

Posterior Cerebral Artery Aneurysms
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Dr. med. Felix Göhre

University of Helsinki 2016

VERLAG JANOS STEKOVICS

for my parents, Gerd and Reinhilde Göhre

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List of Original Publications

I

Goehre F, Jahromi BR, Hernesniemi JA, Elsharkawy A, Kivisaari R, von und zu Fraunberg M, Jääskeläinen JE, Lehto H, Lehecka M: Characteristics of Posterior Cerebral Artery Aneurysms: An Angiographic Analysis of 93 Aneurysms in 81 Patients. *Neurosurgery*. 75(2):134-143, 2014.

II

Goehre F, Jahromi BR, Lehecka M, Lehto H, Kivisaari R, Andrade-Barazarte H, Ibrahim T, Párraga RG, Ludtka C, Meisel HJ, Koivisto T, von und zu Fraunberg M, Niemelä M, Jääskeläinen JE, Hernesniemi JA: Posterior Cerebral Artery Aneurysms – Treatment and Outcome Analyses of 121 Patients. (*World Neurosurgery* – in press)

III

Goehre F, Lehecka M, Jahromi BR, Lehto H, Kivisaari R, Hijazy F, Nayeb L, Sugimoto T, Morishige M, Elsharkawy A, von und zu Fraunberg M, Jääskeläinen JE, Hernesniemi JA: Subtemporal Approach to Posterior Cerebral Artery Aneurysms. *World Neurosurgery*. 83(5):842-851, 2015.

IV

Goehre F, Jahromi BR, Elsharkawy A, Lehto H, Shekhtman O, Andrade-Barazarte H, Munoz F, Hijazy F, Mekhkamov M, Hernesniemi JA: Lateral supraorbital approach to ipsilateral PCA-P1 and ICA-PCoA aneurysms. *Surgical Neurology International*. 6:91, 2015

Abbreviations

BA	Basilar artery
CT	Computed tomography
CTA	Computed tomography angiography
DSA	Digital subtraction angiography
ECA	External carotid artery
ELANA	Excimer laser-assisted nonocclusive anastomosis
H&H	Hunt and Hess
ICH	Intracerebral hematoma
IVH	Intraventricular hematoma
LSO	Lateral supraorbital approach
MRI	Magnetic resonance imaging
MRA	Magnetic resonance angiography
MTT	Mean transit time
PCoA	Posterior communicating artery
P1	P1 segment of posterior cerebral artery
P1/2	P1/P2 junction of posterior cerebral artery
P2	P2 segment of posterior cerebral artery
P2A	Anterior P2 segment of posterior cerebral artery
P2P	Posterior P2 segment of posterior cerebral artery
P3	P3 segment of posterior cerebral artery
P4	P4 segment of posterior cerebral artery
PCA	Posterior cerebral artery
PdistAs	Aneurysms of P3 and P4 segment of posterior cerebral artery
SAH	Subarachnoid hemorrhage
SCA	Superior cerebellar artery
SCTTA	Supracerebellar transtentorial approach
SDH	Subdural hematoma
SV	Saphenous vein

Abstract

Objective:

Aneurysms of the posterior cerebral artery are rare vascular lesions. The overall incidence is less than 1%, representing around 7% of posterior circulation aneurysms. Due to this low incidence, most of the institutional series on PCA aneurysms are small and contain less than 25 patients. Only one other series comparable in scope to ours has been previously published. The presented study analyzes and describes the characteristic features of PCA aneurysms as well as investigates the relevant treatment strategies and their outcomes. A particular focus is in the description and analysis of PCA aneurysms treated from a subtemporal approach and the presentation of an associated aneurysm treatment from a lateral supraorbital approach.

Patients and Methods:

We reviewed 121 patients diagnosed with 135 PCA aneurysms, all of whom were treated between 1980 and 2012 at two Finnish neurosurgical units (Department of Neurosurgery at the University of Eastern Finland, Kuopio and Department of Neurosurgery at the University of Helsinki). Additionally, twelve historical (pre-1980) cases were presented. Detailed analyses of cerebral angiographies were conducted for 93 PCA aneurysms in 81 patients. A further subgroup analysis of 34 patients diagnosed with 37 PCA aneurysms treated via subtemporal approach was also performed.

Results:

Of the 121 patients with 135 PCA aneurysms, 52 (39%) aneurysms were ruptured and 83 (61%) unruptured. The following distribution along the PCA segments was observed: P1 segment (n=53), P1/2 junction (n=39), P2 segment (n=28), and P3 segment (n=15); no P4 segment aneurysms were found. Saccular aneurysms were more common than fusiform PCA aneurysms (76% vs. 24%). The detailed angiographic analysis showed that the me-

dian aneurysm size was 7 mm for ruptured PCA aneurysms and 4 mm for unruptured aneurysms. Saccular aneurysms (n=69, 74%) had a characteristic dome projection for each location: P1 segment, upward (67%); P1/P2 junction, anterior/upward (80%); P2 segment, lateral (67%); and P3 segment, posterior (50%). The following treatment results at 1-year follow-up were achieved for patients with: unruptured PCA aneurysms (n=19; 12 good outcomes, 63%; 6 moderate, 31%; 1 poor, 5%), ruptured PCA aneurysms (n=27; 10 good, 37%; 9 moderate, 33%; 8 poor, 30%), and patients with complex neurovascular pathologies and PCA aneurysms (n=96; 42 good, 43%; 40 moderate, 42%; 14 poor, 15%). Analyzing the subtemporal approach we found that most complications were not related to the subtemporal approach itself but to the specific nature of the PCA aneurysms treated and the chosen strategy. The most common (12 out of 34; 35%) serious complication in this series was an ipsilateral PCA infarction after parent vessel occlusion.

Conclusion:

PCA aneurysms are infrequent vascular lesions that are often associated with other vascular pathologies. Most ruptured PCA aneurysms are smaller than 10 mm and distally located. The saccular PCA aneurysms have a typical dome orientation at each PCA segment. Microsurgery and endovascular treatment are effective options for the occlusion of PCA aneurysms. As a result, individual treatment strategies are required. Despite commonly adequate vessel collateralization of the distal PCA territory, preservation or reconstruction of the parent vessel is crucial for favorable treatment outcomes. The subtemporal approach is favorable for the treatment of PCA aneurysms in proximity to the tentorium. Frontolateral approaches allow the treatment of proximal PCA aneurysms and ipsilateral anterior circulation aneurysms inside the Circle of Willis.

1. Introduction

Subarachnoid hemorrhage caused by the rupture of an intracranial aneurysm is a life-threatening event and results in fatality in approximately 40% of all cases [Nieuwkamp 2009, Steiner 2013]. Meta-analyses show that around 2% of the population carries an intracranial aneurysm [Nieuwkamp 2009, Steiner 2013]. Different risk factors for aneurysm rupture have been identified such as female gender, smoking, older age, hypertension, aneurysmal size and location, and genetic predisposition [Feigin 2005, Juvela 1993, Juvela 2000, Juvela 2003, Linn 1996, Raaymakers 1998, Rinkel 1998].

Aneurysms of the posterior cerebral artery (PCA) are rare and have an overall incidence of less than 1%, representing roughly 7% of all the aneurysms of the posterior circulation [Drake 1996, Honda 2004, Locksley 1966]. Different, closely related classifications of the PCA segments and affecting aneurysms have been published [Saeki 1977, Seoane 1997]. The most frequently used classification is that of Zeal and Rhoton [Zeal 1978], who divided the artery into four main segments (P1 to P4). We consider aneurysms of the P1 segment and the P1/2 junction as proximal PCA aneurysms belonging to the Circle of Willis and aneurysms of the P2, P3, and P4 segment as distal PCA aneurysms.

The microsurgical treatment of PCA aneurysms is technical and demanding. During the last three decades, endovascular techniques have evolved rapidly [Ciceri 2001, Hallacq 2002, Molyneux 2002, Wiebers 2003, van Rooij 2006] and PCA aneurysms that are located close to the brainstem are treated using endovascular techniques at many institutions [Lavine 2007, Cicerini 2001, Halaq 2002, Rooij 2006]. Due to their low incidence, most of the institutional series on PCA aneurysms are small and analyze fewer than 25 cases. The largest collection of PCA aneurysms was reported by Drake et al. (1996) and focuses on the surgical treatment and outcome of 125 PCA aneurysms [Drake 1996].

The present study is based on the retrospective analysis of 121 patients with 135 PCA aneurysms, along with 12 historical cases, treated at the Department of Neurosurgery at the University of Helsinki between 1954 and 1979. The aim of this study is to provide new information about the special anatomical features of PCA aneurysms and to analyze the applied treatment strategies for the further development of PCA aneurysm management.

2. Literature Review

2.1 Intracranial Aneurysms

2.1.1 Incidence and Prevalence

Intracranial aneurysms are present in 2–4% of the general population [Jellinger 1979, Hausepian 1958, Vernooij 2007, Vlack 2011]. Posterior circulation aneurysms are infrequent with an incidence of 7–10% and PCA aneurysms are particularly rare with an incidence of 0.5–2.3% [Ciceri 2001, Drake 1979, Drake 1996, Hallacq 2002, Locksley 1966, Orita 1994, Seoane 1997, Sakata 1993, Terasaka 2000, Yasargil 1984, Zeal 1978]. Multiple aneurysms were found in 18–44% of all intracranial aneurysm carriers [Inagawa 1990/1990, Wilson 1989]. The general incidence of subarachnoid hemorrhage is 6–10/100,000 per year [Anderson 2000, Linn 1996]. Older studies show that in Finland and Japan the incidence rate is much higher at 16–20/100,000 per year [Fogelholm 1993, Inagawa 2001, Ohkuma 2002, Sivenius 2004, Stegmayr 2004]. In recent years the incidence of SAH has decreased dramatically and, at least in Southern Finland, appears to be the same as the frequency reported for western countries. The decline may be due to dietary habits and a dramatic decrease in smoking.

2.1.2 Diagnosis and Imaging of Intracranial Aneurysms

Symptoms

Key symptoms of a ruptured aneurysm are a sudden and severe headache, loss of consciousness, confusion, seizures, nausea, vomiting, cranial nerve disturbance, double vision, light sensitivity and neck stiffness.

Unruptured aneurysms are usually asymptomatic; however, symptomatic unruptured aneurysms can present the following symptoms: pain above and behind the eye, a dilated pupil, a

drooping eyelid, visual disturbance, double vision, numbness, weakness or paralysis of one side of the face.

Imaging

Nowadays SAH is usually diagnosed by computed tomography (CT) during the first 6 hours after the incident or lumbar puncture [Backes 2012, Perry 2011]. Computed tomography and magnetic resonance imaging can indicate major vascular lesions on predilection sites. For detailed analysis of the cause of bleeding, further angiographies by computed tomography angiography (CTA) (Fig. 1), magnetic resonance angiography (MRA) or digital subtraction angiography (DSA) is required [Chen 2009, Papke 2007, Villablanca 2005, Wang 2013].

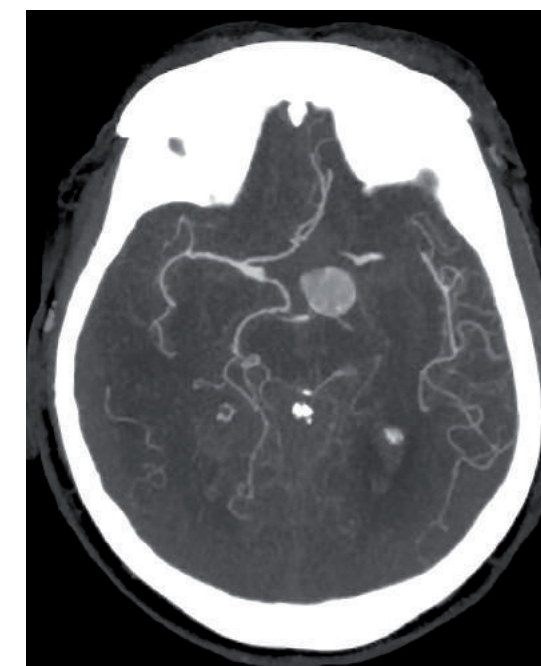


Figure 1. Axial CTA of a ruptured left saccular PCA-P1/2 junction aneurysm.

2.1.3 Risk Factors

The annual rupture rate for previously diagnosed unruptured aneurysms is around 1% [Juvola 2013]. There are modifiable risk factors such as smoking, drug abuse and heavy alcohol consumption [Juvola 2001, Korja 2014]. Non-modifiable risk factors include female gender, older age, arteriosclerosis, head injury and infectious disease [Feigin 2005, Juvola 2001, van Gijn 2007]. Treatable risk factors include hypertension and postmenopausal decreased estrogen levels [Feigin 2005, Juvola 2001, van Gijn 2007].

Congenital factors are various inherited connective tissue disorders, polycystic kidney disease, coarctation of the aorta and cerebral arteriovenous malformations [Chapman 1992, Connolly 2003, Debette 2014, Meisel 2000]. Risk factors for aneurysm rupture are current smoking, aneurysm size above 7 mm, hypertension, alcohol abuse and aneurysm growth in follow up examinations [Bonita 1986, Knekt 1991, Korja 2014, Lindekleiv 2012, Taylor 1995].

2.1.4 Morphology

Aneurysms are basically divided into saccular and fusiform aneurysms. Most intracranial aneurysms are saccular, having a shape reminiscent of a sac, pouch or berry. They mostly originate in close relation to arterial bifurcations or major perforating branches from the parent vessel. Fusiform aneurysms are spindle shaped dilations of the artery wall; dilations greater than 1.5 mm from the normal vessel lumen are considered to be aneurysms [Fleming 2004]. Fusiform aneurysms are uncommon compared to saccular aneurysms [Anson 1996, Dashti 2007]. Fusiform aneurysms can be further subclassified into dissecting (acute and chronic) and atherosclerotic aneurysms based on pathological mechanisms. The vessel course and the lengths of the affected segment allow for an aneurysm description of either dolichoectatic or serpentine. Dolichoectatic aneurysms present a combination of dilatation, elongation and tortuosity of the affected segment; serpentine aneurysms contain

thrombotic material. Large (15–25 mm) and giant (>25 mm) aneurysms are very rare, representing only around 5% of all aneurysms [Lonjon 2015].

2.1.5 Histology and Aneurysm Wall Degeneration

The wall of an intracranial vessel consist of three layers: an inner layer of endothelial and smooth muscle cells (tunica interna, intima), a middle layer with smooth muscle cells (tunica media, media) and an outer layer of connective tissue (tunica externa, adventitia). The internal elastic lamina, located between the tunica interna and tunica media, further stabilizes the artery wall.

Frösen et al. (2004) have analysed the wall structures of intracranial aneurysms and described four wall types of intracranial aneurysms: Type A, an endothelialized wall with linearly organized smooth muscle cells; Type B, a thickened wall with disorganized smooth muscle cells; Type C, a hypocellular wall; and Type D, a thin thrombosis-lined hypocellular wall. Wall types C and D are predominant in ruptured saccular aneurysms. In general, the walls of ruptured saccular aneurysms feature apoptosis, de-endothelialization, luminal thrombosis, smooth muscle cell proliferation and T-cell and macrophage infiltration [Frösen 2004].

Endothelial defects, blood clotting and intraluminal thrombus formation are associated with aneurysm wall degeneration and subsequent aneurysm rupture [Frösen 2004, Kataoka 1999]. Endothelium or tunica media injuries or defects lead to consequential pathological changes in the self-repair of these layers [Newby 2000]. In response to the respective loads, self-remodeling results in partial wall stabilization [Frösen 2012, Sawabe 2010]. However, these are not always effective [Bäck 2010, Bruno 1998, Frösen 2004, Kataoka 1999, Mignatti 1993]. Apoptosis processes are substantially involved in the degeneration of the wall [Chou 2008, Guo 2007, Miller 2011, Sakaki 1997]. Inflammatory cellular responses play an important role in aneurysm wall degeneration [Chyatte 1999, Crompton 1966, Frösen 2004, Kataoka 1999]. Oxidized LDL accumulates in the aneurysm wall of most rup-

tured saccular aneurysms. Saccular aneurysm wall remodelling is associated with changes in growth factor receptor expression. Protein kinases and complementary activation by multiple activators are involved in the degeneration of the aneurysm wall and the consecutive growth and rupture of intracranial aneurysms [Laaksamo 2008, Tulamo 2006/2010/2010].

In fusiform acute dissecting aneurysms, an internal elastic membrane defect is what leads to an intramural haematoma. In the chronic phase of these aneurysms, there is an intramural thrombus formation and neovascularization, resulting in new haemorrhages within the affected vessel wall [Nakatomi 2000]. The aneurysm wall is a heterogeneous and dynamic system with a continuous remodelling process.

2.1.6 Genetics

Intracranial aneurysms are associated with heritable connective tissue diseases like autosomal dominant polycystic kidney disease, Ehlers-Danlos syndrome type IV and neurofibromatosis type I [Chapman 1992, Debette 2014]. The risk of having an intracranial aneurysm is higher in families having two or more members already affected by an intracranial aneurysm. This is also associated with an increased risk of SAH [Ronkainen 1993/1999]. The "Nordic Twin Study" shows that genetic factors play only a minimal role in the etiology of aneurysmal SAH [Korja 2010]. The association of gene loci 8q and 9q with intracranial aneurysms has been previously described [Helgadottir 2008, Yasuno 2008, Bilguvar 2008]. However, these gene loci are also associated with other cardiovascular pathologies in general. Based on family history, association with heritable connective tissue diseases and personal risk factors, an active screening with non-invasive vascular imaging (MRA) is justified for this population.

2.2 Aneurysmal Subarachnoid Hemorrhage

2.2.1 Incidence of Aneurysmal Subarachnoid Hemorrhage

Subarachnoid hemorrhage causes 5–10% of all strokes [Johnston 1998, Kosierkiewicz 1994]. The incidence of subarachnoid hemorrhage is around 6–10 persons per 100,000 annually [Anderson 2000, Linn 1996]. Subarachnoid hemorrhage can be caused by aneurysm rupture (80% of cases), AVM rupture (5%) or an unknown cause (15%) [Ronkainen 1992, Schievink 1997]. The general mortality associated with subarachnoid hemorrhage is 30–40%, followed by a high percentage of patients (around 20%) who subsequently require assistance for daily living [Nieuwkamp 2009, Steiner 2013]. The occurrence rate in younger, working age individuals is high and results in a significant loss of productive life years [Johnston 1998, Rivero-Arias 2009].

