Magnetic resonance angiographic evaluation of circulus arteriosus cerebri (circle of Willis): a morphologic study in 100 human healthy

Magnetic resonance angiographic evaluation of circulus arteriosus cerebri (circle of Willis): a morphologic study in 100 human healthy subjects / C.Macchi;C.Catini;.F.Cerini;M.Gulisano;P.Pacini;F.Cecchi;L.Corcos;E.Brizzi.. - In: ITALIAN JOURNAL OF ANATOMY AND EMBRYOLOGY. - ISSN 1122-6714. - STAMPA. - 101:(1996), pp. 115-123.

IT. J. ANAT. EMBRYOL. Vol. 101, n. 2: 115-123, 1996

Magnetic resonance angiographic evaluation of circulus arteriosus cerebri (circle of Willis): a morphologic study in 100 human healthy subjects

Claudio Macchi */ - Claudio Catini * - Cerini Federico **** - Massimo Gulisano * Paolo Pacini * - Francesca Cecchi *** - Leonardo Corcos *** and Enzo Brizzi ***

* Department of Human Anatomy and Histology, University of Florence, Italy
** Pro-Juventute Foundation Don C. Gnocchi, Pozzolatico, Florence, Italy
*** Prosperius Institute, Florence, Italy
**** Scuola di Sanità, Aeronautica Militare

Key words: Circle of Willis, MRA.
Supported by a grant from MURST through the Institute of Physical Education of Florence

SUMMARY

The Circle of Willis was studied by Magnetic Resonance Angiography in 100 healthy subjects. In 41% of these cases, the arteries were arranged in the classically described way. In 21%, hypoplasia of the posterior communicating as. was noted, while in 13% the posterior cerebral as. were found to originate from the internal carotid a.. In 9% of these cases, three anterior cerebral as. were present. In 3% the anterior communicating artery could not be identified, while the left posterior communicating artery was hypoplastic. In 2% the absence of a posterior communicating artery was associated with the origin of a posterior cerebral a. from the internal carotid. In another 2% the anterior cerebral as. were partially fused, and in yet another 2%, hypoplasia of both an anterior and a posterior cerebral a. was present. The remaining seven cases (7%), all different from each other, represented combinations of the above described variations. Statistical analysis indicated that anomalies occurred more commonly on the left than on the right side. The morphology of the Circle of Willis could not be correlated with either sex or Body Index.

INTRODUCTION

The circulus arteriosus cerebri (circle of Willis) consist of: two anterior cerebral as., the anterior communicating a., two middle cerebral as., two posterior cerebral as., two posterior communicating as. In order to understand its great morphological variability (Adachi, 1928), it should be considered that develops through complex embryological transformations (Pensa and Favaro, 1935; Ruggiero et al., 1987). Initially, the internal carotid is the only artery supplyng the brain mass. It divides into two terminal branches, anterior and posterior. The former branch represents the anterior cerebral a., while the latter gives rise to the posterior cerebral a.. Subsequently, the two anterior cerebral aa., which until this period were independent and parrallel, join via the anterior communicating a.. The two caudal branches of the internal carotid join to form a single trunk (the basilar a.), and the two vertebral as. make their appearance (Testut and Latarjet, 1972). The anterior portion of the caudal branch of the internal carotid a. then undergoes atrophy, diminishing in size, while the vertebral a. enlarges and takes its position, causing the blood-flow in to change its course in a rostral direction. The anterior portion of the caudal branch, which precedes the emergence of the posterior cerebral a., continues to atrophy and becomes the posterior communicating a..

Following Thomas Willis, this arterial circuit has been morphologically studied by many researchers (Mori, 1883; Peli, 1902; Cavatorti, 1908; Elliot-Smith, 1909; Adachi, 1928; Deker and Hipp, 1958; Mortillaro and Crivelli, 1963; Fazio et al., 1970; Orlandini, 1970; Gulisano et al., 1982; Ruggiero et al., 1987). Particularly, it is interestying to note that the study of Orlandini et al. (1985), performed in cadavers, agrees with echo-color-doppler studies (Macchi and Catini, 1994), demonstrating a mean caliber of the main aa. of the circle lower than previously reported. Moreover, many Authors emphasized the importance of morphological study of the circle of Willis from a clinical point of view (Krayembul and Jasargil, 1957; Kudo, 1968; Livanaien, 1973; Schmitt, 1978; Krayembul et al., 1979; Kitahara et al., 1979; Contamin et al., 1983).

