CANADIAN IONM NEWS
Official Newsletter of CANM

Message from the PRESIDENT

Hello Colleagues,

This represents my last message as CANM President as we welcome Dr. Jamie Johnson (Foothills Medical Centre, Calgary) as our new President in September. I have been a CANM member since 2011 and have seen this small group bloom into an internationally recognized organization with education being the focal point of our message. The CANM/Michener course is something we can be proud of but it only represents the start of CANM's vision. By emphasizing education and training we have advocated for IONM interpretation by in situ neuromonitorists, with or without advanced degrees, to augment the intraoperative triad of professionals managing the surgical patient (Surgery, Anesthesiology, Neuromonitoring). The slightly radical idea of "The Expert in the Room" collaborating with the surgical team has met with opposition as many new ideas and concepts do. Yet our notions of training and education have similarities that are comparable to the development and acceptance of the Physician Assistant role. Nevertheless Albert Einstein did wisely say: Great spirits have always found violent opposition from mediocre minds. CANM has not been immune to this idiom unfortunately.

Planning out lofty future directions for CANM is an easy task. The difficulty is in the implementation. This organization is at a time when our goals need to be re-evaluated. Do we have enough members to engage new programs, such as the Certification Exam? How do we attract those IONM people who prefer to give their precious membership dollars to larger organizations that seemingly offer more for their money? How do we re-ignite interest from neuromonitorists and former CANM members in Vancouver, Toronto or Montreal? These are among the challenges that lay ahead for the next Executive Committee to prioritize and resolve.

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There are advantages and disadvantages to being part of a smaller organization like CANM. In a small organization your voice is typically heard but everyone (or almost everyone) needs to pitch in. What we should not lose sight of is that through CANM we are attempting to be the stewards of our profession in Canada; to recognize the needs of the members and direct the practice of IONM to improve the quality of our work to benefit our patients.

CANM continues to grow in knowledge and experience if not so much in membership numbers. If you perform neuromonitoring in Canada you should be part of CANM. You can help direct your profession, have your voice heard and your questions answered. Be a player not a spectator!

Thank you to all those CANM members for 2019. We appreciate your support.

Yours,

Marshall Wilkinson BSc (Hon), MSc, PhD
President, CANM & Neurophysiologist
Section of Neurosurgery
Health Sciences Centre
Winnipeg, MB

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YEAR 1 September 2019 to August 2020

1	Clinical Sciences for IONM	SEP - DEC
2	Basic Principles of IONM	JAN - APR
3	IONM Modalities I	APR - JUL

YEAR 2 September 2020 to August 2021

4	IONM Modalities II	SEP - DEC
5	Considerations for IONM	JAN - APR
6	Advanced Topics in IONM	MAY - AUG

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Dear Colleagues,

Please do not forget to mark your calendar for the Annual CANM Symposium September 20 and 21 in Winnipeg, Manitoba. Registration is now open and we anticipate another successful two day meeting. The key note speaker this year will be Dr Stan Skinner as well as a roster of expert talent in IONM, anesthesiology, neurosurgery and neurology.

In addition to our line-up of speakers we will also set time for the always popular case presentations. These brief reports can present unusual cases, perplexing results or interesting observations. If you have some interesting cases please consider sharing them with us. We can all learn from rare or unusual events.

For the explorers amongst us consider visiting The Exchange District which is about 4 blocks from the conference hotel. The Exchange highlights Heritage-protected turn of the century architecture which houses many excellent restaurants and dispensaries of libation. I also mentioned in a previous communication The Forks Historic site and the Museum for Human Rights as other touring options in close proximity to the conference hotel.

The Exchange District.

Image courtesy of Tourism Winnipeg.

For those that are planning to attend we look forward to seeing you. For those that remain unsure it's not too late to sign up!

See you in September everybody!

Marshall Wilkinson BSc (Hon.), MSc, PhD
President CANM

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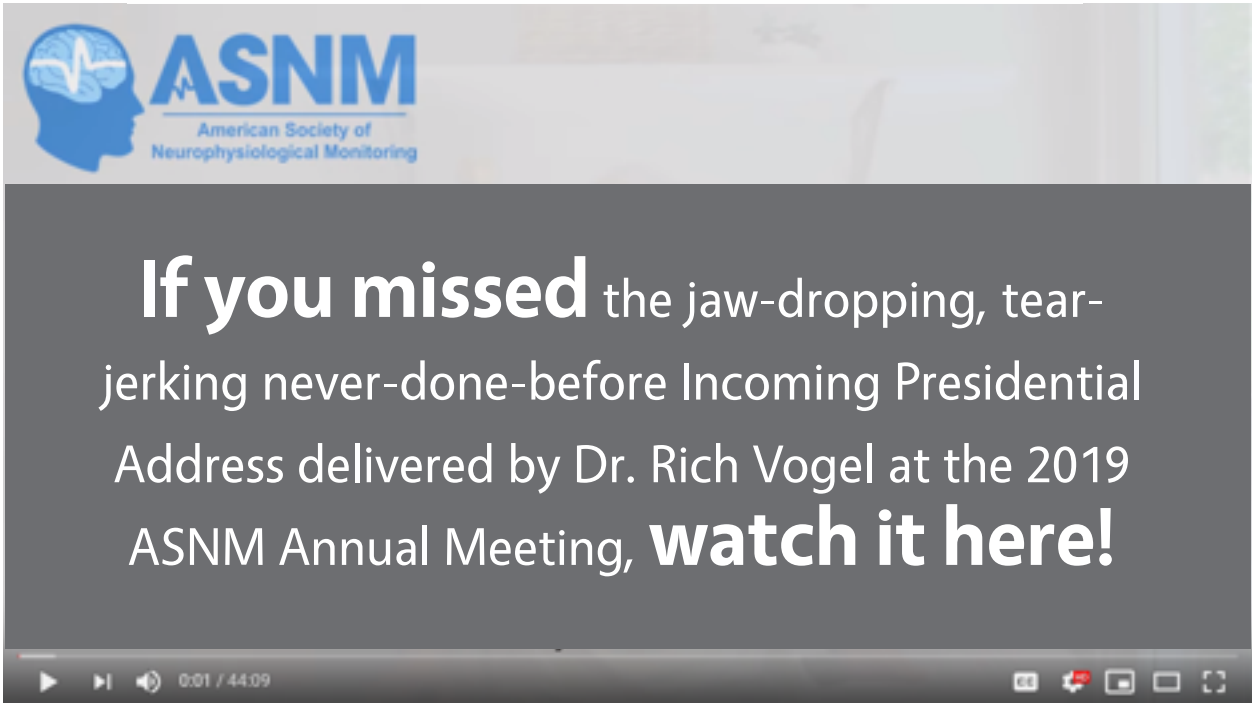

