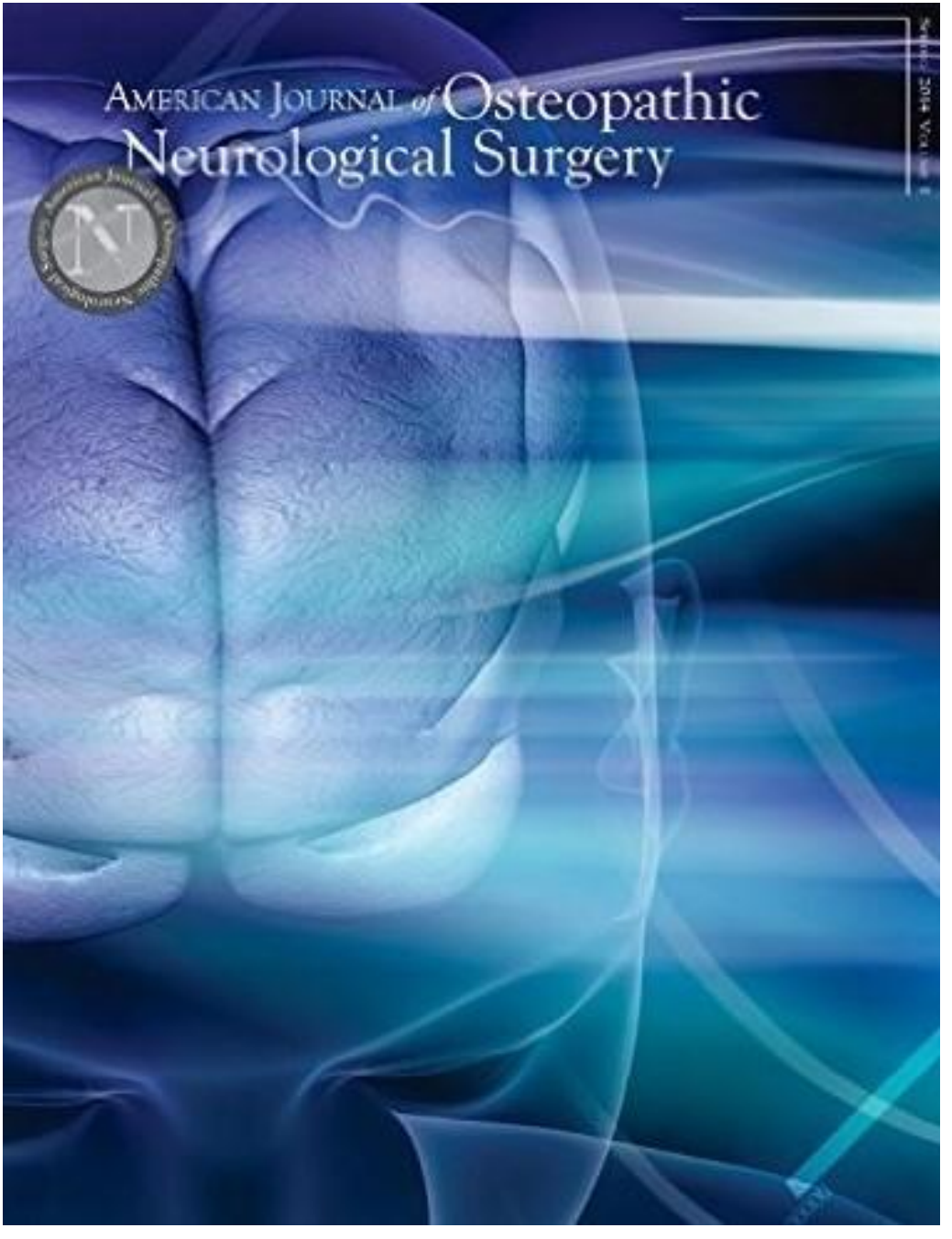
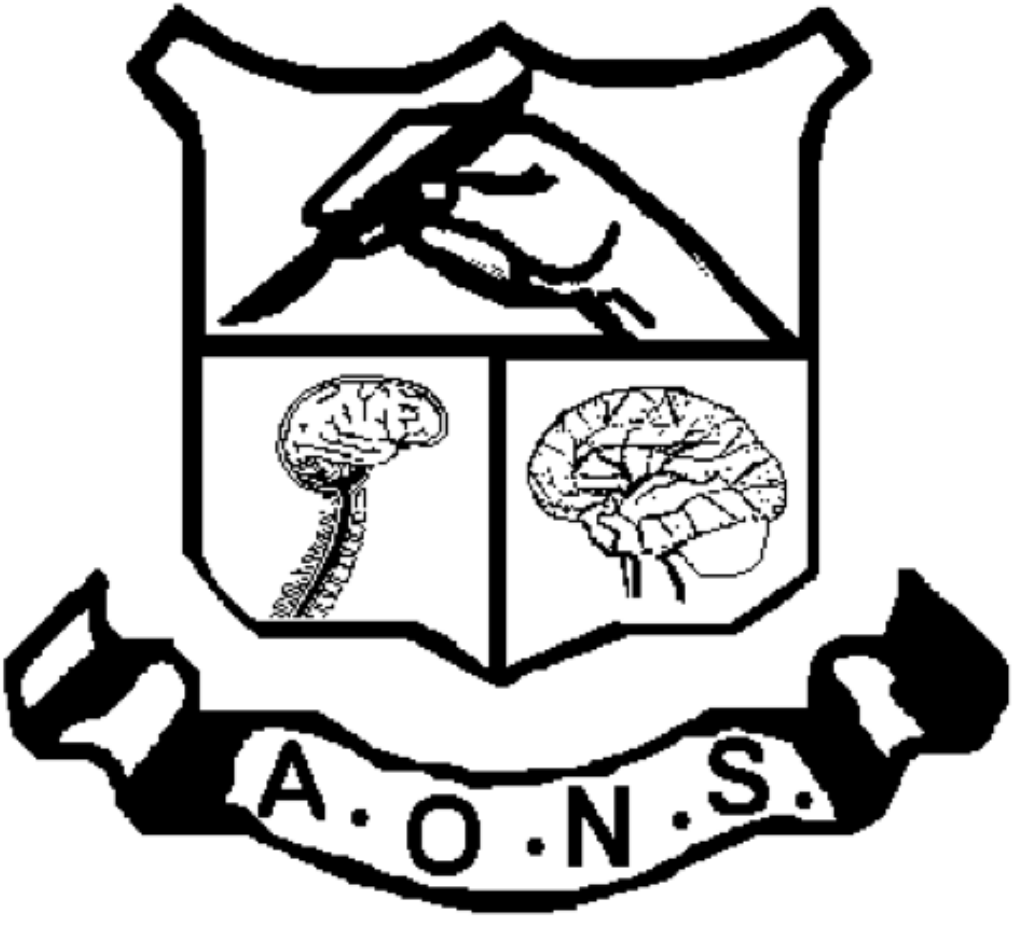

AMERICAN JOURNAL of Osteopathic
Neurological Surgery

AMERICAN JOURNAL OF OSTEOPATHIC NEUROLOGICAL SURGETY



INSTRUCTIONS FOR AUTHORS

Papers submitted should be original documentation, including photographs. The papers should be single column, double-spaced. The title should be in title case and bold, followed by Authors, degree, organization and city, state.

The papers should contain an abstract and be separated into sections with bold typing of the section title. The page set-up should be 0-6.5 inches. Paragraphs should be indented 0.5 inches. All tables should be submitted separate from the paper. If possible make the tables up to 3 inches wide so that they could fit into a column. This will allow quicker scanning and preparation.

References should be numbered, tab, name of authors. Title of paper. Journal. Year volume:pages.

All papers, articles, and correspondence should be directed to:

Dan Miulli drdmnsx@compuserve.com
Or Nicholas Qandah nqandah@yahoo.com
Or Gary Simonds grsimonds@carilionclinic.org

Letter from the Editors 4
Letters to the Editors 5
Retrospective Study of Heparin Administration for Ischemic Stroke
when there is an IV-TPA Contraindication 7
Rotatory Subluxation: Experience from The Hospital for Sick Children
Type of activity: Case Series and Literature Review 11
Incidence of Enlarging Intracranial Hemorrhage on Repeat Head CT 17
Iatrogenic Atlantoaxial Osteomyelitis Causing Paresis and Respiratory
Compromise: A Case Report and Review of Literature 21
Cervical Facet Fractures Associated with Focal Kyphotic Deformity:
Predicting the Need for Surgical Intervention 25
Primary Thoracolumbar Rudimentary Meningocele:
A Case Report and Review of the Literature 29
Brain Tumor Response to Stereotactic Radiosurgery: An Institution's
Experience in Treating Brain Tumors with CyberKnife
Robotic Radiosurgery 33
Closed Reduction of Facet Dislocation: Technical Note 39
Endovascular Management of a Spontaneous Subclavian Artery
Pseudoaneurysm Dissection: Vertebral Artery Sacrifice 43
Osteopathic Neurosurgery News 45
Surgical Nuances
Recurrent Disc Herniation Surgery The Easy Way 47
Socioeconomic Issues
Ethics in "Everyday" Neurosurgery 49

INTRODUCTORY LETTER FROM THE EDITORS

Greetings! Welcome to the all-new American Journal of Osteopathic Neurological Surgery. At a time of great proliferation of neurosurgery-related publications, one might question the wisdom of launching yet another neurosurgical journal. Indeed, the modern neurosurgeon is besieged with an over-abundance of information through so many various media. However, we have no intention of competing with, or reproducing the efforts of, other publications on the market. Instead we wish to produce a truly unique, interesting, and vibrant product that will actually be picked up and read regularly.

Furthermore we are committed to opening up a forum for osteopathic neurosurgeons (and others) to share research, concepts, concerns, viewpoints, techniques, experiences, etc. We want to create a venue for osteopathic residents to participate in, and publish, scholarly works. We want to spur osteopathic neurosurgery into an era of increased scholarly exchange and discourse.

The journal will routinely publish several original peer-reviewed research papers, including award winning resident papers national meetings. In addition to the research papers, included in each issue will be a review article on a salient neurosurgical topic. This will include a review of the current literature and any associated guidelines.

The journal will also include articles on operative nuances, neurosurgical product reviews, socioeconomic reports, textbook reviews, and a classified section featuring relevant courses, recruitments, and recruiters. There will be articles addressing the unique interests and concerns of medical students and residents. There will also be a section serially featuring one of the residency programs.

Circulation for the journal will include all practicing and training osteopathic neurosurgeons, and all osteopathic medical schools.

We hope that this enterprise excites and interests members of the osteopathic neurosurgery community and beyond. We will need your support and enthusiasm to make this work. Please contact us with suggestions, submissions, critiques, and articles.

Dan Miulli
Nick Qandah
Gary Simonds

LETTERS TO THE EDITOR

LETTER TO THE EDITORS

WHY RESEARCH IS CRITICAL IN NEUROSURGERY RESIDENT EDUCATION

Physicians in training learn and practice research "To formulate, ingrain, and measure, a method of thought, investigation, and evaluation necessary for physicians to have multi-lateral information exchange and communication with experts in areas of scientific and medical discovery, knowledge, and analysis, in order to continuously and efficiently improve human health and patient care."