Nowadays, it is generally accepted that the anatomical study of the circle of willis is of great clinical and prognostic utility, given the prevalence of disease, such as atherosclerotic occlusion (Weber et al., 1977; Spencer, 1987) in the cranic circulatory district. The morphological pattern is especially important with regard to the establishment of emodynamically valid compensatory arch (Brice et al., 1964; Fazio, 1969; Flanagan et al., 1977; Brown and Johnson, 1982) and to the pathogenesis of cerebral hemorrhage (Nenci, 1945; Aaslid and Nornes, 1984) and aneurysms (Slany, 1938; Bartholow, 1943; Tardini, 1954). In such perspective, the present research aims to evaluate the morphology of the circle of Willis in a significant healthy population. With this purpose, Magnetic Resonance Angiography (MRA) appeared a suitable instrument since it represents a non-invasive method and provides high quality image definition without need for contrast agents.

MATERIAL AND METHODS

One hundred healthy subjects (50 men, 50 women) aged between 20 and 82 years were studied. According to the etical committee instructions, each patient gave informed consent to be included in the study. The patients had no evidence for: vaso-occlusive disease of the supraaortic trunks, diabetes, hypertension, ischemic or hemorragic brain lesions demonstrable by magnetic nuclear resonance (MNR). Each subject underwent cerebral MRA, including examination of the parenchyma and vessels. A Philips Gyroscan T5-NT instrument was used. The following parameters were also registred for each patient: sex, age and Body Index (BI), obtained by the formula: BI = weight/height. The morphologic variants of the circle of willis were classified in groups. Possible differences between males and females, and between right and left side, as regards the occurrence of anomalies and BI was investigated by the evaluation of correlation coefficient (Cr).

RESULTS

Fig. 1 is a schematic drawing of the morphologic variants of all circle of Willis examined. The pattern classically described as «normal» (type A) constituted only 41% of the cases (*fig. 2*). In 21% of considered cases (type B), hypoplasia of the posterior communicating aa. was present. Such hypoplasia was bilateral (type B1) in 12% and monolateral (type B2) in 9%; the latter 9 hypoplasiae were subdivided as follows: 6 were left-sided and 3 were right-sided. In 13% (type C), the principal anomaly regarded the posterior cerebral as., which originated from the internal carotid a.. In 5 out of these 13 cases (type C1), this anomaly occurred monolaterally (4 on the left, 1 on the right side); in 4 cases the anomaly occurred bilaterally, [3 subjects had moderate hypoplasia (type C2) and 1 had marked hypoplasia (type C3) of the segments connecting the basilar a., which represent the pre-communicating segment of the posterior cerebral as.]; in 2 cases (type C4), apart from a posterior cerebral a. originating from the internal carotid (the left in both cases), there was hypoplasia of the posterior communicating a. (on the left side in 1 case, on the right side in 1 case); in the remaining 2 cases, a posterior cerebral a. arising from the left internal carotid was lacking of any connection with the basilar a. (type C5).

In 9% of the examined cases, 3 anterior cerebral vessels were present (type D). Out of these 9 cases, 2 were otherwise «normal» (type D1) and 2 were associated with hypoplasia of the posterior communicating a. (2 on the left, 1 on the right side) and with a posterior cerebral a. originating from the internal carotid (2 on the left, 1 on the right side) (type D2). In 2 cases, 2 of the 3 anterior cerebral vessels originated from the right anterior cerebral a., while a thin segment joined them to the left anterior cerebral a. (type D3). Two type D cases were associated with hypoplasia of the left posterior communicating a. (type D4), and in the remaining case, the left posterior cerebral a. originated from the internal carotid a., the left anterior cerebral a. was hypoplastic, and the 3 anterior cerebral vessels originated from the right anterior cerebral a. (type D5).

