

# AMERICAN JOURNAL *of* Osteopathic Neurological Surgery



Spring 2014 Vol. 52, No. 1

# Table of Contents AJONS 2017

AJONS Welcome

Instructions for Authors

Winners of the 2017 Neurosurgery Residency Paper Contest

Day to Day Stressors Faced by Neurosurgical Residents

Gary Simonds, MD

**Kyphoplasty Patient-centered Outcomes via Questionnaire**

Daniel Carr, DO

Ethics in “Everyday Neurosurgery”- The Challenge of “Patient Depravity”

Gary Simonds, MD

**Clinical Outcomes in Acute Hypertensive Cerebral Intraparenchymal Hematoma in Patients on Meth Amphetamine Versus Age Adjusted Control**

James AD Berry, DO

**Beside Intracranial Hematoma Evacuation and Intraparenchymal Drain Placement for Spontaneous Intracranial Hematoma Larger than 30 cc in Volume: Institutional Experience and Patient Outcomes**

Tyler Carson, DO

**Cold Hands, Warm Heart But Loss of Surgical Dexterity**

Aaron Danison, DO

**Size of Pituitary Tumor at Surgery Effects Outcome Morbidity and Mortality**

Robert Dahlin, DO

**Boxing for Parkinson’s Disease?**

Gary Simonds, MD

**An approach Using the Occipital Parietal Point for Placement of Ventriculoperitoneal Catheters in Adults**

Jason Duong, DO

fMRI in the Courtroom

Gary Simonds, MD

**Retro Odontoid Pseudo Tumor with Cervical Medullary Compression: A Case Report**

Samer Elfallal, DO

**Put Down the Weights! The Effects of Gross motor Activity on Surgical Dexterity**

Aaron Danison, DO

**The Rate of Adjacent Segment Disease in Combined Anterior and Posterior Lumbar Interbody Fusion: a Retrospective Cohort Study and Review of Current Literature**

Thuy M. Nguyen, DO

**Surgical Deep Wound Infection is Not Related to Intra-Operative Gross Bacterial Contamination in Spinal Instrumentation Surgery**

Jordan Synkowski, DO

**Factors Such as Sodium Level Affecting Return to Hospital Within 30 Days After Traumatic Brain Injury**

Bailey Zampella, DO

**A Case Report of a Treatment Paradigm for Dural Arteriovenous Fistulas: The Statistically Unreported Use of Both Endovascular as Well as Microsurgical Treatment in a Cognard IV Borden III Dural Arteriovenous Fistula Not Described in Recent Literature**

Eric Bialaski, DO

**Use of the Acutrak 4/5 Headless Fully Threaded Variable Pitch Compression Screw for Odontoid Fixation in Type II Odontoid Fractures: A Technical note and Case Series compared with the standard Technique in a Single Institution**

Xin Xin, DO

## EDITOR'S PAGE

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

Welcome to the American Journal of Osteopathic Neurological Surgery and the American College of Osteopathic Surgeons Neurosurgical Discipline. This volume is composed of the Residents' annual papers that were submitted but not published elsewhere. It is therefore dedicated to the future Neurosurgeons and their education. All papers were reviewed by the peer review committee and selected for awards. The papers submitted are excellent, representing some of our talented colleagues. Issues will be published quarterly. I hope that this issue will spread the knowledge of our residents and our section. We will continue to solicit annual papers and all papers submitted at the annual meeting. This is your Journal paid for by your annual dues. This issue is available on our website [AOANeurosurgery.org](http://AOANeurosurgery.org). This is your organization; please support it as you can.

Thank you,

Dan Miulli, D.O, F.A.C.O.S  
Co-Editor In Chief

## INSTRUCTIONS FOR AUTHORS

Papers submitted should be original documentation, including photographs. The papers should be single column, double-spaced in WORD format. 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, correspondence can be submitted to the:

Dan Miulli, DO, FACOS American Organization of Neurological Surgeons

The AJONS is produced and published jointly by the AONS & ACOS-NSD.



## Winners of the 2017 Neurosurgery Resident Annual Paper Award

Joseph Georges, DO

Handheld Confocal Laser Endomicroscopic Imaging Utilizing Tumor-Specific Fluorescent Labeling to Identify Experimental Glioma Cells in Vivo

Philadelphia College of Osteopathic Medicine, Philadelphia, PA

James Berry, DO

The Clinical Efficacy of Mesenteric Lift Osteopathic Manipulative Therapy to Relieve Constipation in Traumatic Brain Injury Patients who are Intubated in the Intensive Care Unit

Riverside University Health System, Riverside, CA

Daniel Carr, DO

Kyphoplasty Patient-centered Outcomes via Questionnaire

St. Johns Providence Hospital, Southfield, MI

The 10 minute talks about the winning papers were presented by each resident on Thursday October 12, 2017 at 11 AM, the first day of the Neurosurgical Discipline lectures. The awards for the first place paper is \$1500. The award for the second place paper is \$1000 and the third place winner will receive \$500. The awards will be presented during the Neurosurgical Reception Thursday October 12, 2017.

The Neurosurgical Resident's Annual Paper Awards are presented by the Neurosurgical Discipline for the best end of year paper submission. Please congratulate these individuals and come see them present their hard work.

## **Day to Day Stressors Faced By Neurosurgical Residents**

Gary Simonds MD MCHDS, Cara Rogers DO, Chris Busch DO, Mike Benko DO, John Fraser MD, Wayne Sotile PhD

Carilion Clinic- Virginia Tech Carilion Neurosurgery, Roanoke, VA

### **Introduction**

For the past 5 years, Neurosurgeon Gary Simonds, and Clinical Psychologist and renown authority on physician wellness Wayne Sotile, have studied and worked with the Neurosurgery team at Carilion Clinic-Virginia Tech Carilion School of Medicine (CC-VTC) on work stress, burnout, and resilience. They focused particularly on the neurosurgery residents, believing them to be especially subjected to the psychosocial stressors of the field. Their work culminated in a book on the subject entitled Building Resilience in Neurosurgical Residents: A Primer.

Neurosurgical residents function in a high-end, high stakes, rapidly changing, multi-tasked environment fraught with inescapably poor outcomes, human tragedies, and unrelenting demands. Residents are tasked with ingesting huge volumes of scientific knowledge and developing exquisite technical skills whilst caring for a broad palette of patients including those with severe head injury, cerebral aneurysm ruptures, stroke, spine fractures, spinal cord injuries, children with brain tumors or congenital spina bifida, and so much more.

While studying the day-to-day plight of the residents, Drs. Sotile and Simonds surveyed the residents extensively and also asked them to tally the various stressors they faced. Results were collated and used as sources for introspection and open discussion. These observations were also used to frame coping strategies for specific and general challenges and stressors.

### **Methods**

An aggressive resilience program was conducted with the CC-VTC Neurosurgery team, with special focus on the residents. This included monthly 2-3 hour group meetings, and resilience; monthly informal resilience dinners; one on one sessions, weekly resilience exercises, and multiple resilience research studies. The various sessions were used to extensively explore the stressors and challenges of neurosurgery, the psychological fallout including burnout, and many strategies and methods of combating the stressors and their effects and building resilience.

For intermittent periods of time (usually 2 weeks) CC-VTC neurosurgical residents were asked to carry hand-held counters at all times during their workdays. In two week increments, they were asked to tally exposure to a series of specific psychosocial entities and stressors. Other stressors were assessed through survey and interview. Results were collated and studied.

### **Results**

Over 4 years, ten residents participated. Exposure to 12 (out of over 60 identified) distinct psychosocial entities and stressors was evaluated.

#### **UNPLEASANT INTERACTIONS**

Residents were asked to tally the number of unpleasant interactions they encountered with hospital coworkers (including nurses, other residents, faculty, technicians) over two-week periods (this was repeated twice). The lowest tally for unpleasant interactions recorded over a two-week period was 20; the highest was 90; with a mean of 65.

#### FREE TIME AT HOME

Residents were asked to record the amount of “free time” at home they experienced during week nights. Free time was defined as time when they were not addressing neurosurgical issues (preparing for surgeries, studying, reading), or other essential caretaking tasks (cooking, bathing, cleaning, eating, finances, etc). This essentially represented time that could be used for pursuit of hobbies, relaxing, socializing, meditating etc. The mean amount of free time reported by residents was less than one hour per night, with a lowest average of 30 minutes.

#### AVERAGE NIGHT’S SLEEP

Residents were asked to record their hours of sleep on non-call nights (nights where they addressed emergent and urgent issues for the neurosurgery service). The lowest average night’s sleep was 4 hours; the maximum was 7 hours (one resident). Most residents reported less than 5 hours average sleep per night over the two-week period assessed.

#### EXPOSURE TO MORTALITY

Residents were asked to tally how many times a week they interacted with patients who were dying or were at “significant risk” of dying (averaged over several weeks). Average number of interactions per resident was 65 per week. The PGY2 level resident (who performed the most hospital rounds) averaged 120 interactions per week.

#### URGENT/EMERGENT INTERACTIONS

Residents were asked to count the number of urgent or emergent interactions they attended to on during a night’s call (6:00 PM to 6:00 PM). Average volume was 38 interactions (phone calls, patient visits) with a high of 68. Average number of emergent, full-patient consultations was 9 with a high of 21.

#### CRUCIAL CONVERSATIONS

Residents were asked to keep track of critical conversations with patients and families. These would include conversations about severe injury or critical illness. Residents averaged 80 such conversations per week with a high of 130.

#### DENIGRATING COMMENTS

Residents were asked to track how many times a coworker spoke to them in a rude, hostile, and/or denigrating fashion. Average was 40 incidents per week with a high of 80.

#### COMPLIMENTS

Residents were asked to tally how many compliments they received from any of their coworkers in a week. The average was less than 2, with several residents reporting none.

## EMOTIONAL STATE ON CALL

Residents were surveyed as to their most common emotional states while on night call. The overwhelmingly most common emotion experienced was loneliness. This was followed (in descending order of frequency) by mild depression, being harried, frustration, angered, excitement, fatigued, accomplished, annoyed, angered, and under appreciated.

## SOCIAL ISOLATION

Residents were surveyed about contact with friends, relatives, and close relations. Only 25% reported engaging routine contact with family and friends. The remainder reported drastic reduction in contact as their residency progressed. All reported a sense of sadness and guilt over reduction in contact.

## THE OUTSIDE WORLD

Residents were surveyed about their response to the world outside of the hospital.

- All reported that, when they were able to experience it, interacting with people outside of the hospital setting engendered a "surreal experience."
- They reported a difficulty accepting that those around them could actually be happy and healthy.
- Every resident surveyed reported feeling like "a stranger in a strange land."

Collectively, residents reported the following observations;

- Discomfort with the unhurried pace of life outside the hospital
- Low frustration tolerance for poor service and inattention
- Shock when encountering friendly and helpful strangers
- Discomfort with how trivial conversations and concerns seemed to be outside of the hospital
- A lack of camaraderie with people outside of the hospital setting -- a lack of anyone with whom they could share the "gallows humor" that bonds the resident cohort

- All residents reported that the unease they felt in the outside environment lead to a longing to be back in the hospital after a brief period of being "out there."
- All reported guilt at leaving the hospital setting for too long.
- All residents also reported an overhanging sense of doom-- that disaster, tragedy, and illness awaited the happy healthy people around them; that the smiling person at the adjacent table may be the next desperately broken trauma victim to be wheeled into the emergency room.

## SCHOLASTIC AND COST OF LIVING DEBT

Residents were asked to estimate their current debt, including collegiate and medical school tuition loans and living expenses. Debt ranged from \$200,000 to \$600,000.

## Discussion

Becoming modern-day physician is a protracted and arduous enterprise. Few enter medical training fully appreciating what they are getting themselves into. Even fewer exit unscathed. Arguably, residency brings the peak years of subjective stress. Our findings indicated that during neurosurgery residency, hours are long, workloads are massive, stakes are high, social

standing is low (within the system at least), lost sleep and fatigue is the rule, humiliation is frequent, approbation is non-existent, and many are exposed daily to tragedy, mayhem, futility, suffering, hostility, depravity, sadness, and more.

Arguably, neurosurgical residents represent an extreme end of the “bell shaped curve,” when it comes to such tests of human emotional and psychological endurance and resilience. Expectations are astronomical, the workloads are insurmountable, the stakes cannot be higher, the academic demands are non-stop, and the exposure to tragedy borders on the intensity of warfare.

The effects of this constant barrage of demands and stressors are clear: Over 50 percent of physicians experience burnout and/or other forms of maladaptive coping. Research has shown that unabated physician suffering puts patient care at risk. It is our hope that a better understanding the tableau of stressors faced by physicians in training will lead to creative interventions that will both help to ameliorate this suffering and foster the development of coping skills that will bolster resilience during the residency years and beyond.

We believe that our study of this small group of neurosurgical residents elucidated many stressors that are likely universal and yet insidious and pervasive in residency training across specialties. The atmosphere surrounding the residents is hardly nurturing and conducive to growth. Heaped upon a frightening level of exposure to death, critical illness, tragedy, crucial conversations, and constant demand is a wealth of slights, disparagements, hostile admonitions and more. The stark absence of compliments is startling and frankly, embarrassing.

The residents’ desire to head back into the hospital and their pervasive sense of doom when encountering healthy happy people is chilling. The residents had essentially “normalized” the abnormal world of a major modern tertiary medical center, and had begun to see the normal happy outside world as deviant (to the norm). This process needs to be reversed. Although it is critical for the residents to seek to make the best of it, the hospital is not a normal place. The sights, sounds, and smells will always be markedly abnormal. Interactions will always be “crucial” and stressed. Time will always be compressed and task-laden. The outside world should be a place of refuge and repair. Residents should be encouraged to drink it all in and allow themselves to feel good there. They should savor their time there and seek to bolster their relationships there.

Perhaps the most important aspect of this study was its effect on the residents who served as our subjects. The residents’ recordings of the stressors they faced lead to open and supportive discussion about the “psychosocial underbelly” of their residency experience. Open discourse served to dissipate some of the sting of this process, and opened the way to discuss healthy methods for remaining resilient. Rather than simply complaining, the residency group was encouraged to find new ways of addressing their stressors. The overall response to this process of identifying, openly discussing, and creatively problem-solving coping options was very positive, and the process continues today, after five years.

We have catalogued many of the stressors and many strategies to ameliorate their effects in *Building Resiliency in Neurosurgical Residents- A Primer* (available on Amazon). We also discuss our observations in podcasts available on iTunes: [Surviving Residency \(and Beyond\)](#). We will soon be publishing an accompanying book directed at all residents and young physicians entitled *The Thriving Physician*.

Please send us your experiences and methods of stress management via [garysimonds@gmail.com](mailto:garysimonds@gmail.com)

## **Kyphoplasty Patient-centered Outcomes via Questionnaire**

Daniel Carr, D.O.<sup>a</sup>, Jennifer Kanack, M.D.<sup>a</sup>, Alicja Sobilo, M.D.<sup>a</sup>, Stephanie Falatko, D.O.<sup>a</sup>, Beverly C. Walters, M.D., MSc., FRCSC<sup>b</sup>, Ryan Barrett, D.O.<sup>a,c</sup>

a. St John Providence Hospital, Department of Surgery, Section of Neurosurgery  
16001 W 9 Mile Rd, Southfield, MI, 48075

b. Department of Neurosurgery, University of Alabama at Birmingham

c. Michigan Spine and Brain Surgeons  
22250 Providence Drive, Suite 601, Southfield, MI 48075

Disclosures and Funding: None

### **Abstract**

**Aim/Background:** To assess patient centered outcomes among adults with compression fractures treated by kyphoplasty.

**Methods:** A 3-question survey was administered via telephone to patients who had a kyphoplasty procedure performed from 2008-2011.

**Results:** One hundred fifty one patients completed the telephone satisfaction survey. Of these, 95.4% of respondents said the procedure was tolerable, 82.8% had full or partial pain relief and 66.2% would have the procedure again.

**Conclusions:** Large randomized and observational evidence support the use of kyphoplasty in osteoporotic and malignant compression fractures. Based on our survey, patients believe kyphoplasty is a tolerable procedure that produces full or partial pain relief and would undergo the procedure again if needed.

**Key Words:** kyphoplasty, vertebroplasty, compression fracture, patient satisfaction

### **Introduction**

Cement augmentation of vertebral bodies began in 1987 with the treatment of vertebral hemangiomas by Gailbert et al<sup>1</sup>. In 2001, kyphoplasty was introduced as a novel method of augmenting vertebral bodies with cement<sup>2</sup>. In 2009, two studies of vertebroplasty versus sham procedure concluded that vertebroplasty was unsuccessful<sup>3,4</sup>. Kyphoplasty usage declined significantly in 2009, presumably secondary to the results published in these two studies<sup>5</sup>. Despite these reported outcomes, anecdotal success with kyphoplasty continued among individual surgeons. Other publications subsequently appeared in the literature that directly contradicted the results from the sham studies, including one randomized controlled trial<sup>6</sup> and one large observational study<sup>7</sup>. Results from these studies have highlighted kyphoplasty's ability to decrease subjective measures such as back pain<sup>6,7,8,9</sup>, improve quality of life<sup>6,9</sup>, reduce physical disability and decrease mortality<sup>10,11,12</sup>. The effect has been studied mainly in the osteoporosis literature but has also shown effectiveness in the groups of patients suffering from pathological fracture secondary to malignancy<sup>13</sup>. The economic impact of kyphoplasty has been studied and shown to reduce healthcare utilization<sup>14</sup>, shorten hospital stay<sup>10, 11</sup>, decrease outpatient follow up visits<sup>15</sup>, and reduce narcotic use<sup>7</sup>. Currently, healthcare reimbursement is dependent upon not only standardized measures of success but also high patient satisfaction. Current research supports the use of kyphoplasty from a functional and economic standpoint

but looking at patients' perspective in medical research is important to complete the overall picture of efficacy. To date in the kyphoplasty literature, only one paper, the 2-year follow up from the FREE paper, has mentioned patient satisfaction using a 20-point Likert scale<sup>9</sup>. They mention statistical significance but do not elaborate on the subject. The objective of our survey was to assess patient-centered outcome measures using specific questions directed at procedure tolerability, pain relief, and willingness to undergo the same procedure again to show the benefits of kyphoplasty not only objectively, but also subjectively from the patients' perspective.

## **Materials and Methods**

### *Patients*

Patients were included in this study if they were >18 years old, with an acute compression fracture confirmed by MRI or nuclear bone scan, and had a kyphoplasty performed. Patients were identified using a coding query from clinic and hospital electronic medical record. All patients who had undergone a kyphoplasty procedure from 2008-2011 were identified. Demographic data from these patients were obtained through electronic medical records. The social security numbers of the patients were checked against the Social Security Death Index (<http://www.genealogybank.com/gbnk/ssdi/>). The deceased patients were identified and excluded from the study. The indication for kyphoplasty was assessed using the medical records as well as pathological information from bone biopsy. They were separated into osteoporotic/spontaneous fractures, fractures related to biopsy-proven malignancy, or traumatic fractures.

### *Kyphoplasty*

Patients were eligible for kyphoplasty based on Magnetic Resonance Imaging or Nuclear Bone Scan demonstrating an acute compression fracture, hyperintensity on STIR sequences and hypointensity on T1 sequences suggesting edema, as well as clinical findings of intractable back pain despite non-operative treatment. All patients identified had a kyphoplasty performed by unipedicular, bipedicular or extrapedicular approach depending upon surgeon preference. All kyphoplasty was performed using the Kyphon Balloon Kyphoplasty system (Medtronic Spine, LLC, Sunnyvale, CA, USA).

### *Survey*

The included patients were contacted through telephone numbers obtained in the demographic data of their electronic health record. The "Kyphoplasty Telephone Satisfaction Survey", a simple three-question survey, was administered to the patient. Institutional review board approval was obtained prior to data collection. Each question is available in Figures 2, 3 and 4. No family member was allowed to take the survey for the patient. If the patient was unable to complete the interview through the telephone they were excluded from the survey and study. If the patient was unavailable for conversation or unreachable, two more attempts were made, for a total of three attempts, before the patient was counted as unreachable and excluded.



## Results

Four hundred ninety two patients were identified from the coding query. Of these, 173 patients were excluded due to identification on the Social Security Death Index as being deceased. Three hundred nineteen patients remained. Nine patients refused to participate in the questionnaire and were excluded. One hundred and fifty nine were unreachable or unable to complete the questionnaire. The remaining 151 of available 310 alive participants were reached and completed the survey, a response rate of 48.7%.

This patient cohort of respondents represents a typical variety for a private practice physician performing these procedures. Full characteristics of the respondents are presented in table 1. The majority of the patients were Caucasian females. The age range was from 26-101, with an average age of 74.3. The most common level requiring kyphoplasty was L1, followed by T12 and then L2. Overall, 61.3% of fractures were at the thoracolumbar junction (T10-L2). Most patients (130 of 151) had either one or two levels treated. No patient had more than three levels performed at one time. The cause for surgery was mainly osteoporotic or spontaneous fractures, which accounted for 72.0% of all patients.

Overall, 95.4% of respondents said the procedure was tolerable. When asked regarding pain relief, 82.8% of respondents had partial or full pain relief from the procedure, with 55.0% overall stating “yes” to the pain relief question. When asked whether they would have the procedure again, 66.2% of respondents stated “yes”. Full survey results are listed in table 2.

## Discussion

This simple questionnaire study showed, from the patient perspective, that treating compression fractures by balloon kyphoplasty is a tolerable procedure that results in subjective pain relief. Based on our findings, most patients’ perspective on kyphoplasty is that given another compression fracture, they would opt for re-operation in the form of kyphoplasty.

Since the two studies in 2009 that showed no benefit of vertebroplasty over sham surgery, there have been large studies specifically regarding kyphoplasty which have contradicted this finding. While there have been many smaller non-randomized studies<sup>8,14</sup>, two major studies<sup>6,7</sup> and a systematic reviews<sup>16</sup> have shown objective decrease in pain, improvement of quality of life and decrease in physical disability from kyphoplasty as compared to conservative management<sup>6,7</sup>.

The patient population presented is similar to the only previous large kyphoplasty specific studies. The FREE trial had 149 patients in their kyphoplasty group of which 77% were female, the SWISS observational study 69.6% female, whereas our study contained 81% female. The average age of our patients was 74.0 while FREE had an average age of 72.2 and the SWISS study, 69.4.

Overall, 58.3% of patients had one fracture treated, compared with 67% for FREE and 77.1% for the SWISS study. The number of patients with two fractures treated was higher in our population at 27.8% than previously mentioned studies. Differences in these numbers may

exist as many of our patients had more than one surgery within our four-year collection period, while the FREE study had only one surgical intervention, while it is not clear in the SWISS study if patients were treated in multiple surgeries. Most of the fractures treated were in a similar area to previous studies; 61.3% were treated at the thoracolumbar junction (T10-L2) in our respondents as compared to 59% in the FREE trial.

Our stratification of patients was similar to the stratification in the SWISS study. Spontaneous fracture was noted in 72.2% of our patients. Osteoporosis was noted in 83.5% of SWISS patients. Trauma was the cause in 14.6% of our patients, while the SWISS study had 12.2%. Finally cancer or pathologic fracture was the cause of 7.3% of our patients and 4.3% of SWISS patients.

The FREE study also collected patient satisfaction data based on a 20 point Likert scale and noted statistical significance to from 1 month to 24 months post operatively. The data presented here show similar results and attempt to build on the FREE results. The questionnaire used in this study was aimed at gathering additional and more specific patient-centered outcomes on kyphoplasty.

Our questionnaire is subject to recall bias. The patients who had procedures in 2008 were called in 2013, thereby introducing approximately 5 years between time of procedure and questionnaire administration. When breaking down the data to compare years, patients who had the procedure in 2008 had the same overall trend in answer choice, with one exception. Patients in 2008 responded “somewhat” to pain relief question 2 53.8%, and “yes” only 28.6% of the time. This trend was reversed in all following years. This finding could be a result of improved surgeon skill over time or recall bias as described above. A perceived limitation of this study may be the lack of objective data such as ODI, RM scale, VAS scale, but we were only attempting to elicit the patients’ individual perspective using patient-centered outcome data.

## **Conclusion**

Since kyphoplasty’s inception, a rocky road has lead from individual anecdotal success to large randomized and observational evidence supporting its use in selected populations. In a changing healthcare environment, it is paramount that patient satisfaction is high among selected procedures. Our population of patients has now shown that kyphoplasty is also well-tolerated, effective, and desirable, based on individual patient perspective.

## **References**

1. Gailbert, P, Deramond, H, Rosat, P, Le Gars, D. [Preliminary note on the treatment of vertebral angioma by percutaneous acrylic vertebroplasty]. *Neurochirurgie*. 1987;33(2):166-8
2. Lieberman IH, Dudeney S, Reinhardt MK, Bell G. Initial outcome and efficacy of “kyphoplasty” in the treatment of painful osteoporotic vertebral compression fractures. *Spine* 2001;26:1631–1638

3. Buchbinder R, Osborne RH, Ebeling PR, et al. A randomized trial of vertebroplasty for painful osteoporotic vertebral fractures. *N Engl J Med*. 2009;361(6):557-68
4. Kallmes DF1, Comstock BA, Heagerty PJ, et al. A randomized trial of vertebroplasty for osteoporotic spinal fractures. *N Engl J Med*. 2009;361(6):569-79
5. Goz, V, Errico, TJ, Weinreb, JH, et al. Vertebroplasty and kyphoplasty: national outcomes and trends in utilization from 2005 through 2010. *Spine J*. 2013; S1529-9430(13)00707-9
6. Wardlaw D, Cummings SR, Van Meirhaeghe J, et al. Efficacy and safety of balloon kyphoplasty compared with non-surgical care for vertebral compression fracture (FREE): a randomised controlled trial. *Lancet*. 2009;373(9668):1016-24
7. Heubschle, L, Borgstroom, F, Olafsson, G, et al. Real-life results of balloon kyphoplasty for vertebral compression fractures from the SWISSspine registry. *Spine J*. 2014;14(9):2063-77
8. Dong R, Chen L, Tang T, et al. Pain reduction following vertebroplasty and kyphoplasty. *Int Orthop*. 2013;37(1):83-7
9. Boonen S, Van Meirhaeghe J, Bastian L, et al. Balloon kyphoplasty for the treatment of acute vertebral compression fractures: 2-year results from a randomized trial. *J Bone Miner Res*. 2011;26(7):1627-37
10. Chen, AT, Cohen, DB, Skolasky, RL. Impact of Nonoperative Treatment, Vertebroplasty, and Kyphoplasty on Survival and Morbidity After Vertebral Compression Fracture in the Medicare Population. *J. Bone Joint Surg. Am*. 2013;95(19):1729-1736
11. Edidin, AA, Ong, KL, Lau, E, Kurtz, SM. Mortality Risk for Operated and Nonoperated Vertebral Fracture Patients in the Medicare Population. *J Bone Miner Res*. 2011 Jul;26(7):1617-26
12. Lange, A, Kasperk, C, Alvares, L, Sauermann, S, Braun, S. Survival and Cost Comparison of Kyphoplasty and Percutaneous Vertebroplasty Using German Claims Data. *Spine*. 2014;39(4):318-26
13. Berenson J, Pflugmacher R, Jarzem P, et al. Cancer Patient Fracture Evaluation (CAFE) Investigators. Balloon kyphoplasty versus non-surgical fracture management for treatment of painful vertebral body compression fractures in patients with cancer: a multicentre, randomised controlled trial. *Lancet Oncol*. 2011;12(3):225-35
14. Grafe IA, Da Fonseca K, Hillmeier J, et al. Reduction of pain and fracture incidence after kyphoplasty: 1-year outcomes of a prospective controlled trial of patients with primary osteoporosis. *Osteoporos Int*. 2005;16(12):2005-12
15. Kasperk C, Hillmeier J, Nöldge G, et al. Treatment of painful vertebral fractures by kyphoplasty in patients with primary osteoporosis: a prospective nonrandomized controlled study. *J Bone Miner Res*. 2005;20(4):604-12
16. Papanastassiou ID, Phillips FM, Van Meirhaeghe J, et al. Comparing effects of kyphoplasty, vertebroplasty, and non-surgical management in a systematic review of randomized and non-randomized controlled studies. *Eur Spine J*. 2012;21(9):1826-43

**TABLE 1**

Table 1 - Patient Characteristics	
Sex	
Male	29

Female	122
Race	
Caucasian	101
African American	12
Asian	1
Unreported	37
Age	
<50	5
50-69	42
70-89	98
>90	6
Cause of Fracture	
Spontaneous/Osteoporotic	109
Malignancy	11
Trauma	22
Unknown	9
Number of Levels	
1	88
2	42
3	12
4	4
5	2
6	0
7	2
8	0
9	1
Levels	
T2	1
T3	3
T4	4
T5	3
T6	6
T7	12
T8	13
T9	10
T10	8
T11	23
T12	40
L1	50
L2	38
L3	19
L4	15
L5	14

--	--

**TABLE 2**

Table 2 - Questionnaire Results	
Q1. Was the procedure to inject cement into your fracture tolerable?	
Yes	144
No	7
Q2. Was the pain in your back relieved by the procedure to inject cement into your fracture?	
Yes	83
Somewhat	42
No	26
Q3. Would you have the same procedure again?	
Yes	100
Not Sure	27
No	24

**Figure 1** Decision Tree for Survey

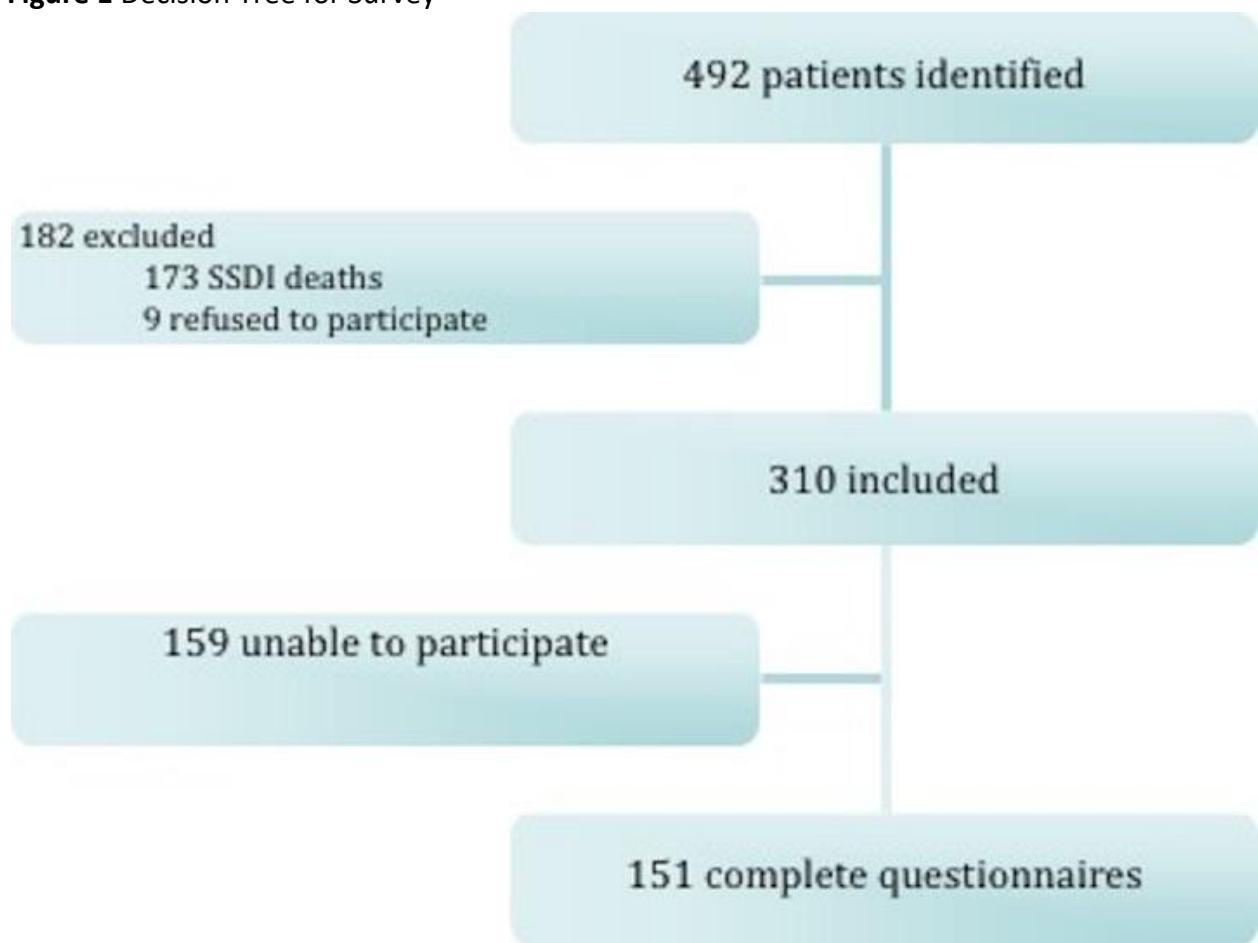
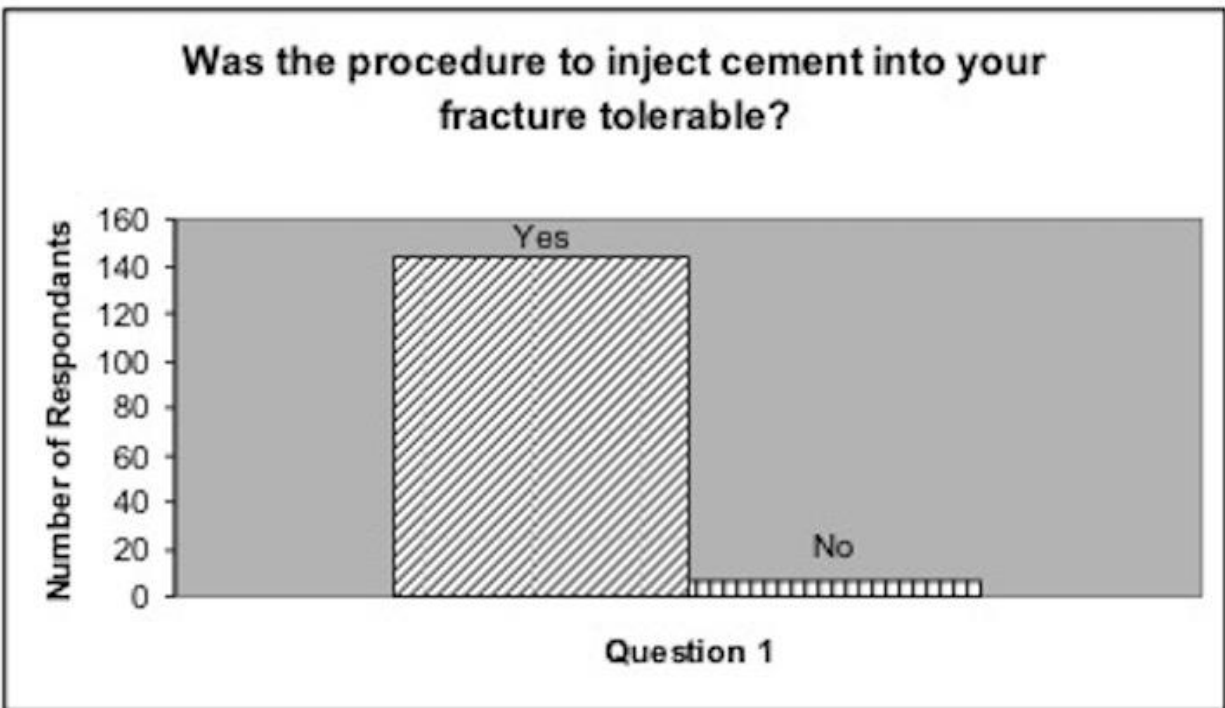


Figure 2 Question 1 of telephone questionnaire.

