Jefferson™ Hospital for Neuroscience

Vol 4, Issue 1 | June 2008

JHN JOURNAL

Inside

Experimental and Computer Models

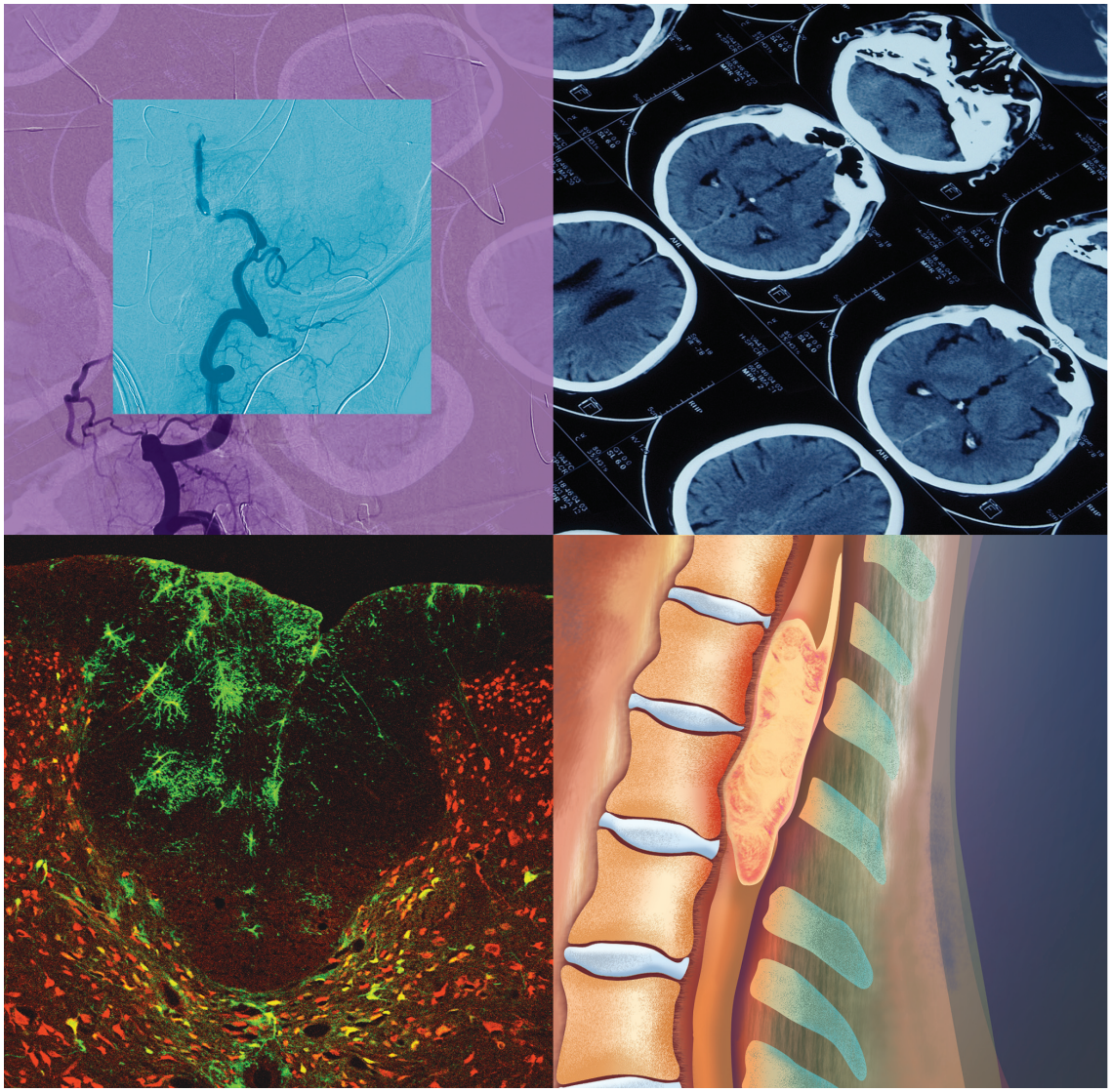
Spinal Cord Stimulation

Primary Stroke Centers

Endovascular Cure

Occult Spinal Ependymoma

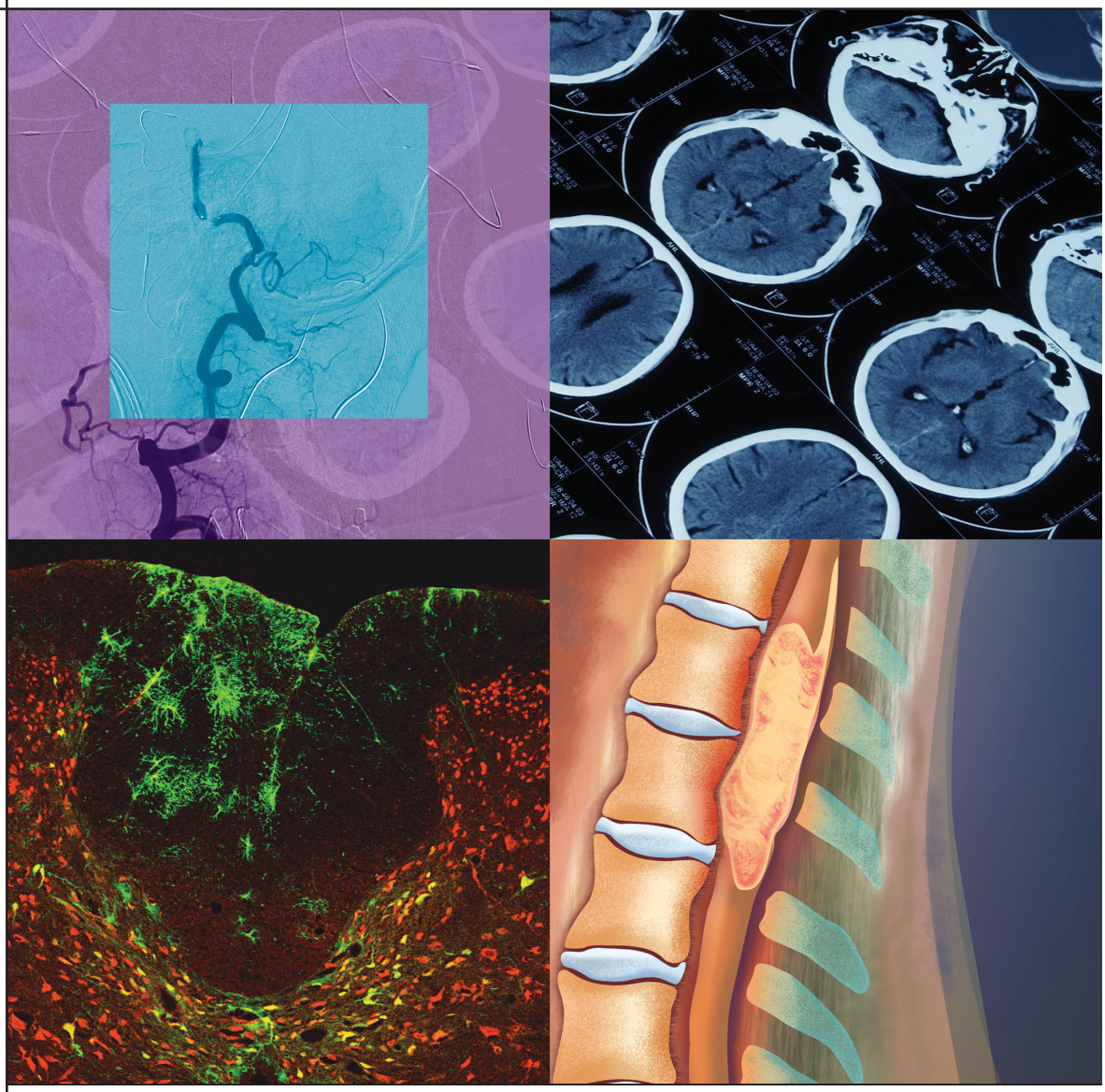
Neuroform Stent Placement



A publication of Thomas Jefferson University, Department of Neurological Surgery

JHN JOURNAL

Vol 4, Issue 1 | June 2008



A publication of Thomas Jefferson University, Department of Neurological Surgery

Chairman/Editor-in-Chief
Robert H. Rosenwasser, MD, FACS, FAHA

Editor
Mitchell G. Maltenfort, PhD

Assistant
Janice Longo

Graphic Design
JeffGraphics
Denise Hansen

Cover
Digital Composition and Illustration,
Artist: Denise Cotter

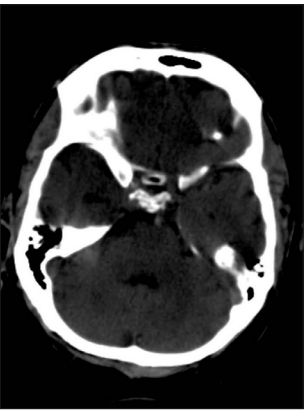
General Information

Correspondence, inquiries, or comments may be submitted to the Editor, JHN Journal, 909 Walnut Street, 3rd Floor, Philadelphia, PA 19107 or email at JHNjournal@jefferson.edu

© 2007 Thomas Jefferson University, All Rights Reserved.

ISSN 1558-8726

Table of Contents



Articles

Understanding Neurosurgery Through Experimental and Computer Models

Ciro Randazzo, MD, Aditya S. Pandey, MD, Erol Veznedaroglu, MD 2

Spinal Cord Stimulation for Control of Pain

Steven Falowksi, MD 5

Primary Stroke Centers: Their Role and Impact on Acute Stroke Management

Carissa C. Pineda MD, Jeannee Birch RN, BSN 10

Case Reports

Endovascular Cure of a "Locked-In" Patient

Meryl A. Severson, III, MD, Aditya S. Pandey, MD, Erol Veznedaroglu, MD 13

Hemorrhage into an Occult Spinal Ependymoma After Epidural Anesthesia

Peter G. Campbell, MD, Ashwini D. Sharan, MD, James S. Harrop, MD, Alexander R. Vaccaro, MD, John K. Ratliff, MD 15

Signal Drop on MRA Imaging of the Internal Carotid Artery after Neuroform Stent Placement

Aditya S. Pandey, MD, Meryl Severson, MD, Robert Rosenwasser, MD 18

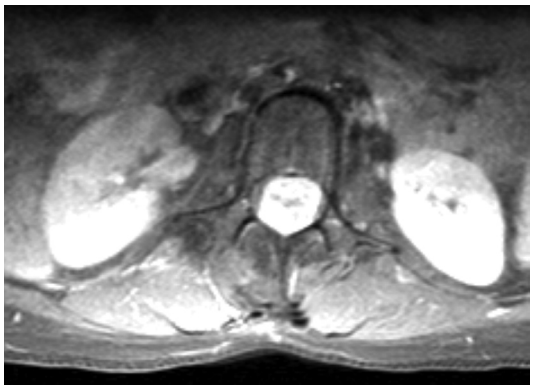
Departmental Information

Contact Information 20

Research Studies

Published Articles 21

Ongoing Research Studies 23



Understanding Neurosurgery Through Experimental and Computer Models

Ciro Randazzo, MD, Aditya S. Pandey, MD, Erol Veznedaroglu, MD

Department of Neurological Surgery, Thomas Jefferson University, Philadelphia, Pennsylvania

Introduction

Endovascular neurosurgery is an evolving field, with the goal of treating neurological disease with minimal invasion of the body. The current approach is to deliver focused therapies via catheters traveling through the vascular tree. Refinement and advancement of these techniques requires not just new ideas, but new ideas that hold up when tested through the scientific method.

Before clinical trials can begin, ethics and law demand that the ideas are tested first under experimental models. One benefit of these pre-clinical tests is the early identification of side effects. Another is the opportunity to practice and polish surgical technique in advance of human surgery.

Historically, the best models of human systems have been similar systems in appropriate animals. Like human care, animal use is governed by strict laws and regulations. Violating these rules can not only result in criminal penalties, they can also make the research worthless: an animal poorly cared for may have physical symptoms that mask or confound the response to treatment.

As computing power has increased, mathematical models have become more popular. The advantages of computational models include relative costs and relative speed. No animals, drugs or equipment have to be purchased, just a computer and software. The disadvantage is that the computational model can only represent those aspects of the modeled system that are understood and quantifiable.

This article is an overview of how both animal and computational models have been used to approximate neurovascular conditions by researchers seeking to explore treatment options.

Animal Models

Aneurysm

Dog

A common animal model for aneurysms is the canine, in which aneurysms are surgically constructed most often through the use of venous pouches. This model was first used in 1954 by German and Black[1]. Shin and Niimi later revised it[2] to allow the creation of four kinds of aneurysm—bifurcation, side-wall, arterial stump and small branch, each with different hemodynamics—in one dog. The canine models have been consistently used due to their ease of handling and anesthesia, the similarity of the blood vessel size to that in humans, and the reproducibility of the studies. However, the canine has a tendency toward rapid lysis of clots, which limits its validity as a model for testing thrombosis of aneurysms after embolization[3, 4]

Rabbit

A rabbit model of bifurcation aneurysms has been described by Spetzger and Reul.[5] Although the rabbit has a similar blood clotting system to that of the human—an advantage missing in the dog—this model was limited due to high rates of pneumonia, post-operative neurologic complications, and peri- and post-operative mortality. There was also a high likelihood of inadvertent vessel occlusion seen on angiography after creation of the aneurysms. Only about half of the procedures yielded surgically created aneurysms which could be treated by coil embolization.

Pig

Venous pouch aneurysms have also been created on the common carotid artery in swine[6] An advantage of the swine model is that spontaneous rupture of aneurysms, like those seen in humans, can occur[7]; experimental aneurysms in other animal models do not tend to rupture spontaneously. To construct the model, an anastomosis is created between the common carotid artery and the external jugluar vein. Retaining sutures are used to create a pouch in the proximal draining external jugular vein. After the animal heals for one to two weeks, an endovascular balloon can be placed angiographically to close the fistula, thus creating a fusiform vein pouch aneurysm which can subsequently be used for testing embolization devices.

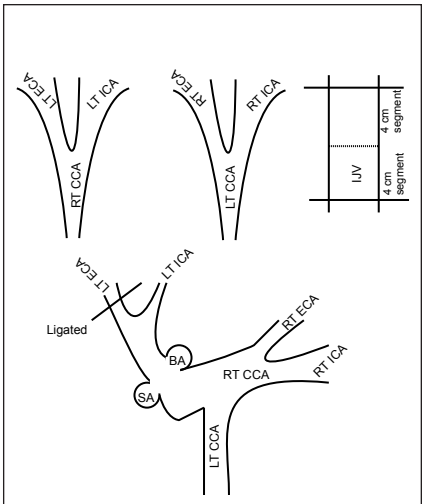


Figure 1

Canine aneurysm model

Rat

Rats are popular in research for simple economic reasons: they are cheap to purchase and cheap to house. Many experimental studies require a comparison of treatments in several subjects; a large number of dogs or pigs would be prohibitively costly, and we have already seen that rabbits had a poor experimental yield. In a study of twenty rats, ten receiving Gugliemi detachable coils and ten receiving biodegradable polyglycolide coils[8], the aneurysms were created by permanent occlusion of the common carotid artery (CCA) just proximal to its bifurcation, creating an arteriotomy between the two ligatures and directly depositing segments of coils into the CCA. Although this model was useful to test the response of arteries to the deposited segments of coils, it was

limited because the end-artery did not have flow or anatomical characteristics similar to aneurysms. Vein-pouch aneurysm models have also been described in rats.[9,10]

Monkey

Because monkeys are so closely related to humans, they would be considered the most accurate model for most systems. In an effort to more closely mimic the anatomy and hematologic clotting characteristics of the human, Tenjin and Ueda developed a primate aneurysm model.[11] They used femoral vein pouches to create venous pouch aneurysms bilaterally on the carotid arteries of Japanese monkeys. They noted a success rate of 74% at two weeks in creating their aneurysms.

