



TEXAS NEUROLOGICAL SOCIETY

Broca's Area

The Voice of Texas Neurology

Summer 1999

Surgical Treatment of Essential Tremor

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Introduction

Advances in stereotactic and neuroimaging techniques that allow for greater accuracy and less surgical morbidity have made surgical interventions appealing in essential tremor (ET). Stereotactic procedures like thalamotomy and thalamic stimulation are safe and effective forms of therapy in some patients.

The intended target for tremor is the VIM nucleus of the thalamus. The current concept of the connectivity of the basal ganglia-thalamocortical motor loop involves basal ganglia connections from the globus pallidus through the anterior part of the VL (Voa) nucleus of the thalamus to the supplementary motor cortex (area 6). A second pathway consists of fibers from the contralateral dentate nucleus of the cerebellum through the posterior part of the VL (Vop and VIM) thalamic nucleus to the motor cortex (area 4). This second pathway may explain the efficacy of VIM thalamic lesions for the treatment of tremor of many etiologies.

Thalamotomy

Initial reports regarding the benefits of thalamotomy in ET indicated improvement in the contralateral side of the lesion in greater than 90% of patients. Although assessments were often qualitative rather than quantitative, the reported studies and present clinical anecdotal experience indicate that unilateral thalamotomy is an effective treatment. The percentage of patients who have shown improvement in ET following thalamotomy has been reported to range from 78% to 100%.

Long-term follow-up studies indicate the benefits of thalamotomy continue in a majority of the patients. Post-operative complications have included numbness, dysarthria, gait disturbance, weakness, dysequilibrium, and cognitive problems. Persistent morbidity occurs in less than 10% of patients. One of the main concerns of ablative thalamic surgery is the risk of speech difficulty associated with bilateral thalamotomy.

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Words from the President Martin Heitzman, M.D.

This is my first message to you as your President. Following on from past presidents who are good at what they do, it is both easier and more difficult than starting from scratch. It is easier because the way is paved for you, and harder because you haven't driven this particular car before. Please bear with me as we move forward towards Y2K.

In 1996, Dr. Ernesto Infante and in 1997, Dr. J. Edward Garrison addressed the issue of electromyography (EMG) being performed by non-physicians. They worked diligently to ensure that EMG remained in the hands of qualified doctors. Their efforts were in vain, as the ultimate decision, made by the Texas Attorney General's Office now allows non-physicians to perform EMG's.

Another area of clinical concern relates to non-neurologists making inaccurate neurological diagnosis. These include radiculopathy, plexopathy, and various entrapment syndromes. Some of these physicians, with little or not training in electrodiagnostic medicine, are performing EMG studies. Once the diagnoses are made, the treating physician is in a position to perform a series of blocks, and if these fail, often surgery.

This problem was addressed in an article by Dr. Peter Dyck et al, published in the journal *Neurology*, July, 1998: "Ten Steps In Characterizing and Diagnosing Patients with Peripheral Neuropathy. Dyck pointed out that physicians (neurologists) must have considerable background information about peripheral nerve anatomy, physiology, pathology and clinical neurology to arrive at a correct diagnosis. The authors do not believe physicians assistants or even many general

physicians can expertly use this 'Ten Step' method in evaluating patients with suspected peripheral nerve disorders.

At present, there are other neurological issues. While these issues may appear to be more subtle than the EMG problem, many would describe them as of greater significance. Some physicians have begun to use Quantitative Sensory Testing (QST) to reinforce their diagnoses. These physicians do not appear to be aware of some of the limitations of QST. In the May, 1999, issue of *Neurology*, an editorial was published by ten nationally known experts on peripheral nerve disorders. This article describes the significant limitations of QST.

The concept of limited use of QST was reinforced at a meeting of the American Association of Electrodiagnostic Medicine in October, 1998, in Orlando, Florida. A half-day session was devoted to small fiber neuropathy and QST. The faculty concluded that QST is inconsistent and often invalid and should be considered investigational until the clinical validity can be well established with known normative values.

A position paper on this topic is in preparation by the American Academy of Electrodiagnostic Medicine in conjunction with the American Academy of Neurology. It is expected to be published within the next year.

