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**Endovascular Treatment  
of  
Dural Intracranial Arteriovenous Fistulae**

**INAUGURAL-DISSERTATION**

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## 1. SUMMARY

Embolization for the treatment of Cranial Dural Arteriovenous Fistulae (CDAVF) was developed as an alternative to open surgical approach and soon appeared as a more attractive treatment mainly due to less invasiveness.

Nevertheless, still many aspects regarding etio-pathogenesis, pathophysiology, natural history as well as treatment strategies, effectiveness of endovascular approach, acceptable rate of complications and embolic materials, need exploration, better understanding and answers based on real evidence.

Trying to contribute mainly in the aspects related to the treatment, we studied retrospectively a consecutive series of 154 adult patients treated endovascularly during last 15 years. Sixty-seven were women and 90 men with age ranged from 22 to 82 years (mean 57 y). The interval from presentation to treatment was recorded in 61 patients and ranged from 1 week to 10 years (mean 16.2 m). Potentially predisposing factors were identified in 11% of patients. One third of the patients were presented with aggressive symptoms. Ten patients had multiple shunts (6.3%). Twenty-five of the lesions were particularly extensive and complex, whereas the rest were located in various sites with the transverse and/sigmoid sinuses being the most frequent location (35%) followed by the cavernous plexus (25%) and the tentorium cerebelli (7.5%).

Regarding classification according to the venous drainage, 74 cases were Borden (B) grade 1, 32 cases Borden grade 2 and 51 cases grade 3. Regarding location according to the epidural space 52 patients were associated with the Ventral Epidural (VE) space (33%), 45 patients with the Dorsal Epidural (DE) space (29%), 38 patients with the Lateral Epidural (LE) space (24%), 17 patients with both VE and DE spaces (11%) and 5 patients with both DE and LE spaces (3%).

Almost all (97.3%) B1 lesions had a benign presentation with the 25% of them having a venous outlet abnormality (VOA – thrombosis, stenosis, varix). Fifty per cent of those with aggressive presentation had a VOA. Two thirds of

the B2 lesion had benign presentation with 52% having a VOA, whereas 72% of the rest with an aggressive presentation had a VOA. The vast majority (80.4%) of the B3 lesions had an aggressive presentation with half of them having a VOA.

Most of the VE lesions had a benign presentation (94.3%). Similarly 62.3% of the DE had a benign presentation. In contrary 86% of the LE lesions had an aggressive presentation. All lesions (100%) of the LE group had cortical venous drainage and showed a male predominance.

A total of 189 operations were performed in 154 patients (average 1.2). The transarterial approach was applied in 85% of the operations, the transvenous approach in 9% and both approaches in 6% of the operations. NBCA (glue) was used as the only or main material in 65% of cases.

The early angiographic results consisted of 68% complete occlusion, 20.5% subtotal occlusion, 8.5% extensive occlusion and 3% partial occlusion. Concerning early clinical outcome, disappearance of symptoms was observed in 24% of the patients, improvement in 11.7%, no new symptoms in 59%, temporary new symptoms in 2.6% and permanent new morbidity in 2.6% of the patients due to operative complications.

One hundred and fifty-one patients were followed-up with MR imaging and clinical examination (range 1m to 15 years) for a mean of 2.9 years. Complete occlusion was obtained in 87% of the patients. Three patients were lost to follow-up. According to the venous drainage pattern: 86% of the benign group and 88% of the aggressive group showed complete occlusion. Clinically 96% of patients had a good outcome (no symptoms/improvement/no new symptoms in case of aggressive presentation with neurological deficit).

Two cases underwent additional surgery and no case had additional radiosurgery.

The above-described management with its particular aspects described in the main body of the study yielded results characterized by high success and low complication rates. These results justify the use of endovascular operation as the treatment of choice for CDAVFs.

## 2. INTRODUCTION and HISTORICAL PERSPECTIVE

Yasargil noted that Rizzoli, in 1873, described an arteriovenous malformation supplied by the occipital artery with drainage to the transverse sinus that involved the dura matter. <sup>(287)</sup>

Cranial dural arteriovenous fistulas as a distinct entity were first described in the early 30's by Sachs and subsequently by Tonnis.<sup>(1,2)</sup> However the description of spontaneous DAVF as distinct from traumatic direct shunt came much later in the early 50's by Verbiest and Fincher. <sup>(3,4)</sup>

In the early 60's van de Werf reported the first case of a congenital high flow dural arteriovenous shunts in a child.<sup>(5)</sup>

During that decade several groups reported on the topography of these lesions describing those in the cavernous sinus,<sup>(6)</sup> sigmoid and transverse sinuses,<sup>(7,8)</sup> and anterior cranial fossa.<sup>(9)</sup>

At that time intracranial hemorrhage as presenting manifestation of a dural shunt was attributed to some pial compartment of the lesion.<sup>(7,9,10)</sup>

In the late 1960's groups from both Europe and US started to explore systematically dural arteriovenous shunts, with external carotid catheter angiography <sup>(11-14)</sup> and generally the diagnosis of dural arteriovenous (AV) shunts presented significant advancement.<sup>(15,16)</sup>

In the mid-1970s the group of Lariboisiere establish the first angioarchitectural classification of dural arteriovenous shunts <sup>(17,18)</sup>

Although a topographical and angioarchitectural classification was introduced, no clear distinction between anatomical factors predisposing to a benign or aggressive clinical presentation was still apparent. <sup>(15,19,20)</sup>

The assumption that clinical symptoms could be related to the venous drainage patterns appeared in 1972, when Houser et al <sup>(16)</sup> in their publication correlated angiographic features with clinical symptoms and concluded that intracranial hemorrhage occurred when venous drainage was directed to the pial veins.

Subsequently, Kosnick et al <sup>(21)</sup> observed that dural AV shunts, could induce neurologic symptoms due to characteristics related to their

angioarchitecture.

These publications led to a better understanding of vascular anatomy and behaviour of these lesions. Later DAVFs draining into the cortical veins were categorized as a separate entity <sup>(22)</sup> and were described more in detail and classified correlating their patterns of venous drainage with clinical presentation in 1978 by Djindjian et al. <sup>(18)</sup> They proposed that cortical venous drainage might produce aggressive neurologic symptoms and hemorrhage.

During the 1980's, many authors reported the particular risk for hemorrhage from dural AVFs located at the floor of the anterior cranial fossa and at the tentorium cerebelli. <sup>(23-26)</sup> Gradually also more evidence for the acquired nature of these lesions appeared. <sup>(27)</sup>

During that period more authors published also on the role of cortical venous drainage in dural shunts and its correlation with neurological manifestation and natural history of the disease. <sup>(51)</sup>

In 1984, Malik et al <sup>(25)</sup> in their review of 223 previously reported cases although concluded that lesions related to large dural sinuses are less likely to bleed than lesions with restricted dural outflow, they failed to notice the role of the pattern of venous drainage.

In 1986, Lasjaunias et al <sup>(28)</sup> presented a meta-analysis of 191 cases and made a step further by analyzing the mechanism of neurologic manifestations and concluding that central nervous system symptoms seem to be related to passive venous hypertension. Furthermore detailed descriptions of the vascularization of the cranial dura by Lasjaunias helped to further understand the potential for endovascular approach to DAVS and the associated risks <sup>(29,30)</sup>

In 1990, Awad et al <sup>(31)</sup> reviewed 360 cases reported in the literature and 17 of their own cases and compared the angiographic features of 100 aggressive cases with 277 benign cases. They concluded that cortical venous drainage (CVD), variceal or aneurysmal venous dilatations, and galenic drainage were features heralding aggressive neurologic signs.

Early approaches to treatment consisted of surgical ligation of the ECA, resection of the fistula in the region of the cavernous sinus, and transarterial

embolization of ECA feeders with particles. For fistulas of the transverse and sigmoid sinuses, surgery had become the main way of treatment by the mid 1980s.<sup>(32)</sup> Later some new surgical<sup>(33)</sup> or endovascular therapeutic techniques have been described. In 1984, Viñuela et al and Lasjaunias P et al reported their series of patients with spontaneous dural arteriovenous shunts treated with transarterial embolization with polyvinyl alcohol (PVA) particles or isobutyl cyanoacrylate (IBCA).<sup>(34,30)</sup>

In 1987, Halbach et al<sup>(35)</sup> reported the results of transarterial embolization in patients with dural fistulas of the transverse and sigmoid sinuses. In 1986 the same group began using a transvenous route of embolization in selected patients and in 1989 they published a couple of reports on the transvenous embolization of dural fistulas as the option of first resort in endovascular treatment of cavernous dural fistulas and elsewhere when applicable.<sup>(36)</sup>

Recently Davies and van Dijk identified morphological subgroups with different natural histories, facilitating patient selection and helping establish therapeutic goals in each subgroup.<sup>(37-39)</sup>

One of the last contributions of P. Lasjaunias, which added a lot to our understanding of this disease, was his study on comparative venous anatomy at the level of the spine and skull and the introduction of a new classification system of cranial dural arteriovenous fistulae based on the craniospinal epidural venous characteristics.<sup>(40)</sup>

### **3. EMBRYOLOGY and VASCULAR ANATOMY of the CRANIAL DURA Matter**

The brain and spinal cord of animals are covered by one or more layers of connective tissue, which are called the meninges, from the Greek word meninx, which means membrane. In fishes, only a single layer is present. Amphibians and reptiles have two meningeal layers. In mammals and birds, three meningeal layers are present. The outermost layer is the dura mater (means „hard mother“) and is actually composed of two layers: an inner layer enclosing the central nervous system and an outer layer that lines the inside

of the skull. The use of the word “mother” to describe these membranes comes from an ancient notion that they were the origin, or mother, of all membranes in the body. <sup>(41)</sup>

The cranial meninges originate from several sources such as the prechordal plate, the parachordal mesoderm and the neural crest. At 5 weeks of development the loose mesenchyme around the brain forms the primary meninx. At 7 weeks, the cranial pachymeninx and leptomeninx are distinguishable. <sup>(42)</sup>

The dura matter covering the convexity of the cranial vault (membranous bone) has a different origin than the dura covering the skullbase (cartilagenous bone). As a result of this difference, different relationships and interactions between bone, dura and venous system should be expected.

Dural sinuses are large venous collectors located between two layers of the dura matter. Septations often occur within the sinuses and result in separate venous channels adjacent to each other.

There are no dural sinuses in the midline of the skullbase or at the spinal cord level. In these locations only venous plexiform channels in the epidural space are present. These are the cavernous plexus (the so-called sinus), the clival plexus and the plexus along the anterior aspect of the foramen magnum in continuation with the ventral spinal venous plexus.

Other structures tightly related to the dura are the transdural segments of the cerebral veins just proximal to their estuary to a sinus. They are found near the superior sagittal sinus (SSS) the transverse sinus close to the tentorium, the middle cranial fossa and lamina cribiformis. CDAVFs often affect this part of the dural system.

The venous sinuses change with age. Few months after birth the cavernous plexus (primarily not part of the cerebral venous system) „captures“ the sylvian vein offering to the brain an alternative venous drainage, whereas the jugular bulb continues to mature after birth and after the regression of the occipital and marginal sinuses. <sup>(43)</sup>

Electron microscopy studies have shown that there is a continuous layer of cells between the inner table of the bone and the subarachnoid space. The outermost (periosteal) dural side is attached to the skull by extensive amounts of intertwining extracellular collagen. This attachment is particularly strong at the cranial base and the sutures. Contained within this layer are fibroblasts, osteoblasts, nerve fibers, and dural vessels. In contrast, the inner (meningeal or juxta-arachnoid) dural side has proportionately more fibroblasts and less collagen. It separates from the periosteal dura to form the walls of the larger sinuses. Immediately below the meningeal aspect of the dura is an easily disrupted dural barrier cell layer, which, compared with the other dural layers, contains little collagen.

Regarding the dural microvasculature there are two important works who focused on the morphology and microstructure of the intrinsic dural vessels by Kerber and Roland <sup>(44,45)</sup>

There are three sets of sub-branches that arise throughout the course of all main meningeal arteries. These numerous primary anastomotic arteries (100–300  $\mu\text{m}$  in diameter) are interconnected with one another and with short secondary anastomotic arteries (20–40  $\mu\text{m}$  in diameter). Another set of sub-branches the penetrating arterioles (5–15  $\mu\text{m}$ ) contributes to an extremely rich capillary network on the inner (juxtaarachnoid) side of the meningeal dura. The last set of arteries arises throughout the course of the main meningeal branches to directly supply the adjacent skull. It should also be noted that a random network or plexus of venous lakes and crevices is found within all of the dura. Kerber and Newton reported also the existence of probably plentiful arteriovenous shunts in the midportion of the dura. No later publications have reported on similar finding.