In 3% of the cases, the absence of the anterior communicating a. was associated with hypoplasia of the left posterior communicating a. (type E). In 2% of the cases, absence of the posterior communicating as. was associated with a left for: vaso-occlusive disease of the supraaortic trunks, diabetes, hypertension, ischemic or hemorragic brain lesions demonstrable by magnetic nuclear resonance (MNR). Each subject underwent cerebral MRA, including examination of the parenchyma and vessels. A Philips Gyroscan T5-NT instrument was used. The following parameters were also registred for each patient: sex, age and Body Index (BI), obtained by the formula: BI = weight/height. The morphologic variants of the circle of willis were classified in groups. Possible differences between males and females, and between right and left side, as regards the occurrence of anomalies and BI was investigated by the evaluation of correlation coefficient (Cr).

RESULTS

Fig. 1 is a schematic drawing of the morphologic variants of all circle of Willis examined. The pattern classically described as «normal» (type A) constituted only 41% of the cases (*fig. 2*). In 21% of considered cases (type B), hypoplasia of the posterior communicating aa. was present. Such hypoplasia was bilateral (type B1) in 12% and monolateral (type B2) in 9%; the latter 9 hypoplasiae were subdivided as follows: 6 were left-sided and 3 were right-sided. In 13% (type C), the principal anomaly regarded the posterior cerebral as., which originated from the internal carotid a.. In 5 out of these 13 cases (type C1), this anomaly occurred monolaterally (4 on the left, 1 on the right side); in 4 cases the anomaly occurred bilaterally, [3 subjects had moderate hypoplasia (type C2) and 1 had marked hypoplasia (type C3) of the segments connecting the basilar a., which represent the pre-communicating segment of the posterior cerebral as.]; in 2 cases (type C4), apart from a posterior cerebral a. originating from the internal carotid (the left in both cases), there was hypoplasia of the posterior communicating a. (on the left side in 1 case, on the right side in 1 case); in the remaining 2 cases, a posterior cerebral a. arising from the left internal carotid was lacking of any connection with the basilar a. (type C5).

In 9% of the examined cases, 3 anterior cerebral vessels were present (type D). Out of these 9 cases, 2 were otherwise «normal» (type D1) and 2 were associated with hypoplasia of the posterior communicating a. (2 on the left, 1 on the right side) and with a posterior cerebral a. originating from the internal carotid (2 on the left, 1 on the right side) (type D2). In 2 cases, 2 of the 3 anterior cerebral vessels originated from the right anterior cerebral a., while a thin segment joined them to the left anterior cerebral a. (type D3). Two type D cases were associated with hypoplasia of the left posterior communicating a. (type D4), and in the remaining case, the left posterior cerebral a. originated from the internal carotid a., the left anterior cerebral a. was hypoplastic, and the 3 anterior cerebral vessels originated from the right anterior cerebral a. (type D5).

In 3% of the cases, the absence of the anterior communicating a. was associated with hypoplasia of the left posterior communicating a. (type E). In 2% of the cases, absence of the posterior communicating as. was associated with a left

