The Circle of Willis (CW) is a circulatory anastomosis between carotid and vertebral arterial systems, maintaining an adequate cerebral perfusion (1, 2, 3).

The anomalies of the vessels which comprise it include persistence of primitive vessels that, normally, come back during early gestation and arteries that are still present in adulthood, but have an early involution (4).

The importance in the recognition of Anatomical Variations of the Circle of Willis (AVCW) lies in the fact that they can be confused with pathological findings and that their presence is relevant in the planning of surgical procedures. Besides, many studies...
show the increase in incidence of aneurysms associated with AVCW (5, 6, 7, 8).

AVCW have characteristics that can be recognized with Magnetic Resonance Angiography (MRA), which becomes an essential tool in their evaluation (9, 10, 11).

Variations, classification and frequency of presentation

1- Variations and anomalies of the Internal Carotid Artery (ICA)

1 A) Dorsal persistence of the ophthalmic artery: In early gestation there are ventral and dorsal ophthalmic arteries. Generally the ventral is the only one that persists originating in the supraclinoid segment of the ICA and going into the orbit through the optic canal. The anomaly is the persistence of the dorsal ophthalmic artery originating in the cavernous segment of the ICA and going into the orbit through the superior orbital fissure.

2- Variations and anomalies of the Anterior Cerebral Artery (ACA) and the Anterior Communicating Artery (ACoA)

2 A) Carotid-ACA Anastomosis: The ACA originates from the ICA, at the same level of the ophthalmic artery, it ascends in a medial pathway between the optic nerves and it produces anastomosis with the union of the A2 segment of the ACA and ACoA.

2 B) Fenestration of ACA and ACoA.

2 C) Duplication of ACoA.

2 D) Persistence of the primitive olfactory artery: The proximal portion of the ACA follows an anterior-inferior-medial pathway through the olfactory tract and it turns in a posterior-superior way. This anomalous ACA is known as persistent primitive olfactory artery. Normally, this artery involutes and becomes a recurrent artery of Heubner.

2 E) Azygos ACA: Complete fusion of the segment A2.

2 F) Bi-hemispheric ACA: Asymmetrical A2 segments, one is hypoplastic and the other is hyperplastic, which send branches to both hemispheres in a dominant way.

2 G) Trifurcated ACA: Anomalous branch originating from the ACA creating a trifurcation.

2 H) Absence of ACoA.

2 I) Unilateral absence of the segment A1.

3- Variations and anomalies of the Middle Cerebral Artery (MCA)

3 A) Duplication of MCA.

3 B) Accessory MCA: Small artery originating in the ACA at the level of segment A1.

3 C) Early bifurcation of MCA: The MCA bifurcates within the first centimeter of its origin.

3 D) Fenestration of the MCA.

4- Variations and anomalies of the Posterior Cerebral Artery (PCA) and the Posterior Communicating Artery (PCoA)

4 A) Duplication of the PCA and the PCoA.

4 B) Branch of the PCA originating in the hyperplastic choroidal artery: The proximal anterior choroidal artery is extremely long simulating a duplication of PCoA.

4 C) Fenestration of PCA.

4 D) Unilateral or bilateral absence of hypoplasia of the segment P1: The ipsilateral PCA originates directly from the ICA. It is called fetal origin of the PCA.

4 E) Unilateral or bilateral hypoplasia of the PCoA.

5- Variations and anomalies of Vertebrobasilar (VB) Arteries

5 A) Fenestrations of VB Arteries.

5 B) Duplication of basilar artery.

6- Variations of Cerebellar Arteries

6 A) Anterior Inferior Cerebellar Artery (AICA) + Posterior Inferior Cerebellar Artery (PICA): AICA and PICA fusion together and create a fenestration.

6 B) AICA/PICA hypoplasias.

6 C) Duplication of the Superior Cerebellar Artery (SCA).

6 D) Origin of the SCA from the proximal segment of the PCA: They both create a common branch.

6 E) Fenestration of SCA.

7- Intracranial Carotid-Vertebral Anastomosis

These anastomoses are normal in the fetus and can persist into adulthood with an incidence of 1%.

