Apert syndrome (AS) is one of at least nine disorders considered members of the FGFR-1,-2, and -3-related craniosynostosis syndromes. Nearly 100% of individuals diagnosed with AS have one of two neighboring mutations on Fgfr2. The cranial phenotype associated with these two mutations includes coronal suture synostosis, either unilateral (unicoronal synostosis) or bilateral (bicoronal synostosis). Brain dysmorphology associated with AS is thought to be secondary to cranial vault or base alterations, but the variation in brain phenotypes within Apert syndrome is unexplained. Here we present novel 3D data on brain phenotypes of mice each carrying one of the two Fgfr2 mutations associated with Apert syndrome. Our data suggest that the brain is primarily affected, rather than secondarily responding to skull dysmorphogenesis. Our hypothesis is that the skull and brain are both primarily affected in craniosynostosis and that shared phenogenetic developmental processes affect both tissues in craniosynostosis phenotypes.