Helmets and synostosis

To The Editor: We read with concern the recent article by Sood and coworkers (Sood S, Rozzelle A, Shaqiri B, et al: Effect of molding helmet on head shape in nonsurgically treated sagittal craniosynostosis. Clinical article. J Neurosurg Pediatr 7:627–632, June 2011). The authors report normalization of the cranial index in 4 patients with sagittal synostosis by using only helmet therapy and conclude that “surgery may be unnecessary in some patients if molding helmets are just as effective.” We think this statement is both misguided and misleading and has the potential to harm children with this condition.

The authors defend their controversial treatment by arguing that the medical indications for operative treatment of single-suture craniosynostosis are largely unfounded. They trivialize the relevance of increased intracranial pressure (ICP) associated with single-suture synostosis by observing that operative intervention does little to normalize ICP and that such procedures have not been demonstrated to affect cognitive outcomes. In fact, the incidence of elevated ICP in sagittal synostosis has been reported widely in the literature. Rates of elevated ICP for isolated sagittal synostosis are historically considered to be at least 5%–25%, but recent studies have shown elevated ICP in 81% of untreated older children.1,2,5–7 Studies have also clearly demonstrated that cranial expansion effectively returns the pressure to normal.5 Furthermore, the observation that development scores do not improve following operative intervention does not imply that there is no derived benefit. Renier and Marchac1 noted worse developmental outcomes in patients who had cranial expansion at a later date than those treated earlier. Studies demonstrating no change in developmental and cognitive function following operative release could be reasonably interpreted as follows: these parameters might have worsened without surgery. Thus, most craniofacial surgeons and neurosurgeons would disagree with the statement that the rationale for operative intervention “remains cosmetic,” and this statement simply ignores years of literature in the field of craniosynostosis.

The use of a helmet orthosis to redirect growth in the absence of suture release also raises significant concerns. As Sood et al. note, the orthotic restrains anterior-posterior expansion and “redirects the growth to the squamosal suture.” In the absence of suturectomy, the fused parietal bones cannot widen and the vertex of the cranium will remain narrow. The only physiological mechanism by which the fused parietal bones could conceivably widen would be bone remodeling. However, remodeling occurs far too slowly to effect significant shape change in the brief time that the authors utilized the helmet. Consequently, the only area in which cranial expansion could occur is in the temporal bones. Lateral displacement of the temporal bones will lead to an improved cephalic index, but such a temporal bulge without parietal widening produces an unusual head shape. All of the apical pictures and CT scans in their article demonstrate persistent parietal narrowing and bilateral temporal bulge. A frontal view would be most demonstrative of this resultant shape abnormality but was not included for any patient. It is not simply enough to normalize an anthropometric number without normalizing the shape of the head.

Even if this cranial shape correction were acceptable, it is dubious whether temporal expansion alone provides enough room for brain growth. Skeptics of suturectomy and postoperative helmet therapy have raised concerns about the restrictive effects of hemming on cranial expansion and the possibility that the craniotomy site will re-fuse and negate the benefit of the procedure. Indeed, Sood et al. rely on this latter argument to justify not releasing the suture prior to hemming. However, recent evidence from our group confirms that neither concern is valid. Our study appears in the same edition of the Journal of Neurosurgery: Pediatrics and confirms that there was a significant jump in percentile head circumference following suturectomy and helmet therapy for sagittal synostoses, which persisted for at least 2 years after the procedure.4 In addition, we have seen that the craniectomy site does not typically re-fuse (as was observed following historical suturectomy without hemming) but instead forms a neosuture that allows subsequent cranial expansion. Thus, lateral expansion of both the parietal and temporal bones after suturectomy and hemming is robust enough to more than compensate for any restriction of growth in the sagittal plane. Most traditional open cranial expansion procedures also produce an increase in head circumference percentile. Consequently, any treatment that does not lead to such a change (such as that presented by the authors) raises concerns that there is still inadequate cranial volume. The absence of endocortical scalloping at the time of open decompression does little to allay these apprehensions since this is an unreliable marker for pressure in very young patients.

We believe that Sood et al. have provided little data to support the effectiveness of their technique to adequately correct the aesthetic concerns, and more importantly they have ignored the issue of intracranial hypertension in synostosis by dismissing it as a non-issue. The indications for operative treatment of single-suture synostosis are still subject to debate, but it is dangerous to dismiss years of clinical experience regarding the effect of craniosynos-
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tosis on ICP and to treat an unnaturally closed cranial suture by restricting compensatory growth at the patent sutures. If the authors were correct that operative intervention is not necessary, it would be great for children with this condition. However, if they are wrong—and there are several reasons to believe that they are—they are putting children at significant risk by proposing such a treatment. We recommend that the authors consider institutional review board approval before treating any more patients in this fashion.

Mark R. Proctor, M.D.
Boston/Harvard Medical School
Boston, Massachusetts

Gary F. Rogers, M.D., J.D., M.B.A., M.P.H.
Children’s National Medical Center
Washington, DC

Disclosure

The authors report no conflict of interest.

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RESPONSE: Drs. Proctor and Rogers have raised 2 concerns. The first concern is that of increased ICP. It seems to us that they are suggesting surgery for patients who have sagittal craniosynostosis to prevent the possibility of subsequent increased ICP. In their endeavor to push this agenda, they have misquoted the recent paper by Seruya et al. as reporting an intimidating 81% incidence of increased ICP in untreated isolated sagittal suture craniosynostosis (SSC). It is misunderstandings like this that sometimes go down the literature and become historically accepted facts. In fact, Seruya and colleagues’ paper documented an 81% increased rate of ICP in patients clinically suspected of intracranial hypertension and not in the entire group of untreated patients. We agree, how-

Fig. 1. Front face view of patient showing lack of temporal bossing from the use of a molding helmet.