Dear Colleague:

Welcome to the second edition of Abington Annals. This issue features seven articles on an array of neurovascular topics.

Our Neurosciences Institute this year marked several important developments:

- Our comprehensive stroke center offers full service care to stroke patients, from time-sensitive diagnosis in the emergency trauma center through advancements in neuro-interventional care and our dedicated neuro intensive care units.
- We entered our third year of 24/7 neuro-interventional expertise on staff.
- We are providing more intravenous rT-PA and intrarterial rT-PA as well as other advanced new interventional procedures for treating stroke.
- We offer the latest therapies for treating AVM's, vein of Galen malformations and cerebral and spinal arteriovenous fistulas (AVF's).
- Have added leading-edge MRI neuroimaging capabilities.
- We opened our Neuro Intensive Care Unit, staffed by a neuro-intensivist and nurses with specialty training and a neurointermediate care unit.
- We are utilizing new, sophisticated technology for monitoring brain tissue oxygen.
- Finally, our one-call transfer service (215-481-8181) offers immediate care for neuro patients who can benefit from being transferred to Abington Memorial Hospital for advanced, high level care.

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Abstract: Giant cell arteritis is the most common form of systemic vasculitis in adults. Patients usually present with headache, and visual symptoms and have elevated erythrocyte sedimentation rate. It has been reported that 3%-4% of patients with giant cell arteritis develop ischemic events secondary to vertebral artery stenosis or occlusion. The mainstay of therapy of giant cell arteritis is high dose steroid and/or methotrexate. We present a case of a 76-year-old female initially presented with intermittent double vision, mild headache, and unremarkable magnetic resonance imaging and magnetic resonance angiography of the head and neck. She was diagnosed and treated for ocular myasthenia. She was readmitted two months later with imbalance, worsening headache and workup suggested bilateral cerebellar infarction, complete occlusion of left vertebral artery and a high-grade stenosis of the right vertebral artery. Erythrocyte sedimentation rate and C-reactive protein were elevated. Temporal artery biopsy demonstrated changes consistent with giant cell arteritis. During the course of the treatment with corticosteroids and immunosuppressant patient developed dysarthria, left facial droop, and left hemiplegia and was found to have complete occlusion of both vertebral arteries. She was emergently taken for revascularization of occluded segment using angioplasty and stent.
placement. Patient had significant improvement of neurological symptoms within three days after procedure and continued to improve during hospitalization. Endovascular treatment of vasculitis affecting the intracranial vessels is not yet established. We report our experience with successful treatment of complete occlusion of vertebral artery secondary to Giant cell arteritis using endovascular intracranial angioplasty and stent placement.

**Key Words:** Giant cell arteritis, vertebral arteritis, intracranial angioplasty, intracranial stent placement, vertebral artery occlusion

**Introduction:** Giant cell arteritis (GCA) is a granulomatous vasculitis of large- and medium-sized arteries. Predominantly it affects the superficial temporal arteries, but other blood vessels such as ascending aorta, its branches and the vertebral arteries may be affected. Involvement of extradural internal carotid or vertebral arteries is usually responsible for acute ischemic strokes. Ischemic complications are a result of either thrombosis or severe occlusion with subsequent reduction of cerebral blood flow. The management of GCA mainly involves medical treatment such as high dose corticosteroids and immunosuppressant like methotrexate which can be used as steroid sparing agent. Endovascular angioplasty has been considered as an alternative or additional treatment in vasculitis such as Takayasu’s arteritis and Kawasaki’s disease since they involve large sized proximal blood vessels, especially brachiocephalic artery or subclavian artery. Favorable outcome after angioplasty and stenting for vasculitis involving peripheral arteries secondary to GCA has been reported in only two case reports. We present a case of a woman who was initially diagnosed with ocular myasthenia, developed progressive bilateral cerebellar infarction and bilateral vertebral artery occlusion despite being on high dose steroid and immunosuppressant. Due to acute onset of left hemiplegia, she was emergently treated with intracranial angioplasty and stent placement with complete recanalization of the vertebral artery and the reversal of the symptoms.

**Case Report:** A 76-year-old female with a past medical history of hypertension, diabetes mellitus type 2, hyperlipidemia and bilateral ptosis of unknown etiology (s/p surgical correction), presented with complaints of intermittent double vision, bilateral eye pain and new onset of mild frontal and temporal headache for two months. On admission, labs were unremarkable except for erythrocyte sedimentation rate (ESR) of 42 mm/hr and C-reactive protein (CRP) of 4mg/l. An extensive workup including magnetic resonance imaging and magnetic resonance angiography (MRI/MRA) of the head was unremarkable. She was diagnosed with ocular Myasthenia Gravis and was treated with oral Prednisone and Pyridostigmine. Two months later she was admitted for vertical diplopia, headache, lightheadedness and gait imbalance. She had tenderness in bilateral temporal regions. Labs were consistent with serum sodium of 125 meq/l, ESR of 51 mm/hr, CRP of 28 mg/l. MRI of the brain was consistent with bilateral acute cerebellar infarctions. MRA of the head and neck showed complete occlusion of the left vertebral artery at the V3-V4 junction. There was a high grade stenosis of the right vertebral artery at the V3-V4 junction. There was irregularity throughout the course of the bilateral vertebral arteries. She underwent bilateral temporal artery biopsy which showed inflammatory infiltrates, consisting of multinucleate giant cells and lymphocytes in the elastic lamina, consistent with GCA. Diagnostic cerebral angiography revealed high grade up to 80% stenosis of the right vertebral artery secondary to vasculitis/arteritis. Patient was treated with pulse dose steroid therapy (Solumedrol 1 gm intravenous for three days), followed by oral Prednisone (50 mg daily). She was discharged in a stable condition to a rehabilitation center. Three weeks later, she presented with worsening balance and a fall. MRI of the head showed progression of cerebellar infarctions. MRA showed complete occlusion of bilateral vertebral arteries. She was started on Azathioprine (50 mg daily) and Prednisone (50 mg daily) was continued. Patient underwent cerebral angiography which confirmed the findings of the MRA (Figures 1a, 1b). In addition, angiography also demonstrated retrograde filling of the basilar artery up to mid-segment from bilateral posterior communicating arteries (Figure 2). Within 24 hours patient developed an acute onset of dysarthria, left facial droop and left hemiplegia (NIHSS 13). Systolic blood pressure was 120 – 130 mmHg. Blood pressure was then induced...
up to 180 mmHg, which stabilized her symptoms. Plavix 300 mg and aspirin 325 mg were given.

Patient was emergently taken to the angiography suite to revascularize the occluded segment. A femoral approach was used and a 6 French sheath was placed. Through the sheath, a 6 French Envoy guide catheter (Cordis Endovascular, Miami Lakes, FL) was advanced and positioned at the origin of the right vertebral artery. Through the guide catheter an Excelsior SL-10 microcatheter (Boston Scientific, Natick, MA) was first advanced over a Transcend 0.014 inches microwire (Boston Scientific, Natick, MA) and then over a Synchro 2.0.014 inches microwire (Boston Scientific, Natick, MA). There was difficulty in crossing the occlusion through the microwire, the wire was removed and through the microcatheter Nicardipine 2mg (Cardene®, PDL pharma) was infused. The microwire was attempted to pass again through the occlusion and this time the wire passed successfully. The microcatheter was then advanced and positioned in the left posterior cerebral artery. A Synchro 2.0.014 inches exchange length microwire was then advanced and positioned in the posterior cerebral artery. The microcatheter was then exchanged with a Gateway 1.5 mm x 9 mm (Boston Scientific, Natick, MA) angioplasty balloon which was placed across the V3-V4 junction of the vertebral artery and inflated at two different points. Post-angioplasty angiographic images demonstrate recanalization of the vertebral artery. The balloon catheter was then removed and a Wingspan 3 mm x 20 mm stent (Boston Scientific, Natick, MA) was positioned and deployed. Because of the irregularity noted not only in the V3-V4 segment but there was progressive narrowing in the V2 and V1 segment thus a second Wingspan stent 3.5 mm x 20 mm stent was placed in the V2-V3 junction and deployed. A third Wingspan stent 4.0 mm x 20 mm was positioned in the V2 segment and deployed. A final Wingspan stent 4.5 mm x 15 mm was deployed at the origin of vertebral artery. Multiple injections after deployment of the stent showed the patency of the stent (Figures 3a, 3b). Patient had significant improvement of neurological symptoms within three days after procedure and continued to improve during hospitalization. She was started on Cyclophosphamide and Azathioprine was discontinued. Her neurological examination after one month showed antigravity strength in left upper and lower extremities normal sensory examination, mild dysarthria (NIHSS 3). CT scan of the head demonstrated small right pontine infarction and stable bilateral cerebellar infarctions.

