Progressive Neuroscience

A publication for physicians produced by the Institute for Neurosciences at Winthrop-University Hospital

• The Secret Life of Bleach in the Brain
• The Importance of Electromyography in Diagnosing CIDP
• Motion-Sparing Laminoplasty for Cervical Spondylotic Myelopathy
To Our Colleagues:

The high-quality treatment patients require, and the standard of excellence their physicians demand, are fundamental to the vigorous culture of the Institute for Neurosciences at Winthrop-University Hospital.

Harnessing the power of multidisciplinary collaboration, our world-class subspecialists provide dynamic, leading-edge care and are involved in robust bench research, as well as significant clinical trials. Almost daily, we translate notable new insights into advanced clinical management of the complex, life-altering diseases that affect the central nervous system.

Once again, Progressive Neuroscience features our neurologists’ and neurosurgeons’ best practices. This issue focuses on:

- Enlightening basic research into the causes of neurodegenerative diseases
- The important role of electromyography in diagnosing chronic inflammatory demyelinating polineuropathy
- The use of advanced brain imaging to widen the window of opportunity for stroke victims
- The challenges faced by pediatric neurosurgery patients when they become adults
- Surgical decompression and fusion for spinal cord compression due to malignancy
- A review of cervical laminoplasty for cervical spondylotic myelopathy

Our charge is to continue to obtain excellent outcomes for your patients. And the strong, collaborative relations we have established with our referring physicians are critical to achieving this goal.

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Research conducted in several laboratories at Winthrop-University Hospital is aimed at discovering some of the causes of Alzheimer’s disease (AD), Parkinson’s disease (PD) and amyotrophic lateral sclerosis (ALS).

Despite the attention given to the role of genetic mutations in the etiology of these diseases, alterations in the genetic code account for only a small number of cases of these conditions. This suggests that environmental or lifestyle factors may play a significant role in degenerative diseases of the central nervous system.

The idea that environmental factors may cause or contribute to the development of neurological diseases stems from the observation that farm workers in the 20th Century had a much greater risk for PD than the general population. Subsequent studies linked the use of agricultural chemicals to the incidence of PD in this population. Some of these substances are now being used to induce PD in experimental animals to further our understanding of this disorder. These poisons, however, are not likely to account for PD in the general population, which as a rule does not farm.

Brain-Generated Poisons

One intriguing possibility for the origins of the poisons that cause Parkinson’s disease is the brain itself, which produces a variety of toxicants, including reactive oxygen species. These species arise from normal metabolism and are usually kept in check by various antioxidants and enzymatic processes. This system of checks and balances, however, is disrupted in the diseased brain, leading to an excess of reactive oxygen species. While an excess of molecules such as superoxide, peroxide and the hydroxyl radical can account for the death of neurons, this mechanism does not explain the death of the specific groups of neurons characteristic of diseases such as PD or AD.
In the neuroscience laboratory at Winthrop, we hypothesized that the excess of reactive oxygen species reacts with neurotransmitters to generate toxins that target the cells, which take up these neurotransmitters. Since PD is caused by the loss of neurons that contain dopamine in the substantia nigra, we hypothesized that in PD, reactive oxygen species react with dopamine to produce toxins, which are taken up by dopaminergic neurons.

**Bleach in the Brain**

One of the most interesting reactive oxygen species formed in the brains of PD and AD patients is hypochlorous acid or bleach. We discovered that the reaction of bleach and dopamine produces products — collectively termed chlorodopamine — which kill dopamine-containing nerves and induce the symptoms of PD in experimental animals.

Chlorodopamine also forms a dark pigment. The substantia nigra contains neuromelanin, a dark pigment responsible for iron binding. PD results in the deposition of free iron in the substantia nigra. The most likely source of this iron is damaged or tainted neuromelanin.

We have been exploring the possibility that pigment, generated from the reaction of bleach and dopamine, accumulates in the brain and supplants neuromelanin in the substantia nigra.

The bleach-derived pigment does not bind iron. Thus, the replacement of neuromelanin by this pigment would lead to the deposition of iron. This metal promotes the generation of reactive oxygen species, which plays a crucial role in the development of PD.

These studies indicate that chlorodopamine may contribute directly to PD by killing the dopamine-containing neurons in the substantia nigra, and indirectly by causing iron to deposit in this region of the brain.

**Aggregates in the Brain**

Pigments form in the brain by a process of aggregation. This process begins with the polymerization of molecules into globular structures that aggregate. For example, neuromelanin begins as dopamine, which polymerizes in spherical bodies that aggregate. This sequence of events is poorly understood, and we have developed a new technique for studying the aggregation process.

Aggregation is also a characteristic of AD and ALS. Amyloid protein aggregates in AD as does superoxide dismutase in ALS. Our current investigations are aimed at studying the aggregation of superoxide dismutase. This protein is aggregated in both the inherited and sporadic forms of ALS. Our studies are aimed at investigating whether the disease-causing mutations alter the rate at which aggregation proceeds. If this is indeed the case, our observations may explain how mutations in superoxide dismutase cause ALS.