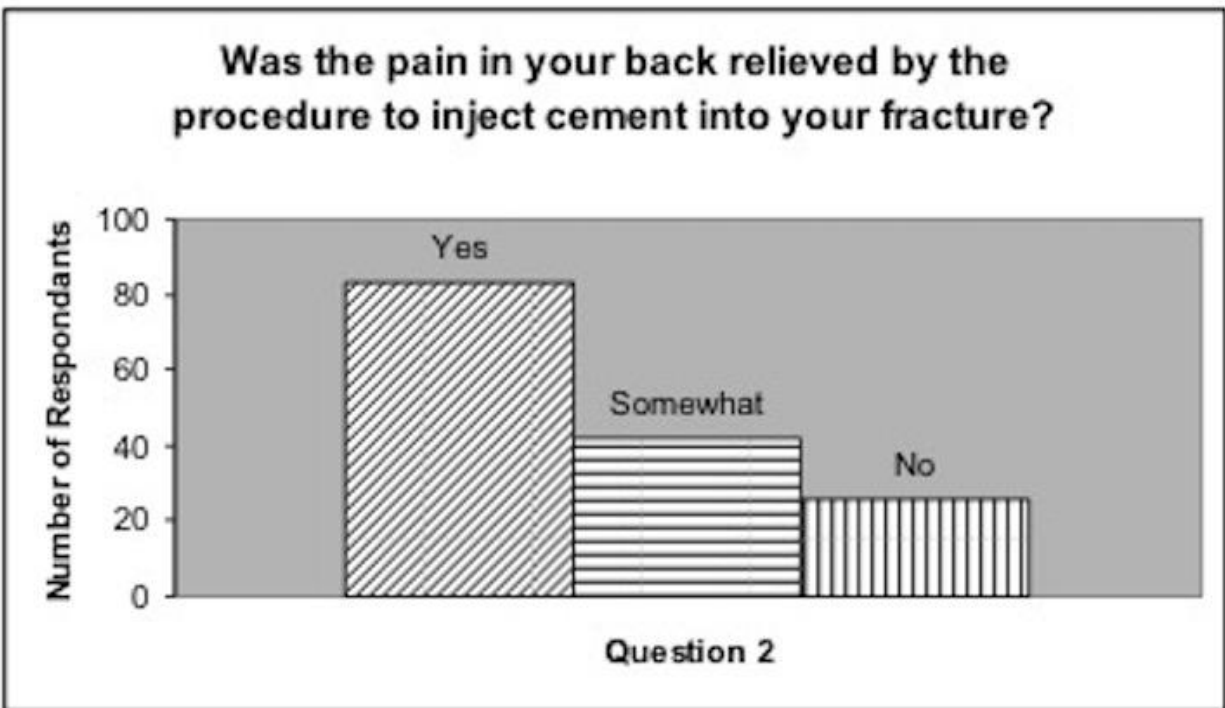


☒ Yes

☐ Somewhat

☐ No

Figure 3 Question 2 of telephone questionnaire.



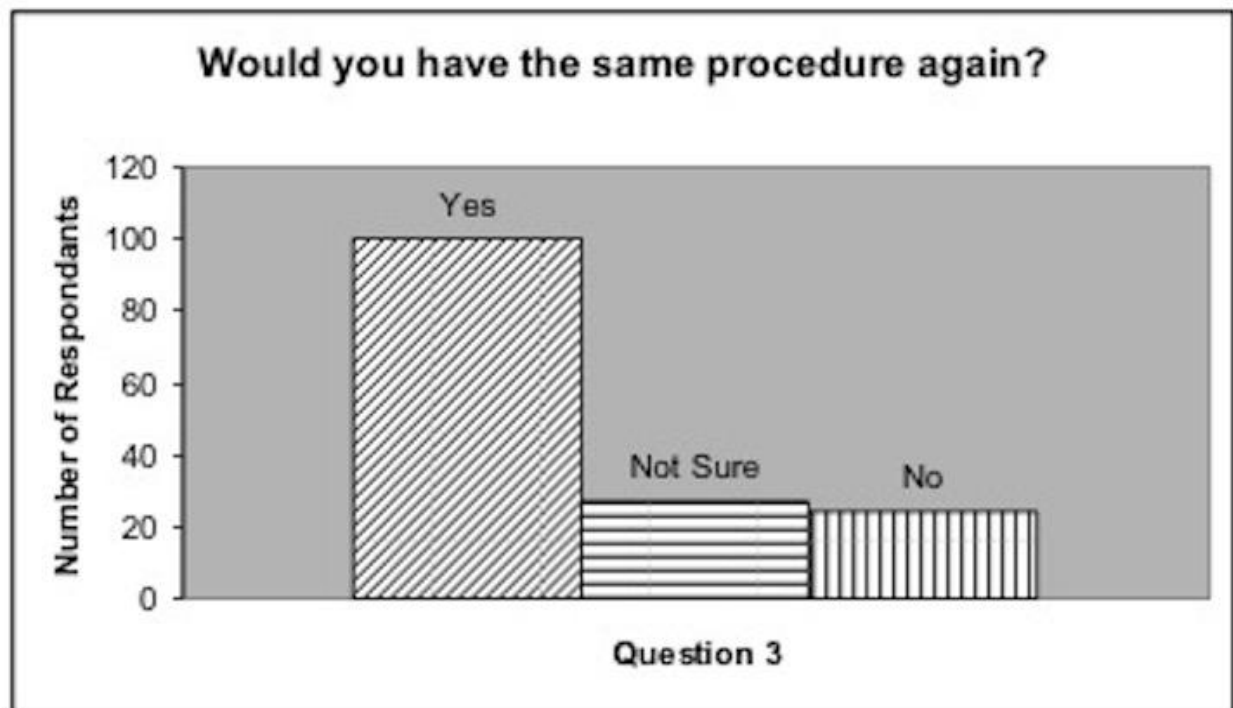
☒ Yes

☐ Not Sure

☐ No



Figure 4 Question 3 of telephone questionnaire.



☒ Yes

☐ Not Sure

☐ No

## **Ethics in “Everyday Neurosurgery” - The Challenge of “Patient Depravity”**

Gary Simonds MD MHCDS, Cara Rogers DO, Chris Bush DO, Greg Howes DO, Eric Marvin DO  
Carilion Clinic- Virginia Tech Carilion Neurosurgery, Roanoke, VA

The following is an excerpt from a book in progress about the ethical decisions made routinely by neurosurgeons and neurosurgical trainees. By “ethical decisions” we are not referring to the more classic dilemmas, presented in ethics courses as study cases due to their complexity or rarity, or to those requiring ethics committees to be adjudicated. Rather we are referring to hundreds of judgment calls a neurosurgeon makes in his or her daily work.

We set out to study these dilemmas and challenges by tallying them over a several month period. We found, however, that they came so fast and furious we could not keep count. We decided therefore to pick several representative issues and study them in further depth. Most are not particular to neurosurgery, and are likely universally encountered by all providers. We feel that they are so frequent and so protean that they have a potentially erosive effect on providers- an often unnoticed source of internal struggle and stress. We hope that exploring these issues will help dissipate some of the related stress. What follows is a discussion of one such ethical challenge.

### **“Patient Depravity” and Labeling**

Typical medical providers were the epitome of “good boys” and “good girls” whilst growing up. They worked hard and they unwaveringly “towed the line” throughout their development and schooling. If they had not, they would not have reached where they are now professionally. That means they “kept their noses clean.” They probably studied in the library on many of the nights their friends were out carousing. They probably worked jobs to support their schooling. They never failed to achieve stellar grades. They probably were in advanced classes since grade school. They probably had countless extracurricular sports and service clubs on their resumes. They stayed out of jail. They sacrificed and gave of themselves. People of authority and responsibility liked them and wrote recommendations for them. They were polite and poised. They were good people.

But then comes a dosing of the “real world.” A dosing that few Americans can even imagine. Most of these good boys and girls end up training at major medical centers- often in big cities- usually featuring massive Emergency Rooms and Level 1 Trauma Centers. They are plunged into a whole new world. The world of a big time medical center is one of a disproportionate representation of human ignorance, dysfunction, psychopathology, and even depravity. We say disproportionate because said individuals tend to disproportionately sustain repeated multiple trauma, rare infections, severe illnesses, overdoses, assault, gunshot wounds, stabbings, hypothermia, malnutrition, and other horrors, than do average citizens.

In such centers these young doctors encounter literal murderers, drug dealers, rapists, child abusers, addicts, gang-members, and the like, on a routine and regular basis. Stories are endless but a few of illustrative examples will drive home the point. John Smith was an addict who “shot up” on a local bridge. He toppled over the railing, fell down an embankment, broke

his neck, and was paralyzed from the triceps down. He lay half submerged in mud for several days surviving on rancid water and the odd insect he could catch with his mouth. He arrived covered in larvae, pustules and boils; hypothermic and in withdrawal. Joe Smith (no relation) was a gang member drug dealer caught selling drugs in another gang's territory. They shot him execution style in the lower neck intentionally paralyzing him. They then threw him in a dumpster behind his mother's house. There he lay for several days surviving on rainwater and any material that once may have been an edible entity. He arrived in similar condition to the above described John Smith. Mary Smith broke her neck in a motor vehicle accident after drinking a bottle of Tequila. She underwent reparative surgery. The day she was released from the hospital, she drove herself home. On the way, she stopped in at a bar. She got drunk and subsequently was involved in another car accident. She totally tore apart her surgical repair, necessitating even more complicated and involved spinal surgery. Maddy Smith was pregnant ten times in ten years. Her first child was born with a severe myelomeningocele. Despite repeated admonitions from her physicians about the risk of further children with spina bifida, or worse, Maddy refused any and all prenatal care. Eventually, seven of her ten children were born with profound cases of spina bifida. Bill Smith was running away from the police when he tumbled down an embankment. He was immediately paralyzed by a severe cervical-thoracic fracture dislocation. He underwent emergency surgery at 02:00 in the morning and experienced a near-miraculous total return of function by the next day. Later in the evening his surgeon came to the room to check on him, and perhaps enjoy a moment of gratitude. Instead, Bill ripped into the surgeon with a blistering invective filled with expletives that "would make a sailor blush." The reason, the team of doctors caring for Bill would not let him out of the ICU to go smoke cigarettes off of the hospital property. Frank Smith got out of jail and headed to a bar to celebrate. He then drove a stolen car down a cul-de-sac, jumping a curb, and plowed into the front porch of a house where two young children were playing. He lay in a trauma bed adjacent to the two toddlers both of whom required emergent brain surgery (who thankfully recovered quite nicely). Joe Smith beat his girlfriend and her toddler child before being stabbed repeatedly by a second boyfriend, three out of the four requiring emergent surgery. Sherry Smith underwent surgery for brain abscesses. An indwelling central line ("pic line") was placed for the many weeks of IV antibiotics she would need. Upon release she used her pic line to inject crushed up analgesic pills and other substances. She returned in extremis with sepsis from her infected pic line.

It is a fair guess to assume that most medical trainees have only rarely if ever experienced people like this, and certainly not to the frequency, degree, and intensity that now becomes their norm. The typical resident, E.R. physician, Trauma Surgeon, Neurosurgeon (as well as many other specialists) experience similar patients almost every night they are on call, sometimes several in a row. How does one who has "towed the line" and "walked the straight and narrow" their entire lives respond to and handle such beings so divergent from themselves? What defense mechanisms will come into play? How can they relate to people whose life experience are so foreign to their own? Can they indeed respect the patient? Can they feel the sacred bond of humanity that helps drive so many health care providers? Is it possible to feel empathy for someone so vastly different?

The risk engendered here is that provider will start making judgments about the character of their patients. Judgments that are non-contributory to the situation; that is, the medical condition at hand. It is so easy in cases such as these. These beings indeed seem so foreign. There is no common ground. They don't fit the mental maps of the provider in any way. Feelings of disdain, disgust, and even fear easily well up. Categorizing the patient as a miscreant is therefore somehow soothing- "this person is an aberration, they are nothing like me." The next step is easy- "not only are they nothing like me, they are less than me- this is why they have ended up in the state they are in." Now the Rubicon of judgment has been crossed to help explain the tragedy and mayhem unfolding in front of the provider. It is a natural progression- experienced by providers on a daily (or nightly) basis.

It is especially natural when the patient is critically ill or horrifically injured. Think about it. As a provider, there has to be a sense of vulnerability whenever one is exposed to what seems like random mayhem. How many times can one muse "sheesh, that could be me lying there in the trauma bay with my body all broken" before it wears one down, interferes with one's ability to function in a critical situation? So, as a defense mechanism it is marvelous to find a reason for the madness. It is particularly effective when the victim can be seen as bringing the mayhem upon him or herself- "oh, he is in such bad shape because he is a drug addict..... a criminal..... a nut case....", etc. In fact, one of the most common tags to a rendition of a motor vehicle accident victim's mechanism of injury is "but he was drunk!" For example: "this is a 37 year old male involved in a single car roll over and ejection who has two broken femurs, a broken pelvis, three thoracic vertebral fractures, a flail chest, and a closed head injury..... But he was drunk!" Everyone in the trauma bay shakes their head in disgust and derision (as if no one in the room has ever been drunk before). This horrible tragedy occurred due to this patient's failure as a rational contributing human being.

Such a qualifier obviates our sense of vulnerability and likely allows us to proceed on dealing with the horror show at hand- detaching ourselves from the stark reality of the situation. But there is a risk. By doing so we are judging the patient and in essence devaluing him or her as a human being. We are essentially saying that "he brought this disaster upon himself and he is lucky to have us caring for him." And somewhere in our deep recesses we may also be thinking: "he is a lesser person than I." Life (and death) just cannot be so sickeningly arbitrary and cruel as it seems in the Emergency Room, Operating Rooms, Trauma Bays, and Cancer Wards- someone (usually the patients themselves) must have caused this through ill will, bad character, or abject stupidity. If not, are we next?

But this can be a very slippery slope. What if the woman with the brain tumor on the operating room table today is not a criminal or a drug addict? We can end up searching for smaller and smaller faults, personality quirks, foibles, to ascribe to our patients. He may not be a rapist or a thief, but how could he be so stupid to dive into the shallow end of a pool? Stupidity, laziness/sloth (as proven by unemployment), drug seeking (as proven by asking for analgesics too often) and, of course obesity, are favorite targets of our contempt. Sometimes, It seems that the patient's greatest offense is simply being old ("a horrible stroke- well, what did she

expect- she is 87??!!"). Depravity, character flaws, undesirableness seem to be in the eyes of the beholder.

As human beings, we are all fallible. Thus when it does come to be our turn to lie in a hospital bed, won't there be elements of our personalities, our pasts, our characters that could be seized upon by our care givers to devalue our worth? Might it affect how we are attended to, how we are cared for? So, when we inevitably make judgments about our own patients, could it possibly affect how we care for them? Are we quick to label a patient (if the labeling was not already done for us- "oh, he is another crack-head" (with requisite eye-roll). With respect to the job at hand does it really matter if the patient was a member of a gang or a minister just returned from helping earthquake victims?

From neurosciences perspective, this is an area well worth studying. How pervasive is patient labeling? How does our perception of patient character affect our processing about his or her disorder? How is care affected by judgments about patient worthiness? How much time is spent with the undesirable patient versus the "citizen" patient? What is our psychological and physiological response to a patient horribly injured? Is it modified by our perception of his or her character? What is the fallout for the provider of not distancing oneself from the humanity of the patient? What brain centers are required to successfully wade into a trauma bay and maintain one's calm and focus?

# **Clinical Outcomes in Acute Hypertensive Cerebral Intraparenchymal Hematoma in Patients on Meth Amphetamine Versus Age Adjusted Control**

James AD Berry, DO

Arrowhead Regional Medical Center, Colton, CA

## **Abstract**

### **BACKGROUND**

Uncontrolled elevations in systolic blood pressure has been identified as the leading risk factor causing cerebrovascular accidents both ischemic and hemorrhagic stroke. Hypertension is not only an underlying etiology but also a factor which causes an intraparenchymal hematoma to expand or re-bleed. The use of meth amphetamine causes a sustained and refractory increase the systolic blood pressure and cause permanent damage to the cardiovascular system. Hypertension can chronically affect small penetrating arteries deep within the brain parenchyma by causing stiffening of the vascular wall, a condition known as hypertensive antipathy. Intraparenchymal hematomas disrupt normal brain tissue architecture and accumulate until the pressure of the hematoma causes the bleeding vessels to collapse and become occluded. However if the systolic blood pressure remains high it can force the pulsating vessel to continue to bleed as it exceeds the collapsing pressure of the hematoma. It has long been consensus that the use of intravenous antihypertensive agents in the setting of acute hypertensive intraparenchymal hematoma can lower systolic blood pressure and improve clinical outcomes. This study aims to assess the clinical outcomes pertaining to morbidity and mortality in age adjusted patient populations who experience acute hypertensive intraparenchymal hematoma on meth amphetamine versus control.

### **STUDY DESIGN**

This is a single institution retrospective study which accessed the records of patients who were admitted to our Level 1 Certified Primary Stroke Center for acute hypertensive cerebral intraparenchymal hematoma on meth amphetamine versus age adjusted control. The study primarily assessed the morbidity, mortality, length of hospital stay, length of ICU stay, Glasgow Outcomes Scale and Glasgow Coma Scale at 3, 7, 14, 30 and 60 days. The need for acute neurosurgical operative and procedural intervention was assessed, e in addition to the efficacy of different intravenous anti-hypertensive agents administered on arrival in acutely lowering systolic blood pressure to less than 140 in one hour.

### **METHODS**

Between September of 2013 and February of 2017, 41 patients aged less than 60 with an acute hypertensive cerebral intraparenchymal hematoma presented to our institution's level I primary stroke center that were admitted under code stroke protocol. The institutions electronic medical records were accessed retrospectively to obtain all points of data under the guidelines of our institutions institutional review board. Patients with aneurysmal or significant vascular malformations were excluded from this study. A urine drug screen identified which patients test positive for amphetamines.

### **RESULTS**

During the period of this study 41 patients under the age of were admitted with a diagnosis of hemorrhagic stroke. Twenty of these patients were positive for Methamphetamines, and twenty one patients were negative for methamphetamines. The age distributions, in both

groups, were from the 30's to the 60's with approximately the same number of patients by age in each group. 8 out of the 20 (40%) of the methamphetamine positive Hemorrhagic stroke patients died. While only 1 of the 21 (4.8%) of the hemorrhagic stroke patients who were not methamphetamine positive died. This demonstrates that the risk of death from a hemorrhagic stroke is over 8 times greater for a person who is Methamphetamine positive as compared to the same age person who is not methamphetamine positive. The Chi Square of 7.424 gives this study a P of 0.0069 indicating a greater than 99 percent chance that the results are statistically significant. Furthermore, 8 of the 20 (40%) of the methamphetamine positive patients required surgical intervention, whereas only 4 of the 21 (19%) of the non-methamphetamine positive patients required surgical intervention. The requirement for surgery (a surrogate measure of severity) demonstrates that Methamphetamine positive patients with hemorrhagic stroke are at more than twice the risk (2.1 times the risk) of requiring surgical intervention as compared to the non-methamphetamine positive patient of the same age group. The Chi Square of 2.17 gives this study a P of 0.14 indicating an 86 percent chance that the results are statistically significant. The GCS score on discharge (another surrogate measure of severity) demonstrates that only 8 out of 20 (40%) of the Methamphetamine positive patients with hemorrhagic stroke had a GCS of 14 or 15 indicating a good outcome. Whereas, 18 of the 21 (86%) of the non-methamphetamine positive hemorrhagic stroke patients were discharged from the hospital with a GCS of 14 or 15. This indicates that a favorable outcome as defined as a GCS score of 14 or 15 is over twice as likely (2.15 times as likely) with a non-methamphetamine positive hemorrhagic stroke patient as with a methamphetamine positive hemorrhagic stroke patient. The Chi Square of 9.227 gives this study a P of 0.002 indicating a greater than 99 percent chance that the results are statistically significant.

#### CONCLUSIONS

People who use Methamphetamine and are positive for Methamphetamine when they have a hemorrhagic brain bleed have a worse outcome as is illustrated with a higher death rate, require more surgical interventions and have a lower GCs score on discharge from the hospital and final Glasgow Out Come Score.

#### Introduction

Hypertension (1) is the number one risk factor which predisposes acute cerebrovascular accidents including both ischemic and hemorrhagic strokes. Hypertension is not only an underlying etiology but also a factor which causes an intraparenchymal hematoma to expand or re-bleed. A wide range of epidemiological studies show that Hemorrhagic Strokes account for approximately 15 to 20% of all cerebrovascular accidents. The incidence of acute hypertensive cerebral intraparenchymal hematoma has increased in recent years primarily in the 16 to 49 age group secondary to abuse of hypertension inducing stimulant illicit drugs such as methamphetamine and cocaine (2).

Hypertension can chronically affect small penetrating arteries deep within the brain parenchyma by causing stiffening of the vascular wall, a condition known as hypertensive angiopathy. The use of Meth Amphetamine (3) has been proven to significantly increase the risk of developing cerebrovascular and cardiovascular disease. Meth Amphetamine (4) causes

the release of a powerful vasoconstrictor endothelin which can cause a sustained increase in systolic blood pressure.

Intraparenchymal hematomas disrupt normal brain tissue architecture and accumulate until the pressure of the hematoma causes the bleeding vessels to collapse and become occluded. However, if the systolic blood pressure remains high it can force the pulsating vessel to continue to bleed as it exceeds the collapsing pressure of the hematoma. It has long been consensus that the use of intravenous antihypertensive agents in the setting of acute hypertensive intraparenchymal hematoma can lower systolic blood pressure and improve clinical outcomes (5). Previous studies (6) have shown a direct correlation between hypertensive cerebral intraparenchymal hematoma size, location and presenting Glasgow Coma Scale on the morbidity and mortality. The use of methamphetamine (7) can cause an increased incidence of prolonged cardiac dysfunction including neurogenic stunned myocardium after hemorrhagic stroke. The neurosurgical management of acute hypertensive cerebral intraparenchymal hematoma (8) can often involve the use of external ventricular drain, particularly when the ventricular system becomes involved.

Older patients with Hemorrhagic Stroke are more likely to have a poor outcome than younger patients. Therefore previous studies comparing methamphetamine positive patients (who are usually younger in age) with non-methamphetamine patients who are usually much older, does not give an accurate comparison of outcomes. It even gives the misleading false assumption that methamphetamine may be cerebral protective. Therefore, the comparison of outcomes between methamphetamine positive and negative hemorrhagic stroke patients should be studied in patients of the same age, in order to remove age as a confounding variable.

## **Methods**

Consecutive patient's admitted to our institution's level I certified primary stroke center between September of 2013 and February of 2017 with acute hypertensive spontaneous cerebral intraparenchymal hematoma were evaluated in a retrospective collection of data. This study was approved by the Arrowhead Regional Medical Center's Institutional Review Board and patient consent was waived because no identifying information was presented and the study was labeled IRB Exempt. The search was conducted using our institutions certified level I primary stroke center's electronic medical record data base and neurosurgical service census using the ICD-10 Code I61.9 which is Nontraumatic intracerebral hemorrhage and the ICD-9 code 432.9 Unspecified intracranial hemorrhage.

Forty one of the one hundred and forty two patients identified met eligibility requirements for participation in the retrospective study (28.8%). Twenty of these patients were positive for Methamphetamines, and twenty one patients were negative for methamphetamines.

Three patients were excluded from the study as they presented with systolic blood pressures of less than 140 mmHg and had not received any anti-hypertensive medications prior to arrival.



De-identified electronic medical records were used to collect the data which included the presenting Glasgow Coma Scale, Glasgow Outcome Scale, Length of Hospital Stay, Morbidity and Mortality at 3, 7, 14, 30 and 60 days, the need for operative and procedural neurosurgical intervention, the efficacy of intravenous ACE inhibitors, Beta Blockers, Hydralazine and Calcium Channel Blockers in acute lowering systolic blood pressure to less than 140 mmHg within the first hour after administration of these agents. Measures of morbidity included the need for procedures such as endotracheal intubation, tracheostomy, percutaneous gastrostomy feeding tubes, central venous lines, arterial lines, Licox Bolt intraparenchymal monitors, need for intubation, incidence of acquiring hospital infections such as ventilator associated pneumonia, urinary tract infections, clostridium difficile colitis and others. The patients ultimate disposition was assessed which included discharge home versus discharge to acute rehab, skilled nursing facility or death was assessed.

The intravenous anti-hypertensive agents used by the admitting service of neurological surgery and in the emergency department as part of the code stroke protocol include the following:

The primary intravenous Angiotensin Converting Enzyme Inhibitor used at our institution in hypertensive hemorrhagic stroke is Enalapril: Dosage 0.625 mg IV initially, if initial dose is not effective within twenty minutes repeat a total of 2.5 mg IV every 6 hours prn Systolic Blood Pressure greater than 140 mmHg

The primary intravenous Beta-Blocker used at our institution in hypertensive hemorrhagic stroke is Labetalol: Dosage Labetalol 10 mg IV every 10 minutes prn to maintain Systolic Blood Pressure less than 140 mmHg not to exceed 300 mg/day

The primary intravenous Calcium Channel Blocker used in our facility in hypertensive hemorrhagic stroke is Nicardipine: Dosage Nicardipine is administered in a drip dosed at 0.1mg/ml to maintain systolic blood pressure less than 140 mmHg and titrated appropriately. The patients on methamphetamine were far less likely to respond to an intravenous antihypertensive within the first hour (18%) compared with (79%) in the control group. Furthermore the amphetamine positive group was more likely to present with a higher initial systolic blood pressure reading, the majority of which exceeded 200 mmHg.

The primary intravenous vasodilator anti-hypertensive use at our facility in hypertensive crisis is Hydralazine: Dosage Hydralazine 10 mg IV q 6 hours for systolic blood pressure greater than 140 mmHg. This agent (9) was not used as it can theoretically increase intracranial pressure.

## **Results**

During the period of this study 41 patients under the age of were admitted with a diagnosis of hemorrhagic stroke. Twenty of these patients were positive for Methamphetamines, and twenty one patients were negative for methamphetamines. The age distributions, in both groups, were from the 30's to the 60's with approximately the same number of patients by age in each group. 8 out of the 20 (40%) of the methamphetamine positive Hemorrhagic stroke patients died. While only 1 of the 21 (4.8%) of the hemorrhagic stroke patients who were not

methamphetamine positive died. This demonstrates that the risk of death from a hemorrhagic stroke is over 8 times greater for a person who is Methamphetamine positive as compared to the same age person who is not methamphetamine positive. The Chi Square of 7.424 gives this study a P of 0.0069 indicating a greater than 99 percent chance that the results are statistically significant. Furthermore, 8 of the 20 (40%) of the methamphetamine positive patients required surgical intervention, whereas only 4 of the 21 (19%) of the non-methamphetamine positive patients required surgical intervention. The requirement for surgery (a surrogate measure of severity) demonstrates that Methamphetamine positive patients with hemorrhagic stroke are at more than twice the risk (2.1 times the risk) of requiring surgical intervention as compared to the non-methamphetamine positive patient of the same age group. The Chi Square of 2.17 gives this study a P of 0.14 indicating an 86 percent chance that the results are statistically significant. The GCS score on discharge (another surrogate measure of severity) demonstrates that only 8 out of 20 (40%) of the Methamphetamine positive patients with hemorrhagic stroke had a GCS of 14 or 15 indicating a good outcome. Whereas, 18 of the 21 (86%) of the non-methamphetamine positive hemorrhagic stroke patients were discharged from the hospital with a GCS of 14 or 15. This indicates that a favorable outcome as defined as a GCS score of 14 or 15 is over twice as likely (2.15 times as likely) with a non-methamphetamine positive hemorrhagic stroke patient as with a methamphetamine positive hemorrhagic stroke patient. The Chi Square of 9.227 gives this study a P of 0.002 indicating a greater than 99 percent chance that the results are statistically significant.

## **Conclusions**

People who use Methamphetamine and are positive for Methamphetamine when they have a hemorrhagic brain bleed have a worse outcome as is illustrated with a higher death rate, require more surgical interventions and have a lower GCO score and lower GCS score on discharge from the hospital.

The patients whose systolic blood pressure was acutely lowered to less than 140 mmHg had a lower incidence of declining GCS and increase in size of their intraparenchymal hematoma on the repeat CT Head without contrast. The preliminary data suggests that intravenous Nicardipine drip was the most efficacious at acutely lowering the systolic blood pressure to less than 140 mmHg within one hour of administration.

Cerebrovascular accident (10) is currently the fifth leading cause of death in the United States of America, and hemorrhagic stroke accounts for almost one out of every five strokes.

The patients who presented with acute hypertensive cerebral intraparenchymal hematoma that had their systolic blood pressure lowered to less than 140 mmHg within the first hour of arrival had lower morbidity and mortality 3, 7, 14, 30 and 60 days in addition less total length of hospital stay, length of ICU stay and less percentage increase in the size of their acute cerebral intraparenchymal hematoma on the first repeat CT Head without contrast.

