TO THE EDITOR: We read with great interest the paper by Orman et al.2 (Orman G, Wagner MW, Seeburg D, et al: Pediatric skull fracture diagnosis: should 3D CT reconstructions be added as routine imaging? J Neurosurg Pediatr 16:426–431, October 2015). The authors investigated the efficacy of combining 3D CT images with 2D CT images and found it useful in the diagnosis of linear skull fractures in all children. They also showed increased specificity with 3D CT images in the interpretation of linear fractures in children younger than 2 years of age. We also believe that 3D CT images should be added as part of routine imaging because in a daily pediatric emergency care unit practice most of the head CT images are interpreted by residents and clinicians on admission and missing skull fractures can lead them to overlook associated intracranial injuries not evident on the first head CT scan. Missing fractures can also preclude the performance of MRI as an alternative method to show subtle associated traumatic brain injuries.4

One point we would like to make is that although the authors emphasized that linear fractures can be missed on 2D CT when they are within the plane of the image reconstruction, they did not evaluate the role of the orientation of the fracture between readers. Linear skull fractures that are parallel or nearly parallel to the section orientation can be missed on the interpretation of CTs even in older children and sometimes even by experienced readers.3 The use of 3D CT images is helpful not only in the diagnosis of parallel-oriented linear skull fractures but also in differentiating them from vascular canals (Fig. 1 left).

The other point we would like to make is that children younger than 2 years old may show abnormalities in head shape. Neonatal calvaria may be abnormal in shape due to pressure on the head during childbirth. Plagiocephaly without craniosynostosis (posterior deformational positional plagiocephaly) associated with sleeping position (sleeping on back), congenital torticollis, abnormal vertebra, and neurological deficits is common in infants.3,5 Premature fusion of one or more sutures, craniosynostosis, isolated or rarely associated with syndromes, may exist in patients with head trauma. In patients with an abnormal head shape, the symmetrical nature of sutures may not be appreciated on 2D CT images. As radiological differ-

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**FIG. 1.** Left: Three-dimensional CT image obtained in a 6-year-old girl who fell off her bicycle, showing a parallel-oriented linear skull fracture (arrows) of parietal bone. Arrowhead indicates a vascular canal that is more clearly defined on the 3D CT image. Right: Three-dimensional CT image obtained in a 1-month-old boy who fell from a couch. The patient has an abnormal head shape without craniosynostosis (positional plagiocephaly). There is a linear skull fracture of the left parietal bone on the vertex that is parallel to the imaging plane (arrows). Figure is available in color online only.
entiation between suture lines and linear skull fractures is mainly based on the bilateral and fairly symmetrical nature of the major sutures, adding 3D CT images really helps us to improve diagnostic confidence (Fig. 1 right). Furthermore, there are some associated vascular canals and enlarged emissary veins associated with syndromic craniosynostosis that can be misdiagnosed as fracture. In brief, combining 2D and 3D CT images would be very helpful to decrease false-negative and false-positive diagnoses in infants with an abnormal head shape.

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References

Disclosures
The authors report no conflict of interest.

Response
We appreciate the interest in our article by Drs. Pekcevik and Sahin, who raised some interesting points.

The first point is that the role of the orientation of the fracture was not evaluated in our study. Drs. Pekcevik and Sahin are correct. In our study all linear fractures regardless of their orientation were evaluated by all readers. The study was not designed specifically to evaluate the differences in the orientation of linear skull fractures between the readers, although this could be pursued as a future study. Linear fractures on 2D CT can be missed when they are within the plane of image reconstruction, and the addition of 3D images should alleviate this problem.

As a second point, Drs. Pekcevik and Sahin mention that linear fractures parallel to the section orientation can also be missed in children older than 2 years of age and by experienced readers. In keeping with our study, we agree with Drs. Pekcevik and Sahin that linear skull fractures, regardless of the age of the patient or the experience of the reader, can be missed if they are in an orientation parallel to the section. The addition of 3D data to the 2D CT data set gives the reader increased confidence, as sutures and other nonfracture-related linear lucencies such as vascular canals can be easily followed and distinguished from linear fractures. However, our data showed 1) that the addition of 3D data to the 2D CT data set significantly increased specificity in the diagnosis of linear skull fractures only in children younger than 2 years of age, and 2) that the greatest increase in the specificity of the diagnosis of linear skull fractures was for the less experienced reader.

