Neurosurgical Forum

LETTERS TO THE EDITOR

Stereotactic subcaudate tractotomy: Knight stood on 3 giants' shoulders

TO THE EDITOR: I read with fascination Marchi and colleagues¹² account of Geoffrey Knight's development of stereotactic subcaudate tractotomy and commend its authors for their detailed explanation of both its anatomy and his scientific rationale (Marchi F, Vergani F, Chiavacci I, et al: Geoffrey Knight and his contribution to psychosurgery. *J Neurosurg* [epub ahead of print June 17, 2016. DOI: 10.3171/2016.3.JNS151756]). However, to isolate the man and his operation from key British stereotactic and functional neurosurgeons of that post-war era is to lobotomize the story of its richness.¹⁶ The contributions of a trio of his pioneering contemporaries should be mentioned. Firstly, Sir Hugh Cairns who established the Nuffield Department of Surgery at Oxford, where I trained.¹⁵ Secondly, Sir Wylie McKissock who founded neurosurgery at Atkinson Morley Hospital, now part of St. George's Hospital in London, where I work.¹ Finally, F. John Gillingham in Edinburgh, whom I came to know first as my patient and then as a friend and inspiration in his final years.¹⁷

Rather than being first performed by Foltz and White in 1961 as the authors suggest, anterior cingulotomy, also known as dorsal or rostral cingulotomy, was first performed for the treatment of psychiatric disorders in 1948 in Oxford by Cairns, albeit freehand before the advent of stereotactic surgery. Cairns extirpated by craniotomy and interhemispheric approach the anterior 4 cm of supragenual cingulate gyrus (Brodmann's Area 24). He published long-term outcomes from 24 of 29 treated patients with psychiatric disorders, showing improvements in many without detrimental changes in behavior and finding the treatment least effective in psychoses and most useful in "certain types of mental illness marked by obsessions, tension and anxiety, in which the basic personality is preserved."²⁰

As with Knight's subgenual cingulate region target, Cairns' dorsal anterior cingulate target has also been vindicated by a vast scientific literature from animal models and human brain imaging implicating the structure as dysfunctional in most psychiatric disorders and also in chronic pain. A recent open-label clinical trial from our Oxford team has suggested that it is a novel target for deep brain stimulation in otherwise neuromodulation-refractory central pain.²

Gillingham, with the Parisian neurosurgeon Gerard Guiot, pioneered awake, freehand pallidotomy for Parkinson's disease in 1953, utilizing a subfrontal approach to the anterior perforated substance that interrupted the ansa lenticularis.⁷ He treated 1 patient in Edinburgh in 1955 and a second in 1957 and reported long-term improvements in tremor, rigidity, and quality of life. However, wishing to avoid the demanding subfrontal approach, he modified Guiot's stereotactic parasagittal approach to an occipital one, developing the Guiot-Gillingham stereotactic apparatus in the process.⁶ In 1960 he published results from stereotactic "thermal electrocoagulation lesions of the globus pallidus, internal capsule and thalamus either separately or in combination" in a further 58 patients operated upon from 1957 to 1959.⁵ It is likely that Knight was audience to one of Gillingham's many presentations of framed stereotactic lesioning via subfrontal, parasagittal, and occipital approaches, and he adopted Gillingham's stereotactic subfrontal approach half a decade later for his first subcaudate tractotomy in 1961.

McKissock was born the same year as Knight and died at the same age. An assiduous British proponent of neurosurgery for psychiatric disorders,¹⁸ he favored a freehand approach to the frontal lobe from above.¹⁰ He described the rostral leukotomy in 1951 as a rejoinder to Freeman and Watt's transorbital "ice-pick" leukotomy, which he considered to contravene "established aseptic surgical principles."^{13,14} McKissock's immense practice covering swathes of South England and Wales and his reputation for extraordinary surgical speed inculcated a peripatetic service visiting other hospitals with his surgical instrument set in his car, drawing parallels with Freeman.¹ In 1961, Tooth and Newton reported that 10,365 leukotomies were performed in the United Kingdom between 1942 and 1954,¹⁹ and it has been suggested that McKissock alone may have performed one-quarter of these procedures. McKissock was less convinced by the merits of stereotactic over freehand approaches in reducing complications than Knight. The two presented their freehand outcomes back to back in 1959, by which time McKissock reported having performed nearly 3 times as many psychosurgeries as Knight.^{11,13}

It is therefore great testament to Knight that the Atkinson Morley neurosurgeon Alan Richardson—profoundly influenced by his mentor McKissock, as all around the great man were—developed a stereotactic approach for his psychiatric procedures.¹⁰ He combined Knight's subcau-

date tractotomy with Foltz and White's selective rostral cingulotomy to invent the procedure of limbic leukotomy in the early 1970s.^{8,9} Like Knight's subcaudate tractotomy, Richardson's limbic leukotomy continues to be performed in carefully selected cases refractory to medical treatment.¹⁵

Gildenberg has suggested 4 tenets that characterize the field of functional neurosurgery: 1) the need to be innovative, 2) that functional neurosurgery is a science, 3) that functional neurosurgeons work as a community not in isolation, and 4) that there is also appreciation for the insight and courage of true pioneers.⁴ Knight worked not in isolation but within such a neurosurgical community, and the centers that these 3 British giants of neurosurgery founded continue to exemplify such principles.