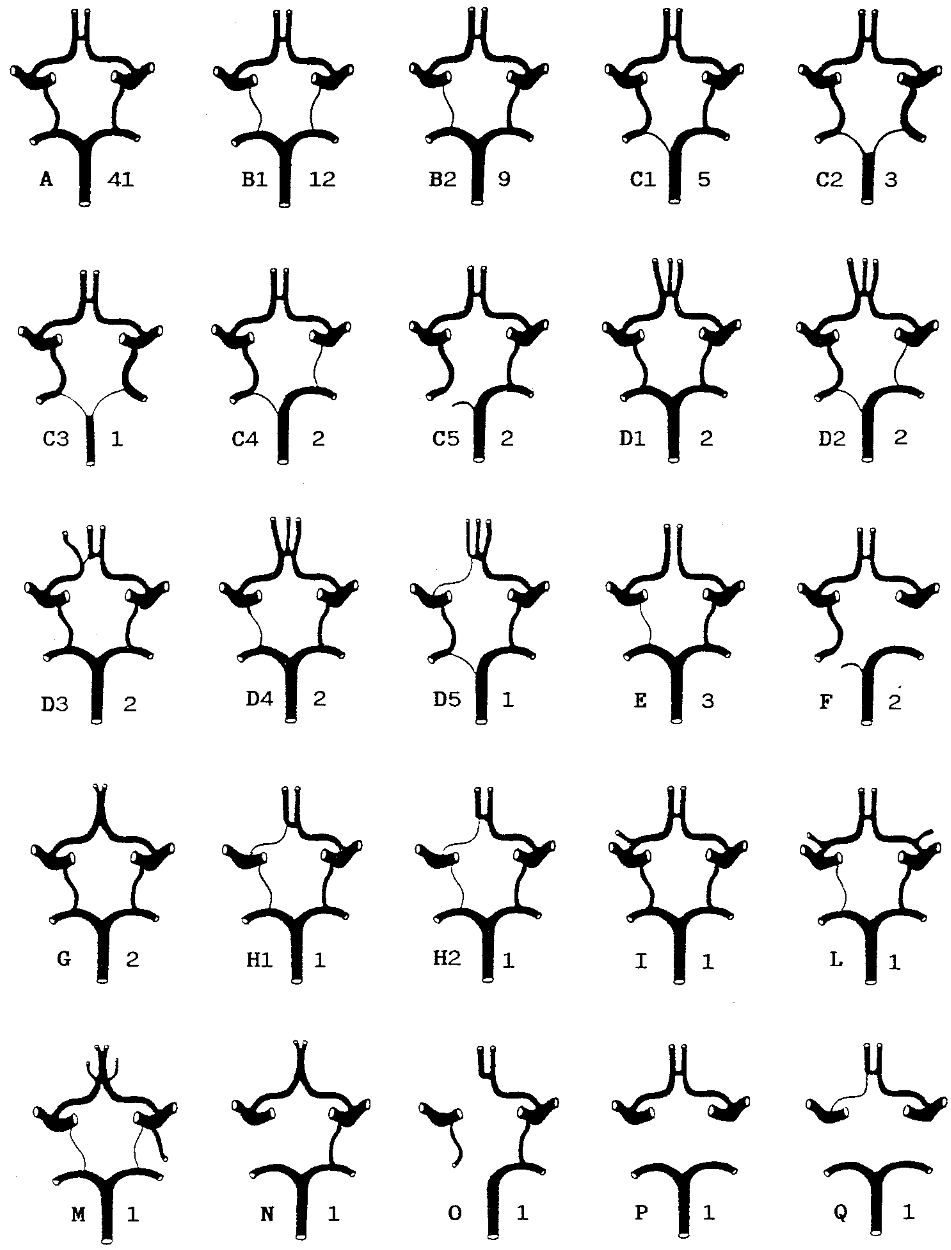

Fig. 1 — schematic drawing of the morphologic variants of the Circle of Willis. The symbol on the left side of each icon represents the morphologic type, wile the number on the right side, the percentage.

communicating a. and fusion of the anterior cerebral as. for a certain lenght (type N); 5) the absence of the left posterior communicating a., which originated from the internal carotid (reduced in caliber with respect to the controlateral a.) and the presence of a left cerebral a. that originated from the right one, with no connections between the as. of the two sides (type O); 6) the absence of the two posterior communicating as. (type P); and 7) the absence of the two posterior communicating as. and hypoplasia of the first tract of the left anterior cerebral a., which at the level of the anterior communicating a. originated from the controlateral vessel, as in types H1 and H2 (type Q).

The statistical evaluation of the morphology of the Circle of Willis with respect to side, sex, and BI demonstrated that anomalies were more frequent in the left than in the right side. In fact the Student's T test led to a probability that the prevalence was casual lower than the confidence limit ($Pt < 0.02$); the difference was, therefore, statistically significant. The most frequent anomaly was the hypoplasia of the posterior communicating a. (34%), which occurred in a highly significant way on the left side ($Pt < 0.01$). As concerns the frequency of the anomalies in males and in females, there were no statistically significant differences ($Pt < 0.05$). Lasty, no statistically significant correlation was demonstrated between the occurrence of arterial anomalies and the BI. The evaluation of r coefficient led to a probability that the correlations were casual higher than the fixed limit ($Pr < 0.05$). Figures 2 and 3 demonstrate the MRAs of a normal case (type A) and case O (type O).