Finally, as their third point, Drs. Pekcevik and Sahin suggest that the evaluation of sutures can be challenging in children with an abnormal head shape. We agree that an abnormal head shape in children with conditions such as craniosynostosis would certainly affect the evaluation of the symmetry of sutures. In our study, we did not specifically evaluate children with craniosynostosis or conditions such as achondroplasia where prominent emissary veins or vascular channels can mimic linear fractures. Our study has shown that the use of 2D+3D CT combined demonstrates increased sensitivity in the diagnosis of linear skull fractures in all children and increased specificity in children younger than 2 years of age, regardless of head shape, age, or underlying syndromal abnormality.

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Pediatric sports-related concussions

TO THE EDITOR: The paper by Ellis et al.1 in the September issue (Ellis MJ, Leiter J, Hall T, et al: Neuroimaging findings in pediatric sports-related concussion. J Neurosurg Pediatr 16:241–247, September 2015) serves to illustrate the need for “updating” our physician and athletic management community, including the families and individuals with concussion/mild traumatic brain injury (mTBI), on the newer objective radiological brain injury evaluations available today. The retrospective chart review, diagnostic conclusions, and follow-up by a single neurosurgeon appear to have reflected out-of-date evaluative procedures. Ellis et al. reviewed data acquired in 36 patients (2% of 151 patients diagnosed with sports-related concussions [SRCs]) who underwent neuroimaging before referral to a specialty clinic. MR images were obtained in patients with focal neurological findings or symptoms or symptoms lasting more than 1 or 2 months, and those with abnormal CT findings. The abnormal CT findings

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included skull fractures, intracranial hemorrhage, arachnoid cyst, and suspected hemorrhage into an arachnoid cyst. MRI revealed intraparenchymal hemorrhage, sylvian fissure arachnoid hemorrhage, nonhemorrhagic contusion, demyelinating disease, posterior fossa arachnoid cyst, cerebellar volume loss, and nonspecific white matter changes.

The authors correctly concluded that CT scans yielded no signs of traumatic injury to structures of the brain in most cases of SRC, and that CT should be limited to patients whose symptoms suggest possibility of skull fracture or intracranial hemorrhage. These notions are correct and point primarily to structural bony or skull lesions, lesions of supportive structures of the brain, vascular injuries, or bleeds. For these other-than-brain-tissue injuries, CT and MRI are and were appropriate. However, the authors wrote that no positive findings were found, that no actual brain damage or injury was documented, and that CT and regular MRI scans ordered to study an injured brain from an SRC are of no value except to find the structural defects and bleeds as mentioned in the study. One may question if these studies are of no value in actual brain tissue injury, and are they ordered only to show other structural lesions potentially accompanying mTBI/concussion since actual brain lesions are not found by plain CT and MRI? If these non–brain tissue injuries are present (or not!), then the next logical step is to order a study that will show actual accompanying brain tissue damage in order to make the injury “visible.”

In order to meet the urgent improvement need in treatment and evaluation of concussion/mTBI, we, as care providers, must leave behind the inappropriate practice and mindset that concussion/mTBI represents a symptom-only diagnosis and a symptom-only treatment. We must help others overcome the same struggle! The standard appears to be that “if there are symptoms, then treat, and if the symptoms are resolved, then the individuals can return to their respective activity.” This, my dear colleagues, is wrong and offers a lower standard of care than, for example, stroke, which is also a brain injury, although sometimes potentially more serious than concussion/mTBI. Nonetheless concussion/mTBI treatment offers a lesser standard of care than for stroke! Stroke standard-of-care therapy involves the acquisition of many scans, serially, until the lesion is resolved, with potential physical and cognitive losses as sequelae, as are potential in mTBI/concussion.

