Neurosurgical Forum

LETTERS TO THE EDITOR

Can muscle hypertrophy cause entrapment neuropathy?

TO THE EDITOR: I read with interest the article by Wilson et al.⁸ (Wilson TJ, Tubbs RS, Yang LJS: The anconeus epitrochlearis muscle may protect against the development of cubital tunnel syndrome: a preliminary study. *J Neurosurg* [epub ahead of print February 12, 2016. DOI: 10.3171/2015.10.JNS151668]).

The cubital tunnel is formed by a groove between the olecranon and the medial epicondyle (ME), usually covered by a fascial sheet known as the cubital tunnel retinaculum (i.e., the Osborne fascia).⁵ Occasionally the cubital tunnel retinaculum is replaced by a muscle tissue—i.e., the anconeus epitrochlearis muscle that is often regarded as a cause of ulnar neuropathy at the elbow (UNE) In their report, Wilson et al.⁸ retrospectively examined the prevalence of this muscle found during surgery for the UNE and compared it to the prevalence in arms of control patients reported in the literature. In their series of 168 UNE patients, they found a prevalence of 5.4%, which was significantly lower ($p < 0.001$) than the 15.5% reported in 634 control arms from previous MRI and cadaver studies.⁸ The authors concluded that the presence of the anconeus epitrochlearis muscle seems an unlikely causative factor for UNE. Furthermore, they argued that its presence may actually have a protective role against the UNE.⁸

In our series of 221 patients we found UNE to consist of 2 focal neuropathies.^{3,4} The first occurs 2–3 cm distal to the ME. Because we found ulnar nerve constriction on ultrasonography in 38% of these arms,³ we concluded that this type of UNE is caused by ulnar nerve entrapment under the humeroulnar aponeurosis (HUA),³ which is the distal expansion of the cubital tunnel retinaculum. As we found UNE at this location mainly in dominant arms of older farmers, miners, carpenters, and so on,⁴ we reasoned that in these patients UNE is caused by decades of hard manual labor that transforms the HUA into a thick fibrous band. Because the anconeus epitrochlearis muscle stretches 2–3 cm proximal to the HUA, it is difficult to attribute to it any role in the pathogenesis of this UNE variety, known in the literature as “a cubital tunnel syndrome.”⁵

The second, more common type of UNE occurring in 85% of arms in our series was found at the ME or up to 2 cm proximal to it in the retroepicondylar (RTC) groove.³

We observed it almost exclusively in the nondominant arms of younger administrative workers and students.⁴ As no nerve constriction was found in any of the arms affected at this location,³ we believe that in these arms, ulnar neuropathy is caused by exogenous nerve compression, probably most importantly while using a computer mouse with the dominant arm and leaving the nondominant arm lying pronated on the desk.⁴ Because the anconeus epitrochlearis stretches between the ME and the olecranon, it may well protect the ulnar nerve at this location.

I agree with Wilson et al. that “entrapment neuropathies commonly occur as nerves pass beneath rigid ligamentous structures,”⁸ as this is also the case with ulnar nerve entrapment under the HUA (i.e., the cubital tunnel syndrome). However, I am reluctant to accept their suggestion of “a much rarer scenario involving compression of a nerve by a hypertrophied muscle secondary to overuse.”⁸ My opinion is based both on my personal experience and on my general reasoning. Although at my institution over 8000 electrodiagnostic evaluations are performed each year, in more than 20 years of practice I have not come across a single patient with a nerve entrapment unequivocally proven to be caused by muscle hypertrophy (e.g., pronator teres syndrome). Moreover, I am reluctant to believe that a rather soft hypertrophic muscle may actually cause entrapment of a much tougher peripheral nerve. In my opinion nerve entrapment within or beneath the muscle can only occur due to tough fibrous bands. As we did not find ulnar nerve entrapment proximal to the RTC (i.e., under the arcade of Struthers) or distal to the HUA (i.e., at the deep flexor pronator aponeurosis)⁵ in any of our 221 UNE patients, entrapment by tough fibrous bands is, at least for the ulnar nerve, probably much rarer than their frequent citation in the literature would suggest.