Arteriovenous Malformations (AVMs)

Pig

Pig vessels approximate the size of human vessels, their coagulation profile is similar to that in the human and they are fairly simple to anaesthetize. With these advantages, the pig model is the most commonly used and described model of cerebral AVMs. The swine, and other mammals including sheep, goat, ox and cat, provide blood supply to the brain via an arterial network of vessels, known as the *rete mirabile*, which is surrounded by the cavernous sinus at the base of the brain. Because the *rete mirabile* is a purely arterial network (as seen in other mammals including sheep, goat, ox and cat), a valid AVM model is created as an arterio-venous fistula formed by isolating and anastomosing the external jugular vein and common carotid artery on one side of the neck.[3,4] The creation of this fistula with occlusion of carotid artery branches has been shown to increase flow and pressures across the rete[12-14] and result in histologic changes to vessel walls very similar to those seen in human cerebral AVMs.[15] This model has been used repeatedly for training in endovascular techniques, to evaluate nascent embolic materials and for histologic evaluations of radiosurgical induced changes.[16,17]

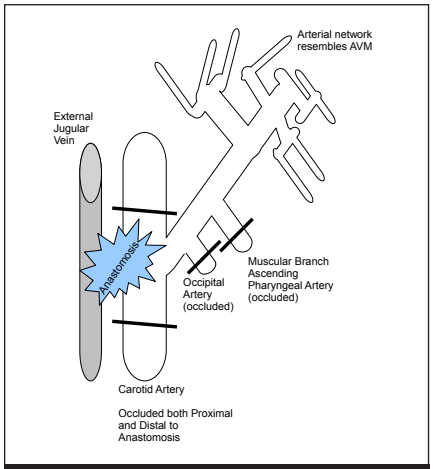


Figure 2
Swine AVM model

Dog

The only canine AVM model described is also the only completely intracerebral AVM model. Pietilä et al.[18] created an arteriovenous fistula (AVF) by interposing a segment of superficial temporal artery between a main branch of the middle cerebral artery and the sagittal sinus. They concurrently implanted a muscle graft supplied by a branch of the fistula onto the ischemic area of the brain. Their intent was to have the ischemia serve as an angiogenic stimulus and thus mimic the angiogenesis that occurs in human AVMs. The authors found that many other aspects of this model mimicked human AVMs, including: thickening and fibrosis of the wall of the draining vein; angiogenesis surrounding the area of the created AVM with new vessels demonstrating pathological wall changes; gliotic brain tissue formation and areas of ischemia; hemorrhage and thrombus formation in the brain tissue and vessels surrounding the created nidus. The authors did not however, report any attempts at experimental treatments of this model.

Rat

In the rat, Bederson[19] created an extracranial arteriovenous fistula with occlusion of the contralateral vein. This was first created in an attempt to determine if a dural AVM would form as a result of sinus thrombosis. This model has been further modified to evaluate cerebral hypoperfusion[20], the role of radiotherapy in the treatment of AVFs/AVMs[21], and the role of sinus hypertension and thrombosis in the development of dural AVMs.[22]

Vasospasm

Cerebral vasospasm continues to be one of the major causes of morbidity and mortality following anuerysmal subarachnoid hemorrhage. Megyesi et al. published a review of fifty-seven in vivo animal models of cerebral vasospasm dating back to 1928.[23] These models employed rats, rabbits, cats, pigs, canines and primates. 72% used a technique in which blood was instilled around blood vessels to cause vasospasm and the remainder used a technique whereby they punctured or lacerated a vessel to allow blood to escape and thus induce vasospasm. Some of these models involve open cranial surgery to puncture the vessels, while others have implemented perforating the vessel from the luminal side by using an endovascular approach. They also describe extracranial models of vasospasm although they do point out that these models are controversial because their validity is questionable.

Simulations

Aneurysm

In a joint project between the departments of Neurosurgery and Aerospace Engineering, Kim et al.[24] used a computational fluid dynamics software package to assess the risk of aneurysm rupture when porous stents were used as the sole treatment. Such models are reliable to the point where they can predict angiograms.[25] A model of a wide-necked basilar trunk aneurysm was constructed based on the patient's CT scans. The aneurysm was modeled untreated and treated with 1-3 stents from various manufacturers. The model predicted that these stents could suppress complex blood flow associated with ruptured aneurysms in another simulation study[26], although the size of the effect depended on the specific geometry and on how well the stents were deployed and that a single stent would probably be insufficient to ensure a good outcome.

Arteriovenous Malformations (AVMs)

Hademenos and Massoud created a mathematical model based on electrical networks.[27, 28] This model was designed to allow an analysis of changes in AVM hemodynamic and biophysical characteristics, and individual parameters could be manipulated to determine changes in flow characteristic and likelihood of rupture or hemorrhage after various treatments.

Kerber and Hecht[29] implemented on a personal computer (with an Intel 286 microprocessor!) a mathematical model which represented the AVM as a collection of 1000 channels, each of which must be embolized to "cure" the simulated AVM. They later[30] constructed a physical model from a sponge with waxed tubes representing inflow (arterial feeders) and outflow tracts (draining veins). These models were intended to help guide the development of new neurointerventional techniques. The physical model made it further possible to test liquid embolic materials using microcatheters, and to teach endovascular embolization techniques in a no-risk situation.

Applications in Training

In an ongoing effort to promote patient safety and enhance skills training, recent advances in technology have contributed to the development of non-animal simulation technology. These simulators enhance learning fundamental skills in procedures such as basic suturing, laparscopic surgery and endovascular treatments. As the technologies improve, the fidelity of these models has improved the reliability of these systems. For example, Wong et al.[31] describe a 3D virtual reality system integrated with simulated manipulators for training in craniotomies and aneurysm clipping. A variety of clips and patient aneurysms were scanned into the simulator database so the user could learn how and why to combine different clips with different surgical approaches. Another group[32,33] used the same system to train clinicians in minimally invasive surgical techniques.

The development of *haptic* technologies, which provide touch and pressure feedback, have significantly improved the tactile experience of simulators.[34, 35] Currently in use at the Jefferson Hospital for Neuroscience (JHN) is the Procedicus VIST™ simulator. This is a virtual reality simulator system that uses haptic feedback and metric assessment.[36, 37] It can be used to acquire and hone basic and complex neuroendovascular skills. The system utilizes a mannequin and two computer screens along with actual endovascular equipment. The computer allows the user to choose from a variety of different programs representing different pathologies, e.g. saccular aneurysms, fusiform aneurysms, carotid stenosis.

The simulator at JHN has been used by neurointerventional technologists, residents and fellows to develop proficiency in various procedures. This leads to increased benefits for practitioners, as they have improved skills and confidence when performing procedures on patients. The simulator has also improved patient safety as simulators have been shown to improve performance in endovascular procedures.[38]

Conclusion

The new era of evidence-based management of disease will continue to demand increased justification from physicians for the treatments they use. At the same time, ethical constraints limit the early testing of new technologies on patients. Animal models, when designed, validated and used appropriately, can be an invaluable resource in the development of life-saving technologies and techniques.

One reason to model a biological system is that construction of the model proves enough understanding to reproduce the system. It is presumably that same understanding that makes neurosurgical repair of the patient possible. A potential advantage of computational models is the opportunity to perform many virtual experiments in less time than it would take to perform one real experiment, but this depends on the power of the computer relative to the complexity of the model. An extension of this is using a computer to design optimized surgical plans and project the likely outcomes for patients in advance of surgery. The potential of computer models is restricted by the bottom limit of our ability to observe physiological processes and represent them on computer: as yet, we may not even have approached that limit.

References

1. German, W.J. and S.P. Black, Experimental production of carotid aneurysms. N Engl J Med, 1954. 250(3): p. 104-6.
2. Shin, Y.S., et al., Creation of four experimental aneurysms with different hemodynamics in one dog. AJNR Am J Neuroradiol, 2005. 26(7): p. 1764-7.
3. Massoud, T.F., et al., Experimental saccular aneurysms. I. Review of surgically-constructed models and their laboratory applications. Neuroradiology, 1994. 36(7): p. 537-46.
4. Massoud, T.F., et al., Experimental models of bifurcation and terminal aneurysms: construction techniques in swine. AJNR Am J Neuroradiol, 1994. 15(5): p. 938-44.
5. Spetzger, U., et al., Microsurgically produced bifurcation aneurysms in a rabbit model for endovascular coil embolization. J Neurosurg, 1996. 85(3): p. 488-95.
6. Guglielmi, G., et al., Experimental saccular aneurysms. II. A new model in swine. Neuroradiology, 1994. 36(7): p. 547-50.
7. Byrne, J.V. and N. Hubbard, A novel two-stage technique for construction of experimental aneurysms. AJNR Am J Neuroradiol, 2004. 25(2): p. 319-21.
8. Abrahams, J.M., et al., Biodegradable polyglycolide endovascular coils promote wall thickening and drug delivery in a rat aneurysm model. Neurosurgery, 2001. 49(5): p. 1187-93; discussion 1193-5.
9. Kirse, D.J., et al., Construction of a vein-pouch aneurysm at a surgically created carotid bifurcation in the rat. Microsurgery, 1996. 17(12): p. 681-9.
10. Young, P.H. and M.G. Yasargil, Experimental carotid artery aneurysms in rats: a new model for microsurgical practice. J Microsurg, 1982. 3(3): p. 135-46.
11. Tenjin, H., et al., Effect of Guglielmi detachable coils on experimental carotid artery aneurysms in primates. Stroke, 1995. 26(11): p. 2075-80.
12. Siekmann, R., et al., Modification of a previously described arteriovenous malformation model in the swine: endovascular and combined surgical/endovascular construction and hemodynamics. AJNR Am J Neuroradiol, 2000. 21(9): p. 1722-5.
13. Murayama, Y., T.F. Massoud, and F. Vinuela, Transvenous hemodynamic assessment of experimental arteriovenous malformations. Doppler guidewire monitoring of embolotherapy in a swine model. Stroke, 1996. 27(8): p. 1365-72.
14. Murayama, Y., T.F. Massoud, and F. Vinuela, Hemodynamic changes in arterial feeders and draining veins during embolotherapy of arteriovenous malformations: an experimental study in a swine model. Neurosurgery, 1998. 43(1): p. 96-104; discussion 104-6.
15. Massoud, T.F., Experimental arteriovenous malformations modeling in laboratory sheep versus swine. AJNR Am J Neuroradiol, 2000. 21(5): p. 985-90.
16. Massoud, T.F., et al., Laboratory simulations and training in endovascular embolotherapy with a swine arteriovenous malformation model. AJNR Am J Neuroradiol, 1996. 17(2): p. 271-9.
17. Jahan, R., et al., An arteriovenous malformation model for stereotactic radiosurgery research. Neurosurgery, 2007. 61(1): p. 152-9; discussion 159.
18. Pietila, T.A., et al., Animal model for cerebral arteriovenous malformation. Acta Neurochir (Wien), 2000. 142(11): p. 1231-40.
19. Bederson, J.B., et al., Intracranial venous hypertension and the effects of venous outflow obstruction in a rat model of arteriovenous fistula. Neurosurgery, 1991. 29(3): p. 341-50.
20. Hai, J., et al., A new rat model of chronic cerebral hypoperfusion associated with arteriovenous malformations. J Neurosurg, 2002. 97(5): p. 1198-202.
21. Altschuler, E., et al., Radiobiologic models for radiosurgery. Neurosurg Clin N Am, 1992. 3(1): p. 61-77.
22. Herman, J.M., et al., Genesis of a dural arteriovenous malformation in a rat model. J Neurosurg, 1995. 83(3): p. 539-45.
23. Megyesi, J.F., et al., In vivo animal models of cerebral vasospasm: a review. Neurosurgery, 2000. 46(2): p. 448-60; discussion 460-1.
24. Kim, M., et al., Quantification of hemodynamic changes induced by virtual placement of multiple stents across a wide-necked basilar trunk aneurysm. Neurosurgery, 2007. 61(6): p. 1305-12; discussion 1312-3.
25. Cebral, J.R., R.S. Pergolizzi, Jr., and C.M. Putman, Computational fluid dynamics modeling of intracranial aneurysms: qualitative comparison with cerebral angiography. Acad Radiol, 2007. 14(7): p. 804-13.
26. Cebral, J.R., et al., Characterization of cerebral aneurysms for assessing risk of rupture by using patient-specific computational hemodynamics models. AJNR Am J Neuroradiol, 2005. 26(10): p. 2550-9.
27. Hademenos, G.J. and T.F. Massoud, An electrical network model of intracranial arteriovenous malformations: analysis of variations in hemodynamic and biophysical parameters. Neurol Res, 1996. 18(6): p. 575-89.
28. Hademenos, G.J., T.F. Massoud, and F. Vinuela, A biomathematical model of intracranial arteriovenous malformations based on electrical network analysis: theory and hemodynamics. Neurosurgery, 1996. 38(5): p. 1005-14; discussion 1014-5.
29. Hecht, S.T., J.A. Horton, and C.W. Kerber, Hemodynamics of the central nervous system arteriovenous malformation nidus during particulate embolization. A computer model. Neuroradiology, 1991. 33(1): p. 62-4.
30. Kerber, C.W., S.T. Hecht, and K. Knox, Arteriovenous malformation model for training and research. AJNR Am J Neuroradiol, 1997. 18(7): p. 1229-32.
31. Wong, G.K., et al., Craniotomy and clipping of intracranial aneurysm in a stereoscopic virtual reality environment. Neurosurgery, 2007. 61(3): p. 564-8; discussion 568-9.
32. Kockro, R.A., et al., A collaborative virtual reality environment for neurosurgical planning and training. Neurosurgery, 2007. 61(5 Suppl 2): p. 379-91; discussion 391.
33. Stadie, A.T., et al., Virtual reality system for planning minimally invasive neurosurgery. Technical note. J Neurosurg, 2008. 108(2): p. 382-94.
34. Scalese, R.J. and S.B. Issenberg, Effective use of simulations for the teaching and acquisition of veterinary professional and clinical skills. J Vet Med Educ, 2005. 32(4): p. 461-7.
35. Scalese, R.J., V.T. Obeso, and S.B. Issenberg, Simulation technology for skills training and competency assessment in medical education. J Gen Intern Med, 2008. 23 Suppl 1: p. 46-9.
36. Neequaye, S.K., et al., Identification of skills common to renal and iliac endovascular procedures performed on a virtual reality simulator. Eur J Vasc Endovasc Surg, 2007. 33(5): p. 525-32.
37. Neequaye, S.K., et al., Endovascular skills training and assessment. J Vasc Surg, 2007. 46(5): p. 1055-64.
38. Aggarwal, R., et al., Virtual reality simulation training can improve inexperienced surgeons' endovascular skills. Eur Vasc Endovasc Surg, 2006. 31(6): p. 588-93.