In the spring of 1996, Dr. Tom Hutton, editor of Broca's Area, stated "the TNS and its members need to maintain vigilance to assure that patients obtain quality EMG services and that EMG does not become trivialized by unsophisticated and untrained practitioners". This philosophy needs to be applied to the areas mentioned above relating to clinical diagnosis, routine EMG and QST.

In the constitution of the TNS, Article II describes six objectives of the society. All six of these items are *(continued on page 9)*

Neurosurgery: A Personal Experience

Gage Van Horn, M.D.

As neurologists, many of us have recommended neurosurgery for our patients, yet few of us have experienced such surgery. On November 11, 1998, I underwent a retro-mastoid craniectomy with microv-ascular decompression of the facial nerve, the so-called Jannetta procedure, done in Pittsburgh by Dr. Peter Jannetta. It was a positive experience, one that has so far led to a "cure" of my hemifacial spasm. Tom Hutton, editor of Broca's Area, asked me to relate my experience in these columns.

I have had left-sided hemifacial spasm for about five years. For the first two years, I could not make the diagnosis. The only symptoms were slight twitching of the left orbicularis oculis, lasting several minutes, induced by intense taste or by chewing. My first spasms began about three years ago. These recurred every three to four days, lasted 5-10 seconds, and involved most of the facial muscles on the left side. My left eye would close and the left side of my face would pull. Since these were so infrequent, I ignored them, and hoped that they would go away, or at least remain the same.

I hid the diagnosis from my wife and my colleagues. Only rarely would a spasm occur in the presence of someone that could make the diagnosis, and I would turn the involved side away, not showing my entire face until the spasm passed. One of our perceptive residents, now a neurophysiology fellow, told me that when I pimpled him during rounds that my left palpebral fissure would narrow. He thought I was just giving him the evil eye. My chairman, Jim Ferrendelli, told me that I had a slight left facial paresis for the six years he has known me.

Over the past two years the spasms

occurred more frequently and their duration increased. Some would last 2-3 minutes, with a frequency of 5-10 per hour. On a white-winged dove hunt in early September, 1998, the spasms occurred every time I pulled the trigger, which did not help my already faulty aim. Until that time, I could count on getting a spasm only when I chewed my morning cereal or when I put eye drops in my left eye. By mid-September, almost anything would induce a spasm: brushing my teeth, shaving, eating, any emotional experience (especially laughing) looking up, whistling, bright sunlight, or voluntarily by closing my eyes tightly or showing my teeth. The spasms were especially annoying when delivering lectures. I reached the point where I just told my audience I had the problem, and just tried to ignore the spasms. Driving was also trying, since bright light or glare usually initiated a spasm. Something had to be done.

After telling my wife, and getting my primary care provider to send me to a specialist, I consulted a neurologist. I chose Jim Willmore, because I thought he had more expertise in using the drugs useful in retarding the spasms. Jim found nothing abnormal on neurological examination, ordered an MRI and initiated baclofen. The MRI was an experience in itself. I am not claustrophobic, but I can certainly see why some patients cannot even contemplate having the procedure without being heavily sedated.

My MRI showed that the left vertebral artery was somewhat aberrant, lifting and compressing the left seventh nerve. My films are now on display in the radiology teaching files. Joel Yeakley, our Chief of Neuroradiology, said my anomaly was the best example of a cause for hemifacial spasm that he had ever seen. What a distinction!

Baclofen, in doses of up to 20 mg tid did nothing to reduce the frequency or severity of the spasms. It was (*continued on page 10*)

Young Investigators' Award Winners

Many thanks to all of the applicants who competed for this year's TNS Young Investigators' Award. The selection committee ran smoothly under the leadership of TNS Vice President Clifton Gooch, M.D., with committee members: Richard Homan, M.D., Texas Tech University HSC, Lubbock; John Slopis, M.D., UT Medical School, Houston; Richard Barohn, M.D., UT Southwestern Medical Center, Dallas; David Sherman, M.D., UTHSC, San Antonio, and John Calverley, M.D., UTMB, Galveston. The first and second place winners received cash awards and the opportunity to present their papers at the TNS Winter Conference in February.