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This section is devoted to celebrating the accomplishments of members of our Canadian IONM community and recognizing them for their contributions and achievements no matter how big or small. Please join us in congratulating the following CANM Superstars.

CANM Superstar

Sam Strantzas

Toronto, ON

Along with two anesthesia colleagues, Sam published a seminal document on Intraoperative Neuromonitoring in Pediatric Spinal Surgery in BJA Education. A link to this publication can be found on the CANM website homepage.

David Houlden, Chantal Turgeon, & Nathaniel Amyot

Ottawa, ON

The IONM team from Ottawa recently published an article on flash VEP in Canadian Journal of Neurological Science. A link to this publication can be found on the CANM website homepage.

Laura Holmes & Samuel Strantzas

Toronto, ON

The recently published article Responding to Intraoperative Neuromonitoring Changes During Pediatric Coronal Spinal Deformity Surgery in Global Spine Journal is the work of Laura, Sam, and the spinal surgeons at SickKids.

https://journals.sagepub.com/doi/full/10.1177/2192568219836993

Are YOU a CANM Superstar?

Do you KNOW a CANM Superstar?

CANM Superstars are members of the Canadian IONM community who we would like to recognize for their contributions, but we need your help! Please send us the accomplishments that should be celebrated in the next issue of Canadian IONM News by submission to **info@canm.ca**

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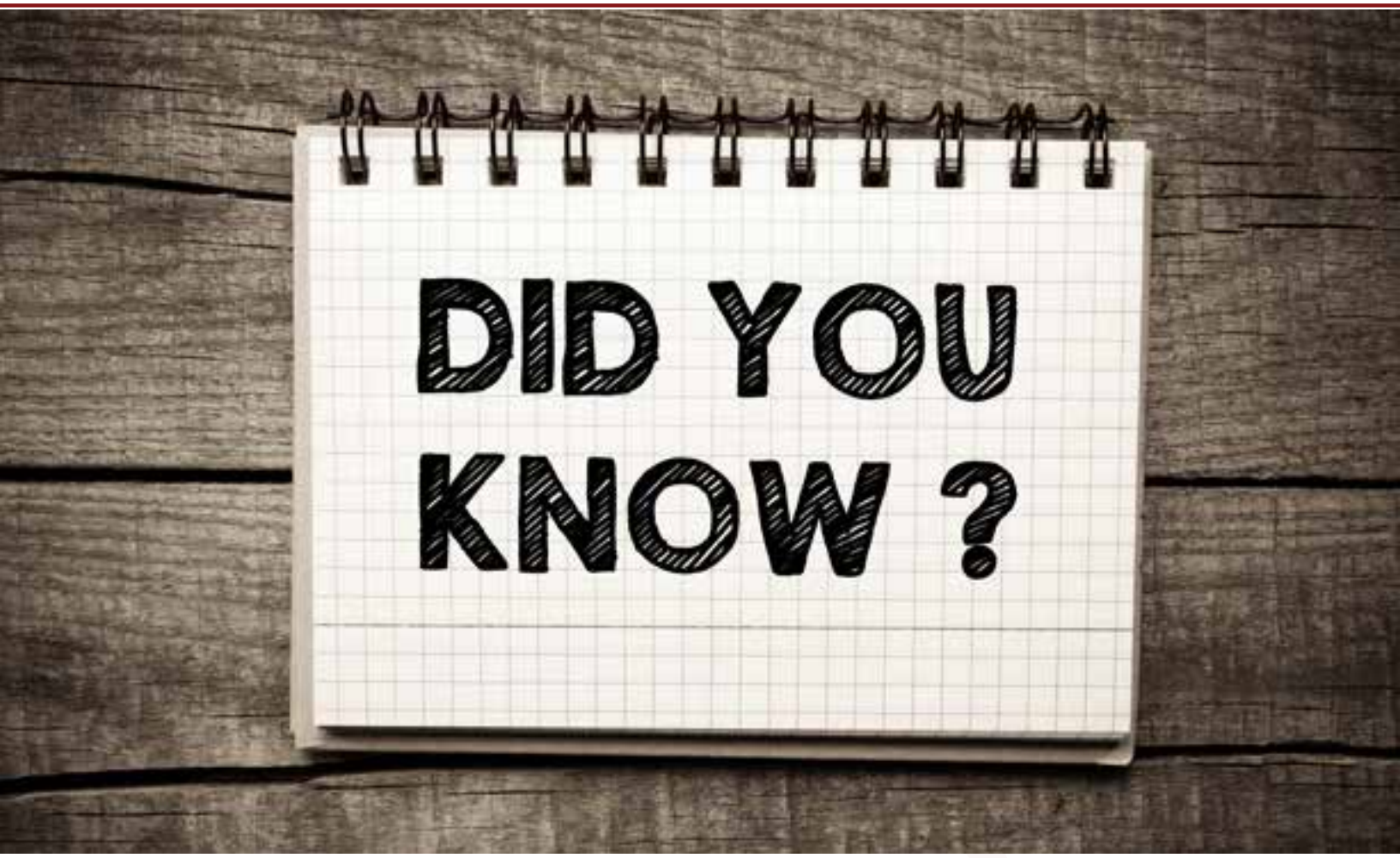

Free

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The Scoliosis Research Society (SRS) offers webinars that are free to non-members. Anyone interested can register for the live sessions or view previously recorded sessions. Participants in the live sessions are eligible for AMA PRA Category 1 credits (ie. CME credits).