Physicians are professionals, and like all learned individuals, they must continuously evaluate and correct their procedures, behaviors and methods of practice. Research understanding, a foundation of medicine, teaches physicians to communicate scientifically with most individuals, navigate through increasing amounts of data that may be clouded by the poor application of scientific methodology or analysis, choose key facts within reams of material, formulate opinions, convert those thoughts into reproducible actions, and then measure the quality of those actions. Medicine is constantly evolving, constantly developing methods of patient investigation, understanding and care. A physician can never stagnate and remain a viable practitioner. They must strive to understand their patients and the patients' medical conditions but the greatest obstruction to discovery is not ignorance- it is the illusion of knowledge.

Students, residents, and others devote hours to patient care, as well as all core competencies whether it is medical knowledge, interpersonal and communication skills, professionalism, practice-based learning and system-based practice. Each one of these requires an analytical method of inquiry, a skill that must be learned, a skill that must be developed and a skill that must be mastered. When physicians perfect research skills they perfect the ability to be the best practicing physician.

However, it is difficult to simply absorb the ability to critically use research methodology; it must be taught as part of a structured curriculum consisting of didactics, demonstration, and doing. Although, students and residents receive research orientation at the beginning of each academic year, they need mentors, and motivation. They need lists of available trials and clinical experiments that can be started or combined with basic science projects. Projects can be initiated by writing a clinical question, describing a method to answer the question, discussing the project with a mentor, re-writing the method, discussing it with a research coordinator, setting a timeline, and meeting monthly with mentors to discuss the project that was put into action.

Research will become one of the pillars of learning when institutions of higher learning assist clinical mentors by formulating a research organization with a director, a library to access knowledge databases, a research coordinator for editing, disseminating information, funding opportunities and applications, and a biostatistician for a review of methodology, analysis, and teaching.

Understanding and performing quality research provides students and residents the tools to propel quality medical care into the community and into the future.

Dan Miulli, DO, FACOS

ORIGINAL ARTICLES

Retrospective Study of Heparin administration for ischemic stroke when there is an IV-TPAcontraindication: A Safety Analysis

Douglas Stofko, DO, Erol Veznedaroglu, MD, FACS; Kenneth Liebman, MD, FAGS
Stroke and Cerebrovascular Center of New Jersey, Capital Health System, Trenton, New Jersey

Abstract
Background: Most patients who are diagnosed with an ischemic stroke arrive after the 3 hour time frame allowed for IV TPA administration. Currently, there are not any well documented and researched immediate treatment options to begin as these patients are being transferred or in a waiting phase for images or surgery. This study is looking to fill the void of administering intravenous weight based

heparin as a treatment option for patients who present within 24 hours post ischemic stroke but with a contraindication to IV TPA,
Methods: The study was designed to test whether an anticoagulation regimen of intravenous heparin, in patients with a contraindication to IV TPA, administered within 24 hours of an acute ischemic stroke could be effective and safe. A retrospective review of 257 patients, 223 which received intravenous heparin within 24 hours post ischemic stroke. Pretreatment National Institutes of Health Stroke Scale (NIHSS) and detailed medical history were analyzed. Heparin was started with no bolus and infused at a rate to maintain activated partial thromboplastin time ration 2.0 to 2.5 x control for average of 4 days. The primary endpoint was to analyze patient outcome. This will be done by analyzing length of stay, medical complications during hospital stay, location of discharge compared to pre-stroke residence and NIHSS pre and post admission. Safety end points are bleeding complications, which will be examined until patient improves or is discharged
Results: A total of 257 stroke patients were included. 223 (86.8%) received only IV heparin. Days on IV heparin ranged 1-18 days with an average of 4 days. Length of stay was 1-36 days with an average of 8 days. 52 patients received endovascular procedures (23.3%), 2 patients received subsequent endovascular or cranial procedures (0.9%), 2 patients received only cranial procedures (0.9%) and 167 patients received no surgery (74.9%).
Conclusion: As can be deduced from a survey of the literature, there is a necessity for published data regarding immediate treatments for patients presenting with an IV WA contraindication. Administration of intravenous heparin within 24 hours of an ischemic stroke seems to be safe and warrants further investigation.

Key Words: cerebral ischemia I anticoagulation 11 stroke I heparin

Introduction

While heparin is widely used for treatment of acute stroke, currently there are few clinical trials that have proven efficacy of intravenous heparin in the treatment of stroke (2, 7). Long term studies have been done to show that intravenous heparin administration within 3 hours of acute nonlacunar hemispheric cerebral infarction has shown benefit based on long term outcomes (2). However, the remaining data is sparse and tends to allow long intervals from the onset of stroke to the initiation of heparin or focus on heparin dosing following IV TPA (5, 8, 10, and 14). Nonetheless,

heparin has been shown to prevent thrombus propagation which is a potential reason for progressive stroke (1, 4). Currently, there are not any well documented and researched immediate treatment options available to patients not eligible to receive IV TPA. As can be seen from the survey of literature, there is a necessity for published data regarding immediate treatments for patients presenting with an IV TPA contraindication.

Methods

We retrospectively (January 2009 - April 2011) analyzed consecutive patients diagnosed with acute ischemic stroke who did not receive IV TPA but received intravenous heparin within 24 hours of, ischemiic stroke onset.

Out of 257 consecutive, stroke patients at our stroke center, 223 patients met the inclusion criteria. The primary, end points of the study were to analyze patient outcome and safety. Patient outcome was evaluated by analyzing length of stay, medical complications during hospital stay, location of discharge compared to pre- stroke residence and pre- and post admission NI HSS. Safety was evaluated by early bleeding complication rates.

Inclusion criteria consisted of patients 18-90 years old with the diagnosis of acute ischemic stroke, symptoms of stroke onset could be narrowed to a specific time and included patients who had a contraindication to IV TPA. Exclusion criteria were any patients who received IV TPA or diagnosed with a hemorrhagic stroke.

Patients matching inclusion criteria were administered weight based intravenous heparin, with no initial bolus, within 24 hours of stroke symptoms, Partial thromboplastin time (PTT) ratio was obtained 6 hours after infusion was started and then everyday to maintain a PTT goal of 2.0 to 2.5 times the control. Heparin infusion was immediately discontinued in case of any hemorrhage. After admission cat scan (CT) of the head, further CT's where only checked on an as needed basis when cerebral bleeding was suspected. Extracranial bleeding was recorded and intravenous heparin discontinued when clinically indicated.

Results

Of 257 consecutive acute ischemic stroke patients, from January 2009 to April 2011 at one single stroke center, 223 patients (86.8 %) met the inclusion criteria. Included were 100 women and 123 men, with a mean age of 66 years (range, 20 — 91).

A total of 257 stroke patients were identified of which 223 received only IV heparin. Days on IV heparin ranged 1-18 days with an average of 4 days. The average PTT was 73 (range, 25-91). 52 patients received endovascular procedures (23.3%), 2 patients received subsequent endovascular or cranial procedures (0.9%), 2 patients received only cranial procedures (0.9%) and 167 patients received no surgery (74.9%).

Received Endovascular &	**2**	0.90%
Received only **Cranial**	2	0.90%
Received Endovascular	52	2.ngici
Received NO Surgery	167	74.89%

Patient outcome was analyzed by evaluating length of stay, location of discharge and N1HSS pre and post ischemic stroke. Length of stay was 1-36 days (with one outlier staying 77 days) with an average of 8 days. Table 1 shows location of discharge compared to pre-stroke residence. 107 patients (48%) where discharged to rehab from home pre-stroke, 42 patients (18.8%) admitted from home were subsequently discharged to home, while another 22 patients (9.9%) were discharged to home with home care. 12 patients (5.4%) died.

Hemorrhagic complications are seen in table 2. Hemorrhagic complications of the brain were differentiated into asymptomatic or symptomatic according to the guidelines of the NINDS trial (12). Symptomatic hemorrhage was seen in 2 patients, while 8 patients had asymptomatic hemorrhage.

Extracranial hemorrhage occurred in the gastrointestinal tract (5 patients), groin site of femoral sheath (2 patients), hematuria (1 patient), rectal bleeding (1 patient), rectus sheath hematoma (1 patient) and carotid hematoma (1 patient). No deaths resulted from extracranial bleeding.

Table 1

Pre Stroke Location	Discharge Location	Total	%
Assisted	Rehab	[redacted]	0.45
Home	Home	42	18,83
Home	Home Care	22	9.87
Home	Hospice	7	3.14
Home	Hospital	6	2.69
Home	Passed	[redacted]	5.38
Home	Rehab	[redacted]	47.98
Home	Unknown	1	0.45
Homc Care	Rehab	1	0.45
Hospital	Hospital	3	i 35
Rctiab	Kebab	5	[redacted]
Rehab	Horne Care	1	0.45
Rehab	Hospice		0.90
Hospital	Rehab	1	0.45
Long term	Rehab	2	0.90
Nursing	Hospice	2	0.90
Nursing	Nursing	1	0.45
Nursing	Rehab	[redacted]	0.90
Unknown	Hospice	1	0.45
[redacted]	Rehab	4	1.79

Hemorrhagic Complications of the Brain: Table 2

	N (%)
Symptomatic	*2* (0.3%6)
Fatal	2 (C.9%)
Non Fatal	0
Asymptomati	8 (8.0%)

Discussion

Our study suggests administering intravenous weight based heparin as a treatment option for patients who present within 24 hours post ischemic stroke but with a contraindication to IV TPA is both safe and warrants further investigation. Currently, the evidence on administering intravenous heparin to patients with acute ischemic stroke is scant. Unfortunately, randomized controlled trials have not studied heparin in patients who have no other treatment options.

The utility of heparins in ischemic stroke is based on the principal that it prevents thrombus propagation. Thrombus formation, propagation or embolization causes new neurological worsening or deficits in 20 — 30% of ischemic strokes (3). Also, heparin has proven to have ant-inflammatory properties, which is known to take place after ischemic stroke (6). With the anti-inflammatory effects of heparin and prevention of thrombus propagation, the use of IV heparin would seem to be a rationale treatment for acute stroke. However, few studies have shown a net clinical benefit to intravenous heparin administration. The majority of the published data does not emphasize heparin administration within 24 hours. The International Stroke Trial (1ST), the largest heparin trial, heparin was administered subcutaneously and not intravenously, therefore PTTs were not closely monitored (10). Long term, randomized controlled trials have been done to show that intravenous heparin administration within the first 3 hours of acute non lucunar hemispheric cerebral infarction has helped patient outcomes based on the 90 day modified Rankin Scale (mRS). However, patients in this time frame would have been available for IV TPA therefore not addressing the patient population that would not be eligible for this option. In the TOAST trial, a low molecular-weight hepannoid was given by intravenous infusion within 24 hours of acute stroke symptoms for 7 days resulting in reduced recurrent strokes and very favorable outcomes were significantly higher in patients given hepranoid vs placebo (14).

Intravenous heparin as well as IV TPA administration has been associated with an increased risk of symptomatic intracranial hemorrhages. However our rate at 0.90% for symptomatic intracranial hemorrhages was significantly lower than previous reported for both intravenous heparin and IV TPA thrombolysis. Other trials of thrombolysis have reported higher intracranial hemorrhage rates but the thrombolysis was given up to 6 hours from onset of symptoms. No other information is available for intravenous heparin clinical trials (5,8).