Spinal Cord Stimulation for Control of Pain

Stephen Falowski, MD

Department of Neurological Surgery, Thomas Jefferson University, Philadelphia, Pennsylvania

Introduction

Spinal cord stimulation (SCS) is an adjustable, non-destructive procedure which delivers therapeutic doses of electrical current to the spinal cord for the management of neuropathic pain. The most common indications include post-laminectomy ("failed back surgery") syndrome, complex regional pain syndrome (CRPS), ischemic limb pain, and angina. Other reported applications include visceral/abdominal pain, cervical neuritis pain, spinal cord injury pain, post-herpetic neuralgia, and neurogenic thoracic outlet syndrome.

History

Advances in technology have driven the popularity of the field. Initially, the contact combinations could only be hardwired, and could not be reprogrammed after the pulse generator was implanted. In the beginning, only radio-frequency (RF) driven passive receivers were available. In the mid-seventies, the first implantable pulse generator powered by a lithium battery was introduced into the market. In 1980, the first percutaneous electrode was produced, and this could be reprogrammed non-invasively through an external transmitter.[1]

Mechanisms of Action

SCS began when Melzack and Wall[2] noted that stimulation of large peripheral nerve fibers could block the sensation of pain. In 1967, Shealy[3] inserted the first dorsal column stimulator in a human suffering from terminal metastatic cancer. Later approaches implanted electrodes via a laminectomy in the subarachnoid space, between the two layers of the dura, or in the epidural space.[4-6] Subsequently, less invasive percutaneous techniques were introduced.[7]

The exact mechanisms of action of SCS remain unclear, although computer modeling work has shed some light on the distribution of complex electrical fields within the spine.[8-13] Animal studies suggest that the SCS triggers the release of serotonin, substance P, and GABA within the dorsal horn.[14, 15] It is not known whether stimulation of different sites—peripheral nerves, dorsal columns, or supra-lemniscal pathways—will trigger equivalent mechanisms of action.

Indications

SCS has been successfully used for a variety of pain conditions (Table I). Experience suggests that, in selected patients, SCS can produce at least 50% pain relief in 50-60% of the implanted patients. Interestingly, with the proper follow-up care, these results can be maintained over several years.

Complex Regional Pain Syndrome (CRPS)

The implementation of SCS in individuals with CRPS type I is more difficult than with any other patients groups. There is risk of aggravating the original pain or causing a new pain at the implanted hardware site is greater than with any other diagnostic category mentioned. The CPRS-affected areas may be too widespread for effective stimulation**.**

In 1989, Barolat et al. reported reduction of pain in ten out of thirteen patients implanted. No patients in that series were made pain free but all ten reported a definite difference from stimulation.[16] In 1997, Kumar et al. reported on twelve patients with permanently implanted leads[17]: eight patients enjoyed near complete resolution of their symptoms and four also maintained good relief.

Kemler et al. reported 23 additional cases with 78% of the patients reporting improvement.[18] A later study[19] compared patients randomized to SCS to those treated with physical therapy. In the SCS group, 67% of patients experienced significant pain relief which persisted at 6 months. However, no functional improvement was observed in either group. A 5-year follow-up on the SCS group indicated that the effects of SCS diminished over time for these patients.[20]

Oakley and Weiner reported a prospective study of 19 patients with CRPS implanted with spinal cord stimulation systems.[21] Of the ten patients in whom detailed long-term efficacy data was available, three reported full relief from their pain and seven, partial beneficial relief.

Three additional prospective studies without matched controls have been reported.[21-23] Two of the studies reported success rates with an 84% overall success rate. The third study by Calvillo et al.[22] reported a significant improvement in pain scores and a >50% reduction in narcotic use by 44% of subjects. In eight retrospective studies the overall success rate was 84% 192 patients.[24]

Post-laminectomy syndrome aka Failed back surgery syndrome (FBSS)

Post-laminectomy syndrome is a broad term which can include pain localized to the lower lumbar region or the buttocks, persistent radicular pain, or diffuse lower extremity pain. Most published series distinguish between back and leg pain, but seldom define the details of the pain syndromes. SCS is accepted in the treatment of leg pain, but its widespread use for relief of pain in the lower lumbar area still remains to be defined.

A technical challenge has been to provide stimulation in the low back, where paresthesia is often replaced over time by an unpleasant segmental band of stimulation from the thoracic roots. Previous pioneering work by Jay Law[25, 26] has shown that stimulation in the low back can be obtained only if one uses multiple arrays of closely spaced bipolar electrodes at T9-T10. North et al. have shown that one single quadripolar electrode in midline has the ability to stimulate the axial low back.[27] Flanking the cathode by lateral anodes also appears to increase the discomfort threshold theoretically.[28]

Marchand et al.[29] conducted a prospective randomized controlled study examining patients with at least one prior surgery for chronic back pain secondary to trauma. Each patient used a SCS and acted as his or her own control.

Although a small trial, with only eight patients, pain scores were significantly reduced with SCS compared to placebo stimulation.

The longitudinal studies by North showed that in patients with post-surgical lumbar arachnoid or epidural fibrosis without surgically remediable lesions, SCS provided at least 50% pain relief in 53% of patients at 2.2 years.[30] North et al. also conducted a study randomizing patients with FBSS to either repeat back surgery or SCS surgery,[33] allowing crossover after six months. Ten of fifteen patients crossed over from back surgery to SCS, while only two of twelve patients crossed over from SCS to back surgery.

Turner et al.[31] systematically reviewed a total of 41 articles from 1966 to 1994 and noted that approximately 50-60% of patients with post-laminectomy greater than 50% pain relief was attained from the use of SCS. In 1996, Burchiel et al. conducted a prospective multi-center study with one year follow-up and also reported 55% successful stimulation.[32]

Angina

There are well documented reports in the literature revealing uniformly good results using SCS to relieve anginal pain.[34-38] Further, the results have been maintained in long term follow-up and have been substantiated by a reduction in the intake of nitrates as well. Interestingly, other findings have supported the evidence that SCS has effects that go beyond pain relief. The observations that there is less ST segment depression and that the exercise capacity, the time to angina and the recovery time all improve with stimulation may suggest that there is a reduction in ischemia. In a positron emission tomography study, a redistribution of myocardial flow in favor of ischemic parts of the myocardium has been demonstrated as a long term effect of spinal cord stimulation, both at rest and after pharmacologic stress induction.[39]

Vulink et al. conducted a prospective study on quality of life changes in patients with refractory angina pectoris implanted with SCS. They found that both the pain and the health aspects of quality of life improved significantly after 3 months of SCS. Further, social, mental and physical aspects of quality of life were found improved after one year of SCS.[40]

Hautvast et al.[41] implanted SCS in patients with stable angina pectoris and randomized them. One group's remained inactivated while the other group was instructed to use the stimulator three times per day for one hour and with any angina attack. At 6 weeks, compared with controls, the treatment group had increased exercise duration and time to angina, and decreased anginal attacks and sublingual nitrate consumption. Also, observed was a decrease in ischemic episodes on EKG, as well as a decrease in observed ST segment depressions on exercise EKG. There was an increase in perceived quality of life and decrease in pain.

Mannheimer et al.[42] randomized 104 patients accepted for CABG to receive either CABG (n=51) or SCS (n=53) in the ESBY study. This study demonstrated that patients randomized to SCS showed a greater than 30% improvement in Nottingham Health Profile (NHP) scores compared with baseline, which was significant and comparable to the improvement shown by patients randomized to CABG.[43] These results were consistent on follow up after four years. It is important to know that the five-year mortality of 27.9% in the ESBY study was similar between those receiving SCS and those who received CABG, with no difference in the percentage of cardiac deaths. The ESBY study showed that cardiac events were similar across the groups, but that there was significantly more cerebrovascular events observed in the CABG group.

Both groups experienced a significant reduction in both the number of angina attacks and the consumption of nitrates. There was no significant intergroup difference regarding these parameters. In another prospective study of 104 patients who underwent SCS implantation for refractory angina pectoris there was a significant decrease in angina episodes at rest, angina episodes with activity, and total angina episodes [44]

DeJongste et al.[36] randomized seventeen patients with angina to an active treatment group (i.e. SCS implantation) and a control group. The control group was followed for two months followed by SCS implantation. Both groups were followed for a total of 12 months. This study also revealed a statistically significant reduction in the incidence of angina attacks and in the consumption of nitrates.

Five additional studies are reported to be prospective but without matched controls.[36, 40, 45-48] Each of these revealed significant benefit from spinal cord stimulation. The benefit indices ranged from reduction in angina attacks, decrease nitrate consumption, decrease in NYHA grade and improvement in NHP grade.

Andersen et al. reported that out of 45 patients treated with SCS for anginal pain, there were three who had also survived a myocardial infarction[49]: all three patients noticed the pain to be different and unrelieved with SCS and all patients correctly guessed that the pain was due to a myocardial infarction. The authors concluded that SCS reduces the severity of anginal attack, but was unable to suppress the conduction and perception of cardiac pain signals which act as alarm signals of cardiac distress. Similarly, Murray et al. have shown that SCS for refractory angina is effective in preventing hospital admissions without masking the ischemic symptoms or leading to silent infarction.[50]

How SCS reduces angina is unclear. Hautvast et al.[41] demonstrated no significant changes in heart rate variability after 6 wks, concluding that autonomic modulation of heart rate may not be the mechanism of action. There SCS may reduces myocardial ischemia via homogenization of myocardial blood perfusion.[39,51] SCS can improve lactate metabolism in the heart muscle, and lactate metabolism, oxygen demand and blood flow in the coronary sinus.[52] SCS does not effect variability in heart rates or cardiac arrhythmias.[35, 53, 54]

We do not know whether the pain relief is due to direct depression of the nociceptive signals in the spinal cord or whether there is secondary gain from a reduction in the ischemia.[55,56] Foreman has shown that dorsal column stimulation inhibits the activity of spinothalamic tracts cells evoked by activation of the cardiac sympathetic afferents or by intracardiac bradykinin.[14] On the other hand the stimulation might producing a prolonged inhibition of the hyperactive sympathetic system, as was shown experimentally in the rat by Linderoth et al.[15] The most appropriate electrode location for the treatment of angina pectoris is most likely the lower cervical and upper thoracic region, although some have reported successful higher cervical placements.[42] Another consideration is continuous versus cyclical use of SCS. In practice, patients using SCS for angina pectoris often use a low intensity stimulation for several hours per day for prophylactic purposes.[57] Recently, a randomized control study demonstrated improvement in functional status and symptoms in treatment arms with conventional or sub threshold stimulation in comparison to a low output placebo treatment arm.[58]

Abdominal/Visceral Pain Syndromes

Approximately 20% of the population in United States have abdominal pain. There are many etiologies for abdominal pain including

gastrointestinal, genitourinary, musuloskeletal and nervous systems. Treatment modalities have included cognitive- behavioral, physical, and pharmacological therapies. Other more invasive therapies have extended to include celiac plexus blocks and celiac ganglia destruction. Some studies have demonstrated some localization in the spinal cord for visceral pain secondary to malignancy. Midline myelotomy through the dorsal columns at the level of T10 has shown success in eight patients with refractory pelvic cancer pain. This was also demonstrated in animal studies where dorsal column activity was observed in pelvic visceral nociception.[59]

Several studies have demonstrated the benefit of SCS in abdominal visceral disease. Ceballos et al.[60] demonstrated reduction in pain scores and decrease in narcotic use in a patient treated for mesenteric ischemia. Krames et al.[61] described a patient treated for irritable bowel syndrome who was developing escalating pain and diarrhea. In the first 6 months there was a subjective decrease in pain on a 10-point scale from 9/10 to 2/10, with only two diarrhea episodes and with significant reduction in pain medications. There was some return of pain after ten month follow-up, but still a significant reduction in diarrhea. Khan et al.[62] reported on the largest series with nine patients with refractory abdominal pain, all of whom had a significant improvement in pain scores as well as decreased narcotic use at six to eight month follow up.

Tiede et al. described treatment of refractory abdominal pain in two patients. Both patients had a significant history including multiple abdominal surgeries and failed conservative measures. Each patient had an element of postprandial abdominal pain with associated nausea and vomiting. In both patients the leads were placed at the T2 level with significant improvement in pain, decreased narcotic use and increase functioning, such as return to work[63]. Kapur et al.[64] recently described relief of abdominal pain associated with colchicine intolerant or resistant patients with familial Mediterranean fever by placement of the electrodes at the lower thoracic levels.

More recent studies have looked at the treatment of visceral pelvic pain with reference to the dorsal columns and spinal cord stimulation. Kapural et al.[65] reported on the value of neurostimulation for chronic visceral pelvic pain in six female patients with the diagnosis of long-standing pelvic pain. These patients had a history of endometriosis, multiple surgical explorations, and dyspareunia. At an average follow up of 30 months there was a significant decrease in the pain score with an average of more than 50% pain relief, with a decrease in opiate use.

Visceral innervation follows the embryologic origin and location of the viscera and is arranged in viscerotomes, analogous to cutaneous dermatomes.[66] The viscera obtain their innervation via the sympathetic and parasympathetic pathways. The parasympathetics carry their afferents to anterior and posterior vagal trunks and are therefore not as amendable to spinal cord stimulation. The sympathetics carry nociceptive information from the viscera to spinal nerve roots making them a more viable target. The sympathetic afferents in the lower six thoracic and the upper three lumbar spinal segments have been shown to transmit painful impulses from the viscera [67].

Equipment (Electrodes and pulse generators)

There are various implantable technologies existing for SCS. These include trial percutaneous electrodes, permanent percutaneous electrodes, permanent plate electrodes, totally implantable rechargeable and non-rechargeable pulse generators (IPG) and radiofrequency (RF) driven pulse generators.

Percutaneous electrodes

Percutaneous electrodes can be inserted without much dissection and can easily be removed in the implanting physician's office. During implantation, these electrodes can be advanced over several segments in the epidural space, allowing testing of several spinal cord levels to assess for optimal electrode position.

Contemporary percutaneous electrodes are slim electrodes, only a few millimeters in diameter and containing four or eight contacts (referred to as either quadripolar or octopolar electrodes). Choosing the particular electrode entails deciding how many segments of the spinal cord are to be covered, with larger spacing allowing broader coverage. Alternatively, closer spacing allows better steering and electric field shaping. Additionally, multiple parallel electrodes and different configuration matrices can be constructed which can create extremely focused electrical fields.