The notion that the brain generates poison is an emerging idea developed by a select group of research scientists, including those in Winthrop’s neuroscience laboratories. These studies could have a significant impact on our understanding of the major neurodegenerative diseases.

For more information, call the Institute for Neurosciences at 1-866-NEURO-RX or visit www.winthrop.org.

**REFERENCES**

Chronic inflammatory demyelinating polyneuropathy (CIDP) is a rare, acquired immune-mediated inflammatory disorder of the peripheral nervous system, which is too often under-recognized and under-treated. Nevertheless, this crippling disease can be managed medically, and it is important to diagnose and treat it early to prevent axonal loss and improve function. If left untreated, patients with CIDP often develop significant disabilities.

**Diagnosis & Treatment**

According to widely used criteria developed by the Inflammatory Neuropathy Cause and Treatment (INCAT) group, only clinical presentation/findings and electromyography are required for a definitive diagnosis; testing of cerebrospinal fluid and obtaining a nerve-biopsy specimen are not mandatory.

Nerve-conduction studies reveal the cardinal features of demyelination, including partial motor-nerve conduction block, reduced motor-nerve conduction velocity, prolonged distal motor latencies and prolonged F-wave latencies. Additionally, the dispersion of compound muscle action potential has been identified as a highly sensitive diagnostic criterion.

Effective treatment of CIDP includes corticosteroids, plasmapheresis and intravenous immunoglobulin (IVlg). While chemotherapeutic and immunosuppressive drugs have been used as secondary agents, reliable data on their efficacy from randomized, controlled trials are not available. Physical therapy may improve function and mobility.

As with other demyelinating conditions, each case of CIDP is different. Some patients experience one episode and spontaneous recovery, others recover partially with relapsing episodes, and others can be chronic progressive. The long-term prognosis
appears to vary according to the time at which therapy is initiated and the degree of associated axonal loss.

Case Report

A 20-year-old woman had a history of left Bell’s palsy and left-hand weakness. The symptoms resolved within six months, followed a year later by a three-month period of left-hand numbness, which also resolved. At those times, MRI imaging and electromyography were normal.

Five years following her last episode, she presented with left-hand weakness, which had developed over three months. In addition, she had had left foot numbness for three weeks, as well as numbness in the right pinky, right lower lip and chin for four days.

A general physical examination revealed no abnormalities. A neurological examination showed decreased pin prick and light touch on the right lower chin and marked weakness of the left hand, particularly finger extensors and flexors. She had mild weakness of left wrist flexors and extensors, as well as decreased pin prick and light touch in the left hand. Deep tendon reflexes were reduced in both upper extremities and in the bilateral knee jerks. Ankle jerks were absent, plantar reflexes were flexor, and coordination and gait were normal.

Suspecting multiple sclerosis (MS) and/or peripheral neuropathy, her physician ordered a full-panel blood test, including CBC, comprehensive metabolic panel, thyroid function, ESR, rheumatoid factor, antinuclear antibody, antineutrophil antibody, serum protein electrophoresis and Lyme titer. Results were normal.

A brain MRI showed non-specific signal abnormalities in the white matter of the occipital horns and right atrium. MRI of the whole spine (Fig 1) showed foci of abnormal signals in the spinal cord at C2, C5-6, T1-2, T3-4 and T10-11 on T2 weighted sequences. Enhancement involved the C5-6 and T3-4 lesions, as well as portions of cauda equina. Elevated cerebrospinal fluid protein was found, but cell count, glucose, oligoclonal band and culture were normal.

When laboratory findings and imaging studies did not confirm the existence of MS or any other specific disease, an extensive electromyographic assessment was conducted. Bilateral median, ulnar, radial, peroneal and tibial motor conduction, as well as median, ulnar, radial, superficial peroneal and sural sensory studies revealed several abnormalities, including absent left ulnar sensory response and abnormalities in multiple motor nerves, which consisted of conduction block, conduction slowing and waveform dispersion of motor responses (Fig 2).

The study of the left radial nerve to extensor indicis proprius showed low amplitude of compound response with a conduction block amounting to 89% in the forearm. The left median motor response to abductor pollicis brevis muscle showed a nerve conduction block and abnormal temporal dispersion after median nerve stimulation at the elbow. Left ulnar motor response to abductor digiti minimi showed reduced motor amplitude with conduction block (47%) after ulnar nerve conduction below the ulnar groove, as compared with the amplitude after stimulation at the wrist. Similar findings — but to a lesser extent — were seen in the right median and right ulnar nerves. F waves and H reflexes were absent, with the exception of normal right tibial F waves. These findings, together with the patient’s clinical presentation, characterized CIDP. CNS lesions are seen in 5% of CIDP patients. This patient had multiple spinal cord lesions.

