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Kathleen Mears Memorial Lecture:
Personal Accountability: Your Key to Survival in Health Care Reform*

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ABSTRACT. Over the past thirty years the rising cost of healthcare has produced changes in reimbursement strategies. Continually, pressures are placed on the practitioners to reduce the length of the patient hospital stay and provide services in a high quality, risk free, cost effective manner. Following the implementation of diagnostic related groups (DRGs) in the 1980s and Managed Care in the 1990s we are now faced with embracing and surviving the Affordable Health Care Act-H.R.3590 (HHS 2013) that is linking reimbursement to quality outcomes. In short, financial constraints in the funding of health care will once more alter the patterns of delivery and challenge the practitioners to maintain superior care.

As Neurodiagnostic Professionals this new reform offers another opportunity to review our process of care and the Neurodiagnostic labs role in the delivery of healthcare. For success, close examination of routine workflows, recognizing and solving existing delivery limitations, developing team care coordination, and increasing the neurodiagnostic professionals profile within the work environment will be required.

Embracing your role in this overall process will most likely demand more paperwork, changing protocols, learning and implementing new policies, accepting new work schedules, implementing new quality standards, and pursuing additional education or credentials. Unlike never before more emphasis will be placed on measuring and reporting on the
quality of the care we deliver in our labs, intensive care units, and operating rooms.
Become “Accountable”; to manage costs and care.

KEY WORDS. Accountable Care Organization (ACO), diagnostic related group (DRG), healthcare analytics, Patient Protection and Affordable Health Care Act (ACA), quality outcomes, team care coordination, workflow process, VALUE.

INTRODUCTION

If you have been asked lately by your hospital administrator, company chief operations officer, or supervisor to reduce costs you are not alone. Virtually every health system around the country is engaged in cost-reduction initiatives as a result of the Patient Protection and Affordable Care Act (H.R.3590) initiated in 2014 (Karash 2013). Of concern is the shift toward a reimbursement system that removes the fee-for-service (quantity) to a reimbursement and incentive system based on an overall quality experience for the patient. This total experience will touch all facets of the patient from centralized management of patient care to technologies used for online patient communications with their caregivers (HHS 2013). To achieve success the collection and analysis of healthcare analytics becomes increasingly important.

While healthcare providers and healthcare facilities maybe crunching the same kind of data they have considered for many years; such as evaluations of needed full time equivalents (FTEs) and suppliers to reduce costs, the reality may be a reduction in costs while still maintaining quality without reducing healthcare workers will only occur if you look at how healthcare is delivered or more specifically the workflow process (Hughes 2008). As a manager, just sending out an email to make this happen doesn’t work but requires significant effort in collecting the data, evaluating the data, and then redesigning the workflow processes to improve care. Ultimately acceptance from the team for a change both in workflow process and new approaches to patient care will need to occur for success. Change is always difficult but involving the team in the process early, breaking it down into manageable “chunks” and recognizing the need to change makes the process easier.

First let’s examine some facts that lead our nation’s leaders to initiate a change in the healthcare system.

BACKGROUND

Some United States healthcare facts:

- Life expectancy of American men and women is less than other industrialized nations (NRC and IOM 2013).
Although nearly 46 million Americans are uninsured, the United States spends more on health care than other industrialized nations, and those countries provide health insurance to all their citizens (OECD 2013).

The Institute of Medicine (IOM) estimates that 44,000 to 99,000 patients die in hospitals each year due to medical errors (NRC and IOM 2013).

Medical errors are the eighth leading cause of death in the United States (NRC and IOM 2013).

The United States ranks last out of 16 industrialized countries on a measure of mortality amenable to medical care (deaths that might have been prevented with timely and effective care) (Pitney Bowes 2012, OECD 2013).

One in every five patients is readmitted to a healthcare facility within 30 days of discharge (NRC and IOM 2013).

Even as healthcare spending per capita has increased in the United States over the last three decades, the nation has fallen behind 12 other wealthy nations in 15-year survival for men and women at ages 45 and 65 (Budryk 2014).

The current healthcare system’s per capita spending ($8,508) is far greater than that of the second-most expensive system, Norway, which spends $5,669 per capita (Budryk 2014).

This sobering information is evidence and suggests the need to improve the health of patients across America but solving the multiple issues related to the delivery of care is a more complex task. For years, healthcare consumers have been used to demanding advance diagnostic testing after hearing about it from friends, the media, or even direct-to-consumer advertising. In turn, our physicians have gone along with these demands due to patient expectations, fear of overlooking anything that could conceivably contribute to a lawsuit, and lack of patient or physician accountability for cost (Rao and Levin 2012). Imperative however, in the new reimbursement model of the ACA is the need to examine our workflow process to reduce inappropriate, unnecessary, wasteful, or redundant and repeated testing and treatments. Within the three Titles of the ACA is the payment of incentives to healthcare facilities, physicians, and physician groups for their success in providing the best, most effective care to patients, not the most care.

One of the strategies to obtain this success is the adoption by many healthcare facilities and physician groups, to form Accountable Care Organizations (ACOs). While now not a new concept, for it was first introduced in 2006 by Elliot Fisher, M.D., MPH, the Director of the Center for Health Policy Research at Dartmouth Medical School, it is one of the ways healthcare providers have begun to manage costs and quality and other shortcomings in the US Healthcare System (McClellan et al. 2010). The goals for ACOs are to improve the experience of care for individuals, improve the health of patient populations, and lower the per capita costs for
taking care of patients; these being in-line with those of the ACA. It is estimated at the time of this publication that over 31 million Americans are receiving their care coordinated through an ACO (Gandhi and Weil 2012). While this is just a small percent of the US population it is noted that 45% of Americans live within driving distance to an already formed ACO and have the purchasing power to choose this type of care over multiple care providers in different clinical practices or care facilities (Accountable Care Facts 2014). The guiding principles within these organizations are to practice healthcare with focus on:

- Preventive care
- Care team coordination
- Common electronic health records
- Treatment based on evidenced-based proof
- Day or night access

Since their inception as early as 2009, approximately 488 ACOs have formed and have more than doubled the number from June 2012 (Petersen et al. 2013). While there are many different models of ACOs no single model has emerged as the most successful. They are all, however, focused on changing the way care is delivered.

WHERE AM I IN THE CURRENT HEALTHCARE REFORM?

Published in the ASET newsletter in the summer of 2013 in the Interest Section Briefings was a discussion on how new healthcare reforms will affect the neurodiagnostic profession. Margaret Hawkins, R. EEG/EP T., CNIM, CLTM posed the question to the Interest Section leaders based on the following comment. “Many techs are concerned that they as professionals and direct providers of health services are getting lost in bottom lines, productivity reports, and efficacy studies. They are wondering how to maintain respect within their institutions and be acknowledged for the unique role they play in caring for neurological patients” (Hawkins 2013). The comments from the Interest Section leaders were quite diverse; however, I found Susan Agostini’s, R. EEG/EP T., CLTM, Epilepsy Monitoring section leader and an ASET Board of Trustee, response motivating and relevant. Her response, I invite you to read, stemmed solely from the concept of creating value within the healthcare facility for what neurodiagnostic professionals do for the patient (Agostini 2013). Here, I share her concept and elaborate the need to become or maintain your value to your organization for long-term employment stability.

The online Merriam-Webster Dictionary (2013) defines value as: usefulness or importance; to rate highly; to have in high esteem; to hold in respect and estimation; to appreciate; to VALUE one for his works.
Using VALUE as an acronym the labels are:

- Visibility/Volunteer
- Accountability
- Learning
- Uniqueness
- Extraordinary

### Visibility/Volunteer

While it is easy to say, “be more visible in the hospital” the reality may be that as one individual working in a lab setting you feel somewhat anonymous. Yes, you may know the charge nurse in the operating room or that fantastic, caring neonatal nurse in the NICU by name but in reality your overall impact to impress upon them your value takes time to develop. Their ability to trust you as a healthcare provider who made a difference in the care of their patient is somewhat undermined when the diagnostic EEG report comes back with an interpretation of “clinical correlation advised.”

In essence Visibility is marketing and marketing should educate your healthcare community on what you do, how you do it, the impact it has on the patients you serve, and why your services are superior to the services down the street. With routine Internet usage by healthcare consumers the whole dynamics of marketing have changed and allow every neurodiagnostic laboratory the opportunity to promote and educate the public about the importance of their services and provide statistics on how neurodiagnostic procedures impact patient outcomes. Prior to the 2013 ASET Annual Conference in Reno, Nevada, I obtained a list of the attendees for the meeting. Using a statistical random sample I choose a list of facilities consisting of University, Community, For Profit, and Not-For-Profit hospitals from the attendees in the audience. I visited their Internet sites for evidence of Visibility using a variety of key search words that included EMU, Epilepsy, Seizures, EEG Lab, Neurophysiology Lab, Neurodiagnostics Lab, Clinical Neurophysiology Lab, and END Lab. The results were quite disappointing. Of the hospital websites surveyed only sixty (60%) percent had direct contact information such as a phone number for scheduling or questions listed for the Laboratory. Only five hundreds (0.05%) of a percentage had the hours of operation of the lab which were listed as 8 AM to 4 PM Monday to Friday. Certainly, if I was looking for Saturday services for my child I wouldn’t contact that facility. Again, only five hundreds (0.05%) of a percentage had a technologist listed as the healthcare provider you would meet with twenty-five (25%) percent not mentioning any staff. Some of the hospitals surveyed provided Epilepsy Monitoring Services. With these services, the mention of the technical staff was more frequent but not always present as evidenced below:
“A comprehensive, multidisciplinary treatment approach, featuring epileptologists, neuropsychologists, neuroradiologists, epilepsy neurosurgeons, and epilepsy nursing.”

“Neurologists, neurosurgeons, nurse specialists, EMU technicians, epilepsy fellows, residents, medical students, nursing students, psychologists, counselors, and social workers may all be involved in the care of each EMU patient.”

“...epilepsy team members, which include neurodiagnostic technicians and highly skilled nurses and physicians.”

Don’t you think “neurodiagnostic technicians” are also “highly skilled” not just nurses and physicians?

On one of the websites I found EEG services under Radiology and Imaging Services as observed below from the website:

“Radiology and Imaging Services
- Computed Tomography (CT)
- Diagnostic Imaging (X-ray)
- Electroencephalography (EEG) Lab
- Interventional Radiology/Angiography”

That being said the question arises; do we as a profession have a branding or imaging problem? Do healthcare consumers really know who we are and what we do? Certainly, this hospital doesn’t even know that EEG is not radiology or imaging services. While we have relied on our national organizations like ASET – The Neurodiagnostic Society and the American Board of Registration of Electroencephalographic and Evoked Potential Technologists (ABRET®) to help educate the consumer on who we are and market our unique abilities as neurodiagnostic professionals, it’s ultimately our responsibility to be Visible within our own hospital. Of particular note, one hundred (100%) percent of the websites I visited had a separate webpage for their sleep lab with extended operational hours, patient instructions for where to go and what to expect, direct contact information, achievements noted, the comment about the uniqueness of their services or new service-lines recently added, lab accreditation information, and their published research papers and current research efforts. Certainly, we should all be challenged to become more Visible on our hospital webpage.

Not only is it important to be Visible on the hospital webpage but also within the hospital community. “Visibility brings credibility and credibility brings trust” (Agostini 2013). Options might include presenting to the nurses, residents, and fellows during their required continuing education seminars on the use of EEG in the diagnosis and treatment of intractable epilepsy, or the role of transcranial motor evoked potentials (TCMEPs) in anterior cervical decompression and fusions, or the use of EEG in the diagnosis of seizures versus dementia in the elderly, or the use of cEEG
in therapeutic hypothermia patients. The list goes on and on. Certainly, a well presented lecture will go a long way in establishing you and your department as competent healthcare providers and educate them past the role of the bedside EEG which is what most of the hospital community sees you do. Other options might be to participate in a hospital sponsored support group for patients and parents of children with epilepsy. Explaining how to prepare for and what happens during an EEG recording could go a long way in ultimately making sure your parents and pediatric patients are prepared for their study and now less likely to miss their appointments. There are many reasons parents and patients don’t show up for their appointments. Have you done a recent survey of your patients to know why they don’t show-up on time or at all? If you are not in a position to create and implement your own survey, partner with a department (more Visibility) and collaborate for a generic survey to get your answers.

With the new emphasis on quality care, volunteering to be on a “quality team” outside your lab or to head-up the team within your laboratory would also bring exposure to what you do and how accountable you are in doing it. Establishing yourself as a member of the team and not just the “gal or guy that hooks up electrodes” is an important step toward accountability.

**Accountability**

Now more than ever before, numbers will drive the system. All across the nation hospitals are engaging in studies of safety, effectiveness, and the value of the services they offer. Getting started now with looking at how you do business, analyzing your results, reporting your outcomes, and sharing the outcomes with the hospital to prove your effectiveness and worthiness to the organization will be paramount in your success. Examine the lab workflow process for waste of technologist time, for example. With labor costs one of the largest expenses for most hospitals, decreases in productivity or not using your staff efficiently and effectively will be detrimental and expose them to layoffs if the hospital is benchmarking output/full-time equivalent (FTE) to other institutions. If your data indicate the department wastes twenty-five (25%) of each FTE each workday waiting for hospital transport to deliver an inpatient, the decision to look at different workflow models is economically an easy conclusion. Using an average neurodiagnostic technologist’s salary from the 2011 ASET Neurodiagnostic Profession Salary and Benefits Survey (ASET 2011), this wasted time would equate to $62.50/workday/FTE in salary (not including overtime, on-call pay, shift differential pay, or benefits). Adding this up over a year rapidly converts to over $13,000/FTE in lost time. If you have more than three (3) FTEs in the lab this wasted time is equivalent to almost one entire FTE (perhaps an assigned shared transport employee) not to mention that the technologist cannot complete
other responsibilities such as inpatients in the ICU due to waiting for patients to arrive in the lab. A conservative estimate would be that any lab could reduce the wait cost nearly fifty percent (50%) by performing recordings in the patient’s room versus the laboratory. While in the past technologists were hesitant to take equipment from the lab into patient rooms due to the “electrically hostile” environment, with current technology EEG amplifiers are capable of recording “noise” free EEG data making this excuse nonexistent.

Not only do you get Visible when venturing out of the laboratory but you increase patient safety by reducing the likelihood of patient falls during the transport process. According to the Agency for Health Care Policy and Research, approximately 700,000 to 1 million preventable falls occur each year in hospitals (Currie 2008). Research shows that close to one-third of these falls are preventable (Cameron et al. 2012). Rates of falls vary, however, with the type of unit. Among the highest fall rates occurring in the neuroscience (6.12 to 8.38 falls/1000 patient-stays) and medical (3.48 to 6.12 falls/1000 patient-stays) units (Fischer et al. 2005). Are these not the very patients that we would be transporting? Ultimately, while you are coordinating the care of the patient you are achieving multiple levels of patient centered care. First, a reduction in costs; second, interacting with nursing and other caregivers on the floors making yourself Visible; and third, providing care in a safer environment with expedited service and perhaps providing an earlier diagnosis or shorter length of stay. The importance of recognizing and offering suggestions to solve the existing delivery limitations of your services will ultimately save cost.

Obviously, there are many issues to consider but letting the statistical data guide the decision will achieve success. Let’s look at another high risk area in the delivery of patient care in a neurodiagnostic lab; needle stick injury (NSI). NSI either in the lab or more appropriately in the operating room (OR) while delivering intraoperative neuromonitoring is not uncommon, therefore; a more deliberate approach to prevent this is required. I think most of us have “it-won’t-happen-to-me attitude” and resist making changes to their daily routines simply because old habits are hard to let go. If, however, you are not going to worry about yourself consider your colleagues as potential targets for NSI; the anesthesiologist, nurse anesthetist, or that overzealous charge nurse who you have told a million times not to remove the needles.

Of significance is 384,325 needle stick and sharps-related injuries occur every year to healthcare workers in the United States with costs ranging from $500 to $3000/case for treatment (Panlilio et al. 2004). Sero-conversion or the development of detectable specific antibodies to microorganisms in the blood serum as a result of exposure is likely with Hepatitis B as high as 30%, with Hepatitis C as high as 1.8%, and with the least likely being HIV as low as 0.3% (CDC 2001). The CDC estimates the total sum of combined medical and work productivity costs for NSI is $188.5 million in the United States per year of which $107.3 million is direct medical costs and $81.2 million is lost-work productivity (O’Malley et al. 2007). Considerations to
reduce the risk, however, can be little effort but largely effective and mostly range from documentation of a policy and procedure to persistent loud communication. Start with communication at the beginning of the surgical case to those in the OR of the number of needles you placed in the patient and your intent to count them “out” at the end of the surgical case. The charge nurse will certainly now understand and respect the fact that you have to count them for she/he is also obligated for “counts” that are complaint with the Joint Commission hospital standards. No sponges left in a patient, no needles either. Her/his eagerness to help you take them out just got less when she/he realizes they will be responsible if one is left in by accident because you couldn’t remove it and count it yourself. If you work with the same team, they are most likely aware of where many of the needle locations are according to the surgical procedure. For your protection, however, if you add unusual needle placements communicate this as additional sites making everyone aware of further potential for NSI. Lastly, be true to your word, count them in and count them out and provide in your medical documentation the number for Joint Commission standard compliance. Consequently, you just took a step forward and gained some respect in the OR by demonstrating your infection control practices instead of just “yanking out” the electrodes because you’re in a hurry to get out of the room.

What about hospital acquired infections, (HAI)? Is there anything in the lab workflow process that can be changed or updated to decrease the likelihood of HAI? Unfortunately, approximately 1.7 million Americans will develop a HAI this year costing the US healthcare system up to $45 billion (Zimlichman et al. 2013). HAI costs 2.8 times more per patient and increases hospital stay by 2.5 times (Zimlichman et al. 2013). Do you feel your lab has any impact or control over these costs?