Returning to the role of the anconeus epitrochlearis muscle, it is easy to imagine that an additional layer of muscle overlying the ulnar nerve in the bony RTC groove might provide some degree of protection against the external compression, as the results of Wilson et al. suggest.⁸ However, strictly speaking, the anconeus epitrochlearis does not protect the ulnar nerve against cubital tunnel syndrome⁵ but, rather, against much more common external compression in the RTC groove.

It is interesting to note that the role of the anconeus epitrochlearis in the causation of UNE in many respects reflects a similar recent evolution in our understanding

of another factor traditionally believed to cause UNE, ulnar nerve dislocation (i.e., luxation) at the elbow.¹ As for the anconeus epitrochlearis, recent studies also found a similar occurrence of ulnar nerve dislocation in arms with and without UNE.^{2,7} To underscore similarity even further, there are also some data indicating that complete ulnar nerve dislocation may even have a protective effect. It may function as a natural equivalent of surgical anterior transposition of the ulnar nerve, effectively relieving ulnar nerve strain during protracted elbow flexion.⁶

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Disclosures

The author reports no conflict of interest.

Response

No response was received from the authors of the original article.

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Published online July 15, 2016; DOI: 10.3171/2016.3.JNS16612.

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Cranioplasty after decompressive craniectomy

TO THE EDITOR: We read with keen interest the article by Schwarz et al.⁴ (Schwarz F, Dünisch P, Walter J, et al: Cranioplasty after decompressive craniectomy: is there a rationale for an initial artificial bone-substitute implant? A single-center experience after 631 procedures. *J Neurosurg* 124:710–715, March 2016).

Decompressive craniectomy is a life-saving option for patients with medically refractory elevated intracranial pressure due to various pathologies. Cranioplasty using an autologous bone flap or artificial bone graft substitute is done for cosmetic, mechanical, and therapeutic purposes. The authors reported statistically significant higher rates of bone resorption in the cohort of patients who underwent cranioplasty using autologous bone graft compared to those in whom an artificial bone substitute was used. However, the authors reported a lower risk of complications when bone resorption was not taken into account as a complication. The authors concluded that younger age (< 30 years age), shunt dependency, and bone flap fragmentation are independent risk factors for bone resorption and need for reoperation, and they recommended initial artificial bone substitute in patients younger than 30 years of age.

Wound infection after cranioplasty is a known factor associated with bone flap resorption.³ The rate of infection following cranioplasty is quite high, up to 10%.¹ Hence, it will be important to know the infection rates in the reported series to eliminate the confounding effect of infection.

Resorption of the bone flap is a major issue in the pediatric age group following cranioplasty, especially in young children up to an age of 7 years.² The authors described 3 age groups (0–30, 31–60, and 61–90 years) for comparison. It would probably be more helpful for the authors to describe the number of pediatric patients and offer a comparison between pediatric and adult patients.

The follow-up duration was 6 months in a majority of patients, and a long-term follow-up period is warranted to establish a true picture of the complications of autologous bone and artificial bone graft, because complications as late as 6 years postoperatively have been reported following cranioplasty.^{1,5}

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Disclosures

The authors report no conflict of interest.

Response

We thank Dr. Dash and colleagues for their careful analysis of our paper.

First, they state that the rate of wound infection after cranioplasty is quite high and such infection is a known risk factor for bone flap necrosis. In our study only 1 patient had a wound infection that resulted in a microbiologically confirmed infection of the bone flap. On this basis, we do not share the opinion of Dash and colleagues.

Second, the writers state that bone flap resorption is a major issue in children up to 7 years of age. After reviewing our data, only 3 relevant patients with 4 surgeries were identified. Two patients experienced bone flap resorption, and both had additional risk factors for bone flap resorption, like multi-fragmented bone or a ventriculoperitoneal shunt in situ. Due to the low number of patients in this group, statistical analysis is not possible.