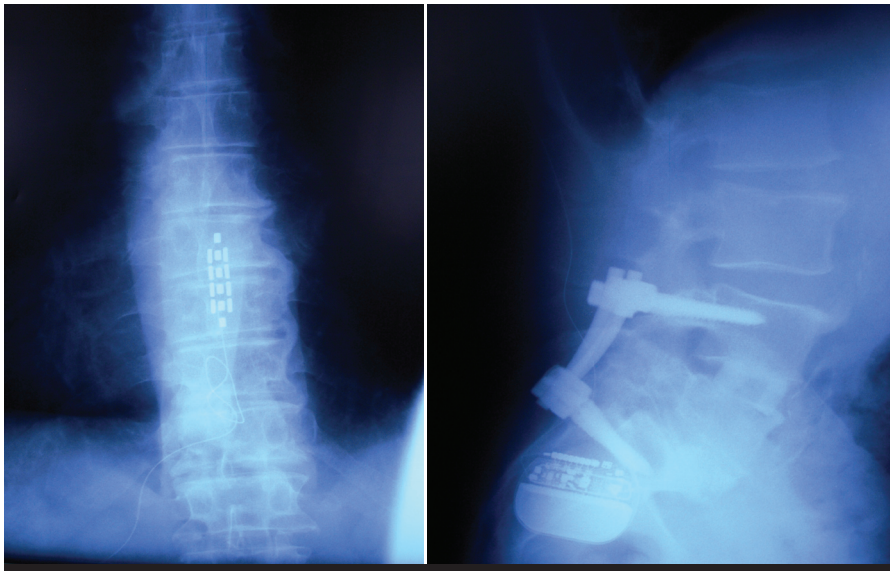


Figure 2

The above graphic on the left shows a patient who had an L4,5 and L5,S1 anterior posterior fusion for back and leg pain from lumbar spondylolisthesis. She continued to have both back and leg pain despite a successful lumbar reconstruction. An ANS (Advanced Neuromodulation System) tripole electrode was implanted in the thoracic spine. There are three columns of electrodes. The middle covers the back and the lateral columns provide stimulation into the legs. The electrode was placed in the operating room with fluoroscopy and intra-operative EMG monitoring under general anesthesia.

Plate electrodes

Plate-type electrodes (or ribbon electrodes, paddle electrodes, or laminotomy electrodes) require a surgical procedure, laminotomy, and implantation under direct vision. Implantation under direct vision may be safer in the upper thoracic and cervical areas, where there is a risk of damaging the spinal cord with the large bore Touhy needle. Most implants can be done through a skin incision between 2.5 cm and 4 cm long, depending on the size of the patient and spinal anatomy. The amount of bony removal is usually minimal.

The main advantage of plate electrodes is their greater inherent stability, with less to migrate. Plate electrodes are also more energy efficient. Multiple arrays or different electrode configurations can be constructed with plate electrodes. As with percutaneous leads, there are varying lengths, and shapes—such as curved leads and hinged leads, all designed to help facilitate insertion and tailor the electrode selection to the patient. They are a preferred option in the case of previous spine surgery at the implant levels.

North et al. have published on comparison between plate and percutaneous electrodes.[68] Laminectomy electrode placement, although more invasive than percutaneous placement, yielded significantly better clinical results in patients with failed back surgery syndrome at up to 3-year follow up. Clinical success was defined as at least 50% pain relief and patient satisfaction with treatment. Secondary outcome measures were ability to perform various activities of daily living, neurological function, and analgesic use. There is some theoretical evidence that shaping of the electrical field is possible with even more complex electrode arrays. Holsheimer et al.[69] concluded that the transverse tripolar system enabled finer control of paresthesia. Electrical field steering could change the paresthesia area completely. Using transverse tripolar configurations increases the threshold for stimulation of dorsal roots. This results in a wider therapeutic range, wider paresthesia coverage, and a greater probability to fully cover the painful area with paresthesia.

Rechargeable and Non-rechargeable Pulse Generators and Radio-Frequency Receivers

Electrical stimulation consists of rectangular pulses delivered to the epidural space through implanted electrode via a power source. Two basic types of systems are currently available: an Internal Pulse Generator (IPG; also called the battery) or a radiofrequency (RF) coupled pulse generator with an implantable receiver. The later has largely fallen out of favor due to the inconveniences of having an external power source. However, the advent of the totally implantable, rechargeable pulse generator has surmounted the power requirement issues, which were previously the main RF advantage.

The totally implantable pulse generator contains a lithium battery. Activation and control occur through an external transcutaneous telemetry device. The IPG can be turned on and off through a small controller which the patient can carry. The controller also allows some control over the stimulation parameters. More extensive control of the unit can be achieved through a small portable unit which can be programmed by the physician. Life span of the battery varies with usage and with the utilized parameters (voltage, rate, pulse width, etc.). Most patients can expect that the battery will last from 2.5 to 4.5 years. Available lithium powered pulse generators allow stimulation with fine control of stimulation amplitude, pulse width and rate. Replacement of the battery requires a surgical procedure which is usually performed on an outpatient basis.

Radio frequency (RF) driven systems, consist of a passive receiver, implanted subcutaneously, and a transmitter which is worn externally. An antenna applied to the skin in correspondence of the receiver is connected to the transmitter, which sends the stimulation signals transcutaneously. RF systems have the inconvenience of having to wear the antenna and the radio-receiver, replacing the batteries on a regular basis, and ensuring proper contact of the antenna on the skin. These issues may be critical for individuals with limited upper-limb motor function. Other patients, particularly individuals who have reflex sympathetic dystrophy (RSD), may not tolerate the antenna taped to the skin. However, what one loses in convenience, however, is gained in power and flexibility. Currently only RF systems can provide a stimulation rate up to 1,400 Hz. This might be beneficial in some patients with neuropathic chronic pain syndromes[70] as well as in patients with extrapyramidal motor disorders.

Complications

With the proper expertise, permanent complications are rare.[71] The most serious complications are paralysis or other severe neurological deficits, which are risks inherent in any spine surgery. Infection of the implanted hardware has occurred with a 3-5% rate. Persistent pain at the implant site has been seen in about 5% of patients. Recalcitrant cerebrospinal fluid leakage has been encountered in a few patients, requiring multiple surgical revisions. Breakage or malfunction of the implanted hardware, particularly the electrodes and the subcutaneous extension cables has been encountered in about 10% of the implanted systems. Painful stimulation, necessitating either repositioning or removal of the electrode, has also been reported in a number of cases.

Conclusions

The treatment of chronic pain remains challenging. Spinal cord stimulation has been performed for over 30 years, and slow but steady progress with this technology has been made. As the equipment and stimulation parameters are improved, selection criteria have been better defined and are slowly being expanded. More importantly, experience in the technique and the equipment has made SCS a much more reliable and safe modality. Like all the modalities performed for chronic pain management, its results are favorable. It is important to remember that the goal of neurostimulation is to reduce pain, rather than to eliminate pain. It has been shown to have a 50% improvement in pain relief, and reduce the use of more medications Very few other invasive modalities can claim this success rate with a few years of follow-up.

Careful follow-up of the patients is necessary for successful long-term satisfaction. Equipment related problems can arise at any time after implantation, such as discomfort at the pulse generator/radio receiver site, electrode breakage or migration, infection, etc., and an open dialogue with the patients is vital for the continuing successful implementation of the modality. Spinal cord stimulation has earned a well established and firm role in contemporary chronic pain management.

References

1. Waltz, J.M., Computerized percutaneous multi-level spinal cord stimulation in motor disorders. Appl Neurophysiol, 1982. 45(1-2): p. 73-92.
2. Melzack, R. and P.D. Wall, Pain mechanisms: a new theory. Science, 1965. 150(699): p. 971-9.
3. Shealy, C.N., J.T. Mortimer, and J.B. Reswick, Electrical inhibition of pain by stimulation of the dorsal columns: preliminary clinical report. Anesth Analg, 1967. 46(4): p. 489-91.
4. Hoppenstein, R., Electrical stimulation of the ventral and dorsal columns of the spinal cord for relief of chronic intractable pain: preliminary report. Surg Neurol, 1975. 4(1): p. 187-94.

5. Larson, S.J., et al., A comparison between anterior and posterior spinal implant systems. Surg Neurol, 1975. 4(1): p. 180-6.
6. Lazorthes, Y., J.C. Verdie, and L. Arbus, [Anterior and posterior medullary analgesic stimulation, using a percutaneous implantation technic]. Acta Neurochir (Wien), 1978. 40(3-4): p. 277-83.
7. Dooley, D. Percutaneous electrical stimulation of the spinal cord. in Assoc Neurol Surg. 1975. Bal Harbour, FL.
8. Coburn, B., Electrical stimulation of the spinal cord: two-dimensional finite element analysis with particular reference to epidural electrodes. Med Biol Eng Comput, 1980. 18(5): p. 573-84.
9. Coburn, B., A theoretical study of epidural electrical stimulation of the spinal cord--Part II: Effects on long myelinated fibers. IEEE Trans Biomed Eng, 1985. 32(11): p. 978-86.
10. Coburn, B. and W.K. Sin, A theoretical study of epidural electrical stimulation of the spinal cord--Part I: Finite element analysis of stimulus fields. IEEE Trans Biomed Eng, 1985. 32(11): p. 971-7.
11. Holsheimer, J., et al., Significance of the spinal cord position in spinal cord stimulation. Acta Neurochir Suppl, 1995. 64: p. 119-24.
12. Holsheimer, J. and J.J. Struijk, How do geometric factors influence epidural spinal cord stimulation? A quantitative analysis by computer modeling. Stereotact Funct Neurosurg, 1991. 56(4): p. 234-49.
13. Holsheimer, J. and W.A. Wesselink, Effect of anode-cathode configuration on paresthesia coverage in spinal cord stimulation. Neurosurgery, 1997. 41(3): p. 654-9; discussion 659-60.
14. Foreman, R.D., et al., Effects of dorsal column stimulation on primate spinothalamic tract neurons. J Neurophysiol, 1976. 39(3): p. 534-46.
15. Linderoth, B., et al., Dorsal column stimulation induces release of serotonin and substance P in the cat dorsal horn. Neurosurgery, 1992. 31(2): p. 289-96; discussion 296-7.
16. Barolat, G., R. Schwartzman, and R. Woo, Epidural spinal cord stimulation in the management of reflex sympathetic dystrophy. Stereotact Funct Neurosurg, 1989. 53(1): p. 29-39.
17. Kumar, K., R.K. Nath, and C. Toth, Spinal cord stimulation is effective in the management of reflex sympathetic dystrophy. Neurosurgery, 1997. 40(3): p. 503-8; discussion 508-9.
18. Kemler, M.A., et al., Electrical spinal cord stimulation in reflex sympathetic dystrophy: retrospective analysis of 23 patients. J Neurosurg, 1999. 90(1 Suppl): p. 79-83.
19. Kemler, M.A., et al., Spinal cord stimulation in patients with chronic reflex sympathetic dystrophy. N Engl J Med, 2000. 343(9): p. 618-24.
20. Kemler, M.A., et al., Spinal cord stimulation for chronic reflex sympathetic dystrophy--five-year follow-up. N Engl J Med, 2006. 354(22): p. 2394-6.
21. Oakley, J. and R. Weiner, Spinal cord stimulation for complex regional pain syndrome: a prospective study of 19 patients at two centers. Neuromodulation, 1999. 2: p. 47-50.
22. Calvillo, O., et al., Neuroaugmentation in the treatment of complex regional pain syndrome of the upper extremity. Acta Orthop Belg, 1998. 64(1): p. 57-63.
23. Ebel, H., et al., Augmentative treatment of chronic deafferentation pain syndromes after peripheral nerve lesions. Minim Invasive Neurosurg, 2000. 43(1): p. 44-50.
24. Cameron, T., Safety and efficacy of spinal cord stimulation for the treatment of chronic pain: a 20-year literature review. J Neurosurg, 2004. 100(3 Suppl Spine): p. 254-67.
25. Law, J.D., Targeting a spinal stimulator to treat the 'failed back surgery syndrome'. Appl Neurophysiol, 1987. 50(1-6): p. 437-8.
26. Law, J.D., Clinical and technical results from spinal stimulation for chronic pain of diverse pathophysiologies. Stereotactic & Functional Neurosurgery, 1992. 59(1-4): p. 21-4.
27. North, R.B., et al., Spinal cord stimulation electrode design: A prospective randomized, controlled trial comparing percutaneous and laminectomy electrodes. Stereotact Funct Neurosurg, 1999. 73(1-4): p. 134.
28. Sharan, A., et al. Selective Dorsal Column Activation With Three Column Electrode Arrays Using Percutaneous and Paddle Leads. in 2007 AANS Annual Meeting. 2007. Washington, DC.
29. Marchand, S., et al., The effects of dorsal column stimulation on measures of clinical and experimental pain in man. Pain, 1991. 45(3): p. 249-57.
30. 5-year follow-up after spinal cord stimulator implantation. Neurosurgery, 1991. 28(5): p. 692-9.
31. Turner, J.A., J.D. Loeser, and K.G. Bell, Spinal cord stimulation for chronic low back pain: a systematic literature synthesis. Neurosurgery, 1995. 37(6): p. 1088-95; discussion 1095-6.
32. Burchiel, K.J., et al., Prospective, multicenter study of spinal cord stimulation for relief of chronic back and extremity pain. Spine, 1996. 21(23): p. 2786-94.
33. North, R.B., D.H. Kidd, and S. Piantadosi, Spinal cord stimulation versus reoperation for failed back surgery syndrome: a prospective, randomized study design. Acta Neurochir Suppl, 1995. 64: p. 106-8.
34. Augustinsson, L.E., Spinal cord electrical stimulation in severe angina pectoris: surgical technique, intraoperative physiology, complications, and side effects. Pacing Clin Electrophysiol, 1989. 12(4 Pt 2): p. 693-4.
35. de Jongste, M.J., et al., Effects of spinal cord stimulation on myocardial ischaemia during daily life in patients with severe coronary artery disease. A prospective ambulatory electrocardiographic study. Br Heart J, 1994. 71(5): p. 413-8.
36. de Jongste, M.J., et al., Efficacy of spinal cord stimulation as adjuvant therapy for intractable angina pectoris: a prospective, randomized clinical study. Working Group on Neurocardiology. J Am Coll Cardiol, 1994. 23(7): p. 1592-7.
37. Mannheimer, C., et al., Epidural spinal electrical stimulation in severe angina pectoris. Br Heart J, 1988. 59(1): p. 56-61.
38. Sanderson, J.E., et al., Epidural spinal electrical stimulation for severe angina: a study of its effects on symptoms, exercise tolerance and degree of ischaemia. Eur Heart J, 1992. 13(5): p. 628-33.
39. Hautvast, R.W., et al., Effect of spinal cord stimulation on myocardial blood flow assessed by positron emission tomography in patients with refractory angina pectoris. Am J Cardiol, 1996. 77(7): p. 462-7.
40. Vulink, N., et al., The effects of spinal cord stimulation on quality of life in patients with therapeutically chronic refractory angina pectoris. Neuromodulation, 1999: p. 33-40.
41. Hautvast, R.W., et al., Spinal cord stimulation in chronic intractable angina pectoris: a randomized, controlled efficacy study. Am Heart J, 1998. 136(6): p. 1114-20.
42. Mannheimer, C., et al., Electrical stimulation versus coronary artery bypass surgery in severe angina pectoris: the ESBY study. Circulation, 1998. 97(12): p. 1157-63.
43. Ekre, O., et al., Long-term effects of spinal cord stimulation and coronary artery bypass grafting on quality of life and survival in the ESBY study. Eur Heart J, 2002. 23(24): p. 1938-45.
44. Di Pede, F., et al., Immediate and long-term clinical outcome after spinal cord stimulation for refractory stable angina pectoris. Am J Cardiol, 2003. 91(8): p. 951-5.
45. Andersen, C., Complications in spinal cord stimulation for treatment of angina pectoris. Differences in unipolar and multipolar percutaneous inserted electrodes. Acta Cardiol, 1997. 52(4): p. 325-33.
46. Bagger, J.P., B.S. Jensen, and G. Johannsen, Long-term outcome of spinal cord electrical stimulation in patients with refractory chest pain. Clin Cardiol, 1998. 21(4): p. 286-8.
47. Eliasson, T., et al., Safety aspects of spinal cord stimulation in severe angina pectoris. Coron Artery Dis, 1994. 5(10): p. 845-50.
48. Sanderson, J.E., et al., Spinal electrical stimulation for intractable angina--long-term clinical outcome and safety. Eur Heart J, 1994. 15(6): p. 810-4.
49. Andersen, C., P. Hole, and H. Oxhoj, Does pain relief with spinal cord stimulation for angina conceal myocardial infarction? Br Heart J, 1994. 71(5): p. 419-21.
50. Murray, S., et al., Spinal cord stimulation significantly decreases the need for acute hospital admission for chest pain in patients with refractory angina pectoris. Heart, 1999. 82(1): p. 89-92.
51. Gonzalez-Darder, J., V. Gonzalez-Martinez, and P. Canela-Moya, Cervical Spinal Cord Stimulation in the Treatment of Severe Angina Pectoris. Neurosurgical Quarterly, 1998. 8: p. 16-23.Augustinsson, L.E., T. Eliasson, and C. Mannheimer, Spinal cord stimulation in severe angina pectoris. Stereotactic & Functional Neurosurgery, 1995. 65(1-4): p. 136-41.
52. Andersen, C., Does heart rate variability change in angina pectoris patients treated with spinal cord stimulation? Cardiology, 1998. 89(1): p. 14-8.
53. Hautvast, R.W., et al., Effect of spinal cord stimulation on heart rate variability and myocardial ischemia in patients with chronic intractable angina pectoris--a prospective ambulatory electrocardiographic study. Clin Cardiol, 1998. 21(1): p. 33-8.
54. Meller, S.T. and G.F. Gebhart, A critical review of the afferent pathways and the potential chemical mediators involved in cardiac pain. Neuroscience, 1992. 48(3): p. 501-24.
55. Thamer, V., et al., Pain and myocardial ischemia: the role of sympathetic activation. Basic Res Cardiol, 1990. 85 Suppl 1: p. 253-66.
56. Norrsell, H., et al., Effects of spinal cord stimulation and coronary artery bypass grafting on myocardial ischemia and heart rate variability: further results from the ESBY study. Cardiology, 2000. 94(1): p. 12-8.
57. Eddicks, S., et al., Thoracic spinal cord stimulation improves functional status and relieves symptoms in patients with refractory angina pectoris: the first placebo-controlled randomised study. Heart, 2007. 93(5): p. 585-90.
58. Hirshberg, R.M., et al., Is there a pathway in the posterior funiculus that signals visceral pain? Pain, 1996. 67(2-3): p. 291-305.
59. Ceballos, A., et al., Spinal cord stimulation: a possible therapeutic alternative for chronic mesenteric ischaemia. Pain, 2000. 87(1): p. 99-101.
60. Krames, E. and D. Mousad, Spinal cord stimulation reverses pain and diarrheal episodes of irritable bowel syndrome: a case report. Neuromodulation, 2004. 7: p. 82-88.
61. Khan, Y., S. Raza, and E. Khan, Application of spinal cord stimulation for the treatment of abdominal visceral pain syndromes: case reports. Neuromodulation, 2005. 8: p. 14-27.
62. Tiede, J.M., et al., The use of spinal cord stimulation in refractory abdominal visceral pain: case reports and literature review. Pain Pract, 2006. 6(3): p. 197-202.
63. Kapur, S., H. Mutagi, and J. Raphael, Spinal cord stimulation for relief of abdominal pain in two patients with familial Mediterranean fever. Br J Anaesth, 2006. 97(6): p. 866-8.
64. Kapural, L., et al., Spinal cord stimulation is an effective treatment for the chronic intractable visceral pelvic pain. Pain Med, 2006. 7(5): p. 440-3.
65. Ness, T.J. and G.F. Gebhart, Visceral pain: a review of experimental studies. Pain, 1990. 41(2): p. 167-234.
66. Mitchell, G., Anatomy of the autonomic nervous system. 1953, Edinburgh: E & S Livingstone.
67. North, R.B., et al., Spinal cord stimulation electrode design: a prospective, randomized, controlled trial comparing percutaneous with laminectomy electrodes: part II-clinical outcomes. Neurosurgery, 2005. 57(5): p. 990-6; discussion 990-6.
68. Holsheimer, J., et al., Clinical evaluation of paresthesia steering with a new system for spinal cord stimulation. Neurosurgery, 1998. 42(3): p. 541-7; discussion 547-9.
69. Alo, K., et al. The treatment of refractory reflex sympathetic dystrophy with higher frequency tripolar dual octrode spinal cord stimulation: a case report. . in Abstracts of the 4th International Congress of the INS. 1998.
70. Barolat, G., W. Peacock , and L. Staudt, Pain and spasticity, in Spine Surgery: Techniques, Complications Avoidance and Management, E. Benzel, Editor. 1999, Churchill Livingstone. p. 863-876.