2.2.2 Fatality of Aneurysmal Subarachnoid Hemorrhage

Mortality

Unselected historical analyzes provide crucial information about the natural history of aneurysmal SAH fatalities [Pakarinen 1967]. The first day mortality rate is very high at 32%, and rises steadily over time to 46% within the first week, 56% within the first month and 60% after 6 months [Pakarinen 1967].

Even today, the mortality rate is very high at around 40% despite modern diagnostic and treatment methods [Nieuwkamp 2009, Steiner 2013]. One reason is fatality resulting from the initial bleeding [Fogelholm 1993]. About 12–15% of patients in western countries are unable to reach medical treatment before their resulting death [Huang 2002, Pobereskin 2001].

Rebleeding

A second hemorrhage occurs in 4–12% of patients during the first 24 hours [Inagawa 1987, Juvola 1989, Kassell 1983, Pakarinen 1967, Starke 2011].

The first 6 hours are especially critical [Starke 2011]. During the next four weeks the daily rebleeding risk remains at 1–2%. The rebleeding risk during the first year decreases to 3% annually.

Vasospasm

Cerebral vasospasm is defined as delayed reactive contraction of the affected vessels, which reduces the distal blood flow. The incidence is high at 50–75% and around 50% of these patients are clinically symptomatic [Inagawa 1990, Kassell 1985]. Around 15% of patients with SAH are affected by permanent neurologic deficits from cerebral vasospasm [Haley 1992, Longstreth 1993] and a fatal outcome is also possible. The critical time period is between the 4th and 12th day after the initial bleeding [Weir 1978]. Vasospasm can be detected by transcranial Doppler ultrasound (TCD), CTA, DSA and CT perfusion imaging. In particular, the initial amount of SAH in the cisterns allows prediction of vasospasm [Fisher 1980]. An active treatment can reduce the rate of ischemic complications related to cerebral vasospasm. Nimodipine has been shown to improve neurologic outcome and decrease the incidence of cerebral vasospasm [Dorhout Mees 2007]. Classically, the "triple H therapy" of hypervolemia, induced hypertension and slight hemodilution is used as the treatment basis after occlusion of the ruptured aneurysm [Lee 2006].

Hydrocephalus

Treatment requiring hydrocephalus occurs in around 10–30% of patients following SAH [de Oliveira 2007, Erixon 2014]. The subarachnoid blood clot can disturb CSF circulation and resorption. This can lead to an acute hydrocephalus, which is a leading cause of intracranial hypertension in the early phase after aneurysm rupture. The acute hydrocephalus can be detected in the primary CT scans and should be treated immediately.

Medical Complications

ECG abnormalities following SAH are common and include: prolonged Q-T interval, inverted t-waves and increased U waves. Cardial dysrhythmia may

cause some of the sudden deaths following SAH. Acute SAH can cause a neurogenic pulmonary edema with an increased risk for pneumonia [Weir 1978]. Hyponatremia and natriuresis are other common problems following SAH. Hyponatremia should be corrected by fluid replacement, not by fluid restriction, due to decreased plasma volume.

2.3 Microsurgical Anatomy and PCA Aneurysms

2.3.1 Microsurgical PCA Anatomy

The posterior cerebral artery is the terminal branch of the basilar artery and supplies the posterior aspects of the hippocampus, thalamus, midbrain, choroid plexus and a part of the lateral cerebral surface [Adamczyk 2014, Erdem 1993, Kocaeli 2013, Margolis 1972, Milisavljevic 1986, Percheron 1973, Zeal 1978]. During embryological development, the posterior cerebral artery arises from the internal carotid artery (ICA) [Padget 1948, Zeal 1978]. Several different closely related classifications of the PCA have been proposed [Saeki 1977, Seoane 1997]. The most frequently used is that of Zeal and Rhoton [Zeal 1978] who divided the artery into four main segments (Fig. 2).

The P1 segment is localized between the origin of the PCA at the bifurcation of the basilar artery and the posterior communication artery (PCoA). The cerebral peduncle forms the medial border of the P1 segment (Fig. 3, 4); the oculomotor nerve runs laterally and inferiorly to the P1. The fetal configuration consists of a dominant PCoA and has been observed in 16–22% of brains in cadaveric studies [Padget 1948, Párraga 2011, Saeki 1977, Zeal 1978]. The average length of the P1 is 7.7 mm (range 4–20 mm) according to Párraga et al. [Párraga 2011]. Thalamoperforating branches originate mostly from the medial third of the P1 segment and also posteriorly from the lateral third, with an average of 3 branches (range 1–10 branches) [Párraga 2011]. Additionally, the short and long circumflex arteries usually arise from the P1 segment.

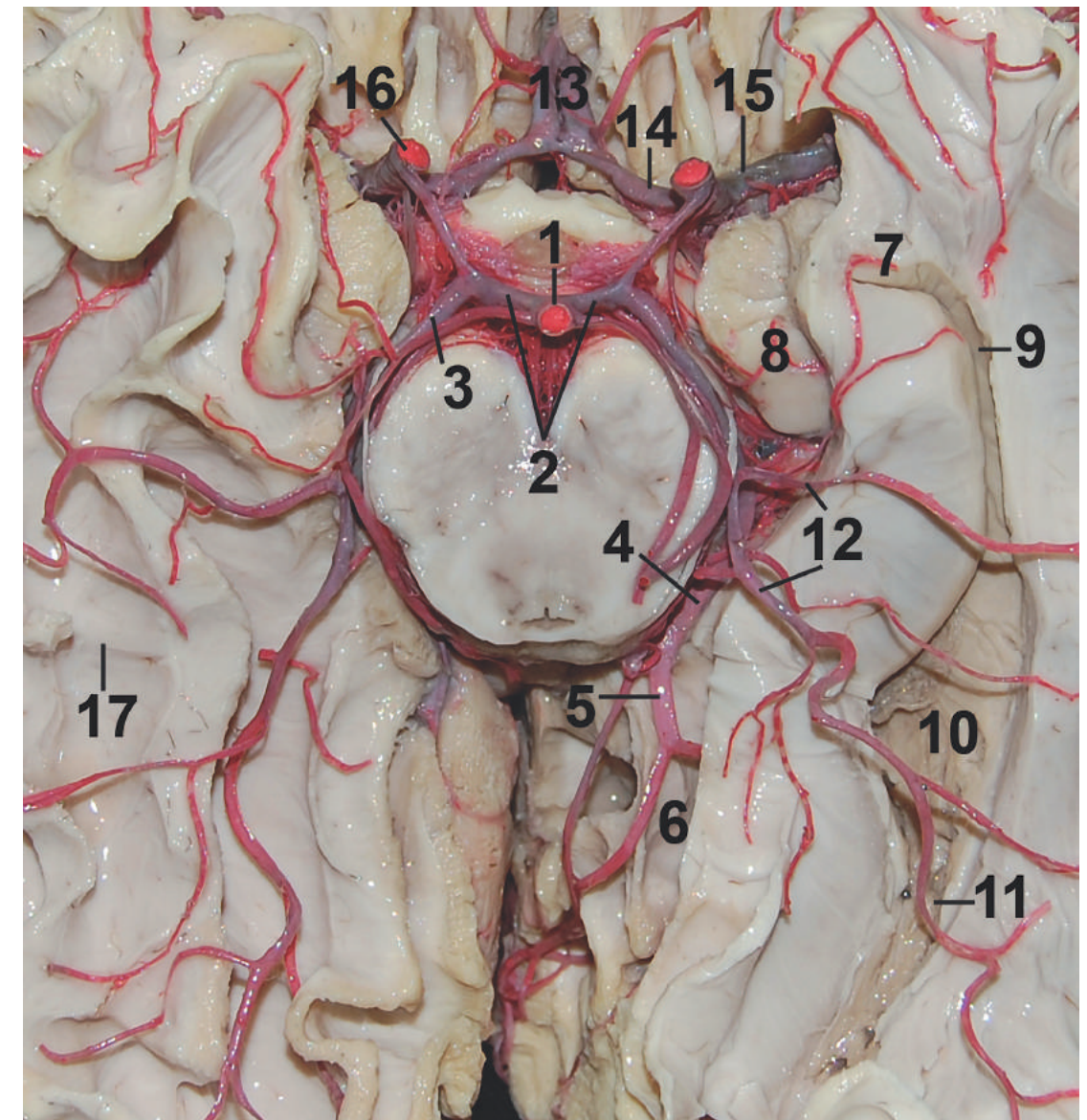


Figure 2. Dissection of the basal surface of the brain in Klingler's technique under preservation of the PCA. The brainstem is sectioned at the level of the pontomesencephalic junction. The basal surface of the left temporal lobe is dissected to disclose the inferior longitudinal fasciculus, hippocampus, calcar avis, atrium, and temporal and occipital horns of the lateral ventricles. The arcuate fibers are seen where the gray matter was removed. (1, BA trunk; 2, PCA-P1 segment; 3, PCA-P2A segment; 4, PCA-P2P segment; 5, PCA-P3 segment; 6, Calcarine fissure with calcarine artery, PCA-P4 segment; 7, Amygdala; 8, Hippocampus head; 9, Temporal horn; 10, Ventricular atrium; 11, Occipital horn; 12, Temporal arteries; 13, ACA-A2 segment; 14, ACA-A1 segment; 15, MCA-M1 segment; 16, ICA; 17, Arcuate fibers)

[provided by Dr. Richard Gonzalo Párraga with personal approval, Reprinted from Publication: Párraga RG, Ribas GC, Andrade SE, de Oliveira E: Microsurgical anatomy of the posterior cerebral artery in three-dimensional images. *World Neurosurg.* 2011;75:233–257, with permission from Elsevier].

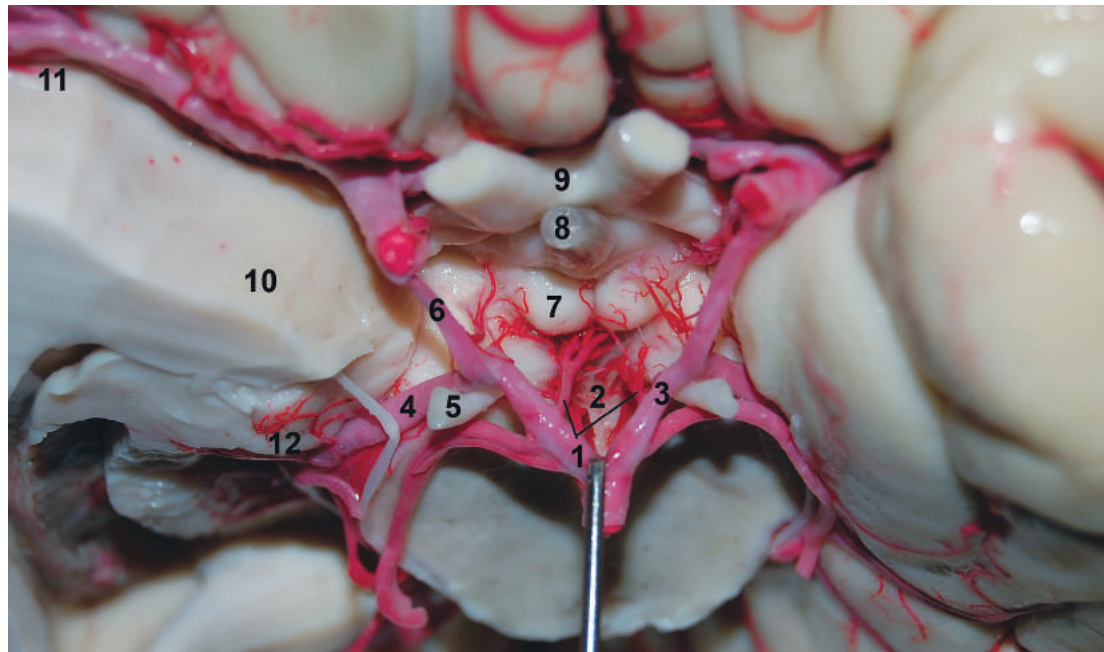


Figure 3. Anteroinferior view of the brain with dissection of the temporal lobe exposing the amygdala. The basilar bifurcation is retracted with a dissector to showcase the thalamoperforating arteries originating along the superior surface of the right P1 segment and posterior surface of the left P1 segment, then traveling posteriorly toward the posterior perforated substance. The mamillary perforating branches arise along the middle third of the posterior communicating artery. (1, Thalamoperforating arteries; 2, Posterior perforated substance; 3, PCA-P1 segment; 4, PCA-P2A segment; 5, Oculomotor nerve; 6, Posterior communicating artery; 7, Mamillary body; 8, Pituitary stalk; 9, Optic chiasm; 10, Amygdala; 11, Limen insulae; 12, Hippocampus head)

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The P2 section consists of the segment extending from the PCoA to the point where it leaves the midbrain and runs through the peduncular and ambient cisterns (Fig. 5). Zeal and Rhoton further subdivided this segment into the anterior (P2A) and posterior (P2P) subsegments. The anterior subsegment begins at the PCoA and ends at the most lateral aspect of the cerebral peduncle [Párraga 2011]. The average length of the P2A subsegment is 23.6 mm (range 18–30 mm) with an average diameter of 1.7 mm (range 1–3 mm) [Párraga 2011]. The posterior subsegment (P2P) begins where the PCA resumes its course along the posterior edge of the lateral surface of the midbrain. The average

length of the P2P subsegment is 1.4 mm (range 9–25 mm) with a diameter of 1.4 mm (range 0.8–2 mm) [Párraga 2011]. Perforators from this segment include the peduncle perforating arteries and thalamogeniculate arteries. The medial posterior choroidal arteries arise mainly from the P2A segment, as does the first cortical branch. The lateral posterior choroidal arteries arise from the P2P subsegment. In addition, the inferior temporal arteries also arise from the P2 segment.

The PCA segment between the inferior temporal arteries and the origin of the parieto-occipital and calcarine arteries constitutes the P3 segment. The perforators arising from this segment are the infe-

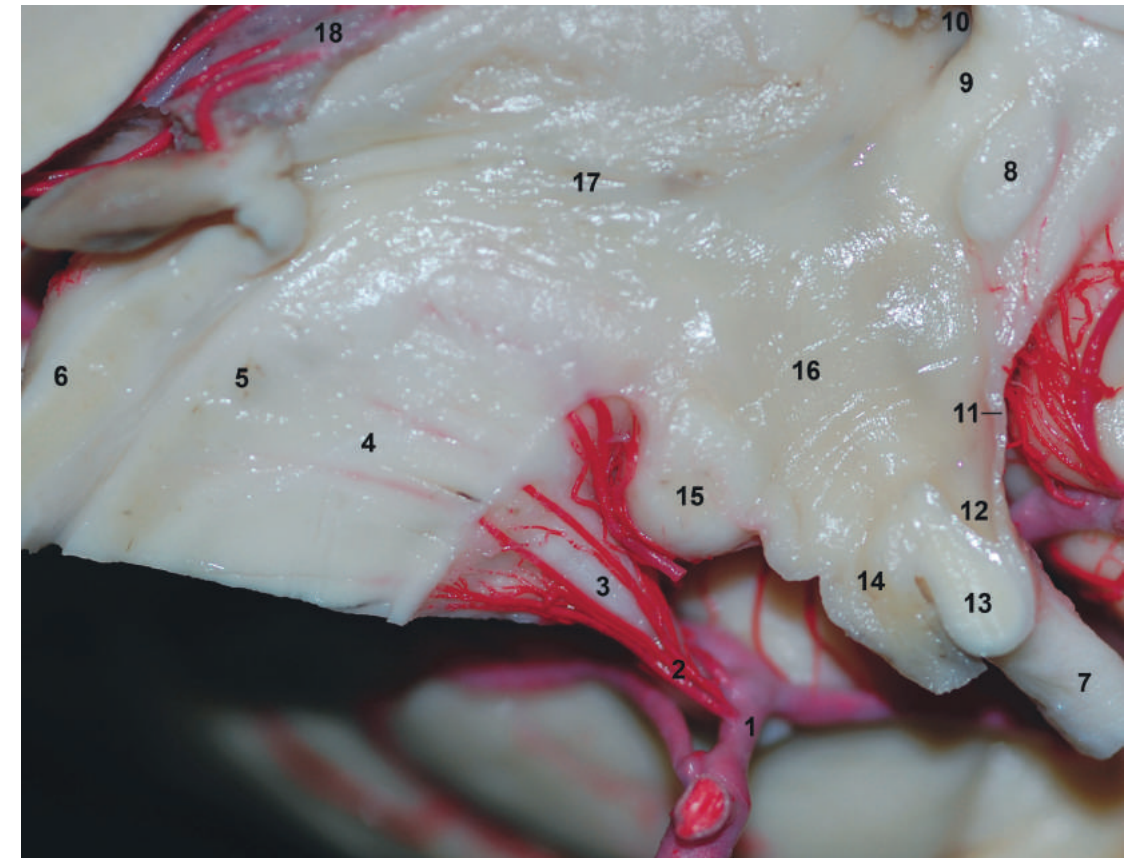


Figure 4. A view of the medial surface of the left hemisphere with magnification of the third ventricle and thalamoperforating arteries traveling toward the mesencephalic periaqueductal gray substance. (1, PCA-P1 segment; 2, Thalamoperforating arteries; 3, Oculomotor nerve; 4, Mesencephalic tegmentum; 5, Periaqueductal gray substance; 6, Quadrigeminal plate; 7, Optic nerve; 8, Anterior commissure; 9, Fornix column; 10, Foramen of Monro; 11, Lamina terminalis; 12, Chiasmatic recess; 13, Optic chiasm; 14, Infundibular recess; 15, Mamillary body; 16, Third ventricle; 17, Hypothalamic sulcus; 18, Velum interpositum cistern)

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rior temporal branches and the posterior pericallosal artery. This segment has an average length of 19.8 mm (range 9–30 mm) and an average diameter of 1.1 mm (range 0.5–1.5 mm) [Párraga 2011]. The point where the left and right posterior cerebral arteries are closest is known as the “collicular point”. This distance has been estimated to range from 2–35 mm [Margolis 1972, Párraga 2011, Zeal 1978]. The majority of the cortical PCA branches at the

lateral cerebral surface typically have a diameter of 0.4–0.6 mm [Zeal 1978].