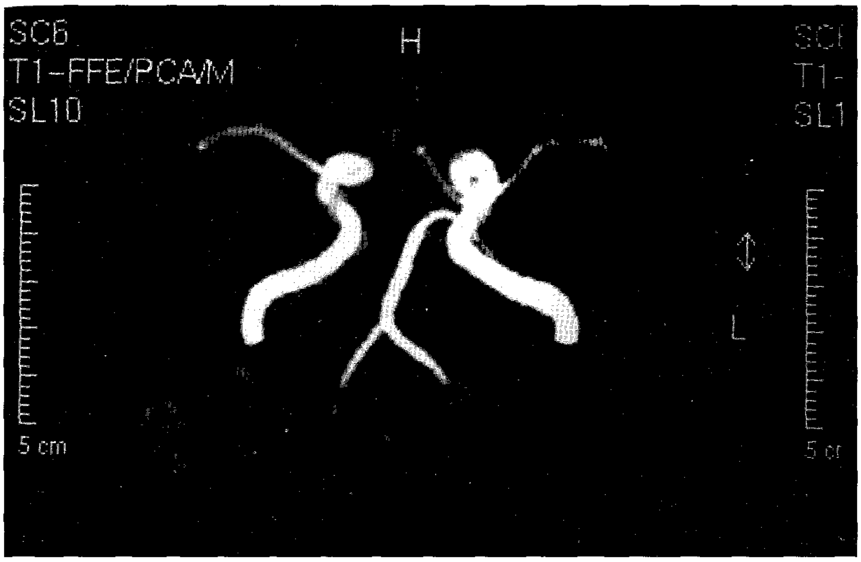

Fig. 3 — magnetic resonance imaging: type O.

DISCUSSION AND CONCLUSION

It is interesting to note that, in the present study, asymmetric and anomalous patterns prevail (59%). Such finding disagrees with the values reported by many anatomists (Peli, 1902; Cavatorti, 1908; Chiarugi, 1912; Mortillaro and Crivelli, 1963; Orlandini, 1970; Orladini et al., 1985) with the exception of Adachi (1928). Moreover, the statistically significant prevalence ($Pt < 0.02$) of the asimmetries and anomalies on the left side, as compared to the right one, should be pointed put. Such prevalence is even greater ($Pt < 0.01$) if one considers hypoplasia of the posterior communicating a., the most frequently observed anomaly: this pattern is present in 34 cases, bilaterally in 21 and monolateral in 13. This observation agrees with the reports of Lanz and Wachsmuth (1979), although these Authors refer lower values (22%). Another frequent anomaly is the origin of a posterior cerebral a., to the internal carotid (19% cases, 15 of which were monolateral). The frequency of this anomaly was also underlined by Mitterwallner (1955), who attributed the origin of the posterior cerebral a., to the internal carotid in 30% of cases, and by Adachi (1928), who reported a percentage of 28% while Lanz and Wachsmuth (1979) reported lower percentages (10%). This finding is well-explained, since in the course of its development the posterior cerebral artery originates from the internal carotid artery, only lateral entering in communication with the basilar artery, and communication with the basilar artery occurs only later.

As concerns the anterior cerebral artery, Wollsclager (1974) reported bilateral hypoplasia in 3.2% and monolateral hypoplasia in 4.1-14% while Tonnis and Schiefer (1959) and Krayenbuhl et al. (1979) described aplasia, respectively, in 0.5 and 1.1%. On the contrary, in the present study, aplasia of the first tract occurred in 1% of cases, hypoplasia of the first segment in 4% of cases, and fusion in the first segment in 3% of cases. However, in all cases the hypoplastic anterior cerebral a. in the first segment was well compensated by means of the controlateral anterior cerebral a., after the anterior communicating a. Furthermore, in 10% of cases, 3 anterior cerebral as. were present, always after the anterior communicating a.

As concerns the anterior communicating a., many variants were described (Adachi, 1928). In this study it was present in 97% and absent in 3%. These values are intermediate between those reported by Sedzimir (1959), (absence of the artery which ranging from 0.5 to 1%), and those of Krayenbuhl and Jasargil (1957), describing absence of anterior communicating a. in 6-8% of subjects. With regard to the middle cerebral a., the present research failed to demonstrate any significant asymmetry or anomaly of this vessel, except in two cases in which small accessory arteries were present. This finding completely agrees with Adachi (1928).

