Midfacial growth and surgically induced growth arrest

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Introduction

Midfacial growth arrest is a common occurrence in patients who have had cleft palate operations but surprisingly, children whose cleft palates have not been surgically repaired have nearly normal midfacial growth [1,2]. The mechanisms responsible for surgically induced midfacial growth arrest are unknown. We hypothesized that the midpalatal suture complex acts as a midfacial growth center. We examined the molecular signals and mechanical forces involved in forming this presumptive growth center then tested whether surgical disruption of the suture complex in neonatal mice contributed to growth arrest of the midface.

Materials and Methods

The distribution of Wnt and Hedgehog responsive cells during midpalatal suture development were mapped using *Axin2*$^{LacZ/+}$, *Gli1*$^{LacZ/+}$, and *Ptc1*$^{LacZ/+}$ mice. FE modeling was used to generate strain maps of the forces acting on palate development and on the palate after surgery. Micro-CT, histology, qRT-PCR, and immunohistochemistry were used to quantify changes.

Results

The midpalatal suture forms between two intramembranous bones that in response to suckling and tongue forces, transform into endochondral growth sites, similar to the growth plates found on long bones. The prenatal distribution of Hedgehog and Wnt responsive cells suggests that this transformation involves both signaling pathways. The resulting cartilage growth plates of the midpalatal suture complex act as growth centers and mucoperiosteal denudation of the site in postnatal animals results in a growth arrest and premature fusion of the suture (Figs. 1,2).

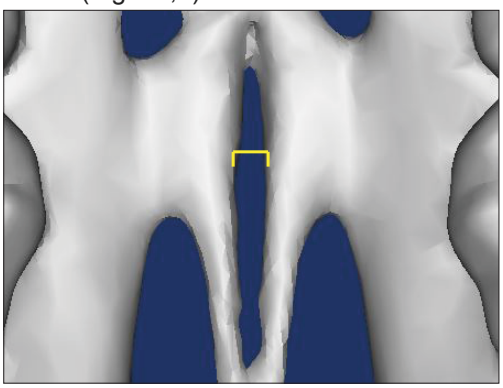

Figure 1. Micro-CT of intact palate; yellow bracket demarcates the midpalatal suture.

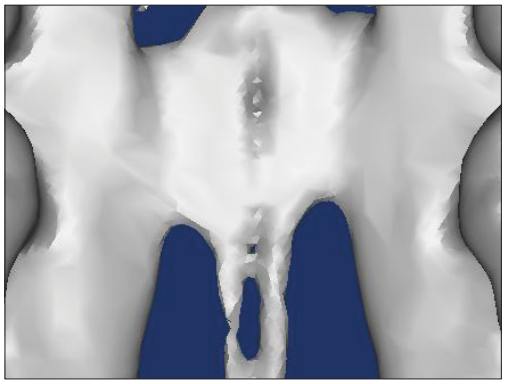

Figure 2. Micro-CT of injured palate.

Conclusion

The midpalatal suture complex develops between the palatal processes that begin as intramembranous bones but transform into endochondral growth plates in response to mechanical forces. Raising a flap disrupts these growth plates and is sufficient to arrest growth. Although the suture complex regenerates, subsequent skeletal growth is compromised. These data strongly suggest that disruption of suture complexes, which have intrinsic growth potential, is a contributor for midfacial growth arrest associated with palatal reconstruction.

References

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2. Savaci N, Hoşnuter M, Tosun Z, Demir A. Maxillofacial morphology in children with complete unilateral cleft lip and palate treated by one-stage simultaneous repair. Plastic and Reconstructive Surgery 2005;115: 1509-17.

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