Once the diagnosis was confirmed, intravenous immune globulin therapy (IVIg) was initiated, and the patient responded quickly. After five days, her motor and sensory function improved significantly. She was able to hold objects and extend her fingers. Three months later she returned to work.

Conclusion

CIDP improves with treatment. If the disease is diagnosed and treated early, axonal loss can be curtailed, and many patients can enjoy significant improvement in quality of life.

Therefore, suspicion of CIDP based on clinical presentation should prompt the performance of extensive nerve conduction studies, which often reveal characteristic findings of the disease to support an accurate, timely diagnosis.

For more information call the Institute for Neurosciences at 1-866-NEURO-RX or visit www.winthrop.org.

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Every four minutes, someone dies as a result of a cerebrovascular accident (CVA) — the nation’s third leading cause of death and the leading cause of long-term disability.1,2

Ischemic stroke, defined as the sudden reduction of blood flow to an area of the brain, results in a corresponding loss of neurologic function. Cerebral ischemia is characterized by decreased perfusion pressure, which prolongs the mean transit time (MTT) of brain tissue blood flow. The process of autoregulation induces capillary dilation in an attempt to maintain a constant cerebral blood flow (CBF). As a result, cerebral blood volume (CBV) in the penumbral tissue — the still-viable tissue contiguous to the damaged area — is maintained with vasodilation. However, in the infarct core, autoregulation mechanisms are overwhelmed and lost, and CBV is diminished.

The goal of thrombolytic therapy for ischemic stroke is to restore normal cerebral blood flow to the tissue at risk. Despite great efforts in stroke research — and significant improvements in care within the last two decades — the available therapies remain intravenous recombinant tissue plasminogen activator (t-PA) — the only FDA-approved medication for the treatment of acute stroke — and endovascular techniques, such as advanced clot retrieval systems.

Narrow Window of Opportunity

To prevent devastating consequences and permanent brain damage, acute ischemic stroke interventions must occur as soon as possible after onset of symptoms. Administered within 3-4.5 hours, t-PA can recanalize an occluded vessel, improving neurological outcome. However, the narrow window of opportunity and the potential for hemorrhagic complications limit effective use to an average of 4%-5% of stroke patients.3,4

It has been hypothesized that IV t-PA and other reperfusion therapies, such as intra-arterial t-PA or the use of clot retrieval systems, such as MERCI® and Penumbra®, can be administered safely beyond the tight time frame to patients with sufficient salvageable tissue. In such instances, treatment is not directed to the already irreversibly damaged core, but toward tissue in the penumbra with the potential for recovery.
According to the literature, penumbra is present in about 80% of stroke patients who arrive early to the emergency room/hospital; up to 44% may retain the salvageable tissue even after 18 hours. However, if perfusion is not restored, these “capricious zones” of potentially salvageable tissue can eventually disappear. The rate of penumbral disappearance depends on many factors, including the degree of CBF reduction, severity of obstruction, presence of residual/collateral flow, duration of ischemia, admission glucose, temperature, systolic blood pressure and hematocrit.6

**Identifying Salvageable Tissue**

While we can postulate that patients who present with potentially salvageable tissue can still benefit from t-PA after the 3-4.5 hour limit, how do we quickly and reliably identify these fortunate individuals? Answer: With rapid assessment through the use of advanced neuroimaging modalities, such as those used by the brain-imaging specialists in Winthrop-University Hospital’s Institute for Neurosciences:

- **Non-Contrast CT (NCT)** provides structural information as well as the ability to exclude intracerebral hemorrhage. However, it cannot reliably differentiate between irreversibly damaged brain tissue and penumbral tissue.
- **CT Angiography (CTA)** can identify a large vessel occlusion or stenosis in the neck or in the circle of Willis. It is an especially valuable modality used as soon as a patient with a suspected stroke presents to the emergency room.
- **CT Perfusion (CT-P)**, which allows for absolute perfusion calculation, as well as perfusion monitoring over time, assesses the delivery of blood to the brain parenchyma and can provide information about the amount of brain tissue already infarcted (infarct core), compared with the total area of decreased perfusion. A significant mismatch between infarct core and penumbra may imply the presence of salvageable brain tissue, and reperfusion therapy can be considered.
- **MR Perfusion (MR-P)**, another widely used technique for stroke imaging, offers semi-quantitative comparison of both brain hemispheres. Typical multimodal stroke MRI protocol consists of DWI/ADC, FLAIR, Gradient Echo and MRA sequences. MRA provides information on vessel patency, while Gradient Echo provides detailed information about intracerebral hemorrhages — even visualizing microbleeds not seen on NCT.
- **Positron Emission Tomography (PET)**, which facilitates imaging and quantification of ischemic stroke pathophysiology, is widely viewed as perhaps the most definitive modality in evaluating the penumbral. PET produces quantitative measurements of CBF, cerebral metabolism of oxygen (CMRO2) and cerebral oxygen extraction fraction (OEF), and early research has indicated that an increased OEF is a signature of ischemic penumbra.6
- **Single-Photon Emission Tomography (SPECT)** can also be used to identify penumbra. With the injection of the tracer, Technetium-99, 2-D or 3-D brain images can be reconstructed to reveal areas of reduced perfusion. It is believed that the tracer signals can indicate ischemic penumbra within the first 3-6 hours of symptom onset. It also can demonstrate collateral flow and phenomena such as diaschisis, which is reduced flow at a location remote to the stroke.6