## References

1. Mukete BN, Cassidy M, Ferdinand KC, Le Jemtel TH. Long-Term Anti-Hypertensive Therapy and Stroke Prevention A meta-analysis. *Am J Cardiovasc Drugs* 2015 August 15(4):243-57
2. Koivunen RJ, Satopaa J, Strbian D, Haapaniemi E, Niemela M, Tatlisumak T, Putaala J. Incidence, risk factors, etiology, severity and short-term outcome of non-traumatic intracerebral hematoma in adults. *Eur J Neurol*. 2015 Jan;22(1):123-32
3. Huang MC, Yang SY, Lin SK, Chen KY. Risk of cardiovascular disease and stroke events in Meth Amphetamine Users: A 10-Year follow up. *J Clin Psychiatry* 2016 Oct;77(10): 1936-1403
4. Seo JW, Jones SM, Hostetter TA, Iliff JJ, West GA Methamphetamine induces the release of endothelin *J Neurosci Res*. 2016 Feb'94(2):170-8
5. Lattanzi S, Cagnetti C, Provinciali L, Silvestrini M. How should we lower Blood Pressure after Cerebral Hemorrhage? A systematic review and Meta-Analysis. *Cerebrovasc Dis*. 2017 Feb 28;43(5-6):207-213
6. Gupta VP, Garton AL, Sisti JA, Christophe BR, Lord AS, Lewis AK, Frey HP, Claassen J, Connolly ES Jr. Prognosticating Functional Outcome Following Intracerebral Hemorrhage: The ICHOP Score. *World Neurosurgery* 2017. 101:577-583.
7. Krishnamoorthy V, Wilson T, Sharma D, Vavilala MS, Prolonged cardiac dysfunction after intraparenchymal hemorrhage and neurogenic stunned myocardium. *A & A Case Reports* [08 Oct 2015, 6(1):3-5
8. Kirmani AR, Sarmast AH, Bhat AR. Role of external ventricular drainage in the management of intraventricular hemorrhage; it's complications and management. *Surg Neurol Int* 2015 Dec 23;6:188
9. Overgaard J, Skinhoj E, A paradoxical cerebral hemodynamic effect of hydralazine. *Stroke* 1975 Jul-Aug;6-(4):402-10.
10. Burwell S, Frieden T, Rothwell C. Centers for Disease Control and Prevention annual report on health status. 2015 US National Center for Health Statistics. CDC Report Atlanta, GA

## **Bedside Intracranial Hematoma Evacuation and Intraparenchymal Drain Placement for Spontaneous Intracranial Hematoma Larger than 30 cc in Volume: Institutional Experience and Patient Outcomes**

Tyler Carson D.O., Vladamir Cortez D.O., Dan E. Miulli D.O.

Department of Neurosurgery, Arrowhead Regional Medical Center, Colton, CA

Nontraumatic intracranial hemorrhages (ICH) occur for a number of reasons including uncontrolled hypertension, amyloid angiopathy, anticoagulant use, cerebrovascular disease, tumors, migraines and those that occur after invasive procedures. Bleeding is usually short-lived and is tamponaded by anatomical and physiological means however is associated with a 30 day morbidity and mortality of 60% and 30% respectively.<sup>1</sup> Elevated blood pressure defined as a systolic pressure greater than 140 mmHg is seen in 75% of patients with acute ICH and strict control of blood pressure is paramount in prevention of delayed rebleeding.<sup>2</sup> The ICH score described by Hemphil in 2001 gives us a good predictive factor for the predicts 30 day mortality.<sup>3</sup>

Many attempts to classify surgical indications for evacuation of ICH have been and continue to be studied. The international surgical trial in intracerebral hematoma (STICH) looked at the outcome of 1033 patients from 83 centers in 27 countries patients treated with early surgery (open craniotomy) vs. initial conservative treatment.<sup>4</sup> Ultimately, the STICH trial showed no significant difference between early surgical treatment and non-surgical treatment groups as a whole. However, there was a subset of the early surgical group which seemed to have a better outcome than conservatively treated patients. These patients had supratentorial ICH that came within 1cm of the surface. To further investigate these findings the STICH II trial was performed which looked at early surgery (within 48 hours) vs. initial conservative treatment specifically for patients with ICH with volume of 10-100 ml, within 1 cm of surface, without IVH and a GCS motor score of 5-6 and GCS eye opening score of  $\geq 2$ .<sup>5</sup> The results showed there was no increase in death or disability at 6 months between groups and there was a small benefit in overall survival.

Clearly there is a subset of patients that benefit from evacuation of hematoma. To that end the MISTIE and MISTIE-II trials (Minimally Invasive Surgery Plus Recombinant Tissue type Plasminogen Activator for Intracerebral Hemorrhage) looked to employ a minimally invasive approach to clot evacuation in hopes to decrease the clot size and perihematoma edema (PHE) more effectively than medical treatment alone.<sup>5,6</sup> Currently the MISTIE III trial is underway and will determine if the reduction in PHE and clot size results in improved neurologic outcome. Here at ARMC we have employed similar techniques, but adapted them to be performed bedside in an ICU setting rather than under general anesthesia in the operative room.

### **Objective**

The Goal of this study is to discuss the institutional experience and patient outcomes over the past 3 years in placement of bedside intraparenchymal drains for intracranial hematomas larger than 30 cc in size.

### **Methods**

Beginning in October 2014 our institution began placement of intraparenchymal drains for patients with intracranial hematomas greater than 30 cc in volume and not related to aneurysmal or AVM rupture. With this study a retrospective review of patient data was performed to determine the effectiveness of IPH drain placement. The primary outcome measure was improvement in GCS from presentation to post treatment. Secondary outcome measures were reduction in clot size, actual versus predicted mortality, re-bleeding associated with catheter placement or after rtPA administration and catheter misplacement requiring repositioning.

The technical placement of the IPH drain utilizes bony anatomical landmarks referenced from CT head to localize the entry point for trajectory of drain placement. The entry point is also chosen keeping in mind where the clot comes closest to the cortical surface, preferably 2-3cm from the Sylvian fissure, midline, or venous sinuses and avoiding eloquent brain tissue such as motor strip. Hair is clipped and sterile preparation of surgical site performed. After local anesthetic and appropriate conscience sedation is administered a 3 cm incision carried down to the cranium. Using the hand twist drill 2 holes are created in same incision ~ 2 cm apart one hole directed toward center of IPH. The dura is opened at both holes. A brain needle with stylet is inserted into clot keeping in mind trajectory and depth at which clot will be encountered based on CT imaging. Once clot has been accessed, the tract is slowly dilated by rotating the needle in a progressively wider circular motion. A Frazier suction tip with stylet is inserted along the tract then the stylet is removed. The wall suction is then connected to the Frazier tip and turned on to 90-120 mmHg suction. The clot is then aspirated until approximately 50% of clot remains based on the output from wall suction. The suction is then turned off and Frazier sucker removed. A trauma style ventricular catheter is then passed down the tract into center of hematoma and tunnel > 5cm from insertion site. The drain is secured to skin and incision is closed. The catheter is connected to blub reservoir but left off suction initially. A post procedure CT Head is obtained to ensure catheter is in the center of the hematoma cavity. If no active bleeding is noted and catheter is in an acceptable position then 2 mg rtPA are administered through the catheter immediately upon return to ICU and the drain is clamped for 1 hour then opened to bulb suction. The CT head is repeated after 12 hours and if no increase in size of hematoma we proceed with administration of 2 mg rtPA per catheter every 12 hours clamping for 1 hour after each administration. It is important to maintain strict SBP goal of <130 mmHg during rtPA administration . The administration of rtPA is continued until output from the catheter is minimal or CT head showing clot size < 15 cc volume.

## **Results**

A total of 12 patients were treated from October 2014 to December 2016, see Table 1. Informed consent was obtained from family member. All patients were treated in the ICU at Arrowhead Regional Medical Center via the method described above. All procedures were performed by a neurosurgery resident. Of the 12 patients, 6 patients had the procedure performed by a single surgeon. The remaining 6 were performed by various surgeons. One patient had care withdrawn per family request after the drain was placed and was withdrawn from the study.

The average number of days treated with the drain was a mean 6.4 days and median 5 days. The number of days treated varied based on the resolution of the hematoma.

The patient's level of consciousness was tracked throughout the hospital course via Glasscow Coma Scale score (GCS). The median initial GCS on arrival was 8T. The lowest GCS that received treatment was a patient who presented as a GCS 5T and the highest was a GCS 12. The median GCS after treatment was 11T. The GCS on arrival and post treatment was compared using the student t-test and P value was calculated to be 0.094 which did not reach statistical significance.

The average clot size on admission was 70.87cc with a range of 26.25 cc to 113 cc. After treatment the average clot size was reduced to 15.95cc, a reduction of 76.9% on average. See figures 1 & 2 which show presenting CT head, CT head post drain placement and CT head post treatment course. The student t-test was performed and p-value was calculated to be 0.0000035 which was statistically significant.

Additionally, all patients showed decreased 30-day mortality when compared to predicted 30 day mortality based on ICH score on arrival. There was one incidence of rebleeding which stabilized and treatment was continued. There were 2 incidences of drains requiring repositioning.

## **Conclusion**

The benefit of evacuation of ICH hematomas remains a controversial subject. Some argue that evacuation is only indicated in life threatening situations such as impending herniation due to the morbidity and mortality associated with craniotomy without evidence of significant neurologic improvement. Other studies indicated that a reduction in mass effect from evacuation of ICH results in decreased perihematoma edema (PHE) and decreased secondary injury similar to the viable penumbra in the case of ischemic stroke. Ideally the answer to both cases is a minimally invasive surgical approach that reduces morbidity and mortality yet results in reduction of PHE. The MISTIE trial attempts to address this with minimally invasive surgical clot evacuation. Our study takes this concept a step further by performing minimally invasive clot evacuation in an ICU setting at the patient's bedside. By performing this surgery bedside we have been able to limit our patient's exposure to general anesthesia, which in many cases results in patient remaining intubated post-op and the sedative effects of general anesthetics. The results for our series of 12 patients shows a trend towards improvement in GCS after treatment with minimally invasive intraparenchymal clot evacuation and drain placement at bedside, though it did not reach statistical significance. There was a reduction in clot size after treatment, which was statistically significant. In addition, a single case of re-bleeding was noted and 2 cases of catheter placement that required repositioning. These, however did not affect the reduction in clot size post treatment nor result in a drop in mental status. In addition, the 30-day mortality actually observed in our patients was lower than that estimated using ICH score

Based on our experience this procedure can be safely performed at the bedside. The use of AXEM or other frameless stereotaxy may be beneficial in the novice surgeon performing this procedure to avoid misplacement of the catheter. This procedure was extremely effective in reducing clot size, and data trends indicated there may be benefit to improvement in neurologic status. Ultimately more patients need to be treated with a longer follow-up period to determine the effectiveness of this technique.

## References

1. Gioia L, Kate M, Dowlathshahi D, et al. Blood pressure management in acute intracerebral hemorrhage: current evidence and ongoing controversies. *Current opinion in critical care* 21(2):99-106, April 2015
2. Qureshi AI, Ezzeddine MA, Nasar A, et al. Prevalence of elevated blood pressure in 563,704 adult patients with stroke presenting to the ED in the United States. *Am J Emerg Med* 2007;25:32-38.
3. Hemphill JC 3rd, Bonovich DC, Besmertis L, Manley GT, Johnston SC. The ICH score: a simple, reliable grading scale for intracerebral hemorrhage. *Stroke*. 2001 Apr. 32(4):891-7
4. Mendelow AD, Gregson BA, Fernandes HM, Murray GD, Teasdale GM, Hope DT, Karimi A, Shaw MD, Barer DH; STICH investigators. Early surgery versus initial conservative treatment in patients with spontaneous supratentorial intracerebral haematomas in the International Surgical Trial in Intracerebral Haemorrhage (STICH): a randomised trial. *Lancet*. 2005; 365 (9457): 387–97.
5. Mendelow, A David et al. Early surgery versus initial conservative treatment in patients with spontaneous supratentorial lobar intracerebral haematomas (STICH II): a randomised trial. *The Lancet* , Volume 382 , Issue 9890 , 397 – 408
6. Mould, W. A., et al. (2013). "Minimally Invasive Surgery Plus Recombinant Tissue-type Plasminogen Activator for Intracerebral Hemorrhage Evacuation Decreases Perihematomal Edema." *Stroke* 44(3): 627-634.
7. Morgan T1, Zuccarello M, Narayan R, Keyl P, Lane K, Hanley D. Preliminary findings of the minimally-invasive surgery plus rtPA for intracerebral hemorrhage evacuation (MISTIE) clinical trial. *Acta Neurochir Suppl*. 2008;105:147-51.

Age	M/F	Etiology	Days tx	ICH sc	GCS initial	GCS post	Clot Adm	Clot post tx	Chng Clot%	Mort D30
49	M	BG Hemorrhage	10	3	8T	13	81.2	15.2	81%	No

57	M	BG Hemorrhage	5	3	8T	10T	56.79	8.68	85%	No
77	M	BG Hemorrhage	8	2	9T	11T	95	12.64	87%	No
53	M	BG Hemorrhage	4	3	8T	11T	72.6	12.2	83%	No
69	F	BG Hemorrhage	4	2	12	14	59.3	15.6	74%	No
18	F	Venous infarct	5	N/A	5T	6T	54.4	9.2	83%	No
73	M	BG Hemorrhage	5	2	10	11	113	23.74	79%	No
58	M	BG Hemorrhage	10	3	6T	10T	80	14.85	81%	No
20	F	Venous infarct	4	N/A	8	14	26.25	9.66	63%	No
43	M	BG Hemorrhage	9	3	6T	11T	54	25	54%	No

Table 1

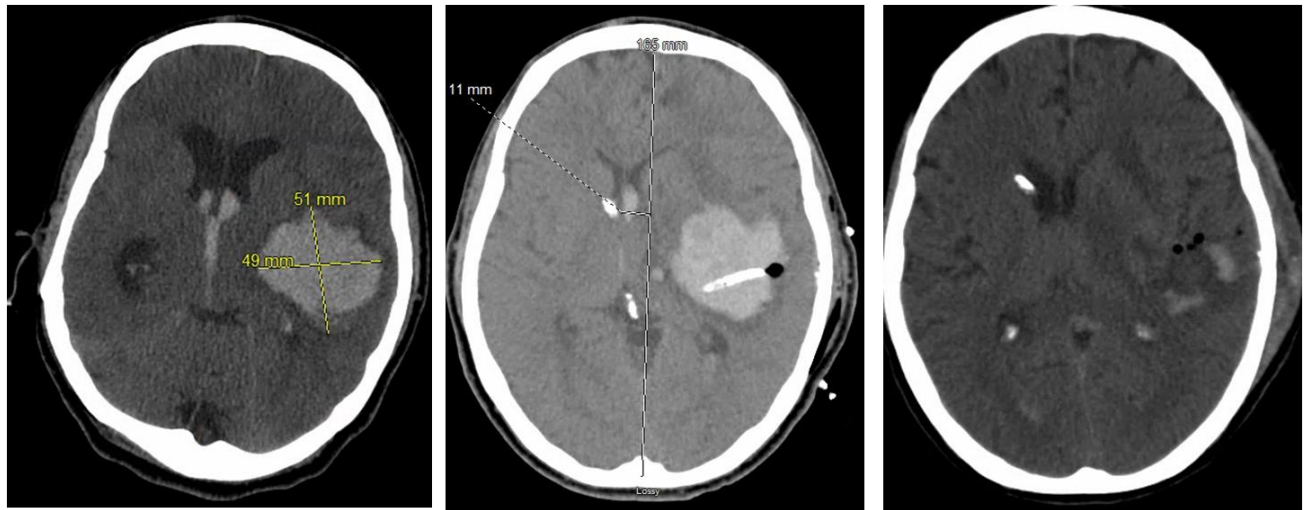


Figure 1





Figure 2

## **Cold Hands, Warm Heart But Loss of Surgical Dexterity**

Aaron Danison DO, Cara Rogers DO, Jonathan McNeal DO, Chris Busch DO, Brendan Klein DO, Gary Simonds MD MHCDS

Carilion Clinic-Virginia Tech Carilion Neurosurgery, Roanoke, VA

### **Abstract**

#### **INTRODUCTION**

We are studying surgeon fine motor dexterity and conditions and circumstances that may affect it. We are hoping to better understand factors that may negatively affect surgical fine motor dexterity, such that they may be mitigated in the interest of minimizing complications due to technical error. This study reports our findings on the effects of hand temperature on fine motor dexterity.

#### **METHODS**

Subjects practiced and underwent fine motor control assessments on a computerized assay system (MLS). In separate sessions they were then subjected to hand cooling and warming. They were then retested on the MLS system.

#### **RESULTS**

Hand warming had no effect on fine motor performance. Hand cooling had significant deleterious effects on fine motor dexterity.

#### **CONCLUSIONS**

Circumstances surrounding the employment of surgical fine motor activities may significantly impact a surgeon's dexterity. Consideration must be given to mitigating such factors. This might include maintaining room temperature at a level most comfortable for the surgeon.

### **Introduction**

Demonstration and quantification of variables that affect fine motor performance could have a significant impact on how surgical procedures are scheduled and performed. We are evaluating a number of variables that may affect surgical dexterity. In another study, we demonstrated that gross motor activity just prior to fine motor tasks caused significant decrement in dexterity. In this study we looked at the effects of heating and cooling of the hands (ambient temperature was not varied).

We believed that cold might have a significant detrimental impact on fine motor control. There is a tendency to keep operating rooms at low temperatures due to the donning of impervious surgical gowns and often lead aprons. Anecdotally, many surgeons have noted diminished fine motor control in particularly cold rooms. We used the test battery Motor Performance Series (MLS), part of the Vienna Test Series, to study the effects of these variables on fine motor dexterity.

### **Methods**

We utilized the MLS Motor Performance Series of the Vienna Test Series by Schuhfried to assess fine motor dexterity. This is a modular test that utilizes Edwin Fleishman's factor analysis of manual dexterity. It consists of a panel with various contact surfaces and the subject uses a stylus to perform static and dynamic tasks. We tested each subject's dominant and non-

dominant arm in the Steadiness, Aiming, Tapping, and Line Tracking tasks. These tests measure the accuracy and precision of movement, steadiness, finger dexterity, and speed of finger, wrist and arm movements

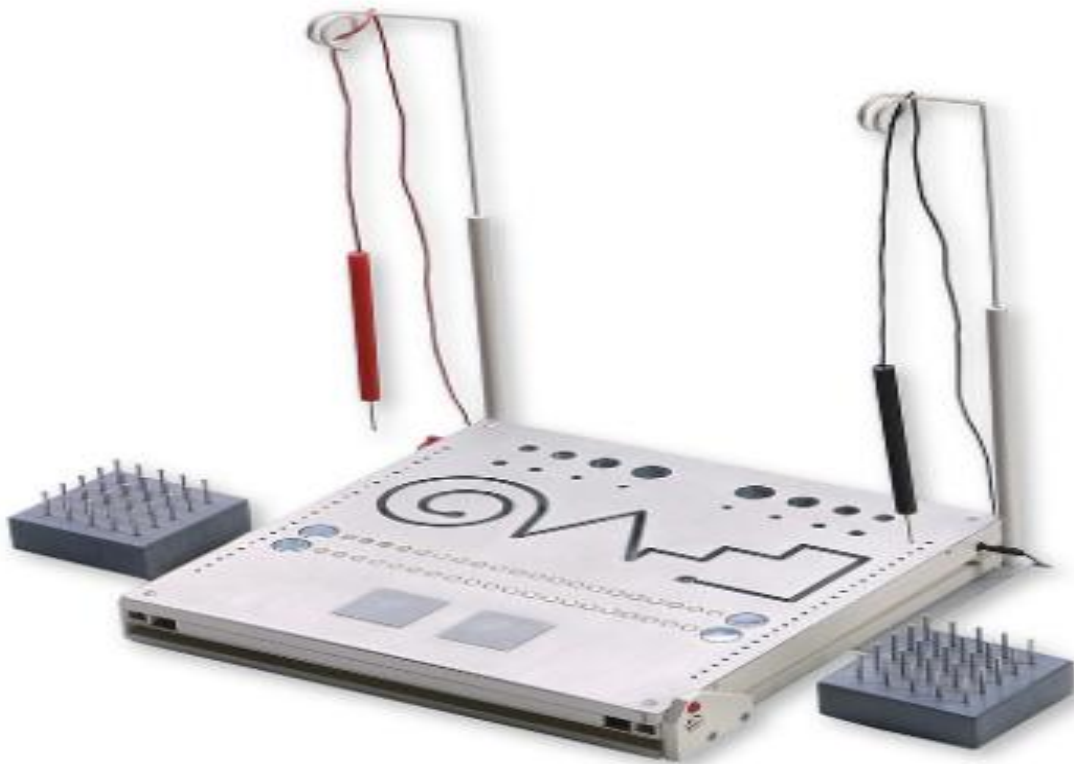
Subjects were all residents, physician assistants, or medical students at our parent institution.

The first phase of the study includes participants performing the six fine motor tasks on MLS until they achieve a uniform/reproducible performance in both hands.

#### Cold/Warm Hands

Subjects performed the six fine motor tasks on the MLS system at various hand temperatures. To decrease hand temperature, participants placed their hands in cold ice water for 1 minute followed by testing on the MLS. After a 5 minute break, the participants then placed their hands in a warm water bath followed by heat blanket. Testing on the MLS was then completed. Hand temperature was measured before and after each individual test. All data was collected at baseline representing normothermia, and after cold/hot challenges. Values at the various temperature challenges were compared with the normothermia data.

Results of the pre and post cooling/heating fine motor activities were scored and analyzed with descriptive statistics.



## **Results**

A total of 26 medical personnel participated in this study. Each participant was tested with both of his or her right and left hands. There were 10 female participants and 16 males representing various levels of surgical training: 9 people with minimal training, 7 with 1- 2 years, 4 with 3-5 years, 6 with 6+ years of training. There were 9 medical students, 3 PAs, and 14 residents. The average age was 31. All participants completed the baseline examination, testing after cooling and heating protocols. All but one study subject was reported right hand dominant.

Steadiness was significantly affected by the cold protocol. Average baseline error rates (Right/Left hand) were 19.04/33.52, which increased to 27.69/26.04 after cooling ( $p$  value  $< 0.03$ ). In contrast, heating did not negatively impact error rates. The average error after heating right/left hand was 24.5/32.4 which did not significantly change from baseline ( $p$  value 0.13).

Aiming: the average error during normothermia was 1.4 across all groups. The average error rate after cooling was 1.31 and after heating was 1.42. Duration of the test was on average 9 seconds. There was no statistically significant change in the number of errors for both hands after temperature variation ( $p$  value 0.36).

During the line tracking task, there was not a significant change in error rates, error durations or precision when comparing normothermia baseline testing to the cooling and heating protocol ( $p$  value 0.17 and 0.30 respectfully). Interestingly, while testing hand speed during the tapping task, we did observe an increase in the total number of hits after varying temperature. Participants increased the average number of hits after both cooling and heating when compared to their normothermia testing but this did not reach significance.

## **Discussion**

In a separate study on the effects of gross motor activity on dexterity and this study on the effects of hand temperature on dexterity, we found that there are indeed relatively straight forward factors that can affect fine motor control and dexterity. We feel that this may have a significant impact on surgical dexterity.

In our previous study we were able to demonstrate that gross motor activities have a statically significant impact on the speed, reproducibility and precision of fine motor activities specifically during steadiness and aiming tasks. Activity lag time and error rates increased after gross motor activities.

In this study, we were able to demonstrate that hand temperature variation has an effect on fine motor dexterity as well, as measured by the MLS. Participants made significantly more errors while trying to maintain steadiness. This study did not test true core temperature cooling but nonetheless demonstrated a decrement in performance even with just the cooling of hands. Clearly, neurosurgery requires great steadiness and precision and these functions should be considered when ambient operating room temperatures are set.

Interesting, effects were seen across multiple test parameters more commonly among female participants compared with males. Females on average had a lower basal temperature before the cooling protocol was initiated which led to lower hand temperatures after cooling.

With greater temperature decreases, activity lag time and error rates increased. Hand/finger speed, reproducibility, and precision of fine motor movements remained intact after both moderate cooling and heating, although a trend to more poor performance was seen with cooling.

Demonstration and quantification of the effects of different modifiable variables of surgical fine motor performance is important because it may be possible to manipulate these variables to improve fine motor dexterity and improve surgical outcomes. So far we have shown that muscle fatigue produced by gross motor movements has a significant negative impact on fine motor dexterity. This study suggests that cold will also have a detrimental effect. We will follow this study up with a similar evaluation of performance in relationship to core temperature.

Primary testing on background noise and its effects on fine motor control are underway. Future plans include examining the effects of sleep deficit, caffeine, core temperature lowering, mental fatigue, persistent engagement in conversation, frequent interruptions/distractions, emotional fatigue, exercise, and even alcohol.

## **References**

1. Fleishman, E.A., Quaintance, M.K. and Broedling, L.A. (1984). Taxonomies of Human Performance: The description of human tasks. Orlando, FL: Academic Press, Inc.
  2. Kalisch T, Wilimzig C, Klebiel N, Dinse H. Age related attenuation of dominant hand superiority. PLoS One. 2006. 1:e90.
  3. Neuwirth W, Benesch M. 2012. Manual: Motor performance series. Schuhfried GmbH, Austria. Version 29.
  4. Magill, R. (2007). Motor learning and control; concepts and applications. (9th edition ed., pp. 2-22). New York, NY: McGraw-Hill companies inc.
  5. Schmidt, R., & Wrisberg, C. (2004). Motor learning and performance, third edition. (3rd ed., pp. 25-48). Champaign, IL: Human Kinetics.
  6. Slack PS, Coulson CJ, Webster K, Proops DW. The effect of operating time on surgeons' muscular fatigue. Ann R Coll Surg Engl 2008; 90: 651-657.
  7. Snow, S. (2010). Skills school fundamental ball skills. (pp. 3-4). US Youth Soccer
- Vanwasshenova, K. (2013, July 21st). Activities that help develop motor skills. Retrieved from <http://uofmhealthblogs.org/childrens/activities-that-help-develop-motor-skills>

## **Size of Pituitary Tumor at Surgery Effects Outcome Morbidity and Mortality**

Robert Dahlin, DO

Arrowhead Regional Medical Center, Colton, CA and Riverside University Health Systems  
Moreno Valley, CA

### **Objective**

In an effort to communicate functionally among medical professionals, the standard nomenclature that has evolved to describe pituitary adenomas is one that is based on size. Adenomas less than 10mm are described as microadenomas, adenomas greater than 10mm are macroadenomas, adenomas greater than 30mm are large adenomas, and adenomas greater than 40mm are considered giant pituitary adenomas. In an ideal classification scheme, each category would correlate with a clinical significance. While it would seem logical that increasing tumor size would be associated with increased challenge of removal and associated complications, there is a scarcity of literature that investigates this relationship. To date there has been only one study that concluded that increased size was correlated with increased mortality<sup>9</sup>, while other studies have not had sufficient power to evaluate this.

The current literature is equivocal as to whether there exists a statistical difference between complications of adenoma resection of all sizes vs giant adenomas. One problem with the current literature is that large series of pituitary resections do not specify complication rates based on size and hence the data is grouped together and unable to be compared to studies specifically of giant adenomas. Another problem with the literature is that most papers specifically analyzing complications of giant have a rather small sample size limiting statistical significance.

When evaluating data collected from studies of patients undergoing resection of pituitary adenomas, it does seem that there might be an increased rate of complications in patients with giant adenomas. In the current literature, the rate of diabetes insipidus (DI) ranges from 1.7%-9.8%,<sup>13, 7</sup> vs 6.6%-80%<sup>1, 14</sup> in the giant adenoma literature. The rate of CSF leak 0.32%-5.6%<sup>13, 7, 15</sup> versus 0.0%-16.7%<sup>14, 11</sup> in the giant adenomas. The rate of meningitis 0.08%-7.8%<sup>15, 21, 5</sup> vs 0.0%-5.5%.<sup>11</sup> The mortality rate ranges from 0.07%-0.45%<sup>7, 9, 13</sup> vs 0.0%-16%<sup>5</sup> in giant adenoma series. Previous studies that have evaluated risk factors for increased morbidity and mortality have identified an age greater than 65<sup>7, 9</sup>, and performing a resection through a transcranial approach as predictors of increasing morbidity and mortality. There have been two studies that sought to address whether the size of the adenoma correlates with mortality; one study found an increased rate of mortality 3.7% vs 0.1%<sup>9</sup> and the other did not achieve statistically significant result due to being underpowered<sup>5</sup>.

While most case series will discuss their operative mortality, to date no study has attempted to address functional outcome by either pre and post-operative Glasgow coma scale, Glasgow outcome scale, or Karnofsky performance score (KPS). Gilberto et al retrospectively reviewed 12 patients who underwent resection of either large or giant pituitary adenomas and within their study group only 25% of patients had a KPS of 100 at final follow-up. However this study

did not record pre-op KPS for comparison<sup>4</sup> and as such one cannot infer which patients improved, worsened or remained the same post-operatively.

Knowledge of a patient functionality post-operatively and their risk factors is important as it will not only guide patient discussions about surgery, but will help guide surgical decision making to decrease morbidity if risk factors are identified. This study was undertaken to evaluate for possible correlations between tumor size and their associated surgical challenges and complication rates, as well as to determine whether characteristics of either the patient or tumor, other than tumor size, correlated with a change in functional status.

## **Methods**

Research approval was performed by the IRB at Arrowhead Regional Medical Center. A search of the neurosurgical database that is maintained at the hospital was performed to identify all patients undergoing resection of a pituitary adenoma consecutively from 2009-2015. Data to be recorded included the patients age at time of surgery, presenting symptoms and Glasgow Coma Score (GCS), GCS at discharge or 7 days post-operatively, GCS at 6 months, size of the pituitary adenoma, imaging characteristics of the tumor prior to resection, post-operative complications, and patient mortality. Primary outcomes were mortality and 6 month GCS. The only exclusion criteria for the study was insufficient documentation of the above data points; as such the study included patients of any age with a pathologically proven diagnosis of pituitary adenoma resected by any surgical approach.

Imaging characteristics measured included the superior/inferior diameter of the tumor measured by a mid-sagittal MRI from the floor of the sella turcica to the superior end of the tumor. Anterior posterior dimensions were obtained by measuring the maximal diameter of the tumor in the plane that is perpendicular to the line used in measuring the superior/inferior diameter. Maximal medial/lateral diameter was calculated as the maximal medial lateral diameter of the tumor on axial view MRI. The presence of pre-operative hydrocephalus was evaluated for on pre-operative MRI And the presence of mass effect on the brainstem as evidence by deflection of the normal brainstem structures was also recorded from pre-operative MRI. All measurements were made by a single observer R.D.

Statistical analysis was performed using SPSS version 20. Chi-square analysis was performed to determine differences between qualitative data and independent student's T test was chosen to evaluate for the difference between quantitative data with a  $p < 0.05$  being considered statistically significant.

## **Results**

Two clinical determinations are important when reviewing the data. Does there exists a size after which complications become more frequent; and after identifying those patients who did not do well, are we able to identify risk factors that seemed to portend a worse prognosis?

In reviewing the data with regards to size; with macro adenomas defined as adenomas greater than 1cm but less than 4cm, and giant adenomas being defined as adenomas greater than 4cm

in maximal diameter, there were a total of 5 giant adenomas in our series of patients. There was no difference in age or gender between patients with macro or giant adenomas. Patients with giant adenomas were however more likely to present with a cranial nerve (CN) palsy on ( $P = 0.019$ ), altered mental status ( $P = 0.0001$ ), and hydrocephalus ( $P = 0.002$ ). On MRI, patients with a giant adenoma were also more likely to have mass effect on the brainstem ( $P = 0.020$ ). There were no differences in the rates of visual changes, apoplexy, or endocrine dysfunction on presentation. Postoperatively, macro adenomas had a post op day 7 GCS of 14.22 vs 11.0 ( $P = 0.253$ ), and a 6 month post op GCS of 14.13 vs 10.80 ( $P = 0.276$ ). There was an increased rate of stroke in patients with giant adenomas ( $P = 0.019$ ).

There was no difference in the rate of post-op transient DI, permanent DI, CN palsies, hemorrhage or mortality.

