see the last patient of the day. A young woman, in her late 20's stood up to shake my hand. The first glance of this polite woman who nicely introduced herself revealed tired eyes and sad face. Her smile was brief, eye contact short, and handshake weak. Although I still did not know about her problems, I could sense she was troubled with something.

As we live in the era of electronic medical records, I had to spend first few minutes to navigate through the patient's presenting complaints and past history prior to starting my own evaluation. Patient stated: "My problems shortly followed an uneventful C-section three years ago. I developed groin pain that never went away, despite the fact that I was told by my doctors that it would go away by three months, then three more months, then by Christmas. It was only becoming more present, ever so negatively affecting quality of my life in every possible aspect. I had to stop any physically demanding activities, was not able to play with my baby, and was not able to have intercourse due to pain. My husband was initially very supportive, lately became quite frustrated, especially as some of the many doctors I saw said to me in his presence "It is in your head, get rid of it"! As my mother had similar groin pain related problems after hysterectomy, I thought, "It is in the family; something is really wrong with me..."

"In the meantime," patient continued, "I had countless doctors visits and interventions, including several ultrasounds, CT scans, blood works and few diagnostic laparoscopies, all revealed nothing abnormal with me." I had to interrupt her to clarify what "few laparoscopies" meant. She said she had five or six laparoscopies and at least that many CT's! She continued by saying: "Pain management physician was prescribing me pain medications, sometimes they would take an edge away, but pain would routinely always be there, and would be back. Local injections to groin area and spinal injections would have vague, mixed, if any, results. I had to go to different pharmacies to pick up my pain medications as I started to feel embarrassed picking them up at my local store since pharmacy staff would recognize me and ask, so Ms. X; is it Percocet or Dilaudid this time? Due to aforementioned, I became depressed; my family life is at the edge of divorce, I feel I have no life, please help me! Few years ago you have operated on my neighbor's relative who had groin pain following inguinal hernia repair with mesh, and have cured him, I will pray you can do something similar for me," she finished.

By this time desperation in patient's teary eyes was obvious, as was my frustration with the fact that nobody in three years gave patient any valid diagnosis or explanation why she had all these problems. Her physical exam indicated peripheral nerves being involved in her agony, while 65 min peripheral nerve surgery done four weeks later eliminated her three year long suffering.

*It was concerning that the patient had 5-6 CT scans exposing her to repeated radiation, and equally troubling was to hear that she underwent 5-6 diagnostic laparoscopies, all without adding anything to her problem solution. Although her doctors have to be complimented for not abandoning her, at the same time her management should have been redirected early, preventing such extensive diagnostic ordeal. Once chronic post-op pain continued beyond expected post-op period for a given procedure, and once baseline specialty evaluation by the providers involved in direct patient's care revealed no credible explanation to the patient's chronic pain, parallel referral to the pain specialist and to a peripheral nerve surgeon should have been initiated. The first one was to provide appropriate supportive care, while latter one to help identifying exact anatomical reason for patient chronic pain, if of a nerve origin.*

**Patient II.** Young man presented in the clinic with his parents due to 11-month-old foot drop following knee dislocation. Patient confirmed that the foot drop was immediately present after injury and persisted ever since, despite corrective orthopedic intervention for torn meniscus. Since the injury, the patient was under the care of an orthopedic surgeon who provided referral to physical therapy. Despite physical therapy and supportive AFO splint, foot drop persisted.

I asked the patient if he discussed persistent foot drop concerns with his orthopedic surgeon, he said yes, and was told: "The nerve will take about an inch per month to grow, so be patient." The same was reiterated to me upon subsequent three-month office visit intervals." When I asked the patient what made him seek my opinion at this time, he replied that he thought something was wrong and was afraid his nerve was not going to come back. Unfortunately, the patient was right as very precious time for nerve decompression intervention was lost. The end result was subsequent requirement for much more invasive and less inferior procedure (Tibialis Posterior tendon transfer) short of permanent foot drop. The extent of professional limitations and personal frustrations this young patient will have to endure for the rest of his life, due to this "Let's wait and see treatment," is simply overwhelming...

