



Neurosurgical Forum

LETTERS TO THE EDITOR

Methodology of intraoperative recordings of SSEPs from the thalamocortical tract

TO THE EDITOR: We read with great interest the article by Alexandratou et al.¹ (Alexandratou A, Virjee RI, Ghare A, et al. Intraoperative stimulation mapping of thalamocortical tracts in asleep and awake settings: novel electrophysiological, anatomical, and tractographic paradigms. *J Neurosurg*. Published online January 27, 2023. doi:10.3171/2022.12.JNS221689). From an anatomical and a neurosurgical perspective, this is a very well-documented study. We congratulate the authors on their attempt to preserve the thalamocortical pathway during surgery around the somatosensory cortex, as this is still an unresolved challenge during neuro-oncological surgery. However, we write to express some concerns regarding the neurophysiological data presented, namely the recordings of somatosensory evoked potentials (SSEPs) from the presumed thalamocortical tract (TCT) recorded by a handheld bipolar probe. We wish to address two critical aspects of the neurophysiological data the authors presented.

First, the neurophysiological recordings of SSEPs from the TCT are not entirely convincing. The authors reported TCT SSEPs with a peak latency of 19.6 msec (case 1) and 24.6 msec (case 2). These are not adequate latencies for SSEPs recorded from the TCT following elicitation by stimulation of the median or ulnar nerve.² It is widely accepted that physiological peak latencies of SSEP recordings from the thalamus are around 18 msec and from the primary somatosensory cortex (S1) are around 20 msec (N20: 18.84–21.78 msec).³ Even if the tumor is located close to the S1, these long latencies do not correlate with the neurophysiological marker of the TCT. If the tumor might have an influence on the latency of SSEPs, this should be confirmed or rejected by recordings of cortical SSEPs. That could be done with SSEP recordings either from the scalp or directly from the surgically exposed sensory cortex, but the authors did not present these data in their paper. Furthermore, the authors reported TCT SSEPs from the lower extremity with a latency of 29.2 msec (case 3, Table 1 in their paper), which in our opinion is too short for an SSEP elicited from the tibial nerve (P37: 38.56–44.05 msec).^{2,3}

Even though the patients in cases 2 and 3 underwent

surgery while awake and peripheral nerve stimulation was done with a low stimulation intensity, the authors could have attempted simultaneous subcortical recordings from the bipolar probe and, in a second recording, channel cortical SSEPs from a strip electrode placed on the post-central gyrus.⁴ Figure 3 in their paper shows that those channels were available but unplugged during the mapping procedure.

Second, to differentiate noise from neurophysiological signals, such as SSEPs, it would have been useful for the authors to demonstrate evoked potential repeatability by superimposing two traces from separate trials or the repeatability of the response in a cascade mode.⁵ However, they did not show the repeatability of the signals that they recorded except in case 1 (Figure 3 in their paper). Although the authors claim that the signals in case 2 in Figure 3 show the repeatability of recordings, careful analysis of their traces cannot confirm reproducible signals.

Again, we congratulate the authors on their approach to integrating complex neurophysiological guidance into their neurosurgical resection strategy. Certainly, up to now, neurosurgeons and intraoperative neurophysiologists have put less focus on preserving the sensory system including the TCT. Thus, the study by Alexandratou et al. is an important step forward in the preservation of sensory function in glioma surgery. However, we would also like to emphasize that intraoperative signal analysis can be challenging and easily misinterpreted. The methodology needs to be developed further to make it neurophysiologically robust. Currently, we are not convinced that the data presented can be confirmed to be signals recorded from the TCT.

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Disclosures

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Response

We thank Deletis and colleagues for their interest in our work and our article. They acknowledge that mapping and preserving the thalamocortical pathways is a significant challenge, requiring continuing efforts to protect sensory systems. We would also add that the physiological importance of the sensory system and its integration to effective motor planning and motor execution have received limited attention.