While there have been contributions to the evidence base by studies such as 1ST, and TOAST and even fewer contributions of heparin administration within 24 hours, there is still no large published trial of monitored, unfractionated heparin administered within 24 hours in acute ischemic stroke. Our published data supports that a future randomized controlled trial would be safe and is needed to validate the use of intravenous heparin in patients with contraindication to IV TPA.

References

1. Berge E. Heparin for acute ischaemic stroke: a never-ending story? *Lancet Neurology.* 20076: 381-382.
2. tamerlingo, MD, et al. (2005). Intravenous Heparin Started Within the First 3 Hours After Onset of Symptoms as a Treatment jar Acute Nonlacunar Hemispheric Cerebral infarctions. *Stroke,* 36 (11): 2415- 2419.
3. Caplan LR_ Resolved· Heparin May Be Useful in Selected Patients With Brain Ischemia. *Stroke.* 2003; 34: 230-231.
4. Caplan LR. Treatment of 'progressive" stroke. *Stroke.* 1991; 22: 694-695,
5. terebral Embolism Study Group. Immediate anticoagulation of embolic stroke: a randomized trial *Stroke.* 1983: 14: 668-66.
6. Chamorro, A. Immediate anticoagulation in Acute Focal Brain Ischemia Revisited_ *Stroke,* 2001 ;32: 577578.
7. thamorro el al. for the RAPID Inverstigators. The RAPID anticoagulation prevents ischemic damage
8. Duke RJ, Bloch AF, Turpie ACG, Trebilcook R, sayer N. Intravenous Heparin for the Prevention of stroke prevention ci stroke progression in acute partial stable stroke: a randomized contmlled trio!. *Ann Intern Med.* 1986; 105: 825-828.
9. Gubitz G, Counsel! C, Sandercock P, Signorini D. Anticoagulants for acute ischemic stroke (Cochrane review). In: *The Cochrane Library,* issue 2, 2000_ Oxford, UK.
10. International Stroke Trial Collaborative Group. The International Stroke Trial (1ST): a randomized trial of aspirin, subcutaneous heparin, both, or neither among 19 435 patients with acute ischemic stroke, *Lancet* .1997; 349: 1569-1581.
11. Muir, Keith (2008). The PREVAIL Trial and Low-Molecular-Weight Heparin for Prevention of Venous Thromboembolism. *Stroke,* 3972174-2176.
12. National Institute of Neurological Disorders and Stroke tTPA Stroke Study Group. Tissue plasminogen activator for acute ischemic stroke. *N Eng/ J Med.* 1995; 332: 15E11-1587
13. Sobesky, Jan, et al. (2007). The Cologne Stroke Experience: Safety and Outcome in 450 Patients Treated with Intravenous Thrombolysis. *Cerebrovascular Diseases,* 24, 56-65
14. The Publication Committee for the Trial ORG 10172 in Acute Stroke Treatment (TOAST) Investigators. Low-molecular heparinoid, ORG 10172 (danaparoid), and outcome after acute ischemic stroke: a randomized controlled trial. *AMA .* 1998;'279: 1265-1272.

Rotatory Subluxation: experience from The Hospital for Sick Children Type of activity: Case series and literature review

Alexandra D. Beier, DO'. Shobhan Vachhrajani MD[2], Simon Bayerl[3], Claudia Y. Diaz Aguilar-[1], Maria Lamberti-Pasculli, RN[2], James M. Drake, MBBCh., MSc., FRCSC[2]

[1] Section of Neurosurgery, St John Providence Health System, Southfield, MI, USA
[2] Division of Neurosurgery, The Hospital for Sick Children, University of Toronto, Ontario, Canada
[3] Charite Universitatsmedizin Berlin, Germany
[4] Escuela de Medicina Ignacio A. Santos, Tecnologico de Monterrey, Campus Monterrey, Nuevo Leon, Mexico

Key Words: rotatory subluxation, rotatory fixation, atlantoaxial rotatory subluxation
Running Head: Rotatory subluxation: The Hospital for Sick Children

Abstract

Purpose: Diagnosis and management of atlantoaxial rotatory subluxation (AARS) is challenging clue to variability in clinical presentation. Although several treatment modalities have been employed, there remains no consensus on the most appropriate treatment. We explore this issue in our nine-year series of AARS.
Methods: Records of patients diagnosed radiographically and clinically with AARS between May 2001 and March 2010 were retrospectively reviewed. 0140 patients identified, 24 patients were male and were on average 8.5 years old (range 15 months to 16 years). Etiologies of AARS included trauma, congenital abnormalities, juvenile rheumatoid arthritis, post-infectious, post-surgical, and cryptogenic. Four patients had dual etiology. Symptom duration was variable: 29 patients had symptoms for less than four weeks, five patients had symptoms between four weeks and three months, and six patients had symptoms for three months or more.
Results: Treatment with a cervical collar was sufficient in 21 patients. One patient failed collar management and reduced with halter traction. Seven patients underwent initial halter traction, however four of these patients

progressed and required halo traction. Two patients were placed in a halo upon presentation due to the severity of rotatory subluxation; both required subsequent operative fusion. One patient required upfront decompression and fusion due to severe canal compromise and myelopathy. All patients requiring operative fusion presented subacutely.
Conclusion: Management of AARS is variable due to the spectrum of clinical presentation. Those presenting acutely without neurological deficit can likely be managed in a collar; those who are irreducible or present with neurological deficit may require traction and/or surgical fixation. Patients presenting subacutely may be more prone to requiring operative intervention.

Introduction

Atlantoaxial rotatory subluxation (AARS) occurs when the primary joint of rotation becomes disrupted and the atlas rotates out of synch to the axis. AARS can be seen with traumatic events, upper respiratory infections, congenital conditions associated with ligamentous laxity or vertebral anomalies, and can also occur spontaneously.[3-6,10]

Although current treatment strategies include observation, traction, halo placement and operative fusion, there is no consensus on the best management approach. Some advocate operative fusion for persistent, chronic or recurrent cases of AARS, also termed atlantoaxial rotatory fixation (AARF) by some authors.[2,1°,13] Others advocate that long-term traction can be successful for chronic or recurrent AARF. There appears to be a trend toward conservative management for acute injuries presenting in less than three weeks, and operative fusion for chronic injuries that present greater than three months after the onset of symptoms. Controversy exists, however, for those patients presenting between three and twelve weeks.[1,6,13]Minimizing the number of operative fusions would be ideal, however prolonged symptoms, the inability to achieve reduction, and recurrence of AARS have been indications for fusion, lending to an operative fusion rate of approximately 30%.[6,13]We present a nine-year series of patients diagnosed and treated for atlantoaxial rotatory subluxation at The Hospital for Sick Children, spurred by our observation that our fusion rate is much lower than commonly reported.

Methods

In compliance with the Research Ethics Board at The Hospital for Sick Children, we reviewed medical records of patients who were diagnosed with AARS and treated by the department of neurosurgery at The Hospital for Sick Children from May 2001- March 2010. Patients had to be younger than 18 years of age at presentation, have a radiographic and clinical diagnosis of AARS, and had to be managed by the neurosurgery service. Exclusion criteria included radiographic diagnosis without clinical symptomatology and sole management by other departments.

Results

A total of 40 patients, 24 of them male, met our inclusion criteria. The average age at presentation was 8.5 years (range 15 months - 16 years). Etiologies for rotatory subluxation included: trauma (n=20), congenital abnormalities (8), juvenile rheumatoid arthritis (1), post-infectious (7), post-surgical (2), and cryptogenic (6); four patients had dual etiologies. The most common presenting symptom was neck pain with limited range of motion (n=35). Other presenting symptoms included limited range of motion only (2), cranial nerve palsy (1), ataxia (1) and respiratory failure (1). Twenty-nine patients had symptoms for less than four weeks before presentation. Five patients had symptoms between four weeks and three months, and six patients had symptoms for three months or more. The average duration of symptoms prior to presentation was approximately three months, with a median of three days.

With regards to initial management, 22 patients were treated in a collar alone for a range of one day to seven weeks. Only one patient failed management after three weeks in a collar; this patient subsequently reduced with halter traction. Seven patients were initially treated with home nocturnal halter traction of 1-21 days duration. The weight used varied from 3-9lbs. Three successfully reduced and were then followed in a collar for three to eight weeks. The other four patients progressed while in halter traction (range one day to one month); halo traction was successful in achieving reduction in this subgroup. Two patients were placed in a halo upon presentation due to the severity of their rotatory subluxation. One of these patients experienced continued subluxation upon ambulation despite halo immobilization and therefore underwent operative fusion. This patient was subsequently found to have juvenile arthritis. The second patient received halo traction followed by

decompression and fusion due to marked rotatory subluxation and canal compromise. In one patient, surgical decompression and fusion was performed upon initial presentation due to severe spinal canal compromise associated with a one to two month history of motor regression and increasing ataxia. Finally, eight patients were observed without a collar or traction. The goal of treatment in all patients was improvement of pain and reduction of subluxation. This was obtained in 37 patients, whereas in three patients the rotatory subluxation appeared to be longstanding, fixed and without instability.

Discussion

The atlantoaxial (AA) joint can account for up to 60% of the neck's total rotation. This occurs due to the support of two main ligaments: the transverse ligament that prevents the forward subluxation of the atlas on the axis during head flexion and the alar ligaments that prevent excessive rotation.[2,7,11] Authors have found that at approximately 65 degrees of rotation, the spinal canal at the atlas can be reduced to approximately 7 mm in diameter, due to complete bilateral dislocation of the articular processes, causing severe cord compromise. With rotation of 45 degrees and 5 mm anterior subluxation of the atlas on the axis, the cord can be narrowed to 12 mm, also causing cord compromise.[2] Additionally, Villas et al have shown that atlantoaxial rotation as little as 36 degrees, which is probably within the normal range of motion, can be associated with excessive contact loss of the articulating facets of C1 and C2.[14]

The immature pediatric cervical spine, especially the AA region, is subjected to higher torque and sheer forces that make this region more susceptible to injury with resultant instability.[12] Several factors predispose this region to injury including underdevelopment of spinous processes, horizontally oriented facet joints, weak neck musculature, immature bone ossification, a larger head to body mass ratio, and ligamentous laxity. In addition, the fulcrum of motion in the pediatric spine is
located at the C2-3 level, much higher than in adults where it is located in the lower cervical spine.[6,12,13]

Rotatory deformities of the AA joint are typically transient and easily managed, However on occasion, they can be resistant to treatment and cause torticollis. Such persistent rotation was termed rotary fixation of the AA joint by Wortzman and Dewar in 1968.[15] The preferred term by Fielding was rotatory

fixation since the fixation of the atlas on the axis may occur with subluxation, dislocation, or when the relative positions of the atlas and axis are still within the normal range of rotation.[2] The current literature unfortunately comprises a variety of definitions and terminology for this entity ranging from persistent rotation to pathological "stickiness".[2,8] Of note, many initial definitions and classifications were based on older technology including open mouth radiographs, lateral radiographs, and cineroentgenography.[2] Pang et al simplified the definition by classifying atlantoaxial rotatory fixation (AARF) as "inclusive of all gross departures from the normal rotational relationship between the atlas and axis. Further classification of the subtypes of AARF can then be predicted on a finer differentiation of the 'degree of pathological stickiness' ".[8]

A number of published reports have evaluated treatment strategies for AARS. Fielding initially presented his series of patients from which the classification arose in 1977. He reported on 17 patients, average age of 20.6 years, who had an average delay in diagnosis of 11.6 months. Surgical arthrodesis was performed in 13/17 (76%) patients. Fielding proposed that if a patient has rotatory fixation, the stability of the atlantoaxial joint might be compromised and therefore recommended traction initially to reduce the subluxation, followed by immobilization in a Minerva jacket for three months. They found that the risk of recurrence was greater with longstanding fixation of greater than three months and this was best treated with surgical fusion.[2] This has since been echoed by several studies.[5,10,13]

In 1989, Phillips and Hensinger presented their series of 23 children with rotatory subluxation. All children were hospitalized for halter traction. Halo traction was used when halter traction was ineffective. Patients were placed in a cervical collar once reduction had been achieved. Surgical stabilization was used for persistent deformity in three children (13%). The recommended management strategies were based on symptom duration: for those with less than one week duration, a soft collar and bed rest for one week was implemented; patients were hospitalized for traction if reduction was not achieved. Patients with symptoms lasting between one week and one month were hospitalized for traction followed by post-reduction immobilization for four to six weeks. Symptom duration of greater than one month led to traction, attempted for up to three weeks, followed by arthrodesis in symptomatic patients who did not achieve successful reduction."