**Conclusion**

Until recently, effective treatment of stroke was seriously limited by time. Now — on a case by case basis — advanced neuroimaging technology and techniques, can potentially expand the treatment time window and help us make more informed decisions based upon brain physiology, not just the clock. This information helps us individualize stroke therapy for improved patient outcomes.

**REFERENCES**

Commentary: As Adults, Pediatric Neurosurgery Patients Require a New Cadre of Specialists

By John A. Grant, MD
Pediatric Neurosurgeon
Winthrop-University Hospital

A 65-year-old woman — active and still employed — presented with headache, dizziness and sleepiness following multiple unsuccessful attempts to repair a ventriculo-peritoneal shunt she had had for many years. Diagnosed with hydrocephalus at age nine, she had surgery to implant a shunt, which diverted the flow of cerebrospinal fluid (CSF) from her brain into her abdominal cavity, where it was absorbed.

While she had responded well as a child, over the intervening 50+ years, she had had her shunt replaced several times. (Shunts are not perfect devices, with complications including mechanical failure, infection, obstructions and the need to lengthen or replace the catheter as the patient matures.) At this point, in her sixth decade, this woman had spent almost a year growing increasingly incapacitated because of her shunt problems.

Finally, since her disorder had developed during childhood, she decided to consult a pediatric neurosurgeon, who diagnosed her current condition as aqueductal stenosis — one of the most common causes of hydrocephalus. An advanced procedure — endoscopic third ventriculostomy (ETV) — was performed. Obviating the need for a shunt, this approach involves introducing a neuroendoscope into the brain to view the ventricular surface and enable the creation of a tiny hole in the floor of the third ventricle that allows the CSF to bypass the obstruction and flow toward a site of reabsorption around the surface of the brain.

Following the ETV, the patient’s symptoms resolved, and she was able to return to work and normal activity within three weeks.

This case illustrates a growing concern and increasing public health problem in the US. With our rapidly improving technology, techniques and knowledge, we understand that many complicated conditions of the brain and spinal cord — once considered “pediatric” in nature — can, in fact, continue as difficult struggles, challenging patients throughout life.

We understand that many complicated conditions of the brain and spinal cord — once considered “pediatric” in nature — can, in fact, continue as difficult struggles, challenging patients throughout life.

The wide range of pediatric neurosurgical disorders includes brain tumors, spasticity, movement disorders, intractable epilepsy, hydrocephalus, craniosynostosis, head injuries and spina bifida, as well as other cranial malformations and spinal deformities.

Children with such conditions, who had successful neurosurgery, are graduating from pediatric care, and it is becoming increasingly difficult for them to see practitioners with the required training and expertise, as few adult neurosurgeons are comfortable treating these young people.

Why?

The wide range of pathophysiological mechanisms, clinical characteristics and management approaches to the disease processes experienced by children with congenital nervous system anomalies are substantially different from those faced by adults. What’s more, the conditions are further complicated by the challenges of growth and development, and as these patients mature, their problems become more complex.

While many pediatric neurosurgery patients can now have normal or near-normal life spans thanks to medical advances — such as the development of effective shunting in the treatment of hydrocephalus — rarely can their conditions be corrected with a single surgical procedure. That being the case, pediatric neurosurgeons face the often-daunting and complex dilemma of how to provide the best care for their patients as they transition from the pediatric to the adult population.

For example, children treated for conditions such as spina bifida (myelomeningocele) and hydrocephalus do not “outgrow” their difficulties. They continue to require specialty care for their complex problems as they mature. While most receive intense, coordinated care as youngsters, they are unlikely to find the same type of comprehensive care as adults.

Consequently, many adults, who once had successful pediatric neurosurgery either for congenital or acquired problems, find themselves coping with chronic ailments that are either sequelae of their initial conditions or delayed complications stemming from earlier treatments.

These adult patients are finding it difficult to access neurosurgical specialists with the experience to bridge the pediatric and adult environments and facilitate the transition of their complex care. At Winthrop-University Hospital’s Institute for Neurosciences, we are prepared and skilled in helping such individuals transition into adult care. However, nationwide, the development of a robust, well-trained cadre of neurosurgical specialists — who are equally comfortable working with children as well as adults — is imperative.

For more information call the Institute for Neurosciences at 1-866NEURO-RX or visit www.winthrop.org.

REFERENCES

Cervical spondylotic myelopathy (CSM) is a progressive degenerative condition that leads to gradual loss of nerve function and is the most common cause of non-traumatic, spastic paraparesis and quadriplegia.