Deletis and colleagues express some concerns regarding the electrophysiological recordings against published reference data, particularly the recorded latencies and reproducibility of our recordings. Their comments and observations are reasonable and clearly stated. Many experienced neurophysiologists are likely to make the same observations. Therefore, we appreciate the opportunity to address these concerns.

Our peak recorded latencies were 19.6 msec for our asleep case (case 1) and 24.6 and 29.2 msec for our awake cases (cases 2 and 3, respectively). The authors note that published physiological SSEP latencies are approximately 20 msec from the S1. Our peak latency is within published thresholds for the asleep case (case 1), slightly prolonged by 4.6 msec for the first awake case (case 2), and shorter for the lower limb (case 3). A critical difference is that pre-

viously published peak latencies were derived from scalp or strip electrodes. Both techniques are based on statistically averaging thousands of traces from large brain regions. In contrast, we obtained individual recordings from 3 individual patients, using specific points no larger than 5 mm, directly within the TCT. Such recordings have not been previously reported and have now become available. In addition, all 3 cases had substantial tumor infiltration of the S1, as well as displacement and involvement of the superior TCT. We are not proposing new latency thresholds for the SSEPs, but rather documenting our specific findings from within the TCT when displaced and affected by large tumor volumes.

The authors also suggest that concurrent cortical SSEP recordings from a strip electrode would have been helpful. We appreciate the suggestion, and this can certainly be considered by our group and other groups when replicating our methods. However, we note two caveats. First, the strip electrode will, again, statistically average recordings from a large brain area as opposed to providing focal recordings from a 5-mm region of the TCT. Second, and specific to our cases, most of the S1 was resected, as shown in the postoperative MRI and intraoperative videos. Therefore, the interpretation of SSEPs from the partially or completely resected S1 is unlikely to be straightforward.

The authors also refer to reproducibility in cases 2 and 3. We are glad they accept reproducibility in the asleep case (case 1). Reproducibility is a function of averaging and supramaximal stimulus intensity.¹ It is a common standard in intraoperative neurophysiology to average 100 traces and then cascade. However, keeping the stimulus longer and applying supramaximal signal intensity is particularly painful in awake patients. Our aim, as well as that of all groups, is to achieve maximal, safe tumor resections with minimal possible discomfort or impact to patients.

In summary, we appreciate the opportunity to address the questions by Deletis and colleagues, as they are reasonable and likely to be asked by other neurophysiologists. Our paper has described our paradigm in detail and includes a lengthy video with continuous screen recordings, a concurrent picture-in-picture demonstration of the surgical cavity, and points of contact within the cavity and adjacent to the S1, so our methodology and techniques can be analyzed and replicated.

Our individual recorded latencies represent the first of such data from within the affected TCT. They should not be compared to physiological latencies based on averaging large brain regions derived from scalp or strip electrodes. This would constitute a comparison of incomparable electrophysiological paradigms.

Concurrent interpretation of strip electrodes when the S1 is affected, or indeed resected, is likely to be problematic, but we welcome the suggestion. Finally, applying supramaximal intensity in awake patients is not practical or permissible. However, we are encouraged by the anatomical, highly accurate tractographic reconstruction of the TCT based on our positive stimulation sites (Figure 2 in our paper).

We maintain trust in our data and in our paper's closing sentence: "Further studies in larger cohorts may help to

validate this technique and improve detection and protection of the TCTs.” We invite Deletis and colleagues and other groups worldwide to help in this direction.