Historically, neurodiagnostic labs have used reusable surface electrodes for EEG, evoked potentials, nerve conduction studies, and other studies in the inpatient and outpatient populations. These surface electrodes are manufactured using expensive metals such as gold and silver which makes them as single-use items not cost effective. While the argument for disinfection and reuse can be made, the contrary argument of reusing surface electrodes is that it leads to cross contamination of communicable diseases. In a study from Germany, patients underwent conventional resting ECG procedures using vacuum ECG systems (Ambu 1999). All electrodes were cleaned and disinfected according to manufacture guidelines before and between each patient. At patients 1, 2, 5, and 10 the electrodes were tested for presence of colini forming units. Upon testing, between the first and second patients there were 400 staphylococcus epidermidis (SE) colonies and 40 micrococcus (MC) colonies in the samples despite the best efforts for cleaning and disinfection between uses. After the fifth and the tenth patients both the amounts of SE and MC colonies were higher than 400 and additional fungi types were found. These included the pathogenic candida albicans and trichophyton (Ambu 1999). This study demonstrates
the potential of non-disposable electrodes to transmit contamination through poor standard cleaning procedures despite one’s best efforts to clean them.

While in this study multi-resistant staphylococcus aureus (MRSA) was not tested for its presence, MRSA will reside on inanimate objects (Mayo Clinic Staff 2012). Therefore, the potential of non-disposable electrodes as a source of transmission from patient-to-patient is also likely for MRSA. I have heard many technologists state that in the presence of a highly contagious disease they dispose of the electrodes as biohazard at the end of a single or serial recording(s). The infectious status of patients is not always known at the time of an initial recording; thus, making the risk for patient-to-patient transmission via contaminated electrodes unknown to the caregiver at the time of the study. Inherently, the unknown increases the risk of cross contamination. While many labs exercise sterilization techniques such as Sterrad® (Ethicon, Inc., Irvine, California, USA) or autoclave, it may be simpler and just as cost effective to use disposable electrodes. In today’s marketplace the cost and reliability of disposable electrodes makes them an attractive alternative to reusable electrodes. In addition, the time spent on disinfecting and preparing for sterilization of reusable electrodes is equivalent to 25% of one FTE. This time might be better spent providing services. Research efforts have demonstrated a reduction in infection rates of grid telemetry patients of 100% when using single-use electrodes in an epilepsy monitoring unit during an 18 month period (Finnegan 2007). If you were the patient which would you prefer? According to the American Medical Association survey 94% of patients would prefer to go to a hospital that uses the latest technology available for preventing the spread of infection (Johnson and Johnson 2011). Switching to disposable electrodes for all neurodiagnostic studies will require a financial analysis and a shift in culture to spend more to ultimately spend less.

Lastly, the National Coalition of Health Care, the Institute for Healthcare Improvement, and The Joint Commission have focused on reduction of medical errors across the many disciplines of medicine for years. The problem of medical errors is not due primarily to a lack of knowledge. Rather, the chief culprit is generally the inadequate dissemination and implementation of ideas and practices we know work. Healthcare professionals are anxious to deliver safe care. When shown effective methods for changing systems to make them safer, doctors, nurses, pharmacists, and other healthcare professionals are eager to embrace them (Adams et al. 2000). Is reducing medical errors part of your daily focus as a neurodiagnostic technologist?

In 2012 the Johns Hopkins University’s Armstrong Institute for Patient Safety and Quality looked at errors in the adult ICU as cause of preventable mortality and morbidity (Winters et al. 2012). They estimated based on autopsy-confirmed research that twenty eight percent (28%), more than one in four patients had at least one missed diagnosis at death. In eight percent (8%) of these patients, the missed
diagnosis, infection, or a vascular event was serious enough to have caused the death and if known, likely would have changed treatment. In short, critical, vascular, and ischemic events are going unrecognized in the ICU which is preventable neurologic complications if recognized prior to permanent damage or death. Currently the measures used in ICUs to access brain blood flow are either indirect or direct. The indirect measures are jugular venous oxygen saturation (SjvO₂) and the Licox® Brain Oxygen Monitoring System (Integra® LifeSciences Corporation, Plainsboro, New Jersey, USA) which is partial pressure of brain tissue oxygen (PbtO₂) in the cells. While PbtO₂ is commonly used, it is invasive and not therefore practical in all patients. The direct measures are the use of the Bowman Perfusion Monitor® (BPM) (Hemedex, Inc., Cambridge, Massachusetts, USA), also invasive; the xenon computerized tomography (CT) which is costly and not available in all facilities; and most recently the use of intracranial microdialysis again, invasive, expensive, and not available in all facilities.

So what has this all to do with neurodiagnostics? Increasingly it is becoming clear that secondary neurological complications, such as brain ischemia, are not limited to patients with primary neurological injury but also is seen in the medical and/or surgical ICU populations (Foreman and Claassen 2012). These ICU patients are not likely to have invasive brain monitors in place to detect secondary brain injury at the time when permanent damage can still be prevented. By using continuous EEG (cEEG) however, much like continuous cardiac function monitoring, it is possible to have real-time detection of compromise in cerebral blood flow (CBF) and subsequent brain metabolism within minutes. With this information the ability to diagnosis, treat, and reverse impending secondary neurologic complication occurs rapidly not hours later or not at all (Sharbrough et al. 1973). In high risk patients such as those after vascular or cardiac surgery, during refractory hypotension, sepsis, or encephalopathy, the opportunity exists to reduce the medical error of not recognizing a secondary injury by using a non-invasive, inexpensive existing test procedure that provides a real-time look at the function of the brain (Foreman and Claassen 2012).

While ischemia in the ICU is overlooked and of concern, the most common reason to provide cEEG in the ICU is for detection of nonconvulsive seizures (NCS) and nonconvulsive status epilepticus (NCSE) in the comatose patient. With the incidence of NCSE in the critically-ill as high as 37%, it is evident that using cEEG, the only way to detect NCSE, will reduce patient mortality and morbidity and improve patient outcome by guiding effective treatment (Bleck 2012). Unlike a traditional bedside 20 to 30 minute EEG which is one point in time and interpreted hours to days after the recording, cEEG provides for uninterrupted assessment that triggers immediate treatment. Once first-line treatment has been initiated, cEEG identifies the effectiveness of the anticonvulsant therapy and guides patient care management for additional pharmacological support if the seizures are refractory (Jordan 1993).
But does every patient have access to cEEG? While other departments of the hospital have expanded and grown offering 24/7/365 care, the traditional neurodiagnostic lab still operates on a daytime schedule with limited on-call services. According to the 2011 ASET Salary and Benefits Report, 54% of the facilities that responded do not offer access to 24/7/365 EEG services (ASET 2011). Given clear evidence of the benefits of cEEG we can no longer ignore the need of 24/7 access. The neurodiagnostic community needs to initiate new clinical programs to provide services to the ICU and the ER that rely on evidence-based medicine that changes intervention or therapy to improve patient outcomes. In general, the medical/legal system has supported new monitoring technology when the cost of the monitoring is less than the incidence of a problem it detects times the cost of the problem (Drislane et al. 2008).

**Learning/Knowledge**

While focusing on taking care of patients more effectively is principle, it is not achievable without each neurodiagnostic technologist being the best that they can be. This means the need to increase your knowledge base by completing an undergraduate education and proving competency by becoming credentialed. It is no longer about being an individual neurodiagnostic technologist but about being a member of a healthcare team. Without the educational backing your effectiveness to compete within the working environment is diminished. Certainly, there are exceptions but for the most part without a formal education broadening the ability to think analytically and to effectively express ideas through oral and written communication, the power to gain the respect and trust you deserve may be out of reach.

If 20 years ago there had been a mandatory entry requirement of a bachelors degree into the field, licensure, and/or our laboratories were providing services 24/7/365 into all areas of the hospital including the ICUs, ORs, and ERs, our acceptance as health care professionals among our peers would be much farther along. They might even know who we are, what we do, and put us on their website.

Despite being behind, the need remains to educate and offer suggestions toward patient care management by being able to discuss the neurodiagnostic viewpoint among the team members. To do this effectively it means not only the initial education but subsequent continuing education staying abreast of current literature and new technologies to bring thoughtful change and innovation to the workplace; and to envisage that change and innovation into completed projects.

**Uniqueness**

An additional provision of the Affordable Care Act is the reporting to the public not only on the ability of the facility to achieve quality care but on the overall patient
satisfaction (HHS 2013). The ever so savvy healthcare consumer will certainly review the statistics; ask the outcome questions and make an informed decision regarding any elective admission to their local hospital or healthcare clinic. A quick Internet search will calm the pre-surgical patient consumer if the hospital participates in the many elective national quality improvement programs such as The American College of Surgeons National Surgical Quality Improvement Program (ACS NSQIP®) (ACS 2013). As participants in these types of quality programs, hospitals are required to track the outcomes of inpatient and outpatient surgical procedures and collect data that direct patient safety, the quality of surgical care improvements, and the overall patient experience (ACS 2013). These unique reported quality measures provide patients with assurances of the competency of the hospital to deliver services. Do you have a unique patient care service and how it is delivered in your lab that is measurable and reportable?

While medical and surgical outcomes data, studies on safety and effectiveness, and audits on staffing are important it can’t get into the way of listening to the patient and considering their opinions and viewpoints about how their care is delivered. Should overall patient satisfaction not be of good quality, a hospital may find themselves a victim of social media backlash. According to Healthcare Communication News (2013) the number two most common complaint patients make on social media is, “The service is bad”. With 41% of patients saying social media affects their choice of health care providers, it’s important to have a presence online and to monitor what is being said (Healthcare Communication News 2013). Remember however, social media alone can’t solve public perception problems. The best thing to do when you get complaints about bad service is to fix the service. When you hear folks complaining in the lab waiting area about specific staff or the length of the wait time don’t ignore them but investigate their concerns and assure them you want to do everything to correct the problem. While it seems like common sense, acknowledgement of the patient’s complaint and directing that concern to upper management as appropriate should reduce negative social media critique. Make it a point to find out who in your workplace is responsible for responding to negative social media complaints you may see on the Internet about your facility. Report these complaints as your responsibility to your patients. Customer service is always key; no matter what business.

Extraordinary

Despite all the metrics that providers are spending millions of Information Technology dollars on, it really comes down to providing care from the heart or doing the right thing: more commonly known as patient centered care.

Would you walk past a small child on the street that appears to be alone without asking if they are lost or need help? If not, then why would you pass a patient clearly
looking confused for where their appointment is without offering help with directions or accompanying them to the right location? Being extraordinary means taking the next step or going the extra mile. Doing just that and changing their approach to patients is Cleveland Clinic. In 2012 they instituted a new expectation among staff in their facilities called “no pass zones” (Theiss 2012). For example, if a patient’s call light is on and you are the first person passing by the room, no matter whom you are or what your role, it is your responsibility to go in and see what is going on and how you can help. I wonder how many times people pass by rooms because they are focused on the day at hand versus the hand that feeds them, the patient. As more patient consumers have high-deductible insurance plans and take on more of their own treatment costs, they will be looking for that quality – the extraordinary for the right price.

Now remember those ACOs we spoke about earlier. How did they do in their first year of delivering care? All 32 of the Pioneer ACOs met their 33 different quality metrics and performed well on cancer screenings, blood pressure control, and cholesterol control for diabetes patients. Twenty-five of the 32 had success in reducing hospital readmission rates. More than a third reduced costs, producing cumulative savings of more than $87 million and saving Medicare nearly $33 million (Petersen et al. 2013). While there is still much controversy about ACOs long-term future, a change was needed to focus on quality of care versus quantity of care.

CONCLUSION

While hospitals are not novice to market-focused strategies to boost positive images such as advertising how many minutes to wait for ER service; they need to consistently focus on the quality of the services they deliver in terms of error reduction and quality improvement and advertise this as well. Overall, safety pays. Decreasing medication errors, eliminating patient falls, reducing needle stick injury, decreasing nosocomial infection, recognizing and treating impending ischemic events before permanent injury ensues, recognizing and treating seizures when they occur versus hours or days later collectively add to our success as a healthcare system. Evaluate your role in the process. Large projects to small daily patient centered efforts all play a part in benefiting our patients and our health care system.

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Laryngeal Nerve Monitoring

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ABSTRACT. Intraoperative neurophysiological monitoring of the vagus and recurrent laryngeal nerves is increasingly used during thyroidectomy, parathyroidectomy, skull base surgery, and cervical discectomy with fusion. Monitoring can assist in nerve localization and in reducing the incidence of neural trauma. To be effective, however, monitoring must be correctly implemented and the results interpreted based on an in-depth understanding of technique and the surgical structures at risk. Because “poor monitoring is worse than no monitoring” all members of the surgical monitoring team must have training specific to laryngeal recording to maximize its benefit and minimize pitfalls. This publication will review pertinent anatomy and neurophysiology as well as technical and interpretative factors.

KEY WORDS. Cervical spine surgery, endotracheal electrodes, intraoperative neurophysiological monitoring, recurrent laryngeal nerve, superior laryngeal nerve, thyroidectomy, vagus nerve.

INTRODUCTION

Injury to the recurrent laryngeal nerve (RLN) has long been a concern during thyroidectomy and is the most common reason for malpractice litigation in endocrine surgery. Historically, the RLN was considered so fragile that surgeons were traditionally taught to avoid it. It was believed that simply dissecting in the nerve’s vicinity could lead to injury via direct trauma or disruption of its vascular supply. Surprisingly, a directive to do the opposite later countermanded this philosophy: routinely
identify the nerve surgically. Management of the RLN has now reached a third stage: active identification in conjunction with intraoperative neurophysiological monitoring (IONM). Intraoperative monitoring has gained wide acceptance in many surgical procedures that place nerves, spinal cord, or brain at risk. Monitoring of the RLN is increasingly used during thyroidectomy, parathyroidectomy, skull base procedures, and cervical disectomy with fusion. It is of particular benefit in cases with difficult anatomy due to prior surgery, large volume tumor/goiter, or history of radiation therapy. Recently, monitoring of the external branch of the superior laryngeal nerve (SLN) has also been advised. To be effective, however, monitoring must be correctly implemented and the results interpreted based on an in-depth understanding of technique and the surgical structures at risk. Poor monitoring is worse than no monitoring (Kartush 1989, Kartush and Bouchard 1992). Therefore, the purpose of this publication will be to review pertinent anatomy, neurophysiology, and technical and interpretive factors to optimize the benefits of monitoring the laryngeal nerves.

**MONITORING, TRAINING, AND COMPETENCY**

Complex, multimodality monitoring often involves numerous individuals including the surgeon, anesthesiologist, technologist, and an interpreting neurophysiologist (either present in the operating room or in constant real-time communication at a remote site). Each individual has an important role to play. A poorly trained technologist or neurophysiologist who conveys inaccurate information can inadvertently mislead the surgeon. Similarly, a surgeon with inadequate training in neurophysiologic monitoring may not be able to assimilate and act upon correct neurophysiologic information. Expertise in one modality does not assure expertise in all modalities. One must understand the dissimilarities, for example, between monitoring the facial nerve and monitoring the recurrent laryngeal nerve (Kartush and Lee 2013). Furthermore, even within a single modality, there may be significant differences based upon the procedure. Experience, for example, in monitoring the facial nerve during parotidectomy would be helpful but not sufficient for monitoring the facial nerve during acoustic tumor resection where understanding the effects of cerebrospinal fluid current shunting and cerebellar retraction are essential for proper interpretation.

Simple, single modality procedures using devices specifically designed for direct surgeon feedback (e.g., electromyography [EMG]) are often performed by the surgeon with or without the assistance of a technologist. When monitoring alone, the surgeon is duty-bound to assure that he or she has had proper training for both the technical and interpretive aspects specific to the monitoring modality employed. Regrettably at this juncture, no standardized educational curriculum exists for training all individuals involved in monitoring but, fortunately, educational assessment exams have at least evolved on the technical side. For example, the American Board of Registration of Electroencephalographic and Evoked Potential Technologists
LARYNGEAL NERVE MONITORING

(ABRET) credentials technologists via the Certification Examination in Neurophysiologic Intraoperative Monitoring (CNIM). ASET – The Neurodiagnostic Society has published National Competency Skill Standards for Performing Intraoperative Neurophysiologic Monitoring (ASET 2011). Neurophysiologists can be accredited on the interpretive aspects of monitoring by the American Board of Neurophysiologic Monitoring (ABNM). The American Society of Neurophysiologic Monitoring (ASNM) is a multidisciplinary society that promotes standards for all individuals involved in monitoring, including surgeons. The ASNM has published position statements on proper use of many IONM modalities (ASNM 2010) as well as practice guidelines for the IONM supervisor (Skinner et al. 2014).

Uniform monitoring training and certification have not yet evolved for surgeons but there is a clear need given that surgeons must always interpret the laryngeal monitoring data – and often perform the entire EMG technical set up, as well. Consequently, residency training programs must begin to formalize their IONM curriculum and develop departmental protocols in order to maximize the efficacy and safety of monitoring. There is a precedent for this in that many surgical departments already require special training and documentation should a surgeon wish to use new, complex, or higher risk procedures such as lasers or robotic assisted surgery. Publications such as those by International, German, and Taiwan Intraoperative Monitoring Study Groups have begun to bring key information to thyroid surgeons – and are likewise of value to all personnel monitoring laryngeal nerves (Dralle et al. 2008, Chiang et al. 2010, Randolph et al. 2011).

Historically, it was the facial nerve that was first monitored intraoperatively more than a century ago. Facial nerve monitoring has evolved to become a common procedure in many operations where the nerve is at risk. Given its demonstrated efficacy, the National Institutes of Health (NIH) in 1991 published a Consensus Statement recommending that the facial nerve be routinely monitored during acoustic tumor resection (NIH 1991, Hong and Kartush 2012, Kircher and Kartush 2012, Porter et al. 2013). In contrast to the facial nerve, the benefits of recurrent laryngeal nerve monitoring using current technologies are less convincing most likely due to: a) the monitoring techniques used to record the response (surface versus intramuscular recording), b) suboptimal methodologies employed by both technologist and surgeon (passive versus active evoked monitoring), and 3) the relatively low incidence of RLN injury (in the realm of 1%) requires a large sample size before studies can show the true benefits of IONM. These factors will all be examined. Table 1 summarizes some of the literature’s divergent conclusions on the present effectiveness of RLN monitoring during thyroid surgery.

Routine use of IONM can lead to behavior modification of the surgeon’s technique. Monitoring provides feedback that certain surgical maneuvers, traditionally considered proper in the past, may indeed be traumatic, e.g., blunt versus sharp dissection. If mechanically evoked potentials are elicited during dissection, the surgeon is
Table 1. Literature review of laryngeal monitoring efficacy.

