Clin Neuroradiol (2019) 29:375–378
https://doi.org/10.1007/s00062-018-0737-6

CORRESPONDENCE

# Reversal of Cortical Venous Reflux in Dural Arteriovenous Fistula with Change in Blood Pressure

**Christine A. Meadows[1] · Andrew Carlson[2] · Peter Vorobieff[3]**

Received: 5 September 2018 / Accepted: 19 October 2018 / Published online: 6 November 2018


## Case Report

Dural arteriovenous fistulas (DAVF) are acquired vascular malformations involving the dural venous sinuses [1]. Management and treatment of these lesions is complex, and often guided by the presence of cortical venous reflux (CVR) [2]. Although benign fistulas (without CVR) may progress to more aggressive fistulas at a rate of approximately 1% per year, and 10% of fistulas may spontaneously resolve, we are not aware of any previous report of fluctuation in CVR due to hemodynamic changes [2].

The patient was a 61-year-old male who presented with partial seizures with secondary generalization and pulsatile tinnitus. He was found to have an superior sagittal sinus (SSS) dural arteriovenous fistula (DAVF) with cortical venous reflux (CVR) (Fig. 1). Because of the presence of CVR and the patient's neurologic symptoms, the decision was made to treat the lesion. During the treatment procedure, there was lack of distal access to the fistula, and transarterial embolization was not thought to be feasible because a dysfunctional segment of the SSS could not be clearly defined for venous sacrifice. Thus, surgical disconnection was performed. Although this procedure initially seemed to be successful based on intraoperative angiography, the patient returned for the 6-month angiogram with recurrent pulsatile tinnitus and had significant recanalization of the fistula. At this time, there was bilateral CVR in multiple veins along a segment of the SSS (Fig. 2). At the time of this diagnostic angiogram, the patient's systolic blood pressure ranged from 150–160 mm Hg. Because of what was now interpreted to be a clearly dysfunctional segment of the sinus, the decision was made to sacrifice that segment. At the time of the treatment angiogram (approximately 3 weeks later), the patient was relatively hypotensive under anesthesia with the systolic blood pressure ranging from 80–100 mm Hg. The angiogram revealed no sign of cortical reflux. The veins which were refluxing from the external carotid artery injection during the diagnostic angiogram were now draining anterograde into the sinus from the internal carotid injection (Fig. 2).

Because of this new finding, no dysfunctional segment could be defined, so transarterial embolization through a middle meningeal branch was attempted. This resulted in successful filling of a parallel channel in the SSS. The remainder of that channel was occluded with coils transvenously to the level of the first normal draining vein, resulting in angiographic cure (Fig. 3).

The patient remained neurologically intact and was discharged home the next day. A 6-month angiogram showed no sign of recurrence.

## Discussion

Due to their benign natural history, most cases without CVR are managed conservatively [3]. When CVR is present, a yearly mortality of 10.4% has been reported with annual hemorrhage risk of 8.1% and neurologic deficit risk of 6.9% [2]; however, in asymptomatic cases even with CVR, the yearly hemorrhage rate may be a low as 1.4–1.5% according to more recent data [3].

This is the first reported case of reversal of CVR with changes in blood pressure. The authors are aware of one other case of a patient treated with venous sinus stenting which resulted in resolution of outflow obstruction and reversal of CVR as expected [4] but we believe our case to be a very different mechanism. A mechanism of spontaneously resolving outflow obstruction or stenosis seems unlikely due to the lack of observed angioarchitectural changes and the short time between diagnostic angiogram and treatment.