**Discussion:** GCA is the most common form of systemic vasculitis in adults. The incidence of GCA varies widely in different populations, from less than 0.1 per 100,000 to 33 per 100,000 persons, aged 50 years and older. It is an inflammatory disease of large and medium sized arteries that affect internal elastic lamina through the pathway of activation of CD4+ Th cells to unknown antigen. It has tropism to arteries with well-developed elastic membrane. Most commonly it affects aorta and its main extracranial branches but in some cases it affects intracranial arteries, such as vertebral, basilar and anterior cerebral arteries. Intracranial vasculitis responsible for acute ischemic stroke in patients with GCA most commonly presents as vertebral artery narrowing and occasionally complete occlusion. Inflammatory process mostly involves extradural part of the vertebral
artery (V2 V 3 segment), but there have been few case reports of involvement of intradural portion (V3-V4 junction).\textsuperscript{4, 5}

Even though corticosteroid and immuno-suppressive therapy like Methotrexate are the mainstay of treatment of GCA, cerebral arteritis has been reported refractory to medical therapy in most cases. Out of eight cases found in the literature, only two had favorable outcome.\textsuperscript{2, 3} Endovascular treatment is not widely used to relieve the symptoms associated with vasculitis in GCA. Angioplasty has been successfully used only as alternative treatment in management of ischemic complications secondary to axillary arteritis in two patients with GCA.\textsuperscript{8, 9} both had a favorable outcome.

We report the first known case of angioplasty and stent placement for the treatment of vertebral artery occlusion in a patient with GCA. Our patient developed complete occlusion of the bilateral vertebral arteries with cerebellar infarction despite being on aggressive medical treatment which improved after balloon angioplasty and stent placement. There are two cases reported of vasculitis secondary to GCA in which angioplasty and stenting were utilized to treat refractory limb ischemic symptoms. In the first case balloon angioplasty of the axillary artery was performed to relieve ischemic symptoms in upper extremities.\textsuperscript{8} In the second case balloon angioplasty of superior mesenteric artery relieved the symptoms of abdominal angina that were persistent after prednisone and cyclophosphamide treatment.\textsuperscript{9} Acute ischemic events secondary to vertebral artery stenosis/occlusion have been reported in 3-4 % of patients with GCA.\textsuperscript{3, 5} In most cases GCA involving vertebral arteries has been refractory to medical treatment and has a fatal outcome. Endovascular intracranial angioplasty and stent placement is a potential alternative treatment option in such cases.

**Conclusion:** Endovascular intracranial angioplasty and stent placement presents an alternative method of treatment for ischemic cerebral complications secondary to giant cell arteritis involving vertebral arteries refractory to medical treatment.

Benign Nerve Sheath Tumors

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Abstract: Benign nerve sheath tumors present a challenge for the Neurosurgeon because they often present primarily with pain, and little motor or sensory deficit. Successful removal requires knowledge of the gross and microscopic anatomy, careful dissection under magnification, and the use of nerve action potential recording to distinguish involved fascicles from those uninvolved. Complete removal with sparing of nerve function should always be the goal. Sub-total resection or biopsy is associated with a high incidence of symptom persistence requiring additional surgery that has both a lower success rate, and a higher complication rate.

We present the case of two patients who presented with schwannomas of the median nerve in the forearm. Both had pain; one also had mild hand weakness. Both underwent total resection using intraoperative nerve monitoring and had good outcomes, with resolution of their pain, no motor and only temporary sensory deficits, and return of normal motor strength.

(Key Words: Schwannomas, nerve action potential, sensory deficit, Neurofibroma, Benign tumors)

Introduction: Schwannoma and neurofibroma are the two most common types of benign tumors that grow from peripheral nerves. The classification of these tumors had been controversial, but for the purposes of this discussion, schwannomas are considered when the pathologic specimen shows benign neoplasia of Schwann cells, the myelin producing cell of the peripheral nervous system. They typically arise from a single nerve fascicle. Neurofibromas arise from the perineural fibroblast, and intertwine themselves within several fascicles, making excision more difficult. At times the distinction between the two blurs, and the term neurolemoma is used, an inclusive term referring to the neuroectodermal origin of these neoplasms.

The surgical goal is straightforward; excise completely and preserve the nerve. En bloc removal of the tumor with the parent nerve, incomplete resection, or biopsy is unnecessary and ill advised. The use of pre-operative imaging with magnetic resonance imaging (MRI), intraoperative assistance with ultrasound imaging and nerve monitoring, operative magnification, and understanding of the anatomy allows for safe and complete removal in the majority of cases.

Although a variety of other tumors, both benign and malignant, can affect the peripheral nerve, the general surgical concepts discussed for these two most common tumors can be applied to all tumor types.

Case Report: Case #1:

A 47-year-old male presented originally in 2006 with a seven-year history of pain in the right shoulder radiating into the forearm and hand associated with burning
dysesthesias in the first and second digits and subjective hand weakness, confirmed on exam as 4/5 weakness of the grip. His past history was significant for a spinal Schwannoma removed from T11 in 1996. An MRI of the cervical spine showed age appropriate changes, and two electromyographies (EMG) were normal. An MRI of the brachial plexus was ordered and was normal. He was treated with Neurontin, but there was little improvement. He returned in 2009 with the same symptoms and findings. On exam it was noted that eliciting his bicep reflex produced focal tenderness. Palpation of the proximal volar forearm just distal to the antecubital fossa revealed a mass with a strongly positive Tinel sign. He had none of the cutaneous stigmata of Neurofibromatosis. A repeat EMG was negative, but an MRI of the forearm revealed a mass lesion with the appearance of a nerve sheath tumor (Figure 1). Surgery was performed under general anesthesia. Intra-operative EMG and nerve action potential (NAP) monitoring were used. A six-centimeter incision was opened directly over the palpable mass. Infiltration of the wound with local anesthesia was not used so as not to obscure palpation of the lesion, but was injected at the close of the procedure. The tumor was located deep to the Flexor Carpi muscle groups, in continuity with the Median nerve (Figure 2). Continuous EMG monitoring revealed no changes throughout the course of the operation. NAP monitoring directly from the exposed nerve was used to confirm nerve transmission in non-parent nerve fascicles (Figure 3). Stimulating and recording electrodes, shaped like a ‘J’ allowing cradling of the nerve, were placed respectively, proximally and distally to the tumor. Under high power magnification, the traversing fascicles were dissected away from the tumor until only the parent nerve fascicle, entering and leaving at the poles, remained (Figure 4). NAP recording from this fascicle was silent, with no action potential able to be recorded from the distal fascicle. The parent fascicle was divided at the poles of the tumor and it was removed in its entirety. Microscopic pathology revealed a fibromembranous cellular structure, and a neurolemoma was diagnosed.

Post-operatively, the patient’s pain resolved, and his strength returned to normal. Mild parasthesias of the first and second digits not present before surgery, disappeared by three months after surgery.

**Case #2:**

A 41-year-old female presented with a mass in her distal right forearm associated with several months of hand pain and paresthesias into digits two, three, and four. An attempt to drain the lump by her physician was unsuccessful and increased her pain. Examination revealed a small, hard mass on the volar wrist just proximal to the transverse carpal ligament. Palpation produced tenderness and a Tinel’s sign into the median innervated fingers. Strength and sensation were normal. An MRI of the wrist revealed an enhancing lesion of the median nerve proximal to the carpal tunnel. At surgery, the tumor was found coming from the inferomedial surface of the median nerve (Figure 3). Intrafascicular dissection, using EMG and NAP monitoring, revealed the boarders of the tumor, and separated the uninvolved fascicles from the parent one (Figure 5).

