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Brain Commissural Anomalies

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1. Introduction

The human brain commissures include the corpus callosum (neocortical), the anterior commissure (paleocortical), the fornix (archicortical) [the hippocampal commissure (also called commissure of psalterium Davidi or David's lyre in the older literature)] (Raybaud, 2010**)** and the posterior commisure (Keene, 1938). The largest of the commissures in advanced mammals is the corpus callosum that holds its name from its compactness (Raybaud, 2010) which develops embryologically in intimate relationship to the hippocampal formation, fornix, septum pellucidum, and cingulate gyrus (Swayze et al., 1990). It has already been accepted that the commissural fibers are important for transfer of complex cognitive information between the brain hemispheres (Zaidel, 1994; van der Knaap & van der Ham**,** 2011) and coordinated transfer of information is essential for the cerebral functions (Moldrich et al., 2010). In normal condition, commissural fibers must be actively guided across the midline to reach their targets in the contralateral hemisphere. When the underlying mechanisms regulating the guidance of commissural fibers fail, pathological dysgenesis of one or more commissures ensues. It is suggested that a complex set of cellular and molecular mechanisms regulate commissural development (Ren et al., 2006). Malformation of the corpus callosum is a various condition, which can be observed either as isolated form or as one manifestation in the context of congenital syndromes (Schell-Apacik et al., 2008). Based on survey of 596 network families, the most frequently clinical findings reported about agenesis of the corpus callosum are developmental delay, visual problems, language delay, seizures, and muscle- tone issues (Schilmoeller & Schilmoeller, 2000). Furthermore, agenesis of the corpus callosum results in disabilities in social cognition that appears to be secondary to deficits in complex cognitive operations such as reasoning, concept formation, and problem solving (Doherty et al., 2006). Also, there is no evidence that individuals with partial agenesis of the corpus callosum have better outcomes than individuals with complete agenesis the corpus callosum (Moes et al., 2009). Although, the embryology, anatomy, functions, anomalies and molecular mechanisms of the human brain commissures have been extensively studied over the past years. However a need to an overall and new collection on the basis of the other recent studies was seriously felt. Therefore this chapter is to provide a collection of the fundamental principles of the embryogenesis, organization, congenital malformations of brain commissures. The chapter presents new information about prevalence, the brain disorders associated with commissural anomalies, the etiology and the pathogenetic mechanisms that have been understood in recent years in this issue in the neurosciences.

2. Embryology of the brain commissures

2.1 Embryology of the corpus callosum

The corpus callosum is a new phylogenetic acquisition of the placental mammals (Raybaud, 2010) that develops from anterior to posterior pattern **(**Richards et al., 2004**)** through a process of interhemispheric midline fusion with groups of specialized midline glial guiding the callosal fibers to the other side (Raybaud, 2010). The corpus callosum begins to differentiate as a commissural plate (Rakic & Yakovlev, 1968).within the dorsal third of the lamina terminalis at about 39th embryonic day (Sarnat, 2007). The primitive lamina terminalis corresponds to the closing point of the anterior neuropore. Its dorsal part grows and forms the lamina reunions (6-8 intra uterine weeks). From ventral to dorsal, the lamina reunions **(Fig. 1)** gives rise to the area praecommissuralis (origin of the anterior commissure), to the primordium hippocampi (10 intra uterine weeks,fornix), and to the massa commissuralis (10 intra uterine weeks., corpus callosum) (Destrieux et al., 1998).The plate acts as a passive bed for axonal passage and provides a preformed glial pathway to guide decussating growth cones of commissural axons (Silver et al., 1982).In the human embryo the genu of corpus callosum begins to develop around 8th week after conception (Giedd et al., 1996) and inter-hemispheric crossing fibres begin to transverse the massa commissuralis in this region at 11 to 12 weeks post-conceptional age(Griffiths et al., 2009) and progress caudally, forming the body (corpus) and the splenium (Rakic & Yakovlev, 1968), so that at 18 weeks' gestation the genu and body are detected cleanly; but the splenium is thin and not fully developed (Malinger & Zakut ,1993).The last part of the corpus callosum that to form at the weeks 18-20 post-conceptional ages is the rostrum (Griffiths et al., 2009; Destrieux et al., 1998). It is reported that the adult morphology of corpus callosum is achieved by 16.4 weeks (115 days) (Loser & Alvord, 1968) so that it is clearly identified. The studies have shown that the linear association are between the corpus callosum length, thickness and width, with age before **(** Achiron & Achiron, 2001) and after birth **(**children and young people aged 4 to 18 years) **(** Giedd et al., 1996; Pujol et al., 1993). The length of the corpus callosum increases a 10-fold during gestation and rapid growth of thickness increases during the period between 16 and 20 weeks' gestation. Additionally, the maximal growth of the corpus callosum width and thickness was observed between 19 and 21 weeks' gestation, while the growth of its length appeared to be constant. Further growth is accelerated until 21–22 weeks and then remains stable throughout the rest of gestation. This rapid development of the fetal corpus callosum depends on the first phase of neuronal migration (Achiron, 2001) and follows the expansion of the hemispheres, in a rostro-caudal and then dorso-ventral circular movement (Destrieux et al., 1998).The studies have been shown that, Although the basic structure of the corpus callosum is completed by 18–20 weeks' gestation, but continues to increase in size over the third trimester (Malinger & Zakut, 1993) and grows dramatically during the first 2 postnatal years (Keshavan et al., 2002).The results of these evolutions correspond to axonal elimination and myelination and progressively changing pattern of callosal connections of the newborn and infant into the adult pattern. In spite of the development of the corpus callosum from anterior to posterior pattern ,the preoligodendrocytes are thought to appear first in the genu and splenium (Huppi et al., 1998) and attains the adult levels by the age of 10 years (Yakovlev & Lecours, 1967).Of course ,the Magnetic resonance imaging studies indicate that the maturation in the corpus callosum may be more protracted.Differences in the size and form of the corpus callosum in adults have been shown to relate differences in hemispheric representation of cognitive abilities (Witelson, 1989).
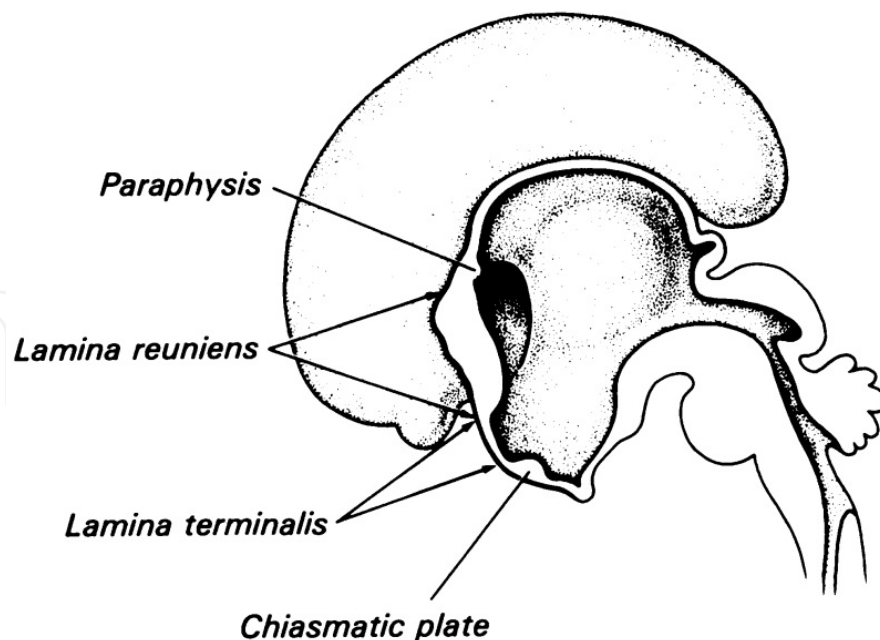
Fig. 1. Rostral midline telencephalon at approximately 7 week's gestational age. Thickening of dorsal aspect of thin rostral wall of telencephalon (primitive lamina terminalis) represents lamina reuniens of His, which will eventually form precursors of corpus callosum and anterior commissure (Barkovich & Norman, 1988)

2.2 Embryology of the anterior commissure

The anterior commissure contains the paleopallial **(**Lamantia & Rakic, 1990**)** and **t**he neocortical parts (Guénot, 1998). It is phylogenetically, the oldest of the great forebrain commissures (Raybaud, 2010; Griffiths et al., 2009). At the 8th week of development ,the early fibres of the anterior commissure appear laterally, gradually ,these fibers come nearer to the midline at the week 9, cross the midline at the week 9 (Bayer & Altman, 2006) or week 10 **(**Rakic & Yakovlev, 1968**;** Griffiths et al., 2009**)**. Crossing fibres can be detected in the area praecommissuralis by 10 weeks post-conceptional age (12 weeks post-last menstrual period) in the lamina reunions **(Fig. 1)** (Griffiths et al., 2009) and at 13th gestational week both the anterior commissure and optic chiasm are well developed (Ren et al., 2006**)**. The progression of these fibers is facilitated by commissural cellular glial tunnels that provide axonal guidance cues for them along their path (Katz et al., 1983; Lent et al., 2005). During these processes the anterior commissure is surrounded by a glial fibrillary acidic protein [+]/ vimentin [+] glial tunnel and a tunnel of GFAP- and VN-positive glial cells with TN between the cell bodies from 12th week post conceptional age until at least 17th week postovulation (Lent et al., 2005).

2.3 Embryology of the fornix

The fornix begins as two fiber bundles arising in the area of praecommissuralis and passes dorsally into the hippocampal primordium of the lamina reunions **(Fig. 1)**. The fornices pass towards the medial wall of their epsilateral hemispheric vesicle (Griffiths et al., 2009) as early as week 8 **(**Bayer & Altman, 2006) or week 9 (Rakic & Yakovlev, 1968) and diverge as they do so (Griffiths et al., 2009). By weeks 10–11(13 weeks post-last menstrual period) some

of the fornical fibers cross the midline and form the early hippocampal commissure (Griffiths et al., 2009). Glial fibrillary acidic protein -expressing glial cells were seen surrounding the fornix as of 16–17 weeks postovulation (Lent et al., 2005). The early fornix is a short and slightly curved bundle that contains more hippocampal-septal or septohippocampal fibers that connecting the hippocampus with the septal area (Vasung et al., 2010). Myelination of the fornix is first evident near term, with strong myelin basic protein immunoreactivity that presents in the angular bundle, alveus, and fimbria and relatively scant immunoreactivity in the nascent perforant pathway.Myelination in the hippocampus increases in childhood until adolescence, after which the pattern to stay in the same condition (Arnold & Trojanowski, 1996).