Subach et al in 1993 evaluated 20 children with AARS. Five patients were managed in a collar with adjunctive nonsteroidal anti-infiammatories. Halter traction was utilized in 15 patients. Nonoperative management failed in six patients (30%) requiring fusion due to recurrence or unsuccessful reduction.[13] In contrast to other reports, their study did not include congenital spine abnormalities, patients with Down syndrome or juvenile rheumatoid arthritis. Additionally, halo immobilization was not utilized.

Pang et al in 2005 presented their series of AARF. Their 35 patients were classified according to their proprietary classification system. Patients were deemed to either have AARF or be in a diagnostic grey zone (DGZ) after undergoing three full cervical computed tomography (CT) scans. All AARF patients were subject to traction regardless of duration. Halter traction was initiated for patients with symptoms less than three months, with reduction maintained in a Guilford brace. Cranial traction was used for patients with symptoms greater than three months duration or for patients who failed halter traction. Traction was not maintained beyond 2.5 weeks. These patients were immobilized in a halo vest after reduction. For patients falling into the DGZ, a soft collar, analgesics and muscle relaxants were used for two weeks. If this was unsuccessful, halter traction was initiated. Of 32 patients, six (19%) underwent surgical fusion.[9]

Our study found that the average length of symptom duration prior to presentation was approximately three months, with a median of three days. This skew suggested that this population could be pathophysiologically divided into distinct groups: an acute group (symptom duration of less than four weeks), a subacute group (symptom duration between four weeks and three months), and a chronic group (symptom duration of three months or greater). (Table 1)

Analysis of the chronic patients (N=6) revealed that three patients did not require active intervention and were therefore observed. These patients all had minimally symptomatic, non-progressing, and fused RS. Another patient in this group had a mild head tilt for three years with very mild limitation of rotation; this patient was also observed. The other two chronic patients presented after previous laryngeal infections. Both were initially treated with halter traction, followed by halo traction and vest placement, and finally a collar, which was

subsequently weaned. Of note, none of the patients in this subgroup required operative fusion.

In the subacute group (N=5), three patients underwent operative fusion. As previously mentioned, one patient presented with motor regression with accompanying severe cord compression, whereas the second was diagnosed with juvenile arthritis and had continued subluxation after halo placement. The third patient had underlying cerebral palsy and presented with pneumonia leading to respiratory failure. Investigation revealed severe cord compression. The patient was placed in halo traction followed by operative decompression and fusion. The other two patients in this group were treated with halter traction followed by a collar. One of these two patients recurred after being immobilized in the collar for one month and required halo traction for reduction. A halo vest was subsequently used for three months, followed by a collar for eight weeks.

In the acute patient group (N=29), no patient required operative fusion. Three patients were placed in halter traction. Of these three, one did not reduce, requiring
halo traction and vest placement. The other two patients managed in halter traction were successfully transitioned to a collar. Of 22 patients initially managed in a collar, only one patient progressed requiring halter traction. Four patients were observed.

In our series, all of the patients requiring operative fusion presented subacutely. Historically patients in this subgroup appear to have been most variably managed. This group likely comprises patients with progressive etiologies that, in the absence of an inciting event, do not present to medical attention until significant subluxation and neural element damage has occurred. Their underlying pathophysiology makes non-operative management unsuccessful, and their monitored progression prompts surgeons to operatively fuse in an attempt to prevent further neurological compromise. The patients in this group should be closely observed for this reason. This is in contrast to the acutely presenting patients who often incur a traumatic event and present early in the course of their disease. Likely these patients have less derangement of normal anatomical supports, and as such are more easily maintained in a cervical collar.

Interestingly, we did not observe the typical need for operative intervention in the chronic subgroup of patients. There may be several reasons for this, the most obvious of which is that only six patients presented chronically. Additionally, this group seems to represent unique pathophysiological entities, comprising such slowly developing conditions that autofusion and stabilization occurs by the time they present for evaluation. Operative fixation in these patients cannot be justified. Patients with laryngeal infections likely have a normal anatomical substrate and therefore respond to non-operative forms of management.

Finally, only three patients (7.5%) underwent operative fusion, a figure significantly less than previous studies have reported. Several reasons may be implicated. First, the diagnosis was much less delayed overall than in previous reports.[2,9,13] As mentioned above, this is due to the majority of patients presenting acutely. Additionally, two previously published series excluded patients with congenital bony abnormalities, Down syndrome and juvenile rheumatoid arthritis, all of which typically increase the rate for surgical intervention.[9'13] Of our patients that underwent fusion, one had Down syndrome, one was diagnosed with juvenile rheumatoid arthritis and one had cerebral palsy. As such, the fusion rate in our series would have been essentially zero had we excluded these patients.

We must acknowledge the limitations inherent in the retrospective nature of this study. Recommendations for management strategies are difficult to develop based on review of treatment decisions made by treating surgeons, as these are often based on specific clinical scenarios and personal preference. It was also difficult to assess exactly how rotatory subluxation was radiographically diagnosed, as there were no standardized criteria for evaluation.

Conclusion

We present our recent experience in managing AARS. We operatively fused a lower proportion of patients than previously reported, and believe that the majority of patients will reduce and can be stabilized in a non-operative manner. All three patients requiring operative fusion presented subacutely; these patients likely represent a distinct pathophysiological group in whom thorough investigation and close monitoring is required.

References

1. Burkus JK, Deponte RJ: Chronic atlantoaxial rotatory fixation correction by cervical traction, manipulation, and bracing. J Pediatr Orthop 6:631-635, 1986
2. Fielding JW, Hawkins RJ: Atlanto-axial rotatory fixation. (Fixed rotatory subluxation of the atlanto-axial joint). J Bone Joint Surg Am 59:37-44, 1977
3. Harma A, Firat Y: Grisel syndrome: nontraumatic atlantoaxial rotatory subluxation. J Craniofac Surg 19:1119-1121, 2008
4. Hicazi A, Acaroglu E, Alanay A, Yazici M, Surat A: Atlantoaxial rotatory fixation-subluxation revisited: a computed tomographic analysis of acute torticollis in pediatric patients. Spine (Phila Pa 1976) 27:2771-2775, 2002
5. Ishii K, Chiba K, Maruiwa H, Nakamura M, Matsumoto M, Toyama Y: Pathognomonic radiological signs for predicting prognosis in patients with chronic atlantoaxial rotatory fixation. J Neurosurg Spine 5:385-391, 2006
6. Martinez-Lage JF, Martinez Perez M, Fernandez Cornejo V, Poza M: Atlanto-axial rotatory subluxation in children: early management. Acta Neurochir (Wien) 143:1223-1228, 2001
7. Pang D, Li V: Atlantoaxial rotatory fixation: Part 1--Biomechanics of normal rotation at the atlantoaxial joint in children. Neurosurgery 55:614-625; discussion 625-616, 2004
8. Pang D, Li V: Atlantoaxial rotatory fixation: part 2--new diagnostic paradigm and a new classification based on motion analysis using computed tomographic imaging. Neurosurgery 57:941-953; discussion 941-953, 2005
9. Pang D, Li V: Atlantoaxial rotatory fixation: part 3-a prospective study of the clinical manifestation, diagnosis, management, and outcome of children with alantoaxial rotatory fixation. Neurosurgery 57:954-972; discussion 954- 972, 2005
10. Phillips WA, Hensinger RN: The management of rotatory atlanto-axial subluxation in children. J Bone Joint Surg Am 71:664-668, 1989
11. Rinaldi I, Mullins WJ, Jr., Delaney WF, Fitzer PM, Tornberg DN: Computerized tomographic demonstration of rotational atlanto-axial fixation. Case report. J Neurosurg 50:115-119, 1979
12. Samartzis D, Shen FH, Herman J, Mardjetko SM: Atlantoaxial rotatory fixation in the setting of associated congenital malformations: a modified classification system. Spine (Phila Pa 1976) 35:E119-127, 2010

13. Subach BR, McLaughlin MR, Albright AL, Pollack IF: Current management of pediatric atlantoaxial rotatory subluxation. Spine (Phila Pa 1976) 23:2174-2179, 1998
14. Villas C, Arriagada C, Zubieta JL: Preliminary CT study of C1-C2 rotational mobility in normal subjects. Eur Spine J 8:223-228, 1999
15. Wortzman G, Dewar FP: Rotary fixation of the atlantoaxial joint: Rotational atlantoaxial subluxation. Radiolology

Incidence of Enlarging intracranial Hemorrhage on Repeat Head CT

Leslie Lyness D.O.
Philadelphia College of Osteopathic Medicine, Philadelphia, PA

Abstract

Objective: To review the literature on traumatic Intraparenchymal Hemorrhage (IPH), to evaluate the incidence of increase size of intracranial Hemorrhage (ICH) that appears on the second CT scan, and to predict the risk of an increase in the hemorrhage size by type of hemorrhage.

Background: In a retrospective chart review of 73 patients at a single Level 1 Trauma Center, the size of the ICH (subdurals, epidurals, and contusions), age, sex, and time differential between the initial CT scan and the second CT scan was evaluated. A total of 73 patient images from 2008 and 2010 were reviewed, of those, 39 meet the criteria to *be* included in the review. 3 patients were evaluated more than once due to multiple types of hemorrhages, and therefore, the total number of hemorrhages evaluated was 43. 12/43 (27.9%) bleeds were considered to be larger in size with a significant change in the size of the hemorrhage, and of these, 4/12 (33.3%) images had measurements that were larger on the 2" CT, but the radiologist read the scan as "stable". 31/43 (72.1%) images showed no significant change on the second CT, and of these, 14 (45.2%) hemorrhages were actually smaller. 19/43(44.2%) bleeds were contusions; 10/19 (52.6%) of the contusions increased in size on the second Cr. 3/43 (7.0%) bleeds were [OH; 1/3 (33.3%) increased in size. 21/43(48.8%) bleeds were SOH; 1/22 (4.5%) increased in size.