The pathologic diagnosis was Neurofibroma, however the microscopic description showed “a circumscribed spindle cell lesion composed of interlacing fascicles of Schwann cells,” suggesting a Schwannoma was the more accurate diagnosis. Following surgery her pain and paresthesias resolved, and her strength and sensation were normal.
Discussion: Fascicle sparing surgery for peripheral nerve tumors is not a new surgical technique, but because these tumors are uncommon, many surgeons, including many neurosurgeons, have little experience with them. The two patients described here presented with relatively small lesions amenable to en bloc resection taking only the non-functional parent fascicle, determined both by the magnified anatomic appearance at surgery and the electrophysiological evidence provided by EMG and NAP monitoring.

Schwannomas uniformly have a single parent fascicle entering and leaving at the tumor poles. Neurofibromas more commonly have several that appear at the poles, and NAPs become essential in determining if any are functional. If so, a redoubled attempt should be made to trace these fascicles through the tumor and spare them. If impossible, the surgeon has the option of resecting them and using a cable graft to bridge the gap.

Large tumors are at times difficult to fully isolate from the enveloping traversing fascicles without causing undo retraction on them. EMG monitoring will indicate if this is occurring, and the surgeon should use intratumoral debulking to reduce its size and allow for easier exposure of the deeper tumor surfaces. An ultrasonic aspirator is useful in this setting, although it is usually accompanied by bleeding that can be controlled with low power bi-polar coagulation to avoid heat injury to the adjacent nerves. The latter, if it occurs, is a cause of persistent pain and paresthesias in the distribution of the nerve. In rare cases, neuropathic pain occurs in addition to incisional pain after tumor removal. This is treated aggressively with medication, physical therapy, and sensory desensitization. Although it usually resolves, it came become a permanent and disturbing complication of peripheral nerve tumor surgery.

Preoperative pain often disappears quickly. Numbness and paresthesias take longer, usually several months to resolve completely, and in some cases may persist indefinitely, although it usually lessens. Weakness is treated with physical therapy.

Conclusion: Benign peripheral nerve tumors, because they are uncommon, present a challenge to many surgeons. Curative excision can be achieved in most cases, using careful microscopic technique and intraoperative nerve monitoring. Surgeon experience with both is essential, and will allow for nerve sparing and normal limb function. A sound knowledge of the nerve anatomy and its surrounding structures is necessary. Good outcomes are the rule when these principles are applied to these tumors.

Figure 4 (Left): Case 1. The tumor has been completely dissected away from the traversing fascicles leaving it attached only to the parent fascicle.

Figure 5 (Right): Case 2. Instruments holding back the uninvolved fascicles showing the isolated tumor and the parent fascicle exiting the distal pole of the tumor.

Introduction: A spinal cerebrospinal fluid (CSF) leak is a condition caused by tear or violation of the dura, and can occur anywhere within the spinal axis. Causes include blunt or penetrating trauma, post operative, post lumbar puncture, post epidural injection, spontaneous or even post extreme valsalva, such as in weightlifting. Untreated CSF leaks can cause spontaneous intracranial hypotension syndrome, which is manifested by postural headaches that improve when recumbent, dizziness, neck pain and stiffness, photophobia, vomiting and hearing loss.

Sequelaes of untreated CSF leaks included CSF fistulas, for which 25-50% of patients can develop meningitis. Up to 85% will resolve on their own after one week, the rest after six months. For those that remain symptomatic, a targeted epidural blood patch could be performed.

Diagnosis of a spinal CSF would include performing a Computed Tomography (CT) cisternogram, injecting radionuclide or nonionic contrast in the subarachnoid space followed by either nuclear imaging or thin section, high resolution multislice CT through the spinal axis. Both these techniques...
include creating an additional defect in the dura, potentially causing another leak and an exacerbation of the symptoms.¹

This case report describes a noninvasive alternative to accurately diagnosing the location and cause of the leak.

**Case Report:** A 36-year-old female with past medical history of Sjogren’s syndrome presented after a fall on ice, landing on her back and hitting her head. The patient experienced severe midline lumbosacral pain that was non-radiating but was able to drive herself home. Over the next 24 hours she developed headaches that were getting progressively worse and was admitted with a diagnosis of post traumatic head injury. CT scan of the head, cervical and thoracic spine in the Emergency Trauma Center was unremarkable. The patient was neurologically intact and her vital signs were stable. Over the next several hours her headaches worsened and she developed nausea and vomiting. Intravenous caffeine cocktail was started without significant relief; she then underwent Magnetic Resonance Imaging (MRI) of the brain which was unremarkable. Her low back pain persisted with positional headaches. Intracranial hypotension was suspected secondary to a post traumatic CSF leak. A CT myelogram was contemplated, but to avoid further violation of the subarachnoid space, an MR myelogram was performed. On a 1.5 Tesla Philips Intera MRI, sagittal and axial T2 fast spin echo (FSE) images, Single Short Fast Spin Echo (SSFSE) axial heavily weighted T2 images (TE 1000 and TR 8000), and Maximum Intensity Projections (MIPs) were performed of the entire spine. Cervical and thoracic imaging demonstrated no abnormality.

Images of the lumbosacral spine demonstrated a 3.5 x 2.6 x 1.2 cm septated midline perineural cyst at the S2 level (Figures 1, 2). This was felt to be congenital. Surrounding the cyst however, was fairly extensive extravasation of the CSF into the epidural space and surrounding soft tissues (Figures 3, 4). It was felt that the direct impact on the cyst from the fall caused it to rupture, causing a CSF leak. The patient was sent for an imaging directed blood patch. Approximately 20 ml of blood was injected into the epidural space at the sacral level. Immediately following the procedure the headaches, nausea and vomiting were resolved. The patient returned to work soon after with no further symptoms except for some mild midline sacral pain.

**Conclusion:** Our case demonstrated that MR myelography is an excellent non-invasive modality and could be a good non-invasive alternative to diagnose post traumatic cerebrospinal fluid leaks and can accurately guide therapy.

**Introduction**: Intracranial hypertension is a common feature of multiple pathologic processes affecting the brain parenchyma and its vessels, including focal pathology such as hematomas in different compartments (parenchymal, subdural or epidural), or more diffuse processes such as cerebral edema from traumatic brain injury (TBI), wedge-shaped cytotoxic-edema due to ischemic stroke, or even the infiltrating vasogenic edema remaining after a parenchymal hematoma has begun to decay and resorb. All these processes share in common the Monroe-Kellie Doctrine, which posits that within the rigid vault of the skull there is finite room for the contents, so the intracranial pressure (ICP) corresponds to the number of items trying to occupy the space. Typically, the contents are: A) Blood (both arterial and venous), B) brain, and C) cerebrospinal fluid (CSF), which occupy their respective spaces to exert a force against the inner table of skull, which is reflected backwards because the skull is (usually) a rigid box which lacks compliance.

An additional body (i.e. hematoma, swelling, tumor, pus, or extra CSF) increases the pressure and challenges the ability of the venous and CSF systems to compensate for that pressure by themselves being displaced into the venous circulation or lumbar spine cisterns. When the capacity to compensate approaches exhaustion, this threatens to culminate in herniation, whereby the intracranial contents shift mechanically down the pressure gradient, crushing or twisting/torquing structures which are perhaps remote from the initial injury and not yet irreversibly injured. The infamous “unilateral blown pupil” is the harbinger of herniation, menacing that the temporal lobe uncus will herniate over the tentorium and into the midbrain, twisting and stretching it at the expense of the patient’s mortality and future independence. Additionally, as the pressure in the skull rises with the decline in compliance, so too rises the resistance to blood flow until a state of oscillating flow occurs, where blood moves into the cranial vault during systole, but is pumped back out towards the heart during diastole because the intracranial pressure is higher than the diastolic pressure.