2.4 Embryology of the posterior commissure

The posterior commissure can be seen in stage 12 mm embryos as a large of fibers and until stage 25-37 mm (about 7-8 weeks), it is presented as a very well-developed commissure. During these stages the attachment of the fibers to the cells of the subcommissural organ is still continuing. Some of the fibers connect with the subcommissural organ, others with the thalamus and tegmental region of the embryos of 25-37 mm. Myelination of the posterior commissure begins about the 14th week of development and proceeds to develop in the various fibres in the following order: (1) at the 14th week a few myelinated fibres are found in the ventral part of the commissure, and also in the nucleus chiefly connected with this group of fibres, the nucleus of the posterior commissure. (2) at about the 24th week myelination is found in the fibres connecting with the subcommissural organ and the medial longitudinal fasciculus (Keene, 1938). Recent researches have revealed that development of both the posterior commissure and the underlying subcommissural organ are tightly related to one another and that these structures are under the control of regulatory genes such as *Pax2*, *Pax5*, *Pax6* and *Msx1* (Estivill-Torrús et al., 2001).

3. Anatomical organization of the brain commissures

3.1 Anatomy of the corpus callosum

The corpus callosum holds its name from its compactness; it is the largest of the commissures in advanced mammals (Raybaud, 2010). Although, this commissure has proven to be an important structure in the human brain, it is possible to live without this white matter structure (van der Knaap & van der Ham, 2011). The corpus callosum consists mainly of myelinated axons of various sizes (Griffiths et al., 2009), a certain amount of non-myelinated fibers (Tomasch, 1954), neuralgial cells and a certain number of blood vessels that connect the homologous regions of cerebral cortex of both hemispheres (Griffiths et al., 2009) from the anterior commissure anteriorly to the hippocampal commissure posteriorly (Raybaud, 2010). The callosal commissural neurons are located predominantly in intermediate cortical layers (Richards, 2004). The corpus callosum can be subdivided into several functionally and morphologically distinct sub regions which are arranged according to the topographical organization of cortical areas (Witelson, 1989); the small comma-shaped rostrum tucked under the genu (Griffiths et al., 2009) genu, truncus or midbody and splenium in sequential order from anterior to posterior (Witelson, 1989; Griffiths et al., 2009). The isthmus usually appears as a mild focal narrowing found where the fornix joins

the corpus callosum (Velut et al., 1998; Hofer & Frahm, 2006) which contains, connecting fibers of motor, somatosensory and primary auditory areas (Aralasmak et al., 2006; Raybaud, 2010; Aboitiz & Montiel, 2003; Aboitiz et al., 1992; Buklina, 2005; Fabri et al., 2005). The upper surface of the corpus callosum is lined with the indusium griseum (gray velum) (Jea, 2008). The rostrum of the corpus callosum extends anteriorly from the anterior commissure to the posterior inferior aspect of the genu and commonly assumed to be the last callosal segment to develop (Kier et al., 1997); its fibers are likely to connect the fronto-basal cortex (Velut et al., 1998; Hofer & Frahm, 2006). The genu (knee) is a thickened part of the corpus callosum, so named because of the sudden alteration in orientation; it is located between the rostralis and the callosal body. It forms the anterior boundary of the septum pellucidum and its fibers take part the formation of the forceps minor that connect the prefrontal cortex, the anterior cingulate area (Hofer & Frahm, 2006) and higher order sensory areas (Aralasmak et al., 2006; Raybaud, 2010; Aboitiz & Montiel, 2003; Buklina, 2005; Fabri et al., 2005). Its ventral part contains the fibers of the ventro-medial prefrontal cortex; its dorsal part includes the fibers of the dorso-lateral prefrontal cortex (Velut et al., 1998). The callosal body is the horizontal portion that extends from the genu to the point where the fornix abuts the undersurface of the corpus callosum. It borders the septum pellucidum superiorly. The fibers of the callosal body extends laterally between the cingular bundle superiorly and the occipito-frontal fascicle inferiorly and across the anterior radiations of the thalamus and forming the roofs of the lateral ventricular bodies. They connect the precentral cortex (premotor area, supplementary motor area), the adjacent portion of the insula, and the overlying cingulate gyrus mostly (Velut et al., 1998; Hofer & Frahm, 2006).The commissural fibers of the isthmus connect the pre- and postcentral gyri (motor and somatoisensory strips) (Velut et al., 1998; Hofer & Frahm, 2006) and the primary auditory area (Aboitiz, 2003; Aboitiz, 1992). The splenium is the thickest portion of the corpus callosum. It protrudes in the ambient cistern and overhangs the tectal plate, while the vein of Galen sweeps around it. Its morphology is extremely variable, from rounded to flat. It should be located above or just at the line drawn along the third ventricular floor (Widjaja et al., 2008). Fibers of the splenium form the forceps major and participate in the tapetum, or sagittal stratum, in the lateral wall of the posterior cornu of the lateral ventricle. It contains the commissural fibers for the posterior parietal cortex, the medial occipital cortex (Aboitiz, 2003; Velut et al., 1998; Hofer &Frahm, 2006), which connects visual areas in the occipital lobe (Aralasmak et al., 2006; Raybaud, 2010; Aboitiz & Montiel, 2005; Aboitiz et al., 1992; Buklina, 2005; Fabri et al., 2005) and the medial temporal cortex (Aboitiz, 2003; Velut et al., 1998; Hofer &Frahm, 2006). In regard to callosal size and width, most of the articles have shown that callosal size to be directly related to the number of interhemispheric connections (Bloom & Hynd, 2005) and vary between individuals and between sexes (Luders et al., 2010; Aboitiz et al., 1992; Junle et al., 2008; Clarke & Zaidel, 1994; Hasan et al., 2008). Additionally, Age related thinning of the corpus callosum is often reported (van der Knaap & van der Ham, 2011), however, these findings are still controversial.

3.2 Anatomy of the anterior commissure

The anterior commissure (Fig. 2. A, B) in humans is classically composed of two distinct tracts, the anterior (olfactive limb) and posterior limbs (temporal limb) (Patel et al., 2010; Mitchell et al., 2002; Peltier et al., 2011). The anterior limb forms an open "U" and those of
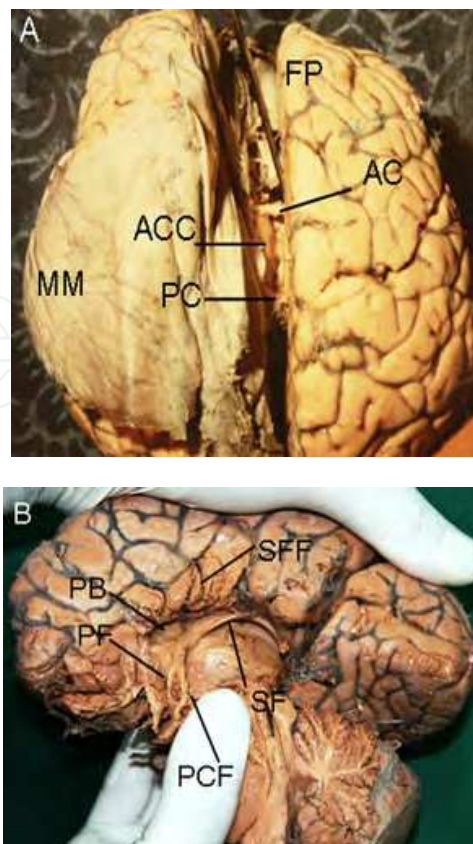
Fig. 2. Photographs of the brain in the superior view (A) and midsagittal plane (B) showing an absence of the corpus callosum (ACC), septum pellucidum, cingulum sulcus, interthalamic adhesion and hippocampal commissure.The anterior (AC) and posterior commissure (PC) are seen within the hemispheres. Other abbreviations in this figure: FP, frontal pole; MM, meningeal membrane; SF, separated fornix; PF, precommissural fornix; PCF, postcommissural fornix; PB, Probst bundle.

the posterior limb make an apposed flattened "M" shapes when viewed in the axial plane (Mitchell et al., 2002). The anterior limb is much smaller and varies considerably in size between subjects, it contains the small bundles of fibers that leave the main bulk of the commissure at the level of the anterior perforated substantia and connects the olfactory bulbs (Di Virgilio et al., 1999), their nuclei (Di Virgilio et al., 1999; Mitchell et al., 2002) and the inferior posterior orbital gyri (Patel et al., 2010; Di Virgilio et al., 1999). In addition, the small numbers of axons are detected in the anterior limb that crossing the midsagittal plane which is believed to convey fibers to territories others than the temporal cortex. Fibers of the posterior limb, which form the major and neocortical portion of the anterior commissure (Di Virgilio et al., 1999), travels within the basal parts of the putamen, the caudate nucleus and below the anterior border of the globus pallidus (Di Virgilio et al., 1999; Turner, 1979; Peltier et al., 2011) into the temporal cortex and projects to the amygdale (Turner, 1979; Di Virgilio et al., 1999; Jellison et al., 2004; Patel et al., 2010), (basolateral nucleus) (Martínez-Lorenzana et al., 2004), temporal pole (Jellison et al., 2004; Patel et al., 2010) parahippocampal, inferior temporal and fusiform gyri (Di Virgilio et al., 1999; Johnston et al., 2008; Demeter et al., 1985). The remaining fibers (a few fibers) of the posterior limb travel into the occipital lobe (Di Virgilio et al., 1999) and intermingle with other fasciculi in various directions to form a dense 3D network (Peltier et al., 2011). Also, additional afferent

fibers from the occipital cortex (Patel et al., 2010; Di Virgilio et al., 1999) precentral gyrus and central fissure have been detected through the posterior limb (Di Virgilio et al., 1999).

