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RADIOLOGICAL STUDY OF THE MORPHOLOGICAL VARIATIONS OF CIRCULUS ARTERIOSUS CEREBRI IN LIBYAN PEOPLE

Thesis submitted in partial fulfillment for Master Degree in Anatomy

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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

يَا أَيُّهَا الإِنسَن مَاغَرَّك بِرَبِّك الْكَرِيم (6) الَّذِ خَلَقَك فَسَوَّاك فَعَدَلَك (7) فِيِّ صُورَ مَّاشَاء رَكَّبَك (8)

صدق الله العظيم

سورة الإنفطار

Dedication

For Allah's sake

I dedicate this work to my parents, to my brothers, sisters and their kids

and to you.

Acknowledgements

It would not have been possible to finish this thesis without the help and support of the kind people around me, to only some of whom it is possible to give particular mention here.

Above all, I would like to thank my parents, brothers and sisters who have given me their unequivocal support throughout, as always, for which my mere expression of thanks likewise does not suffice.

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For any errors or inadequacies that may remain in this work, of course, the responsibility is entirely of Satan and my own.

Contents

List of Abbreviations

- A1: Precommunicating segment of anterior cerebral artery.
- A2: Postcommunicating segment of anterior cerebral artery.
- ACA: Anterior cerebral artery.
- AcomA: Anterior communicating artery.
- AVM: Arteriovenous malformation.
- BA: Basilar artery.
- CT: Computed tomography.
- CTA: Computed tomography angiogram.
- DM: Diabetes mellitus.
- FTP: Fetal type posterior circle of Willis.
- HTN: Hypertension.
- ICA: Internal carotid artery.
- MCA: Middle cerebral artery.
- MIP: Maximum intensity projection.
- MR: Magnetic resonance.
- MRA: Magnetic resonance angiogram.
- MRI: Magnetic resonance imaging.
- PTA: Persistent trigeminal artery.
- P1: Precommunicating (peduncular) segment of posterior cerebral artery.
- P2: Postcommunicating (crural) segment of posterior cerebral artery.
- P3: Quadrigeminal segment of posterior cerebral artery.
- PCA: Posterior cerebral artery.
- PcomA: Posterior communicating artery.
- SCA: Superior cerebellar artery.
- SCP: Selective cerebral perfusion.
- SD: Standard deviation.
- SPGR : Spoiled gradient recalled acquisition.
- SPSS: Statistical package for the social sciences.
- 3D TOF MRA: Three dimensional time of flight magnetic resonance angiogram.
- TIA: Transient ischemic attacks.
- VA: Vertebral artery.

Introduction

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aim of work.

Introduction and aim of work

Circulus arteriosus:

The circulus arteriosus is a large arterial anastomosis which unites the internal carotid and vertebrobasilar systems. It lies in the subarachnoid space within the deep interpeduncular cistern, and surrounds the optic chiasma, the infundibulum and other structures of the interpeduncular fossa (Warwick and Williams, 2008).

The circulus arteriosus which also called circle of Willis after **Sir Thomas Willis** who was the first scientist to describe it in 1644 and his description was the classical textbook description of circle of Willis (Van der Zwan et al, 1993; Hartkamp and Van der Grond, 2000) which consists of Anteriorly, the two anterior cerebral arteries ACA, which are derived from the internal carotid arteries of both sides, and are joined together by the small anterior communicating artery (AcomA). Posteriorly, the two posterior cerebral arteries (PCA), which are formed by the division of the basilar artery (two terminal branches) and each artery of them is connected to the ipsilateral internal carotid artery by a posterior communicating artery (PcomA). In the majority of instances, the posterior communicating arteries are very small, however, and a limited flow is possible between the anterior and posterior circulations. This is important because the primary purpose of the vascular circle is to provide anastomotic channels if one vessel is occluded (Warwick and Williams, 2008).

There is considerable individual variation in the pattern and caliber of vessels which

make up the circulus arteriosus. Although a complete circular channel usually exists, one vessel is usually sufficiently narrowed to reduce its role as a collateral route.

Cerebral and communicating arteries individually may all be absent, variably hypoplastic, double or even triple. The circle is rarely functionally complete (Warwick and Williams, 2008).

Since Thomas Willis has just described the circulus arteriosus and the scientists became fond of the anatomy and variation of this circle and thus numerous studies have been carried out. These studies demonstrated that the circle of Willis is one of the most variable parts of human vascular system. And from an ontogenetic point of view indicating random variability of the cerebral vessels (Van der Zwan et al, 1993). Many variations have been recognized in different segments of the circle of Willis such circles in which certain segments are hypoplastic, absent, duplicated or even triplicated (Hartkamp and Van der Grond, 2000).

Commonly, the diameter of the precommunicating part of the posterior cerebral artery is larger than that of the posterior communicating artery; in which case the blood supply to the occipital lobes is mainly from the vertebrobasilar system. Sometimes, however, the diameter of the precommunicating part of the posterior cerebral artery is smaller than that of the posterior communicating artery, in which case the blood supply to the occipital lobes is mainly from the internal carotids via the posterior communicating arteries (PcomA) (Warwick and Williams, 2008), A variation known as a fetal-type posterior cerebral artery (FTP) (Hartkamp and Van der Grond, 2000); which is due to the embryological development where in early fetal stages, the posterior cerebral arteries originate from the internal carotid arteries. Later in fetal development, the posterior cerebral arteries attach themselves to the apex of the basilar artery leaving behind the smaller posterior communicating arteries.

However, when the posterior cerebral arteries remain attached to the internal carotid arteries, this result in the fetal origin of the posterior communicating arteries (Zachariah et al, 2005)

There are two variants of the fetal type posterior circle of Willis which are full and partial, and there is another variant called the transitional variant of posterior circle of Willis which is intermediate between both fetal type and the adult type posterior circle of Willis. In the full variant of FTP the P1 segment (which is the precommunicating segment of posterior cerebral artery) is absent. In partial variant of FTP the P1 segment is hypoplastic, and in the transitional variety both posterior communicating and P1 are almost of the same caliber. And all of these fetal type variants could be unilateral or bilateral, and in case of bilateral full variant of FTP it implies near 100% dependence on the carotid arteries for cerebral blood supply, with minimal or no dependence on the vertebrobasilar system, rendering the subject less well-equipped with potential collateral pathways (Zachariah et al, 2005).

The brain is a vital organ with a high metabolic activity and it demands approximately 15% of the cardiac output and consumes about 25% of the total oxygen consumption of the body (Warwick and Williams, 2008), thus Collateral circulation in the brain is important for maintaining a sufficient level of cerebral blood flow in case of obstructive disease in the main afferent arteries. This arterial network consists of extra-cranial and intra-cranial routes. The intracranial collateral vessels comprise the so-called primary collaterals, consisting of the arterial segments of the circle of Willis, which are used in case of acute need (Van Raamt et al, 2006). Where the primary purpose of circle of Willis is to provide an anastomotic channel if one vessel is occluded (Warwick and Williams, 2008) and to connect the circulation in the right and left hemispheres with the circulation in the anterior and posterior

hemispheres (Zachariah et al, 2005) i.e. carotid and vertebrobasilar systems of both sides to ensure a well maintained cerebral perfusion, and the secondary collaterals such as the ophthalmic artery and the leptomeningeal vessels, which develop after an ischemic stimulus when the primary collaterals are insufficient.

There is increasing evidence that a well functioning complete circle of Willis plays an important protective role against cerebral ischemia in patient with carotid occlusive disease (Hoksbergen et al, 2000), and the collateral ability of the circle is believed to be dependent on the size of its component vessels (Hartkamp et al, 1999). So this suggests, while an individual with one of these variations may under normal circumstances suffer no ill effects, there are certain pathological conditions which can present a risk to the person's health and increase the possibility of suffering an ischemic stroke when compounded with the effects of an anatomical variation (Moore et al, 2005).

Furthermore some surgical, neurosurgical and interventional radiological procedures which require an afferent artery to be temporarily or permanently occluded, relying for brain perfusion during the operations or the procedures on the integrity of circle of Willis (Hartkamp and Van der Grond, 2000; Moore et al, 2005; Merkkola et al, 2006; Alawad et al, 2009). Also because the hemodynamic of the circle is influenced by the variations in the caliber of the communicating arteries and in the segments of the anterior and posterior cerebral arteries which lie between their origins and their junctions with the corresponding communicating arteries (Warwick and Williams, 2008). And those anatomical variations might therefore pose different hemodynamic challenges to cerebral blood flow and might thus possibly evoke the formation of aneurysms (Chen et al, 2004).

Aim of work:

The aim of the study is to find out the common variations in cerebral vessels constituting circle of Willis in Libyan people.

Introduction and aim of work

Circulus arteriosus:

The circulus arteriosus is a large arterial anastomosis which unites the internal carotid and vertebrobasilar systems. It lies in the subarachnoid space within the deep interpeduncular cistern, and surrounds the optic chiasma, the infundibulum and other structures of the interpeduncular fossa (Warwick and Williams, 2008).

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hemispheres (Zachariah et al, 2005) i.e. carotid and vertebrobasilar systems of both sides to ensure a well maintained cerebral perfusion, and the secondary collaterals such as the ophthalmic artery and the leptomeningeal vessels, which develop after an ischemic stimulus when the primary collaterals are insufficient.

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Furthermore some surgical, neurosurgical and interventional radiological procedures which require an afferent artery to be temporarily or permanently occluded, relying for brain perfusion during the operations or the procedures on the integrity of circle of Willis (Hartkamp and Van der Grond, 2000; Moore et al, 2005; Merkkola et al, 2006; Alawad et al, 2009). Also because the hemodynamic of the circle is influenced by the variations in the caliber of the communicating arteries and in the segments of the anterior and posterior cerebral arteries which lie between their origins and their junctions with the corresponding communicating arteries (Warwick and Williams, 2008). And those anatomical variations might therefore pose different hemodynamic challenges to cerebral blood flow and might thus possibly evoke the formation of aneurysms (Chen et al, 2004).

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The aim of the study is to find out the common variations in cerebral vessels constituting circle of Willis in Libyan people.

Review

Of

Literature.

REVIEW OF LITERATURE

CIRCULUS ARTERIOSUS ANATOMY:

The circulus arteriosus (circle of Willis) is a large arterial anastomosis which unites the internal carotid and vertebrobasilar systems. It lies in the subarachnoid space within the deep interpeduncular cistern, and surrounds the optic chiasma, the infundibulum and other structures of the interpeduncular fossa. Anteriorly, the anterior cerebral arteries, which are derived from the internal carotid arteries, are joined by the small anterior communicating artery. Posteriorly, the two posterior cerebral arteries, which are formed by the division of the basilar artery, are joined to the ipsilateral internal carotid artery by a posterior communicating artery.

In the majority of instances, the posterior communicating arteries are very small; however, a limited flow is possible between the anterior and posterior circulations. This is important because the primary purpose of the vascular circle is to provide anastomotic channels if one vessel is occluded. There is considerable individual variation in the pattern and caliber of vessels which make up the circulus arteriosus. Even if a complete circular channel exists, one vessel is usually sufficiently narrowed to reduce its role as a collateral route. Cerebral and communicating arteries individually may all be absent, variably hypoplastic, double or even triple. The circle is rarely functionally complete.

Components: The circle of Willis is an interconnecting arterial polygon that surrounds the ventral surface of the diencephalon adjacent to the optic nerves and tracts the normal circle of Willis is comprises of the following vessels:

1- The two internal carotid arteries (ICAs).

2- The horizontal A1 segment of both anterior cerebral arteries.
3- The anterior communicating artery (AcomA).
4- The two posterior communicating arteries (PcomA).
5- The horizontal P1 segments of both posterior cerebral arteries.
6- The basilar artery (BA).

The ICAs, ACAs, AcomA and their branches are sometimes termed the anterior circulation; the basilar bifurcation, the PCAs and PcomAs are collectively termed as the posterior circulation (Saeki and Rhoton, 1977).

Branches: several small vessels arise from the circle of Willis to supply the optic chiasma and tracts, infundibulum, hypothalamus, and other important structures at the base of brain (Saeki and Rhoton, 1977).

Cerebral arteries:

The distal internal carotid artery usually terminates by bifurcating into anterior and middle cerebral arteries. The posterior cerebral arteries typically arise from the basilar artery, less commonly from the ICA (Osborn, 1980).

Anterior cerebral artery (ACA):

It is the smaller of the two terminal internal carotid artery branches (Muller et al, 1991). ACA has several major segments, each of which has important branches.

1- Horizontal A1 segment extends medially from the ACA origin to its junction with AcomA. Deep perforating branches, the middle lenticulostriate arteries, arise from the A1 segments and pass cephalad through the anterior perforated substance. These small vessels usually supply the head of caudate nucleus and anterior limb of the internal capsule (Ghika et al, 1990).

2- A2 segment. This segment includes the ACA from its junction with AcomA to its bifurcation into the pericallosal and the callosalmarginal arteries. The A2 segment courses cephalad in the cistern of the lamina terminalis and curves around corpus callosum genu (Osborn, 1980).

Anterior communicating artery (AcomA):

The AcomA is technically part of the circle of Willis, not a true ACA branch. However, most investigators treat ACA and AcomA as a single complex (Nathal et al, 1992). Perforating branches, small perforating AcomA branches are almost invariably present. These tiny but nevertheless important vessels may supply parts of the lamina terminalis and hypothalamus, anterior comissure, fornix, septum pellucidum, paraolifactory gyrus, the subcallosal region, and the anterior part of the cingulated gyrus. Occasionally these branches may even supply part of the medial surface of the cerebral hemisphere beyond the callosal genu (Vincentelli et al, 1991). Although rarely seen on routine cerebral angiograms, they sometimes can be visualized on super-selective studies and are often encountered during microsurgical approaches to AcomA aneurysms (Nathal et al, 1992).

Posterior cerebral artery (PCA):

In most cases the PCAs originates from the basilar artery bifurcation. The major PCA segments and their branches are as follows (Gerber et al, 1993).

1- Precommunicating (P1or peduncular) segment. This is a short segment of PCA which extends laterally from its origin at the basilar bifurcation to its junction with PcomA. Posterior thalamoperforating arteries arise from the basilar bifurcation and from P1 segments, to supply the diencephalon and midbrain (Barkhoff and Valk, 1988).

2- Ambient (P2 or crural) segment. This perimesencephalic segments extends from the PCA-PcomA junction posteriorly around the midbrain, coursing above the trochlear nerve and the tentorial incisura. Its major branch is the lateral posterior choroidal artery which can originate either from P2 or proximal cortical branches. It supplies the posterior thalamus and lateral ventricle choroidal plexus.

3- Quadrigeminal (P3) segment. This segment of PCA runs behind midbrain within Quadrigeminal plate cistern (Osborn, 1990).

Vertebrobasilar arterial system:

The vertebral arteries usually originate from their respective subclavian arteries; the left vertebral artery is usually the dominant vessel (Schronz C et al, 1986). Each vertebral artery passes superomedially through the foramen magnum. Within the posterior fossa, usually anterior to the medulla, the vertebral arteries unite to form the basilar artery (Osborn, 1990). The basilar artery courses cephalad in front of the pons and terminates in the interpeduncular cistern by dividing into the posterior cerebral arteries. The basilar artery averages about 3 cm in length and 1.5 to 4 mm in width (Wilson et al, 1954); diameter greater than 4.5 mm should be considered abnormal (Smoker et al, 1986).

Embryology of the circle:

At the 4 to 5.7 mm stage of the embryonic life (28–30 days), the ICA, which develops as a cranial extension of the paired dorsal aorta, is formed (Okahara et al, 2002). Paired longitudinal neural arteries appear along the hindbrain and coalesce to form the basilar artery at the 5 to 8 mm stage. The caudal divisions of the ICA anastomose with the neural arteries to become PcomAs. At the 40 mm stage (8 weeks) the PCAs are extensions of the PcomA. The vertebrobasilar system

develops and thus participates in the supply of the PCA through the segment between the basilar artery and the post communicating part of the PCA, the P1 segment. In that phase, the component vessels of the circle of Willis all have the same caliber. In the remaining fetal period, the circle develops into one of three variants: an adult configuration, a transitional configuration or a fetal (embryonic) configuration (Padget, 1948). In the adult configuration, the P1 segment has a larger diameter than the PcomA. In the transitional configuration, the PcomA and P1 have an equal diameter. Both the basilar artery and the ICA thus contribute equally to the PCA. The fetal or embryonic configuration is the variant in which the P1 is smaller than the PcomA and the ICAs are the main blood suppliers to the occipital lobes. It has been shown that these variations in morphology arise during fetal brain development (Van Overbeeke et al, 1991). And during this period, the frequency of adult and fetal configurations increases, while the number of transitional configurations decreases (Van Raamt et al, 2006).

A study of fetal and neonatal circles of Willis resulted in the conclusion that the variations of this segment were the result of developmental modification especially with increased functional demands in connection with the rapid growth of the occipital lobes (Van Overbeeke et al, 1991). Others believed that the arterial variations and abnormalities have fetal characteristics and that they are the result of a genetically established pattern (Vasovic et al, 2002).

Role of the circle:

The primary purpose of the vascular circle is to provide anastomotic channels if one vessel is occluded, where it enables inter-hemispheric flow through the anterior communicating artery and in two directions through the two PcomAs (Liebeskind 2003). Where the collateral circulation in the brain is important for

maintaining a sufficient level of cerebral blood flow in case of obstructive disease in the main afferent arteries. This arterial network consists of extra-cranial and intra-cranial routes. The intracranial collateral vessels comprise the so-called primary collaterals, consisting of the arterial segments of the circle of Willis, which are used in case of acute need, and the secondary collaterals such as the ophthalmic artery and the leptomeningeal vessels, which develop after an ischemic stimulus when the primary collaterals are insufficient (Liebeskind, 2003), the leptomeningeal collateral indicates a poor hemodynamic status, in which collateral flow through the circle of Willis was not sufficient (Muller and Schimrigk, 1996 and Hofmeijer et al, 2002).

While an individual with one of the variations of circle of Willis may suffer no ill effects under normal circumstances, the reduced ability for collateral blood flow via the circle of Willis compounded with certain pathological conditions can present a health risk, in particular the possibility of suffering an ischemic stroke (Petty et al, 2000). The collateral potential of the circle of Willis is believed to be dependent on the presence and size of its component vessels (Schomer et al, 1994 and Mull et al, 1997). Like in patients with ICA occlusion and a well-functioning AcomA, the collateral supply from the PcomA to the deprived hemisphere is thought to fell to zero when its diameter was set at levels, 0.5 to 0.6mm (Cassot et al, 1995; Dickey et al, 1996 and Hoksbergen et al, 2000). Therefore, the term hypoplasia may be reserved for those vessels that cannot supply collateral flow (Hoksbergen et al, 2000). Patients with ICA occlusion demonstrate a high prevalence of collateral flow through the anterior circle of Willis and significantly increased diameters of the communicating channels. Patients with bilateral ICA occlusion relied on collateral flow via the posterior circle of Willis and demonstrated a bilateral increase in posterior communicating arteries diameters (P

0.05). Thus PcomAs are more important in cases of bilateral ICA occlusions than it is in cases of unilateral ICA occlusion (Schomer et al, 1994).