As said, the study provides support to consider a different modality to make the actual brain injury more knowable. Greenwald et al. found that, even in the absence of symptoms and with patient nonreporting of symptoms, with more thorough cognitive testing the symptoms can be made more visible, and any re-injury or the extension of a current injury is obviated by more rest. In this instance, more thorough psychological testing is appropriate and potentially “brain saving.” Furthermore, comprehensive psychological testing is Step 1 in making the injury more visible, despite the absence or disappearance of symptoms or patient nonreporting. However, psychological testing, no matter how thorough or prolonged, may only make concussion/mTBI symptoms apparent. How do we make the injury more visible after we are eventually unable to show it through psychological testing? Psychological testing does not provide objective proof that the injured brain is healed; it merely shows symptoms.

It is an absolute fact that if/when there are symptoms after concussion/mTBI, then there is an organic brain injury. However, we as physicians and other caregivers also know that when the symptoms of an injury or illness are gone, injury or disease may still be present. This is the likely case in concussion/mTBI. And so it becomes a task for all in the chain of evaluation and care of concussed patients of all ages to seek and demand a higher standard of care than simply resolution of symptoms.

This potential is available today but sadly not often used. It appears that some physicians are not aware of the current literature on how to make brain injuries other than stroke or structural/skull injuries more visible. Diffusion tensor imaging (DTI) is an objective radiological modality currently available to study actual organic brain injury after a concussion/mTBI, and it is fairly easily performed by radiologists, if only asked for by you, my dear colleagues! The literature on DTI in brain injury and axonal tractography is robust, and many papers support its use.4 Narayana et al. have suggested that after 90 days postinjury DTI can detect no further brain damage in white matter tracts. What is the average waiting time for symptomatic or asymptomatic concussed/mTBI patients to return to prior or activities? Niogi and Mukherjee have written that “unlike computed tomography or conventional magnetic resonance imaging, DTI is sensitive to microstructural axonal injury, the neuropathology that is thought to be the most responsible for the persistent cognitive and behavioral impairments that often occur after mTBI.” In the excellent review of mTBI and the use of DTI by Shenton et al., the authors used the term “post-concussive syndrome” instead of “chronic mTBI” and this seems appropriate. The point is that the more often DTI (or any other potential objective radiological study) is demanded by those in the treatment chain (ER docs, sports docs, primary care docs, orthopods, internists in the ICU, neurologists, neurosurgeons, etc.), the better and more technologically sophisticated DTI and these studies will become, or they could even morph into an entirely different technology. However, DTI is what we have now, today, and its effectiveness in detecting an injury in patients with concussion/mTBI is robustly supported in the literature, as underscored here by just a few examples drawn from supportive literature. There are likely a few naysayers, but the need to use this tool and make it part of an improved and expected standard-of-care protocol far outweighs any specious or scientifically unfounded objection to it. Our athletes (young and old, professional and amateur), our soldiers, our first responders, those with accidental concussions of all types, must be helped by our making the injured brain more visible, with and without presence of symptoms. DTI ought to be a part of the standard of care, to be performed serially, just as scanning is for stroke. Many concussion/mTBI patients sustain multiple brain injuries in their activities and have been given symptomatic to symptom-free care, which is not an acceptable standard of care or treatment end point.

I have a concern that the title of this paper may mislead many physicians and others in the chain of care to believe that no neuroimaging modalities are of use in concussion/
mTBI. This is simply not the case, given the availability and relative ease of specialized MRI (i.e., DTI) and potentially other objective tests. Ellis and colleagues reviewed a series of patients and offered a hugely impactful and influential result based on seeing inappropriate and outdated radiological modalities that have allowed an unacceptable standard of care. Their results must be a beginning of turning to objective testing to show that the injured/concussed brain is healed, and not just merely symptom free. The paper gives welcome support to other evaluative methods than regular CT and regular MRI for mTBI/concussion in a pediatric population.

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References

Disclosures
The author reports no conflict of interest.

Response
We thank Dr. Griffin for his letter, which provides a review of our recent article and highlights a number of important issues and knowledge gaps in the field of concussion and mTBI.