3.3 Anatomy of the fornix

The fornix **(Fig. 2. B)** provides bidirectional connectivity between the hippocampus and subcortical structures (Swanson & Cowan, 1977; Cassel et al., 1997). It contains the main efferent bundle (Carpenter, 1991) of large fibers connecting the hippocampal formation to the mamillary body (Atlas et al., 1986) and anterior thalamic nuclei. It is also has afferent cholinergic tracts from the septal nuclei and a smaller amount pathways from other basal forebrain to the hippocampus and entorhinal cortex, respectively (Gaffan et al., 2001; Mesulam et al., 1983; Ridley et al., 1996; Selden et al., 1998). The most of the fibres in the fornix begin from the subicular cortex and the pyramidal cells of the hippocampus. Those fibres converge into a discrete bundle as the fimbria at the medial surface of the alveus of the head of the hippocampus (Standring, 2005). The fimbria on the anterosuperior curvature of the hippocampus (Chance et al., 1999) lies to posterior end of thalamus, then arcs posterosuperiorly and medially to form the crus of the fornix (Atlas et al., 1986). Beneath the splenium (Chance et al., 1999), about 20% of the fibres (Lamantia & Rakic, 1990) cross the midline between the fornical crura at a point known as the commissure of the fornix (Chance et al., 1999; Lamantia & Rakic, 1990). Anteriorly, upon reaching the septum pellucidum on the midline and under the corpus callosum the crura meet to form the body of the fornix (Atlas et al., 1986; Lamantia & Rakic, 1990). Most text-books state that the two fornices merge but evidence from MR imaging indicates it is more accurate to say that they join but always maintain an obvious, separate identity (Griffiths et al., 2009).There, they course in the lower margin of the pellucidal leaves until they reach the superioranterior edge of the foramen of Monro (Lamantia & Rakic ,1990). As they descend, above the interventricular foramina the body of the fornix diverges into right and left fascicles which split into a precommissural fornix and a posterior commissural fornix (the columns of the fornix) near the anterior commissure (Williams et al., 1989). In each side the column or posterior commissural fornix (Carpenter, 1991; Meibach & Siegel, 1977) [hippocampo-mammillary tract (Lamantia & Rakic, 1990)] which contains the majority of the fornical fibres (Carpenter, 1991; Meibach & Siegel, 1977) and the fibres from the subicular area (Lamantia & Rakic, 1990) bend ventrally in front of the interventricular foramina and caudal to the anterior commissure, to join the anterior thalamus and hypothalamus (Atlas et al., 1986), predominantly to mamillary body. The precommissural (hippocampo-septal tract) contains the remaining portion of the fornical fibres which arising from the cornu ammonis (Lamantia & Rakic, 1990; Meibach & Siegel, 1977) and the subiculum (some of the fibres) and terminate exclusively in the (Meibach & Siegel, 1977) septum area (Chance, 1999) and septal nuclei (Meibach & Siegel, 1977; Lamantia & Rakic, 1990). The distribution of neurons contributing to the fornix in rhesus monkeys (Macaca mulatta) have been shown that the medial fornix originates from cells in the caudal half of the subiculum, the lamina principalis interna of the caudal half of the presubiculum, and from the perirhinal cortex (area 35). The intermediate portion of the fornix originates from cells in the rostral half of the subiculum and prosubiculum, the anterior presubiculum (only from the lamina principalis externa), the caudal presubiculum (primarily from lamina principalis interna), the rostral half of CA3, the EC (primarily 28I and 28M), and the perirhinal cortex (area 35). The lateral parts of the fornix arise from the rostral EC (28L only) and the most rostral portion of CA3.

Subcortically, the medial septum, nucleus of the diagonal band, supramammillary nucleus, lateral hypothalamus, dorsal raphe nucleus, and the thalamic nucleus reuniens all send projections through the fornix, which presumably terminate in the hippocampus and adjacent parahippocampal region (Saunders & Aggleton, 2007). In conclusion, it is apparent that schizophrenia and to some extent gender have an influence on the neuroanatomy of the fornix (Church et al., 1999).

3.4 Anatomy of the posterior commissure

The posterior commissure **(Fig. 2. A)** extends from the region of the pineal recess to the tectal commissure. Its caudal end corresponds with the position of the orifice of the mesocoelic recess. It contains the coarse and fine fibres. The coarse fibres lie close to the ventricular roof and also skirt the mesocoelic recess, whereas the fine ones occupy a position nearer to the exterior, and are continued into the tectal commissure. thus the cephalic part of the commissure consists of ventral coarse fibres and fine dorsal ones, and the caudal part has a more complicated arrangement of fibres, due to the forward folding of the roof of the mid-brain in that region. The following connexions for the posterior commissure are reported: a) the coarse fibres directly connect with the nucleus of the posterior commissure and also indirectly through the nucleus of the posterior commissure or interstitial nucleus with the ipsilateral medial longitudinal bundle, b) Other fibres, chiefly coarse ones, connect with the regions of the tegmentum and the capsule of the red nucleus, c) fine fibres situated in the dorsal part of the commissure connect with the thalamus, d) the commissure consisting of horizontal fibres which may be traced in a lateral direction, it is thought that this connexion may be striatal,or possibly cortical, e) a small connexion with the habenular ganglia, and the habenulo-peduncular tracts, h) a fine connection with the pineal gland is also established (Keene, 1938). Also, studies in rat have demonstrated that the activity of the subcommissural organ depends on serotoninergic fibers originated in the raphe nuclei, some of which reach the subcommissural organ through the posterior commissure (Mikkelsen et al., 1997). In the chick brain, the tract of the posterior commissure emerges in the caudal pretectum as the first transversal tract. It is formed by dorsally projecting axons from neurones located in the ventral pretectum, and by ventrally projecting axons from neurones located in the dorsal pretectum (Ware & Schubert, 2011).

4. The vessels of the brain commissures

4.1 The arteries of the brain commissures

4.1.1 The arteries of the corpus callosum

The blood supply to the corpus callosum originates from both of the arterial systems of the brain; the carotid system and the vertebral-basilar system.

4.1.1.1 The carotid system

The carotid system contributes mainly to this supply via the pericallosal artery (Kakou et al., 1998; Wolfram-Gabel et al., 1989; Türe et al., 1996) which is the main artery of the corpus callosum (Wolfram-Gabel et al., 1989; Türe et al., 1996). It curves around the genu and continues posteriorly along the dorsal surface of the corpus callosum (Yasargil, 1984). Its posterior extension followed a cork-screw-like tortuousity, anastomosed with the posterior

pericallosal artery in the splenial region, and formed the dense portion of the pericallosal pial plexus within the callosal sulcus. Usually, some of the branches arising from this network circle around the splenium and joint with branches of the medial posterior choroidal arteries in the tela choroidea of the third ventricle. In addition to pericallosal artery, the anterior communicating artery accessorily contributes to it by an inconstant artery called median artery of the corpus callosum (Wolfram-Gabel et al., 1989; Türe et al., 1996). The pericallosal artery gives rise to four types of branches that supply the corpus callosum, these are the callosal artery; cingulocallosal artery; long callosal artery; and recurrent cingulocallosal artery. **The callosal arteries** are thin branches which directly supply the indusium griseum and the superficial surface of the corpus callosum in the midline (Kahilogullari et al., 2008; Türe et al., 1996**). The cingulocallosal arteries** bring the chief supply to the corpus callosum. These arise from the inferolateral aspect of the pericallosal artery and run laterally into the callosal sulcus, where they are divided into three arterial subgroups (Türe et al., 1996) which supply the corpus callosum, the cingulate gyrus and the radiation of the corpus callosum.The cingulocallosal arteries anastomosing with each other and with branches arising from the subcallosal, median callosal and long callosal arteries to form the pericallosal pial plexus. **The long callosal artery** is found almost in half of the hemispheres, it is an another branch arising from the pericallosal artery, courses parallel with it in the callosal sulcus and has multiple branches that contributed to the pericallosal pial plexus (Kahilogullari et al., 2008; Türe et al., 1996) .The artery ends in the body of the corpus callosum or in the medial longitudinal striae at the splenium and anastomosis with the posterior pericallosal artery of the same hemisphere or is crossed the midline and anastomosed with the posterior pericallosal artery of the opposite hemisphere, both within the callosal sulcus in the splenial region. **The recurrent cingulocallosal artery** is a thin branch, arises from major cortical branches of the pericallosal artery: It courses on the medial surface of the cingulate gyrus toward the callosal sulcus, present in 45% of the subjects (Türe et al., 1996) and contributed to the pericallosal pial plexus (Kahilogullari et al., 2008; Türe et al., 1996). In addition to the pericallosal artery, the perforating branches of **the anterior communicating artery** participate in providing blood supply to the corpus callosum.The hypothalamic artery (which do not supply the corpus callosum); subcallosal artery; and median callosal artery spring from these branches. In 80% of the specimens, either the subcallosal artery or the median callosal artery are present and contributed to blood supply of the corpus callosum, especially to the anterior portion. **The subcallosal** artery is a major contributor to the blood supply of the medial portions of the rostrum and genu of the corpus callosum.**The median callosal artery** is present in 30% of the specimens an anatomical variations. This artery followed the same course as that of the subcallosal artery and supplies the same structures, except that its distal extension reached the body and frequently even the splenium of the corpus callosum (Kahilogullari et al., 2008; Türe et al., 1996; Kakou et al., 1998).

4.1.1.2 The vertebral-basilar system

The vertebral-basilar system contributes to the blood supply of the corpus callosum by the terminal and choroidal branches of the posterior cerebral artery (Wolfram-Gabel et al., 1989; Türe et al., 1996). The posterior cerebral artery is divided into four segments: the end segment of which comprises the posterior extension of the posterior cerebral artery that runs along or inside both the parieto-occipital sulcus and the distal part of the calcarine fissure and gives the parieto-occipital and calcarine arteries (Párraga et al., 2010). The posterior

cerebral artery contributes in providing blood supply to the corpus callosum by the posterior pericallosal artery (also known as the splenial artery), in particular the splenial portion, in all hemispheres. It arises from the main trunk of the parieto-occipital artery or its precuneal branch (52%) of the third segment of the posterior cerebri artery (32%), the calcarine artery (7%), the temporo-occipital artery (7%), or the second segment of the posterior cerebri artery (2%). In addition to the posterior pericallosal artery, a very fine artery that contributed to the blood supply of the splenium is observed in 25% of the hemispheres. It origins from the precuneal branch of the parieto-occipital artery, the hippocampal artery, the medial posterior choroidal artery, or the lateral posterior choroidal artery. It has been named this artery the "accessory posterior pericallosal artery (Türe et al., 1996).

4.1.2 The arteries of the anterior commissure and the fornix

The medial portion of the anterior commissure and the column of the fornix, are supplied by the small perforating branches of the hypothalamic arteries(Türe et al., 1996; Dunker & Harris , 1976) and the remaining anterior cerebral artery proximal to the anterior communicating artery (Dunker & Harris , 1976). The hypothalamic arteries arise from the posteroinferior aspect of the anterior communicating artery (Türe et al., 1996). Also, the inferior branch of the posterior pericallosal (Türe et al., 1996) and lateral posterior choroidal arteries supply the crus of the fornix.