<table>
<thead>
<tr>
<th>Paper</th>
<th>Nerves at Risk (Monitored/Unmonitored)</th>
<th>Benefit of IONM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Randolph et al. 2004</td>
<td>Palpation and Medtronic ETT/NIM®</td>
<td>586 Yes</td>
</tr>
<tr>
<td>Ulmer et al. 2008</td>
<td>EMG electrodes and surface stick on electrodes/vagal electrodes and Outcome</td>
<td>32 Yes</td>
</tr>
<tr>
<td>Petro et al. 2006</td>
<td>EMG electrodes/NIM®</td>
<td>60 Yes</td>
</tr>
<tr>
<td>Thomusch et al. 2004</td>
<td>Direct/indirect stimulation and Outcome</td>
<td>15,403 Yes</td>
</tr>
<tr>
<td>Chiang et al. 2008</td>
<td>EMG electrodes/NIM® and Outcome</td>
<td>173 No</td>
</tr>
<tr>
<td>Tomoda et al. 2006</td>
<td>Palpation and Outcome</td>
<td>2197 No</td>
</tr>
<tr>
<td>Schneider et al. 2009</td>
<td>Stick on electrode/vagus anchor</td>
<td>78 Yes</td>
</tr>
<tr>
<td>Beldi et al. 2004</td>
<td>Stick on electrode and Outcome</td>
<td>429 No</td>
</tr>
<tr>
<td>Chan et al. 2006</td>
<td>Stick on electrodes and Outcome</td>
<td>271 No (but author recommends for high risk cases)</td>
</tr>
<tr>
<td>Hermann et al. 2004</td>
<td>EMG electrodes and Outcome</td>
<td>502 No (helpful but does not predict postoperative outcome)</td>
</tr>
<tr>
<td>Robertson et al. 2004</td>
<td>Retrospective, cohort Medtronic ETT/NIM® and Outcome</td>
<td>116 No</td>
</tr>
<tr>
<td>Thomusch et al. 2002</td>
<td>Prospective, multicenter EMG electrode</td>
<td>2483/4640 Yes</td>
</tr>
<tr>
<td>Dionigi et al. 2009</td>
<td>Prospective, randomized minimally-invasive ETT/NIM®</td>
<td>55/57 No</td>
</tr>
<tr>
<td>Barczynski et al. 2009</td>
<td>Prospective, randomized EMG electrodes</td>
<td>1000/1000 Yes</td>
</tr>
<tr>
<td>Shindo et al. 2007</td>
<td>Retrospective Medtronic ETT/NIM®</td>
<td>427/257 No</td>
</tr>
<tr>
<td>Loch-Wilkinson et al. 2007</td>
<td>Palpation</td>
<td>100/40 No</td>
</tr>
</tbody>
</table>
alerted to the fact that they may be causing microtrauma to the nerve and thus their technique can be immediately modified. In addition, the proper use of electrical stimulation can also modify the surgeon’s dissection by actively “mapping” or confirming the location of nerves early during the surgical dissection even before nerves become visible to the surgeon.

Initially, RLN monitoring was performed by placing needle EMG electrodes into the vocal cords via direct laryngoscopy or transcutaneously. Difficulties in achieving this accurately and safely led to a search for alternative means. As discussed in detail in the historical section below, RLN monitoring is now typically performed by attaching surface recording electrodes to an endotracheal tube (ETT) due to its close proximity to the vocal cords. It is important to understand that while this commonly used technique has many practical advantages, the use of surface electrodes is a compromise that may increase the possibility of false-negative and false-positive errors. Furthermore, “hitchhiking” the laryngeal electrodes onto the endotracheal tube engenders potential risks to the airway. Consequently, it is important that surgeon, anesthetist, and monitorist all have training specific to laryngeal recording to maximize its benefit and minimize its disadvantages. There are significant differences in the recording modes of laryngeal versus facial EMG. Consequently, experience in facial nerve monitoring cannot be assumed to be sufficient to perform laryngeal nerve monitoring without additional training.

CRANIAL NERVE X ANATOMY AND PHYSIOLOGY

The vagus nerve, Latin for “the Wanderer”, is the longest of the twelve cranial nerves and contains fibers that subserve many different functions. Its viscero-motor parasympathetic and viscero-sensory fibers supply the entire aero-digestive tract

<table>
<thead>
<tr>
<th>Study</th>
<th>Nerves at Risk (Monitored/Unmonitored)</th>
<th>Benefit of IONM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chan et al. 2006</td>
<td>Stick on electrode</td>
<td>501/499</td>
</tr>
<tr>
<td>Yarbrough et al. 2004</td>
<td>Retrospective Hook-wire electrodes via direct laryngoscopy</td>
<td>52/59</td>
</tr>
<tr>
<td>Brauckhoff et al. 2002</td>
<td>Retrospective Children</td>
<td>91/78</td>
</tr>
</tbody>
</table>

ETT – endotracheal tube; NIM® (Medtronic, Minneapolis, Minnesota, USA); EMG – electromyography.
while its somatosensory and somato-motor branches innervate the palate, pharynx, and larynx. End-organs below the neck have dual innervation by both ipsi- and contralateral nerves, making trauma to one nerve clinically insignificant. In contrast, because of its highly complex and strictly unilateral innervation of pharynx and larynx in the neck, unilateral injury typically causes considerable functional impairment of the larynx.

The recurrent laryngeal nerve branches off the vagus at the level of the thoracic inlet, loops in an anterior to posterior direction around the aortic arch on the left and the subclavian artery on the right to then ascend in the tracheoesophageal groove towards the larynx (Figure 1). Its terminal branches enter the larynx between cricoid and thyroid cartilage at the cricothyroid joint before branching to supply the intrinsic laryngeal muscles. Anatomic variations along the RLN are common. Proximal bifurcation or even arborization along its course towards the laryngeal muscles has a reported incidence of 20 to 95% (Miller and Spiegel 2008). If dissection is carried along one branch assuming it is the main RLN, one or more branches or even the majority of the main motor trunk may be inadvertently sacrificed. This is of significant importance both clinically and specific to interpreting the results of RLN stimulation. Kandil et al. (2011) reported that 34% of their patients had extralaryngeal bifurcation of the RLN with the motor fibers routinely in the anterior branch while the posterior branch contained only sensory fibers. Such anomalies demonstrate the importance of frequent electrical stimulation which can neurophysiologically alert the surgeon that a nerve visible in the common trachea-esophageal location may not be a normal RLN but in fact be an aberrant sensory division. Under these circumstances, the enlightened surgeon-monitoring team who encounter what anatomically appears to clearly be the RLN but does not respond to stimulation will first exclude technical malfunction and then electrically map other more anterior and inferior areas for the branch that carries motor fibers to the vocal cords. In a similar way, “non-recurrent” recurrent laryngeal nerves can also be electrophysiologically identified. In these rare instances, the RLN does not originate inferiorly in the chest but takes a more direct path originating from the vagus nerve in the neck.

Histologically, peripheral laryngeal branches of the recurrent laryngeal nerve from healthy individuals contain small, medium, and large fibers. The large fibers with thick myelin represent efferent neurons for motor innervation of the intrinsic laryngeal musculature (Dahlqvist et al. 1986). Whereas the intracranial segment of the vagus lacks a protective sheath, the ultrastructure of the peripheral nerve has thick connective tissue (epineurium, perineurium, and endoneurium) shielding its nerve fibers. The unmyelinated intracranial portion of the nerve makes it exquisitely sensitive to both electrical and mechanical stimulation. In contrast, peripheral nerves like the RLN, with its motor thick fibers and the thick neural sheaths are much less sensitive. Contrasting facial and RLN responses during monitoring, a key difference is a reduced responsiveness of the RLN to mechanical stimulation. The proximal facial
nerve will readily elicit EMG signals with even minor surgical manipulation whereas it requires a much higher “strain” to mechanically depolarize the RLN. When the surgeon cannot rely on mechanical evoked potentials during dissection, electric stimulation of the nerve becomes even more critical in mapping the nerve’s location and repeatedly re-assessing the nerve’s function which can dynamically change due to

micro-trauma, traction, and de-vascularization. We refer to the use of frequent electrical stimulation as “active” monitoring, whereas “passive” monitoring relies only on detecting mechanically evoked trauma potentials.

The superior laryngeal nerve is composed of an internal and external branch. The internal branch innervates the sensory fibers of the larynx while the external branch (EBSLN) primarily innervates the cricothyroid muscle, which increases tension of the vocal folds allowing higher, sustained pitches. The SLN is a small nerve with quite a variable course which may increase the chance of inadvertent iatrogenic injury particularly when dissecting the superior thyroid vessels. It may be injured in up to 58% of patients but the incidence is likely underestimated due to the more subtle effects of SLN injury versus recurrent nerve injury (Teitelbaum and Wenig 1995). In 80% of patients, the SLN may lie in a sub-fascial plane of the inferior constrictor muscle making it difficult to visualize by conventional means. But fortunately for surgeons who use electric stimulation to actively monitor (versus only passively relying on mechanically evoked responses), the SLN can be readily identified neurophysiologically by stimulating at the superior pedicle even if the nerve cannot be seen, e.g., due to a sub-fascial location (Lennquist et al. 1987, Friedman et al. 2002).

Clinically, unilateral RLN trauma typically leads to hoarseness from a single paralyzed vocal cord – while bilateral trauma may lead to life-threatening airway obstruction (Figure 2). The paralyzed vocal cord will be in a paramedian position on laryngoscopy and will not move towards the center to close the glottis during phonation, nor move laterally with deep inhalation. With one functional vocal fold the airway is wide but the voice will be breathy (hoarse) because air will leak through the incompetent glottis. If both RLNs are injured, the laryngoscopic exam will show both folds in a paramedian position. Bilateral paralysis causes a raspy voice but, more importantly, due to lack of vocal fold abduction during deep inhalation, stridor is heard and the patient will have symptoms of shortness of breath with exertion. Furthermore, the combined sensory and motor paralysis can lead to aspiration of food and liquids resulting in pneumonia.

Injury of the SLN is frequently undiagnosed as clinical findings may be subtle. On laryngoscopy, the vocal fold of the injured SLN may be seen as slightly elevated compared to normal. Generally the voice is clear but the patient will be unable to maintain a high pitch tone due to loss of cricothyroid muscle function which acts to tense the vocal cords. Furthermore, the patient may experience coughing and choking when swallowing, particularly with thin liquids. This is because the SLN also provides sensation to the supraglottic region above the vocal folds.

Injury of the vagal nerve will lead to more pronounced symptoms as several of the mechanisms to protect the airway are compromised by the combined loss of both RLN and SLN function. First, ipsilaterial SLN causes the supraglottis to be insensate
allowing food or liquids to trickle into the larynx without being noticed and, second, the RLN injury creates a paralyzed vocal cord which prevents closure of the vocal folds. This combination is further exacerbated by the simultaneous paralysis of other vagal nerves that innervate the pharyngeal musculature above the glottis. These normally lead the food bolus into the posteriorly located esophagus rather than the anteriorly positioned trachea. Vagal nerve paralysis leads to loss of pharyngeal nerve function resulting in pooling of foods that can “spill” into the larynx when the food bolus is not “squeezed” into the esophagus. The consequence of these multiple dysfunctions markedly increases the risk of aspiration of food and liquids into the trachea resulting in recurring aspiration pneumonia.

The incidence of avoidable vagus nerve injury is unknown but high, proximal vagal injuries (e.g., skull base), especially when associated with concomitant IXth nerve injury can result in severe dysphonia, dysphagia, and aspiration. And yet, in a large published case series of skull base procedures performed by experienced neurosurgeons, the author comments that the vagus was monitored infrequently “due to the demanding and complex nature of monitoring this nerve” (Topsakal et al.
2008). Dysphonia is most commonly caused by RLN injury during neck surgery, particularly the thyroid. In 2009, almost 60,000 thyroidectomies were performed in the United States (Vashishta et al. 2012). The incidence of RLN injury during thyroid surgery lies between 1 to 6%. In revision surgery or surgery for malignant thyroid lesions RLN injury is known to be much higher, with 8% being reported as permanent. Fortunately, most RLN injuries are transient and recover within six months but even temporary vocal cord paralysis can lead to major complications such as aspiration pneumonia.

Accurate documentation of RLN function is imperative when collecting and analyzing data. The gold standard to assess vocal cord function is to perform preoperative and postoperative direct laryngoscopy, and when indicated, videostroboscopy and EMG. Simply relying on patients verbalizing a complaint after surgery may fail to detect partial injuries or those that are well compensated by movement of the contralateral vocal cord. Many past studies failed to obtain laryngoscopic documentation of vocal cord function and thus the true incidence of RLN injury has almost certainly been under-reported.

HISTORY OF LARYNGEAL MONITORING

Laryngeal recording methods have evolved over the last three decades. Initially, using principles from facial EMG, needles were placed into the true or false vocal cords by direct laryngoscopy or percutaneously through a transcricoid approach. The use of intramuscular needle electrodes had the advantage of robust EMG signals – but there were also significant disadvantages due to the specific microenvironment of the larynx. A critical factor leading away from intramuscular needle recording was the practical fact that, while direct laryngoscopy was a common procedure for Otolaryngologists, it was rarely in the armamentarium of General Surgeons (for thyroidectomies) or Neurosurgeons (for skull base procedures). Therefore, an initial alternative to laryngoscopic placement of vocal cord electrodes was transcricoid approaches to the larynx by placing long needle electrodes through either the skin or after through the cricothyroid membrane once it had been exposed surgically (Spahn et al. 1981, Lipton et al. 1988, Alon and Hinni 2009) (Figure 3). When correctly placed, excellent laryngeal EMG could be obtained – but disadvantages also prevented this method from becoming a standard. Most importantly, the transcricoid approach entails “blind” electrode placement by the surgeon who can only estimate the final location of the electrode tips after penetrating the cricothyroid membrane. Poor placement could result not only in false negative errors but there are reports of the needle electrodes penetrating through the vocal cords to then puncture the subglottic endotracheal tube balloon resulting in the need for urgent reintubation.
Straight needle electrodes placed via laryngoscopy were also problematic because they not uncommonly became displaced during the operation, for example, when the head was placed in hyperextension. This displacement was especially of concern because once the laryngoscope was removed, the larynx was no longer visible – thus unrecognized electrode displacement led to false negative monitoring errors. In an attempt to reduce electrode displacement, hooked tip electrodes were tried which did reduce displacement, but the barb-like hooks often traumatized the vocal cords when removed at the end of the procedure. Infection and bleeding into the vocal cords are yet other concerns of needle electrodes.

Nonetheless, apart from RLN monitoring, carefully placed needle electrodes may have value for improving specificity when monitoring the SLN’s target muscle, the cricothyroid (Inabnet et al. 2009). Once the surgical field is opened, needle electrodes can be placed under direct visualization into the cricothyroid muscle. This accurate recording technique readily allows detection of electrically evoked responses of the SLN when the surgeon is about to dissect the superior pole of the thyroid gland. Stim probes or stimulating dissection instruments can be used to map the location of the SLN prior to ligation of vessels in the area. This is of particular importance for the times when the SLN is in an invisible, sub-fascial plane.

**FIG. 3.** Transcricothyroid placement of intramuscular needle electrodes into the vocal cords is possible once the operative field has been opened. The final position of the needle electrodes, however, can only be estimated. (Reprinted with permission from John Wiley and Sons from Alon and Hinni 2009.)
In the past, maintaining a laryngoscope or video-equipped endoscope in place throughout the procedure has been attempted primarily to directly visualize movement of the vocal cords; secondarily as a means to assess the position of needle or surface electrodes. Such visualization can be of benefit to determine if there is a recording error (e.g., if a laryngeal twitch is visualized in response to electric stimulation despite no recorded EMG, a technical recording error is confirmed). The technique, however, is impractical as the principle means of continuous, ongoing observation of small vocal cord movements when procedures may last for hours.

Therefore, despite the acknowledged superiority of intramuscular needle electrodes for EMG recording in general, the multitude of practical problems specific to RLN monitoring has led most surgeons to settle on surface recording of the laryngeal response as an acceptable compromise. Ease of surface electrode placement has, at this time, trumped superior EMG needle electrode recording. While this compromise is understandable based on the foregoing history, the intraoperative monitoring clinician and surgeon must both keep the advantages and disadvantages of this compromise clearly in mind. Limitations and pitfalls of surface recording will be elaborated below.

ANESTHETIC CONSIDERATIONS

As with all EMG monitoring, long-acting paralytics must not be administered by anesthesia nor should topical viscous lidocaine be used during intubation. Neuromuscular blockade of 50% or more will obliterate mechanically evoked neurotonic discharges and markedly increase stimulation thresholds (Daube 1991). Low doses of neuromuscular blocking agents may allow recording of electrically evoked responses but abolition of the smaller mechanically evoked responses is too significant of a disadvantage to allow their use except under extenuating circumstances.

Because surface laryngeal electrodes are attached to endotracheal tubes, it is essential that the anesthesia team be trained in the proper placement of the tube to avoid false negative and positive monitoring errors. As detailed later, surgeon, anesthesiologist, and monitorist all have an obligation to understand the proper use and precautions of the monitoring device they select. This is especially true for factory-adhered electrodes because the endotracheal tube selected by the factory may not have been the ideal choice of the anesthetist who may have to modify or compromise their technique in order to use this particular tube. According to the package insert of one of the most commonly used factory-installed laryngeal electrodes, anesthetists are prohibited from performing common airway maneuvers such as suctioning through these endotracheal tubes – nor may they use a tube exchanger. Furthermore, they may be required to use a bite guard to prevent the anesthetized patient from inadvertently collapsing the tube and compromising the airway. The package inserts are an important source of information that should be clearly understood by all the
stakeholders. Likewise, package inserts are an important source of information for user-installed electrodes that must be appropriately placed to optimize position on the tube as well as minimize the chance of electrode displacement.

SURGICAL DISSECTION OF THE RLN

Certain maneuvers and specific locations during thyroid or parathyroid surgery place the RLN at increased risk for injury. Suboptimal positioning, exposure, and visualization will make every surgery more challenging and error-prone. Safe dissection is maximized with adequate exposure, clear identification of landmarks, and a bloodless operating field. Classically, the surgeon maximizes exposure with a wide dissection around the thyroid gland, but minimally invasive techniques including use of endoscopes and robots are increasingly being used to minimize scar and postoperative pain. In so doing, intraoperative monitoring may become even more important in locating and preserving the laryngeal nerves.