As you are aware, the field of concussion has long held the notion that SRC is a functional injury and not associated with any structural injury to the brain. As such, most consensus position statements suggest that conventional neuroimaging studies such as CT and MRI show normal findings and thus offer little value to the evaluation and management of individuals who have sustained a concussion or mTBI. Unfortunately there is little empirical evidence to support these conclusions. While our preliminary study detected traumatic abnormalities in a small proportion (11%) of pediatric SRC patients who underwent imaging, it is misleading to assume that because the majority of these studies were normal that they did not provide clinical value to the treating physician and the patient. As illustrated in the study, children and adolescents who present with prolonged or worsening neurological symptoms following head injuries may also harbor other neurological conditions that, in many cases, can only be detected and appropriately managed following the judicious use of structural neuroimaging.

We agree that we have an obligation to our patients to look beyond treating individual symptoms and offer a more comprehensive, multidisciplinary approach to concussion and TBI. Thankfully, the appropriate use of physical examination tools and graded aerobic treadmill testing has increasingly permitted us to classify patients with acute SRC and post-concussion syndrome (PCS) and direct them toward emerging evidence-based therapies that target the pathophysiological mechanisms governing individual symptoms. In view of the fact that concussion is, as Dr. Griffin correctly points out, an “organic brain injury,” it is incumbent on leaders in the field to continue to promote the value of comprehensive care by a multidisciplinary team of TBI experts working within their scope of practice and training. We agree that these teams should always include clinical neuropsychologists, the only professionals with optimal training in the administration and interpretation of neuropsychological testing that can assist in confirming complete neurocognitive recovery in concussion patients.

We also agree that there remains an urgent and persistent need for neuroimaging assessment tools that can make concussion “more visible” and provide valuable biomarkers that impact the clinical management of individual concussion patients. While there has been an explosion of neuroimaging studies in SRCs utilizing advanced techniques such as DTI, task-based and resting-state functional MRI, and resting cerebral blood flow, none of these technologies have advanced beyond experimental use. As Dr. Griffin points out, alterations in white matter integrity have been demonstrated in groups of patients with mTBI, but they have also been observed in those who have sustained subconcussive head impacts, and in some cases DTI indices have failed to discriminate between subject groups with and without PCS. While we hope that future technical refinements will help clarify inconsistencies observed among DTI studies in SRC and mTBI, at present the failure of this modality to provide reliable and consistent quantitative biomarkers that can be used to diagnose, classify, prognosticate, or confirm recovery in individual concussion patients leads the authors to question how routine use of this technology would impact clinical decision-making in our patients. On the other hand, emerging work suggests that MRI-based cerebrovascular reactivity mapping may hold promise to help visualize and quantify the functional alterations that mediate PCS symptoms in individual patients, but more work remains to be completed to validate this technique, especially in adolescents.

Lastly, we acknowledge the reader’s concerns regarding the title of our recent article, the conclusion, and the current standard of concussion care. As we indicated, the decision to order neuroimaging studies, and include certain sequences, is impacted not only by patient factors, but
also by the clinical judgment and experience of the multidisciplinary team, which undoubtedly contributes to wide variability in the standard of care across centers and health care providers. Nonetheless, we believe the findings of this preliminary study clearly support the conclusion that currently validated clinical neuroimaging studies are normal in the majority of pediatric SRC patients imaged, but in selected cases these studies can provide clinically valuable information that impacts the management of children and adolescents presenting with neurological symptoms following concussion and TBI.

References

Filum terminale in tethered cord syndrome

TO THE EDITOR: I have read with great interest the clinical article by Thompson et al. (Thompson EM, Strong MJ, Warren G, et al: Clinical significance of imaging and histological characteristics of filum terminale in tethered cord syndrome. J Neurosurg Pediatr 13:255–259, March 2014). The importance of this well-documented paper stems from their assertion that surgical intervention is highly effective in reversing the signs and symptoms of properly diagnosed (that is, “true”) tethered cord syndrome (TCS)—at least 90% of their cases.