#### Vessels of the cranial vault dura

Some of the dura lining the skull vault may be supplied by the transosseous branches of two scalp arteries: the occipital and posterior auricular; however, most of it is supplied by the middle meningeal artery. The middle meningeal

artery divides into four main branches from frontal to temporo-occipital. These branches are 400–800  $\mu\text{m}$  in diameter. A dural vein is often larger than the adjacent artery, and frequently, the veins almost encircle the artery, creating what has been called a meningeal sinus. This structure can connect the superior sagittal sinus with the sphenoparietal sinus. Most of the convexity meningeal vein-pairs enter the superior sagittal or transverse sinuses. Some form a plexus 1–2 cm in width, the lateral lacunae, can be found along the lateral aspects of the superior sagittal sinus. This plexus receives emissary veins from the scalp, diploic veins from the skull, and occasionally cerebral veins. (47)

#### Vessels of the skull base dura

Three main arteries supply the dura of the skull base. The internal carotid, vertebral, and external carotid arteries. Their vascular territories are variable. The internal carotid artery supplies the sellar and parasellar region via the meningohypophyseal and inferolateral trunks. The ophthalmic artery gives rise to the ethmoidal branches that supply the cribriform region and the anterior frontal meningeal branch that supplies the anterior fossa. The vertebral artery contributes to the dura of the posterior fossa via two branches directly, the posterior (often coming from the PICA) and anterior meningeal, and one branch indirectly, the subarcuate branch arising from the AICA. Three external carotid artery branches contribute to the supply of the skull base dura. The ascending pharyngeal artery supplies the dura around and in the jugular foramen and contributes to the clival, odontoid arch plexus, and cerebellar pontine plexus. The maxillary artery gives rise to the middle meningeal, which supplies the middle and anterior cranial fossa. The occipital artery supplies a variable region of the dura in the posterior fossa via the mastoid and other meningeal branches.

#### Vessels of the falx cerebri and cerebelli

The vasculature in the falx cerebelli, falx cerebri, and tentorium lies within the two layers of the dura.

Along the attachment of the falx cerebri to the convexity, the anterior and posterior paramedian arteries form a prominent arcade. These arteries interconnect at the midline across the superior sagittal sinus and are particularly large when one middle meningeal artery is hypoplastic. The sinuses do not constitute an arterial barrier, so both meningeal arteries can cross the midline. The inferior margin of the falx cerebri is supplied by the anterior and posterior cerebral arteries. The ophthalmic artery supplies ethmoidal (anterior meningeal) branches to the crista falciformis, the anterior attachment of the falx cerebri in the cribriform region. The vertebral artery supplies the posterior meningeal artery and its terminal branches to the falx cerebelli and part of the falx cerebri. The maxillary artery contributes through the middle meningeal to the supply of superior falx cerebri.

The meningeal veins that drain the falx cerebelli and cerebri are inconsistent and unnamed, but they enter the adjacent sinuses. The most consistent features are the presence of a large vein connecting the superior sagittal sinus to the proximal inferior sagittal sinus in 10% of specimens and a large venous lake at the dorsal portion of the inferior sagittal sinus in 30% of specimens <sup>(47)</sup>

### Vessels of the tentorium

The multiple patterns of vascular supply to the tentorium are complicated because the lower and upper surfaces are supplied by different arteries. Regarding the inferior surface the supply is coming from the basilar, superior cerebellar, occipital, and posterior meningeal branch from the vertebral arteries.

For the upper tentorial surface there is supply from six branches. The posterior cerebral may supply through the so-called Schechter-Davidoff branch. The internal carotid artery contributes supply via the marginal (called also Bernasconi-Cassinari) and basal tentorial branches. The vertebral artery may supply the posterior meningeal branch. Three external carotid artery branches contribute vessels to the tentorium dura. The ascending pharyngeal, the middle meningeal artery and the occipital artery. <sup>(48-51)</sup>

The complexity of the vascular supply of the dura requires that any angiographic assessment of dural lesions requires visualization of the circulation of both vertebral, both internal carotid, and both external carotid arteries for most of the locations.

#### **4. ETIOPATHOGENESIS and PATHOPHYSIOLOGY of CDAVFs**

Most of the authors agree on the acquired nature of these lesions. <sup>(15, 18, 27, 31, 32, 44, 52-66)</sup> in the adult population. Moreover, laboratory studies have demonstrated that sinus thrombosis <sup>(53, 62, 65, 67-73)</sup> combined with venous hypertension <sup>(52, 63, 66, 74-85)</sup> reliably produces CDAVFs. This in turn leads to tissue hypoxia and activation of angiogenesis, <sup>(80,86,87)</sup> through various angiogenic factors <sup>(88)</sup> leading to eventual formation of DAVFs. Hypercoagulable states predispose patients to venous sinus thrombosis and can occur after acquired conditions such as trauma or infection and in pregnancy or during oral contraceptive therapy or in genetic conditions that result in thrombophilia as factor V Leiden mutation, prothrombin G20210A mutation, and protein C and protein S deficiencies.<sup>(79, 83, 84, 89-102)</sup>

The exact mechanisms triggering the formation and progression of arteriovenous dural shunts in patients with preexisting sinus thrombosis is unknown. Seems that sinus thrombosis is a precipitating factor in the pathogenesis of DAVF only if it has induced venous hypertension. <sup>(103)</sup>

DAVF formation is more likely to be associated with venous hypertension than with sinus thrombosis as Chen et al reported recently. Based on their experiments they proposed that venous hypertension might induce chronic regional hypoperfusion and vascular endothelial growth factor (VEGF) expression. Angiogenesis in response to venous hypertension, hypoperfusion, and angiogenic factors could promote collateral venous drainage, which serves to reverse the venous hypertension and hypoperfusion. However, if the chronic congestion of the venous outflow leads to prolonged secretion of VEGF, MMPs (matrix metalloproteinases),

and other humoral factors, which stimulate vascular endothelial proliferation and excessive angiogenesis in the dura mater around the sinus, the meningeal capillaries may connect to dural sinuses, or connection of the arteries and veins in the dura mater occurs to initiate arteriovenous shunting.

(104)

Neonatal cases <sup>(5, 105-109)</sup> indicate a congenital origin and a different variety for the children population.

However reports referring to links between specific genes and sporadic arteriovenous fistulas have been also appeared <sup>(110)</sup>

As a comprehensive summary seems that several known but mainly unknown causes can induce angiogenesis which will connect the osteodural arteries with the sinuses, regional dural veins and/or cortical veins depending among other factors on the location of the lesion. Changes in the venous structure promoting thrombosis can occur before, during or after the angiogenic process. The resulting lesion will be manifested with various symptoms depending on the interaction of several interassociated factors, the location, the extent and hemodynamics of the shunt, the collateral venous circulation and the time –rate of progression being some of the apparent ones. <sup>(111,112)</sup>

## **5. EPIDEMIOLOGY and NATURAL HISTORY of CDAVFs**

In relation with other vascular pathologies we know that CDAVFs account for 10% to 15% of all intracranial arteriovenous malformations <sup>(13)</sup>

Recently, an epidemiological survey of the detected brain vascular malformations was conducted with detection rates per 100 000 person-years 2.0 (95% CI, 1.8 to 2.3) for dural fistulas, cavernous malformations, Vein of Galen malformations, and venous malformations <sup>(113)</sup>

Another study including only DAVF cases in Japan yielded a detection rate of 0.29 DAVF cases per 100,000 adults per year. Furthermore, unlike Europe and North America where cases of transverse-sigmoid sinus DAVF are

predominantly detected, in Japan, a higher number of cases of cavernous sinus lesions are detected, indicating racial difference in the presentation of DAVF. <sup>(114)</sup>

A previous survey from Scotland reported a crude detection rate (per 100,000 adults per year) of 0.16 (95% CI, 0.08 to 0.27) for dural AVMs <sup>(115)</sup>

Recently and departing from different point of view Geibprasert et al presented the results of a multiethnic study on the potential differences in demographic, angiographic and clinical characteristics of different types of DAVFS in Europe, South America, and Asia trying to find out whether the same clinical profile for lesions located in the ventral (VE), dorsal (DE) or lateral (LE) epidural spaces, is present in different ethnicities. Interestingly they reported that DE-shunts in the Asian population were more aggressive secondary to a higher rate of venous outlet alteration (VOA) with associated CVR. <sup>(116)</sup>

The natural history of CDAVFs or otherwise their clinical course once identified, has been addressed last years by several publications.

From previous sporadic reports we know that CDAVFs can present spontaneous regression. At least 15 cases have been presented in the literature <sup>(27, 117-126)</sup> whereas in a recent study aiming to define the incidence and clinical characteristics of patients with DAVFs showing spontaneous angiographic pattern conversion a percentage of 12.5% of spontaneous occlusion of the shunt was reported. <sup>(127)</sup>

Luciani et al proposed that two types of spontaneously regressing dural AVFs must be distinguished: posttraumatic and spontaneous. The mechanisms accounting for all dural AVF regressions remain unclear. A direct occlusion of the intradural shunts, rather than dural sinus thrombosis, could be responsible for this process in some cases. <sup>(117)</sup>

Cranial dural arteriovenous fistulas (DAVFs) can be classified into benign or aggressive, based on their patterns of venous drainage. Benign lesions do not have cortical venous drainage and are presented with benign symptoms

non directly related to the CNS, as tinnitus, venous congestion in the orbita with or without cranial nerve deficit, headache etc. Nevertheless it is not clear whether this initially benign appearance persists over time. Satomi et al.<sup>(128)</sup> studied the clinical course of 117 consecutive patients with benign lesions (with palliative treatment of 44 of them) and found in a median follow-up period of 27.9 months that 98% of this population preserved a „benign“ level of disease whereas in two cases (2%) managed conservatively CVD developed. In both of these cases the conversion from benign to aggressive DAVF was associated with spontaneous progressive thrombosis of venous outlets.

From the same group of Toronto University and with an expanded patient cohort a recent publication<sup>(127)</sup> reported that studying the evolution of patients with benign lesions found that spontaneous angiographic conversion (defined as complete occlusion of a pre-existing DAVS or conversion of a benign into an aggressive lesion on follow-up DSA) without any treatment, was 12.5% for spontaneous occlusion of the shunt and 4% for conversion to an aggressive lesion from benign DAVF, concluding that DAVFs are dynamic disorders, which will show chronological progression.

Furthermore, seems that the natural history of aggressive CDAVFs is even more complex. Previous and recent studies on the events later than the initial presentation were performed by Duffau et al.,<sup>(132)</sup> Brown et al.,<sup>(68)</sup> Davies et al., van Dijk et al.,<sup>(37, 38)</sup> Soderman et al.,<sup>(133)</sup> Strom et al.,<sup>(134)</sup> and Bulters et al.<sup>(135)</sup>

The Duffau et al study focused on a short period of 20 days post-presentation event. Brown et al followed patients for a longer time (mean of 6.6 years) however, they did not studied separately the patients with CVR. The van Dijk study examined an expanded patient cohort initially published in 1997 by Davies

et al. and they calculated for a group of 20 patients an annual mortality rate of 10.4%, an annual risk for hemorrhage or nonhemorrhagic neurological deficit during follow-up 8.1% (4.5 times the annual rate of 1.8% reported by Brown et al) and 6.9%, respectively, resulting in an annual event rate of

15.0%

Almost simultaneously another study from the Swedish group published a similar study based on the records of 85 patients with dural arteriovenous shunts with CVD. They found, for those patients who presented with hemorrhage an annual risk for hemorrhage of 7.4% and in those not presenting with a hemorrhage a risk of 1.5%. They concluded that the natural course of DAVFs with CVD is most probably more benign than previously proposed, in particular for patients with a presenting event other than an intracranial hemorrhage.

More recently one more study by Strom et al. pointed out that the previous studies on the CDAVFs with CVD included patients the great majority (80-100%) of whom had an aggressive manifestation whereas several such lesions including 17 of the 28 DAVFs in their series, are discovered incidentally or present with benign symptoms. By studying separately these two groups of different actual presentation they found an apparent less aggressive clinical course (1.4% annual event rate) for the lesions with benign manifestation and a very high risk of new ICH or non-hemorrhagic neurological deficit (19.0% per year) for the group of lesions that have been already presented aggressively. Interestingly, they noticed that although they share venous drainage with the cortical circulation, lesions with CVD and benign manifestation may lack or have less severe cortical venous hypertension. This may be attributable to less flow through the fistula, more efficient collateral pathways to uninvolved dural sinuses, or other factors at a microvascular level.

Lately Bulters et al. trying to explain differences between these studies and to help estimate the risk of specific fistulae carried out a study of 70 patients harbouring 75 CDAVFs with CVD and found in 90.1 years of follow up a crude annual risk of hemorrhage of 8.9%. Untreated lesions had a risk of 12.7%. Partial treatment reduced the risk of haemorrhage to 4.7%. The presence of a varix increased the risk of haemorrhage seven fold (3.5% versus 26.6%). Patients presenting with a haemorrhage (20.4%) or non-haemorrhagic neurological deficit (22.2%) had a higher risk of haemorrhage

than those with a benign presentation (4.3%).

Although the information presented above should not affect the management of DAVF patients who are considered at high risk at the present time, it does prompt discussion about the need for aggressive treatment for all DAVF patients with CVD and the potential further heterogeneity of the group. <sup>(136,137)</sup>

## 6. CLASSIFICATIONS

Djindjian and colleagues <sup>(18)</sup> proposed the first comprehensive classification of intracranial dural AVFs based on radiological anatomy in 1977; this classification used location as the main identity of the lesion. This scheme has subsequently been modified by Cognard et al. <sup>(130)</sup> In their series of 205 patients they were able to show a relationship between type and aggressive presentation. Later a similar, but simplified, version of this classification was proposed by Borden, et al. <sup>(62)</sup> They proposed their classification as a predicting tool for lesion behavior and how it might serve as a rationale for treatment <sup>(129)</sup> (Table 1).

Recently a publication by Geibprasert et al proposed a new classification of the CDAVFs. <sup>(40)</sup> According to the concept presented the craniospinal epidural spaces can be categorized into 3 different compartments related to their specific drainage role of the bone and central nervous system. CDAVFs developed in different epidural compartments constitute groups of lesions with different venous drainage pattern, the ventral epidural, dorsal epidural, and lateral epidural groups.