Finally, the authors do not mention that most of Knight's patients called him either "Professor Knight" or "Sir Geoffrey," presumably because even though he was neither, he ought to have been.³ The myth that Knight was knighted has crossed the Atlantic and pervades functional neurosurgery folklore to this day. I witnessed a compelling lecture in April 2016 by G. Rees Cosgrove, at a masterful lesioning course deftly assembled as ever by raconteur Marwan Hariz, where subcaudate tractotomy was explained and several references made to "Sir Geoffrey." I am sure that Professor Cosgrove, Professor Hariz, and the article's senior author, Professor Ashkan, would agree that he ought to have been Professor Knight. Perhaps Sir Hugh and Sir Wylie might even have concurred that he should have been honored Sir Geoffrey.

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Disclosures

The author reports no conflict of interest.

Response

We read with great interest the comments made by Pereira with regard to our original paper on the life of Geoffrey Knight and his contribution to the field of psychosurgery. It is fair to acknowledge the role that other prominent British neurosurgeons had before and along with Knight in developing new techniques for the surgical treatment of psychiatric disorders, and we agree that the contribution of giants such as Sir Hugh Cairns, John Gillingham, and Sir Wylie McKissock cannot be overemphasized. To that extent, the comment by Pereira stands as a useful and interesting complement to our article.

However, rather than recollecting the history of the birth and early stages of functional neurosurgery (and psychosurgery) in the United Kingdom, of which more exhaustive accounts have already been reported in the literature,² the aim of our paper was to illustrate the individual contribution made by Knight in this particular field. As we stressed, his major contribution was in the pursuit of a less-invasive surgical procedure, in an effort to identify a more precise functional target while minimizing the risk of complications. We agree that on this regard his view was quite different from that of his contemporary McKissock, who preferred open surgery to a stereotactic approach. And again, the view of Knight was destined to be more influential in the following years, when stereotactic lesioning and then stereotactic deep brain stimulation (DBS) virtually replaced open surgery in functional neurosurgery.

The other major aspect that we wanted to stress in our paper, and this is again unique to the work of Knight, is the choice of target for his subcaudate tractotomy, which corresponds largely to the subgenual cingulate region targeted today in DBS for depression.¹ It is of interest that Knight realized that the target was in fact represented by the white matter connections of this region, which appears to be at the center of a network involving limbic, frontal and temporal regions, as recently reported by our group in an anatomical study combining dissections and tractography.³

As with reference to the titles, academic or regal, we left any discussions on these out of our paper. After all, it is great men who make the titles and not titles that make the men!

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INCLUDE WHEN CITING

Published online November 4, 2016; DOI: 10.3171/2016.7.JNS161635.

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Screening protocol for blunt cerebrovascular injury

TO THE EDITOR: We thank Tso et al.⁵ for their paper on a screening protocol for blunt cerebrovascular injury (BCVI) (Tso MK, Lee MM, Ball CG, et al: Clinical utility of a screening protocol for blunt cerebrovascular injury using computed tomography angiography. *J Neurosurg* [epub ahead of print April 22, 2016. DOI: 10.3171/2016.1.JNS151545]). We read the paper with a lot of interest and have a few questions.

The authors assessed the utility of a CT angiography (CTA) screening protocol for BCVI detection and concluded that a defined protocol may be of clinical benefit. It is important to note that antiplatelet agents were used safely in 75% of patients with BCVI without new hemorrhage. Adopting the protocol increased the use of any vascular

imaging from 5% in the preprotocol era to 8% postprotocol. However, the reduction in ischemic complications could simply be attributable to earlier and increased use of treatment, and the conclusion that adopting the CTA protocol could be responsible does not seem to be substantiated since the BCVI detection rate remained unchanged before and after adopting the protocol.

In addition to an increased use of CTA studies, the significant difference seems to be the decreased use of MR angiography after adoption of the protocol. Magnetic resonance angiography has been shown to have lower sensitivity for the detection of BCVI, as the authors themselves have stated. In the preprotocol era, did the authors find missed BCVI cases that had stroke despite vascular imaging? In addition, imaging was performed after significant delay in the preprotocol era, and some patients may have had resolution of the imaging findings by Day 8.

The authors stated that modified Denver criteria were used to select patients for CTA after adoption of the protocol. However, almost 20% of patients with risk factors did not undergo any vascular imaging. Can the authors share under what circumstances the protocol was not followed?

