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ORIGINAL ARTICLE - FUNCTIONAL NEUROSURGERY - MOVEMENT DISORDERS

# Arachnoid bands and venous compression as rare causes of hemifacial spasm: analysis of etiology in 353 patients

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## Abstract

**Background** Hemifacial spasm is usually caused by arterial compression at the root exit zone of the facial nerve. However, other etiologies have been reported. The aim of this study was to analyze the frequency of other causes of hemifacial spasm.

**Methods** Our prospectively maintained hemifacial spasm database containing all patients who underwent microvascular decompression (MVD) for hemifacial spasm from 2002 to 2018 was reviewed. All offending structures were identified and recorded by the surgeon at the time of surgery. Additionally, the operative videos were analyzed retrospectively.

**Results** MVD was performed in 353 patients. Arterial compression was the main cause of hemifacial spasm in 341 (96.9%) patients. Combined venous-arterial compression was seen in 7 (2.0%) patients. In one patient, the compression was from a large vein. In two patients, no compression was found. One patient who suffered from Bell's palsy many years previously had severe synkinesis and the other had facial tics. In two patients, the spasm was caused due to strangulation of the facial nerve by arachnoid bands. Long-term follow-up of more than 18 months was available in 249 patients with total resolution or near total resolution of spasms in 89.96% of patients.

**Conclusions** In most patients with hemifacial spasm, arterial vessels are involved in compressing the facial nerve. Purely venous compression is rarely encountered. We report for the very first time arachnoid bands strangulating the nerve as a cause for hemifacial spasm without involvement of any vessel.

**Keywords** Hemifacial spasm · Etiology · Arachnoid band · Microvascular decompression · Outcome · Endoscope-assisted microsurgery

## Introduction

Hemifacial spasm is characterized by progressive, involuntary, irregular, clonic or tonic movement of the muscles innervated by the facial nerve [31]. In most cases, the etiology is attributed to arterial compression at the root exit zone of the facial nerve. However, other etiologies that may cause hemifacial spasm have been reported [32]. Hemifacial spasm can occur due to other rare causes including all kinds of space-occupying lesions in the cerebellopontine angle. In addition, certain processes in the brainstem can also result in symptoms. These include gliomas, demyelinating neurological disorders such as multiple sclerosis or brainstem infarction [26, 31, 32].

In this study, we analyzed data from our database in order to identify the etiology of hemifacial spasm.

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## Methods

### Data collection

The local ethics committee approved this retrospective study. Our prospectively maintained hemifacial spasm database containing all patients who underwent microvascular decompression (MVD) for hemifacial spasm from 2002 to 2018 was retrospectively reviewed regarding the cause for facial nerve

root compression and outcome. Patient data were prospectively collected including preoperative clinical picture, magnetic resonance (MR) imaging findings, operative notes, intraoperative videos, and postoperative outcome at last follow-up. Additionally, the operative videos were analyzed retrospectively to confirm the intraoperative findings. Routine follow-up was done both by outpatient visits and telephone calls. The compression of the facial nerve was assessed radiologically and confirmed intraoperatively by endoscopic visualization of the facial nerve root exit zone (REZ). Etiological factors were identified and analyzed.

Subgroup analysis regarding outcome was performed based on cause of hemifacial spasm. The outcome was divided into 4 grades: (1) excellent (hemifacial spasm absent), (2) good (hemifacial spasm> 90% resolved), (3) fair (hemifacial spasm 90-50% resolved), and (4) poor (hemifacial spasm< 50% resolved). Ninety percent improvement means that no visible spasms occurred or rarely slight spasms were observed that did not occur daily.

### Operative technique

All surgeries were performed via an inferior retrosigmoid approach in the supine position under brainstem auditory evoked potentials and facial electromyography monitoring [9, 10]. An endoscope-assisted microscopic technique as described elsewhere was applied [11, 25]. The endoscope was used to inspect the course of the facial nerve from the pontomedullary sulcus to the entrance into the internal auditory canal. Decompression was achieved by interposing shredded Teflon or by transposing the vessel with a Teflon or Gortex sling which was fixed to the basal dura with a suture or aneurysm clip. All MVDs were performed by the senior author (HWSS) who prospectively recorded the compression pattern at the time of surgery.