Three main observations of the venous development of the brain and spinal cord during embryology contributed to the generation of this new classification:

1. The venous system of the notochord and corresponding sclerotome extends from the basisphenoid (cavernous plexus) to the sacrum and gives rise to the ventral epidural drainage group. It collects the blood from

spongiosus bony structures and has no primary role in the drainage of the central nervous system.

2. The dorsal epidural venous space is normally poorly developed at the spinal level. Presence of dorsally located dural sinuses intracranially is therefore the major difference between the venous systems of the brain and spine. Their formation is linked to the appearance, during evolution, of the dural falces and tentorium and associated with the development of the paleo- and neopallial structures. These sinuses result from the confluence in the epidural space of 2 different venous systems: the osseous system draining the cranial vault and the leptomeningeal system draining the brain.

3. The veins draining the central nervous system (both spinal and cranial) are not related to the peripheral nerves, as are the arteries; these “emissary-bridging veins” join the lateral epidural venous spaces as connecting drainage system. This leptomeningeal venous drainage has no direct confluent communication with the ventral and dorsal epidural venous plexuses, which drain primarily the skull and spine.

They suggested that DAVFs in these 3 different areas will drain according to the specific role of the venous system of each region, unless there are associated conditions such as constraint placed on the venous outlet or high-flow arteriovenous shunts. These venous outlet restrictions (ie, occlusion or stenosis) can be either adjacent to the shunting area, most commonly from thrombosis, and/or remote from the shunt.

In the VE group the following locations are encountered: “vertebral body,” basioccipital, sigmoid sinus, petrous pyramid, basisphenoid and adjacent sphenoid wings, and related dural structures.

In the DE group, the following DAVF locations are encountered: “dorsal spinal epidural DAVS,” marginal sinus (dorsal portion), medial occipital sinus, torcular, transverse and accessory epidural sinuses, and superior sagittal sinus.

In the LE group, the following locations are encountered: spinal dural arteriovenous shunts, marginal sinus (lateral portion- foramen magnum) with

the emissary-bridging vein to the condyloid vein, falcotentorial (vein of Galen), petrosal and basitentorial, Breschet sinus, paracavernous region (embryonic tentorial sinus remnants), intraorbital shunts, and lamina cribiformis.

### Multiplicity

Although extensively spreading fistulas over a large territory (as those of the transverse-sigmoid sinuses), are not infrequent, real multiple DAVFs at distinct sites are relatively rare. The incidence of multiple lesions was 6.7% and 8.1 in two large series of patients with DAVF respectively. <sup>(16, 52, 60, 70, 145-148)</sup>

Some explanations for the development of multiple DAVFs have been proposed. One is based on the hypothesis that the venous drainage of a CDAVF may cause turbulent flow or stagnation in the distant venous sinus, resulting in thrombosis of the sinus and development of other DAVFs. Thrombosis of a dural sinus at several sites caused by, for example, hypercoagulative state may also lead to the development of multiple DAVFs.

As only venous hypertension unaccompanied by sinus thrombosis can cause the development of DAVF, elevated sinus pressure caused by initial DAVF can also result in the formation of multiple new DAVFs at other sites. <sup>(149)</sup>

Van Dijk et al <sup>(150)</sup> in a group of 284 patients found multiple shunts in 8.1% of all cranial dural AVFs. Multiplicity was associated with a higher percentage of CVD, yielding a higher risk for hemorrhage.

## **7. PATIENTS and METHODS**

This series includes 157 consecutive adult patients who were evaluated and treated at our institution in a 15 years period between January 1995 and December 2009. The primary purpose of this study was to evaluate the results of the endovascular treatment. Patients treated before 1995 were excluded because of difficulties to collect complete data. A retrospective analysis of hospital and outpatient charts and angiographic and imaging studies was undertaken. The angiographic-operative images of 62 patients (39%) could not be accessed; therefore the data for those patients were collected from the history chart, operative report and CT-MR imaging. Data

about Age, Sex, Interval from symptoms to treatment, Presence of Single or Multiple lesions, potential Predisposing factors, Presentation, Imaging other than angiogram findings, Location, Arterial Supply, Venous drainage, Classification according to the venous drainage pattern, Classification according to the development of the epidural venous anatomy, Venous outlet abnormalities (as thrombosis, stenosis or other), Management (endovascular, surgical, radiosurgical, combination or conservative), kind of Anesthesia, Approach (transarterial, transvenous, other or combination), Materials used for embolization, No of Sessions per patient, Immediate postoperative Angiographic result, Early postoperative Clinical outcome, Operation Complications, Peri-operative medications administered, Co-morbidities, Follow-up period either clinical alone or in combination with imaging modality (MRI, DSA), and Outcome Changes during Follow-up were collected.

## **8. RESULTS**

All patients underwent selective catheter angiography to document the diagnosis and reveal the particular features of the fistula. Patients treated by endovascular embolization underwent superselective arteriography before treatment and control arteriography immediately after treatment as well as postoperative MRI before discharge.

There were 67 women and 90 men. Their age ranged from 22 to 82 years, with a mean age of 57 years.

The interval from initial presentation of symptoms presumably related to the fistula, until treatment was recorded in 61 patients and varied from 1 week to 10 years, with a mean interval of 16.2 months.

Factors that could be considered as predisposing were recorded in only 17 patients (11%). History of head surgery related to the location of the fistula was reported in 4 patients, severe upper respiratory infection in 3 patients, documented sinus thrombosis in one patient, incompletely embolized pial arteriovenous fistula in one patient. In 8 cases a history of head injury was recorded. In 3 of them a severe trauma was referred 6, 10 and 30 years

before, respectively.

In 47 patients the fistula was presented with tinnitus alone and in another 13 patients tinnitus was combined with headache and dizziness (38% combined). In 31 patients (20%) the lesion was manifested with hemorrhage, either SAH or ICH or IVH or combination (one of these patients presented exophthalmus and chemosis prior to the bleeding). Thirteen patients had orbital signs alone as exophthalmus and chemosis. In another 9 patients the orbital signs were accompanied by tinnitus with (in 5 pts) or without headache. In another 11 patients the orbital signs were combined with diplopia due to ophthalmokinetic nerve palsy whereas 3 more patients presented nerve palsy and tinnitus alone without any orbital manifestation. From the group of patients with prominent orbital manifestations (13+9 pts), 4 patients presented impaired visual acuity (1 with glaucoma), whereas from the group of ophthalmokinetic nerve palsy plus orbital signs (11 pts) 2 patients presented impaired visual acuity (1 with glaucoma). It total 23% of the patients presented some combination of "orbital" symptoms. Fourteen patients (9%) presented focal neurological disorder attributed to local - regional venous congestion or mass effect from a large varix. In this group, 3 patients with progressive cervical myelopathy are included. Five patients presented only with epilepsy. Two patients presented with congestive venous encephalopathy syndrome, 1 patient with dementia, 2 more patients with headache only, 1 patient with prominent frontal veins accompanied by dizziness and occasional tinnitus. For 2 patients the lesion was discovered incidentally and for another 3 patients no data in relation to the clinical presentation were found.

### 8.1 Classification of the CDAVFs in our series

Classification of CDAVFs according to location is sometimes a very difficult task first because dural fistulae often extent to a certain length along adjacent sinuses and second because what we call transverse sinus for instance as a distinct location with defined limits is actually just a segment of a continuous draining system with often morphologically undistinguishable

borders with the superior sagittal sinus or the ipsilateral tentorial adhesion. In complex and extensive lesions the determination of the exact location is often even more problematic. Yet the definition of multiplicity may be questionable in such lesions. According to that and in order to preserve accuracy we list our material according to the exact location avoiding round groups.

### *8.1.1 According to topography*

Twenty-seven patients had a dural fistula limited to the right or left Transverse sinus, 9 patients had a fistula in the Sigmoid sinus, 15 patients in ipsilateral Transverse and Sigmoid sinuses, 24 in the Cavernous plexus (3 of them bilateral), 10 in the Tentorium (either supra- or infra-tentorial), 7 in the Convexity (either supra- or infra-tentorial) without involvement of sinuses, 7 in the Jugular bulb, 4 in the foramen Magnum, 5 in the Torcula, 3 in the Ethmoid area, 2 in the Hypoglossal canal, 2 in the SSS, 7 sub or infratentorial (petrosal), 2 with involvement of Transverse and Superior Sagittal sinuses, 2 in Sigmoid sinus and Jugular bulb, 2 in the Jugular bulb and Hypoglossal canal, 2 in the Torcula and Tentorium, 2 in the Transverse sinus and Torcula, 2 Temporobasal, 1 in Transverse sinus and contralateral Sigmoid sinus, 1 in bilateral Transverse sinuses, 1 in Transverse sinus and posterior fossa, 1 in Transverse sinus bilaterally, Torcula and Occipital sinus, 1 in Transverse sinus, Sigmoid sinus and SSS, 1 in Jugular bulb, Sigmoid sinus and IPS, 1 in Jugular bulb and IPS, 1 in Jugular bulb, Sigmoid sinus and Hypoglossal canal, 1 in Torcula and Straight sinus, 1 in the Tentorium and Falx, 1 in the SSS and Tentorium, 1 in the Convexity and the Transverse-Sigmoid sinus transition, 1 in the Convexity and SSS, 1 in the Cavernous plexus and petrosal apex, 1 in the Optic nerve sheath, 1 connecting the Ophthalmic Artery to the Ophthalmic vein, 1 in the Cerebellomedullary cistern, 1 Temporopolar, 2 in the Sphenoid wing, 1 Osteodural of the Orbital apex and Sphenoid wing, 1 in the Jugular bulb as predominant location with the whole base involved, 1 in bilateral Jugular bulb, foramen Magnum, Clivus and Cavernous plexus.

### *8.1.2 According to venous drainage*

The number of each type of venous drainage according to Borden and Cognard classification is summarized in table 2.

Eighty-three patients (53%) presented CVD in the initial angiographic investigation whereas almost one third of the whole population had exclusively CVD.

### *8.1.3 According to the epidural space*

Fistula localization was divided into the three groups, ventral epidural (VE), dorsal epidural (DE) and lateral epidural (LE) groups. In 52 patients the CDAVF was located in the VE group, in 45 patients in the DE group and in 38 patients in the LE group. Sixteen patients had a fistula extending into both VE and DE spaces, 6 patients in both DE and LE.

The clinical presentation in relation to the above classifications is presented in the table 3.

We defined aggressive symptoms those related to either focal or global neurological disturbance attributed to a CNS disorder hemorrhagic or non-hemorrhagic (paresis, aphasia, ataxia, seizures etc) whereas we describe as benign symptoms those related to local factors outside or non directly related to the CNS, as venous congestion in the orbita with or without cranial nerve deficit, tinnitus, headache etc.

One hundred and one patients (2 patients with incidentally discovered lesions are included) were presented with benign symptoms and 53 patients with aggressive symptoms

There were 3 cases with unknown presentation and the following angiographic features: one belonging to the LE group with B3CIII grades, one to the DE group with B1CI grades and one DE+VE, B2CIIb.

Regarding epidemiologic characteristics of the populations carrying lesions in the various epidural spaces we found some differences. In the LE group 27

of 37 patients (73%) were men and the mean age was 54.4 years. In the VE group the 31 of 54 patients (57%) were females and the mean age was 61.3 years. In the DE group 26 of 45 patients (58%) were men and the mean age was 55 years. In the VE+DE group 8 of 15 patients (53%) were men and the mean age was 57.7 years. In the DE+LE group all patients were men with mean age 47.4 years. As we mentioned in the introduction, children were not included in this study.

Analysing the relation between venous drainage according to Borden classification, clinical presentation (benign versus aggressive) and venous outlet abnormality (VOA – Thrombosis, Stenosis related or not to partial thrombosis, Dilatation, Varix) we found that 29.5% of lesions grade B1, which were presented with benign symptoms, had some kind of VOA. In the B2 group a 52% of the benign presentation subgroup had some type of VOA whereas a 70% of the aggressive presentation group had some type of VOA. The group of B3 grade with aggressive presentation consisted of 41 patients with half of them having a VOA and most of these being a varix (80% present – in 2 cases in combination with Stenosis or Thrombosis of the venous outlet).

Analysing the relation between venous epidural spaces, clinical presentation and venous outlet alteration we found that in the VE group 5.7% of the patients presented with aggressive symptomatology. In addition, 29% of the cases with benign presentation had some VOA whereas the 66% of the cases with aggressive presentation had some VOA. In the DE group, 37.7% of the patients presented with aggressive symptoms and the 46% of the benign presentation cases had some VOA whereas in the aggressive presentation subgroup the 70% had some VOA. In the LE group 86% of the patients were presented with aggressive symptoms and the 42% of the aggressive presentation cases had a VOA all of them being a varix (in one case combined with stenosis). None of the group of patients with lesions in both VE and DE spaces was presented with aggressive symptoms, whereas half of them presented some VOA, mostly stenosis.

All shunts of the LE group presented CVD (100%) and 37% of the patients had a VOA. Ten patients of the VE group had CVD (20%), 6 of them with downstream sinus thrombosis or post thrombotic changes as stenoses, 1 VE without VOA but high flow and 3 without any detected VOA (30% of those with CVD). In total, 23 patients of the DE group had CVD (49%). Of them 14 patients had a VOA, 7 patients were without VOA and 1 case without VOA but high flow.

