20th International Conference on

Pediatrics & Primary Care

September 03-04, 2018 | Zurich, Switzerland

Telencephalic flexure and malformations of the lateral cerebral (Sylvian) fissure

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After sagittal division of the prosencephalon at 4.5 weeks of gestation, the early fetal cerebral hemisphere bends or rotates posteroventrally from seven weeks of gestation. The posterior pole of the telencephalon thus becomes not the occipital but the temporal lobe as the telencephalic flexure forms the operculum and finally the lateral cerebral or Sylvian fissure. The ventral part is infolded to become the insula. The frontal and temporal lips of the Sylvian fissure, as well as the insula, all derive from the ventral margin of the primitive telencephalon, hence may be influenced by genetic mutations with a ventrodorsal gradient of expression. The telencephalic flexure also contributes to a shift of the hippocampus from a dorsal to a ventral position, the early rostral pole of the hippocampus becoming caudal and dorsal becoming ventral. The occipital horn is the most recent recess of the lateral ventricle, hence most vulnerable to anatomic variations that affect the calcarine fissure. Many major malformations include lack of telencephalic flexure (holoprosencephaly, extreme micrencephaly) or dysplastic Sylvian fissure (lissencephaly, hemimegalencephaly, schizencephaly). Although fissures and sulci are genetically programmed, mechanical forces of growth and volume expansion are proposed to be mainly extrinsic (including ventricles) for fissures and intrinsic for sulci. In fetal hydrocephalus, the telencephalic flexure is less affected because ventricular dilatation occurs later in gestation. Flexures can be detected prenatally by ultrasound and fetal magnetic resonance imaging and should be described neuro-pathologically in cerebral malformations.

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Clinical Pediatrics: Open Access
ISSN: 2572-0775

Primary Care 2018
September 03-04, 2018

Volume 3

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