When comparing patients who continued to have a declined GCS at 6 months versus patients who either improved neurologically or remained at their baseline, there was no difference in age or sex between the two groups. Furthermore, there was no difference between the two groups in the rate of apoplexy, vision changes, endocrine changes, cranial nerve palsies, or hydrocephalus on presentation. The average age of patients who experienced a decline in mental status vs no change was 41.25 vs 46.5, ( $P = 0.558$ ). There was a difference in the rate of presentation with altered mental status ( $P = 0.006$ ). The mean maximum diameter of the adenoma in patients who experienced a decline in mental status was 41.25 vs 29.83 ( $P = 0.325$ ). Other dimensions such as maximum caudal cranial diameter, medial-lateral diameter, and anterior-posterior diameter were not significantly different. Patients with mass effect on the brainstem were also more likely to experience a decline in mental status ( $P = 0.005$ ). Postoperatively, patients who had a decline in mental status were more likely to have been found to have had either an ischemic stroke ( $P = 0.0001$ ), or new intraparenchymal hemorrhage ( $P = 0.013$ ).

	All Patients N = 28	Declined Group N = 4	No change Group N = 24	P Value
% Male	17 / 28	3 / 4	14 / 28	0.527
Average Age	45.75	41.25	46.5	0.558
Presenting Symptoms				
Vision Change	25 / 28	3 / 4	22 / 24	0.318
Hydrocephalus	2 / 28	1 / 4	1 / 24	0.134
Altered Mental Status	3 / 24	2 / 4	1 / 24	0.006
Cranial Nerve Palsy	3 / 24	1 / 4	2 / 24	0.318
Apoplexy	5 / 28	0	5 / 24	0.314
Endocrine changes	5 / 28	0	5 / 24	0.314
Initial GCS	14.96	15	14.96	0.691
GCS at 7 days	13.64	5.75	14.96	0.010
GCS at 6 months	13.54	4.75	15	0.002
MRI Characteristics				



Max Diameter	31.46mm	41.25mm	29.83mm	0.325
AP Diameter	23.46mm	27.5mm	22.79mm	0.619
ML diameter	24.18mm	35.25mm	22.33mm	0.143
CC diameter	30.21mm	41.25mm	28.38mm	0.277
Mass effect on Brainstem	6 / 28	3 / 4	3 / 24	0.005
Post-op complications				
CSF leak	0	0	0	-
Meningitis	0	0	0	-
Stroke	3 / 28	3 / 4	0 / 24	0.000
Hemorrhage	1 / 24	1 / 4	0 / 24	0.013
New CN deficit	1 / 24	0 / 4	1 / 24	0.678
Transient DI	8 / 28	1 / 4	7 / 24	0.864
Permanent DI	3 / 28	0 / 4	3 / 24	0.454
Mortality	2 / 24	2 / 4	0 / 24	0.000

Table 1: Declined group is defined as patients whose examination was conducted at 6 months post-op. No change group is defined as patients who either improved neurologically or remained at neurologic baseline.

## Conclusion

It is important to identify risk factors associated with the resection of pituitary adenomas as it will affect the discussion surgeons have with the patient and may ultimately affect the way surgeons approach their resection. Previous studies have identified age greater than 65 as a risk factor for poor prognosis. Our study however had only one patient fall into that category limiting statistical for that subgroup. Surgical approach was also a risk factor identified in previous studies, with craniotomies being found to have a higher rate of morbidity and mortality. Tumors resected via a craniotomy typically have characteristics that make them poor candidates for resection via other methods due to the inability to achieve maximal resection from either its size or pattern of spread which makes parts of the tumor inaccessible through a transnasal approach. This pattern was reflected in our study in which only two craniotomies had been conducted, with both being performed on patients with giant adenomas.

There was a trend towards patients experiencing a decline in GCS post-operatively as the size of the adenoma increased. This was however not statistically significant.

Patients who experienced a decline in mental status were more likely to present with altered mental status, and found to be more likely to have had either a post-op ischemic stroke or intraparenchymal hemorrhage.

We theorize that this may be due to the tumor either investing itself in key structures of the brain, or causing mass effect on critical structures such as arteries in the circle of Willis. A tumor that is either invested in, adherent to, or are significantly displacing these arteries, can

theoretically lead to either injury or spasm of these arteries during surgical resection. As an analog way to measure investment of the tumor into these structures we evaluated whether the tumor caused either mass effect on the brainstem or hydrocephalus. This decision was made as a tumor that causes either of these to occur radiographically must be pushing against and displacing the arteries of the circle of Willis.

In our study we found that indeed patients with mass effect on the brainstem were statistically more likely to experience a decline in mental status ( $P = 0.005$ ) or death ( $P = 0.005$ ).

The question then remains: Does the size of the tumor cause an increase in the rate of the complications or is it merely correlated with complication rates? Our data would indicate that increasing adenoma size is associated with increasing surgical risk due to qualities inherent to increasing adenoma size such as their propensity to present with altered mental status, and mass effect on the brainstem. One can presume that the presence of altered mental status is a way of implying size of a tumor as it would require either significant mass effect of the tumor on the brain, or compression of arteries. Our study results at this time should be taken with caution however. While we found that patients with altered mental status and mass effect on the brainstem were statistically more likely to experience a decline in mental status, this was achieved with a low number of patients. Further, as we did not have many patients who underwent craniotomy as a means of resection, we were unable to control for this confounding variable. At this time, future studies will need to be performed to determine whether these variables do indeed correlate with post-operative morbidity and mortality.

## References

1. Goel A, Nadkarni T. Surgical Management of Giant Pituitary Tumours - A Review of 30 Cases. *Acta Neurochir*. 1996; 138:1042-1049
2. Murat Mu A, Cansever T, Adem Yilmaz A, et al. Surgical Results of Large and Giant Pituitary Adenomas with Special Consideration of Ophthalmologic Outcomes. *World Neurosurgery*. 2011; 76 [1/2]: 141-148
3. Guo F, Song L, Bai J, et al. Successful treatment for giant pituitary adenomas through diverse transcranial approaches in a series of 15 consecutive patients. *Clinical Neurology and Neurosurgery*. 2012; 114 885– 890
4. Leung GKK, Law HY, Hung KN, Fan YW, Lui WM. Combined simultaneous transcranial and transsphenoidal resection of large-to-giant pituitary adenomas. *Acta Neurochir* . 2011; 153:1401–1408
5. Garibi J, Pomposo I, Villar G, Gaztambide S. Giant pituitary adenomas: clinical characteristics and surgical results. *British Journal of Neurosurgery* 2002; 16(2): 133–139
6. Gondim JA, Almeida JP, Albuquerque LA, Gomes EF, Schops M. Giant Pituitary Adenomas: Surgical Outcomes of 50 Cases Operated on by the Endonasal Endoscopic Approach. *World Neurosurgery*. 2014; 82 [1/2]: 281-290
7. Liu J, Li C, Xiao Q, et al. Comparison of Pituitary Adenomas in Elderly and Younger Adults: Clinical Characteristics, Surgical Outcomes, and Prognosis. *JAGS*. 2015; 63:1924–1930
8. Juraschka K, Khan OH, Godo BL, et al. Endoscopic endonasal transsphenoidal approach to large and giant pituitary adenomas: institutional experience and predictors of extent of

resection. J Neurosurg. 2014; 121:75–83

9. Barzaghi LR, Losa M, Giovanelli M, Mortini P. Complications of transsphenoidal surgery in patients with pituitary adenoma: experience at a single centre. Acta Neurochir; 2007. 149: 877–886

10. Paiva Neto MA, Vandergrift A, Fatemi N, et al. Endonasal transsphenoidal surgery and multimodality treatment for giant pituitary adenomas. Clinical Endocrinology. 2010; 72, 512–519

11. Koutourousiou M, Gardner PA, Fernandez-Miranda JC, Paluzzi A, Wang EW, Snyderman CH. Endoscopic endonasal surgery for giant pituitary adenomas: advantages and limitations. J Neurosurg . 2013; 118:621–631

12. Cusimano MD, Kan P, Nassiri F, et al. Outcomes of Surgically Treated Giant Pituitary Tumours. Can J Neurol Sci. 2012; 39: 446-457

13. Raikundalia MD, Pines MJ, Svider PF, et al. Characterization of transsphenoidal complications in patients with acromegaly: an analysis of inpatient data in the United States from 2002 to 2010. International Forum of Allergy & Rhinology. 2015; 5:

14. Nakao N, Itakura T. Surgical outcome of the endoscopic endonasal approach for non-functioning giant pituitary adenoma. J of Clinical Neuroscience. 2011; 18:71–75

15. Mortini P, Barzaghi R, Losa M, Boari M, Giovanelli M. Surgical Treatment of Giant Pituitary Adenomas: Strategies and Results in a Series of 95 Consecutive Patients. J Neurosurgery. 2007; 60:993–1004

16. Ho R, Huang H, Ho J. The Influence of Pituitary Adenoma Size on Vision and Visual Outcomes after Trans-Sphenoidal Adenectomy : A Report of 78 Cases. J Korean Neurosurg Soc. 2015; 57 (1) : 23-31

17. Komotar RJ, Starke RM, Raper DMS, Anand VK, Schwartz TH. Endoscopic endonasal compared with microscopic transsphenoidal and open transcranial resection of giant pituitary adenomas. J Pituitary. 2012; 15:150–159

18. Xue-Fei S, Yong-Fei W, Shit-Qi L, et al. Microsurgical treatment for giant and irregular pituitary adenomas in a series of 54 consecutive patients. British J of Neurosurgery. 2008; 22(5): 636–648

19. Sinha S, Sharma BS. Giant pituitary adenomas – An enigma revisited. Microsurgical treatment strategies and outcome in a series of 250 patients. British Journal of Neurosurgery. 2010; 24(1): 31–39

20. Sinha S, Sarkari A, Mahapatra AK, Sharma BS. Pediatric giant pituitary adenomas: are they different from adults? A clinical analysis of a series of 12 patients. Childs Nerv Syst. 2014; 30:1405–1411

21. Xiang Z, Anmin L, Shengyu Y, et al. Transsphenoidal microsurgical removal of large pituitary adenomas. Chinese Medical Journal. 1998; 111(11) 963-967

## **Boxing for Parkinson's Disease?**

Gary Simonds MD MHCDs, Cara Rogers DO, Chris Busch DO, Lisa Apfel MD, Harald Sontheimer PhD, Sean Simonds DPT  
Carilion Clinic, Roanoke, VA

### **A Novel Approach**

John is a 53 year-old former Marine and is who is becoming progressively debilitated by Parkinson's Disease. He manifests the illness in its full form- diffuse muscle rigidity; trouble initiating movement (bradykinesia); tremor so pronounced he cannot drink coffee from a cup lest he spill the contents all over himself; a "festinating" gait where he tends to shuffle faster and faster to keep his feet under a forward tipping body (postural instability). For the past two months, twice a week for 45 minutes, he has undergone boxing training with his Physical Therapist. First the heavy bag, then footwork, then a fast bag, and finally sparring. By the end of each session, he is more upright, his motions quicker and more fluid, balance more sound, the tremor dampened.

John is taking part in a novel approach to therapy for patients affected by Parkinson's Disease. A boxing program of all things. Patients are regularly put through the ropes of a vigorous boxing work out, and the majority show significant improvement in their symptoms. The improvement usually sustains itself for at least 3 to 5 days and then symptoms slowly return.

### **Elements of the Disease**

Parkinson's Disease (PD) involves systems within deep regions of the brain that are involved in modulation of movement (the basal ganglia and diencephalon). Beyond direct motor commands emanating from the primary motor cortex, the brain employs multiple parallel motor systems to effect movement. Such systems integrate intended movement commands with multiple other inputs in order to help make movement more smooth, fluid, and accurate. The basal ganglion/diencephalon structures constitute such a system that interfaces with the primary motor cortex, the premotor and supplemental motor regions, and other external inputs. The system acts somewhat like a graduated "brake" on initiation of movement. In Parkinson's Disease, critical neurons of the system die off in the Pars Compacta of the Substantia Nigra (in the midbrain). These neurons connect to the striatum (Caudate and Putamen) via the Nigrostriatal tract. They use dopamine as their neurotransmitter. Dopamine seems to affect the basal ganglion/diencephalic motor system by easing up or releasing the aforementioned "brake" on cortical motor commands. In a healthy being, the dopaminergic nigro-striatal system serves to "release the brakes" and allow initiation of movement. In PD, dopamine is significantly depleted in the basal ganglia, the brakes are left "on", and there results significant inhibition of movement.

Symptoms begin to manifest when approximately 80 percent of the dopaminergic neurons are lost from the system. Symptoms often develop first on one side of the body but eventually become bilateral. They include rigidity, bradykinesia, tremor, postural instability, and mask like facies (lack of expression). As the disease progresses it robs patients of their ability to get around and care for themselves with frequent falls and the need for significant support in

activities of daily living. Dopamine is also involved in various psychosocial functions of the brain. It is heavily represented in reward and reinforcement systems of the brain. Emotion homeostasis is often affected in PD with depression being very common. The majority of patients develop dementia late in the disease manifested with loss of executive function but not necessarily memory. Despite all this, patients tend to live to near normal limits.

It is not clear why the dopaminergic cells of the substantia nigra are subject to loss. There is a feeling that it may be less of a truly pathological process than it is an effect of aging in a vulnerable cell population. Dopamine metabolism and the pacemaker-like high-energy function of the substantia nigra neurons may result in an excess of harmful free-radicals within the cells. Free radical dynamics then may lead to cascades of other harmful processes. Pathologically, the cells build up alpha-synuclein in what are known as Lewy Bodies. Eventually, the majority of nigral striatal dopaminergic cells and connections are lost.

No treatment has been demonstrated to slow or obviate the process of PD although, ironically, cigarette smoking and caffeine seem to have some degree of protective effects. The bulk of current treatment is oriented to replenishing dopamine within the system (and generally throughout the brain). A dopamine precursor (Levodopa) is administered in a pharmaceutical form (Carbidopa) and is at first very effective in limiting the motor symptoms of the disease. Over time its effects lessen and patients experience “off periods” when the circulating level of Levopopa drops and PD symptoms intensify. In addition, iatrogenic persistent diffuse super-physiological levels of central dopamine (via administration of Levodopa) lead to other motor control side effects such as dyskinesias.

Another major form of treatment is the implantation of a deep brain stimulator. This involves surgically implanting an electrode into the basal ganglion or the diencephalon (the Globus Pallidus Interna or the Sub-thalamic Nucleus respectively). The electrode is driven by a distant pacemaker implanted just under the skin, usually in the chest. Through mechanisms not completely understood the passage of small electrical current into these regions of the brain tends to be very effective in controlling the motor symptoms (tremor, bradykinesia, rigidity) on the opposite side of the body. Thus, electrodes are often eventually placed on both sides of the brain.

Neither L-Dopa therapy or deep brain stimulator therapy prevents the gradual progression of PD. Motor symptoms tend to progress over time to the level of being debilitating. Postural instability leads to falls. Depression is common and dementia becomes an important component.

### **Exercise and Parkinson's Disease**

Of late, several studies have demonstrated that regular exercise in PD patients may carry symptomatic benefit. Various types of exercise have been invoked including weight training, dancing, Tai Chi and more. Multiple limb, high frequency exercise has been felt to be more effective. Benefits may be both PD-specific or general. PD specific benefits are manifested by improved motor function in all categories (tremor, rigidity, bradykinesia, postural stability).

These benefits may be secondary to increased diffuse cerebral dopamine release (or other related neurotransmitters) but is not clear. Repetitive cognitive rehearsal of diminished automated motor functions is thought to promote neuroplasticity at the synaptic and connectivity levels. General benefits include better conditioning, increased flexibility and range of motion, balance practice, and increased self-esteem. The last of these cannot be overestimated.

### **Boxing and Parkinson's Disease**

Boxing training seems in some ways an ideal form of exercise therapy for PD. It involves all extremities with rapid range of motion movements and rapid adjustments in balance and posture. Indeed, boxing therapy has been demonstrated to significantly improve all motor components in most patients with mild to moderate PD. What is more, it is fun, and positive emotional responses with increased self-esteem and diminished depression indices have been demonstrated amongst patients partaking in boxing therapy. Interestingly, benefits appear to last well beyond the exercise period. These benefits have been noted for up to three weeks after several sessions.

Sean Simonds is a Doctor of Physical Therapy who runs Specialized Physical Therapy of North Carolina, in Asheville N.C., and has treated over 150 Parkinson's patients using boxing as a principle therapy. He notes moderate to significant improvements in motor indices in the majority of his patients. He feels that the regimens can be worked into a patient's daily activities without a "coach" after several sessions. What has struck him beyond the motor effects however has been the profound emotional impact the program has had for his patients. People are often fooled into thinking PD patients lack engagement and commitment to their own care due to the blank facial expressions and slow movements so often encountered. What Dr. Simonds found however has been profound engagement, with patients begging for more vigorous work outs. He notes that several patients outlast him in sessions. What is more, the PD patients brim with pride from gaining physical skills through their own hard work. This as opposed to the sense of helplessness with the inexorable loss of skills, and the passivity of depending on medical and surgical therapies.

Dr. Simonds has noted improvement in the following indices in patients undergoing the PD Boxing Regimen, with improvements lasting as long as one-week post-session:

- Increased measured gait speed, step height, stride length
- Decreased freezing and festinating, decreased fall risk
- Improved TUG, BERG, and DGI (Functional Outcome Measures)
- Increased ABC Parkinson's Confidence Scale, increased reported confidence, decreased reported depression
- Increased reported willingness to go out in public/do hobbies/try new skills
- Increased facial expression and vocal abilities
- Improved measured ability to perform ADLs

The following are direct quotes from Parkinson's patients participating in the Boxing Sessions:

"Originally I came because my neurologist told me I had to, now, I'm going to beat Parkinson's."

"You have truly earned the nickname Sultan of Suffering, but I love you for it."

"I just went mountain biking for the first time since my diagnosis 10 years ago."

"Every time I come in here I get my butt kicked, but I'm never going to stop coming in. This is the best thing I've ever done."

"Nothing has helped me this much without negative side effects. This is the best medicine I've ever been prescribed."

"That's the first time I've smiled in a loooong time."

### **A Typical Boxing Session**

5- 10 minutes: Basic warm up: bike, treadmill, rower, and/or jump rope

20 minutes: Basic Boxing Skills/Speed Drills: boxing combos, focus mitts, speed bag, double end coordination bag, footwork, agility ladder, etc. no major breaks, full intensity

20 Minutes: Power Drills: heavy bag, plyometrics, max power hits, etc. no major breaks, full intensity

10 Minutes: Major Motor Work: Weightlifting, lunges, squats, chin ups, push-ups, etc.

10- 20 minutes: Stretch/cool down

### **References**

1. Ahlskog, Ph.D. M.D. J. Eric. Does vigorous exercise have neuroprotective effect in Parkinson's disease? American Academy of Neurology, Neurology 2011, pp 288-294, July 27, 2011.
2. Combs, Stephanie A., et. al. Boxing Training for Patients with Parkinson's Disease: A Case Series. Physical Therapy, Vol. 91-No. 1, pp. 1-11, January 2011
3. Hirsch, MA., et al. Exercise and neuroplasticity in persons living with Parkinson's disease. European Journal of Physical and Rehabilitation, Vol. 45-No. 2, pp. 215-228, June 2009.
4. Lin CH, Sullivan KJ, Wu AD, Kantak S, Winstein CJ. Effect of task practice order on motor skill learning in adults with Parkinson disease: a pilot study. Phys Ther. 2007 Sep;87(9):1120-31. Epub 2007 Jul 3.
5. Ridgel, Angela L., et al. Forced, not voluntary, exercise improves motor function in Parkinson's disease patients. Neurorehabilitation and Neural Repair, vol. 23-No. 6, pp. 600-608, July/August, 2009.
6. National Parkinson's Foundation. What role does exercise play in the management of PD?
7. Umphred, D. Neurological Rehabilitation. 5<sup>th</sup> ed. Mosby and Elsevier. 2007
8. Kornhuber, NH. Motor functions of cerebellum and basal ganglia: the cerebellocortical saccadic clock, the cerebellonuclear hold regulator, and the basal ganglia ramp (voluntary speed smooth movement) generator, Kybernetik 8:157, 1971.

9. Lisa M. Shulman, MD; Leslie I. Katzel, MD, PhD; Frederick M. Ivey, PhD; John D. Sorkin, MD, PhD; Knachelle Favors, MPH; Karen E. Anderson, MD; Barbara A. Smith, RN, PhD; Stephen G. Reich, MD; William J. Weiner, MD; Richard F. Macko, MD. Randomized Clinical Trial of 3 Types of Physical Exercise for Patients With Parkinson Disease. *JAMA Neurol.* 2013;70(2):183-190. doi:10.1001/jamaneurol.2013.646.
10. Laurie A King, Fay B Horak. Delaying Mobility Disability in People With Parkinson Disease Using a Sensorimotor Agility Exercise Program. *Physical Therapy.* DOI: 10.2522/ptj.20080214 April 2009
11. Dr Giselle M Petzinger, MD Beth Fisher, PhD, Sarah McEwen, PhD, Jeff A Beeler, PhD, John P Walsh, PhD, Michael W Jakowec, PhD. Exercise-enhanced neuroplasticity targeting motor and cognitive circuitry in Parkinson's disease. *The Lancet Neurology.* Volume 12, No. 7, p716–726, July 2013. DOI: [http://dx.doi.org/10.1016/S1474-4422\(13\)70123-6](http://dx.doi.org/10.1016/S1474-4422(13)70123-6)
12. Harald Sontheimer. *Diseases of the Nervous system.* Academic press. 2015









## **An Approach Using the Occipital Parietal Point (OPP) for Placement of Ventriculoperitoneal Catheters in Adults.**

Jason Duong, DO<sup>1</sup>, Dan Miulli DO<sup>1</sup>, Fanglong Dong, PhD<sup>2</sup>, Andrew Sumida MSIV<sup>3</sup>,

<sup>1</sup>Neurosurgery Department, Riverside University Health System

<sup>2</sup>Graduate College of Biomedical Sciences, Western University of Health Sciences

<sup>3</sup>College of Osteopathic Medicine of the Pacific, Western University of Health Sciences

**Keywords:** ventriculoperitoneal shunt, intracranial catheters, cranial approaches, occipital parietal point, keen's point, Frazier's point, hydrocephalus

### **Abstract**

**Introduction:** Ventriculoperitoneal shunts (VPS) have been widely used in the management of hydrocephalus. No general consensus on cranial entry points has been established for the adult population. We compare the known conventional posterior and frontal approaches with our proposed occipital parietal point (OPP) analyzing its associated outcomes.

**Methods:** An IRB approved retrospective chart review was conducted on patients at Arrowhead Regional Medical Center between 1999 and 2016. Patient's age, reasons for hydrocephalus, cranial entry points, and clinical outcomes (optimal placement, blood loss, OR time, malfunctions or infections) were abstracted. Chi-square analyses were conducted to identify the association between treatment and clinical outcomes.

**Results:** 93 adults ( $\geq 18$  years old) patients were included in the final analysis, average age was  $40.8 \pm 15.6$  years, with 57.0% had catheters placed utilizing the OPP, and 43.0% using conventional landmarks. OPP had less rates of suboptimal placement ( $p=0.0469$ ), and was less likely to develop a mechanical malfunction (5.7% vs 12.5%,  $p=0.2441$ ), though the difference was not statistically significant. External ventricular drains (EVD) prior conversion to an internalized VPS had increased risk for infection (11% versus 8 %, with  $P=0.650$ ) but due to our power was not statistically significant.

**Conclusion:** OPP can reduce the rates of catheter malposition, avoiding re-operations and its associated comorbidities. The OPP may not only be as safe as the conventional landmarks, but more optimal in long term outcomes. Utilizing the results to further characterize the natural history of adult VPS, future studies can investigate the pathological causes of hydrocephalus and its correlation with shunt failure and infection rates.

### **Introduction**

Ventricular shunting has been widely used in the management of hydrocephalus, especially seen within the pediatric population. This procedure diverts cerebrospinal fluid from the ventricular system and subarachnoid space to an alternative location i.e. peritoneum, pleural, or atrial space<sup>1</sup>. Each year there were 38,200–39,900 admissions, 391,000–433,000 hospital days, and total hospital charges of \$1.4–2.0 billion for pediatric hydrocephalus<sup>2</sup>, and while shunts have revolutionized the treatment of increased intracranial pressure, they also have their fair share of complications<sup>3,4</sup>. In the Shunt Design Trial, only 61% of patients did not have a shunt malfunction at 1 year follow up, and 47% at 2 year follow up<sup>5</sup>. This has led to continual investigation and research in determining ways to reduce the risks of shunt malfunction or infection. A post hoc analysis of data for risk factors for failure within the pediatric population

indicated that the etiology of the hydrocephalus, position of the ventricular catheter tip, and local environment of the catheter are most common<sup>6,7</sup>.

Ventricular catheter approaches have been investigated for optimal outcomes with mixed results. Shunts inserted via the frontal or anterior region functioned significantly longer than parietal inserted shunts,<sup>7</sup> while posterior approach groups 'survived' slightly longer without malfunction or becoming infected compared to anteriorly placed shunts<sup>8</sup>. Even to date, a randomized controlled trial of anterior versus posterior entry sites is being analyzed to reduce partial or full shunt replacements and its associated co-morbidities<sup>9</sup>.

While predictors of shunt malfunction have been mostly studied in the pediatric population, there is little literature addressing factors and outcomes with ventricular shunt systems in adult patients with hydrocephalus. Like pediatrics, no consensus regarding optimal intracranial catheter placement has been established. Therefore, this current study explores the pathology, rate of malfunction and infections of ventricular shunting system within the adult population. We also want to evaluate a proposed catheter approach at an occipital parietal point (OPP) by a single surgeon, and compare it to conventional posterior and frontal approaches.

Obstruction of the ventricular catheter is the most common reason for mechanical shunt failure in both pediatrics and adults<sup>10</sup>, and it can be caused by proteinaceous cerebrospinal fluid, debris, and more often obstruction by the ventricular wall or choroid plexus. Therefore, optimal catheter placement can reduce the rate of proximal failure<sup>11</sup>. While frameless stereotaxic or ultrasonography guidance of the catheter can reduce the rate of obstruction in adults<sup>12</sup>, we propose an approach using alternative anatomical landmarks with the free-hand technique to achieve similar results.

Traditionally used *anterior* (Kocher's), *posterior* (Frazier's), and *parietal* (Keen's) approaches for ventricular catheters have been utilized for entry and placement of the ventricular catheter. With the frontal approach, a bur hole is placed a Kocher's point which is found 1 cm anterior to the coronal suture, and 2 to 3 cm off to midline entering through the lateral horn to the foramen of Monro<sup>1</sup>. Frazier's point is 6 cm superior to the inion and 3 cm off midline, with the catheter approaching from a superior lateral to inferior midline direction. Finally, Keen's point is 3 cm above and 3 cm behind with the direction of the catheter aiming perpendicular to the cortex and slightly cephalic. Our proposed OPP is one half the distance of a line between Frazier's and Keen's point, where when measured on the CT scan is the most medial access point to the ventricles in a straight line and only 2 cm from the ependymal surface. The optimum length is premeasured on computed tomography or MRI of the brain.

## Methods

After obtaining institutional IRB approval, a retrospective chart review was conducted on patients who were admitted into Arrowhead Regional Medical Center (ARMC) between 1999 and 2016, a level II trauma center and acute care teaching facility located in San Bernardino County, CA<sup>13</sup>. Inclusion criteria included patients 18 years or older, that either had a new ventriculoperitoneal shunt placed or a proximal catheter revision. Patient data such as age,

intracranial pathology for hydrocephalus, intracranial catheters placed using the OPP versus conventional points, and associated clinical outcomes (optimal placement, blood loss, OR time, malfunctions or infections) were abstracted from the medical record. Suboptimal placement of ventricular catheters is crossing the septum pellucidum over midline, tip in contact with the ventricular walls or choroid plexus, or intraparenchymal placement. Optimal placement was ipsilateral to the site of entry or towards midline superior to the Foramen of Monroe. Chi-square analyses were conducted to identify the association between treatment and clinical outcomes.

## Results

A total of 93 adults ( $\geq 18$  years old) patients were included in the final analysis. The average age was  $40.8 \pm 15.6$  years, with half (57.0%) had catheters placed utilizing the OPP, and the other (43.0%) using conventional landmarks. Among patients who used the OPP, only 15.1% had suboptimal placement, which is significantly less frequently than the conventional landmarks (32.5%,  $p=0.0469$ ). Additionally, the OPP group was less likely to develop a mechanical malfunction (5.7% vs 12.5%,  $p=0.2441$ ), though the difference was not statistically significant. No statistically significant difference was detected on infection (9.4% vs 10%,  $p=0.9272$ ), median OR time (85 vs 85 minutes,  $p=0.5464$ ), and average blood loss volume (18.3 vs 26.3 ml,  $p=0.2037$ ) between the OPP and conventional landmarks. In evaluating our cohort, 45 had an external ventricular drains (EVD) prior conversion to an internalized VPS, and they also had increased risk for infection (11% versus 8 %, with  $P=0.650$ ) but due to our power was not statistically significant.

## Discussion

In adults, poor catheter placement was found to be the strongest predictor of shunt failure in a population of ages 0-91 with median age of 44<sup>14</sup>. Shunt malfunction tends to occur after migration of the catheter tip into the frontal brain substance as the ventricles decrease in size, or migration of the catheter itself towards the body of the ventricle into the choroid plexus<sup>3,7,15</sup>. Yamada SM et al. analyzed 52 patients and demonstrated that optimal shunt position to prevent proximal obstruction was to be within the anterior center of the lateral ventricle and above the foramen of Monroe<sup>16</sup>. Our study demonstrated that with freehand catheter placement using the OPP and freehand technique, the catheter was more likely to end in optimal positions than the conventional entry points (85% versus 67% respectively,  $p = 0.0469$ ).

The rate of shunt malfunction was also less compared to conventional entry sites (5.7% versus 12.5%,  $p = 0.2441$ ), however, not able to provide statistical significance due to our sample size. Shunt malfunction has a detrimental to the patient both clinically and financially, and to our national health care system. Hydrocephalus places the patient at the risk of severe disability, even death if left untreated in a timely manner. Shunting procedures then cost our national health care system more than 1 billion each year, where predominantly the costs arise from complication and revision rates<sup>17-19</sup>. Therefore, optimal positioning is a crucial step in reducing the associated risks of hydrocephalus as well as having a positive economic impact on healthcare.

Our rates of infection had no difference between the OPP and conventional landmarks, as well as OR time or average blood loss. As the only change in surgical technique is the location of catheter placement, we did not expect a drastic change from available literature in the adult population. Korinek et al. demonstrated that the male sex and previous external ventricular drains (EVD), revisions, CSF leaks, and longer operation times were risks of long term mechanical dysfunction and infection.<sup>10</sup> Our overall infection rate is 9.6%, consistent with current literature. In evaluating our cohort, 45 had an external ventricular drains (EVD) prior conversion to an internalized VPS, and they also had increased risk for infection (11% versus 8 %, with  $P=0.650$ ) but due to our power was not statistically significant. With infection-related cost per 100 de novo shunts placed was \$162,659 in adult patients<sup>20</sup>, additional measures can be explored to relieve that financial burden.

The majority of adult patients who required a VPS were due to trauma (25.8%) causing communicating hydrocephalus, which included traumatic subarachnoid hemorrhages, intraparenchymal subdural, epidural hemorrhages, gunshot wounds, and those who required a decompressive craniectomy for refractory intracranial hypertension. The next common causes of hydrocephalus requiring VPS in our adult population are tumors (17.2%) then followed by intraventricular hemorrhages (8.6%).

Being a retrospective review, a disadvantage we can run into is the selection bias due to the inability to achieve randomization. The OPP is selected and preferred by a single neurosurgeon, but as a teaching institution even with guidance, the operative experience can vary by the lead senior resident physician at the time. VPS placed with other entry points are performed by more than one neurosurgeon which was not controlled due to the retrospective nature. Certain exposures or extent of degenerative pathologies may not have been able to be controlled.

