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لم ترد بالأصل

THE EFFECT OF ALCOHOL ON POSTNATAL DEVELOPMENT OF CORPUS COLLOSUM IN ALBINO RATS

Thesis

Submitted For Partial Fulfillment Of Master Degree
In Basic Medical Science "Anatomy"

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2000

بسم الله الرحمن الرحيم

وقل رب زدني علما

صدق الله العظيم

(من الآية ١١٤ سورة طه)

TO THE MEMORY OF MY FATHER,

MY MOTHER

MY HUSBAND

&

MY SON KAREEM

CONTENTS

AIM OF THE WORK

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Anatomy and development of the Corpus Callosum:

The corpus callosum is the largest commissure of the brain, that connects the two cerebral hemispheres. It lies at the bottom of the longitudinal fissure, Snell (1997). It consists of a mass of one hundred million commissural fibers each of which extends from cortex to cortex between symmetrical parts of the two hemispheres, McMinn (1994). For purpose of description, it is divided into the rostrum, the genu, the body and the splenium. The majority of the fibers of corpus callosum interconnect symmetrical areas of the cerebral cortex. Because it transfers information from one hemisphere to another, the corpus callosum is essential for learned discrimination, sensory experience and memory, Snell (1997). The rostrum is the thin part of the anterior end of the corpus callosum, which is prolonged posteriorly to be continuous with the upper end of lamina terminalis. The genu is the curved anterior end of the corpus callosum that bends inferiorly in front of septum pellucidum. The body of the corpus callosum arches posteriorly and ends as the thickened posterior portion called the splenium, Snell (1997). The fibers of the corpus callosum extend to all parts of the cerebral cortex. In a horizontal section the fibers of the genu are seen arching forwards on each side to the frontal cortex. This appearance gives them the name forceps minor, McMinn (1994) and Williams, *et al.* (1989).

Similarly, the fibers of the massive splenium curve backward symmetrically to the occipital cortex, forming the forceps major. Between forceps minor and forceps major the fibers of the corpus callosum spread out to the cortex on the lateral surface of the hemisphere. As they turn down into the temporal lobe they form the lateral wall of the inferior and posterior horns of the lateral ventricle, where they are known as the tapetum, McMinn (1994).

Yakovlev and Lecours (1967), revealed that myelination of corpus callosum become grossly evident at about the fourth postnatal month and proceeds anteriorly from splenium toward the genu. They also reported that callosal myelination continues through the end of the first decade and suggested that slow myelination proceeds even after this stage.

Gilles, *et al.* (1983), noted that myelination of the corpus callosum is microscopically visible at about the time of birth.

Patricia and Merrilla (1985), reported in mice (BALB/c strain) that the first callosal axons cross the midline by day 17 post conception and attains the adult size range by day 26 post conception but in mice (C57 strain) animals develops slightly earlier.

P. Berbel, *et al.* (1988), studied the develop of corpus callosum in cats and divided it into three phases:

1- Embryonic development from embryonic day 38 (E38) to embryonic day 58 (E58). At E38 only a part of body is formed, at E53 and E58 the corpus callosum was still very short, but its different parts (genu, body and splenium) had formed. During this phase all callosal axons were unmyelinated, the most common cell type were microglia, gitter cells and astrocytes which contained vacuoles with electron dense inclusions suggesting phagocytic activity.

2- Early postnatal development from postnatal day 4 (P4) to postnatal 26 (P26). In this period the corpus callosum was much longer, premyelinated and myelinated axons were seen. In this period there was a substantial axonal loss, also the most common cell type were microglia, gitter cells and astrocytes. Gitter cells however decreased in number from P18 onword. From P15 onword oligodendrocytes were seen. microglia, gitter cells and some astrocytes still showed vacuoles suggesting phagocytic activity.

3- Late postnatal development from P39 to P150. In this period the corpus callosum grew dramatically in length and thickness. However myelination is not complete at this age. During this phase microglia, astrocytes and oligodendrocytes were seen. Gitter cells were no longer found in the corpus callosum. Unlike the preceding phases the majority of microglia and astrocytes no longer showed vacuoles or inclusions suggesting phagocytosis.

Barkovich, *et al.* (1988), evaluated the normal development of corpus callosum in human using MR imaging during the first year of life. They found that the corpus callosum during the first month of life is uniformly thin. In second month a growth spurt occurs in genu, followed by a similar growth spurt in splenium between 4-6 months of age. The corpus callosum has an adult appearance by about 8 months of age.

Stephanie, *et al.*, (1989), reported that women tended to have a smaller cross sectional callosal area, a larger fraction of corpus callosum area in the posterior fifth of corpus callosum and more slender corpus callosum.

Valk and Van der knapp, (1989), noted that the development of callosal myelin is associated with a jump in the cross sectional area.

Claude, *et al.*, (1990), reported that in rats the corpus callosum contains 4.4 $\times 10^6$ axons at birth. This number increases to 11.4 $\times$ 10^6 by 5 days of age and remains constant until at least P60. Also they noted that in rats callosal axons cross the midline in two waves, one starting at about gestational day 18.5, the other at about P0 animal.

Hiroki S. and Douglas (1992), reported that in mice the first callosal axons emerged from their cortical cells of origin at about 15.5 days after conception, the axon growth rate was faster for those from frontal cortex than occipital cortex.

Aboitiz, *et al.*, (1992), studied the densities of fibers of different sizes in different regions of corpus callosum. They reported that thin fibers are most dense in the genu and decrease in density posteriorly towards the body, where they reach a minimum. Towards the splenium, the density of thin fibers increases again, but in the posterior pole of the callosum the density decreases locally. Large diameter fibers show a pattern complementary to that of thin fibers, having a peak of density in the body and a local increase in posterior pole of corpus callosum.