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Guest Article from ASNM President

As the new President of the American Society of Neurophysiological Monitoring (ASNM), it is my honor to accept this invitation to write an article for the Canadian IONM News. I'd like to take this opportunity to share my views of the IONM profession (in the USA), and tell you about my vision for the future of the ASNM. My goal is to help you find the same hope and optimism that I have in these particularly challenging times.

Before I go on, I should tell you that portions of this article were adapted from my incoming Presidential Address that I delivered at the ASNM Annual Meeting in May of 2019. **The Presidential Address link to:** https://www.youtube.com/watch?feature=youtu.be&v=ZmS2I3jAM0I&app=desktop is available on YouTube. It's 45 mins long, but viewers with the attention span to watch the entire speech will find it quite rewarding.

I thought I might begin by telling you a little about myself, perhaps to provide some context for my comments and help you to see the human side of me, assuming we don't already know each other. I am a PhD neurophysiologist from Philadelphia, Pennsylvania. I've been working in the field of IONM for just over 10 years, and I'm a certified Diplomate of the American Board of Neurophysiologic Monitoring (D.ABNM). My favorite cases involve functional mapping of the brain and spinal cord. If there's a such thing as a claim to fame in this profession, I suppose mine came in the form of a blog (neurologiclabs.com) that I wrote until late 2017 when I finally closed it down. If you were one of my nearly two thousand monthly readers, I'd like to say thank you!
Over the course of my career, I've worked in both private practice and large corporate contexts. Such is the life of IONM in the USA. In addition to being a neurophysiologist, I've managed IONM teams, trained many budding neurophysiologists, developed education programs, worked on quality assurance, lectured widely, and otherwise devoted much of my spare time to the voluntary and often lonely endeavor of advancing this profession. These experiences, and many others, have given me a unique perspective because I've seen IONM all across the USA, and around the world. I see much of what we've done well, and where we've failed, and I'm acutely aware of the many formidable challenges that we face both nationally and internationally. This perspective has allowed me to build a vision for the future – at least of my little corner of the world here in the USA – and I've never shied away from sharing my views about the need for radical change in IONM. I guess that's how I ended up President of the ASNM.

My first job as President, even before I took office, was to build on my vision for the future of the ASNM by developing some goals to work toward. In order to do that, I had to look back at the history of the Society. How did we come to be today's ASNM, and are we on the right track? I also examined the context in which we presently function, which is modern IONM in the USA. What is going well, and what is not, and what must the ASNM do to lead this profession through these challenging times to ultimately build a better future for our members and the patients for whom we care?

Guest Article from ASNM President

In answering these questions and many others I developed the following Strategic Goals for the ASNM to focus on:

1. **Quality:** through education, guidelines and standards,
2. **Competency:** through credentialing and certification,
3. **Advocacy:** through patient education about who we are,
4. **Representation:** through positions we take, and
5. **Reputation:** through ethical conduct, collaboration and diversity.

From this point, I'd like to expand on each Goal to provide some background and context so you can see why I think we're heading in the right direction.

Quality is an important goal for me because I don't think we do a good job delivering high quality care in the USA, at least not consistently. Pervasive apathy throughout the field means that few people belong to societies, attend conferences or take continuing education seriously. We have no standards for education and training for IONM in the US. Almost all IONM is on-the-job training. Finally, our remote supervision model, while functional, leaves much to be desired. One way that we can tackle these problems is through education. As the only society in the US solely dedicated to IONM, we must be the go-to experts. While "education" has always been our core product at the ASNM, I believe our portfolio must be the best in the world. So, a big focus of mine is to ensure that our meetings, symposia, webinars and other content are an exceptional educational experience for practitioners at all levels.

In an effort to advance quality IONM, we must continue to publish evidence-based Guidelines. Where there is sufficient evidence to develop such guidelines and standards for the purpose of defining best clinical practices in the context of evidence-based medicine, we will continue our endeavor to publish and update such guidelines and standards as blueprints for the delivery of high quality patient care.

Competency is an important goal because we need a true benchmark to determine whether or not IONM practitioners have the education, training and experience to monitor specific cases and patient populations. For a number of reasons, we are failing in the US to demonstrate such competencies in any meaningful way. The ASNM is neither a credentialing body, nor a certification body, but that doesn't mean we simply wash our hands of this. First, the ASNM will engage relevant certification bodies to identify places where we need to raise the bar. Second, the ASNM will stand ready to assist certification bodies with any support they need to develop tests and methods for vetting clinical competency in IONM. Furthermore, as hospitals may have questions about how, when and why to credential neuromonitoring personnel, we will stand ready to offer guidance.

Advocacy is often something we overlook, but we must do a better job at educating patients about IONM because we work mostly behind the scenes. Many patients don't know about IONM until a technologist arrives at their bedside to perform an exam in the hours before surgery. The ASNM will continue important work being done in collaboration with the North American Spine Society (NASS), we're now joining forces with ASET's Neurodiagnostic Awareness and Patient Advocacy (NAPA) initiative, and we're updating our website to inform and educate patients about IONM.