The proportion of patients that had at least 1 BCVI risk factor did not change between the pre- and postprotocol period (10.7% vs 10.0%, respectively). The number of patients who underwent vascular imaging did increase from 5% to 8%, and the unscreened patients with BCVI risk factors decreased from 51.2% to 19.6%. However, the number of diagnosed BCVI cases decreased slightly from 0.9% to 0.5%, despite the use of 64-slice CTA and greater CTA sensitivity. Do the authors have an opinion on this?

The authors stated that none of the patients with a negative CTA subsequently had a stroke. Were these patients routinely followed up? Significant concerns have been raised about the sensitivity of CTA, even as current-technology digital subtraction angiography was performed postprotocol in 7 patients, 3 of whom had BCVI.³ Were the other cases false-positive on CTA? If so, did this finding raise concern about the other cases also being false-positive? Some centers have reported very high false-positive rates with CTA.⁴

A total of 17% of patients with BCVI did not have at least 1 of the screening risk factors. Were these patients all in the preprotocol era? Were they symptomatic BCVI patients? The latter seems unlikely given the numbers in the authors' Tables 1 and 2.

We would also ask the authors to share if there was a reason that the treatment was not initiated until 5.6 days before and 2.7 days (mean) after the diagnosis of BCVI. Previous literature has shown that many strokes in BCVI occur early after trauma.¹

Significant heterogeneity remains in the literature about the use of CTA screening for BCVI, and its cost-effectiveness requires further study.²

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Disclosures

The authors report no conflict of interest.

Response

We thank Wu and colleagues for showing interest in our recent publication regarding CTA screening for BCVI. We address the authors concerns in the order they were presented.

After implementation of the CTA protocol, imaging was performed earlier in patients diagnosed with BCVI (2.5 vs 8.2 days) and medical treatment was started earlier after BCVI diagnosis (2.7 vs 5.6 days). Earlier treatment may have contributed to the decrease in ischemic complications, but the timing of treatment was not the independent variable in our study. We compared 2 groups of patients (before and after implementation of the CTA protocol), and we believe that the CTA protocol may have changed the management strategy for BCVI and that the earlier treatment initiation was just a by-product of having an established screening protocol. At our institution, physicians who actively manage trauma patients have become much more aware of BCVI and its potential morbidity and mortality. In fact, residents rotating through the trauma service are given pocket-sized laminated cards listing all the high-risk criteria that indicate a need for CTA for BCVI screening.

In our study, we did not observe any patient having a negative vascular imaging study who then proceeded to have an ischemic stroke because of a missed BCVI diagnosis. We acknowledge that the delayed vascular imaging in the preprotocol era may have led to spontaneous resolution of some BCVIs, especially Grade 1 and 2 vascular injuries.^{1,2}

We do not know why the CTA protocol was not always followed despite the presence of BCVI risk factors. We suspect that some patients may have had legitimate reasons such as renal failure or prior anaphylactic reaction to contrast but that the majority were just simply missed.

On a trauma population basis, the rate of BCVI diagno-

sis was similar before and after CTA protocol implementation (0.9% vs 0.8%). This was an unexpected finding, as we would have predicted an increased rate of BCVI diagnosis with a CTA protocol in place and with improved CT resolution. We do not have an explanation for this. The data are the data.

As mentioned in our discussion, trauma patients with BCVI were not routinely followed up as outpatients. Of the 3 patients postprotocol who had prior CTA followed by digital subtraction angiography (DSA), 1 patient had the DSA study to rule out a ruptured intracranial aneurysm in the setting of a suspicious pattern of subarachnoid hemorrhage, 1 patient underwent DSA to confirm the BCVI finding on CTA, and 1 patient underwent DSA despite a normal CTA, resulting in the identification of a BCVI but no ischemic complications. A false-positive CTA was not identified in the postprotocol era. There were also 4 postprotocol patients who underwent DSA as the only vascular imaging modality.

Table 3 showed that 26% (6/23) and 8% (2/24) of BCVI patients did not have any of the BCVI risk factors in the preprotocol and postprotocol eras, respectively, and this difference was not statistically significant. All 6 of the preprotocol BCVI patients without risk factors developed ischemic complications, whereas the 2 postprotocol BCVI patients without risk factors did not develop ischemic complications.

It was not clear from the health records why treatment for BCVI was delayed.

We believe screening for BCVI with CTA is important. Having a formalized institutional screening protocol will help raise awareness among physicians dealing with trauma patients and potentially help reduce the risk of BCVI-related ischemic complications.

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INCLUDE WHEN CITING

Published online November 11, 2016; DOI: 10.3171/2016.8.JNS161942.