*"Let's wait and see" approach is rather very frustrating subject for any peripheral nerve surgeon. Those not following the 'expected' nerve regeneration time frame, with this approach unfortunately literally have their destiny sealed with permanent functional deficits, for life! Certainly it is a judgment call if someone needs nerve exploration right away, or decompression at six or 12 weeks following nerve deficit, but the evidence-based data strongly point to an early over late/delayed nerve intervention if aiming*

## Featured This Fall - ASPN Past Presidents: Pearls and Insights

towards positive outcome. These data range from Cajal, who was awarded a Nobel Prize for explaining nerve degeneration events upon nerve injury, to few decades old explanations related to the role of neuromuscular junction and muscle atrophy, and to some newer data focusing on Schwann cell role in nerve regeneration. Considering the existence of strong evidence based data, this is why it is devastating for someone with an acute nerve deficit (like foot drop in this patient) to be allowed to continue with his/her functional deficit, simply due to dogmatic belief that the nerve will (for sure?!) regenerate an inch per month following injury. Once this "Let's wait and see treatment" produces no response it can be very difficult, if at all, to reverse nerve injury a year later.

Separate but equally difficult issue is that this scenario puts an additional pressure on the evaluating peripheral nerve surgeon. Despite the fact that the surgeon should remain professional and only serve as doctor trying to help, he/she needs to be very careful when choosing words for discussion with the patient as this situation can easily trigger malpractice claim.

**Patient III.** A phone call in the OR from a very reputable colleague from different town with a request to see his family member gave me a sense of urgency so I managed to open the appointment for her next day. Both parents accompanied this colleague student who was visibly anxious at the time of initial evaluation. Some eight months ago she had fallen on a glass and injured her hand. She had pain and numbness in the hand. The relatively small puncture wound distal to the wrist crease was closed in the ER. However the pain and numbness continued for two more months. A neurologist, who happened to be the father's friend ordered NCS, confirming loss of sensation distal to the injury. Subsequently, the same neurologist ordered an MRI (now already some four months since the injury). It identified no foreign body, and confirmed some nonspecific but expected scarring in deep SQ tissues within the zone of injury. Her treating doctor made the decision to re-evaluate her with another MRI in three months, when she is back home from college, if the problems continue. As she came home, now some eight months after the injury, complete wasting of the dominant hand ulnar nerve innervated intrinsic muscles was obvious, making the patient's parents seek opinion with me at this time.

As I first evaluated her, it was obvious that she initially suffered the nerve damage and that unfortunately the required treatment for that injury did not take place. The first available OR time for this elective surgery was some three weeks later, making it nearly nine months since the injury. Intra-operatively, complete transection of the motor branch of ulnar nerve was confirmed, requiring reconstructive intervention in form of autograft from the posterior interosseous nerve. Unfortunately, despite reconstructive nerve surgery, patient's muscle wasting and weakness were not reversed; further exposing patient to functional challenges that required suboptimal tendon transfer surgery.

*An acute functional nerve deficit (either motor or sensory) following sharp penetrating injury should be considered the result of a nerve injury until proven otherwise. Wound exploration with attention to potentially injured nerve remains priority in searching for causative answer. If this nerve evaluation does not happen while patient is being evaluated by physicians in the ER, then patient should be referred to a peripheral nerve surgeon who can make decision if and when intervention is needed. The role of MRI is minimal, if any in these scenarios as the clinical exam alone can tell more than NCS and MRI combined. Unfortunately, these considerations were all ignored in this case, serving as another example of excessively unjustified long observation resulting with intrinsic muscle wasting, with permanent consequences.*