Guest Article from ASNM President

Representation is always an important goal, but I've taken a different approach to this initiative. The ASNM is not focused on legislative actions such as coding and licensure. Other societies are tackling those issues and we will stand ready to support the efforts of our sister societies in any way we can. Instead, we will focus on representing neuromonitoring in other ways; ways in which we are well positioned to speak for the profession. As an example, the ASNM is working to provide this profession with the tools they need to move the needle on hot button issues that jeopardize our standing as an ancillary medical service and jeopardize patient care. So, for example, I'm focused on combatting the problem of outsourced IONM groups being classified as vendors by hospitals and made to wear inappropriate attire in the clinical setting. This may just be a problem in the USA, but I see it as a major patient safety issue that needs critical attention. So, we're representing the profession in different ways.

Reputation is perhaps the most important goal to me as President. Why do I care what people think about us? The reason is because our reputation is what drives membership and collaboration. The more members we have, the more influential we become. And, the more people are willing to work with us to advance this profession, the more success we will find in this endeavor.

Without question, holding our members to the highest standards of ethical practice, and collaborating with other societies, are both critical to advancing our reputation, but I want to highlight one of my most exciting initiatives: Diversity. I feel that we've always had difficulty getting women and minorities to join our leadership. I don't think it is a problem with our society or Board; rather, it's more of a pervasive problem in our profession of IONM. In order to examine these issues further, I've launched a Diversity Taskforce, which is being run by Dr. Tara Stewart. As Dr. Stewart quickly pointed out, the issues go well beyond women and minorities. Indeed, the mission statement that Tara developed made this task force bigger than I could have envisioned, and I'm really excited about the work that she's doing to get this task force off the ground.

One of the things that many people don't know about the ASNM is that we were founded in 1990 as a multidisciplinary society; our membership composed of and representing everyone who had an interest in IONM. The same is still true today. Our members consist of neurophysiologists, anesthesiologists, surgeons, neurologists, neuroscientists, audiologists, chiropractors, neurodiagnostic technologists, and many other clinical and scientific backgrounds. Beyond that, our membership also consists of people who work in medical billing, scheduling, biomedical engineering, HR, supply chain, and other administrative support roles for IONM. So, we are open to everyone without regard for education, training, role, etc. In some ways, diversity has always been at the core of who we are, and now we are expanding what it means to focus on diversity.

So, those of are the Strategic Goals that I established as President of the ASNM. These goals are not mine for the year, however, they are meant to set the direction of the ASNM for years to come. Obviously, I've just scratched the surface in the present article, but there's a lot more that we're working on, and I'm amazed at what we've already accomplished in the first two months. If you are interested in learning more about the ASNM, or want to get involved, please feel free to contact us at asnm@affinity-strategies.com

Rich Vogel, PhD
President
American Society of Neurophysiological Monitoring

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Can strength-duration analysis identify

the afferent limb of the lateral spread response in hemifacial spasm?

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Hemifacial spasm (HFS) is a cranial nerve hyperactivity disorder causing unilateral involuntary and irregular twitching of the facial muscles innervated by the facial nerve (FN). HFS is successfully treated using microvascular decompression (MVD) surgery. While the pathophysiological mechanism of HFS remains debatable, the success of MVD surgery implies an essential peripheral nerve role in HFS. Nevertheless, a central nervous system mechanism is involved as well as excitability changes within the facial motor nucleus causing generalized hyperactivity in the FN pathways are evident[1,2,3]. The lateral spread response (LSR) is a classic example of facial nerve pathway hyperexcitability and is readily obtainable in the anesthetized patient. This signature neurophysiological response is demonstrated by electrical stimulation of one branch of the facial nerve and observing the evoked electromyographic activity from muscles innervated by different facial nerve branches. The fundamental question of how neuro-vascular compression causes changes within the central nervous system remains unsolved. One possibility is that antidromic nerve impulses, presumably produced by the arterial pulsing on the FN, provide stimuli that modify the excitability of facial motor neurons. The lack of recurrent collaterals on facial nerve axons makes modifications to nuclear activity possible via antidromic conditioning[4]. It is also possible that trigeminal nerve afferents contribute to excitability changes in the facial nucleus. Coupling between trigeminal and facial nerve branches occur at several places on the face[5,6] and synaptic coupling between the trigeminal and facial nerve systems are well known (for e.g. the blink reflex[6,7]. There have also been reports in HFS patients whereby a functional link between trigeminal afferents and the generation of the LSR have been suggested[8,9,10].

For LSR generation the efferent pathway is undoubtedly the FN while the afferent segment remains unknown.

As described above 2 options are possible:

1) the antidromic signal along motor axon invades the facial nucleus to evoke the LSR from facial muscles; or **2)** the stimulus (or stimulus response) activates trigeminal afferents creating a pathologic reflex arc[8]. Facial muscles do not contain muscle spindles so sensory feedback from the facial musculature is entirely mediated by the trigeminal system[6].

Axons have different thresholds to electrical stimulation depending on several factors including the diameter of the axon fibre, the degree of myelination, expression of ion channels among others[11]. In human median nerve studies, stimulus response characteristics have shown that excitability distinctions can be made between the sensory and motor axons[12].

Strength-duration analysis is one method used to study these axonal characteristics. This method determines the threshold for motor or sensory responses at different stimulus durations (pulse width). From this analysis the rheobase and chronaxie, can be determined and compared. The rheobase is the minimal current needed to evoke a response at a theoretically infinite pulse width. From the rheobase, the chronaxie is defined as the stimulus pulse width at twice the rheobase current. The chronaxie is a function of the axonal membrane time constant and can shed light onto the myelination status, excitability state or possibly differentiate functional identity of the nerve(s) under study. In mixed nerves such as the median nerve, both sensory and motor strength-duration characteristics can be distinguished by stimulation studies using the same locus[12].