## Results

### Patient demographics

A total of 353 patients suffering from hemifacial spasm underwent MVD in our institution from 2002 to 2018. We offered surgery for all patients even when the symptoms were mild, but the patients felt uncomfortable. Some patients were suffering tremendously even from mild spasms. However, most patients had severe functionally impairing spasms. The only exclusion criteria was a high risk for general anesthesia because of comorbidity. There were 136 (38.5%) male and 217 (61.5%) female patients. The symptoms were left-sided in 211 and right-sided in 142 patients. The mean age was 54.78 years ranging from 22 to 81 years. The mean duration of the spasms was 7.78 years ranging from less than 1 to 35 years. Mean follow-up was 51.64 months ranging from 0 to 194 months.

In 249 patients with a follow-up of at least 18 months, the cause of compression and degree of improvement were analyzed.

### Compression pattern and outcome

The causes of compression in all patients were collected in Table 1. Additionally, we had one patient with hemifacial spasm caused by an epidermoid who was excluded from the study because no MVD was performed, but a tumor resection which resulted in complete resolution of the spasms. Arterial compression was the main cause of hemifacial spasm in 341 (96.6%) patients with different combinations of vessels. The most frequent offending vessel was posterior inferior cerebellar artery (PICA) (122, 34.6%) followed by anterior inferior cerebellar artery (AICA) (90, 25.5%) and VA (11, 3.12%). Complex arterial compression was seen in 118 patients. The most frequently observed complex compression was a combination of AICA + VA (41, 11.6%) followed by PICA + VA (38, 10.8%), AICA + PICA (26, 7.37%), AICA + PICA + VA (12, 3.4%), and AICA + BA (1, 0.28%). The outcome in the arterial compression group in patients with at least one and half year follow-up ($n$ = 240) was excellent, good, fair, and poor in 194 (80, 83%), 24 (10.0%), 18 (7.50%), and 4 (1.66%) respectively. PICA was the most common cause of compression in 83 patients, where total resolution occurred in 66 patients and near total resolution in 8 patients.

Combined venous-arterial "sandwich" compression was detected in seven (2.0%) patients. A vein was compressing the facial nerve together with AICA and PICA in four (1.1%) and three (0.9%) patients respectively. In one patient, the vein was located above the facial nerve. In six cases, the vein was located between brainstem and facial nerve running

**Table 1** Cause of compression

| Cause of compression | Number (= 353) |
|---|---|
| VA | 11 (3.12%) |
| PICA | 122 (34.6%) |
| PICA + vein | 3 (0.85%) |
| PICA + VA | 38 (10.8%) |
| AICA | 90 (25.5%) |
| AICA + vein | 4 (1.1%) |
| AICA + BA | 1 (0.28%) |
| AICA + VA | 41 (11.6%) |
| AICA + PICA | 26 (7.37%) |
| AICA + PICA + VA | 12 (3.4%) |
| Arachnoid band | 2 (0.57%) |
| Vein | 1 (0.28%) |
| No compression | 2 (0.57%) |