The importance of accurate TCS diagnosis is further emphasized by earlier assertions that surgery is ineffective in patients with signs and symptoms and even anatomical abnormalities that are similar to those of TCS but attributable to other causes. Therefore, in considering treatment options, it is essential to avoid diagnostic misinterpretations and to distinguish true TCS from TCS-mimicking disorders.

To enhance understanding as a basis for TCS diagnosis, Thomson and colleagues explored clinical and anatomical abnormalities in pediatric TCS patients with an emphasis on MRI and histological studies of the filum terminale (FT) and on the statistics of multiple clinical findings. Their study confirmed that the filum in these patients was filled by fibrous or fibroadipose tissue (the latter always the component of fat). The filum in these patients lacked normal elasticity and were exerting a traction effect on the spinal cord.

My concern is directed at their introductory statement that the pathophysiology of TCS is unknown. In fact, much is known about TCS pathophysiology and such knowledge enhances our ability to properly diagnose TCS and to define treatment. The key fact is that neurological deficits in patients with true TCS result from a lesion due to tethering (that is, stretching) of the spinal cord with the lesion localized cephalic to the level of the inelastic FT.

Insights into TCS pathophysiology have been derived from anatomical and physiological studies. For example, links between embryology and the development of spinal dysraphism are supported by findings of tufts of hair in the lumbar area, an elongated cord and thickened filum, as well as aberrant nerve fibers in the filum. These findings suggest additional clues that can be used
to initiate a differential diagnosis between TCS and TCS-mimicking disorders.\textsuperscript{10} Further, our findings of a postero-medially displaced filum and conus on MRI\textsuperscript{13,17} also assist in confirming a TCS diagnosis, as does intraoperative stretch testing of the filum.\textsuperscript{14,18} Although reflectance dual wavelength spectrophotometry for oxidative metabolism study\textsuperscript{10,12,22} is not practical for practicing neurosurgeons because of the extra technological time required during surgery, additional information about TCS pathophysiology has been derived from experimental and clinical studies, which have shown impaired oxidative metabolism in the tethering-induced stretched region of the spinal cord and its reversal after untethering.\textsuperscript{14,18,21}

With these insights into TCS pathophysiology, it has been possible to categorize patients with spinal dysraphism and occult TCS (modified from Yamada et al.\textsuperscript{15}). Category 1 is considered true TCS. This category includes patients in whom the caudal end of the spinal cord is attached by an inelastic structure such as a fibrous or fibroadipose filum, sacral myelomeningocele (MMC), or caudal lipomyelomeningocoele (LMMC).\textsuperscript{3,15} Neurological dysfunction seen in patients in Category 1 derives from pathophysiology attributable to stretching of the conus and/or lumbar segments of the spinal cord.

Category 2 is considered partial TCS. This category includes patients with a small dorsal or transitional LMMC,\textsuperscript{3,15} those with a small MMC attached to the dorsal aspect of the conus or lowest lumbar segments, and those with fibroadipose filum and conus.\textsuperscript{4,14} In these patients, neurological dysfunction cespidual to the lesion is reversible, whereas dysfunction in the affected segments is only moderately reversible or nonreversible,\textsuperscript{13} the latter because of fibrous or fibroadipose invasion into the cord parenchyma.

Category 3 includes patients with a TCS-mimicking disorder that results from a large MMC, LMMC, or dermoid, which covers most of the lumbosacral area. Because of extensive fibrous or fibroadipose invasion into the cord, no neurological reversibility is expected in these patients. Pang and colleagues’ chaotic LMMC corresponds to this category.\textsuperscript{8} According to our experience, the spinal cord after removing a large lumbosacral LMMC showed no redox response from reflectance spectrophotometry,\textsuperscript{11} indicating that this is an example of a TCS-mimicking disorder.

Category 4 includes patients with a large MMC or LMMC associated with a barely developed lumbosacral cord. Studies show that there is no functional reversibility in these patients.

In summary, the study by Thompson et al. adds important insights into the diagnosis of TCS and should be well considered by those providing clinical management of this disorder.