The P4 segment consists of the terminal cortical branches such as the parietooccipital arteries, the calcarine arteries and the cortical branches. The P4 vessels supply the cuneus, the posterior aspect of the lingual gyrus and the occipital pole, including the primary visual cortex. The parietooccipital artery is one of the major terminal branches and lies in or

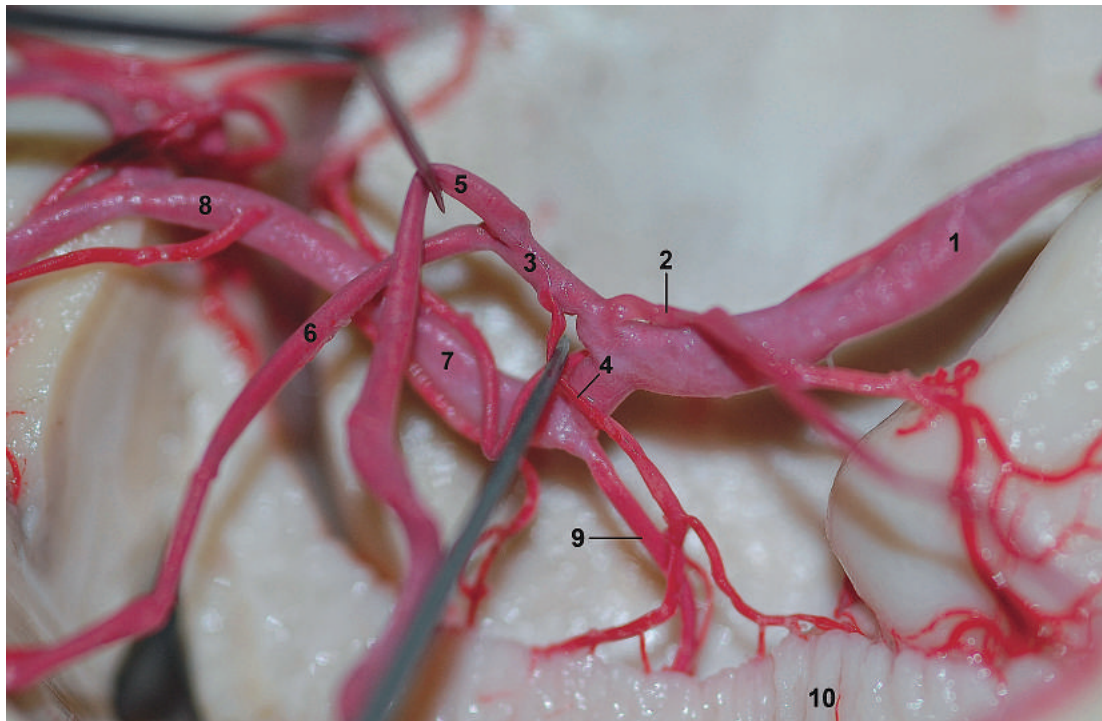


Figure 5. Inferior view showing the common temporal artery originated at the transition between the P2A and P2P segments. The hippocampal artery originates from the common temporal artery; the posterolateral choroidal artery from the P2P segment. (1, P2A segment; 2, anterior temporal artery; 3, common temporal artery; 4, hippocampal artery; 5, medial temporal artery; 6, posterior temporal artery; 7, P2P segment; 8, P3 segment; 9, posterolateral choroidal artery; 10, dentate gyrus)

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along the parietooccipital sulcus. The posterior pericallosal artery usually originates from the parietooccipital artery that anastomoses with the anterior pericallosal artery. A second terminal branch is the calcarine artery and runs in or along the calcarine sulcus to supply the cuneus and the lingual gyrus, as well as the visual cortex.

Important collaterals to the PCA territory come from the anterior pericallosal artery (ACA), the supracerebellar artery (SCA), anterior choroidal artery (ICA) and superior temporal branches (MCA).

2.3.2 Symptoms Associated to PCA Aneurysms

Most patients with PCA aneurysms are symptomatic of SAH following aneurysm rupture. The distribution pattern can be different between proximal (Fig. 6A) and distal (Fig. 6B) PCA aneurysms. PCA aneurysms are often associated with other intracranial vascular lesions such as: moyamoya, arterial occlusions, multiple aneurysms and AVM [Drake 1996, Honda 2004, Sawada 1998]. Therefore, many PCA aneurysms were detected by vascular imaging for these lesions. Symptoms of ischemia are relatively often associated with thrombosed large and giant aneurysms

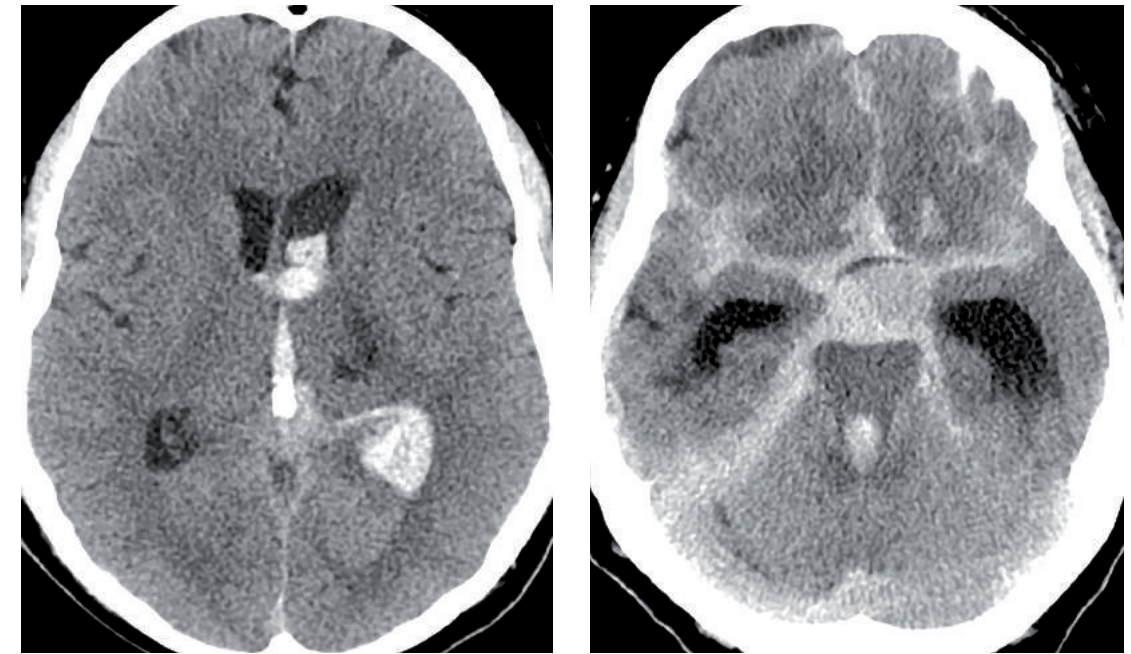


Figure 6. SAH caused by a left PCA-P3 (A) and a PCA-P1/2 junction aneurysm rupture (B).

[Drake 1996]. Due to the close anatomical relationship, affection of the oculomotor nerve is also a common sign, especially for proximal PCA aneurysms. Symptoms of visual disturbance, memory disturbance, seizures and temporal pain have been described for PCA aneurysms with mass effect [Drake 1996, Honda 2004].

2.3.3 Characteristics of PCA Aneurysms

Previously reported series have a high proportion of large and giant PCA aneurysms. Additionally, fusiform PCA aneurysms were commonly observed [Drake 1996]. In general, the proportion of proximal PCA aneurysms is predominant [Drake 1996, Yonekawa 2011].

2.4 Historical Aspects

Following the development of cerebral angiography by Moniz in 1933, Krayenbühl was able to angiographically diagnose the first posterior circulation aneurysm in 1941, located at the basilar artery [Krayenbühl 1941, Moniz 1933]. The first direct treatment for an angiographically diagnosed aneurysm was carried out by the team of DeSaussure in 1956 [DeSaussure 1958]. However, the microsurgical treatment of posterior circulation aneurysms was significantly influenced by Charles Drake, who summarized his experience together

with Sydney Peerless and Juha Hernesniemi in 1996 (Fig. 7) [Drake 1988/1996, Kassell 1999].

Furthermore, we must remember that J. Ausman and T. Sundt, Jr. contributed to the development of revascularization procedures applied to the posterior circulation (Fig. 8) [Ausman 1976/1981/1981, Kelly 1989, Sundt 1978/1981/1982]. They described extracranial-intracranial bypass procedures to the posterior circulation, bridging procedures between SCA and PCA as well as even presenting high flow augmentation in the form of an ICA-SV-PCA bypass in 1982 [Sundt 1981/1982].



Figure 7. Charles G. Drake

Sydney J. Peerless

Juha A. Hernesniemi

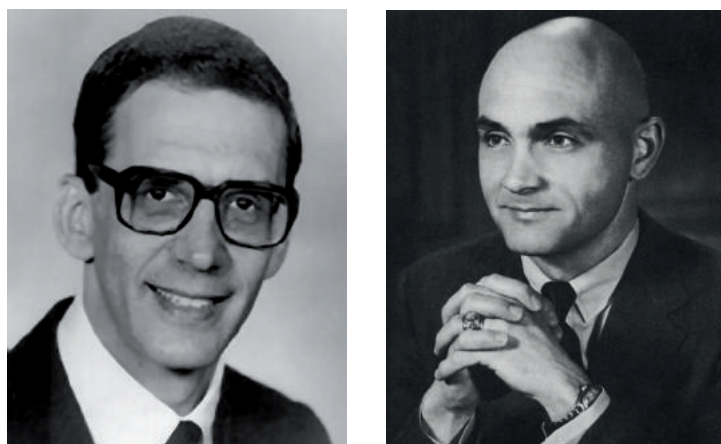


Figure 8. James I. Ausman

Thoralf M. Sundt Jr.

2.5 Treatment Strategies for PCA Aneurysms

Nowadays various open microsurgical and endovascular methods are available for the occlusion of PCA aneurysms. Clip occlusion, wrapping, proximal occlusion, trapping, trapping after bypass bridging and aneurysmorrhaphy of these aneurysms is possible by open microsurgery. Using endovascular techniques such as direct coil occlusion, balloon assisted coiling and stent assisted coiling, proximal occlusion, aneurysm coiling with parent vessel occlusion and combinations with bypass procedures are well described. Due to the low incidence of PCA aneurysms, it is an accurate assessment that comparative trials are not feasible.

2.5.1 Direct Microsurgical Treatment of PCA Aneurysms

The PCA aneurysm configuration and exact location in relation to skull base structures is particularly important when choosing the microsurgical approach. Each of approaches described hereafter are demanding due to the deep anatomical location of the PCA and the narrowed surgical corridor.

Irrespective of the approach selected, the following key points are always important:

- avoidance of injury to the temporal lobe from retraction,
- prevention of injury to the venous system,
- prevention of injury to the brainstem perforators,
- securement of a sufficient perfusion of the PCA territory while preventing PCA infarctions.

2.5.1.1 Frontolateral Approach to PCA Aneurysms

Several frontolateral approaches are described for the treatment of the proximal PCA aneurysms such as the pterional approach [Yasargil 1976, Yonekawa 1999, Yonekawa 2011], lateral supraorbital or extended lateral supraorbital approach [Goehre 2015, Hernesniemi 2005], anterior temporal approach [Goehre 2016, Sundt 1990] and orbitozygomatic approach [Gerber 1993, Hakuba 1986]. For the treatment of multiple intracranial aneurysms in particular, these frontolateral approaches provide access to the intracranial ICA, the ACA-A1 segment, the ACoA complex and proximal ACA-A2 segments, the MCA-M1, the MCA-bifurcation and the M2 segments.

Positioning, Skin Incision, & Craniotomy

The patient is placed in the supine position with the head rotated between 30° and 45° to the con-

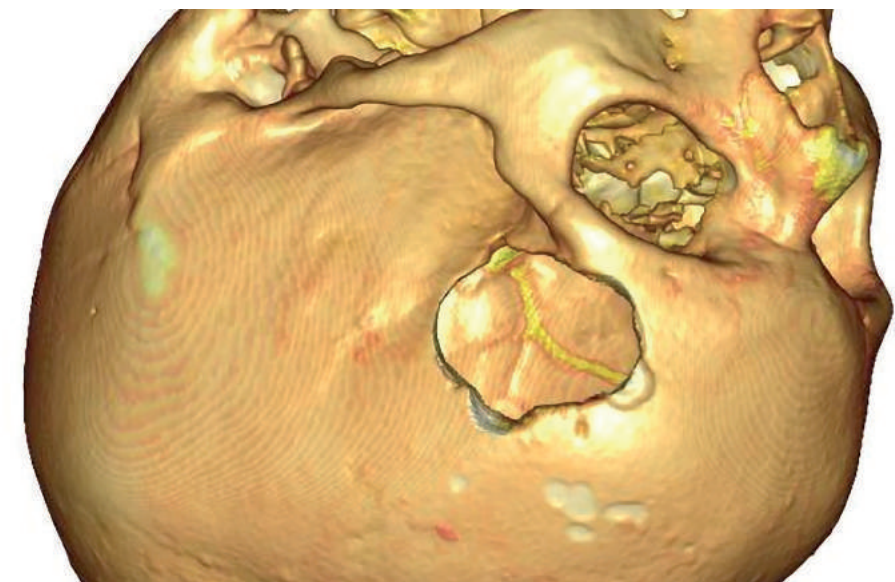


Figure 9. 3D CT of LSO for a left-sided approach.

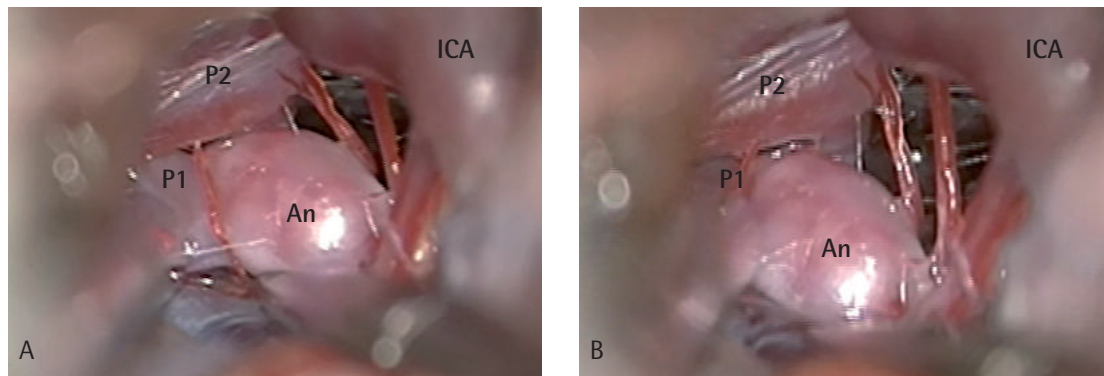
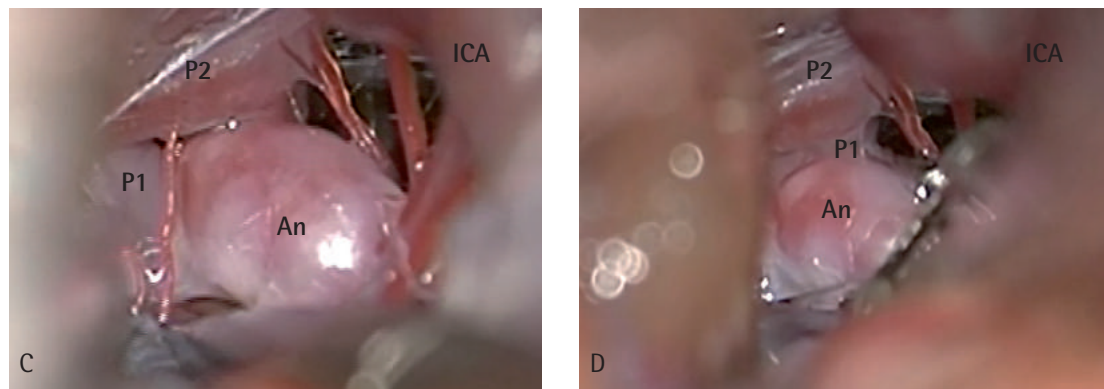


Figure 10. Mobilization of small perforators (A, B) during dissection of a PCA-P1 aneurysm from a left lateral supraorbital approach.



PCA-P1 aneurysm dissection (C) and clip occlusion (D) in the left carotid oculomotor triangle (An, Aneurysm; ICA, internal carotid artery; P1, P1 segment; P2, P2 segment).

tralateral side. Head and shoulders are slightly elevated above heart level. A skull clamp is used for head fixation. The skin is marked in a curved, oblique line just behind the hairline, approximately 8-10 cm long and 3 cm above the zygoma. The skin incision is made to detach a one-layer skin-muscle flap from the cranium. A left-sided lateral supraorbital craniotomy is presented in Figure 9.

Intracranial Dissection

With these frontotemporal approaches, the sylvian fissure must be widely opened to obtain enough space for the aneurysm dissection in this deep surgical corridor. Sometimes an anterior or posterior clinoidectomy is necessary to improve binocular

visualization [Yonekawa 1997]. For somewhat more distal PCA aneurysms, a selective amygdalohippocampectomy can be necessary for exact visualization of the aneurysm [Yonekawa 2011]. In particular, the transylvian route is preferable for high-lying proximal PCA aneurysms. Additionally, frontotemporal craniotomies allow access to most ipsilateral anterior circulation aneurysms. Exposure of the aneurysm neck can be more difficult from a frontotemporal view than a subtemporal view as the aneurysms of the P1 segment and P1/P2 junction typically project in the cranial or anterior direction. Treatment of PCA-P1/2 junction aneurysms is complicated by the involvement of the P1, P2 and PCoA in the aneurysm's formation and the ne-

cessity to preserve all three vessels. If one of the efferent vessels has to be sacrificed, the other must be preserved. During the microsurgical management of proximal PCA aneurysms, preservation of all perforating branches is crucial (Fig. 10).