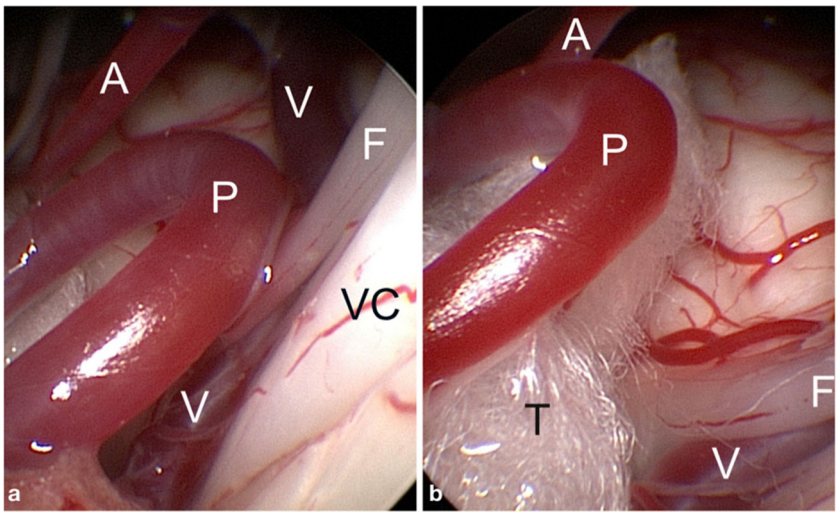


**Fig. 1** Endoscopic view showing a sandwich compression of the facial nerve (F) by a vein (V) and PICA (P). AICA (A) and vestibulocochlear nerve (VC) are seen as well (**a**). After decompression of PICA (P) with shredded Teflon (T) the facial nerve (F) is free (**b**). The vein was not dissected or decompressed with Teflon. Nevertheless, the patient was without spasms immediately after surgery (follow-up 33 months)

between facial and vestibulocochlear nerve down to the pontomedullary sulcus. Because of this unfavorable course, the veins could not be decompressed sufficiently. All attempts to mobilize these veins failed. Furthermore, we did not coagulate the veins because of the unpredictable sequelae. Only in one patient, a little piece of shredded Teflon could be interposed between facial nerve and vein. This patient is without spasms 59 months after surgery. In all other patients, only the arteries were mobilized and decompressed (Fig. 1). Three patients had an excellent and one patient a good outcome (follow-up 33–71 months). In two patients with a large vein running between facial nerve and brainstem where the vein was flattening the facial nerve at the REZ, the outcome was fair and poor respectively. The patient with the poor outcome had additionally a very narrow cistern (Fig. 2). The AICA was decompressed, but he still needs botox injections to control the spasm 98 months after surgery. The other patient improved after PICA decompression and had a fair outcome after 71 months follow-up (Fig. 3).

A purely venous compression was seen in one patient only. It was a large vein, running between facial and vestibulocochlear nerve down to the pontomedullary sulcus. Together with its side branch, it compressed the facial nerve (Fig. 4a). After separating the vein from the facial nerve, a flattening of the nerve was clearly seen (Fig. 4b). The facial nerve was decompressed by transposing the vein laterally with the aid of a Teflon sling which was fixed with a stitch to the dura between jugular foramen and internal auditory canal (Fig. 4c). The patient had been spasm-free immediately after the surgery (follow-up 88 months).

No compression was found in two patients. One patient did not mention to us that he had facial palsy (Bell's palsy) several years previously. The spasms looked very similar to hemifacial spasm, but were obviously synkinesis after facial nerve damage. Not surprisingly, he did not improve. The other patient was misdiagnosed as hemifacial spasm and after surgery it was proven to be facial tics which were verified by absence of lateral spreads. She did not improve as well.

In two (0.6%) patients, the hemifacial spasm was caused by an unusual strangulation of the facial nerve at the REZ by arachnoid bands. Endoscopic inspection with a 45° endoscope confirmed that there was no vascular compression. In neither case was there a history of meningitis, head injury, or

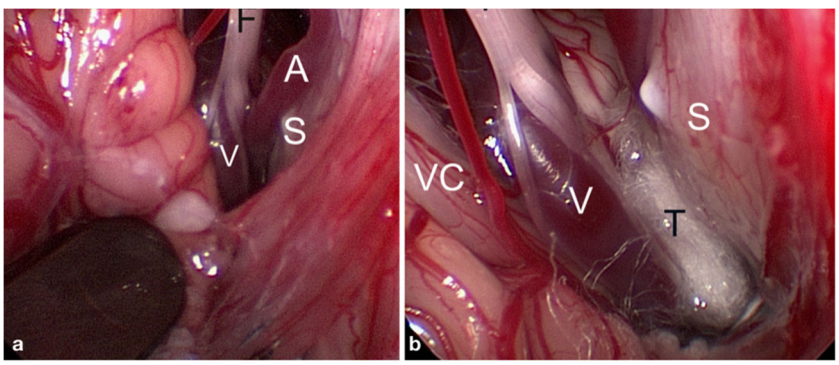


**Fig. 2** Endoscopic view showing a sandwich compression of the facial nerve (F) by a vein (V) and AICA (A) (**a**). Note the extremely narrow subarachnoid space between brainstem and skull base (S). The AICA was transposed anteriorly and fixed with a Teflon (T) pledget (**b**). The vein was not dissected or coagulated because of the risk of facial palsy or venous congestion. The patient had a poor outcome 98 months after surgery