## 8.2 Multiplicity

Multiplicity in extensive dural fistulas was often difficult to detect. We defined multiplicity as clear presence of distinct arteriovenous shunts.

One hundred forty-two (95%) patients demonstrated a single fistula, 10 patients had multiple lesions (in 2 of them the whole skull base was involved) 5 patients had bilateral lesions (3 at the cavernous plexuses and 2 at the transverse sinuses).

According to Epidural space location there were 16 patients with fistulas extending in both Ventral and Dorsal epidural spaces and another 5 patients with lesions in Dorsal and Lateral epidural spaces.

## 8.3 Technical Aspects

### *8.3.1 Embolization Procedure*

All procedures were performed on a biplane angiographic unit. Percutaneous femoral artery catheterization was used to achieve access in all patients. Patients were not systematically heparinized. Standard coaxial techniques were used. The Valavanis catheter 5F was constantly used as both angiographic and guide catheter, which was navigated, into the appropriate parent vessel (external carotid artery, internal carotid artery, or vertebral artery). Microcatheters were then advanced into the selective branches, and superselective angiography was performed on the target branch or branches for embolization. Analysis of pre-embolization angiogram routinely involved identification of the following: 1) arterial feeders, 2) the fistulous connection,

3) venous outflow, 4) normal sinuses, 5) venous pouches, 6) visible and occult extracranial-to-intracranial anastomoses, and 7) visible and occult supply to cranial nerve vasculature. Knowledge of the occult anastomoses is especially important because these connections can remain angiographically silent until the moment the embolic material is injected.

An Elite 1.5F flow-directed microcatheter was subsequently navigated over a microwire (Mizzen preferably or lately Mirage .008) to reach the distal aspect of the pedicle supplying the DAVF. Microcatheter angiography was then performed to confirm optimal positioning. The microcatheter was then flushed with 10 mL of Dextrose 0.5. Glue NBCA was typically used for embolization usually in high dilution permitting better penetration to the venous side of the shunt. The mixture of glue and Lipiodol was agitated mechanically for at least 3-5 minutes before use to obtain homogeneity.

Selected branches were embolized with NBCA using plain x-ray. A small amount of initial reflux usually would not influence the progress of injection. Excessive reflux, however, was avoided to prevent the occlusion of proximal normal vessels and to make the removal of the microcatheter feasible. Whenever unwanted reflux or flow into non-targeted areas was observed, injections were briefly held or stopped. These pauses seemed to allow glue to polymerize at those areas, diverting the newly injected glue deeper forward.

### *8.3.2 Anaesthesia*

Fourteen of the first patients of this series were operated under local anesthesia. Soon it became clear that general anesthesia was necessary mainly for the safety of these operations as this is valid for most of the endovascular operations today. One patient was operated on under sedation and the rest operations were undertaken under general endotracheal anesthesia. Routine postoperative care was performed. All patients except the acute bleedings were maintained normotensive in an ordinary wardroom. Perioperative medications were used on a case-by-case basis.

### 8.3.3 Sessions

Overall, 189 procedures were performed to treat 154 patients, with an average of 1.2 sessions per patient and max 4 sessions per patient. One hundred and thirty-two patients (86%) were treated with a single endovascular procedure whereas 22 patients were treated with staged procedures, of them 13 patients were embolized in 2 sessions, 6 patients were treated in 3 sessions whereas for 3 patients 4 sessions were required. Three of our patients have been treated before (in a total of 6 embolization sessions for all 3 patients) in other institutions.

### 8.3.4 Approaches - Transarterial, Transvenous, Combined

In one hundred and sixty-one (85%) sessions the transarterial approach (TA) was exclusively used. In 3 of them balloon assistance was used for protection of the parent vessel during occlusion of tiny internal carotid feeders. In 17 sessions (9%) the transvenous (TV) approach was used exclusively. This was applied mainly for cavernous plexus lesions and occasionally for some lesions in the transverse or/and sigmoid sinus. Combination of TA and TV approach in the same session was required in 11 cases. In one of the above cases with cavernous plexus lesion a transvenous approach was tried out but due to stenosis of the angular artery it was aborted and only TA approach was used.

### 8.3.5 Embolic Materials used for Embolization of CDAVFs

In 45 of 154 lesions, NBCA alone was used. In another 55 lesions NBCA was used with PVA as supplementary material for feeders, which persisted after glue injection and were not selectively approachable. Therefore in 100 patients (65%) glue was used as the only or main embolic material. In another 27 cases (17.5%) pertaining either to lesions with both very tortuous and diffuse supply (this was the case of 11 transverse sinus shunts with main supply from distal occipital branches) or to lesions with minimal supply from tiny and/or dangerous feeders, solely PVA was used. In 15 cases (10%), mainly referring to cavernous plexus lesions, Coils were used as the only embolic material. A combination of the above three materials (NBCA, Coils,

PVA) was used in 12 cases. PVA was delivered in 5 of the 12 cases, in all of them as supplementary material. In 6 of the 154 lesions balloon assistance was employed.

#### 8.4 Applications and Goals of endovascular treatment

##### *8.4.1 Curative Treatment*

Endovascular treatment should be considered the treatment of choice for CDAVFs since can be a curative treatment for the majority of cases as long as a permanent embolic agent can be pushed to the venous collector of the lesion or a venous approach is applicable. This was achieved in the 68% of our cases.

##### *8.4.2 Palliative Treatment*

Palliative treatment should be reserved for very extensive lesions without a chance of complete eradication or for lesions, which involve either long and tortuous arterial feeders without a possibility of approaching the shunt zone to deliver a permanent embolic agent or/and a venous approach is not feasible.

##### *8.4.3 Preoperative Treatment*

Practically this is not an application of the endovascular treatment. In our series in one case and only after failure of palliative treatment of a very extensive shunt to control the patient's symptoms in the long-term, a surgical occlusion of the transverse-sigmoid sinuses was performed, combined with a venous by-pass connecting the transverse sinus proximal to its occlusion with the jugular vein. In 1 more case surgical intervention was also employed after a subtotal endovascular occlusion, aiming to a complete surgical eradication of the lesion.

#### 8.5 Results of Endovascular Treatment

### *8.5.1 Angiographic results*

Angiographic results were defined the immediate results in the final control angiography.

Complete occlusion or anatomic cure of the AVF was strictly considered the absence of any arteriovenous shunt in the late angiographic phases. As subtotal occlusion, was defined an above of 90% occlusion rate and an even faint opacification of the venous drainage in the late arterial phase. Extensive occlusion was considered an occlusion of approximately 70-90% of the shunt. Below 70% occlusion of the shunt was reported as partial occlusion. We should notice that some cases with an angiographically demonstrated subtotal occlusion at the end of embolization, presented a picture of thrombosis of their shunt and draining vein/s in the subsequent MR imaging. In 92 of our patients (60%) a complete occlusion in one session was obtained. Of the 40 remaining patients who underwent just one embolization session, 25 patients (16%) had a subtotal angiographic occlusion, 11 patients (7%) had an extensive occlusion and 4 patients had a partial occlusion.

The remaining 22 patients had multiple embolization sessions.

Of the 13 patients who underwent 2 sessions, 8 patients demonstrated a complete angiographic occlusion in the final angiogram of the second session. In the remaining 5 patients with 2 sessions, a subtotal occlusion was obtained in 4 patients and an extensive occlusion in 1 patient.

Of the 6 patients who underwent 3 embolization sessions, a complete angiographic occlusion was achieved in 3 patients. In 2 patients a subtotal occlusion was achieved after the third session and in 1 patient an extensive occlusion was achieved after the third session.

Of the 3 patients who underwent 4 embolization sessions, a complete angiographic occlusion was achieved in two. In the third patient a subtotal angiographic occlusion was achieved in all four sessions.

By summarizing the above results, 105 patients (68%) presented a complete angiographic occlusion of their AVF, 32 patients (20.5%) presented a subtotal occlusion, 13 patients (8.5%) presented extensive occlusion and 4

patients (3%) had a partial occlusion (one intensive, one palliative and one aborted because of a complication). The exact number of patients with subtotal angiographic occlusion who demonstrated a thrombosis of the shunt and draining veins in the immediate post-operative MR was not precisely reported and therefore not mentioned here.

Considering the angiographic results in relation with the venous drainage pattern, we obtained a complete occlusion in 47 of the 74 patients (63.5%) with benign lesions (Borden grade 1) whereas a complete occlusion was achieved in 58 of the 83 patients (70%) with aggressive lesions (Borden 2 & 3).

In 41 of the 105 achieved complete angiographic occlusions, NBCA was the only embolic material, whereas in 14 complete occlusions, coils were the only embolic material. In 32 achieved complete angiographic occlusions a combination of acrylic glue and PVA (in most of the cases as supplementary agent) as the only embolic materials were employed, whereas in another 4 complete occlusions another combination of materials was used. In 14 of the complete angiographic occlusions, PVA was employed as the only embolic material.

#### *8.5.2 Early Clinical outcomes*

Regarding the early clinical outcome, a clinical cure was defined by complete resolution of signs and symptoms attributed to the fistula, within days after treatment. Patients were considered clinically improved if their symptoms significantly diminished after treatment without development of new symptoms or signs as a result of a complication.

We defined immediate clinical outcome the clinical condition of the patient during the period of postoperative hospitalization. For most of our patients who were presented without severe symptoms this period corresponded with a time period of few days to 1 week in average.

Ninety-one of our patients did not present a new neurological deficit postoperatively (NND). This refers to the patients who either were suffering of

some neurological deficit due to previous bleeding/ischemia or the presenting symptom was not permanent in nature (epileptic fit, for instance) or it was too early to be evaluated during the immediate postoperative period (as dementia or nerve paresis for instance).

Thirty-seven of our patients showed complete disappearance of their presenting symptom (tinnitus in most of these cases).

Eighteen patients had clear improvement of their presenting symptoms (mainly orbital symptomatology) during immediate postoperative days.

Four of our patients presented temporary new symptoms. Two patients presented a new mild diplopia (in one of them due to III<sup>n</sup> paresis), 7 and 12 hours postoperatively correspondingly, which subsided after 24 hours approximately. One more patient presented a worsening of a preexisting decreased visual acuity. The fourth patient presented a new and only epileptic fit few hours postoperatively.

One patient presented hemiplegia as a new and permanent neurological deficit postoperatively. One patient presented headache and vomiting connected to edema in the cerebellum after embolization of a fistula draining into cortical cerebellar veins. He was managed by craniectomy of the posterior fossa and antiedematous treatment but he developed some cerebellar infarcts. One more patient presented X and XII nerve paresis after embolization of a Jugular foramen DAVF.

One last patient who developed spasm of his SCA (feeder of the AVF) presented a vermis infarction after post-embolization surgery aiming to complete elimination of the shunt.

#### 8.6. Complications of Endovascular Treatment

There were 4 cases of permanent neurological deficit (2.6%) due to an operative complication and 4 cases of temporary neurological manifestations (2.6%) most of them not clearly related to an operative complication. There was no mortality connected to an operative complication in this series.

The first case of permanent neurological deficit is related to an unwanted glue migration through a non-opacified collateral, the artery of the foramen

lacerum to the ICA and then to MCA territory, which resulted to a severe infarction of the MCA. The second case with permanent deficit is related to an infarction of cerebellar vermis after spasm of the SCA, which occurred during incomplete embolization and after subsequent surgical operation for elimination of the lesion. The third case with permanent morbidity was a patient with X and XII nerve paresis after embolization of a jugular-hypoglossal foramen lesion.

One last patient with a torcular DAVF and cerebellar cortical drainage with varix vein presented two days after the embolization cerebellar signs and edema with later infarction presumably due to venous thrombosis. A decompressive craniectomy was performed plus anti-edematous treatment and the patient showed improvement during ensuing weeks.

Two patients presented temporary diplopia few hours after embolization presumably related to paresis of ophthalmokinetic nerves. The first patient improved rapidly after 24 hours whereas the second patient recovered over several days. One AVF was located at the transverse and sigmoid sinus, was managed exclusively with PVA and clival branches of the ICA were involved and selectively catheterized. Similarly the other case was an ethmoidal AVF with supply from bilateral ophthalmic artery, was managed exclusively with PVA and the ethmoidal arteries were involved and selectively catheterized. The diplopia was attributed to temporary ischemia of the cranial nerves.

One more patient with optic nerve sheath DAVF and orbital symptoms with decreased visual acuity, while he showed clear improvement of the orbital symptoms (chemosis, exophthalmus), he presented a worsening of visual acuity after one day. The lesion was managed exclusively with transarterial approach and glue injection without complication detected during injection or in the control angiography.

One more patient with a transverse-sigmoid sinus AVF who presented an epileptic fit for the first time postoperatively did not suffer of any detected operative complication.

None of the patients with a benign lesion presented a complication and

related morbidity.

### 8.7 Follow-up

As a general rule the first postoperative imaging with CT was performed in the same day or the next day after the operation. MR was undertaken during the first 2-3 days after the operation.