Conclusion: The incidence of increase in size of ICH is greatest for intraparenchymal contusions (52% of contusions increase in size) and least for subdural hematomas (4.5%). in a literature review of Traumatic Brain Injury and IPH, despite the incidence of increase in size of hemorrhages, if a patient has a normal neurological exam, the increase in size of the ICH, regardless of type, rarely, if ever leads to neurosurgical intervention.

Introduction

The incidence of closed head injury is approximately 200 per 100,000 which amounts to nearly half a million patients annually. Each year, approximately 400,000 pts with head injury are admitted to the hospital for evaluation[2], and the financial burden in the United States for Traumatic Brain Injury (TI31) is estimated to be 100 billion dollars annually[3]. The study of choice for evaluation of any patient with a traumatic brain injury is a CT scan of the head; this study is a relatively quick and easy way to evaluate for the presence of intracranial hemorrhage and skull fractures. There has been some debate over the last few years as to the need for repeat serial CT scans in TM. This study reviews the literature on repeat Head CT in relation to traumatic intracranial hemorrhage, and retrospectively reviews ICH at a Level 1 Trauma center in order to determine the incidence of enlarging ICH on repeat CT.

Methods

At a single Level 1 Trauma center, a retrospective review of 73 head trauma patient images from 2008 and 2010 were analyzed for type of hemorrhage, age, sex, and time differential between the initial and repeat CT scan. The intracranial hemorrhages studied included Subdural Hematomas (SDH), Epidural Hematomas (EDH), and Intraparenchymal Contusions. Subarachnoid hemorrhages and tentorial subdural hematomas were excluded from the study due to the fact that with these hemorrhages it is sometimes difficult to quantify the initial size and amount of enlargement of the hemorrhage. Of the 73 patients, 39 met the criteria to be included in the review. Exclusion criteria were subarachnoid hemorrhage, tentorial suladurals, if only one CT was performed, and if the pt subsequently went to surgery based on the initial CT scan. Three patients had more than one type of ICH (2 patients had 2 types of hemorrhages — SDH as well as a contusion, and one patient had a SDH along with both frontal and temporal contusions), therefore, of the 39 patients, 43 hemorrhages were evaluated. For each image, the measurement of the hemorrhage was performed by the author. If there was > 1.5mm difference in the ***size*** of the hemorrhage, then the hemorrhage was considered to be increased in size. The images were checked with the radiologists reading and in 4 cases the hemorrhages were measured as larger, but the radiologist's reading was "stable", In one case, the hemorrhage was read "larger, but the measurement was not increased in size.

Results

CT scans of 39 patients were reviewed and of these 39 patients, 43 ICH's were evaluated owing to 3 patients with multiple types of hemorrhages. 31/43 (72.1%) images showed no significant difference in the size of the hemorrhage, and of these 14/31 (45.2%) were actually smaller on the second CT scan. 12/43 (27.9%) of the hemorrhages were considered to be larger, with > 1.5mm increase in the size of the bleed, however, 4 of these hemorrhages (33.3%) were read by the radiologist as "stabler' on repeat CT. In breaking down the images in types of hemorrhages, 19 (44.2%) were contusions, 3 (7%) were epidural hematomas, and 21 (48.8%) were subdural hematomas. Ten out of 29 contusions (52.6%) increased in size on the second CT, 1/3 (33.3%) of the epidural hematomas increased on the second CT, and 1/22 (4,5%) subdural hematomas increased on the second Cr (Appendix 1). The majority of patients were male (71.8%) and there was approximately an even distribution of patients 65 years of age or older versus less than 65. Seventeen patients greater than or equal to 65 years old and 22 less than 65 years old (See Table 1). For each type of hemorrhage, there were more men than women, and hemorrhages affected patients less than 65 years old except for the subdural hemorrhages in which the age grouping was about the same (See Table 2).

The average time between image one and two was 8.7 hours with the range being from 3 hours to 22 hours_ The Median number was 7 hours. In looking at the hemorrhages that increased in size, the range was 3 —18 hours with the average being 7.3 hours. There were two images in this group of 11 scans that were outliers in terms of time between scans (18 hours and 14 hours) while the average of the other 9 scans in this group was 5.4 hours. These two scans were of patients where the only injury was a contusion, This shorter average time between scans may indicate that due to injury or the appearance of the injury on the initial CT scan, the Neurosurgical staff believed these injuries were somehow more concerning in nature and an earlier repeat scan was indicated.

TABLE 1

	Total (39 patients)	Percentage
Male	28	**71.8%**
Female	**11**	**28.2%**
Greater or equal to 65 yr	**17**	**43.6%**
Less than 65 yr old	**22**	**56.4%**

TABLE 2

	Contusion (# of patients)	Epidural Hematoma (ft of patients)	Subdural Hematoma (It of patients)
Male	**14**	**2**	**16**
Female	**5**	**1**	5
Greater or equal to 65 yr old		1	11
less than 65 yr old	**13**		10

Discussion

In the course of following patients with Traumatic Brain Injury, it is difficult to predict what will worsen over time either clinically or radiographically just from observing the intracranial injury diagnosed on the initial CT scan. There have been articles published concerning this topic, with conclusions made about prognostic factors and who should have repeat CT scans. Although in some cases, the results prove to be conclusive, the majority of Trauma centers that follow the limited established literature are small which is likely due to the retrospective nature of most of the studies. In terms of prognostic factors influencing the likelihood of progressive hemorrhagic injury, Oertel looked at 142 patients with a mean GCS of 8 of which 60 had progressive hemorrhagic injury. 51% of intraparenchymal hemorrhage, 22% of EDH, 17% of subarachnoid hemorrhages (SAH), and 11% of SDH progressed. In analyzing the prognostic factors that would lead to progressive injury, male sex, older age (> 50 years old), increased PTT (partial thromboplastin time), shorter time from injury to initial CT scan, and the absence of hypotension post-injury correlated positively[4]. Chang also looked at risk factors for intraparenchymal hemorrhage progression in his study of 113 head trauma patients with 229 acute IPHs. In this retrospective study, 4% of bleeds were smaller, 58% were unchanged, and 38% were bigger. Progression of IPH growth was independently associated with the presence of SAH, SOH, and the initial size of the parenchymal hemorrhage, with the strongest predictor of IPH progression being the presence of SAH[5]. In his study, the absence of growth of a hemorrhage on the second CT scan predicted a lack of further growth, and he concluded that in most cases, 1PH growth is seen early in the post-injury phase. He further recognized that factors strongly associated with surgical intervention were worsening Glasgow Coma Scale {GCS), significant IPH growth (**>** 5cm^3), and effacement of cisterns on the initial CT scan**.**

Two retrospective studies by Chao and Figg look at the utility and efficacy of routine or serial CT scans in head injured patients. Chap's study looked at the utility of routine CT to predict the need for surgical intervention in blunt head injured patients. Sixty four of 198 patients in the study (323%) had worsening hemorrhages on their follow-up CT scans but only those with a worsening neurological exam had an invasive procedure as a result[6], He concluded that a routine serial CT head in a patient with an unchanged or normal neurological exam would not lead to invasive neurological intervention. Figg also looked at whether serial CTs would prompt surgical intervention in patients with GCS 8 with severe head injury and an intracranial pressure monitor. His results showed that no urgent surgical intervention was performed based on serial CT scans in severe head injured patients who were non- surgical candidates based an the initial and repeat CT scans'. He also took into account the cost of performing these serial CT scans as they relate to radiologist charges, cost of nursing and transport personnel time, and the actual cost of the CT: the cost per CT was $655 per scan.

Carlos and Sifri both performed prospective studies on the topic of repeat CT scans in brain injured patients. Carlos looked at 100 patients in a 9 month period who had SAH, IPH, SETH, EDH, and contusions, of these patients, 68 (68%) underwent 90 repeat CTs. Routine scans were done on 90% of the patients; that is they had no change in the neurological exam which prompted the repeat scan, and 10% had a repeat CT scan after a change mental status or the clinical exam. Of the 90% who underwent routine scan without a clinical change, 26% of the scans were better, 51% were the same, and 23% were worse; no neurological intervention was performed based on these scans. Of the 10% who had a clinical change, 22% of the scans were better, II% unchanged, and 61% were worse. The conclusion of this prospective study is that if there is no change in the patient's neurological exam, the use of a repeat Cr is not supported[8]. Sifri's study looked at the value of a repeat CT scan in patients that had minimal he ad injury who had a normal exam. All patients in this study had a GCS of 5 13 with post-traumatic amnesia or loss of consciousness. 130 patients meet criteria and had a repeat CT within 24 hours of admission. At the time of repeat CT scan 76% of patients had a normal neurological exam and based on the repeat scan, none of the patients required a change in management or neurosurgical intervention. Also, none had a subsequent delayed neurological deterioration for the remainder of their hospital stay. Thirty one (24%) of patients had an abnormal neurological exam at the time of the repeat scan and 2 patients (6%) required neurosurgical intervention as a result (both had an acute change in mental status in the Emergency Department 2-3 hours after the initial CT scan). Rased on this prospective study, Sifri concluded that repeat CT's on patients with minimal head injury and a normal exam is not indicated.

In comparing the results of this study with the results of the aforementioned studies the risk of increasing hemorrhage falls in the 20% - 30% range. When comparing this study to Oertel's study, we have similar results in that contusions increase in size about 50% of the time, but in his study, 22% of EMI increased in size compared to my 33% and his SDH increased in size compared to my 4.5%. This could be a factor of study size, his being 142 patients to my 43. Also, in Ortel's study, the basis of hemorrhage size was based on the Neuroradiologist reading, and there may be discrepancy between what the Neuroradiologist sees as a stable or progressive scan as is evident in my study where 4 scans were read a stable, but if measurements were actually taken, there is proof that the bleed is in fact bigger. Another factor that, in all these studies, may alter the results is that CT scans are usually done in 5rnm slices, and that repeat CTs hardly ever reproduce the same cut us seen on the previous scan. This of course can lead to false reading of the size of a hemorrhage.

Conclusion

The use of CT scans as a tool to diagnose head trauma is paramount, but in this current health care environment, the need for conserving medical costs is becoming a great issue. Prospective studies have been done to show that serial CT scans on patients with minimal head trauma a normal neurological exam are not indicated, yet repeat imaging is still being done routinely. From a review of the literature, the following conclusions can be drawn: if a patient has a normal neurological exam and there is no clinical change during his hospital stay, a repeat CT scan is not indicated, In severe head injured patients with intracranial pressure monitors, serial CT scans are not indicated, if however, there is a change in intracranial pressure, a CT is indicated. In terms of evaluating patients with IPH and determining which patients may have progression of their hemorrhage, factors which may predict worsening of the hemorrhage are male sex, older age, a short time from injury to initial CT, and a prolonged PTT. If a patient has an associated SAH, SDH, or large IPH on initial CT, these correlate with progression of IPH. Future studies should be directed at evaluating the presence of anti-platelet/anti-coagulation factors to predict progression of ICH, and the effect of using reversing agents to halt hemorrhagic progression.