Considered a significant source of spinal cord dysfunction in the over-55 population, CSM currently affects an estimated 50% of men and 33% of women by age 60,\(^2\) and with the expanding elderly population, that figure is expected to increase.

Resulting from compression of the cervical spinal cord, CSM involves a range of degenerative changes that occur over time. Multiple static and dynamic mechanical factors play a significant role in the pathophysiology, including degeneration of the cervical disc and subsequent collapse of discal space, osteophyte formation, spinal cord ischemia and trauma. Although rare, ossification of the posterior longitudinal ligaments can also lead to severe myelopathy.

### Signs & Symptoms

“CSM signs and symptoms develop slowly and can vary greatly among patients. The condition is characterized by long periods of disability with intermittent episodes of neurologic decline,” said neurosurgeon Sachin Shah, MD, who specializes in spine surgery and has written a peer-reviewed article about CSM.\(^3\)

Presenting clinical features depend on the duration of the condition since onset and the severity of spinal cord or nerve root compression. Spastic gait and leg stiffness are the most common — and often earliest — signs, with upper extremity numbness and hand weakness, generally occurring as the condition progresses. Other common symptoms include neck pain, headache, and referred pain in the shoulder, arm, forearm, and hand — typically unilateral, but sometimes bilateral. Hyperreflexia, clonus, Babinski and bowel and bladder dysfunction may also occur.

### Diagnosis

“Such debilitating and persistent symptoms can seriously diminish quality of life — particularly among the elderly,” explained Dr. Shah. “But pinpointing a CSM diagnosis can be challenging, since several neurologic conditions, including amyotrophic lateral sclerosis, multiple sclerosis, primary spinal cord tumors, syringomyelia, and extramedullary conditions, can present similarly.”

To diagnose CSM, the patient’s clinical history and neurological findings must be correlated with imaging studies. While basic radiographs can demonstrate osteophyte formation, MRI and CT are the diagnostic procedures of choice — essential to quantifying the degree of stenosis in the spinal cord, evaluating the ligaments and intervertebral discs and ruling out differential diagnoses.

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Sachin Shah, MD
Neurosurgeon
Winthrop-University Hospital

### Treatment

Approximately 40% of CSM patients will deteriorate if left untreated.\(^4\) “Traditionally, CSM has been treated conservatively with traction and immobilization. However, there’s increasing evidence that early surgical intervention improves long-term functional recovery,” Dr. Shah said.

There are several surgical approaches to CSM. Choosing the most appropriate one depends on the patient’s clinical condition and radiological findings. Regardless of the selected technique, the goal is to decompress the spinal cord and provide room for recovery, thus halting symptom progression.

While the optimal surgical technique for CSM remains controversial, the longstanding technique for decompression caused by CSM has been laminectomy with fusion. However, laminoplasty, with preservation of the posterior elements and without fusion, is becoming more widespread. “The management of multilevel cervical stenosis has undergone an evolution over the past century. Early treatments with multilevel laminectomies resulted in initial neurological improvements but long-term results in some cases were disappointing.”\(^5\)

Today, cervical laminoplasty employs the “open door” technique for multilevel decompression without removing the laminae. The complex, motion-sparing procedure involves making an incision along one side of the laminae and creating a hinge at the junction of the laminae with the lateral mass. This elevates the spinal canal, widens the diameter, provides more space for the spinal cord and relieves the pressure. A spacer is usually inserted to hold the spinal canal open.

Patient selection and careful attention to contraindications are critical to success. Factors that must be considered in selecting appropriate patients include the presence of multilevel stenosis and MRI findings consistent with myelopathy. Contraindications include kyphotic cervical pathology, previous posterior cervical surgery, ossification of the ligamentum flavum and epidural fibrosis.\(^6\)

With carefully selected patients, open-door laminoplasty is safe and effective and ideal for multilevel stenosis. It can be performed quickly and with minimum blood loss, thus minimizing risks.\(^7\)

“CSM can lead to significant clinical morbidity, especially among elderly, debilitated patients,” said Dr. Shah. “It is a challenging condition to diagnose and treat, but as we gain greater understanding of the processes, we’re beginning to see that timely diagnosis and laminoplasty can have a significant impact on functional recovery.”

For more information call the Institute for Neurosciences at 1-866NEURO-RX or visit www.winthrop.org.

### REFERENCES

Case Report: Patient with Malignant Spinal Cord Compression Resumes Normal Activities after Surgery

By Vladimir Dadashev, MD
Neurosurgeon
Winthrop-University Hospital

A 64-year-old male, had a history of thoracotomy for lung cancer in 2000, followed by chemotherapy. When the treatment was completed, a full-body CT indicated he was disease free. Throughout the therapy, he continued to work at his job.