2.5.1.2 Subtemporal Approach to PCA Aneurysms

The subtemporal approach is favorable for the treatment of PCA aneurysms in proximity to the tentorium. The approach was initially developed by Drake in 1961 for the treatment of basilar aneurysms and later adopted for PCA aneurysms. Different authors have subsequently contributed to the further development of this approach [Hernesniemi 2005/2005, McLaughlin 2014, Terasaka 2000, Zador 2010].

Positioning

The patient is placed in the lateral park bench position. To facilitate minimal retraction of the temporal lobe it is necessary to release 50 to 100 ml of cerebrospinal fluid (CSF) by lumbar drainage.

Skin Incision

A horseshoe-shaped skin incision is made starting 1 cm anterior to the tragus, above the zygomatic

arch, and curving posteriorly around the earlobe until reaching the high-line between the porion and asterion. A one-layer skin-muscle flap is then everted. The Sugita frame provides strong retraction in the basal direction via its spring hooks. The temporal muscle is dissected down to the origin of the zygomatic arch. During dissection, the external auditory canal should be left intact. The skin is typically thin over this region. If performing an external carotid/internal carotid bypass using the STA as a bypass donor, the skin incision can be varied.

Craniotomy

One burr hole is placed at the cranial border of the planned bone flap. A curved blunt dissector is used to carefully detach the bone from the underlying dura. A 3-4 cm bone flap is detached with a craniotome (Fig. 11). The first cut is made in the anterior direction to the base of the middle fossa; the second cut is made posteriorly towards the floor of the middle fossa. Finally the bone is thinned down along the basal border of the temporal bone and the bone flap is cracked. Holes for tack-up sutures are drilled at the cranial border of the craniotomy. The craniotomy is then widened basally by removing bone in the temporobasal direction using a high-speed drill to expose the floor of the middle fossa

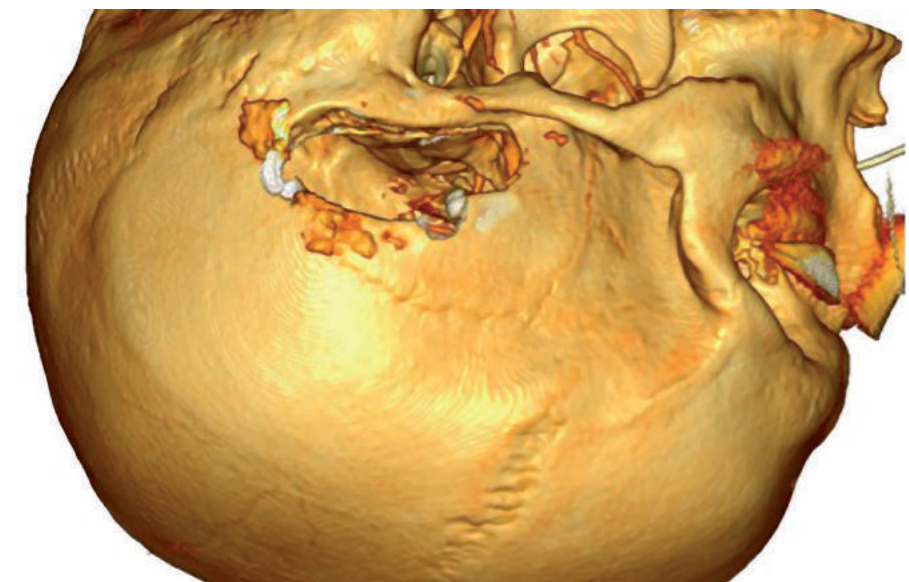


Figure 11. 3D CT of temporal craniotomy for a left subtemporal approach.

so that no ridges obstruct the view towards the subtemporal region. The craniotomy must be modified slightly to accommodate the exact aneurysm location.

Intracranial Dissection

The dura is opened via a curved flap with a caudal base and the dural edges are elevated over the craniotomy dressings. The secret to proper utilization of the subtemporal approach lies in gaining rapid access to the tentorial edge without heavy compression of the temporal lobe. This is where cisterns are opened to release additional CSF, allowing relaxation of the brain. The spinal drain can be closed at this point. The temporal lobe should be elevated close to the temporal pole. The dissection proceeds posteriorly, with great care taken not to excessively stretch the bridging veins. Abrupt retraction or elevation of the middle portion of the temporal lobe risks tearing of the vein of Labbé, which would result in swelling of the temporal lobe and venous infarction. Once the temporal lobe is mobilized and elevated with the tentorial edge visible, a retractor is placed to retain space for further advancement. We prefer a relatively wide retractor that provides a large surface area without focal pressure points. The elevation of the uncus with the retractor exposes the opening to the interpeduncular cistern and the third nerve [Yasargil 1976]. The subtemporal standard view is presented in Figure 12. The third nerve can be mobilized by cutting the arachnoid bands surrounding it, however palsy can develop even with minimal manipulation. By contrast, in some patients even prolonged manipulation of the third nerve does not lead to any signs of postoperative palsy. Even with the uncus retraction, the opening into the interpeduncular cistern remains narrow. It can be widened by placing a straight micro clip at the edge of the tentorium, in front of the insertion and the intradural course of the fourth nerve, lifting the tentorial edge upwards [Hernesniemi 2005]. If this procedure does not provide a wide enough corridor, we partially divide the tentorium for better exposure. The cut, perpendicular to the tentorial edge and measuring about 10 mm in length, is made posterior to the

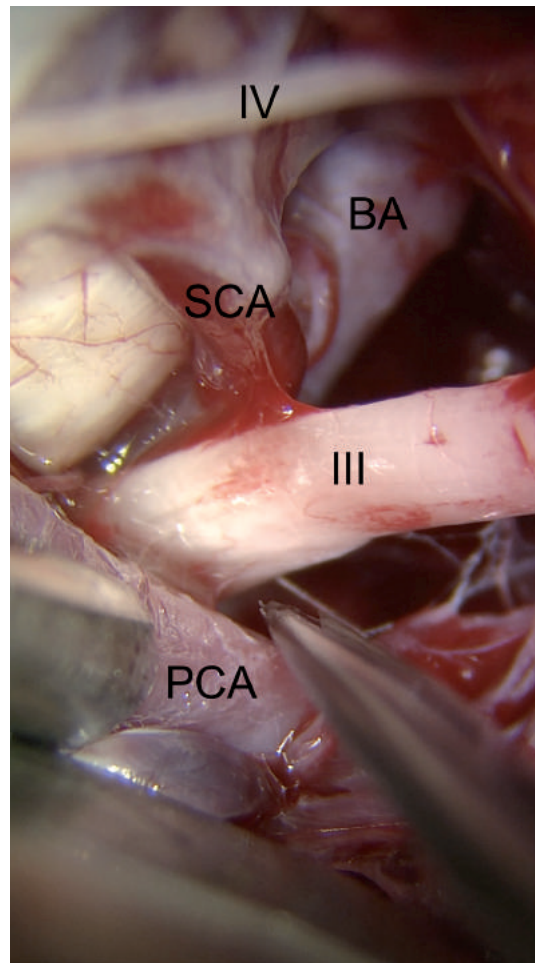


Figure 12. Standard view from a left subtemporal approach (III, oculomotor nerve; IV, trochlear nerve; BA, basilar artery; PCA, posterior cerebral artery; SCA, superior cerebellar artery).

insertion of the fourth nerve. The tentorial flap is then fixed with a small aneurysm clip to allow better exposure of the upper portion of the basilar artery [Hernesniemi 2005]. Venous bleeding after the first tentorium incision can be stopped by the injection of glue into this first small opening.

Vessel and Aneurysm Management

In cases with a low-lying P1 origin at the basilar bifurcation, exposure of the tentorium is absolutely necessary and a more posterior approach with a larger bone flap is preferred. The arachnoid is

opened via a wide incision beginning above the fourth nerve and the superior cerebellar artery (SCA), below the third nerve and the side of mid-brain, and continuing forward to free the carotid and posterior communication arteries. A careful water dissection technique described by Toth et al. in 1987 is useful when entering the narrow arachnoid layers [Nagy 2006, Toth 1987]. Oculomotor nerve paresis is the second most common complication of open microsurgical management of upper posterior circulation aneurysms. This is related to the close proximity of the PCA to the oculomotor nerve. Direct mechanical irritation and disturbance of the vascularization of the oculomotor nuclei and fascicles is possible. The frontotemporal approach allows a slightly better trajectory for the protection of the third nerve compared to the subtemporal approach. The paresis usually regresses after a period of several weeks to a few months. Due to the different surgical trajectory, the trochlear nerve can interfere with the surgical field of the subtemporal approach (Fig. 13). Intraoperative monitoring of the third and fourth nerves can prevent permanent damage [Zhou 2012].

Prior to clipping, perforating arteries must be protected carefully during preparation and exposure of the aneurysm. In cases of SAH, this preparation is hindered by blood clots. Before clipping the aneurysmal neck, the perforating branches must be carefully separated. Often a complete dissection of the aneurysm dome is not advisable. Larger P1-P2 junction aneurysms can be challenging in that P1, P2 and posterior communicating arteries may be involved in the aneurysm formation, necessitating cautious clipping to preserve the integrity of all three vessels. The P2 segment is hidden under the parahippocampal gyrus as it winds around the side of the midbrain. As the P2 is followed out from its anterior extremity, it is usually possible to elevate this gyrus gently with the tip of the retractor to locate the P1 branching and the neck of the aneurysm. Occasionally, it is necessary to remove a centimetre or so of the parahippocampal gyrus in order to expose a highly placed or more complex aneurysm lying on the upper midbrain in the choroidal fissure. Temporary clipping is recom-

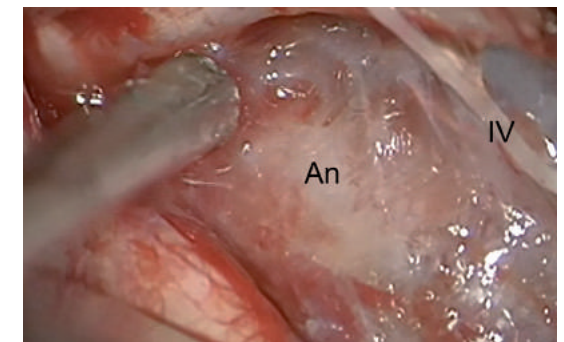


Figure 13. Mobilization and preservation of the right trochlear nerve from the sack of a PCAP1/2 junction aneurysm using a right subtemporal approach (IV, trochlear nerve; An, aneurysm).

mended during dissection around the aneurysm (Fig. 14). The duration of temporary clipping should be short and caution is advised with atherosclerotic vessels. Temporary clips should not interfere with the final clipping procedure. While placing temporary clips, care must be taken to avoid accidentally injuring the perforating arteries.

After the final clip has been placed, the distal temporary clip is removed first to evaluate if there is any bleeding from a potentially incompletely clipped aneurysm. A safe clip replacement is possible whereby the clip is released without actually being removed. While slowly placing the final clip, the perforating arteries should be closely observed to avoid injury. In the case of difficult neck inlay into the clip where temporary clipping is not possible, it can be useful to induce a transient cardiac arrest using a rapid bolus of intravenous adenosine (20–25 mg) [Luostarinen 2009]. After pilot clip placement, the dissection around the aneurysm can be extended if necessary. Aneurysm puncture and down coagulation is used to shrink the aneurysm before placing the final permanent clip. This extends the overall view of the dissection area and helps prevent injuries to perforating arteries and branches. The technique of permanent clip placement is well described [Kamiyama 2010, Lawton 2010]. An example for tandem clipping of a giant PCA-P2 aneurysm is given in Figure 15.

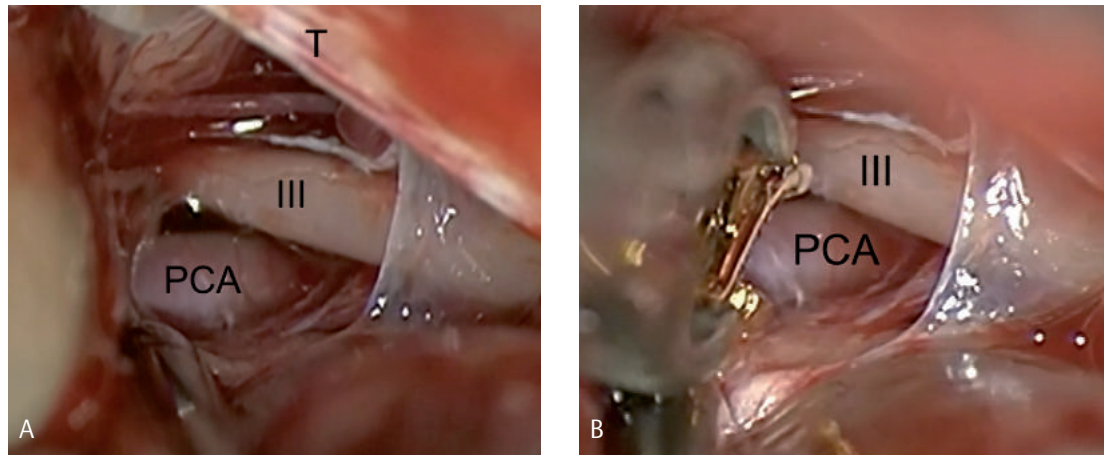


Figure 14. Exposure (A) and temporary proximal occlusion (B) of the anterior P2 segment through a left subtemporal approach, in proximity to the oculomotor nerve (III, oculomotor nerve; PCA, posterior cerebral artery; T, tentorium).

For immediate intraoperative control of the aneurysm, use of intraoperative indocyanine green (ICG) videoangiography has been mandatory at our institution since 2006, and allows real-time imaging of the vascular system [Dashti 2010, Raabe 2003/2005]. Care should also be taken that the aneurysm clips do not interfere with the cranial nerves.

Closure

The entire closing procedure is performed under microscopic view and completed in layers. However a water-tight closure is necessary. During initial

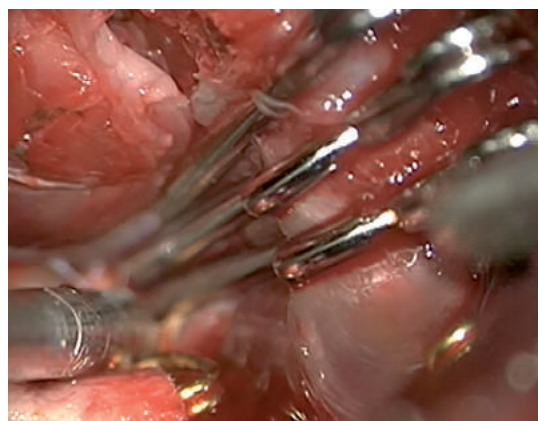


Figure 15. Tandem clipping of a large PCA-P2 aneurysm using a right subtemporal approach.

temporobasal drilling, very often some of the air cells of the temporal bone are opened. This necessitates meticulous closure at the end of the surgery to prevent postoperative CSF leak. Utilization of Tachosil® can be helpful in this case [Kivelev 2015]. Sealing the air cells with a part of the temporal muscle flap everted over the bony edge and sutured to the dura is one option; other methods include using fat graft, fibrin glue, and bone wax.

2.5.1.3 Posterior Approaches to PCA Aneurysms

Far distal PCA aneurysms on the P3 and P4 segments require different approaches. In 1984 Yasargil described the parasagittal parietal-occipital craniotomy in sitting position for the dissection and clipping of distal PCA aneurysms. Although far distal PCA aneurysms at the P4 segment are extremely rare, they are easily accessible from an occipital interhemispheric approach [Ishibashi 1989, Hashimoto 2000, Ito 1998, Orita 1994, Yamahata 2010]. Figure 16 shows a right-sided parasagittal parietooccipital craniotomy. Later Yonekawa et al. in 2011 described the supracerebellar transtentorial approach in sitting position for the microsurgical treatment aneurysms of the PCA-P2P and PCA-P3 segments of the PCA [Yonekawa 2001/2011]. Both approaches allow the application of OA-PCA by-

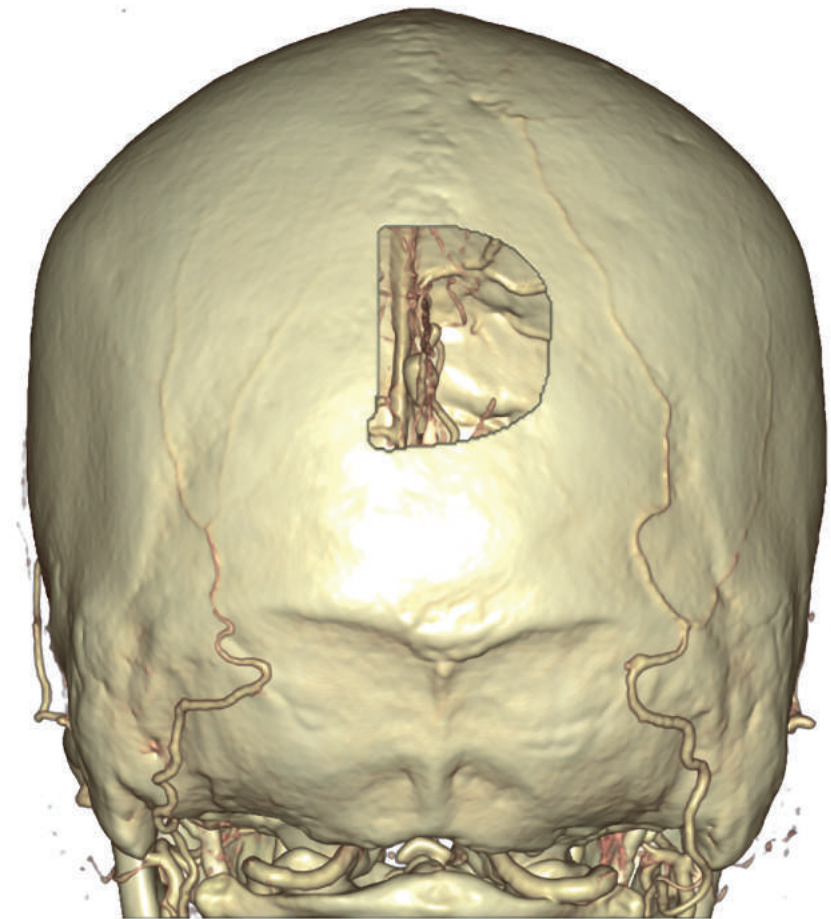


Figure 16. Right sided parasagittal parietooccipital craniotomy.