Primary Stroke Centers: Their Role and Impact on Acute Stroke Management

Carissa C. Pineda MD[1] and and Jeannee Birch RN, BSN[2]

Department of [1]Neurology and [2]Department of Performance Improvement, Thomas Jefferson University, Philadelphia, Pennsylvania

The management of acute ischemic stroke has been recognized as a significant medical problem. Stroke remains to be the third leading cause of death and the leading cause of long-term disability; 80% of all strokes are ischemic (a blood clot disrupts blood flow), and the rest are hemorrhagic (a blood vessel ruptures in the brain). Nearly 1 in 15 deaths in 2003 were the result of a stroke.[1] Approximately 750,000 new strokes occur annually, of which 250,000 result in the deaths in the United States alone. The latest estimates for stroke costs total to about $30 billion in direct costs, and $20 billion are in indirect costs.[2] If nothing is done about this disease, the annual incidence of strokes is expected to reach 1.1 million by the year 2015.

Despite recent advances in its diagnosis and treatment, a population of the general public still have difficulty recognizing stroke symptoms, and are unaware that stroke is indeed a medical emergency.[3] The two major causes of delay in treatment are mainly failure to appropriately recognize the symptoms that indicate stroke (numbness, dizziness, confusion, or headache), and failure to access a medical facility that is equipped to handle acute strokes. In an effort to raise awareness, and the level of care provided to stroke patients, stroke center standards were created and hospitals that met the strict criteria were then selected. However, many hospitals do not have the necessary infrastructure and organization needed to triage and treat patients with stroke rapidly and efficiently.[4]

Seeing that the model of trauma centers were effective both in the rural and urban settings, the concept of acute stroke centers were then explored. The Brain Attack Coalition, which is a multidisciplinary group involved in stroke care, set out to establish guidelines for stroke centers. The role of a *Primary Stroke Center* is to be able to stabilize and provide emergency care for the acute stroke patients. Depending on the complexity of the case, they would either admit the patient, or transfer them to a comprehensive stroke center. On the other hand, the *Comprehensive Stroke Center* should be able to provide complete care to any complex patient; they should have the necessary infrastructure, both in personnel and equipment.

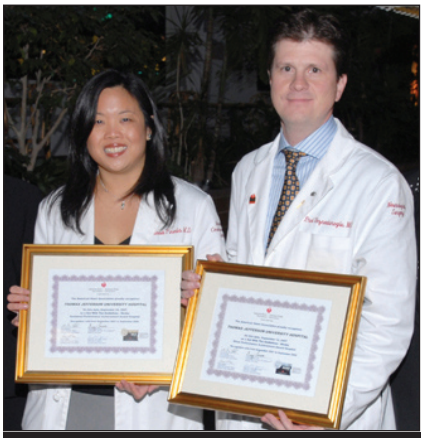
The main goals and benefits of a certified stroke center are the following:

- Improve the efficiency of patient care
- Provide access to the most up to date stroke therapies, which ultimately will lead to fewer peristroke complications
- Increase the number of patients discharged home than to skilled nursing facilities
- Decrease the length of hospital stay.

By accomplishing all of the above, the cost to the healthcare system will be reduced, and hopefully, patient satisfaction should also increase.

Although advances in stroke treatment have moved forward over the past years, the main limitation remains to be the prompt and proper treatment. Thrombolytic therapy, which has been FDA approved for over 10 years now remains to be the mainstay of acute treatment, however, only about 3% of patients with acute strokes are being treated. Another important goal of stroke centers is to provide access to these acute therapies.

The Brain Attack Coalition and American Stroke association have developed a framework of requirements needed to establish a Primary Stroke Center. These elements are based on the use of standardized method of delivering evidence based clinical care with known improvement of patient outcome. The Joint Commission has recognized the effectiveness of these measures and has based their criteria for Primary Stroke Center Certification on the recommendations listed in the accompanying table.[6-8]



Drs. Erol Veznedaroglu (Neurosurgery) and Carissa Pineda (Neurology) with the Gold and Silver Performance Awards from the American Heart Association for maintained high standards in care and management of stroke patients.

In this decade, the integration of multidisciplinary approach in the treatment of strokes will occur within the structure of a stroke center. Establishing a Stroke Center requires coordination of efforts throughout the Health System. Even hospitals with limited resources can positively impact the outcome of a stroke patient. Smaller healthcare centers can develop a "Stroke Network" with larger healthcare centers to provide comprehensive care. The most crucial component of stroke care is time—"Lost time equals lost brain" is the slogan, as brain damage progresses the blood flow is cut off. Rapid evaluation and referral with "Treat and Transfer" protocols can be established at any hospital that has the ability to provide Emergency Services. With new therapies emerging for stroke treatment, this could significantly impact all patients having access to cutting edge stroke care.

The American Heart Association's Performance Awards for hospitals is based on 85% compliance for patients in each of the following

items. The Silver Award is given to institutions who meet goals for 12 months and the Gold Award, for 24 months.

- Percent of acute ischemic stroke patients who arrive at the ED within 120 minutes (two hours) of onset of stroke symptoms who receive IV t-PA within 180 minutes (three hours) of onset of stroke symptoms
- Percent of ischemic stroke or TIA patients who receive antithrombotic medication within 48 hours of hospitalization
- Percent of ischemic stroke or TIA patients discharged on antithrombotics (e.g., warfarin, aspirin, other antiplatelet drug)
- Percent of ischemic stroke or TIA patients with atrial fibrillation who are discharged on anticoagulation therapy (warfarin/Coumadin or heparin/heparinoids) unless an absolute or relative contraindication exists
- Percent of patients at risk for DVT who received DVT prophylaxis by the second hospital day
- Percent of ischemic stroke or TIA patients with LDL >= 100 mg/dL OR on cholesterol reducer prior to admission who are discharged on cholesterol reducing drugs
- Percent of smokers who receive smoking cessation advice or medication (e.g., Nicoderm or Zyban) at discharge

These steps are all based on published evidence assessing modifiable risk factors which affect the likelihood of stroke.[9, 10] Any one of these risk factors can double the chance of a stroke recurring.

Acute Stroke Team

This is composed of physicians and health care professionals with experience and proper training in management of acute strokes. They should be available 24 hrs a day to evaluate patients with symptoms suggestive of an acute stroke. The institution should have a Stroke notification system in place with documented expected response times.

Written Care Protocols

One of the major key elements in developing good outcomes is through the implementation of protocols. As such, protocols should be based on well established Standards of Care and national guidelines. This should include emergency care of ischemic/hemorrhagic stroke patients. Studies have shown that by adhering to protocol guidelines, more patients were properly screened for tPA. Not only was there a substantial increase in the proportion of patients receiving thrombolytic therapy, but rather, there was also a decrease in the rates of symptomatic ICH.[5] If the patient is deemed to be complex, a protocol should be in place for those requiring transfers to a comprehensive center—a "Treat and Transfer" pathway should be initiated.

Emergency Department

The ED serves as the gateway between the patient and the hospital. It is important that emergency personnel are trained in recognizing, diagnosing, and treating all types of acute stroke. There should be highly organized means communication amongst the EMS, ED personnel and the Acute Stroke Team. The ED should be able to provide proper documentation of patient's history, procedures, and qualifications for thrombolytic therapy.

Stroke Unit

This is a dedicated unit staffed by health care personnel (vascular neurologists, stroke-trained nurses, physical/occupational/speech therapists, case managers, social workers) who are trained to care for patients with cerebrovascular diseases. This unit should also be equipped with infrastructure such as continuous telemetry, and the capability for blood pressure monitoring. There has been a big difference noted in the care received by stroke patients in the stroke unit setting as compared to the general medical floors. Patients in stroke units had an 8% reduction in length of stay, and 7% increase in going back to living at home, and a 17% reduction in death rates. The cost of forming and operating stroke units will vary depending on size and location. Again, there is a need for written care protocols for standardization of care.

Neurosurgery

During the acute phase of a stroke, patients may need a neurosurgical procedure, thus, a primary stroke center must be able to provide neurosurgical care within 2 hours if clinically necessary. If this is not met, the patient should be transferred to a medical facility with an available neurosurgeon who is experienced in treating different types of acute strokes. A protocol is necessary to Treat and Transfer patients with the need for comprehensive stroke care.

Neuroimaging

In order to have a proper stroke diagnosis, it is vital for a primary stroke center to have imaging capability 24 hours a day. The ideal door to imaging time is within 25 minutes of order entry, and must be interpreted within 20 minutes.

Hospital and Administrative Support

There should be a designated Medical Director of Stroke Center with expertise and training in Cerebovascular Diseases. Commitment from administration of hospital/health system is important in providing support and funding for the needed resources to maintain the ability to deliver high quality and efficient care to acute stroke patients and the community.

Laboratory Services

This should be available 24 hrs. a day (CBC, Chemistry and Coagulation studies) The chest x-ray and EKG must be completed within 45 minutes of patient's arrival

Outcome/Performance Improvement:

Primary Stroke Centers must be able to develop specific stroke performance measures to be monitored by Performance Improvement Department, there should be ongoing efforts with Stroke Team and Performance Improvement to improve stroke care by utilizing benchmarking and performance measures data

Educational Programs

In order to keep up with new developments in stroke care, the stroke team professional staff is required to maintain 8 hours CME (or its equivalent) credits per year. In addition to this, the stroke center should be able to offer education to the community at least twice a year in relation to Stroke recognition, prevention, and treatment.