✉ Andrew Carlson
AndrewCarlson@salud.unm.edu

[1] University of New Mexico School of Medicine, 1 University of New Mexico, Albuquerque, NM, USA

[2] Departmentof Neurosurgery, MSC10 5615, 1 University of New Mexico, Lomas Blvd. NE, Albuquerque, NM 2211, USA

[3] Department of Mechanical Engineering, 1 University of New Mexico, Albuquerque, NM, USA

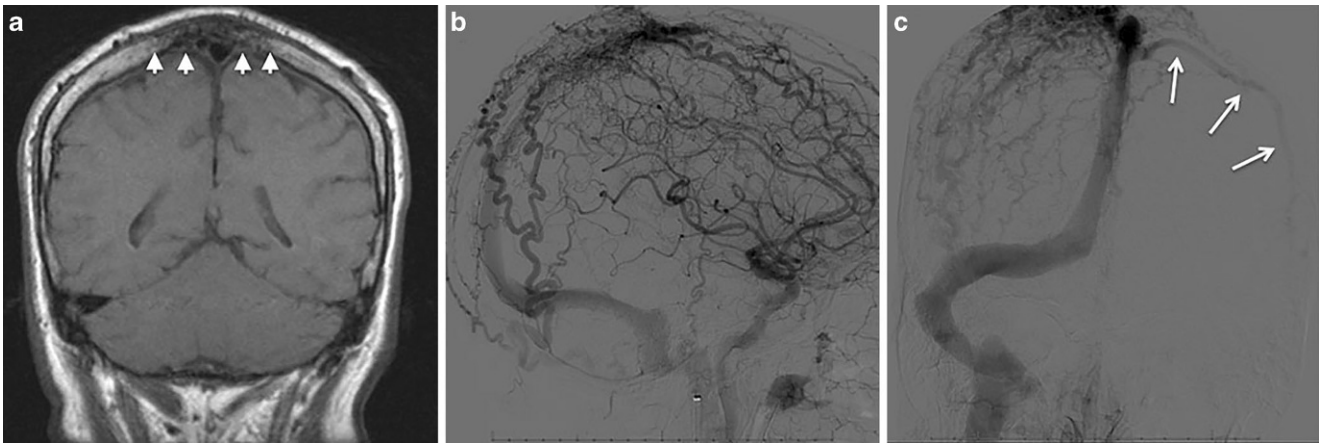


**Fig. 1** Imaging of the fistula at presentation. **a** Coronal noncontrasted T1 weighted image showing multiple flow voids in the region of the superior sagittal sinus (*arrowheads*). **b** Lateral angiogram in late arterial phase of the right common carotid artery showing an extensive network of vessels from the external branches draining into the region of the superior sagittal sinus with extensive shunting into the sinus consistent with dural arteriovenous fistula. **c** Anterioposterior projection of right external carotid angiogram in late arterial phase. Clear reflux into a large cortical vein on the left is noted (*arrows*)

