INTRODUCTION

Craniosynostosis: modern treatment strategies

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In this issue of *Neurosurgical Focus*, we sought articles that provided new insights regarding our understanding of the pathophysiology, evaluation, and management of patients with the various forms of craniosynostosis.

Kosty and Vogel summarize the available basic research on important topics relating to the molecular underpinnings of both sporadic and syndromic craniosynostoses. In their article, they offer possible future molecular therapies that may aid in postoperative treatment by promoting suture patency and preventing early recrudescence. Taking advantage of medical therapies, Kosnik-Infinger et al., with the same senior author as the previous paper, report on a small case series of patients with congenital hypophosphatasia who were treated perioperatively with enzyme replacement therapy.

Christian et al. perform a systematic review of the available literature from 7 studies and summarize the incidence of postoperative intracranial hypertension following surgical correction of craniosynostosis. The authors report a higher incidence of intracranial hypertension in spite of surgical correction in patients with sagittal synostosis and patients with nonsyndromic craniosynostosis undergoing remodeling without orbital advancement. The ability to identify children at risk for impaired skull growth and intracranial hypertension was explored by Rijken et al. This group reported on the value of the occipitofrontal circumference in predicting intracranial volume in patients after repair of syndromic and complex craniosynostosis repairs. In spite of severe cranial deformities, the authors report a strong correlation between occipitofrontal circumference and intracranial volume for all forms of syndromic and complex craniosynostoses. The strongest correlations were observed with Apert and Muenke syndromes.

Readers will also find novel basic articles within this *Neurosurgical Focus* issue. Bonfield and colleagues report on the effect of surgery on diffusion tensor imaging in rabbits with familial coronal craniosynostosis, a model of bilateral coronal craniosynostosis. The authors found greater white matter abnormalities, which worsened over time in affected rabbits compared with wild-type controls. Furthermore, affected rabbits that underwent early sutu-

rectomy had no significant differences from wild-type controls, suggesting that surgical treatment prevents the deleterious effects of craniosynostosis.

Included in this issue are several works focused on technical surgical considerations in the treatment of craniosynostosis. Courtemanche et al. from the University of British Columbia detail their "cranial orbital buttress technique" for metopic and unilateral coronal craniosynostosis, which provides biomechanical stability of the advancement without the need for expensive plates and screws. The authors describe their surgical nuances and offer the advantages of this less resource-intensive technique.

Guzman et al. from Switzerland take full advantage of modern software and hardware by creating a modernized version of the Marchac templates using 3D imaging techniques and printing. They have utilized virtual mirroring techniques and superimposed patient images to age-matched controls. Using these virtual models, the authors could preoperatively fabricate individualized surgical templates. These techniques move toward creating standardized, yet individualized, repairs.

Arko et al. at the Children's Hospital of Philadelphia describe their experience using spring-mediated cranial remodeling after sagittal suturectomy in 22 patients. The authors compared their results to the pooled results from previously published series. Using their minimally invasive technique, the authors report excellent improvements in the cranial index with significantly longer intensive care unit stays but shorter overall hospital stays and less blood loss.

Mehta et al. describe a difficult and instructive case of upward transtentorial herniation related to a craniofacial repair in a child with Muenke syndrome. The authors postulate that this occurred as a result of excessive loss of supratentorial CSF leading to a remote cerebellar hemorrhage. The loss of CSF was believed to have occurred during the postoperative period via a subgaleal Hemovac, which was thought to be draining CSF. The authors detail the postoperative events and surgical procedures that ensued in an effort to manage this difficult case.

<http://thejns.org/doi/abs/10.3171/2015.3.FOCUS15133>

INCLUDE WHEN CITING DOI: 10.3171/2015.3.FOCUS15133.

DISCLOSURE The authors report no conflict of interest.