Different surgical techniques are employed based on training and experience – some approach the gland starting superiorly while others choose an inferior approach. Nonetheless, the nerve is often at greatest risk at the thyrohyoid ligament (“Berry’s ligament”), a tight band stabilizing the thyroid. Dissection typically begins after ligation of the middle thyroid vein and separation of the superior pole, allowing the gland to roll medially. Identification of the RLN may be undertaken at this time or the RLN may be sought inferiorly within the “RLN triangle”, with its boundaries along the trachea, carotid sheath, and the undersurface of the retracted thyroid, as described by Lore et al. (1977). Dissecting parallel to the course of the nerve will minimize stretch injuries of the RLN. Once identified it is traced towards its entrance site into the larynx near Berry’s ligament. Meticulous dissection is required along this ligament as the nerve passes under or through a tight fascial band. Small vessels within the ligament will frequently cause minor bleeding but cautery should be avoided in this area to prevent thermal injury to the RLN. If this approach fails, the superior approach can be used identifying the RLN at its entry into the larynx, dissecting the nerve from medial to lateral.

RECORDING ELECTRODES

For monitoring the RLN, contact surface electrodes are attached to an endotracheal tube either by the user or at the factory – each method having its distinct advantages and disadvantages. Intubation technique is critical, not only to align the electrodes with the surface of the vocal cords, but to assure proper use in order to minimize risks to the airway. Because these electrodes are not intramuscular, slight malposition of the surface electrodes can result in missing the laryngeal EMG (a false negative error) – or inadvertently recording from other nearby muscles (e.g.,
inferior pharyngeal constrictors) which could result in a false positive error. It is possible to design pinpoint laryngeal electrodes (LE) to contact only the vocal cords, which would restrict recording predominately from the thyroarytenoid muscles within the cords. Such precision, however, would then miss recording the compound motor action potentials from many of the other important laryngeal muscles, especially the posterior cricoarytenoid muscles which are key abductors of the vocal cords. Consequently, most LEs are designed to be broad enough, or multiple in number, to contact a large portion of the laryngeal inlet. Some RLN’s “arborize” or split into multiple branches before entering the larynx near Berry’s ligament. Stimulating one such branch while only recording from the thyroarytenoid muscles might miss the fact that a branch to the posterior cricoarytenoid muscles has been encountered. Thus, as is true in other types of EMG intraoperative monitoring, recording should be selective enough to avoid inadvertent recording of non-target muscles – while sensitive enough to sample a sufficient population of muscles in the area of interest.

As noted, surface electrodes are of two basic types: either factory installed unto an endotracheal tube, or an adhesive type applied by the clinician to an endotracheal tube of the anesthetist’s choosing. A commonly used factory-installed LE, the NIM® EMG tube, is manufactured by Medtronic (Minneapolis, Minnesota, USA) (Figure 4). Factory installation provides a secure connection of the LE to the NIM® tube. Conversely, the anesthetist is restricted to using a wire reinforced tube selected by the factory with only three size choices: 6, 7, and 8 mm internal diameter (ID). Half sizes have not been made available in the United States.

These prefabricated Medtronic NIM® tubes were initially criticized for being too flexible. The company responded by stiffening the tube with a reinforcing wire on the inner lumen surface. The stiffening wire, however, has proven susceptible to rare collapsing or unraveling which may result in airway obstruction. Inadvertent biting on the tube by the patient can lead to collapse of the endotracheal tube lumen.

**FIG. 4.** Medtronic NIM® laryngeal monitoring endotracheal tube. (Reprinted with permission from Jack M. Kartush, M.D.)
(Rengasamy and Ortega 2004, Kartush and Lee 2013). Therefore biting or common anesthesia maneuvers such as suctioning or using a tube exchanger must be avoided to prevent unraveling of the wire which may then extend beyond the tube which can then create an obstructive fibrin clot (Evanina and Hanisak 2005). Other special features of the NIM® tube such as its slightly longer length as well as the silicone elastomer cuff have also been reported to cause airway obstruction and pneumothorax (Leissner et al. 2007, Kim et al. 2010, Sundaram 2010, Capra et al. 2012). Additional problems with overinflated cuffs leading to similar airway emergencies have been described with this tube (Oysu and Demir 2011) (Figure 5A, B, C, D).

Thus, special precautions to avoid airway obstruction with the factory installed electrode include: 1) use of a bite block (Figure 6), 2) avoidance of suctioning through the endotracheal tube with a catheter, and 3) abstaining from use of a tube exchanger. Once again, it is strongly emphasized that users familiarize themselves with the manufacturer’s recommendations for use.

It is not widely known that the NIM® endotracheal tubes have a larger outer diameter than the average endotracheal tube of the same numbered size (which represents the ID, internal diameter). This fact in conjunction with the absence of half sizes, may lead the anesthetist to use a larger than expected diameter endotracheal tube which may increase the risk of laryngeal trauma. The NIM® tubes as well as all user applied LE devices are contraindicated for prolonged intubation. Consequently, if a patient must remain intubated after surgery, the endotracheal tube with attached LE should be replaced.

Other options for surface electrodes include taped-on designs, which are attached to conventional endotracheal tubes. Some examples include Rea’s design by Neurovision™ Medical Products (Ventura, California, USA) (Figure 7A) and those by Magstim Neurosign® Surgical (Carmarthenshire, Wales, UK) (Figure 7B). They are of lower cost and there are no additional concerns about airway safety because the anesthetist selects whichever endotracheal tube they believe is best for their particular patient. These laryngeal electrodes accommodate full and half sized endotracheal tubes. There are no limitations to the anesthetist on suctioning through the tube or using a tube exchanger. The electrodes should be taped approximately 1 to 2 cm above the cuff. A water-based lubricant (e.g., K-Y® jelly, Johnson and Johnson, New Brunswick, New Jersey, USA) may be used after the electrodes have been applied, not before. As with factory-installed electrodes, surgeon, anesthesiologist, and monitorist must familiarize themselves with proper use of the stick-on electrodes. Figure 8 provides an example of the proper method to attach one such user-applied electrode. During very long cases (e.g., skull base tumors), it is possible for the adhesive to become less adherent resulting in slippage from the tube. While such electrode slippage has never caused any reported airway problems, the accuracy of EMG recording may be diminished if they slip inferior or superior to the glottis. Conversely, if the electrodes simply lateralize from the ET tube, their position may
FIG. 5. If not properly protected, the reinforcing wire within the Medtronic NIM® monitoring tube can unravel causing airway obstruction. A demonstrates wire unraveling beyond the endotracheal tube (ET) tip with formation of an obstructive fibrin clot. (Reprinted with permission from Jack M. Kartush, M.D.) B demonstrates wire unraveling within the endotracheal tube. (Reprinted with permission from the AANA Journal from Evanina and Hanisak 2005). In C the arrow indicates kinking of the Medtronic NIM® monitoring tube may cause airway obstruction. (Reprinted with permission from Lippincott William & Wilkins from Rengasamy and Ortega 2004.) In D, the left photo shows normal ET tube where balloon maintains patent lumen. The right photo shows how a prolapsing balloon of a NIM® tube can cause obstruction of the endotracheal tube by deflecting the tip onto the tracheal wall. (Reprinted with permission from the American Medical Association from Capra et al. 2012.)
FIG. 6. The risk of NIM® endotracheal tube obstruction can be reduced by proper placement of “bite guards” (such as these created out of gauze) to prevent the patient’s unconscious biting down onto the tube. (Reprinted with permission from Jack M. Kartush, M.D.)

FIG. 7. User-applied laryngeal electrodes: A) Neurovision™ Medical Products (Ventura, California, USA). B) Magstim Neurosign® Surgical (Carmarthenshire, Wales, UK).
actually move closer to the vocal cord muscles thereby improving recording of the adjacent EMG responses. Newer stick-on electrodes have overlapping adhesive to reduce the possibility of becoming detached.

In order to avoid missing important EMG responses, clinicians must assure that the laryngeal electrode is in as close proximity to the vocal cord muscles as possible. If the endotracheal tube is placed too shallow, too deep, or rotated, recording can be compromised.

As noted, a major concern of using surface electrodes to record EMG potentials is that poor contact of the electrodes to the vocal cords will adversely impact the accuracy resulting in difficult to measure low amplitude responses – or even completely missed responses. Furthermore, even a perfectly positioned endotracheal tube may not allow perfect apposition of the surface electrode to the vocal cords.
Laryngeal electrodes are traditionally flush against the endotracheal tube yielding a circular surface in the midst of the larger triangular glottic inlet. This geometric mismatch often leads to sub-optimal contact of the electrode to the vocal cords. A commonly recommended way of compensating for this is to use an overly large endotracheal tube to force the electrodes closer to the cords. However, an oversized endotracheal tube can cause trauma during intubation and place undue pressure on vocal cords and trachea. Balloons were considered long ago as a method to push the electrodes closer to the cords – but fear of glottic and sub-glottic trauma prevented that method from being accepted.

To solve this problem, a new self-optimizing laryngeal electrode has been designed to gently fill the triangular glottic space atraumatically: Lantern Laryngeal Electrode (Neurosign® Surgical, Carmarthenshire, Wales, UK) (Figures 9 and 10). Like the unfolding of a Chinese lantern, the Lantern Laryngeal Electrode gently fills the triangular glottic space to optimize vocal cord contact without having to rely on an over-sized endotracheal tube.

SUPERIOR LARYNGEAL NERVE MONITORING

In contrast to the RLN, paresis of the superior laryngeal nerve (SLN) may lead to more subtle symptoms that are more likely to be missed. Unlike the RLN, the SLN enters the thyroid from a superior branch of the vagus nerve. In addition to sensory function, it innervates the ipsilateral cricothyroid muscle which helps maintain and increase vocal fold tension. The incidence of SLN injury during thyroid surgery has been reported between 28% and 58% but the incidence is likely underestimated due to the more subtle effects of SLN injury versus recurrent nerve injury (Teitelbaum and Wenig 1995, Lifante et al. 2009) A slight elevation of the ipsilateral vocal fold and the inability to maintain a high pitch tone may be the only findings on exam and might only be noted by a professional voice user, such as a singer. As noted previously, sensory anesthesia resulting from SLN injury may lead to silent aspiration and pneumonia, especially in a patient with other morbidities. Recently, attention has

![Image of Lantern Laryngeal Electrode](image-url)

**FIG 9.** Lantern Laryngeal Electrode (Magstim Neurosign® Surgical, Carmarthenshire, Wales, UK). (Reprinted with permission from Jack M. Kartush, M.D.)
been focused on reducing the risk not only to the RLN but to the SLN (Barczynski 2013).

The most common method to measure recurrent laryngeal nerve responses uses a surface electrode on an endotracheal tube. Because the SLN primarily innervates the cricothyroid muscle which is not in contact with this electrode, responses may be missed. However, in 68 to 83% of patients of patients, there is a communicating nerve from the SLN that provides additional innervation to thyroarytenoid muscle within the vocal folds (Sañudo et al. 1999, Maramillo et al. 2003). Consequently, stimulation of the SLN may in fact register a response on an endotracheal tube electrode that has leads anteriorly in contact with the vocal folds by means of this secondary mechanism causing tensing of the vocal cords – rather than contraction of the cricothyroid muscle. Because this vocalis muscle contraction is not present in all patients, endotracheal tube electrodes alone cannot be relied upon to detect all SLN responses. Consequently, it is best to complement the endotracheal tube electrode either by a) visual inspection of the contraction or b) by placing a needle electrode into the muscle once the cricothyroid space has been surgically exposed.

**Technical Aspects – Electrode Position**

Close contact of the electrode to the vocal musculature is critical to maximize surface EMG recording. Positioning too deep, too shallow, malrotated, or too loose a contact will lead to recording errors. The endotracheal tube must be of the appropriate size. If the tube is too small, the surface electrodes will have insufficient contact

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**FIG. 10.** Standard versus self-optimizing laryngeal electrodes. Left: Standard flush mounted laryngeal electrodes with incomplete vocal cord contact. Right: Lantern Laryngeal Electrode with dynamic flowering electrodes atraumatically optimizes contact with vocal cords. (Reprinted with permission from Jack M. Kartush, M.D.)
with the vocal fold resulting in inaccurate EMG recordings. During intubation the anesthesiologist places the endotracheal tube such that the color-coded location of the laryngeal electrodes is centered at the midline of the vocal cords. To assure optimal positioning, attention must be paid to tube depth, rotation, and diameter.

Unintended movement of the endotracheal tube may occur when the head is positioned; rotated for skull base procedures or hyper-extended for thyroid surgery. Such movement may displace the laryngeal electrodes therein compromising recording of the EMG response. If the head must be adjusted it may be helpful to re-visualize the glottis following re-positioning using a laryngoscope, flexible endoscope, or newer iterations such as the GlideScope® (Verathon Inc., Bothell, Washington, USA).

With commonly used laryngeal electrodes, there may be a 15% incidence of false negative responses. In a study of 173 nerves monitored during thyroid surgery malpositioning of the tube (and therefore the electrodes) was suspected to be the most common cause of failure to recognize a nerve injury (Chiang et al. 2008). With exposure of the larynx in the neck, Randolph et al. (2004) noted that suspected false negative errors can be cross-checked by stimulating the presumed nerve while directly palpating the laryngeal twitch response in the tracheo-esophageal groove. If a laryngeal twitch can be palpated despite absence of an EMG response, a technical problem is confirmed and must be corrected.

While it is important for electrode impedance to be low (typically below 5 kOhms), low impedance does not equate to proper electrode position; “good tissue contact” in an unwanted position (too deep in the trachea or within the esophagus) will also yield low impedance. Similarly, a “tap test”, commonly performed with intramuscular facial muscle electrodes, provides no information on whether laryngeal electrodes are in a proper position.

Nerve Stimulation

Stimulation can be performed with either bipolar or monopolar stimulators (Kartush et al. 1987). While bipolar stimulation is most specific, monopolar stimulators are superior for mapping the general location of a nerve especially when it is not clearly visible (Kartush et al. 1985). The stimulator can be a simple monopolar probe or an electrified instrument as has been advocated for years by Kartush (1989) and Silverstein (1991). By electrifying the instruments that surgeons already use, surgeons are more likely to actively (versus passively) monitor during critical dissection therein not only continuously mapping the location of the nerve but also re-assessing its integrity after potentially injurious maneuvers. It is possible for the surgeon and monitoring teams to electrify their own instruments but these should be a) insulated to prevent current from shunting away into adjacent tissue or fluid, and b) securely connected to the stimulator cord to prevent inadvertent detachment. The insulated
Kartush Stimulating Instruments (KSI) (Magstim Neurosign® Surgical, Carmarthenshire, Wales, UK) are disposable tools with which the surgeon can dissect and stimulate simultaneously (Figure 11). They are available in the form of both: a) micro-instruments and b) a fine stimulating hemostat optimized for thyroid and parotid surgery. (Reprinted with permission from Jack M. Kartush, M.D.)

FIG. 11. Kartush Stimulating Instruments (KSI) (Magstim Neurosign® Surgical, Carmarthenshire, Wales, UK) are disposable tools with which the surgeon can dissect and stimulate simultaneously. They are available in the form of both: a) micro-instruments and b) a fine stimulating hemostat optimized for thyroid and parotid surgery. (Reprinted with permission from Jack M. Kartush, M.D.)

Kartush Stimulating Instruments (KSI) (Magstim Neurosign® Surgical, Carmarthenshire, Wales, UK) are disposable tools with which the surgeon can dissect and stimulate simultaneously (Figure 11).

The RLN and SLN nerves may respond to current intensities as low 0.3 mA – but when first mapping the location of the nerve, it is best to start at approximately 2.0 mA to assure an initial response before rapidly titrating the current level down as soon as the nerve has been identified. The stim level chosen at any given time is dependent upon distance from the nerve as well as the surrounding tissue. Like using a minesweeper, once the general location of the nerve is identified, progressively lowering of the stim level allows enhanced spatial resolution. Mapping, for example, with 3.0 mA when stimulating through a centimeter of soft tissue will pose no risk to the nerve. Conversely, an unjustified fear of “excessive stimulation” which might prompt a surgeon to only map at 0.5 mA markedly increases the chance of false negative errors.

Cautery should be avoided adjacent to nerves. When necessary, bipolar cautery is preferable to monopolar cautery due to its more focused electrical and thermal effects. At other times, alternative hemostatic options may be preferred including pressure and topical adrenaline or thrombin. Whenever a risky surgical maneuver has
been performed adjacent to a nerve, it is prudent to re-stimulate the nerve proximal to this area to assure the nerve has maintained neural integrity. Deferring neural integrity testing until the very end of surgery may prove that the nerve has been compromised – but by this time, the surgeon may no longer be aware of which maneuver, and at which location, the nerve may have been injured.

Brief, intermittent RLN and vagal nerve stimulation are well tolerated without any reported adverse effects (laryngeal, cardiovascular, or respiratory) when performed with commonly used pulsed stimulation below 4.0 mA (typically 1.5 mA). The quality of the recorded signal should be confirmed at the beginning of each procedure by obtaining a “baseline” EMG recording from vagal nerve stimulation and then from the RLN in the tracheo-esophageal groove. Such proximal baseline testing is essential to confirming proper system set up as well as the absence of complicating anesthetic factors (neuromuscular blockade or transient lidocaine-induced nerve paralysis). Furthermore, the amplitude of these initial responses can be used as a baseline to determine if later responses have deteriorated (increased stimulation needed for threshold or complete loss of signal).

The stimulus intensity should be titrated – i.e., an initial high stimulus level (e.g., 2.0 to 3.0 mA) can be helpful for initial nerve mapping but once the nerve is identified, lower levels of stimulation allow greater precision. The RLN typically has a threshold of approximately 0.3 to 0.5 mA. It is best, however, to stimulate at higher intensities because even small amounts of blood and soft tissue will cause enough current shunting that a false negative error may occur. Consequently, once the RLN is identified, stimulus levels of 1.0 to 2.0 mA are commonly used.

In addition to baseline proximal stimulation, the surgeon should map distally to exclude anomalies such as an arborized nerve or a “non-recurrent” recurrent laryngeal nerve. As discussed above, it is important to realize that an arborized RLN typically will have its motor division anteriorly rather than in the expected posterior trachea-esophageal location. Under these circumstances, electrical stimulation is critical in assisting the surgeon in recognizing these anomalies.