In short, as long as the skull is a rigid box, there is only so much room to tolerate an expanding new process, such as hematoma, tumor, pus, or cerebral edema, so management decisions are guided by the goal of decreasing intracranial contents by medical or mechanical means, increasing systemic pressure and blood flow to overcome the increasing intracranial resistance, or increasing the compliance of the intracranial compartment by removing one or more sides of the rigid box so it is less rigid (craniectomy). CT scan of the head highlights the case of a diffuse subarchnoid hemorrhage (Figure 1) and a cerebral angiography demonstrating no filling of the intracranial vessels due to elevated intracranial pressure (Figure 2).
Factors influencing intracranial pressure:

a) Hypoxia and hypotension: Although there are no large clinical trials randomizing patients to “allow hypotension and hypoxia” versus “avoid hypotension and hypoxia,” both hypoxia and hypotension are strongly associated with poor outcome after brain injury, and so the Brain Trauma Foundation has recommended monitoring blood pressure and treating patients to avoid a systolic pressure <90mm Hg. Likewise, oxygenation should be monitored and hypoxia <90% should be avoided. Although the injury pathophysiology differs in acute ischemic stroke, the American Stroke Association Guidelines identify similar thresholds for management, defining a systolic blood pressure <100 mm Hg and oxygen saturation <92% as thresholds for management in acute ischemic stroke.

b) Hypercarbia: It is generally agreed that an impaired mental status threatens the security of the airway, the principle outcome of which is impaired oxygenation and ventilation of CO2. There are no randomized clinical trials of endotracheal intubation in acute brain injury, nor are there randomized trials of permissive hypercarbia. However, it is well-accepted that hypercarbia (i.e., from hypoventilation) provokes a reflex intracranial vasodilatation, which can increase the intracranial blood volume and consequently worsen intracranial hypertension. Checking an arterial blood gas in the patient with known or suspected intracranial hypertension can allow for the identification of hypercarbia and its rectification. (The normal PCO2 range is 35-45.)

Basic bedside management:
The very most barebones basics of ICP management are usually easy to put into practice with little risk: 1) Head elevation to 30-45 degrees ought to increase jugular venous drainage while simultaneously decreasing hydrostatic pressure in the arteries. 2) Mechanically ensuring midline head position and no jugular venous constriction should maximize venous drainage, promoting metabolic waste removal and decreasing intracranial hypertension. Clinical pearl: beware the tape from the endotracheal tube in patients with intracranial hypertension—crimping the jugular veins has never been reported to help control ICP. 3) Attention to analgesia and anxiolysis can decrease overall arousal and its contribution to metabolic demand. 4) Temperature is thought to raise the cerebral metabolic rate by 10% per 1 degree Celsius, so ensuring normothermia ought to decrease metabolic debt and cumulative ischemic burden by decreasing cerebral demand for oxygen and glucose, as well as the need for waste removal (control of metabolic acid waste). More aggressive maneuvers for temperature control such as a cooling blanket or iced saline infusion, paralyzing and sedating a patient to avoid shivering are not trivial, but conservative measure such as turning on a cooling fan, putting ice packs in the armpits, and administering Tylenol frequently can be performed without much fanfare.

Medical management:
The workhorse of medical management of ICP is mannitol, the prototypical osmolar agent for brain dehydration. It has multiple proposed mechanisms, including dragging the extra water (edema) out of the brain across the intact blood brain barrier, just as salt would affect a slug, or for that matter, sugar would affect a piece of “cured” salmon. Driving up the serum sodium by decreasing total body water should decrease intracranial water content, leaving
more room for the brain and less opposition to the flow of blood to feed it. Multiple mannitol dosage regimens have been proposed, including between 0.25-1g/kg IV q6 hrs, or “enough”, not to exceed “too much.” “Too much” is difficult to assess, since we have no direct, rapid laboratory assessment of the serum mannitol dose. However, serum osmolality is an indirect assessment, with “osmolar gap” likely the best. It is feared that if the osmolar gap exceeds 20, then the risk of harm from renal injury begins to rise. Another respectable player in the hyperosmolar arsenal is hypertonic sodium chloride, with hypertonic sodium lactate gaining some penetrance in the literature. To date, in trials of hyperosmolar treatment, all trials have compared apples to oranges, while no published trial yet has compared equivalent osmoles to osmoles. It would seem the question for the hour is “are x osms of hypertonic saline the same, better or worse than the same number x osms of mannitol?” Certainly, such data are awaited. In the meantime, we are left with “use enough and not too much.” Helpful guidelines are not to exceed 20mosms of mannitol, as calculated using the osmolar gap. The absolute serum sodium and serum osms can be misleading for reasons including serum glucose as well as lipids. Mannitol has the added benefit of being a diuretic, so if diuresis is a desirable side effect of hyperosmolar treatment, then mannitol is helpful (i.e. patient with heart failure). At the same time, with mannitol one must be very careful not to ignore intravascular volume depletion via osmotic diuresis, as mannitol could culminate in hypovolemia and consequent inadequate cerebral perfusion if blood volume loss through urine output is not carefully repleted, the exact opposite effect usually desired by practitioners ordering mannitol infusions. If, on the other hand, one favors augmented perfusion pressure, hypertonic saline of multiple tonicities pulls water from the interstitial and intracellular spaces to amplify the intravascular volume.

In the patient who is comatose, and in whom one has chosen to employ a pressure monitor for therapeutic guidance, how low should the ICP be, and/or how high should the cerebral perfusion pressure (CPP) be? These are questions without widely accepted, evidence-based answers. A reasonable reference comes from the Brain Trauma Foundation, which has found a gentle evidence-based compromise between European (Lund) and American (Rosner) schools of thought in that the CPP should not be allowed to drop below 50mmHg, while the CPP should not be augmented above 70mmHg due to appearance of harm (ARDS) associated with hypervolemia. We are left with “keep it between 50-70mmHg, but don’t raise it above 70mmHg.”

Hyperventilation: It is widely accepted that blowing down the PCO2 can cause reflex intracranial vasoconstriction, shunting a tiny fraction of intracerebral arterial blood out of the head and lowering ICP. Hyperventilation may be useful as a stop gap measure in order to buy the patient some time for a ride to the OR for decompression or perhaps a central line for hypertonic saline administration, but it is not sustainable and should not be the first line or second line therapy for anyone trying to control ICP. There is reasonable randomized-controlled evidence that sustained hyperventilation worsens clinical outcome.3 This is likely for the same reason it controls ICP acutely: vasoconstriction works transiently to decrease intracranial volume/pressure, but it opposes cerebral perfusion, and inadequate perfusion leads ultimately to cytotoxic cerebral edema and neuronal injury.

Physiologic suppression using sedation: One strategy to protect the brain from pressure and metabolic challenges such as ischemia is to turn it off, like a bear in hibernation which does not feel the pangs of hunger while it is asleep. Physiologic suppression using sedation can be achieved with multiple sedating medications, all culminating in the electrographic state called “burst suppression.” Unfortunately, all the sedatives commonly used for achieving burst suppression also cause significant concomitant increased demand on artificial life support, including mechanical ventilation, pressor support to keep the patient out of hemodynamic shock, and occasionally ileus. The Brain Trauma Foundation has reviewed the available evidence and recommended that high-dose barbiturate administration is recommended to control elevated ICP refractory to maximum standard medical and surgical treatment.4 In other words, “barbs” are useful “when all else fails”.

Physiologic suppression using neuromuscular blockade (drug-induced paralysis) has not been addressed in a randomized fashion.

Physiologic suppression using hypothermia: The Brain Trauma Foundation has commented on the role of prophylactic hypothermia, but refrained from commenting on the use of therapeutic hypothermia for increased ICP due to inadequacy of studies reporting its use. Essentially, the
BTF does not support prophylactic hypothermia for mortality benefit, though they suggest it may have a functional outcome benefit.

Surgical management:
The bread and butter of the neurosurgeon to remove the bad humors, in this case the extra mass. Unfortunately, much of practice is not based on large randomized clinical trials, which are the gold standard of evidence-based medicine. CSF can be removed easily via an extraventricular drain, and we know intracranial hypertension culminates in poor outcomes if left to rise without check, so the practical rule of thumb is do the easy thing to control it first. CSF drainage is easy, usually. There are no randomized data to support CSF drainage in intracranial hypertension, but many would argue that if you suspect and are going to measure ICP, do it with a device which can also drain the fluid (as compared to a device which can monitor without providing a concurrent therapy). If there is an epidural hematoma or symptomatic subdural hematoma, take it out. Intraparenchymal mass removal is more complicated. STICH, the largest randomized trial of parenchymal hematoma removal in primary hemorrhage did not demonstrate a net outcome benefit of surgical evacuation; however, retrospective subgroup analysis suggested statistical benefit when the hematoma border was <1 cm from the skull, presumably relatively easy to drain without causing collateral brain damage. In the case of cytotoxic cerebral edema after ischemic stroke, pooled randomized controlled trial analysis revealed that early (<48 hrs) decompressive hemicraniectomy (DHC) after malignant cerebral edema provided not only a survival benefit, but also increase in the percentage of patients with good functional outcome. That DHC can statistically increase functional outcomes in addition to survival argues that it is more than just a “salvage” intervention. The counter-argument against DHC is that patients who might have died from herniation after malignant cerebral edema were saved, only to spend the remainder of their days dependent in a nursing home. In TBI with diffuse cerebral edema, unfortunately, there are no published randomized trial data yet re: the “ifs” or “whens” of decompressive hemicraniectomy, so it remains a matter of opinion whether it should be a first, second, or third tier intervention in the treatment of intracranial hypertension. Is it a last-ditch effort to “salvage” the patient with “refractory” intracranial hypertension, or should it be used long before other intensive medical maneuvers are considered? In the absence of randomized data, we must settle for opinions and observations, such as that decompressive craniectomy, deemed by some to be “definitive” pressure reduction, lowers cumulative ischemic burden and decreases therapeutic intensity levels after TBI.

