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M Forsting, K M Resch, R von Kummer and K Sartor

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Balloon Occlusion of a Giant Lower Basilar Aneurysm: Death Due to Thrombosis of the Aneurysm

Michael Forsting,[1] Klaus M. Resch,[2] Rüdiger von Kummer,[1] and Klaus Sartor[1]

Endovascular balloon embolization therapy for neurovascular lesions was first described by Serbinenko in 1974 [1]. With improved technology, including detachable balloons, minicatheters, permanently solidifying agents, and real-time digital subtraction angiography, it is now possible to treat intracranial arterial aneurysms from an endovascular approach in selected cases [2, 3]. In the case reported here, the patient had a giant aneurysm without a definable neck at the junction of both vertebral arteries; he was treated by bilateral endovascular occlusion of the vertebral arteries distal to the origin of the posterior inferior cerebellar arteries. Upon thrombosis of the aneurysm, the patient developed signs of severe brainstem ischemia and ultimately died. At autopsy, occlusion of perforating arteries that arose from the aneurysm sac was found to be the reason for the fatal outcome of endovascular therapy in this patient.

Case Report

This 29-year-old man had a 6-month history of slowly progressive sixth nerve palsy and recent onset of rapidly progressive hearing loss on the left. CT showed a nearly spherical enhancing mass lesion measuring 2.5 × 2.7 cm in the left cerebellopontine angle. Cerebral angiography revealed the presence of a giant basilar aneurysm at the junction of the vertebral arteries (VA). The aneurysm opacified on injection of either VA and had no definable neck (Fig. 1A). Superselective digital angiography (DSA) with a Tracker microcatheter (Target Therapeutics, San Jose, CA) placed into the aneurysm did not show any perforating arteries arising from the aneurysm sac (Fig. 1B).

A team of neurosurgeons and neuroradiologists determined that the aneurysm was unclippable. The hearing loss that had occurred during the preceding 3 weeks suggested growth of the aneurysm. It was thus decided to treat the aneurysm, if feasible, by bilateral VA occlusion via an endovascular approach. First, a test occlusion was done of both VAs distal to the origin of the posterior inferior cerebellar artery (PICA) while the patient was monitored continuously. Under local anesthesia, 9-French vascular sheaths (Terumo Corp., Tokyo) were placed in both femoral arteries. Following this, 5000 units of heparin was administered intravenously for systemic anticoagulation to prevent thrombus formation within the coaxial catheter system. After selective placement of an 8.0-French catheter into the proximal right VA, a 2.2-French microcatheter with a nondetachable silicone balloon (NDSB; Interventional Therapeutics Corp., San Francisco) at its tip was advanced into the intracranial portion of the right VA distal to the PICA origin. Upon inflation of this balloon, contrast injection into the *left* VA via a second selectively placed 5-French catheter (Terumo Corp., Tokyo) revealed unchanged filling of the aneurysm. A second nondetachable balloon was then introduced into the *left* VA, again distal to the PICA origin. Test occlusion of the right VA for 45 min and then of both VAs for another 15 min was tolerated well by the patient; neurophysiologic monitoring did not reveal any changes indicative of subclinical ischemia of the brainstem.

One week later both VAs were permanently occluded under general anesthesia, while the patient was monitored continuously. Again via a bifemoral approach, a detachable balloon (DSB; Interventional Therapeutics Corp., San Francisco) was placed into the right VA distal to the PICA origin and the test occlusion was repeated by inflating the silicone balloon with nonionic contrast material (Omnipaque, Schering AG, Germany). During the ensuing 30 min all monitored neurophysiologic parameters remained unchanged. The balloon was then deflated, reexpanded with HEMA (3-hydroxyethyl methylacrylate; Interventional Therapeutics Corp., San Francisco), and detached 60 min later. Up to this time no neurologic changes had occurred. A similar procedure was then performed on the left side. During this second test occlusion, a 5-French catheter was placed into the left internal carotid artery. Carotid angiography revealed good retrograde filling of the basilar artery. Because of this excellent collateral flow and unchanged evoked potentials, occlusion of the left VA was considered to be safe and the second balloon was detached 60 min after it was filled with HEMA.