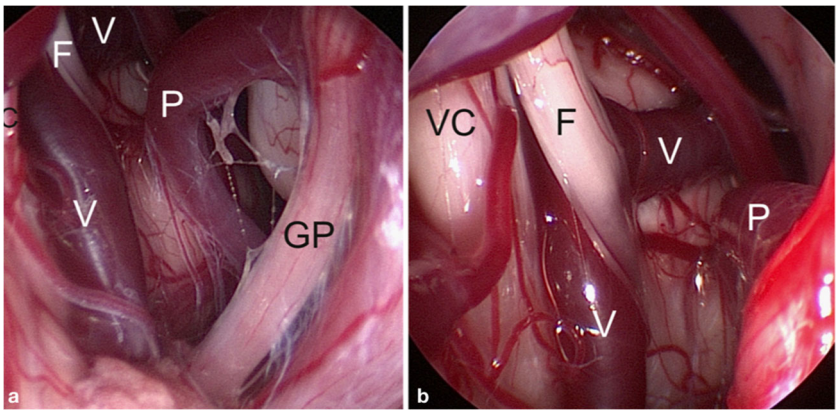


**Fig. 3** Endoscopic view showing a sandwich compression of the facial nerve (F) by a vein (V) and PICA (P). VC vestibulocochlear nerve, GP glossopharyngeal nerve (**a**). The PICA was already decompressed simply by arachnoid dissection. The close-up shows the tiny side branches of the vein draining the brainstem. The vein was not mobilized or coagulated because of the risk of facial palsy or venous congestion (**b**). The patient had a fair outcome 94 months after surgery.

hemorrhage. The first patient was a 41-year-old male who had complained of hemifacial spasm for 10 years. He presented with a slight facial palsy (HB 2) because of previous botulinum toxin injections. Time-of-Flight (TOF) and Constructive interference in steady state (CISS) MR imaging revealed no vascular compression (Fig. 5a, b). Surgical exploration showed a small vein running close to the facial REZ above the nerve and a small arteriole, but these vessels did not cause any compression However, a little bit more distally a clear strangulation of the facial nerve by several arachnoid bands became apparent. (Fig. 5c, d). The arachnoid bands were cut with microscissors to release the nerve (Fig. 5e, f). Immediately after surgery the spasm disappeared. The patient had no complaints 26 months after surgery.

The second patient was a 32-year-old male who had suffered from hemifacial spasm for 20 years. TOF and CISS MR imaging showed no vascular compression, but an asymmetry of the facial nerve when compared with the other side (Fig. 6a, b). The left facial nerve seemed to be anteriorly displaced. Endoscopic inspection revealed a severe strangulation of the facial nerve by arachnoid bands (Fig. 6c). On the dorsal side of the nerve, an unusual gliotic band was identified which seemed to pull the nerve anteriorly (Fig. 6d). The bands and the fibrotic perineurium were cut. Careful elevation of the nerve showed significant damage of the nerve at the area of the former strangulation (Fig. 6e). The final inspection showed the released nerve (Fig. 6f). The patient was spasm-free immediately after surgery. Nine days later, he developed a delayed facial palsy (HB 6). Fortunately, the palsy resolved completely 50 day after surgery. He was doing well without spasms 15 months after surgery.

**Overall outcome** The overall outcome of 249 patients with a minimum follow-up of 18 months is shown in Table 2. 198 (79.52%) patients were spasm-free. A total of 26 (10.44%) patients had a good and 19 (7.63%) a fair outcome. In 6 (2.41%) patients, we observed a poor outcome. The reason for a poor outcome was mostly a structural damage of the facial nerve due to prolonged severe compression by elongated vessels and in one patient mentioned above a combination of severe venous compression.