A week prior to being seen at Winthrop-University Hospital’s Emergency Department, he began to experience back pain, issues with his gait and urinary problems, which prompted a visit to his urologist, who considered his medical history, and sent him directly to the ER. There, a thorough examination revealed that his upper extremities were moving well, with normal reflexes, but movement in his lower extremities was limited and extremely hyperreflexic and hyparspastic.

Since his lower extremities and bladder function were affected, spinal cord compression below the cervical spine was suspected. Emergency CT and MRI exams confirmed that he had two sites of likely metastasis to his spine — at T4 and L1.

At T4, a lesion had completely replaced the vertebral body and extended into the spinal canal, causing critical circumferential compression of the spinal cord. Additionally, the tumor had replaced normal bone, destabilizing the spine. At L1, while there was no compression of the spinal cord, a mass had replaced a significant portion of the vertebral body, causing overall spinal instability.

Given his medical history, presenting symptoms and the results of the imaging exams, this patient was immediately hospitalized and prepared for emergent surgical decompression of the spinal cord.

Serious Complication

Nearly 13,000 cancer patients in the US develop metastatic spinal cord compression (MSCC) each year; lung cancer metastasis accounts for 15%-20% of this serious complication,1 with MSCC patients often experiencing incapacitating pain, paraparesis or paralysis and incontinence. To prevent irreversible neurological damage, they require timely diagnosis and prompt intervention.

With the use of today’s advanced MRI technology, diagnosis of MSCC can be rapid and precise. Moreover, during the last decade, advancements in open and minimally invasive surgical techniques have substantially enhanced the management of MSCC. After considering general health, ability to tolerate surgery and goals of therapy, surgery is recommended for patients with progressive neurological deficits, vertebral column instability, radioresistant tumors and intractable pain.2

In the groundbreaking study3 that changed our understanding and treatment paradigms of MSCC, patients were randomized to radiation alone vs. surgical decompression with stabilization — when necessary — followed by radiation. After an early analysis, the trial was halted because results favored the surgical group significantly.

More surgery patients were able to maintain their ability to walk (42/50, 84%), compared to those in the radiotherapy group (29/51, 57%). Also, patients who had surgery retained the ability to walk for a much longer period of time (122 vs.13 days). Of the 32 patients who could not walk prior to treatment, 10/16 (62%) in the surgery group resumed walking, compared to 3/16 (19%) who received radiation alone. Additionally, the secondary endpoints were met using the ASIA and Frankel neurological assessment scores (Fig 1).

At Winthrop-University Hospital, the patient first underwent T4 transpedicular decompression surgery with stabilization, utilizing titanium screws and rods. This approach is preferred when the malignancy involves the dorsal aspect of the vertebral body, especially when the disease extends into the pedicle and compresses the cord. He did well postoperatively, and a week later, minimally invasive stabilization was performed for the L1 lesion. This technique employs percutaneous placement of screws and rods through half-inch incisions.

Following the surgeries, the patient had radiation to his thoracic and lumbar spine, followed by another course of chemotherapy.

MSCC is an alarming, highly serious complication of cancer, which should be prevented as much as possible by early detection and speedy intervention. While there is no cure, in select patients, we can reduce the physical and emotional devastation of paraplegia and enable patients to retain their independence and improve the quality of their lives.

When last seen, the patient was continent, walking without assistance and reporting he had recently played golf.

For more information call the Institute for Neurosciences at 1-866-NEURO-RX or visit www.winthrop.org.

REFERENCES
Stroke Team Achieves “Gold Level” in Get with the Guidelines® Program

Members of Winthrop’s Stroke Team and Administration celebrate the Hospital’s Get with the Guidelines® Program Gold Level quality achievement award for stroke patient care.

Comprehensive Epilepsy Center Attains Level 4 Designation

Members of Winthrop’s Comprehensive Epilepsy Center Team and Administration celebrate designation as a Level 4 Epilepsy Center.
Contributing Surgeons & Physicians

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Dr. Mark Stecker is Board Certified by the American Board of Psychiatry and Neurology in Neurology, and by the American Board of Clinical Neurophysiology, in EEG/epilepsy and intra-operative neurophysiologic monitoring. His special clinical interests are EEG/epilepsy and intra-operative neurophysiologic monitoring. His research interests center on the response of peripheral nerve to ischemia, the properties of electrodes and information theory. Prior to his appointment as Chairman of Neuroscience at Winthrop, he was Associate Chair for Neurology in the Department of Neuroscience at Marshall University in Huntington, West Virginia, where he was also a Professor of Neuroscience. His postgraduate training includes a residency at The Mount Sinai Medical Center in New York, where he was Chief Resident. He received his medical degree from Columbia University’s College of Physicians and Surgeons. Dr. Stecker has published numerous articles in professional journals. He is past President of the Nassau County Medical Society and serves on the Board of Directors of the New York State Neurosurgical Society.