References

1. American Heart Association. Heart Disease and Stroke Statistics–2006 Update; American Heart Association; 2006.
2. Stroke Treatment and Ongoing Prevention Act of 2001. S. 1274/H.R. 3431 (2001)
3. Dion, J. Management of Ischemic Stroke in the Next Decade: Stroke Centers of Excellence. Journal of Vasc. Interventional Radiology 2004; 15:S133-S141
4. Alberts, M. et al. Recommendations for the Establishment of Primary Stroke Centers. JAMA, 2000; 283:3102-3109
5. Katzan et al. Quality Improvement and Tissue-Type Plasminogen Activator for Acute Ischemic Stroke: A Cleveland Update Stroke, 2003; 34:799-800
6. Adams, R, Acker, J et. Al. Recommendations for Improving the Quality of Care Through Stroke Centers and Systems: An Examination of Stroke Center Identification Options. Stroke, 2002; 33:e1-e7
7. Schwamm, L.H., Pancioli, A, et al. Recommendations for the Establishment of Stroke Systems of Care, Recommendations from the American Stroke Association's Task Force on the Development of Stroke Systems., Stroke 2005;36:690-703
8. The National Institute of Neurological Disorders and Stroke (NINDS) rt-PA Stroke Study Group; A Systems Approach to Immediate Evaluation and Management of Hyperacute Stroke : Experience at Eight Centers and Implications for Community Practice and Patient Care. Stroke 1997;28:1530-1540
9. Sacco, R. L., Adams, R., et al. Guidelines for Prevention of Stroke in Patients With Ischemic Stroke or Transient Ischemic Attack. Stroke 2006;37:577-617
10. Goldstein, L. B., Adams, R., et al. Primary Prevention of Ischemic Stroke. Stroke 2006;37:1583-1633

Endovascular Cure of a "Locked-In" Patient

Meryl A. Severson, III, MD, Aditya S. Pandey, MD, Erol Veznedaroglu, MD

Jefferson Hospital for Neuroscience, Philadelphia, PA

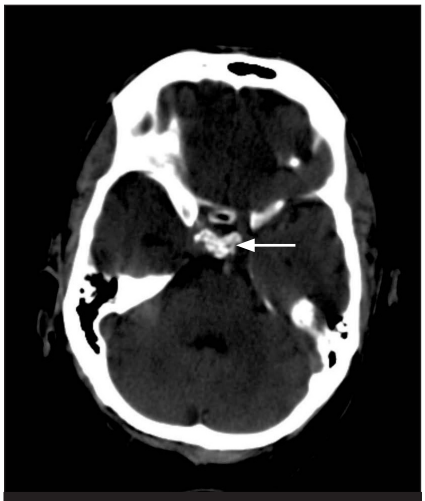


Figure 1. Non-contrast computed tomography scan of the head showing a hyperdense basilar artery (arrow).

We report the case of a 60 year-old right-handed gentleman who became acutely "Locked-In" several days after a lower extremity orthopedic procedure. He underwent emergent endovascular treatment and had complete resolution of his neurologic findings except for mild dysarthria and dysphagia. Endovascular intervention for posterior circulation thrombosis is highly effective when patients are treated within 24 hours.

Case

FC is a 60 year-old right-handed gentleman that underwent uncomplicated total knee arthroplasty and was started on low dose warfarin therapy for deep venous thrombosis prophylaxis. On postoperative day number two the patient had an acute change in his neurologic status. A computed-tomography scan of his head was obtained showing a hyperdense basilar artery. He was emergently transferred to our institution.

On arrival the patient was lethargic but able to follow commands with eye blinks only. His pupils were equal and reactive but he only had vertical gaze. The right extremity minimally withdrew to stimulation. Computed-tomography angiogram of the head showed a dominant right vertebral artery with occlusion just distal to the posterior inferior cerebellar artery origin extending to just below the superior cerebellar arteries.

The patient was taken emergently to the Endovascular Neurosurgery Suite and the right femoral artery accessed. The right vertebral artery was selectively catheterized and digital subtraction angiography performed revealing complete occlusion of the right vertebrobasilar junction without distal runoff or PCA filling. A Hyperglide balloon was positioned at the VB junction and seven inflations were performed, revealing a focal stenosis at the junction. A Gateway 4 balloon was positioned next and inflated to six atmospheres of pressure. Subsequent angiographic runs revealed complete resolution of the thrombus and filling of both PCAs and SCAs, as well as the basilar perforators. A Wingspan 4x5x20 stent was then deployed at the location of the stenosis resulting in complete resolution of the stenosis and thrombus. Additional angiographic runs showed distal occlusions bilaterally in the P4 segments; these were treated by placing an SL-10 microcatheter in the P4 segments and injecting 150,000 units of Urokinase on the right and 200,000 units on the left.

Post-procedurally the patient remained intubated for less than twenty-four hours. His neurologic exam improved to normal except for mild dysarthria and dysphagia and a right upper extremity drift without associated weakness. Magnetic resonance imaging of his brain revealed acute infarcts in the left pons, right occipital lobe, right dentate nucleus, both mesial temporal lobes, and the right posterolateral thalamus. He was gradually mobilized with the assistance of physical therapy/occupational therapy and eventually discharged in good condition.

Figure 2. Digital subtraction angiography AP projection of a selective right vertebral injection showing complete obstruction of flow at the right vertebrobasilar junction.

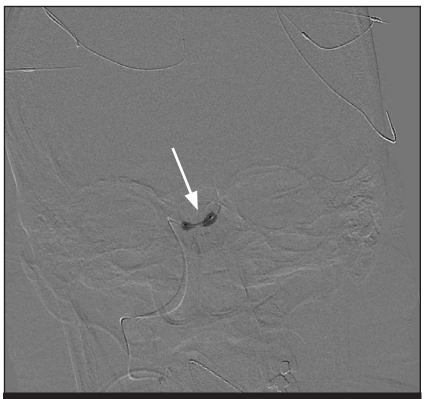


Figure 3. Digital subtraction angiography AP projection demonstrating inflation of the Hyperglide balloon at the right vertebrobasilar junction. Note the microcatheter extending into the proximal basilar artery and the "waist" (arrow) on the balloon reflecting the location of the obstruction.

Discussion

Unlike anterior circulation thromboses, the time window for posterior circulation thrombosis treatment extends up to 24 hours after the ictus. Cases of complete improvement in an adolescent treated 20 hours[1] and a child almost 50 hours after symptom onset[2] are

Figure 4. Digital subtraction angiography LAT projection of a right vertebral injection after Hyperglide balloon inflation. Note the improved filling of the basilar artery. A Gateway balloon has been advanced to the vertebrobasilar junction.

reported. However, we recommend treating up to 24 hours post-ictus only. An additional study reviewed outcome predictors for basilar artery thrombosis and found that recanalization after thrombolysis and low NIH stroke scale at admission were associated with a favorable outcome; initiating thrombolysis early after ictus as well CT evidence of a hyperdense basilar artery were positively associated with recanalization rates.[3] "Locked-In" Syndrome resulting from acute basilar artery thrombosis is curable but requires the patient be quickly diagnosed and transferred expeditiously to a specialized center with a highly experienced endovascular neurosurgical team.

References

1. Kirton A, Wong JH, Mah J, Ross BC, Kennedy J, Bell K, Hill MD. Successful endovascular therapy for acute basilar thrombosis in an adolescent. Ped. 2003 Sep: 112(3 Pt 1): e248-51.
2. Grigoriadis S, Gomori JM, Grigoriadis N, Cohen JE. Clinically successful late recanalization of basilar artery occlusion in childhood: what are the odds? Case report and review of the literature. J Neurol Sci. 2007 Sep 15;260(1-2):256-60.
3. Arnold M, Nedeltchev K, Schroth G, Baumgartner RW, Remonda L, Loher TJ, Stepper F, Sturzenegger M, Schuknecht B, Mattle HP. Clinical and radiological predictors of recanalisation and outcome of 40 patients with acute basilar artery occlusion treated with intra-arterial thrombolysis. J Neurol Neurosurg Psychiatry. 2004 Jun; 75(6):857-62.

Case Report: Hemorrhage into an Occult Spinal Ependymoma after Epidural Anesthesia

Peter G. Campbell, MD[1], Ashwini D. Sharan, MD[1], James S. Harrop, MD[1, 2], Alexander R. Vaccaro, MD[1, 2], John K. Ratliff, MD[1, 2]

[1]Department of Neurological Surgery, Thomas Jefferson University Hospital, Philadelphia, PA
[2]Department of Orthopedic Surgery, Thomas Jefferson University Hospital, Philadelphia, PA

Originally published in The Open Surgery Journal (vol. 2, pp. 18–20, 2008), an Open Access Journal (Bentham Science Publishers Ltd.). Republished with permission.

Abstract

Summary of Background Data: Five cases of hemorrhage into a spinal neoplasm after spinal or epidural anesthesia are reported in the literature. Presentation ranges from severe low back pain to acute cauda equina syndrome.

Methods: A case study of a patient who hemorrhaged into an intradural, extramedullary spinal cord mass was performed. A detailed literature review is also provided.

Results: A 27 year old female underwent epidural anesthesia for Cesarean section delivery. She presented with a 3 week history of increasing low back pain with bilateral radiculopathy. Imaging studies revealed a large hemorrhagic intradural mass compressing the lower conus medullaris and cauda equina, which operatively was confirmed to be a myxopapillary ependymoma.

Conclusions: We report a case of hemorrhage into a previously unrecognized ependymoma after epidural anesthesia. Underlying tumors may rarely complicate regional anesthesia in the lumbar spine.

Keywords
Ependymoma, epidural anesthesia, spinal anesthesia, hemorrhage, myxopapillary ependymoma.

Abbreviation list
MRI: Magnetic resonance imaging

Introduction

Epidural anesthesia is a procedure which is well tolerated and has a low incidence of adverse events. In performing caesarean sections, regional anesthesia (spinal or epidural) is the preferred modality for anesthetic delivery[7]. Although rare with continuous epidural anesthesia, epidural hematomas have been reported to occur with an incidence between 1:150,000 and 1:190,000[10]. An underlying bleeding diathesis has been implicated as a causative factor. We present the sixth reported case of hemorrhage into an occult intradural neoplasm after spinal or epidural anesthesia. Similar lesions have not been reported in the recent spine literature.[1, 2, 3, 5, 8]

Case Report

A 27-year-old gravida 1, para 0 patient underwent a Caesarean section with epidural anesthesia without difficulty. She was discharged home with a healthy infant on post operative day two. Three days after the procedure the patient noted a moderate degree of low back pain, which was initially attributed to ligamentous strain. Conservatively therapy, consisting of bed rest and analgesics, failed to provide symptomatic relief. The symptoms persisted and the patient progressed over the following three weeks, noting worsening low back pain radiating to the legs. The patient then presented to the emergency department, where she described severe low back pain radiating into both lower extremities. Her past medical history was insignificant. She denied any history of coagulopathy, heavy menses or any contributory family history. A thorough neurological exam revealed intact motor strength sensation, with normal deep tendon reflexes and an absent Babinski's sign. A rectal exam revealed normal anal sphincter tone and volition.

Figure 1. Sagittal T1 Lumbar Spine MR Imaging post gadolinium reveals an enhancing mass at the T12 - L1 spinal levels with heterogeneous but intense enhancement of the lesion and an irregular central foci of nonenhancement. The lesion fills the spinal canal, and appears to arise from the terminal portion of the spinal cord.

Investigations

No hematological abnormalities were noted on laboratory examination. MR Imaging of the lumbar spine with and without gadolinium revealed an oblong intradural mass at T12-L1 with intense peripheral enhancement and irregular central nonenhancement was noted (Figures 1-3). Complete MR imaging of the neuroaxis showed no additional lesions.

Figure 2. Sagittal T2 Lumbar Spine MR Imaging reveals an oblong intradural mass 6.1 x 1.9 x 1.7 cm in the cranio-caudal, transverse, and anterioposterior dimensions. Bright imaging CSF appears above and below the lesion. Again, the lesion appears to begin at the terminal portion of the spinal cord.

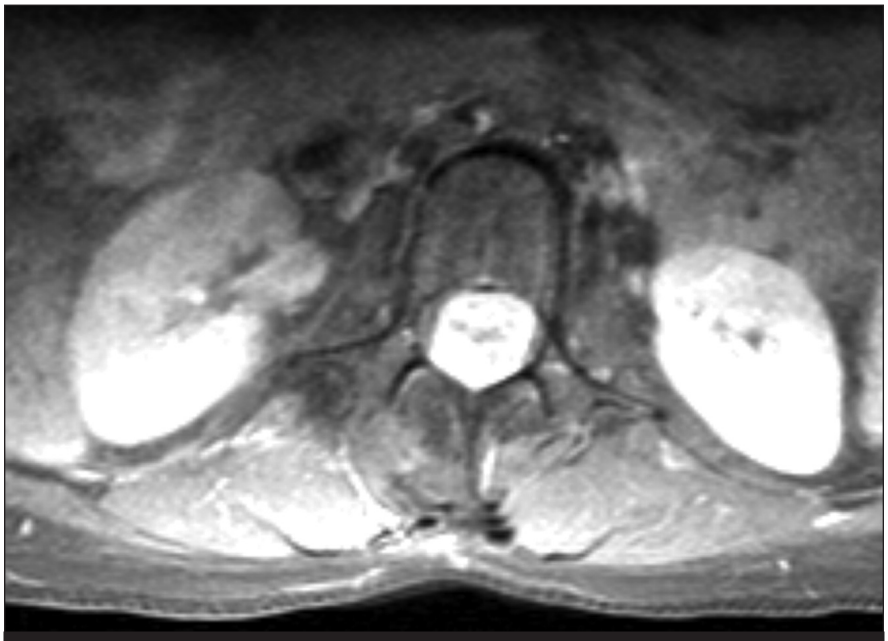


Figure 3. Axial T1 MRI lumbar spine MR Imaging post gadolinium at the level of L1 showing the intradural mass. The entire spinal canal is occupied by the heterogeneously contrast enhancing mass.

Treatment

The patient underwent a T12 to L2 laminectomy. Intraoperatively, an intradural mass adherent to the cauda equina and filum terminale with some intratumoral hemorrhage was removed. A gross total resection was accomplished.

Pathology

Final pathologic analysis of the surgical specimen revealed a myxopapillary ependymoma.

Postoperative Course

The patient made a full recovery. She manifested initial urinary retention, which resolved with the discontinuation of narcotics. Postoperative MR Imaging revealed no evidence of residual tumor.

Discussion

Extramedullary spinal cord tumors may be intradural, extradural, or a combination of the two. Extradural tumors account for roughly 30% of spinal cord neoplasms while intradural extramedullary tumors are responsible for approximately 40 to 50%.[4] Intradural spinal cord tumors are uncommon with an incidence of about 3–10 per 100,000 individuals. These tumors occur predominately in the third and fourth decades of life.[9] The most common extramedullary spinal cord tumors are nerve sheath tumors, meningiomas and filum terminale ependymomas. Ependymomas are the most common gliomas of the lower cord, conus and filum terminale; the myxopapillary histological variant is most common. Approximately 40% of all spinal ependymomas arise within the proximal intradural filum terminale.[4] Back pain is the most common presenting symptom, with neurological compromise rare secondary to the adaptive compressibility of the surrounding structures. Mork and Loken report that in 82% of patients with spinal ependymoma, symptoms were present for more than 1 year before the diagnosis was achieved.[6] Treatment for these lesions is typically en bloc surgical resection.[9] Radiation therapy is reserved for recurrent disease or aggressive histopathology, with regular radiographic follow-up recommended. In cases of subtotal resection, radiotherapy may be required.

Epidural anesthesia is an effective procedure with a low incidence of complications. Neurological compromise following epidural anesthesia, while highly uncommon, is the most concerning potential sequela of the procedure. Epidural hematomas may rarely occur during continuous epidural anesthesia, with a reported incidence between 1:150,000 and 1:190,000.[10] The majority of these cases are believed due to a primary coagulopathy.