**Patient IV.** A nice 67-year-old lady presented in the office with long standing peripheral neuropathy problems. As she was introducing herself I noted bilateral muscle wasting in the first web space. She stated that she was diabetic for the past 5 years but had noted paresthesia of her fingers few years earlier. Eight months earlier she had NCS that showed: "generalized peripheral neuropathy and ulnar nerve entrapment at the elbow." Two months later, a local surgeon performed ulnar nerve decompression at the elbow that left the patient with excruciating pain and minimal improvements. She was even more worried since she started to develop similar symptoms in the other arm. When asked if she had discussed her concerns with her surgeon, she said she did but got no real answer and did not want to see him again since he had told her that he "usually perform the nerve surgery only few times a month or less". Patient continued: "when I asked him why he did not send me to someone who does the nerve surgery every day, he replied: "Why should I send you to someone else, I am an orthopedic surgeon, we do this nerve surgery anyway." Patient finished by saying how she later learned through some friends that he mostly did joint replacements for the past 15 years, and had a rather nice reputation as a joint surgeon in town.

Exam confirmed advanced neuropathy with intrinsic wasting, some 18-20 cm long inner elbow scar, numbness in the distribution of MABCN, and tenderness over elbow scar. Intervention required removal of MABCN neuroma, re-do UN-decompression (both deep FCU fascia distally and intermuscular septum with internal brachial fascia proximally not released at all during first surgery), and appropriate UN related surgery at the wrist. Considering her intrinsic atrophy was present for years, no baby sitter jump graft was considered. Subsequently, UN at the elbow and wrist on opposite extremity were later addressed accordingly.

*It is important to acknowledge that diabetics have three times higher incidence of peripheral neuropathy when compared to general population and that about half of all diabetic develop neuropathy (5x general population). Failure to recognize these facts can easily contribute to wasting an important window of opportunity for intervention once compression neuropathy is identified.. Equally important, altered glucose metabolism due to*

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*pathophysiological changes in diabetics, including glycosilation of collagen, negatively affect both nerve itself and the surrounding structures around the nerve (tunnels). This contributes to the vulnerability of the nerve due to increased pressure at anatomically known nerve compression sites, which could be responsible for paresthesia in early phases of diabetes.*

*The most common dogmatic mistake made by those treating patients for neuropathy-prone chronic conditions, primarily diabetes, is to assign patients paresthesia (numbness, burning, pins & needles sensation in hand/ fingers and/or foot/toes) simply to "diabetes," excluding relevant anatomical compression as being potentially responsible for those symptoms. Once these symptoms are present despite optimal medical treatment, medical provider should have very low threshold to initiate proper work up for superimposed compression neuropathy in diabetic patients. Again, when confirmed, patient would most benefit from subsequent evaluation by a peripheral nerve surgeon.*

**Patient V.** A middle-aged woman presented with chronic headaches, migraines and occipital neuralgia for the past 16 years; ever since her car accident with whiplash injury. Upon entering the exam room, she had sunglasses on, room lights were turned off, spoke very softly and was hardly turning her head/neck towards me. Review of her previous treatments, hospitalizations, ER visits and list of medications consumed about 20 minutes. Careful examination and nerve blocks identified specific peripheral nerves in the occipital region as the major cause of her symptoms. Carefully planned operative treatment successfully addressed those problems that hunted this lady for the past 16 years.

*As I was totally pleased with her very nice response to surgery, at three months post-operative visit, she appeared visibly angry. As I was still trying to decode reasons for her anger, not even few minutes into the follow-up evaluation, she asked me how many of these surgeries have I done and for how long have I been doing this "headache surgery." I replied nearly 10 years and some nearly 2000 surgeries. She literally screamed: "Why was I allowed by my doctors to suffer for so long, why nobody referred me to you earlier ... all these years in pain, why?"*

### Author's Commentary

Examples like these are not numbers, stories or cases; these are real people, possibly any one of us in the future. The true numbers of human sufferings as presented in these five scenarios are not known, although one should not overlook that they are still very much present around us. We often quote to our patients high percentages of good outcomes, while unfavorable negative percentages cited are usually mostly incidental and of much smaller values. The problem is that if negative outcome is present in even 1-2% range that equals 100% failure rate for those 1-2 patients! Considering that "1-2 %" can be any one of us, these relatively small negative numbers become much more significant than initially appeared to some. At the same time, the extent to

which patients' lives are negatively affected, speaking both in personal and professional norms are not well known. Even the socioeconomic burden to the society as a result of the negative impact of these events on the patients' quality of lives is grossly under reported and underestimated.