## **Conclusion**

We demonstrated that utilizing the OPP can reduce the rates of catheter malposition, essentially avoiding future operations and its associated comorbidities reducing personal and financial burdens. The technique can be performed freehand without the need of stereotactic guidance, reducing operating time and additional costs. Also, we found that risks of shunt infection are increased to having an EVD in place prior, consistent with previous literature. Our review was also able to further provide additional data on the natural history of VPS in adults, raising areas that can further be investigated.

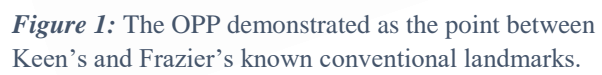
## References

1. Badhiwala JH, Kulkarni AV. Ventricular Shunting Procedures. In: Winn H, ed. *Youmans and Winn Neurological Surgery*. 4th ed. Elsevier; 2016:1615-1629.
2. Simon TD, Riva-Cambrin J, Srivastava R, et al. Hospital care for children with hydrocephalus in the United States: utilization, charges, comorbidities, and deaths. *J Neurosurg Pediatr* 2008;1(2):131-137. doi:10.3171/PED/2008/1/2/131.
3. Sekhar LN, Moossy J, Guthkelch AN. Malfunctioning ventriculoperitoneal shunts. Clinical and pathological features. *J Neurosurg* 1982;56(3):411-416. doi:10.3171/jns.1982.56.3.0411.
4. Colak A, Albright AL, Pollack IF. Follow-up of children with shunted hydrocephalus. *Pediatr Neurosurg* 1997;27(4):208-210.
5. Drake JM, Kestle JR, Milner R, et al. Randomized trial of cerebrospinal fluid shunt valve design in pediatric hydrocephalus. *Neurosurgery* 1998;43(2):294-303; discussion 303.
6. Drake JM, Kestle JR, Tuli S. CSF shunts 50 years on--past, present and future. *Childs Nerv Syst* 2000;16(10-11):800-804. doi:10.1007/s003810000351.
7. Albright AL, Haines SJ, Taylor FH. Function of parietal and frontal shunts in childhood hydrocephalus. *J Neurosurg* 1988;69(6):883-886. doi:10.3171/jns.1988.69.6.0883.
8. Bierbrauer KS, Storrs BB, McLone DG, Tomita T, Dauser R. A prospective, randomized study of shunt function and infections as a function of shunt placement. *Pediatr Neurosurg* 1990;16(6):287-291.
9. Whitehead W. A Randomized Controlled Trial of Anterior Versus Posterior Entry Site for Cerebrospinal Fluid Shunt Insertion | PCORI. *Patient-Centered Outcomes Research Institute*. Available at: <http://www.pcori.org/research-results/2014/randomized-controlled-trial-anterior-versus-posterior-entry-site-cerebrospinal>. Accessed April 25, 2017.
10. Korinek A-M, Fulla-Oller L, Boch A-L, Golmard J-L, Hadji B, Puybasset L. Morbidity of ventricular cerebrospinal fluid shunt surgery in adults: an 8-year study. *Neurosurgery* 2011;68(4):985-94; discussion 994. doi:10.1227/NEU.0b013e318208f360.
11. Villavicencio AT, Leveque J-C, McGirt MJ, Hopkins JS, Fuchs HE, George TM. Comparison of revision rates following endoscopically versus nonendoscopically placed ventricular shunt catheters. *Surg Neurol* 2003;59(5):375-9; discussion 379.
12. Wilson TJ, McCoy KE, Al-Holou WN, Molina SL, Smyth MD, Sullivan SE. Comparison of the accuracy and proximal shunt failure rate of freehand placement versus intraoperative guidance in parietooccipital ventricular catheter placement. *Neurosurg Focus* 2016;41(3):E10. doi:10.3171/2016.5.FOCUS16159.
13. Arrowhead Regional Medical Center. *Arrowhead Regional Medical Center*. Available at: <https://www.arrowheadmedcenter.org/>. Accessed May 31, 2017.
14. Jeremiah KJ, Cherry CL, Wan KR, Toy JA, Wolfe R, Danks RA. Choice of valve type and poor ventricular catheter placement: Modifiable factors associated with ventriculoperitoneal shunt failure. *J Clin Neurosci* 2016;27:95-98. doi:10.1016/j.jocn.2015.07.026.
15. Whitehead WE, Riva-Cambrin J, Kulkarni AV, et al. Ventricular catheter entry site and not catheter tip location predicts shunt survival: a secondary analysis of 3 large pediatric

hydrocephalus studies. *J Neurosurg Pediatr* 2017;19(2):157-167.  
doi:10.3171/2016.8.PEDS16229.

16. Yamada SM, Kitagawa R, Teramoto A. Relationship of the location of the ventricular catheter tip and function of the ventriculoperitoneal shunt. *J Clin Neurosci* 2013;20(1):99-101. doi:10.1016/j.jocn.2012.01.041.
17. Stone JJ, Walker CT, Jacobson M, Phillips V, Silberstein HJ. Revision rate of pediatric ventriculoperitoneal shunts after 15 years. *J Neurosurg Pediatr* 2013;11(1):15-19. doi:10.3171/2012.9.PEDS1298.
18. Pham ACQ, Fan C, Owler BK. Treating pediatric hydrocephalus in Australia: a 3-year hospital-based cost analysis and comparison with other studies. *J Neurosurg Pediatr* 2013;11(4):398-401. doi:10.3171/2013.1.PEDS12233.
19. Shannon CN, Simon TD, Reed GT, et al. The economic impact of ventriculoperitoneal shunt failure. *J Neurosurg Pediatr* 2011;8(6):593-599. doi:10.3171/2011.9.PEDS11192.
20. Parker SL, McGirt MJ, Murphy JA, Megerian JT, Stout M, Engelhart L. Cost savings associated with antibiotic-impregnated shunt catheters in the treatment of adult and pediatric hydrocephalus. *World Neurosurg* 2015;83(3):382-386. doi:10.1016/j.wneu.2014.06.010.





**Figure 1:** The OPP demonstrated as the point between Keen’s and Frazier’s known conventional landmarks.

## **fMRI in the Courtroom**

Gary Simonds MD MHCDs, Cara Rogers DO, Chris Busch DO, Michael Benko DO, Zev Elias MD  
Carilion Clinic, Roanoke, VA

### **Tom Cruise- Neuroscientist**

Imagine a movie courtroom scene where the villain has bribed a series of witnesses to lie on his behalf in a murder trial. It is clear he is literally going to get away with murder. But in walks a bespectacled Tom Cruise- the devilishly handsome yet cerebral forensic neurosurgeon-neuroscientist. He takes the stand and proceeds to tell the Judge and jury that his fMRI studies of the defendant demonstrate that he fully recognized images of the crime scene, although the defendant completely denied he had ever laid foot there. The fMRI also demonstrated an off-the-scale emotional attachment to the murder weapon. It also demonstrated a semantic and visual processing sequences that were 100% consistent with murderers and not innocent people. The processing also indicated that the defendant was likely to kill again. Finally, fMRI testing of all the defendant's alibi witnesses demonstrated that to a man, they were all openly lying. The villain leaps across the table, grabs the bailiff's gun and shoots Cruise. The filming slows down to a speed that you can see the bullet in flight speeding towards our intrepid neurosurgeon-neuroscientist as he contorts his body Matrix-like to avoid its path. Well, you can take it from here....

Far-fetched? Perhaps not. Momentum is gaining for the employment of fMRI within the American criminal legal system. Such usage of what is still clearly an inexact assessor of cerebral function is almost inevitable to one degree or another. In such, it opens up a series of issues- some for neuroscientists to ponder, some for legal scholars, and some for philosophers.

### **The Unreliability of Memory**

Legal standards for conviction of a crime depend on multiple factors, not the least of which is "proof" that the defendant indeed committed the crime, followed by the fact that the defendant made a choice to commit the crime. Forgive us, lawyers out there, for perhaps stating this awkwardly, we do recognize that the second component mentioned here is very vague and opens up a can of worms but is useful for further discussions in this piece- so please don't sue us!

Proof that someone committed the crime must be to the level of being "Beyond a Reasonable Doubt" or at least to a level of being supported by a "Preponderance of Evidence", and here lies the rub. These standards can be very hard to meet. Traditionally, multiple convergent eye witness accounts of a crime have been a gold standard, short of an un-tampered videos. Reliability of memory streams, particularly in high-stress situations has, however, been demonstrated to be extremely questionable. This is evident in the overturning of hundreds of previous eye-witness based convictions in the past couple of decades by DNA evidence.

### **Science to the Rescue**

Thus if there is a dearth of video capture of a crime, and eye-witness accounts are unreliable, who can the criminal justice system turn to? The answer more and more is Science. Science is

asked to deliver incontrovertible evidence in the courtroom, because so many other methods have proven to be undependable- particularly if they are predicated on individual human testimony. Science is seen as the ultimate impartial arbiter. Science isn't affected by flaws in memory and malfeasance. This is not a new concept. Scientific measures have been heavily employed in evidence collection and processing for many decades – think of the evolution of crime scene investigation. In fact, many TV shows depicting the process place half the action within laboratories with requisite bubbling Erlenmeyer Flasks and electron microscopes.

Admission of scientific evidence is based on a principle known as the “Daubert Standard.” Loosely this requires scientific evidence to be testable and reproducible, to have a definable error rate, to have been subject to peer review, to have associated standards and controls in its execution, and to be accepted by the scientific community. Furthermore, scientific evidence must be both reliable and relevant. “Scientific evidence” in criminal law, however has proven to be fallible and periodically its “sacred cows” have come to be debunked and rendered inadmissible- witness for example the judicial fate of bite mark evaluations and polygraphs.

### **The Promise of fMRI**

Like Tom Cruise in the scenario depicted above, onto the scene struts functional magnetic resonance imaging (fMRI). Two decades of study employing the technology has led to a wealth of literature on cerebral function and processing. Patterns of cerebral activation have led to significantly improved understanding of how the brain works and, more importantly perhaps for this discussion, how it dysfunctions in various disease and disorder states. Furthermore, neuroscientists are probing the heretofore uncharted universe of what the brain is actually thinking. Cerebral circuitry involved in various functions is implicated by detection of increased blood flow to various regions. Presumably increased blood flow indicated increased activity in (or use of) these regions.

Now, think of the legal ramifications. What if we can assess the validity of eyewitness testimony? What if we can tell exactly what a defendant is thinking with regards to a crime? What if we can tell for sure whether he or she is lying? What if we can tell if a defendant had no choice but to commit a crime, or had no way to control an impulse to commit such crime? What if we could tell whether a convicted criminal was destined to commit a crime again? The impact of definitive neuro-scientific evidence to the criminal justice system is difficult to fully fathom- both for the prosecution and the defense. It's premature infiltration of the system is highly predictable.

### **The Modern Polygraph**

A wealth of study has demonstrated relatively reproducible detection of willful lying on fMRI testing. Rather than evaluating a subject's physiological response to lying (as in a polygraph), fMRI actually captures the brain's creation of a lie, and it appears that it does so in a somewhat stereotyped manner. If this could be broadly applied in criminal law, imagine the ramifications. Find a suspect for a crime, ask them if they committed it under fMRI, and voila- a conviction!

There may be implications for the civil court as well. Besides determining lies, researchers feel they can define pain and suffering via fMRI. Imagine a car-accident lawsuit- the plaintiff claims horrific pain and suffering but the fMRI shows no evidence of either- voila, a huge reduction in settlement!

Thus far, the study of lying in the laboratory has been very ordered, controlled, and contrived. The field of lying out in the real world of normal human interaction, let alone criminal, has got to be like the wild wild west- think of a suspect combining a series of truths with one lie. And what about the suspect who has come to believe their own lies? With mega-data assessment of finer and finer cerebral processing, perhaps definitive patterns will be identified, but for now study of lie detection has to be akin to the evaluation of a single wildebeest in a zoo as opposed to one in a wide open ecosystem out on the Serengeti. Yet, our current ability to determine whether a suspect or witness is lying without fMRI is essentially zero. As one forensic neuroscientist put it “any reliable hint that a subject is lying would be a big step in the right direction.”

### **Criminal Memory**

Let’s keep peering into the future by exploring a subject’s recollection of the past. What if we could detect the reliability of an eye witness’s account? Neuroscientists using fMRI can detect in subjects whether they have seen a particular image or object in the recent past. What if refinement of the technology could lead to reliable identification of recognition in a suspect of a crime scene or murder weapon. What if we could tell without a doubt that a witness is picking the correct suspect out of a police line-up? What if an alibi could be disproven by lack of recognition/response to a place, an activity, a conversation that had allegedly taken place?

### **Predisposition and Mitigating Circumstances**

OK, a suspect is definitively proven to have committed a crime. Could there be mitigating circumstances? Was the suspect capable of comprehending the ramifications of his or her actions? Is he or she even competent to stand trial? Could he/she control his/her impulses? There is a wealth of fMRI study of brain function and processing in psychological and functional disorders. Researchers feel more and more comfortable that they can identify disorders such as Schizophrenia and Sociopathy with great reliability using fMRI. Various types of processing are significantly and reproducibly distorted. Some researchers feel therefore that a psychopath can be identified by fMRI alone, and that their brain function is significantly impaired- unable to process empathy, unable to control impulses.

What are the legal ramifications of a “physiological” definition of disordered processing in a criminal? fMRI has already been entered into criminal sentencing testimony in the hopes of affecting sentencing. Should fMRI evidence be allowed to mitigate a crime? Should it result in modified sentencing? Should the conviction be overturned? Should the suspect receive treatment rather than imprisonment? Could a court order treatment?

See the collision between the scientific and the legal worlds?

## **Mind Reading?**

Take the last concept one step further. Say we know a convict is a sociopath, that he has no empathy, that he has no violent behavior impulse control. Say we know with little doubt that he will commit his crime again. What do we do? What might come into play if fMRI could tell us relatively reliably about a convicted criminal's likelihood of recidivism? Would we/should we block the criminal from ever re-entering society? Would we need to create a new "Devil's Island" for such definitive "pre-recidivists?"

Let's go even further. What if we can read a witness's or a suspect's mind? Researchers have crudely reconstructed pictures and even movies recently viewed by subjects using fMRI. How credible then would it be to someday reconstruct a crime scene from the mind of a suspect or a witness? Could remote memories someday be called up and viewed by criminal investigators, Judges and juries? Oh heck, let's go one step further- could we peer into the mind of a criminal and see if he is planning a crime yet to happen? And that, brings us back to Tom Cruise movies doesn't it?

## **Current Use**

Burned by decades of acceptance of polygraphs in the courtroom, there has definitely been some reticence in allowing fMRI testimony in the courtroom. But admissibility is regional, dependent on the local system and Judges, and fMRI evidence has been employed in cases throughout the country. Indeed, there are businesses that offer fMRI scanning to defendants to assist in their cases. Increased employment of the technology particularly on the defendant side is inevitable. This is problematic in that the reliability "in vivo" of fMRI study is quite questionable. Furthermore, studies have demonstrated that lay people (i.e. juries) are quite susceptible to neuroscientist testimony. Thus, entry of fMRI evidence as presented by an "expert" neuroscientist may have disproportionate influence on a jury even if the evidence is rather insubstantial.

## **Current Pitfalls**

The employment of fMRI in the modern courtroom is problematic. Most studies of brain function and processing via fMRI are necessarily very controlled and contrived in order to limit variables. Unlimited variables are liable to create nothing but noise, yet the world outside the "torpedo tube" of the 3 tesla (or more) MRI is a jumble of a million stimuli, thoughts, decisions, incoming data, out-going actions, emotional supercharging, interruptions, multi-tasking and the like. What is more, data generated from fMRI is collated and aggregated to obtain average functional responses. The criminal justice system generally is focused on one individual. Translation of the general to the individual is fraught with potential inaccuracies. Furthermore, at its current status fMRI may tell us something about the subject's current cerebral function but it cannot tell us anything about its function at the time of the crime.

Another critique is that current fMRI fidelity is still very regional and gross (within the brain), whereas cerebral functional connectivity is microscopic and hyper-complex. Real understanding therefore of processing may still be a long way from our grasp. We generate vague understandings of processing, but are only really scratching the surface. This is illustrated by the

significant overlap of brain regions invoked for various physiological and pathological functions. This has allowed some critics to call fMRI science “the new phrenology.” Furthermore, statistical algorithms employed in fMRI study can be rather gross. This was satirized by an infamous report of an fMRI evaluation of the brain of a dead salmon. The dead fish was “shown” pictures and asked to describe its emotional response. fMRI demonstrated a cerebral activation pattern.

Forensic employment of fMRI is further complicated by the fact that it can be “gamed.” Researchers have been able to significantly disrupt evaluations of multiple functions by training their attentions or thoughts on functions other than what is being tested. For example, in studies testing for recognition of an object, typical activation patterns were disrupted by the subject concentrating on a single component of the image screen. In other studies, micromovements of a finger disrupted tested activation patterns. Surely other patterns of gaming the process will be figured out by the always resourceful criminal element, and broadly disseminated within its fraternity.

After all, we are dealing with mentation here. We may tell a subject what we want them to think or focus upon, but we thus far cannot control what they are doing on the inside. They may purposefully create thoughts and memories that block the real ones, or act as red herrings, or create new internal realities. We could indeed find it rather difficult to discern between the intentionally created internal reality of a crafty criminal and that of an innocent suspect. What is more, remember that memory traces are notoriously imprecise. What about the criminal or the witness whose memory traces become more and more distorted by time, television, discussion, emotion, and questioning session after questioning session? The criminal and the witness may develop a memory stream that they completely believe, and yet is false. How will an fMRI discern between truth and lie when the subject remembers only a distorted reality? Is this truly a case of perception being reality?

### **Conscience and Predeterminism**

A final fascinating and perhaps frightening note on this subject is the philosophical ramifications of fMRI and the law. If we feel that we can reliably track cerebral function (and dysfunction) to the point that we can detect lies, track experiences, and perhaps predict behaviors; and if we feel we can understand the circuitry of bad behavior (criminal in this case) and absolve certain individuals from consequences because they had no real choice in their selection of said behavior; aren't we surrendering to a neurobiochemical predeterminism of human behavior in general? We are faced again with the insoluble questions of what is consciousness and then what is conscience (both I presume required to hold anyone accountable for their actions)? If all human behavior is predetermined by our circuitry and its activation how is there innocence or guilt in any action? How does law apply? What is the Law's role and purpose? Who determines it, and based on what principles? Who should be held accountable and who should not be? Taken a step further to the other end of the spectrum- what about good behavior? Why should industry and responsibility and dedication and kindness and leadership and thoughtfulness be celebrated or rewarded above any other behavior or lack thereof?

We plan to go to the movies and see if indeed Tom Cruise has the answers.

## References

1. Will Neuroscience Radically Transform the Legal System? Hank Greely. Slate
2. Daniel D. Langleben and Jane Campbell Moriarty. Using Brain Imaging for Lie Detection: Where Science, Law and Research Policy Collide. *Psychol Public Policy Law*. 2013 May 1; 19(2): 222–234. Published online 2012 Sep 17. doi: 10.1037/a0028841 PMID: PMC3680134 NIHMSID: NIHMS409229
3. Science in court: Head case. Virginia Hughes. *Nature* 464, 340-342 (2010) | doi:10.1038/464340a
4. Deceiving the Law, Editorial. *Nature Neuroscience*. <https://www.nature.com/articles/nn1108-1231>
5. Anthony Wagner, Richard J. Bonnie, BJ Casey, Andre Davis, David L. Faigman, Morris B. Hoffman, Owen D. Jones, Read Montague, Stephen J. Morse, Marcus E. Raichle, Jennifer A. Richeson, Elizabeth S. Scott, Laurence Steinberg, Kim Taylor-Thompson, Gideon Yaffe. fMRI and Lie Detection A Knowledge Brief of the MacArthur Foundation Research Network on Law and Neuroscience (2016). <http://static.vtc.vt.edu/media/documents/SSRN-id2881586.pdf>
6. Weighing the Admissibility of fMRI Technology Under FRE 403: For the Law, fMRI Changes Everything – and Nothing. Justin Amirian. *Fordham Urban Law Journal* Volume 41, Number 2 2015 Article 1 SPECIAL EDUCATION IN URBAN SCHOOLS: IDEAS FOR A CHANGING LANDSCAPE
7. Neurolaw. Wikipedia
8. Joshua Buckholz, David Faigman. Promises, promises for neuroscience and law. *Current Biology* Volume 24, Issue 18, 22 September 2014, Pages R861- 867 <https://doi.org/10.1016/j.cub.2014.07.057>
9. Craig M. Bennett, Abigail A. Baird, Michael B. Miller, and George L. Wolford. Neural correlates of interspecies perspective taking in the post-mortem Atlantic Salmon: An argument for multiple comparisons correction. <http://prefrontal.org/files/posters/Bennett-Salmon-2009.pdf>

## Retro Odontoid Pseudo Tumor with Cervical Medullary Compression: A Case Report

Samer Elfallal DO and Wissam Elfallal DO

Beaumont Hospital, Trenton, MI

### Introduction

The disease entity of retro odontoid pseudo tumor may over time enlarge and cause cervical medullary compression leading to a progressive myelopathy from extrinsic compression.<sup>1</sup> This mass may also be known as an odontoid pannus or phantom tumor.<sup>2</sup> In rare instances there may be development of pannus-related cysts that may lead to further compression of the upper spinal cord and lower medulla, as was seen in our case. In the past prior authors have suggested treatment through a trans oral approach with direct removal and decompression.<sup>3</sup> In recent literature, more surgeons have been trending towards upper cervical spinal fusion with or without laminectomy for indirect decompression and treatment of the pannus.<sup>4</sup>

### Case Report

A 97-year-old man presented to our neurosurgical service with an inability to ambulate, global paresis of upper and lower extremity, atrophy of the intrinsic hand muscles with long track signs noted in the upper extremity. After work-up was completed, the patient was found to have large odontoid pannus causing compression at the cranial cervical junction. We also noted a cyst associated with this pannus causing significant mass effect on the medulla leading to a T2 hyperintense signal at the medulla consistent with myelomalacia (figure 1). Patient had diffuse spondylosis of the cervical spine along with central stenosis at the 4-5 and 5-6 level. MRI of brain and CT angiogram were obtained and were unrevealing for other pathology. The posterior odontoid mass was found to be due to his rheumatoid arthritis.

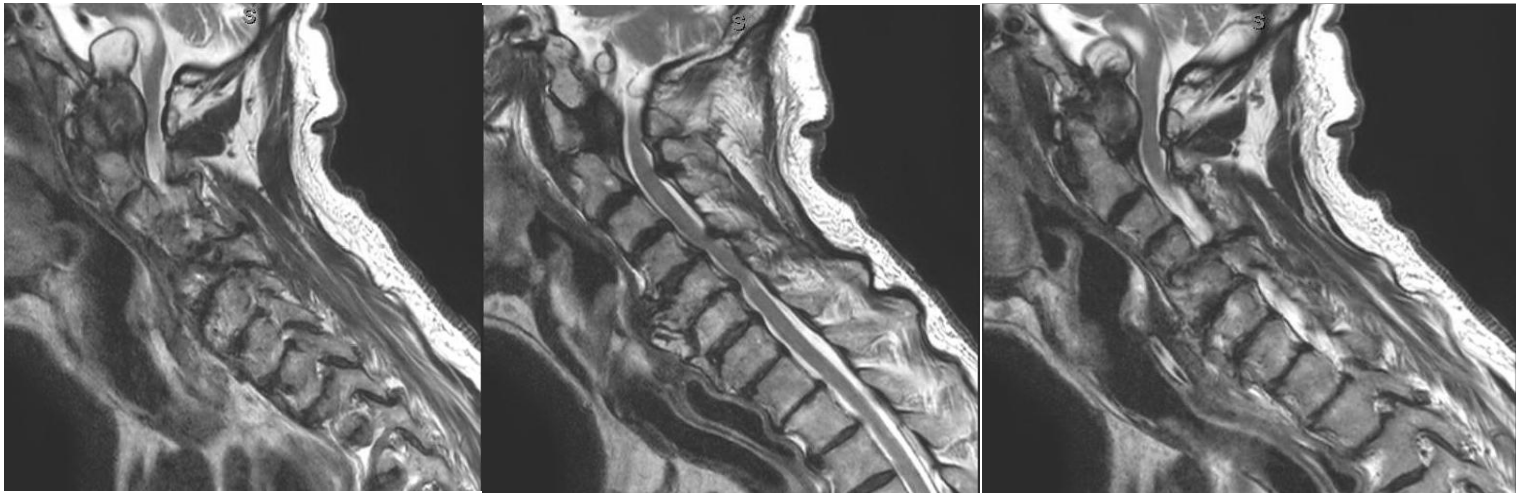


Figure 1: pre-operative MRI A. sagittal T2 with cystic component with medullary compression. B. T2 sagittal MRI showing spondylosis and central stenosis from C4-6. C. T2 sagittal with odontoid pannus.



## Interventions

After discussion with family, it was decided that the patient would undergo posterior cervical arthrodesis with instrumented fixation from C1-6 with decompressive laminectomy from C4-6 to treat associated stenosis. Intraoperative neuro monitoring was utilized along with fluoroscopy for localization and lateral mass screw placement. No signal changes were noted intraoperatively and patient was successfully extubated. Post-operative x ray is provided (figure 2). Patient was placed in our ICU for post-operative monitoring. Patient tolerated procedure well and his hospital course was uncomplicated. He was sent for a short course of inpatient rehabilitation.

## Outcome

Patient was followed up for two years. There was improvement in the patient's clinical symptoms of motor weakness and he was eventually able to ambulate without assisted device. The patient tolerated the procedure without any associated surgical or medical complications. Radiographic evaluation at the two-year mark showed marked reduction in the pannus and associated cyst with resolution of T2 hyperintense signal within the medulla (figure 2). To the best of our knowledge there are no reports of such a large odontoid pannus with an associated cyst and medullary compression.

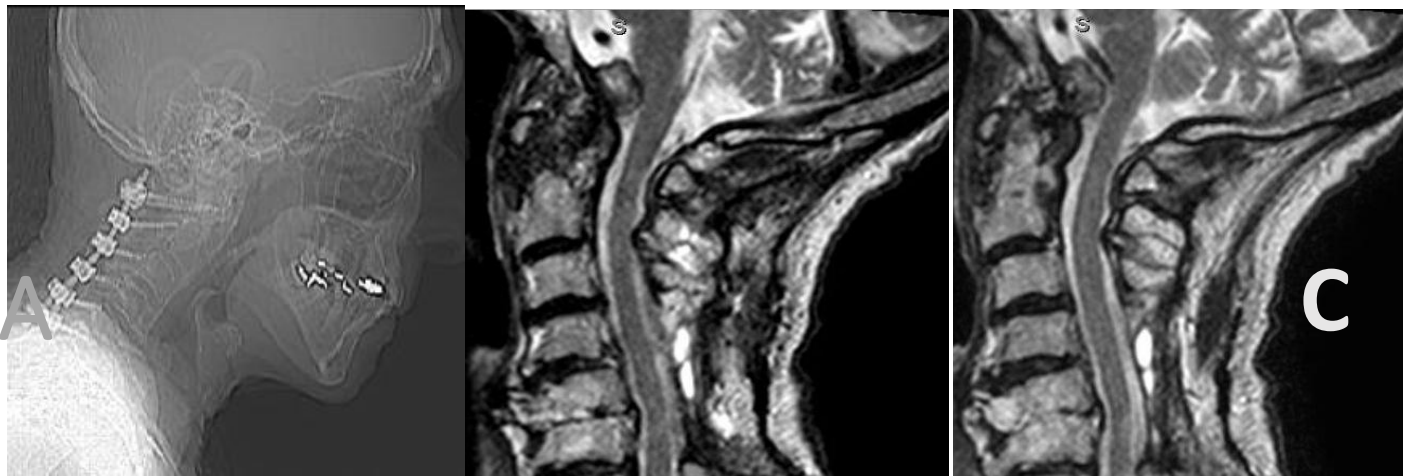


Figure 2: A. x-ray showing C1-6 posterior fusion. B. T2 sagittal MRI showing reduction in cyst size and resolution of signal within medulla. C. T2 MRI showing odontoid process with reduction in pannus size.

## Discussion

Retro odontoid pseudo tumor or odontoid pannus can be seen with inflammatory disease entities such as rheumatoid or psoriatic arthritis, and in the non-inflammatory settings, for example chronic dialysis, post-traumatic pseudo arthrosis, and degenerative disease.<sup>4</sup> The primary proposed mechanism is chronic atlanto-axial instability which leads to the development of this pannus. This produces an inflammatory process with fibrocartilaginous mass formation in the posterior odontoid space which over time can lead to direct spinal cord compression.<sup>5</sup> However, there are a few instances where no atlantoaxial instability is noted on

imaging and yet there still may be development of this mass lesion.<sup>6</sup> In our case, the patient had an underlying inflammatory spondyloarthropathy, but was under no active treatment. In general, overall incidence is not known but a recent 11-year case series review found that incidence of retro odontoid pannus in patients with symptomatic atlanto-axial instability with associated risk factors including rheumatoid arthritis, os-odontoideum, dens fracture and Morquio syndrome was 23.2% .<sup>7</sup>

In the past, authors have recommended a direct form of decompression with removal of the odontoid pannus. Surgical treatments included anterior trans oral odontoidectomy and posterior transdural resection.<sup>8</sup> Recently, it has been proposed to surgically fuse the posterior spine, primarily at the C1 to C2 junction. Authors have reported good radiographic and clinical outcomes that have obviated the need for a transoral anterior approach.<sup>4</sup> In our case, fusion not only included C1 and C2, but was extended to the lateral masses of C6. Decompressive laminectomy was completed at the C4 to 6 levels to treat the associated central canal stenosis. Our surgical intervention showed good outcome with resolution of the pannus compression on the medulla and radiographic reduction of the mass. The use of C1-2 fusion is a known safe treatment for atlanto-axial instability and has also been extended as a treatment for painful arthritis.<sup>9</sup> Some authors have recommended occipital cervical fusion, to treat associated occipital cervical instability and adjacent segment disease. This indirect treatment also lead to radiographic improvement with good functional outcome.<sup>5, 10</sup> There is also literature supporting the use of C1 laminectomy for indirect decompression and in some cases it lead to reduction in pannus size. This strategy may be useful in cases where patients may not tolerate prolonged surgeries with larger blood loss and lengthy anesthesia exposure.<sup>11</sup> Concern with completion of C1 laminectomy without fusion may lead to further atlanto-axial instability, but a reduction in neurologic symptoms can be feasibly obtained without progressive instability in most cases.<sup>12</sup>

## **Conclusion**

Retro odontoid pseudo tumors or odontoid pannus is a disease process characterized by formation of a posterior mass with possible cystic formation, which may lead to marked compression on vital neuronal structures at the cervico-cranial junction. Certain risk factors have been associated with an odontoid pannus including rheumatoid arthritis, Os odontoneum, or malunion of odontoid fracture. The basis of this disease process is believed to be due to atlanto-axial instability. However, this may not be true for all cases, since it has been found that indirect techniques in the form of C1 laminectomy have helped reduce pannus size. In our case, we proposed cervical fusion and avoided a transoral approach due to our patient's comorbidities and inability to tolerate this procedure. After our two-year follow-up, the pannus and associated cyst had significant reduction with resolution of cord signal change within the medulla. We support the use of posterior fixation for treatment of this disease process. Further investigation in the pathophysiology of this disease process to help create a more unified treatment approach is needed.