Rarely, hemorrhage from an occult spinal mass occurs in an asymptomatic patient receiving epidural anesthesia. The first case of intratumoral bleeding in an asymptomatic patient after attempted epidural anesthesia was reported in 1984. A patient developed low back pain, bladder dysfunction, worsening motor and sensory function in both legs three days after epidural analgesia.[8] Since then, four additional cases involving hemorrhage into an occult neoplasm by regional lumbar spine anesthesia have been reported. Three cases involve epidural anesthesia [1,3,5] and one was caused by a subarachnoid injection of anesthetic.[2] In each of these cases, the underlying lesion was an ependymoma. In our case and one other the only complaint prior to neurosurgical intervention was severe low back pain with increasing bilateral radicular

Table 1. Reports of Hemorrhage into a Neoplasm After Regional Spinal Anesthesia

Author	Year	Patient	Surgery	Anesthesia	Tumor
Antoniadis *et al.*[1]	1985	79, female	Removal of femoral osteosynthesis	Epidural	Ependymoma
Bredtmann *et al.*[2]	1989	58, male		Subarachnoid	Ependymoma
Jaeger *et al.*[3]	2002	28, female	Cesarian section	Subarachnoid	Ependymoma
Martin *et al.*[5]	1992	31, female	Cesarian section	Epidural	Ependymoma
Roscoe et al.[8]	1984	24, female	Cesarian section	Epidural	Ependymoma

pain.[5] In the remaining reports, a component of lower extremity weakness and bowel or bladder dysfunction was noted. More severe presentations are reported: Jaeger et al. detailed a similar case with a lesion in a comparable area and described neurological deficits progressing to paraplegia within 24 hours.[3] Previously reported cases are reviewed in Table 1.

Conclusion

Occult spinal masses should be considered in the differential diagnosis in a patient with new onset neurological deficits or unusual pain complaints after spinal or epidural anesthesia. MR imaging of the lower thoracic and lumbar spine may be useful in evaluation of these symptoms.

References

1. Antoniadis G. Richter HP. Schachenmayr W. Hemorrhage into a spinal ependymoma after peridural anesthesia. Neurosurgery. 1985; 16(5): 669-71.
2. Bredtmann RD, Wright J, Weissflog M. A space-occupying hemorrhage following spinal anesthesia in the presence of an undiagnosed small spinal tumor (ependymoma). Reg Anaesth. 1989; 12(2): 38-40.
3. Jaeger M, Rickels E, Schmidt A, et al. Lumbar ependymoma presenting with paraplegia following attempted spinal anaesthesia. Br J Anaesth. 2002; 88(3): 438-40.
4. Kuntz C, Shaffrey CI, Wolcott WP. Approach to the Patient and Medical Management of Spinal Disorders. In: Winn HR, editor. Youmans Neurological Surgery, Vol 4. Philadelphia, Saunders. 2004: 4289-4326.
5. Martin HB, Gibbons JJ, Bucholz RD. An unusual presentation of spinal cord tumor after epidural anesthesia. Anesthesia & Analgesia 1992; 75(5): 844-6.
6. Mork SJ, Loken AC. Ependymoma: a follow-up study of 101 cases. Cancer. 1977; 40(2): 907-15.
7. Ng K. Parsons J. Cyna AM. Middleton P. Spinal versus epidural anaesthesia for caesarean section. Cochrane Database of Systematic Reviews, 2, CD003765, 2004.
8. Roscoe MW, Barrington TW. Acute spinal subdural hematoma. A case report and review of literature. Spine. 1984;9(7):672-5.
9. Stein BM, McCormick PC. Spinal intradural tumors. In Wilkins RH, Rengachary SS, eds. Neurosurgery, Vol 2. New York, McGraw-Hill. 1996: 1769-1781.
10. Wulf H. Epidural anesthesia and spinal haematoma. Can J Anaesth. 1996;43:1260-1272.

Case Report: Signal Drop on MRA Imaging of the Internal Carotid Artery after Neuroform Stent Placement

Aditya S. Pandey, MD, Meryl Severson, MD, Robert Rosenwasser, MD

Department of Neurological Surgery, Thomas Jefferson University Hospital, Philadelphia, PA

Introduction

Magnetic resonance angiography (MRA) is an important tool in evaluating the patency of vessels which have previously been stented. Neuroform stents (Boston Scientific, Natick, MA, U.S.A.) are utilized to provide a scaffold across the neck of an aneurysm. These stents are synthesized from Nitinol (nickel and titanium) and thus cause minimal distortion upon imaging with MRA. Patients who have undergone Neuroform stent assisted coiling of aneurysms are routinely followed with MRA to delineate stenosis of the stented segment of vessel as well as recurrence of the aneurysms. While numerous reports show that Neuroform stents do not lead to MRA imaging artifact, we report of a case where the utilization of the Neuroform stent led to a signal drop out at the site of the stent upon evaluation with MRA and thus led to further invasive radiological procedures.

Materials and Methods

This study is a case report identifying Magnetic resonance imaging artifact associated with the use of Neuroform stents. Data was collected from the patient's chart, operative report, and imaging studies.

Case Report

The patient presented in this report is a 55 year old right handed female who underwent screening MRA of the head due to a family history of subarachnoid hemorrhage. This revealed a 5 mm left carotid wall aneurysm. Subsequently, cerebral angiography revealed a broad based 6 mm by 4 mm left posterior carotid wall aneurysm (Figure 1). The angioarchitecture of the aneurysm made it necessary to place a stent in order to prevent coil prolapse into the parent vessel.

Due to technical difficulty associated with the tortuousity of the vessel, a Neuroform-3 stent was prematurely deployed into the lacerum portion of the left internal carotid artery (ICA). However, another neuroform-3 stent was able to be placed across the neck of the posterior carotid wall aneurysm. Postoperatively, the patient remained neurologically intact and was maintained on plavix therapy to prevent thromboembolic events. We allowed the stent to adhere to the vessel over a period of eight weeks at which time the patient underwent coil embolization of the aneurysm without any complications (Figure 2).

Four weeks post coiling, the patient presented to the emergency room complaining of right sided numbness and tingling. A computed tomographic (CT) scan of the head was unrevealing of any pathology. An MRI/A of the neck and head was performed to delineate whether a carotid dissection with associated thromboembolic event was the causation for the patient's symptoms (Figure 3). This revealed a change in the caliber of the left ICA at the level of the petrous carotid. While certainly this could represent susceptibility artifact from the deployed stent, no such cases have been reported in the English literature. Thus the above mentioned imaging results led to the patient being admitted to the hospital and subsequent prophylactic anticoagulation. A CTA of the head was performed to better understand the exact location and length of the dissected segment of vessel (Figure 4). Due to the surrounding bone associated with the petrous carotid artery, the CTA was not revealing in delineating whether the MRA findings represented artifact or actual vascular dissection. Thus it was essential to perform cerebral angiography which revealed no evidence of dissection or recurrence of the previously coiled aneurysm (Figure 5).

Discussion

Use of self expanding Nitinol stents has revolutionized the treatment of intracranial aneurysms as well as intracranial atherosclerotic disease. MRI/A have been shown to be of great value in evaluating restenosis of a treated vessel or recanalization of treated aneurysms where these stents have been utilized. A multitude of reports in the English literature have revealed that MRI/A creates minimal artifact when imaging vessels with these stents. In fact, there are no reports of MRA associated signal drop out from imaging of vessels which have been stented with Neuroform stents.

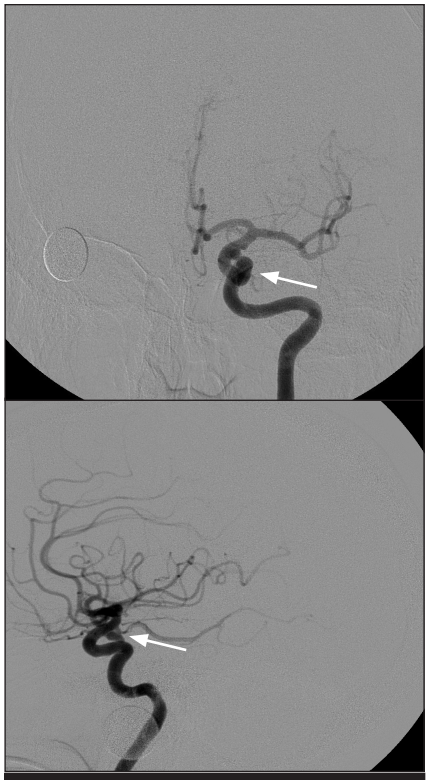


Figure 1. Cerebral angiography: early arterial phase. Anterior-Posterior (A) and Lateral (B) projections revealing a left posterior carotid wall aneurysm.

Our case report exemplifies that MRA imaging can indeed create a signal drop out at the site of Neuroform stent placement thus creating a false image of possible dissection of the vessel. Such an artifact can lead to further treatments and imaging which not only increase the cost burden to society but also can increase the morbidity to the patient. While we did perform a computed tomographic angiography (CTA) of the head, it was of no value as the artifact from the petrous bone prevented a clear delineation of whether a dissection existed or not.

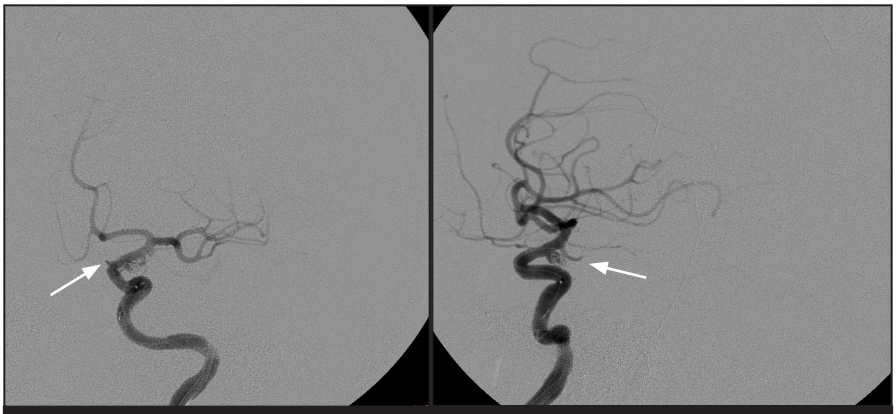


Figure 2. Left ICA injection: early arterial phase. AP (A) and Lateral (B) projections post Neuroform stent placement and coil embolization of the posterior carotid wall aneurysm.

Figure 4. Computed tomographic angiography (CTA) of the head revealing the left petrous ICA not well visualized due to artifact created from the petrous bone.

Figure 3. Magnetic Resonance Angiography (MRA) of the head revealing a left petro us carotid artery dissection which was later proved to be signal drop out from the presence of a Neuroform stent.

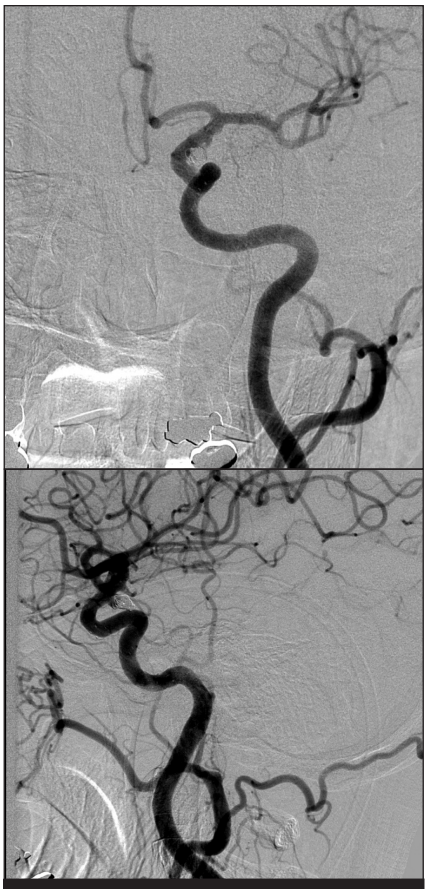


Figure 5. Cerebral angiography: left common carotid artery injection early arterial phase. AP (A) and Lateral (B) projections revealing no evidence of carotid dissection at the site of Neuroform stent placement.

Neuroform-3 stents can create MRI associated artifact/signal drop out and such this should be taken into account when imaging vessels which house such stents. While MRA with gadolinium or CTA head can better visualize the pathology, cerebral angiography remains the gold standard in evaluating vessels which have undergone stenting.

Departmental Information

Thomas Jefferson University Department of Neurosurgery

909 Walnut Street 2nd and 3rd Floors
Philadelphia, PA 19107
Telephone: 215-955-7000
www.Jefferson.edu/Neurosurgery

Faculty

Robert H. Rosenwasser, MD, FACS, FAHA
Professor and Chair

David W. Andrews, MD, FACS
Professor and Vice Chair

Deborah L. August, MD, MPH

William A. Buchheit, MD, FACS
Professor Emeritus & Former Chair

Stephen J. Dante, MD, FACS
Assistant Professor

James J. Evans, MD
Assistant Professor

Jon Glass, MD
Associate *Professor of Neurology and Neurological Surgery*

James S Harrop, MD, FACS
Associate Professor

Larry A. Harshyne, Jr, PhD
Instructor

Kenneth M Liebman, MD, FACS
Assistant Professor

Jewell L. Osterholm, MD, FACS
Professor Emeritus and Former Chair

John K. Ratliff, MD, FACS
Associate Professor

Ashwini D. Sharan, MD
Assistant Professor

Elisabeth J. Van Bockstaele, PhD
Professor and Vice Chair for Research

Erol Veznedaroglu, MD, FACS
Associate Professor

Research
Department of Neurosurgery

Clinical Research Publications In Peer-reviewed Journals: January 2007 through June, 2008

- Asadi-Pooya, A.A., Sharan, A., Nei, M., Sperling, M.R. Corpus callosotomy. Epilepsy and Behavior. In press, 2008
- Harrop, J.S., Jeyamohan, S., Sharan, A., Ratliff, J., Flanders, A., Maltenfort, M., Falowski, S., Vaccaro, A. Acute cervical facture or congenital spinal deformity? Journal of Spinal Cord Medicine. 31(1): 83-87, 2008
- Veznedaroglu, E., Koebbe, C.J., Siddiqui, A., Rosenwasser, R.H. Initial experience with bioactive cerecyte detachable coils: impact on reducing recurrence rates. Neurosurgery. 64(4): 799-805, 2008
- Fayssoux, R.S., Tally, W., Sanfilippo, J.A., Stock, G., Ratliff, J.K., Anderson, G., Hilibrand, A.S., Albert, T.J., Vaccaro, A.R. Spinal injuries after falls from hunting tree stands. Spine Journal. 8(3): 522-528, 2008
- Patel, A.A., Brodke, D.S., Pimenta, L., Bono, C.M., Hilibrand, A.S., Harrop, J.S., Riew, K.D., Youssef, J.A., Vaccarro, A.R. Revision strategies in lumbar total disc arthoplasty. Spine. 33(11): 1276-1283, 2008
- Ortiz, R., Stefanski, M. Rosenwasser, R., Veznedaroglu, E. Cigarette smoking as a risk factor for recurrence of aneurysms treated by endosaccular occlusion. Journal of Neurosurgery: Spine. 108(4):649-654, 2008
- Andrews, D.W. Currrent neurosurgical management of brain metastases. Seminars in Oncology. 35(2):100-107, 2008
- Fassett, D.R., Harrop, J.S., Vaccaro, A.R. Evidence on magnetic resonance imaging of Brown-Séquard spinal cord injury suffered indirectly from a gunshot wound: Case report. Journal of Neurosurgery: Spine. 8(3):286-287, 2008
- Chandrasekar, T., Sharan, A.D., Sperling, M.R. Postoperative auras and the risk of recurrent seizures. Epilepsy Research. 78(3-Feb):195-200, 2008
- Sperling, M.R., Nei, M., Zangaladze, A., Sharan, A.D., Mintzer, S.E., Skidmore, C., Evans, J.G., Schilling, C.A., Asadi-Pooya, A.A. Prognosis after late relapse following epilepsy surgery. Epilepsy Research. 78(1):77-81, 2008
- Falowski, S., Celii, A., Sharan, A. Spinal Cord Stimulation: An Update. Neurotherapeutics. 5(1):86-99, 2008
- Zangaladze, A., Sharan, A., Evans, J., Wyeth, D.H., Wyeth, E.G., Tracy, J.I., Chervoneva, I., Sperling, M.R. The effectiveness of low-frequency stimulation for mapping cortical function. Epilepsia. 49(3):481-487, 2008
- Lee, J.Y., Vaccaro, A.R., Schweitzer Jr., K.M., Lim, M.R., Baron, E.M., Rampersaud, R., OÌˆner, F.C., Hulbert, R.J., Hedlund, R., Fehlings, M.G., Arnold, P., Harrop, J., Bono, C.M., Anderson, P.A., Patel, A., Anderson, D.G., Harris, M.B. Assessment of injury to the thoracolumbar posterior ligamentous complex in the setting of normal-appearing plain radiography. Spine Journal. 7(4):422-427, 2007
- Fassett, D.R., Jeyamohan, S., Harrop, J. Asymptomatic Cervical Stenosis: To Operate or Not?. Seminars in Spine Surgery. 19(1):47-50, 2007
- Curry, J.M., Cognetti, D.M., Harrop, J., Boon, M.S., Spiegel, J.R. Cervical discitis and epidural abscess after tonsillectomy. Laryngoscope. 117(12):2093-2096, 2007
- Dvorak, M.F., Fisher, C.G., Aarabi, B., Harris, M.B., Hurbert, R.J., Rampersaud, Y.R., Vaccaro, A., Harrop, J.S., Nockels, R.P., Madrazo, I.N., Schwartz, D., Kwon, B.K., Zhao, Y., Fehlings, M.G. Clinical outcomes of 90 isolated unilateral facet fractures, subluxations, and dislocations treated surgically and nonoperatively. Spine. 32(26):3007-3013, 2007
- O'Leary, O.F., Bechtholt, A.J., Crowley, J.J., Hill, T.E., Page, M.E., Lucki, I. Depletion of serotonin and catecholamines block the acute behavioral response to different classes of antidepressant drugs in the mouse tail suspension test. Psychopharmacology. 192(3):357-371, 2007