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White matter fiber tract architecture and ventricular surgery

TO THE EDITOR: In their recently published laboratory investigation, Güngör and colleagues⁹ meticulously explore and illustrate the topographic relationship of various cerebral white matter pathways to the ventricular compartments, using the fiber microdissection technique and MR tractography studies (Güngör A, Baydin S, Middlebrooks EH, et al: The white matter tracts of the cerebrum in ventricular surgery and hydrocephalus. *J Neurosurg* 126:945–971, March 2017). They further attempt to correlate their findings to possible neuropsychological deficits resulting from the injury of discrete fiber tracts that are either transgressed during ventricular surgery or displaced in case of ventricular enlargement.

Undoubtedly, the authors, through the very detailed morphological description of the relevant white matter pathways, especially of the superior longitudinal and arcuate fasciculi, and the thorough correlation of the brain surface anatomy to the deep corresponding fiber tract architecture and ventricular topography, make a valuable contribution to current literature and provide a very useful anatomical-parenchymal roadmap for ventricular surgery. Linking white matter dissections to MR tractography aids in a better understanding of the intricate subcortical anatomy and enhances the paper's readability. The significance of an accurate 3D neuroanatomical knowledge to surgical planning and potentially to clinical outcome, resulting from the incorporation of laboratory microanatomical dissection of white matter tracts to modern neurosurgical education, is once again strongly emphasized.

However, there are some technical, anatomical, and surgical comments that have to be highlighted to enhance the manuscript's clarity. The authors, in their effort to vividly illustrate the complex subcortical fiber tract anatomy in relation to the ventricular compartments, have heavily edited most of the included fiber dissection photographs with picture software. Although this may aid in a better demonstration, and possibly understanding, of the relationship of multiple white matter pathways with regard to the ventricles, it indeed seriously distorts the normal fiber tract anatomy and configuration encountered during standard laboratory dissections. In our opinion, using "unenhanced" photographs of cadaveric dissections has a higher scientific and educational value, even if strict anatomical accuracy can sometimes be slightly compromised.^{14,15}

The authors, furthermore, state in the *Results* section that "Removing the posterior part of the posterior crus of the anterior commissure exposes the occipital and temporal thalamic radiations (optic radiations) running lateral to the temporal horn and inferior two-thirds of the atrium." Although this statement is based on a concrete anatomical rationale, it has to be stressed that the relevant fiber bundles—i.e., the posterior part of the anterior commissure, the posterior part of the occipitofrontal fasciculus, the retrolenticular part of the internal capsule, and the inferior longitudinal fasciculus—that blend at the posterior temporal and occipital areas to form the single entity known as the "sagittal stratum" cannot be individually differentiated

and thus demonstrated by using the Klingler's technique, since they share the same direction and orientation, with no demarcation point between them.^{13,14,21,23} The paper's senior author (Dr. Rhoton), in one of his earlier seminal anatomical studies entitled "Three-dimensional microsurgical and tractographic anatomy of the white matter of the human brain,"⁷ which is also used in the reference list of the current laboratory investigation, has explicitly clarified this issue, coming to the same conclusion that "Differentiation among all of these layers of fibers via the dissection technique is impossible" and further saying in the same paragraph, "This explains the inclusion of all these fasciculi in a single layer, identified as the sagittal stratum in several anatomic works in which the fiber dissection technique was used" (*Results* section, page 997, paragraph 5).

Additionally, in their *Results* section, Güngör and colleagues, continuing the description of the lateral to medial white matter dissection process and trying to expose the callosal radiations, state, "Removing the sagittal stratum and corona radiata exposes the callosal fibers." Again, although this—theoretically—seems as the next dissection step in the entire procedure, practically when performing a lateral to medial white matter dissection it is indeed very difficult to adequately separate the fibers of the internal capsule from the callosal radiations, since their respective directions meet at a nearly perpendicular angle. This anatomical observation, in combination with the fact that the relevant fibers at this level exhibit a high density and tight intermingling, renders the proper identification and demonstration of the callosal fibers extremely difficult, if not impossible. Thus, due to these reasons and to thoroughly expose, understand, and illustrate the different parts of the callosal radiations, the anatomical dissection should be ideally directed from the medial to the lateral aspect of the brain. This is advocated by several previous anatomical studies^{14,18,26} and also by the paper's senior author (Dr. Rhoton) in his earlier work.⁷

The authors subsequently discuss their findings with regard to the possible neuropsychological symptoms associated with the majority of the applied operative corridors to the lateral ventricles and also make an extensive list, linking every approach to the underlying parenchymal lesion, the related injured fiber tract(s), and the expected postoperative deficit(s). Particularly, regarding the anterior and posterior operative ventricular variants, the authors seem to focus their discussion mostly on the consequences/deficits that could—theoretically—result from the injury of individual fiber bundles en route to the ventricles with little reference made to the major and most common postoperative neuropsychological symptoms encountered following each of the listed approaches. Accordingly, the relevant literature included in the paper's reference list is based mainly on the field of functional neuroanatomy (over 30 references) with little evidence coming from studies on surgical and neuropsychological outcomes of clinical series (References 22, 70, 74, 77, 81, 82, 95, and 119). Although this academic approach has a strong scientific background it indeed moves away from the practicality that the everyday neurosurgical practice entails.