7 A) Proatlantal Artery.

7 B) Persistent Hypoglossal Artery: Anastomosis between ICA and the low position of the basilar artery through the hypoglossal canal.

7 C) Persistent Otic Artery.

7 D) Persistent Trigeminal Artery: It originates in the
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precavernous or cavernous segment of the ICA and it connects itself with the medial portion of the basilar artery.

Results

- 68 patients with anatomical variations.
- 14 different types of anatomical variations:
  4 D) Fetal origin of PCA: 17 (25%).
  6 B) AICA/PICA hypoplasia: 15 (22%).
  4 E) Absence or hypoplasia of PCoA: 11 (16%).
  2 I) Absence of segment A1: 4 (6%).
  7 D) Persistent trigeminal artery: 4 (6%).
  2 E) Azygos ACA: 3 (4%).
  3 C) Early bifurcation of MCA: 3 (4%).
  6 C) Duplication of SCA: 2 (3%).
  6 D) Joint origin of SCA and PCA: 2 (3%).
  2 B) Fenestration of ACoA: 2 (3%).
  2 C) Duplication of ACoA: 2 (3%).
  2 G) Trifurcation of ACA: 1 (2%).
  4 A) Duplication of PCoA: 1 (2%).
  4 C) Fenestration of PCA: 1 (1%).

Fig. 1: Variations and anomalies of ICA, ACA and ACoA.

A) Persistent dorsal ophthalmic artery (arrow). a: ICA; b: vertebral artery; c: basilar artery; d: ophthalmic artery; e: anterior cerebral artery (ACA); f: middle cerebral artery (MCA); g: posterior cerebral artery (PCA).
B) Left Carotid-ACA Anastomosis (arrow).
C) Fenestration of ACoA (black arrow) and Fenestration of ACA (white arrow).
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Fig. 2:

Variations and anomalies of ICA, ACA and ACoA.
A) Duplication of ACoA (arrow).
B) Persistent primitive olfactory (arrow). a: ICA; b: vertebral artery; c: basilar artery; e: ACA; f: MCA; g: PCA; h: anastomosis of ACA.
C) Azygos ACA (arrow).
D) Bihemispheric ACA (arrow).
E) Trifurcated ACA (arrow).
F) Absence of ACoA (arrow).
G) Absence of the segment A1 (arrow).

Fig. 3:

Variations and anomalies of MCA and PCA.
A) Duplication of MCA (white arrow); Proximal (thick black arrow) and Distal (thin black arrow) Accessory MCA; Early bifurcation of MCA (white arrowhead); Fenestration of MCA (black arrowhead).
B) Duplication of PCA (arrow).
C) Branch of the PCA originating from the Hyperplastic Choroidal Artery (arrow). a: ICA; b: vertebral artery; c: basilar artery; e: ACA; f: MCA; g: PCA.
Variations and anomalies of PCA, ACoP and VB.
A) Fenestration of PCA (arrow).
B) P1 Hypoplastic Absence (arrow).
C) ACoP Hypoplastic Absence (arrow).
D) Fenestration of Vertebral (white arrow) and Basilar (black arrow) Artery.
E) Duplication of Basilar Artery (arrow).

Variations of Cerebellar Arteries.
A) AICA + PICA (arrow).
B) AICA Hypoplasia (arrow).
C) SCA Duplication (arrow).
D) Joint origin of SCA and PCA (arrow).
E) SCA Fenestration (arrow).
Fig. 6: Carotid Vertebral Anastomosis. Proatlantal Artery Type I (white arrow); Proatlantal Artery Tipo II (thick black arrow); Persistent Hypoglossal Artery (thin black arrow); Persistent Otic Artery (white arrowhead); Persistent Trigeminal Artery (black arrowhead).

Fig. 7: Based on 190 patients to identify AV.
1- Patients with AV: 68 (36%).
2- Patients without AV: 122 (64%).