References

1. Valadka, AB. Injury to the Cranium. *Trauma.* 4th Edition. New York: McGraw-Hill; 2000
2. Zwienenberg, M_ Injuries to the Central Nervous System. *ACS Surgery Principles and Practices.* Elmwood Park, NJ. WebMD Corporation; 2002
3. Sosin, OM. Trends in death associated with traumatic brain injury, 1979 through 1992: success and failure. *LAMA.* 1995
4. Oertel, M. Progressive Hemorrhage after Head Trauma: Predictors and Consequences of the Evolving Injury. *Journal of Neurosurgery.* Vol 96, Jan., 2002
5. Chang, EF. Acute Traumatic Intraparenchymal Hemorrhage: Risk Factors for Progression in the Early Post-Injury Period. *Neurosurgery.* Vol 58, April, 2006

6. Chao, A. Utility of Routine Serial Computed Tomography for Blunt Intracranial Injury. *Journal of Trauma injury, infection, and Critical Care.* Vol 51, Number 5, Nov., 2001
7. Figg, RE. Clinical Efficacy of Serial Computed Tomographic Scanning in Severe Closed Head Injury Patients. *Journal of TrOL11170 Injury, Infection, and Critical ow.* Vol 55, Number 6, Dec, 2003
8. Carlos, vrt. Does Routine Serial Computed Tomography of the Head Influence Management of Traumatic Brain Injury? A ProspectiVe Evaluation. *Journal of Trauma Injury, Infection, and Critical Care.* Vol 57, Number 5, Nov., 2004
9. Sifri, ZC. A Prospective Evaluation of the Value of Repeat Cranial Computed Tomography in Patients with Minimal Head Injury and an intracranial Bleed. *Journal of Trauma Injury, Infection, and Critical Care.* Vol 61, Number 4, Oct., 2006

Iatrogenic Atlantoaxial Osteomyelitis Causing Paresis and Respiratory Compromise: A Case Report and Review of Literature

Eric Marvin, D.O., Jonathan E. McNeal, D.O., Nick Qandah, D.O. Gary Simonds, M.D., Carilion Clinic Neurosurgery, Roanoke Virginia

Case report

Abstract: Cervical osteomyelitis (CVO) is uncommon, with infection isolated to the atlantoaxial spine being even more rare. Catastrophic complications can result from such destructive processes and urgent identification and treatment is essential in the prevention of neurological deficit, worsening of an already identified deficit, and respiratory compromise We describe a case of iatrogenic atlantoaxial osteomyelitis with rapidly progressing deficits and respiratory compromise, treated with antibiotics and posterior occipital-cervical stabilization.

Case Report

Our patient is a 60 y/o male with a past history of heavy ETOH and tobacco use. He was reported as having a five week history of neck pain, extending into both of his shoulders. He was being managed conservatively by an outside physician with pain medication, muscle relaxers as had received a cervical epidural steroid injection one week prior. The patient presented to an outside facility with a four day history of progressive dysphagia, weight loss, and right sided paresis. He denied fevers or chills. Computed tomography was performed and revealed a destructive process resulting in a fracture dislocation at C1-2 with a 4-point fracture of the atlas, a fracture line through the base of the odontoid and a significant rotatory component with eccentricity to the right causing moderate canal stenosis (Figure 1). There was no obvious soft tissue swelling, abscess or fluid collection seen on soft tissue windows. A CT scan of the brain failed to reveal any acute process. CT scans of the

chest, abdomen and pelvis as well as a nuclear bone study were all negative for other pathological process. The patient's pacemaker precluded magnetic resonance imaging.

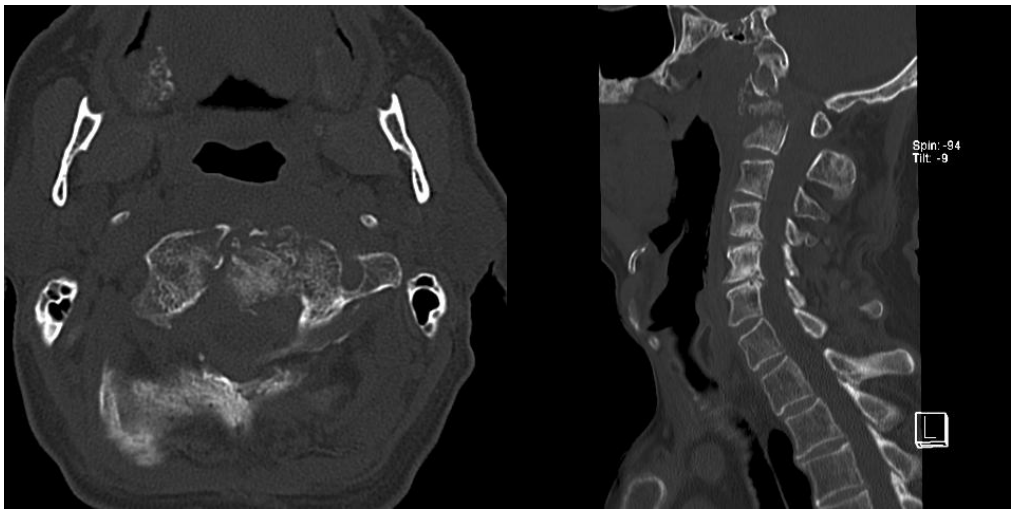


Figure 1

Initial examination revealed no acute distress. Vital signs included a temperature of 96.3, heart rate 77, respirations 20, and blood pressure 185/98. Neurologic exam revealed 5/5 in all major muscle groups on the left side. On the patients right upper extremity, he had 4-/5 strength in all muscle groups including his deltoid, biceps, triceps, wrist flexors and extensors, intrinsic and grip. His lower extremity strength on the right ranged between 4-/5 to 4/5. Sensation to light touch was intact. Reflexes were symmetric throughout at 2+/4. There was no evidence of Hoffman's sign or clonus. Pertinent lab values included a WBC of 17,000 and sedimentation rate of 62 mm/hr.

The patient was placed in the neurointensive care unit with a rigid cervical orthosis and under cervical spine precautions. Later that day, the patient began to experience respiratory distress evident by desaturation, tachypnea and accessory muscle use. Note was made during intubation of an edematous upper airway resulting in difficulty placing the endotracheal tube. The patient was subsequently placed in Gardner-Wells tongs and serial x-rays confirmed good cervical alignment. The neurological exam remained unchanged after this event.

The patient was taken to the OR where an occipital to C5 posterior cervical fusion was performed with placement of lateral mass screws (Figure 2). Intraoperatively, bony destruction was noted of the C2 lamina and frank pus was discovered upon dissecting laterally at the C1-2 level. Cultures revealed oxacillin sensitive Staphylococcus aureus. The patient was extubated on post-operative day number two and placed on six weeks of intravenous antibiotics. The patient was doing well four months postoperatively, with no further signs of infection, no pain, and had regained full strength in his right arm and leg.

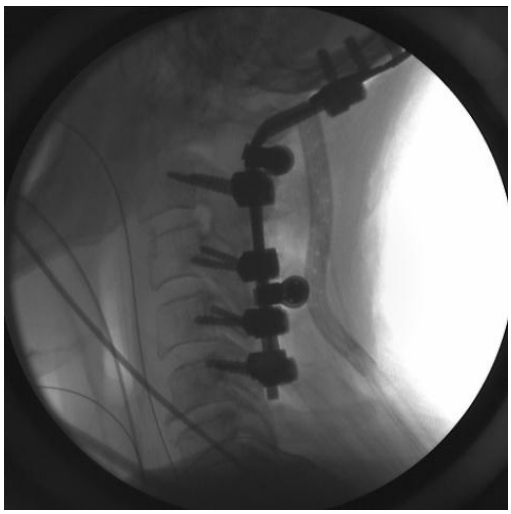


Figure 2

Discussion

Cervical vertebral osteomyelitis is most commonly seen in the fifth to seventh decade of life and interestingly, a significant majority occurs in males (66-87%).[i] CVO is termed "pyogenic" when there is a bacterial etiology causing purulence and a predominantly neutrophilic response[ii], and is thought to be secondary to iatrogenic, spontaneous or hematogenous spread. The most common route of infection is hematogenous spread[iii,iv,xiii]. Iatrogenic etiology is rare, but has been reported, usually in association with epidural abscesses[v,vi,vii,viii,ix,x,xi,xii]. As of 2004, the overall incidence of pyogenic vertebral osteomyelitis was only 1 per 100,000 people,[xiii] however, this number is on the rise[xiii,xiv]. Though this increase may be due in part to changes in social behavior and an aging population, the frequency of immunosuppression (AIDS, treatment of neoplastic disease) as well as the prevalence of chronic disease and intravenous drug use certainly contributes.[xxxi] The established risk factors of CVO reflect this phenomenon and include alcoholism, dental work, liver disease, diabetes, previous surgery, cervical trauma, renal insufficiency, heart disease, tuberculosis, distant focus of infection, and can be seen in up to 27% of IV drug users[xiii,i,xv,xvi,xvii,xviii,xix,xx,xxi.] In addition, spinal infections tend to be associated with a substantial mortality rate. Though treatment has improved over recent years, the death rate of spinal infections continues to be approximately 20%.[xxii]

Whereas the thoracic and lumbar vertebrae are affected in 35 and 40% of cases of vertebral osteomyelitis respectively,[xiii] the cervical spine is the least common site, representing only 3-4% of total cases[i]. Atlantoaxial involvement is even uncommon, accounting for only 0.7% of all spinal infections.[xxiii,xxiv] The literature reflects the rarity of this entity. A Medline (PubMed) search using the text "Cervical Osteomyelitis" resulted in 489 articles, while an inquiry using the text "Atlantoaxial Osteomyelitis" yielded only 13 results.

Clinical presentation of CVO are often non specific in the early stages, thus a high sense of suspicion should be utilized in those with cervical pain and associated risk factors. Pain is the most common symptom and is present in almost all patients with CVO.[xxv] Only 50% will have fever.[xxvi,xxi] Laboratory markers should include WBC, ESR, and CRP. Blood and urine cultures are mandatory, and can be helpful with initiation of antibiotic treatment. CT with 3D reconstruction have the capacity to best evaluate bony destruction, however MR imaging with contrast remains the gold standard for evaluating CVO with 96% sensitivity[xxvii]. As was illustrated by our patient, in the high cervical spine, osteomyelitis and atlantoaxial instability should receive urgent attention. Deficits can range from neck pain with or without radiculopathy to quadriplegia and respiratory compromise.[xxi] Because of the decreased diameter of the cervical canal, neurologic symptoms are not uncommon and present in up to 60% of cases of CVO.[xiii]

Treatment of cervical osteomyelitis is usually stratified into one of two groups: surgical and nonsurgical management. Nonsurgical management is usually indicated for patients without neurological deficit, good pain control, no associated epidural abscess and no significant deformity[i,xvii,xix,xxi,xxviii,xxix]. However, in the absence of these criteria, surgical intervention has been shown to be effective in improving or returning normal neurological function as well as substantially controlling pain in 66-83% of patients[i,xix,xxi,xxx,xxxi]. There remains controversy surrounding the placement of instrumentation and graft material into an infected bed. However, there have been several reports that indicate surgical instrumentation is safe and effective in an unstable, vertebral osteomyelitis.[xxxi,xxxii,xxxiii,xxxiv,xxxv,xl,xxxvi,xxxvii] Outcomes in patients with associated epidural abscesses are often influenced by the patients age, mass effect, location, surgical findings and initial clinical presentation.[xxxviii] Because the anterior elements of the spine are often the focus of the infection, in conjunction with the frequency of associated epidural abscess, anterior surgical approaches (+/- posterior instrumentation) have become the mainstay in treatment of pyogenic vertebral osteomyelitis.[xxxix,xl] However, we feel that in atlantoaxial instability due to cervical osteomyelitis, without significant anterior compression of the cord, a posterior approach can be safe and effective in the definitive treatment of atlantoaxial osteomyelitis.