Another interesting aspect recognized by Dash and colleagues was the issue of follow-up. The median follow-up period in our cohort was 6 months. We share their view that the rate of bone flap necrosis rises with a longer follow-up. To this end, we will reevaluate this study cohort in a few years, to obtain a longer-term view of patient outcomes.

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INCLUDE WHEN CITING

Published online September 30, 2016; DOI: 10.3171/2016.5.JNS161142.

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Sudden death following cranioplasty: vacuum suction to blame?

TO THE EDITOR: We read with interest the article by Sviri³ (Sviri GE: Massive cerebral swelling immediately after cranioplasty, a fatal and unpredictable complication: report of 4 cases. *J Neurosurg* 123:1188–1193, November 2015) and the recent correspondence by Dr. Honeybul² regarding the article, in which Dr. Sviri reported a case series of 3 patients who suffered from sudden death after cranioplasty.

An interesting observation made by Dr. Sviri was with regard to the use of closed vacuum suction drains, which he believed contributed to sudden shifts in intracranial pressure (ICP). We would also like to share our experi-

FIG. 1. Axial (A–C, E and F) and 3D reconstructed (D) head CT scans. **A:** Initial scan performed demonstrating a large, left-sided basal ganglia ICH that was surgically evacuated. **B:** Postsurgical evacuation of ICH. **C:** Scan obtained 2 months after evacuation showing the sunken craniectomy site. **D:** 3D planning scan for cranioplasty plate fabrication. **E:** Scan after cranioplasty in the immediate postoperative period. **F:** Scan after removal of titanium cranioplasty and bilateral decompressive craniectomies.

ence with a similar case in a 31-year-old, previously fit and well man, admitted to our institution with a large left-sided basal ganglia intracerebral hemorrhage secondary to amphetamine use (Fig. 1). This patient underwent evacuation of a left-sided basal ganglia intracerebral hematoma (ICH) followed by decompressive craniectomy after an uncontrolled rise in ICP. He eventually recovered well with a dense right-sided weakness but was conversant and obeying commands. The patient underwent an elective titanium cranioplasty at 14 months and the procedure itself was uncomplicated. Intraoperatively, the brain was noted to be slack and a closed vacuum suction drain was placed in the subgaleal space prior to closure.

Postoperatively, the patient was slow to recover consciousness and a repeat CT scan and CT angiography demonstrated widespread diffuse brain edema with no evidence of carotid dissection or venous infarction. The patient was taken back to the operating room for bilateral decompressive craniectomies, but the ICP continued to rise despite medical and surgical management, and the patient died shortly after. A postmortem examination was performed that demonstrated extensive thrombosis of the superior sagittal sinus, but it was believed that this was a consequence of the massive cerebral swelling rather than the primary cause.

There are several features of this case that were similar to those described by Dr. Sviri. Our patient was a young man with a significantly sunken craniectomy site at the time of surgery; our case differs only in the technical aspect of bone flap reconstruction (we used a 3D titanium plate rather than autologous bone). Dr. Honeybul recently also wrote to propose impaired cerebral autoregulation as a cause of massive cerebral swelling. While we agree that patients with severe cerebral insults are likely to have impaired autoregulation, the fact that sudden-onset massive swelling after cranioplasty (fortunately) remains rarely observed indicates additional factors at work beyond impaired autoregulation.¹

It is also interesting to note that 11 of 13 cases (including this case) reported in the literature were young males who underwent decompressive craniectomies from a variety of insults. This would appear to lend further tangential evidence of a clinical syndrome in young males. Although we have no definitive evidence that suction drainage played a role in inducing cerebral edema, we support the view that that significant shift in the intracranial compartment pressure brought about by cranioplasty and suction drainage may play a role in cerebral edema⁴ and thus we also have changed our practice and advocate turning on gentle suction drainage only after the patient has awakened postoperatively.

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Disclosures

The authors report no conflict of interest.

Response

I would like to thank Kenny K. H. Yu and Kaushik Ghosh for their response and case presentation. The case presented by Yu and Ghosh is a case of sudden death following cranioplasty surgery in a patient who had a significant sunken brain and showed remarkable recovery after intracerebral hemorrhage. They suggested this syndrome mainly involved young male patients and that impaired regulation is not solely responsible for the massive brain swelling after cranioplasty.