More specifically, regarding the anterior interhemispheric transcallosal approach, most of the available clinical evidence indicates that permanent impairment of at-

tentional function, persistent deficits in verbal and recent memory, or postoperative disconnection syndrome and behavioral disturbance are very rarely observed and tend to occur in patients who have sustained extracallosal damage.^{1,3,5,12,16,24,25} Moreover, the incidence of transient recent memory deficit is reported to be around 10%, thus rendering the transcallosal route a safe and effective approach for ventricular lesions.^{1,5} With respect to the anterior frontal transcortical route, the major and most commonly encountered causes of surgery-related morbidity are reported to be the presence of subdural fluid collections^{2,11,22} and postoperative seizures,^{2,4,16} due to the involved middle frontal gyrus corticotomy. Deficits such as spatial neglect, memory disturbance, agraphia, and facial apraxia are extremely rare.^{2,4,6,16} Furthermore, since we tend to perform this approach on the nondominant hemisphere, speech and language deficits are again very rarely seen. Transient postoperative hemiparesis, which is mentioned by the authors as a possible consequence of both transcallosal and transcortical variants, arises mainly from the ligation of parasagittal veins and is not usually due to parenchymal violation.^{1,12}

Lastly, as far as the superior parietal lobule approach is concerned (either transcortical or through the intraparietal sulcus),^{8,10,19} it has to be stressed that its relevant subcortical operative corridor allows entry to the ventricle through the medial part of the roof of the atrium, which is known to be devoid of optic radiation fibers.^{9,17,20} Based on our microanatomical white matter dissections at the Athens Microneurosurgery Laboratory, we can argue that regardless of how far laterally the entry point of the surgical trajectory is placed on the cerebral surface, if the trajectory is kept within the boundaries of the superior parietal lobule, the atrium is always accessed through its roof, thus avoiding injury to the optic pathway. To hit the optic radiations, the subcortical dissection would have to be directed toward the lateral atrial wall and thus inferior to the intraparietal sulcus, within the boundaries of the inferior parietal lobule. We therefore disagree with the authors on their argument that through this approach the surgeon might risk potential damage to the optic pathway.

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Disclosures

The authors report no conflict of interest.

Response

We thank Dr. Koutsarnakis et al. for their interest in our recent study on the white matter tracts in ventricular surgery and hydrocephalus. In their letter to the editor, these authors aimed to highlight several technical, anatomical, and surgical comments to enhance our manuscript's clarity. Most of these comments warrant further explanation in order to correct several misconceptions.

These authors initially claimed that heavily edited fiber dissection photographs in our study resulted in serious distortion of the normal fiber tract anatomy. Furthermore, they questioned the scientific and educational value of the manuscript due to insufficient use of “unenanced” photographs of cadaveric dissections. We must repudiate this accusation. The Rhoton Microneurosurgical Laboratory at the University of Florida has a long history of producing some of the best 2D and 3D dissection images in the world, including several recent fiber-tract anatomy dissection studies.^{2,3,7,9–11} This has been possible because of the quality of specimens we are able to procure through the Florida State Anatomic Board; our long-established protocol for handling, preparing, and photographing specimens; and the meticulous quality of dissections performed by neurosurgeons in the laboratory. In this paper, we have maintained that tradition, resulting in photographs in which the fiber tracts can be easily seen and understood by readers. The only editing done to these photographs beyond text labeling was the addition of color overlay placed on certain fiber tracts in Figs. 4D, 9E, and 15D. The goal of these overlays is to clearly distinguish the relevant anatomy. No aspect of the anatomy has been changed or otherwise enhanced in any way.

Koutsarnakis et al. also made an assertion that the fiber bundles of the sagittal stratum cannot be individually differentiated and demonstrated by Klingler's technique, quoting one of Dr. Rhoton's previous articles.⁶ We disagree with the authors and strongly believe that segregating the sagittal stratum through the fiber dissection technique is possible. To isolate different layers of the sagittal stratum, one can progressively distinguish each of the fiber layers along the sagittal plane in regard to orientation, course, and terminations. The most superficial layer, the inferior fronto-occipital fasciculus, passes through the ventral part of the external capsule/claustum sheet beneath the posteroinferior part of insula, courses through the temporal

stem, and extends backward toward the extrastriate areas on the occipital lobe. The second layer consists of the posterior crus of the anterior commissure, and this layer can be followed from medial to lateral by defining the stem of the anterior commissure beneath the globus pallidus. Strikingly, the clockwise turn of the anterior commissure and its distal extension posteriorly through the temporal stem—toward the sagittal stratum—can be delineated in almost all hemispheres. The third layer that can be differentiated within the sagittal stratum is the optic radiations, specifically, fibers arising from the lateral geniculate body (LGB) passing to the upper and lower banks of the calcarine fissure. At this point, the fiber dissection technique not only displays fibers arising from the LGB that constitute the posterior thalamic radiations (accompanying the retrolenticular internal capsule), but also clearly pinpoints its anterior loop (Meyer's loop) in relation to the temporal horn of the lateral ventricle. Finally, lateral to medial dissection deep to the sagittal stratum differentiates obliquely oriented vertical fibers, the tapetum, extending from the splenium of the corpus callosum and separating the stratum above from the temporal horn and below from the inferior two-thirds of the atrium.