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Director, MS Care Center  
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Dr. Malcolm Gottesman specializes in the treatment of multiple sclerosis (MS), and is the founder of Winthrop’s comprehensive MS Care Center. The Center conducts original clinical research and participates in state-of-the-art clinical trials. Dr. Gottesman was also instrumental in establishing the Hospital’s Stroke Program and Neuroscience Intensive Care Unit. He is Board Certified in Psychiatry and Neurology. His postgraduate training includes a residency in neurology at Long Island Jewish Medical Center, where he was Chief Resident. He also completed an internship and residency in psychiatry at Boston University Medical Center. Dr. Gottesman received his medical degree in an accelerated BS-MD program joint-ly sponsored by Rensselaer Polytechnic Institute and Albany Medical College. He has published numerous articles in professional journals and presents at national and international conferences. Dr. Gottesman received an MS Leadership award from the Long Island MS Society.

Vladimir Dadashev, MD  
Neurosurgeon  
516.255.9031

Dr. Vladimir Dadashev specializes in the treatment of degenerative diseases of the spine and brain tumors. Dr. Dadashev utilizes both minimally invasive and traditional open approaches for the treatment of spinal stenosis, sciatica, disc herniation, fractures, spinal instability and back pain. Some of these treatments include microdiscectomy, laminectomy and spinal fusion, as well as innovative techniques, such as 3-D navigation and neuroendoscopy. His postgraduate training includes a residency and internship at Emory University School of Medicine in Atlanta, where he served as Chief Resident in his final year and won “The Best Paper Presentation” on the use of novel 3-dimensional endoscopy in transsphenoidal pituitary surgery from the Georgia Neurosurgical Society. He earned his medical degree from Wayne State University School of Medicine in Detroit with High Distinction, where he was elected to Alpha Omega Alpha, the medical honor society. Dr. Dadashev has authored numerous articles and book chapters, including the most recent chapter on “Treatment of Disk and Ligamentous Diseases of the Cervical Spine” in Youmans Neurological Surgery, 6th edition. He has presented extensively at professional meetings.
**John A Grant, MD**  
Pediatric Neurosurgeon
516.255.9031

Dr. John Grant, a Board Certified neurosurgeon, specializes in pediatric neurosurgery, and has an interest in vascular neurosurgery, as well as epilepsy surgery. His postgraduate training includes a Fellowship in Pediatric Neurosurgery at Children’s Memorial Hospital in Chicago and a neurosurgery residency at the Neurological Institute of New York at Columbia University. Dr. Grant completed general surgery internships in Dublin and at Johns Hopkins Hospital in Baltimore. He earned his medical degree from the Medical School of the Royal College of Surgeons in Ireland, where he was an Arthur Jacob Scholar. Dr. Grant served as Professor and Chairman of the Department of Neurosurgery at the University of Kansas Medical Center’s School of Medicine from 2004 to 2011. He received the inaugural A. Todd Davis Outstanding Physician Award at Children’s Memorial Hospital. Dr. Grant has written on pediatric and congenital neurosurgery, as well as the history of neurosurgery and head trauma. He has been a member of the editorial board of Pediatric Neurosurgery since 2006 and has presented widely at international professional meetings. Dr. Grant visits Haiti regularly where he has a long-term commitment to caring for children with hydrocephalus and congenital malformations.

**Thomas Jeitner, PhD**  
Lead Scientist, Applied Bench Core  
516.663.2654

Dr. Thomas Jeitner is a neurochemist focusing on neurodegenerative diseases. He was recruited by Winthrop-University Hospital to initiate a research program on amyotrophic lateral sclerosis (ALS). The laboratory specializes in high performance liquid chromatography (HPLC) and protein chemistry studies. Prior to joining Winthrop, he conducted research on Parkinson’s disease, Huntington’s disease and Alzheimer’s disease at Cornell University, the Medical College of Albany and the Medical College of Wisconsin. Dr. Jeitner earned a Doctorate in Experimental Pathology from the University of Sydney in Australia.

**Feliks Koyfman, MD**  
Vascular Neurologist  
Director, Neurovascular Laboratory  
516.663.4325

Dr. Feliks Koyfman, a vascular neurologist and Director of Winthrop’s Neurovascular Laboratory has a special interest in stroke prevention and the use of imaging modalities in acute stroke treatment and workup. His postgraduate training includes Fellowships in Advanced Vascular Neurology and Imaging, as well as Vascular Neurology at Boston University Medical Center, where he also completed a residency in neurology. He earned his medical degree from the Stony Brook University School of Medicine. Dr. Koyfman has co-authored many abstracts and articles, including “Pelvic Magnetic Resonance Venography for Detection of Deep Vein Thrombosis in Young Patients with Cryptogeneric Stroke and Patent Foramen Ovale” published in Stroke and “Acute Stroke, Catheter Related Venous Thrombosis, and Paradoxical Cerebral Embolism: Report of Two Cases” in the Journal of Neuroimaging. He also presents frequently at professional meetings.