- Pandey, A.S., Koebbe, C., Rosenwasser, R.H., Veznedaroglu, E. Endovascular coil embolization of ruptured and unruptured posterior circulation aneurysms: Review of a 10-year experience. Neurosurgery. 60(4):626-636, 2007
- Smith, H.E., Rynning, R.E., Okafor, C., Zaslavsky, J., Tracy, J.I., Ratliff, J., Harrop, J., Albert, T., Hilibrand, A., Anderson, G., Sharan, A., Brown, Z., Vaccaro, A.R. Evaluation of neurologic deficit without apparent cause: The importance of a multidisciplinary approach. Journal of Spinal Cord Medicine. 30(5):507-517, 2007
- Chaudhry, S., Sharan, A., Ratliff, J., Harrop, J.S. Geriatric Spinal Injury. Seminars in Spine Surgery. 19(4):229-234, 2007
- Campbell, P.G., Harrop, J.S. Incidence of fracture in adjacent levels in patients treated with balloon kyphoplasty: a review of the literature. Current Reviews in Musculoskeletal Medicine. 1:61-64, 2007
- Schweitzer Jr., K.M., Vaccaro, A.R., Harrop, J.S., Hurlbert, J., Carrino, J.A., Rechtine, G.R., Schwartz, D.G., Alanay, A., Sharma, D.K., Anderson, D.G., Lee, J.Y., Arnold, P.M. Interrater reliability of identifying indicators of posterior ligamentous complex disruption when plain films are indeterminate in thoracolumbar injuries. Journal of Orthopaedic Science. 12(5):437-442, 2007
- Vadera, S., Harrop, J.S., Sharan, A.D. Intrathecal granuloma and intramedullary abscess associated with an intrathecal morphine pump. Neuromodulation. 10(1):6-11, 2007
- Vadera, S., Ratliff, J., Brown, Z., Patel, A., Harrop, J.S., Sharan, A., Vaccaro, A.R. Management of Cervical Facet Dislocations. Seminars in Spine Surgery. 19(4):250-255, 2007
- Pandey, A., Rosenwasser, R.H., Veznedaroglu, E. Management of distal anterior cerebral artery aneurysms: A single institution retrospective analysis (1997-2005). Neurosurgery. 61(5):909-916, 2007
- Musacchio, M., Patel, N., Bagan, B., Deutsch, H., Vaccaro, A.R., Ratliff, J. Minimally invasive lumbar laminectomy via a dual-tube technique: evaluation in a cadaver model. Surgical Neurology. 67(4):348-352, 2007
- Fassett, D.R., Harrop, J.S., Maltenfort, M., Jeyamohan, S.B., Ratliff, J.D., Anderson, D.G., Hilibrand, A.S., Albert, T.J., Vaccaro, A.R., Sharan, A.D. Mortality rates in geriatric patients with spinal cord injuries. Journal of Neurosurgery: Spine. 7(3):277-281, 2007
- Showalter, T.N., Andrel, J., Andrews, D.W., Curran Jr., W.J., Daskalakis, C., Werner-Wasik, M. Multifocal Glioblastoma Multiforme: Prognostic Factors and Patterns of Progression. International Journal of Radiation Oncology Biology Physics. 69(3):820-824, 2007
- Patel, A.A., Vaccaro, A.R., Martyak, G.G., Harrop, J.S., Albert, T.J., Ludwig, S.C., Youssef, J.A., Gelb, D.E., Mathews, H.H., Chapman, J.R., Chung, E.H., Grabowski, G., Kuklo, T.R., Hilibrand, A.S., Anderson, D.G. Neurologic deficit following percutaneous vertebral stabilization. Spine. 32(16):1728-1734, 2007
- Harrop, J.S., Hanna, A., Silva, M.T., Sharan, A. Neurological manifestations of cervical spondylosis: An overview of signs, symptoms, and pathophysiology. Neurosurgery. 60(1 SUPPL.):0-0, 2007
- Patel, N., Bagan, B., Vadera, S., Maltenfort, M.G., Deutsch, H., Vaccaro, A.R., Harrop, J., Sharan, A., Ratliff, J.K. Obesity and spine surgery: Relation to perioperative complications. Journal of Neurosurgery: Spine. 6(4):291-297, 2007
- Ratliff, J., Anand, N., Vaccaro, A.R., Lim, M.R., Lee, J.Y., Arnold, P., Harrop, J.S., Rampersaud, R., Bono, C.M., Gahr, R.H. Regional variability in use of a novel assessment of thoracolumbar spine fractures: United States versus international surgeons. World Journal of Emergency Surgery. 2(1), 2007
- Patel, A.A., Vaccaro, A.R., Albert, T.J., Hilibrand, A.S., Harrop, J.S., Anderson, D.G., Sharan, A., Whang, P.G., Poelstra, K.A., Arnold, P., Dimar, J., Madrazo, I., Hegde, S. The adoption of a new classification system: Time-dependent variation in interobserver reliability of the thoracolumbar injury severity score classification system. Spine. 32(3):S14-S20, 2007
- Whang, P.G., Vaccaro, A.R., Poelstra, K.A., Patel, A.A., Anderson, D.G., Albert, T.J., Hilibrand, A.S., Harrop, J.S., Sharan, A.D., Ratliff, J.K., Hurlbert, R.J., Anderson, P., Aarabi, B., Sekhon, L.H.S., Gahr, R., Carrino, J.A. The influence of fracture mechanism and morphology on the reliability and validity of two novel thoracolumbar injury classification systems. Spine. 32(7):791-795, 2007
- Vaccaro, A.R., Hulbert, R.J., Patel, A.A., Fisher, C., Dvorak, M., Lehman Jr., R.A., Anderson, P., Harrop, J., Oner, F.C., Arnold, P., Fehlings, M., Hedlund, R., Madrazo, I., Rechtine, G., Aarabi, B., Shainline, M. The subaxial cervical spine injury classification system: A novel approach to recognize the importance of morphology, neurology, and integrity of the disco-ligamentous complex. Spine. 32(21):2365-2374, 2007
- Yadla, S., Ratliff, J.K., Harrop, J.S. Whiplash: diagnosis, treatment, and associated injuries. Current Reviews in Musculoskeletal Medicine. 1:65-68, 2007

Research
Department of Neurosurgery

Clinical Research Ongoing Studies

- The Wingspan™ Stent System and Gateway™ PTA Balloon Catheter, a humanitarian use device
- A single blind, randomized evaluation of dilute Betadine solution irrigation versus normal saline irrigation in the prevention of postoperative posterior instrumented spinal surgeries
- A multicenter retrospective study of the effectiveness, safety and treatment characteristics of subcutaneous neurostimulation (SQS) in patients with chronic pain
- Radiographic evaluation of intersegmental correction of cervical kyphosis with combined anterior interbody grafting and posterior instrumented arthrodesis
- Factors predicting the need for a ventricular peritoneal shunt (VPS) in patients with cerebellar hemorrhage
- Magnetic resonance analysis of postsurgical temporal lobectomy and correlation to seizure outcome
- Management of pericallosal artery aneurysms at Thomas Jefferson University
- Cordis ENTERPRISE™ Vascular Reconstruction Device and Delivery System: a humanitarian use device
- Changing impedances over time with spinal cord stimulation systems
- Identifying abnormal neurocognitive circuits in intractable epilepsy
- Neurological deterioration after spinal cord injury
- Magnetic resonance imaging of cervical spondolytic myelopathy: correlation between findings and outcome
- Incidence and outcome of patients with spinal fracture and ankylosing spondylitis
- FlexiCore Intervertebral Disc versus circumferential lumbar spinal fusion for the treatment of discogenic pain unresponsive to conservative treatment associated with degenerative disc disease
- Magnetic resonance imaging of spinal cord injury: correlation with clinical findings and outcome
- A prospective, multi-center, randomized controlled study to compare the spinal sealant system as an adjunct to sutured dural repair with standard of care methods during spinal surgery
- Carotid revascularization endarterectomy versus stent trial (CREST)
- In vitro pilot study to determine the capacity of glioma cells and metastatic intracranial tumor cells to cross-prime dendritic cells and induce a measurable anti-tumor cytotoxic T cell response
- Retrospective chart review of fractionated stereotactic radiotherapy for the treatment of residual craniopharyngioma
- The use of expandable vs non-expandable cages in vertebral body replacement surgery: a retrospective review of outcomes at Jefferson
- Retrospective review of surgical complications of occipital nerve stimulator systems
- Surgical anastomosis of the superficial temporal artery to the middle cerebral artery (STA-MCA) when added to the best medical therapy for carotid occlusion
- Humanitarian use device: Neuroform Microdelivery Stent
- Role of brain tissue oxygen monitoring in detection of cerebral vasospasm
- Determinants of Hunt Hess grade: a retrospective analysis of the last 100 patients admitted with subarachnoid hemorrhage
- Perimesencephalic subarachnoid hemorrhage: the role of repeated cerebral angiography
- prospective glioma patients archival database including parallel clinical, radiographic, histopathological, and unsupervised microarray analyses

- A prospective national study to molecularly and genetically characterize human gliomas: the glioma molecular diagnostic initiative (GMDI) a retrospective review of the radiographic and prognostic qualities of anaplastic astrocytoma subtypes
- The role of increased CSF production in clinical and radiologic vasospasm
- The use of fractionated stereotactic radiotherapy for the treatment of optic nerve sheath meningiomas: a retrospective medical record review
- A randomized, clinical trial of unruptured brain arteriovenous malformations (ARUBA)
- Surgical treatment of pituitary tumor-transition from open to endoscopic approach
- Overall experience of pituitary tumor patients at Thomas Jefferson University
- Growth of the cranial base program-Otolaryngology/ Neurosurgery experience
- The relationship of pre-existing junctional c2-c3 fusion with odontoid fracture
- A multi-center retrospective cervical facet fracture/subluxation study
- Assessment of intracranial aneurysm shape as an indicator of rupture risk
- A pilot study comparing various electrode and program settings using the Eon IPG
- A retrospective review of the correlation between increased T2 weighted signal on cervical spine MRI to myelopathic clinical signs
- A retrospective chart review of cases done at Thomas Jefferson University: the failure rate in surgical treatment with fixation of c1-c2 fracture
- A retrospective chart review of TJUH patients who obtained epidural injections; looking at the post procedural complications
- A phase i/ii dose-ranging study to evaluate the safety, tolerability, and pharmacokinetics of BA-210 and the neurological status of patients after a single, extradural application of Cethrin (BA-210) during surgery for acute thoracic and cervical spinal cord injury
- Micrus® Cerecyte™ registry: a registry to evaluate outcomes at one year post-embolization with the Micrus® Cerecyte™
- A multicenter retrospective chart review, reviewing the safety and efficacy of the enterprise stent in patients with intracranial aneurysm
- Retrospective 5 year review of follow up angiogram on patients with cerebral aneurysms post clipping
- A prospective, multicenter, randomize, study comparing the use of Healos to autograft in a transforminal lumbar interbody fusion (TLIF) approach
- Use of discarded tissue from frontal and temporal lobectomy
- A multicenter retrospective chart review, reviewing the safety and efficacy of the enterprise stent in patients with intracranial aneurysm
- A retrospective review of post-treatment hemorrhage rates of patients with arterio-venous malformations
- A comprehensive retrospective review of anterior corpus callosotomy outcomes at TJUH, a substudy of previously reviewed cases
- Clinical outcome in closed head injury: a retrospective review of patients that received transfusion (FFP) versus patients who were not transfused
- Atlanto-occipital dislocation injuries (AOD): a retrospective review of management and outcome
- A retrospective and prospective review of peri-operative complications in patients undergoing spine surgery
- Outcome of patients with ruptured aneurysm who underwent endovascular coiling procedure and evacuation of intracerebral clot
- A pilot research study of Coumadin versus Plavix for the treatment of carotid and/or vertebral artery dissection

20th Annual

Pan Philadelphia Neurosurgery Conference

Friday, December 5, 2008
Save the Date

The Union League of Philadelphia
Philadelphia, PA

We welcome former faculty and alumni members as we celebrate the 20th Anniversary of the PAN Philadelphia Neurosurgery Conference in acknowledgement of the original Course Directors:

- William A. Buchheit, MD, FACS
- Eugene S. Flamm, MD, FACS
- Jewell L. Osterholm, MD

Featuring:

- Daniel F. Ryan III, Esquire

Course Directors

Robert H. Rosenwasser, MD, FACS, FAHA
Professor and Chair, Neurological Surgery
Division of Neurovascular Surgery and
Endovascular Neurosurgery
Jefferson Medical College,
Thomas Jefferson University

M. Sean Grady, MD, FACS
Professor and Chair, Neurosurgery
University of Pennsylvania
School of Medicine

Christopher Loftus, MD, DHC (Hon.), FACS
Professor and Chair, Neurosurgery
Assistant Dean for International Affiliations
Temple University
School of Medicine

Lectures and Case Studies in the following areas:

- Spinal Cord Injury
- Brain Tumors
- Neurovascular

Information will become available in the coming weeks online at
http://jeffline.jefferson.edu/jeffcm

Jefferson Medical College of Thomas Jefferson University is accredited by the ACCME to provide continuing medical education for physicians.

Jefferson™ Hospital for Neuroscience

School of Medicine TEMPLE UNIVERSITY

University of Pennsylvania Health System