A higher percentage of complete circles in living patients with ICA obstruction compared with the autopsy populations as described by (Alpers and Berry, 1963; Riggs and Rupp, 1963). The differences in circle of Willis configurations found in these studies may therefore indicate the protective role of the circle of Willis in survivors of ICA occlusive disease (Hartkamp et al, 1999). Some authors concluded that the relative risk of hypoperfusion infarctions was significantly higher in cases with a nonfunctioning AcomA (Miralles et al, 1995 and Schomer et al, 1994). However, others concluded that the presence of a large ipsilateral PcomA was the only feature that correlated significantly with the absence of a watershed infarction (Hartkamp et al, 1999). Patients with variants of the circle with efficient collateral circulation have a lower risk of transient ischemic attack (TIA) and stroke than that of patients without such collaterals (Henderson et al, 2000 and Hoksbergen et al, 2003). On the other hand an incomplete circle of Willis increases the risk of TIA during carotid clamping in patients with contralateral ICA occlusion (Lee et al, 2004). A higher percentage of circle of Willis variants has been reported in the mentally ill and in patients with cerebrovascular catastrophy, indicating a possible linkage, the one being consequent on the other. knowledge of the normal size of these vessels may also be of use to the surgeon in assessing the feasibility of shunt operations and in the choice of patients (Kamath, 1981).

Hence the circle functions as an anastomotic channel and offers a potential shunt under abnormal conditions such as might occur during an occlusion or spasm (Rogers and Lambert, 1946), thus from this fact extra efforts is to be taken to improve brain protection in aortic arch reconstructions and during carotid endarterectomy where brain perfusion during these operations is mainly dependent on the integrity of the circle of Willis, and a considerable number of patients are

still at risk for neurologic injuries (Spielvogel et al, 2005 and Merkkola et al, 2006). On the contrary though there is a complete circle in 90% of cases, in most, one of the vessels is sufficiently narrowed to impair its role as a collateral route (Fields and Weibel, 1965). Angiographic evidence supports defective or absent circulation in about a third of such cases (Alawad et al, 2009).

Variations in the circle as a whole:

This vessel structure enables inter-hemispheric flow through the anterior communicating artery and in two directions through the two PcomAs (Liebeskind 2003; Van Raamt et al, 2006). Where it functions as an anastomosis and offers a potential shunt under abnormal conditions such as might occur during an occlusion or spasm (Rogers and Lambert, 1946 and Kamath 1981). It has been shown to exhibit many kinds of anatomical variations (Kayembe et al, 1984). And these anatomical variations of the circle of Willis are common (Wolpert, 1997 and Kim et al, 2002). The typical circle of Willis was defined with respect to its components as a closed circuit (Kayembe et al, 1984).

Many studies have reported that hypoplasia of vessels as the commonest anomaly in the circle of Willis (Fetterman and Moran, 1941; Alpers et al, 1959; Riggs and Rupp, 1963; Alpers and Berry, 1963; Reddy et al, 1972). Whereas these workers have reported abnormal diameters most commonly in the PcomAs, followed by the posterior cerebrals, others observed them more commonly in the PCAs than in the PcomAs (Windle, 1887; Fawcett and Blachford, 1905).

Normal variants: A complete circle of Willis in which no component vessel is absent or hypoplastic is seen in only 20% to 25% of cases (Saeki and Rhoton, 1977). 21.30% (Chen et al, 2004). (Macchi et al, 1996 and Hartkamp et al, 2000) demonstrated that the typical circle seen in 41%, others reported among the general

population, approximately 50% have a complete circle of Willis (Alpers et al, 1959; Hartkamp et al, 1998 and Van Raamt et al, 2006), where possible variations can include underdeveloped or completely absent blood vessels (Moore et al, 2005; Moore et al, 2006).

In (Riggs and Rupp, 1963; Lazorthes et al, 1979; El Khamlichi et al, 1985 and Eftekhar et al, 2006) works more than 50% of the cases were complete circle with no hypoplasia. Other investigators found higher percentages of 72% as reported by (Alawad et al, 2009), and 90% as mentioned by (Fields and Weibel, 1965) who also commented though there is a complete circle in of cases, in most, one of the vessels is sufficiently narrowed to impair its role as a collateral route (Fields and Weibel, 1965). Angiographic evidence supports defective or absent circulation in about a third of such cases (Alawad et al, 2009).

In comparative studies between circles of normal subjects and circles of people with circlus arteriosus aneurysms it was found that the incidence of atypical circles in the aneurysm and control series were 63% and 48% respectively (Alpers and Berry, 1963 and Kayembe et al, 1984). And at other comparative studies between circles of normal subjects and circles of people suffering ICA occlusive disease patients demonstrated a significantly higher percentage of entirely complete circle of Willis configurations in patient versus normal control (55% versus 36%, P50.02), and the percentage of complete circle of Willis configurations was highest on the symptomatic side (P50.001). No trend was found in the completeness of the circle of Willis with respect to severity of the carotid lesion (Hartkamp et al, 1999). Other comparative study on 3D TOF MRA reported 36% show an entirely complete circle configuration in control group versus 55% in patients with significant internal carotid stenosis. It is likely that the (inherent) inclusion of survivors of ICA obstruction may have resulted in the selection of a subgroup from the entire population with ICA lesions, favoring patients in whom a

complete circle configuration and/or larger diameters were a pre-existent feature (Hartkamp and Van der Grond, 2000).

Where the collateral potential of the circle of Willis is believed to be dependent on the presence and size of its component vessels (Mull et al, 1997; Schomer et al, 1994). Patients with ICA occlusion demonstrated a high prevalence of collateral flow through the anterior circle and significantly increased diameters of the communicating channels. Patients with bilateral ICA occlusion relied on collateral flow via the posterior circle and demonstrated a bilateral increase in PcomAs diameters (P,0.05) (Hartkamp et al, 1999).

In another study it was found that an incomplete circle of Willis increased the risk of TIA during carotid clamping in patients with contralateral ICA occlusion (Lee et al, 2004).

The common variations in the circle:

1. Absent anterior communicating artery.
2. Doubled anterior communicating artery.
3. Triplet anterior communicating artery.
4. Absent Rt. A1.
5. Hypoplastic Rt. A1.
6. Absent Lt. A1.
7. Hypoplastic Lt. A1.
8. Azygotic anterior cerebral artery.
9. Absent Rt. PcomA.
10. Hypoplastic Rt. PcomA.

11. Absent Lt. PcomA.
12. Hypoplastic Lt. PcomA.
13. Absent Rt. P1.
14. Hypoplastic Rt. P1.
15. Absent Lt. P1.
16. Hypoplastic Lt. P1.
17. Transitional type Rt. PcomA.
18. Transitional type Lt. PcomA.

Variations in the anterior circle:

There is a considerable variation in the presence of the arterial segments of the circle of Willis. On the anterior part the AcomA or one of the A1 segments of the ACA can be missing or hypoplastic (Hartkamp et al, 1998; Alpers et al, 1959 and Van Raamt et al, 2006). Where anatomic variations in the ACA – AcomA complex are present approximately on one third of anatomic dissection (Nathal et al, 1992); Other studies found a range of percentages as follows (Macchi et al, 1996) demonstrate that complete configuration of the anterior part was present in 90%, (Lee et al, 2004) found it was the most frequent type 80%, (Chen et al, 2004) found 78.30% of cases had complete anterior parts of the circle of Willis, (Hartkamp et al, 1998 and Merkkola et al, 2006) reported complete anterior circulation in 74% and others found lesser percentages of 60% of subjects with complete anterior circle (Miralles et al, 1995).

Some studies compared the percentage of complete anterior circle of Willis among patient suffering ICA occlusion and normal control people and found that complete anterior circulation at patient to control percentage 88% to 68% (Hartkamp et al, 1999), with a statistically significant diameter increase in the anterior channels.

Thus, the AcomA appears to be of more importance in unilateral ICA occlusion (Hartkamp and Van der Grond, 2000). Patients with unilateral ICA occlusion demonstrated a high prevalence of collateral flow through the anterior circle of Willis and significantly increased diameters of the communicating channels (Hartkamp et al, 1999); which is also consistent with (Hoksbergen et al, 2000) where they reported that cross flow through anterior circulation was insufficient in 5%.

Similar studies found that the frequency of complete pattern of the anterior part of the circle in control group 43% was lower than that of ICA occlusion suffering group (Lee et al, 2004). And percentage of complete anterior circle configuration for an elderly subgroup of population aged more than 60 years was 68% on 3D TOF MRA, all of these finding might suggest protective role of AcomA collateral flow (Hartkamp and Van der Grond, 2000).

Variations in ACA:

anatomic variations in the ACA are present in a considerable percentage of anatomic dissection. Common variations are hypoplastic or absent A1 segment, seen in 5% to 18% (Nathal et al, 1992; Van Raamt et al, 2006). And azygus ACA which is a solitary unpaired vessel that arises as a single trunk from the confluence of the horizontal A1 segment of the right and left ACAs (Schick and Rumbaugh, 1989). A true azygus ACA is rare and is often associated with other intracranial anomalies such as lobar holoprosencephaly and saccular aneurysm (Cennamon et al, 1992). More commonly, a bihemispheric ACA sends a variable number of branches to the contralateral hemisphere. Here, separate right and left ACA vessels are present, but one is dominated and sends branches to both hemispheres, whereas the other is hypoplastic and may terminate in an orbitofrontal or frontopolar

branch. Other ACA variants such as infraoptic origin and A1 fenestration or duplications are uncommon. Infraoptic ACA origin and duplicated ACA are associated with an increased incidence of saccular aneurysm (Cennamon et al, 1992; Suzuki et al, 1992). Fenestrated arteries have the same incidence of intracranial aneurysms as other circle of Willis bifurcation points (Sanders et al, 1993). And a definite correlation between asymmetric proximal segments of the anterior cerebral artery and aneurysm of the AcomA (Kayembe et al, 1984). And others obtained the same results (Wilson et al, 1954 and Stehbens, 1963).

In contrast of these literatures others found lesser percentages of variations at ACA; where hypoplasia of the ACA occurred in 0.7% (Alawad A et al, 2009). And even more (Eftekhar et al, 2006) found no hypoplasia of precommunicating part of the left anterior cerebral artery A1 nor aplasia of the A1.

Variations in AcomA:

There are many variations noted in AcomA which include missing (aplastic) or hypoplastic (Van Raamt et al, 2006), duplicated, triplicated, fenestrated or plexiform AcomA (Kayembe et al, 1984). Variations could be at shape, length or caliber; where coefficient of variation at the length was the greatest for the AcomA (Kamath et al, 1981). And the smallest luminal diameter allowing for cross-flow through the AcomA was 0.4 mm (Cassot et al, 1995; Dickey et al, 1996 and Hoksbergen et al, 2000). AcomAs were of normal morphology and caliber in 45% (Merkkola et al, 2006). Other studies show hypoplastic AcomA to represent 10% of cases (Riggs and Rupp, 1963) and 11% of cases (El Khamlichi et al, 1985), it was seen in less than 5% in (Lazorthes et al, 1979) study and 3% in study of (Fisher, 1965).

Various studies found different figures of aplasia of AcomA where it was found to represent 22% (Merkkola et al, 2006) , 10% (Kim et al, 2002), 2.1% (Piganiol et al, 1960; Macchi et al, 1996 and Alawad et al, 2009), 1% (Piganiol et al, 1960; Eftekhar et al, 2006). Duplication of AcomA was seen in 10% of all cases (Nathal et al, 1992). Other studies denied any variations at AcomA where it was found in all cases 100%, with no duplication or triplication (Baptista, 1964; Perlmutter and Rhoton, 1976 and Hoksbergen et al, 2000).

Regarding the function of AcomA it was found that the relative risk of hypoperfusion infarctions was significantly higher in cases with nonfunctioning AcomA (Miralles et al, 1995). And regarding the relation of fenestrated AcomA with aneurysms at circlus arteriosus 50% of circles of Willis with Fenestrated AcomA showed aneurysms in AcomA. 40% of the aneurysms in circles of Willis with Fenestrated AcomA were localized in different places such as BA, VA, ICA bifurcation and anterior choroidal artery. 60% of circles with median ACA show aneurysms in AcomA, 20% of which were had asymmetric ACA also. The remaining aneurysms were located in other sites. 90% cases of circles of Willis with Asymmetrical ACA have aneurysms on AcomA; 70% of aneurysms in AcomA occurred on the thicker side or midline and 20% on the thinner side (Kayembe et al, 1984).

Variations in the posterior circle:

Posterior component of circle of Willis shows a greater percentage and variability of variants where (Fetterman and Moran, 1941; Alpers et al, 1959 and Reddy et al, 1972) have reported hypoplasia of vessels is the commonest anomaly in the circle of Willis and abnormal diameters most commonly in the PcomAs, followed by the PCAs, others (Windle, 1887; Fawcett and Blachford, 1905 and

Warwick and Williams, 1973) observed them more commonly in the PCAs than in the PcomAs. And a significant inverse relationship existed between the diameters of the PCAs and PcomAs of the same side (Kamath, 1981).

A low percentage of complete posterior circle of Willis of 25.44% (Chen et al, 2004); 26% -24% (Lee et al, 2004), Where as others reported a higher values 52% (Hartkamp et al, 1998), 50% (Saeki and Rhoton, 1977), 49% (Macchi et al, 1996) and 47% (Hartkamp and Van der Grond, 2000). In another study estimating blood flow through circle of Willis (Hoksbergen et al, 2000) reported that the cross flow through the posterior circulation was insufficient in 45% of cases. In other studies comparing the percentage of completeness of posterior circle of Willis in patient with internal carotid artery occlusive disease and control group, a complete posterior configuration was demonstrated in 63 % of patients, compared to 47 % in controls (P = 50.04) (Hartkamp et al, 1999; Hartkamp and Van der Grond, 2000), and at another similar study a high percentages of complete posterior circles (74%) was seen at the diseased group (Miralles et al, 1995).

Variations in PCAs:

Many studies found variable figures of percentages of variants of PCAs. Abnormally narrow diameter was most frequently seen in the PCAs (Warwick and Williams, 1973), and PcomAs (Rogers and Lambert, 1946). A significant inverse relationship existed between the diameters of the PCAs and PcomAs of the same side (Kamath, 1981). Common variants include hypoplasia, aplasia and FTP. Where hypoplasia of the PCA occurred in 2.1% (Alawad et al, 2009), aplasia of PCA also occurred in 2.1% (Macchi et al, 1996; Alawad et al, 2009). FTP with hypoplastic or absent P1 segment occurred in 17% (Haring et al, 1993). On the

other hand (Eftekhar et al, 2006) reported no aplasia of P1 was seen. Arteriovenous malformations AVMs were found in (1.4%) in the PCA (Alawad et al, 2009).

Variations in PcomA:

The PcomAs demonstrate the greatest variation in diameter, where abnormally narrow diameter was most frequently seen in the PCAs and PcomAs. And a significant inverse relationship existed between the diameters of the PCA and PcomA of the same side **(**Kamath, 1981).

Those variations are found unilaterally and bilaterally also. Common variants include hypoplasia, aplasia, infundibular dilation of PcomA and fetal type variants. Different figures of these variants were published by different investigators; of which unilateral and bilateral hypoplasia of PcomAs were the most common (Eftekhar et al, 2006). Hypoplasia of one or both PcomAs 34%, infundibular dilatation at the PcomA origin from the ICA are present in 10% of cases (Haring et al, 1993). And in 33% of cases both PcomA were hypoplastic (Eftekhar et al, 2006). According to the study (Kim et al, 2002) the PcomA may be hypoplastic or absent on one or both sides of the brain in about 25% to 30% of patients.

Regarding aplasia of PcomA (missing PcomA) in 3% both right and left PcomAs were absent. absent PcomAs on both sides seen in 3%, on the left side seen in 3%, and 4% on the right side (Eftekhar et al, 2006). Absence of right PcomA in 2.2% and left PcomA in 0.85% (Fawcett and Blachford, 1905; Fisher, 1965 and He et al, 2007) which show aplasia of PcomA is more common on the left side, other studies show the opposite where in study of (Windle, 1887 and Piganiol et al, 1960) the right PcomA was absent in 4.5%, left PcomA was absent in 6.5% and at

study of (Alawad A et al, 2009) aplasia was observed in 3.5% & 0.7% on the left and right side respectively.

(Schomer et al, 1994) observed that the presence of a large ipsilateral PcomA was the only feature that correlated significantly with the absence of a watershed infarction. In cases of bilateral ICA occlusion patients demonstrated a statistically significant bilateral diameter increasing in the PcomA, hence a conclusion of PcomAs dominant role in bilateral ICA occlusions (Hartkamp and Van der Grond, 2000). Which indicate that the PcomA is more important in cases of bilateral ICA occlusions than it is in cases of unilateral ICA occlusion (Schomer et al, 1994; Hartkamp et al, 1999).

Variations in Rt. side and Lt. side of the circle:

Various studies at circle of Willis compared the percentages of variations between the right side of the circle and the left side of the circle and various results; (Eftekhar et al, 2006) found neither hypoplasia nor aplasia of precommunicating part of the left anterior cerebral artery LA1, neither they found hypoplasia of precommunicating part of the left posterior cerebral artery LP1. (Fawcett and Blachford, 1905) reported absence of PcomA in 2.2% on the right side and in 0.85% on the left side. (Eftekhar et al, 2006) found absence of PcomA on the left side in 3%, and 4% on the right side and hypoplasia of RPcomA in 16% and on LPcomA in 11% of cases. Kamath, 1981 reported that internal carotid arteries and the left anterior cerebral artery did not exhibit abnormal form throughout the study. On the other hand many authors were in disagreement with the former results, where (Macchi et al, 1996; Alawad et al, 2009) observed anomalies of ACA, PcomA and ICA were found to occur more on the left. In study of (Windle, 1887) the right PcomA was absent in 4.5%, left PcomA was absent in 6.5% of cases.

(Alawad et al, 2009) reported PcomA 3.5% and 0.7% on the left and right side respectively, and in other literatures it is found to range from 2.2% as reported (Fisher, 1965) to 4.5% as reported (Piganiol et al, 1960) on the right side and from 0.85% as reported (He et al, 2007) to 6.5% as found (Piganiol et al, 1960) on the left side which conclude aplasia of PcomA is more common on the left side. And regarding fetal type posterior circle of Willis it was seen in 26% on the right side and 28% on the left side (Eftekhar et al, 2006).

Fetal variants:

These include fetal type posterior circle of Willis and trigeminal variant.

A- Fetal type posterior circle of Willis (FTP).