It is unclear whether autoregulation impairments are associated with this syndrome, and there is no way to prove it. I can speculate that the atmospheric pressure prevents the brain from resuming a normal autoregulation balance, but as it is a very rare complication I am not sure this impairment can apply to all patients after decompressive craniectomy. Definitely, this syndrome is associated with elimination of the atmospheric pressure applied on the brain for a long period of time and it might also be aggravated with the vacuum effect of the draining system.^{1,2} As for the prevalence of male patients in this syndrome, I must say that most of the patients who undergo decompressive craniectomy are male as they are obviously much more involved than females in accidents and violent activity. Therefore, I am not sure we can presume that this syndrome is typical for young males.

This syndrome is fatal and the outcome is devastating both to family members as well as to the surgeon. Therefore it is important to acknowledge it and to discuss it with patients and family members prior to surgery. I personally found myself subjected to two lawsuits and two investigations by the Ministry of Health looking for negligence, and these cases shadow my career as a young attending neurosurgeon. I do hope my paper as well as your case presentation will support other “unlucky” neurosurgeons in understanding this fatal, unexpected complication.

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INCLUDE WHEN CITING

Published online October 7, 2016; DOI: 10.3171/2016.4.JNS16977.

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Neurosurgery skills training laboratories and curriculum: a supplement to Halstedian practice

TO THE EDITOR: We have read with great interest the article by Liu and colleagues¹ (Liu JKC, Kshetty VR, Recinos PF, et al: Establishing a surgical skills laboratory and dissection curriculum for neurosurgical residency training. *J Neurosurg* 123:1331–1338, November 2015). The authors aptly described the practical problems and their solutions for establishing a skills training cadaver laboratory to train neurosurgery residents and visiting fellows in a premier academic institution. They also highlighted the need for a curriculum for residents as per their competency and seniority level in the training. In our earlier publications, we have also shared our experience in establishing a skills training cadaver laboratory, along with details of its infrastructure and the design and follow-up response of low- and high-fidelity skills training modules to supplement a traditional Halstedian training program with competency-based training of neurosurgery residents in the All India Institute of Medical Sciences (AIIMS), New Delhi, India. The Neurosurgery Education and Training School (NETS) at AIIMS was established in 2008 with interdepartmental and interinstitutional collaboration. Skills training modules consist of structured modular approaches for neuroanatomy, microneurosurgery, neuroendoscopy, high-speed drilling, and spine instrumentation. Modules are categorized into task-based submodules and procedure-based submodules for the purpose of standardization and validation.^{2,4,5} Having shared the same experience, we recommend our papers to the authors.^{2–9}

In this era of minimally invasive endoscopic, microscopic, and robotic surgery, several forces are making it difficult for operating theaters to be the predominant venue for the acquisition of primary technical skills. Increasing financial and medicolegal constraints, increasing demand for maximum possible proficiency in surgical care, and a decreasing patient/resident ratio have adversely affected neurosurgical training. Cadaver dissection is the closest simulation of the real operating experience. The authors should be congratulated for their effort in establishing such a laboratory since it demands significant interdepartmental collaboration, financial assistance from intramural and extramural resources, and manpower to manage equipment and record keeping. The first step of finding a proper place and procuring donated and refurbished instruments was the most difficult task in our experience too, but we have realized that most of the surgical exposures can be done with the basic surgical set and a decent microscope. The authors' detailed description of the especially efficient utilization of the cadaver for maximum possible learning and a seniority-based training schedule will be helpful in the standardization of the training curriculum. They have shared their valuable experience in procuring instrument

sets, which will be significantly useful for establishing such laboratories in other teaching institutions as well.¹

One specific concern with such a training curriculum is the objective assessment of its translation into real surgical practice. In our experience, the residents and fellows believed that they most benefitted from practice in high-speed drilling, followed by practice in microsurgery and endoscopy. Since the evaluation parameters were subjective, we agree with the authors' belief in the need for more experience and objective evaluation for their validation. Positive results from the incorporation of a skills training curriculum can be regarded as a value base for such programs. This would help in achieving the professionalism imperative for high-end branches like neurosurgery and proven competency at the end of training program.^{2,3–5}

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Disclosures

The authors report no conflict of interest.