Patients were usually evaluated at yearly intervals after treatment. Some patients who presented complete angiographic cure of their lesion in the final post-operative angiogram were checked regularly in approximately 1 year with MR and then in approximately 2 years and in case of confirmation of the initial result no other control was performed. In most of the cases, an individualized approach regarding follow-up was applied. In total, 151 patients were followed-up for 439 patient-years (mean 2.9 years ranging from 1 to 15 years). For patients with known incomplete occlusion or signs of recurrence either clinical or imaging the strategy was defined according to the specific symptomatology in combination with the MR imaging. In case of either severe symptoms or suspicion of CVD or both, then a DSA was performed and further embolization was applied. Four patients with an angiographic complete occlusion in the initial embolization session presented recurrent clinical symptoms during follow-up and underwent DSA and further embolization. One of them presented with a new bleeding. In 3 of these 4 re-embolized patients a complete occlusion was achieved whereas in one a subtotal occlusion was obtained. Five more patients with an initial angiographic complete occlusion presented MR findings indicating a persisting shunt in their regular imaging follow-up without clinical manifestation. They are just followed-up. Thirty-five patients with an incomplete occlusion in the last embolization presented a disappearance of vascular abnormalities (arterial feeding vessels, shunt or dilated veins) indicating a persisting DAVF in the follow-up MR examination (one after additional surgery – by-pass). For 92 patients the clinical and MR follow-up confirmed the known complete occlusion of their lesion, whereas for 11 patients the MR follow-up imaging confirmed the known incomplete

occlusion of their shunt; one patient of this group with persistent CVD presented cerebral bleeding and died. The remaining 10 patients had no clinical worsening and 5 of them had no evidence of CVD, therefore further follow-up and neither new embolization nor DSA control were suggested. Three recently treated patients with complete occlusion achieved, have no follow-up examination yet, whereas one more patient with complete angiographic occlusion died few weeks after his admission due to a severe brainstem bleeding which was the presentation mode of his AV shunt.

In total, of the 151 embolized and followed-up patients, 134 patients (4 recently treated patients without f-u yet are included) demonstrated a complete occlusion (88.7%) of their shunt and 17 patients an incomplete occlusion (either subtotal, extensive or partial); one patient with incomplete occlusion died as a result of a re-rupture of the CDAVF during f-up and one patient with complete occlusion of his CDAVF died as a result of his initial brainstem hemorrhage.

In total 3 patients were lost to follow-up; Two of them carried a benign lesion (Borden grade 1) with an initial extensive occlusion in the post-operative angiogram and the third patient had a Borden grade 2 lesion with an elimination of the CVD in the post-operative angiogram (Table 4).

#### 8.8 Other therapeutic modalities – Combinations

In this series only 2 patients both with Cavernous plexus lesions, who had mild orbital symptoms and slow flow in the diagnostic angiogram were treated with compression. One more patient diagnosed with MR in combination with the clinical picture, was discovered with spontaneous thrombosis of a Cavernous plexus fistula in the angiogram. In this consecutive series of patients no other therapeutic modality alone or in combination was applied prior to embolization, which was the first line choice for all cases. In one case of extensive and complex DAVF in the area of the Transverse sinus and after four embolization sessions resulting in subtotal occlusion, by-pass surgery with occlusion of the sinus was employed. In a

second case and after incomplete embolization, surgical operation was undertaken aiming to the complete elimination of the lesion. No patient was referred or treated by radiation.

## 9. DISCUSSION

Many publications have been appeared in the literature during the last 25 years regarding CDAVFs. These include angiographic predictors of hemorrhage<sup>(16, 22, 24, 25, 31, 71, 79, 130, 143, 151, 152)</sup> natural history<sup>(28,31,37,38,68,79,132-135)</sup> classification<sup>(15,18,40,62,71,130)</sup> but also various techniques and materials for treatment and results<sup>(34-36,38,61,167,174,221,224-286)</sup>.

Spontaneous or traumatic sinus thrombosis seems to be one the primary events, triggering the stimulation of angiogenesis and perhaps engorgement of the native microscopic arteriovenous channels that normally exist within the dura mater<sup>(18, 22, 47,62,129,130, 154-157)</sup>

A history of trauma<sup>(158-165)</sup> previous surgery<sup>(20,59,16-169)</sup> or clinical states associated with hypercoagulability, including infection<sup>(170-172)</sup> pregnancy<sup>(30,119,173-175)</sup> are reported in the literature. Nevertheless the largest percentage of cases, as in our series do not refer any such predisposing factors. Factors that could be considered as predisposing were recorded in only 17 patients (11%).

### 9.1 Angioarchitecture of CDAVFs

The term angioarchitecture refers to the angiographically demonstrable vascular elements composing a CDAVF and includes the feeding arteries, the shunt area, the draining veins and any associated vascular abnormalities either involved in the aetiopathogenesis process or secondarily induced by the lesion.

#### *9.1.1 Feeding arteries*

The arterial aspect of the dural fistula looks more interesting for planning of

treatment than for their development and manifestation. This is particularly illustrative of the importance of the venous aspect in the understanding of CDAVFs. The location of the lesion determines the arteries, which supply the shunt in a more or less predictable way. Variations in the combination of expected arterial feeders apparently reflect the normal variations of the territory covered by each individual artery. The feeding arteries in the vast majority of the cases are dural branches of the external carotid artery and/or internal carotid artery and vertebral artery. Skin and transosseous branches of the external carotid artery, primarily from the distal occipital artery and secondarily from the posterior auricular artery and superficial temporal artery often contribute to the shunt. Sometimes pre-existing but small dural branches of Superior Cerebellar artery, Posterior Cerebral artery and Anterior Inferior or Posterior Inferior Cerebellar arteries are observed having significant contribution to the shunt, which is usually located in the tentorium, torcula and Transverse sinus. This was observed in 11 of our patients with 7 lesions located at the tentorium area, 3 in Transverse sinus area and 1 in foramen magnum. All of them except one had CVD and aggressive presentation. In a minority of cases non-dural, cortical branches of the internal carotid and vertebral arteries are also involved. In our series this was observed in 4 cases (part of the previously 11 cases mentioned). Some noteworthy though not common characteristics of those cases were, multiple embolizations in one, extensive lesion with dementia in another and previous craniotomy in the other two.

Dural shunts supplied even by dilated and/or multiple dural or cortical branches of the internal or vertebral arteries have no significant hemodynamic effect on the brain.

On the other hand, symptoms potentially explainable by meningeal arterial steal of dural arteries supplying the cranial nerves are rarely referred. In our series no symptomatology attributed to such a mechanism was recorded.

Arterial aneurysms flow-related or not are practically not encountered. This is most probably related to the fact that dural arteries lie in the outermost, periosteal layer of the dura surrounded by connective tissue.

A distinct group of lesions are the so-called osteodural AVFs which are fed by meningeal arteries and lie actually within the bone either in the convexity or in the skull base. <sup>(55)</sup>

### 9.1.2 Shunt zone

Although the arterial network of the dura of the cranial vault and base, primary and secondary is actually located in the periosteal layer, the penetrating arteries cross the border between outer and inner layer and fall into the capillary bed, which lies, on the inner surface of the dura. <sup>(44)</sup> The vasculature in the falx cerebelli, falx cerebri, and tentorium lies within the opposing meningeal layers of the dura that have invaginated to form these partitions. <sup>(138,45)</sup> It is not clear if the capillary network of the falx and tentorium lies on the inner surface or not.

In addition, histopathological reports and studies of DAVFs in serial sections have been scanty. We know from them that arteriovenous fistulas consist of small shunts between dural arteries and dural veins, which lie in the wall of the dural sinus, rather than direct communications between dural arteries and the lumen of the dural sinus. <sup>(52,53,65, 139-142)</sup>

What we do not know is on which aspect of the dural layers the shunt zone lies, therefore in which microanatomic environment it is developed. In relation with this, it is interesting to notice that epidural hematoma is not included in the spectrum of the clinical manifestations of a non-traumatic CDAVF. This could imply that a dural “nidus” is not structurally directly associated with the periosteal side of the dura matter, but apparently this is not the case since several lesions located in the skull base area present a clear bony involvement. In any case the exact location of the shunting zone should be expected to depend on the structure of the dura and its relationship with the adjacent bone and leptomeninges. An arteriovenous shunt located in a convexity dural site should be different in terms of structure, evolution and clinical manifestation in comparison with a shunt located in the wall of a dural sinus, in the free margin of the tentorium, in the wall of a venous plexus or in

the transdural segment of a cortical vein.

Histopathological studies on CDAVF specimens revealed that the essential abnormality was a connection between the dural arteries and the dural veins within the venous sinus wall, through small vessels averaging approximately 30  $\mu\text{m}$  in diameter. By using several staining methods, they confirmed that the vessels were part of the venous system having an incomplete smooth muscle layer and lacking internal elastic lamina.<sup>(46)</sup>

### *9.1.3 Venous drainage*

The venous drainage of a CDAVF can employ a dural vein, a dural sinus or just a part or channel of a dural sinus,<sup>(143, 144)</sup> a cortical vein or a combination of the above. Employment of only dural veins represents the simplest form of a CDAVF venous drainage. The most usually encountered type of venous drainage is the one through a dural sinus. Sinuses can present morphological abnormalities (VOA) as occlusion corresponding to sinus thrombosis, stenosis related or unrelated to previous thrombosis, dilation or mixed changes located either in the proximity of the lesion or in a remote site. Moreover, venous sinuses and particularly the SSS normally present a multicompartmental structure and often one or some of these channels are exclusively involved in the drainage of the lesion offering thus a chance for superselective transvenous obliteration of the shunt leaving the sinus patent.

Cortical venous drainage can give to the venous system a phlebotic appearance with slow or even reversed flow or can present significant varices or/and stenoses.<sup>(153)</sup> These different patterns of appearance represent the degree of adaptation of the venous system and are determined by the time and progress rate of the shunt, the amount of the flow and the intrinsic capability related to the collateral pathways of the system to compensate the retrograde flow. Otherwise, CDAVFs developed in relation to the 3 epidural areas will predictably drain according to the specific role of the venous system of each region, unless there are associated conditions such as constraint placed on the venous outlet or high-flow arteriovenous shunts or other unknown factors that contribute to the re-direction of the flow to

alternative draining channels.

## 9.2 Association with other vascular or neoplastic lesions

Only limited information is available concerning association of CDVAFs and intracranial aneurysms. After an initial description in 1987<sup>(176)</sup> only some sporadic reports have appeared. <sup>(177-180)</sup> Suzuki et al reported an incidence of 13% in their series of consecutive 46 patients with dural AVFs. They were rather associated with shunts of the anterior cranial fossa and cerebral convexity.

Few case reports have focused on the coexistence of CDAVFs and moya-moya phenomena speculating on a potentially shared association with dural angiogenesis <sup>(181-183)</sup>

Dural and pial arteriovenous fistulas are uncommonly associated. Most of the few reports come from the Toronto group. They proposed that venous steal effect in the dural sinus secondary to the high-flow dural arteriovenous shunt induced the pial arteriovenous fistulas. <sup>(184-187)</sup>

And last, it is known the association between meningiomas and other benign or non-benign tumors with CDAVFs. Several publications have focused on the coexistence and potential etiologic relation between the two entities. <sup>(188-196)</sup>

In our series we observed 5 patients with a coexistent vascular malformation or anomaly. One patient suffered of multiple cavernomas of the cerebellum associated with a tentorial-torcular DAVF. One patient had a cavernoma, a DVA and a venous hemangioma of the maxilla associated with a temporopolar CDAVF. Two patients had associated pial AVFs (one of them with multiple dural and pial shunts along the SSS) and one patient had a DVA.

One more patient suffered of HHT with a family history and a polmonary shunt operated on in the past.

We also observed 5 patients with neoplastic disease, 3 of them being a meningioma topographically associated with the DAVF and one patient with Multiple Myeloma with a paraganglioma which is an extremely rare association <sup>(288)</sup>.

### 9.3 Presentation

Regarding the clinical presentation, a correlation between the venous pattern of CDAVFs and an aggressive clinical course has been established. <sup>(16,18,21-31)</sup> Fifty-one of 53 (96%) of our cases with aggressive clinical presentation had a CVD in the angiogram. The remaining 4% corresponds to 2 patients, one of them presented initially with SAH and treated partially elsewhere and the other one with a SSS located lesion presented with one single episode of epilepsy. Only a 61.5% of patients who were found with a CVD in the diagnostic angiogram had presented an aggressive clinical behaviour. As the presence of a bruit is frequently associated with CDAVFs involving the transverse-sigmoid sinus <sup>(21,33, 80, 170, 218)</sup> in our series 81% of the patients with lesion involving transverse and/or sigmoid sinus were presented or were complaining for tinnitus. Tinnitus was also a characteristic symptom for lesions located in the Jugular foramen. Fifteen of sixteen patients (94%) with lesions involving the Jugular foramen had tinnitus as one of the presenting symptoms. Also 9 of 26 patients (35%) with lesion involving the Cavernous plexus and 2 of the 3 patients (65%) with lesion of the Sphenoid wing had tinnitus among other symptoms at presentation. All 3 cases with dementia-like syndrome related to venous congestion-hypertention were patients with lesions of Transverse-Sigmoid sinus. Furthermore, drainage of cavernous sinus CDAVFs into the orbital plexus produces the "red eye" spectrum of symptoms from a mild chemosis and proptosis to severe exophthalmos with ophthalmoplegia and glaucoma <sup>(219-221)</sup>

In 21 of 24 patients with Cavernous plexus DAVF this spectrum of symptoms was the main clinical manifestation whereas 3 of the patients were presented with tinnitus and diplopia with (in 2) or without headache. For 5 other patients

with lesions located not in the cavernous plexus these signs constituted the presenting picture. In 2 of these patients the lesion was located in the jugular-hypoglossal foramen, in one patient in the temporopolar dura and in another 2 patients in the orbita.