Conclusion

Atlantoaxial osteomyelitis is an uncommon entity. There should be a high level of suspicion in patients undergoing interventional procedures that present with increasing occipital and cervical pain with associated risk factors. Prompt diagnosis and treatment can defer significant neurologic and respiratory morbidity. The goals of surgery in atlantoaxial osteomyelitis should include debridement and removal of purulence, restoration of neurological function as well as spinal stabilization and restoration of alignment. In addition, there should be a focus on the prevention of further deformity, pain and neurological deficit.[xxi] We feel that posterior

instrumentation in the absence of significant anterior pathology can accomplish these goals in appropriate patients, with acceptable outcomes.

References

[1] Barnes, B., Alexander, J., Branch, C. Cervical osteomyelitis: A brief review. Neurosurg Focus 17(6):Ell, 2004

[1] Tandon N, Vollmer DG: Infections of the spine and spinal cord. Winn HR (ed): Youmans Neurological Surgery, ed 5. Philadelphia: Saunders, Vol 4, pp 4363–4394, 2004

[1] Saban KL, Ghaly RF: Spinal cervical infection: a case report and current update. J Neurosci Nurs 30:105–109, 114–115, 1998

[1] Yazawa S, Ohi T, Sugimoto S, et al: cervical spinal epidural abscess following acupuncture: successful treatment with antibiotics. Intern Med 37:161–165, 1998

[1] Cooper AB, Sharpe MD. Bacterial meningitis and cauda equina syndrome after epidural steroid injections. Can J Anesth 43: 471-474, 1996

[1] Huang RC, Shapiro GS, Lim M, Sandhu HS, Lutz GE, Herzog RJ. Cervical epidural abscess after epidural steroid injection. Spine 29*:*E7-E9, 2004

[1] Knight JW, Cordingley JJ, Palazzo MG. Epidural abscess following epidural steroid and local anaesthetic injection. Anaesthesia 52:576-578, 1997

[1] Elias M. Cervical epidural abscess following trigger point injection. J Pain Sympt Manage 9:71-72, 1994

[1] Rathmell J, Lake T, Ramundo M. Infectious risks of chronic pain treatments: Injection therapy, surgical implants, and intradiscal techniques. Regional Anesthesia & Pain Medicine 31: 41(4), 346–352 , 2006

[1] Reid PJ, Holman PJ. Iatrogenic pyogenic osteomyelitis of C-1 and C-2 treated with transoral decompression and delayed occipitocervical arthrodesis. Case Report. J Neurosurg Spine 7(6):664-8, 2007

[1] Falagas ME, Bliziotis IA, Mavrogenis AF, Papagelopoulos PJ: Spondylodiscitis after facet joint steroid injection: A case report and review of the literature. Scand J Infect Dis 38:295–299, 2006

[1] Waldman SD: Cervical epidural abscess after cervical epidural nerve block with steroids. Anesth Analg 72:717–718, 1991

[1] Acosta FL Jr, Chin CT, Quiñones-Hinojosa A, Ames CP, Weinstein PR, Chou D. Diagnosis and Management of Adult Pyogenic Osteomyelitis of the Cervical Spine. Neurosurg Focus 15: 17, 2004

[1] Jensen AG, Espersen F, Skinhoj P, et al: Increasing frequency of vertebral osteomyelitis following Staphylococcus aureus bacteremia in Denmark 1980–1990. J Infect 34:113–118, 1997

[1] Belzunegui J, Del Val N, Intxausti JJ, et al: Vertebral osteomyelitis in northern Spain. Report of 62 cases. Clin ExpRheumatol 17:447–452, 1999

[1] D'Elia JA, Cooppan R, Younger D, et al: Cervical osteomyelitis. JAMA 241:29, 1979

[1] Dimar JR, Carreon LY, Glassman SD, et al: Treatment of pyogenic vertebral osteomyelitis by anterior debridement and fusion followed by delayed posterior spinal fusion. Spine 29:326–332, 2004

[1] Ghanayem AJ, Zdeblick TA: Cervical spine infections. OrthopClin North Am 27:53–67, 1996

[1] McHenry MC, Easley KA, Locker GA: Vertebral osteomyelitis: long-term outcome for 253 patients from 7 Cleveland area hospitals. Clin Infect Dis 34:1342–1350, 2002
[1] Pinckney LE, Currarino G, Highenboten CL: Osteomyelitis of the cervical spine following dental extraction. Radiology 135:335–337, 1980
[1] Schimmer RC, Jeanneret C, Nunley PD, et al: Osteomyelitis of the cervical spine: a potentially dramatic disease. J Spinal DisordTech 15:110–117, 2002
[1] Keenen TL, Benson DR: Differential diagnosis and conservative treatment of infectious diseases, in Frymoyer JW (ed): The Adult Spine: Principles and Practice, ed 2. Philadelphia: Lippincott-Raven, pp 871–894, 1997
[1] Malawski SK, Lukawski S. Pyogenic infection of the spine. Clin Orthop 272: 58-66, 1991
[1] Yamane K, Nagashima H, Tanishima S, Teshima R. Severe rotational deformity, quadriparesis and respiratory embarrassment due to osteomyelitis at the occipito-atlantoaxial junction. J Bone Joint Surg Br 92(2): 286-8.
[1] Sapico FL, Montgomerie JZ: Vertebral osteomyelitis. InfectDis Clin North Am 4:539–550, 1990
[1] Sapico FL, Montgomerie JZ: Pyogenic vertebral osteomyelitis: report of nine cases and review of the literature. Rev Infect Dis 1:754-776, 1979
[1] Modic MT, Feiglin DH, Piraino DW, et al: Vertebral osteomyelitis: assessment using MR. Radiology 157:157-166, 1985
[1] Lam CH, Either R, Pokrupa R: Conservative therapy of atlantoaxial osteomyelitis. A case report. Spine 21:1820–1823, 1996
[1] Sasaki K, Nabeshima Y, Ozaki A, Mori H, Fujii H, Sumi M, et al. Septic arthritis of the atlantoaxial joint: case report. J SpinalDisord Tech 19:612–615, 2006
[1] Busche M, Bastian L, Riedemann NC, Brachvogel P, Rosenthal H, Krettek C: Complete osteolysis of the dens with atlantoaxial luxation caused by infection with Staphylococcus aureus: a case report and review of the literature. Spine 30:E369–E374, 2005
[1] Rezai AR, Woo HH, Errico TJ, et al: Contemporary management of spinal osteomyelitis. Neurosurgery 44:1018–1026, 1999
[1] Dietze DD Jr, Fessler RG, Jacob RP: Primary reconstruction for spinal infections. J Neurosurg 86:981–989, 1997
[1] Przybylski GJ, Sharan AD: Single-stage autogenous bone grafting and internal fixation in the surgical management of pyogenic discitis and vertebral osteomyelitis. J Neurosurg Spine 94:1–7, 2001
[1] Safran O, Rand N, Kaplan L, et al: Sequential or simultaneous, same-day anterior decompression and posterior stabilization in the management of vertebral osteomyelitis of the lumbar spine. Spine 23:1885–1890, 1998
[1] Rath SA, Neff U, Schneider O, et al: Neurosurgical management of thoracic and lumbar vertebral osteomyelitis and discitis in adults: a review of 43 consecutive surgically treated patients. Neurosurgery 38:926–933, 1996
[1] Ogden A, Kaiser M. Single-stage debridement and instrumentation for pyogenic spinal infections. Neuorsurg Focus 17 (6): E5, 2004
[1] Lee M, Wang M, Fessler R, Liauws J, Kim D. Instrumentation in patients with spinal infection. Neurosurg Focus 17 (6): E7, 2004

[1] Khanna RK, Malik GM, Rock JP, Rosenblum ML: Spinal epidural abscess: evaluation of factors influencing outcome. Neurosurgery 39:958–964, 1996
[1] Cahill DW, Love LC, Rechtine GR: Pyogenic osteomyelitis of the spine in the elderly. J Neurosurg 74:878–886, 1991
[1] Quinones-Hinojosa A, Jun P, Jacobs R, Rosenberg W, Weinstein P. General Principles in the Medical and Surgical Management of Spinal Infections: a Multidisciplinary Approach. J Neurosurg 17 (6): E1, 2001

OPERATIVE NUANCES

Original article by seasoned surgeon on an operative nuance. Eventually, may mimic theme of the journal, ie; "Deformity Surgery."

Recurrent Disc Herniation Surgery The Easy Way

Gary Simonds, MD
Carilion Clinic, Roanoke, VA

Few operations cause as much trepidation in our junior residents as do redo disc surgeries. There are many who feel far more comfortable correcting a kyphosis or clipping an aneurysm than they do wading into a recurrent disc herniation. The prospect of marching through scar to manipulate attenuated dura seems so very daunting, and so guaranteed one or more dural tears- with nerve roots irreversibly surging out and epidural bleeders letting loose. Indeed, I have seen many a senior surgeon turn the procedure into a several hour, complex, microscopic undertaking or, on the other end of the spectrum, a "peek and shriek" minimal intervention.

Ironically, I have always found the operation to be fast and rather enjoyable. Oddly, I have seen few discussions of the technique I employ, but I know several a colleagues who employ it.

The central premise of the technique is to essentially ignore the anxiety-provoking scar that is invariably cemented to sections of the exposed dura, and the creation of rliable anatomic plains that circumvent the scarring.

After the fascial opening a subperiosteal paraspinous muscle dissection is performed using cob pereosteal elevators rather than a bovie (the elevators seem to develop the appropriate plain better). Excess scar over the interlaminar space is removed with a Leksell rongeur in the plain of the lamina.

With the previous surgical site exposed, bovie elctrocautery is employed just adjacent to the laminectomy defect to create a definitive clean bone surface. The adjacent bone-scar interface is then developed using a small, sharp, cupped curette. The curette is used to separate the scar off of the bone right at the laminar defect from the previous surgery. This is usually executed over the facet joint but can also be performed at the pars

interarticularis if it is sufficiently cleaned. The bone-scar interface is then expanded and exploited using the curette. A dissection plain in the interface usually opens up quite readily and the plain is expanded to the region of the inferior pars.

Lamina and medial facet redisual from the previous surgery is removed where necessary for good lateral recess decompression. The key though is to remove facet and lamina bone right to the pedicle of the traversing nerve root (eg L5 at L4-5). The laminar removal should become "flush" with the pedicle.