Definition:

Embryologically, the posterior cerebral arteries originate from the internal carotid arteries. Later in fetal development, the posterior cerebral arteries attach themselves to the apex of the basilar artery leaving behind the smaller posterior communicating arteries. However, when the posterior cerebral arteries remain attached to the internal carotid arteries, this results in the fetal origin of the posterior communicating arteries (Zachariah et al, 2005). There are some types of the fetal variant circle of Willis, which are complete, partial, transitional, unilateral and bilateral. In a complete FTP whether was unilateral or bilateral, the P1 segment is not visualized on CTA or MRA or does not fill after injection of contrast in a vertebral artery in conventional angiography. A partial FTP whether was unilateral or bilateral, is when the P1 segment is smaller than the PcomA (hypoplastic P1). In transitional posterior circle configuration, the PcomA is as large as the P1 segment

(Van Raamt AF et al, 2006). The bilateral complete FTPs is called the primitive-type embryonic derivation (Kameyama and Okinaka, 1963).

Clinical importance of fetal variants:

The leptomeningeal vessels develop between the anterior (ACA), middle (MCA) and posterior cerebral arteries (PCA) when there is an ischemic insult. They can represent an important connection between the internal carotid artery (ICA) and the vertebrobasilar system. Leptomeningeal collaterals can develop in the majority of circle of Willis configurations. However, in the fetal variant (FTP), makes leptomeningeal collaterals between the ICA and the vertebrobasilar system impossible to develop since both the MCA and the PCA are connected to the internal carotid system and not to the vertebrobasilar system. In this situation an obstruction of the ICA cannot be compensated by the development of leptomeningeal vessels between the PCA and the MCA as both of them are derived from the same vessel and the tentorium prevents cerebellar vessels from connecting to the PCA territory. Bilateral fetal origin implies near 100% dependence on the carotid arteries for cerebral blood supply, with minimal or no dependence on the vertebral arteries for the same, rendering the subject less well-equipped with potential collateral pathways if flow limiting carotid stenosis or occlusion develop (Zachariah et al, 2005). Thus an important consequence of the fetal variant of the circle of Willis could be an increased stroke risk in patients with obstructive arterial disease, therefore patients with fetal type PCA could be more prone to develop vascular insufficiency. This prompted more revision of literatures concerning fetal-type PCA (Van Raamt et al, 2006).

Some authors studied posterior segment of circles of Willis on fetal and neonatal brains, they concluded that variations of the posterior segment were the result of

developmental modification especially with increased functional demands in connection with the rapid growth of the occipital lobes (Van Overbeeke et al, 1991).

Percentages of fetal variants in different studies:

(Van Raamt et al, 2006) Found that in case of partial FTPs, 11–29% of the study participants had a unilateral FTP, and 1–9% a bilateral FTP. When the definition of a complete FTP was used, 4–26% had a unilateral FTP and 2–4% a bilateral FTP. (Zachariah et al, 2005) found that the incidence of unilateral fetal type is around 15%, and hence the incidence of bilateral fetal type cerebral arteries is expected to be significantly lower. (Eftekhar et al, 2006) found that the diameters of 26% of P1s on the right side and 28% on the left side (average 27%) were smaller than the PcomAs on the same side (FTP).

B- Carotid vertebrobasilar anastomosis and trigeminal variant.

Carotid-vertebrobasilar anastomosis they are anomalies represent persistent embryonic circulatory patterns channels between embryonic aorta (which eventually form the caudal carotid artery) and the paired longitudinal neural arteries (which eventually form the basilar and the vertebral arteries) may fail to regress, resulting in a congenital carotid basilar or vertebral anastomosis (Reynolds et al, 1980).

The most common carotid-basilar anastomosis is the persistent primitive trigeminal artery PTA. This anomaly is present in 0.1% to 0.6% of cerebral angiograms. Bilateral PTAs is extremely rare (Okada et al, 1992). A PTA arises where the ICA exits the carotid canal and enters the cavernous sinus. It then runs backwards along the trigeminal nerve or crosses over or through the dorsum sellae before joining the

basilar artery (Schuierer et al, 1990 and Ohshiro et al, 1993). A PTA is usually associated with small PcomAs and vertebral arteries and a hypoplastic basilar artery caudal to the anastomosis (Osborn, 1980). PTAs have an increased incidence of intracranial aneurysms and vascular malformation (Fortner and Smoker 1988). Congenital absence of one or both ICAs is rare (Quint and Silbergleit 1992). Inersellear intercarotid communicating arteries are common if one ICA is absent. Although this is an uncommon anomaly, it is important to identify its presence when considering cerebrovascular or trans-sphenoidal surgery (Udzura et al, 1988).

Variations and hemodynamics:

Many studies demonstrated that the circle of Willis is one of the most variable parts of the human vascular system. From an ontogenetic point of view, indicating a random variability of the cerebral vessels (Padget, 1944; Padget, 1948 and Van der Zwan et al, 1993). The collateral potential of the circle of Willis is believed to be dependent on the presence and size of its component vessels (Schomer et al, 1994; Mull et al, 1997 and Hartkamp et al, 1999). The peripheral resistance of the major cerebral arteries and consequently the flow patterns both of which are hemodynamic factors, which are suggested to predominantly determine the form and size of the cerebral vascular system (Van der Zwan et al, 1993). On the contrary deterioration of cerebral hemodynamic induces diameter enlargement as a necessity for survival, this process of diameter enlargement is also known as remodeling (Hartkamp and Van der Grond, 2000).

Fluid dynamic mathematical models have been developed to study the effect of collateral artery diameter on cerebral blood flow after ICA stenosis or occlusion (Cassot et al 1995; Viedma et al, 1997). A threshold of 1 mm to define hypoplasia or inadequacy of collateral vessels has been widely used in anatomic studies

(Battacharji et al, 1967; Gomes et al, 1986 and Tulleken and Luiten, 1987). The collateral artery threshold diameters allowing for cross flow through the primary collateral arteries of the circle of Willis is between 0.4 and 0.6 mm (Hoksbergen et al, 2000). Thus the artery was defined as normal if its diameter was at least 0.5 mm. The artery was defined as compromised if its diameter was less than 0.5 mm. The artery was defined as missing if it was not present in either the permanent cast or the angiography (Merkkola et al, 2006). The smallest luminal diameter allowing for cross-flow through the AcomA was 0.4 mm (Cassot et al, 1995 and Dickey et al, 1996). In all model simulations of the circle of Willis that were developed to investigate the function of the circle of Willis as anastomosing system, the relation between peripheral resistance and the size of the segments of the circle was also negated (Kramer, 1912; Kufahl and Clark, 1985 and Hillen et al, 1986).

Moreover, in patients with ICA occlusion and a well functioning AcomA, the collateral supply from the PcomA to the deprived hemisphere fell to zero when its diameter was set at levels, 0.5 to 0.6 mm (Dickey et al, 1996). Patients with unilateral ICA occlusion demonstrated a high prevalence of collateral flow through the anterior circle of Willis and significantly increased diameters of the communicating channels. Patients with bilateral ICA occlusion relied on collateral flow via the posterior circle of Willis and demonstrated a bilateral increase in posterior communicating artery diameters (P,0.05) (Hartkamp et al, 1999). In clinical studies, an increased risk of ischemic cerebral infarction after ICA occlusion (Schomer et al, 1994) and an increased risk of brain stem ischemia after basilar artery occlusion (Steinberg et al, 1993) have been associated with PcomA diameters 0.1 mm. Theoretically the circulation to the left hemisphere would be sufficient in most patients undergoing an operation of the aortic arch. In 14% of subjects at a threshold of 0.5 mm and 17% at a threshold of 1mm the circulation would have been insufficient (Merkkola et al, 2006).

Only in a minority of studies was a threshold of 0.5 mm used to define hypoplasia of collateral arteries (Fetterman and Moran, 1941; Kamath, 1981). The varying definitions of hypoplasia of circle of Willis collaterals and the different populations studied have resulted in a large variability of anomalous or incomplete circles of Willis throughout the literature (Baptista, 1964; Fisher, 1965 and Battacharji et al, 1967). Hence the term hypoplasia may be reserved for those vessels that cannot supply collateral flow (Hoksbergen et al, 2000).

Variations and aneurysms:

It has long been suspected that variations in the circle of Willis may play some role in the development of cerebral aneurysms (Riggs and Rupp, 1943; Alpers and Berry, 1963; Stehbens, 1963). Some studies have found a correlation between cerebral aneurysms and certain variations of the circle (Horikoshi et al, 2002). Some authors (Wilson et al, 1954) reported that atypical configurations of the circle of Willis were present in 95% of their aneurysm series, and others reported the incidence to be 79% (Eftekhar et al, 2006). Many studies showed that the incidence of variations was significantly higher in the aneurysm series than in the control circles without aneurysm. There was a definite correlation between asymmetric proximal segments of the anterior cerebral artery and aneurysms of the anterior communicating artery, and a tendency to correlation was found in the case of asymmetric posterior communicating arteries and aneurysms on the internal carotid artery-posterior communicating artery junction (Kayembe et al, 1984).

Many figures of percentages of this association were resulted from different studies like, 77% of aneurysms in AcomA occurred on the thicker A1 side or midline and 33% on the thinner A1 side. In the case of Asymmetrical PcomA, 64% of aneurysms were located in AcomA and 36% in ICA-PcomA. All aneurysms in

ICA-PcomA occurred on the thicker side. Statistical comparison between the two ratios showed a significant correlation between Asymmetrical ACA and AcomA aneurysms (P < O.O5). A tendency to correlation was found in the case of Asymmetrical PcomA and ICA PcomA aneurysms (Kayembe et al, 1984). In the study of (Alpers and Berry, 1963), the incidence of atypical circles in the aneurysm and control series were 63 and 48% respectively.

A definite correlation is found between Asymmetrical ACA and aneurysms in AcomA ($p < 0.05$) (Wilson et al, 1954; Stehbens, 1963 and Cassot et al, 1995). Asymmetrical PcomA and ICA-PcomA aneurysms showed a tendency to correlation ($p < 0.10$) (Cassot et al, 1995) which is also supported by results of (Kayembe et al, 1984). aneurysms occurred always on the thicker side of the ICA-PcomA junction in the case of Asymmetrical PcomA, while in the case of Asymmetrical ACA, such a difference was not clear (Montake et al, 1976). The incidence of variations in the circle of Willis was shown to be significantly higher in the aneurysm series than in the control (Kayembe et al, 1984).

This clear correlation between the variations of the circle and cerebral aneurysms, leads one to the assumption that the variations are a factor in the occurrence of aneurysms. Although the association of variations and aneurysms had been used as an argument in favor of a congenital theory of aneurysmal development (Padget, 1944). It should be interpreted in terms of the hemodynamic stress caused by variations (Cassot et al, 1995). Finally, although it is still controversial to (El Khamlichi et al, 2001; Kim et al, 2003) there are studies that demonstrate the existence of ethnic differences in the incidence of intracranial aneurysms (Tay et al, 1971 and Nogueira, 2002).

Variations and stroke:

The primary purpose of the vascular circle is to provide anastomotic channels if one vessel is occluded, where it enables inter-hemispheric flow through the anterior communicating artery and in two directions through the two PcomAs. Hence the collateral circulation in the brain is important for maintaining a sufficient level of cerebral blood flow in case of obstructive disease in the main afferent arteries (Liebeskind, 2003). It functions as an anastomosis and offers a potential shunt under abnormal conditions such as might occur during an occlusion or spasm (Rogers and Lambert, 1946 and Kamath, 1981).

Patient with unilateral occlusive disease of ICA shows 88% of complete anterior circle in comparison with 68% of complete anterior circle in normal population and demonstrated a statistically significant diameter increases in the anterior channels. This indicates that the AcomA is of more importance in unilateral ICA occlusion. Whereas in bilateral ICA occlusion patients demonstrated a statistically significant diameter increases in the PcomA bilaterally, hence conclude the PcomAs play a dominant role in bilateral ICA Occlusions (Hartkamp and Van der Grond, 2000).

Patients with carotid lesions have a significantly (P50.02) higher percentage of entirely complete circle configurations than did controls. The percentage of complete circle of Willis configurations was highest on the symptomatic side (P50.001). It is likely that the inclusion of survivors of ICA obstruction may have resulted in the selection of a subgroup from the entire population with ICA lesions, favoring patients in whom a complete circle configuration and/or larger diameters were a pre-existent feature (Hartkamp and Van der Grond, 2000).

Many studies demonstrate higher percentages of complete circles in living patients with ICA obstruction compared with the autopsy populations as described by

(Alpers and Berry, 1963 and Riggs and Rupp, 1963). The differences in circle of Willis configurations found in these study populations may therefore indicate the protective role of the circle of Willis in survivors of ICA occlusive disease (Hartkamp et al, 1999).

In case of FTPs, the ICA covers a larger area to provide with blood than in the normal non-FTP configuration of the circle of Willis. It is probable that patients with ICA obstruction and a full FTP more often encounter ischemic problems than patients with a normal circle, in which the PcomA is preserved and leptomeningeal vessels can develop between the carotid and the vertebrobasilar system. Patients who also have a missing contralateral A1 segment, thus having to feed the area of the ACAs, an MCA and a PCA with one ICA could be even more at risk. Therefore patients with fetal-type PCA could be more prone to develop vascular insufficiency (Van Raamt et al, 2006).

Where possible variations can include underdeveloped or completely absent blood vessels. While an individual with one of these variations may suffer no ill effects under normal circumstances, the reduced ability for collateral blood flow via the circle of Willis compounded with certain pathological conditions can present a health risk, in particular the possibility of suffering an ischemic stroke. The importance of understanding the sensitivity of cerebral perfusion on circle of Willis geometry arises from the fact that in the western world stroke is the third largest cause of death after heart disease and cancer, and is the largest cause of serious long-term disability (Petty et al, 2000). There is thus a growing importance in the understanding of factors that increase the risk of stroke and how blood is distributed throughout the brain (Moore et al, 2006).

The relative risk of hypoperfusion infarctions is significantly higher in cases with a nonfunctioning AcomA (Miralles et al, 1995). However, the presence of a large ipsilateral PcomA was the only feature that correlated significantly with the

absence of a watershed infarction (Schomer et al,1994). In clinical studies, an increased risk of ischemic cerebral infarction after ICA occlusion (Schomer et al, 1994) and an increased risk of brain stem ischemia after basilar artery occlusion have been associated with PcomA diameters 0.1 mm (Steinberg et al, 1993).

An incomplete circle of Willis increases the risk of transient cerebral ischemia during carotid clamping in patients with contralateral ICA occlusion (Lee et al, 2004). Which led to more efforts to be taken to improve brain protection in aortic arch reconstructions, and despite of that a considerable number of patients are still at risk for neurologic injuries. These can be either global or focal in nature. Global insult may be reflective of generalized ischemia or attributable to inadequate or absent perfusion (Spielvogel et al, 2005). On the other hand patients with variants of the circle with efficient collateral circulation have a lower risk of transient ischemic attack (TIA) and stroke than that of patients without such collaterals (Henderson et al, 2000).

Previous studies have proposed correlation between variants of the circle of Willis and some cerebrovascular diseases (Kayembe et al, 1984 and Horikoshi et al, 2002) and also differences in incidence of these diseases in different populations (Ramamurthi, 1969; Tay et al, 1971; So et al, 1979; Nogueira 2002 and Flaherty et al, 2005;). It seems that different distribution of variations of the circle of Willis may partially explain the different incidence of some of the cerebrovascular diseases in different ethnic or racial groups. The incidence of ischemic stroke is different among different populations, especially blacks and Hispanics compared with whites (White et al, 2005).

The study of variations in the anatomy of the cerebral arterial circle may thus partially explain differences in the incidence of some of the cerebrovascular diseases in different ethnic or racial groups (Eftekhar et al, 2006).

Variations and sex:

Some studies found that there is no statistically significant difference between the frequency of variation between two sexes (Macchi et al, 1996 and Alawad et al, 2009); Other studies where the authors could find sex linked differences in anatomical variations of circle of Willis and a statistically significant correlation between sex-linked differences and aneurysm distribution (Horikoshi et al, 2002).

Variations and ethnicity:

The incidence of ischemic stroke is different among different populations, especially blacks and Hispanics compared with whites, differences in incidence of these diseases in different populations is well noticed (Flaherty et al, 2005; White et al, 2005), thus the study of variations in the anatomy of the cerebral arterial circle may partially explain differences in the incidence of some of the cerebrovascular diseases in different ethnic or racial groups. Occurrence of such variants in different ethnic or racial groups has not been well compared yet (Eftekhar et al, 2006). some studies have proposed correlation between variants of the cerebral arterial circle and some cerebrovascular diseases (Kayembe et al, 1984; Henderson et al, 2000; Horikoshi et al, 2002 and Hoksbergen et al, 2003).

(Eftekhar et al, 2006) in his study of the anatomical variations in the cerebral arterial circle of the Iranian males found they were not significantly different to those of more diverse populations and thus conclude there is no evidence suggesting that the distributions of the variations of cerebral arterial circle differ in different populations.

Variations and handedness:

Many studies find different figures of percentages of the variations in the circle of Willis between the right and left circulations, these figures might explain the dominance of one hemisphere over the other. Where some studies (Orlandini, 1970; Warwick and Williams, 1973) found that the majority of cases show a greater length, and a smaller diameter in the right half of the circle. Hence, blood flow will be better in the left half, and thus the left hemisphere has on the whole a better blood supply. This is in keeping with the dominance of the left hemisphere and the commoner occurrence of right handedness, whereas earlier observations by (Windle, 1887 and Mitterwallner, 1955) suggested that the blood supply to the left half of the brain was on the whole, less complete than that to the right side.

Abnormal narrowing of vessels was a commoner occurrence on the right side than on the left side. This may be related in some way to the need for a better blood supply to the left hemisphere. The only artery which showed a smaller average diameter on the left side, coupled with a higher incidence of abnormal diameters on the left side, was the posterior communicating artery. Since a significant inverse relationship existed between the diameters of the posterior communicating and posterior cerebral arteries of the same side, a smaller posterior communicating on the left would be associated with a larger posterior cerebral on that side, thus ensuring better blood supply to the left hemisphere from two sources, thus the left cerebral hemisphere appeared to enjoy a better blood supply (Kamath, 1981).

Aplasia of PcomA was found to be 4% on right, 3% on left and 3% on both sides (Eftekhar et al, 2006). Other studies of circle of Willis at males (Fawcett E and Blachford, 1905) have reported absence of right PcomA in 2.2%, left PcomA in 0.85% and absence of both PcomAs never observed in males. In (Windle, 1887)

study, right PcomA was absent in 4.5%, left PcomA was absent in 6.5% of cases and both PcomAs were absent in 1.5% of cases.

In other study of (Alawad et al, 2009) aplasia of the PcomA 3.5% & 0.7% on the left and right side respectively and also anomalies of ACA, ICA were found to occur more on the left, which is in disagreement with the previously reported literature where aplasia of the PcomA occurred more on the left side.