One of the last and important contributions <sup>(222)</sup> of P. Lasjaunias was the new classification of CDAVFs according to the anatomy of the epidural venous system and the location of the fistula. In that publication by Geibprasert et al <sup>(40)</sup> a new step was made towards a better understanding of the anatomical characteristics and the clinical manifestations of such lesions. They found that the ventral epidural group showed a female predominance, more benign clinical presentations, lower rate of cortical and spinal venous reflux, and no cortical and spinal venous reflux without restriction of the venous outflow. The dorsal epidural group had a lower mean age and a higher rate of multiplicity. The lateral epidural group presented later in life with a male predominance, more aggressive clinical presentations, and cortical and spinal venous reflux without evidence of venous outflow restriction. Therefore, dural arteriovenous shunts predictably drain either in pial veins or craniofugally depending on the compartment involved by the dural arteriovenous shunt. Associated conditions (outflow restrictions, high-flow shunts) may change that draining pattern. The significant differences between the groups of the new classification support the hypothesis of biological and/or developmental differences in each epidural region and suggest that CDAVFs are a heterogeneous group of lesions

In our series most of the above observations were confirmed.

The VE group of lesions had the more benign presentation with only 5.7% of them presenting with aggressive symptomatology. The female predominance of 57% did not reach statistical significance.

Regarding the relation of the DE group with multiplicity, in fact all 20 cases with multiple lesions had a DE component, 15 of them with an additional VE component and 5 of them with a LE component. The age features of the DE

group could not be compared due to non-inclusion of children in the present series.

The LE group presented a male predominance (73%) as in Geibprasert et al series. The 86% of them had an aggressive presentation and 58% of the aggressive lesions had no VOA.

There were also a percentage (approx. 1/3) of patients in groups DE and VE, where a CVD was observed without any indication of high flow or restriction in the venous drainage downstream to the shunt either due to stenosis or thrombosis (Table 4).

The fact that nearly 40% of the lesions with CVD were presented with non-aggressive symptoms may indicate that these particular lesions might have a different natural history presumably due to their intrinsic capacity to compensate the hemodynamic stress of the arteriovenous shunt.

#### 9.4 Indications for Endovascular Treatment

The basic decision-making process regarding active treatment versus conservative treatment (compression when applicable, or observation) is based on the patient's neurologic and general condition at presentation, the evaluation of natural risks according to the angioarchitecture of the lesion and the anticipated results of a conservative approach versus potential risks or the different treatment modalities.

The patient's neurologic and general condition at presentation plays an important role. Patients presenting with ICH sometimes require emergent hematoma evacuation. In our series for none patient such an operation was necessary. Depending on the hemodynamic conditions the entire shunt and draining veins may be fully or partially compromised or thrombosed in the early acute phase. Regardless of the indication for urgent embolization, cerebral angiography should be repeated after resorption (or evacuation if indicated) of a hematoma to demonstrate the true angioarchitecture of the lesion. Embolization in a patient being investigated angiographically in the

acute phase of cerebral hemorrhage is indicated if a pseudoaneurysm, varix, CVD or severe venous congestion is being identified.

Since cranial dural arteriovenous fistulae represent a heterogeneous group of pathologies with different angioarchitecture and risks, the indications for treatment cannot be the same for each group or case. The primary goal of treatment of a cranial dural arteriovenous fistula with cortical venous drainage is the prevention of severe neurologic deficits either as a potential result of a rupture or as consequence of cortical venous congestion and infarction. On the other side the primary goal of a benign cranial dural fistula is the alleviation of persistent and irritating symptoms as tinnitus or more disabling ones as orbital symptoms – chemosis and exophthalmus - with or without nerves palsy and glaucoma.

The patient's tolerance to otherwise mild symptoms and his attitude towards active or conservative treatment play often a decisive role in the management of benign lesions. Generally, conservative treatment and follow-up in benign lesions with mild or none symptoms is the management of choice.

### 9.5 Diagnosis and Follow-up

Six-vessel angiography (bilateral internal, external carotid, and vertebral arteries) is the examination of choice for diagnosing and classifying <sup>(197-199)</sup>. The ability to define the arterial and venous patterns significantly increases the diagnostic value of angiography compared with computed tomography or magnetic resonance imaging <sup>(200-206)</sup>

Magnetic resonance angiography and venography provide a noninvasive method for defining dural arterial and venous anatomy <sup>(77,207,208)</sup> but the techniques have significant limitations. Resolution is suboptimal compared with that of conventional angiography, and flow dynamics of the lesion are poorly defined. Despite its small but definite risk <sup>(209)</sup> cerebral angiography remains the "gold standard" for assessing CDAVFs.

However the modality that can be used for follow-up of treated patients is a

different issue. As recent publications <sup>(210-211)</sup> and our experience have shown MR angiography techniques offer a reliable and non-invasive alternative to the digital angiogram. In our institution conventional MR imaging, MR angiography (3D-TOF-MRA), and source images, have been used for the visualization of intracranial DAVFs. The crucial question that could be discussed with regard to the follow-up imaging modality is whether such an MR imaging can reliably rule-out the presence of a small and slow flow residual or the presence of CVD. Based on the published reports the answer is apparently negative at least for the first part of the question. However, in an asymptomatic patient such a small and slow flow shunt is rather benign without clinical significance and simply requires follow-up to exclude progression to a higher-grade lesion. Therefore, the absence of evidence of any residual requires either the verification of digital subtraction angiography, which can then safely lead to the suspension of further controls, or alternatively the repetition for several years of the non-invasive MR control. Regarding the second part of the question it is evident that dilated cortical veins that usually signify CVD can be reliably detected by MRI-A. Non-dilated or slightly dilated cortical veins involved in a DAVF certainly have been observed but again they can be detected with flow sensitive MR techniques and moreover their significance in terms of risk for aggressive manifestations is questionable. In any case, at least at the moment DSA remains the gold standard imaging not only for diagnosis but also for follow-up. <sup>(212-214)</sup>

Other authors have proposed that duplex sonography can be used as screening tool with high specificity (99%) and moderate sensitivity (51% to 63%) for diagnosis of CDAVFs or with high diagnostic sensitivity and reliability for detecting DAVF in patients with pulsatile tinnitus. <sup>(215,216,217)</sup>

In our series all patients underwent a 6-vessel cerebral DSA at the stage of initial diagnosis. All patients underwent brain MRI-A for evaluation in addition to the diagnostic CT scan performed nearly in all acutely presented cases. In 43% of patients an initial diagnosis was made based on the MR findings. In 10 cases the initial diagnosis was made by U/S and was confirmed by either

MRI or/and DSA. In 5 cases only after DSA was the lesion revealed. For the rest of the cases no clear data were found.

### 9.6 Treatment and Complications

The decision of how to deal and whether to operate on a CDAVF should be based on the patient's clinical presentation, natural history, lesion angioarchitecture, and location of the lesion. Certain locations (i.e. lateral epidural space) and/or angiographic findings (i.e. presence of cortical venous drainage) are associated with a risk of hemorrhage and demand treatment.

The primary goal of treatment should be elimination of the cortical venous reflux if any and furthermore complete obliteration of the lesion. As experience has shown, complete and permanent obliteration of a CDAVF by endovascular approach is achieved by complete obliteration of the venous component of the lesion. This is a crucial aspect of the treatment of CDAVFs and requires that the embolic material promote the complete thrombosis of the venous part of the shunt. It explains also why some materials as PVA fail to induce permanent cure and present a high rate of recanalization. In some cases, however, palliative treatment for control of symptoms in cases of very complex and/or extensive lesions is a reasonable alternative. To relieve the symptoms, especially for patients in poor general medical condition, a palliative endovascular procedure even with a non-permanent embolic agent offers in some cases an acceptable therapeutic option with inconsiderable risk. Generally the risks of endovascular treatment include mainly infarction of cranial nerves and stroke whereas the limitations are related to the difficulty to access very tortuous arterial feeders (transarterial approach) or very dilated and fragile venous collectors (transvenous approach) <sup>(140, 223-237)</sup>

We reviewed all publications focusing to the endovascular treatment of cranial dural arteriovenous fistulas during last 25 years, which included more than 20 cases each (Table 5). We found 27 publications <sup>(35, 132, 238-262)</sup> in the English literature until 2009 presenting a total of 1213 cases of CDAVFs

treatment. Only 3 of these publications included more than 100 patients (135,141,150 patients respectively) with all the rest 24 publications with less than 60 patients each. These series are very heterogeneous in relation with almost all relevant data as location, venous drainage pattern, management, approaches and materials used. Therefore proper statistical analysis is not really applicable. Nevertheless, some rough conclusions can be drawn.

Regarding location of CDAVF, 14 publications reported on single-location lesions (6 of them in Cavernous plexus, 6 in Transverse-Sigmoid sinuses, one in Anterior fossa and one in the Tentorium) whereas the remaining 13 publications included lesions from various locations. Twenty-three of these papers reported data about venous drainage pattern. Three of them included only cases with cortical venous drainage whereas the twenty series included cases with various venous drainage patterns. In 14 of the publications data about venous outlet abnormalities are reported. In the vast majority thrombosis of the involved sinus is reported as the prevalent venous alteration.

Regarding treatment, which is the main purpose of this study, 973 of the 1213 cases were treated only with embolization (80%). Five publications refer the transarterial approach as the only approach used in the endovascular treatment of the included cases. In 18 series both transarterial and transvenous or combination were used. Only 13 publications reported data about number of sessions needed for achieving the goal of endovascular treatment. The mean number of embolization sessions ranged from 1 to 2.5 sessions per patient.

The materials used were reported in details in 19 series. In 15 series a combination of materials as NBCA, PVA and Coils were used in the majority of the cases treated. In only 4 of them a single material was used (1 series with coils as exclusive material and 3 series with onyx).

The immediate post-procedure angiographic outcome is described in 20 publications. From the single-location series the complete occlusion rate in the cavernous plexus lesions ranged from 37% (this percentage reached the 89% during a follow-up period of 2-16 months without additional interventions) to 90%. The complete occlusion rate for transverse-sigmoid sinus lesions ranged from 36% to 52%. Whereas for tentorium and anterior fossa the percentage of complete immediate angiographic occlusion was not reported. From the mixed-location series the complete occlusion rate ranged from 20% to 91%.

The early clinical outcome is incompletely described in 20 publications. In the single-location group series the percentage of early clinical cure ranged from 30% to 87%. This percentage reached the 96% after a 1-4 months follow-up period in one series of cavernous plexus lesions. For the mixed-locations group the percentage of early clinical cure ranged from 30% to 83%.

The operative complication rate and the related neurological morbidity ranged from zero to 23% transient and 12.5% permanent neurological deficit. Both high percentages of transient and permanent morbidity were appeared in series with Onyx as the main or exclusive embolic material. No deaths were reported.

Only 13 publications provided, though incomplete, clinical or imaging follow-up data. The follow-up interval ranged from 3 months to 56 months.

Our series has certain particular features. It is one of the largest series of DAVF including lesions from various intracranial locations, focusing on endovascular treatment, operative results and complications and having a long-term follow-up. It comprises of 154 consecutive patients treated exclusively endovascularly with the exception of 2 cases where surgical intervention was also employed aiming to the complete eradication of the lesion and 3 cases where conservative treatment was applied. In another case of torcular DAVF a craniectomy was applied in the early post-operative

period, aiming to a decompression of the posterior fossa and not to the elimination of the lesion itself.

If we examine the patients endovascularly treated in the present series divided in two groups, the “benign” group (Borden gr 1) and the “aggressive” group (having CVD) comprised of 71 and 83 patients respectively, then we should notice that 63.5% of the “benign” group had an anatomic cure in the post-operative DSA without complication and permanent morbidity. On the other hand, 70% of the patients of the “aggressive” group had an anatomic cure in the post-operative angiogram with all permanent-morbidity cases belonging to this group.

During the follow-up period 86% of patients with benign lesions demonstrated a complete occlusion (2 were lost to f-up) whereas 88 % of the aggressive group demonstrated a complete occlusion (one was lost to f-up). Therefore, the total complete occlusion rate was 87%.

Otherwise, 128 (83%) of the 154 patients treated endovascularly presented either clinical cure or no new neurological deficit in case of preexisting deficits. Another 18 patients presented clear early improvement of their symptoms and 4 more patients at discharge after subsidence of post-operative temporary neurological manifestations. Four patients presented a clinical worsening related to a complication. One patient died during the initial hospitalization due to his presenting ICH. Two patients presented cerebral bleeding during f-up. The first patient had a subarachnoid-subdural hemorrhage without new neurological deficit whereas the second patient died. In total, 96.7% of our treated patients presented clinical cure or improvement of the presenting symptoms at discharge, without new neurological deficits.

Four patients (2.6%) suffered a complication. In one of these cases, spasm of the superior cerebellar artery was only obvious before the end of the procedure. The immediate post-embolization employment of an open

surgical approach aiming to a complete elimination of the lesion and resulting in a cerebellar infarct makes the exact estimation of cause and effect difficult. Nevertheless we included this case in the endovascular treatment-related complications due to the potential relation of cerebellar infarct with the spasm of the SCA. These percentages of successful treatment are among the highest with an associated complication rate among the lowest.

### 9.7 Materials used and long-term result

Recently a new embolic material called Onyx (ev3 Neurovascular, Irvine, CA) have been developed and is reported as a kind of "gold standard" at least for the embolization of CDAVF.