Continuous Vagal Evoked EMG

As noted, a stretched or bluntly traumatized recurrent laryngeal nerve is much less likely to display mechanically evoked EMG potentials compared to the proximal facial nerve. Consequently, frequent electrical stimulation is essential in maximizing the benefits of IONM during thyroid surgery. In addition, researchers have been exploring the possible benefits of continuous vagal stimulation while monitoring the ongoing vocal fold EMG. This is simply a variant of the long established principle behind somatosensory evoked potentials where stimulation of a peripheral sensory nerve evokes a cortical evoked response. Similarly, the monitorist obtains a baseline
response and looks for changes in amplitude and latency that may signal neural trauma.

Recently, stimulating electrodes have been developed which remain in contact with the vagus nerve in the carotid sheath throughout the procedure (Figure 12). A repetitive stimulus triggers an ongoing EMG response. Changes of the compound muscle action potential amplitude and latency can be followed (Ulmer et al. 2008, Schneider et al. 2009). Research is underway in many centers to determine if this technique may provide greater sensitivity and thus an earlier warning when the RLN has been traumatized directly or by de-vascularization. Numerous electrodes have been suggested to wrap around the vagus nerve in an attempt to assure that the stimulating electrode remains secure whilst the surgeon’s attention is elsewhere. Users should use great caution to secure these devices, however, because of the possibility of vagal nerve injury or even avulsion should the electrode wire become inadvertently pulled away. Furthermore, surgical dissection to expose the vagus nerve requires dissection of the carotid artery and jugular vein – while the risk of hemorrhage is very low in experienced hands, it should be kept clearly in mind. Consequently, we are evaluating alternate means of reliable vagal stimulation while ameliorating the risks of carotid sheath dissection.

We speculate that this technique of Continuous Vagal Evoked EMG (CV-EMG) may in fact have significant advantage if applied to RLN monitoring when used during cervical spine surgery (e.g., anterior cervical disectomy and fusion [ACDF]). Because of the aforementioned unreliability of detecting RLN trauma via mechanically evoked potentials, typical RLN techniques have not proved very effective in monitoring during ACDF (Dimopoulos et al. 2003). Under these circumstances, the greatest concern is typically the persistent pressure applied to the RLN between the surgical retractor and the endotracheal cuff. If this indirect trauma can be better
detected by CV-EMG, the operative team can be alerted to either relax the retractors or temporarily deflate the endotracheal cuff to minimize RLN ischemia.

**Direct or Indirect Nerve Injury**

During electrocautery, most dedicated nerve monitors temporarily deactivate the loudspeaker and the EMG oscilloscope. This is an important feature that prevents the surgeon from being startled by a raucous blast of artifact coming through the loudspeakers every time cautery is used. This feature is rarely available in multimodality monitoring equipment that, as a workaround, requires great attentiveness by the monitorist to “ride the volume knob”, i.e., to manually turn the volume up and down as needed.

When automatic muting is engaged, the monitor cannot detect nerve injury if it occurs from direct cautery or thermal spread. Even complete electrosurgical ablation of the nerve would not elicit a signal and, therefore, it is important to use frequent electrical stimulation to confirm functionality after cautery adjacent to a nerve. In contrast, gradual thermal injury in the cerebellopontine angle from adjacent laser use can lead to a slow increase of the baseline. Electrocautery should never be used simultaneously with the nerve stimulator because current can travel back through the stimulator and into the monitoring device. If cautery is required during the dissection, the use of bipolar cautery or the use of modern alternatives such as the Harmonic scalpel (Ethicon Endo-Surgery, Inc., Cincinnati, Ohio, USA) may limit thermal spread.

**Pitfalls: False Positive and Negative Errors**

To minimize the risk of injury the surgeon and monitorist must be aware of interpretive errors. Besides a true positive response, where after nerve stimulation an expected EMG response is triggered, false positive and negative errors must be distinguished. Clear understanding and use of the correct terminology of false positive and negative errors in scientific publications is necessary. In the current literature these errors are described with various terms. It is crucial to use proper definitions to avoid confusion (Chan and Lo 2006).

A **true positive response** means that the nerve stimulator elicits a muscle response as a result of stimulation of its innervating nerve. A positive response should always be confirmed at the beginning of the case (i.e., baseline testing) prior to close neural dissection.

A **false positive response** is an EMG signal that was not triggered by the target nerve stimulation but caused by stimulating either a structure in proximity to the nerve (leading to cross activation) or direct muscle stimulation. A false positive error at the larynx may occur, for example, when the pharyngeal constrictors are inadvertently stimulated; the large muscle response may be carried to the laryngeal surface...
electrodes and misinterpreted as an RLN response. This error appears to be increased if the endotracheal tube has been inserted deep into the trachea as well as when high levels of stimulation are used that create volume conducted responses or artifact.

An increased EMG baseline (i.e., trains of EMG activity) could represent neural trauma – but it is important to exclude other causes especially by correlating the responses to the ongoing surgical events. For example, in neurotologic surgery at the cerebellopontine angle, cool irrigation often results in trains of EMG activity that do not represent trauma. During RLN monitoring, periodic fluctuations of the baseline may in fact indicate a cuff leak (e.g., from 15 to 80 mV), particularly when there is a corresponding EMG response synchronized with ventilation. In contrast, if there is a large, irregular baseline shift, it may indicate that the patient’s anesthesia may be “light” and the patient’s vocal cords are contracting voluntarily. These causes of EMG baseline change need to be differentiated from mechanically evoked EMG due to neural trauma.

A false negative response has no EMG response despite stimulation of the target nerve. If muscle twitches along the larynx can be palpated when stimulating the vagus or RLN in the absence of an EMG response, a technical problem should be assumed. There appears to be an approximately 15% incidence of false negative responses with commonly used surface laryngeal electrodes. Randolph et al. (2004) have noted that suspected false-negative errors can be cross-checked by directly palpating the laryngeal twitch response in the tracheoesophageal junction. This false negative error should lead to a check of the entire system.

If monitor and probes appear to be functional, the impedance is suitably low and electrode position appears correct, an improper “stimulus ignore period” may be the cause (Figure 13). This intentional silencing of the monitor’s loudspeaker prevents the surgeon from hearing responses created by the stimulus artifact. It should be understood that this feature is similar to, but distinct from, automatic muting during cautery. However, the duration of intentional muting needs to be set appropriate to the anticipated conduction time based on the distance between where the nerve is stimulated and the muscle that is being recorded. Unpublished research by the senior author (JMK) in 1982 led to establishing the factory setting of the original NIM® monitor’s stimulus ignore period at 3.1 msec for monitoring the facial nerve during otologic surgery. However, in contrast to the facial nerve, given that the markedly shorter nerve length of the RLN is associated with a shorter conduction time, many users were not aware that the stimulus ignore period needs to be shortened for thyroid surgery (Kartush and Lee 2013). To prevent inadvertent filtering of a response rather than just stimulus artifacts during thyroid or parathyroid surgery, the stimulation suppression filter should be lowered from the default duration of 3.1 msec (e.g., otologic procedures) to approximately 2.35 msec. Each device and each situation may require different values that each team should assess and establish. Note, too, that the site of stimulation is another important factor: stimulating in
the trachea-esophageal groove will have a longer conduction time than stimulating distally at Berry’s ligament. Shortening the ignore period further can sometimes be helpful in avoiding inadvertent silencing of true responses especially for very distal stimulation – but too short of an ignore period will allow the stimulus artifact to be heard which can confuse or distract the surgeon and monitorist.

Anesthetic causes should also be excluded when troubleshooting monitoring. Avoiding long acting muscle relaxants typically allows normal mechanical and electrical evoked EMG recording – however, in some rare individuals with atypical pseudocholinesterase activity, they may have very prolonged muscle paralysis despite induction with short acting muscle relaxants e.g., succinylcholine (Bojanic et al.

FIG. 13. Stimulation of the recurrent laryngeal nerve (RLN) nerve close to the larynx (e.g., Berry’s ligament) may result in a response with a very brief latency, for example, less than 3 msec. Stimulus filters are helpful in ignoring stimulus artifact but if not used judiciously, can result in masking of both stimulus and response, causing a false-negative error. In this case, the response peak occurred at 1.75 msec but was detected because the technologist shortened the stimulus filter’s time window from the factory default of 3.1 msec. (Reprinted with permission from Jack M. Kartush, M.D.) L-Voc – left vocal cord; R-Voc – right vocal cord.
A normal response to RLN stimulation during early baseline testing confirms appropriate nerve and muscle function. When this fails, troubleshooting should include a train of four (TOF) peripheral nerve stimulation test to determine if neuromuscular blockade has in fact been reversed. In thyroid surgery, lidocaine local anesthetics are rarely infiltrated near the RLN. However, during ear and parotid surgery, clinicians must be aware that lidocaine inadvertently affecting the facial nerve (e.g., through a tympanic membrane perforation), may temporarily paralyze the nerve rendering monitoring useless. In this instance, the false negative error occurs not due to failure of the muscles to contract but failure of the nerve to depolarize secondary to temporary chemical paralysis.

Increased thresholds to stimulation may be seen after micro-trauma, the presence of overlying tissue, liquids in the operative field, desiccation, or a malpositioned electrode within the larynx.

Assessment of the RLN’s integrity at the end of the procedure is best made by stimulating the vagus nerve or the most proximal location of the RLN in the tracheoesophageal groove. Should nerve injury be suspected, it is critical that the epicenter of neural trauma be localized by stimulating both proximal and distal to the lesion. Stimulating only distal to the site of a fresh injury will evoke a normal response – and if not cross-correlated to proximal stimulation (which would show loss of signal or a reduced response) will result in a false positive error.

As an illustration in thyroid surgery, if the nerve is injured in the mid tracheoesophageal groove, an assessment of nerve integrity requires stimulation at either the vagus nerve or the RLN in the proximal trachea-esophageal groove, not just distally at Berry’s ligament.

A recurrent nerve anomaly such as a bifurcation could lead clinicians to erroneously conclude that monitoring failed. For example, consider a bifurcated nerve wherein the motor fibers are typically in only the anterior branch. If the surgeon only stimulates the posterior branch in the expected location within the tracheal groove and has no response, they will mistakenly conclude that the RLN has been injured. When the patient awakes with normal RLN function, they will now mistakenly conclude that monitoring failed, whereas in fact, the anomaly led to user error. There are two important lessons to learn here. The first is that not every apparent monitoring failure is a failure of the recording electrodes or device – user error must also be considered. The second is that while anomalies may, by definition, be infrequent or rare, the surgeon and monitoring team must anticipate these by establishing a consistent protocol that reduces the chance that they may become ensnared in such a pitfall. In this instance, the clinical correlate is that while proximal stimulation of RLN and/or vagus is indeed critical, it is also essential that stimulation be used from proximal to distal along typical and atypical RLN paths to determine the nerve’s location as well as exclude anomalous anatomy.
Table 2. Kartush laryngeal nerves monitoring protocol.

Kartush Laryngeal Nerves Monitoring Protocol

<table>
<thead>
<tr>
<th>Steps</th>
<th>Check Box</th>
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| 1     | Consult with anesthesia:  
|       | • Avoid long acting muscle relaxants & lidocaine on endotracheal tube  
|       | • Proper placement of Laryngeal Electrodes |
| 2     | Demonstrate absence of neuromuscular blockade:  
|       | • Train of four  
|       | • RLN/SLN/Vagal nerve stimulation within the operative field |
| 3     | Assure the monitor’s loudspeaker is set at a volume sufficient to be heard over the operating room’s ambient noise |
| 4     | Check electrode impedance & Stim Ignore setting |
| 5     | Check stimulus current flow through soft tissue |
| 6     | Obtain baseline RLN/SLN/Vagal nerve response to distal stim |
| 7     | Electrically map the location of target nerves within the operative field  
|       | • RLN/SLN/Vagal nerve stimulation  
|       | • Selectively use monopolar, bipolar or Stimulating Instruments  
|       | • Titrate stim current based on nerve location and surrounding tissue (cartilage, soft tissue, blood) |
| 8     | Map location of SLN. Record response via LE or cricothyroid electrode |
| 9     | Consider monitoring continuous vagal evoked laryngeal responses  
|       | • Establish repetitive stimulation of the Vagal nerve  
|       | • Monitor the ongoing EMG responses for amplitude reduction and latency increases |
| 10    | Obtain a final proximal nerve response to stim prior to closure  
|       | • Vagus  
|       | • Proximal RLN |
| 11    | Generate a brief written report (can be within Operative Report):  
|       | • Confirm adherence to protocol  
|       | • Highlight key events |

GUIDELINES AND CHECKLISTS

Over the last century, physicians have been reluctant to accept written clinical practice guidelines believing that they would result in “cookbook medicine”. These were believed to take away the ‘art and science’ of Medicine and lead to fodder for plaintiff’s lawyers. However, the last two decades have shown a significant cultural shift. Sparked by reports of the Institute of Medicine and the World Health Organization, more than a thousand clinical guidelines have been published to improve patient safety. The airline industry has long learned that even experienced pilots benefit from protocols and checklists due to the complexity of their work environment – and the
dire consequences of errors. Consequently, the senior author (JMK) has long promulgated a Facial Nerve Monitoring Protocol with an associated Checklist to aid in following the protocol. An analogous checklist has been created to assist clinicians in monitoring laryngeal nerves (Table 2).

All checklists represent a balance between complexity versus simplicity. The latter tends toward being comprehensive but difficult to use while the latter is incomplete but much more likely to be used in clinical practice. The Laryngeal Nerves Monitoring Protocol is not intended to be all encompassing but a “pre-flight” and “intra-flight” checklist intended for the knowledgeable practitioner. It can also serve as the basis for a hospital, department, or monitoring service company’s Policy and Procedure Manual. Furthermore, for self-monitoring surgeons who in the past have overlooked generating any type of IONM written report, such a checklist can serve not only as a reminder to perform all the recommended steps but also as a rapid means of establishing documentation of the monitoring procedure.

**SUMMARY**

Monitoring the laryngeal nerves during surgery can assist in nerve location and in reducing the incidence of neural trauma. All members of the surgical monitoring team must have training specific to laryngeal recording to maximize its benefit and minimize pitfalls. Active monitoring entails frequent use of electric stimulation rather than passively awaiting responses after trauma has occurred. Stimulating dissection instruments are a useful adjunct to detect the location of nerves prior to a mechanical insult. As improved methods are adopted, the efficacy of laryngeal nerve monitoring will increase. For additional information, refer to the monitoring course “IONM 109: Skull Base Surgery and Cranial Nerve Monitoring” at http://aset-edu.org/.

**DISCLOSURES**

Dr. Kartush was principal designer of the NIM® monitor, past consultant to Medtronic, founding president of the American Society of Neurophysiologic Monitoring, and is a consultant to Magstim Neurosign® Surgical, a manufacturer of intraoperative monitoring products. Dr. Naumann indicates no disclosures.

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Use of Somatosensory Evoked Potentials to Detect and Prevent Impending Brachial Plexus Injury during Surgical Positioning for the Treatment of Supratentorial Pathologies

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ABSTRACT. Somatosensory evoked potentials (SSEPs) are widely utilized for the intraoperative detection and prevention of nerve conduction injuries. Their use in identifying position-related injuries to the brachial plexus in patients undergoing supine craniotomies for the treatment of supratentorial pathology is not well documented. This case series describes three instances of unilateral upper extremity SSEP changes in patients positioned for supine craniotomies. In all three cases SSEP responses improved after repositioning. None of the patients exhibited new neurological deficits post-operatively. This case series highlights the importance of vigilant monitoring in the period after final positioning and demonstrates the usefulness of SSEPs as a tool to aid in the early detection and prevention of impending position-related nerve injury.

KEY WORDS. Amygdalohippocampectomy, craniotomy, diabetes mellitus, hypertension, intraoperative neurophysiological monitoring, obesity, positional brachial plexus injury, somatosensory evoked potentials, supratentorial lesion.
INTRODUCTION

Somatosensory evoked potential (SSEP) monitoring has become a standard of care for the detection and prevention of iatrogenic injury to the nervous system. The early identification of significant SSEP changes has been shown to be effective in reducing the incidence of post-operative neurological deficits (Nuwer et al. 1995). The criteria for significant changes in SSEP waveforms are well established and can be identified by a 50% drop in amplitude or a 10% increase in latency from baseline values. Significant changes can occur as a result of surgical manipulation, changes in anesthesia, hypoperfusion, or positioning.

Neurologic injury related to positioning is a well-documented perioperative complication accounting for 16% of all adverse anesthetic outcomes resulting in major injury legal claims (Cheney et al. 1999). Injuries to the brachial plexus make up 19 to 34% of these claims placing them as one of the most common injuries reported (Cheney et al. 1999, Fritzlen et al. 2003). SSEP monitoring has been documented to be an effective indicator of impending, position-related nerve injury during anterior cervical spine procedures (Schwartz et al. 2006), prone lumbar decompression/fusions (Kamel et al. 2006), prone scoliosis surgery (Labrom et al. 2005), and supine transaxillary approach thyroidectomies (Davis et al. 2011). Intraoperative SSEP changes have also been recently described after supine positioning for the surgical treatment of acoustic neuromas and other infratentorial lesions (Anastasian et al. 2009, Jellish et al. 2013).

The documented evidence of brachial plexus injury during positioning for the surgical treatment of supratentorial pathology is more limited. The purpose of this paper is to present a case series illustrating the benefits of SSEPs in the early detection of potential supine position-related brachial plexus injury in three patients undergoing craniotomy for the treatment of supratentorial pathology.

CASE REPORT 1

Patient 1 is a 42-year-old male with a history of intractable seizures of gelastic semiology. His workup, which included an epilepsy monitoring unit (EMU) admission, positron emission tomography (PET) scans, and depth/subdural electrode monitoring, demonstrated the seizures primarily arising from the right temporal cortex. Other pertinent medical history included hypertension (HTN), diabetes mellitus (DM), and obesity with a body mass index (BMI) of 41 kg/m². He was consented for a right cortical amygdalohippocampectomy.

The patient was brought to the operating room where general anesthesia was administered with an induction dose of propofol and maintained in a steady state using an expired concentration of 0.8% sevoflurane as well as intravenous (IV) infusions of remifentanil and dexmedetomidine. He was positioned supine with a roll...
placed under the right shoulder and thorax. His head was turned approximately 45 degrees to the left and secured in a Mayfield 3 point pin system. All pressure points were padded. Taping was added to secure the patient to the bed and to aid in the positioning of the right shoulder.