4.2 The veins of the brain commissures

The venous drainage of the corpus callosum is essentially via callosal and callosocingulate veins empty into the deep venous system of the brain (Kakou et al., 1998; Wolfram-Gabel & Maillot , 1992). Most of these veins pass caudally and anastomoses together at the central level of the corpus callosum and form the subependymal veins and are collected by the septal and the medial atrial veins. All these veins are tributaries of the internal cerebral veins (Wolfram-Gabel & Maillot, 1992**).**

5. Functional correlation of the brain commissures

It has long been accepted that the commissural fibres are important for transfer of complex cognitive information (Zaidel, 1994; van der Knaap & van der Ham, 2011). In this issue the corpus callosum has an important role than other commissures. The corpus callosum involves in lower-level processes (Schulte & Müller-Oehring, 2010), transferring sensory information (Banich, 1998), interhemispheric visuomotor integration (Banich, 1998; Schulte & Müller-Oehring, 2010; Mordkoff & Yantis, 1993)**,** hemispheric specialty (Doron & Gazzaniga, 2008) and contribution in development of higher-order cognitive functions (Gazzaniga, 2000; Doron & Gazzaniga, 2008). So, the corpus callosum is needed to maintain an integrated sense of self with regards to body awareness and planning of actions. (Uddin , 2011), as in regard to visuomotor integration, the integration of perception and action by the corpus callosum promoting a unified experience of the way that we perceive the visual world and prepare our actions (Bloom & Hynd, 2005). It appears that the corpus callosum employs a differentiated role with callosal areas transmitting different types of information depending on the cortical destination of connecting fibers (Bloom & Hynd, 2005). The anterior corpus callosum is necessary for awareness of initiation of goal-directed movements and subjective feelings of agency (Uddin, 2011) and associate with inhibitory functions in situations of semantic competition (Stroop) and local-global interference (Bloom

& Hynd, 2005); in addition to intact fronto-parietal cortical functioning (Uddin, 2011) . The posterior corpus callosum integrity seems proved for maintaining a sense of limb ownership, as this region interconnects parietal areas involved in self-body representation (Uddin, 2011). Also, it connects temporo-parietal and occipital cortical regions in related with facilitation functions from redundant targets and local-global features. It is reported that an intact (posterior) corpus callosum and interaction between ipsilateral and contralateral hemispheres are required for coordination of the hand movements (Eliassen et al., 1999). Additionally, it is suggested that the posterior callosal area associated with the superior colliculi connect visual extrastriate areas as the key structures for interhemispheric neural coactivation explaining visuomotor integration between hemispheres (Iacoboni et al., 2000). A study has shown that lesions of the posterior or mid-body corpus callosum or complete commissurotomy conflict intermanual coordination; injuries of the posterior corpus callosum and parietal cortical areas cause the alien hand sign; and lesions of the frontal lobe or anterior corpus callosum results the anarchic hand (Aboitiz et al., 2003).Studies in acallosal and split brain patients have revealed that the absence or loss of the corpus callosum integrity contributes to impairment in sensory and cognitive integration (Fabri et al., 2001; Yamauchi et al., 1997) and large individual differences in interhemispheric transfer among split-brain patients (Zaidel et al., 2003). In split-brain patients, however, several investigators have noted that transfer of some types of visual information is usually spared (Eviatar & Zaidel, 1994; Uddin et al., 2008).The condition that cortical commissures are no longer available some information can be transferred between the hemispheres through subcortical pathways (Funnell et al., 2000) by the subcortical coordination of cortical networks (Uddin et al., 2008). In regard to involvement of the corpus callosum in lower-level visuomotor functions, split-brain research indicates that the corpus callosum acts in an inhibitory fashion within a subcortico-cortical network (Corballis et al., 2002; Roser & Corballis, 2003), while recent research on callosal degradation without disconnection have shown cooperative role for the corpus callosum (Schulte & Müller-Oehring, 2010) in conscious perception (Marzi et al., 1996; Müller-Oehring et al., 2009). In addition to mentioned functions, recently the enhanced redundancy gain (co-activation model) (Bucur et al., 2005; Schulte et al., 2006; Turatto et al., 2004) and mediate interhemispheric processing advantages (Corballis et al., 2003; Iacoboni et al., 2000; Roser & Corballis, 2003) a possible role for the corpus callosum are reported. In regard to this question, how the corpus callosum mediates this transfer? There are, two contrasting theories of interhemispheric interaction in the literature, excitatory and inhibitory messages, although there is more evidence to support the notion that the corpus callosum plays an excitatory function in interhemispheric communication rather than an inhibitory function, there is some evidence that inhibition occurs. The nature of functions may occur at different times depending on the task or may even occur simultaneously to achieve an interhemispheric balance between component brain functions (Bloom & Hynd, 2005). How the corpus callosum regulates this transfer of information between cortical areas seems uncertain (van der Knaap & van der Ham, 2011).

6. Interhemispheric transfer time

Consumed time of transfer time of information between hemispheres is shorter and more equal for women than men (Moes et al., 2007) and is faster from right-to-left than from left-to right (Barnett & Corballis, 2005; Iwabuchi & Kirk, 2009). The causes of these differences may be; faster axonal conduction in the right hemisphere relative to the left (Barnett & Corballis, 2005) or the degree of hemispheric specialization (Nowicka et al., 1996; Rugg &

Beaumont, 1978) more gray matter relative to white matter in the left hemisphere than in the right (Gur et al., 1980); other anatomical differences between both hemispheres. It appears that the ratio of gray and white matter may be underlying functional asymmetry (Schulte & Müller-Oehring, 2010). A correlation between callosal connectivity and prolonged interhemispheric transfer time have been reported in split-brain patients and in acallosal patients (Iacoboni et al., 2000; Mooshagian et al., 2009; Paul et al., 2007; Reuter-Lorenz et al., 1995; Roser & Corballis, 2002).

7. Brain commissural anomalies

7.1 Malformations of the corpus callosum

It is observed in a variety of conditions that disrupt early cerebral development, including chromosomal and metabolic disorders, as well as intrauterine exposure to teratogens and infection (Paul et al., 2007). Callosal agenesis can be detected prenatally by routine sonography, for which the important signs include absence of the cavum septum pellucidum, colpocephaly, high-riding third ventricle, and widening of the interhemispheric fissure (Tang et al., 2009). On the basis of the known embryology of the corpus callosum, two primary or "true" types and two secondary types of callosal abnormalities have been documented. The two types of true agenesis of the corpus callosum include (1) defects in which axons form but are unable to cross the midline because of absence of the massa commissuralis and leave large aberrant longitudinal fiber bundles known as Probst bundles **(Fig. 3. A, B)**, along the medial hemispheric walls; and (2) defects which the commissural axons or their parent cell bodies fail to form in the cerebral cortex (Sidman & Rakic, 1982). The former, probably the most common type of agenesis of the corpus callosum, occurs in BALB mice and all agenesis of the corpus callosum syndromes, in which Probst bundles are
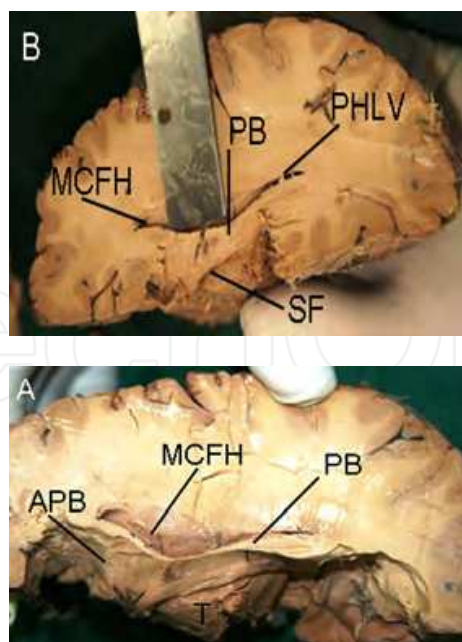

Fig. 3. Photographs of transverse sections of the cerebral hemisphere (A, B) showing the Probst bundle (PB) and medially concave frontal horn (MCFH). Other abbreviation in this figure (B): APB, anterior part of the Probst bundle; T, thalamus; SF, separated fornix; PHLV, posterior horn of the lateral ventricle.

seen. The latter occurs in Walker- Warburg syndrome and other types of lissencephaly, in which Probst bundles are generally not seen.The two types of secondary of callosal abnormalities include (1) absence of the corpus callosum associated with major malformations of the embryonic forebrain prior to formation of the anlage of the corpus callosum; and (2) degeneration or atrophy of the corpus callosum, which results in striking thinning that may again be mistaken for true agenesis of the corpus callosum (Dobyns, 1996). Its incidence in the general population is 3-7 per 1000 birth; in children with developmental disabilities is 2-3 per 100 (Grogono, 1968; Jeret et al., 1985; Glass et al., 2008), among patients undergoing cranial magnetic resonance imaging at a tertiary care referral institution was determined to be 0.25% (Hetts et al .,2006). Also, a population-based survey indicates that the combined prevalence of agenesis and hypoplasia of the corpus callosum before age 1 year is only 1.8 per 10,000 live births (Glass et al., 2008). In addition to mentioned incidences, a epidemiologic study in Hungary has been shown that the overall birth prevalence of total or partial agenesis and hypoplasia involved 2.05 per 10,000 live births, including 2.73 per 10,000 among boys, and 1.33 per 10,000 among girls. The birth prevalence of total and partial agenesis of the corpus callosum involved 1.02 per 10,000 live births, with 1.36 per 10,000 among boys and 0.66 per 10,000 among girls. The birth prevalence of hypoplasia of the corpus callosum involved 1.02 per 10,000 live births, with 1.36 per 10,000 among boys, and 0.66 per 10,000 among girls. The male/female sex ratio was 2.2 for both total or partial agenesis and hypoplasia of the corpus callosum (Szabó , 2011).The morphological anomalies of the corpus callosum may be **agenesis (complete and partial), dysgenesis (Fig. 4), hypoplasia** and **hyperplasia** (Raybaud, 2010; Yousefi & Kokhei, 2009; Hetts et al .,2006; Hanna , 2011).

Fig. 4. Corpus callosum is markedly dysgenic; the genu (long Stright arrow) and body (short straight arrows) are present, but splenium and rostrum are absent. Anterior cornmissure (curved arrow) is present and of normal size (Barkovich & Norman, 1988).