Now the curette is again employed to easily separate the scar/dura from the medial and inferior pedicle. The pedicle is followed to the floor of the canal- the nerve root is displaced medially and the separation maintained with a cottonoid. Once the nerve root is displaced medially, the surgeon knows that there is no neural tissue between he or she at the pedicle and the cephalad disc space. There will be no neural structure between the point of dissection (pedicle-floor junction) and the disc space- and all scar and epidural tissue up to the disc space can be cauterized with bipolar and cut with scissors or knife with impunity.

Once at the disc space one often encounters the herniation surrounded by a wall of scar although free fragments are not unheard of. The scar is entered sharply and the herniated material is removed. The nerve root may be stuck down with scar and I generally will sharply dissect the root and lateral dura off of the floor to assure better mobilization. Removal of material from the disc space is conducted to the surgeon's desire.

Often the herniation turns out to be more medial and the nerve root is stubborn and does not want to budge medially. Under these circumstances I will enter the disc space lateral to the adhesion to the dura. With "up-biting" pituitary rongeurs the offending fragment(s) can almost always be accessed and removed and the nerve root become freely mobile.

Shards of scar are often stuck to the lateral and anterior nerve root and dura. I may tug on them but will not fight them. Sometimes they must just be left but they do no harm.

The procedure rarely takes more than 40 minutes and scar tissue is never dissected or manipulated. The thick scar over the dura is, for all intents and purposes, ignored.

This method has worked very well through the years with excellent relief of radiculopathy and almost no occasions of dural tears.

RESIDENT'S CORNER

News, discussion, controversy, important information relevant to, or about, osteopathic residents and residencies.

IS A NEUROSURGICAL FELLOWSHIP NECESSARY?

Eric Marvin, DO

Cailion Clinic, Roanoke VA

After six or seven years of intensive training in a neurosurgery residency, it is inconceivable that any more neuroscience knowledge, or surgical acumen, could possibly be packed into a trainee's head. Yet, there is a definitive trend for more surgeons to acquire fellowship training on the heels of their neurosurgical residencies. How necessary is this? How beneficial are the extra 6 months to 2 years of neurosurgical "finishing school?"

There is no "on size fits all" answer to this question. The answer depends on several factors; quality of the residency, desire for specialization, targeted future market, academic interests, confidence level, and others. For the most part, an extra period of training is seldom detrimental other than to one's finances and, possibly, one's mental health (we are all physically and psychically exhausted at the end of our residencies).

What a fellowship offers is essentially a "sheltered workshop" where a neophyte neurosurgeon can hone skills as a bona fide surgeon, but still enjoy and benefit from some degree of supervision. Furthermore the surgeon becomes truly proficient and formidable in at least one component of his or her chosen field. Fellowship also enters the trainee into a "club" of surgeons who readily recognize him or her as a colleague and fellow expert. This sort of networking is always a benefit in the politically charged world of Neurosurgery.

Another related facet that cannot be overlooked is the gravitas that a fellowship adds to the CV of a graduate of an Osteopathic Neurosurgery Residency program. Out in the real world the vast majority of neurosurgeons are allopaths and ignorance and prejudice abound reference their osteopathic colleagues. Qualification for the above mentioned sub-specialty clubs goes a long way to tear down the artificial barriers between the two worlds.

Fellowship training must certainly be entertained by those who wish to enter academic neurosurgery. Extra qualification and expertise, and a specialized research interest, is almost a prerequisite now.

Another consideration is the market one anticipates entering. If the market is wide open and craving general and trauma neurosurgeons then a fellowship is not necessary. If the market is tight and the competition is steep, or a prospective practice needs strengthening in a specific subspecialty field, a fellowship diploma could become indispensible.

In considering a fellowship one also has to be realistic about his or her training. Is the trainee strong in various necessary neurosurgical disciplines. Is he or she particularly lacking in an area that they wish to participate in fully? If one's training was strong and varied, a fellowship may be superfluous.

It is incumbent on neurosurgical residency programs to graduate trainees who are comfortable with the grand majority of neurosurgical disorders and associated procedures. Unfortunately, not all programs hit this mark. For this and many of the reasons catalogued above, a fellowship in a neurosurgical subspecialty should be considered by all graduating residents. It is not a crime to demure. On the other hand, much can be gained by an extra year.

WHAT DOES IT TAKE TO BE A NEUROSURGEON

Jonathan McNeal, DO
Carilion Clinic Neurosurgery, Roanoke, VA

Offended by the top tiered salary that a former partner of mine commanded, physicians of other specialties would somewhat jovially harass him at the lunch table. He would calmly feint a movement towards his inner jacket and retort "I just happen to have an application for residency here in my jacket pocket if you think neurosurgery is such easy money." The words were arresting in their weight and world-weariness. Every one recognized the supreme demands neurosurgery placed upon its trustees and trainees.

Neurosurgery is unique amongst the medical specialties for its rigorous nature, its acuity of disease, its necessary demand for perfection, its need for empathy and compassion, its precision and control of mind and body, its insistence on vigilance, and its exquisite timing of disaster- when least desired or prepared for. What it takes to be a neurosurgeon is surely blood, sweat, toil, and tears. But also humanity, humanity on a level that few can conceive.

Neurosurgery is a medical specialty that attempts to address ALL structural disorders of the central and peripheral nervous system; that is anything that physically impacts the nervous system be it a compressive blood clot, an expanding tumor, swelling from injury, dislocated vertebral bone against the spinal cord, pain caused by pressure against nerves, cut nerves, crushed nerves and so much more. Neurosurgeons deal in the practical, they are people with "street smarts"; "if I do this to the patient, this will happen."

Neurosurgeons are people of action. Things go bad quickly in the nervous system and decisions have to be made on the fly, often with limited information available. Errors of commission are often favored over errors of omission.

Neurosurgery is still a "calling" and it is certainly a "profession." It is not a specialty for shift-workers. Neurosurgery days are hard and long and are often filled with seemingly arbitrary patient outcomes. It is not for the weak of spirit or heart. It is physically and emotionally exhausting.

BUT, Neurosurgey has to be the single most rewarding field of medicine. There are no patients more in need, or more grateful for even failed efforts. It is a field that shifts below one's feet, almost daily. No other field is more affected by advancement in technology. No other field has further to go.

Neurosurgery begins with a six to eight year residency. Technically this is limited to 80 hour work weeks- but don't fool yourself, the eighty hours are filled to the last second (and readily extended to 88 hours in many programs). Then the real work begins. In your free time your are expected to master neuroanatomy (remember that from first year??), neurophysiology, neuroradiology, neuropharmacology, neuro-oncology, neuro-pathology, neurology (yes , you are responsible for all the diseases that they cover), and every approach, indication, risk, benefit, procedure, of every neurological surgery out there.

The first couple of years of residency are generally focused on pre and post-operative care. The resident spends more time in the ICU's and on the wards than actually in the operating room. Call is demanding with much of the action beginning late at night. In the middle years, the resident spends much more time in the operating room and develops surgical judgment and critical thinking. Many residencies incorporate up to 18 months of solid research time into the middle years. This is often spent on "bench research" in various neuroscience laboratories. In the last two years the residents surgical acumen is sharpened to a point with the most difficult and challenging brain and spine cases. In the Chief Resident year full responsibility for a neurosurgery service is assumed and the resident manages and directs a full team of health care providers in the care of their patients.

Generally residencies are high-powered, competitive, and exceptionally fast-paced. Yet an esprit grows amongst the participants that is unmatched in any other discipline of medicine.

Once out of training one faces a profession of late evenings, unscheduled disasters, missed dinners, three o'clock in the morning craniotomies. The typical practicing neurosurgeon spends about two and a half to three days a week in clinic and the remainder in the operating room. In between they manage a complement of hospitalized patients. Call remains very challenging and is often one in three to one in five nights and weekends. A typical day often starts at 0645 and goes to after 6 p.m.. Clearly the profession is demanding and certainly it is often inconvenient.

However, if you are good, you will find many a day when you walk into a room where all are losing their heads and you calmly sort the mess out to the benefit of a patient in need, and where even the sickest patient recognizes your exquisite preparation and skill instituted in their care.

In the end, you will be considering data from the most technically advanced evaluations in history. You will make profoundly impactful decisions. You will employ every fiber of your concentration, intellect, critical thinking, and fine motor

control to get your patient through surgery. You will be a leader in your institution and in your community. You will save lives, and lose lives. You will often be the soul arbitrator for the neurological well being of your patient.

If you feel this is your calling- WE WANT YOU. Call your nearest residency program and have a look. We hope that you join us!

For a more gentile evaluation of what is involved in becoming a neurosurgeon please refer to the adjacent AANA publication; “So, you want to be a neurosurgeon?”:

http://www.neurosurgerywins.org/career/SYWTBANS.pdf.

PRODUCT REVIEW

Submitted article on a piece of equipment, product line, system.

Koros Rotatable Rongeurs

Gary Simonds, MD
Carilion Clinic Neurosurgery, Roanoke, VA

I date back to a neurosurgical era where “indirect decompression” was as foreign a concept as a Hudson brace or a 120 hour work week is to today’s resident. Anterior cervical discectomies were performed using a Cloward interbody graft or equally as satisfying, with no graft at all. We used to speak of some intrascapular “settling pain” but the procedure was just as efficacious at relieving radiculopathy then as is any variant employed today.

This being said, I tended to be very compulsive about the aggressive removal of marginal and foraminal osteophytes. The anticipation of disc space collapse and/or graft settling made me obsessive about leaving a wide open foramen at the end of the procedure. Furthermore, in myelopathic cases I never felt comfortable until a near partial-corpectomy was affected on the marginal spurs. Kerrison rongeurs were employed aggressively across the disc space superiorly and inferiorly and well out into the foramina. The disc space could be rather narrow however and manipulating the jaws of the rongeur into a 45- 90 degree angle to the disc space could be quite a challenge.

Several years ago after having changed parent institutions I was struggling with a large osteophyte and the scrub tech inquired whether I wanted Dr. Martin’s (Scott Martin, a legend at Geisinger Clinic) “greek thing.” Upon inquiring as to the nature of this instrument I was handed a Koros rotatable kerrison-type punch. I have used the instrument in ACDFs ever since. I have been quite surprised at how few of my colleagues are aware of the device. Everyone who has tried it has become instantly

addicted. I must say that I only use it for ACDFs but I am sure that it could be effectively employed in many more procedures.

The key to the instrument is rotatable shaft that allows for an angle of the biting jaws reference to the handle. The angle can be set and reset with ease – just the thumb manipulation of a levered trigger. Thus, 30, 45, 90, 120, 180 degree bites (and everything in between) can be easily obtained with the handle and the surgeon's hand still in a comfortable, and retractor free, neutral position. This is particularly useful in an ACDF where 90 degree bites with respect to the disc space puts the surgeon's hand over the chest and retractors using a standard Kerrison punch.

The instrument can be a bit cantankerous and has had to be sent for repairs on occasion, and it is best for the surgeon to learn the mechanism because it often comes up from central supply in pieces. But these considerations are a small price to pay for the amazing freedom of utility the instrument option. I defy the reader to give the instrument a try and not be hounding their operating room coordinators to purchase a series.

\

\