This is why examples of some bad practices and dogmatic beliefs as outlined here need to be eliminated when dealing with patients suffering from chronic pain, nerve injuries, headaches, or any other neuropathy.

Medical colleagues and all surgical specialists dealing with nerve related problems following any surgery or trauma would benefit from acknowledging evidence-based available algorithms related to nerve problems. Patients with nerve problems should be referred to a peripheral nerve surgeon in a timely fashion so patients can get an appropriate care for their peripheral nerve problem. Ignorance of available protocols and late referrals or no referral of patients with neuropathy, chronic pain or nerve injuries unfortunately, only further contribute to patient's aggravation, prescription of wrong subsequent treatments and development of permanent functional or psychological deficits and pain syndromes. On the other hand, considering that every doctor holds a license to practice "Medicine and Surgery" in any given state, this license is translated as an entitlement to any surgeon to do peripheral nerve surgery regardless of whether they have proper training, or true nerve surgery expertise, or if they perform nerve surgery only occasionally. This is even a bigger problem as there are no structured nerve surgery fellowships, thus many specialists end up doing nerve surgery based on entitlements and limited scattered exposure during their residencies. This, unfortunately, adds to surgery failure and contributes to the development of unnecessary complications, all negatively adding to patient's poor quality of life and increase in the socio-economic burden to the society.

Considering aforementioned, peripheral nerve surgeons, not only primarily having a privilege of helping patients with peripheral nerve problems, but also they have long way of educating non-nerve surgeons and medical providers about consequences of ignoring the basic principles of peripheral nerve surgery. At the same time ASPN, as the official assembly should continue to encourage and educate all involved in management of patients with neuropathies, nerve injuries, headaches and chronic pain and the critical role of timely referral and application of proper peripheral nerve surgery principles and techniques in managing patient with nerve problems. Patients, themselves, should continue to wisely choose their surgeons, based on his/her true expertise in nerve surgery. At the end, it is about team approach and multidisciplinary treatment that will be of the most benefit to any patient with nerve problems.

## 2014 ASPN Program Chair Report



As program chair for the 2014 ASPN annual meeting, I am would like to invite you and your families to the ASPN annual meeting in Kauai, Hawaii, January 10-12, 2014. We have planned an extraordinary program to combine education, networking and social activities at the Grand Hyatt Kauai Resort & Spa.

To allow more time to enjoy the beautiful island of Kauai, we will begin early each morning and adjourn early in the afternoon. Friday morning will include an integrated program with the AAHS; instructional courses, combined paper session and a panel on “Radial Nerve Transfer vs. Tendon Transfer: What’s Best When, What’s The Evidence.” We will also have an ASPN scientific paper session, an interactive poster session and the invited speaker, Dr. Jaishri Blakeley. Saturday morning will include AAHS/ASPN/ASRM instructional courses, outstanding paper session and invited speaker, Dr. Peter Galpin. On Sunday morning, the early morning instructional courses will be followed by an ASPN/ASRM panel on “Enhancing Outcomes in Facial Reanimation Utilizing a Multi-Modality Approach; Present and Future.” The morning will also include ASPN scientific paper sessions, Presidential Address, a panel of Past Presidents and conclude with the ASPN business meeting.

The program will be an exciting, educational and fun learning experience and we will have activities at the resort for you to enjoy with your family and friends. The weather and venue will be fantastic, and I look forward to seeing you in Kauai! Aloha!