Conclusion: Intracranial hypertension is a common feature of multiple forms of brain injury, ranging from ischemic to hemorrhagic stroke, as well as traumatic brain injury, which is perhaps the best studied with respect to treatments affecting intracranial volume and pressure. Many important therapeutic questions have yet to be answered in a solid, evidence-based fashion, such as “hypertonic saline versus mannitol.” Probably the maxim “do the easy things first” is one with some wisdom behind it: raising the head of the bed, ensuring adequate oxygenation and resuscitation, as well as dehydrating the brain and/or removing a bone flap to increase intracranial compliance are probably less fraught with complications than trying to achieve neuroprotection via complete physiologic suppression with drugs (barbiturates or other sedatives), neuromuscular blockade, and therapeutic hypothermia.

Extending the Treatment Window for Patients Who Wake Up with Acute Ischemic Stroke

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Abstract: Stroke is the leading cause of disability and third leading cause of death in the US. Intravenous recombinant tissue plasminogen activator therapy is only instituted to patients who present within 3 hours of the time of onset and now with extended window up to 4.5 hours in selected group of patients. Intra-arterial therapy can be instituted up to 8 hours from the time of stroke symptom onset, but with the utilization of newer radiological techniques especially computed tomographic angiography and perfusion magnetic resonance diffusion and perfusion scans, intra-arterial therapy may be used for selected patients who wake up with stroke symptoms. We present a case of an 86-year-old woman who woke up with right hemiplegia, right visual field loss and aphasia (National Institute of Health Stroke Scale 20). Head CT was suggestive of a left middle cerebral artery occlusion with a large perfusion deficit. The patient was treated with intra-arterial thrombolytic therapy with complete recanalization. Her symptoms improved significantly and she was discharged to a rehabilitation unit. This case shows the importance of recanalization and clinical improvement in selected group of patients.

(Key Words: Intravenous recombinant tissue plasminogen activator, Intra-arterial thrombolysis, Wake-Up Stroke, Merci Clot Retrieval Device, Computed Tomography, Angiography, Perfusion scan)
**Introduction:** Stroke is the leading cause of disability and the third leading cause of death in the United States. It affects approximately 795,000 individuals annually, translating into about one stroke every 40 seconds. Eighty-seven percent of strokes are ischemic and 13% are hemorrhagic. In the United States, more than 4 million people have survived stroke but they are limited with some form of neurological impairment or disability. In the United States, the overall economic impact of stroke exceeds $45 billion annually. There is a persistent goal amongst practitioners to prevent, emergently treat and maximize recovery for stroke.¹

**Case Report:** An 86-year-old woman woke up with right arm and leg weakness, right visual field loss, and language impairment. The patient was brought by Emergency Medical Services (EMS) to Abington Memorial Hospital. Her initial National Institute of Health Stroke Scale (NIHSS) was 20. Because of the unknown time of onset, the patient was not considered a candidate for intravenous rt-PA. The patient underwent CTA/CTP, which showed a significant perfusion mismatch (Figure 1), demonstrating a large area of ischemic tissue at risk. She was emergently taken to the Neurovascular Lab for recanalization. A 6 French sheath was placed and through the sheath, a 6 French Neuron guide catheter was advanced and was positioned in the left internal carotid artery. Cerebral angiogram showed complete occlusion of the inferior division of the middle cerebral artery (TIMI 0) (Figures 2a, 2b). Then, through the guide catheter, a Distal Access catheter (DAC) was advanced and positioned in the supraclinoid segment of the internal carotid artery. Through the DAC catheter, a Merci retrieval catheter was advanced over a Synchrho 0.014” microwire. Through the microcatheter intra-arterial Tenecteplase (3mg) was instituted. Then a V 2.0 firm Merci Clot Retriever Device was advanced and after 2 passes the clot was retrieved with complete recanalization (TIMI III) (Figures 3a, 3b, 3c). Total time to restore cerebral blood flow was 1.5 hours. The patient remained in the Neuro Intensive Care Unit for 24 hours. Her neurological examination over a 24-hour period improved rapidly. The patient was discharged to acute rehabilitation three days after procedure. At the time of rehab discharge to home (one week), the patient demonstrated minimal disability in language skills only, was able to move all extremities and had normal vision (NIHSS 2). Secondary stroke risk reduction

**Figures 2a, 2b:** Cerebral angiogram; anterior-posterior and lateral view of the left internal carotid artery, demonstrating complete occlusion of the inferior division of the left middle cerebral artery, marked by arrows.
including antiplatelet therapy in conjunction with the treatment and management of hypertension and hyperlipidemia was performed.

**Discussion:** Recognizing stroke symptoms and seeking emergency treatment is vital to functional stroke recovery. The only Food and Drug Administration (FDA) approved therapy for acute ischemic stroke is intravenous recombinant tissue plasminogen activator (rt-PA), which has been formally approved for administration within 3 hours of symptom onset. The dim reality is that < 5% of stroke patients actually receive intravenous rt-PA nationally.

**New extended window: 3-4.5 Hours:**

The European Cooperative Acute Stroke Study (ECASS-3) trial represents an important advancement in the treatment of acute ischemic stroke, showing benefit of rt-PA in the 3-4.5 hour time window. The results of this study are consistent with the results from pooled analyses of prior trials. Based on this, the American Stroke Association now recommends treatment with IV rt-PA up to 4.5 hours after stroke onset for appropriately selected patients (Class 1 Recommendation, Level of Evidence B).2,4

**Intra-arterial Thrombolysis: 3-8 Hours:**

The emergence and utilization of intra-arterial (IA) therapies and mechanical clot retrieval procedures have also offered more options to decrease cerebral damage. Intra-arterial thrombolytic therapy and mechanical clot retrieval procedures have extended the treatment window 3-8 hours and in some cases >8 hours such as basilar artery occlusion.1

**Wake Up Strokes:**

Approximately 16% to 28% of ischemic stroke patients wake up with their deficits. The last seen-normal time is defined as the time the patient went to sleep, which places these patients outside the window for the intravenous thrombolytic therapy. With the institution of new radiological techniques, it is possible now to effectively treat the selected group of patients.3 Computed tomography angiogram and perfusion (CTA/CTP) provides crucial information reflecting cerebral blood volume (CBV), cerebral blood flow (CBF) and mean transit time (MTT) within seconds. With the identification of perfusion mismatch, intra-arterial therapy can be administered.1

**Conclusion:** Our case demonstrates that advanced neuroimaging may facilitate selection of appropriate patients for interventional stroke management even in those cases where the patient wakes with symptoms and the time of onset in unknown. With quick recognition of stroke symptoms, notification of 911 and triage to a comprehensive stroke center that provided advanced stroke therapies, this “Wake Up Stroke” patient was offered a chance to recover from very severe disability.

Abstract: This paper presents the case of a patient diagnosed with an acute ischemic stroke while in the hospital. It discusses both the pathophysiology associated with ischemic stroke, as well as diagnostic measures used in determining ischemic stroke. Finally the paper discusses care of the patient diagnosed with acute ischemic stroke from the role of the nurse practitioner.