First place was awarded to Suleiman Kojan, MD., for his paper, "Saint Louis Encephalitis- A Review of 11 Cases in the 1995 Dallas Epidemic." Dr. Kojan is a resident in the Department of Neurology, Pathology and Radiology, University of Texas Southwestern Medical School, Dallas, Texas.

Second place went to Tim Benke, M.D. for his paper, "Two Molecular Mechanisms for Hippocampal LTD Determined by the Recent History of Synapses: A Decrease in AMPA Receptor Conductance and in their NSF-Dependent Surface Expression." Tim Benke is first year Neurology resident in the Child Neurology program at Baylor College of Medicine in Houston. Prior to completing two years of pediatric training in the Department of Pediatrics at Baylor, he was a postdoctoral research assistant in the laboratory of Dr. Graham L. Collingridge in the Department of Anatomy at the University of Bristol. He received his M.D. and Ph.D. from Baylor College of Medicine, his M.S. in Electrical Engineering from Rice and his B.S. in Biomedical Engineering and Chemistry from Vanderbilt University.

The winning abstracts are below.

"Saint Louis Encephalitis- A Review of 11 Cases in the 1995 Dallas Epidemic."

by Suleiman Kojan, MD

Objective: St. Louis encephalitis is a common arboviral infection occurring in epidemic patterns in the south-central and midwestern United States.

Methods: We studied 11 patients with SLE during the 1995 epidemic in Dallas.

Results: We noticed several features that have not received prominent attention in previous studies. Through aggressive use of electroencephalography, we diagnosed seizures in a larger percentage of our patients, and one had non-convulsive status epilepticus. Two patients had abnormal MRI scans. Finally, 18% of our patients had co-infection with HIV, suggesting that HIV-positive patients are higher risk for symptomatic SLE infection.

Two Molecular Mechanisms for Hippocampal LTD Determined by the Recent History of Synapses: A Decrease in AMPA Receptor Conductance and in their NSF-Dependent Surface Expression."

by Tim Benke, M.D., Ph.D.,

Andreas Luthi Mary Palmer, Ramesh Chittajallu, Timothy A. Benke, Jeremy Henley, John T.R. Isaac and Graham L. Collingridge.*

Knowledge of how synapses alter their efficiency of communication is central to the understanding of learning and memory. The most extensively studied forms of synaptic plasticity are long-term potentiation (LTP) and its counterpart, long term depression (LTD) of AMPA receptor-mediated glutamatergic excitatory synaptic transmission (Bliss and Collingridge, 1993; Nicoll and Malenka, 1995; Bear and Abraham, 1996). It has been shown recently that LTP involves a rapid increase in unitary conductance of (continued on page 8)

Essential Tremor Surgery

(continued from page 1)

Thalamic stimulation

Due to concerns relating to side effects and complications associated with bilateral ablative surgery, stimulators were implanted for chronic stimulation of the VIM. Since the late 1980s a number of investigators have reported the safety and efficacy of chronic thalamic stimulation in ET. The efficacy of thalamic stimulation is similar to thalamotomy however, the risk of complications appears to be lower than thalamotomy.

Thalamotomy versus thalamic stimulation

A major decision facing the clinician is whether a patient with disabling drug-resistant tremor should undergo thalamotomy or thalamic stimulation. There are several advantages of thalamic stimulation over thalamotomy: reversibility (i.e., minimal destructive lesion); adaptability (the ability to change stimulus parameters to increase efficacy or reduce side effects); and the ability to perform bilateral operations with less risk of permanent morbidity. The disadvantages of stimulation include: increased expense related to the cost of the system; implantation of foreign material, which increases the potential of infection or inflammatory reaction; the future need to replace batteries and other hardware; and the time and effort required to optimize stimulus parameters. Further studies are required to assess which of the two procedures might be more helpful.

Patient selection

ET patients with disabling medication-resistant tremor are reasonable candidates for these stereotactic procedures. Contraindications include marked cognitive problems or an unstable medical diagnosis that would increase surgical risk significantly. Some patients may not be able to tolerate the surgery due to severe anxiety (prompted by lying flat with their head bolted to the stereotactic frame).

Suggested reading:

Ohye C, Hirai T, Miyazaki M, Shibasaki T, Nakajima H. Vim thalamotomy for the treatment of various kinds of tremor. *Appl Neurophysiol* 1982;45:275-280.