Christine B. Novak, PT, PhD  
2014 ASPN Program Chair



*We Look Forward to Seeing You in Kauai, Hawaii!*



# 2014

## AAHS ASPN ASRM ANNUAL MEETINGS

American Association for Hand Surgery

*January 8-11, 2014*

American Society for Peripheral Nerve

*January 10-12, 2014*

American Society for Reconstructive Microsurgery

*January 11-14, 2014*

Grand Hyatt Kauai, Kauai, Hawaii

JOINTLY  
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Thursday, January 9, 2014

2:00pm – 4:00pm

ASPEN 2013 Council Meeting (not for credit)

Friday, January 10, 2014

6:00am – 8:00am

Breakfast with Exhibitors

6:30am – 7:45am

AAHS/ASPEN Combined Instructional Courses

### 117 Offering The Wide Awake Alternative To Your Patients; How and Why To Do It

Chair: Peter C. Amadio, MD

Instructors: Elisabet Hagert, MD, PhD; PC Ho, MD; Amanda Higgins, BScOT, OT; Donald H. Lalonde, MD

This course will review the indications and technique of “wide awake” surgery in the hand from the perspective of hand surgeons and hand therapists.

### 118 Simplifying Kienböck’s Disease: Diagnosis and Treatment

Chair: Greg I. Bain, PhD, MBBS, FRACS, FAA

Instructors: Randip R. Bindra, MD, FRCS; Steven L. Moran, MD; David J. Bozontka, MD

This Instructional Course will cover the assessment of the patient with Kienböck’s disease including advanced imaging and wrist arthroscopy. The latest treatment options of the management of Kienböck’s Disease, including the role of Osteotomy, Vascularised bone graft and Limited wrist fusions will be discussed.

### 119 Vascular Disorders of the Upper Extremity: Diagnosis and Management

Chair: Michael S. Murphy, MD

Instructors: James P. Higgins, MD; Michael A. McClinton, MD; Andrew Tyser, MD; Brian T. Carlsen, MD; Joy MacDermid BScPT, PhD

This course will cover complex vascular problems of the upper extremity, present diagnostic, and treatment dilemmas. Few hand surgeons outside of major referral centers garner extensive experience in their management. As a result many hand surgeons are not as familiar or facile with their treatment alternatives. This course will also review the pertinent vascular anatomy, with an emphasis on critical variations. Appropriate use of diagnostic testing including office based alternatives, capabilities of the vascular lab and radiographic studies will be presented. Finally, common vascular problems and their treatment options, both surgical and nonsurgical, will be reviewed utilizing a case based format.

### 120 The Scaphoid- Treatment of the Entire Spectrum: From Fresh Fractures, to Stable Scaphoid Nonunions, to Unstable Scaphoid Nonunions with AVN and Humpbacked Collapse

Chair: Allen T. Bishop, MD

Instructors: William B. Geissler, MD; Eric P. Hofmeister, MD; Michael J. Morhart, BSc, MSc, MD; Paul Brach, MS, PT, CHT

The entire spectrum of scaphoid pathology will be presented from the acute fracture stage, to stable nonunions, progressing to unstable scaphoid nonunions with AVN and carpal instability (humpbacked collapse). Each of the faculty members will be presented a typical set of images from each of these various stages of scaphoid fracture pathology, and then will walk the audience through their preferred method of treatment for each of those particular stages. A combination of various treatment techniques will be presented ranging from percutaneous fixation to arthroscopic assisted internal fixation, arthroscopic guided bone grafting, and ORIF with a variety of vascularized bone grafts. A treatment continuum will be stressed, emphasizing that the selected treatment plan must be highly individualized to the patient’s specific fracture / nonunion with respect to elapsed time from injury, location – waist vs. proximal pole, stability (displacement / angulation), presence of cystic resorption and proximal pole perfusion/ viability.