Theories explaining variations in the circle:

Many theories were suggested to explain the variations on this arterial circle where (Milenkovic et al, 1985) based on previous works and his own observations postulated that genetic factors are probably responsible not only for the development of the circle but also for its caliber, also (Vasovic et al, 2002) believed that the arterial variations and abnormalities have fetal characteristics and that they are the result of a genetically established pattern, others who studied posterior segment conclude that variations of this segment were the result of postnatal developmental modification (Van Overbeeke et al, 1991) whereas (Lazorthes et al, 1979) suggested that the variation of the segment calibers was due to the amplitude of the neck movements in the later years of life and (Hillen et al, 1986) indicated that variation of the circle is based on a delicate hemodynamic tuning of all segments and that the relations between diameters of segments exists without reference to flow changes in the afferent arteries during movement of the neck.

These studies demonstrated that the circle of Willis is one of the most variable parts of the human vascular system. From an ontogenetic point of view, indicating a random variability of the cerebral vessels (Padget, 1944; Padget, 1948 and Van der Zwan, 1993).

Material

&

Methods.

Material and methods

A-Subjects:

86 Libyan subjects were collected from cases scheduled to undergo MRI brain or contrast enhanced CT brain in the Diagnostic Radiology Department in Triploi Medical Center (T.M.C) and Al Khadra Hospital. Patients included were with no history of cerebrovascular disease, DM nor HTN, in the period of September, October and November 2010. Age ranges from 10 - 70 years (mean 41.4 years), subjects were divided into four groups , each group interval is 15 years. Among the subjects of the study only two subjects were left handed.

Out of 86 subjects included in the study, 47 subjects were females (54.7%) and 39 subjects were males (45.3%) as shown in table 1 and figure 1.

Table 1: Distribution of cases according to sex.

Sex	No.	%
Male	39	45.3
Female	47	54.7
Total	86	100

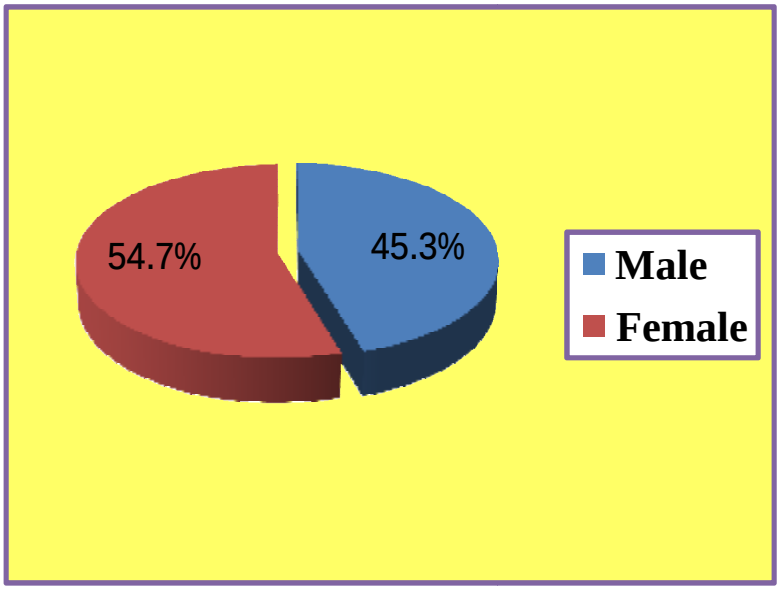

Fig. 1: Distribution of cases according to sex.

The subjects of this study were categorized into 4 groups according to their age, where each group interval is 15 years as shown in table 2 and figure 2. Figure 3 show sex cross tabulation with age groups.

Table 2: Distribution of cases according to age group.

Age group	No	%
10-24	15	17.4
25-39	28	32.6
40-54	17	19.8
55 or more	26	30.2
Total	86	100.0

Mean =41.9 years. SD = 17.2 years. Median =39.5 years.

Mode =35years. Minimum age = 10 years. Maximum age = 70years.

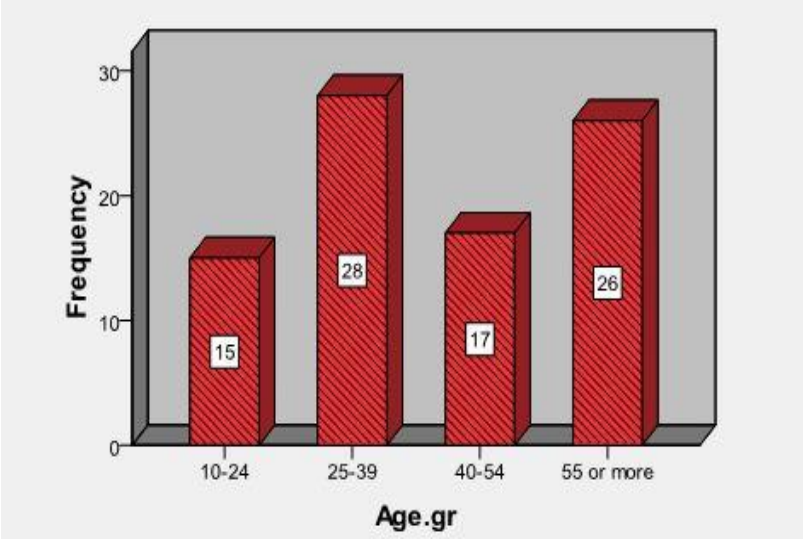

Fig.2: Distribution of cases according to age group.

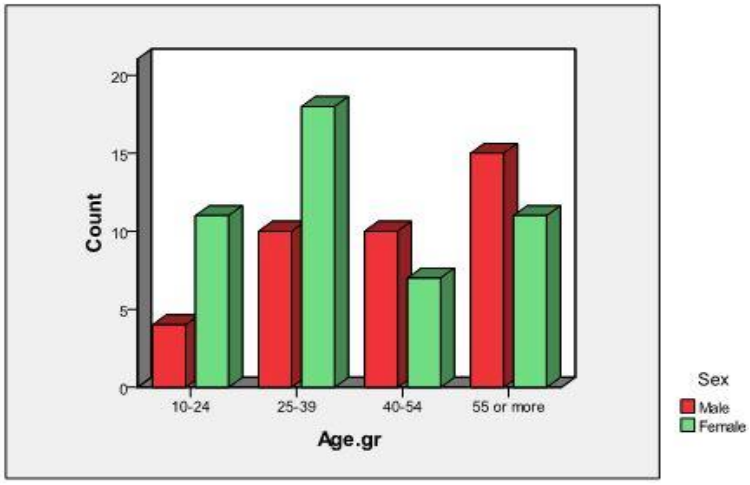

Fig. 3: Sex-age groups cross tabulation.

The eighty six subjects of this study underwent either CTA or MRA, 50 cases (58.1%) underwent MRA and 36 cases (41.9%) underwent CTA as shown at table 4 and figure 4.

Table 3: Distribution of cases according to modality.

Modality	No.	%
MRA	50	58.1
CTA	36	41.9
Total	86	100

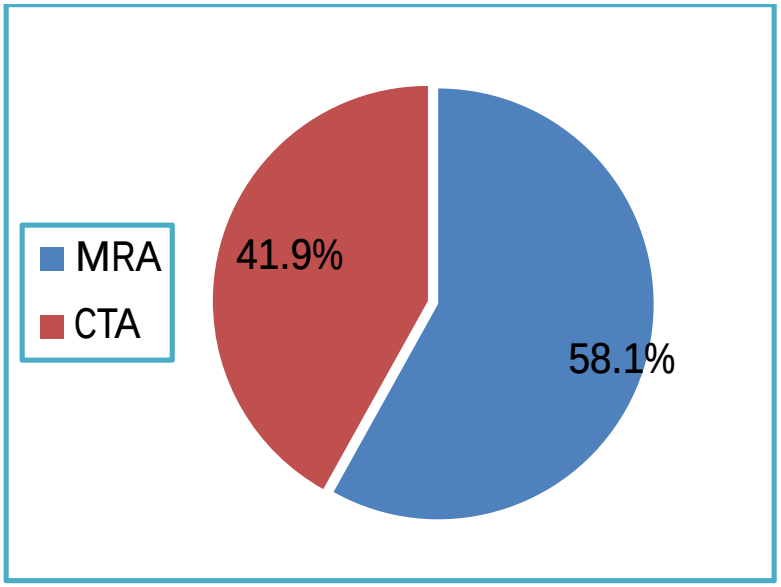

Fig. 4: Distribution of cases according to modality.

According to the handedness, 84 0f cases were right handed and only 2 cases were left handed as shown at table 4 and figure 5.

Table 4: Distribution of cases according to handedness.

Handedness	No.	%
Right	84	97.7
Left	2	2.3
Total	86	100

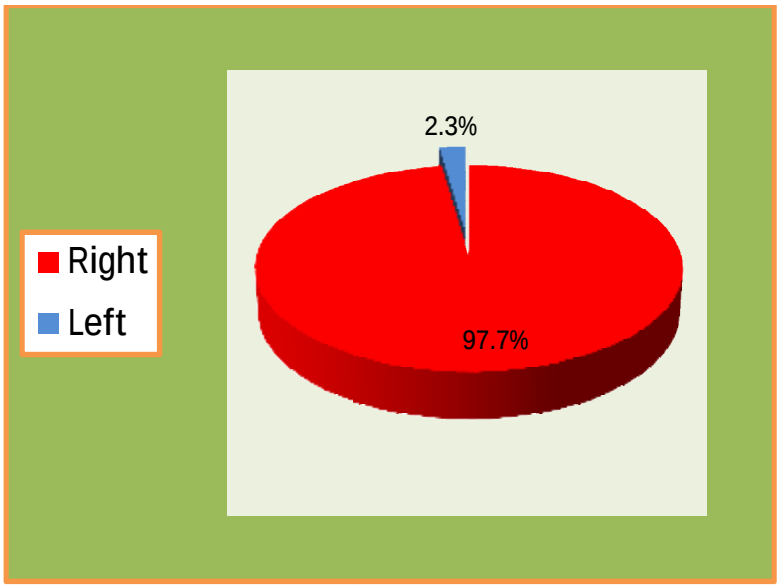

Fig. 5: Distribution of cases according to handedness.

B- Modalities and techniques:

1- Magnetic Resonance Angiography (MRA):

The three-dimensional time-of-flight (3D-TOF) MR Angiograms of the circle of Willis were obtained with the sequence of spoiled gradient-recalled acquisition (SPGR) using a 1.5-tesla MR scanner (GE Medical Systems). Imaging parameters were 45 msec repetition time (TR), 4.9 msec echo time (TE), 20˚ flip angle, 256-256 matrix in a 14cm field of view, and 42mm slab with 60 serial axial slices of 0.7mm thick. The total imaging time, including acquisition of the survey image and positioning, was approximately 10 minutes. These axial source images were post-processed by the maximum intensity projection (MIP) algorithm to produce twenty projections rotating about the section axis and one axial image (projection images). All component vessels of the circle of Willis were accessed by measuring the diameter on the individual MIP images. Kodak Dryview 8700 Laser imager and CODONICS HORIZON GS Multimedia imager were used. Whenever there is a doubt in determining the diameter of one vessel due to overlapping vessels in the MIP images, the TOF source images are then reviewed on the advanced workstation (HP xw8400) with Multisync LCD 1990 sxi screen.

2- Computed Tomography Angiography (CTA):

The three dimensional CTA angiograms of circle of Willis were obtained by using a 64 Slice PHILIPS CT with BRILLIANCE 190P screen. Imaging parameters were: Helical set, KV 120, mA 250, Rotation time 0.5 sec., Slice thickness 0.5mm, soft algorithm, Scanning time 40 sec., and Scanning delay of 16 sec., Pitch 0.9. The images were underwent post-processing multiplanar reconstructing in BRILLIANCE 190P workstation with slice thickness up to 20mm by MIP (maximum intensity projection) and VRT (volume rendering technique) techniques with bone removal property and

clipping technique. The scan was starting from the aortic arch and extending to vertex in cases of brain CT scans and up to frontal air cell level in cases of neck CT scans. The nonionic contrast Guerbet Labitridol XENETIX 300mg/ml IV contrast, was injected into the Cubital vein by MeDRaD Stllant CT injection system (Dual head), With 80 ml contrast and 40 ml saline (total 120 ml) with injection rate 4cc per second.

According to the vessel size seen on 3D angiograms, the component vessels constituting circle of Willis were regarded as normal, hypoplastic or absent. Vessels, which were visualized as continuous segments of at least 0.8mm, were considered present (normal). Those smaller in diameters than the half of their contra-lateral corresponding were considered as hypoplastic. Vessels, which were un-visualized or visualized as non-continuous segments, were considered as absent and their diameters were regarded as zero. The anterior communicating artery was considered as normally patent if the segment of anterior communicating artery was clearly visualized or junctions of A1 and A2 segments were in close contact and therefore were not separable with each other on 3D angiograms. Hypoplastic A1 segment of anterior cerebral artery was defined when the diameter of the artery was less than half of the contra-lateral A1 segment. In a case of posterior communicating artery or P1 segment of posterior cerebral artery, hypoplasia was defined when the diameter of the artery was less than half of contralateral P1 segment or posterior communicating artery, respectively.

Fetal type posterior circle of Willis were considered as complete if P1 was completely absent, partial if P1 is hypoplastic and a transitional type posterior circle of Willis, if both P1 and PcomA were of the same caliber. The prevalence of each anatomic variant was calculated. And statistics were done.

C-Statistical analysis:

Results were expressed as mean ± standard deviation (SD) or number and (%). Categorical data was compared using Chi-square test. Statistical analysis was performed with the aid of the statistical package for the social sciences (SPSS) computer program (version 18 windows). P value <0.05 was considered significant.

Results.

Results

Results:

The collected cases were studied and were compared with the control figure of the circle resembled in figure 6; and were classified according to their configuration into a classical group where all of the vessels are present with the expected caliber and morphology, and a variant group which show one or more variations at presence, caliber or morphology of the vessels of the circle as shown in table 5 and figure 8.

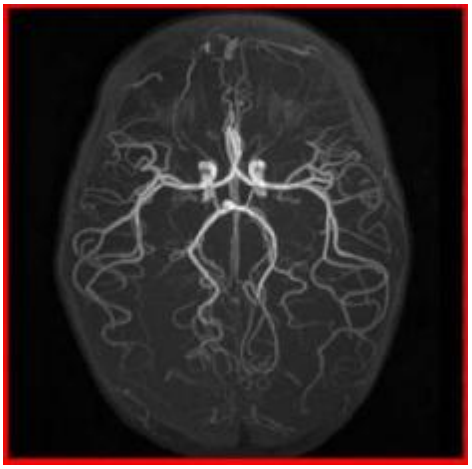

Fig. 6: 3 D MRA showing classical circle of Willis (control). Malamateniou C et al**,** 2009.

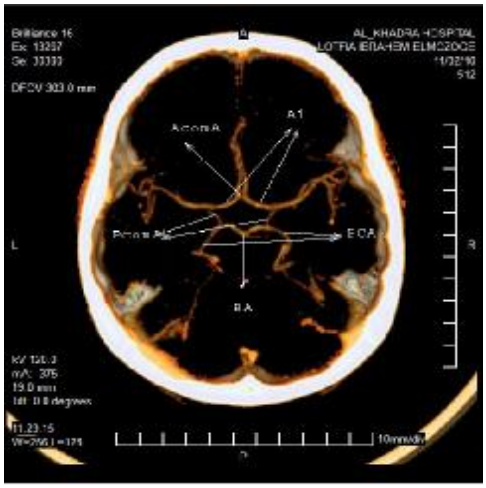

Fig.7: CTA showing a classical circle of Willis.

Table 5: Categorization of subjects according to circle configuration.

Circle configuration	No	%
Classical	31	36.0
Variant	55	64.0
Total	86	100.0

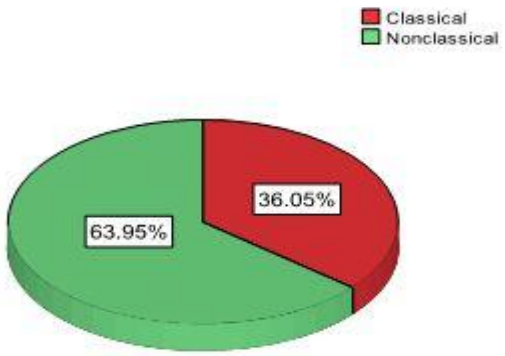

Fig. 8: Categorization of subjects according to circle configuration.

Variations - age groups cross tabulation:

Cases population were grouped into 4 age groups and the percentages were calculated between the classical cases and variant cases among the age groups. Which show 10-24 years age group 53.3% of cases were variants, 25-39 years age group 21.4% of cases were classical, 40-54 years age group 52.9% of cases were not classical, and in 55-70 age group 38.5% of cases were classical with non statistically significant p value; as shown at table 6 and figure 9.

Table 6: Distribution of cases (cross tabulation) according to age group and circle configuration.

Age group / years		Configuration		Total
		Classical	Non classical	
10-24	Count	7	8	15
	% within Age gr.	46.7%	53.3%	100.0%
25-39	Count	6	22	28
	% within Age gr.	21.4%	78.6%	100.0%
40-54	Count	8	9	17
	% within Age gr.	47.1%	52.9%	100.0%
55 or more	Count	10	16	26
	% within Age gr.	38.5%	61.5%	100.0%
Total	Count	31	55	86
	% of Total	36.0%	64.0%	100.0%

X^2 = 9.706 df = 6 p =0.138 (Non-significant).

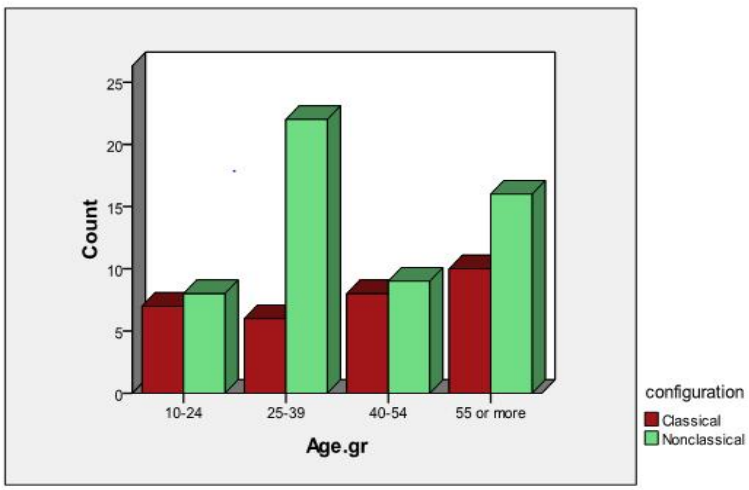

Fig. 9: Distribution (cross tabulation) of subjects according to age group and circle configuration.

Variations - sex cross tabulations:

Cases were (cross tabulated) distributed according to sex and configuration of the circle, where among 39 male cases 13 cases (33.3%) show classical configuration of the circle and 26 cases (66.7%) show sort of variation, and among 47 female cases 18 cases (38.3%) were classical and 29 cases (61.7%) were variant. Chi square testing was applied with no statistical significance was resulted. As shown in table 7 and figure 10.