Another criticism was related to the low probability of separating the fibers of the internal capsule from the callosal radiations. Dr. Koutsarnakis et al. offered a medial to lateral dissection in order to understand and illustrate the different parts of the callosal radiations. We contradict most parts of these remarks and believe that lateral to medial dissection is capable of separating the internal capsule from the callosal radiations. In this regard, we have already discussed the distinct orientation and course of the tapetal fibers underneath the sagittal stratum, creating a callosal compartment that is relatively easy to expose. More anteriorly, external and internal capsules come together and form the corona radiata along the depth of the superior limiting sulcus of the insula and above. During lateral to medial dissection, differentiation of deep corona radiata fibers (internal capsule) from those of callosal fibers is straightforward at the region anterior to the rolandic (central) and the central insular sulci. The horizontal, somewhat S-shaped, orientation of the anterior and parts of superior thalamic radiations can be demonstrated deep to the internal capsule at this region. Further medial dissection at the region anterior to the rolandic (central) and the central insular sulci deep to thalamic radiations exposes the convex callosal fibers above the body of the lateral ventricle. It is also possible to separate the callosal fibers from the posterior limb of the internal capsule at the region behind the rolandic (central) and the central insular sulci. At this more posterior region, callosal fibers typically extend outward above the body of the caudate nucleus deep to intermingled internal capsule and thalamic radiation fibers and can be followed all the way back to the “forceps major,” which most often passes to the cuneus at the medial part of the cerebrum. Thus, we are confident that fibers of the internal capsule can be separated from the callosal radiations via not only medial to lateral dissection, but also through the lateral to medial approach, all of which can be complemented with MR tractography studies.

Regarding the comments on neuropsychological conse-

quences of different surgical approaches to the ventricles, Koutsarnakis et al. stated that our academic approach, although based in a scientific background, lacks sufficient evidence from surgical and neuropsychological outcomes of clinical series. We have cited more than 40 clinical studies (References 5, 6, 9, 13, 16, 17, 20, 21, 22, 29, 32, 35, 37, 39, 45, 55, 57, 62, 63, 70, 73, 74, 75, 81, 82, 84, 86, 87, 89, 94, 95, 97, 103, 104, 105, 107, 112, 114, 118, 119, 124, and 125) and discussed all possible mechanisms related to sensorimotor and neuropsychological consequences of both transcallosal and transcortical routes, including those mentioned by the authors. More specifically, postoperative hemiparesis, agraphia, language impairments, facial apraxia, memory disturbance, and spatial neglect were all discussed, although neuropsychological outcomes were ignored or underestimated as rare empirical observations in most clinical series.^{1,4,5,8}

Koutsarnakis et al. likewise argued that the superior parietal lobule approach, through the perimeter of the intraparietal sulcus, creates no potential risk of damage to the optic pathways. On the basis of anatomical dissections, we believe that even slight lateral deviation from the surgical route through this approach may encounter the optic radion fibers on the inferior two-thirds of the lateral wall of the atrium, possibly via the way deep to the inferior parietal lobule.

Consequently, we share the same view with the authors in regard to the significant positive potential role of accurate 3D neuroanatomical knowledge to surgical planning and eventually to better clinical outcomes. In this vein, incorporation of laboratory microanatomical white matter dissection to modern neurosurgical education should once again be emphasized.

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Acknowledgments

This study was conducted, written, and submitted under the guidance of Albert L. Rhoton Jr., MD. The authors reserve their deepest respect and admiration for their mentor for his supervision over the years. We deeply appreciate the continuous support provided by Robin Barry and Jessica Striley with the manuscript.

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Published online February 3, 2017; DOI: 10.3171/2016.9.JNS162239.

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Volume management after subarachnoid hemorrhage

TO THE EDITOR: We read with interest the article by Obata and colleagues⁵ (Obata Y, Takeda J, Sato Y, et al: A multicenter prospective cohort study of volume management after subarachnoid hemorrhage: circulatory characteristics of pulmonary edema after subarachnoid hemorrhage. **J Neurosurg** 125:254–263, August 2016), describing hemodynamic changes after ruptured aneurysm treatment by using a PiCCO-plus device (Pulsion Medical Systems) in a prospective multicenter cohort study. The authors found an incidence of pulmonary edema of 25.5% in patients with subarachnoid hemorrhage (SAH). Patients who developed pulmonary edema presented with a higher World Federation of Neurosurgical Societies (WFNS) grade and were older. Moreover, the authors found a decrease in cardiac index in the pulmonary edema group, and, at the time of PiCCO catheter insertion, the mean serum brain natriuretic peptide levels were higher than those in the non-pulmonary edema group in the early phase. The authors concluded that pulmonary edema occurring in the early phases after SAH is caused by cardiac failure, whereas pulmonary edema occurring in the delayed phases after SAH is caused by inflammatory (i.e., noncardiogenic) conditions.