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Identifying the brand and model of implanted shunts for revision planning in hydrocephalus

TO THE EDITOR: Ventriculoperitoneal shunt (VPS) surgery is a challenge for neurosurgeons. As a result of infection or mechanical failure, revision is required in 50% of cases 1–2 years and in 80% of cases 10 years after implantation.^{1–3} The very promising results of a recent nationwide large-scale study conducted by Mansoor et al.⁴ to determine the full scope of shunt surgery in patients with hydrocephalus could help neurosurgeons standardize treatment and improve outcomes (Mansoor N, Gulati S, Fredriksli OA, et al. Epidemiology and practice variations

of shunt surgery for hydrocephalus: a nationwide registry-based study. *J Neurosurg*. Published online February 3, 2023. doi:10.3171/2022.12.JNS222083). Currently, it is hard to trace the manufacturers and brand names of VPSs worldwide.⁵ Therefore, the interregional practice variations in overall shunt and revision surgery that Mansoor et al.⁴ found may be partially related to whether the attending neurosurgeons could identify the brand and model of the inserted shunt before revision surgery.

The attending neurosurgeon's knowledge of the inserted shunt's brand name may affect the revision surgery plan. By obtaining this information, the surgeon can better decide which parts of the shunt need to be changed and ensure the compatibility of the parts in the revision. Different features of each brand (e.g., one piece or three pieces; a fixed or programmable valve; a distal catheter with an open end, multiple holes, simple slits, or graphite-coated slits) can provide advantages and disadvantages in the management of an individual patient.^{1–3,6–8} In some cases, it may be sufficient to simply replace the valve or distal catheter with limited surgery; however, with one-piece shunts, the entire device should be replaced. Notably, if the valve is programmable, adjusting the program noninvasively may be the only action required.

When a patient with a VPS is admitted to a hospital in a different region or country from that where the initial surgery took place and the surgical records are unavailable, the surgeon performing the revision can only gain a vague idea about the brand of the implanted shunt either radiologically or from the patient's knowledge. COVID-19 has brought additional challenges for neurosurgeons.⁹ At the peak of the pandemic, people were unable to travel, and perhaps patients with VPSs had to be treated where the attending neurosurgeons could not access their medical records. Unfortunately, the healthcare system worldwide is increasingly focused on financial profitability, and quantity is treated as more valuable than quality, thus neglecting patient follow-up and deviating from medicine's primary mission of helping patients.¹⁰

In conclusion, it is essential to have access to the previous surgical records of patients with a VPS, including the brand and model of the shunt, to be able to provide the most appropriate treatment. Therefore, a worldwide database of these patients is needed, similar to that used to monitor COVID-19 vaccine applications by brand name.

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Response

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Rotterdam CT score

TO THE EDITOR: We read with interest the article by Kashkoush et al.¹ (Kashkoush AI, Potter T, Pettitt JC, et al. Novel application of the Rotterdam CT score in the prediction of intracranial hypertension following severe traumatic brain injury. *J Neurosurg.* 2023;138[4]:1050-1057).

Severe traumatic brain injury (TBI) often results in high mortality and morbidity. It is crucial to have a prediction model in the management of intracranial hypertension (ICTN) in patients with severe TBI. Rotterdam CT scoring serves as a CT classification model for grouping patients with TBI based on numerous CT characteristics.² Kashkoush et al. investigated the application of the Rotterdam CT score in patients with severe TBI. Their

study found that the Rotterdam CT score was predictive of ICTN in patients with severe TBI. Moreover, they showed that the diagnostic accuracy of the model was improved with the addition of sulcal effacement at the vertex on head CT scans.¹

However, their study has some limitations. First, serial follow-up CT is crucial because previous studies have shown that changes in Rotterdam CT scores over time may serve as a prognostic indicator of TBI and can help determine which patients need decompressive craniectomy.² Therefore, serial CT scans might provide the progressive information for evidence-based determination of surgical decompression.³ Moreover, the authors did not provide information regarding patients' alcohol consumption. Alcohol consumption significantly reduces the Glasgow Coma Scale score in a dose-dependent manner in patients with moderate and severe TBI and with Rotterdam CT scores of 1–3.⁴

Taken all together, the study by Kashkoush et al. provided a novel application of the Rotterdam CT score for the prediction of ICTN following severe TBI. Future large-scale studies including prospective serial CT scans of the head are warranted to completely validate the Rotterdam CT score, especially in patients at high risk of ICTN, as well as to provide evidence-based medical management of severe TBI.