Response

We thank Drs. Suri and Tripathi for their interest in and

evaluation of our article. We note that the authors' papers on their experience with the NETS in the All India Institute of Medical Sciences came to press shortly after the submission of our article.^{1,2} We cordially acknowledge the authors' work in carefully detailing the establishment of a surgical skills laboratory, from obtaining and preserving cadaveric specimens to acquiring the necessary surgical instruments.² In addition, the authors have described the use of NETS in creating a simulation protocol and a training center for both practicing neurosurgeons and neurosurgeons in training.¹ Whereas our paper aimed to provide individual training programs with a template for establishing a thorough surgical skills curriculum with the efficient use of minimal funding, the authors have demonstrated the possibilities of what can be achieved with assistance from federal funding. With this effort, the authors have taken a large step toward the creation of a nationwide standardized system of neurosurgical skills training.

As the authors note, the next step in the development of skills training is an objective form of assessment. Surveys from trainees and faculty, such as those reported in our paper as well as in the communication noted by the authors, are subjective and cannot be easily translated into quantifiable means of surgical skills improvement. After several years of experience with the surgical skills curriculum, we have noted that there is a distinction between the curriculum providing a benefit for preparation and practice of rare complex approaches, and the development of manual surgical dexterity. Although cadaveric dissection is highly useful for the development of basic surgical techniques, it has been traditionally seen as a means of refining more advanced and difficult approaches. It allows for a more complete training experience, but gaps in comfort with various approaches will probably remain. It is impractical to expect training programs to provide neurosurgical trainees with adequate exposure to every type of procedure that they may encounter in their future practice. Providing a nationwide training center and curriculum similar to the one described by the authors is a step toward closing that gap. Since the resources necessary to develop a standardized nationwide system would be significant and probably only possible in the distant future, we propose a different approach in that the surgical skills lab is utilized as a means of quantifying the development of surgical dexterity. Using dexterity as a benchmark for surgical skills development may be a better method of demonstrating that the trainee has developed the skill sets necessary to perform unfamiliar surgical cases in their future practice. We are currently working on the development of objective tools to quantify surgical dexterity and how it correlates with progression in a training program and application in the operating room.

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INCLUDE WHEN CITING

Published online September 30, 2016; DOI: 10.3171/2016.4.JNS161010.

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Enlargement of the middle meningeal artery

TO THE EDITOR: We read with keen interest the article by Takizawa et al.¹⁰ (Takizawa K, Sorimachi T, Ishizaka H, et al: Enlargement of the middle meningeal artery on MR angiography in chronic subdural hematoma. *J Neurosurg* **124**:1679–1683, June 2016) regarding the increased diameter of the middle meningeal artery (MMA) in patients with chronic subdural hematoma (CSDH). We commend the authors for their study.

It would be interesting to know whether MR angiography (MRA) is performed in all patients with CSDH as a routine at the authors' center or if there were some specific reasons for obtaining an angiogram in these patients. The authors have listed various reasons for obtaining an angiogram in 13 patients; however, the reasons for obtaining MR angiograms in the other patients have not been mentioned.

As the authors have mentioned in the limitations of their study, the signal intensity of arteries on MR angiograms obtained using the time-of-flight (TOF) method depends on flow velocities and flow directions. Therefore, measurements of MMA diameter on MR angiograms may not reflect the exact diameter of the MMA. Moreover, MR angiograms are prone to susceptibility artifacts at the air-bone interface, arterial overlap, and pulsation-induced artifacts.¹

The evolution of CSDH is known to be associated with multiple factors. Defective neovascularization with rebleeding and absorption of plasma proteins into the subdural space, localized coagulopathy, and defective clot formation contribute to the evolution of CSDH.^{4–6,11} The absence of a statistically significant difference between the diameters of bilateral MMAs in cases of bilateral CSDHs of unequal size and the authors' finding that hematoma size was not correlated with the diameter of the MMA may also highlight the fact that the aforementioned factors probably play a more important role in the evolution of CSDH.

