

The perforating arteries located in the quadrilateral space are supplied by the arteries that border the space. The perforating arteries leaving the VA, the vertebrobasilar junction or even the basilar artery if the vertebrobasilar junction is lower than the foramen caecum of Vicq d'Azyr adopt a lateral transverse or oblique course over the pyramid; the branches of these either penetrate the medulla in the preolivary sulcus, or continue over the olive to perforate the retroolivary sulcus (Figures 2–6). AICA gives off descending perforating branches, which usually penetrate the medulla supra- or retro-olivary (Figures 1, 5, 6 and 9). At the level of the pontomedullary sulcus, the transverse or oblique perforators sent off by the basilar or vertebral arteries course and branch superficially to those perforators descending from the AICA (Figure 9). The perforating arteries can describe more or less defined loops.

Discussion

There is insufficient data in the literature related to the microanatomic features of the perforating branches of the vertebral artery [23]. It has been shown that PICA, AICA and VA occasionally compress the medulla oblongata and that patients with essential hypertension are associated with neurovascular compression of the RVLM at the root entry zone of the ninth and tenth cranial nerves, in clinical observations and magnetic resonance imaging (MRI) studies [13]. However, the microvascular layers inside the quadrilateral space we defined here at the level of the RVLM were overlooked as being possible offending vessels able to trigger the tonic sympathetic discharge from the neurons of the RVLM.

Consistent with our results, the location of the origin of the supra- and retro-olivary perforator arteries may vary: the preolivary sulcus is primarily supplied by the anterior spinal artery, the upper portion of the posterior olive is supplied with perforators by the VA, AICA and basilar artery (BA), while the middle and lower portions of the posterior aspect of olive were fed by the VA and PICA [24].

Experimental studies using rats indicated that pulsatile compression of the RVLM increases arterial pressure by enhancing sympathetic outflow [13]. These results would indicate rather the first two vascular layers over the RVLM, which are arterial and thus pulsatile, than the deep venous one, as being involved in the essential hypertension pathogeny. Noteworthy, the relation/contact of the vessels in the superficial vascular layer of the quadrilateral space (VA, AICA or PICA) with the RVLM may not be direct, but mediated by the arterial perforators and the veins inside the respective space.

Major vessels, arteries and veins, but also small-unspecified arteries, are taken into consideration as possible offending vessels of the trigeminal nerve in the cerebellopontine angle [25]. Similarly, small vessels should be taken into account when the vascular relations of the RVLM and the root entry zone of the ninth and tenth cranial nerves are considered as sites of vascular contacts or compressions.

Conclusions

Microanatomical studies of the vascular relations of the RVLM are able to complete the findings of imaging studies, which are limited by spatial and contrast resolution of imaging techniques. The offending vessels of the RVLM could be any of the vessels inside the quadrilateral space. Major vessels, such as the VA, AICA or PICA should not be viewed as the only possible offending vessels at this level. The perforators and the venous layers in the quadrilateral space should also be better evaluated.

Acknowledgments

This study was supported by the Sectoral Operational Programme Human Resources Development (SOP HRD), financed from the European Social Fund and by the Romanian Government under the contract number POSDRU/89/1.5/S/64153 (author #1).

References

- [1] Naraghi R, Geiger H, Crnac J, Huk W, Fahlbusch R, Engels G, Luft FC, *Posterior fossa neurovascular anomalies in essential hypertension*, Lancet, 1994, 344(8935):1466–1470.
- [2] Menzel C, Geiger H, *Neurovascular contact of cranial nerve IX and X root-entry zone in hypertensive patients*, Hypertension, 2001, 37(6):E25.
- [3] Hohenbleicher H, Schmitz SA, Koennecke HC, Offermann R, Offermann J, Zeytounchian H, Wolf KJ, Distler A, Sharma AM, *Neurovascular contact of cranial nerve IX and X root-entry zone in hypertensive patients*, Hypertension, 2001, 37(1):176–181.
- [4] Nicholas JS, D'Agostino SJ, Patel SJ, *Arterial compression of the retro-olivary sulcus of the ventrolateral medulla in essential hypertension and diabetes*, Hypertension, 2005, 46(4):982–985.
- [5] Coffee RE, Nicholas JS, Egan BM, Rumboldt Z, D'Agostino S, Patel SJ, *Arterial compression of the retro-olivary sulcus of the medulla in essential hypertension: a multivariate analysis*, J Hypertens, 2005, 23(11):2027–2031.
- [6] Jannetta PJ, Segal R, Wolfson SK Jr, *Neurogenic hypertension: etiology and surgical treatment. I. Observations in 53 patients*, Ann Surg, 1985, 201(3):391–398.
- [7] Jannetta PJ, Segal R, Wolfson SK Jr, Dujovny M, Semba A, Cook EE, *Neurogenic hypertension: etiology and surgical treatment. II. Observations in an experimental nonhuman primate model*, Ann Surg, 1985, 202(2):253–261.
- [8] Wilkins RH, *Neurovascular compression syndromes*, Neurol Clin, 1985, 3(2):359–372.
- [9] Yamamoto I, Yamada S, Sato O, *Microvascular decompression for hypertension – clinical and experimental study*, Neurol Med Chir (Tokyo), 1991, 31(1):1–6.
- [10] Hoff JT, *Neurovascular compression and essential hypertension*, J Neurosurg, 1992, 77(1):101–102.
- [11] Naraghi R, Gaab MR, Walter GF, Kleineberg B, *Arterial hypertension and neurovascular compression at the ventrolateral medulla. A comparative microanatomical and pathological study*, J Neurosurg, 1992, 77(1):103–112.
- [12] Morimoto S, Sasaki S, Miki S, Kawa T, Itoh H, Nakata T, Takeda K, Nakagawa M, Kizu O, Furuya S, Naruse S, Maeda T, *Neurovascular compression of the rostral ventrolateral medulla related to essential hypertension*, Hypertension, 1997, 30(1 Pt 1):77–82.
- [13] Morimoto S, Sasaki S, Miki S, Kawa T, Itoh H, Nakata T, Takeda K, Nakagawa M, Naruse S, Maeda T, *Pulsatile compression of the rostral ventrolateral medulla in hypertension*, Hypertension, 1997, 29(1 Pt 2):514–518.
- [14] Naraghi R, Schuster H, Toka HR, Bähring S, Toka O, Oztekin O, Bilginturan N, Knoblauch H, Wienker TF, Busjahn A, Haller H, Fahlbusch R, Luft FC, *Neurovascular compression at the ventrolateral medulla in autosomal dominant hypertension and brachydactyly*, Stroke, 1997, 28(9):1749–1754.