# Basilar bifurcation aneurysm surgery

Basilar bifurcation aneurysm were considered inoperable until Charles George Drake reported 4 cases in 1961 [1] , with larger series reported later [2].

Anatomical factors such as size and projection of the aneurysm, distance between the aneurysm neck and the dorsum sellae, and location of the basilar bifurcation contribute to surgical complexity [3].

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Direct operative management of basilar bifurcation aneurysms is one of the most challenging procedures in the realm of vascular neurosurgery, as these lesions are deeply situated in the interpeduncular region and maintain an intimate relationship with important anatomical structures. Direct clipping of aneurysms generally represents the gold standard of surgical treatment, as it allows exclusion of the aneurysmal sac from the circulation, evacuation of aneurysmal contents for decompression, and preservation of efferent flow.

## Timing

Initial experience tended to favor allowing basilar tip aneurysms to “cool-down” for ≈ 10–14 days after SAH before attempting surgery to permit cerebral edema to subside. More recently, early surgery for these aneurysms has been advocated as for anterior circulation aneurysms [4].

However, some surgeons still recommend waiting ≈ 1 week [5] , and most would agree that if there are obvious technical difficulties because of aneurysm size, configuration or location of the aneurysm, that early surgery may not be appropriate. Also, if during the craniotomy it becomes apparent that cerebral edema is impairing the exposure, the operation should be aborted and attempted again at a later date.

## Approaches

see Basilar bifurcation aneurysm approaches.

## Large broad-based basilar apex aneurysm

Large, broad-based basilar apex aneurysms involving multiple arterial origins are complex lesions commonly not amenable to direct clipping or endovascular management.

BA proximal (Hunterian) occlusion with extracranial-to-intracranial bypass is a supported strategy if one or both posterior communicating arteries are small. Hunterian ligation risks sudden aneurysm thrombosis and thromboembolism in the perforator rich BA apex. There currently exists no guidelines for antiplatelet and anticoagulant therapy following Hunterian ligation for complex BA apex aneurysm treatment.

Ravina et al presented in 2018 a literature review and an illustrative case of an 18-year-old man who presented with progressive headaches and was found to have a large, unruptured BA apex aneurysm involving the origins of bilateral superior cerebellar and posterior cerebral arteries. Given the small posterior communicating arteries and complexity of the aneurysm, proximal BA occlusion with unilateral superficial temporal artery-to-superior cerebellar artery bypass was recommended. Despite antiplatelet treatment with acetylsalicylic acid pre- and postoperatively, the patient developed acute ischemia of the brainstem and cerebellum as well as an embolic left temporal lobe infarct. The patient received dual antiplatelet therapy starting postoperative day 6 following which he experienced no new infarcts and made a significant neurologic recovery. The current evidence suggests that proximal BA occlusion in complex BA apex aneurysm cases is thrombogenic and can be especially dangerous if thrombosis occurs suddenly in aneurysms without pre-existing intraluminal thrombus. Dual antiplatelet therapy during the first postoperative week presents a possible strategy for reducing the risk of ischemia due to sudden aneurysm thrombosis [6].

# References

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