Such great anatomical variability is of acute interest, even if one considers that stenotic lesions of the carotids and vertebral as. are mainly located in the extracranial segment (Hass et. al., 1968). Thus the circle of Willis represents the most important compensatory system of the intracranial arterial pressure and blood distribution. Aplasia or hypoplasia of its vessels could limit the possibility of collateral arch in a poorly-perfused area. The perfect knowledge of the normal anatomy is fundamental in the elevation of the single patient and for a correct prognosis of vasoocclusive disease and for surgical planning. MRA is a realiable, non invasive methodology, and has very good imaging sensivity; moreover it implies very low risk and minimal disturbance for the patient. For all these reasons, MRA deserves special consideration, appearing to be most suitable technique for studying healthy subjects, which is unavoidable in anatomical research.

Arrived 26/6/96. Accepted 3/7/96.

REFERENCES

Aaslid R, Nornes H. 1984. Musical murmurs in human cerebral arteries after subarachnoid hemorrage. J Neurosurg, 60: 32-36.

Adachi B. 1928. Das Arteriensystem der Japaner. Maruzen, Kioto.

Bartholow JR. 1943. Congenital aneurysm of the cerebral arteries: an embryology study. Archs Path, 35: 819-831.

Brice JG, Dowsett DJ, Lowe RD. 1964. Haemodinamic effects of carotid artery stenosis. Brit Med J, 11: 1363-66.

Brown PM, Johnston KW. 1982. The difficulty of quantifying the severity of carotid stenosis. Surgery, 92: 468-73.

Cavatorti P. 1908. Il tipo normale e le variazioni della base dello encefalo. Monitore. Zool Ital, 19: 248-58.

Chiarugi G. 1912. Istituzioni di anatomia dell'uomo. Vol. 2: 1a ediz., Milano, Sel 88: 120.

Contamin F, Mignot B, Wesley C, Ollat H, Brandley M, Henry-Biabaud E. 1983. Occlusive arteriopathy of the circle of Willis with outlined «moya-moya» network in a woman with migraine under oral contraception. Regressive course. Sem Hop Paris, 59: 1387-1392.

Deker K, Hipp E. 1958. Der Basale Gefsskranz. Morfologie und Angiographie. Anat Anz, 105: 100-116.

Dorndorf W, Ganshirt H. 1972. Die Klinik der Arteriellen zerebralen gefabverschlusse. In Granshirt H (Hrsg). Der Hirnkreislauf, Thieme, Stuttgard, 512-650.

Elliot-Smith MA. 1909. Note on anomalus anastomosis between the internal carotid and the basilar artery. J Anat phisiol, 43: 310-11.

Fazio C. 1969. Haemodinamic Factors in the Patogenesis of Brain Infarct. Europ Neurol, 2: 76.

Fazio C, Fieschi C, Regesta C. 1970. Frequenza e ruolo delle variazioni del poligono di Willis nella apoplessia cerebrale. Sist Nerv, 22: 286-92.

Flanigan PD, Tullis JP, Streeter VL, Frey WG, Stanley JC. 1977. Multiples subcritical arterial stenosis: effect on postenotic pressure and flow. Ann Surg, 186: 663-668.

Gulisano M, Zecchi S, Pacini G, Orlandini GE. 1982. The behaviour of some human arteries as regards the correlated circumference: a statistical research. Anat Anz, 152: 341-357.

Hass WK, Fields WS, North RR, Kasse NE, Bauer RB. 1968. Joint study of extracranial arterial occlusion. Arteriography, techniques, sites and complications. J Amer Med Ass, 203: 9961-8.

Iivanainen M, Vuolio M, Halonen V. 1973. Occlusive disease of intracranial main arteries with collateral networks (moya-moya disease in adults). Acta Neurol Scand, 49: 307-332.

Kitahara T, Ariga N, Yamura A, Makino H, Maki Y. 1979. Familiar occurrence of moya-moya disease: report of three japanese families. Neurol Neurosurg Psychiatr, 42: 208-214.

Krayenbühl HA, Jasargil G. 1957. Die Vascularen Erkankungen im Gebiet der A. Vertebralis und A. Basilaris. Eine Anatomische und Pathologische, Klinische und Neuroradiologische Studie. Thieme, Stüttgart.