Jankovic J, Cardoso F, Grossman RG, Hamilton WJ.

Outcome after stereotactic thalamotomy for parkinsonian, essential and other types of tremor. *Neurosurgery* 1995;37:680-687.

Koller WC, Pahwa R, Busenbark K, et al. High frequency unilateral thalamic stimulation in the treatment of essential and parkinsonian tremor. *Ann Neurol* 1997;42:292-299.

Benabid AL, Pollack P, Gervason C, et al. Long-term suppression of tremor by chronic stimulation of the ventral intermediate thalamic nucleus. *Lancet* 1991;337:403-406.

Thanks to Winter Conference Supporters

Many thanks to the following supporters who made this year's Winter Conference possible.

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Winter Conference '99 a Success

Gary L. Tunell, Md.
1999 Program Chairman

The 1999 Winter Conference far exceeded my highest expectations! I was extremely pleased by all speakers, the "Grand Rounds" quality of the lectures, and the succinct and practical nature of the presentations. I was also very pleased that our speakers stayed for the entire meeting and mingled with the Society as well as attending the social events. I certainly would invite all speakers again, and we got our money's worth on their presentations and added stature of our society by having the chiefs of all Neurology Departments attend and participate.

I am happy with the attendance we obtained; the entire state of Texas was well represented at the meeting. The hotel accommodations were outstanding, and the Marriott Capitol will get my vote for hosting next year's Conference. They were very accommodating to Rachael, and the conference hall and projection facilities were of the highest caliber.

The "Support Your Future Partner" project was a success. Ten neurology residents were sponsored from the academic centers across Texas. Next year I will assign each resident a host who will introduce the residents to society members throughout the Conference. This will encourage their mingling with other Society members.

As always, Rachael Reed did a stellar job in coordinating the event. I rely on Rachael heavily for the minutia of details that are necessary for this Conference. With her assistance, I look forward to the year 2000 program along with co-chair Richard Brower.

The meeting was a financial success for the Society, largely because of sponsorship by pharmaceutical companies. As you

know, this allowed us to keep fees to the Society at a bare minimum: \$75.00 for 14 hours of CME credit. We had complaints from some exhibitors about not having enough exposure time to the attendees. We will work to rectify that complaint since we truly depend on their sponsorship to keep the fees at this bargain rate.

The Ethics Hour was an excellent presentation and Rev. Meyer will be invited back next year, perhaps to speak at the Saturday luncheon with the Society and spouses in full attendance.

We also received excellent feedback regarding the extra curricular activities at the Welcome, Esther's Follies, Dr. Phil Berry and Cactus Pryor. I think it will be very difficult to improve for next year's meeting, although our efforts have already begun.

I am confident that our new president, Dr. Martin Heitzman, will lead us into a great year in 1999-2000. He has ideas about being more proactive on organ donation and working with other organizations to promote the high standard of practice of neurology across the state of Texas.

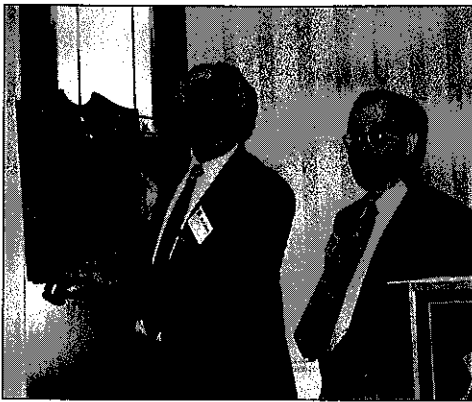


Visit the TNS Website

Visit the TNS website for membership information, registration forms for TNS meetings and information about the TMA Neurology section. Also included on the website are *Broca's Area* and other information about the Texas Neurological Society.

Visit our website and bookmark it for the future:

<http://www.texmed.org/tns/default.htm>



Dr. Phil Leonard, outgoing president welcomes Dr. Martin Heitzman, president for 1999-2000.

TNS Winter Conference '99

February 19 -21, 1999
Austin



(Above) Three founding members celebrated the TNS 25th anniversary: Sheff Olinger, M.D., Walter Buell, M.D. and Harold Skaggs, M.D.



