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The conflicting vessels in hemifacial spasm: Literature review and anatomical-surgical implications

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#### ABSTRACT

The conflicting vessels in hemifacial spasm: literature review and anatomical-surgical implications. Since several decades, it has been established that so-called primary hemifacial spasm is linked to neurovascular conflicts in the facial nerve, especially its root exit zone (REZ). Based on our review of the detailed publications of literature (2489 patients), together with our own series (340 patients), the responsible vessels found at surgery were: the posterior inferior cerebellar artery in 47.2%, the anterior inferior cerebellar artery in 45.9%, the vertebro-basilar artery in 17.5%, another (smaller) artery in 11.7%. Participation of veins was very diversely estimated according to series: 4.9% on average. Multiple neuro-vascular conflicts in a same individual were frequently observed, in the order of 20 to 30% according to authors, 37% in our series. Also, abnormal conformation of the posterior fossa may play a role, such as flatness of the posterior fossa or exiguity of the cerebello-pontine angle cistern. Whatever, most neurovascular conflicts are located at brainstem and/or ventrocaudally to the facial REZ, in the order of 95% of the patients. The anatomical location and conformation of the compressive vessel(s) are crucial in determining the difficulties to identify the responsible conflict(s) and to perform effective and safe decompression. Main difficulties are encountered in cases with arteriosclerotic megadolicho-vertebrobasilar artery, at brainstem, especially when PICA and/or AICA come in association, or for neurovascular conflict(s) located at the cisternal or the intrameatal portions of the facial root. Later ones can be alone or in addition to NVC at brainstem/REZ.

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Dandy was the first to bring attention to the link between hyperactive symptoms and compression of cranial nerves in the cerebellopontine cistern, not only by tumors but also by elongated arteries [1]. His hypothesis was based on observations in patients affected with trigeminal neuralgia and operated on for tumor removal or juxta-pontine rhizotomy. However, Dandy did not reach the point to propose pure vascular decompression in these patients harbouring neuro-vascular-compression (NVC).

In the late forties, cases with apparently primary hemifacial spasm (HFS) due to vascular compression were reported in several publications [2–4].

The real pioneer of the concept of vascular decompression for NVC was James Gardner [5]. Same year, 1962, Gardner and Sava reported their experience in 19 cases of HFS operated on by a non-lesioning decompression in the eleven patients where an arterial abnormal vessel was found [6]. Then Peter Jannetta in

\* Corresponding author. *E-mail address:* philippe.mercier@univ-angers.fr (P. Mercier). 1970 introduced the modern micro-vascular decompression (MVD) operation [7] and popularized it through a large number of publications, followed by Jules Hardy in Montreal [8] and later on by many disciples all over the world.

The present work consisted of getting maximum information on primary HFS from literature through the PubMed system and its well-documented articles, together with our own patients' series.

#### 1. The various conflicting vessels

Overall, based on the general literature overview, it appears that an offending vessel was found in almost all the patients who underwent MVD for HFS, exploration being reported negative in 1 to 2% according to authors, none in our own series that totalized 340 cases. These percentages are perhaps underestimated, due to the fact that patients in whom clear-cut NVC was found on MRI did not undergo a MVD and therefore from posterior fossa exploration. Overall, arteries were considered the conflicting vessels in nearly all the cases. A vein was considered as possibly responsible in approximately 10%, either in association with an artery or more rarely as

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## Table 1Distribution of conflicting vessels in hemifacial spasm.

Only first surgery and series with details	AICA	PICA	VBA	Other arteries	Veins with arteries	Veins alone	Other causes or nothing found	Remarks
Auger et al. (1986) [10], n=54 pts	20 pts (37%)	14 pts (26%)	9 pts (16.6%)	7 pts (15%)	3 pts	(5.5%)	No = 1	
Huang et al. (1992) [11], n = 310 pts	121 pts (39%)	71 pts (23%)	11 pts (3.5%)	102 pts (32.8%) 132 pts (20.4%)	2 pts	(0.6%)	No = none	
Barker et al. (1995) <mark>[12]</mark> , n = 648 pts	229 pts (35.3%)	442 pts (68.2%)	157 pts (24.2%)	Lab = 19 (2.9%) No named 109 (16.9%) SCA = 4 (0.6%)	19 pts	(2.9%)	No = none	
Shin et al. (1997) [13], n=261 pts	169 pts (64.8%)	73 pts (27.9%)	52 pts (19.9%)	3 (1.2%) Not specified	NA	NA	NA	Multiple conflicts (13%)
Samii et al. (2002) [14], n = 145 pts	38 pts (26.2%)	105 pts (72.5%)	31 pts (21.4%)	0	2 pts (1.4%)	6 pts (4.1%)	No = none	
Naraghi et al. (2007) [15], n=25 pts	15 pts (60%)	7 pts (28%)	3 pts (12%)	0	NA	NA	No = none	
Park et al. (2008) [16], n = 236 pts	175 pts (74.2%)	84 pts (35.6%)	47 pts (15.7%)	0	0	1 pt (0.4%)	No = none	
Campos and Kaufmann (2008) [17], <i>n</i> = 115 pts	49 pts (43%)	36 pts (31%)	27 pts (23%)	NA	NA	3 pts (2.6%)	NA	Multiple conflicts (38%) Overall conflicts at REZ in 97% In cistern 3%
Sindou and Keravel (2009) [18], <i>n</i> = 240 pts	94 pts (39%)	163 pts (67.8%)	66 pts (27.5%)	0	19 pts (7.9%)	2 pts (0.8%)	No = none	Overall conflicts
Zhong et al. (2014) [19], n = 393 pts	141 pts (36,7%)	204 pts (51.9%)	35 pts (8.9%)	51 pts (12.9%)	3 pts (0.7%)	3 pts (0.7%)	NA	At REZ in 93% In cistern in 7%
Lee et al. (2015) [20], n = 108 pts	69 pts (63.6%)	45 pts (41.6%)	20 pts (18.5%)	None	None	None	NA	Multiple conflicts (24%)
Soriano-Revuelta et al. (2015) [21], <i>n</i> = 194 pts	147 pts (75.8%)	12 pts (6.2%)	10 pts (5.1%)	35 pts (12.8%)	NA	NA	NA	

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