## Long-term outcome according to type of compression

The range of improvement according to different types of compression is shown in Table 2. In arterial compression,

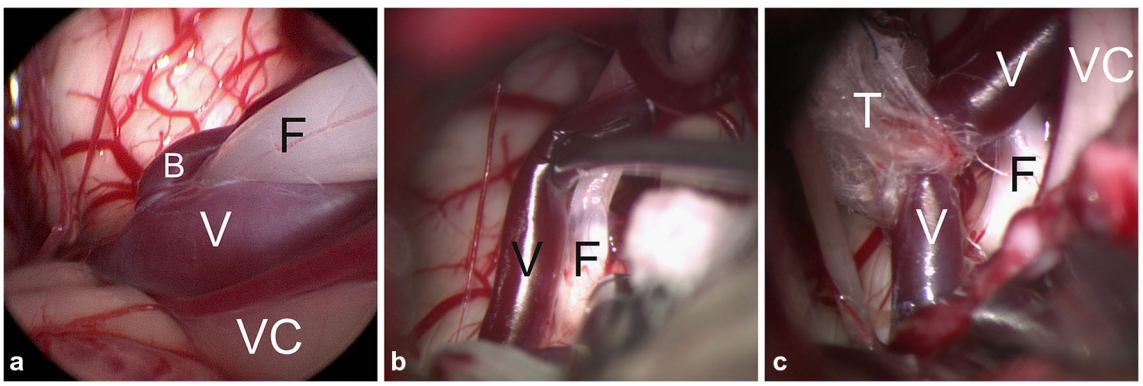


**Fig. 4** Endoscopic view showing a purely venous compression of the facial nerve (F) by a major vein (V) and its side branch (B). VC vestibulocochlear nerve (**a**). After the vein (V) had been dissected, the thinned facial nerve (F) was seen (**b**). After decompression of the facial nerve (F), the vein (V) was transposed with the aid of a Teflon sling (T) which was sutured to the basal dura (**c**). The patient had an excellent outcome immediately after surgery (follow-up 88 months)