(Left) Dr. John Calverley and Dr. Danny Ray Bartel visit during a break in the exhibit hall.

The "Support Your Future Partner" project welcomed residents from academic centers in Texas to attend the TNS Winter Conference and meet with members.



Welcome New Members

Active Members

The following new TNS members were approved for active status during the Winter 1999 Conference.

- Richard J. Barohn, M.D.**
Dallas, TX
- Stephen N. Brooks, M.D.**
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- Gabriel Diaz, M.D.**
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- Tommy Yee, M.D.**
McAllen, TX

Associate Member

- Kelvin A. Samaratunga, M.D.**
Austin, TX

Resident Members

The following Resident members were approved during the Winter Conference,

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- Yanko A. Yankov, M.D.**
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- Hui-Juan Zhang, M.D.**
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Broca's Area

A periodic newsletter of the Texas Neurological Society

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President's Message

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important, but the first one appears to be especially significant. This states "The purpose of this society is to promote the interest of patients with neurological disease by supporting the development and delivery of quality medical care to these patients, and by opposing things adverse to their interest"

In order to maintain the highest of

standards in the practice of neurology, input is requested from the membership relating to any ideas that could be implemented to maintain these standards. Consideration needs to be given to forming a committee or panel to review records when necessary and devise methods to correct any deficiencies, thereby maintaining the highest standards of neurological care. Should you wish to share you comments, I would love to hear from you.

Neurosurgery (continued from page 3)

gradually tapered and Neurontin 400 mg tid was substituted. The Neurontin helped a little, but I still had up to 5 spasms per hour lasting up to 3 minutes. I took my films to our Chief of Neurosurgery, Dr. Guy Clifton, and asked his advice. He told me about his own experience with trigeminal neuralgia about six years ago. He said that Tegretol helped his symptoms, but he experienced many toxic side effects. He consulted Peter Jannetta and underwent successful surgery in Pittsburgh. He advised that I also talk with Jannetta. Guy told me that he had only performed micro-vascular decompression on five or six patients and that he would not operate on me. Bill Maggio, our skull base neurosurgeon, told me essentially the same thing: consult Peter Jannetta. I e-mailed and then called Peter Jannetta. I have known Peter for the last 30 years or so. I was on the neurology faculty at the University of Pittsburgh when Peter became Chairman of Neurosurgery in 1971.

The first time I heard about retromastoid micro-vascular decompression for hemifacial spasm, I was more than skeptical. I attended Peter's presentation to the AAN in the early 70's where he was openly ridiculed for proposing such drastic measures for such a benign process. Now, the Jannetta procedure is well accepted, although movement disorder specialists consider Botox injections and anticonvulsant drugs the treatments of first choice for hemifacial spasm. Peter replied that he was sorry about the HFS, but that he had a very effective surgical procedure for this malady. He told me that most medications were ineffective and Botox was not a cure. He said that the longer one waited to have definitive surgery, the more likely that permanent ephaptic transmission would occur, rendering surgery less effective.

I soon learned that no scientific proof existed for any of the treatment options for

HFS, and that I would have to make up my mind based on anecdotal experiences. My decision to have the surgery was buttressed by Peter telling me that my left-sided hearing loss of many years might also respond to microsurgical decompression.

My wife Virginia and I flew to Pittsburgh on November 9, for preoperative testing scheduled the next day. I signed a surgical consent form and was told to report to Presbyterian Hospital at 5:30 a.m. I reported to the hospital, paid an initial co-pay, went to the anesthesia pre-operative area and changed into a hospital gown. Memory blurs after that.

I recall meeting the anesthesiologist after two intravenous lines had been established. She reached over me, turned a stopcock and injected about two ml of a colorless solution into the IV. My next memory was that of waking up in the recovery room with a full bladder and a dry mouth. I had expected to have a Foley in place when I awakened. Instead I was given a urinal but couldn't urinate and had to be cathed (1200 cc) with much relief. Sometime later that day, after another straight cath, a foley was inserted, which stayed in for another 24 hours. Bill Fields, my former boss and frequent surgical recipient, told me that there is usually no discomfort associated with Foley placement. The problem came when the Foley was removed. He was right.