Krayenbuhl HA, Jasargil P, Huber P. 1979. Zerebrale angiographie fur klinik und praxis. Thieme Stuttgart.

Kudo T. 1969. Spontaneus occlusion of the circle of Willis: a disease apparently confined to japanese., Neurology, 18: 485-496.

Lanz T, Wachsmuth W. 1979. Praktische Anatomie, Springer, Berlin, p. 1327.

Macchi C, Catini C. 1994. The measurement of the calibres and blood flow velocities of the arteries of the circle of Willis: a statistical investigation of 120 living subjects using transcranial colordoppler ultrasonography. It J Anat Embryol, 99, 1: 9-16.

Mitterwallner F. 1955. Variationsstatistische Untersuchungen an den basalen Hirngefaben. Acta Anat, 24: 51-88.

Mori A. 1893. Sulla disposizione delle arterie della base dell'encefalo nei normali e negli alienati. Monitore Zool Ital, 4: 179-202.

Mortillaro F, Crivelli G. 1963. Le variazioni e le anomalie del poligono di Willis. Folia Hered Pathol, 13: 355-384.

Nenci G. 1945. Anomalie, varietà ed ipoplasie del circolo di Willis come fattori patogenetici meno noti dell'emorragia cerebrale. Arch De Vecchi, 8: 267-9.

Orlandini GE. 1970. La circonferenza rettificata delle principali arterie della base dell'encefalo: ricerca statistica su 100 casi umani. Arch It di Anat Embriol, 75: 49-79.

Orlandini GE, Ruggiero C, Zecchi S, Gulisano M. 1985. Blood vessel size of circulus arteriosus cerebri (circle of Willis): a statistical research on 100 human subjects. Acta Anat 123: 72-76.

Peli G. 1902. Il calibro delle principali arterie della base dell'encefalo nei sani di mente e negli alienati. Bull SCI Med, 73: 537-47.

Pensa A, Favaro G. 1935. Trattato di anatomia umana sistematica. Utet, Torino, II 44-51.

Ruggiero C, Zecchi Orlandini S und Orlandini GE. 1987. Morphometrie des hinteren Teiles des Circulus arteriosus Willisi. Verh Anat Ges, 81: 401-402.

Schmitt HP. 1978. Die Bedeutung von Varianten im Bereich der Arteriae Vertebrales und der arteria Basilaris fur Forensische Medizin. 2, Rechtsmed, 73: 23-28.

Sedzimir CB. 1959. An angiographic test of collateral circulation through the anterior segment of the circle of Willis. J Neurol Neurosurg Psychiat, 22: 64-68.

Slany A. 1938. Anomalien des Circulus Arteriosus Willisi in iher Beziehung zur Aneurysmen Bildungen an der Hirnabasis. Wirchows Arch. 301: 62-71.

Spencer MP. 1987. Haemodinamics of arterial stenosis. Ultrasonic diagnosis of cerebrovascular diseases, 1a ediz. Dordrecht: Nijhoff 117-46.

Tardini A. 1954. Anomalie anatomiche, variazioni metriche strutturali delle arterie del «Circulus Willisi Anterior» in rapporto alla patogenesi degli aneurismi. Riv Anat Patol Oncol, 8: 521-76.

Testut L, Latarjet A. 1972. Trattato di anatomia umana. 5a Edizione, Torino, Utet pp. 313-317.

Tonnis W, Schiefer W. 1959. Zirkulationsstorungen des Gehirns im Serienangiogramm. Berlin, Springer pp. 514-523.

Weber G, Grechi G, Colafranceschi M. 1977. Esistono o no interdipendenze tra ipoplasia arteriosa e arteriosclerosi? Saggio di una ricerca a livello dell'arteria comunicante posteriore nel circolo di Willis. Arch De Vecchi, 48: 29-35.

Wollschlager PB, Wollschlager B. 1974. The anterior cerebral-internal carotid and middle cerebral internal carotid arteries. Acta Radiol Diagn, 5: 615-620.

Address:
Prof. Claudio Catini
Dipartimento di Anatomia Umana e Istologia
Policlinico di Careggi
50134 - Firenze - Italia
Phone: +39 55 437 9500; Fax: +39 55 410 084