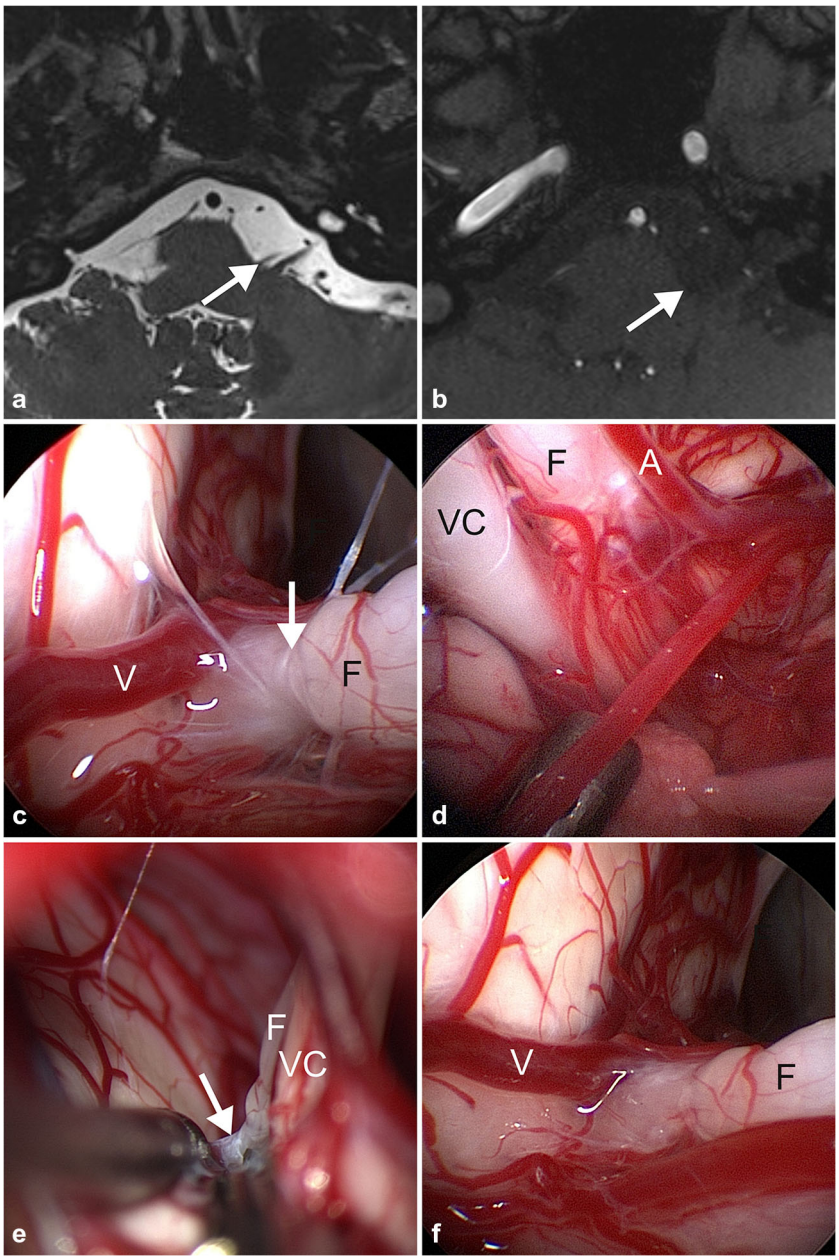


**Fig. 5** Axial CISS (**a**) and TOF (**b**) MR images showing no vascular contact at the facial REZ (arrow). Endoscopic inspection showed clearly a strangulation of the facial nerve (F) by arachnoid bands (arrow). A small vein (V) was running above the nerve, but not causing compression (**c**). Endoscopic inspection of the ventral surface of the facial nerve (F) down to the pontomedullary sulcus revealed no vascular compression. The small AICA branches (A) did not cause facial compression. VC vestibulocochlear nerve (**d**). The bands were cut under microscopic view (arrow) (**e**). Final endoscopic inspection showed the released facial nerve (**f**). The patient was immediately spasm-free after surgery (follow-up 26 months)

the best long-term outcome occurred after MVD of complex AICA-PICA compression with total resolution of spasms in 94.44% of patients (17 out of 18 patient), and the worst outcome in the combination of AICA and vein with 66.67% of patients experienced at least 90% improvement with no spasm free patients in this group.

## Discussion

### Summary of the key results

Our study shows that although arterial compression is usually the cause of hemifacial spasm, other very rare causes exist [5, 19, 21, 23, 26]. To the best of our knowledge, we report for the first time cases of hemifacial spasm caused by arachnoid bands which strangulated the facial nerve at the REZ. After simply cutting the arachnoid bands, the spasms disappeared in both patients. Pure venous compression is also exceedingly rare. The compression pattern is very variable in hemifacial spasm. The most frequent offending vessel is PICA followed by AICA, but often a complex compression is encountered with a combination of two or three vessels.

### Venous compression

Venous compression was reported in most of the large clinical studies [3, 16, 28, 33]. Purely venous compression in