We studied the axons mediating the mentalis M wave and the simultaneously activated o. oculi LSR after stimulation of the marginal mandibular branch of the FN in 22 patients undergoing MVD for HFS. The mentalis M wave is entirely represented by motor FN axons while the LSR has an unconfirmed afferent and a motor FN efferent. Using the mentalis M wave as a control we compared the strength-duration properties of the M wave to the LSR to uncover any axonal excitability differences which might indicate afferent pathway identity.

Methods

This study was approved by our institutional review board (B2017:082) and patients were included after providing signed and informed consent.

All recording and stimulation electrodes were affixed to the patient's spasm side after induction of anesthesia. Paired 12 mm stainless steel subdermal needle electrodes (Medtronic) were placed into the o. oculi (upper and lower), o. oris, mentalis and masseter muscles for spontaneous EMG activity assessment, LSRs and mentalis M wave. For study data the LSR was achieved by stimulation of the mandibular branch of the FN and measuring the LSR from the o. oculi. Stimulation of the marginal mandibular FN branch occurred via paired 20 mm surface electrodes (Cadwell) directed along the FN on the border of the mandible. The cathode was placed 2 cm from the mentalis recording electrode position and the anode 2 cm proximally. The stimulus produced an orthodromically activated M wave from the mentalis and a simultaneous, antidromically directed, LSR from the o. oculi.

For strength-duration analysis of the M wave thresholds were determined at 40% of the maximum responses[13] as this is typically on the linear portion of the stimulus response curve ***(see Figure 1)***. Maximum responses (M wave and LSR) were derived from stimuli delivered using pulse durations of 1.0 ms. Because the LSR demonstrated a steep stimulus response curve ***(see Figure 1)*** we defined these thresholds as a

response of at least 20 µV in 50% of the stimulus trials similar to the method of Abalkhail et al.[14]. Maximum M wave and LSR were determined in each case and from this threshold target levels were established. Using target thresholds of different amplitudes has been shown not to affect the calculation of the chronaxie in human median nerve studies [12,15].

Strength duration studies used stimulus pulse widths of 0.05, 0.1, 0.2, 0.3, 0.4, 0.5, 0.75 and 1.0 ms delivered at ≤ 1 Hz. Data were displayed, stored and analyzed using Cadwell Elite work station running Cadwell Cascade neuromonitoring software. All EMG signals were filtered between 100 – 3000 Hz and the signals audibly amplified when necessary. The threshold stimulus values were reported as the actual current delivered by the electrode. Study measurements were conducted prior to dural opening as LSR responses may decrease or disappear at this stage of the operation[16]. After periods of bipolar cautery, collection of stimulus response data was suspended for at least 60 seconds to minimize the potential influence these currents might have on the study results.

In median nerve studies the current versus pulse duration relationship decays hyperbolically according to the formula of Weiss[17, cited in 11]. To obtain the chronaxie, the stimulus charge (µC) delivered [charge = current (mA)*pulse width (ms)] was plotted against the pulse duration (ms). According to Weiss[17] this linear relationship allows calculation of the chronaxie, where chronaxie (ms) = y int/slope = **µ**C/µC ms-1. The slope of this plot also describes the rheobase which is defined as the minimal stimulus current required to evoke a response using an infinitely long stimulus pulse width (18). From the rheobase, the chronaxie is theoretically defined as the stimulus pulse duration at twice the rheobase current[18].

For each patient, delivered threshold current vs stimulus pulse width plots were generated and fit to a 3 parameter hyperbolic decay function using SigmaPlot 13 curve fitting algorithm. From this, a charge vs. pulse width plot was derived and the chronaxie and rheobase empirically determined from the subsequent linear regression of each patient's data. Comparison of mean chronaxie and rheobase values were performed in SigmaPlot using a one-tailed t-test. Significance was assumed when $p < 0.05$.

We have previously determined that inhalational anesthetics can inhibit or block the LSR[16]. In addition, the chronaxie of human corticospinal axons has been shown to be significantly altered in the presence of the inhalational anesthetic sevoflurane[19]. Therefore during acquisition of the study data a total intravenous anesthesia was utilized.

Results

A total of 26 patients were recruited for this study but paired data from both the M wave and LSR were not obtainable in 4 people. The data from 22 patients was included for analysis.

Maximum responses

In order to fulfill a target level response for the M wave threshold (40% of maximum) the maximum response was determined. ***Figure 1*** shows an example of the stimulus response relationship from the mentalis M wave (A) and o. oculi LSR (B)

simultaneously acquired following stimulation of the marginal mandibular branch of the FN using a stimulus pulse width of 1.0 ms. The M wave response fit well to a sigmoidal function with an R2 = 0.99. In contrast the LSR (B) rose rapidly to a maximum peak below the midpoint of the M wave curve and typically decreased to a steady level with further increases in stimulation current. This response did not conform well to any common dynamic fit option and was a dominate feature for the LSR. The mean (± SEM) maximum amplitudes for the M wave and LSR from the study cohort (n = 22) were 3154 ± 496 μV and 203.2 ± 31.6 μV respectively ($p < 0.001$, one-tailed t-test).

Threshold responses

Figure 2A shows the mean strength-duration testing for both the mentalis M wave and LSR from 22 patients. Both data sets fit the 3 parameter hyperbolic decay with an adjusted R2 = 0.999.

The mean charge (μC) versus pulse width analysis is shown in Figure 2B. The linear regression r2 = 0.997 for the M wave and 0.999 for the LSR. From the linear regression plot the calculated chronaxie was 0.31 ms and 0.28 ms for the M wave and LSR, respectively. When individual chronaxies were averaged the mean (± SEM) chronaxie for the M wave was 0.34 ± 0.03 ms, while LSR chronaxie was 0.33 ± 0.04 ms. Chronaxies determined by the two methods were in agreement within the margin of error. The mean chronaxie data were not significantly different ($p = 0.42$, one-tailed t-test). For rheobase values statistical comparison demonstrated a significant difference between the M wave (8.0 ± 1.0 mA) and the LSR (5.7 ± 0.7 mA; $p = 0.03$, one-tailed t-test).