This article is important, being one of the few to report the use of hemodynamic PiCCO-plus monitoring for early identification of pulmonary edema in patients with SAH. Nevertheless, we have some questions regarding the validity of the conclusions, as insufficient information is presented on the methods used and because the incidence of pulmonary edema is extraordinarily high.

What surprised us is the study population: having 204 patients in a time frame of 43 months reflects an accrual rate of fewer than 5 patients per month from 9 different institutions, which amounts to 1 patient per 2 months per center. We wonder whether this low accrual rate provides enough of a routine to recommend such complex monitoring in these critically ill patients. Furthermore, the number of enrolled patients implies that a significant selection bias has taken place, but no information is provided on the volume of SAH patients treated per center and which patients were not included. This impression of selection bias is further strengthened by the observation that the authors found a staggeringly high percentage of patients who developed pulmonary edema (25.5%) compared with that reported in the literature (8%–23%).^{1,4,7–9} Moreover, it would be very interesting to be informed about the standard protocol(s) used in the participating centers for SAH patients, especially regarding fluid volume management. What would have strengthened this article is a discussion regarding influences that led to this high percentage.

It is remarkable that more patients were treated with triple-H therapy (hypertension, hypervolemia, and hemodilution) than there were patients with delayed cerebral ischemia, suggesting a kind of “prophylactic” triple-H therapy, although triple-H therapy has been shown not to be superior to normovolemic fluid therapy in the prevention of delayed cerebral ischemia. The high percentage (22%) of patients who developed pulmonary edema with a WFNS Grade I seems to support this assumption, as pulmonary edema is a known complication of hypervolemia.^{2,3}

No information was presented about patients’ outcomes after 30 days in *Results*, although this parameter was mentioned in *Methods*. The definition of morbidity used (“any result other than satisfactory recovery”) also seems a very subjective measure, easily prone to bias. Moreover, the mortality rates of 7.7% and 5.8% in the pulmonary edema group and non-pulmonary edema group, respectively, were quite low compared with percentages reported in the literature.^{4,6}

In conclusion, the additional use of hemodynamic PiCCO-plus monitoring might well be a useful tool to reduce the risk of developing (secondary) pulmonary edema in selected patients after SAH, but without further information we think that this is a premature conclusion.

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Disclosures

The authors report no conflict of interest.

Response

We thank Dr. Post and colleagues for their interest in our study and for their comments on our paper.

We reported that 25.5% of patients with SAH developed pulmonary edema. This incidence of pulmonary edema was slightly high, as was pointed out. Here, we discuss possible causes for this outcome. First, patients with severe SAH grades were brought to emergency and critical care centers and underwent surgery. Of these patients, those for whom hemodynamic monitoring would be useful in the management of their postoperative condition were enrolled in our study by attending physicians from each participating institution. In addition, the neurosurgical specialists themselves treated the patients, even after surgery for SAH. Thus, there was no standard protocol, even for fluid volume management, for patients with SAH. Second, the patients who were enrolled in our study underwent hemodynamic monitoring for 10 consecutive days after surgery. Hence, there may have been unintentional selection bias.

In such patients with SAH, we investigated the incidence and cause of pulmonary edema by using hemodynamic monitoring with PiCCO-plus pulse contour analysis and other clinical data. Therefore, we concluded that pul-

monary edema occurring in the early and delayed phases after SAH was caused by cardiac failure and inflammatory conditions.

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Published online January 27, 2017; DOI: 10.3171/2016.9.JNS162321.

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Approaches to the ventricular atrium

TO THE EDITOR: We read with interest the recent article by Jeelani and colleagues³ (Jeelani Y, Gokoglu A, Anor T, et al: Transtentorial transcollateral sulcus approach to the ventricular atrium: an endoscope-assisted anatomical study. *J Neurosurg* [epub ahead of print June 24, 2016. DOI: 10.3171/2016.3.JNS151289]).

In this interesting laboratory investigation, Jeelani et al. describe the well-known supracerebellar transtentorial transcollateral approach (STTS)^{2,4} to the atrium through a retrosigmoid craniotomy in cadaveric specimens. They also include the use of the endoscope to enter and identify the regional anatomy of the atrium in order to augment visibility and enhance surgical maneuverability. Undoubtedly, by introducing the three-quarter prone position coupled with the retrosigmoid corridor and the use of the endoscope to perform the STTS approach for accessing atrial lesions, the authors make a valuable contribution to the current neurosurgical armamentarium, since, at least theoretically, the risk of air embolism is greatly reduced. They also, very prudently, at the end of the article comment on the limitations of their study, clearly stating that the extrapolation of this approach to real clinical settings should be done with great caution. Obviously, it should also only be done by surgeons with vast clinical experience and thorough neuroanatomical knowledge.