A final angiogram showed both vertebral arteries occluded, with excellent retrograde filling of the basilar artery (Fig. 1C). No protamin sulfate was given to reverse the action of heparin. The patient left the angiography suite in a stable condition. Eight hours later he suddenly became tetraplegic and was only able to make vertical eye movements. MR imaging on the first and third days (Figs. 1D and 1E) after the intervention revealed thrombosis of the aneurysm as well as ischemic infarction of the pons and medulla oblongata. Five days later, the patient died of sudden cardiac arrest. An autopsy with detailed anatomic examination of the vertebrobasilar system revealed complete thrombosis of the aneurysm. Neither balloon had changed position, and the basilar artery was patent. The left anterior interior cerebellar artery (AICA) and three pontine branches arose from the aneurysm sac and were completely thrombosed (Figs. 1F and 1G).

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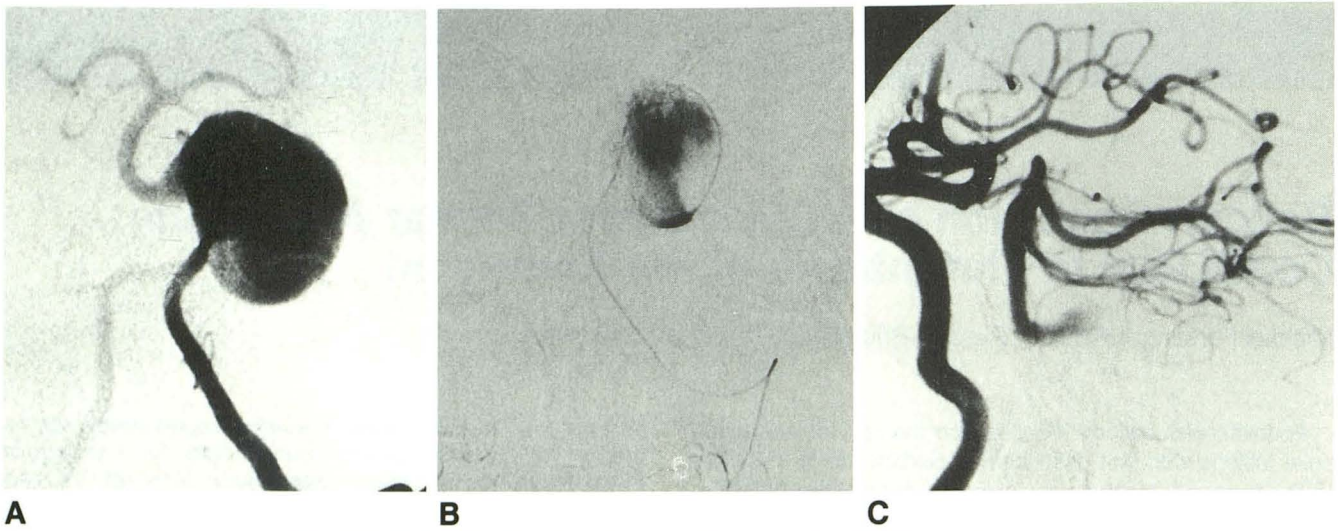
[1] Department of Neuroradiology, University of Heidelberg, Kopfklinikum, Im Neuenheimer Feld 400, D 6900 Heidelberg, Germany. Address reprint requests to M. Forsting.

[2] Department of Neurosurgery, University of Heidelberg, Heidelberg, Germany.



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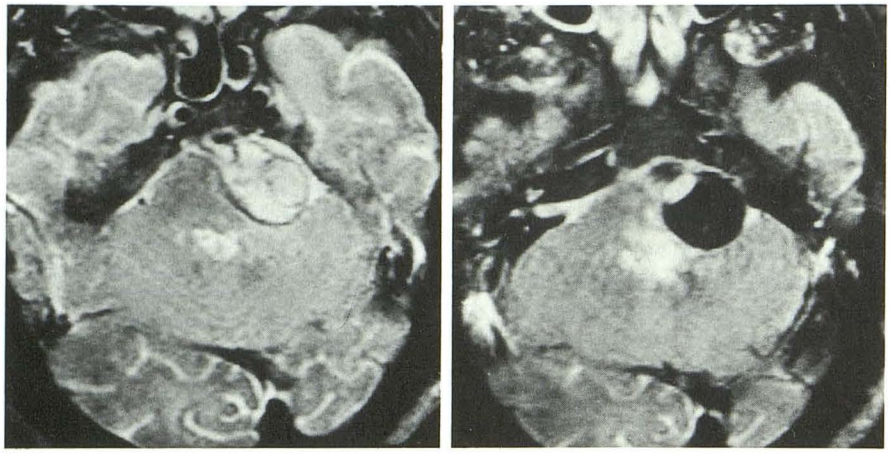