### 121 Update on Congenital Hand Differences

Chair: Scott H. Kozin, MD

Instructors: Hilton P. Gottschalk, MD; Joshua M. Abzug, MD; David T. Nestcher, MD; Wendy Tomhave, OT

Congenital hand differences are challenging problems to the surgeon. A variety of reconstruction options are available with consideration of both form and function. This course will be case-based discussions of a myriad of congenital hand problems with an emphasis on surgical reconstruction.

### 122 Relative Motion Splints: A Simple Solution to Many Hand Problems

Chair: Wyndell H. Merritt, MD

Instructors: Michael W. Neumeister, MD; Julianne Howell, PT, CHT; Gwendolyn van Strien, MSc, PT; Melissa Hirth, OT

This course will address the rationale for use of relative motion splinting that permits immediate active motion following various hand injuries and repairs. Its well-established use for long extensor repair will be discussed and reviewed, and the panelists’ experience using this concept in less well-established conditions will be presented using both flexor and extensor relative motion splinting for conditions such as acute and chronic boutonniere, flexor tendon and nerve repair, acute and chronic sagittal band rupture, caput ulnar syndrome and digital joint stiffness. Participants will be encouraged to provide their input, insights and experience on the use of relative motion splinting in their own practice.

### 123 Making Carpal Tunnel Simpler: Things To Do and Not To Do

Chair: Warren C. Hammert, MD

Instructors: Loree K. Kallianen, MD; Scott F. Duncan, MD, MPH; Allen van Beek, MD; Kristin A. Valdes, OTD, OTR, CHT

This course will discuss methods to simplify treatment of carpal tunnel syndrome, focusing on optimizing outcomes. The role of electrodiagnostic studies, anesthetic options, and management of patients who return to the office with symptoms of CTS will be reviewed.

### 124 Nerve Transfers to Improve Hand Function in Tetraplegia

Chair: Ida K Fox, MD

Instructors: Steve K. Lee, MD; Allan J. Belzberg, MD

Cervical spinal cord injury resulting in tetraplegia is a devastating injury with substantial limitations to independent hand and upper extremity function. Nerve transfers provide the opportunity to utilize otherwise untapped resources to restore hand function and independence. This course will present the strategies for nerve transfer in tetraplegia to improve hand function with specific focus on patient selection, surgical technique and outcomes.

8:00am – 9:15am

AAHS/ASPEN Concurrent Abstract Scientific Session

9:15am – 9:45am

Coffee Break with Exhibitors

10:00am – 10:10am

ASPEN President and Program Chair Welcome (not for credit)



Allan J. Belzberg, MD  
ASPEN President



Christine B. Novak, PT, PhD  
ASPEN Program Chair

10:10am – 10:45am

Scientific Paper Session 1

10:45am – 11:30am

President’s Invited Lecture

Jaishri Blakeley, MD



11:45am – 12:45pm

Joint Concurrent AAHS/ASPEN Panel: Radial Nerve Transfer vs. Tendon Transfer: What’s Best When, What’s The Evidence

Moderator: Robert Spinner, MD

Panelists: Thomas Tung, MD; Allen T. Bishop, MD; David C. Ring, MD; Milan Stevanovic, MD; Christine B. Novak, PT, PhD

Radial nerve paralysis is a relatively common condition that offers surgeons different options for operative intervention, often with favorable outcomes. This course will review time honored procedures such as nerve grafting and tendon transfers as well as newer ones involving nerve transfers. Surgical techniques, outcomes, and rehabilitation for each will be reviewed.

12:45pm – 2:00pm

ASPEN Poster Session (not for credit)

Moderators: William Kuzon, MD; Steven McCabe MD

## Saturday, January 11, 2014

**6:30am – 8:00am**      **Breakfast with Exhibitors**

**6:30am – 12:00pm**    **AAHS/ASPN/ASRM Combined Day**

**12:00pm – 2:00pm**    **Scientific Paper Session 3  
Lunch (not for credit)**

**6:00pm – 7:30pm**    **ASPN/ASRM Welcome Reception**

Cost: 1 ticket included in ASPN and ASRM registration.