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Response

We would like to thank Hsu and Hueng for their comments regarding our study. We agree that obtaining serial

head CT scans is important given that changes in serial CT scans may lead to fluctuations in Rotterdam CT scores, which could alter clinical decision-making regarding interventions such as decompressive hemicraniectomy.¹ Furthermore, previous work has demonstrated that serial head CT scans can be used to monitor the intracranial progression of severe head injuries in patients who are managed without intracranial pressure monitoring.^{2,3} However, the aim of our paper was to predict which patients will develop ICHTN based on the initial CT scan of the head, which is useful in the triage and management of patients in larger trauma systems. Our paper calls for further study to determine the optimal frequency of repeat imaging, type of monitoring (neurological examinations vs serial imaging vs hemodynamic monitoring), and timing of monitoring for this patient population.

With respect to the authors' second point, we did not include the effect of alcohol consumption on Glasgow Coma Scale because this was beyond the scope of our study. The aim of our study was to investigate the association between Rotterdam CT score and the risk of developing ICHTN following severe TBI. To the best of our knowledge, there is no evidence to suggest that alcohol consumption is associated with ICHTN, which was the primary outcome of our study. Therefore, we believe that including alcohol consumption as a variable in our analyses would not significantly impact our results and conclusions.

We thank the authors for their critique of our study and agree that more large-scale prospective studies are needed to further elucidate the relationship between Rotterdam CT score and ICHTN events after TBI.

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Ventriculosinus shunts for hydrocephalus in adults

TO THE EDITOR: We read with great interest the research by Munthe et al.¹ on investigating the patency of a novel ventriculosinus (VS) shunt for the treatment of hydrocephalus by diverting CSF to the internal jugular vein (Munthe S, Pedersen CB, Poulsen FR, et al. Ventriculosinus shunt: a pilot study to investigate new technology to treat hydrocephalus and mimic physiological principles of cerebrospinal fluid drainage. *J Neurosurg*. Published online April 21, 2023. doi:10.3171/2023.3.JNS222858). In this single-center prospective interventional study, 12 adult patients underwent placement of the VS shunt investigational outlet device at the junction of the jugular foramen. The shunt remained patent and functioned well in 10 patients (83.3%) at the 6-month follow-up evaluation, and no occlusion of the internal jugular vein or thrombosis occurred. We congratulate the authors on their excellent results and appreciate their valuable contribution to the treatment of hydrocephalus.

Since the 1950s, hydrocephalus has been treated with CSF shunting to extracranial compartments, most commonly the peritoneal cavity or right atrium of the heart. However, because of the siphoning effect, ventriculoperitoneal (VP) or ventriculoatrial (VA) shunting results in non-physiological CSF diversion with high complication and revision rates in the long-term follow-up. The introduction and improvement of antisiphoning or programmable shunt devices have not significantly reduced the revision rate or extended shunt durability.^{2,3} Moreover, the expenses associated with CSF shunting and subsequent revision surgery as well as follow-up care are found to be costly, with an estimated annual cost of \$1.1 billion in the United States.⁴ To obtain physiological CSF diversion, neurosurgical pioneers have attempted to shunt CSF toward the dural sinuses, taking advantage of the antisiphoning effect of the internal jugular veins.⁵ In a VS shunt, the shunt system is short in length and confined to the skull, making the procedure technically less complex and invasive. VS shunting avoids the siphon effect and overdrainage-related complications through maintaining a decreased pressure difference and establishing a near-physiological CSF pathway. Shunt revisions and various complications resulting from peritoneal drainage can also be eliminated.