After positioning and registration of the neuro-navigation system, baseline SSEPs were obtained. Stimulation sites included bilateral ulnar nerves (intensity 20 mA, stim rate 4.1 Hz, phase duration 0.3 msec) and bilateral posterior tibial nerves (intensity 40 mA, stim rate 4.1 Hz, phase duration 0.3 msec). Stainless steel recording electrodes were placed at A1, C3’, C4’, Cz, and Fz according to the International 10–20 System. Cortical recording montages included C3’-C4’, C3’-Fz, and C4’-Fz montages for the upper limb SSEPs. Lower limb SSEPs were recorded using C3’-C4’ and Cz-Fz montages. An A1-Fz channel was utilized for monitoring of subcortical potentials for both upper and lower SSEPs. Cortical waveforms were identifiable from all limbs and these were established as baselines (Figure 1A). Subcortical potentials were identified and reproducible from the upper limbs only.

Within eight minutes of baselines being established, and prior to the surgical incision, the right ulnar nerve SSEP responses were abolished (Figure 1B). Left ulnar

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**FIG. 1.** Ulnar nerve somatosensory evoked potential (SSEP) waveforms recorded during a craniotomy for amygdalohippocampectomy. **A.** Baseline potentials established post-positioning. **B.** Pre-incision loss of right upper extremity (UE) waveforms including cortical and subcortical control potentials. **C.** Recovery of right UE SSEPs after repositioning of the arm. uV – microvolts; div – division; msec – millisecond.
nerve SSEP and bilateral posterior tibial nerve SSEP responses remained unchanged. The anesthesia team was consulted, and they confirmed that no significant changes to the anesthetic regime had occurred. The attending surgeon was immediately notified and the patient’s positioning was reassessed under the drapes. The positioning of the right arm and neck appeared adequate. As a precautionary measure, towels were placed under the right shoulder to help support the arm and relieve any possible stretch being applied to the brachial plexus on that side. Within five minutes of this additional support being provided the right ulnar nerve SSEPs began to recover. The SSEPs recovered in full to baseline within 20 minutes (Figure 1C) and remained stable throughout the remainder of the procedure. The mean arterial pressure (MAP) remained stable at 70 to 80 mmHg throughout the duration of SSEP changes.

Postoperatively the patient had no subjective complaints of upper extremity neurologic injury. He remained afebrile and seizure-free throughout the rest of his hospital admission and was discharged to home on post-op day four. At his two week follow-up he continued to be seizure-free and had no detectable deficits in his right upper extremity.

**CASE REPORT 2**

Patient 2 is a 52-year-old female with a history of medically refractory right temporal lobe epilepsy. Her workup included an EMU admission and demonstrated a seizure focus emanating from the right temporal lobe. Her past medical history was significant for obesity with a BMI of 59.6 kg/m². Her documented medical history did not include DM or HTN. She was consented for a right cortical amygdalohippocampectomy.

The patient was brought to the operating room where general anesthesia was administered using an induction dose of propofol, and was subsequently maintained using an expired concentration of 1.2% sevoflurane as well as IV infusions of remifentanil and dexmedetomidine. The patient was positioned supine with a towel roll placed under the right shoulder and thorax. Her head was positioned into approximately 45 degrees of extension and rotated approximately 30 degrees to the left side, secured in a Mayfield 3 point fixation system. All pressure points were padded. Taping was added to secure the patient to the bed and to aid in the positioning of the right shoulder.

After positioning and registration of the neuro-navigation system all applicable recording electrodes were placed. Seventeen minutes after final positioning baseline ulnar nerve and posterior tibial nerve SSEPs were established as described above. Waveforms were identifiable from all limbs with the exception of both cortical and subcortical responses from the right upper extremity (Figure 2). All technical and anesthesia related factors were quickly ruled out and the certified registered nurse anesthetist as well as the attending surgeon were notified.
The patient’s positioning was reassessed. The patient’s right shoulder was felt to be inadequately supported by the shoulder roll, allowing for excessive extension and lateral rotation of that joint. In addition, tape which had been placed across the right acromioclavicular joint to aid in shoulder girdle depression was assessed to be too restrictive. The securing tape was removed and the arm was repositioned to a more anatomically-correct position. Towels were placed under the right shoulder to help maintain the alignment. Within six minutes of the repositioning cortical waveforms from stimulation of the right upper extremity were recorded. Over the course of the next hour the right upper extremity amplitudes became symmetrical compared to those from the left upper extremity, and remained so for the remainder of the case (Figure 3). The MAP remained stable at 80 to 90 mmHg throughout the duration of SSEP changes and positioning interventions.

Postoperatively the patient had no subjective complaints of upper extremity neurological injury. She remained afebrile and seizure-free throughout the remainder of
her hospital admission and was discharged to home on post-op day three. At her two week follow-up she had no detectable deficits in the right upper extremity.

CASE REPORT 3

Patient 3 is a 63-year-old female with a past medical history of HTN, obesity (BMI of 48.4 kg/m²), and stroke who experienced a sudden onset headache and loss of vision in the left eye. An angiogram demonstrated a ruptured aneurysm at the A1/A2 junction on the left. The patient was consented for surgical treatment consisting of craniotomy and clipping of this complex aneurysm and was brought to the operating room as an urgent case. Following a propofol induction, general anesthesia was maintained using an expired concentration of 0.8% sevoflurane as well as IV infusions of propofol, remifentanil, and phenylephrine. The patient was placed supine
with a towel roll under the left shoulder and thorax. Her head was turned approximately thirty degrees to the right and secured in a Mayfield 3 point fixation head holder. All pressure points were padded, and silk tape was used to secure the patient to the bed. Tape was not used to maintain shoulder girdle depression in this case.

After the patient was positioned and all applicable neuromonitoring leads were placed, initial SSEPs were obtained. Clear waveforms were identified from all extremities, and these were established as baselines (Figure 4A). Approximately 20 minutes after these baselines were established, there was a complete loss of both cortical and subcortical potentials from the left upper extremity (Figure 4B). Right upper extremity and bilateral lower extremity SSEP responses remained at baseline. The surgeon and certified registered nurse anesthetist were alerted. The anesthesia team confirmed that no anesthetic changes had occurred to explain this loss, so the

![SSEP Waveforms](image-url)

**FIG. 4.** Ulnar nerve somatosensory evoked potential (SSEP) waveforms recorded during a craniotomy for aneurysm clipping. **A.** Baseline potentials established post-positioning. **B.** Loss of both cortical and subcortical left upper extremity (UE) waveforms. **C.** Recovery of left UE SSEPs after repositioning of the arm. uV – microvolts; div – division; msec – millisecond.
left arm position was assessed. The arm appeared to be adequately supported. As a precautionary measure several towels were inserted to more fully support the shoulder. There was a clear improvement of the SSEPs from the left arm as soon as this support was provided (Figure 4C).

The signals remained satisfactory for approximately 18 minutes until again the waveform evoked from the left upper extremity was noted to be completely abolished. The attending surgeon was again alerted, and the attending anesthesiologist was asked to assess the patient’s position under the drapes. Again, there appeared to be no positioning issues with the patient’s arm. In light of the ongoing SSEP changes, however, the anesthesiologist determined that the shoulder needed to be better supported. Egg crate foam and towels were inserted to both elevate and internally rotate the shoulder. Following this intervention, the left upper extremity SSEPs were noted to improve and fully return to baseline over the course of the next thirty minutes. The signals remained satisfactory throughout the remainder of the procedure (Figure 5). The MAP remained stable at 75 to 85 mmHg throughout the duration of SSEP changes and positioning interventions.

The patient awoke with no new neurological deficits in the left arm. Her hospital course was complicated by ongoing vasospasm in the left cerebral vasculature. She remained febrile for an extended period and was discharged on post-op day 14 to acute rehab. At the time of her discharge the patient reported only baseline weakness in the left upper extremity that had been present pre-operatively due to a remote stroke history.

**DISCUSSION**

Intraoperative nerve injury can be attributed to a number of different mechanisms including compression, ischemia, lacerations, and stretch among others. It is the normal anatomy of the brachial plexus and its surrounding structures that makes the plexus particularly vulnerable to stretch injury. The brachial plexus is attached to firm fixation points both proximally at the pre-vertebral fascia and distally at the axillary fascia (Uribe et al. 2010). Any position which lengthens the distance between the two endpoints and forces the brachial plexus past its maximum physiological length (MPL) may be expected to increase the risk for stretch related injury. Tensile, electrophysiologic examinations of nerve tissue in animal models suggest longitudinal strains of only 5 to 10% past MPL are enough to produce significant conduction deficits (Rickett et al. 2011). Deficits from strains of less than 10% past MPL have been associated with complete acute recovery, while strains that exceed this threshold are more likely to be associated with only partial conduction recovery (Li et al. 2007).

Conduction deficits produced in minimal strain situations as one might see in instances of mild stretch appear to occur through ischemic processes rather than
physical disruption of neural fibers. Blood flow in animal peripheral nerves is reduced by 70% when an 8.8% longitudinal strain is applied (Driscoll et al. 2002). At this level of strain full flow is restored when the neural tissue is relaxed. A longitudinal strain of 15% has been shown to completely arrest interneural blood flow with minimal recovery of flow evident upon release of the tension (Ogata et al. 1986). Patient positioning which places the anatomically-fixated brachial plexus past its MPL challenges the ability of vascular tissue in the endoneurium to adapt to the stresses to which it is being subjected.

Supine positioning minimizes strain on the brachial plexus. A classic supine positioning technique for cervical spine cases, for example, includes the head and neck slightly extended, a shoulder roll placed in midline below the neck, and shoulders taped down to maximize radiographic views. In a study of 1000 consecutive spine cases only 2 out of 110 subjects (1.8%) positioned in this manner experienced SSEP
changes due to the positioning (Kamel et al. 2006). Similarly, a retrospective review of over 3,800 anterior cervical discectomies showed intraoperative evidence of evolving positional injury in only 69 cases (1.8%) (Schwartz et al. 2006). Both of these studies demonstrate the relative safety of the classic supine position.

Craniotomies for supratentorial lesions may at times require modifications to the standard supine positioning technique in order to optimize the surgical access. All of the patients in this series were positioned supine with a roll under the shoulder and thorax to “bump up” the patient on the operative side. Their heads were turned 30 to 45 degrees of rotation contralateral to the surgical side, and the arms were tucked to the side. Shoulder taping with traction was applied across the acromioclavicular joint to maximize surgical access in both amygdalohippocampectomies, but not for the aneurysm clipping.

In all three cases, inspection of the patient positioning after the identification of SSEP changes demonstrated that the shoulder was poorly supported by the towel roll, allowing for shoulder extension, external rotation, and retraction of the scapula away from the fixated and turned head (Figure 6). Intervention consisted of inserting additional supporting materials to minimize shoulder extension and external rotation. In the two patients undergoing amygdalohippocampectomies the tape depressing the shoulder girdle was also removed. These actions served to restore the proper anatomical relationship between the cervical spine and the shoulder complex (Figure 7). Improvement in the SSEPs after these interventions established a cause and effect relationship between malpositioning and impending neurapraxia in all three cases.

FIG. 6. Sub-optimal positioning allowing the shoulder to extend and externally rotate away from a fixed head positioned in contralateral rotation/lateral-flexion. This position increases the risk of iatrogenic neuropathy. The arrow demonstrates the directions of longitudinal strain across the brachial plexus.
The combined joint positions of contralateral head rotation/lateral-flexion and shoulder external rotation with extension/scapular retraction have the potential to place a longitudinal strain on the brachial plexus (Figure 6). In a study of awake, healthy subjects, the addition of cervical contralateral lateral-flexion to subjects who were already positioned with the shoulder externally rotated and with forces applied over the acromioclavicular joint to depress and fixate the shoulder girdle was found to significantly increase subjective complaints of UE pain and paresthesia (Coppieters et al. 2002). In one study impending brachial plexopathy identified through neurophysiological changes was most commonly encountered when the shoulder was taped and traction was applied to depress the shoulder girdle (Schwartz et al 2006).

Pre-existing medical co-morbidities may place certain patients at increased risk of sustaining an intraoperative peripheral nerve injury. A retrospective review of over 380,000 cases established a significant association between hypertension (HTN) and intraoperative neuropathy with HTN being present as co-morbidity in 34% of all cases with injury (Welch et al. 2009). HTN can reduce nerve blood flow by up to 25%, and has been shown to cause neural ischemia, conduction slowing, and axonal atrophy in animal models (Gregory et al. 2012). The results of another study found smaller sensory action potentials amplitudes without corresponding latency changes in human subjects with hypertension, suggesting that functional changes to the endoneural microcirculation may lead to hypoxic axonal neuropathy (Edwards et al. 2008).
Patients with diabetes mellitus (DM) are 2.4 times more likely to develop a nerve injury during surgery than patients without the co-morbidity (Welch et al. 2009). The pathophysiology of DM, while multifaceted, does include structural changes to endoneural vasculature (Sugimoto et al. 2010). Periods of even brief ischemia that were insufficient to cause nerve fiber damage in normal neural tissue have been shown to cause demyelination and axonal degeneration in diabetic animal models (Nukada et al. 2011). Both sensory nerve action potential and compound muscle action potential conduction velocities have been shown to slow significantly in diabetic nerve compared to healthy nerves when both are subjected to mild ischemic stresses (Wang et al. 2004).

Patients with underlying clinical or subclinical peripheral neuropathies related to HTN and DM already have neural tissue predisposed to blood flow impairment. These patients in the operating room with a malpositioned upper extremity may show a decreased tolerance for longitudinal strain across the brachial plexus since the patency of blood vessels within the nerve sheath is further impaired during longitudinal stretching of nerve tissue. Only one of the patients in this series carried diagnoses of both HTN and DM. Two out of three patients were hypertensive, although it should be noted that the patient who did not have a diagnosis of HTN was clinically hypertensive during much of her hospital admission.

Large body habitus has also been identified as a potential predisposition to position-related iatrogenic injury. A study of 1506 patients found a significant difference in the mean body weight of patients who developed intraoperative neuropathy compared to those who did not (Warner et al. 1999). A more recent prospective cohort observational study of 65 patients positioned with contralateral lateral rotation of the head combined with shoulder girdle depression demonstrated a significant effect of body mass index (BMI) on the incidence of brachial plexus injury post-operatively (Jellish et al. 2013). All of the patients in this series shared morbid obesity as a significant medial co-morbidity with an average BMI of 49.7 kg/m².

**CONCLUSION**

These cases demonstrate that patients undergoing craniotomy in the supine position can be at risk for brachial plexopathy, especially in the presence of medical co-morbidities such as obesity, diabetes, or hypertension. While a limited case series such as this does not justify the need for routine use of somatosensory evoked potentials during all supine craniotomies, the findings do highlight the importance of vigilant monitoring in the interval after final positioning when SSEPs are already being utilized as an adjunct intraoperative monitoring tool. This case series serves to further demonstrate the usefulness of somatosensory evoked potentials as a tool to aid in the early detection and prevention of impending position-related nerve injury.
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Mesial Temporal Lobe Epilepsy: A Distinct Electroclinical Subtype of Temporal Lobe Epilepsy

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ABSTRACT. Mesial temporal lobe epilepsy is a common subtype of temporal lobe epilepsy. Its most common cause is hippocampal sclerosis, which contributes to its distinct electroclinical phenotype that is seen commonly in the epilepsy monitoring unit setting. The common electrophysiological data show anterior temporal interictal sharp waves as well as rhythmic theta activity in the same localization. While the electrophysiological data can at times be misleading, its stereotyped and characteristic semiology can often allow for accurate diagnosis on its own. As patients with mesial temporal lobe epilepsy often fail medical therapy, surgical therapy can be considered. Early accurate diagnosis in these patients is essential for optimal care.

KEY WORDS. Abdominal aura, automotor seizures, EEG, epilepsy, hippocampus, mesial temporal lobe, semiology.

INTRODUCTION

Epilepsy is a common neurological diagnosis, affecting 70 million people worldwide (Brodie et al. 2012). However, the diagnosis of epilepsy is a heterogenous group consisting of multiple types of both generalized and focal epilepsy. It is vital to correctly classify the epilepsy diagnosis, as the etiology of the epilepsy diagnosis can impact prognosis (Wiebe and Jette 2012). By obtaining a precise history, most pertinently a detailed analysis of the seizure semiology, one can often correctly classify the type of epilepsy nearly 50% of the time. If the history remains unclear, video review of an ictal event in an epilepsy monitoring unit (EMU) can provide a
significant amount of information. If the diagnosis is still uncertain, electroencephalography (EEG) can further increase the correct classification of diagnosis to nearly 80%.

Of all the focal epilepsies, temporal lobe epilepsy is the most common localization (King et al. 1998). Temporal lobe epilepsy comprises a vast array of clinical syndromes, but one specific type, mesial temporal lobe epilepsy (MTLE), remains the single most common indication for epilepsy surgery (Berg 2008). The specific electrophysiological, pathological, and clinical signatures of mesial temporal lobe epilepsy will now be further reviewed.

**NATURAL HISTORY**

Mesial temporal lobe epilepsy has been linked to hippocampal dysfunction (Tatum 2012). Hippocampal sclerosis is a pathological finding of atrophy and gliosis of the hippocampus and has been linked as both a cause of epilepsy as well as the result of ongoing uncontrolled seizures (Theodore et al. 1999). There is evidence that febrile status epilepticus causes acute hippocampal injury as demonstrated by magnetic resonance imaging (Shinnar et al. 2012). Moreover, epidemiological data demonstrate the risk of developing epilepsy in people with prior febrile seizures is 10 times greater as compared to the general population (Neligan et al. 2012). MTLE has an onset at three different ages: 5, 15, and 26, with subsequent studies indicating that a later age of onset was found to be a good prognostic indicator for good seizure outcome (Janszky et al. 2004, Aguglia et al. 2011). It generally takes around nine years for a MTLE patient to become medically refractory (Berg 2008). Therefore, early recognition of MTLE is important, as a randomized trial supports early surgical intervention for those medically refractory patients (Engel et al. 2012).