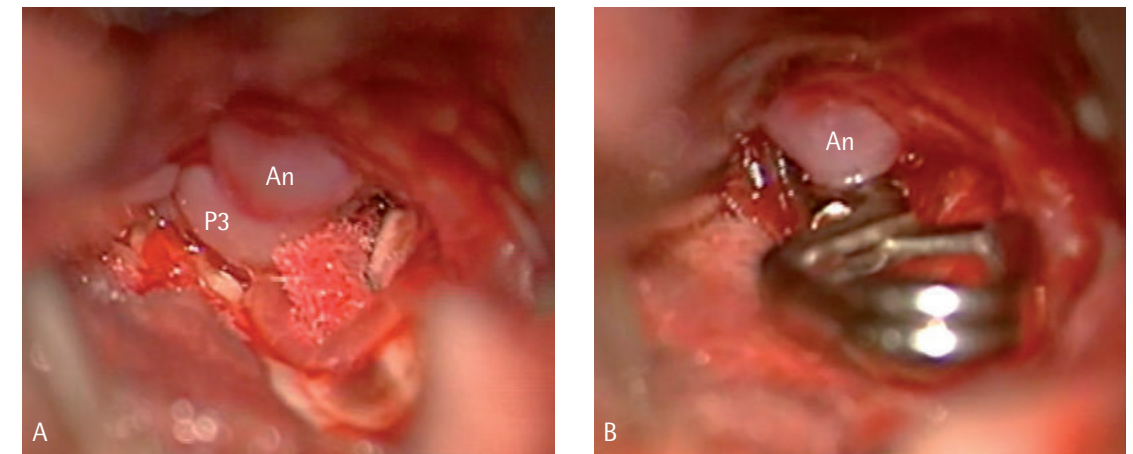


Figure 17. Dissection (A) and clipping (B) of a P3 segment aneurysm (An) using a right posterior interhemispheric approach.

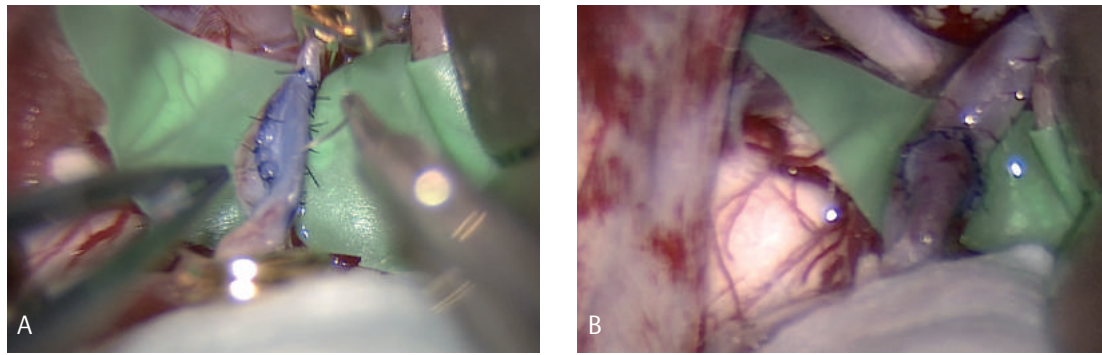


Figure 18. Application of a microanastomosis (A) for a STA-P2 bypass (B) through a left subtemporal approach.

passes for the revascularization of distal PCA territory [Vishteh 1998, Yonekawa 2011]. Figure 17 show the microsurgical view from a posterior interhemispheric approach for the clip occlusion of a right-sided P3 segment aneurysm.

2.5.1.4 Revascularization Procedures Applied to PCA Aneurysms

Revascularization procedures of the PCA are considered to be effective for the prevention of cerebral ischemic infarctions, including the distal PCA area [Chang 2010, Goehre 2016, Kalani 2014, Kazumata 2011, Mohit 2007, Saito 2006, Sekhar 2001, Sundt 1982, Takeuchi 2015, Touho 1995, Vishteh 1998, Yasargil 1969/1970/1977, Yonekawa 2011, Zador 2010]. Multiple bypass procedures with the PCA as the recipient vessel have been described, such as the OA-PCA [Chang 2010, Kazumata 2011 Touho 1995, Vishteh 1998], STA-PCA (Fig. 18) [Goehre 2016, Hopkins 1982, Takeuchi 2015, Ulku 2010], MMA-PCA [Ustun 2006] and the ECA-RA-PCA or ECA-VA-PCA bypass as high flow bypass procedures [Reyes 1995, Sundt 1982/1986, Sundt III 1987, Shi 2011, Sekhar 1999, Tanikawa 2006]. The visibility of the arterial wall can be improved by the use of pyocyanium blue (Fig. 19) [Kamiyama 1993]. The SCA-PCA anastomoses are described as in situ techniques. A side-to-side technique as well as an end-to-side (Fig. 19) technique is possible [Saito 2006, Sundt 1982].

The vessel diameters are shown in Table 1. However, specific complications like bypass failure and intracranial hematomas have also been described [Chang 2010]. The dimensions of the vessel calibers are also summarized in Table 1. Even application of ELANA bypasses is possible [Langer 2005, Tulleken 1993/1998]. The depth of the surgical corridor from a subtemporal approach amounts to 50–65 mm according to own measurements. In this way, this anastomosis is substantially different from a superficial vascular reconstruction and special training is required [Yonekawa 1999]. The intraoperative assessment of the bypass flow can be tested by indocyanine green videoangiography and ultrasonic perivascular flow probe [Charbel 1998, Raabe 2003/2005, Woitzik 2005].

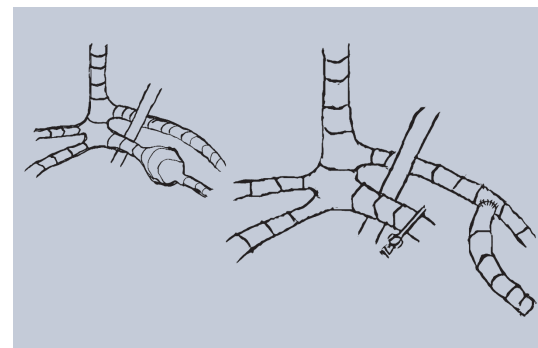


Figure 19. PCA-SCA end-to-side anastomosis after aneurysm resection (schematic drawing) according to Sundt, Jr. (1982).

Table 1. Vessel diameters for revascularization of the PCA [Kawashima 2005/2011].

	Vessel Diameter (mm)
Posterior cerebral artery (PCA)	
Anterior P2 segment	2.13 ± 0.38
Posterior P2 segment	1.73 ± 0.33
P3 segment	1.67 ± 0.16
Superior cerebellar artery (SCA)	
Lateral pontomesencephalic segment (single trunk)	1.51 ± 0.12
Occipital artery (OA)	
At the digastric groove	2.05 ± 0.48
At the level of the superior nuchal line	2.01 ± 0.45
Superficial temporal artery (STA)	
At the level of zygoma	1.93 ± 0.48
External carotid artery (ECA)	
Cervical portion	5.75 ± 0.94

2.5.2 Endovascular Treatment of PCA Aneurysms

Endovascular interventions of the PCA are usually done under general anesthesia and systemic heparinization [Ciceri 2001, Hallacq 2002]. Saccular PCA aneurysms of the P1 and P1/2 junction are suitable for the direct GDC coil embolization with parent vessel preservation [Ciceri 2001]. For wide neck PCA aneurysms, a balloon assisted technique or stent assisted coiling can be helpful to avoid encroachment of the coils in the vessel lumen of the parent artery (Fig. 20) [Arat 2002, Ciceri 2001, Huang 2013]. A combined occlusion of the aneurysm and the parent artery is often required for the treatment of PCA aneurysms distal to the P1/2 junction (Fig. 21); this is possible by detachable coils (Fig. 22), histoacryl and detachable balloons [Ciceri 2001, Cotroneo 2007, Eckard 2000, Hallacq 2002, van Rooij 2006]. The authors consider that most patients are able to compensate the parent vessel occlusion by collateral flow, but ischemic complications occur in up to 28% [Ciceri 2001], 50% [Cotroneo 2007] or 78% [Kashiwazaki 2011] of cases. However, the long endovascular route to

the terminal segments of the posterior circulation and a tortuous path can aggravate or prevent endovascular access.

2.5.3 Outcome after Posterior Cerebral Artery Aneurysm Treatment

2.5.3.1 Radiological Outcome

Regarding the literature, it appears that the rate of incomplete occlusion of PCA aneurysms is very low [Drake 1996, Hallacq 2002]. The reason for this is that when an aneurysm was considered as difficult for neck clipping or endovascular occlusion, another option such as parent vessel occlusion was used for complete exclusion of the aneurysm formation from the circulatory system [Cotroneo 2007, Drake 1996, Hallacq 2002, Kashiwazaki 2011]. In general, the rate of parent vessel occlusions is with 43–100% high for PCA aneurysms, which is due to their characteristic features such as a high incidence of fusiform shape [Cotroneo 2007, Drake 1996, Hallacq 2002, Kashiwazaki 2001]. Since the adoption of stent assisted coiling the frequency of parent vessel occlusion has decreased for this type of aneurysm

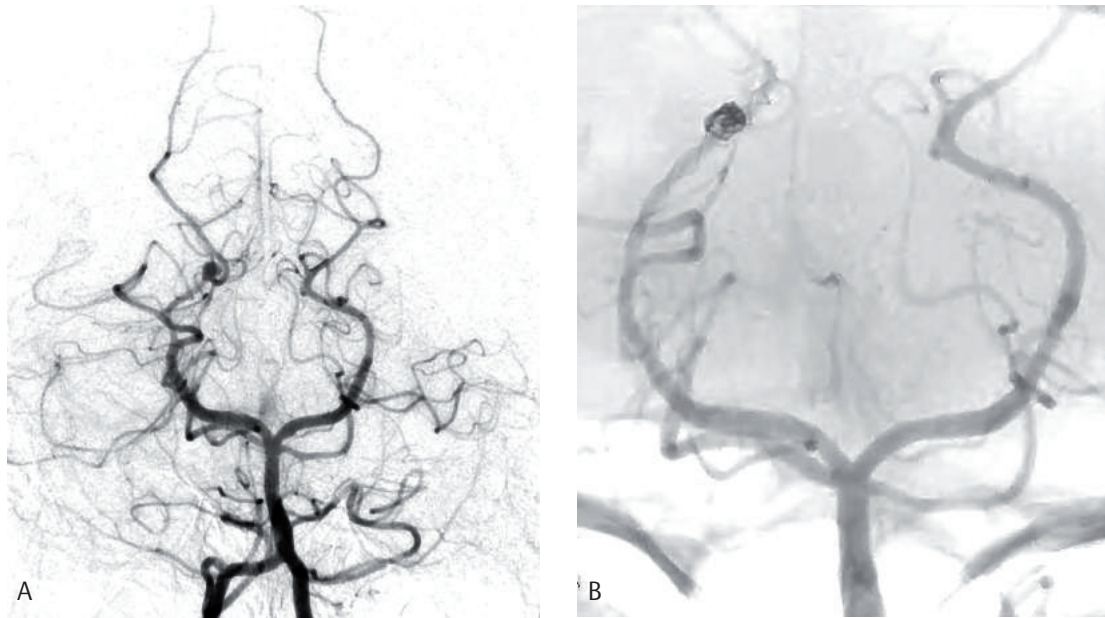


Figure 20. A fusiform PCA-P3 segment aneurysm (A) is occluded with a GDC coil under stent protection of the PCA (B).

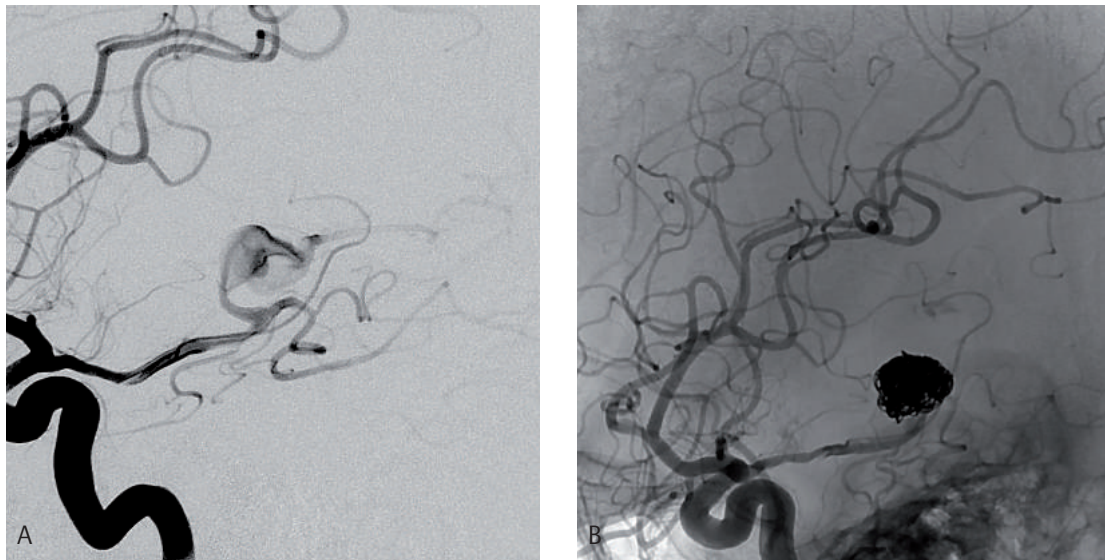


Figure 21. GDC occlusion of a fusiform PCA-P3 segment aneurysm (A) with parent artery occlusion (B) on a fetal left PCA.

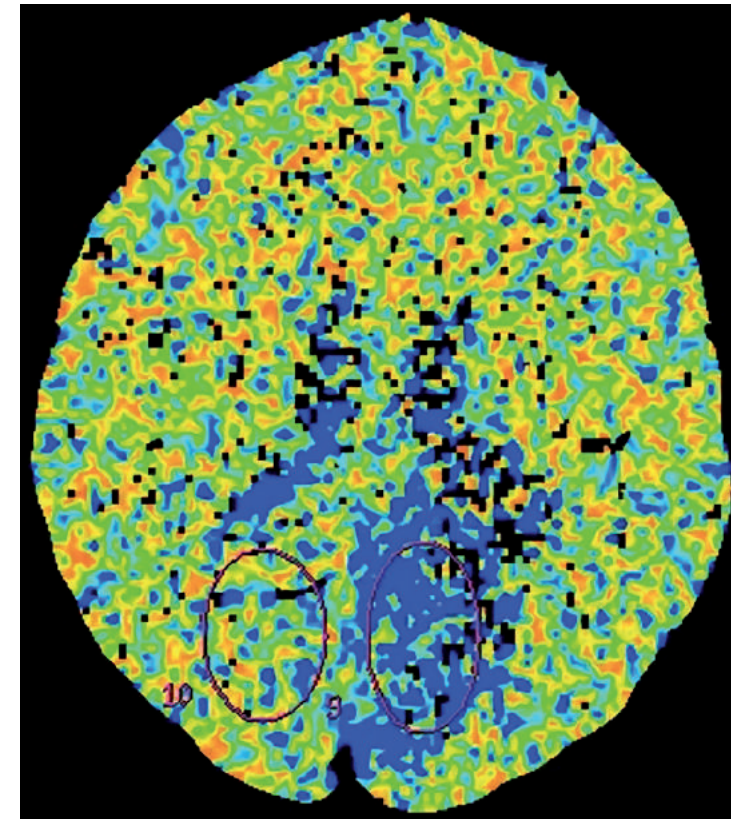


Figure 22. Decreased perfusion (blue; MTT) of the left PCA territory after endovascular parent vessel occlusion.

regarding to endovascular treatment [Huang 2013]. However, incidence of ischemic infarction (Fig. 22) following parent vessel occlusion is high at 50–78% in all series that included consequent follow up, even when the infarct area was not large [Cotroneo 2007, Kashiwazaki 2011]. However, in case of bypass application MRI imaging with 4D-flow techniques allows for less invasive bypass flow evaluation [Sekine 2015].

2.5.3.2 Clinical Outcome

Early reports on conservative and surgical management of PCA aneurysms show unfavorable but also good treatment outcomes [Bertrams 1968, Drake 1969, Hunt 1967]. As an example, the first

results of Drake and Amacher from 1969 were presented: The treatment of 8 patients with PCA aneurysms show that one patient died, one had a poor outcome caused by contralateral hemiplegia and ipsilateral oculomotor palsy, and another patient suffered a monoparesis and a oculomotor paresis. The other five patients had a good or excellent outcome.

It is problematic to give a general overview of the treatment results due to the low incidence of PCA aneurysms; most of the series are small, with fewer than 25 cases and analyzed using different evaluation standards. Table 2 provides a summary of PCA aneurysm series with more than 10 cases.

The following is a summary of the treatment results of the five most rigorous reports on PCA aneurysms, each with more than 30 patients. Drake

Table 2. Series on PCA aneurysms.

Author	Year	Cases	Treatment
Drake et al.	1996	125	surgical
Zhitao et al.	2010	42	surgical
Chang et al.	2010	33	surgical and endovascular
Wang et al.	2015	30	surgical
Taylor et al.	2003	30	surgical and endovascular
Sanai et al.	2008	27	surgical and endovascular
Kim et al.	2013	25	surgical and endovascular
van Rooij et al.	2006	22	endovascular
Park et al.	2015	21	surgical and endovascular
Kashiwazaki et al.	2011	21	endovascular
Hamada et al.	2005	21	surgical, endovascular and conservative
Yonekawa et al.	2011	20	surgical
Ciceri et al.	2001	20	endovascular
Li et al.	2008	18	endovascular
Seoane et al.	1997	15	surgical
Gerber et al.	1992	15	surgical
Roh et al.	2008	13	endovascular
Kitazawa et al.	2001	11	surgical
Sakata et al.	1993	11	surgical
Luo et al.	2012	10	endovascular
Coert et al.	2007	10	surgical and endovascular
Honda et al.	2004	10	surgical and conservative
Hallacq et al.	2002	10	endovascular

et al. (1996) report roughly 78% good or excellent results among their 125 patients (75 patients following SAH) [Drake 1996]. Chang et al. (2010) from the Barrow Institute report good or excellent results in 29 of 33 cases (87%), however 3 cases (9%) were fatal and 9 patients (27%) had an initial SAH [Chang 2010]. Taylor et al. (2003) analyzed 30 patients and reported that 8 of 18 patients (44%) with unruptured PCA aneurysms and 6 of 12 patients (50%) with ruptured PCA aneurysms achieved a good outcome after one-year follow-up; however, one patient died during treatment [Taylor 2003].