Discussion

The present study was conducted to determine if the strength-duration properties of axons mediating the LSR were distinguishable from those axons producing the mentalis M wave. By comparing excitability properties between the mentalis M wave and o. oculi LSR it would be theoretically possible to make inferences as to the afferent pathway mediating the LSR. This is the first study to determine the strength-duration properties of the LSR pathway and compare this to a well defined FN response (M wave) in HFS patients during MVD surgery. The afferent pathway involved in the LSR is unknown, although antidromic signalling along the FN tends to be the favored hypothesis[1, 20]. Alternatively, some investigators have suggested the trigeminal nerve provides the afferent pathway for LSR generation in HFS (8,9,10). Intuitively trigeminal nerve involvement in processing afferent information from the face makes sense as this is a major role for this cranial nerve under normal circumstances. However, if the LSR possessed strength-duration properties equal to the M wave then the pathway mediating the LSR would likely be facial nerve mediated. This was the primary outcome of this investigation and our most logical conclusion. We observed that the strength-duration properties of the LSR were virtually identical to the M wave suggesting an antidromic FN afferent pathway for the LSR.

Although the chronaxies of the LSR or the mentalis M wave were similar we did note significantly different rheobases. The rheobase difference is a consequence of the amplitude of the target threshold responses. As figure 1 shows, the M wave at 40% of maximum was much higher in amplitude compared to the LSR and particularly so for LSR at threshold. The relationship between target response amplitude and rheobase has also been noted by others studying the median nerve [12,15].

Using charge vs. pulse width plots it is possible for the slopes (equivalent to rheobase) of the measured responses to be different yet maintain similar chronaxies as shown in figure 2B. Thus the mean chronaxie for the LSR was essentially equal to that of the M wave despite the differences in rheobase and target threshold levels used for analysis. Unlike rheobase, the chronaxie is not influenced by threshold responses of varying amplitude [15,21].

Studies of median and tibial nerves have shown that motor and sensory axons are distinguishable based on strength-duration measurements[12,15,22]. Of particular relevance for the present study was the application of strength-duration analysis to the H reflex where Ia afferents of the tibial nerve have demonstrably different chronaxies compared to the α motor axons in the same nerve[23]. At first glance it would seem that the LSR mimics a sensory mediated response similar to the H reflex[7]. ***Figure 1*** shows that the LSR peaks well before the M wave and that the response decreases and plateaus at peak M wave stimulus intensities. At stimulus intensities at or near peak M wave responses, the decrease in LSR amplitude is likely due to action potential collisions along the FN, possibly with F waves. This is similar to H wave behavior. During the course of our stimulus-response testing we occasionally observed the LSR with little or no mentalis M wave generated, although this was not a consistent feature. Generally an M wave response coincided with the appearance of the LSR as in ***Figure 1***. H wave-like characteristics of the LSR have been noted previously by Misawa and colleagues [9] who stimulated the zygomatic FN branch and observed o. oris LSR responses. Investigations using Botox treatment of the spasm side o. oculi led to decreases in the LSR derived from lower face muscles [8] as well as decreased F wave responses from the mentalis [10]. These results were interpreted as support for trigeminal involvement in the mediation of the LSR and the modulation of the facial nucleus (via F wave inhibition). Our data, however, are not consistent with the conclusions of these investigators.

Strength-duration analysis has been conducted in awake HFS patients in the clinical laboratory but LSR per se was not investigated. Krishnan et al. [22] stimulated the temporal facial nerve branch and measured the strength-duration properties from the orthodromically activated nasalis muscle in HFS volunteers. The mean chronaxie measured using this protocol was 0.55 ms and the corresponding rheobase was 3.0 mA in these HFS patients. In a separate study using normal volunteers this group stimulated the marginal mandibular branch (at the anterior angle of the mandible) and conducted strength-duration analysis on the depressor angularis oris (DAO) muscle [24]. They reported a chronaxie of 0.4 ms which was closer to our mean chronaxie determination of 0.34 ms from the mentalis in our HFS patients ***(0.31 ms from Figure 2)***.

These data represent the first description of the basic excitability properties of one LSR pathway. We sought to determine if the strength-duration properties of the o. oculi LSR and the simultaneously acquired mentalis M wave were distinguishable. Our results indicate that the chronaxies determined from the LSR and M wave were virtually identical suggesting that the LSR is likely mediated by antidromic afferent signalling along the FN. We cannot exclude however, that trigeminal afferents possess similar strength-duration attributes as the FN and further excitability studies of trigeminal containing pathways is warranted.

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Dear colleagues,

It is a great pleasure to announce that the next event in a series of very successful meetings of the ISIN society will be held in Vienna. It is an honour to be your host and we are finalizing a fascinating program as well for the Educational Course as for the Scientific Meeting of the ISIN.

Techniques in surgery are under fast development, not only influencing the indications of surgery but also challenging the safety of the surgical interventions. Intraoperative neurophysiology offers an additional safety net for the neurological outcome in neurosurgery, orthopaedic surgery and many other fields of surgery. Thus, neurophysiological monitoring has become an essential tool of intraoperative neuroprotection.

The Educational Course will follow the modular structure of the ISIN educational program, that offers a comprehensive didactic concept. This year we will focus on surgery in brainstem areas and surgery on the peripheral nervous system. For surgeons, neurophysiologists, neurologists, anaesthesiologists and technicians, as well as for students in medicine and neuroscientists is a unique advantage to learn from a distinguished faculty of experienced neurophysiologists and surgeons about basics and application in high risk surgery.

The Scientific Congress will be a platform for communication for all related medical and scientific fields. It serves a roundtable for interprofessional exchange between scientists, clinicians, technicians and the medical industry. We invite participants from all over the world, to contribute their experiences and to communicate their requests within an interdisciplinary scientific environment.