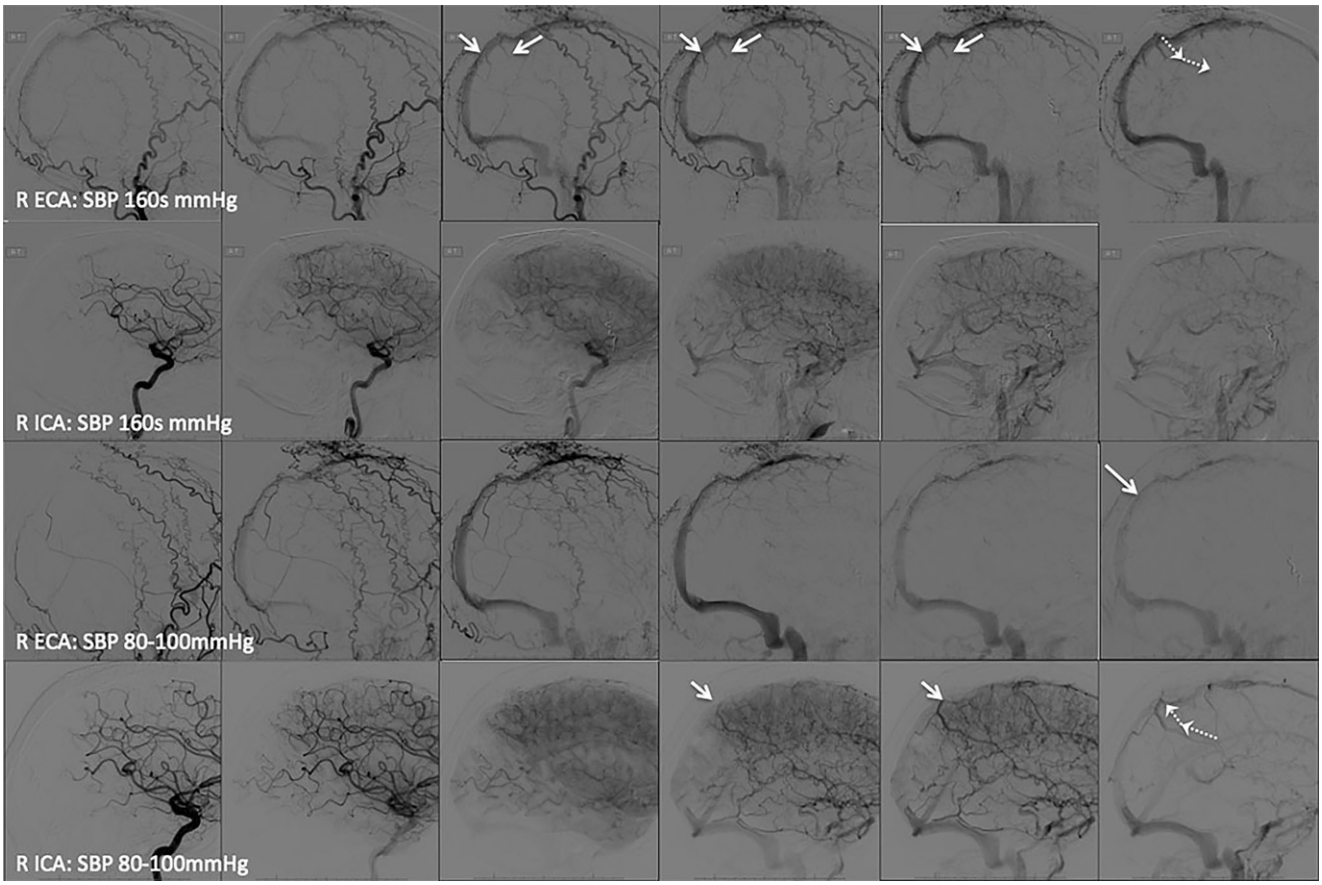


**Fig. 2** Reversal of reflux with changes in blood pressure. *Top row*: right external carotid artery (*ECA*) lateral angiogram from arterial (*left*) through venous phases (*right*). There is retrograde filling into several cortical veins from the posterior superior sagittal sinus (*arrows*). The *dotted arrow* points out the direction of filling of a prominent parietal vein. *Second row*: right internal carotid artery (*ICA*) lateral angiogram, again showing early arterial through venous phases. The veins noted in the above injection are not seen due to the reflux from the external carotid. *Third row*: right ECA lateral angiogram as above but now with lower systolic blood pressure (*SBP*) during anesthesia. The previously noted parietal vein reflux is not seen (*arrow*). *Bottom row*: right ICA lateral angiogram as above at lower blood pressure, now with normal venous drainage through the same parietal vein as previously seen on the external injection (*arrow*). The *dotted arrow* shows the direction of filling in the vein which is now reversed (normal)