Ischemic Stroke Case Study:
The patient is a woman in her late 70s who presents to the hospital with left lower quadrant abdominal pain. Her past medical history includes: hypertension, coronary artery disease, dysrythmia, hypothyroidism, high cholesterol, and cardiac arrest. Her surgical history includes a four vessel Coronary Artery Bypass Graft (CABG) and defibrillator placement. The patient has residual left arm weakness after a cardiac arrest. Computed Tomography (CT) of the abdomen showed diverticulum with a fistula. It was evident that the patient would need surgery based on CT scan results, although further workup was needed based on her extensive past medical history. The patient was immediately started on a regimen of antibiotics, deep vein thrombosis prophylaxis, and proton-pump inhibitor prophylaxis, along with her prescribed oral medications.

The patient was then worked up for her medical issues for the next several days before undergoing a diverting loop colostomy. The day after surgery, nursing staff noticed a change in the patient when attempting to get the patient out of bed to the chair. The patient had paralysis on the left upper extremity, decreased movement on the left lower extremity, while her right side remained with full strength. The patient complained of an occipital headache and a pupil check revealed unequal pupils. She also had a left-sided facial droop and a left-sided visual field deficit. CT angiogram of the brain was ordered, as an MRI was contraindicated for this patient due to the presence of her defibrillator. CT angiogram revealed a new large perfusion mismatch in the right middle cerebral artery territory without discrete cut off noted in the parent vessel. The CT scan also revealed a moderate narrowing of the distal brachiocephalic artery. Patient was not considered a candidate for intrarterial mechanical thrombectomy as there was no vessel occlusion noted. After CT scan, the patient was transferred to the intensive care unit, where the course of treatment was extensively discussed by various members of the medical team, including the surgeon, neurologist and interventional neurologist. It was decided that this patient was a candidate for intravenous recombinant tissue plasminogen activator (rt-PA), despite the fact that the patient had a loop colostomy the day before. Although typically a recent surgery prevents patients from receiving rt-PA, the physicians concurred that this surgery was minor and the benefits of using rt-PA outweighed the risk. Unfortunately, neurological symptoms persisted in this patient despite intervention occurring within three hours. The patient remained with facial droop, field deficit on the left side, decreased movement on the left lower extremity, and no movement in the left upper extremity. A repeat CT scan revealed infarction in the middle cerebral artery distribution.
An Introduction to Stroke:

Stroke is a condition in which cerebral blood flow is compromised to the point where it causes neurological symptoms in the patient. There are two major categories for stroke: hemorrhagic stroke and ischemic stroke. Hemorrhagic strokes most often occur at the site of aneurysm or arteriole rupture. Damage from hemorrhagic strokes can often be more devastating because regions of the brain not in direct contact with the hemorrhage can be affected due to cerebral edema, cerebral compression, and increased intracranial pressure.

Ischemic stroke can be divided into two major categories: embolic and thrombotic. An embolic stroke occurs when a blood clot lodges in an artery of the brain that is too narrow for the clot to pass through and blocks the flow of blood. Embolic strokes often originate from the heart, as plaque can break off from the endocardium in conditions such as atrial fibrillation, endocarditis, valvular prosthesis, and myocardial infarction. A thrombotic stroke occurs due to a narrowing of cerebral blood vessels from atherosclerotic plaque buildup within the vasculature. Often, a thrombotic stroke is preceded with a transient ischemic attack, which is reversible neurological symptoms that last several minutes. Both embolic and thrombotic strokes present with neurological symptoms based on the vessel occluded. The severity of symptoms associated with ischemic stroke can depend on a variety of factors, including the patient's blood pressure, the presence of collateral blood flow, the vessel affected, and the anatomy of the vessels.

Epidemiology and Statistics Related to Stroke:

Stroke is the third leading cause of death among people in the United States, and it is the greatest cause for long-term disability within the United States. Every year, 795,000 people are afflicted with strokes, with 185,000 of those strokes being recurrent attacks. Men have a greater risk of having a stroke at a younger age than women, but the rate of overall strokes is higher in women due to the fact that statistically women live longer. African American individuals are approximately twice as likely to have a stroke as compared to Caucasian individuals, and Mexican Americans are also more likely to have both ischemic and hemorrhagic strokes than Caucasians. It is estimated that the majority of strokes in the United States are ischemic at 87 percent, and the remaining 13 percent are hemorrhagic intracerebral and subarachnoid strokes.

There are a variety of factors that influence the incidence of stroke. Patients who have atrial fibrillation have a five times greater likelihood of experiencing stroke than those who do not. Smoking doubles the chance of stroke and studies have also shown an increased incidence of ischemic stroke in post-menopausal women taking hormonal replacement therapy. Hypertension is one of the most major risk factors for both hemorrhagic and ischemic stroke. People with blood pressure measurements less than 120/80 are half as likely to experience a stroke as those with higher readings. Transient Ischemic Attacks have also been shown to be a major risk factor for stroke as the risk of stroke is 10 percent in the week following a transient ischemic attack. Finally, the risk of stroke increase to 2.4 times more likely in those that are pregnant and in the six weeks post-delivery than those that are not.

The cost of both direct and indirect care related to strokes is estimated at 68.9 billion dollars this year so far. Beyond the high costs associated with stroke, there are also physical and emotional residuals that result from strokes. Many patients may experience a lifetime of disabilities such as aphasia, hemiparesis, inability to perform activities of daily living, and inability to walk. The long-term emotional, cognitive, and physical impact on patients and their families is difficult to measure.

Pathophysiology of Stroke:

The major contributor to ischemic stroke is atherosclerosis. Atherosclerosis occurs in the endothelium of the large arteries of the neck and brain. It occurs after injury to endothelial cells due to a variety of factors, including: mechanical, inflammatory, and biochemical insults. Plaque develops as a result of endothelial injury and increases the turbulence of blood in the area of damage. Plaque development provides a spot for platelet aggregation to occur. This platelet aggregation can eventually be significant enough to block the vessel completely or rupture off, leaving the patient susceptible for embolic consequences.

Once ischemic stroke has occurred and an area of the brain has lacked perfusion, a loss of neurons occurs. Damage to these critical cells results in a stroke lesion, which is an area of cell death. It is thought that injury from stroke is a result of astrocytes not being able to support the metabolic needs of the neurons during this critical period. When a stroke lesion occurs, the area surrounding the lesion called the penumbra is also at risk for damage. The penumbra is moderately hypoperfused during a stroke and is at risk to become necrotic if intervention is not taken to help restore blood flow to the area.

Clinical Manifestations:

An ischemic stroke can manifest itself in a variety of ways, including motor function abnormalities, communication problems, affect, intellectual functioning, and spatial-perceptual alterations. Sometimes, the symptoms of stroke can be confused with a variety of other neurological conditions, including brain tumors, encephalopa-
Diabetes, atrial fibrillation, and congestive heart failure are all modifiable risk factors for stroke.11

Asymptomatic carotid stenosis can also be considered a modifiable risk factor, as patients discovered to have this condition may choose to undergo carotid endarterectomy to correct the issue, thus reducing the chances of stroke. Smoking is yet another modifiable risk factor that nearly doubles the risk of stroke in patients.3 Some other modifiable risk factors for stroke include use of oral contraceptives that contain high dose estrogen, post menopausal hormonal therapies, alcohol consumption, drug use, physical inactivity, obesity, and poor diet.3 Finally, patients that have hypercoagulation disorders and disorders like Sickle Cell Disease have an increased incidence of stroke.3

Risk Factors for Stroke:

There are multiple modifiable and non-modifiable risk factors for stroke. Some of the non-modifiable risk factors for stroke include a patient’s age, race, gender, and family history.3 Among the modifiable risk factors, the most important is hypertension, yet it is often not properly treated and under detected.2 Also, coronary artery disease is a major risk factor for stroke, which placed this particular patient at great risk.10 Beyond this patient’s past medical history of coronary artery disease, she also had a history of high cholesterol, which has been shown in congruence with hypertriglyceridemia to increase the risk of stroke.11

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Diagnostic Testing for Stroke:

Before providers make a radiologic diagnosis of ischemic stroke, they must first do a quick and accurate physical assessment, take a medical history, and establish the onset of the symptoms. Due to the narrow timeframe that thrombolytic therapy can be given post ischemic stroke, it is absolutely essential that tests and physical exam be performed in a timely manner. The National Institute of Health Stroke Scale is a test which can be completed in five to eight minutes to test patients for symptoms of stroke. After physical examination patient can undergo a diffusion/perfusion weighted Magnetic Resonance Imaging (MRI). Unfortunately, due to time constraints and patient contraindication such as in the case of a pacemaker, MRI is not feasible for most patients. Often, the chosen method for ischemic stroke diagnosis is a non-contrast head CT or a CT angiogram to evaluate the vasculature of the brain. Both of these tests can be performed in a fraction of the time it takes to perform an MRI and offer the advantage of being less sensitive to motion disturbances than MRI. Non-contrast brain CT scans often do not detect areas of ischemia, but are sensitive enough to detect other brain abnormalities, including acute hemorrhage, lesions, and abscesses.4

Performing a CT angiogram of the brain is quite valuable in the diagnosis of ischemic stroke, even more of an asset than a traditional non-contrast head CT scan. CT angiogram images are able to give more valuable information in the diagnosis of ischemic stroke, including the extent of infarction, collateral circulation, and the penumbra.6 CT angiogram aids physicians in deciding the method of treatment for the patient, as mechanical clot retrieval or intra-arterial thrombolysis may be a viable option for the patient based on the results of the angiogram.9

One of the major barriers that can hinder the test being performed is patient cooperation. If the patient is combative, light sedation may be necessary in order to complete the test with minimal patient movement. Interpretation of the results of the CT angiogram can come from both radiologists and interventional neurologist. The results may be looked at as the test is being performed in order to make a quick diagnosis and perform the necessary interventions for the patient.