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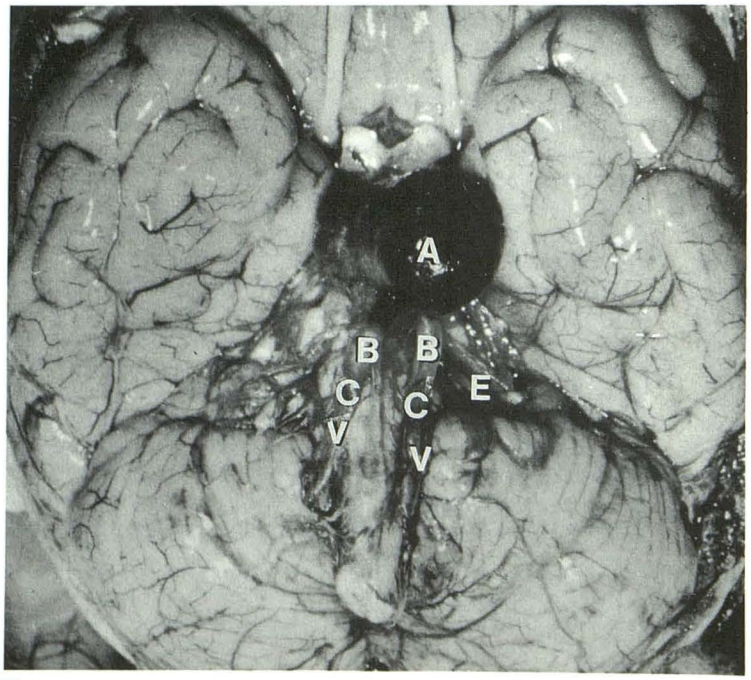

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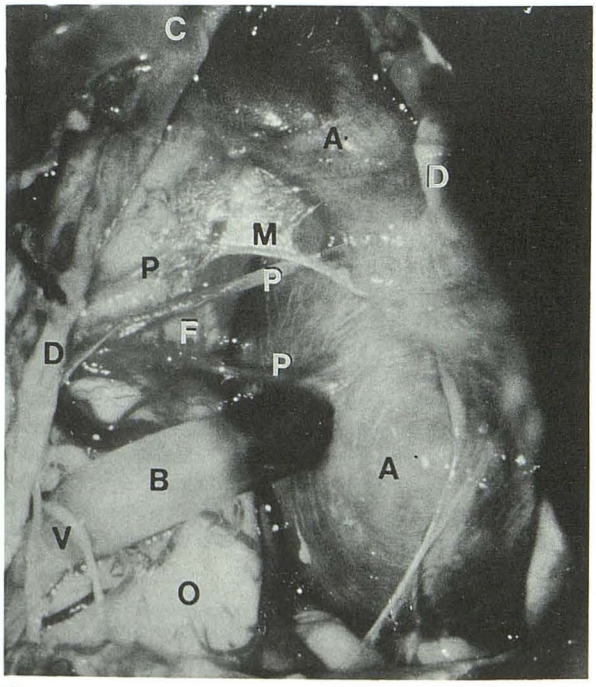

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Even in retrospect, we were unable to detect these arteries on the angiogram.

Discussion

The accepted method of treating cerebral aneurysms is surgical clipping of the aneurysm neck. The direct surgical approach to giant aneurysms of the vertebrobasilar system is one of the most difficult operations to perform. Dandy's experience of "the fastest death I have ever seen" [4] has additionally deterred many surgeons from treating giant vertebrobasilar aneurysms. Morbidity and mortality rates from 25% to over 50% have been reported in the literature [5, 6]. Endovascular techniques such as balloon occlusion may be a therapeutic alternative to surgery [3, 7–9]. Bilateral vertebral occlusion to initiate thrombosis within an aneurysm by eliminating the direct blood inflow and thereby changing the pressure and turbulence within the lumen was first recommended by Drake [5]. Pelz et al. [10] pointed out that before performing vertebral occlusion, it is necessary to show collateral blood flow to the brainstem via the posterior communicating arteries (PComA). These authors found strong trends indicating that patients with at least one large PComA do better than those with two small ones. Fox et al. [11] recommended a total test occlusion time of 15 min. In their study, eight of 67 patients did not tolerate the test occlusion, and signs of cerebral ischemia appeared between 30 sec and 7 min after the occlusion.