## References

1. Sze G, Brant-Zawadzki MN, Wilson CR, Norman D, Newton TH. Pseudotumor of the craniovertebral junction associated with chronic subluxation: MR imaging studies. *Radiology*. 1986;161:391–394.
2. Suetsuna F, Narita H, Ono A, Ohishi H. Regression of retroodontoid pseudotumors following C1 laminoplasty. *J Neurosurg Spine*. 2006;5:455–460. doi: 10.3171/spi.2006.5.5.455.
3. Crockard HA, Pozo JL, Ransford AO, Stevens JM, Kendall BE, Essigman WK. Transoral decompression and posterior fusion for rheumatoid atlanto-axial subluxation. *J Bone Joint Surg Br*. 1986;68:350–356.
4. Barbagallo GMV, Certo F, Visocchi M, Palmucci S, Sciacca G, Albanese V. Disappearance of degenerative, non-inflammatory, retro-odontoid pseudotumor following posterior C1–C2 fixation: case series and review of the literature. *Eur Spine J*. 2013;22(Suppl 6):879–888. doi:10.1007/s00586-013-3004-1.
5. Chikuda H, Seichi A, Takeshita K, et al. Radiographic analysis of the cervical spine in patients with retro-odontoid pseudotumors. *Spine (Phila Pa 1976)*. 2009;34:E110–E114.
6. Kakutani K, Doita M, Yoshikawa M, et al. C1 laminectomy for retro-odontoid pseudotumor without atlantoaxial subluxation: review of seven consecutive cases. *Eur Spine J*. 2013;22(5):1119–1126. doi:10.1007/s00586-013-2681-0.
7. Park JH, Lee E, Lee JW, et al. Postoperative regression of retro-odontoid pseudotumor after atlantoaxial posterior fixation: 11 years of experience in patients with atlantoaxial instability. *Spine (Phila Pa 1976)*. 2017 Apr. doi: 10.1097/BRS.0000000000002222. [Epub ahead of print]
8. Fujiwara Y, Manabe H, Sumida T, Tanaka N, Hamasaki T. Microscopic Posterior Transdural Resection of Cervical Retro-Odontoid Pseudotumors. *Journal of Spinal Disord Tech*. 2015;28(10):363–369. doi:10.1097/BSD.0000000000000335.
9. Payer M, Luzi M, Tessitore E. Posterior atlanto-axial fixation with polyaxial C1 lateral mass screws and C2 pars screws. *Acta Neurochir (Wien)*. 2009 Mar;151(3):223–9; discussion 229. doi: 10.1007/s00701-009-0198-4.
10. Landi A, Marotta N, Morselli C, Maronqui A, Delfini R. Pannus regression after posterior decompression and occipito-cervical fixation in occipito-atlanto-axial instability due to rheumatoid arthritis: case report and literature review. *Clin Neurol Neurosurg*. 2013 Feb;115(2):111–6. doi: 10.1016/j.clineuro.2012.04.018.
11. Kakutani K, Doita M, Yoshikawa M, et al. C1 laminectomy for retro-odontoid pseudotumor without atlantoaxial subluxation: review of seven consecutive cases. *Eur Spine J*. 2013;22(5):1119–1126. doi:10.1007/s00586-013-2681-0.
12. Takemoto M, Neo M, Fujibayashi S, et al. Clinical and Radiographic Outcomes of C-1 Laminectomy Without Fusion in Patients with Cervical Myelopathy that is Associated with a Retro-odontoid Pseudotumor. *Clin Spine Surg*. 2016 May 18. [Epub ahead of print]

## **Put Down the Weights! The Effects of Gross motor Activity on Surgical Dexterity**

Aaron Danison DO, Cara Rogers DO, Jonathan McNeal DO, Chris Busch DO, Eric Marvin DO, Gary Simonds MD MHCDS

Carilion Clinic- Virginia Tech Carilion Neurosurgery, Roanoke, VA

### **Abstract**

#### **INTRODUCTION**

We are studying fine motor dexterity and conditions and circumstances that may affect it. We are hoping to better understand factors that may negatively affect surgical fine motor dexterity, such that they may be mitigated in the interest of minimizing complications due to technical error. In this study we evaluate the effects of gross motor activity on subsequent fine motor dexterity.

#### **METHODS**

Subjects practiced and underwent fine motor control assessments on a computerized assay system (MLS). In separate sessions they were then subjected to heavy gross motor exercise (simulating heavy spinal surgery manipulation). They were then retested on the MLS system.

#### **RESULTS**

Gross motor exercise had significant deleterious effects on fine motor dexterity.

#### **CONCLUSIONS**

Circumstances surrounding the employment of surgical fine motor activities may significantly impact a surgeon's dexterity. Consideration must be given to mitigating such factors. This might include eliminating gross motor activity immediately prior to surgical fine motor manipulation.

### **Introduction**

Demonstration and quantification of variables that affect fine motor performance could have a significant impact on how surgical procedures are scheduled and performed. We are evaluating a number of variables that may affect surgical dexterity. In this study we looked at the impact of gross motor activity on subsequent fine motor dexterity.

Subjects participated in gross motor activity to fatigue immediately prior to tasks requiring considerable dexterity. This is particularly of concern to neurosurgeons who often have to switch from gross motor activity of deep muscle dissection and bone removal, to exquisite fine motor activity of intradural microscopic dissection- often in the same procedure. Alternatively, they may go from one or two procedures of significant gross motor activity such as major spinal instrumentation surgery, to a high dexterity microscopic procedure such as brain tumor surgery. We wished to study the impact of gross motor muscular exercise immediately prior to fine motor tasks.

### **Methods**

We utilized the MLS Motor Performance Series of the Vienna Test Series by Schuhfried to assess fine motor dexterity. This is a modular test that utilizes Edwin Fleishman's factor analysis of manual dexterity. It consists of a panel with various contact surfaces and the subject uses a stylus to perform static and dynamic tasks. We tested each subject's dominant and non-dominant arm in the Steadiness, Aiming, Tapping, and Line Tracking tasks. These tests measure

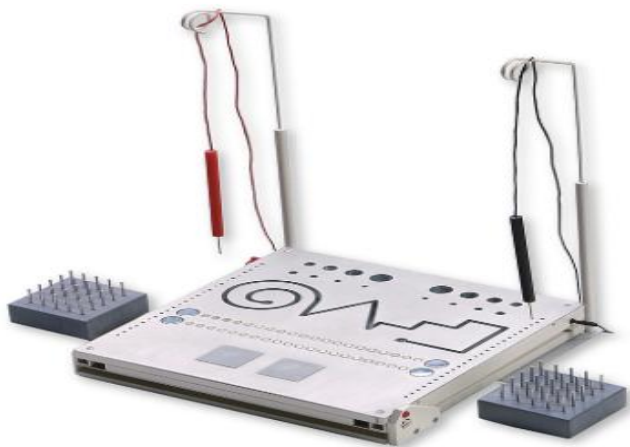
the accuracy and precision of movement, steadiness, finger dexterity, and speed of finger, wrist and arm movements. Subjects were all residents, physician assistants, or medical students at our parent institution.

The first phase of the study includes participants performing the six fine motor tasks on MLS until they achieve a uniform/reproducible performance in both hands.

### Gross Motor

In this study subjects then performed a series of gross motor activities that reproduce the gross motor maneuvers performed in major neurosurgical procedures. Step one included holding 15 pounds of weight at 90 degrees of arm abduction for one minute to simulate retraction of the paraspinous musculature during spinal dissection. The second step was placement and removal of six pedicle screws into solid polymer blocks. In the third step participants closed and opened a handgrip as many times as possible for 2 minutes to simulate the use of Kerrison and Leksell rongeurs in spinal surgery bone removal.

Immediately after the gross motor exercises subjects repeated the six fine motor tasks on MLS system with both hands. Pre and post gross motor activity MLS scores were compared.



### Results

A total of 20 surgical trainees participated in this study. Each participant was tested with both of his or her right and left hands. There was six females and 14 males representing various levels of surgical training: 5 people with minimal training, 5 with 1- 2 years, 6 with 3-5 years, 4 with 6+ years of training. There were four medical students, 2 PAs, 14 residents, and 2 neurosurgeons. The average age was 31. All participants completed the baseline examination, the fatigue exercises followed by the fatigued testing examination. All study subjects were reported right hand dominant.

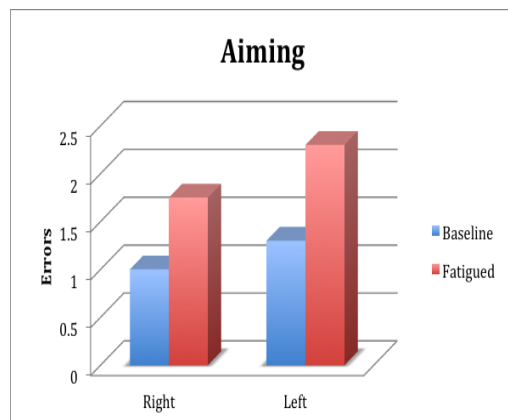
Gross motor activities significantly impacted the speed, reproducibility and precision of fine motor activities.

Aiming: the average error of the right (dominate) hand in aiming tasks was 1 across all groups. The average error rate at baseline was 1.75 on the left. Duration of the test was on average 9 seconds. There was a statistically significant increase in the number of errors for both hands after completing the fatigue protocol (p value < 0.002).

Steadiness was significantly affected by the fatigue protocol. Average baseline error rate of the Right hand/arm was 19.4, which increased to 32.3 after fatigue (p value < 0.001). Similarly, the average error rate of the left hand/arm at baseline was 25.2, which significantly increased to 40.45 after fatigue (p value 0.0001).

During a line tracking task, there was an increase in the number of errors when comparing the baseline and fatigued examinations, but these did not reach significance for the right or left hands, p value <0.13 and 0.82 respectfully. Interestingly, what we did observe was an increase in the speed while completing the task.

The fine motor tasks of repetitive tapping and long pin insertion were not significantly affected by the fatigue protocol, p value 0.31 and 0.92 respectfully. The number of tapping hits and the duration of time needed to place the 20 pins stayed consistent between the two groups.



## Discussion

We found that there are indeed relatively straight forward factors that can affect fine motor control and dexterity. We feel that this may have a significant impact on surgical dexterity.

We were able to demonstrate that gross motor activities have a statistically significant impact on the speed, reproducibility and precision of fine motor activities specifically during steadiness and aiming tasks. Activity lag time and error rates increased after gross motor activities.

Interestingly activity speed increased while the error rates decreased during line tracking activities. We believe this is likely secondary disinhibition caused by motor fatigue. Participates

performed the task quicker with less errors likely secondary to fatigue and less concentration. We would like to believe this does not happen in real life surgical performance but other assays will have to be created to examine this further.

Our study demonstrates the potential effects gross motor activities have on fine motor tasks, which may play a vital role in a surgeon's performance especially in long operative cases. Demonstration and quantification of these should call to question how surgical procedures are scheduled and performed. Procedures requiring exquisite dexterity should not be scheduled immediately after gross motor intense procedures. This may be particularly problematic in a procedure that requires both. Perhaps such procedures should be performed by teams of surgeons- one or more performing the gross motor tasks and a fresh fatigue-free surgeon performing the fine motor tasks. We also question whether mechanical assistance or even robotic technology can improve upon fine motor task performance after gross motor activity.

Of further concern is how long the detrimental effects of gross motor exercise on surgical fine motor dexterity may last. For example, could weight lifting the night before a complex dexterity-dependent procedure impact the surgeons fine motor control of said procedure? Anecdotally, several of our surgeons have noticed such effects. To explore this, we plan to trial periods of exercise at various times before fine motor testing.

We are interested in other factors that might impact surgical dexterity. We have completed a study on hand cooling that also has demonstrated a deleterious effect on dexterity. Primary testing on background noise and its effects on fine motor control are underway. Future plans include examining the effects of sleep deficit, caffeine, core temperature, mental fatigue, persistent engagement in conversation, frequent interruptions/distractions, emotional fatigue, exercise, and even alcohol.

## References

1. Fleishman, E.A., Quaintance, M.K. and Broedling, L.A. (1984). Taxonomies of Human Performance: The description of human tasks. Orlando, FL: Academic Press, Inc.
2. Kalisch T, Wilimzig C, Klebiel N, Dinse H. Age related attenuation of dominant hand superiority. PLoS One. 2006. 1:e90.
3. Neuwirth W, Benesch M. 2012. Manual: Motor performance series. Schuhfried GmbH, Austria. Version 29.
4. Magill, R. (2007). Motor learning and control; concepts and applications. (9th edition ed., pp. 2-22). New York, NY: McGraw-Hill companies inc.
5. Schmidt, R., & Wrisberg, C. (2004). Motor learning and performance, third edition. (3rd ed., pp. 25-48). Champaign, IL: Human Kinetics.
6. Slack PS, Coulson CJ, Webster K, Proops DW. The effect of operating time on surgeons' muscular fatigue. Ann R Coll Surg Engl 2008; 90: 651-657.
7. Snow, S. (2010). Skills school fundamental ball skills. (pp. 3-4). US Youth Soccer
- Vanwassenhova, K. (2013, July 21st). Activities that help develop motor skills. Retrieved from <http://uofmhealthblogs.org/childrens/activities-that-help-develop-motor-skills>

# **The Rate of Adjacent Segment Disease in Combined Anterior and Posterior Lumbar Interbody Fusion: a Retrospective Cohort Study and Review of Current Literature**

Thuy M. Nguyen, DO

Beaumont Hospital, Trenton, MI

## **Introduction**

The origin of anterior lumbar interbody fusion may be credited to Norman Capener in the 1930's, an orthopaedic surgeon from London, England (1,2). This approach was primarily used to treat spondylolisthesis but has evolved to treat a number of pathologies beyond spondylolisthesis including pseudoarthrosis and degenerative disc disease (3). Anterior lumbar interbody fusion has been shown in studies to afford superior restoration of disc height along with lumbar lordosis and a lower risk of dural injury when compared to the posterior approaches; however, there may be longer hospitalization and a higher risk of vascular injury associated with the anterior lumbar interbody fusion. Although there is much in the literature regarding potential complications and rates of complications of the anterior lumbar interbody fusion including studies comparing outcomes between anterior lumbar interbody fusion versus transforaminal lumbar interbody fusions or even posterior lumbar interbody fusions, there is nothing reported in the literature regarding rates of adjacent segment disease as it relates to clinical symptomatology necessitating revision. Here we report on our institutions results on the revision rate of a combined 360 degree lumbar fusion using an anterior lumbar interbody fusion technique along with posterior backup screws through a percutaneous method. We will also review known complications of anterior lumbar interbody fusion as well as complications associated with the transforaminal lumbar interbody fusion.

## **Methods**

A chart review as well as a radiological review was conducted by a single neurosurgery resident done on 140 patients who had both an anterior lumbar interbody fusion as well as a posterior backup fixation done by a single neurosurgeon at one institution, Beaumont Dearborn. Any revision surgery was noted along with the reason for reoperation. The caseload spanned over a course of 6 years with the longest followup being 6 years. Of the 140 patients, only 121 were successfully and properly identified by way of date of birth and the medical record number. Anterior exposure was conducted by one of two separate general surgeons for each patient.

### **Our complications and rates**

Of the 121 patients who received an anterior lumbar interbody fusion with posterior backup utilizing instrumented fixation, we saw seven revision surgeries in six patients. Revisions involved no adjacent segment disease or surgery to extend the fusion due to adjacent segment disease. Our revision surgeries included taking out misplaced facet-pedicle screws due to medial breach with nerve irritation and replacing that construct with interspinous process plating. A case involving nonunion at L3-4 and S1 spinous process fracture in the setting of an L3-4 interspinous process plate and L5-S1 interspinous process plate with L4-5 pedicle screws underwent revision by removing the interspinous process plates and replacing them with pedicle screws from L3 to S1. There was one case where some spine surgeons may argue could be technically adjacent segment disease which



included our patient who had a prior anterior lumbar interbody fusion with posterior pedicle screws from L4-S1 that later required a left-sided sacro-iliac joint fusion. There was one patient who developed an epidural fluid collection and also concurrently had loose pedicle screws from L4-S1 and so an operation was carried out to biopsy the epidural fluid as well as a washout and upsizing the pedicle screws; the cultures grew back staphylococcus aureus. One of our patients had two revisions; this included an index procedure where the patient underwent an anterior lumbar interbody fusion at L5-S1 and later percutaneous pedicle screws from L1-S1. The patient later was found to have intractable new back pain and discovered to have a superior endplate fracture at L1 and so surgery was carried out to extend the fusion to T10. She then later developed pseudoarthrosis and was taken back for repositioning of the left T10 screw while removing bilateral S1 screws and an extension of the construct to the iliac crests bilaterally.

### **Complications of ALIF**

An anterior approach to the lumbar spine is not without its own complications. These may include not only complications that are more familiar with any interbody fusion but also include exposure related complications. Major access related complications include venous injury to the extent that it would need to be repaired and arterial injury. Minor access related complications would include incidental peritoneal openings, infection, leg edema, minor vascular injury not necessitating repair, and retrograde ejaculation. One retrospective review from the United Kingdom reported their complication rates for anterior lumbar interbody fusion via a paramedian retroperitoneal access approaching from the left side. This included a report of 304 consecutive patients who were followed over the course of 10 years. This study excluded patients who underwent anterior lumbar interbody fusion for tumor resection, trauma, revision surgery, and infection. They saw that vascular complications were the most common out of this group occurring in 7.8% of their patients. Interestingly, only 3% of the vascular injuries required repair by a vascular surgeon suggesting that the majority of these vascular injuries either did not need a repair or were minor enough that the spine surgeon was able to fix it (5). Other potential complications from an anterior lumbar interbody fusion include ileus, deep vein thrombosis, adjacent segment disease, as well as wound infection (6). A multicenter study determining associated predictors of complications following single-level anterior lumbar interbody fusion was conducted. This study included an assessment of both medical and surgical complications. They determined that surgical complications were associated with diabetes, corticosteroid dependence, and preoperative transfusion of greater than 4 units. As for medical complications, predictors included weight loss prior to surgery, longer operative times, more severe American Society of Anesthesiologists classification, and preoperative anemia. The surgical complications found in this group included superficial wound infection, deep wound infection, organ infection, wound disruption, graft failure, and peripheral nerve injury. Medical complications included such things as pneumonia, unplanned intubation, pulmonary embolism, ventilation dependence for more than 48 hours, renal insufficiency, urinary tract infection, myocardial infarction, transfusions, deep vein thrombosis, and sepsis (9).

### **Complications of TLIF**

Transforaminal lumbar interbody fusion has been around since the early 1980's and much literature has been published on the approach including complications. One group from the University of Pittsburgh published their own complication database including rates and compared single-level transforaminal lumbar interbody fusion complications and rates to those of two-level transforaminal lumbar interbody fusion and multilevel transforaminal lumbar interbody fusion. Their study showed that the most frequent complication included durotomy effecting 14.3% of their patients. Infection was also found in 3.8% of their patients, screw misplacement occurred in 2.1% of patients, and interbody cage migration happened in 1.8% of their patients. This particular group found that patients with a prior surgery were at greater risk for complications as well as those who would undergo a multilevel operation. This was especially true for cases complicated by durotomy (7). A group out of the Rothman Institute published their complications for single-level transforaminal lumbar interbody fusion as it pertains to patients who had autograft from their iliac crest compared with those who received rhBMP-2. This was a retrospective cohort study and what they found was that the autograft group experienced more frequent complications although this difference did not reach statistical significance. The peri-operative complications seen in the autograft group involved persistent donor-site pain most commonly, lumbar wound infection, donor-site infection, and postoperative radiculitis. Peri-operative complications in the rhBMP-2 group included postoperative radiculitis most commonly, followed by vertebral osteolysis, then lumbar wound infection, and least frequently ectopic bone formation (8). A group out in Minneapolis reported on their retrospective cohort study regarding perioperative complications and rates in obese patients who underwent minimally invasive transforaminal lumbar interbody fusion for degenerative spondylolisthesis. This cohort involved a review of 134 consecutive patients. They further compared the complication rates from this cohort to a non-obese cohort. This study found no difference among intra-operative complications between the two cohorts. When comparing in-hospital complication rates, however, the obese group was found to have a higher rate than their non-obese counterparts. It was revealed that genitourinary complications predominated overall in regards to in-hospital complications. It was also noted that pulmonary and cardiac complications as well as ileus were only present in obese patients. When comparing complication rates between the two cohorts, it was found that obese patients had more frequent complications; this difference reached statistical significance. Wound drainage was found to be the most common complication six months postoperatively.

## **Conclusion**

Ultimately, although the anterior lumbar interbody fusion has been around since the 1930's, there has been much evolution of the procedure itself including instrumentation. There has been a great deal of literature on complications and rates among patients who have undergone anterior lumbar interbody fusion and posterior approaches including transforaminal interbody fusion as well as posterior lumbar interbody fusion. Even though complications and rates of complications have been reported for the anterior lumbar interbody fusion, there is no literature on the risk or rate of adjacent segment disease in a patient undergoing anterior lumbar interbody fusion with posterior pedicle screw fixation. Our group found no instances of clinically detectable or radiographic adjacent segment disease necessitating a revision. This raises many questions and challenges some of the

beliefs we currently hold for adjacent segment disease. Performing an anterior and posterior instrumented construct in the lumbar spine affords a very stiff construct and by historic rhetoric, should be an impetus for adjacent segment disease; however this was not seen in our series. Could this be because percutaneous screw placement decreases posterior tension band damage and limits dissection? Does paraspinal musculature play a role in the prevention of adjacent segment disease? These are questions that are yet to be explored but would lead us closer to better understanding adjacent level disease.

## References

1. Capener, N. (1932), Spondylolisthesis. *Br J Surg*, 19: 374–386. doi:10.1002/bjs.1800197505
2. Durbin FC. Norman Capener, the surgeon. *Annals of The Royal College of Surgeons of England*. 1975;57(6):287-291.
3. Phan K, Mobbs RJ. Evolution of Design of Interbody Cages for Anterior Lumbar Interbody Fusion. *Orthop Surg*. 2016 Aug;8(3):270-7. doi: 10.1111/os.12259. PubMed PMID: 27627708.
4. Phan K, Thayaparan GK, Mobbs RJ. Anterior lumbar interbody fusion versus transforaminal lumbar interbody fusion--systematic review and meta-analysis. *Br J Neurosurg*. 2015;29(5):705-11. doi: 10.3109/02688697.2015.1036838. Epub 2015 May 12. Review. PubMed PMID: 25968330.
5. Quraishi NA, Konig M, Booker SJ, et al. Access related complications in anterior lumbar surgery performed by spinal surgeons. *European Spine Journal*. 2013;22(Suppl 1):16-20. doi:10.1007/s00586-012-2616-1.
6. Qureshi R, Puvanesarajah V, Jain A, Shimer AL, Shen FH, Hassanzadeh H. A Comparison of Anterior and Posterior Lumbar Interbody Fusions- Complications, Readmissions, Discharge Dispositions and Costs. *Spine (Phila Pa 1976)*. 2017 May 25. doi: 10.1097/BRS.0000000000002248. [Epub ahead of print] PubMed PMID: 28549000.
7. Tormenti MJ, Maserati MB, Bonfield CM, Gerszten PC, Moossy JJ, Kanter AS, Spiro RM, Okonkwo DO. Perioperative surgical complications of transforaminal lumbar interbody fusion: a single-center experience. *J Neurosurg Spine*. 2012 Jan;16(1):44-50. doi: 10.3171/2011.9.SPINE11373. Epub 2011 Oct 14. PubMed PMID: 21999389.
8. Rihn JA, Patel R, Makda J, Hong J, Anderson DG, Vaccaro AR, Hilibrand AS, Albert TJ. Complications associated with single-level transforaminal lumbar interbody fusion. *Spine J*. 2009 Aug;9(8):623-9. doi: 10.1016/j.spinee.2009.04.004. Epub 2009 May 30. PubMed PMID: 19482519.
9. Choy W, Barrington N, Garcia RM, Kim RB, Rodriguez H, Lam S, Dahdaleh N, Smith ZA. Risk Factors for Medical and Surgical Complications Following Single-Level ALIF. *Global Spine J*. 2017 Apr;7(2):141-147. doi: 10.1177/2192568217694009. Epub 2017 Apr 6. PubMed PMID: 28507883; PubMed Central PMCID: PMC5415155.

## **Surgical Deep Wound Infection is Not Related to Intra-Operative Gross Bacterial Contamination in Spinal Instrumentation Surgery**

Jordan Synkowski DO, Aaron Danison DO, Jonathan McNeal DO, Josh Prickett DO, Cara Rogers DO, Chris Busch DO, Gary Simonds MD MHCDS  
Carilion Clinic- Virginia Tech Carilion Neurosurgery, Roanoke, VA

### **Abstract**

#### **INTRODUCTION**

Deep wound infection after major spinal surgery is presumed to be secondary to intra-operative deep wound contamination. We hypothesized that were this the case, aggressive deep wound cultures should correlate with downstream deep wound infections.

#### **METHODS**

We serially aggressively cultured the deep wounds of 220 consecutive patients undergoing major spinal surgery just prior to irrigation and closure. Results were then correlated with cultures of patients with post-operative deep wound infections.

#### **RESULTS**

There were 28 post-operative deep wound infections altogether. There were 15 positive intra-operative cultures. Of all patients with positive intraoperative cultures, none went on to sustain deep wound infections. Of all the patients who sustained deep wound infections, none had positive intra-operative cultures.

#### **CONCLUSIONS**

There was no correlation between intra-operative cultures and later deep wound infections. This suggests that deep wound gross bacterial contamination is not a major factor in post-operative deep wound infections.

### **Introduction**

Deep wound infections are the bane of the complex spinal surgeon's existence. Estimates of such infection in major spinal instrumentation surgery range from 5 to 15% of all cases. These infections seldom respond simply to antibiotic therapy. A large proportion will require surgical debridement and "washing out" of the wound with volumes of sterile fluid (often with antibiotics in solution). In fact, many sources advocate the full removal of hardware and prolonged IV antibiotic therapy to fully and permanently eradicate the infection (although this is not universally accepted).

Presumably the infection, which oft presents itself weeks downstream, is secondary to intra-operative bacterial contamination. In major spinal instrumentation surgery there is ample opportunity for such contamination. To better understand the contributing factors to such contamination we launched a longitudinal study of the organisms and potential sources of intra-operative deep wound contamination. Our eventual results called into question our original assumptions and opened up many other avenues of investigation.

### **Methods**

We studied a series of consecutive patients undergoing complex instrumented spinal surgery with the Neurosurgery Department at the Carilion Clinic in Roanoke Virginia. These surgeries

involved fusion and instrumentation of at least three segmental levels in the cervical, thoracic, and/or lumbar spine. Indications for surgery were degenerative instability, traumatic instability, or pathological instability (tumor). All surgeries involved, at least in part, posterior open approaches with sub-periosteal paraspinous dissections for posterior element and lateral mass exposures. Further posterior lateral dissection and partial or full corpectomies were performed when needed. Instrumentation involved multi-segmental pedicle screw and/or lateral mass and rod internal fixation. Fusion involved demineralized bone matrix and/or morselized local bone onlay to the lateral masses and adjacent transverse processes. Interbody spacers and fusion was used in addition when appropriate. All patients received appropriate peri-operative antibiotic therapy (Cefazolin whenever possible).

Prior to closure of the wounds it was our custom to irrigate with 3-6 liters of Bacitracin-Normal Saline solution. After full instrumentation, reduction, internal fixation, and graft onlay; but prior to any irrigation, the deep wound was vigorously cultured. Process of culture was to use two cotton swab applicators and aggressively rub them along all exposed surfaces including the instruments, the graft, and the deep muscles and fat bilaterally. Deep tissue was procured by the vigorous abrasion of the region with the swabs. Swabs were then immediately immersed in medium and sent for full batteries of culture (aerobic, anaerobic, fungal, TB, etc.).

Patients with positive intra-operative cultures were treated with appropriate antibiotic therapy. All Patients were followed for at least one year. Patients presenting with deep wound infections underwent interventional percutaneous aspiration of infected fluids and/or open access and collection of infected material for culture. Patients were treated with appropriate antibiotics for 8 to 12 weeks and/or open debridement and "wash-out." No case required removal of instrumentation. All infections resolved with our regimen.

Intra-operative culture results from the original surgeries were then compared with cultures obtained at the time of diagnosis of the deep wound infections.

## **Results**

220 patients underwent intra-operative deep wound culture during their original spinal instrumentation surgery. Of the 220 procedures, 15 had positive intra-operative cultures. Of the 15 patients with positive intra-operative cultures, zero went on to manifest deep wound infections.

Of the 220 patients undergoing surgery, 28 later presented with deep wound infections. Of the 28 patients presenting with deep wound infections, zero had positive intra-operative cultures at the time of their original surgery.

Of 95 patients classified by BMI as being obese, 16 developed deep wound infections (17%). Of 64 patients classified by BMI as being overweight, 8 developed deep wound infection. (13%). Of 56 patient classified by BMI as being normal weight, 4 presented with deep wound infections requiring wash-out (7%).

Of 89 patients presenting for surgery with traumatic instability, 9 developed deep wound infections requiring wash-out. (10%). Of 28 patients presenting for reoperation after an initial surgery, 3 developed deep wound infections requiring wash-out. (11%). Of 19 patients presenting for surgery with pathological fractures, 5 developed deep wound infections requiring wash-out. (26%). Of 79 patients presenting for elective surgery, 11 developed deep wound infections. (14%)

The most common bacterial organism obtained from deep wound culture at initial surgeries was: Staphylococcus was the most common genus, with Coagulase-negative Staphylococcus being the most common sub-genus group identified.

Bacterial organisms obtained from post-operative wound infections were: Methicillin Staph Aureus (6), Enterobacter (6), Proteus (3), Meth Sensitive Staph Aureus (2), Citrobacter, Enetrococcus, Klebsiella, Prevotella, Serratia, E.Coli, and Propionobacter.

## **Discussion**

Large scale posterior spinal instrumentation surgery affords ample opportunity for bacterial contamination of the somewhat devitalized deep spaces of the dissection. Dozens of large instruments pass in and out of the wound hundreds of times. Glove breaches are common. Limited sterility of imaging modalities (c-arms, O-arms etc.) are passed over and around the wound. Bone is harvested, morselized, and contained for often many hours prior to its implementation. Graft has been demonstrated to carry a bacterial load in a no insignificant number of cases. Hands are used directly in the wound for various tasks. Headlights and loupes are collided over the incision showering the open wound with surgical skin detritus and dust. The room generally features more support personnel coming and going than standard cases (product representatives, monitoring personnel, break- personnel, extra help for the back tables, etc.). The wound is open often for many hours.

We began this study anticipating a large number of cases demonstrating intra-operative deep wound gross bacterial contamination. Our original goal was to obtain baseline descriptions of the rate and organism profile of said contaminations. From there we had planned to study ways to mitigate deep wound contamination. What we witnessed instead was a very low rate of contamination, and a total lack of correlation between contaminating bacteria in the original surgeries and infection-causing bacteria obtained from post-operative infected deep wounds. This would suggest that the predominance of major post-operative infections in spinal instrumentation surgery are not caused by intra-operative deep wound contamination.

A critique of this study could be the methodology of tissue culture. It is asserted that the most sensitive wound cultures are obtained by actual tissue biopsy rather than swab culture. The problem with a biopsy methodology is that it would sample only a single or handful of areas in the wound. These are often massive wounds. We sought to cover the entire region of the open wound including the graft and the instrumentation. The swabs were aggressive and contained tissue from the wound and thus approximated biopsy.

We surgeons are conditioned to believe that we are responsible for wound infections. We are taught that such infections must have something to do with our technique, or tissue retraction, or closure. This is certainly bolstered by a medical legal world that holds us accountable. In fact, there is a whole sub-specialization of litigation attorneys in the field of surgical wound infections. We are so conditioned that we find it difficult to accept the notion that wound infection may be a problem that at least to some degree is out of our control. Yet our other findings, and those of the related literature, suggest that risk of infection is more related to the tissue micro-environmental milieu than it has to do with surgical technique. Thus, medically compromised, obese, and/or trauma patients are more prone to infection by the nature of their tissues than by the actions of their surgeons. Could it be that the wounds are seeded after surgery or by post-operative superficial wound contamination/colonization? Certainly, large prolonged open wounds are ripe for tissue devitalization. This may be why there is an apparent decreased rate of infection in minimally invasive instrumentation procedures. Certainly these procedures have their share of multiple instruments being passed in and out of the wound. But, perhaps the wounds are healthier and less traumatized, and thus more resistant to downstream contamination or bacteremic seeding.

Perhaps our culture method was simply too insensitive to pick up minimal contamination with just a scattering of organisms- that our study argues only against gross contamination. If this is so, it would still argue that the infections are more related to the wound milieu rather than the surgical technique, for no operating theatre and indeed no operation is 100% void of bacterial exposure.

In the end, we are not arguing against aggressive sterile technique. Obviously this is critical for all invasive procedures. What we are forwarding is the notion that deep wound infections in instrumented spine surgery is possibly not related to gross wound contamination, but rather to the health of the tissue of the wound itself. Bacterial entry may indeed be during surgery itself, but also may be post-operative through the incision or through bacteremic seeding. What may be most important is efforts to prevent wound devitalization such as diminished surgical time, decreased cauterization, and minimization of tissue retraction. This last notion becomes quite a challenge nowadays with the epidemic of intra-operative electrophysiologic monitoring which often obviates the use of muscle relaxants. Further tissue protection may be gained by the employment of minimally invasive techniques when possible.