**NEUROANATOMY AND PERTINENT PATHOLOGY**

The mesial temporal lobe (MTL) comprises the hippocampus, parahippocampus (entorhinal and perihinal cortex), amygdala, and dentate gyrus (Wen et al. 1999). The generic pathological cause of epilepsy in MTLE patients is mesial temporal sclerosis; a combination of gliosis and atrophy of all these structures. The most important area within the MTL is the hippocampus. Therefore, hippocampal sclerosis, the gliosis and atrophy of the hippocampus, is the single most common pathology seen in epilepsy surgery series with a distinct magnetic resonance imaging (MRI) signature, although it can be normal appearing on MRI 30% to 40% of the time. The hippocampus is divided into layers of the Cornu Ammonis (CA1 to CA4). These CA layers are further surrounded by the dentate gyrus, which connects via the parahippocampus to a mixture of temporal and extratemporal cortical association areas (Tatum 2012).
There are several important fascicles connecting the mesial temporal region to other brain areas. These include the connection between the two temporal lobes called the intertemporal portion of the anterior commissure, the connection between the two hippocampi called hippocampal commissure or fornix, the connection between the ipsilateral frontal and temporal lobes called the uncinate fascicle, and lastly diffuse associative fibers (Sindou and Guenot 2003). These connections are what allow seizures that begin in the mesial temporal lobe to spread throughout the brain during secondary generalization.

**CLINICAL FEATURES**

The average MTLE patient presents in the fourth decade of life and has likely failed numerous antiepileptic medications. A history of febrile seizures is common, occurring in about one-third of MTLE patients (Berg 2008). However, when discussing clinical features of MTLE, we are mainly discussing semiology. Semiology is a very important tool in the diagnosis of epilepsy. There are two dominant features of MTLE semiology: the abdominal aura and automotor seizures (Lüders 2008, Blair 2012, Tatum 2012). Some type of aura occurs in 96% of MTLE patients, with abdominal auras being the most frequent (French et al. 1993). Abdominal auras are characterized by a sense of nausea or discomfort in the abdominal area. Sometimes this sensation will rise up into the throat (Lüders 2008). The abdominal aura has excellent localizing value to the mesial temporal region, especially when followed by automotor seizures (Henkel et al. 2002). Other common types of aura include the sense of fear, abnormal olfactory hallucinations, or the feeling of déjà vu (French et al. 1993). Fear in particular is an excellent indicator of MTLE (Maillard et al. 2004). Often times the abdominal aura will lead to the automotor seizure. The semiology of an automotor seizure is defined by stereotyped motor movements such as chewing (as seen in oral automatisms) or repetitive, seemingly purposeful hand movements (such as pill rolling movements or manipulating buttons on a shirt). Consciousness is nearly always affected, although if affecting the non-dominant hemisphere, an unimpaired state of consciousness can be seen clinically (Lüders 2008).

Patients with untreated MTLE (electively done in an epilepsy monitoring unit setting when antiepileptic medications are withdrawn) will often have secondary generalization of the seizures. Careful analysis of the semiology during this phase of the seizure can yield additional localizing and lateralizing information. Dystonic posturing is sustained, forceful unnatural posturing of a limb that reliably lateralizes to the contralateral hemisphere (Bleasel et al. 1997). Similarly, a versive seizure is defined as sustained, unnatural turning of the head and body towards one side. This typically occurs just prior to secondary generalization in MTLE and reliably lateralizes to the contralateral side (Chee et al. 1993). The presence or absence of speech is
important. If speech occurs during the seizure, the seizure focus lateralizes to the nondominant hemisphere (most often the right hemisphere) (Yen et al. 1996). Conversely, postictal aphasia or dysphasia points to involvement of the dominant hemisphere (most often the left hemisphere) (Gabr et al. 1989). The last clonic movement, if unilateral, as well as a unilateral postictal nose wipe lateralizes to the hemisphere ipsilateral to the moving limb (Chee et al. 1993, Hirsch et al. 1998).

The actual tonic and clonic phases often do not add much to the localizing or lateralizing of the seizure focus. However, when the tonic phase occurs asymmetrically and the arms make a sign of 4, this is a localizing sign to the hemisphere contralateral to the extended arm. The “sign of 4” occurs when one arm extends and the other arm is flexed creating a 4 with the arms. In fact, this sign of 4 is often part of the typical motor sequence of a temporal lobe seizure. It begins with the above mentioned automatisms and then evolves into the versive seizure, subsequent asymmetric tonic posturing with sign of 4, followed by the onset of the clonic phase of seizure (Tufenkjian and Lüders 2012).

**ELECTROPHYSIOLOGY**

Mesial temporal lobe epilepsy has both a typical scalp and intracranial electrophysiological signature. To begin, it is important to remember what an epileptiform discharge represents from an electrophysiological perspective. A sharp wave or spike occurs when a large number of neurons’ postsynaptic potentials fire in a synchronous manner that disrupts normal brain activity (Tatum 2012).

**Scalp Interictal EEG**

Typical interictal findings are seen in 96% of patients (Williamson et al. 1993) and consist of spikes or sharp waves that localize to the anterior temporal region (F7/F8) in the International 10–20 System of electrode placement (Figure 1). The F7/F8 electrodes are more anterior than one might expect given the typical hippocampal etiology of MTLE. Therefore, these epileptiform waves most likely represent activation of the parahippocampal tissue (Lüders 2008). Along these lines, true hippocampal epileptiform activity is not seen on scalp EEG due to its small area of activation as intracranial EEG has indicated that approximately 10 cm of cortex is required to generate scalp interictal discharges (Tao et al. 2005).

Placement of basal temporal electrodes (i.e., sphenoidal or FT9/FT10 electrodes) can provide additional localizing information that is useful in both scalp EEG (Figure 2) as well as more advanced modalities such as electrical source imaging (ESI) (Cherian et al. 2012). This is especially true in MTLE with bitemporal epileptiform discharges. Bilateral scalp epileptiform discharges are not uncommon, being
noted in around 40% of the patients (Williamson et al. 1993). However, bitemporal intracranial EEG can often localize the seizure onset zone to just one temporal lobe, making resective therapy a viable therapeutic option (Henry et al. 1999). Sphenoidal electrodes can also increase the accuracy of electrical source imaging (Hamaneh et al. 2011). However, it is important to note that if ESI is to be done, analysis of individual spikes seems to be unreliable, but averaging eight or more spikes can produce more reliable results that localize to within 1 cm of the intracranial generator (Wennberg and Cheyne 2014).

Scalp and Intracranial Ictal EEG

There are three well described ictal patterns in MTLE. Ictal patterns are not typically seen at clinical onset due to the small area (the hippocampus) involved in the seizure onset zone of MTLE (Lüders 2008). The first and most typical scalp ictal pattern consists of a progressive build-up of sinusoidal theta/alpha activity in the temporal (F7/F8) or subtemporal (FT9/FT10) electrodes (Figure 3). When this rhythmic theta activity is evident within 30 seconds of clinical onset, the pattern was found...
to be highly correlated with mesial temporal seizure onset, specifically the hippocampus (Risinger et al. 1989, Ebersole and Pacia 1996, Pacia and Ebersole 1997). Intracranial EEG (iEEG) shows a typical pattern of pre-ictal hippocampal spiking followed by an electrodecremental response before giving way to more sinusoidal 12 to 20 Hz rhythms (Pacia and Ebersole 1997) (Figure 4).

The second ictal pattern for MTLE is characterized by irregular 2 to 5 Hz activity that lateralizes well to the affected hemisphere, but is not typically localizing to a specific electrode (Figure 5). Sometimes this activity will eventually evolve into the more typical sinusoidal alpha/theta activity noted above. While occasionally associated with hippocampal onset on iEEG, this ictal pattern was more often noted with temporal neocortical onset (Ebersole and Pacia 1996, Pacia and Ebersole 1997). The third type of ictal onset is when there is no identifiable ictal activity. Rather, there is diffuse, irregular slowing (Figure 6). Again, this third type was occasionally associated with hippocampal onset on iEEG, but more often was seen in temporal neocortical onset (Ebersole and Pacia 1996, Pacia and Ebersole 1997).

In many cases, careful consideration of the above clinical and electrophysiological factors will remove the need for iEEG recording. However, if an incomplete answer

FIG. 2. Basal temporal Sp1 sharp wave in a longitudinal bipolar montage.
FIG. 3. Type 1 temporal lobe seizure pattern typically associated with mesial temporal lobe epilepsy (MTLE). Note the rhythmic theta maximal at FT9.

FIG. 4. Ictal pattern seen emanating from the left hippocampus on intracranial EEG (iEEG). LH – left hippocampal head; LB – left hippocampal body.
is found from this evaluation, iEEG is vital for determining the seizure onset zone, with the realization of the inherent sampling bias of iEEG. Therefore, one should expect to see iEEG onset before clinical onset if in the seizure onset zone. Indeed, when the iEEG onset precedes the clinical onset by >10 seconds, the probability of postsurgical seizure freedom is increased (Weinand et al. 2001). Both depth electrodes and grid/strip electrodes have been used to good effect in iEEG monitoring.
Intracranial Interictal EEG

Sharp waves or spikes are readily identifiable on iEEG and have similar morphological characteristics as seen on scalp EEG (Figure 7). It is important to remember that the number of epileptiform discharges may be significantly increased on iEEG. This is explained by the fact that in order to see activity on the scalp EEG, approximately 10 cm of synchronous neuronal activity is needed. While spikes and sharp waves are also readily identifiable on intracranial EEG, the ability to record faster frequencies than the 1 to 35 Hz seen with scalp EEG offers a unique opportunity for other interictal activity. This activity is termed high frequency oscillations (HFOs) (Jacobs et al. 2012) (Figure 8). There are two types of HFOs: ripples (80 to 200 Hz) and fast ripples (>250 Hz) (Engel et al. 2009). High frequency oscillations were actually first discovered in hippocampal cells as ripples. Ripples are a normal electrophysiological finding and are thought to represent the process of consolidating plasticity and episodic memory (Le Van Quyen et al. 2008). Conversely, fast ripples appear to be uniquely associated with the epileptogenic zone and correlate with hippocampal atrophy. They are thought to represent the synchrony of abnormally firing neurons (Engel et al. 2009, Bragin et al. 2010). HFOs have been shown to occur in the seizure onset zone and not within areas of seizure propagation and are thus thought to possibly be a biomarker of epileptogenicity (Jacobs et al. 2009, Salami et al. 2014). Perhaps more importantly, the resection of tissue containing HFOs has been correlated with better surgical outcomes (Jacobs et al. 2010).

FIG. 7. Intracranial (iEEG) interictal discharge. Note the accompanying Sp2 sharp wave with diffuse cerebral activation. The second discharge approximately one second later that is more focal in distribution does not have a scalp correlate.
(Fast Ripples visible in spike)

FIG. 8. High frequency oscillations (HFOs) as seen with different EEG parameters. Note the change in waveform morphology as slower frequencies are filtered out. Figure adapted from Jacobs et al. 2012 with permission from Elsevier.
Pseudotemporal Patterns and Benign Variants

The other difficulty seen in the electrographic diagnosis of MTLE is pseudotemporal ictal rhythms. These are EEG seizure patterns that have typical rhythmic theta in the temporal regions as in MTLE, but actually represent spread from a distant epileptogenic site that may not be well localized on EEG. One study reported that 11% of patients with an extratemporal MRI lesion had ictal temporal seizure patterns (Rémi et al. 2011). Pseudotemporal ictal patterns have been reported for nearly all anatomic localizations (i.e., orbitofrontal lobe, parietal lobe, occipital lobe, etc.). Additionally, many different developmental abnormalities including nodular heterotopias and hypothalamic hamartomas can cause ictal temporal seizure patterns. Unfortunately, there does not appear to be a morphological difference between true temporal ictal rhythms and pseudotemporal rhythms (Elwan et al. 2013). Therefore, it is imperative to always match a patient’s clinical semiology with the EEG pattern for the most accurate localization of the epileptogenic zone.

Benign variants can be confused for epileptiform discharges and lead to an inappropriate diagnosis of epilepsy. They occur in approximately 3.4% of individuals (Santoshkumar et al. 2009). These waveforms can either be isolated, as seen in benign sporadic sleep spikes or wicket spikes. They can also occur in prolonged runs, as seen in 14 and 6 Hz positive waves, rhythmic temporal theta of drowsiness, or subclinical rhythmic electrographic discharges (Klass and Westmoreland 1985, Santoshkumar et al. 2009) (Figure 9). Awareness of these different waveforms can minimize the possibility of an inappropriate diagnosis of epilepsy.

TREATMENT AND OUTCOMES

In general, around 68% of epilepsy patients will achieve seizure freedom with medical treatment alone (Brodie et al. 2012). However, patients with hippocampal sclerosis have a markedly lower rate of seizure freedom (11 to 42%) with medical treatment alone (Tatum 2012). A randomized controlled trial of early surgical therapy in patients with medically refractory MTLE showed a seizure freedom rate of around 60% (Engel et al. 2012). While surgery remains the cornerstone for treatment of medically refractory epilepsy, it is important to note that some seemingly medically refractory patients may in fact go into seizure remission following introduction of a new antiepileptic drug, all the more relevant as the number of seizure medications available to clinicians increase (Luciano and Shorvon 2007). However, this observation is intriguingly contested by recent studies demonstrating that remission in a medically refractory patient after change to a new antiepileptic drug may actually be the result of the spontaneous and/or periodic remissions seen in the natural course of epilepsy (Wiebe and Jette 2012, Wang et al. 2013).
FIG. 9. Examples of various benign variants: A) benign sporadic sleep spikes, B) left temporal wicket waves, C) 14 and 6 Hz positive spikes, D) six hertz spike-waves, E) rhythmic temporal theta burst of drowsiness, and F) subclinical rhythmic electrographic discharge of adults (SREDA). Reprinted with permission from Elsevier from Santoshkumar et al. 2009.
CONCLUSIONS

In conclusion, MTLE is a very specific subtype of temporal lobe epilepsy with distinct clinical and electrophysiological characteristics. Hippocampal sclerosis is the most common pathological cause of MTLE noted in surgical series and helps to explain the semiology seen. Characteristic semiological signs such as the abdominal aura or automotor seizures remain vital clues to a mesial temporal epileptogenic zone. When these signs are accompanied by the typical ictal 5 to 9 Hz rhythmic activity in the temporal leads, one can be fairly comfortable with the diagnosis of mesial temporal lobe epilepsy. In the end, while some MTLE patients will respond to medical therapy alone, surgical therapy does represent a viable treatment strategy for those patients who do not. The accurate diagnosis and localization of the MTLE epileptogenic zone is essential so that patients may be provided with optimal information when counseling them on their disease.

REFERENCES


The Role of Neuropsychology on an Epilepsy Monitoring Unit: A Peek Behind the “Do Not Disturb” Sign

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ABSTRACT. Neuropsychological services are considered an essential component of specialized epilepsy centers. In such a multidisciplinary setting, neuropsychologists interact regularly with other professionals involved in epilepsy patients’ care. For these other professionals, this article provides an overview of the background of neuropsychologists, the services they provide, and how their findings contribute to the evaluation of the epilepsy patient. Two case examples are included to illustrate how neuropsychological evaluations are employed in the epilepsy monitoring unit setting.

KEY WORDS. Cognitive profile, epilepsy, epilepsy monitoring unit (EMU), epilepsy surgical candidate, neuropsychological evaluation.

INTRODUCTION

The American Academy of Clinical Neuropsychology Practice Guidelines (2007) define clinical neuropsychology as “an applied science that examines the impact of both normal and abnormal brain function on a broad range of cognitive, emotional, and behavioral functions.” Neuropsychologists’ training typically includes a doctoral level degree in psychology (i.e., Ph.D. or PsyD), followed by a clinical internship and post-doctoral fellowship in the science of human behavior as it relates to normal and abnormal functioning of the central nervous system (Hannay et al. 1998). Some
pursue board certification as further demonstration of competence in this specialty area of professional psychology.

In clinical practice, neuropsychologists’ methods include the integration of objective neuropsychological test findings with systematic behavioral observations, interview of the patient and collateral informants, review of medical records, and knowledge of the neuropsychological impact of neurological conditions. Neuropsychological tests are standardized instruments with acceptable levels of reliability and validity designed to quantitatively evaluate cognitive functioning.

The test battery administered to the patient is selected by the neuropsychologist based on the patient’s individual characteristics, their presenting complaints, and the referral question being addressed. The tests are administered in a quiet room in a one-on-one fashion by either the psychologist themselves or by a trained psychometrist practicing under the psychologist’s supervision. The task instructions and scoring procedures are standardized such that the test is administered in the same fashion to every patient.

A comprehensive neuropsychological evaluation assesses a range of different cognitive functions to sample functioning of different brain regions and networks (Jones-Gotman et al. 2010, Lezak et al. 2012) and often requires at least four hours to complete in an epilepsy monitoring unit (EMU) setting. Commonly assessed cognitive domains include:

**Orientation:** The neuropsychological evaluation often begins with an examination of the patient’s basic alertness and orientation by asking them about themselves, their current location, and the time/date. If the patient is unusually somnolent or confused (potentially due to being in a postictal state), further testing may be postponed until they are better able to participate.

**Intellectual functioning:** The majority of intelligence instruments are comprised of multiple subtests assessing several factors such as verbal reasoning, visuospatial abilities, attention/concentration, and processing speed. For example, common verbal subtests ask patients to provide definitions or engage in verbal reasoning tasks which are rated for accuracy. Visuospatial subtests might ask the patient to recreate a geometric design or identify the missing piece of a matrix. Attentional subtests often ask the patient to hold progressively longer strings of information in mind and repeat them back to the examiner. Processing speed subtests are timed with a stopwatch and assess how much of a task a patient can complete in a set time period.

**Academic skills:** These tests assess an individual’s ability to perform such academic tasks as spelling, reading, and arithmetic and are particularly relevant for patients who are students or have a suspected learning disability. Academic measures’ format is similar to “school type tests.” For example, the patient might be asked to complete as many math problems as they can in a set time limit.

**Language abilities:** Language tests typically assess abilities such as confrontational naming, verbal fluency, and auditory comprehension. Naming tests ask the
patient to provide the name for an object presented pictorially or described to them. Verbal fluency tasks ask the patient to name as many words as possible in a set time frame that meet certain criteria (i.e., belong to a semantic category, begin with a certain letter). A frequently utilized receptive language measure involves the patient following a set of increasingly complex commands.

**Learning/memory:** Memory for both verbal and visuospatial stimuli is usually assessed (i.e., word lists, stories, word-pairs, designs, dot locations). On some memory tasks, the patient is asked to learn information over repeated trials, and their ability to later recall that information after a delay is tested. For example, some verbal memory tests ask that the patient listen to a list of words several times, each time repeating back as many of the words as possible to the examiner. Then, after a delay period, they are asked to provide as many of those words as they can remember. A recognition trial is also included in some memory measures, where the patient is asked to identify the previously learned items from a larger set including non-target options.