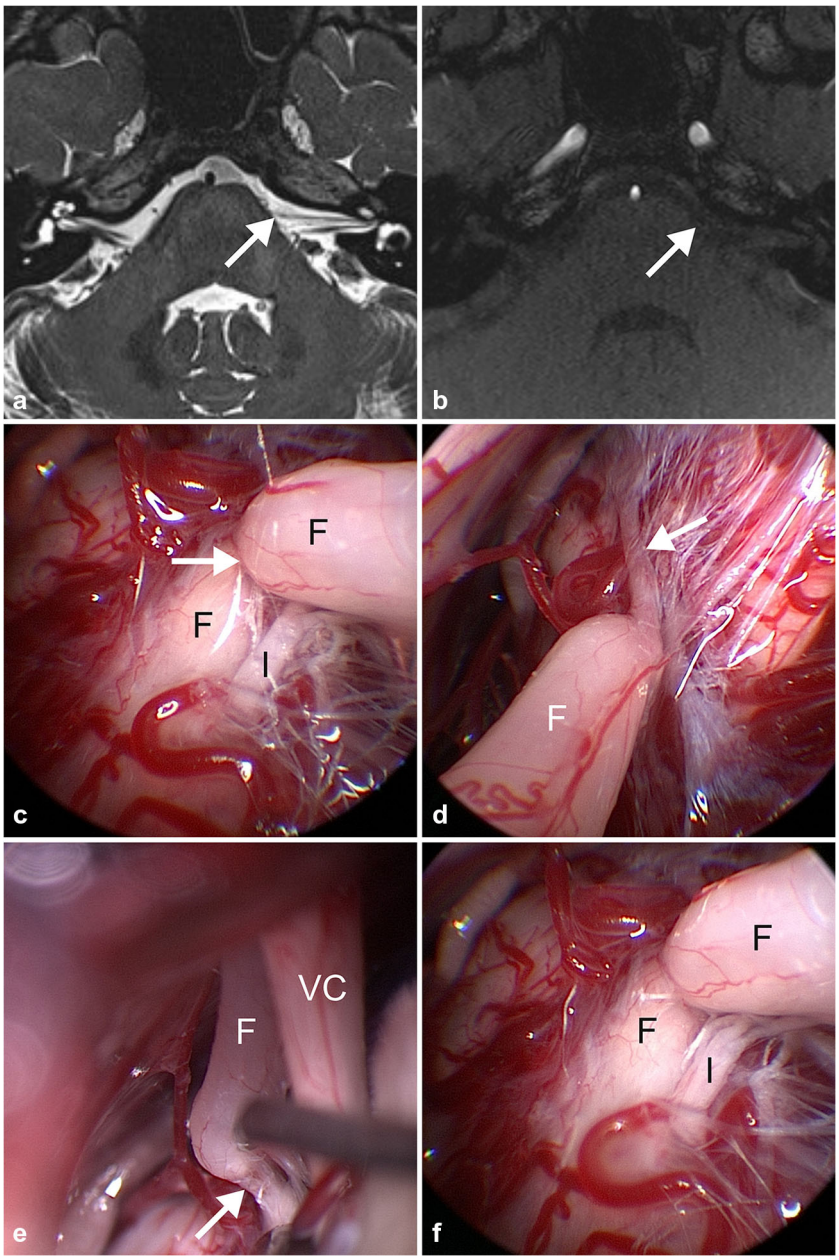


**Fig. 6** Axial CISS (**a**) and TOF (**b**) MR images showing no vascular contact at the facial REZ (arrow). Endoscopic view showed clearly a strangulation of the facial nerve (F) by arachnoid bands (arrow). The intermedius nerve (I) is seen (**c**). An unusual gliotic band (arrow) seemed to pull the facial nerve (F) anteriorly (**d**). After cutting of the bands the severe neural injury (arrow) was clearly seen. VC vestibulocochlear nerve (**e**). Final endoscopic inspection showed the released facial nerve (**f**). The patient was immediately spasm-free after surgery (follow-up 7 months).

hemifacial spasm is rare. In a recent review by Dumot and Sindou, an incidence of 0.1% was given [8]. Han et al. found 5 (0.3%) patients with venous compression in their series of 1642 patients [13] that is in accordance with our series. We saw just one venous compression (0.28%) in our series of 353 patients, but we can confirm that a vein can cause symptomatic compression of the facial nerve. We had an excellent outcome in this patient after transposing the vein with a Teflon sling. However, more frequently venous compression is accompanied by arterial compression, which we saw in seven patients. Typically, there was a sandwich compression with the facial nerve in between vein and artery. In six of these seven patients, only the artery was decompressed which resulted in excellent outcome in three and good outcome in one patient confirming that the artery was the main spasm-causing offender and not the vein. However, in two patients, the outcome was less favorable. In one patient, who had a poor outcome, the facial nerve was compressed at the REZ by a large vein and an AICA loop. The AICA was transposed anteriorly and fixed with shredded Teflon which was placed between brainstem and skull base. In this patient, the brainstem was almost in contact with the skull base due to a very narrow prepontine cistern, which was probably the reason for the failure of our decompression. In the other patient, who had a fair outcome (60% improvement), the PICA was decompressed, but there was a large vein which stretched the

**Table 2** The long-term follow-up of the patients with hemifacial spasm according to different methods of compression

| | | Follow-up outcome | | | |
|---|---|---|---|---|---|
| | | More than 18 months follow-up period | | | |
| | | (n) | | | |
| Cause of compression | *n* (=249) | 100% | 90% | 50% | idem |
| A. vertebralis | 8 | 6 | 0 | 2 | 0 |
| PICA | 83 | 66 | 8 | 7 | 2 |
| PICA + vein | 3 | 2 | 0 | 1 | 0 |
| PICA + VA | 28 | 24 | 4 | 0 | 0 |
| AICA | 66 | 52 | 4 | 8 | 2 |
| AICA + vein | 3 | 0 | 2 | 0 | 1 |
| AICA + VA | 29 | 22 | 6 | 1 | 0 |
| AICA + PICA | 18 | 17 | 1 | 0 | 0 |
| AICA + PICA + VA | 8 | 7 | 1 | 0 | 0 |
| Arachnoid band | 1 | 1 | 0 | 0 | 0 |
| Vein | 1 | 1 | 0 | 0 | 0 |
| No compression | 1 | 0 | 0 | 0 | 1 |
| | 249 | 198 (79.52%) | 26 (10.44%) | 19 (7.63%) | 6 (2.41%) |