Additional adult tickets available @ \$60 each; tickets for children and young adults ages 5 - 17 available @ \$30 each and include 2 drink tickets.

Gather at Shipwrecks Lagoon Beach, to catch up with fellow meeting attendees while being serenaded with Island sounds.

## Sunday, January 12, 2014

**6:30am – 8:00am**      **Breakfast with Exhibitors**

**6:45am – 7:45am**      **Instructional Courses (4)**

### **301 Obstetrical Brachial Plexus Palsy**

Chair: Howard M. Clarke, MD

Faculty: Martijn Malessy, MD, Linda Yang, MD

This course will review the advances in surgical management of obstetrical brachial plexus palsy. Through didactic presentations and interactive case presentations, assessment, surgical management and outcomes will be presented.

### **302 End to Side Nerve Repair**

Chair: Gregory Borschel, MD

Faculty: Fausto Viterbo, MD; Ayato Hayashi, MD

This course will present the scientific basis for the clinical use of end to side nerve repairs. The faculty will present clinical cases in which this technique is used, and discuss the risks, benefits, potential complications and outcomes following the use of the technique.

### **303 Repairing the Nerve Gap: Autograft, Allograft, Neural Tube - Indications & Contraindications**

Chair: Rajiv Midha, MD

Faculty: Paul Cederna, MD; Loree Kallianen, MD; Amy Moore, MD

An interactive format, including case based discussion, will be used to present and debate the appropriate use of nerve autografts, nerve allografts and nerve tubes (conduits) for the repair of nerve injuries.

### **304 Coding Peripheral Nerve Surgery: How to Maximize and Stay Legal**

Chair: Aaron Filler, MD; Christopher J. Winfree, MD

Faculty: TBA

This course will use case based discussion and an interactive format to review coding and billing issues for peripheral nerve surgery.

### **8:00am – 8:45am**      **Joint ASPN/ASRM Panel: Enhancing Outcomes in Facial Reanimation Utilizing a Multi-Modality Approach; Present and Future**

Chair: Michael Klebuc, MD

Panelists: Tessa Hadlock, MD; Elliott Rose, MD; Travis Tollefson, MD; Michael Zenn, MD

This panel will explore an array of surgical and non-surgical techniques that can be employed to improve outcomes with rehabilitation of the paralyzed face. The potential benefit of surgical intervention in early childhood with regard to speech, socialization and spontaneity will be examined. The value and necessity of standardized outcome reporting measures will be addressed. Additionally, postoperative physical therapy techniques and their ability to enhance surgical results will be explored. The potential value of the masseter nerve as a babysitter will be considered and the emerging role of bionics and artificial muscle in the future of facial paralysis will be examined.

**8:45am – 9:15am**      **ASPN/ASRM Scientific Paper Session**

**9:15am – 9:45am**      **Break with Exhibitors**

**9:45am – 10:45am**    **Scientific Paper Session 4**

**10:45am – 11:30am**   **Scientific Paper Session 5**

**11:30am – 12:00pm**   **ASPN Presidential Address**  
Allan J. Belzberg, MD

**12:00pm – 12:45pm**   **Past Presidents Invited Panel**

**12:45pm – 1:30pm**    **Past Presidents Invited Panel**

**1:30pm – 1:45pm**    **Closing Remarks & Awards**

**1:45pm – 2:30pm**    **ASPN Business Meeting & Lunch (ASPN Members Only)**

**2:45pm – 3:45pm**    **ASPN 2014 Council Meeting**

## 2015 Annual Meeting

Dear ASPN Members,

Mark your calendars; the 2015 will be approaching soon. We will be meeting January 21-27, 2015, at the Atlantis Resort in Paradise Island, Bahamas. We are planning a robust scientific program to build on the history of our interactive format of previous ASPN meetings. Continue to visit the ASPN website at [www.peripheralnerve.org](http://www.peripheralnerve.org) for details of abstract submission and meeting registration.

We look forward to seeing you in the Bahamas!