In the 30-patient case series of Wang et al. (2015), 26 patients (87%) had a good result even though 18 patients (60%) suffered from an initial aneurysm rupture and 18 of the aneurysms were large or giant [Wang 2015]. There was a single fatal outcome in this study. Zhitao et al. (2010) analyzed a patient collective of 42 patients with PCA-P2 segment aneurysms, 25 of whom after SAH [Zhitao 2010]. Remarkably, 41 patients (97%) had a good treatment result after a mean follow-up period of 5.8 years.

3. Aims of the study

To describe the characteristic features of PCA aneurysms.
(Study I)

To analyze the treatment strategies and outcomes of PCA aneurysms.
(Study II)

To analyze the treatment of PCA aneurysms via subtemporal approach.
(Study III)

To describe the treatment of PCA aneurysms and ipsilateral anterior circulation aneurysms inside the circle of Willis via lateral supraorbital approach.
(Study IV)

4. Patients, Materials and Methods

The study retrospectively reviewed 133 patients (41 men and 92 women) with 147 PCA aneurysms. All patients were treated between 1934 and 2014 at one of two Finnish neurosurgical centers: the Department of Neurosurgery at the University of Eastern Finland, Kuopio and the Department of Neurosurgery at the University of Helsinki. These institutions are the only neurosurgical centers for all of southern and eastern Finland, serving a population of 2.8 million people. Between 1934 and 2014 a total of 15,300 patients with 19,177 intracranial aneurysms were treated at our institutions. Data was collected with the approval of the local university ethics committee (469/E0/04 HUCH). A commercially available software package was used for data analyses (SPSS for Mac, version 21.0 [2012]; SPSS, Inc, Chicago, Illinois).

Study I

"Characteristics of Posterior Cerebral Artery Aneurysms: An Angiographic Analysis of 93 Aneurysms in 81 Patients" analyzed 81 patients with 93 PCA aneurysms.

Study II

"Posterior Cerebral Artery Aneurysms: Treatment and Outcome Analysis in 121 Patients" analyzed 121 patients with 135 PCA aneurysms.

Study III

"Subtemporal Approach to Posterior Cerebral Artery Aneurysms" analyzed 34 patients with 37 PCA aneurysms treated via subtemporal approach.

Study IV

"Lateral supraorbital approach to ipsilateral PCA-P1 and ICA-PCoA aneurysms" described the lateral supraorbital approach for the treatment of a proximal PCA aneurysm in one patient.

4.1. Publication I – Characteristics of PCA Aneurysms

4.1.1 Patients

The characteristic features of 93 PCA aneurysms in 81 patients were analyzed. These patients were treated between 1980 and 2012 at the neurosurgical centers of the University of Eastern Finland, Kuopio and the University of Helsinki. Patients with a subarachnoid hemorrhage had a median age of 51 years (range: 11–86 years) at diagnosis while patients without had a median age of 45 years (range: 10–88 years) at diagnosis.

4.1.2 Imaging

The PCA aneurysms were diagnosed by conventional digital subtraction angiography (DSA) in 27 patients, by computed tomography angiography (CTA) in 31 patients, and with both methods in 22 patients. One patient was diagnosed by magnetic resonance angiography (MRA) alone. If CTA and DSA were performed, CTA was used for the presented measurements. For DSA analyses a metallic reference was used, as small measurement errors are possible even with localization of pathology close to the central beam.

The following angiographic variables were determined: maximal aneurysm length, width, and shape (fusiform/saccular), neck and parent artery diameter, distance to the basilar bifurcation for P1, P1/2 junction and P2 segment aneurysms, dome orientation of saccular aneurysms and any secondary pouches, and the anatomic relationship to the posterior clinoid process.

For calcified or thrombosed aneurysms the measurements were done from outer wall to outer wall. The widest wall-to-wall distance was used for the calculation of the median aneurysm size. The image analyses were done by two experienced neu-

rosurgeons, one of whom has double specialization in neurosurgery and neuroradiology according to European Union regulations.

4.2 Publication II – Treatment and Outcome of PCA Aneurysms

4.2.1 Analysis

Hospital records and images were analyzed to identify patients with PCA aneurysms. From these resources the following data were collected: patient age, sex, Hunt and Hess grade on admission, treatment modality, time to treatment, occlusion grade, complications, neurological deficits, and cause of death. The modified Rankin Scale (mRS) was used to measure outcomes, evaluated at one-year follow-up. For presentation and analysis of the data, outcomes were categorized into three groups: (i) good (mRS 0–1), (ii) moderate (mRS 2–4), and (iii) poor (mRS 5–6). This represents a more rigorous review than that in "Publication III – Subtemporal Approach to CPA Aneurysms". All patients were examined angiographically for diagnosis via computed tomography angiography (CTA), digital subtraction angiography (DSA), or magnetic resonance angiography (MRA). Radiological images were stored in the hospital's digital archiving system (PACS; AGFA, IMPAX, version 4.5, launched in 1998) while X-ray photographs were housed in the central X-ray image archive. The image analyses were completed by two experienced neurosurgeons, one with double specialization in neurosurgery and neuroradiology. The following radiological data were collected: aneurysm height, neck diameter, dome width, previous treatment (coiling or clipping), and the presence of remnant aneurysm parts following treatment.

4.2.2 Patients

A total of 121 consecutive patients (36 male, 85 female) with 135 PCA aneurysms were identified and further analyzed. Patients were subdivided into nine groups for detailed analyzes: 1) unruptured saccular aneurysms (n=9), 2) unruptured fusiform aneurysms

(n=10), 3) unruptured giant aneurysms (n=7), 4) unruptured PCA aneurysms with associated aneurysms (n=21), 5) ruptured saccular aneurysms (n=20), 6) ruptured fusiform aneurysms (n=7), 7) ruptured PCA aneurysms with associated aneurysms (n=18), 8) SAH from an associated aneurysm rupture (n=17), and 9) SAH from an associated AVM (n=12). In total, 52 patients presented ruptured PCA aneurysms.

4.3 Publication III – Subtemporal Approach to PCA Aneurysms

4.3.1 Patients

Between 1980 and 2012, 34 patients (7 male, 27 female) with 37 PCA aneurysms were operated on via the subtemporal approach. Fourteen of these patients became symptomatic by acute subarachnoid hemorrhage from PCA aneurysm rupture. Eight patients had a H&H grade of 1–2 and 6 patients a H&H grade of 3–4. Six patients had acute hydrocephalus. Two patients had an intracerebral hematoma in the temporal lobe and one in the occipital lobe. Additionally, five patients had intraventricular hemorrhage. Another aneurysm ruptured in two patients, both of whom had a H&H grade of 2. Eighteen patients had no SAH. Twelve patients were diagnosed incidentally; six patients were symptomatic by hemiparesis from mass effect (n=2), headache (n=2), diplopia (n=2) and embolic syndrome (n=1).

4.3.2 Analysis

The clinical data and radiological images were analyzed retrospectively. Clinical variables collected included age, gender, aneurysm rupture state, Hunt and Hess grade, date of surgery, intraoperative procedures, and complications. The complications were divided into approach related complications and aneurysm management related complications. The modified Rankin scale (mRS) was used for the description of the early outcome after 3 months. Good outcome was classified as mRS 0–2, moderate outcome as mRS 3–4, and poor outcome as mRS



Figure 23. Axial CTA shows a left ICA-PCoA (left arrow) and PCA-P1 aneurysm (right arrow).

5-6. Radiological image analyzes used the preoperative and postoperative computed tomography, computed tomography angiography, and digital subtraction angiography images for determination of the aneurysm location, aneurysm size and shape, Fisher grade, intracerebral hematoma, intraventricular hemorrhage, postoperative parent artery occlusion, and postoperative hematomas and infarcts.

4.4 Publication IV – Lateral Supraorbital Approach to PCA Aneurysm

Publication IV was written as a technical note for the presentation of the LSO for the combined treatment of ipsilateral anterior circulation aneurysms.

4.4.1 Patient History and Imaging

Following an ischemic insult, a cervical and intracranial computed tomography angiography (CTA) was performed on a 59 year old female patient. Thereby, an occlusion of the right carotid artery and two intracranial aneurysms was detected (Fig. 23). The patient presented a left ICA PCoA aneurysm (saccular, neck 4 mm, width 6 mm, length 8 mm) and a left sided PCA P1 aneurysm (saccular, neck 2 mm, width 3 mm, length 6 mm).

5. Results

5.1 Characteristics of PCA Aneurysms

Location

Proximal PCA aneurysms are more frequent than distal PCA aneurysms. The collected data has the following distribution: P1As (n=39), P1/P2As (n=25), P2As (n=21), and P3As (n=8). The proximal PCA segments (P1 and P1/2 junction) were affected by 69% (n=64) of all PCA aneurysms and 77% of these were located above the posterior clinoid level. Additionally, 54% of all PCA aneurysms were located at the left PCA.

Symptoms of Unruptured PCA Aneurysms

Analysis included 64 unruptured PCA aneurysms in 53 patients. Vascular imaging for ruptured associated aneurysms show 12 associated PCA aneurysms. Eight patients were diagnosed following symptoms of ischemic strokes, three patients presented oculomotor nerve palsy, and ten patients had AVM-related PCA aneurysms. One patient had a PCA aneurysm and an AVM with feeding arteries from the anterior circulation. Only one patient became symptomatic by mass effect. Eighteen aneurysms were found incidentally.

Anatomic Variations

An ipsilateral fetal PCA configuration was observed in nine patients and a contralateral fetal PCA configuration in six patients. Two patients presented a bilateral fetal CPA configuration. One patient had the entire anterior circulation filling from the contralateral PCoA. One patient with a P1 aneurysm had an ipsilateral middle cerebral artery perfusion from the PCoA. A kinking P1 segment was present in three P1 segment aneurysms and in one P1/2 junction aneurysm.

Parent PCA Diameter

The parent artery diameter was larger for unruptured PCA aneurysms (1.8 mm) than for ruptured

(1.5 mm). This was still the case even when PCA aneurysms from AVM feeding PCAs were removed from the analysis. There was no difference in the mean parent artery diameters between aneurysms of the proximal (P1 and P1/2 junction) and distal (P2 and P3) PCA; the mean diameter was 1.5 mm in both groups.

Involvement of PCA Branches at the Origin

An angiographically visible branch originating from the aneurysm or its base was found in 37 aneurysms (59%), excluding P1/P2 junction aneurysms. In 26 saccular aneurysms these perforating branches originated from the aneurysm base. In fusiform aneurysms, we observed that in seven cases these branches originated immediately proximal or distal from the aneurysm formation. In four cases the perforating branches originated directly from the fusiform aneurysm.

Size and Features of Fusiform PCA Aneurysms

A greater proportion of the fusiform aneurysms were either large (n=1) or giant (n=5) compared with saccular aneurysms: 25% vs 7%. An irregular wall structure was found in four unruptured (25%) and five ruptured (63%) fusiform PCA aneurysms. The P2 segment was particularly affected by fusiform PCA aneurysms (n=9). Only two fusiform P3 segment aneurysms were detected, both of which were ruptured. An overview is given in Table 3.

Size and Features of Saccular PCA Aneurysms

Only 74% (n=69) of the observed PCA aneurysms were saccular; the remaining 26% were fusiform (Tab. 4). The rupture rate of saccular and fusiform aneurysms was comparable at 30% vs. 31%, respectively. The subclassification of unruptured saccular PCA aneurysm (n=48, 52%) was the largest subgroup. Most of the 21 ruptured saccular PCA aneurysms were of small or medium size; 12 (57%)

Table 3. Length and diameter analyzes of 24 fusiform PCA aneurysms.

	P1 aneurysms	P1/2 junction aneurysms	P2 aneurysms	P3 aneurysms	Total
Fusiform aneurysms unruptured, no. (%)	4 (10%)	5 (20%)	7 (33%)	0	16 (17%)
Length [mm]; median (range)	5 (3-8)	10 (2-35)	8 (5-40)	-	8 (2-40)
Diameter [mm]; median (range)	3 (2-14)	10 (2-32)	5 (5-20)	-	5 (2-32)
Fusiform aneurysms ruptured, no. (%)	4 (10%)	0	2 (10%)	2 (25%)	8 (9%)
Length [mm]; median (range)	8 (3-9)	-	7 and 37	12 and 13	8.5 (3-37)
Diameter [mm]; median (range)	4 (2-6)		6 and 33	9 and 10	6 (2-33)

Table 4. Characteristics of 69 saccular PCA aneurysms.

	P1 aneurysms	P1/2 junction aneurysms	P2 aneurysms	P3 aneurysms	Total
Saccular aneurysms unruptured, no. (%)	26 (67%)	11 (44%)	9 (43%)	2 (25%)	48 (52%)
Length [mm]; median (range)	5 (1-12)	5 (2-21)	4 (2-36)	3 and 35	3 (1-36)
Width [mm]; median (range)	2 (2-8)	3 (2-18)	4 (2-36)	3 and 24	3 (2-36)
Neck [mm]; median (range)	2 (1-10)	2 (2-14)	2 (2-9)	3 and 8	2 (1-14)
Saccular aneurysms ruptured, no. (%)	5 (13%)	9 (36%)	3 (14%)	4 (50%)	21 (23%)
Length [mm]; median (range)	5 (2-9)	6 (2-17)	15 (11-21)	6 (3-9)	6 (2-25)
Width [mm]; median (range)	4 (2-4)	6 (2-14)	9 (8-15)	4 (3-6)	4 (2-15)
Neck [mm]; median (range)	3 (2-3)	4 (3-10)	6 (4-8)	3 (3-4)	3 (2-10)

Table 5. Dome orientation of saccular PCA aneurysms.

PCA Segment	Typical Aneurysm Dome Orientation (%)
P1 aneurysms	upward (67%)
P1/2 junction aneurysms	anterior or upward (80%)
P2 aneurysms	lateral (67%)
P3 aneurysms	posterior (50%)

of them were smaller than 7 mm. An irregular wall structure with secondary pouches was found in 15 (22%) of the saccular PCA aneurysms, 10 (66%) of which were ruptured. Saccular PCA aneurysms had a typical dome projection for each segment, as presented in Table 5.

P1 Segment Aneurysms

Most PCA aneurysms were located at the P1 segment (n=39, 42%). Most of these were saccular (n=31, 79%) and unruptured (n=30, 77%). There was no statistically significant size difference between ruptured and unruptured saccular P1 aneurysms (P=.38). The dominant dome projection for saccular aneurysms was upward (n=21, 67%), in continuation of the blood flow from the basilar artery. Half of the eight fusiform P1 segment aneurysms were ruptured and the remainder were unruptured. The four ruptured aneurysms had a somewhat larger average size: 8 mm vs 5 mm.

P1/2 junction Aneurysms

The P1/P2 junction was the second most frequent location for PCA aneurysms, with 25 aneurysms (27%). Most of these were unruptured (n=16, 64%) and saccular (n=20, 80%). The saccular P1/P2 junction aneurysms projected mainly in either an anterior (n=10, 50%) or upward (n=6, 30%) direction. There was only a minor average size difference between the ruptured (n=9) and unruptured (n=11) saccular P1/P2 junction aneurysms: 6 mm vs. 5 mm. All five fusiform P1/P2 junction aneurysms were unruptured. The P1/P2 junction aneurysms had the most complex angioarchitecture of PCA aneurysms in general. In five cases the PCoA originated just proximal to the aneurysm base and in four cases just distal to the aneurysm base. In eight cases the PCoA was completely involved in the aneurysm formation.

P2 Segment Aneurysms

There were 21 P2 segment aneurysms (24%), most of which were unruptured (n=16, 76%). Although saccular aneurysms were more common than fusiform, the proportion of fusiform aneurysms was larger at the P2 segment than the P1 segment

or P1/P2 junction (40% vs. 21% and 20%, respectively). Therefore, the P2 segment was the most common location for fusiform aneurysms (n=9) in our series. There was an average size difference between the ruptured (n=3) and unruptured (n=9) saccular P2 segment aneurysms (15 mm vs. 4 mm; P=.22). Fusiform P2 segment aneurysms affected a longer portion of the parent artery than fusiform aneurysms at other PCA segments. In general, diagnosed P2 segment aneurysms were larger than aneurysms at the other PCA segments. The saccular P2 segment aneurysms mainly projected in the lateral direction (n=8, 67%).

P3 Segment Aneurysms

The P3 segment was the least frequent location for PCA aneurysms, representing only eight aneurysms (9%). Six (75%) of these were ruptured, which represents the highest rupture rate of all PCA segments. Unlike at the P2 segment, the proportion of fusiform aneurysms (25%) at the P3 segment was similar to that of the proximal PCA aneurysms. Both of the fusiform P3 segment aneurysms were ruptured. There was one giant unruptured saccular P3 segment aneurysm. The four ruptured saccular P3 segment aneurysms had a median length of 6 mm, similar to the ruptured saccular P1 segment or P1/P2 junction aneurysms. Three of the six saccular P3 segment aneurysms (50%) projected in the posterior direction.

Associated Aneurysms

Altogether 49 of 81 patients had a total of 98 associated aneurysms. Interestingly, 36 of these 49 patients became symptomatic by an associated aneurysm rupture. The vessel distribution of associated aneurysms is given in Table 6.

PCA Aneurysms in Association with AVM

In ten patients the PCA aneurysm was found on an AVM feeding PCA. In two patients, multiple PCA aneurysms were found. The average age of patients with AVM related PCA aneurysms was less than the average study population (40 vs. 50 years), but this difference was not statistically significant (p=.72). Most of the AVM related aneurysms were located

Table 6. Distribution of 98 associated aneurysms.