facial REZ. Because of the course of the vein, dissection and mobilization of the vein was not possible. There were several small side branches draining the brainstem into this vein. Therefore, we did not dare to coagulate it because of the unpredictable sequelae.

Some authors reported the need of decompression of the facial nerve from the compression exerted by a vein, even if the vein has to be coagulated and cut [13, 19]. Although the complications which could occur from venous sacrifice along the cerebellopontine angle seem to be rare, there is still a risk of venous congestion and infarction that made our surgical preference not to sacrifice these kinds of veins [8, 22].

## Arachnoid strangulation of the facial nerve

We report a rare cause of hemifacial spasm that to our knowledge has not previously been described in the literature. In two patients, the facial nerve was found strangulated by arachnoid bands without any vascular compression. In both patients, the spasm immediately disappeared simply after cutting the arachnoid bands and, in one patient, additionally the fibrotic perineurium. Although hemifacial spasm due to arachnoid cysts within the cerebellopontine angle is described, strangulation of the facial nerve by arachnoid bands has not been reported.

The etiology of the arachnoid bands remains unclear. There was no history of trauma, hemorrhage, or infection in both patients. In the second patient in whom the spasms started at the age of 12, there was obviously a congenital abnormality. The facial nerve seemed to be tethered by a gliotic band which contributed to the severe strangulation. MR imaging clearly showed anterior displacement of the facial nerve at the brain stem.

## Space-occupying lesion-related hemifacial spasm

A variety of rare causes of hemifacial spasm have been previously reported in the literature [12, 14, 18, 27]. Most of these were epidermoid tumors [1, 6, 7, 17] or arachnoid cysts [4, 15, 18, 24, 27, 30]. The incidence of hemifacial spasm caused by a tumor is reported as 0.3 to 2.5% [2, 20, 29]. We have seen only one patient with epidermoid-induced hemifacial spasm, who was free of symptoms after resection. This patient was not included in this series because only MVD was evaluated but not tumor surgeries.

## Absence of compression

A few authors stated that most patients with hemifacial spasm had no apparent etiology [31]. However, our data clearly show that this statement is not true. In all our patients with hemifacial spasm, we found a morphological structure which was responsible for the spasm. Both patients without compression were misdiagnosed by us. One patient presented with severe synkinesis after Bell's palsy, the other with facial tics. MR imaging showed in both cases a close relationship between AICA and facial nerve, but the endoscopic inspection revealed no contact at all. It had been reported that peripheral facial nerve injury or Bell's palsy can result in hemifacial spasm [31]. However, in our opinion, that is not correct. Facial nerve lesions may lead to synkinesis. But synkinesis

occurs during voluntary movements, but not or very spontaneously. Facial tics can be interrupted voluntarily, while hemifacial spasms cannot. Electrophysiology has been helpful in differentiating these conditions from hemifacial spasms by documenting either lack of lateral spread or electrophysiological evidence of old facial nerve injury and synkinesis [28, 31].

## Conclusions

In most patients with hemifacial spasm, arterial vessels are involved in compressing the facial nerve REZ at the brain stem. Purely venous compression is rarely encountered. This is the first report of arachnoid bands strangulating the nerve as a cause for hemifacial spasm without involvement of any vessel.

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### Compliance with ethical standards

**Disclaimer** The content of this manuscript has never been presented or published previously.

**Conflict of interest** Henry W. S Schroeder is consultant to Karl Storz GmbH & Co. KG, Tuttlingen, Germany. The rest of the authors declare that they have no conflict of interest.

**Formal consent** For our prospectively collected database, informed consent was obtained from all individual participants included in the study.

**Ethical approval** All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee (name of institute/committee) and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

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