In our patient, progressive hearing loss was thought to indicate ongoing enlargement of the aneurysm, and the decision to apply endovascular techniques was made jointly by the neurosurgeons and the neuroradiologists. As recommended in the literature, we performed test occlusions demonstrating excellent retrograde filling of the basilar artery. In addition to clinical monitoring we were able to perform continuous neurophysiologic monitoring [12]. All parameters including somatosensory and acoustic evoked potentials remained unchanged during test occlusion. Despite this, the patient died from brainstem ischemia.

Delayed death due to thrombosis of a giant aneurysm has been described before [2, 5], but as far as we know, the cause of such an event has never been explained. It has been speculated that the delayed ischemic complications are due to peripheral embolization from the aneurysm [2]. In our patient, autopsy revealed perforating arteries arising from the aneurysm—branches that subsequently thrombosed, leading to fatal brainstem ischemia. This observation provides important insights: superselective DSA, even with the catheter tip inside the lesion, is not capable of demonstrating perforating arteries arising from the aneurysm sac; the microcatheter is probably not large enough to deliver an adequate volume of contrast material into a giant aneurysm. Even conventional cut-film magnification angiography never was totally reliable in showing the tiny perforating branches of the basilar artery. Thus, there is no radiologic technique capable of ruling out perforating arteries that arise from the aneurysm sac. Endovascular test occlusion or manual vascular compression to evaluate collateral blood flow (Allcock test) is only a diagnostic tool to determine *hemodynamic* tolerance of vessel occlusion.

After changing the pressure within the aneurysm by endovascular techniques it may be useful to heparinize these patients to keep important arteries that arise from the aneurysm sac temporarily patent. This way, collateral channels have more time to establish themselves. In giant aneurysms in which it is unclear whether or not perforating arteries arise from the aneurysm sac, the first aim of the endovascular procedure should not be to achieve rapid thrombosis of the aneurysm but to reduce the jet stream pressure on the wall. On the other hand, it may be dangerous to change the jet stream by doing a staged occlusion—first one VA and later on the second—thereby raising the pressure on one point of the wall. One should also keep the patient anticoagulated for a while, hoping to avoid delayed ischemic complications due to permanent endovascular occlusion. In our case, it probably would have been better if the VAs had been occluded below the PICA origin, allowing two good-sized vessels to maintain flow down the basilar artery. Thrombosis of the aneurysm would probably not have been as rapid nor as complete. Our rationale for occluding above the PICA origin was to protect the cerebellum from ischemia in case of extended thrombosis of the aneurysm.

In conclusion, the clinical outcome of patients with giant aneurysms of the type described is unpredictable, regardless of the mode of treatment. If an endovascular approach is used, even sophisticated monitoring techniques and tolerance tests cannot simulate all hemodynamic situations. A major problem is that such anatomic details as minute perforating arteries arising from the aneurysm itself are invisible on angiograms. A first-look operation may help optimize the endovascular procedure in such cases.

◀ **Fig. 1.—29-year-old man with 6-month history of slowly progressive sixth nerve palsy and recent onset of rapidly progressive hearing loss on left.**

***A*, Left vertebral angiogram, anteroposterior view. Giant aneurysm at vertebrobasilar junction fills from both vertebral arteries without definable neck.**

***B*, Digital subtraction "aneurymography," anteroposterior view, with tip of microcatheter placed into aneurysm, shows absence of vessels arising from aneurysm sac.**

***C*, Left carotid angiogram, lateral view, reveals large posterior communicating artery with excellent retrograde filling of basilar artery, as well as rostral portion of aneurysm after bilateral vertebral artery occlusion.**

***D* and *E*, MR images (2400/90/2) obtained on first (*D*) and third (*E*) day after intervention. High signal within aneurysm in *D* suggests presence of slowly flowing blood. Two days later (*E*), signal has changed to the typical hypointense signal of acute thrombosis of the aneurysm sac. At this time, large ischemic infarction of brainstem is visible.**

***F*, Overview after autopsy: left AICA (E) arises from rostral portion of aneurysm sac (A). Both balloons (B) are in place distal to the PICA origin (C). (V = vertebral artery)**

G, Aneurysm (A) is exposed in situ transorally. Balloon (B) in right vertebral artery (V) is clearly visible, while balloon in left vertebral artery is not seen. Three perforator arteries (P) arising from aneurysm sac are visible. (C = clivus, D = dura, F = pons, M = arachnoidal membrane, O = medulla)

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