Affected Vessel	Quantity of Aneurysms
Middle cerebral artery	21
Internal carotid artery	19
Posterior cerebral artery	12
Posterior communicating artery	13
Anterior communicating artery	11
Distal anterior cerebral artery	9
Basilar artery	7
Superior cerebellar artery	4
Vertebral artery	2

on the proximal PCA in close relation to the basilar artery; the mean distance to the basilar artery was 6 mm. The aneurysm distribution along the PCA segments was as follows: P1 (n=9), P1/2 junction (n=2), and P2 (n=3). The aneurysms had a saccular configuration with a median width of 4 mm. The median parent vessel diameter on AVM related PCA aneurysm was larger than in non-AVM related PCA aneurysms: 2.5 vs. 1.5 mm.

5.2 Outcome after PCA Aneurysm Treatment

5.2.1 Historical Series (1954–1979)

To provide an overview of conservative treatments, we analyzed historical records. There were 12 patients with 12 PCA aneurysms treated before 1980. Of these 12 aneurysms, nine (75%) were ruptured. The distribution of the aneurysms along the PCA segments was: P1As (n=2; 17%), P1/2As (n=8; 66%), P2As (n=1; 8%), P3As (n=1; 8%). Three PCA aneurysms were found in autopsy, nine with conventional angiography. These patients (5 male, 7 female) had a mean age of 43 years (range 23–63 years). All patients were treated conservatively. The follow-up period ranged from 3 months to 34 years in initial survivors. One patient re-bleed after 24 years of conservative treatment and the aneurysm was treated by microsurgical clip occlusion.

5.2.2 Distribution of 135 PCA Aneurysms

The P1 segment was the most affected segment with 53 aneurysms (39%), followed by the P1/2 junction (n=39; 29%), P2 segment (n=28; 21%), and P3 segment (n=15; 11%). The highest rupture rate was in P3 aneurysms (60%), particularly fusiform P3 segment aneurysms (75%). However, the P2 segment was most often affected by fusiform aneurysms (n=10).

5.2.3 Clinical Presentation

SAH was the most common symptom (n=62). An additional IVH was present in 21 patients, ICH in 3 patients, and a SDH in 1 patient. In 17 patients the SAH was caused by associated aneurysm rupture. Twelve patients (10%) had an AVM-related PCA aneurysm. Oculomotor nerve affection was found in seven patients (6%). Six patients (5%) had symptoms of ischemia following embolism. Two patients became symptomatic from mass effect.

5.2.4 Microsurgical Treatment

Sixty-three aneurysms were treated microsurgically; two aneurysms had previously been incompletely occluded by coiling. Aneurysms affected the P1 segment (n=18), P1/2 junction (n=22), P2 segment (n=15), and P3 segment (n=8). Forty-four

aneurysms were saccular and nineteen aneurysms fusiform. The following approaches were used: subtemporal approach, pterional approach, lateral supraorbital approach, and posterior interhemispheric approach for distal aneurysms. Modified temporal approaches were used for two bypass procedures. The following procedures were applied: neck clipping (n=45), failed clipping (n=1), neck clipping after failed endovascular occlusion (n=2), aneurysmorrhaphy (n=1), trapping (n=2, one after failed clipping), proximal occlusion (n=7), parent vessel occlusion after bypass (n=4, two ELANA procedures), and wrapping (n=1). In 23 procedures, associated aneurysms were addressed from the same approach. Incomplete aneurysm occlusion occurred in six cases after neck clipping and in after aneurysm trapping. Oculomotor nerve palsy was observed in nine patients and trochlear nerve palsy in one patient.

5.2.5 Endovascular Treatment

Endovascular treatment was applied for 19 aneurysms affecting the P1 segment (n=8), P1/2 junction (n=4), P2 segment (n=3) and P3 segment (n=4). Only four of these aneurysms were fusiform. Direct coiling (n=15), stent assisted coiling (n=1) and sole stenting (n=2) were the techniques applied. In one case the parent vessel was occluded to occlude three PCA aneurysms. Parent vessel occlusion was used in a second case, where the patient developed an asymptomatic PCA infarction. Five aneurysms were incompletely occluded, two of which were subsequently surgically treated and three followed up. Two patients developed permanent oculomotor nerve palsy.

5.2.6 Conservative Treatment

Consideration for conservative treatment was mainly based on the patient's poor clinical condition (i.e. H&H grade V, n=10). Twenty-nine PCA aneurysms with a size under 4mm were treated conservatively. Most AVM related aneurysms were also treated indirect or conservatively following AVM treatment. One patient died following an ia-

trogenic injury during diagnostic DSA. One patient was treated conservatively due to advanced age.

5.2.7 One-Year Outcome

Patients with Unruptured Saccular PCA Aneurysms (n=9)

Six of nine patients were treated conservatively (Tab. 7). One patient with oculomotor palsy and older age (88 years) was treated conservatively (mRS was 1 after one year with persistent oculomotor palsy). During the first year of follow-up, one patient died following a cardiac infarction. The other patients had aneurysms below 4 mm in size and all of them had favorable outcomes (mRS 0). Three patients were treated microsurgically (Tab. 7). Two patients, including one who underwent bypass bridging, had a good outcome. One patient had a moderate outcome caused by PCA infarction after parent vessel occlusion.

Patients with Unruptured Fusiform PCA Aneurysms (n=10)

Nine patients were diagnosed incidentally. One patient became symptomatic with embolic symptoms. Only one patient with a small aneurysm (<3 mm) was treated conservatively and had a good outcome. All other patients were treated by microsurgical means. Three patients were treated by proximal occlusion and two of them developed a PCA infarction after proximal occlusion. One aneurysm was clipped following failed coiling and one clipped aneurysm required re-operation to trap the aneurysm. Four aneurysms were directly clipped. Three patients developed permanent oculomotor nerve palsy following direct clipping. One aneurysm was trapped after bypass bridging. The overall outcome was good in five cases and moderate in five cases (Table 7).

Patients with Ruptured Saccular PCA Aneurysms (n=20)

All of these patients suffered from SAH. One patient had an additional oculomotor palsy. Four patients were treated conservatively due to poor clinical condition on admission (H&H V, n=3) or advanced

Table 7. One-year outcome of 46 patients with PCA aneurysms.

	Unruptured Saccular PCA Aneurysms (n=9)	Unruptured Fusiform PCA Aneurysms (n=10)	Ruptured Saccular PCA Aneurysms (n=20)	Ruptured Fusiform PCA Aneurysms (n=7)
Treatment, no.				
Microsurgical treatment	3	9	13	2
Endovascular treatment	0	0	3	2
Conservative	6	1	4	3
Outcome, no.				
mRS 0-1 (good)	7	5	8	2
mRS 2-4 (moderate)	1	5	6	3
mRS 5-6 (poor)	1	0	6	2

age and severe cardiac dysfunction (n=1) (Tab. 7). Two patients were treated by direct coiling and one by a stent/coil technique (Tab. 7). Two patients had a good outcome. The other patient developed an anterior circulation infarction and recovered with a mRS of 4 after one year. Microsurgical treatment with direct clip application was applied to 13 patients (Tab. 7). The parent vessel was occluded in five cases. One patient had a poor outcome that later proved fatal after intraoperative aneurysm rupture during clipping from a pterional approach. One aneurysm was incompletely occluded by clipping and the patient died following re-rupture (confirmed in autopsy). The one-year outcome was good in six cases and moderate in four cases.

Patients with Ruptured Fusiform PCA Aneurysms (n=7)

All patients in this subgroup suffered from SAH. Three were in poor clinical condition and treated conservatively. Only one patient survived during the first year. Two were treated successfully by endovascular techniques. In one case, a patient developed a PCA infarction following extended temporary clipping while managing an intraoperative rupture. One patient was treated successfully by proximal occlusion. The outcomes are summarized in Table 7.

Patients with Unruptured Giant PCA Aneurysm (diameter $\geq 25\text{mm}$; n=7)

Three patients became symptomatic via rupture, two via mass lesion, one via thromboembolism, and one was an incidental finding. Six aneurysms were located at the P2 or P3 segment and four presented a fusiform shape. The following treatment methods were applied: proximal occlusion via subtemporal approach (n=3), coil embolization with parent vessel occlusion (n=1), thrombectomy and PCA reconstruction (n=1), complex ELANA bypass (n=1). One patient was treated conservatively following iatrogenic injury during DSA. A ventriculoperitoneal shunt was required in one case. One patient had a good outcome, five had moderate outcomes, and one a fatal outcome (Tab. 8).

Patients with Unruptured PCA Aneurysms with Associated Unruptured Aneurysms (n=21)

Twenty-one patients had multiple aneurysms and unruptured PCA aneurysms (Tab. 8). Thirteen patients had a good outcome and seven a moderate outcome. The remaining patient died following a complication from a diagnostic DSA procedure. Microsurgery was applied to ten patients. In three cases a pterional or lateral supraorbital approach was used to occlude the PCA aneurysm in the same procedure with associated aneurysms. In five patients a two-step strategy was chosen. In two cases

Table 8. One-year outcomes of 75 patients with complex vascular lesions and PCA aneurysms.

	Patients with Unruptured Giant PCA Aneurysms (n=7)	Patients with Unruptured PCA Aneurysms with Associated Unruptured Aneurysms (n=21)	Patients with Unruptured PCA Aneurysm and Ruptured Associated Aneurysm (n=17)	Patients with Ruptured PCA Aneurysms with Associated Unruptured Aneurysms (n=18)	Patients with an Associated AVM (n=12)
Treatment, no.					
Microsurgical treatment	5	10	4	10	2
Endovascular treatment	1	3	3	5	0
Conservative	1	8	10	3	10
Outcome, no.					
mRS 0-1 (good)	1	13	7	5	3
mRS 2-4 (moderate)	5	7	4	9	8
mRS 5-6 (poor)	1	1	6	4	1

an additional posterior interhemispheric approach was used to occlude a distal PCA aneurysm (P3 segment). Flow modulation by a complex bypass procedure was used in one case for the treatment of multiple posterior circulation aneurysms. In three cases, all aneurysms were occluded by endovascular techniques in one interventional session. Proximal occlusion was used in one case to occlude a total of three PCA aneurysms. In eight patients the PCA aneurysm was treated conservatively due to small aneurysm size or poor clinical conditions.

Patients with Unruptured PCA Aneurysms with Associated Ruptured Aneurysms (n=17)

Seventeen patients suffered from associated aneurysm rupture, exhibiting the following Hunt and Hess grade distribution: H&H I (n=3), H&H II (n=7), H&H III (n=1), H&H IV (n=2), and H&H V (n=4). The following intracranial vessels were affected by ruptured aneurysms: MCA (n=5), BA (n=4), ICA (n=3), ACoA (n=3), and VA (n=2). For three patients, endovascular approaches were used to treat all affecting aneurysms. A multi-stage procedure with a microsurgical approach for the PCA aneurysm was used in four cases. Ten patients with

an unruptured PCA aneurysm were treated conservatively after the treatment of the ruptured aneurysm. Following this treatment, one patient was in such poor condition that the cerebral aneurysms could not be treated. A secondary hydrocephalus was treated by EVD in five cases (29%). A permanent shunt was not needed for any of these patients. Seven patients had a good outcome, three patients a moderate outcome, and six a fatal outcome (Tab. 8).

Patients with Ruptured PCA Aneurysms with Associated Unruptured Aneurysms (n=18)

Eighteen patients with ruptured PCA aneurysms and associated aneurysms were treated (Tab. 8). Poor clinical condition (H&H grade V) led to conservative treatment in three cases, with all outcomes being fatal. One suffered from a cardiac infarction following SAH. Microsurgery was applied to ten patients. Ruptured PCA aneurysms were addressed directly in six cases and all reachable aneurysms were occluded from the same approach. Additional surgeries were necessary in four cases. The following outcome results were achieved: good (n=3), moderate (n=6), and fatal (n=1). Five patients

were treated endovascularly. In four cases associated anterior circulation aneurysms were treated by clip occlusion. Two patients recovered with a good outcome and three a moderate outcome.

Patients with an Associated AVM (n=12)

There were 12 patients with associated AVMs; in eight of these cases the aneurysm was located on an AVM-feeding PCA. Direct microsurgical occlusion was considered necessary in only two cases; in one case a ruptured saccular P2 aneurysm was clipped and in the other case a P3 aneurysm was clipped together with a flow-related basilar bifurcation aneurysm. Both cases utilized via subtemporal approach. In six cases the aneurysms were treated indirectly by AVM occlusion. A conservative treatment was chosen for four patients. One patient was in poor clinical condition after AVM rupture. The outcome was good in three cases, moderate in eight cases, and poor in one case. The outcome deficit was primarily related to the AVM and its treatment. Only one patient suffered from a flow-related PCA aneurysm rupture. The outcomes are summarized in Table 8.

5.2.8 Complications and Poor Outcome Analyses

Of the 121 patients with PCA aneurysms (52% ruptured vs. 48% unruptured, but 33% SAH from associated aneurysm rupture), 21 died during the first year and two remained vegetative. Thirteen of these patients arrived under poor clinical conditions (H&H grade V) and were primarily treated conservatively. Five patients had a moderate SAH (H&H III-IV): one died by cardiac infarction, one bled in an infarct area after proximal PCA occlusion, and three died following severe vasospasm and multiple infarctions. Two patients without SAH died following DSA complications and one by a cardiac infarction. Two other patients entered a vegetative state; one of these cases was associated with a ruptured AVM. Six of eleven patients with parent vessel occlusion had a symptomatic PCA infarction. Incomplete aneurysm occlusion occurred in six cases after neck clipping and aneurysm trapping. Nine patients de-

veloped oculomotor nerve palsy following microsurgical treatment and two after endovascular treatment. One developed trochlear nerve palsy after microsurgical treatment.

5.3 Subtemporal Approach to PCA Aneurysms

Characteristics of 37 PCA Aneurysms Treated from a Subtemporal Approach

Thirty-seven PCA aneurysms were treated from a subtemporal route. In particular, the P2 segment was easily approachable in 16 cases, followed by the P1/2 junction (n=10), the p1 segment (n=6) and the P3 segment (n=5). The aneurysm configuration is listed in Table 9.

Aneurysm Management

The specific treatment modalities for saccular and fusiform PCA aneurysms are shown relative to the rupture status in Tables 10 and 11.

Outcome after 3 Months

Of the patients with SAH from PCA aneurysm rupture, ten achieved a good outcome (mRS 0-2), two achieved a moderate outcome (mRS 3-4), and two had a poor outcome (mRS 5-6). One patient died due to hemorrhagic infarction of the PCA territory 17 days after the proximal occlusion of a P3 aneurysm. The other patient with a poor outcome had an initial Hunt and Hess grade of V on admission and remained in a vegetative state.

There were two patients suffering from SAH and unruptured PCA aneurysms. The treatment of the associated PCA aneurysms was uneventful. One patient achieved a good outcome and the other a moderate outcome in regards to the treatment of the primary ruptured aneurysms.

Of the 18 patients without PCA aneurysm rupture, 14 achieved a good outcome (mRS 0-2) and 4 a moderate outcome (mRS 3-4). Two of these patients developed symptoms of a PCA infarction after accidental parent vessel occlusion subsequent to direct clipping of a medium (10 mm) and large (21 mm) PCA aneurysm. One patient with a giant

Table 9. Shape and size of 37 PCA aneurysms operated via a subtemporal approach.

	Ruptured Aneurysms	Unruptured Aneurysms
Saccular aneurysms, no.	9	12
Length (mm); median (range)	9 (5-25)	4 (2-35)
Neck (mm); median (range)	4 (2-8)	2 (2-14)
Fusiform aneurysms, no.	5	11
Length (mm); median (range)	13 (9-37)	8 (5-40)
Diameter (mm); median (range)	11 (5-33)	6 (5-32)

Table 10. Treatment strategies for 23 unruptured PCA aneurysms.

Treatment Modality	Unruptured saccular PCA Aneurysm (n=12)	Unruptured fusiform PCA Aneurysm (n=11)
Clipping	10	4
Wrapping	1	
Proximal occlusion	-	4
Trapping	-	1
Trapping after bypass	-	1
Proximal occlusion after ELANA bypass	-	1
Aneurysmorrhaphy	1	-

Table 11. Treatment of ruptured 14 PCA aneurysms.

Treatment Modality	Ruptured saccular PCA Aneurysm (n=9)	Ruptured fusiform PCA Aneurysm (n=5)
Clipping	8	2
Proximal occlusion	-	3
Trapping	1	-

(35 mm) saccular PCA aneurysm developed a hemiparesis after proximal occlusion and another patient with a giant (40 mm) fusiform PCA aneurysm developed a thalamic infarction by a thromboembolism after an ELANA high flow bypass procedure.

Aneurysm Management Related Complications

The most common complication after PCA aneurysm treatment was the occurrence of postoperative infarctions in the PCA supply area. Our series had four such infarctions after direct neck clipping, six after proximal occlusion, one after aneurysm trapping, and one after a complex ELANA procedure. Small neck remnants were found in three aneurysms after direct neck clipping. One of these patients was retreated by aneurysm trapping using the same approach.

The distribution of complications for each treatment option in total numbers; saccular vs. fusiform is shown in parentheses (n/n).

Approach Related Complications

Two patients with a ruptured PCA aneurysm suffered from postoperative temporal lobe contusions caused by temporal lobe retraction. Cranial nerve palsy was observed in six patients; five of these patients exhibited oculomotor nerve palsy, four of whom recovered well during the first 3 months, and one patient suffered from trochlear nerve palsy. Wound infection, dehiscence, and postoperative CSF leakage were not observed in our series.

Table 12. Treatment associated complications.