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\end{enumerate}
Response

We are grateful for Dr. Yamada’s thoughtful and supportive commentary regarding our paper. His contribution to our best understanding of the pathophysiology of TCS based on changes to oxidative metabolism in the distal spinal cord represents a major advance in the field. Nevertheless, a number of theoretical and practical questions remain.

In his letter, Dr. Yamada outlines 4 categories of TCS or TCS-mimicking anatomy on the basis of a correlation with the proposed underlying pathophysiology. Importantly, these physiologically based categories reflect a significant overlap in anatomical presentation, precluding definitive preoperative categorization based on MRI-defined anatomy alone. Similarly, we have found an imperfect correlation between the presence of TCS and the imaging-derived anatomy of the FT (thickness, fat content, and cord position). Filum terminale histology in patients with presumed TCS also demonstrates a spectrum of abnormality, as reported in our study.

In sum, TCS—whether determined prospectively by imaging and clinical characteristics or retrospectively by FT histology—lacks precise diagnostic boundaries. Patient selection for surgery, while uncertain, is very important to ensure that appropriate interventions result in defined clinical benefit. Further progress in this area will result from the use of validated outcome measurement tools specific to TCS as well as the accumulation of prospective, high-quality registry data.

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Suboccipital decompression without dural opening

TO THE EDITOR: I read with great interest a recently published report by Kennedy et al.1 (Kennedy BC, Kelly KM, Phan MQ, et al: Outcomes after suboccipital decompression without dural opening in children with Chiari malformation Type I. J Neurosurg Pediatr 16:150–158, August 2015). I was very pleased to see this article has the same conclusions we reported in 2002,2 although their series was much larger, consisting of 156 patients compared to our preliminary report of 22 patients.

I wholeheartedly support the conclusions of the article; however, I must disagree on the need for “numerous vertical scoring incisions,” which the authors apply to the outer layer of the dura. During my residency, my pediatric neurosurgery mentor, Luis Schut, MD, demonstrated to me that after bone decompression and sectioning of the atlantooccipital ligament in children and adolescents, the dura visibly expands. He emphasized that in the dura of young patients there are elastic fibers, which are not present in adult patients, in whom the elasticity of the dura is gone and the dura is far more rigid and less expansive. I applied this technique in cases reported on in our manuscript.1 The cultural impact of adult neurosurgery on pediatric neurosurgery is probably what influences the decisions of pediatric neurosurgeons, in needing to open the dura or to score it.

I wish to congratulate the authors on the excellent description of the presurgical, surgical, and follow-up outcomes of their patients. The current report by Kennedy et al. adds to the literature the advantages of not opening the dura and avoiding its subsequent complications. Shweikeh et al. recently emphasized the complication rates of patients who undergo durotomy (2.3%) versus those who do not undergo durotomy (0.8%).3 They also noted the need for longer hospital stays in the patients in whom durotomy is performed.

As more articles are published that emphasize the concept of reducing the complication rate in patients and achieving comparable outcomes in patients who have had only bone decompression compared with those who have undergone durotomy, we may create the behavioral change that is needed so that it can become a sustainable cultural change.

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Disclosures
The author reports no conflict of interest.

Response

We very much appreciate the earlier contributions of Dr. James, as well as his careful reading of our manuscript. We agree that the elastic nature of the dura of young people is an essential factor allowing such good outcomes...
after decompression without the need for opening of the dura. Symptom resolution and syrinx resolution can take time after this type of surgery (data in press), and it could be that the dura, due to its elasticity, continues to expand postoperatively in the absence of the resected bone. Certainly there are many children who will improve without split-thickness durotomies, but given the extremely low risk in our experience of performing these durotomies, with no CSF leaks encountered, we believe the technique could enhance or accelerate the process of dural expansion responsible for symptom resolution.

We believe that further study and open discussion of both the indications for, and techniques employed in, less invasive surgeries will continue to help us provide safer, more effective care for our patients, and we thank Dr. James for his interest.

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INCLuDE wHEN cITING
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