## **Conclusions**

The results of this study suggest that deep wound infections after major spinal instrumentation surgery are not due to gross bacterial contamination of the wound at the time of the surgery. Other factors such as wound tissue viability, and general health status of the patient, may play a much more important role in the entity.

## **References**

1. Steven M. Kurtz, Ph.D, Edmund Lau, M.S., Kevin L. Ong, Ph.D., Leah Carreon, M.D., M.Sc., Heather Watson, Ph.D., Todd Albert, M.D., and Steven Glassman, M.D.

- Infection risk for primary and revision instrumented lumbar spine fusion in the Medicare population. *Journal of Neurosurgery: Spine*. Oct 2012 / Vol. 17 / No. 4 / Pages 342-347
2. Chen SH, Lee CH, Huang KC, Hsieh PH, Tsai SY. Postoperative wound infection after posterior spinal instrumentation: analysis of long-term treatment outcomes. *Eur Spine J*. 2015 Mar;24(3):561-70. doi: 10.1007/s00586-014-3636-9. Epub 2014 Oct 29.
  3. Viola RW<sup>1</sup>, King HA, Adler SM, Wilson CB. Bacterial wound contamination during simple and complex spinal procedures. A prospective clinical study. *Spine (Phila Pa 1976)*. 1997 Oct 15;22(20):2444-50; discussion 2450-1.
  4. Gelalis ID, Arnaoutoglou CM, Politis AN, Batzalexis NA, Katonis PG, Xenakis TA. *Spine J*. 2011 Nov;11(11):1042-8. doi: 10.1016/j.spinee.2011.10.015.
  5. Makama JG, Okeme IM, Makama EJ, Ameh EA. Sterility of C-Arm fluoroscopy during spinal surgery. *Surg Infect (Larchmt)*. 2016 Mar 16.
  6. Biswas D, Bible JE, Whang PG, Simpson AK, Grauer JN. Incidence of microbiological contamination of local bone autograft used in posterior lumbar interbody fusion and its association with postoperative spinal infection. *Spine (Phila Pa 1976)*. 2008 Aug 1;33(17):1913-7. doi: 10.1097/BRS.0b013e31817bb130.
  7. Lee CS, Kang KC, Chung SS, Kim KT, Shin SK. Incidence, prevalence, and analysis of risk factors for surgical site infection following adultspinal surgery. *J Neurosurg Spine*. 2016 Jan;24(1):20-4. doi: 10.3171/2015.3.SPINE14578. Epub 2015 Sep 11
  8. Pullter Gunne AF, Cohen DB. *Spine (Phila Pa 1976)*. 2009 Jun 1;34(13):1422-8. doi: 10.1097/BRS.0b013e3181a03013.
  9. Kurtz SM, Lau E, Ong KL, Carreon L, Watson H, Albert T, Glassman S. *J Neurosurg Spine*. 2012 Oct;17(4):342-7. doi: 10.3171/2012.7.SPINE12203. Epub 2012 Aug 24.
  10. Fang A, Hu SS, Endres N, Bradford DS. Risk factors for infection after spinal surgery. *Spine (Phila Pa 1976)* 30:1460–1465, 2005.
  11. Olsen MA, Nepple JJ, Riew KD, Lenke LG, Bridwell KH, Mayfield J, et al.: Risk factors for surgical site infection following orthopaedic spinal operations. *J Bone Joint Surg Am*90:62–69, 2008
  12. Parker SL, Adogwa O, Witham TF, Aaronson OS, Cheng J, McGirt MJ: Post-operative infection after minimally invasive versus open transforaminal lumbar interbody fusion (TLIF): literature review and cost analysis. *Minim Invasive Neurosurg* 54:33–37, 2011
  13. World Health Organization. [http://apps.who.int/bmi/index.jsp?introPage=intro\\_3.html&](http://apps.who.int/bmi/index.jsp?introPage=intro_3.html&)



## **Factors Such as Sodium Level Affecting Return to Hospital Within 30 Days After Traumatic Brain Injury**

Bailey Zampella, DO

Arrowhead Regional Medical Center, Colton, CA

### **Abstract**

**Objective:** Imbalances in electrolytes, particularly sodium, are common after traumatic brain injury (TBI). Decline in serum sodium levels are a significant cause of morbidity and mortality in the neurocritical patient. We sought to investigate the correlation between sodium levels and return to hospital within 30 days after TBI. **Materials and Methods:** A retrospective review of the Neurosurgery census from a single trauma center from 2016 to 2017 was conducted. 204 patients were identified as being admitted for TBI. From those 204 patients, we identified 10 patients who were re-admitted to the hospital within 30 days after suffering from TBI. Data collection points included demographics, sodium level on initial admission, length of stay (LOS), and sodium level on re-admission within 30 days. **Results:** 50% of patients presented with an increase in their sodium level within 30 days of readmission as compared to their sodium level on discharge; 30% of patients presented with a decrease in their sodium levels within 30 days of readmission after TBI, and 20% of patients had no change in their sodium level. **Conclusion:** There is no statistically significant correlation between sodium levels in patients with TBI who were re-admitted to the hospital within 30 days of discharge. It remains that sodium levels are closely associated with central nervous system dysregulation which is common in traumatic brain injury.

### **Introduction**

Fluid and electrolyte imbalance, particularly sodium, are common after traumatic brain injury (TBI). Fluctuation in sodium levels are particularly common in neurocritical patients due to the major role the central nervous system (CNS) plays in the regulation of sodium and water balance [1]. Decline in serum sodium levels are a significant cause of morbidity and mortality in patients suffering from TBI and may be difficult to diagnose. In non-comatose patients, clinical signs of decline in sodium levels are varied but may include: mild headache, confusion, emesis, changes in mental status, lethargy, respiratory depression, seizure, coma, and even death [2]. These symptoms are typically more common with large changes in sodium levels in a small interval of time.

Sodium fluctuations, specifically hyponatremia, are important in TBI patients as they are one of the most common and most major causes of refractory elevations in intracranial pressure [1]. Patients can suffer from syndrome of inappropriate antidiuretic hormone secretion (SIADH) or cerebral salt wasting (CSW). Clinical features of SIADH are non-specific and typically depend on absolute serum sodium levels and rate of development. Initial symptoms of SIADH may include, but are not limited to, nausea, malaise, headache, lethargy and mild cognitive deficits, and may eventually result in seizures, cardio-respiratory distress and coma [3]. Essential diagnostic criteria for SIADH include: low serum osmolality ( $< 280$  mmol/kg) with

an inappropriately high urine osmolality ( $>100$  mmol/kg) and urine sodium concentration ( $> 30$  mmol/L) [3]. The diagnosis of CSW requires a high index of suspicion and early identification is crucial to appropriate management. The hallmarks of CSW are decreased serum sodium levels ( $< 135$  mEq/L), urine sodium  $> 20$  mmol/L, serum osmotic pressure  $< 280$  mOsm/kg  $H_2O$ , and reduced volume status [4,6]. There is speculation that CSW syndrome is caused by CNS injury leading to disturbances in the parasympathetic and sympathetic nervous system, resulting in renin-aldosterone imbalance leading to subsequent inhibition of urine sodium and water reabsorption [6]. Treatment requires volume resuscitation and correction of sodium.

## Methods

This retrospective, single institution, observational study identified 204 patients that were identified to have suffered traumatic brain injury using an ICD-10 code for traumatic brain injury, traumatic subdural hematoma, traumatic epidural hematoma from 2016-2017. Data collected included demographics, ICD-10 codes, date of first admission, sodium level upon initial admission, date of second admission, length of stay (LOS), and sodium level on re-admission within 30 days. Hyponatremia was defined as a serum sodium level  $< 135$  mEq/L.

## Results

A total of 204 patients were identified as having suffered from TBI at one institution during a one year period. Of those 204 patients, 10 patients were re-admitted to the hospital within 30 days of discharge. Of these 10 patients who were re-admitted within 30 days, 8/10 (80%) were male and 2/10 (20%) were female. Age range was from 20 – 74 years of age, with average being 51.5 years old. The average length of stay during initial hospitalization was 8.5 days.

Upon further analysis, it was found that 5/10 (50%) patients had an increase in their sodium level on readmission as compared to their sodium level on discharge from initial hospitalization. 3/10 (30%) of patients had a decline in their sodium level on readmission after TBI as compared to their sodium level on discharge from initial hospitalization, and 2/10 (20%) of patients had no change in their sodium levels.

Of the patients who had a decrease in their sodium level on readmission, 3/3 (100%) were defined as hyponatremic and presented with symptoms of hyponatremia. 0/5 patients with an increase in their sodium level were considered hyponatremic.

## Conclusion

It is estimated that approximately 42 million people worldwide suffer from mild traumatic brain injury (mTBI) [5]. One of the most common complaints in patients with mTBI is persistent headache, known as post-traumatic headache (PTH). For most patients with PTH, pain management is insufficient and this tends to lead to a poor quality of life. Patient with PTH also tend to have imbalances in their

electrolytes, particularly with hyponatremia. Hyponatremia is unfortunately common in patients with mTBI and must be treated quickly and appropriately.

The results of this investigation show that hyponatremia after mTBI is common, as 100% of patients who had a decline in their sodium levels on readmission met criteria for hyponatremia. Treatment of hyponatremia is urgent, however, understanding why the patient is hyponatremic is the first appropriate step in guiding treatment. Associated symptoms with hyponatremia are most commonly headache and nausea/emesis and this is a common reason why patients present to the emergency department after suffering mTBI. Typically the hyponatremia associated with mTBI is transient and reversible, but it is necessary to determine if the patient is suffering from SIADH or CSW or another electrolyte imbalance causing the hyponatremia.

This study had several limitations. First, it is a retrospective study at a single institution with little control of various factors. Second, a wide variety of ICD-10 codes were included which creates variability in evaluation of patients and proposed outcomes. This study was underpowered and further prospective studies should be conducted to further investigate the effect of sodium levels in mTBI on re-admission within 30 days.

## Reference

1. Human T, et al. Treatment of Hyponatremia in Patients with Acute Neurological Injury. Neurocritical Care Society. DOI: 10.1007/s12028-016-0343-x
2. Greenberg M. Handbook of Neurosurgery. Thieme
3. Dick M, et al. Persistent Syndrome of Inappropriate Antidiuretic Hormone Secretion Following Traumatic Brain Injury. Endocrinology, Diabetes & Metabolism. DOI: 10.1530/EDM-15-0070
4. Taylor P, et al. Cerebral Salt Wasting Following Traumatic Brain Injury. Endocrinology, Diabetes & Metabolism. DOI: 10.1530/EDM-16-0142
5. Levy D, et al. Responses of Dural Mast Cells in Concussive and Blast Models of Mild Traumatic Brain Injury in mice: Potential Implications for Post-Traumatic Headache. Cephalalgia. DOI: 10.1177/0333102415617412
6. Wu X, et al. Diagnosis and Management of Combined Central Diabetes Insipidus and Cerebral Salt Wasting Syndrome after Traumatic Brain Injury. World Neurosurgery. (2016) 88:483-487

## **A Case Report of a Treatment Paradigm for Dural Arteriovenous Fistulas: The Statistically Unreported Use of Both Endovascular as Well as Microsurgical Treatment in a Cognard IV Borden III Dural Arteriovenous Fistula Not Described in Recent Literature**

Eric Bialaski, DO

Beaumont Health System, Trenton Campus

**Introduction:** Dural arteriovenous fistulas are vascular abnormalities in which a shunt exists between an arterial and venous structure contained within the leaflets of the dura and are almost exclusively supplied by branches of the carotid or vertebral artery before becoming intradural. Given the paucity of branches extracranially from the internal carotid artery, the external carotid artery is the artery implicated in these dural arteriovenous fistulas (dAVF). Dural AVFs comprise 10-15% of all intracranial vascular malformations. Some authors extend the definition of arteriovenous malformations (AVMs) to include that of dAVFs; however, the difference between these two entities exists in that dAVFs are true arterial and venous connections via the fistula. 61-66% of dAVFs occur in females and typical age of presentation for these patients is usually in the 5<sup>th</sup> or 6<sup>th</sup> decade from general symptoms such as pulsatile tinnitus to visual changes to cephalgia. Treatment of dAVFs include transarterial or transvenous endovascular embolization, microsurgical ligation and, stereotactic radiosurgery. As we will see below, a novel approach has been employed in the treatment for a Cognard IV Borden III dAVF yet to be described in literature as dual therapy in recent research yet mentioned in texts.

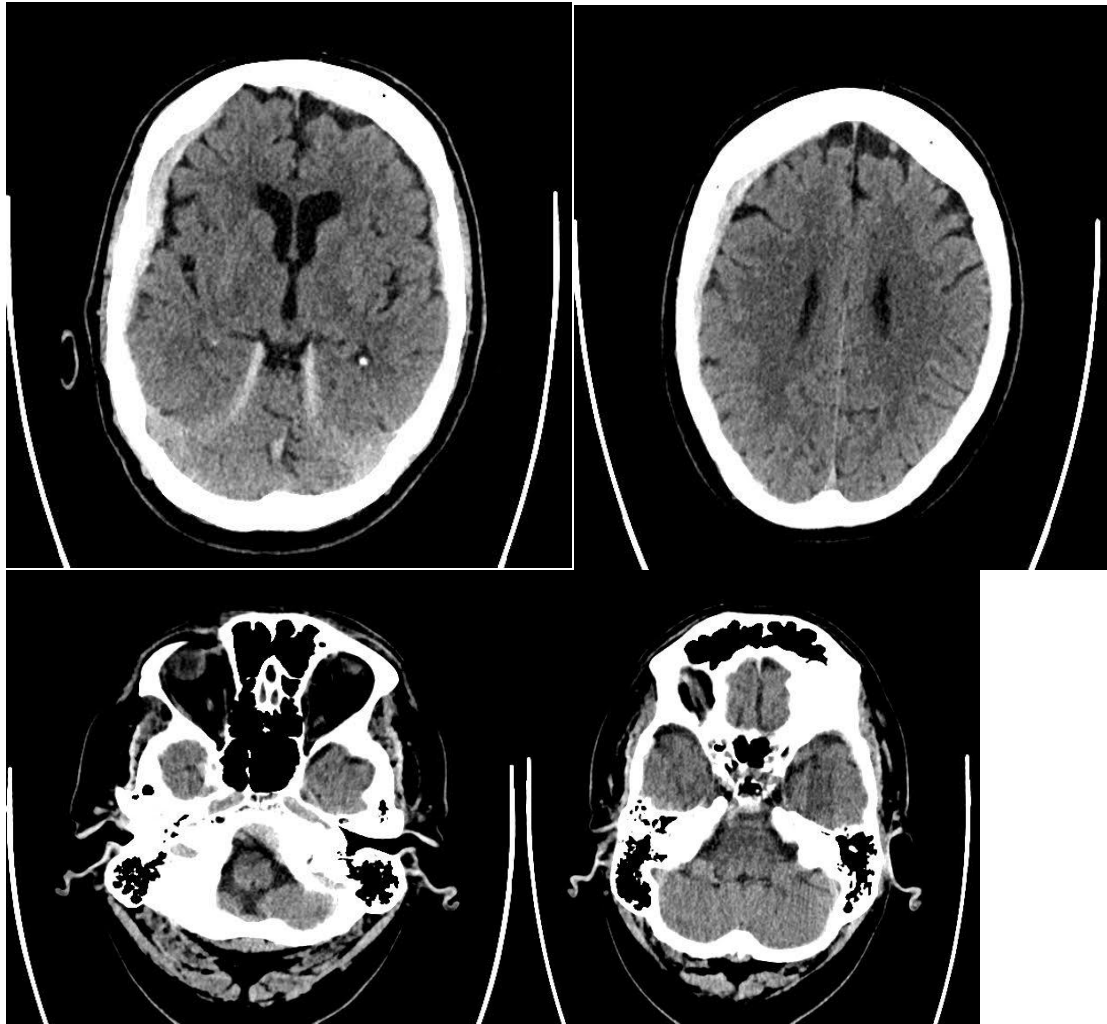
**Case Presentation:** RD is a 73 year old right handed male that initially presented to one of our satellite hospitals with a past medical history significant for atrial fibrillation with an ablative procedure, myocardial infarction, bladder cancer with nephrectomy (pathology was transitional cell), coronary artery disease with a in the RCA, hypertension and, diabetes mellitus presents with the chief complaints of cephalgia, hearing as well visual changes. It is of note that the patient was on Coumadin and aspirin at the time of presentation with a platelet count of 183 and an INR of 1.8. The patient states that he was on his way to a Beaumont facility for testing when he started experiencing the onset of these symptoms. The patient was driving at the time and because of these symptoms, presented to the nearest hospital setting. RD states that the symptoms were a gradual onset over the course of 45 minutes without any history of antecedent trauma. He had never experienced these symptoms in the past and described the hearing change as loss of hearing without lateralization as well as visual changes described as being "foggy". The patient stated that upon arrival to the satellite hospital, his hearing and visual changes abated; however, the cephalgia persisted which he described as being bandlike in the frontal region. The patient underwent a noncontrasted CT head as well as a MRA of the brain and was ultimately transferred to our facility for definitive care. This patient denied current use of tobacco (states he quit 5 years ago after an extensive past history use) and alcohol and states that he works part time as a broker.

**Physical Examination:** General appearance of RD is that of a nontoxic well hydrated male with an appearance appropriate for age. RD has a nonfocal examination where face is symmetric, tongue is midline. There were no cerebellar signs found on examination; dysdiadochokinesia was absent and negative rhombbergs. Visual field testing demonstrated intact extraocular

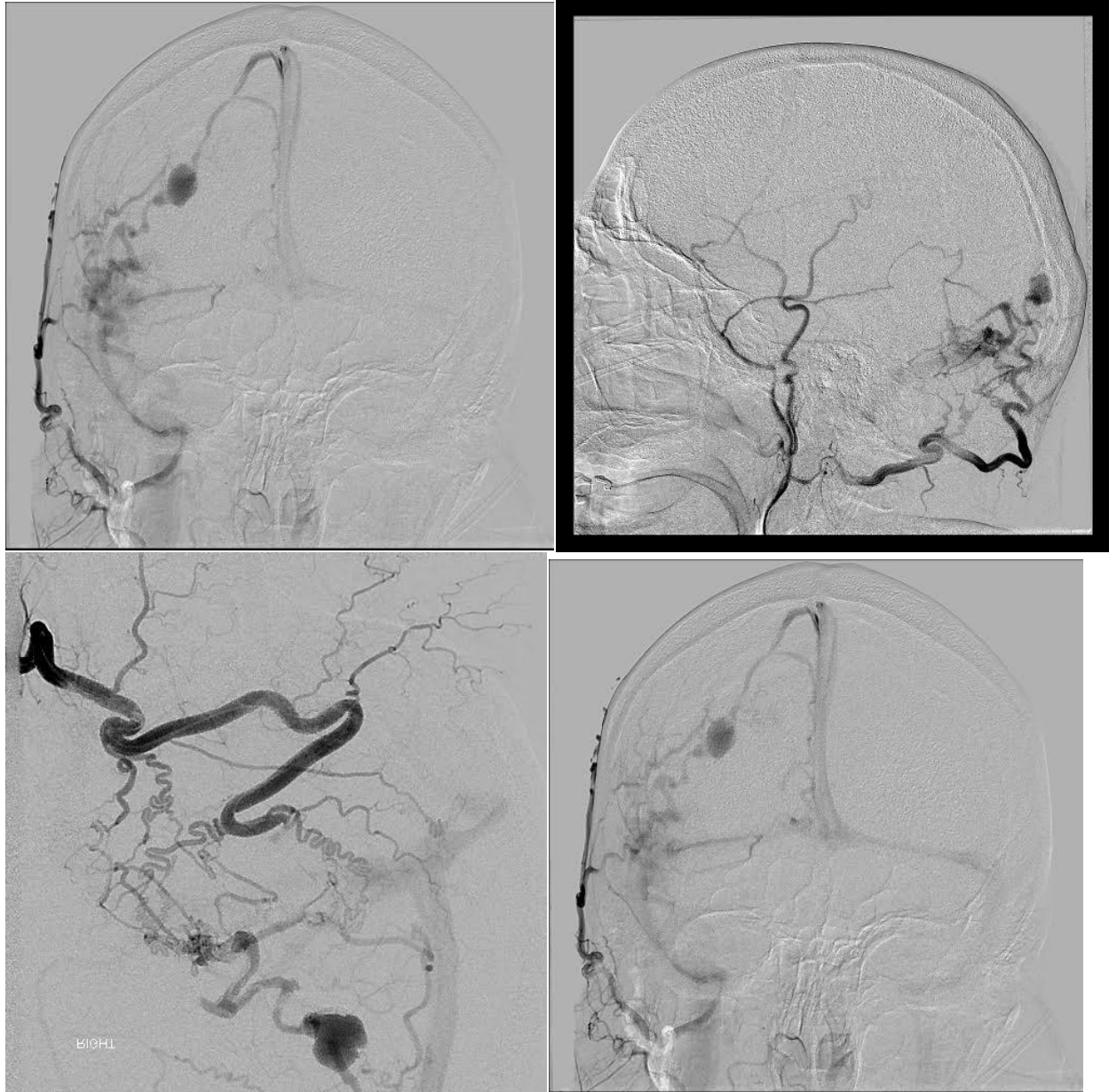
movements without any perceivable visual field deficits. There were no lateralizing hearing deficits at bedside examination. Strength is well preserved in the myotomes C5-T1 as well as L2-S1. Sensation is intact to light touch in the dermatomes of C5-T1 as well as L2-S1 bilaterally. Reflexes at this biceps, brachioradialis, triceps, quadriceps and, Achilles are graded as 2/4. There was no reproducible tenderness in the suboccipital or temporal regions.

**Imaging:** Upon initial arrival to the satellite facility, RD underwent a noncontrasted CT head which was reviewed. Salient findings included bilateral acute subdural hematomas right greater than left resulting in midline shift and mass effect. There was also noted prepontine subdural hematoma as well as hemorrhage extending into the foramen magnum extending down into the cervical spinal cord not well visualized on the CT head. This initial scan was completed upon the day of arrival and in light of his Coumadin coagulopathy, the patient was appropriately reversed with the use of fresh frozen plasma and vitamin K with the cessation of his coumadin. The patient was also transferred to a higher level of care at an adjacent facility where a repeat noncontrasted CT was completed. Findings were recapitulation of the bilateral subdural hematomas right greater than left with slight resolution of the left sided component as well as resolution of the noted prepontine and foramen magnum subdural hematomas. All the while, the patient remained stable neurologically without any focal or lateralizing deficits. Subjective complaints of cephalgia persisted. Given the atraumatic nature of spontaneous subdural hematomas, the patient underwent a MRA of the brain. Salient findings of the MRA consisted of abnormal increased vascularity in the right posterior temporal and occipital areas with a large vascular structure in the right occipital pole measuring 10mm in size. Because of this, RD electively consented to undergo a diagnostic formal cerebral angiogram which required transfer to a stepdown ICU floor at our facility. Chronologically, the patient underwent the formal angiogram 4 days after initial presentation to a satellite hospital.

Diagnostic cerebral angiogram was completed upon arrival to our facility. A focused angiogram pertaining to the abnormalities found on MRA was performed with selective catheterization of the right external and internal carotid arteries and vertebral artery. Findings were consistent with a Cognard type IV Borden grade III dAVF. Treatment was not performed at this time as the purpose for this study was that of diagnosis. Seen on the angiogram from the anterior-posterior and lateral views is the posterior occipital artery and meningeal feeders forming the dural arteriovenous fistula with drainage into the superior sagittal sinus with accompanying venous ectasia. Venous outpouching was measured to be 1.26cm by 1.17cm by 0.86cm in size. Images are represented below.



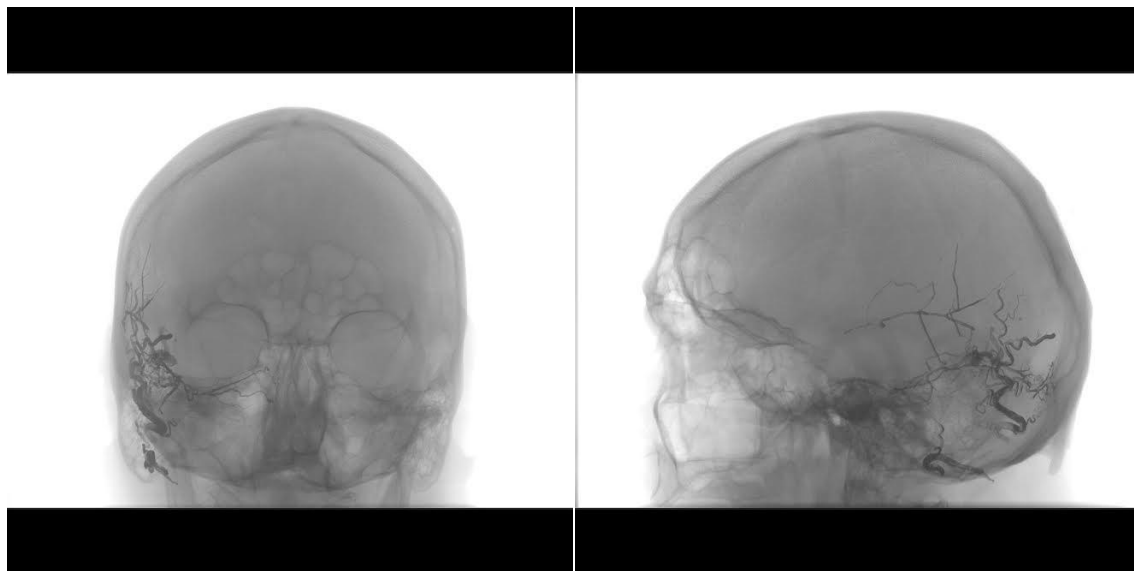
The above pictures are the initial noncontrasted CT head that was performed. Access to outside films were not permitted; hence, these scans are from the patients arrival to our facility prior to performing the diagnostic formal cerebral angiogram. The findings from that study are depicted below.



Findings from above depict on the diagnostic study the presence of a dAVF being fed predominately by the posterior occipital artery with a lesser extent of meningeal feeders draining into the superior sagittal sinus by way of a ectatic venous outpouching. One may raise concern of the possibility that there is a smaller vessel draining into the right transverse sinus.

**Treatment:** After completion of the angiogram and discussion with RD as to what his treatment options were, he underwent transarterial endovascular embolization via onyx technique the following day. Selective catheterization of the right external carotid artery as well as right internal maxillary and middle meningeal arteries was performed and after identifying all components of the fistula, onyx embolization was performed and complete exclusion of the fistula from the normal circulation was achieved. Post onyx application runs were completed to

demonstrate preservation of normal circulation without vessel cutoffs and normal capillary and venous filling stages. Post procedurally, the patient was extubated and transferred back to the stepdown ICU floor in stable neurological condition. With obliteration of flow through this fistula but the fistulous tract still present, the patient then went to the operating room 2 days following the onyx procedure for definitive ligation of these vessels, additional ligation of dural feeders into the right transverse sinus and cauterization of the venous ectasia from the fistula. Coagulation of the dura was performed as well with dural tack up stitches utilized. Post embolization angiography is included below. Surgery was performed in the prone position with the use of Mayfield head pins. Incision was made over the right parietooccipital region in horseshoe fashion with creation of a myocutaneous flap. Free bone flap was elevated just cephalad to the transverse sinus and posterior enough to be in proximity to the superior sagittal sinus. The dura was opened radially and cauterized as it was opened exposing the dural venous channel traversing deep. At this point, microscopic dissection ensued with rupture of the venous ectatic outpouching. Hemostasis was achieved with gentle pressure and noting the proximal and distal boundaries of this at which point cauterization was performed to control the bleeding. After coagulation of feeders into the right transverse sinus, there were no other fistulous tracts that could be seen. Closure was achieved folding the dural leaflets loosely over duragen placed in the subdural space. The bone flap was anchored in place with a titanium plating system and skin closure was achieved in 2 layers.



Postoperatively, RD's recovery process was uncomplicated and was ultimately discharged from the hospital in the following week to an outside nursing facility with the instructions to follow up in 2 weeks time period for suture removal as well as postoperative evaluation. When the patient was discharged, he was noted to be in stable neurologic condition.

**Discussion:** Dural arteriovenous fistulas, as previously noted, are common in females in their 5<sup>th</sup> and 6<sup>th</sup> decade. Typical presenting symptoms are visual impairment with cephalgia from elevated intracranial pressures. Treatment of these vascular lesions is guided by the type of



fistula that is presents. Fistulous connection into a sinus or retrograde flow into a sinus may be observed. Retrograde flow into cortical venous structures, as was the case with RD, require more aggressive treatment with either embolization of surgery and in refractory cases, stereotactic radiosurgery. Courtesy of Cognard (1) and Borden (2), below is a grading scheme used to classify these fistulas:

Borden Classification	
1	Venous drainage directly into DVS or meningeal vein
2	Venous drainage directly into DVS with CVR
3	Venous drainage directly into subarachnoid veins CVR only
Cognard Classification	
I	Venous drainage directly into DVS with antegrade flow
II a	Venous drainage directly into DVS with retrograde flow
II b	Venous drainage directly into DVS with antegrade flow and CVR
II a+b	Venous drainage directly into DVS with retrograde flow and CVR
III	Venous drainage directly into subarachnoid veins CVR only
IV	Type III with venous ectasias of the subarachnoid veins
V	Direct drainage into spinal perimedullary veins
	DVS = Dural Venous Sinus
	CVR = Cortical Venous Sinus

Table 1 adapted from Cognard (1) and Borden (2)

Type I has a benign course while type IIa the sinus reflux caused intracranial hypertension in 20%; type IIb with reflux into veins induced hemorrhage in 10% of cases, type IIa+b found aggressive in 66% with bleeding and/or intracranial hypertension, type III had hemorrhage occur in 40% of patients while type IV was 65%. Type V exhibited progressive myelopathy as it incorporated spinal venous drainage (1).

Given that RD's angiogram demonstrated a Cognard type IV Borden III dAVF, treatment was necessary. Historically, in Schmidek and Sweet (3), Javadpour note that these types of dAVFs need to be treated aggressively with a multidisciplinary approach sometimes requiring transarterial, transvenous and, surgical intervention. They note that, "complete obliteration of the fistula is ideal but not critical because there is increasing evidence that disconnection of the cortical venous reflux into the sinus alone results in long term protection against hemorrhage and neurological deficit" (3). Transarterial approach is the preferred method over transvenous approach for the more aggressive fistulas. The authors contrived a treatment paradigm applicable to Cognard type IV dAVFs that favors the use of transarterial embolization first. If unsuccessful, transvenous approach may then be applied and if still unsuccessful, then surgery. Keeping this paradigm in mind, the current literature that I reviewed fails to mention success with surgical intervention; more to the point, that endovascular techniques are the almost exclusively published techniques in the treatment of dAVFs. Are we accepting the paradigm of

embolization alone at the expense of the patient safety or is what Schmidek and Sweet have to offer in a multidisciplinary approach more apt to offer better patient outcomes?

Published in the handbook of Clinical Neurology in 2017, Mulholland (4) found that “the majority of dAVFs can be treated with either transarterial or transvenous embolization and for those that are not amenable, adjuvant surgery or radiotherapy”. Interesting to note though, De Keukeleire et al, Macdonald et al, Natarajan (7) and, Chen (8) note that their primary mode of treatment is transarterial embolization. Natarajan further refines this to state that if there is cranial nerve involvement, transvenous embolization is the preferred choice. Notably, from these authors; however, we see that these authors (and more globally, the vast amount of currently published literature) reports their success rates with endovascular treatment.

## **Conclusion**

I propose that there is a void in our current literature where we report success rates with both endovascular as well as surgical treatment collectively, as seen as the case with RD.

In 2010, Natarajan (7) found that complete obliteration and cure was achieved in his study with a multimodal approach, namely, 81% of patients were cured. He finds that all surgical cases treated were cured but statistics could not be located. Macdonald reported his success in the Journal of Neuroradiology in 2010 (6) with endovascular approaches and further states that these incompletely treated with onyx embolization may benefit from surgery. Again the point is raised though that there is a paucity of any of literature that exists defining the success of dual combination therapy of onyx embolization and surgery, unlike SRS which was first utilized in 1970's and documented success in 1982 by Barcia-Salorio (9). To this point, there is literature that states success rates with stereotactic radiosurgery (SRS). Chen in 2015 (8) found a success rate of 73% and 58% complete obliteration for cavernous sinus and noncavernous sinus dAVFs respectively using SRS. Gross (10) published his work in 2012 in Neurosurgery focus with his success rates. When combining his research with data from his literature review, 558 dAVFs were treated with SRS with an obliteration rate of 71%. The obliteration rate for cavernous dAVFs was 84%, whereas the rates for transversesigmoid and for tentorial dAVFs were 58% and 59% respectively.