Despite these and other potential advantages, the concern about thrombotic obstruction of the distal catheter positioned in the venous sinus is the reason why VS shunting has not been entertained as a popular option in patients with hydrocephalus. The distal ends commonly drift toward and come into contact with the side wall of the vein and can become encased by endothelial overgrowth in a short period. Munthe et al.¹ introduced a novel distal outlet device consisting of three components: a nitinol frame, polyetheretherketone resistance tube, and silicone catheter. The frame keeps the outlet centrally placed and prevents it from endothelialization. The resistance tube provides built-in flow resistance, maintaining normal intracranial

pressure and avoiding overdrainage. The outcome of this innovative technology is indeed very impressive. The ability to obtain significant improvement with a high 6-month patency rate is worth noting.

We previously reported a case series of 10 patients undergoing temporal-to-frontal horn (TFH) shunt placement for the management of a trapped temporal horn.⁶ This technique effectively shunts CSF from the temporal horn to the contralateral frontal horn. There are substantial interesting parallels between the TFH shunt and VS shunt since both techniques divert CSF back to the intracranial compartment and mimic physiological drainage. A recent comparative cohort study demonstrated that, as a valveless shunt technique and one without an abdominal incision, the TFH shunt is cosmetically favorable, cost-effective, and completely free of overdrainage with equivalent revision rates to a VP shunt.⁷ Nonetheless, the frontal distal catheter still carries the risk of obstruction and shunt failure in long-term follow-up. Based on the experience of Munthe et al. and new technology, we believe that a temporal horn-to-sinus shunt, which evolves from the techniques of TFH and VS shunting, might be a potential alternative treatment for patients if a TFH shunt is not available sometime in the future.

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Response

We are grateful to Lin et al. for their positive feedback and for highlighting the benefits of VS shunts over VA and VP shunts as well as noting the potential of VS shunts for reducing healthcare costs. We are also encouraged to hear that they see an opportunity to use the same technology to expand on their own research for the management of a trapped temporal horn.

In their case series of 10 patients,¹ they successfully diverted CSF from the temporal horn to the frontal horn as an alternative to the usual temporal horn-to-peritoneum standard of care in the interest of avoiding shunt complications and overdrainage. Avoidance of shunt complications and overdrainage were also key drivers in our study. The authors noted that while the TFH shunt method avoided overdrainage, the frontal distal catheter might become obstructed in the long term.

Lin et al. suggest that their TFH shunting method could be improved by using our VS shunt technology to shunt CSF from the temporal horn to the sinus instead, thus still confining the surgery to the skull to avoid overdrainage. We concur that using the VS shunt from our study for this purpose would offer advantages. It would be less invasive than their current TFH shunting method since it would require only one burr hole rather than two, the risk of obstruction would be avoided due to the venous access port outlet design, and the collapse of the temporal horn (overdrainage) would not be a risk as it would drain to the sinus at the same pressure. We are delighted that Lin et al. recognize alternative uses for the technology and share the same enthusiasm for physiological drainage. We will follow their future research with great interest.

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Aggressive cranial dural arteriovenous fistulas: higher obliteration and recurrence rates?

TO THE EDITOR: We read with great interest one of the Consortium for Dural Arteriovenous Fistula Outcomes Research (CONDOR) studies by Abecassis et al.¹ (Abecassis IJ, Meyer RM, Levitt MR, et al. Recurrence after cure in cranial dural arteriovenous fistulas: a collaborative effort by the Consortium for Dural Arteriovenous Fistula Outcomes Research (CONDOR). *J Neurosurg.* 2022;136[4]:981-989). In this study, the risk factors for the recurrence of dural arteriovenous fistulas (dAVFs) after putative cure were identified as tentorial location, cortical venous drainage, and deep cerebral venous drainage. This is the largest follow-up study to determine the rate of dAVF recurrence after initial cure. A delayed long-term angiographic evaluation (at least 1 year after cure) is advised, particularly in cases with recurrence risk factors. This article also discussed in great detail why fistulas recurred after complete penetration of the fistulous point and drainage vein: it is generally accepted to be related to suboptimal casting of the draining vein. The recurrence of fistulas is actually the result of a dAVF that is temporarily angiographically silent but anatomically intact.