Various case reports and small case series have shown successful outcomes following embolization of the MMA in cases of recurrent CSDH.^{2,3,7-9} The readers would be interested to know the number of recurrent cases in the present series and whether the diameters of the MMA in such patients were larger than those in other patients. Also, it would be great if the authors could clarify whether any intervention was performed in such patients, the correlation between the MMA diameters on MRA and those on digital subtraction angiography, and the outcome in such patients.

Once again, we commend the authors' work in the enigmatic field of CSDH.

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Disclosures

The authors report no conflict of interest.

Response

We are grateful to Dash et al. for showing such interest in and taking the time to comment on our article.

During the study period, 251 patients with CSDHs were admitted to our hospital. Computed tomography scanning is the first-line imaging study for patients with CSDHs at our hospital; therefore, MRA was not performed in all patients admitted with CSDH. Among the 251 patients, the primary physician selected MRI or MRA at his or her discretion in 55 patients. Then, we retrospectively evaluated the 55 patients.

A measurement of MMA diameter using MR angiograms has limited accuracy because arterial intensities depend on the flow velocities and flow directions on TOF MR images. To confirm enlargement of the MMA in patients with CSDH, we examined the laterality of MMA diameters in patients with a unilateral CSDH and we compared MMA diameters from an MR angiogram obtained after the onset of CSDH with those on a previous MR angiogram on which a CSDH had not yet been observed.

The size of a CSDH presumably represents a net result of blood supply from the MMA and absorption from the hematoma. No relationship was found between MMA size and CSDH size in this study, which could be explained by the following. The shorter duration of blood supply results in a smaller CSDH, even if the blood supply ability, which possibly relates to the MMA size, is high in a patient with large MMAs. A large MMA size can facilitate an increase in CSDH size, which is also affected by both the age of the CSDH and the blood absorption speed from the hematoma. Furthermore, in this study, only a main trunk MMA was evaluated on MR angiograms, even though dural branches of other arteries could supply blood to a CSDH. Approximately one-fourth of CSDH patients in this series showed small MMAs with a diameter < 1.3 mm, which is the cutoff value for the existence of CSDH. In these CSDH patients with small MMAs, blood supply may be received from the other dural branches rather than the main MMA trunk.

In this series, a few cases of CSDH recurred after operation; therefore, we cannot conclude that there is a relationship between the MMA condition and the recurrence of CSDH. Although interventional embolization of the MMA seems to be an attractive option for a repeatedly recurrent CSDH, no patients in this series underwent intravascular treatment for an intractable CSDH. Both confirmation of the accuracy of MMA evaluation using MRA as compared with digital subtraction angiography and MMA diameter change after surgery, especially in cases of recurrent CSDHs, are future issues for a study of MMA evaluation using MRA in patients with CSDHs.

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INCLUDE WHEN CITING

Published online September 30, 2016; DOI: 10.3171/2016.6.JNS161517.

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Seizures and invasive meningiomas

TO THE EDITOR: I read with interest a recent review by Englot et al.⁶ on symptomatic epilepsy in cases of supratentorial meningiomas (Englot DJ, Magill ST, Han SJ, et al: Seizures in supratentorial meningioma: a systematic review and meta-analysis. *J Neurosurg* 124:1552–1561, June 2016). In their extensive meta-analysis the authors demonstrated that seizures before surgery are observed in 29.2% of patients, with the incidence ranging widely from series to series (from 12% to 76%), and that the strongest predictor of their presence is peritumoral brain edema. Peritumoral brain edema in cases of intracranial meningiomas may be caused by different factors and brought about through various mechanisms, but invasive tumor growth with the disappearance of the arachnoid layer and frequently associated pial blood supply of the tumor is one of the most consistently identified findings.^{2,4,5,8–10,12} Therefore, it is possible that there might be interrelationships between seizures and brain invasion, as was discussed briefly in the referenced article⁶ and the accompanying editorial.¹¹