Cognard et al <sup>(257)</sup> reported a prospective series of 30 patients treated with Onyx with an anatomic cure of 80% achieved and 6.6% complication rate (rebleeding and cranial nerve palsy)

Xian Li et al <sup>(255)</sup> reported a 61.3% complete anatomic cure with clinical complication rate of nearly 20% in a series of 31 patients treated by Onyx.

In 2009 several more small series including 5 to 30 patients have been published with complete occlusion rates ranging from 72% to 96% and clinical complication rates from zero to 10% <sup>(264-266)</sup>

Wang et al published on five cases of an unusual complication with symptomatic Onyx migration to the heart and downstream to the draining vein of the lesions, questioning the consideration that Onyx is a controllable material.<sup>(267)</sup>

In early 2010 several more publications appeared with complete anatomic cures ranging from 62.5% to 92% and clinical complication rate from zero up to 8% <sup>(268-272)</sup>

Guedin et al presented a series of 42 consecutive CDAVF cases with CVD treated by transarterial embolization with NBCA achieving a complete

occlusion rate of 81% and zero morbidity and mortality, aiming to emphasize the importance of transarterial embolization using acrylic glue in the therapeutic management of such lesions, and to compare the results the authors obtained using this treatment with those reported in the literature for Onyx treatment of the same type of dural shunts. <sup>(273)</sup>

Our skeptical attitude towards Onyx is based mainly on the achieved good results with low complications rate and few sessions by using other embolic materials and especially NBCA. Additionally it correlates with our criticism toward a philosophy that seems to inspire the whole process of embolization with Onyx, a philosophy of non-selective attack not only of the lesion but rather all the adjacent vascular structures often in a surprisingly great extent in an attempt to include the lesion in the attacked area, often with an unacceptable rate and/or kind of complications <sup>(274)</sup>

Often an indirect allusion is made that a high angiographic cure rate cannot be achieved without paying a price (anecdotal data), thereby overestimating the value of an imaging result against the actual clinical condition of each individual patient.

As it was mentioned in the results, 27 patients with lesions presenting very tortuous feeders and diffuse supply or lesions with minimal supply from tiny and/or dangerous feeders, PVA was used as the only embolic material throughout their management. One of these patients manifested slight clinical (bruit) or imaging indications of recurrence but no further treatment was suggested. Fourteen other patients with initially obtained complete occlusion plus five more patients with initial incomplete occlusion remained without clinical symptoms and with MR imaging confirming the initial result. Seven patients with incomplete initial occlusion showed complete disappearance of vascular abnormalities indicating presumably a progression to complete occlusion.

## 9.8 Alternative treatment modalities

Gamma knife treatment (GKT) is considered an alternative treatment for CDAVFs. Major disadvantages of this therapy include the time elapsed before obliteration and the possibility that not all shunts will be obliterated. Actually the reported results after 24 months are showing complete occlusion rates between 50 and 75% <sup>(275-277)</sup>

The vast majority of the benign lesions after observation or palliative treatment have a benign course<sup>(128)</sup> with extremely low risk.

Therefore, a curative g-knife treatment for these lesions is unnecessary, because the (long-term) morbidity and mortality rates will most probably not outweigh the natural course of the disease.

Lesions with CVD on the other hand have a contraindication for radiosurgery mainly because its result is not achievable in a timely fashion. Consequently, GKT can be theoretically used only in the treatment of benign CDAVFs with subjectively intolerable symptoms or aggressive CDAVFs not accessible or not responsive to endovascular treatment or surgery.

In our series no case was referred for g-knife treatment because for none of our patients it was judged that further treatment with radiosurgery was necessary or in cases of incomplete occlusion that it would offer any additional benefit for the patient.<sup>(275-277)</sup>

Surgery for the obliteration of DAVFs is frequently associated with higher risks,. <sup>(32, 278-281)</sup>

Some groups have suggested selective surgical disconnection of the draining cortical vein as a minimally invasive and effective treatment for lesions with CVD. Actually, simple surgical disconnection of the cortical venous reflux maybe a good option in the management of patients with Borden type II or even type III superficially located dural arteriovenous fistulas when anatomic features prevent endovascular access, or embolization fails to obliterate the lesion. This procedure is a much smaller surgical undertaking and is associated with fewer complications than attempts to resect or pack the whole fistula, especially if located in the skull base. <sup>(282-286)</sup>

For a minority of cases, a combination of treatments offers a better chance for success. Most of the lesions, however, require only one approach and

this is the endovascular approach. For some particular locations as for example, anterior fossa CDAVFs surgical obliteration alone is sometimes advisable when endovascular approach fails.

## **10. CONCLUSION**

Endovascular embolization is the treatment of choice for the treatment of CDAVFs. High percentage of anatomical cure or subtotal-extensive occlusion of the lesion with even higher rates of excellent clinical results associated with very low operative complication rate can be achieved with few embolization sessions per patient. In our view, these results can be reliably achieved through a philosophy of selective attack of the AV shunt and especially its venous component, mastering a certain permanent embolic material and avoiding both overtreatments and aggressive approaches based on overestimated imaging features and ignoring the actual clinical aspect of the disease.

## 11. TABLES

<i>Borden Classification</i>	<i>Cognard Classification</i>
Type 1: DVS/MV outflow only	Type I: DVS/MV outflow only (anterograde)
	Type IIA: DVS/MV outflow only (retrograde)
Type 2: DVS/MV outflow &CVD	Type IIB: DVS/MV outflow (anterograde) & CVD
	Type IIA+B: DVS/MV outflow (retrograde)&CVD
Type 3: CVD only	Type III: CVD only (no ectasia)
	Type IV: CVD only (ectasia)
	Type V: CVD only (spinal)

Table 1. Summary of classification schemes

Abbreviations: AVF: arteriovenous fistula; DVS: dural venous sinus; MV: meningeal vein; CVD: cortical venous drainage.

Borden	No Patients	Cognard	No Patients
Type 1	74	Type I	39
		Type IIa	35
Type 2	32	Type IIb	10
		Type IIab	22
Type 3	51	Type III	23
		Type IV	22
		Type V	6

Table 2. Venous drainage according to Borden and Cognard classification

CLASS	BENIGN	VOA	AGGRE SSIVE	VOA
B1	71	7 Thr, 6 Ste, 5 Var, 3 Dil	2	1 Ste
CI	36	1 Thr, 4 Ste, 2 Var, 1 Dil	1	1 Ste
CIIa	35	6 Thr, 2 Ste, 3 Var, 2 Dil	1	
B2	21	5 Thr, 5 Ste, 1 Ste+Var	10	2 Thr, 2 Ste, 2 Var, 1 V+S
CIIb	4	2 Ste	5	1 Ste, 1 Var, 1 V+S
CIIab	17	5 Thr, 3 Ste, 1 Ste+Var	5	2 Thr, 1 Ste, 1 Var
B3	9	2 Thr, 1 Ste, 1 Var	41	4 Thr, 14 Var, 1 V+T, 1 V+S
CIII	8	2 Thr, 1 Ste	15	1 Thr, 1 Var, 1 V+T, 1 V+S
CIV	1	1 Var	20	3 Thr, 12 Var
CV			6	1 Var
VE	49	5 Thr, 2 Ste, 4 Var, 1 Ste+ Var, 3 Dil	3	1 Thr, 1 Ste
DE	28	9 Thr, 4 Ste	17	5 Thr, 2 Ste, 3 Var, 1 Ste+Var, 1 V+T
LE	5	1 Var	31	12 Var, 1, 1 V+S
VE+DE	16	1 Thr, 6 Ste	0	
DE+LE	3		2	1 Var

Table 3. The clinical presentation in relation to classifications according to venous drainage and epidural space

Abbreviations: CLASS=classification, C(I to V)=Cognard classification, B(1 to 3)=Borden classification, VOA=venous outlet abnormality, Thr=thrombosis, Ste=stenosis, Dil=dilatation, Var=varix

<b>Post-op</b>	<b>Follow-up</b>	<b>No</b>
CAO	Clinical & MR follow-up confirmed the known CAO. One pt died because of MM	92 pts
CAO	Clinically recurrent symptoms (1 bleeding) → DSA and embolization →(3 Complete, 1 Subtotal)	4 pts
CAO	MR: persisting shunt-no CVD – Clinically: asympt → F-up	5 pts
InAO	MR: occluded shunt - Clinically: asymptomatic → F-up	35 pts
InAO	Clinical & MR follow-up confirmed the known InAO (1 pt with persistent CVD → cerebral bleeding and death). 5 pt with CVD.	11 pts
InAO	Lost to F-up	3 pts
CAO	Recently treated 3 pts have no follow-up, (1 pt died→ brainstem bleeding).	4 pts

Table 4. Follow-up

Abbreviations: CAO: Complete angiographic occlusion, InAO: Incomplete angiographic occlusion

	<b>Gender</b>	<b>Mean Age</b>	<b>CVD</b>	<b>VOA</b>	<b>VOA in CVD</b>	<b>High Flow in CVD</b>	<b>?</b>
<b>VE</b>	57% F	61.3 y	20%	32.5%	60% of CVD	10% of CVD	30% of CVD
<b>DE</b>	58% M	53 y	49%	55.5%	61% of CVD	4.5% of CVD	34.5% of CVD
<b>LE</b>	73% M	54.4 y	100%	37%			

Table 5. Epidural space in relation to gender, age, CVD & VOA

SERIES	No of Pt	M\F	Age (mean/range)	Single/ Multiple	F-u	Predisposing factors	Presentation	Imaging	Location	Venous drainage	Venous outlet alteration	Management	Approach	Materials used	No of Sessions	Angiographic outcome	Early Clinical outcome	Operation Complications	Follow-up Outcome Changes
V. Halbach et al 1987	28	13\15	53y/28y-72y	-	19m	-	H:8 O:20	-	TS:28	-	SIT:18% - 32% "abnormal" venous drainage	C:5 E:17 E+S:6	TA:28 (?)	G:15 P:3 G+P:5	-	C:10 InC:7 (13 if Co with S is included)	C:10 Im:6 S:1 (C:14 Im:8 with Sx)	P:2	-
V. Halbach et al 1987	30	7\22	47y/22y-75y	29/1	31m	P:4	OVH:77% CNP:50% DVA:27% O:47%	-	C:30	IIB:10%	-	Comp:8 E:20 Co:2	TA:22(?)	G:11 P:6 G+P+:5	-	C:18 Im:4	C:18 Im:4	Tr:2 P:1 AC:1	-
Fernand et al 1987	43	15\28	53y/19y-82y	1 bilat	40m	T:7 IH:1 TPh:1	O:43 IH:3	3pt CT normal	TS:41 JB:2	IIA:12 - IIB:4	SIT:20 SIS:2 11T/S?	C:7 E:34 S:2	Not mentioned (TA?)	G:7 P:19 D:27	54	Not mentioned	C:13 Im:30	0	2 pt more no tinnitus (15) - Im:9 - S:9 - W:1
Debrun et al 1988	37	-	-	-	-	-	O CNP DVA	-	C:37	-	Partial thrombosis of C sinus in 28 pts	E:29 S:1 C:3	TA all 37	G:11 P:14 G+P:10	35	C:14	C:14 Im:15		
Urtasun et al 1996	24	9\15	58y/25y-87y	24/0	C:10.8m 96%pt A:12.7m 62%pt	T:4 S:1 M:2	H:2 I:1 Nd:1 S:1 LVH:1 IH:5 O:19	-	TS:20 SS:4	I:13% - IIA:29% - IIB:29% - IIB:29%	75%SIT 25%SIS 33%AT	E:22 Co:2	TV:5 TAV:19, including 2:O	C:13 G+C:11	61 (32TA - 26TV - 3TAV)	C: 71% S: 12% M:17%	83%:C 13%:I 4%:S	Tr:8% P:8% D Un:4%	4% angio C - 8% clinical W
Bavinski et al 1996	30	11\19	54y/18y-85y	-	48.3 m	T:2 I:4	OVH+O:18 H+S+Nd+IH:12	-	TS:14 C:10 JB:1 T:5	IIB/III: 30%	13T 1S 6VD	C:2 E:20 Co:8	TA:26 TAV:2	G+P:2 G+C:2 G:4 C+P:11 G+P+C:3 C+D+F:5 C+P+F:1	57 (54TA - 3TAV)	C:6 InC:22	C:10 Im:11 S:3 W:3 D:1?	P:7% P Un?:3%	5 spont angio C - 4 C after Sx/Rx