Treatment Options for the Ischemic Stroke Patient:

In the case of ischemic stroke, “time is brain.” Thrombolytics, such as rt-PA are given to patients intravenously if the patient meets certain diagnostic criteria and the diagnosis is made within a three-hour window of onset of symptoms.3 The goal behind rt-PA is to bust clots in cerebral circulation in order to reestablish blood flow to an area of ischemia. Studies have shown a direct correlation between the time of rt-PA administration and the overall outcome of the patient; if the patient receives rt-PA quickly, the patient is more likely to have a better outcome clinically as opposed to those who receive rt-PA just within the three-hour window.13 Patients with ischemic stroke may also be started on a regimen of platelet inhibitors or anticoagulants
24 hours after being treated with rt-PA in order to prevent a recurrent stroke.\textsuperscript{3} Beyond the established treatment of intravenous rt-PA and oral agents used to treat ischemic stroke, endovascular treatment is now becoming a standard of care in ischemic stroke management. Endovascular treatments are aimed at restoring blood flow to the brain just as with rt-PA, and are often used when patients do not meet the criteria to receive rt-PA.\textsuperscript{13} Endovascular treatments consist of intra-arterial thrombolytics directly at the site of the occlusion and clot retrieval with the use of mechanical equipment.\textsuperscript{13} The benefits of treating ischemic stroke with endovascular treatments is that attempts can be made outside of the three-hour window, offering a decreased incidence of intracranial hemorrhage that can result from giving intravenous rt-PA too late.\textsuperscript{13}

**Role of the Nurse Practitioner in Ischemic Stroke:**

The most essential role of the nurse practitioner who works in an outpatient setting is education. Ischemic strokes have a variety of modifiable risk factors, and if patients know the proper preventative techniques, the overall incidence of stroke would decrease.\textsuperscript{3} Furthermore, community education should be aimed at learning the symptoms of stroke and the importance of seeking medical attention rapidly so there is a possibility for the patient to receive thrombolytic therapy. Finally, nurse practitioners working in the outpatient setting should properly screen patients for stroke risk including physical examination and testing, and place patients on medications accordingly to control those risk factors.

In the acute care setting, the role of the nurse practitioner is to first and foremost remember the basics of protecting the patient's airway, breathing, and circulation.\textsuperscript{1} Patients who have experienced a stroke may have a decreased level of consciousness, become unable to protect their airway, and therefore require intubation.\textsuperscript{3} Furthermore, just as it is the physician's goal to rapidly assess potential stroke patients, it is also the role of the acute care nurse practitioner to establish onset of symptoms, take a thorough history, and rapidly assess stroke patients so that diagnostic measures can be taken as soon as possible. Elevated blood pressure is common in those patients with ischemic stroke, and anti-hypertensive medication should not be prescribed unless the patient's systolic blood pressure is greater than 220 or mean arterial pressure is greater than 130.\textsuperscript{3} Finally, the acute care nurse practitioner should be in constant communication with the physician to organize the patient's care. Decisions must be made whether intravenous thrombolytics will be used or endovascular treatment will take place. After interventions to reduce ischemia have taken place, the patient must be medically managed with everything from medication regimens, lab work, follow-up scans, nutritional intervention, physical, and occupational rehabilitation. The role of the acute care nurse practitioner is extremely important in being sure that proper medical management is taking place, as the incidence of recurrent ischemic stroke is significant.

**Summary:** Ischemic stroke is a devastating diagnosis that afflicts many patients and families across the United States. Ischemic strokes have a variety of modifiable risk factors, and if patients are properly medically managed, the overall incidence of strokes will be reduced. When a patient shows signs and symptoms of a stroke, it is essential that the patient receive medical attention immediately, as many interventions for ischemic stroke are time sensitive treatments. CT angiograms are often performed to make the diagnosis of ischemic stroke and to determine the best course of treatment for the patient. The goal of the nurse practitioner when it comes to ischemic stroke is to manage the patient's medical care and collaborate with physicians to make sure the patient is receiving proper treatments and rehabilitation services. Finally, the goal of all healthcare practitioners should be to educate patients and their families on risk reduction techniques and the need to seek medical attention immediately at the onset of symptoms.

Development of the Neurointermediate Unit: Goals And Objectives

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Introduction: According to the American Association of Critical Care Nurses, the Synergy Model for patient care allows for the best possible patient outcomes. To follow the synergy model, patient’s needs and characteristics are matched with the nurse’s competencies (American Association of Critical Care Nurses). In keeping the above concept in mind, we established our new Neurointermediate Care (NIC) unit. This article describes the establishment of this new unit, nursing training, goals, and case examples to demonstrate the importance of the unit in patient’s care and outcome. Future goals of this unit are also described.

a) Background: Intermediate level care is an option for less critical patients with nursing care and monitoring needs that no longer meet intensive care unit (ICU) criteria yet does not yet meet criteria for medical-surgical level care. Intermediate units can also be a solution for patient flow. In theory, intermediate units reduce the holds in the Emergency Trauma Center (ETC) and Post Anesthesia Care Unit (PACU). Patients are less likely to be readmitted to the ICU when moved from ICU to Intermediate Care, rather than directly to a medical-surgical level bed. Additionally, there is a reduction in total cost if a patient is in intermediate care rather than ICU. The Neurosciences team felt that a NIC unit was necessary to provide a synergistic model to neurology care at Abington Memorial Hospital. There are better outcomes, fewer complications and shorter length of stay in a synergistic unit. The smaller nurse to patient ratio provides for rapid evaluation and frequent monitoring.

b) Initial planning phase: Developing the NIC unit on 3 Widener West (3WW) at Abington Memorial Hospital was a collaborative approach. The planning committee comprised of nursing leaders from 3WW, PACU, ETC, ICU, catheterization lab, and physicians from Neurology, Neurosurgery and Neurovascular. Nursing was responsible for writing a policy/procedure, fact sheet, admission and exclusion criteria, and nursing requirements for the NIC unit. Admitting privileges were given to Neurology, Neurosurgery and Neurovascular physicians. A neurologic diagnosis is required for admission to the NIC. Nursing care is provided at 3:1 patients to nurse ratio compared to 5:1 patients to nurse ratio on 3WW Neurology Unit.

c) Nursing training and monitoring:
NIC was created on 3WW and consists of three designated rooms. Currently NIC unit patients are evaluated by the National Institute of Health Stroke Scale (NIHSS) every eight hours as well as vital signs and a full neurologic assessment every two hours by the nurses. To prepare for the opening of the NIC, nurses were tested on accuracy of NIH Stroke Scale, neurologic assessment, knowledge of neurologic changes and monitoring requirements. Continual monitoring of nursing assessment accuracy is routinely provided. Continuing education on neurologic topics is scheduled monthly.

d) Outcome statistics: We looked at multiple outcome data, primarily length of the hospital stay. In theory, the intermediate unit should allow patients to move out of the ICU faster thus shortening the length of the
ICU stay. Other data includes the rate of hospital acquired deep vein thrombosis (DVT), aspiration pneumonia, pressure ulcers, urinary tract infection, and falls. The NIC unit officially opened on March 1, 2010. In the first two months, the NIC unit has taken care of a total of 45 patients with great success. Thirty-eight percent patients were discharged home and thirty-three percent were admitted to an acute level rehabilitation. Twenty-nine percent of patients were admitted to other levels of care such as skilled nursing facilities, hospice and psychiatry. Less than 7% (three patients) were readmitted to ICU. Average length of stay in the NIC was two days. The average hospital length of stay was 6.4 days. The most common diagnosis was intracranial hemorrhage. There have been no incidence of aspiration pneumonia, DVT, falls, pressure ulcers, hospital acquired urinary tract infection or central line infections.