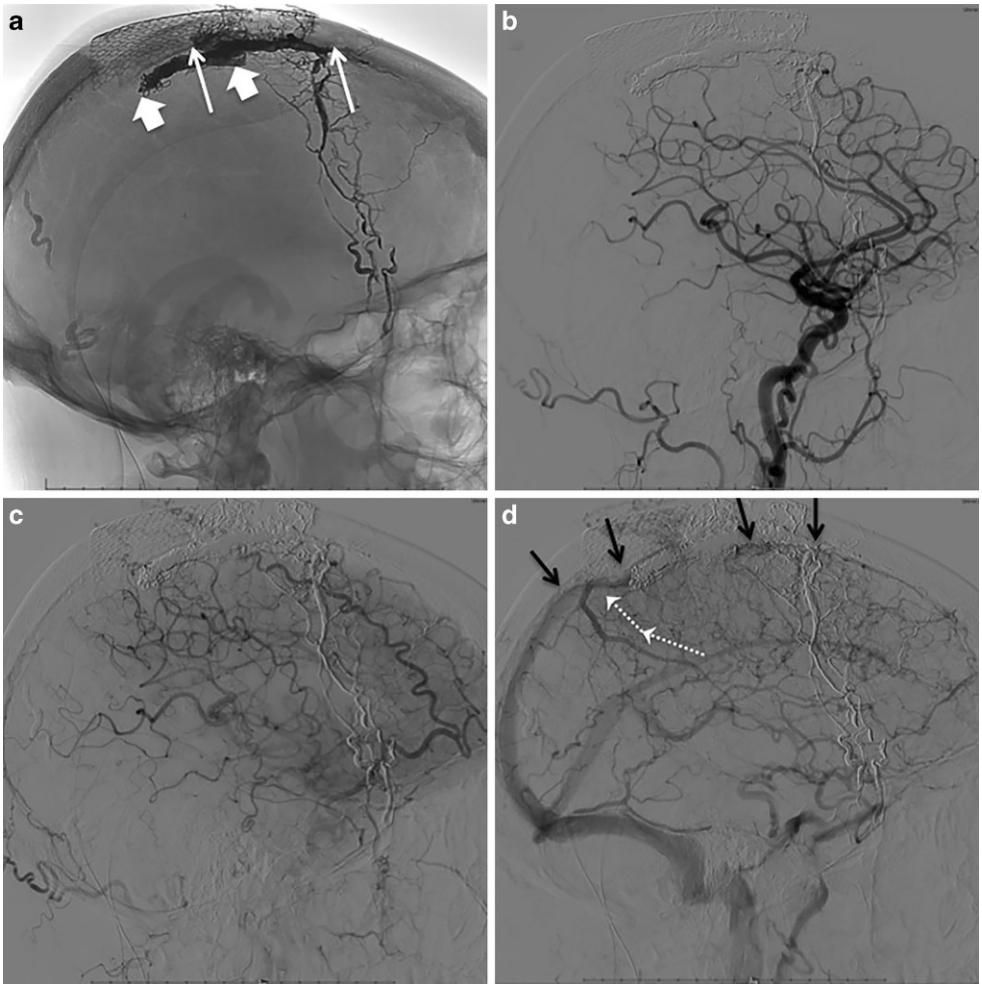


**Fig. 3** Final post-treatment result. **a** Onyx cast (in *black*) is seen filling the middle meningeal artery and the fistula. There was a long parallel channel to the fistula (*narrow arrows*) and only a relatively short segment of the superior sagittal sinus involved in the fistula (*wide arrows*). Note that there is some overlap. **b, c** Right common carotid injection, lateral projection demonstrating angiographic cure of the fistula with no early venous drainage. **d** Venous phase of the right common carotid injection, demonstrating preservation of the large parietal vein, with normal drainage pattern (*dotted arrows*). Note also that the superior sagittal sinus is draining normally anterior and posterior to the area of occlusion (*black arrows*)

Another potential mechanism could be related to distal blood flow differences in the internal and external carotid systems due to intracranial autoregulation.