7.1.1 Complete agenesis of the corpus callosum (Figs. 2. A, B & 5. A)

The agenesis (complete or partial) is one of the most commonly observed features in the malformations of the brain (Chiappedi & Bejor, 2010), a part of many syndromes (Chiappedi & Bejor, 2010; Penny, 2006) and/or somatic anomalies (Barkovich, 2005; Barkovich & Norman, 1988; Marszal et al., 2000; Hetts, 2006). Primary complete agenesis usually occurs earlier in embryologic development, while partial agenesis occurs in later gestation (Penny, 2006). Complete agenesis of the corpus callosum, in which patients do not develop a callosal

structure. It is rarely limited to the callosal structure (Raybaud, 2010) and usually sporadic (Chouchane et al., 1999). This form of anomaly is often associated with defects or absence of the other forebrain commissures (Raybaud, 2010). Most of the patients with complete agenesis and without telencephalic dysgenesis or syndromic features typically have the Probst's bundles (Szriha, 2005). During embryogenesis, the fibres are thought to arrive at the midplane, where they are hindered in their further migration across the midline and then change their direction of growth (Rosenthal-Wisskirchen, 1967) into an anteroposterior direction which leads to the formation of bundles in each hemisphere (Rosenthal-Wisskirchen, 1967; Lee et al., 2004; Hetts et al., 2006; Meyer & Röricht, 1998).The formation of bundles upon the lateral ventricular lumen, giving it a crescentic and a bull's head appearance to the section of the lateral and third ventricles on the coronal view. This bundle is called the bundle of Probst in the literature (Raybaud ,2010) .The Probst bundle may be intermingled with upper border of the separated fornix (Hetts et al., 2006; Meyer & Röricht, 1998; Yousefi & Kokhei, 2009). In this condition, frontally, it has comma (Meyer & Röricht, 1998), a U-turn shape (Ozaki et al., 1987) and the lower area of the anterior portion (radiated fibres) of the Probst bundle is attached to the ventral branches of the precommissure fornix. Posteriorly it forms a thin layer on the upper medial wall of the lateral ventricles (Meyer and Röricht, 1998) and accumulates as an anomalous fascicle below the cingulum (Ozaki et al., 1987) or attaches to the crus of fornix at the beginning of the fimbria (Yousefi & Kokhei, 2009). The etiology of Agenesis of the corpus callosum is heterogeneous, including cytogenetic abnormalities, metabolic disorders and genetic syndromes (Dobyns, 1996). At the molecular level, the process of development of the corpus callosum is complex. These processes rely on intricate cell-to-cell signaling mechanisms. Disruption or desynchronization of these mechanisms could lead to partial or complete callosal agenesis (Prasad et al., 2007).

7.1.2 Partial agenesis of the corpus callosum

Partial agenesis of the corpus callosum **(Fig. 4)** results from an arrest of growth between 12 and 18 weeks of gestation and usually involves the dorsal part or splenium (Kier & Truwit, 1996). It is suggested that a deviation in the normal course of the pericallosal arteries may be the sign of corpus callosal partial agenesis. In such cases the arteries closely follow the contour of the corpus callosum at its anterior part (the genu and the body), but take an upward direction at the level of the missing splenium (Volpe et al., 2006). Additionally, an insult to the developing corpus callosum may inhibit the complete formation of this large commissural bundle and lead to partial agenesis (hypogenesis) of the corpus callosum, when only the early formed portions appear (Szriha, 2005).

7.1.3 Callosal hypoplasia

Callosal hypoplasia is a developmental disorder that may be induced by teratogens (radiation, alcohol) or compression (e.g. intracranial masses, obstructive hydrocephalus (Davila-Gutuerrez, 2002; Paupe et al., 2002) rather than a primary malformative abnormality.Thus, callosal hypoplasia more likely depends upon an external factor affecting the number and size of callosal axons. This is apparently confirmed by a experience since callosal hypoplasia was often associated with additional brain anomalies (Ghi et al., 2010). Hypoplasia and partial agenesis of the corpus callosum may occur in isolation form, in these

conditions, neurological outcome is reported by some to be similar to that in cases with absent corpus callosum1(Moutard et al. ,2003; Mordefroid et al., 2004; Ghi et al., 2010). Callosal hypoplasia include a significant size reduction of the anterior genu, posterior genu (Walterfang, 2008, Walterfang, 2009a; Vidal et al., 2006; Just et al., 2007; Walterfang et al., 2009b; 2009c), isthmus (Walterfang, 2008; Walterfang, 2009a; Vidal et al., 2006; Just et al., 2007; Cao et al., 2010)and the posterior midbody (Cao et al., 2010), a smaller splenium width (Bersani, 2010; Vidal et al., 2006; Just et al., 2007; Hutchinson et al ., 2008), a cyst in the splenium (Bamiou et al., 2007) and a smaller anterior midbody. The anterior midbody is known to increase in size until the late twenties (Bersani, 2010). Other abnormal shapes of the corpus callosum are reported such as global shape due different bending degrees of the callosal body (He et al ., 2010) ; a slit-like left paracallosal lesion extending from the genu towards the splenium (Faber et al., 2010) with an additionally smaller anterior corpus callosum for boys (Hutchinson et al ., 2008). The studies have shown a correlation between illness duration and callosal shape in patients with bipolar disorder. Therefore, the corpus callosum degeneration and axonal loss is repeatedly described in some of the psychiatric disorders (Evangelou et al., 2000; Manson et al., 2006; Warlop et al., 2008; Gadea et al., 2009). Callosal thinning by defective myelination or decreased fiber density, can manifest itself in pathology specific symptoms. Also, a lot of variations are seen in patients in related to age, sex and type of symptoms (van der Knaap, 2011).

7.1.4 Callosal hypertrophy

Hypertrophy of the corpus callosum is a classical marker of neurofibromatosis type 1. It has also been recently identified as a characteristic of a macrocephaly syndrome with polymicrogyria and developmental delay (Pierson et al., 2008). Finally, investigations have shown that the corpus callosum is particularly vulnerable to closed head trauma (Peru et al., 2003). There are evidences that the chromosomes of 8, 11, 13-15 and 18 involvement in abnormal corpus callosum morphogenesis and it can occur as an X-linked (Jeret et al., 1987; Davila-Gutierrez, 2002) or autosomal-recessive condition, or can present as an incidental finding during imaging in apparently normal patients (Davila-Gutierrez, 2002).

7.2 Malformations of the anterior commissure

In the classic commissural agenesis, in about 50% of the cases, the anterior commissure is either absent or too thin to be recognized (Raybaud & Girard, 1998), or apparent but hypoplastic (Raybaud & Girard, 1998; Griffiths, 2009), probably due to the absence of its neocortical component. It is classically mentioned that in some of the cases it may be enlarged, as if compensating for the missing corpus callosum (Raybaud & Girard, 1998; Probst, 1973; Barr Melodie & Corballis Michael, 2002) whereas in other studies, it is reported that this commissure was small (Bamiou et al., 2007; Barkovich & Norman, 1988; Atlas, 1986) in the patients who had complete agenesis of the corpus callosum associated with cranial abnormalities and some of syndromes and small but had a normal configuration in the patient with isolated callosal agenesis (Barkovich & Norman, 1988; Atlas, 1986).The anterior commissure is dislocated in more than a third of the cases (38%), low on the lamina terminalis, halfway between the foramen of Monro and the optic chiasm (Raybaud, 2010). In addition to above anomalies, the unilateral anterior commissure run posterior to the columna fornicis in the brain of a 20 year-old man was reported (Hori, 1997). Association of

the callosal agenesis with absent or hypoplastic of the anterior commissure is most likely the result of either an anomaly of the primitive lamina tenminalis, either of these situations would inhibit the formation of the beds of tissue into which both the commissural and callosal fibers are induced to grow. The presence of normal anterior commissures in those patients with a partially formed corpus callosum suggests that the insult to the brain that disrupts callosal formation occurs after the bed for ingrowth of the anterior commissure is formed (Barkovich & Norman, 1988). Investigations have shown that in adult humans, *Pax6* mutations are associated with cerebral malformations and structural abnormalities of the interhemispheric pathway, with an absent or hypoplastic anterior commissure (Sisodiya et al., 2001). Also, the anterior commissure is reduced in *Pax6cKO* mutants (Abouzeid et al., 2009; Sisodiya et al., 2001). Of interest, there is circumstantial evidence that a hypertrophied anterior commissuer may reflect compensation for the lack of the corpus callosum in terms of interhemispheric transfer function (Fischer et al., 1992).

7.3 Malformations of the fornix

A focus on the fornix abnormalities and their association with hippocampal anomalis may figure importantly in our understanding of the pathophysiology of schizophrenia (Kuroki et al., 2006).

7.3.1 Anomalies of the fornix

The fornical defects associates with other commissural agenesis such as missing of the hippocampal commissure. The accumulation of the fornical fibers in the lower edge of the medial telencephalic medullary velum, the separated fornix **(Fig. 5. A)** (Meyer & Rorich, 1998; Yousefi & Kokhei, 2009), variation in the pattern of distribution (Griffiths, 2009; Yousefi & Kokhei, 2009) of the precommissural fornix to more than three branches **(Fig. 5. A)** on the medial surface of the frontal lobe, thickening one of **(Fig. 5. B)** precommissural fornix branches and continued to curve inferior posteriorly parallel with the posterior commissural fornix, so that is visualized without dissection (Yousefi & Kokhei, 2009), entrapped some fibres of the genu of the corpus callosum with the fornical fibers bundle (Hori, 1997), enter the fornix to the basal forebrain without the normal division, a bulky connection between the anterior parts of the fornices producing a very prominent hippocampal commissure (Griffiths, 2009; Barkovich, 1990) are repoted in literature as anomalies of the fornix. Also, a recent postmortem and a in-vivo studies confirming decreased axonal density (Ozdogmus et al., 2009, Concha et al., 2010) and a near complete absence of unmyelinated axons of the fimbria-fornix bilaterally in the temporal lobe epilepsy and unilateral mesial temporal sclerosis patients (Ozdogmus et al., 2009; Concha et al., 2010) due to the intriguing possibility that a specific subset of projection fibers may be lost in temporal lobe epilepsy (Concha et al ., 2010). Reduced fractional anisotropy and cross-sectional area simultaneous increase mean diffusivity in the fornix in the schizophrenia patients which indicate that fornix abnormalities may be due to either immaturity or degeneration of the fiber tract. These abnormalities may reflect decreased axonal density, axonal damage, or decreased degree of myelination. Atrophy of the fornix is the other condition that is detected in 86% of the temporal lobe epilepsy patients with unilateral hippocampal atrophy and in almost all patients with bilateral symmetrical hippocampal atrophy. This finding suggests that hippocampal atrophy may cause secondary fornix
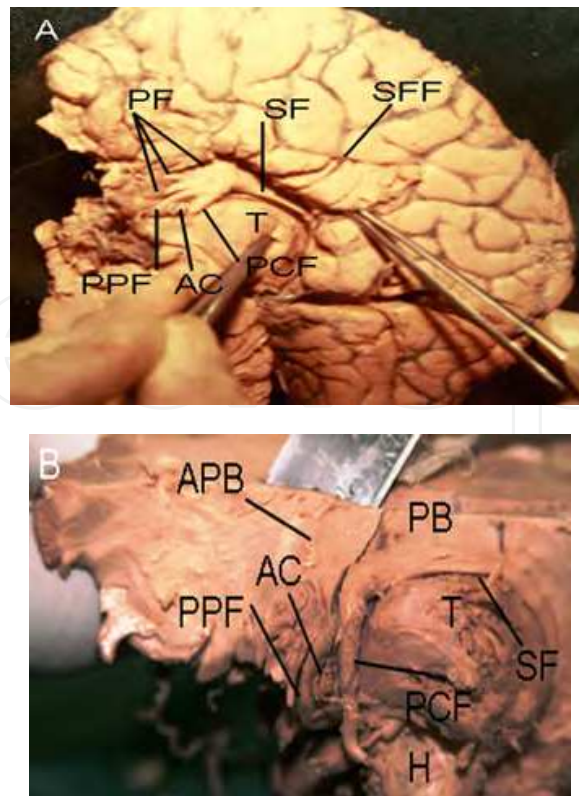
Fig. 5. The photograph of the brain in midsagittal plane (A), exposed of medial aspect of the frontal lobe (B) showing the separated fornix (SF) and associated branches. The precommissural fornix (PF) is abnormal. Other abbreviations in this figure: PCF, postcommissural fornix; AC, anterior commissure; PB, Probst bundle; SFF, sulci as fan-like fashion. APB, anterior part of the Probst bundle; T, thalamus; H, hypothalamus.