**Sachin N. Shah, MD**  
Neurosurgeon  
516.255.9031

Dr. Sachin Shah specializes in complex and revision spine surgery to treat a wide range of conditions, including adult scoliosis, spinal deformities, spinal cord tumors and malformations. Dr. Shah utilizes minimally invasive spine procedures, such as kyphoplasty, X-STOP™ and microdiscectomy, as well as cervical laminoplasty as an alternative to spinal fusion. His postgraduate training includes a Spine Fellowship at the University Of Miami and a residency and internship in neurological surgery at Emory University Hospital in Atlanta. He has written and co-authored numerous articles and book chapters, including “Complications Associated with Lumbar Stenosis Surgery in Patients Older than 75 Years of Age” for Neurosurgery Focus and “Clinical Outcomes Following Cervical Laminoplasty for 204 Patients with Cervical Spondylotic Myelopathy” for Surgical Neurology. Dr. Shah presents extensively at professional meetings.

**Huiying Yu, MD**  
Director, Electrodiagnostic Laboratory  
516.663.4525

Dr. Huiying Yu, Board Certified in Neurology and Electrodiagnostic Medicine, has a special interest in neuromuscular diseases. Dr. Yu’s postgraduate training includes an Electro-myo-gra-phy Fellowship at Harvard University Medical College/ Massachusetts General Hospital, a Fellowship in Physiology at the University of Virginia Medical Center and four years as a postdoctoral researcher in neurobiotechnology at Ohio State University. She has spent time as a research scientist in the Department of Psychiatry at New York University Medical Center, where she also completed a residency in neurology and served as a medical intern. Additionally, Dr. Yu was a neurology resident at the Peking Union Medical College Hospital in Beijing. She earned her medical degree from the Norman Bethune University of Medical Sciences School of Medicine in the People’s Republic of China. Dr. Yu has presented at professional meetings and published numerous articles in peer-reviewed journals, including Clinical Imaging and the Beijing Medical Journal.
Winthrop-University Hospital’s Institute for Neurosciences

Winthrop-University Hospital is a 591-bed teaching hospital located on Long Island in Mineola, NY. A major regional healthcare resource, the Hospital has been a leading healthcare provider for more than a century, dedicated to the integrity, dignity and well-being of every individual. Winthrop offers a full complement of advanced inpatient and outpatient services with a deep commitment to medical education and research.

Physicians and surgeons in Winthrop’s Institute for Neurosciences are pioneering the use of technologically advanced approaches for the diagnosis and treatment of diseases of the brain and spine, including computerized imaging systems, state-of-the-art surgical interventions and the latest generation of medication therapies.

The Institute’s interdisciplinary team includes neurologists; neurosurgeons; neurointensivists; pediatric neurologists and neurosurgeons; neuroradiologists; vascular surgeons; orthopaedic spine surgeons; neuro-oncologists; neuropathologists; neurophysiologists; and specially trained nurse practitioners, physician assistants and nurses. Specialized physical and occupational therapy, social work and other supportive services are also key components of the Institute. The Institute’s experts are up to date on the latest developments in neuroscience and help pave the way for new discoveries through participation in clinical research trials, which enable them to provide patients with access to tomorrow’s most promising therapies.

Programs & Services Offered by the Institute for Neurosciences

Neuroscience Intensive Care Unit
The 14-bed acute care NeuroICU is reserved for patients with serious, complex neurological issues. The focus is on providing continuous monitoring and instantaneous results of critical values, allowing the expert staff, experienced in using advanced technology and providing neurocritical care, to employ aggressive interventions that treat neurological deterioration.

Neurology
- Comprehensive Level 4 Epilepsy Center
- Movement Disorders Program
- Multiple Sclerosis Care Center
- Neurodiagnostic Laboratory

Neurosurgery
- Ankylosing Spondylitis & Spinal Fusion
- Disc Replacement
- Brain Aneurysm Program
- Brain Tumor Program
- Brain & Skull Base Surgery
- Carotid Stenting & Endarterectomy
- Cerebrovascular & Endovascular Surgery
- Chiari Decompression Surgery
- Complex & Minimally Invasive Spinal Surgeries
- Complex Cranial Surgery
- Computer-Assisted Resection of Brain Tumors
- CyberKnife® Radiosurgery
- Endoscopic Pituitary Surgery
- Epilepsy Surgery Program
- Facial Pain/Trigeminal Neuralgia Program
- Image-Guided Spine Surgery
- Kyphoplasty

Neuroangiography
- Aneurysm Coiling & Clipping
- Endovascular Genomic Sequencing
- Interventional Neuroradiology
- Neuroprotection

Pediatric Neurology & Neurosurgery
- Attention Disorders & Learning Disabilities Treatment
- Craniosynostosis Surgery
- Brain Tumor Treatment
- Evaluation & Treatment of Children with Headaches
- Evaluation & Treatment of Neurological Disorders
- Myelomeningocele Surgery

For more information, call the Institute for Neurosciences at 1-866-NEURO-RX.