Table 7: Distribution of cases according to sex and radiological finding.

Configuration of the circle	Male		Female	
	No.	%	No.	%
Classical	13	33.3	18	38.3
Variant	26	66.7	29	61.7
Total	39	100	47	100

$X^2 = 0.063$ df = 1 p =0.801 (Non-significant).

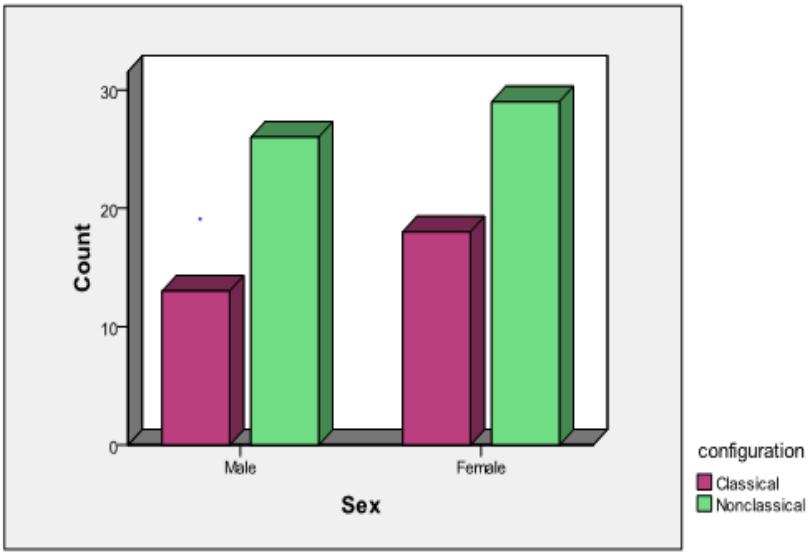

Fig. 10: circle configuration cross tabulated with sex of subject.

AcomA was missed in 2.3% of all cases, 2.5% of males and 2.1% of female cases. Duplicated in 2.3% of the whole cases, 4.2% of female cases and no duplication was seen in males. AcomA aneurysm observed in 1.16% of all cases, 2.5% of male cases and not seen in females. Regarding ACA (A1 segment); right A1 was missed in 2.3% of the entire cases, 5.1% of male cases and no missing was noted in female cases. Right A1 hypoplasia was seen in 2.3% of all cases, 5.1% of male cases and not seen in females. Left A1 was absent at 3.5% of total cases, 5.1% of male cases and 2.1% of female cases; it was hypoplastic at 7% of all cases, 7.7% of male cases and 6.3% of female cases. One male case shows right A1 arising from right MCA. Hypoplastic vertebral artery seen at one male case on the right side. Left P1 gives left superior cerebellar artery in one female. PcomA was missed bilaterally in 31.4% of all cases, 28.2% of male cases and 34.04% of female cases, was missed on the left side in 10.5% of total cases, 7.7% of male cases and 12.7% of female cases, and was absent on the right side in 4.6% of all cases, 5.1% of male cases and 4.2% of female cases. Regarding PCA (P1 segment) it was missed bilaterally in 2.3% of the entire cases, 2.5% of male cases and 2.1% of female cases. Bilateral hypoplasia of (P1) was seen in 1.16% of all cases, 2.5% of male subjects and not observed in female subjects. Right P1was missed in 4.6% of all cases, 2.5% of male cases and 6.3% of female cases. Left P1 was missed in 3.5% of all cases, 2.5% of male cases and 4.2% of female cases. Right P1 hypoplasia noted in 2.3% of all cases, 4.2% in female cases and not observed in males. Left P1 hypoplasia was found in 2.3% of all cases, 2.5% of males and 2.1% of females. Distribution of variations according to sex and type of variation present shown in table 8.

Table 8: Distribution of cases according to sex and variation at circle.

Type of abnormality	Male		Female		Total	
	No.	%	No.	%	No.	%
Bilateral missing of PcomA	11	28.2	16	34.04	27	31.4
missed LPcomA	3	7.7	6	12.7	9	10.5
missed RPcomA	2	5.1	2	4.2	4	4.6
Bilateral missing of P1	1	2.5	1	2.1	2	2.3
Bilateral hypoplasia of P1	1	2.5	0	0	1	1.16
missed RP1	1	2.5	3	6.3	4	4.6
Hypoplastic RP1	0	0	2	4.2	2	2.3
missed LP1	1	2.5	2	4.2	3	3.5
Hypoplastic LP1	1	2.5	1	2.1	2	2.3
Missing LA1	2	5.1	1	2.1	3	3.5
Hypoplastic LA1	3	7.7	3	6.3	6	7
Missing RA1	2	5.1	0	0	2	2.3
Hypoplastic RA1	2	5.1	0	0	2	2.3
AcomA aneurysm	1	2.5	0	0	1	1.16
AcomA duplicated	0	0	2	4.2	2	2.3
Missing AcomA	2	5.1	1	2.1	3	3.5
Basilar artery hypoplastic	1	2.5	1	2.1	2	2.3
Total	34	100	41	100	75	100

Total No of variant cases more than the real percentage of non classical cases because more than one variant might be seen in the same subject.

Variations in the anterior circle:

Anterior circle of Willis (AcomA and both side A1) was studied and variations were recorded where 77.9% of cases showed classical configuration of the anterior circle as in figure 11, 22.1% of cases show variations in the anterior circle of Willis as shown in table 9 and figure 12, out of this 22.1% variant cases 10.4% of variations occurs in the LA1, 4.6% occurs in RA1 and 7% seen at AcomA; with a higher male predominance 30.7% in comparison with 14.9% of female cases.

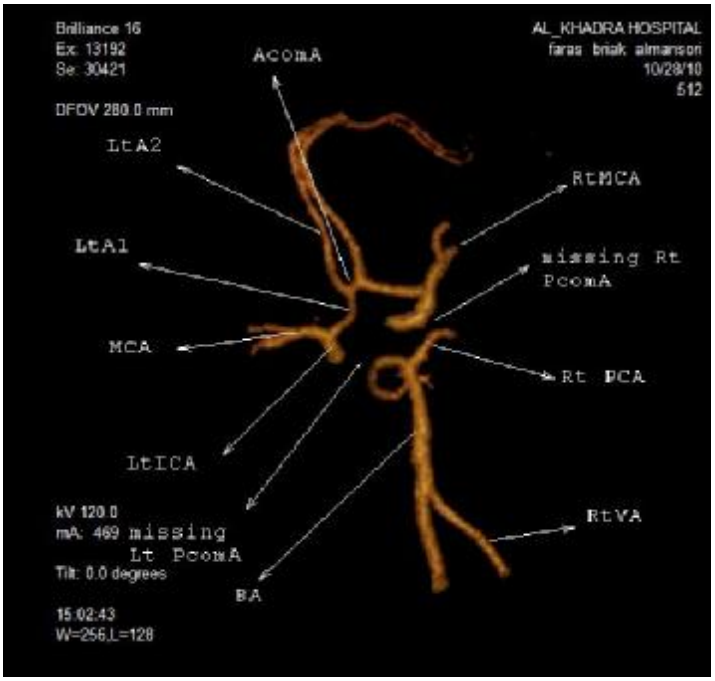

Fig. 11: CTA showing bilateral missing PcomA with classical anterior circle.

Table 9: Configuration of anterior circle of Willis.

Anterior circle	No	%
Classical	67	77.9
Variant	19	22.1
Total	86	100.0

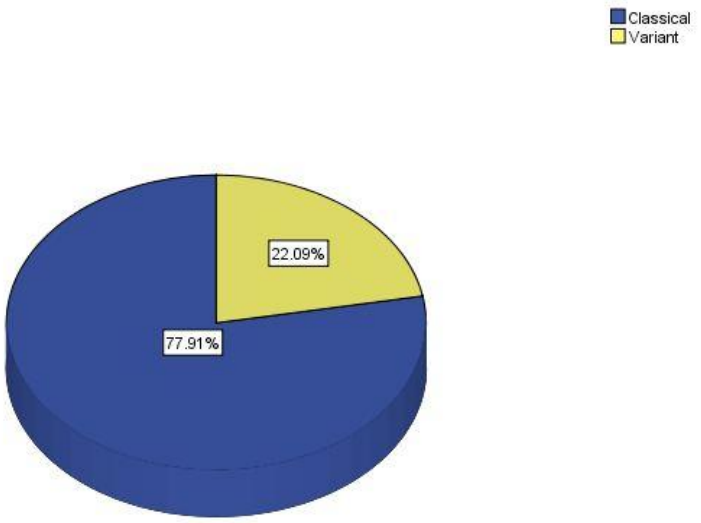

Fig. 12: Configuration of anterior circle of Willis.

Variations in the AcomA:

Anatomy of AcomA was studied where 93% of subjects demonstrated classical anatomy of AcomA as in figure 11, 3.5% of cases show absent AcomA (fig. 13), 2.3% of cases show duplicated AcomA (fig. 14) and 1.2% of subject show AcomA aneurysm (fig. 15); these variations were summarized in table 10 and figure 16.

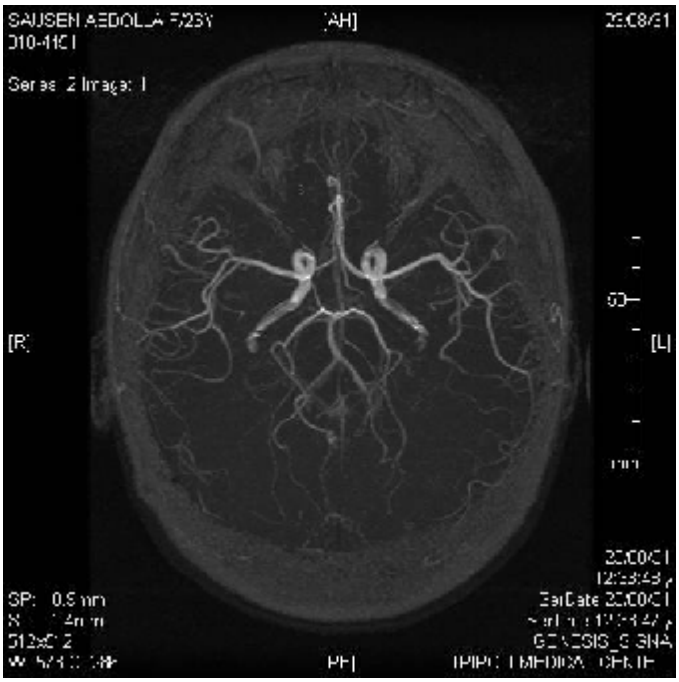

Fig. 13: MRA showing missing LPcomA, missing AcomA.

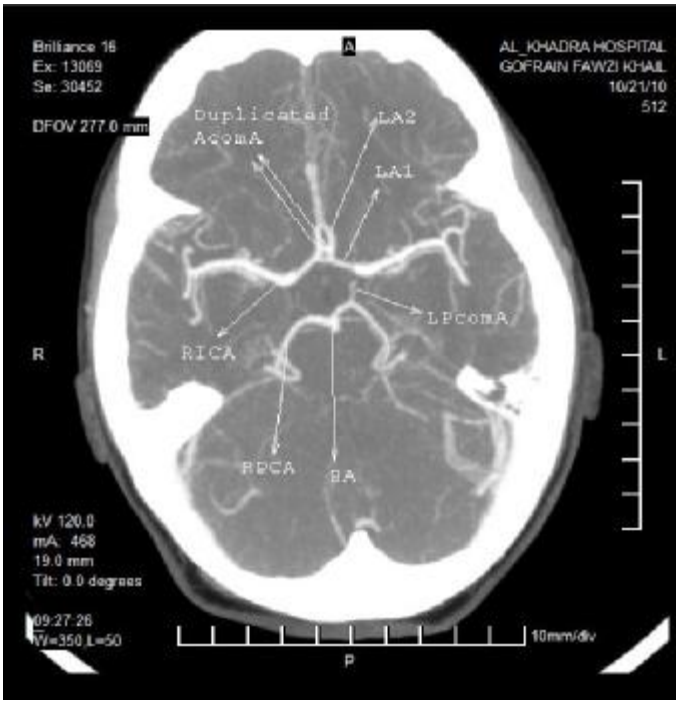

Fig. 14: CTA showing duplicated AcomA and missing RPcomA.

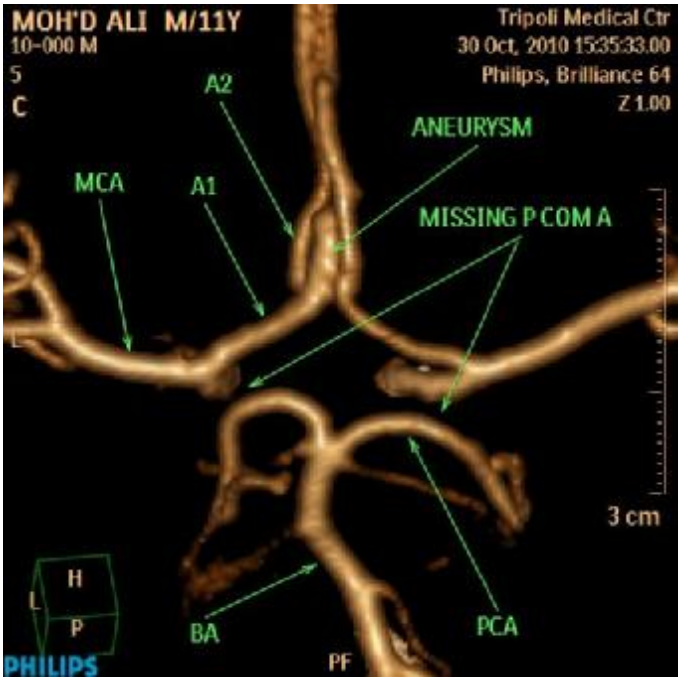

Fig. 15: CTA shows bilateral missing of PcomA, hypoplastic RA1 and AcomA aneurysm.

Table 10: variations in AcomA.

AcomA	No of cases	%
Classical	80	93.0
Absent	3	3.5
Duplicated	2	2.3
Aneurysm	1	1.2
Total	86	100.0

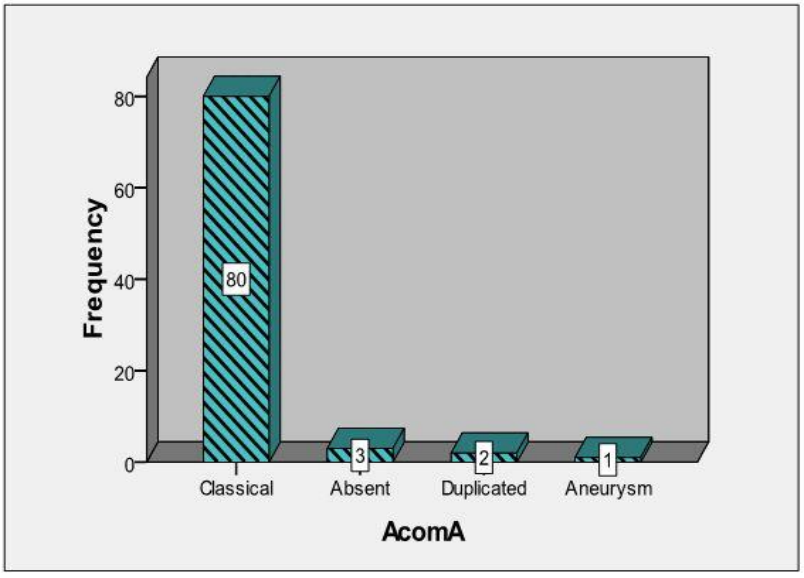

Fig. 16: Variations in AcomA.

Variations in the Rt. A1:

Right anterior cerebral artery RA1 was studied where 95.3% of cases showed classical anatomy of RA1, 2.33% of cases showed hypoplastic RA1 (fig. 17) and in 2.33% of cases RA1 was missing (fig. 18), these variations were summarized in table 11 and figure 19.

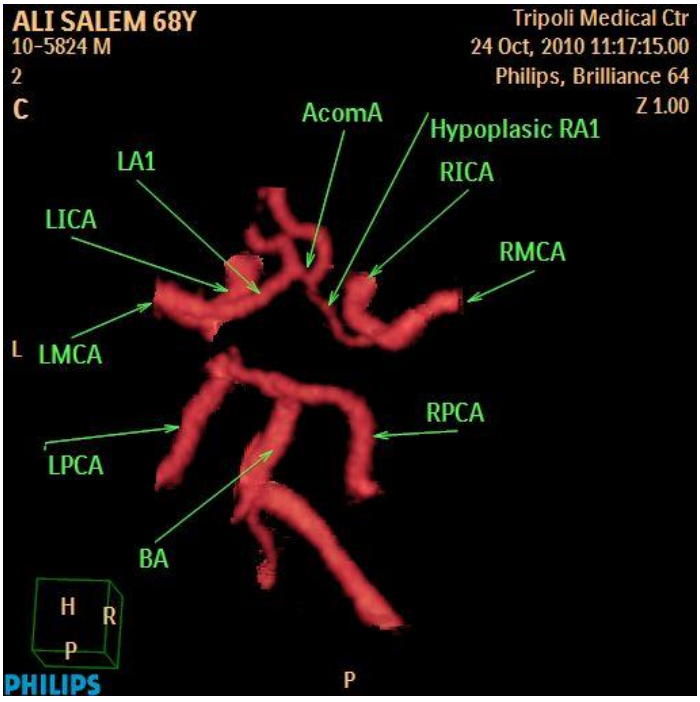

Fig. 17: CTA shows hypoplastic RA1 and bilateral missing of PcomA.

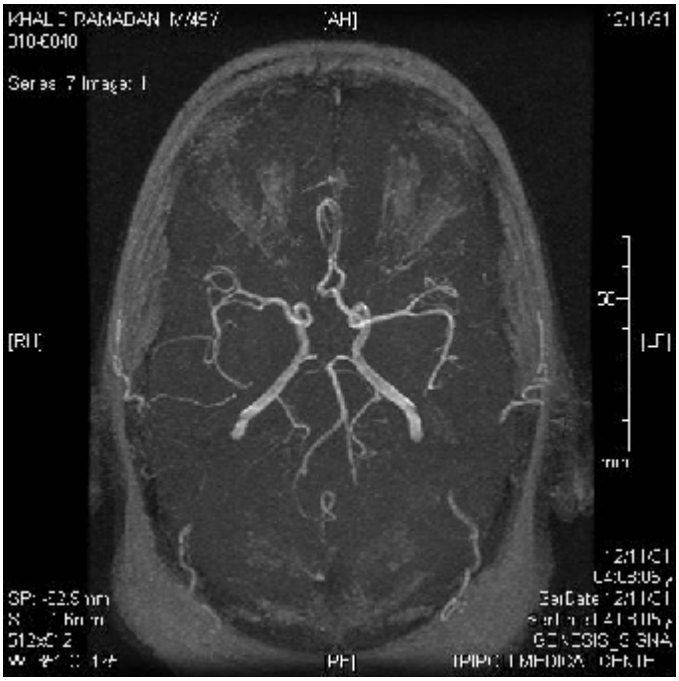

Fig. 18: MRA shows missing RA1, Rt. complete fetal type posterior circle of Willis and missing LPcomA.