atrophy (Concha et al., 2010). In addition to the schizophrenic patients, the fornical anomalies are common in patients with myelomeningocele and Chiari II malformation. These fornical anomalies include; intact but thin, thin and right greater than left, atrophic and left greater than right, left intact and right crus deficient, thin body and crura,, thin crura, defects in crura, bilaterally deficient crus and body and frank defects in the fornices associated with atresia or hypoplasia of crura and body of fornices. In these patients such defects are associated with memory and learning deficits (Vachha et al., 2006). Beyond mechanical stretching of periventricular axons, chronic hydrocephalus has been shown to be associated with microvascular changes in the cerebral white matter, which include capillary compression and calcium-mediated proteolysis that may account for the defects within the limbic fibres (Del Bigio, 2001).

7.3.2 Asymmetry of the fornix

In addition to patients (Baldwin et al., 1994), significant differences in the fornical volume were seen between the right and left sides of the fornix in healthy individual (Zahajszkyet al., 2001).This asymmetry is present in the position of the two columns of the fornix in relation to the septum pellucidum. This difference was seen in most of the subjects caudal located of the left fornical column to the right (Supprian &, Hofmann, 1997). In patients asymmetric volume loss in the fornix is detected on the same side as the abnormal

hippocampus and hippocampal sclerosis, the correlation may be related to the anatomy of this white matter tract. Because some of axons of the fonnix originate in the pyramidal cells of the hippocampus, it is suggested that hippocampal neuronal loss may result in wallenian degeneration and subsequent atrophy of the ipsilatenal fornix (Baldwin et al., 1994). The fornix asymmetry is more likely to be of developmental origin as opposed to secondary alterations (Supprian &, Hofmann, 1997) and the degree of asymmetry between the fomices varied from 41% to 82% (mean, 68%).It is apparent that men have a lower density of fibers in the fornix than women, the density of fibers on the left in men is significantly greater for patients with schizophrenia than those whom total fibre number is not significantly affected by gender or diagnosis (Chance et al., 1999). In regard to the precise course of the fornix in commissural agenesis associated with meningeal dysplasia cases, it is highly variable and posteriorly appears to be influenced by the anatomy of the interhemispheric cysts to a major degree. The fornix maintains a high-riding path as it courses cephalad and does not appear to give a postcommissural branch; instead, the fornix passes more anteriorly than usual before passing posteriorly to enter the basal forebrain (Griffiths, 2009). In children with complete commissural agenesis the path of fornix appears to be remarkably constant, although the fornix travels more laterally than usual away from its partner. Some of the cases may show the Shift of the fornix into a rostroventral direction (Boretius et al., 2009).

7.4 Malformations of the posterior commissure

The hypoplasia (Abouzeid et al., 2009) and absence of the posterior commissure may be associated with other forebrain commissures anomalies (Meyer & Rorich, 1998; Abouzeid et al., 2009) in the patients with *Pax6* (p.R159fs47) mutations (Abouzeid et al., 2009). Experimental studies in the null mutant mice with lacking subcommissural organs or with subcommissural organs alterations have shown that a normal posterior commissure fails to form (Louvi & Wassef, 2000; Estivill-Torrus et al., 2001; Fernandez-Llebrez et al., 2004; Ramos et al., 2004) due to lack of the homeobox gene Msx1 (Fernandez-Llebrez et al., 2004; Ramos et al., 2004). Additionally, in mutant mice lacking the transcription factor PAX6, the posterior commissure fails to develop (Estivill-Torrus et al., 2001).Also, in WEXPZ.En1 transgenic mice in which engrailed-1 is expressed ectopically in the dorsal midline of the diencephalon (Danielian & McMahon, 1996) the posterior commissure development is delayed and frequent errors in axonal pathfinding happen (Louvi & Wassef, 2000). In addition to mentioned anomalies, relocation of the posterior commissure on the subcommissural organs is reported in the one-eyed pinhead mutants of the zebrafish Dario rerio due to slightly displaced of the subcommissural organs from its normal midline position (Hoyo-Becerra et al., 2010).

8. Brain malformations associated with commissural disorders

It has been proposed that the corpus callosum is useful as an indicator of both congenital and degenerative brain disorders in children, since the corpus callosum is formed contemporaneously with many other major telencephalic structures (Barkovich & Norman, 1988; Hetts et al., 2006). Callosal dysgenesis is frequently associated with other central nervous system malformations and /or somatic anomalies (Barkovich & Norman, 1988; Marszal et al., 2000; Hetts et al., 2006) and its defects are rarely isolated (Barkovich & Norman, 1988; Hetts et al., 2006). Since formation of the corpus callosum is complex and this characteristic may

explain why most cases of callosal agenesis are not isolated (Tang et al., 2009). The type, number, and severity of related anomalies, however, are deferent. Brain anomalies associated with commissural disorders can be arranged based on the morphology of the cerebral commissures and associated malformations of the midline, of cortical development, of white matter, and of the diencephalon and rhombencephalon (Hetts et al., 2006).

8.1 Midline anomalies

Midline malformations association with agenesis or dysgenesis of the corpus callosum include **interhemispheric cysts (Figs. 6, 9), lipomas** (Hetts et al., 2006; Byrd, 1990; Johnston, 1934; Probst, 1973, Barkovich et al., 2001; Raybaud, 2010; Truwit, 1990) and **craniocerebral midline defects** (Raybaud, 2010) which can be confirmed by imaging studies (Davila-Gutierrez, 2002). Agenesis of the commissures with interhemispheric cysts is felt to have different causes, possibly related to a meningeal rather than neural disorder (Raybaud, 2010). There are two broad classes of interhemispheric cysts (**Fig. 6**), communicating and non-communicating (Johnston, 1934; Probst, 1973; Barkovich et al., 2001). The communicating cysts are expansions of the ventricular tela choroidea and the non-communicating cyst is multiloculated meningeal cystic dysplasia (Raybaud, 2010; Davila-Gutierrez, 2002). Additionally, another classification of the callosal agenesis with cysts has been advised: type 1, in which there is one single cystic cavity that communicates with the ventricles and subdivides in three subgroups on the bases of being a) with macrocephaly and hydrocephalus, b) with macrocephaly and hydrocephalus associated with a developmental ventricular obstruction (thalamic fusion, hamartoma), and c) with microcephaly (Barkovich et al., 2001). Type 2 refers to the cases where the interhemispheric cysts are multiloculated (Davila-Gutierrez, 2002; Barkovich et al., 2001) and independent from the ventricles; it is subdivided into three subgroups on the bases of being a) hydrocephalus and an essentially normal brain, b) affects girls and is made of multiple cysts different from cerebro spinal fluid with frontoparietal polymicrogyria and periventricular nodular heterotopias and one or two dilated ventricles (Barkovich et al., 2001) and c) with multiloculated cysts, large subcortical heterotopia, and dysmorphic head and brain. However, it needs to be confirmed (Raybaud, 2010). In the form of a single ventricular diverticulation cyst, the commissural agenesis is usually not associated with significant hemispheric dysplasia or malformations of cortical development. The main feature is the markedly expanded tela choroidea, the septum pellucidum, fornices, and bundles of Probst are missing (Raybaud, 2010). Such conditions have been previously described as "septo-optic dysplasia": with total absence of the corpus callosum (Sener, 1993) or agenesis of the corpus callosum with dehiscent fornices (De León et al., 1995). In commissural agenesis with multilocular cysts, most of the cases have cerebral dysplasia. The CT density and the MR signals of some of these cysts commonly are different from those of the cerebro spinal fluid, histological peculiarity to explain protein content different from that of the cerebro spinal fluid, children usually are born with hydrocephalus and the size of the cysts usually increased during gestation (Raybaud, 2010). An association of agenesis or dysgenesis of the corpus callosum with subarachnoidal cysts also have been recognized for example, reported two sisters that presented corpus callosal agenesis, neuralsensory deafness, and subarachnoideal cysts with hydrocephalus, the cysts being located in the pineal region and obstruct the cerebral aqueduct, as an autosomal-recessive trait (Hendriks et al., 1999). Interhemispheric meningeal lipomas are the second meningeal dysplasia which commonly
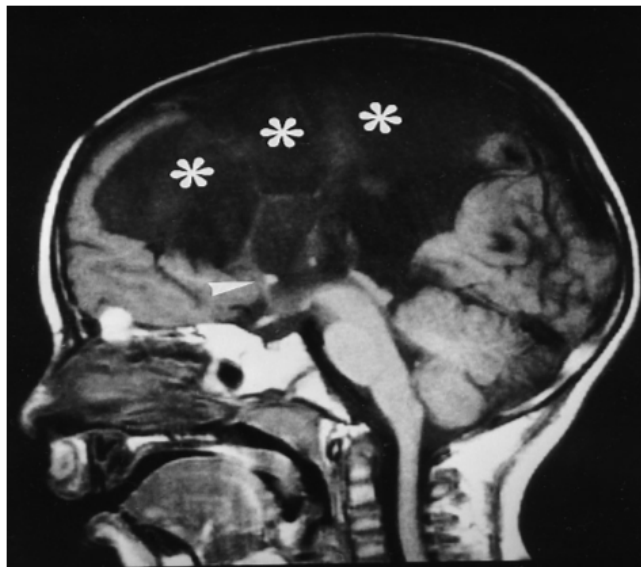
Fig. 6. Midsagittal T1-weighted image shows a complete callosal defect with interhemispheric cyst and cortical dysplasia in a 3-year-old boy, histologically verified as glioependymal (asterisks) (Utsunomiya et al., 1997).