However, there are a few surgical and anatomical issues that need to be stressed in order to enhance the clarity of the current study. When using normal cerebral sulci as surgical corridors to access deep-seated lesions, one should consider 2 important facts. 1) The morphology and topography of the cerebral sulci in terms of their continuity and branching pattern are highly variable,⁶ rendering the proper identification of the relevant sulcus challenging. 2) It is almost always mandatory to open the sulcus widely along its length so as to minimize the ill effects of adjacent normal parenchyma retraction, preserve sulcal vascular anatomy, and maximize the surgeon's operative view and maneuverability.^{1,8} The latter is especially crucial when dealing with intraventricular pathologies with rich blood supply, such as meningiomas, papillomas, and AVMs, where frequently shifting the operative angle is of paramount importance for the safe resection of the lesion

and for meticulous hemostasis. Therefore, a considerable extent of the sulcus needs to be exposed in order to meet the aforementioned objectives. In this context, the working area that the STTS approach offers through the retrosigmoid corridor is quite limited not only for a wide spreading of the collateral sulcus but also for the safe identification of the posterior mediobasal temporal lobe surface anatomy, factors that, as previously stressed, interfere with a safe and effective surgical exploration of atrial lesions.

Additionally, in the event that the surgeon needs to switch from endoscopically assisted surgery to traditional microneurosurgery, it seems quite challenging and time consuming to optimally align the microscope's light through the small cortical opening made on the collateral sulcus, with the difficulty being augmented by the steep angle of the surgical trajectory to the ventricular atrium achieved through the retrosigmoid pathway. This challenge could be significant and could potentially increase the surgical risk of postoperative complications when inadvertent intraventricular bleeding has to be tackled with the traditional use of the surgical microscope and bimanual dexterity.

Furthermore, in Table 2 of their *Discussion* section, the authors provide a very useful summary of the different surgical routes to the ventricular atrium and their potential complications, thus orienting the reader with respect to the different surgical variants. However, in their list of approaches, although the main transgyral operative corridors to the atrium are mentioned, there is a lack of reference to the relevant transsulcal transventricular routes, which shorten the surgical distance while minimizing normal brain transgression-retraction.^{1,7,8} In this context, apart from the collateral sulcus, the surgeon can gain access to the atrium through the intraparietal sulcus by performing the relevant transsulcal approach.^{1,7} A wide opening of the intraparietal sulcus followed by a subcortical dissection along the longitudinal axis of the fundus of the sulcus allows a short, direct, and effective access to the atrium through its roof, thus avoiding the optic radiation, which courses on its lateral wall, and preserving eloquent motor sensory and speech areas, which are located further anteriorly and laterally. Ideally, the entire process can be assisted by image-guided stereotaxy and/or ultrasonography. When compared to STTS, the intraparietal transsulcal approach provides a more straightforward and wider working corridor for surgical manipulations; avoids tentorial incision, with possible venous bleeding or infarct from the tentorial sinus; and allows for quicker and easier positioning of the patient. Potential postoperative complications, such as visuospatial neglect and some forms of ataxia, that result from injury to the parietal lobe are greatly reduced, since normal brain transgression and retraction are kept to minimal through the transsulcal trajectory.

Lastly, in the *Results* section, where the authors describe the regional anatomy identified through the endoscope, they write the following: "Focusing the endoscope on the lateral wall of the atrium, the tapetum was visualized." At this point, it has to be emphasized that in order to study and illustrate the subcortical white matter fiber tract anatomy and architecture in the context of a microneurosurgery laboratory one has to apply Klingler's method

(freeze-thaw process) to formalin-fixed hemispheres followed by the well-known fiber microdissection technique.⁵ This is the only procedure through which subcortical fiber tracts can be dissected and identified in a neuroanatomy laboratory. Particularly, the fibers of the tapetum, due to their deep location and elegant configuration, are very difficult to demonstrate, even with the implementation of the aforementioned techniques.⁵

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Disclosures

The authors report no conflict of interest.

Response

We appreciate the insightful comments from Koutsarnakis et al. and we agree with their observations about the topographic variability of the cerebral sulci on the basal occipitotemporal surface. They have also alluded to an alternative way to get to the ventricular atrium, namely through the intraparietal sulcus, which provides a wide working corridor and eliminates the necessity of transcending the tentorium. Our transtentorial approach is intended to provide a medial corridor to selected lesions that might be difficult to access using standard supratentorial exposures.

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Published online February 17, 2017; DOI: 10.3171/2016.7.JNS161766.

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