In fact, such an association was noted as a collateral result of our prospective study focused on evaluating the role of proton MR spectroscopy (¹H-MRS) in cases of intracranial meningiomas.⁴ The protocol presumed collection of the multiple clinical, radiological, surgical, and histopathological variables, whereas subsequent univariate (but not multivariate) analysis revealed a significant association between invasive growth of the tumor macro-

scopically identified during surgery and the presence of seizures throughout the course of the disease. Relatively tight adhesion of the neoplasm to the pia mater or brain tissue was nearly twice as frequent (82% vs 42%; $p = 0.0308$) in patients with symptomatic epilepsy, but their number was disproportionally small (11 of 100 cases).⁴

For detailed characterization of this finding, an additional assessment of our data was done, which was limited to newly diagnosed convexity and parasagittal meningiomas (49 cases).³ Epileptic seizures before surgery were noted in 9 patients (18%), and their presence was positively associated with the size ($p = 0.0271$) and macroscopically invasive growth ($p = 0.0316$) of the tumor (Fig. 1). The presence of seizures demonstrated 0.89 positive predictive value for pial or brain invasion by the neoplasm.³ Owing to the evident limitations of our study (post hoc analysis of a limited number of cases), the obtained results certainly require further validation in carefully planned prospective investigations with detailed consideration of other factors, including lesion location above the cerebral convexity.⁷ Nevertheless, it seems quite possible that the presence of symptomatic epilepsy in patients with supratentorial meningiomas may be predictive of invasive tumor growth, which may have important surgical and prognostic implications.^{1,2,9}

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FIG. 1. Interrelationships between the size of meningiomas, macroscopically invasive growth, and presence of seizures in the history of disease. Copyright S. Karger AG. Published with permission from Chernov et al: *Stereotact Funct Neurosurg* 91 (Suppl 1):304, 2013.

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Disclosures

The author reports no conflict of interest.

Response

No response was received from the authors of the original article.

INCLUDE WHEN CITING

Published online October 7, 2016; DOI: 10.3171/2016.8.JNS161962.

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Error in the eye of the beholder: crew resource management in neurosurgery

TO THE EDITOR: I read with interest the article by Michalak et al.² (Michalak SM, Rolston JD, Lawton MT: Prospective, multidisciplinary recording of perioperative

errors in cerebrovascular surgery: is error in the eye of the beholder? *J Neurosurg* **124**:1794–1804, June 2016). Much has been written about the lessons that health care providers can learn from aviation. Indeed, many physicians have probably grown weary of hearing about how checklists and automation can save patients from thousands of potential medical errors. But checklists and automation are only part of the story when it comes to the vast improvement in aviation safety that occurred in the 1980s and beyond.

In the preceding decades, there were numerous fatal airplane crashes whose ultimate cause was found to be “pilot error.”³ Most importantly, there is evidence that copilots or other flight personnel recognized errors, dangerous situations, or potential lifesaving solutions, but either did not tell the pilot or were not able to effectively communicate the situation to the senior officer. Airlines realized that one of their substantial problems was a steep hierarchy in the cockpit. Junior officers were unwilling to question the authority of senior pilots, even in situations where it was clear that an error was occurring. The solution to this was Crew Resource Management.¹ Training and standardized procedures were widely adopted throughout commercial aviation to assure a culture of psychological safety and a flattened hierarchy, such that the most junior member of the flight crew not only could, but was required to notify the pilot of dangerous situations. The parallels to the neurosurgical operating room (OR) are obvious, and the need for Crew Resource Management in neurosurgery is huge.

The paper by Michalak et al.² is a novel and very important contribution to our field. The authors conducted a survey of OR teams, asking team members to identify errors (“deviations from optimal care”). For the first time, not only was the attending neurosurgeon surveyed, but residents, circulating nurses, and anesthesiologists were also asked to identify errors. They found that in 31 cerebrovascular cases, a total of 118 errors were reported, 94 of them unique (3.1 errors per case). The attending neurosurgeon identified 25 errors (0.8 errors per case). Most neurosurgeons would likely agree that the attending neurosurgeon is ultimately responsible for the care of the patient and the outcome of the operation. But how can the attending neurosurgeon optimize the outcome if he or she is only aware of one-third of the potential errors during a case?