A hemodynamic explanation is likely. When blood flows at a volumetric rate ($Q$) through a vessel with an approximately circular local cross-section of diameter ($D$), flow is described by the Reynolds number ($Re = 4Q/\pi\nu D$), where $\nu$ is the kinematic viscosity of the blood. At low values of $Re$, the fluid flow through a network of vessels is laminar. In cases where there are changes in the cross-section of the vessel and/or branching, a flow rate increase may lead to an abrupt change in the flow structure. One possible change is formation of recirculating vortices near an expansion/contraction, which would effectively obstruct the flow through the branch where the vortices form. This phenomenon has been observed in abdominal aortic aneurysms at $100 < Re < 300$ and some cases of fistulas [5, 6]. In this case, the Reynolds number is estimated within this range, while the geometry of fistulous vessels certainly manifest considerable complexity so it may be a reasonable explanation that at higher pressure, recirculation zones form and obstruct the normal drainage causing CVR, compared to laminar flow at lower pressure.

The optimal treatment is complete angiographic obliteration of the fistula whether by transarterial, transvenous, or combined routes; however, some authors have argued that selective disconnection of the refluxing veins may be an effective strategy, especially in particularly complex fistulas [1, 7–10]. This current case may introduce some uncertainty in this approach, since it is possible that refluxing veins are not a static problem. This may be particularly relevant at the proximal and distal borders of the fistula where there may be fluctuations between reflux and normal drainage that are dependent on overall blood pressure. This case supports the standard approach of direct occlusion of the fistula when possible compared to selective venous disconnection.

## Compliance with ethical guidelines

**Conflict of interest** A. Carlson, C.A. Meadows and P. Vorobieff declare that they have no competing interests.

**Ethical standards** This article does not contain any studies with human participants or animals performed by any of the authors. For images or other information within the manuscript which identify patients, consent was obtained from them and/or their legal guardians.

## References


1. Barnwell SL, Halbach VV, Higashida RT, Hieshima G, Wilson CB. Complex dural arteriovenous fistulas: result of combined endovascular and neurosurgical treatment in 16 patients. J Neurosurg. 1989;71:352–8.
2. van Dijk JM, terBrugge KG, Willinsky RA, Wallace MC. Clinical course of cranial dural arteriovenous fistulas with long-term persistent cortical venous reflux. Stroke. 2002;33:1233–6.
3. Zipfel GJ, Shah MN, Refai D, Dacey RG Jr, Derdeyn CP. Cranial dural arteriovenous fistulas: modification of angiographic classification scales based on new natural history data. Neurosurg Focus. 2009;26:E14.
4. Weber W, Kis B, Esser J, Berlit P, Kühne D. Endovascular treatment of a dural arteriovenous fistula of the transverse sinus by recanalisation, angioplasty and stent deployment. A case report and follow-up. Interv Neuroradiol. 2003;9:65–9.
5. Finol EA, Amon CH. Blood flow in abdominal aortic aneurysms: pulsatile flow hemodynamics. J Biomech Eng. 2001;123:474–84.
6. Browne LD, Walsh MT, Griffin P. Experimental and numerical analysis of the bulk flow parameters within an arteriovenous fistula. Cardiovasc Eng Technol. 2015;6:450–62.
7. Carlson AP, Taylor CL, Yonas H. Treatment of dural arteriovenous fistula using ethylene vinyl alcohol (onyx) arterial embolization as the primary modality: short-term results. J Neurosurg. 2007;107:1120–5.
8. Carlson AP, Alaraj A, Amin-Hanjani S, Charbel FT, Aletich V. Endovascular approach and technique for treatment of transverse-sigmoid dural arteriovenous fistula with cortical reflux: the importance of venous sinus sacrifice. J Neurointerv Surg. 2013;5:566–72.
9. da Costa LB, Terbrugge K, Farb R, Wallace MC. Surgical disconnection of cortical venous reflux as a treatment for Borden type II dural arteriovenous fistulae. Acta Neurochir (Wien). 2007;149:1103–8.
10. van Dijk JM, TerBrugge KG, Willinsky RA, Wallace MC. Selective disconnection of cortical venous reflux as treatment for cranial dural arteriovenous fistulas. J Neurosurg. 2004;101:31–5.