SERIES	No of Pt	M\F	Age (mean/range)	Single/Multiple	F-u	Predisposing factors	Presentation	Imaging	Location	Venous drainage	Venous outlet alteration	Management	Approach	Materials used	No of Sessions	Angiographic outcome	Early Clinical outcome	Operation Complications	Follow-up Outcome Changes
Olteanu-Nerde et al 1997	30	17\13	52y/17y-82y	-	6m-24m	S:1 AVM:1	H:5  Nd:5  S:1  DVA:1  IH:4  O:30	-	TS all 30	I:60% IIA:16% IIB:16% IIab:3.5% III:3.5%	20% venous stenosis or occlusion	E:22  E+S:8	TA:21 TV:1	P:13  G:9  P+G:6 Bal:1  Co:1	59	C:10(45.5%) InC:12(54.5%) or C:33.3 - InC:66.6%	C:10+8 Im:11  S:1	Tr:2(6.6%) P:1(3.3%)  they state 3/59(5%)	-
Dawson et al 1998	24	9\15	54y/40y-80y	-	30m	T:2 remote	H:1  Nd:2  CNP:1  O:31	-	TS all 24	I:29% Ila &b:50% IIab:16.5%, III: 4.5%	-	E:22  E+S:2?	TA:9  TV:8 TAV:7	... and ethanol	-	-	C:21 (1 with Sx) Im:2  S:1	P:2	-
Shah et al 1999	41	11\30	1y-79y	-	-	-	H:3  CNI:6  O:41	11CT inaccurate/ 10MR showed abnormalities	TS all 41	I:12% - II:49% - III:29% - IV:10%	-	Comp:3  E:25  (2 of them +comp) E+S:13	TA:36 TV:16	G:7  P:25  C:22  Eth:8	52	C:14(37%) InC:28(63%)?	C:34(83%) all modalities	P:6(14.6%) all modalities	Angio C:17(41.4%) all modalities
Friedman et al 2001	23	5\18	57y/33y-79y	-	21m for 17pts	-	H:2  CNI:2  O:22	-	TS all 23	I:52% - IIA:30% - III:17%	SIT:7  SIS:2	E+R:20(87%) R:3	TA all 20	Mainly P (few+ gelfoam & silk)	-	-	C:20(87%) Im:2(9%) S:1(4.5%)	Tr:1(5%)  + Tr1 & P:1 in angios	In 21m for 17p angio/ C:7(41%) - S:4(24%) - M:6(35%)
Kim et al 2002	53	20\3	45y/1m-71y	50/3	25.5 m	T:5  S:1  C:2  I:1  P:1	A:2  H:6 - Nd:4  S:2 -  OVH:27 -  DVA:10 -  CNP:12 -  IH:28 -  O:3	-	TS:12 C:34  JB:1  SS:3  T:1  FM:2	I+IIA:62% - :38%	58.5% T of S and/or V	C:13  E:38  S:1  Co:1	TA:28 TV:7 TAV:4	-	71 (58TA - 9TV -4TAV)	C:33.3%  InC:66.6%	C:33.3% Im:30.7%  SW:25.6%  L:10.2%	Tr:%  P:3.9%  AC:10%	-
Meyers et al 2002	135	36\99	60y/18y-87y	-	56m	P:6	H:2  OVH:125  DVA:46  CNP:92  O:66	-	Call 135	IIAB:30%	-	C:1  E:133	TV:101 TA:32?	C:87% Silk:13% G:3% data for TV appr. only	179	C:121  InC:14	C:121 over a 5.4m mean period	AC:0.7% P:0.7%  O:5%	72 F-u DSA - 11 dead un. - 28 lost after mean 20m

SERIES	No of Pt	M\F	Age (mean/range)	Single/ Multiple	F-u	Predisposing factors	Presentation	Imaging	Location	Venous drainage	Venous outlet alteration	Management	Approach	Materials used	No of Sessions	Angiographic outcome	Early Clinical outcome	Operation Complications	Follow-up Outcome Changes
Tomak et al 2002	22	16\6	50y/35y-80y	-			H:60% Nd:18.5% S:4.5% O:18%	-	T all 22	III:100%	14% sinus thrombosis, 77% venous varices	E:45% E+S:55%	TA:18 TV:4 TAV:1	-	-	-	Exc:77% Good:9% Fair:5% D:9%		
Chung et al 2002	60	22\38	53y/4y-75y	-	14m	T:4 S:1 STh:8 Pinf:1 Ac:3	A:2 H:13 I:1 Nd:2 OVH:48 CNP:49 DVA:1 O:15	30pt CT:70% abnormal 36pt MRI: 81% abnormal	TS:12 C: 34 JB:2 T:3 AF:4 Con:3 Torc:1 Petrous surface:1	I:38% - IIA:5% - IIB:5% - IIAB:13% - III:20% - IV:18%	-	C:13 E:33 S:5 R:5 Co:2	TA:17 TV:11 TAV:5	-	62		C:19 Im:22 S:10 W:7 D:2	Tr:2	
Cheng et al 2003	27	3\24	29y-79y	12 bilateral	26m	-	OVH:26 DVA:7 CNP:5 O:3	-	C all 27	I:48% - IIA:4% - IIB:48%	SIT:9 bilat SIT:3 unilat	E:27	TV:26 TAV:1	C:all	-	C:10 InC:21	C:26 in 1-4m	Tr:2	14 more pts angio cure=24 pts
Klisch et al 2003	25	8\17	57y	-	30m	-	OVH:8 DVA:6 CNP:17 O:9	-	TS:14 C:11	I:48% - IIA:8% - IIB:4% - IIAB:32% - III:8%	-		TA:15 TV:27 (6 with Stent assist)	-	-		C:19 Im:6	Tr:4 AC:3 O:3	
Kim et al 2006	56	11\45	57y/30y-73y	-	13m	-	OVH:30 CNP:26 DVA:7 O:31	-	C all 56	-	-	E all 56	TV:50 TA: 11+6(?)	-	-	C:52% S:23% M+S:25%		Total:11 (19.6%) Tr:9 P:2	C+Im:42(91%) - S:2 - Recur:2
Kirsch et al 2006	141	29\112	67y/23y-85y	-	52m (for 69pts)	-	OVH:94% CNP:54% DVA:28% O:19%	-	C all 141	IIA:88% - III or/and IIAB: 34%	-	E all 141	TA:32 TV:161	P:32 C:161	193	C:81% S:13% M:4% F:2%	C:30% Im:57% S:10% W:3%	Tr:2 AC:9 O:2	55% of pts: C:94.5% - Im:5.5%

SERIES	No of Pt	M\F	Age (mean/range)	Single/ Multiple	F-u	Predisposing factors	Presentation	Imaging	Location	Venous drainage	Venous outlet alteration	Management	Approach	Materials used	No of Sessions	Angiographic outcome	Early Clinical outcome	Operation Complications	Follow-up Outcome Changes
van Rooij et al 2007	29	24\5	54y/24y-77y	-		-	A:4(14%) H:18(62%) S:4(14%) Visual:2(7%) O:1(3%)	CT/ICH:16 pt - SAH:2pt MRI/Dilated veins: 4pt	-	IIAB and/or III 100%	Sinus occlusion: 4(13.8%)	E:14(48%) E+S:7(24%) S:5(17%) R:1(3.4%)  plus 2 spont cure post H and I	-	G+C	-	C:14(66.6%) In:7(33.3%) (these 7 C if Surgery is included)	-	P:1(4.7%)	-
Xianli Lv et al 2008	31	21\10	43y/23y-60y	-	7.8m	-	H:12 Nd:1 OVH:9 O:13	-		I:10% - IIA:35% - III:10% - IV:45%	VS+VD in 51.6% (III & IV)	E all 31	TA:31 TV:1	O	-	C:61.3% InC:28.7%		TR:2 P:2	At F-u GOS 5:90.3%, GOS 4:9.7%
Xianli Lv et al 2008	23	14\9	49y/11y-70y	-	22.1 m	-	H:1 Nd:1 S:1 OVH:14 CNP:8 O:14	-	TS:3 C:17 T:1 AF:2	IIA:74% - IIAB:4.5% - III:17% - IV:4.5%	-	E:23	TA:6 TV:28	C:24 O:4 C+O:6	34	C:21 InC:2	C:12 Im:5 S:5 W:1	Tr:2	8 f-u angio:1 recanal - Cl f-u C:21 - S:1 - W:1
Cognard et al 2008	30	18\12	62.4y/36y-76y	-	3m	TPh:5 T:3	A:2 H:16 I:2 Nd:1 S:4 DVA:1 IH:1 O:1	-	TS:15 SS:4 T:5 FM:1 AF:2 VG:1 FC:1 SphS:1	IIIB:20% - IIAB:13% - III:27% - IV:40%	SIT:10	E:26 E+S:2 E+R:2	TA all 30	O	at least 35	C:24 InC:6	-	P:2	F-u in 23 pts confirmed results
Xianli Lv et al 2009	21	14\7	46y	-	28m	-	H:3 Nd:5 OVH:7 DVA:6 O:12	-	TS:7 C:6 T:4 AF:2 Torc:1 Cerebellar convex:1	-	-	E all 21	TA:21 TV:1	O:15 O+C:6	-	C:14 InC:7	-	Tr:5 P:1	

SERIES	No of Pt	M\F	Age (mean/range)	Single/ Multiple	F-u	Predisposing factors	Presentation	Imaging	Location	Venous drainage	Venous outlet alteration	Management	Approach	Materials used	No of Sessions	Angiographic outcome	Early Clinical outcome	Operation Complications	Follow-up Outcome Changes
Cohen et al 2009	42	19\23	62y	-	26.3 m	-	"Neuro":35% Other:56% OVH:37%	13pt CT:2 abnormal - 26pt MRI:13 abnormal	TS:56.5% Petrous ridge:39% Both:4.5%	I:19.5% - IIA:21.8% - IIB:17.4% - IIAB:24% - III:4.3% - IV:15%	-	C:2.2% E:71.7% S:6.5% E+S:19.5%	-	-	-	C:90%(?)	?	Tr:2 P:1 AC:6	-
Agid et al 2009	24	22\2	57y/3y-77y	22/2	15.8 m	-	A:25% H:46% Nd:8% S:8% O:13%	17pt CTorMR: H:10 - E:4 - Venous dilat:16	AF all 24	III:100% in 46% venous pouch	-	E:11 S:11 R:2	-	-	-	-	Embo: C:7/11	None embo	
Xianli Lv et al 2009	40	29\22	43y/23y-60y	-	8m	-	H:16 OVH:11 CNP:2 O:24	-	-	I:7.5% - IIA:32.5% - III:7.5% - IV:45% - V:2.5%	-	E all 40	TA:40 TV:1	O all 40	-	C:25(62.5%) InC:15		Total:9(22.5%) Tr:4 P:5 State:7.5 P but it appears:10-12.5%	
Kirsch et al 2009	150	71\79	58y/18y-82	9 bilat	13m	-	H:15, I:3 Nd:1, IH:6 DVA:11 S:5, O:150 "org brain dysfunction":3	-	TS all 150	I:31% - IIA:29% - IIB:20% - III:17% - IV:3%	SIT:79 AT:1	E all 150	TA:33 TV:21 TAV:96	G:34 P:64 C:21 G+P:16 G+P+C:5 P+C:8 G+C:1 S:11	50pts:1 session mean: 2.4	C:79(52%) InC:71	C:110 (73%) Im:23 S:13 W:3	Total:13(9%) Tr:10 P:3	46% of pts clin F-u in 13m (m):6 more C & 1 Im. Angio F-u 44% of pts: 33 InC turned to C-2 C recurrent

Table 6

FOLLOW-UP: C: Clinical, A: Angiographic

PREDISPOSING FACTORS: T: Trauma, S: Surgery, M: Medications, C: Congenital, I: Infection, P: Pregnancy, IH: Intracr.Hypertension, TPH: Thrombophlebitis, STh: Sinus Thrombosis, Plnf: Previous infarction, Ac: Acupuncture

PRESENTATION: A: Asymptomatic, H: Hemorrhage, I: Infarction, Nd: Neurodeficit non bleeding/infarction, S: Seizures, OVH: Orbital Venous Hypertension, DVA: Decreased Visual Acuity, CNP: Cranial Nerve Palsy, IH: Intracranial Hypertension, Other: Tinnitus, headache etc

IMAGING: N: Normal, E: Edema, I: Infarction, H: Hemorrhage

LOCATION: TS: Transverse Sigmoid sinus, C: Cavernous plexus, JB: Jugular bulb, SS: Sup Sagittal sinus, T: Tentorium, FM: Foramen Magnum, AF: Anterior Fossa, Con: Convexity, VG: Vein of Galen, FC: Falx Cerebri, SphS: Sphenoparietal Sinus

VENOUS DRAINAGE: AS: Antegrade Sinus, RS: Retrograde Sinus, RC: Retrograde Cortical, RSC: Retrograde Sinus and Cortical, C: Cortical only, CC: Cortical Congestion, S: Spinal

VENOUS OUTLET ALTERATION: SIT: Thrombosis inv. Sinus, SIS: Stenosis of inv. Sinus, AT: Thrombosis of another S, AS: Stenosis of another S, VT: Draining vein thrombosis, VS: Draining vein stenosis, VD: Draining vein dilatation

MANAGEMENT: C: Conservative, E: Endovascular, S: Surgery, R: Radiosurgery, Co: Combination

APPROACH: TA: Intrarterial, TV: Intravenous, TAV: Combined, O: Other (percutaneous, Burr hole etc)

MATERIALS: G: Glue, C: Coils, P: PVA, O: Onyx, D: Dura, F: Fibrin, S: Stent, Eth: Ethanol, Co: some combination

ANGIOGRAPHIC OUTCOME: C: Complete occlusion, S: Significant occlusion, M: Moderate occlusion, SI: Slight occlusion, F: Failure, InC: in the range of S, M, SI

EARLY CLINICAL OUTCOME: C: Cure, Im: Improvement, S: Same, W: Worse, D: Death, L: Lost to f-u

OPERATIVE COMPLICATIONS: Tr: Transient Neurologic Morb, P: Permanent Neurologic Morb, D: Death, Un: Unrelated, AC: Asymptomatic complication, O: Other non Neurologic

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### 13. CURRICULUM VITAE

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 1981-1983 Facolta di Medicina e Chirurgia, University of Naples, Italy and  
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