**Visuospatial abilities:** The visuospatial domain includes tests of visuoconstructional skills, visual scanning and cancelation tasks, visuoperceptual abilities, pattern recognition, and facial recognition. For example, a patient may be asked to copy a complicated design or identify lines of similar angles.

**Executive functioning:** Executive functioning can be a fairly broad domain, including such abilities as attention/concentration, cognitive flexibility, selective attention/response inhibition, organization/planning, problem solving, and concept formation. Examples include tests that ask patients to sort items in order to assess their ability to form abstract concepts, and those that ask patients to shift between different sorting or sequencing concepts.

**Effort:** Effort tests are designed to detect cases in which a patient’s performance suggests suboptimal engagement or motivation to do their best. The psychologist may examine the patient’s performance within the standard test battery for embedded signs of questionable effort and/or may administer freestanding tests.

**Sensorimotor skills:** These include measures of grip strength, fine manual motor speed, dexterity, and sensory functions. For example, the patient may be asked to tap their index finger as quickly as possible for a set time period.

**Psychological functioning:** These instruments can include self-report questionnaires regarding quality of life, which is important in determining the extent of disability an epilepsy surgery candidate is experiencing due to their seizures. Other measures assess personality, social-emotional functioning, adaptive behaviors, and psychiatric symptomatology. Psychiatric issues are often comorbid with epilepsy and may influence an individual’s quality of life and likelihood of becoming seizure-free after surgery (Boylan et al. 2004, LaFrance et al. 2008, Metternich et al. 2009). Video-EEG remains the gold-standard for the diagnosis of epilepsy versus psychogenic nonepileptic events, but personality testing can assist in identifying those EMU
patients with a propensity towards somatization and potential risk for psychogenic etiology for their events (Locke et al. 2011).

Once the testing is complete, an individual patient’s score is compared to normative data, allowing the neuropsychologist to objectively determine whether their performance is within the expected range for someone of their demographic background (i.e., age, gender, education, etc.). The neuropsychologist analyzes their overall pattern of performance relative to the normative group rather than examining how the patient responds to individual test questions. This process yields a cognitive profile for the patient, highlighting their individual strengths and weakness. Areas of deficit can be suggestive of dysfunction in the brain region typically subserving that cognitive domain. For example, verbal learning/memory abilities are often localized to dominant (usually left), mesial temporal lobe functioning.

ROLE OF NEUROPSYCHOLOGY IN EPILEPSY

Neuropsychologists are members of the essential personnel comprising an Epilepsy Center interdisciplinary care team (Labiner et al. 2010). They work with patients who have epilepsy in a variety of settings including outpatient assessment of individual’s neurocognitive status, drug and device studies examining the potential cognitive side effects of products, determining eligibility for school services, disability evaluations, and in EMUs as part of patients’ diagnostic work-ups or presurgical evaluation.

For patients undergoing EMU monitoring, their neuropsychological testing is often conducted bedside during their hospital stay. This practice has the advantage of simultaneous video EEG (VEEG) recording. The cognitive demands of testing can act as a stressor to elicit a seizure in some patients, which is then captured by the VEEG for review by the epileptologist. The VEEG also allows the detection of subclinical epileptiform discharges during the evaluation that can be associated with transient cognitive impairment. Such brief epileptiform events can adversely impact a patient’s test performance (Lee 2010), and might go undetected during an outpatient evaluation.

For patients being considered for epilepsy surgery, the cognitive profile yielded by their neuropsychological evaluation is examined for evidence of lateralized or localized cerebral dysfunction. These findings establish a preoperative baseline and have implications for identifying the seizure focus, prognosis for postoperative seizure freedom, and the risk of postoperative cognitive decline (Potter et al. 2009, Jones-Gotman et al. 2010, Lee 2010). The EMU patients’ neuropsychological evaluation findings are integrated with data from other elements of their work-up (i.e., EEG, video review of their seizure semiology, neuroimaging studies, neurological examination, medical history) at a multidisciplinary conference where their candidacy for
surgery is considered. Potential surgery candidates’ performance on neuropsychological testing provides a unique contribution to their overall assessment, in that it quantifies their behavioral abilities while other techniques evaluate anatomical or neurophysiological abnormalities (Jones-Gotman et al. 2010). A pattern suggesting a focal deficit that is concordant with the EEG identified epileptogenic zone and abnormalities on a magnetic resonance imaging (MRI) scan of the brain can increase confidence in the localization of the seizure focus (Lee 2010). Discordant findings may prompt further investigative studies and can provide further information for the providers who are counseling the patients about the potential risks and benefits of surgery.

For example, an isolated deficit in verbal learning/memory can be suggestive of mesial temporal lobe dysfunction in the language dominant hemisphere. If that finding is coupled with MRI evidence of left mesial temporal sclerosis, EEG demonstrating left temporal seizure onset, and semiology consistent with temporal lobe partial onset seizures, a patient with medically refractory epilepsy would likely be considered a strong surgical candidate. In contrast, a patient with intact pre-operative memory and negative MRI or other discordant findings might be considered in need of Phase II monitoring or additional counseling regarding the potential cognitive risk of proceeding with surgery.

In addition to bedside cognitive testing, neuropsychologists at some epilepsy centers are involved in the development and interpretation of Wada procedures, functional neuroimaging studies (functional MRI [fMRI], magnetoencephalography [MEG]), electrocortical stimulation mapping, and intraoperative mapping. Some also provide interventional services such as psychotherapy or cognitive rehabilitation.

Below are case examples illustrating how the neuropsychological assessments of two patients seen in an EMU setting were integrated into their larger presurgical evaluation. Some details of the cases have been changed to protect the patients’ confidentiality and for clarity of the discussion.

**Case #1:** The first case is an example of how concordant neuropsychological test results can support a patient’s candidacy for epilepsy surgery. Mrs. Jones was a 45-year-old, right-handed female who experienced status epilepticus at age 2 in the setting of meningoencephalitis and later developed possible partial seizures at age 40. Her events as an adult were characterized by an occasional aura of feeling a “whoosh down her body,” followed by 20 to 30 seconds of oral and manual automatism and loss of consciousness. Postictically, she was confused for 1 to 2 minutes, but quickly returned to baseline. At the time of her EMU admission, the frequency of her possible seizures had recently increased to several per day. The patient had previously failed trials of levetiracetam, carbamazepine, and divalproex sodium, and was taking clonazepam at the time of EMU admission.

On interview, Mrs. Jones reported that she had noticed some trouble with her memory around the time her seizures resumed in adulthood, but felt her cognition
had since returned to normal. She did not report any trouble working in a retail position and denied any other functional limitations. She reported a period of depression about six years ago, but no current emotional issues or substance abuse.

Mrs. Jones’ neuropsychological test performance during her EMU admission is outlined in Table 1. The pattern suggested mild restrictions in expressive language abilities (confrontational naming, verbal fluency) and a more significant impairment in verbal learning/memory. Her overall intelligence was measured to be in the average range for her age, with mildly stronger visuospatial than verbal ability levels. The overall pattern was suggestive of dysfunction in the language dominant hemisphere, maximal in the mesial temporal lobe. There was no evidence of significant psychiatric issues.

Mrs. Jones’ EMU admission lasted four days. Her interictal EEG was remarkable for left temporal spike waves, most frequent during sleep. Six stereotyped events were recorded, which were consistent with her typical events and semionically suggestive of epileptic seizures. The ictal EEG did not have any definite electrographic correlate, but subtle generalized slowing was noted postictally.

### Table 1. Case #1 neuropsychological data summary.

<table>
<thead>
<tr>
<th>Cognitive Domain</th>
<th>Qualitative Description of Score</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Intellectual Functioning</strong></td>
<td></td>
</tr>
<tr>
<td>Verbal intellectual ability</td>
<td>Low average</td>
</tr>
<tr>
<td>Perceptual intellectual ability</td>
<td>Average</td>
</tr>
<tr>
<td><strong>Verbal Memory</strong></td>
<td></td>
</tr>
<tr>
<td>Story memory</td>
<td>Moderately impaired</td>
</tr>
<tr>
<td>Word list memory</td>
<td>Moderately impaired</td>
</tr>
<tr>
<td><strong>Visual Memory</strong></td>
<td></td>
</tr>
<tr>
<td>Simple design memory</td>
<td>Average</td>
</tr>
<tr>
<td>Complex design memory</td>
<td>Average</td>
</tr>
<tr>
<td><strong>Language</strong></td>
<td></td>
</tr>
<tr>
<td>Naming</td>
<td>Mildly impaired</td>
</tr>
<tr>
<td>Category fluency</td>
<td>Mildly impaired</td>
</tr>
<tr>
<td>Letter fluency</td>
<td>Mild-moderately impaired</td>
</tr>
<tr>
<td><strong>Executive Functioning</strong></td>
<td></td>
</tr>
<tr>
<td>Cognitive flexibility</td>
<td>Average</td>
</tr>
<tr>
<td>Working memory</td>
<td>Average</td>
</tr>
<tr>
<td>Processing speed</td>
<td>Low average</td>
</tr>
<tr>
<td><strong>Motor Functioning</strong></td>
<td></td>
</tr>
<tr>
<td>Right hand fine motor speed</td>
<td>Average</td>
</tr>
<tr>
<td>Left hand fine motor speed</td>
<td>Average</td>
</tr>
</tbody>
</table>

Qualitative classification of performance based on normative data comparisons; tests administered: Wechsler Adult Intelligence Scale-III, subtest of Wechsler Memory Scale-III, Wide Range Achievement Test-4 Reading subtest, Auditory Verbal Learning Test, Brief Visual Memory Test – Revised, Boston Naming Test, Controlled Oral Word Association Test, Animal Naming, Rey-Osterrieth Complex Figure Copy and Delay trials, Trailmaking Test A and B, Stroop Color Word Test, Finger Tapping, Minnesota Multiphasic Personality Inventory-2.
Mrs. Jones also underwent several neuroimaging studies as part of her presurgical work up. MRI of the brain revealed mild left mesial temporal sclerosis and a positron emission tomography (PET) scan demonstrated decreased left temporal metabolism. Subtraction ictal single-photon emission computed tomography (SPECT) coregistered to MRI (SISCOM) suggested focal hypermetabolism in the anteromedial left temporal lobe/hippocampus during her seizure.

Mrs. Jones was discharged from the hospital on levetiracetam and lacosamide with a diagnosis of intractable partial epilepsy. A left temporal focus was suspected given her interictal EEG, neuropsychological test results, and neuroimaging findings. Her seizure control did not improve with the new medications and she was discussed at epilepsy surgery conference. Intracranial monitoring was recommended due to her non-localizing ictal EEG and she underwent implantation of bilateral temporal depth electrodes and a left temporal subdural strip. The Phase II recording captured eight electrographic events, all of which arose from the left anterior mesial temporal contacts. A sodium amytal evaluation (Wada procedure) demonstrated left hemisphere language dominance, good memory support using the right hemisphere, and only marginal memory performance using the left. Given her Wada results and the verbal memory deficit observed on her neuropsychological testing during her EMU stay, the patient was considered to be at low risk of further memory decline postoperatively. The patient proceeded with a left temporal lobectomy three years ago and her seizures remain in remission with one antiepileptic drug. She has not reported any postoperative cognitive complaints.

**Case #2:** This case is an example of the neuropsychological results playing a prominent role in surgical planning. Mr. Smith was a 21-year-old, right-handed male. His seizures began at age 17. Seizures were stereotyped episodes of déjà vu, nausea, and tunnel vision progressing to loss of awareness. There was occasional secondary generalization of his seizures. Postictally, he was typically confused and had trouble with word finding. Evaluation at that time with routine EEG showed left temporal sharp waves and a lesion in the left hippocampus on MRI. Differential diagnosis included gliosis versus developmental abnormality versus low grade glioma. He was tried on phenytoin (discontinued for rash) and levetiracetam (discontinued for cognitive side effects) prior to his current regimen of lamotrigine and divalproex with lorazepam prn for breakthrough seizures. The left temporal lobe lesion remained stable on serial MRI exams. At the time of admission to the EMU, his seizure frequency had been increasing to several per month.

During his admission, his medications were tapered and discontinued. He underwent activating procedures including hyperventilation, photic stimulation, exercise, and sleep deprivation. There were no interictal abnormalities on EEG. He had one clinical seizure that showed onset over the left frontotemporal region (maximum at F7/T3/T5).
Mr. Smith underwent comprehensive neuropsychological evaluation while in the EMU, prior to the activating procedures and clinical seizure. Subjectively, Mr. Smith felt he was having more problems with memory, concentration, and word finding over the past six months (the same time period seizures were increasing). He was working in a bookstore while also engaged in some college coursework. He had recently taken two “incompletes” in college classes because of his increased seizure frequency. Mr. Smith denied any problems completing his work responsibilities. He was managing his appointments, medications, and finances independently with some external aids such as a calendar and pillbox. He denied any psychiatric symptoms or complaints as well as any history of substance abuse.

Results of his neuropsychological evaluation are described in Table 2. In summary, his cognitive data showed intact verbal memory (which is typically correlated with left temporal lobe functioning) and variability in visual memory. This pattern (stronger verbal and visual memory) was unexpected given the known left temporal

Table 2. Case #2 neuropsychological data summary.

<table>
<thead>
<tr>
<th>Cognitive Domain</th>
<th>Presurgery</th>
<th>Postsurgery</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Intellectual Functioning</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Verbal intellectual ability</td>
<td>High average</td>
<td>High average</td>
</tr>
<tr>
<td>Perceptual intellectual ability</td>
<td>Average</td>
<td>Average</td>
</tr>
<tr>
<td><strong>Verbal Memory</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Story memory</td>
<td>Average</td>
<td>Average</td>
</tr>
<tr>
<td>Word list memory</td>
<td>High average</td>
<td>Average</td>
</tr>
<tr>
<td><strong>Visual Memory</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Simple design memory</td>
<td>Mildly impaired</td>
<td>Average</td>
</tr>
<tr>
<td>Complex design memory</td>
<td>Low average</td>
<td>Below average</td>
</tr>
<tr>
<td><strong>Language</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Naming</td>
<td>Low average</td>
<td>Average</td>
</tr>
<tr>
<td>Category fluency</td>
<td>Below average</td>
<td>Below average</td>
</tr>
<tr>
<td>Letter fluency</td>
<td>High average</td>
<td>Average</td>
</tr>
<tr>
<td><strong>Executive Functioning</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cognitive flexibility</td>
<td>Average</td>
<td>Average</td>
</tr>
<tr>
<td>Working memory</td>
<td>Average</td>
<td>High average</td>
</tr>
<tr>
<td>Processing speed</td>
<td>Low average</td>
<td>Average</td>
</tr>
<tr>
<td><strong>Motor Functioning</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right hand fine motor speed</td>
<td>Below average</td>
<td>Low average</td>
</tr>
<tr>
<td>Left hand fine motor speed</td>
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</tbody>
</table>

Qualitative classification of performance based on normative data comparisons; tests administered: Wechsler Adult Intelligence Scale-IV, subtest of Wechsler Memory Scale-III and IV, Wide Range Achievement Test-4 Reading subtest, Auditory Verbal Learning Test, Brief Visual Memory Test – Revised, Brown Location Test, Boston Naming Test, Controlled Oral Word Association Test, Animal Naming, Rey-Osterrieth Complex Figure Copy and Delay trials, Trailmaking Test A and B, Stroop Color Word Test, Finger Tapping, Personality Assessment Inventory, Toronto Alexithymia Scale, Quality of Life in Epilepsy – 31.
lobe lesion and suggests increased risk of memory decline with standard left anterior temporal lobectomy. Therefore, the neuropsychologist recommended a sodium amytal exam (Wada procedure) to further evaluate memory and language lateralization. Psychological testing revealed possible low-grade depressive symptoms and low self-reported quality of life ratings.

The amytal exam revealed bi-hemispheric support for speech and language functions, but stronger in the left hemisphere than the right. In addition, there was strong support of both verbal and visual memory by the left hemisphere, but poor to adequate support of memory in the right hemisphere. These findings also increased concern regarding Mr. Smith’s risk for post-surgical memory loss.

Mr. Smith was discussed at the epilepsy team surgical conference attended by the neurologists, neurosurgeon, neuropsychologist, and EEG technologists. MRI and EEG evidence suggest good candidacy for resective left temporal lobe surgery. However, the neuropsychological and amytal results suggested high risk for memory loss. It was discussed that the exact nature of his left temporal lobe lesion was never confirmed. That is, it was not known if this was a low grade glioma (which has some risk of malignant transformation in the future) or a benign tumor such as a dysembryoplastic neuroepithelial tumor (DNET). It was recommended that Mr. Smith undergo a biopsy for determination of pathology. If the tumor was a glioma, it would be recommended that he undergo resection due to the concern about malignant transformation in the future. If the tumor was a DNET, it would be recommended that he consider a tissue sparing treatment such as Visualase MRI-guided (Visualase, Inc., Houston, Texas, USA) laser ablation in order to minimize his cognitive risk. Mr. Smith underwent a biopsy with the resulting pathology indicative of a DNET. He proceeded to undergo the laser ablation procedure.

Post-surgically, Mr. Smith has done well. He has had two seizures in the interim, both with possible provocation (missed medication dose and one when ill with gastrointestinal and febrile symptoms). He has gotten married and is in the process of taking over his family business. Mr. Smith denied any cognitive complaints or limitations. He is managing his personal affairs independently. He denied any psychiatric symptoms and feels his energy and motivation have improved. His medication regimen has been reduced to lamotrigine only. Results of his neuropsychological evaluation completed six months post-operatively are outlined in Table 2. In summary, verbal memory (the specific area of risk concern) remains normal. Mr. Smith continues to have some memory asymmetry with weaker visual memory, but this is unchanged to even less prominent than pre-surgery. He is also improved on some speed measures (motor and cognitive), which may reflect his reduced antiepileptic drug regimen. Psychological assessment was within normal with no evidence of any depressive symptoms and improved quality of life ratings compared to presurgery.
CONCLUSION

In summary, a comprehensive epilepsy evaluation entails a collaborative effort involving multiple disciplines and integration of data from multiple sources. To function smoothly, the team members need an understanding of each other’s roles and what each discipline contributes to the process. We have described the methods by which neuropsychological evaluations are conducted and how the findings contribute to the larger evaluation to guide treatment.

REFERENCES