I had expected to have a sore throat from the endotracheal tube, but the only laryngeal/tracheal symptom was mild hoarseness, which disappeared by the next day. When consciousness was more sustained, I realized that I didn't have a facial paresis and that my hearing was unchanged. I was aware of fullness on the left side of my head, but did not experience any pain.

I was told that the surgery involved a quarter-sized diameter retro-mastoid craniectomy, with *(continued on page 11)*

Neurosurgery (continued from page 10)

micro-surgical exploration of the seventh and eighth cranial nerves on the left. The left vertebral artery and two smaller branches compressed my seventh nerve. These were teased away from the nerve and Teflon "pillows" were inserted between the nerve and these arteries. One artery compressed the eighth cranial nerve and this nerve was also decompressed.

Except for mild temperature elevation for the next two days, I had no post-operative complications. I had been warned that I might develop a transient post-operative facial palsy, but that did not occur. After transfer to a monitored intermediate care unit, I slept intermittently for the next 24 hours. The only real pain I had was in the low back, radiating down both legs. I felt like I had contracted the flu, but was told that this symptom was due to meningeal irritation. My neck was sore and moderately stiff for the next few days, but there was little pain over the incision site.

The nurses offered me narcotics, but Tylenol seemed to stop most of the discomfort. I was up in a recliner the evening of surgery and given clear liquids. I ate a regular breakfast the next morning, and was allowed to get up and walk around the room. After the Foley and IV were taken out I was transferred to a regular floor. A shower felt good, and pajamas and robe allowed me to roam the floor.

Forty-eight hours after surgery, the bandage was removed and I was discharged. I was told I could wash my hair, but not soak the wound. The next day I felt well enough to walk around Oakland, the Pittsburg subdivision containing the University. We went out to eat Chinese food that evening, and my wife and daughter left the next day.

I attended a wonderful concert by the Pittsburgh symphony four days post-operatively. My old boss, Henry Higman, took me out to lunch the next day and Peter and

his wife took me out to dinner. Most of the time I stayed in the hotel room catching up on journals. One week after surgery my stitches were removed and I flew back to Houston. I was told to avoid strenuous exercise for the next six weeks, avoid driving for two weeks, and return to work after three weeks. I fudged a little on all of these times.

I have had a few twitches around the left eye and mouth on occasion, but no spasms since the surgery. Therefore the surgery was an unqualified success. My hearing was unchanged, but I wasn't expecting that to improve. I tapered the Neurontin over the next two weeks and went back to work 10 days after surgery.

Gage Van Horn, M.D. is a past president of the Texas Neurological Association. He is a professor in the Department of Neurology, University of Texas Medical School, Houston.

Young Investigator Award Winners

(continued from page 4)

AMPA receptor channels (γ) in the CA1 region of the hippocampus (Benke et al, 1998). Here we report that this increase in γ is always rapidly reversed during depotentiation (Barrionuevo et al, 1980) of pre-established LTP. However, a change in γ is not observed during LTD of naive inputs (de novo LTD; Dudek and Bear, 1992; Mulkey and Malenka, 1992) or during depotentiation when γ was not previously increased by LTP. Intracellular injection of peptide which specifically blocks the postsynaptic interaction between NSP and GluR2 (Nishimune et al, 1998; Osten et al, 1998) blocked the induction of LTD. Therefore two mechanisms exist for decreasing synaptic strength, which are dependent on how and when LTP occurred; a decrease in γ and a removal of AMPA receptors from the synaptic membrane.

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Contributors Wanted

Broca's Area is a forum for Texas Neurologists to share their comments, concerns and knowledge about neurology in Texas.

The best way to share ideas and comments is to present them to the TNS membership through articles and letters published in Broca's Area. The Editor of Broca's Area invites members to submit their comments, concerns or questions which are of interest to the TNS membership.

Reader's Historical Notes & "Neurodotes" Wanted

Broca's Area also welcomes articles from its readers that depict the impact of neurological illnesses on historical events or the development of Neurology as a specialty in Texas or interesting neuro anecdotes.

To submit an article or letter to the editor to Broca's Area, please send your contribution to:

*Tom Hutton, M.D
Editor, Broca's Area,
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Lubbock, Texas 79410*



Take notice of the new TNS logo celebrating the first 25 years of the Texas Neurological Society.