Complication	Clipping (n=25)	Proximal occlusion (n=7)	Trapping (n=2)	ELANA (n=1)	Total
PCA infarction	4 (2/2)	6 (0/6)	1 (0/1)	1 (0/1)	12 (2/10)
Temporal lobe contusion	2 (2/0)	–	–	–	2 (2/0)
Neck remnant	3 (1/2)	–	–	–	3 (1/2)
Intraoperative rupture	1 (1/0)	–	–	–	–
Oculomotor nerve palsy	4 (3/1)	–	–	1 (0/1)	5 (3/2)
Trochlear nerve palsy	–	–	1 (1/0)	–	1 (1/0)

5.4 Lateral Supraorbital Approach to PCA Aneurysms

Using the lateral supraorbital approach we were able to occlude ipsilateral aneurysms of the ICA-PCoA segment and the PCA-P1 segment by clip-occlusion in 55 minutes operation time, skin to skin. The postoperative computed tomography angiography shows a patent posterior cerebral artery. The patient was discharged six days after the surgery with the same neurological state demonstrated preoperatively (mRS of 2), caused by a preexisting right cerebral infarction secondary to an idiopathic right carotid artery dissection.

6. Discussion

6.1 Characteristics of PCA Aneurysms

Aneurysm Distribution

Our analysis confirms the findings of previously published series that saccular proximal PCA aneurysms are more common than distal PCA aneurysms [Ciceri 2001, Drake 1996] as only 32% of PCA aneurysms were found distal to the P1/2 junction. The most affected location in particular was the P1 segment. Similar distribution patterns are described for the anterior cerebral artery (ACA) and the middle cerebral artery (MCA) [Dashti 2007, Lehecka 2008]. In contrast, fusiform PCA aneurysms present a more uniform distribution pattern. Unequal hemodynamic conditions across the different portions of the arteries may play a role in the formation of these saccular aneurysms.

Ruptured PCA Aneurysms

It appears that although distal PCA aneurysms are less frequent, they are also more dangerous as evinced by P3 segment aneurysm rupture rates of up to 75% compared to 38% for proximal PCA aneurysms. However, the mechanism is difficult to explain, as distal aneurysms are located in segments with lower flow. Further structural and transcriptomic investigations are necessary to analyze the wall structure and the degeneration processes in these aneurysms [Frösen 2004, Kurki 2011]. Saccular P1/2 junction aneurysms represent the second largest group among ruptured PCA aneurysms. Turbulent blood flow resulting from two inflow zones and only one outflow may play a role for the formation of these aneurysms, as flow separation and rupture rates are comparable to ACoA and MCA bif aneurysms [Dashti 2007, Hernesniemi 2008].

Size of PCA Aneurysms

Most PCA aneurysms (n=77, 75%) were smaller than 7 mm even when ruptured. Ruptured aneurysms were only slightly larger on average than

those unruptured. In our series, we saw only nine large and seven giant aneurysms, representing 12% of all the PCA aneurysms studied. PCA aneurysms are generally no larger than intracranial aneurysms at other locations [Dashti 2007, Drake 1996, Lehecka 2008, Taylor 2003, Yasargil 1984, Yonekawa 2011]. In our opinion, a different admission policy led to an overrepresentation of large and complex cases compared to previous series.

Fusiform PCA Aneurysms

The proportion of fusiform PCA aneurysms in our series is high at 24%, but falls inside the 19–40% range previously reported [Ciceri 2001, Drake 1996, Hallacq 2002, Taylor 2003]. However, the occurrence rate is very high compared to fusiform anterior circulation aneurysms, where only around 1% of aneurysms had a fusiform shape [Dashti 2007, de Sousa 1999, Lehecka 2008, Pritz 2011, Steven 2007]. The low-flow distal PCA segment in particular shows more fusiform aneurysms than saccular compared to segments with higher blood flow. In general, blood flow velocity in the PCA is much lower than in the anterior circulation [Hennerici 1987]. One could speculate that the anatomical proximity of the PCA to skull structures imparts a predisposition for fusiform aneurysm formation following a skull injury. The P2 segment in particular, which is in close relation to the tentorial edge, was the segment most often affected with fusiform aneurysms.

Anatomical Relationships of PCA Aneurysms

Saccular PCA aneurysms present a characteristic dome orientation at each PCA segment. Their orientation is often in line with the vascular flow or in the direction of the originating branches. Therefore, PCA aneurysms often exhibit a dome orientation toward the surgical view. Additionally, the location of proximal PCA aneurysms is typically relatively high, above the posterior clinoid level. The typical dome projections and positioning with

respect to the skull base are important factors for consideration when planning microneurosurgical approaches and treatment strategies for these rare lesions.

AVM and Multiple Aneurysm Association

Our findings confirm previous reports, which suggest that PCA aneurysms are often associated with arterio-venous malformations (10%) and multiple intracranial aneurysms (55%). This association is particularly influential when planning occlusive therapy. Usually, patients are clinically symptomatic by the AVM. In the case of AVM associated aneurysms, the relevant literature reports shrinkage or disappearance of flow-related aneurysms after complete AVM obliteration [Meisel 2000, Redekop 1998]. Another interesting finding is that all of the AVM related aneurysms, located at dilated and higher flow feeders of AVMs, were saccular.

6.2 Treatment Strategies for PCA Aneurysms

Historical Aspects

Our own historical series reviewed 12 patients treated between 1954 and 1980. These patients all received conservative treatment and demonstrated a high rate of unfavorable outcomes (50%). A case report given by Bertrams in 1968 regarding a patient with a fatal outcome following a giant distal PCA aneurysm rupture reflects our impression of the historic patient records [Bertrams 1968]. Around that point in time the treatment of PCA aneurysms changed from conservative measures to a mostly active microsurgical treatment [Drake 1969, Hunt 1967]. In addition, endovascular techniques have evolved rapidly during the last three decades. As a result, PCA aneurysms closely related to the brainstem are now treated by endovascular techniques at many institutions [Ciceri 2001, Hallacq 2002, van Rooij 2006].

Clinical Presentation

With regard to SAH severity, the overall H&H grade was similar for proximal PCA aneurysms and distal PCA aneurysms. However, five of the nine ruptured

P3 aneurysms led to an IVH or ICH, highlighting the danger of distal PCA aneurysms. Our series showed a lower frequency of cranial nerve disturbances compared to Drake's series on PCA aneurysms (6% vs. 21%). One possible explanation for this difference is the lower rate of large and giant aneurysms in our study (12% vs. 21%) [Drake 1996].

Treatment Strategies for Saccular PCA Aneurysms

Microsurgery and interventional techniques allow for easy access to proximal saccular PCA aneurysms. We have treated 15 patients presenting 15 saccular PCA aneurysms with endovascular techniques, but the primary aneurysm occlusion rate was low at 73%, compared to 89% in the microsurgical group. The relevant literature reports that a complete closure of the aneurysm by endovascular techniques often requires a parent vessel occlusion [Ciceri 2001, Cotroneo 2007, Hallacq 2002]. Most cases of the actual series were treated by microsurgical means, which allows for direct visual control of the important perforating branches and the occlusion of the aneurysm neck. However, the most frequent complications were more dependent on the chosen strategy rather than the treatment approach. In general, quick aneurysm treatment following SAH is recommended [Fogelholm 1993, Peerless 1994]

Treatment Strategies for Fusiform PCA Aneurysms

A fusiform shape is common for PCA aneurysms. The direct microsurgical and endovascular treatment of these aneurysms is aggravated by the origination of perforating branches and the aneurysm shape itself. Direct clip occlusion was possible in only 7 of the 29 fusiform aneurysms (29%), and 2 of these were only partially occluded. Proximal occlusion must be done with great care to prevent devastating PCA infarction. This treatment strategy can only be recommended as an "ultima ratio". In our opinion reconstructive procedures should be discussed for these cases. Despite the risks, a balloon occlusion test can provide helpful information [Chang 2010, Jayakumar 2004, Lawton 1996, Mathis 1995, Originato 1994].

Need for Revascularization

According to the 125 PCA aneurysm series of Drake et al. (1996), it seems that parent vessel occlusion is a safe treatment option for aneurysms distal to the thalamoperforating branches thanks to the collateral supply potential from the anterior circulation by the anterior/posterior choroidal artery and the pericallosal arteries [Drake 1996]. However, we must be more critical of this assumption based on the results of the present series. The most compromising factor on patient outcome was the appearance of PCA infarctions, particularly when proximal occlusion or trapping was the chosen treatment strategy. While causation is not entirely clear, the main difference between our series and Drake's is that we treated mostly non-giant and non-thrombosed PCA aneurysms where collateral circulation had not yet developed prior to treatment.

Revascularization procedures may often be required for successful treatment of non-saccular or complex aneurysms that cannot be treated by direct clipping. Chang et al. from the Barrow Institute recommended a practical treatment algorithm in 2010 following a series of 33 patients with distal PCA aneurysms. In Chang's study bypass procedures were considered helpful in 14 cases but also observed to be associated with specific complications like bypass failure and epidural hematomas.

Microsurgical Approaches to PCA Aneurysms

When choosing the microsurgical approach for intracranial aneurysm surgery the aneurysm configuration, parent vessel course, and exact location in relation to skull base structures and basal cisterns are particularly important [Seoane 1997, Terasaka 2000]. Frontotemporal and subtemporal routes are the most common approaches for PCA aneurysms [Drake 1996, Seoane 1997, Taylor 2003, Terasaka 2000, Yonekawa 2011]. Regardless of the selected approach, the main challenges are:

- avoidance of brain tissue injury from retraction,
- prevention of injury to the venous system (Sylvian veins, the vein of Labbé),

- prevention of brainstem perforator injury,
- securing sufficient perfusion of the PCA territory with prevention of PCA infarction.

Frontolateral Approaches

Segmental allocation is especially important when selecting the surgical approach [Seoane 1997]. To gain microsurgical access to proximal PCA aneurysms (P1 and P2), the following frontolateral approaches are well described: orbitozygomatic approach, anterior temporal approach, pterional and subtemporal approaches [Drake 1996, Gerber 1993, Türe 2003, Yasargil 1984, Yonekawa 2011]. Using these approaches, the proximal sylvian fissure must be opened to obtain enough space for the aneurysm dissection. However, the surgical corridor is deep, narrow, and in close proximity to very sensitive neurovascular structures. Sometimes a selective amygdalohippocampectomy is necessary to obtain enough space [Yonekawa 2011]. The removal of anterior or posterior clinoid can be required to improve the area of binocular vision [Yonekawa 1997/2011]. To approach high-lying P1 segment aneurysms, the transsylvian route is effective. The exposure of the aneurysm neck can be somewhat more difficult from a pterional view point compared with the subtemporal view as aneurysms of the P1 segment and P1/2 junction mainly project in the cranial or anterior direction.

Subtemporal Approach

Drake introduced the subtemporal approach in 1961 for the treatment of basilar aneurysms. This method was later adopted for PCA aneurysms and represents the most direct and efficient route to the PCA [Drake 1961/1996]. The subtemporal approach is relatively simple and fast, avoids extensive bone removal, and therefore belongs in the scope of standard neurosurgical approaches. It also offers good visualization of the interpeduncular and ambient cistern. PCA aneurysms in proximity to the tentorium are well-approachable from a subtemporal route [Hernesniemi 2005, McLaughlin 2014, Terasaka 2000, Uygur 2007, Zador 2010]. The course of the P2 segment, parallel to the tentorium, allows for proximal and distal parent vessel control. How-

ever, far distal PCA aneurysms and aneurysms high above the posterior clinoid process (>3 mm) cannot be reached [Drake 1996, Seoane 1997, Yonekawa 2011].

The main drawback of the subtemporal approach is the required elevation of the temporal lobe and potential problems associated with this movement. This risk is unavoidable, as execution of this method without temporal lobe retraction is not possible. To keep the retraction as minimal as possible, prior release of CSF via spinal drain should provide sufficient room for reaching the tentorial edge and the interpeduncular cistern safely for additional CSF release. A severe brain edema or intracerebral hematoma can make the application of the subtemporal impossible. We have observed related complications in our own series; fortunately only two patients suffered from a temporal lobe contusion, but both also had acute SAH.

After sufficient CSF release the subtemporal approach provides sufficient working space, even for complex microsurgical treatments such as bypass-bridging techniques, trapping, aneurysmorrhaphy, and *in situ* anastomoses between the SCA and PCA [Sundt 1982, Terasaka 2000, Zador 2010]. Additional aneurysms lying on the ipsilateral proximal SCA, PCoA, or the tip of the basilar artery can be treated in the same session using the subtemporal approach.

Posterior Approaches

It is usually not possible to directly approach far distal PCA aneurysms (P3 and P4) using the frontolateral or subtemporal approach. Only when proximal occlusion is deemed necessary are these approaches feasible; the direct treatment of P3 and P4 aneurysms require different approaches. According to Yasargil (1984) the posterior interhemispheric approach is suitable to reach the terminal PCA (P4). To approach deeper locations around the posterior surface of the midbrain, Yonekawa et al. reports that the supracerebellar transtentorial approach in the sitting position is applicable [Touho 1995, Yasargil 1984, Yonekawa 2011].

Treatment Strategies for PCA Aneurysms in Association with Multiple Aneurysms

The treatment of patients with multiple aneurysms is challenging. Regardless, the general rules of aneurysm treatment must be observed for PCA aneurysms:

- the first intervention is focused on the aneurysm most likely to rupture to prevent re-bleeding;
- the most technically secure method is used;
- all reachable aneurysms are occluded during the same procedure, if possible;
- other treatable aneurysms are approached after the acute phase;
- aneurysms without indication for active treatment are observed.

Using these strategies, we were able to manage 54 patients with multiple intracranial aneurysms. Fortunately, frontotemporal craniotomies are favorable for the management of most ipsilateral anterior circulation aneurysms and endovascular techniques allow the occlusion of several aneurysms during a single intervention [Shen 2014, Yasargil 1984].

Treatment Strategies for PCA Aneurysms in Association with AVM

We addressed only three aneurysms of AVM feeding vessels by direct microsurgical treatment. In the case of AVM associated aneurysms, the literature reports shrinkage or disappearance of flow-related aneurysms after complete AVM obliteration [Meisel 2000, Redekop 1998]. The low rupture rate during follow-up supports this decision history. However, longer follow-up periods are necessary. The decision-making process for each of these individual cases is very complex in general.

6.3 Complications

Parent Vessel Occlusion

The occurrence of ischemic infarctions following parent vessel occlusion is one of the most serious

and outcome-affecting complications following PCA aneurysm treatment. Contrary to general opinion and results of comparable studies [Drake 1997, Hallacq 2002], parent artery occlusion is not a safe treatment option for distal PCA aneurysms and can only be recommended as an "ultima ratio" especially in the case of fetal-type PCA [Xu 2015].

Perforator Infarction

The main objective of the treatment of PCA aneurysms is preventing injury to the brainstem and midbrain infarctions. Open microsurgery has the advantage of direct visual control compared to endovascular treatment. ICG videoangiography, as a new intraoperative technique introduced to micro-neurosurgery in 2003, provides real-time images of arterial and venous blood flow and allows an immediate evaluation of the perforating branches. We found only one patient with a thalamic infarction and one patient with a partial temporal lobe infarction.

3rd and 4th Nerves

The proximity of the 3rd and 4th nerves to the surgical field is a frequent cause of problems. Oculomotor nerve irritation is the second most common microsurgical complication in treatment of upper posterior circulation aneurysms, irrespective of the chosen approach [Drake 1996, Horikoshi 1999]. The paresis can be caused by direct mechanical irritation as well as vascular impairment of the oculomotor nuclei or fascicle. A temporary paresis can occur even after only minimal manipulation during dissection. Frontotemporal approaches allow a trajectory that is perhaps slightly more favorable for the protection of the 3rd nerve than the subtemporal approach, but oculomotor paresis is possible in this case as well. The paresis usually regresses after a short period (a few weeks to a few months). Clip application is another situation where oculomotor nerve injury is possible; careful clip verification is of paramount importance.

Trochlear nerve injury is uncommon but possible. The risk of such injury is much higher when using the subtemporal approach compared to the pterional approach, due to the different surgical

angle. Using a subtemporal approach, the 4th nerve runs directly across the surgical field in a nearly horizontal fashion and is much thinner and more sensitive than the 3rd nerve. It is possible to accidentally injure the 4th nerve with an instrument shaft, especially during dissection under very high magnification when the 4th nerve is out of focus or surgical view. Protection of the 4th nerve during surgery is only possible by being aware of its exact position during all steps, especially in the case of SAH.

Direct visual control in combination with intraoperative monitoring of the 3rd and 4th nerves can prevent permanent damage [Zhou 2012].

6.4 Future Aspects of PCA Aneurysm Treatment

Modern vascular imaging will allow for less invasive methods and provide more detailed information about the aneurysm, wall configuration, vascular reserve, and collateral supply. This information will make it easier to decide on the need for revascularization and plan the treatment strategy as a whole. With the progressive development of materials science, new endovascular devices are being made available to our endovascular units. In particular, well-configured saccular aneurysms will be treated mostly by endovascular means. Microsurgical revascularization will gain importance in the treatment of complex lesions. Hybrid procedures, with proximal endovascular occlusion and distal microsurgical flow augmentation, will also be increasingly applied. Clip occlusion will always be available for definitive microsurgical treatment of saccular aneurysms.

7. Conclusion

I

PCA aneurysms are rare lesions that are often associated with other vascular pathologies such as multiple aneurysms and AVM. Most PCA aneurysms are smaller than 10 mm, even when ruptured. Distal PCA aneurysms are more often ruptured than proximal PCA aneurysms. The incidence of fusiform PCA aneurysms is high at 26% and the P2 is the segment most often affected by fusiform PCA aneurysms. Saccular PCA aneurysms typically have a dome orientation in relation to the originating PCA segment.

II

Aneurysms of the PCA are infrequent and often associated with other vascular pathologies. As a result, individual treatment strategies are required. Both microsurgery and endovascular treatment are effective options for the occlusion of PCA aneurysms. Despite commonly adequate vessel collateralization of the distal PCA territory, preservation or reconstruction of the parent vessel is crucial

for favorable treatment outcomes. The rate of intraoperative third nerve injuries shows that solely visual control is insufficient and intraoperative monitoring is recommended.

III

The subtemporal approach allows access to the proximal PCA segments as well as the P2 and anterior P3 segment. In these cases, CSF release is necessary to prevent temporal lobe injuries. Additionally, the subtemporal approach can provide enough space for revascularization procedures. The most frequently observed complications were not related to the approach method.

IV

The lateral supraorbital approach is less invasive than a frontotemporal approach and is suitable for the simultaneous microsurgical treatment of proximal anterior circulation and ipsilateral proximal PCA aneurysms.

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