Another recent CONDOR study by Becerril-Gaitan et al. reported that Borden type II/III was an independent predictor of complete obliteration.² The study found a higher likelihood of complete obliteration among patients with Borden type II/III dAVF (OR 1.59, $p = 0.079$) compared to type I, which may reflect the curative goal and more aggressive treatments used for these high-grade lesions. Aside from the curative goal, we believe that the angioarchitecture of Cognard type III/IV dAVFs is simpler than that of dAVFs in the main sinus region, allowing for easier penetration of the fistulous point and drainage vein.^{3,4} The presence of dAVFs in the main sinus region is required to consider embolizing the fistulas while maintaining sinus patency, which cannot be achieved by occluding the draining vein (sinus). In theory, aggressive dAVFs also have a higher chance of complete obliteration.

However, are aggressive dAVFs more likely to recur after the initial cure? As mentioned in this article,¹ "Each center might have a different definition of cure, and it is not clear in this analysis whether or not thorough casting of the draining vein angiographically was incorporated into that definition for each case, as opposed to simply no residual arteriovenous shunting." It is unknown whether the recurrent fistulas were completely occluded (complete occlusion of the proximal drainage vein). Ambekar et al. also

stated that a patent channel with the Onyx cast may allow for delayed recurrence.⁵ As a result, the goal of complete occlusion is not only to penetrate the drainage veins, but also to completely occlude the proximal drainage veins.

It is difficult to achieve uniform standards in multicenter studies, and the results may be biased. While the study was being carried out, it was necessary to define what was complete embolization. We also believe that a further systematic review on the topic of recurrence is required. We sincerely appreciate the introduction of this study, which leads readers to a more in-depth discussion of dAVF recurrence.

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Disclosures

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Response

We thank Su et al. for their commentary on our report. As they summarize, we found that dAVF with cortical venous drainage (and thus Borden type III lesions) had a higher rate of recurrence after endovascular cure. A more recent investigation from the CONDOR group (Becerril-Gaitan et al.)¹ found that higher-grade dAVFs were more likely to experience complete obliteration upfront. The authors of the letter seem to question whether or not it is possible for higher-grade lesions to experience both higher rates of cure and recurrence as compared to lower-grade (Borden type I) lesions, and suggest that perhaps this dis-

crepancy stems from not uniformly defining angiographic cure across the multiinstitutional cohort.

Based on our study, we know that recurrence of a dAVF occurs exclusively in those lesions treated with an endovascular technique, which has become the first-line modality of treatment for most dAVFs. Indeed, a more aggressive approach to treatment is usually used for higher-grade lesions, not only because they are more commonly ruptured or symptomatic, but also because they are more appropriate anatomical substrates for achieving radiographic cure than Borden type I lesions, in which drainage often involves a functional venous sinus that may or may not tolerate occlusion. As we explain in our report, for the cases in which we observed recurrence, it was not clear whether or not there was definitive occlusion and casting of the immediate draining vein (given that angiographic imaging was not annotated by a centralized reviewer). What is perhaps more relevant and useful to clarify the issue moving forward is to determine whether an incomplete embolization occurs due to 1) premature and inappropriate early termination of the embolization—perhaps due to misinterpretation of the anatomical details of the dAVF, versus 2) an inability to push the embolysate to the location of interest (but surprisingly, resulting in unplanned resolution

of early venous drainage) due to reflux, off-target embolization, or simply cast solidification prior to penetration. The focus then becomes the formulation of a deliberate plan of close angiographic follow-up and/or retreatment. Although the risk of recurrence is identical between the two groups, the level of expertise and understanding of the pathophysiology is obviously more developed for cases in the latter category.

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