Fig. 8: Types of Anatomical Variations found.
1- Fetal origin of PCA: 17 (25%).
2- AICA/PICA hypoplasia: 15 (22%).
3- Absence or hypoplasia of PCoA: 11 (16%).
4- Absence of segment A1: 4 (6%).
5- Persistent trigeminal artery: 4 (6%).
6- Azygos ACA: 3 (4%).
7- Early bifurcation of MCA: 3 (4%).
8- Duplication of SCA: 2 (3%).
9- Joint origin of SCA and PCA: 2 (3%).
10- Fenestration of ACoA: 2 (3%).
11- Duplication of ACoA: 2 (3%).
12- Trifurcation of ACA: 1 (2%).
13- Duplication of PCoA: 1 (2%).
14- Fenestration of PCA: 1 (1%).
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Fig. 9: Fenestration of ACoA: A) 3D TOF sequence in axial view (arrow), and B) 3D MIP reconstruction (arrow).

Fig. 10: Duplication of ACoA: A) 3D TOF sequence in axial view (arrow), and B) 3D MIP reconstruction (arrow).

Fig. 11: Azygos ACA: A) 3D TOF sequence in axial view (arrow), and B) 3D MIP reconstruction (arrow).

Fig. 12: Trifurcated ACA: A) 3D TOF sequence in axial view (arrow), and B) 3D MIP reconstruction (arrow).
**Fig. 13:** Absence of segment A1: **A)** 3D TOF sequence in axial view (arrow), and **B)** 3D MIP reconstruction (arrow).

**Fig. 14:** Early bifurcation of MCA: **A)** 3D TOF sequence in axial view (arrow), and **B)** 3D MIP reconstruction (arrow).

**Fig. 15:** Duplication of PCoA: **A)** 3D TOF sequence in axial view (arrow), and **B)** 3D MIP reconstruction (arrow).

**Fig. 16:** Fenestration of PCA: **A)** 3D TOF sequence in axial view (arrow), and **B)** 3D MIP reconstruction (arrow).
Fig. 17: Fetal origin of PCA: A) 3D TOF sequence in axial view (arrow), and B) 3D MIP reconstruction (arrow).

Fig. 18: Absence of PCoA: A) 3D TOF sequence in axial view (arrow), and B) 3D MIP reconstruction (arrow).

Fig. 19: AICA Hypoplasia: A) 3D TOF sequence in axial view (arrow), and B) 3D MIP reconstruction (arrow).

Fig. 20: Duplication of SCA: A) 3D TOF sequence in axial view (arrow), and B) 3D MIP reconstruction (arrow).
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Discussion

The percentage of normal CW varies widely in the bibliography, with percentages between 38 and 80% (14). According to our experience, a 64% of the patients presented the usual anatomy.

Authors like Riggs and Takahashi (4, 12) describe multiple ways to modify the AVCW and some others take into account extracranial vessels. Therefore, we have unified criteria in a simplified classification based on anatomical territories, since we consider it more didactic.

In the consulted bibliography, the most frequent AVCW correspond to the anterior vascular territory (4, 9, 14), followed by the absence of PCoA. The fetal origin of PCA was the most present variation, comprising the 25% of the total of detected anomalies. AICA/PICA hypoplasia and the absence/hypoplasia of PCoA followed in frequency. There was a lower percentage of anomalies in the anterior vascular territory but with a greater variability in subtypes. As for vertebral-carotid anastomosis, the persistent trigeminal artery was the most frequent in our exploration.

Six patients showed 2 associated AV (9%); that is, more than one alteration; this frequency is inferior to the one published in the international bibliography, which is of 23% (14).

The CW presents a great anatomical variability and to study it, there are multiple evaluation methods, such as MRA and Angio-CT, which have a sensibility and specificity that are comparable to traditional angiographic methods (15).

The use of MRA as a noninvasive method is very useful and optimal for the study of AVCW, permitting visualization in different views, with 3D reconstructions.

Fig. 21: Joint origin of SCA and PCA: A) 3D TOF sequence in axial view (arrow), and B) 3D MIP reconstruction (arrow).

Fig. 22: Persistent Trigeminal Artery: A) 3D TOF sequence in axial view (arrow), and B) 3D MIP reconstruction (arrow).
Bibliography