Table 11: variation in RA1.

RA1	No	%
Classical	82	95.3
Hypoplastic	2	2.33
Absent	2	2.33
Total	86	100.0

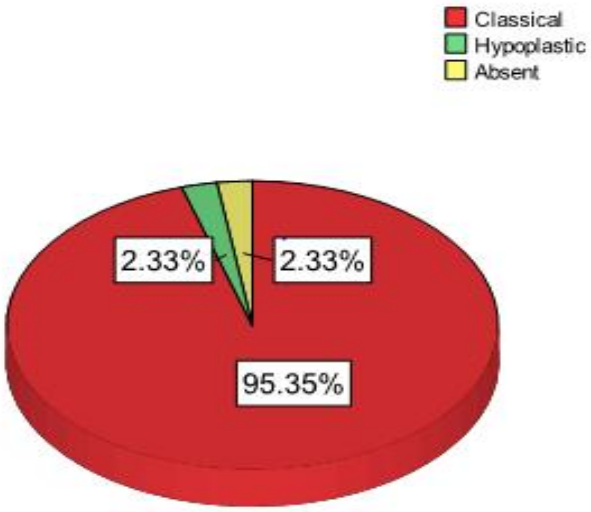

Fig. 19: variations in RA1.

Variations in the Lt. A1:

Left Anterior cerebral artery LA1was studied and found that 89.5% of subject show classical anatomy of LA1, 7% were hypoplastic (fig. 20) and in 3.5% of cases LA1 was missing (fig. 21), these variations were demonstrated in table 12 and figure 22.

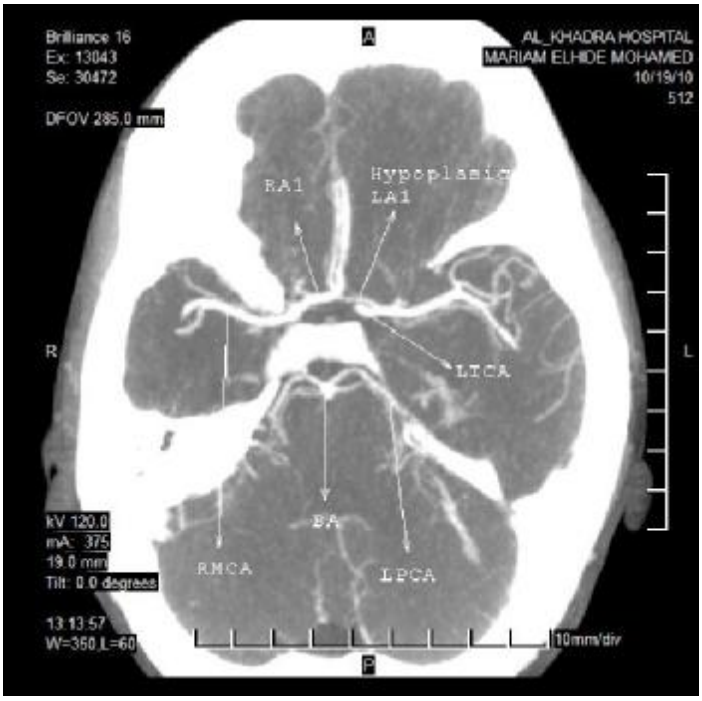

Fig. 20: CTA shows hypoplastic LA1 and bilateral missing of PcomA.

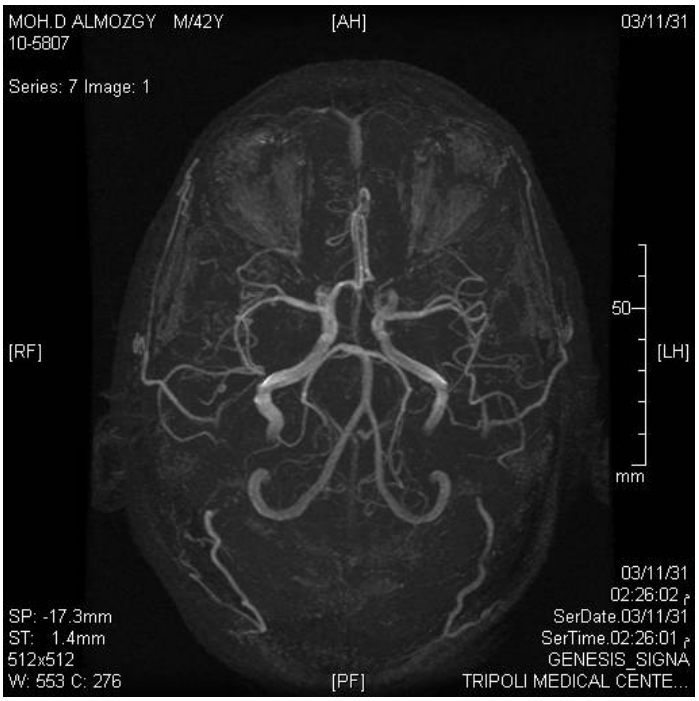

Fig. 21: MRA shows missing of LA1.

Table 12: variations in LA1.

LA1	No	%
Classical	77	89.5
Hypoplastic	6	7.0
Absent	3	3.5
Total	86	100.0

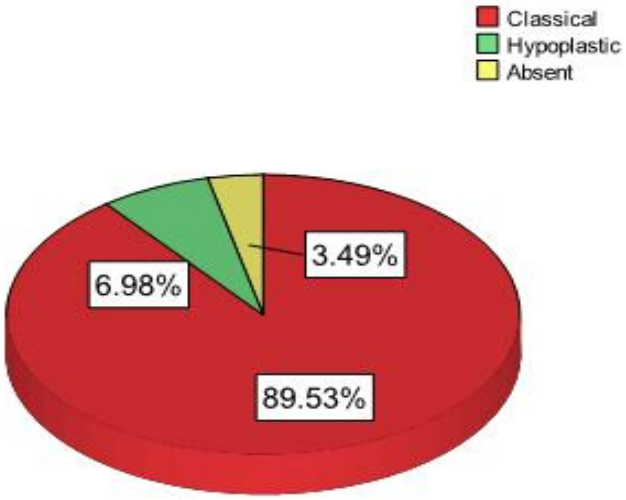

Fig. 22: Variations in LA1.

Variations in the posterior circle:

Posterior circle of Willis (PcomA at both sides and P1 at both sides along with tip of Basilar artery) was studied and found that 39.5% of subjects are of classical anatomy, 60.5% of subjects showed variations as shown in table 13 and fig. 23. PcomA was missing bilaterally in 31.4% of cases (fig. 11) and missing in either side in 16.3% of cases as shown in table 14 and fig. 26. RPcomA was of classical morphology in 48.8% of subjects, hypoplastic in 3.5%, absent in 34.9% (fig. 24) and enlarged in 11% of cases as shown at table 15 and fig. 27. LPcomA was

classical in 46.5% of subjects, hypoplastic in 1.2%, missing in 41.9% (fig. 13 and fig. 25) and enlarged in 10.5% as demonstrated in table 16 and fig. 28. Regarding right posterior cerebral artery RP1 it was classical in 89.5% of cases, hypoplastic in 3.5% and missing in 7% of subjects as shown in table 17 and fig. 29. Which is almost similar to left posterior cerebral artery LP1 percentages which is classical in 90.7% of subjects, hypoplastic in 3.5% and missing in 5.8% of subjects as shown in table 18 and fig. 30. Those variations in posterior circle of Willis showed higher female predominance 70.2% of females and 53.8% of males as shown at table 8.

Table 13: Configuration of posterior circle of Willis.

Posterior circle	No	%
Classical	34	39.5
Variant	52	60.5
Total	86	100.0

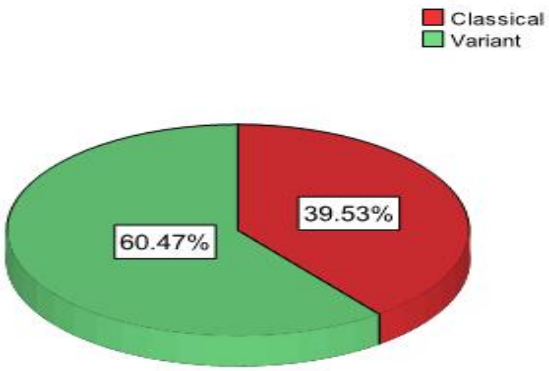

Fig. 23: Configuration of posterior circle of Willis.

Variations in PcomA:

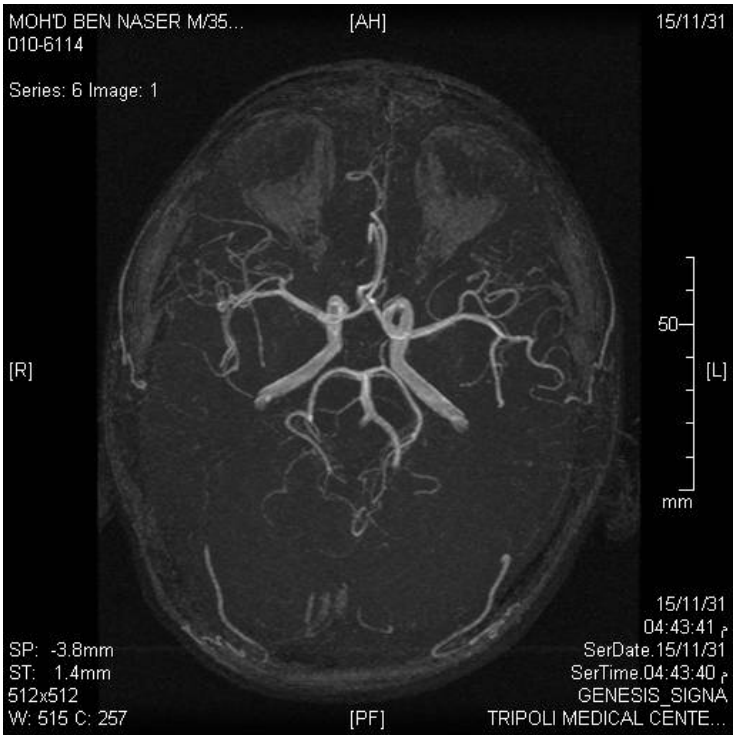

Fig. 24 : MRA shows missing RPcomA.

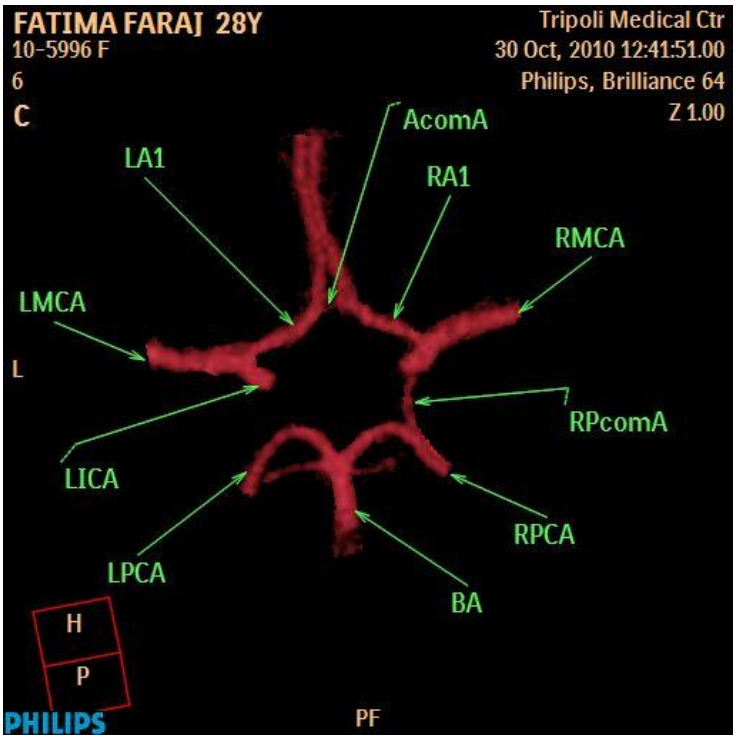

Fig. 25: CTA shows missing LPcomA.

Table 14: variations in PcomA.

PcomA	No	%
Classical	45	52.3
Missing bilaterally	27	31.4
Missed on either side	14	16.3
Total	86	100.0

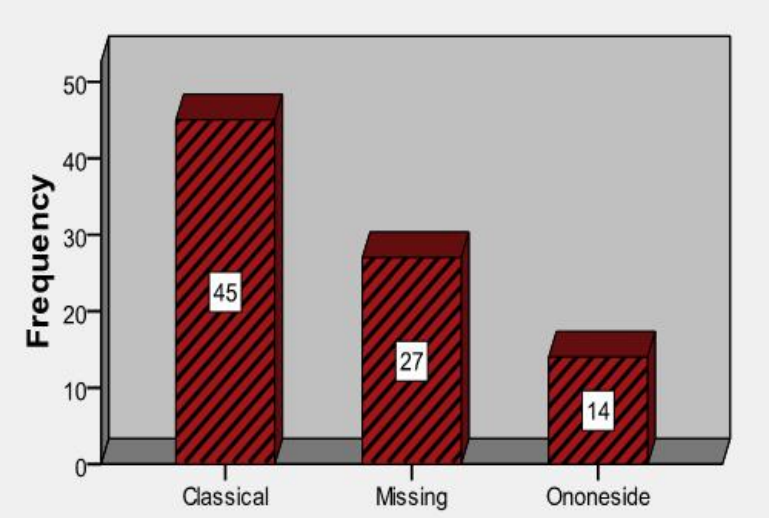

Fig. 26: variations in PcomA.

Table 15: variations in RPcomA.

RPcomA	No	%
Classical	42	48.8
Hypoplastic	3	3.5
Absent	30	34.9
Enlarged	11	12.8
Total	86	100.0

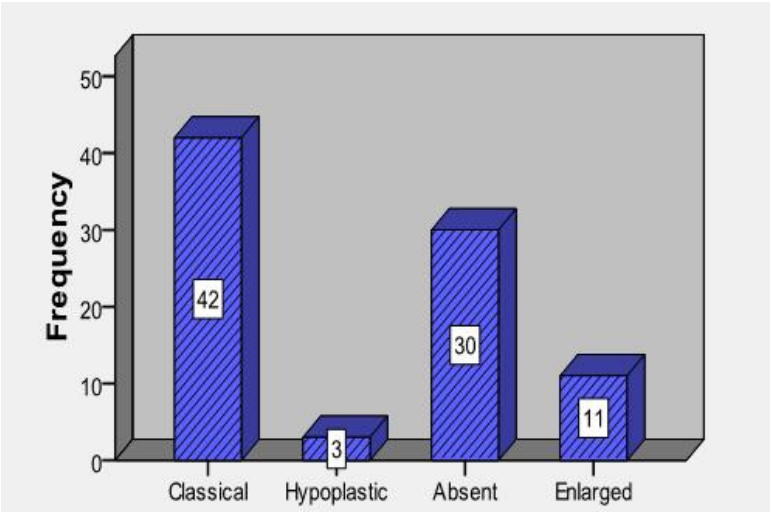

Fig. 27: variations in RPcomA.

Table 16: variations in LPcomA.

LPcomA	No	%
Classical	40	46.5
Hypoplastic	1	1.2
Absent	36	41.9
Enlarged	9	10.5
Total	86	100.0

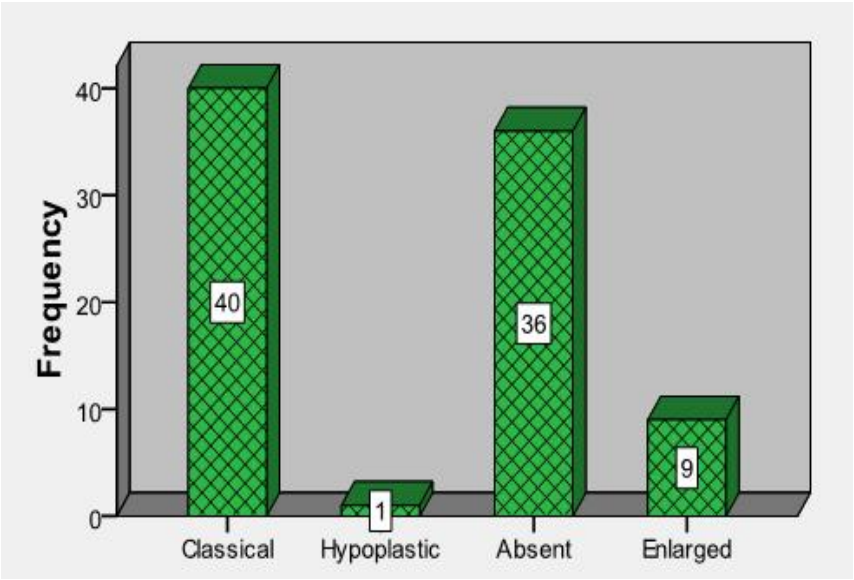

Fig. 28: Variations in LPcomA.

Variations in RP1:

Table 17: variations in RP1.

RP1	No	%
Classical	77	89.5
Hypoplastic	3	3.5
Absent	6	7.0
Total	86	100.0

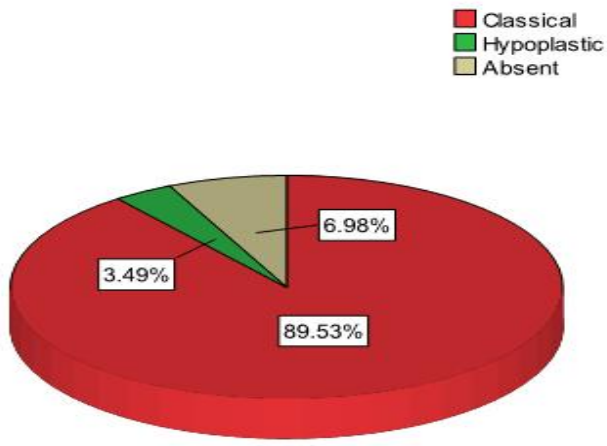

Fig. 29: Variations in RP1.

Variations in LP1:

Table 18: variations in LP1.

LP1	No	%
Classical	78	90.7
Hypoplastic	3	3.5
Absent	5	5.8
Total	86	100.0

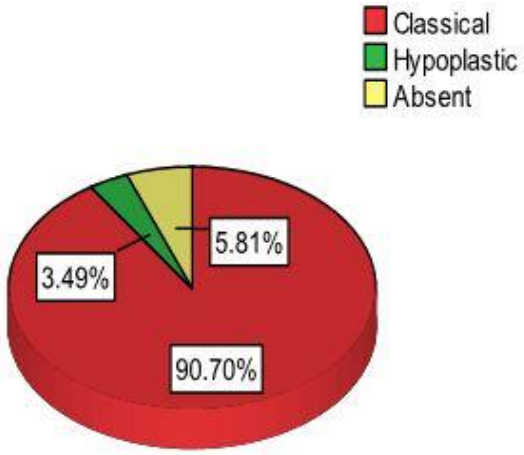

Fig. 30: Variations in LP1.