We invite you to come to a stimulating and vibrant city with lots of historical gems to discover. You may find sources of the rich heritage of the Vienna Medical Faculty in collections, archives and architecture, dating back to the Age of Enlightenment in Austria. Vienna provides a safe harbour for many scientific and international societies to meet and work. As the capital of Austria, it is well connected to all major cities of the world. Music and art serve as the motor that constantly drives the intellectual output of the city. We hope you will be able to enjoy a fruitful convention and explore the touristic highlights in and around this marvellous place in the heart of Europe.

We hope to welcome you in Vienna.

Sincerely,
Dr. Klaus Novak
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Popliteal Fossa Stimulated Somatosensory Evoked Potentials as a Localization Tool for Peripheral Limb Ischemia: **A Case Report**

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Somatosensory evoked potentials (SSEPs) are commonly used during spine surgery to monitor the transmission of electrical current from the peripheral limbs to the somatosensory cortex via the dorsal column-medial lemniscal pathway. When performing lower limb SSEPs, a stimulating electrode is placed over the posterior tibial nerve at the ankle. Recording electrodes are placed in the popliteal fossa to record the nerve action potential and therefore confirm the functionality of the stimulator, as well as over the somatosensory cortex on the scalp. ***(Figure 1)***.

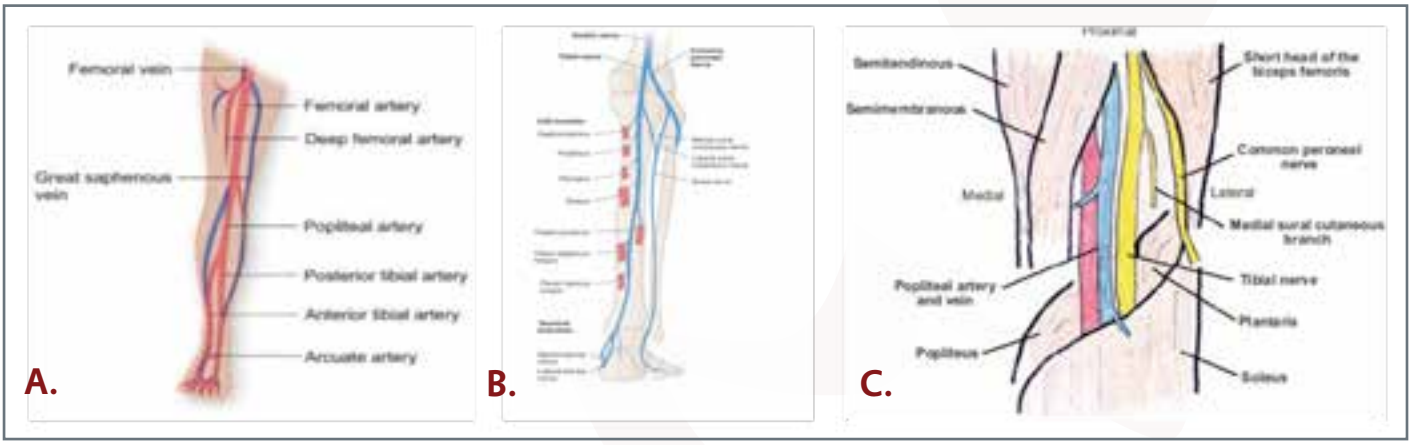

Figure 1: **A.** The pathway of the femoral artery and its branches through the distal leg. **B.** The pathway of the sciatic nerve and its branches through the distal leg. **C.** The popliteal fossa, illustrating the course of the popliteal artery and the tibial nerve.

https://www.texasheart.org/heart-health/heart-information-center/topics/vasculature-of-the-leg/

https://mobilephysiotherapyclinic.in/tibial-nerve-injury/

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Popliteal Fossa Stimulated Somatosensory Evoked Potentials as a Localization Tool for Peripheral Limb Ischemia: **A Case Report**

Patient

The patient is a 73 year old female who underwent a two-stage spine operation on a Jackson table for renal metastasis ***(Figure 2).***

Figure 2: A Jackson Table bottom (left) for supine positioning and top (right) for prone positioning

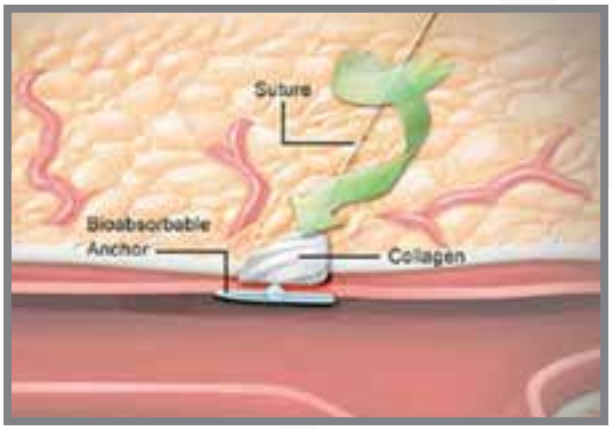

Figure 3: A diagram of the type of Angio-Seal arterial closure system placed in the patient's femoral artery the day prior to spine surgery.

Stage one of the surgery consisted of posterior C2-C6 and T7-T9 decompressions and instrumented fusions in the prone position. Following this, the patient was moved to the supine position using the Jackson table. Stage two involved an anterior C3-C5 instrumented fusion with a C4 vertebrectomy.

The patient had also undergone a vascular procedure resulting in the placement of an Angio-Seal device for femoral artery closure the previous day ***(Figure 3)***. Upon placement of the posterior tibial nerve stimulating electrodes, the patient's distal lower limbs were noted to be bilaterally mildly discolored suggesting poor distal circulation.