Why is this important? When speaking to patients about treatment paradigms and the multidisciplinary approach that Schmidek and Sweet defined, patients may take solace in being presented the statistical success of endovascular embolization and surgery together. Therefore, as before, I see a need for improvement in our research in documenting the success of these 2 therapies combine and may be able to deliver improved patient care by understanding the statistical success behind this.

## **References**

1. Cognard C, Casaco A, Toevi M, Houdart Chiras J, Merland JJ. Dural arteriovenous fistulas as a cause of intracranial hypertension due to impairment of cranial venous outflow. J Neurol Neurosurg Psychiatry. 1998;65:308–16

2. Borden JA, Wu JK, Shucart WA. A proposed classification for spinal and cranial dural arteriovenous fistulous malformations and implications for treatment. *J Neurosurg.* 1995;82:166–79
3. Mohsen Javadpour, M. Christopher Wallace. 2012. Surgical Management of Cranial Dural Arteriovenous Fistulas. *Schmidek and Sweet's Operative Neurosurgical Techniques*, 959-976
4. Mulholland CB, Kalani MYS, Albuquerque FC Endovascular management of intracranial dural arteriovenous fistulas. *Handb Clin Neurol.* 2017;143:117-123. doi: 10.1016/B978-0-444-63640-9.00011-4
5. De Keukeleire K, Vanlangenhove P, Kalala Okito JP, Hallaert G, Van Roost D, Defreyne L. Transarterial embolization with ONYX for treatment of intracranial non-cavernous dural arteriovenous fistula with or without cortical venous reflux. *J Neurointerv Surg.* 2011;3:224–228
6. Macdonald JH, Millar JS, Barker CS. Endovascular treatment of cranial dural arteriovenous fistulae: a single-centre, 14-year experience and the impact of Onyx 18 on local practise. *Neuroradiology*2010;52:387–95
7. Natarajan SK, Ghodke B, Kim LJ, Hallam DK, Britz GW, Sekhar LN. Multimodality treatment of intracranial dural arteriovenous fistulas in the Onyx era: a single center experience. *World Neurosurg.* 2010;73:365–379
8. Chen CJ, Lee CC, Ding D, Starke RM, Chivukula S, Yen CP, Moosa S, Xu Z, Pan DH, Sheehan JP. Stereotactic radiosurgery for intracranial dural arteriovenous fistulas: a systematic review. *J Neurosurg.* 2015 Feb;122(2):353-62. doi: 10.3171/2014.10.JNS14871. Epub 2014 Dec 5.
9. Barcia-Salorio JL, Hernandez G, Broseta J, Gonzalez-Darder J, Ciudad J: Radiosurgical treatment of carotid-cavernous fistula. *Appl Neurophysiol* 45:520–522, 1982
10. Gross BA, Ropper AE, Du R. Cerebral dural arteriovenous fistulas and aneurysms. *Neurosurg Focus.* 2012 May;32(5):E2. doi: 10.3171/2011.12.FOCUS11336.

# **Use of the Acutrak 4/5 Headless Fully Threaded Variable Pitch Compression Screw for Odontoid Fixation in Type II Odontoid Fractures: A Technical note and Case Series compared with the standard Technique in a Single Institution**

Xin Xin, DO. Michael Kakareka, DO., Alan Turtz, MD., Steven Yocom DO  
Cooper University Hospital. Camden, NJ

## **Abstract**

**Study design:** Retrospective review of patients treated at a Level One Trauma Center for acute type II odontoid fracture using 2 different types of odontoid screws. Group A patients were treated with a standard lag screw and Group B patients were treated using an Acutrak 4/5 headless compression screw.

**Objective:** To evaluate the clinical and radiographic results of patients treated with an Acutrak 4/5 headless compression screw for acute type II odontoid fracture, and to demonstrate non-inferiority compared to standard technique.

**Background:** Type II odontoid fracture is a common cervical spine fracture that disproportionately afflicts the elderly. An anterior odontoid screw allows immediate fixation, preserves C1/2 motion, and is highly effective in fracture healing of acute fractures in patients with anatomically favorable fracture morphology and body habitus. The traditional approach uses a lag technique that can be technically challenging and invariably disrupts the C2/3 disk to some degree. The Acutrak 4/5 is a headless fully threaded variable pitch compression screw that has been described once in the literature to successfully treat an odontoid fracture. This is the largest case series on the use of this novel technique. This technique is simpler and allows for a steeper angle of approach, easier reduction, and can help minimize C2/3 disk disruption.

**Methods:** We retrospectively analyzed 44 patients between 2002 and 2014 who underwent an anterior odontoid fixation with either or a traditional lag screw (group A) or Acutrak 4/5 screw (group B) for stabilization of type II odontoid fractures, and report results of 30 patients with follow up data.

**Result:** We identified 14 patients who were treated using the lag screw technique and 16 patients using the Acutrak technique with follow up data. The mean radiographic follow up was 5.1 month and 3.1 month for the lag technique and Acutrak technique, respectively ( $p=0.082$ ). The mean clinical follow up was 6.6 months and 4.9 months, respectively, for Groups A and B, respectively ( $p=0.467$ ). The average age of patients who received the lag screw was 68, and Acutrak screw 79 ( $p=0.1$ ). Overall the fusion/stable fibrous union rate of Acutrak technique was 68% while for the lag technique was 78% ( $p=0.337$ ). Excluding two patients that we learned in retrospect were not good candidate for anterior approach yield as adjust rate of 75% for the Acutrak group ( $P=0.887$ ).

**Conclusion:** Odontoid screw fixation for acute type II odontoid fractures provides an acceptable rate of fracture healing and preserved C1/2 motion. The use of a headless fully threaded variable pitch compression screw such as the Acutrak 4/5 is technically easier and provides an equivalent outcome comparable to the conventional lag screw. Long term follow up with a larger patient cohort would be needed to assess the durability of this technique.

## **Introduction**

Odontoid fractures are common entities in the geriatric population. Fractures of the odontoid account for approximately 20% of all cervical fractures, and approximately 70% of these are type II fractures<sup>1</sup>. It involves a fracture through the base of the dens without involvement of the body. These are unstable fractures and treatment options are controversial as there are currently no standards of treatment or guidelines. Several risk factors have been associated with an increase likelihood that conservative treatment options may fail to achieve a stable fusion. Age > 50 has been associated with an 21 fold risk of increased non-union rate<sup>2</sup>. A fracture gap of > 2mm, odontoid displacement > 5 mm or the inability to obtain acceptable reduction and fracture alignment with a halo or collar are all associated with a high risk of bony non-union and may be indications for surgical fixation<sup>3</sup>.

Two basic surgical approaches for fixation of odontoid fractures include the anterior odontoid screw fixation and posterior cervical atlantoaxial fusion techniques. The optimal surgical approach depends on both clinical and radiographic factors. The anterior approach using an odontoid screw is an attractive option because of benefits such as direct osteosynthesis, preservation of C1/2 motion, and fusion rates ranging from 75 to 100 in all age group.<sup>4,5</sup> However, in the elderly, there is a high rate of failure of initial treatment<sup>6</sup> and the rate of nonunion in odontoid fixation has been reported as high as 75%<sup>7</sup>. Anterior odontoid fixation can be a technically challenging procedure but is a favored approach in patients with a fracture line in the anterosuperior to posteroinferior orientation with an intact transverse ligament, fractures < 6 months old and in patients without severe cervicothoracic kyphosis, osteoporosis or inappropriate body habitus (i.e. barrel chest).



Figure 1 Acutrak 4/5 headless compression screw

There is variability in the anterior odontoid screw fixation technique. Most surgeons use a partially threaded lag screw or a fully thread thru via a lag technique, where the proximal part of C2 is overdrilled, making a glide hole to reduce and stabilize the fracture. There are also cannulated systems using a K wire. The Acutrak 4/5 is a headless, fully-threaded, self tapping, cannulated, tapered screw with variable pitch designed to provide sufficient compressive force across fracture line with insertion (Figure 1). This type of screw is commonly used by orthopedic surgeons for the reduction of scaphoid fractures. In our institution, we use the lag screw for odontoid fixation and since 2009, the senior author (SY) and colleagues have been using a modified technique for odontoid fixation using the Acutrak 4/5 to treat selected patients with acute type II odontoid fractures with good results. There is only one previous case report published in 2013 describing the use of this screw for anterior odontoid fixation<sup>8</sup>. The purpose of this report is to critically analyze both clinical and radiographic outcomes of a series of patients treat with the Acutrak 4/5 screw in anterior odontoid fixation for Type II odontoid fractures compared to our own cohort of patients using the standard lag screw.

## Methods

We retrospectively reviewed data from 44 consecutive patients who were operated on in the Department of Neurosurgery at a regional level 1 Trauma center between the 2002 and 2014 using anterior odontoid screw fixation for traumatic type II odontoid fractures. The study was approved by the Cooper University Hospital IRB. Inclusion criteria include patients with traumatic odontoid fractures with pre and postoperative imaging and clinical follow up data. Pathologic fractures were excluded. Study parameters include age, gender, complications, surgeon, length of radiographic and clinical follow up, timing of surgery (immediate= within 3 days of injury). Management of patients was determined by the treating surgeon as to whether conservative therapy was offered first versus odontoid fixation. Follow up data was obtained from clinic notes and/or radiographic data. Assessment of good outcome was assessed clinically and/or radiographically. Good result included freedom from pain and neurological symptoms with full range of movement in all direction, lack of abnormal motion on postoperative flexion extension x-rays, or evidence of bony fusion. Poor result includes requirement of revision surgery, refractory pain, clinical myelopathy, and/or demonstrated unstable nonunion on flexion extension x-rays.

### Surgical procedure

In brief, all patients were consented for surgery with benefits, risks, and alternatives fully explained. The operation was done in a supine position. General anesthesia was administered. The surgical technique for the exposure is the same for both techniques. A transverse incision along a fold in the neck just medial to the border of the sternocleidomastoid was made. Using Metzenbaum scissors, the skin was undermined. Prevertebral space was exposed using sharp dissection and carried up just to the bottom of C2.

For the standard technique with the lag screw, 2 C-arm were positioned to provide intra-operative fluoroscopic images in opened mouth AP and lateral views centered at C2. A trough was made at the C2/3 disk space with an small annulotomy. An awl was used to make a starting point at the inferior anterior corner of C2. This was then drilled with 2.5mm bit through the distal tip of the fractured fragment for a pilot hole. Then a 3.5mm tap was used to broach nearly the length the C2 fragment followed by a 3.5 to 4.0mm partially threaded lag screwed until it was fully engaged in the fragment with reduction on x-ray. The length of the screw was carefully calculated so that the threads will be distal to fracture line when the screwed is fully tightened in the fragment. The wound is inspected for bleeding and closed in standard fashion.

For the Acutrak technique, except for 3 cases done before 2009, the AP and lateral fluoroscopy was replaced by the O-Arm. After manual reduction prior to fixation, a scan was done to ensure adequate alignment and reduction. The procedure is essentially the same except there is minimal to no C2/3 annulotomy. There was no need to premeasure of the length of screw to ensure the thread will be distal to the fracture line. Typically, a 30mm to 35mm screw was used to span the length of the C2. The pilot hole is drilled using a generic 3.2mm bit drill. We do not use a guidewire or the standard Acutrak cannulated drill. Following the tap of only 1-2cm of proximal C2, a 4/5 Acutrak screw is tightened into the fracture fragment until satisfactory

reduction as it slightly countersinks into the body of C2. To help overcome the shallow angle needed in patients with large chest, we use a customize-made flexible hex screw driver. An intraoperative O-arm spin was then done to verify accuracy of screw placement. If satisfactory, the wound is then closed. The patient is placed in a rigid C-collar. Usually, A CT cervical spine was done by postop day 1.

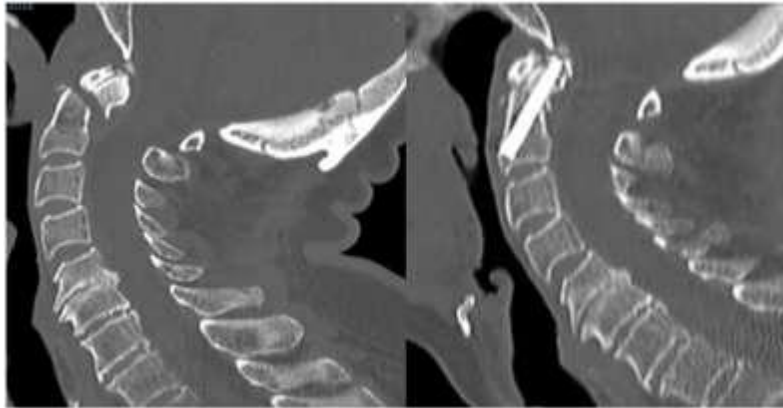


Figure 2 example of a well placed Acutrak screw. Note the proximal aspect of the screw does not disrupt the C2/3 disk space.

#### Statistical analysis

The following information was extracted from the medical record: preoperative neurologic status, surgical complications, symptoms and neurological status at the time of the most recent follow-up along with imaging studies. The patient returned to the outpatient office at 2, 6 and 12 weeks, sometimes 6 months and 12 month post-op depending on the treating surgeon.

Statistical analysis was done by comparing two groups: group A being the lag screw technique cases, and Group B the Acutrak technique cases. The T test was used to compare average age as well as average length of follow up between the lag screw and the Acutrak screw technique cases. Statistical analysis was done comparing results of the surgery, this was done using the Chi Squared test. Univariable analysis was done to test effect of gender, treating surgeon, age at time of operation above or less than 70, timing of surgery (delayed surgery greater 6 week post injury versus early surgery), all using the Chi Squared test.

#### Radiographic interpretation

Image data was interpreted by the surgeon, the lead author, and the reading radiologist. In cases where radiographic imaging were not available, the data was obtained by official read and/or surgeon's interpretation from clinic notes.

### Results

#### Patient demographics

We identified 44 patients that underwent an anterior odontoid fixation for traumatic unstable odontoid fractures at our institution between 2002 and 2014. There were 22 cases that was identified as using the lag screw technique, 19 cases using the Acutrak screw technique, and

three patients there were no data on which screw was used. We found follow-up data on 30 cases. 16 cases were Acutrak cases, 14 were lag screw case.

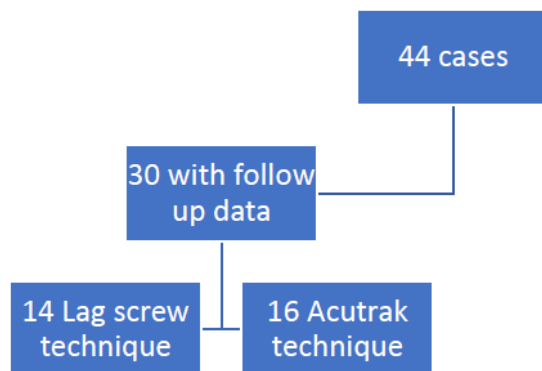


Figure 3 chart search

	Group A:Lag screw	Group B: Acutrak screw	p value
average age	68	79	0.1
mean radiographic follow up	5.1 m	3.1 m	0.082
mean clinical	6.6 m	4.9 m	0.467
female	10 (71%)	8 (50%)	0.23
male	4 (29%)	8 (50%)	
surgeon: SY	9 (75%)	13 (81%)	0.432
surgeon: AT	3 (25%)	2 (12.5%)	
delayed surgery	1 (7%)	3 (18.8%)	0.35
immediate surgery	13 (93%)	13 (71.2%)	

Table 1 patient characteristic between the group A and group B

The average age of patients who received the lag screw was 68, and Acutrak screw 79 with p value of 0.1. The range of age was from 19 to 90. The mean radiographic follow up was 5.1 month and 3.1 month for the lag technique and Acutrak techniques, respectively (p=0.082). The mean clinical follow up was 6.6 month and 4.9 month, respectively (p=0.467). There was also no significant difference in gender between the two groups (p=0.23). There were a total of 6 different neurosurgeons who operated on these 30 patients. However, all but 4 cases were operated on by two senior surgeons, SY and AT. There was no significant difference between the two groups in terms of who the surgeon was. Lastly, there was no significant difference between the two groups in terms of timing of surgery from initial injury. All but two patients were neurologically intact. Patient 19 presented with spinal cord injury. Patient 24 had concomitant severe TBI. Patient 30 was neurologically intact but had a delayed failure of



surgery with loss of reduction and symptomatic instability. He required a posterior C1-2 fusion and did well after.

Patient number	Year	Gender	Age	Results	Complications	Screw type	Subluxation	Imaging following	Clinical follow up	Timing of surgery	Surgeon
1	2002	female	87	good		lag screw		3 months	3 months	immediate*	MT
2	2002	female	89	good/fibrous union		lag screw		6 months	6 months	immediate	AT
3	2002	male	86	good/fusion		lag screw		12 months	12 months	immediate	SY
4	2002	female	82	non union		lag screw		3 months	3 months	immediate	SY
5	2002	female	86	good/fibrous union		lag screw		3 months	12 months	immediate	SY
6	2006	female	85	good/fusion		lag screw		1.5 months	3 months	immediate	JS
7	2006	male	73	good/fibrous union		lag screw		12 months	12 months	immediate*	SY
8	2006	female	69	poor fixation/non union		2 lag screw	yes, retro	4 years		6 week	SY
9	2006	female	89	good/fibrous union		lag screw	yes, retro	12 months	12 months	1 week	SY
10	2008	male	82	good		lag screw	yes, antero	3 months	3 months	immediate*	SY
11	2008	male	47	delayed failure requiring posterior fusion	delayed failure	lag screw		3 month*	3 month*	8 month	SY
12	2008	female	78	good/fusion		2 lag screw	yes, slight retro	3 months	6 months	immediate	AT
13	2009	female	61	good/fusion		lag screw	yes, retro	4 months	8 months	immediate	AT
14	2009	female	87	good		lag screw	no	1.5 months	3 months	immediate	SY

Table 2 Group A: Patient data use lag screw technique

Patient number	Year	Gender	Age	Results	Complications	Screw type	Subluxation	Imaging follow	Clinical follow up	Timing of surgery	Surgeon
15	2008	male	77	non union	trached	Acutrak		4 months		Immediate	SY
16	2009	female	90	good		Acutrak	yes, retro	2 months		Immediate	SY
17	2009	male	74	good/fibrous union		Acutrak	no	8 months	6 months	4 weeks	SY
18	2009	female	63	loss of reduction, required posterior fusion		Acutrak	yes, retro	2 weeks	3 weeks	Immediate	AT
19*	2009	female	89	good/fibrous non union		Acutrak	yes, retro	3 months	2 years	Immediate	SY
20	2010	male	88	loss of reduction/requiring fusion		Acutrak	yes, retro	2 months	2 months	Immediate	SW
21	2010	male	75	good/fibrous union	screw pull out/incidental 6 years later	Acutrak		3 months	3 months	11 weeks	JB
22	2011	female	71	good/fusion	peg	Acutrak		4.5 year	3 months	Immediate	SY
23	2011	male	27	good/fusion		Acutrak		3 months	3 months	Immediate	SY
24*	2011	female	20	failure to reduce/Type III fx		Acutrak		3 months	3 months	Immediate	SY
25	2012	female	81	good		Acutrak		3 months	3 months	Immediate	SY
26	2012	male	17	technical failure to purchase bone		Acutrak	yes, antero	3 months	3 months	immediate	SY
27	2012	male	67	good		Acutrak	yes, retro	3 months	3 months	immediate	SY
28	2013	female	88	good		Acutrak	yes, retro	3 months	3 months	immediate	AT
29	2014	female	80	good		Acutrak		3 months	3 months	immediate	SY
30*	2014	male	73	delayed loss of reduction requiring posterior fusion		Acutrak		1.5 months	1.5 months	6 weeks	SY

Table 3 Group B: Patient data of Acutrak screw cases

Result	Group A: Lag screw	Group B: Acutrak screw	Excluding Pt 24 and 26
good/fusion/stable fibrous union	11 (78%)	10 (63%)	12 (75%)
poor/non union	3 (22%)	6 (37%)	4 (25%)
p value	0.337		0.817

#### Table 4 Surgical outcome

The results of surgery show a good outcome, defined as either fusion or stable fibrous union, in 11 patients (78%) and 10 patients (63%) of the lag screw and Acutrak screw, respectively. This difference is not statistically significant ( $p=0.337$ ). Patient #24 had failure of the operation to reduce the fracture and required a Halo followed by a posterior reduction and fusion. However, this was a type III odontoid fracture with significant ligamentous injury and rotatory and shear component. We believe this fracture would not be adequately reduced from an anterior approach. Patient #26 had an anteriorly displaced coronally oblique fracture that during surgery the screw could not get purchase to the distal fragment and maintain reduction. This case was aborted and the patient was placed in a halo. We believe this was due to the incompatibility of the fragment geometry with anterior reduction approach as well. Excluding patients #24 and #26 yielded a success rate of Acutrak group of 75% ( $P=0.817$ ). Other failures from both approaches include loss of reduction and delayed need for posterior fusion. Patient# 21 had a good result at 3 months using the Acutrak technique, and 6 years later the screw was observed to have partially backed out as an incidental, asymptomatic finding. The patient was offered surgery to remove the screw.

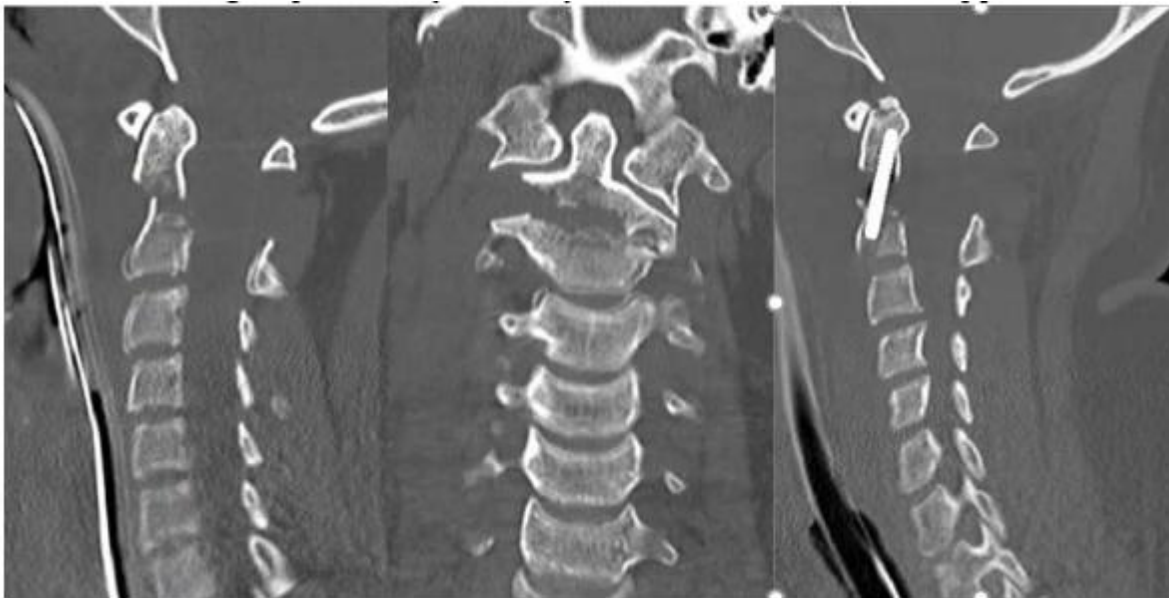


Figure 4 patient #24, type III fracture, unreducible with odontoid screw



Figure 5 Patient #24 after reduction and fixation posteriorly

#### Variables and rates of fusion

Since there was no statistical difference in outcome between the two techniques after adjusting for case selection, we looked at several variables that may have an impact with success of surgery. Specifically, timing of surgery, gender, age greater or less than 70, and surgeon who performed the operation were considered in a univariate fashion.

There was a statistically significant difference in outcome if surgery was delayed greater than 6 week from injury, with only 1 union out 4 cases,  $P=0.019$ . Excluding patient #24 and #26, who are retrospectively deemed technically infeasible for the anterior approach, who both happened to be young, 19 and 17, respectively, age was not a statistically significant variable for fusion rates in this cohort of patient ( $p=0.11$ ). Also, the choice of surgeon ( $p=0.561$ ) nor the patient gender ( $p=0.253$ ) were statistically associated with either outcome.

	Union	Non-union		p
delayed surgery	1	3		0.019
Immediate surgery	21	5		
age>70	18	4		0.111
age<70*	3	3		
Surgeon SY	14	7		0.561

Surgeon AT	4	1		
female	14	4		
male	7	5		0.253

Table 5 Variable and fusion rates

## Discussion

The objective of this paper was to review the clinical and radiographic outcome of the use of the Acutrak 4/5 headless compression screw for unstable type II odontoid fractures at a busy Level 1 Trauma Center. Although fixation of type II odontoid fracture using an anterior odontoid screw is an attractive option with benefits such as direct osteosynthesis and preservation of C1/2 motion, there remain technical and intrinsic fracture patterns that are challenges to successful surgical results. The ideal screw requires “bicortical” purchase from the anterior-inferior C2 to apical cortex. It requires the threaded part of screw to be placed distal to the fracture line if using partially threaded lag screw, and there should be reduction/compression across strong cortical bone. If using a fully threaded screw using a lag technique, the proximal bone need to be over-drilled, ie. a glide hole. The technical challenges include: sizing the correct thread length of lag screw, fully thread screws technique require overdrilling of the proximal C2, small risk of K-wire bending or penetration beyond distal cortex to brain stem in cannulated systems, difficult body habitus such as barrel chest/kyphotic c-spine, and fracture geometry. Poor nutritional status, osteoporosis, age, and delayed timing of surgery has also been cited in the literature for decreased union/healing<sup>9</sup>. Although our study was probably underpowered to show the effect of patients’ age on fusion, it confirmed delayed surgical intervention > 6week from injury was associated with lower rate of fusion (p=0.019).

Theoretical pitfall of anterior odontoid screw includes adjacent segment disruption due to the need for C3 body drilling and C2/3 disk disruption.. Due to its variable pitch of the thread that is proximally shallower and distally more aggressive, as the screw is tightened it functionally acts like a lag screw and results in reduction and compression of the fracture fragment without having to calculate precise measurements dependent on a predicted amount of reduction This screw been widely used in many orthopedic applications including scaphoid fracture. Its headless design allows the screw to be countersunk flush with or even beneath the cortex thus minimizing its profile and decreasing soft tissue irritation. When used as an odontoid screw in our modified technique, there are some notable advantages over the traditional method:

- \* no need to size the threads as needed for a lag screw
- \* no need to overdrill C2 body proximal to fracture as needed in the lag technique
- \* there is bony purchase through the entire length of the screw which is presumed to enhance stiffness
- \* we have found the Acutrak screw to provide more effective reduction of the fracture fragment intraoperatively
- \* the use of the O-arm enhances visualization of the adequacy of pre fixation reduction and post fixation screw accuracy

\* The headless design allows starting point to be more anterior and steeper thus minimizing the need for C2/3 disk disruption and improving the approach angle which may allow instrumentation in barrel chested and kyphotic patients

In this study, we do not have the data to prove its superiority over the traditional method. However, the senior author has used the Acutrak technique exclusively since 2009 due to relative technical ease and advantages without sacrificing good results. As a result, on average the lag screw was used in 3.2 cases per year while the Acutrak screw was used in 3.8 cases a year. We do not have the prevalence data of all type II odontoid fracture patients to assess if the increased use is truly due to better applicability versus increase incidence of the injury being treated in our institution. As seen in table 1, the two groups being compared have similar patient demographics. The actual surgical good result/fusion rate of Acutrak technique was 75% when the 2 patients that we have learned we would not consider odontoid screw candidates because of their injury types were excluded from analysis. Compared to the lag screw technique result of 78%, with a p value of 0.817, this study suggest that there is no statically significant difference in outcome between the two groups . It is also consistent with published rates in the literature and very good for the elderly.<sup>4,5,7</sup>

Biomechanically, the Acutrak screw has been shown to be superior. Wheeler et al. published a study that evaluated and compared the mechanical strength of the Acutrak screw with an AO 4-mm cancellous screw using anatomic cancellous specimen bone and cancellous bonelike foam. This study found that the Acutrak was able to maintain compression after cyclic loading significantly better than the AO 4-mm cancellous screw .The torque that was required to break fragment contact was also significantly greater for the Acutrak screw compared to 2 other screw types.<sup>10</sup> In 2007, Magee et al. published a paper that compared stiffness and load to failure in human cadavers with Type II odontoid fractures that were stabilized with either a lag or an Acutrak screw. This study found that the stiffness and load to failure were greater for the Acutrak models compared to the lag screw<sup>11</sup> The only clinical report of the use the Acutrak in odontoid fracture comes from Tonosu et al<sup>8</sup>. They reported 1 case in using the Acutrak 4/5 headless compression screw for anterior odontoid fixation in a patient with osteopenia with an acute Type II odontoid fracture. Her 3 month follow-up CT cervical spine showed bone union of C2 with no deformity. A CT cervical spine taken 3 years post fixation continued to show bony union with no degenerative changes. Our results show similar results in using the Acutrak 4/5 headless compression screw for anterior odontoid fixation.

Limitations of our study include retrospective analysis, relative small sample size and short follow-up period, lack of complete and consistent follow up data, especially on complications. Radiologic assessment by the treating physician and lead author is prone to observer bias. The long term complication of a screw backing out after 6 years is a potential issue, and long term follow up imaging may be necessary.

## **Conclusion**

A good technical understanding of anterior odontoid fixation for Type II odontoid fractures is imperative as the frequency of these fractures become more common in our aging population.

Our study suggest that the Acutrak 4/5 headless compression can provide equivalent outcome in selected anterior odontoid fixation for acute Type II fractures as compared to our cohort of patient using the traditional lag technique. Larger prospective, randomized trials with long-term follow-up would better determine the efficacy in using the Acutrak 4/5 screw in the anterior odontoid fixation of Type II odontoid fractures.

## References

1. Anderson LD, D'Alonzo RT, 1974. "Fractures of the odontoid process of the axis." J Bone Joint Surg Am 56 (8): 1663-74
2. Lennarson PJ, Mostafavi H, Traynelis VC, Walters BC. Management of type II dens fractures: a case-control study. Spine 2000;25(10):1234–123
3. Chi YL, Wang XY, Xu HZ, et al. : Management of odontoid fractures with percutaneous anterior odontoid screw fixation. Eur Spine J. 2007;16(8):1157–1164
4. Börm W, Kast E, Richter HP, Mohr K. Anterior screw fixation in type II odontoid fractures: is there a difference in outcome between age groups? Neurosurgery 2003;52:1089–1092, discussion 1092–1094
5. Alfieri A. Single-screw fixation for acute type II odontoid fracture. J Neurosurg Sci 2001;45:15–18
6. Hart R, Saterbak A, Rapp T, Clark C. Nonoperative management of dens fracture nonunion in elderly patients without myelopathy. Spine 2025;25:1339–1343
7. Scheyerer MJ, Zimmermann SM, Simmen HP, Wanner GA, Werner CM. Treatment modality in type II odontoid fractures defines the outcome in elderly patients. BMC Surg. 2013 Nov 9;13:54
8. Tonosu J, Watanabe K, Abe H, Higashikawa A, Kato S, Yamada K (2013) Anterior screw fixation for an odontoid fracture using an Acutrak 4/5 screw: A Case Report. Springer
9. Apfelbaum RI, Lonser RR, Veres R, Casey A. Direct anterior screw fixation for recent and remote odontoid fractures. J Neurosurg. 2000;93(Suppl):S227–S236.
10. Wheeler DL, McLoughlin SW (1998) Biomechanical assessment of compression screws. Clin Orthop Relat Res 350:237-245
11. Magee W, Hettwer W, Badra M, Bay B, Hart R (2007) Biomechanical comparison of a fully threaded, variable pitch screw and a partially threaded lag screw for internal fixation of Type II dens fractures. Spine 32 (17): E475-E479