Fetal variants:

Fetal variants of circle of Willis were searched where in 80.2% of cases no fetal variant was seen, 2.3% of subjects showed bilateral complete fetal variant (fig. 34), 4.7% of subjects showed right complete fetal variant (fig. 18 and fig. 31), 3.5% of cases were of left complete fetal variant (fig. 32 and fig. 33), 1.2% subject demonstrated bilateral partial fetal variant, 2.3% of subjects demonstrated right partial fetal variant (fig. 35), 2.3% of cases were of left partial fetal variant (fig. 36), 2.3% of cases showed right transitional fetal variant (fig. 37), 1.2% of cases showed left transitional fetal variant (fig. 38) and bilateral transitional fetal variant was not seen, these data are summarized in table 19.

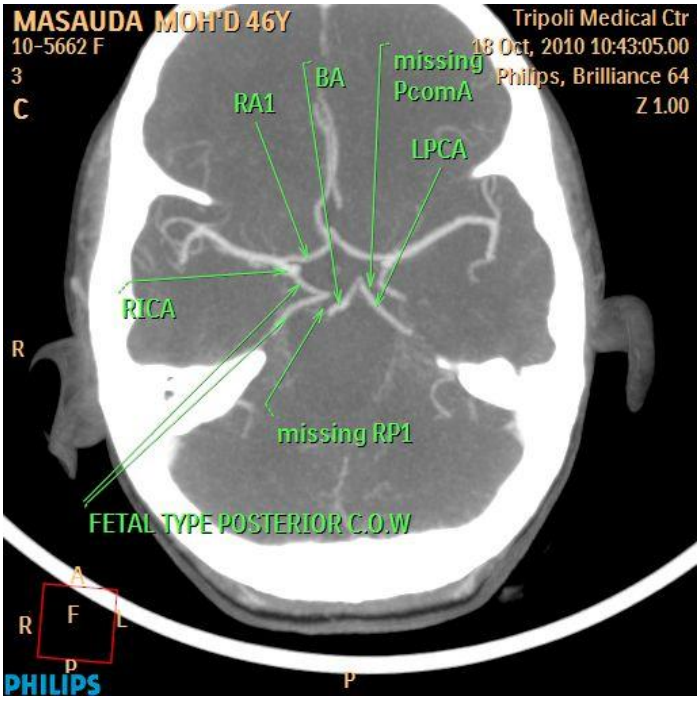

Fig. 31: CTA shows Rt. complete FTP and missing LPcomA.

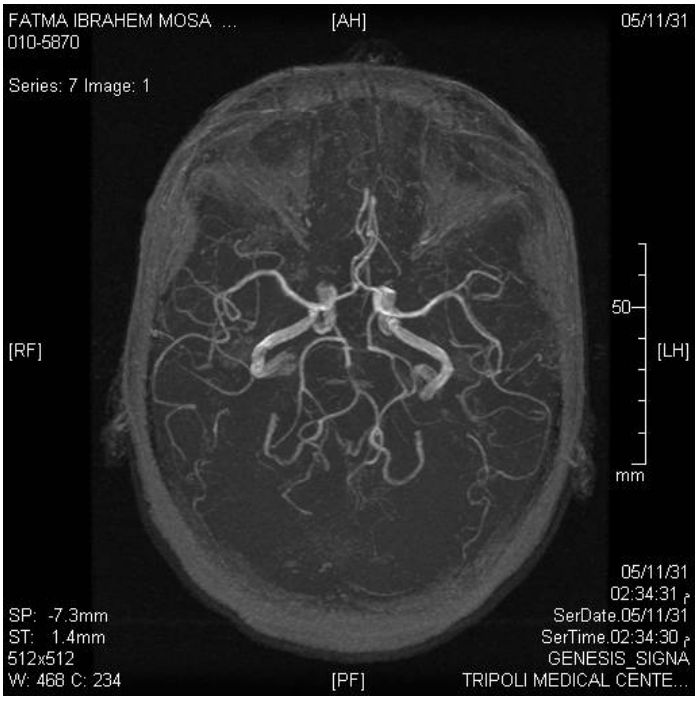

Fig. 32: MRA shows Lt. complete fetal type posterior circle of Willis.

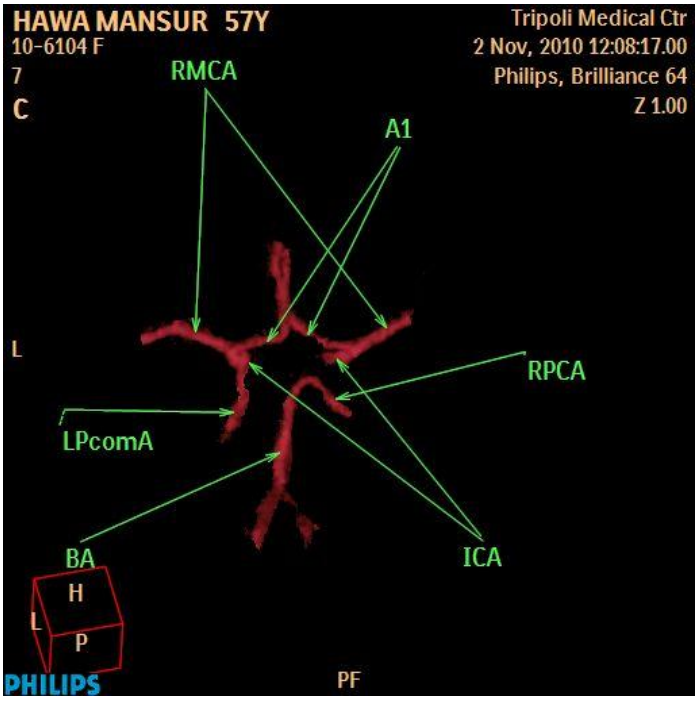

Fig. 33: CTA shows missing RPcomA and Lt. complete fetal type posterior circle of Willis.

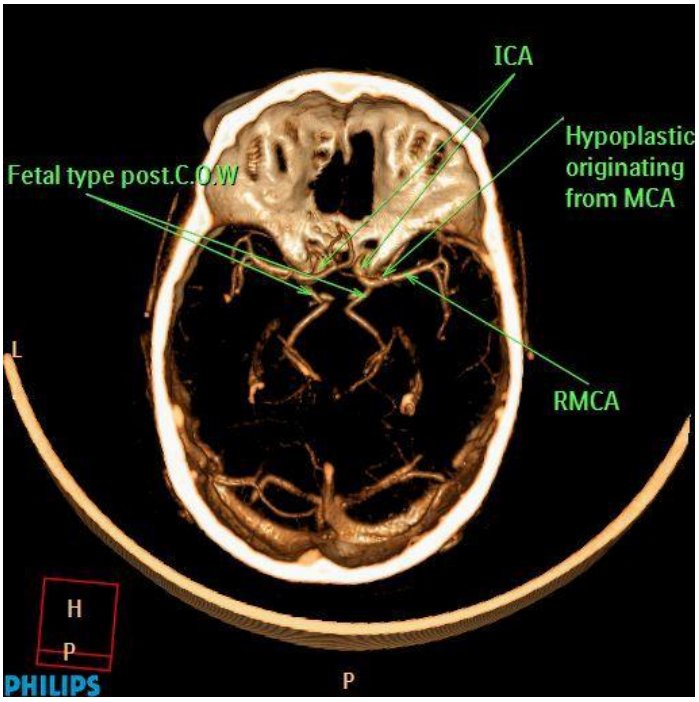

Fig. 34: CTA shows bilateral complete fetal type posterior circle of Willis, hypoplastic RA1.

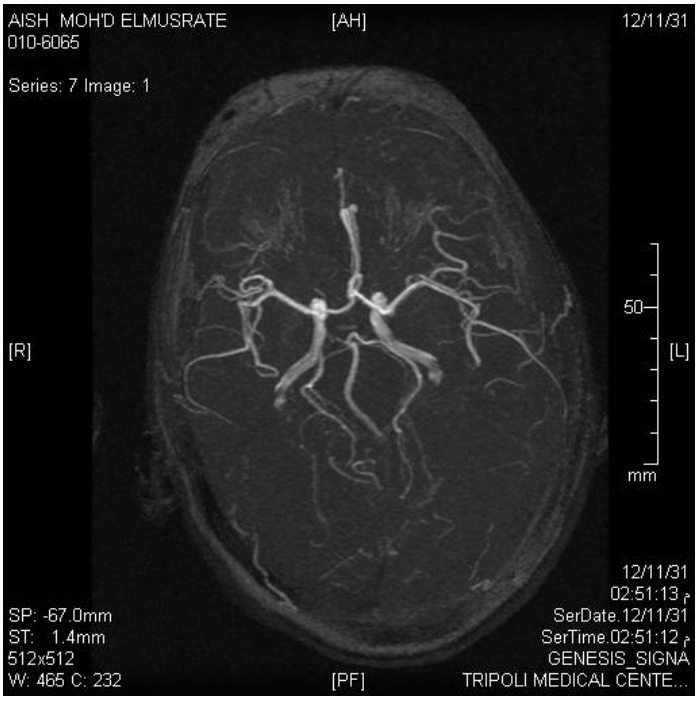

Fig. 35: MRA shows Rt partial fetal type posterior circle of Willis and missing LPcomA.

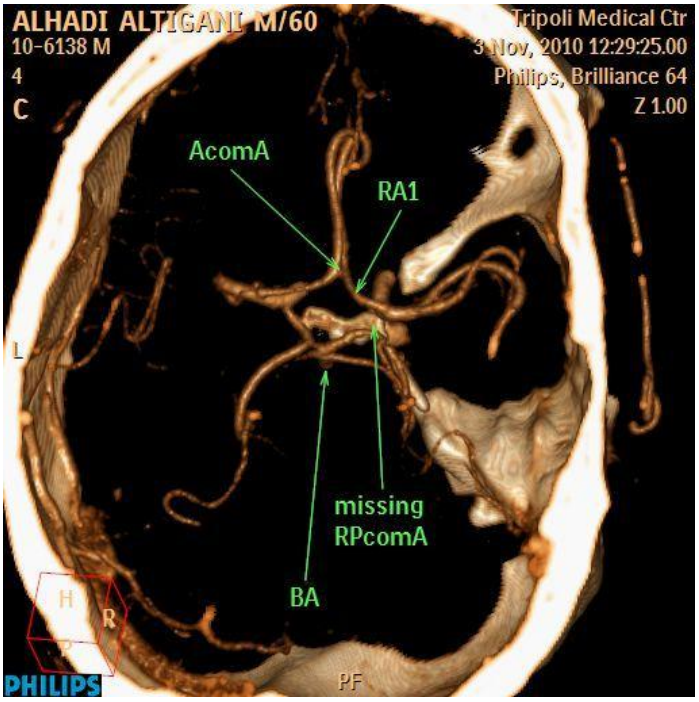

Fig. 36: CTA shows Lt partial fetal type posterior circle of Willis and missing RPcomA.

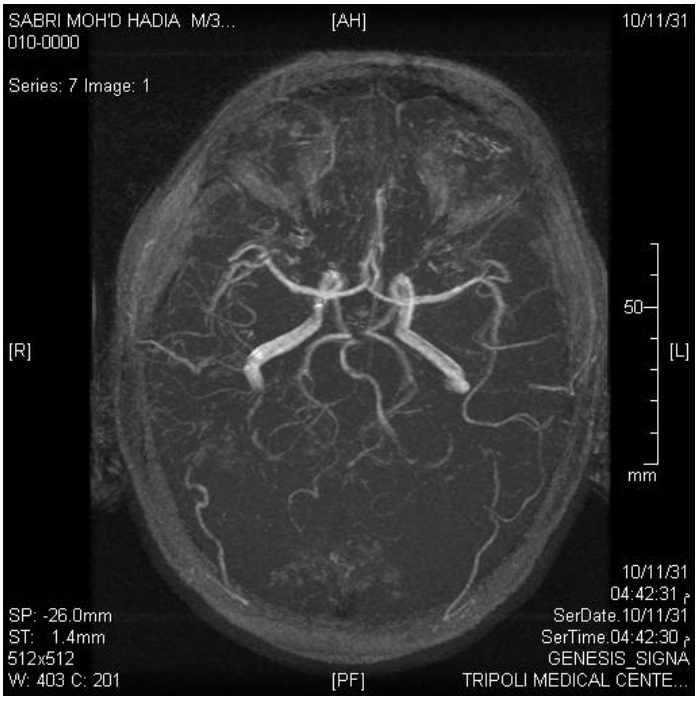

Fig. 37: MRA shows Rt tranisitional fetal type posterior circle of Willis and Lt complete fetal type posterior circle of Willis.

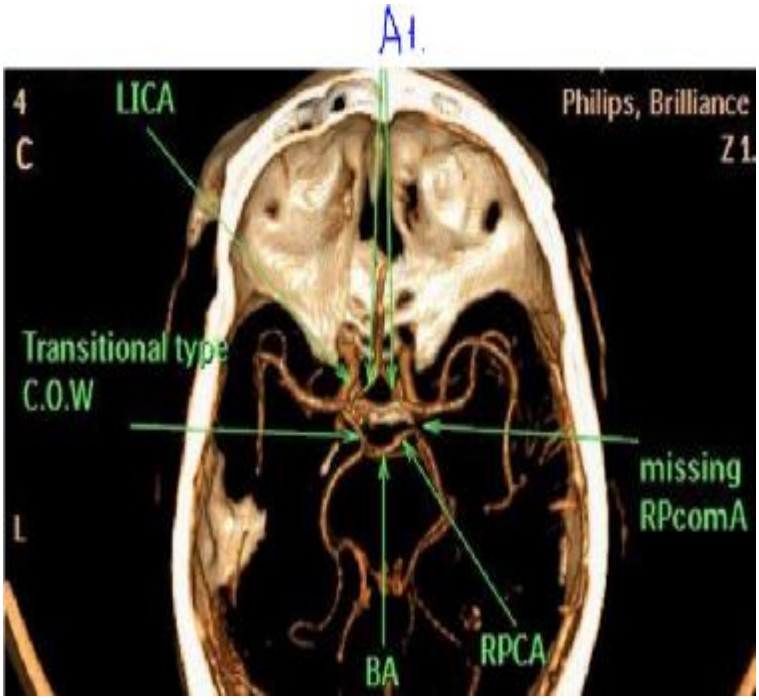

Fig. 38: CTA shows Lt. transitional fetal type posterior circle of Willis and missing RPcomA.

Table 19: Fetal variants of posterior circle of Willis.

Fetal type posterior circle of Willis variants	No	%
Not present	69	80.2
Rt. complete	4	4.7
Lt. complete	3	3.5
Bilateral complete	2	2.3
Rt. partial	2	2.3
Lt. partial	2	2.3
Bilateral partial	1	1.2
Rt. transitional	2	2.3
Lt. transitional	1	1.2
Total	86	100.0

Variations in the Rt. side and Lt. side:

Anatomy of circle of Willis was studied at both sides of the circle of Willis and demonstrated that 51.2% of subjects showed variations in the right side and 57% of subjects showed variations in the left side as shown at table 20 and 21 and figure 40 and 41. These results were cross tabulated with handedness of the subjects as shown at table 22 and 23 and figure 42 and 43. With no statistically significant P value.

Table 20: Configuration of the right side of the circle.

Rt. side	No	%
Classical	42	48.8
Variant	44	51.2
Total	86	100.0

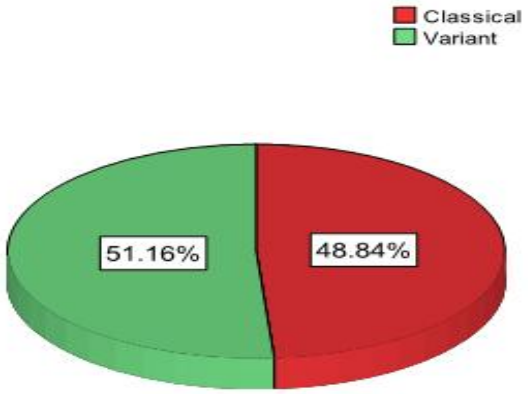

Fig. 39: Configuration of the right side of the circle.

Table 21: Configuration of the left side of the circle.

Lt. side	No	%
Classical	37	43.0
Variant	49	57.0
Total	86	100.0

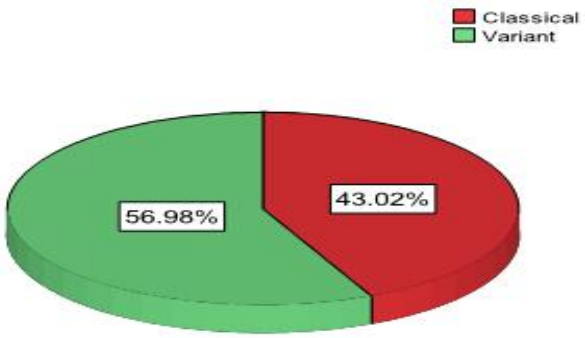

Fig. 40: Configuration of the left side of the circle.

Table 22: cross tabulation between handedness and configuration of the Rt. side of the circle.

	Handedness		Rt. side		
			Classical	Variant	Total
Rt.	Count		41	43	84
	% within Handedness		48.8%	51.2%	100.0%
	% of Total		47.7%	50.0%	97.7%
Lt.	Count		1	1	2
	% within Handedness		50.0%	50.0%	100.0%
	% of Total		1.2%	1.2%	2.3%
Total		Count	42	44	86
		% within Handedness	48.8%	51.2%	100.0%
		% of Total	48.8%	51.2%	100.0%

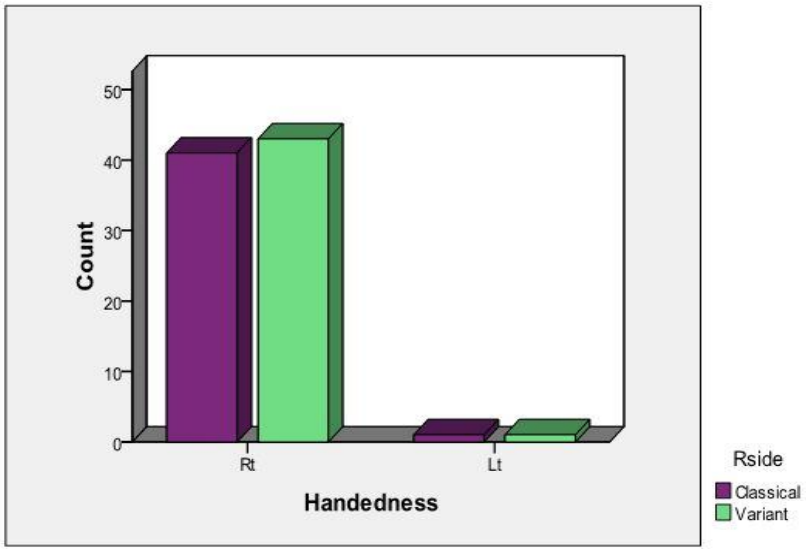

Fig. 41: Cross tabulation between handedness and configuration of the Rt. side of the circle.