associated with a malformation of the commissures (Raybaud, 2010). The most common location being the depth of the interhemispheric fissure where the lipoma often extend toward into the choroid plexuses (Truwit, 1990). The mechanism of the malformative association is not really known. It has been known for some time that, depending on the appearance (tubulonodular or curvilinear) and location (ventral or dorsal) of the lipoma, the dysplasia of the corpus callosum was different, while that the commissural defect does not correlate with the size or shape of the lipoma (dorsal tubulonodular lipoma can be observed with normal callosal morphology). A study, depending on the location of the lipoma, has classified it into four topographic groups: anterior, transitional (or global: covering the callosum from the front to the back), posterior, and inferior (below the hippocampal commissure). The anterior lipoma (15%) is associated with major commissural hypogenesis, the more posterior transitional lipoma (24%) with a complete but hypoplastic commissural plate, the posterior ones (48%) with minor shortening or tapering of the splenium, and the inferior ones (12%), with minor commissural abnormalities only. Craniocerebral midline defects: along the neural tube, commissuration is primarily a basal process, and in cases of commissural agenesis other commissuration defects may be observed anywhere along the ventral cord and brainstem (Raybaud, 2010). In the basal forebrain, other commonly associated defects involve the anterior optic pathway (Raybaud & Girard, 1998) and the hypothalamo-pituitary axis (Raybaud & Girard, 1998). Because the development of the corpus callosum itself is associated with the dorsalization of the hemispheres, other disorders of the dorsalization may be observed, primarily at the level of the cerebellum: a Dandy–Walker malformation (or related defect) is commonly associated with an agenesis of the corpus callosum (Johnston, 1943; Raybaud, 1982). The rare rhombencephalon synapsis is often found in association with septal defects/ septo-optic dysplasia (Michaud et al., 1982; Jellinger, 2002) and obviously the midline skull defects commonly include commissural agenesis or dysgenesis, especially the frontonasal dysplasia (Guion-Almeida et al., 1996; Wu et al., 2007) and the basal, notably sphenoidal cephaloceles (Koenig et al., 1982).

8.2 Malformations of cortical development

All abnormalities of cortical development may be associated with anomalies of the commissures. Migration disorders are probably the most typical (Raybaud, 2010). Periventricular nodular heterotopias (Volpe et al., 2006; Tang et al.,2009; Raybaud, 2010) are commonly found (Raybaud, 2010) and dysplastic-appearing deep gray nuclei characterized by small size, abnormal shape with periventricular nodular heterotopias (Volpe et al., 2006; Tang et al.,2009) see only in delayed sulcation (Tang et al., 2009). Abnormal sulcation associated with commissural anomalies are reported as the most common malformation of cortical development (Hetts et al., 2006; Byrd, 1990; Barkovich & Norman 1988; Tang et al., 2009). Major hemispheric dysplasia with large subcortical heterotopia and cortical dysplasia **(Figs. 7, 9)** are characteristic as well (Raybaud, 2010). Cortical dysplasia (Donmez et al., 2009; Volpe et al., 2006) may be associated with an interhemispheric glioependymal cyst and porencephaly (Utsunomiy et al., 1997), and small porencephalic cysts (Volpe et al., 2006). In addition to above malformations of cortical development, the gyral abnormalities have been described previously in relation to anomalies of the cerebral commissures and abnormal gyral patterns which are characterized either by abnormal, too numerous infoldings or by absent sulcation (Tang et al., 2009). These abnormalities include polymicrogyria (Utsunomiy et al., 1997 ; Hetts et al., 2006; Tang et al., 2009) classic lissencephaly (Hetts et al., 2006: Tang et al., 2009; Volpe et al., 2006, Donmez et al ., 2009), cobblestone lissencephalies (Hetts et al., 2006), schizencephaly (Hetts et al., 2006; Tang et al., 2009), schizencephaly with bilateral frontoparietal holohemispheric clefts (Utsunomiy et al., 1997), pachygyria (Tang et al., 2009), heterotopia pachygyria (Hetts et al., 2006) or diffuse pachygyria (Utsunomiy et al., 1997) and other nonclassified abnormalities (Tang et al., 2009).
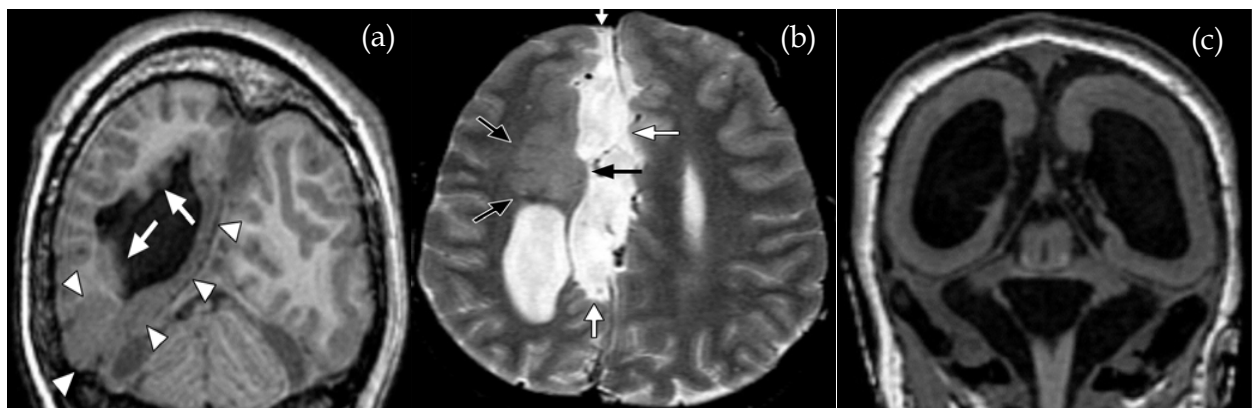

Fig. 7. Anomalies of cortical development of varying extent and severity were found in patients with callosal hypogenesis or agenesis. Coronal (a) T1-weighted image in 4-year-old boy shows periventricular nodular heterotopia (arrows) and dysplastic occipital cortex (arrowheads) in addition to dysplastic cerebellum. Axial (b) T2-weighted image in 17-year-old boy shows dysplastic frontal and cingulate cortex (black arrows) adjacent to interhemispheric cyst (white arrows). Coronal (c) T1-weighted image in 6-year-old girl shows lissencephaly with four-layer (Hetts et al., 2006).

8.3 Brain white matter anomalies

Definition of commissural anomalies is an abnormality of the white matter (Van Essen., 1997).The white matter has been postulated to contribute to normal sulcation. Abnormalities of sulcation may be possibly associated with a decreased volume of white matter, as reported in literature (Hetts et al., 2006). The following sulcal abnormalities have been described in the brains with commissural defects: The sulci of the medial surface of the hemisphere radiated in a fan-like fashion (Meyer & Röricht, 1998; Sztriha, 2005; Yousefi & Kokhei, 2009) towards the lateral **(Fig. 5. A)** wall of the third ventricle (Yousefi & Kokhei, 2009; Sztriha, 2005) without a visible callosomarginal (Meyer & Röricht, 1998; Yousefi & Kokhei, 2009) and cingulate sulcation (Yousefi & Kokhei, 2009; Sztriha, 2005). The parieto-occipital and calcarine sulci cross in the medial surface and enter toward the lateral ventricle and a lack of a well-defined cingulum (Atlas et al., 1986; Yousefi & Kokhei, 2009). Beyond the expected eversion of the cingulum and radial orientation of paramedian gyri that routinely accompany callosal agenesis (Hetts et al., 2006; Yousefi & Kokhei, 2009). Abnormalities of sulcation ranged from overly shallow olfactory sulci to frank hemispheric dysplasia (Hetts et al., 2006). The basis of one theory (Van Essen, 1997) it is possible that the absence of normal connections between hemispheres and formation of aberrant connections within the same hemisphere can delay the formation of primary sulci and perhaps even contribute to the abnormal sulcal morphology seen in so many of the cases. Reductions in extracallosal white matter volume and the presence of moderately or severely reduced extracallosal white matter volume in patients with agenesis of the corpus callosum and patients with hypogenesis of the corpus callosum may represent a primary dysplasia or hypogenesis, with fewer axons forming during development, or a secondary regression, possibly due to retraction of axons that do not find their way across midline to synapse with their homologues and thereby gain the neurotrophic support necessary for survival (Hetts, 1998). The thickness of the mid-body of the corpus callosum positively correlates with volume of cerebral white matter in children with cerebral palsy and developmental delay. Assessment of the thickness of the corpus callosum might help in estimating the extent of the loss of volume of cerebral white matter in children with a broad spectrum of periventricular white matter injury (Panigrahy et al., 2005).

8.4 Abnormal morphology of the lateral ventricle

Anomalies of the lateral ventricle are always seen in association with abnormal sulcal morphology and constantly influence at least the frontal horn and occurrs on the side with the abnormal cortical infoldings. The morphological abnormalities of the lateral ventricle include enlargement of the ventricular atria (Tang et al., 2009), widening, colpocephaly, disproportionate dilatation of the trigones and occipital horns (Bekiesiñska et al., 2004; Atlas et al., 1986; Utsunomiy et al., 1997), keyhole dilatation of the temporal horns which is thought to result from deficient hippocampal formation (Atlas et al., 1986; Utsunomiy et al., 1997), narrow frontal horns (Bekiesiñska et al., 2004), abnormal curvature of the anterior horn **(Fig . 3. A, B)** (Yousefi & Kokhei, 2009) which is explained as secondary deformity of anterior horn (Atlas et al., 1986) and irregularity of the ventricular wall due to periventricular nodular heterotopia, choroid plexus cysts, abnormal brain stem, germinal matrix and intraventricular hemorrhage (Tang et al., 2009). Also, some of the cases with callosal hypoplasia show abnormal cerebrospinal fluid spaces (Bekiesiñska et al., 2004).