A majority of the NIC unit patients are complex requiring treatment and attention not only for their neurological problems but also frequent attention to other co-morbid conditions. A strong interdisciplinary team cares for the NIC unit patients including nursing, physicians, therapists and counselors. Physical therapy has become a strong link with nursing on 3WW as well as the NIC unit. The physical therapist sees the patients on the floor in the room, using the hallways as their track. With nursing close by, if the patient was to decompensate, supplies and support is not far away. These patients also get the opportunity to be seen more than once a day if time allows.

Case examples:

Case 1: A 53-year-old man with a history of untreated hypertension was seen in an outside emergency room for right hemiplegia and aphasia. Computed Tomography scan (CT) of the head confirms large left hemispheric intraparachymal hemorrhage with left ventricular extension. He was transferred to Abington Memorial Hospital for management and care. Patient was admitted to the Neuro ICU. After stabilization and successful extubation, patient was transferred to the NIC unit. During his stay in the NIC unit, patient’s mental status waxed and waned. He would open his eyes to sound and make eye contact but minimal speech, mostly unintelligible sounds. He continued to have a right visual field cut, right facial palsy, and no movement of the right arm and leg. With continued aggressive medical support patient started to improve and participated in physical therapy. He tolerated one to two hours out of bed at a time. Patient was continually assessed for aspiration, DVT, wounds and urinary tract infection. Finally, the patient was accepted and transferred to an acute rehab facility to continue his rehabilitation.

Case 2: A 60-year-old female with a history of headaches and an acoustic neuroma was admitted to the emergency room for complaints of the “worst headache of her life” with photophobia, nausea and vomiting. A CT of the head illustrates a Fisher Grade III subarachnoid hemorrhage and three cerebral aneurysms. She underwent endovascular coil embolization of all three aneurysms by the neurointerventional team. Patient was stabilized and transferred to the NIC unit on day 14 of the treatment with complete recovery. Patient developed an acute onset of severe headache and developed generalized tonic-clonic seizure. Nursing coordinated an immediate transfer to ICU and a CT scan of the head, which showed rebleeding. Because of the urgent attention by the nursing staff patient’s symptoms were rapidly identified and she was retreated in the cath lab. After stabilizing, the patient returned to the NIC unit.

Patient was continually assessed for aspiration, DVT, wounds and urinary tract infection. Patient was considered a falls risk at all times and interventions initiated as appropriate.

Conclusion: Working with the NIC patients with multiple co-morbidities, the neuro nurses have sharpened their neuro assessment skills and critical thinking. The NIC unit staffing comes out of the Neuro unit’s staffing making it necessary to be creative with staffing when the NIC is not full or when census changes rapidly. Over the first two months, the neuro nurses have formed a strong collegial bond with the whole neurologic interdisciplinary team. It is important to the success of the unit and essential for good patient outcomes that the neuro nurses feel empowered to call and question orders, verbalize concern for a patient’s condition and request help. Finally, no one could have predicted the high acuity seen in the NIC unit in the first two months. It has opened the nurse’s eyes to a whole new level of nursing care. Case 2 is a prime example of the rapid evaluation/assessment and necessary treatment.

This case will be used as a learning tool for further nursing education.

Currently 3WW has a remote telemetry monitoring and capability of monitoring Lumbar drains. Looking ahead to the future, to expand the NIC additional monitoring capabilities will be necessary, including central venous pulse (CVP), external ventricular drainage (EVD), radial and femoral arterial lines. Additional patient populations would also be identified. Further education, such as Advanced Cardiac Life Support (ACLS), would be necessary for the nursing staff.

With the addition of the NIC, the neurosciences department has improved the services available to the patients.
1) Cervical Aortic Arch: A Rare Anatomic Variant:  
Presented by: Jennifer Villa Frabizzio, MD

The 86-year-old male patient presented for CT angiogram as part of workup for carotid stenosis. Angiographic images demonstrate the presence of a cervical aortic arch, a rare anatomic variant. Review of the literature suggests that no more than 50 cases have been reported to date.

A cervical aortic arch is defined when the aortic arch extends above the clavicles. Though the exact embryogenesis is uncertain, it is thought to be due to anomalies in the involution and persistence of several of the six pairs of dorsal aortic (brachial) arches, particularly the second, third and fourth arches. Cervical arches can be asymptomatic or present with dysphagia or respiratory symptoms due to compression of the esophagus and trachea from a vascular ring. They may present as a pulsatile supraclavicular mass. Eighty percent are right sided arches, with absence of the aortic knob on the left. The aorta descends normally on the left but extends behind the trachea and esophagus and displaces them anteriorly. The branching pattern varies may be normal or mirror image as in our patient. Our patient also had a moderate stenosis of the left subclavian artery, just adjacent to persistent diverticulum of Kommerall, a remnant of the ductus arteriosus. (Image 1.1).

2) Persistent Trigeminal Artery

*Presented by:* Qaisar A. Shah, MD and Osman S. Kozak, MD

Persistent Trigeminal Artery is the arterial communication between internal carotid and the basilar artery. Its incidence is 0.1–0.2% in general population.

Persistent Trigeminal Artery usually arises from the petrous portion of the internal carotid artery and connects with the distal third of the basilar artery between the origin of superior and anterior cerebellar arteries. The anomaly can be associated with intracranial aneurysm, AVM, CCF and moyamoya, in nearly 25% of cases (Image 2.1).

3) Superficial Temporal Artery Aneurysm

*Presented by:* Qaisar A. Shah, MD and Paul V. O’Moore, MD

A 19-year-old man presented with pulsating and increasing right scalp mass after blunt head trauma a few months previously. Ultrasound and CT angiogram demonstrated a pseudoaneurysm originating from the superficial temporal artery (branch of the external carotid artery) (Image 3.1, Image 3.2).

4) Distal Cerebral Aneurysm

*Presented by:* Qaisar A. Shah, MD, Abington Memorial Hospital, and Yelena Shpigel, MD, Holy Redeemer Hospital

A 74-year-old woman with a history of anti-cardiolipin antibody syndrome was admitted after two episodes of vertigo and diplopia. She was evaluated with cerebral angiogram for known distal middle cerebral artery aneurysm, to identify the size and location for the purpose of anticoagulation.

Cerebral angiogram demonstrated an unusual location of the aneurysm (Image 4.1, Image 4.2), measuring 3.5 mm with wide neck.
5) Mycotic Aneurysm

Presented by: Qaisar A. Shah, MD and Osman S. Kozak, MD

Mycotic cerebral aneurysms are a rare but serious complication of infective endocarditis. Mycotic aneurysms occur in 2% of all patients with infective endocarditis. Ruptured aneurysms lead to high mortality (>90%) in untreated patients.

Clinical course:

Our patient had a ruptured mycotic aneurysm with subarachnoid hemorrhage (Image 5.1). He was treated with microcatheterization of the aneurysm, followed by placement of the coils and complete obliteration of the aneurysm. (Image 5.2a, Image 5.2b). He developed re-growth after two months of the aneurysm treatment (Image 5.3). This was treated with parent vessel occlusion using Onyx with selective WADA testing (Image 5.4).

Image 5.1: Un-enhanced CT scan showing subarachnoid hemorrhage in the left parietal region (arrows).

Image 5.2a: AP and lateral view of the cerebral angiogram demonstrating mycotic aneurysm in the distal left MCA branch. Image 5.2b: Cerebral angiogram, AP and lateral views, showing coil placement within the aneurysm with no filling (arrow). Image 5.3: Lateral view of the cerebral angiogram demonstrating re-growth of the mycotic aneurysm (arrow). Image 5.4: Lateral view of the cerebral angiogram demonstrating treatment with parent vessel occlusion (arrow heads).