Table 23: Cross tabulation between handedness and configuration of the Lt. side of the circle.

		Lt. side		
	Handedness	Classical	Variant	Total
Rt.	Count	37	47	84
	% within Handedness	44.0%	56.0%	100.0%
	% of Total	43.0%	54.7%	97.7%
Lt.	Count	0	2	2
	% within Handedness	.0%	100.0%	100.0%
	% of Total	.0%	2.3%	2.3%
Total	Count	37	49	86
	% within Handedness	43.0%	57.0%	100.0%
	% of Total	43.0%	57.0%	100.0%

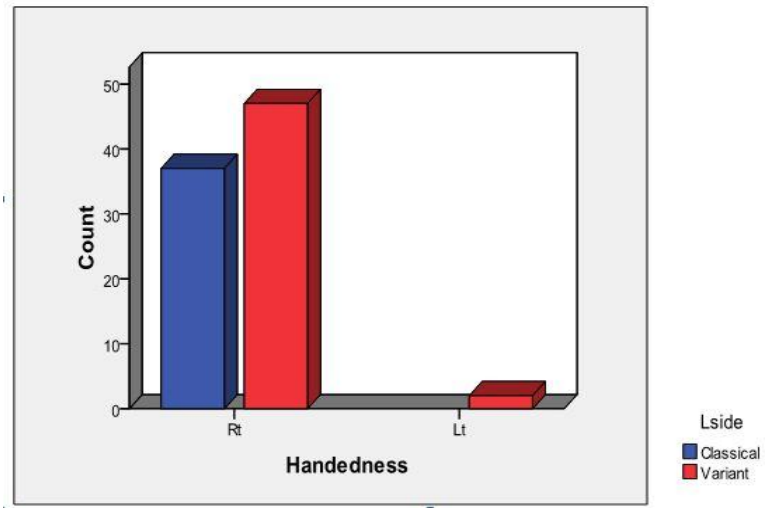

Fig. 42: Cross tabulation between handedness and configuration of the Lt. side of the circle.

Discussion.

Discussion

The present study focusses on the presence of normal anatomical variants of the Circle of Willis by analyzing MRA and CTA of 86 subjects (39 males and 47 females). The results were compared with some published studies. The following vessels constitutes the circle of Willis: the Anterior communicating Artery (AcomA), the precommunicating segment (A1) of the Anterior Cerebral Artery (ACA), the precommunicating segment (P1) of the Posterior Cerebral Arteries (PCA), the posterior communicating arteries (PcomA) and the Internal Carotid Arteries (ICA).

At the present study 36% of subjects showed classical configuration of the circle other studies found range of percentages where Saeki and Rhoton, 1977 and Chen et al, 2004 found it around 21% to 25%, Macchi et al, 1996 and Hartkamp et al, 2000 demonstrated a more concomitant percentages to the present study where they reported that 41% of subjects show classical circles, Hartkamp et al, 1998; Alpers et al, 1959 and Van Raamt et al, 2006 reported a higher percentage of 50% of classical configuration whereas other researchers found a much higher percentages of classical circles where Alawad et al, 2009 reported a percentage of 72% and Fields and Weibel, 1965 reported the percentage of 90% of classical circles.

Regarding A1 at the present study it is found to be classical in 92.4% of subjects and 7.6% of subjects showed hypoplasia or aplasia, these results are within the range mentioned by Nathal et al, 1992 and Van Raamt et al, 2006 where they reported it is seen in 5% to 18%, other studies found lesser percentages of variations in ACA; where hypoplasia of the ACA occurred in 0.7% Alawad et al, 2009. And even more Eftekhar et al, 2006 found no hypoplasia of

precommunicating part of the left anterior cerebral artery A1 nor aplasia of A1. At the present study AcomA was found to be classical in 93% of cases, Merkkola et al, 2006 found it classical in 45% of subjects whereas Baptista et al, 1964; Perlmutter et al, 1976 and Hoksbergen et al, 2000 denied any variations in AcomA. At the present study aplasia was seen in 3.5% of subjects which is in agreement with studies of Piganiol et al, 1960; Macchi et al, 1996 and Alawad et al, 2009. On the other hand aplasia of AcomA was found to represent 22% Merkkola et al, 2006; 10% Kim et al, 2002 but it was only 0.5% Eftekhar et al, 2006. At the present study duplicated AcomA was seen in 2.3% of subjects, Nathal et al, 1992 reported it was seen in 10%. At present study AcomA aneurysm was seen in 1.2%. And in regards with hypoplasia of AcomA at our study it was not recorded, Lazorthes et al, 1979 reported hypoplasia was seen in less than 5% and in study of Fisher, 1965 it was found to be 3% on the other hand Riggs and Rupp, 1963 and El Khamlichi et al, 1985 found that hypoplastic AcomA seen in 10% of cases.

The present study showed that the precommunicating segment of PCA (P1); were bilateral missed in 2.5% of subjects and P1 was bilaterally hypoplastic in 1.6% of subjects these results are in agreement with results of Alawad et al, 2009 who reported that hypoplasia of the PCA occurred in 2.1%, and with the results of Macchi et al, 1996 and Alawad et al, 2009 who found aplasia of PCA also occurred in 2.1% of subjects.

Regarding PcomA it was found to be bilaterally classical in 52.3%, bilaterally missed in 31.4% and missed on either side in 16.3% of cases. RPcomA was found to be classical in 48.8%, missing in 34.9%, hypoplastic in 3.5% and enlraged in 12.8% of cases. LPcomA was found to be classical in 46.5%, absent 41.9%, hypoplastic in 1.2% and enlarged in 10.5% of subjects. Kim et al, 2002 found closer results to the present study where they reported that PcomA may be hypoplastic or absent on one or both sides of the circle in about 25% to 30% of

subjects, on the other hand Haring et al, 1993 and Eftekhar et al, 2006 found that hypoplasia of one or both posterior communicating arteries 34%, of cases. Other authors found that aplasia of both right and left PcomA is around 4.5% (Fawcett, 1905; Fisher, 1965; He et al, 2007, Windle, 1887; Piganiol et al, 1960 and Alawad et al, 2009).

At the present study the difference of occurrence of variations at both sides of the circle was little higher on the left side than on the right side where 48.8% of subjects showed classical configuration of the right side of the circle and only 43% of subjects showed classical configuration of the left side of the circle. These results are concomitant with studies of Windle, 1887; Mitterwallner, 1955 and were in disagreement with Warwick and Williams, 1973; Orlandini, 1970 who found that the majority of cases showed a greater length, and a smaller diameter in the right half of the circle. Also Kamath, 1981 who found abnormal narrowing of vessels was a commoner occurrence on the right side than on the left side and these results were hypothesized to explain dominance of left hemisphere over the right hemisphere.

Variations of anterior part of the circle (both sides A1 and AcomA) in the present study found 77.9% were classical and 22.1% of subjects showed sort of variations. These results are concomittent with the results of Lee et al, 2004; Chen et al, 2004, and to a lesser extent with Hartkamp et al, 1998; Merkkola et al, 2006 where they found classical circles to be seen in 74% of subjects; and are in disagreement with other authors found lesser percentages of classical anterior circles where Miralles et al, 1995 and Nathal et al, 1992 found classical anterior circles seen in 60% of subjects. On the other hand Macchi et al, 1996 found a higher percentage of classical anterior circle where they found it to be 90% of subjects.

The present study found 39.5% classical circles and 60.5% of circles with different variations in the posterior part of the circle (both PcomA and both P1) . These results are in disagreement with Hartkamp et al, 1998; Saeki and Rhoton,1977; Macchi et al, 1996 and Hartkamp and Van der Grond, 2000 where they found higher percentages of classical posterior circle around 50% of subjects, and also in disagreement with Chen et al, 2004 and Lee et al, 2004 where they found lower percentages of classical posterior circle around 25% of subjects. The present study found 52.2% of posterior communicating arteries were absent or hypoplastic whereas 20.9% of precommunicating segment of posterior cerebral arteries were absent or hypoplastic thus the present study revealed that variations in PcomA were more than that on P1, these results are concomitant with reports of Alpers et al, 1959; Fetterman and Moran, 1941; Reddy et al, 1972; Rogers and Lambert, 1946 and in disagreement with reports of Windle, 1887; Fawcett, 1905; Warwick, and Williams, 1973 where they observed variations were more commonly in the posterior cerebrals than in the posterior communicating.

The present study did not reveal any statistically significant difference between the frequencies of variations between the two sexes, which is concomitant with Macchi et al, 1996 and Alawad et al, 2009 and it was in disagreement with the report of Horikoshi et al, 2002 where the authors could find sex-linked differences in anatomical variations of the circle of Willis and a statistically significant correlation between sex-linked differences and aneurysm distribution. Thus they concluded that sex may be one of the potential sources of differences. The present study did not reveal any statistically significant difference between the frequencies of variations in different age groups, these results are in disagreement with Alpers and Berry, 1963; Riggs and Rupp, 1963 where they reported that complete circle of Willis was seen more at survivors of patients with ICA occlusion if compared with autopsy populations.

At the present study fetal variants posterior circle of Willis results were concomitant with the results of Zachariah et al, 2005 who found that the incidence of unilateral fetal type is around 15%. Whereas Eftekhar et al, 2006 found higher results around 27% of vessels having FTP.

Summary

&

Conclusion.

Summary and conclusion

Summary: The circle of Willis is a large interconnecting arterial polygon which enables inter-hemispheric flow through AcomA and in the two directions through the PcomAs where the primary purpose of this vascular circle is to provide anastomotic channels if one vessel is occluded. Cerebral and communicating arteries individually may all be absent, variably hypoplastic, double or even triple.

Objectives*:* To find out normal variations in the circle of Willis in Libyan people. 86 Libyan subjects (39 males (45.3%) and 47 females (54.7%)) with no history of cerebrovascular disease were included in the study. Age ranges from 10 - 70 years (mean 41.4 years), patients are divided into four groups, each group interval is 15 years. 50 patients (22 males and 28 females) underwent 3D TOF MRA of the circle of Willis with the sequence of SPGR using a 1.5-tesla MR scanner (GE Medical Systems); and the other 36 patients (17 males and 19 females) underwent 3D CTA of circle of Willis by using a 64 Slice PHILIPS CT with BRILLIANCE 190P screen.

Statistical analysis: Results were expressed as mean ± standard deviation (SD) or number and (%). Categorical data was compared using Chi-square test. Statistical analysis was performed with the aid of the statistical package for the social sciences (SPSS) computer program (version 18 windows). P value <0.05 was considered significant.

We found that 36% of the cases of the study showed classical configuration of the circle, cross tabulation with age in the different age groups of the study showed no

statistical correlation of circle configuration with the age, circle configuration was cross tabulated with sex found 33.3% of male cases show classical configuration whereas 38.3% of female cases show classical configuration with no significant statistical relation.

22% of subjects showed variations in the anterior circle, LA1 10.4%, RA1 4.6%, AcomA 7%. On the other hand the posterior circle showed 60.5% variations, PcomA was absent bilaterally in 31.4%, it was absent unilaterally in 16.3% (11.6% on the left side and 4.7% on the right side). RPcomA was absent in 34.9%. LPcomA was absent in 41.9%. Regarding RP1 it was hypoplastic in 3.5% and absent in 7% of subjects. LP1 was hypoplastic in 3.5% and absent in 5.8% of subjects.

We found that 19.8% of subjects showed fetal variants, 2.3% bilateral complete fetal variant, 4.7% right complete fetal variant, 3.5% left complete fetal variant, 1.2% bilateral partial fetal variant, 2.3% right partial fetal variant, 2.3% left partial fetal variant, 2.3% right transitional fetal variant, 1.2% left transitional fetal variant and bilateral transitional fetal variant was not seen. The variations in the circle in both sides were as follows 51.2% in the right side and 57% in the left side, with no statistical correlation with handedness of the subjects.

Conclusion: variations in the circle of Willis in Libyan people are found to be in the range mentioned in other studies done at different races, with no statistical difference of occurrence of variations between male and female sexes. Posterior circle of Willis showed the greatest variations if compared with the anterior circle.

Recommendations: preoperative CTA or MRA of circle of Willis should be performed for patients scheduled for carotid endarterectomy, aortic arch reconstructive surgery, and neurointerventional procedures.

Further recommendations: a large scaled study to give more confirmed percentages of variations with the variables age and sex, with large number of left handed

people to evaluate the relation of handedness with configuration of the circle in the right side and the left side. Two series study (with aneurysm and without aneurysm) could give a clear idea about the relation of variations with the aneurysms.

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قسم علم التشريح والأنسجة
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دراسة إشعاعية للتباينات الشكلية في الحلقة الشريانية الدماغية فى المجتمع الليبي

رسالة توطئة للحصول على درجة الماجستير فى علم التشريح

مقدمة من الطبيب

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المعجم العربي

الملخص العربي

دراسة إشعاعية للتباينات الشكلية فى الحلقة الشريانية الدماغية في المجتمع الليبي

حلقة ويلس هي حلقة شريانية تفاغرية كبيرة متعددة الاضلاع تمكن من السريان البيني للدم بين نصفي الكرة الدماغية عن طريق الشريان الموصل الامامي و تمكن من السريان فى الاتجاهين الامامي و الخلفي عن طريق الشريان الموصل الخلفي حيث ان الغرض الاساسي لهذه الحلقة الوعائية هو تزويد قنوات تفاغرية وعائية اذا ما انسد احد الاوعية. الشرايين الدماغية او الشرايين الموصلة كلها او بصورة منفردة قد تكون مفقودة او ضامرة او مزدوجة او ثلاثية الازدواج.

المهام: لتحديد التباينات الشكلية فى حلقة ويلس فى المجتمع الليبي، شملت الدراسة 86 فرد ليبي (39 ذكر (45.3%) و 47 انثى (54.7%)) دون وجود اي تاريخ مرضي وعائي دماغي ... الاعمار تتراوح بين 10 سنوات و 70 عاما (المتوسط 41.4)، المرضى تم تقسيمهم الى اربع مجموعات، كل فترة مجموعة هي خمسة عشرة سنة. خمسون مريض (22 ذكر و 28 انثى) خضعوا للفحص الوعائي بالرنين المغنطيسي لحلقة ويلس باستخدام تقنية 3D TOF MRA لحلقة ويلس باستخدام متتالية SPGR بواسطة ماسح رنين مغنطيسي 1.5 تسلا (GE Medical Systems). وستة وثلاثون مريض (17 ذكر و19 انثى) خضعوا للفحص باستخدام تقنية 3D CTA لحلقة ويلس باستخدام جهاز المسح المقطعي 64 شريحة نوع PHILIPS CT و شاشة عرض نوع BRILLIANCE 190P.

التحليل الإحصائي: النتائج تم التعبير عنها بواسطة المتوسط و الانحراف المعياري و الارقام والنسبة المئوية. المعطيات الفئوية تمت مقارنتها باستخدام مربع كاي. التحليل الاحصائي تم باستخدام برنامج الحزمة الاحصائية للعلوم الاجتماعية SPSS برنامج الكومبيوتر نوافذ- الاصدار 18. واعتبرت قيمة $P<0.05$ على انها ذات اهمية احصائية.

وجدنا ان 36% من الحالات اظهرت التكوين التقليدي للحلقة. من خلال الجدولة البينية بين التكوين و العمر و التى اجريت على جميع الفئات العمرية لم تظهر الدراسة اي علاقة ذات دلالة احصائية للتكوين مع العمر. وكذلك تمت الجدولة البينية لتكوين الحلقة مع الجنس و اظهرت الدراسة ان 33.3% من الذكور يظهرون

التكوين التقليدي للحلقة بينما 38.3% من الاناث يظهرن التكوين التقليدي للحلقة ولم تكن هناك اي علاقة ذات دلالة احصائية .

22% من الاشخاص اظهروا تباينات فى الحلقة الأمامية، الشريان الدماغي الامامي الايسر 10.4%، الشريان الدماغي الامامي الايمن 4.6%، الشريان الموصل الامامي 7%.

ومن الناحية الاخرى اظهرت الحلقة الخلفية 60.5% من التباينات، الشريان الموصل الخلفي مفقود على الجانبين فى 31.4%، و مفقود على احد الجانبين في 16.3% (11.6% على الجهة اليسرى و 4.7% على الجهة اليمنى). الشريان الموصل الخلفي الايمن مفقود في 34.9%. الشريان الموصل الخلفي الايسر مفقود فى 41.9%. وبالنسبة للشريان الدماغي الخلفي الايمن كان ضامرا فى 3.5% من الحالات و مفقود فى 7% من الحالات. وبالنسبة للشريان الدماغي الخلفى الايسر كان ضامرا فى 3.5% من الحالات و مفقود فى 5.8% من الحالات.

كما وجدنا انه 19.8% من الحالات تظهر التباين الجنيني حيث ان 2.3% تظهر تباين جنيني ثنائي كامل، 4.7% تظهر تباين جنيني كامل أيمن، 3.5% تظهر تباين جنيني كامل أيسر، 1.2% تظهر تباين جنيني ثنائي جزئي، 2.3% تظهر تباين جنيني جزئي أيمن، 2.3% تظهر تباين جنيني جزئي أيسر. و 2.3% تظهر تباين جنيني انتقالي ايمن، 1.2% تظهر تباين جنيني انتقالي ايسر، بينما لم تسجل اي حالة للاختلاف الجنيني الانتقالي الثنائي.

التباينات فى الحلقة فى الجهتين كانت كما يلي 51.2% فى الجهة اليمنى و57% فى الجهة اليسرى، دون وجود اي علاقة ذات دلالة احصائية بين يدوية الشخص و التباينات.

الخلاصة: التباينات فى حلقة ويلس فى المجتمع الليبي وجدت فى النطاق المذكور لدراسات اخرى اجريت على اعراق اخرى. دون وجود اي دلالة احصائية لوقوع التباينات بشكل مختلف فى الجنسين. الحلقة الخلفية لويلس اظهرت اكبر نسبة من التباينات اذا ما قورنت مع الحلقة الامامية لويلس.

التوصيات: CTA و MRA لحلقة ويلس قبل الجراحة يوصى ان تجرى للمرضى المجدولين لاجراء عملية استئصال بطانة الشريان السباتي، و عملية اعادة ترميم القوس الوتيني، و الإجراأت التدخلية العصبية.

المزيد من التوصيات: اجراء دراسة اوسع لتعطي نسبة اكثر ثبات للتباينات فى الحلقة مع المتغيرات من العمر و الجنس، و استخدام عدد اكبر من العسراء لتقييم العلاقة بين اليدوية و التكوين للحلقة في الجهة اليمنى و الجهة اليسرى وإجراء دراسة ذات سلسلتين (سلسلة امدم و سلسلة دون امدم) قد تعطي فكرة اكثر وضوح عن علاقة التباينات مع الإمدم.