IONM Data

Baseline traces were obtained preceding the patient's turn to prone. Motor evoked potentials (MEPs) were recorded from bilateral trapezius, deltoid, brachioradialis, abductor pollicis brevis, and abductor hallucis muscles. SSEPs were obtained using bilateral stimulation at ulnar nerves at the wrist and posterior tibial nerves at the ankles. All evoked potentials were present, although the right lower SSEPs were lower in amplitude ***(Figure 4b)***, and the right abductor hallucis MEP was frequently absent (not shown). There was no change in any evoked potentials following the turn to prone ***(Figure 4c)***. Volatile anesthetic was used for induction and the anesthetic regimen was changed to TIVA after patient positioning.

Stage one of the surgery was performed without incident. However, during the final 10 minutes of stage one closure, a decrease in the amplitude of the right lower SSEP of approximately 40% was noted **(Figure 4e)**. At this time, neuromonitoring was paused to facilitate the repositioning of the patient from prone to supine. Volatile anesthetic was utilized during repositioning and turned off again when the patient was supine. Following the repositioning period, which lasted 35 minutes, neuromonitoring was resumed. At this point, a complete loss of the right lower SSEP was observed in all channels ***(Figure 4f)***. The left lower and bilateral upper SSEPs were unchanged. Upon visual inspection, the stimulating and recording electrodes were found to be intact but skin discoloration was evident. The possibility of peripheral ischemia was discussed at length between the surgical, anesthesia, nursing, and neuromonitoring teams. Right lower SSEP stimulation was then moved from the ankle to the popliteal fossa and a robust cortical response was obtained ***(Figure 4g)***, strongly suggesting distal leg arterial ischemia. At this time, the vascular surgeon on-call was paged for a consult.

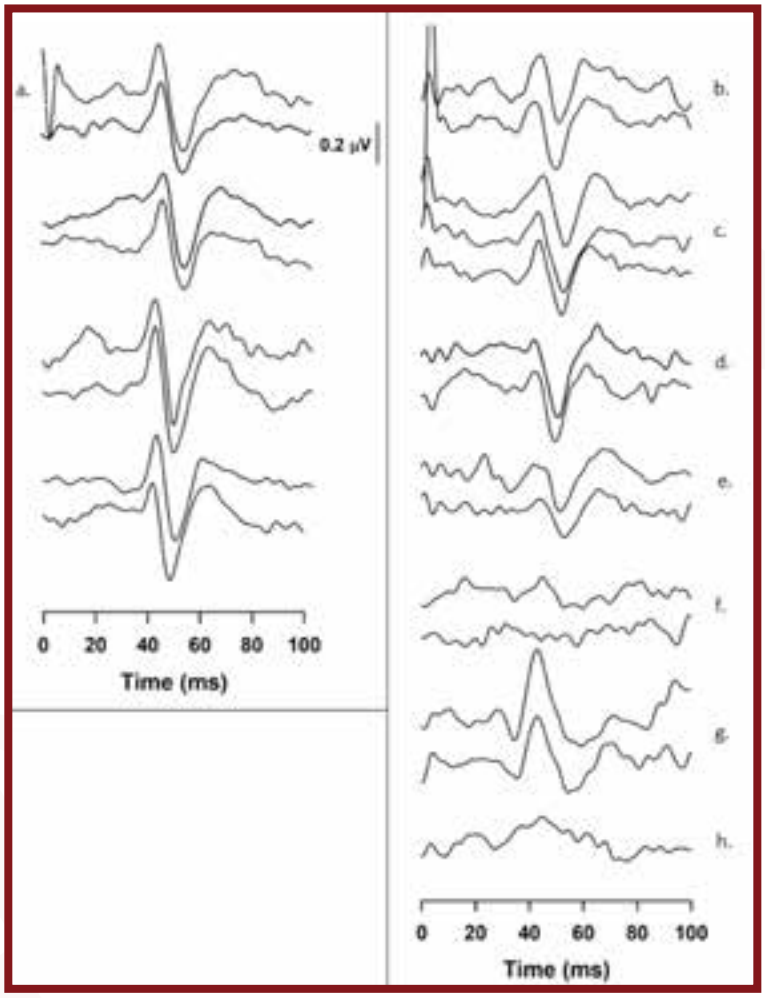

Figure 4: a. Left lower SSEP traces during pre-positioning baseline (top), post-exposure baseline, end of instrumentation, and closing (bottom). b. Right lower SSEP traces during pre-positioning baseline. c. Right lower SSEP baselines after exposure. d. Right lower SSEP traces at the end of instrumentation. e. Right lower SSEP amplitude decrease during closure of stage one. f. Absent right lower SSEP after patient turned to supine position. g. Right lower SSEP with popliteal fossa stimulation. h. Absent right lower SSEP with final test at posterior tibial nerve.

Vascular Procedure

The vascular surgeon arrived and performed a Doppler scan at the right popliteal fossa. A popliteal pulse was observed, but it was significantly weaker than the left popliteal pulse. A right femoral artery embolectomy was performed simultaneously with the anterior cervical decompression and instrumented fusion. After reperfusion of the leg, the vascular surgeon noted a strong right popliteal pulse with Doppler. The embolus was found to be the anchor of the Angio-Seal device placed in the femoral artery the previous day. Because all electrodes had to be removed from the right lower limb for sterility, no post-embolectomy SSEP data was obtained. The total time from the initial SSEP decrease to the beginning of the embolectomy was approximately 180 minutes. Unfortunately, the patient later underwent a below knee amputation due to ischemia.

Conclusion

During spine surgery, SSEP loss is most often attributed to spinal cord ischemia, cord injury, or plexopathy due to patient positioning. This case demonstrates the importance of physical investigation of the electrodes and the patient as a method of troubleshooting. In addition, when the cause of SSEP loss is suspicious of peripheral ischemia, utilizing popliteal fossa stimulation may be a means to localize the embolus.

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