For example, the authors note that there were 9 total contamination errors reported. Attending neurosurgeons reported only 2. It is possible that the attending was not in the room at the time, and thus was not aware of the other 7. Far more concerning is the thought that the attending was present, but was not notified of the contamination event.

The authors found “a paucity of duplicate errors,” and determined that team members were more likely to report errors that were directly related to their role in the case. Although this may seem intuitive, it is an important finding. They conclude that to fully understand OR errors, we must solicit information from all members of the OR team. Their results certainly support this conclusion, but there should be more to the story. As surgeons, we need to do more than understand OR errors. We must recognize that it is human nature to make errors. And we must recognize that the surest way to prevent an individual's error from leading to an adverse event is to provide every member of

the operating team with the full support of all other team members. In brief, we must recognize the importance of Crew Resource Management in our field.

How can we flatten the hierarchy in the OR? How can we assure that the resident, the circulating nurse, even the medical student is comfortable speaking up if she sees an error? Taken further, how can we assure that everyone in the OR knows that he is part of the team caring for the patient on the table, and knows that he has a responsibility to notify the rest of the team if a potentially harmful situation is developing?

To consider these questions for an entire specialty, or even for a hospital, seems daunting. Perhaps this is a change that can occur on an individual level. For example, in my own OR, at the completion of the formal time out, I have added a simple statement: "We are the team caring for this patient today. There are many ways this case can go wrong. I expect everyone here to speak up and inform me if they see a dangerous situation developing. Does everyone agree?" It is a simple step, taking less than a minute, but it establishes the expectations and accountability from every team member. Is this enough? Probably not, but it is a start.

The authors of this paper should be commended for studying this problem and calling our attention to it. If our field is to make the giant steps in safety that we have seen in air travel, we must find answers to these questions. We must begin to systematically apply the principles of Crew Resource Management to the neurosurgical operating theater.

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Disclosures

The author reports no conflict of interest.

Response

We are grateful for Dr. Rocque's insightful response to our article. As Dr. Rocque emphasizes, the vast majority of errors we documented were not found by the attending neurosurgeon, but rather by the nurses, anesthesiologists, and residents involved in each case, underscoring the importance of systems-level thinking. As neurosurgeons, we tend to focus on the technical and decision-making errors we commit. But errors can strike patients from any part of

the health care system that touches them, from anesthesia to hospital bureaucracy. For example, when we reviewed other prospective studies of neurosurgical errors, we found that only 25% were technical in nature, the remaining errors often involving domains outside the neurosurgeon's direct control.^{3,4} To put it in perspective, even if a surgeon perfects his or her craft, patients are still subject to 75% of the errors and adverse events to which they were exposed before.

Improvement in health care systems can occur at many levels, with the OR being of critical importance for neurosurgeons. Improving coordination among the many OR players by using techniques like Crew Resource Management, as suggested by Dr. Rocque, has been shown in other surgical specialties to improve outcomes, and has been suggested for neurosurgery specifically at least as early as 2001.¹ The fact that 15 years have passed and Crew Resource Management is still not widely studied in neurosurgery is disheartening. However, strides are being made to flatten the intraoperative hierarchy and improve teamwork, as suggested by Dr. Rocque. His routine of asking for all those in the OR to speak up if they see a dangerous situation developing is simple and exemplary. At the University of California, San Francisco, our workflow includes introductions of all OR personnel before the case begins, and concludes with structured postoperative debriefings, which can be used to identify issues that occurred during the case. These are just a small subset of the many other safety protocols detailed in our neurosurgical "Culture of Perioperative Safety" video.² In the end, it will take more people like Dr. Rocque, who are committed to improving patient safety and teamwork in the OR, to move our field forward.

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Published online October 7, 2016; DOI: 10.3171/2016.6.JNS161581.

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