Among above abnormal morphology of the lateral ventricle, colpocephaly, a selective ventriculomegaly (**Fig. 8. b**) of the occipital horns more than the frontal or temporal horns of the lateral ventricles, is a common finding in agenesis of the corpus callosum, and it appears that callosal agenesis is probably the second most frequent cause of colpocepbaly after periventricular leukomalacia (Sarnat, 1992). The deficiency of white matter around the occipital horns due to absence of the posterior fornix of the corpus callosum is the reason (Davila-Gutierrez, 2002). Holoprosencephaly (Raybaud, 2010), some subtypes of microcephaly (significantly associated) (Vermeulen et al., 2010) and DCX (doublecortin) related lissencephaly may be associated with callosal agenesis in humans (Kappeler et al, 2007). Of course the agenesis is a defining feature of the ARX (Xp22.13) related lissencephaly with callosal agenesis (Kitamura et al., 2002).
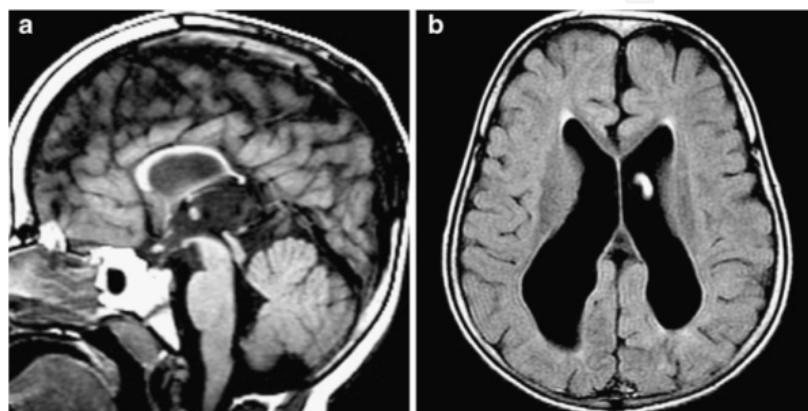

Fig. 8. Diffusely hypoplastic commissural plate with ventriculomegaly, Midline (a) sagittal T1WI.The commissural plate is complete but thin with a tiny splenium, Axial FLAIR (b). Diffuse ventriculomegaly without real evidence of leukomalacia: this points to a global white matter disorder that may be developmental or acquired, not a commissural disorder (Raybaud, 2010).

8.5 The diencephalon and rhombencephalon abnormalities

Abnormalities of the cerebellum (hemispheres, vermis), brainstem, orbits, pituitary, state of white matter myelination, and olfactory (apparatus and sulci which are more frequent in agenesis of the corpus callosum patients) have been reported in many patients with callosal anomalies (Hetts et al., 2006). Additional findings about the cerebellum include cerebellar hypoplasia with lissencephaly (Miyata et al., 2004), small or absent of the vermis, an asymmetric appearance of the fourth ventricle, small or absent of the cerebellum, the small cerebellum with abnormal orientation of the folia and posterior fossa cyst or hydrocephalus. An abnormal brain stem appears as dysplastic, small, compressed; also, dysgenesis of the corpus callosum can occur in association with dysgenesis of the frontal, parietal, and occipital lobes (Kawamura et al., 2002).

9. Syndromes that include commissural dysgenesis as a defining feature

OMIM (Online Mendelian Inheritance in Man, Johns Hopkins University, March 16, 2010) lists 189 specific syndromes in which a commissural agenesis is or may be present (Raybaud, 2010). Also, agenesis/dysgenesis of the corpus callosum has been described with

congenital metabolic diseases (Dobyns, 1989; Kiratli, 1999, Kolodny, 1989). The syndromes of the Aicardi **(Fig. 9),**Acrocallosal, Andermann and Shapiro are characterized by agenesis of the corpus callosum while others are only sporadically associated (Jeret et al., 1987). The CRASH syndrome with clinical features of callosal agenesis, retardation, adducted thumbs, spasticity, hydrocephalus (Yamasaki et al., 1997; Sztriha et al., 2000; Fransen et al., 1997; Weller & Gärtner, 2001) aphasia (Fransen et al., 1997; Weller & Gärtner, 2001) is related to a mutation of *L1* gene at Xq28 (Yamasaki et al., 1997; Sztriha et al., 2000). This gene involves in encoding a cell adhesion molecule which is involved in the fasciculation of the axons, as well as synaptic targeting and cellular migration (Schmid et al., 2008). The Miller-Dieker syndrome and Walker-Warburg syndrome are defined with partial to complete agenesis of the corpus callosum (Davila-Gutierrez, 2002). Walker-Warburg syndrome is the most severe phenotype of the group of the "cobblestone brains" that also includes the Fukuyama and the muscle-eye-brain syndromes characterized by congenital muscular dystrophy and neuronal migration disorder in which there is overmigration of the neurons beyond the pia limiting membrane. The neurons overmigrate and form abnormal arrangements in the cortical and meningeal layers. This disorganization of the tissular pattern and the abnormal extracellular matrix signals in turn results in failure of the white matter to form perfectly. Of the three phenotypes, the Walker–Warburg syndrome is the most severe, irregular (cobblestone) cortical surface, disorganized cortex, thin cerebral mantle with lack of white matter and ventriculomegaly, absence of the commissures; the underdeveloped brainstem often has a Z shape; and the cerebellum is hypoplastic with correspondingly huge posterior fossa cisterns (Raybaud, 2010). Syndromic craniosynostoses (Apert, Crouzon, Pfeiffer mostly) the typical occurrence of corpus callosal dysgenesis and/or septum pellucidum defects may well be intrinsically part of the syndromes **(**Raybaud & Di Rocco, 2007), All syndromic craniosynostoses result from a defect of one of the FGFR genes (FGFR2 on 10q25- q26 for Apert, Crouzon and Pfeiffer; FGFR1 on 8q11.22- p12 for Pfeiffer alsoes (Doherty & Wlash, 1996; Kamiguchi & Lemmon, 1997).
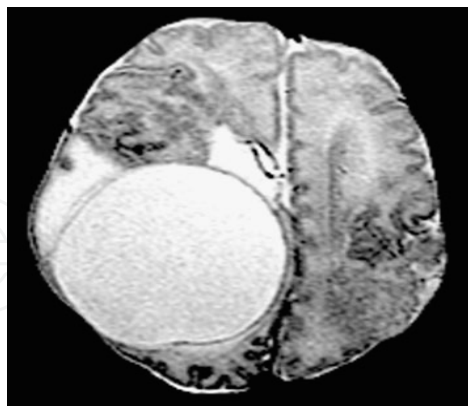

Fig. 9. Aicardi syndrome, newborn girl. Huge right-sided choroid plexus cyst with adjacent parenchymal damage. Note the multiple subcortical heterotopias and cortical dysplasia in the adjacent right frontal lobe and in the left parietal lobe (Raybaud, 2010).

10. Clinical and paraclinical features

Children with isolated form of agenesis or dysgenesis of the corpus callosum are asymptomatic or presented a mild hypotonia (Francesco et al., 2006). The intelligence

(Davila-Gutierrez, 2002) and electroencephalographic (Francesco et al., 2006), usually are normal (Davila-Gutierrez, 2002; Francesco et al., 2006), although these children have a lower capacity for processing somatosensory information (Friefeld et al., 2000). Many of the cases have shown a hypertelorism as mild facial dysmorphism which may be a clue to the neuroanatomic anomaly and further justify neuroimaging studies (Davila-Gutierrez, 2002), a more thorough neurological examination reveals defects in transfer of information. Additionally, the mental retardation can be exist (Serur et al., 1988) and neurodevelopmental outcome reported to be poor in 15–28% of cases (Moutard et al., 2003; Pilu et al., 1993). While in children with associated brain malformations (Francesco et al., 2006), the neurological features relate to the severity and variety of the accompanying cerebral defects (Davila-Gutierrez, 2002) and include epilepsy (Davila-Gutierrez, 2002; Francesco et al., 2006) mental retardation, hydrocephalus, and morphologic and growth abnormalities that vary from hypotonia to severe spasticity, ataxia, autistic behavior, learning disabilities, and behavioral disorders (Davila-Gutierrez, 2002).

10.1 Electroencephalographic features

The most characteristic feature is the continued asynchrony of sleep spindles after 18 months of age. However, this asynchrony is not an overall asymmetry, and the morphology and number of spindles in the two hemispheres are relatively equal over an extended period of stage 2 sleep (Sarnat, 1992).

10.2 Prenatal diagnosis

Laterally displacement of the lateral ventricles and atrium, upward movement of the third ventricle (Comstock et a1., 1985), absence or alteration of the cavum septum pellucidum (Meizner et a1, 1987), high-riding third ventricle, and widening of the interhemispheric fissure (Tang et al., 2009) and posterior ventriculomegaly or colpocephaly have been described as an imaging sign of the corpus callosal malformations (Lockwood et a1., 1988). Also, it is found that when complete corpus callosal dysgenesis exists, the frontal region is small and the cavum septi pellucidi is not evident (Tepper & Zale1, 1996). Male fetuses are more likely to have an isolated agenesis/dysgenesis of the corpus callosum that is considered benign in its clinical expression (Davila-Gutierrez, 2002).

11. Conclusion

It is highly likely that agenesis of the brain commissural might have been developed as a result of an early embryological abnormally growth and development .The commissures formation is a complex process and involved many commissuration factors, so that a isolated commissural agenesis is uncommon, and the abnormality is usually associated with other cerebral or craniocerebral or syndromic defects. A full understanding of the embryological, anatomical and functional of the commissures could us to the diagnosis and handling of these abnormalities.

12. Acknowledgment

13. References

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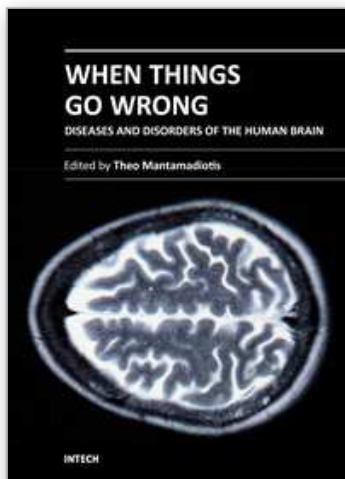
When Things Go Wrong - Diseases and Disorders of the Human Brain

Edited by Dr. Theo Mantamadiotis

In this book we have experts writing on various neuroscience topics ranging from mental illness, syndromes, compulsive disorders, brain cancer and advances in therapies and imaging techniques. Although diverse, the topics provide an overview of an array of diseases and their underlying causes, as well as advances in the treatment of these ailments. This book includes three chapters dedicated to neurodegenerative diseases, undoubtedly a group of diseases of huge socio-economic importance due to the number of people currently suffering from this type of disease but also the prediction of a huge increase in the number of people becoming afflicted. The book also includes a chapter on the molecular and cellular aspects of brain cancer, a disease which is still amongst the least treatable of cancers.

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