Aneurysms of the vertebral and posterior inferior cerebellar arteries

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Academic Dissertation

To be publicly discussed with the permission
of the Faculty of Medicine of the University of Helsinki,
in Lecture Hall 1 of Töölö Hospital
on April 17th 2015, at 12 noon.

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ISBN 978-951-51-0901-9 (paperback)
ISBN 978-951-51-0902-6 (PDF)
http://ethesis.helsinki.fi
Unigrafia, Helsinki, 2015

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II Lehto H, Harati A, Niemelä M, Dashti R, Laakso A, Elsharkawy A, Satopää J, Billon-Grand R, Canato B, Kivisaari R, Hernesniemi J.: *Distal posterior inferior cerebellar artery aneurysms: Clinical features and outcome of 80 patients.* World Neurosurgery, 2014 82(5):702 – 713

III Lehto H, Niemelä M, Kivisaari R, Laakso A, Jahromi BR, Hijazy F, Andrade H, Dashti R, Hernesniemi J: *Intracranial vertebral artery aneurysms: Clinical features and outcome of 190 patients* (World Neurosurgery, accepted)

Abbreviations

ACA..... Anterior cerebral artery
AICA ..... Anterior inferior cerebellar artery
AcomA .. Anterior communicating artery
AVM..... Arteriovenous malformation
AVF ..... Ateriovenous fistula
BA ..... Basilar artery
CCA ..... Common carotid artery
CI ..... Confidence interval
CN..... Cranial nerve
CSF ..... Cerebrospinal fluid
CT ..... Computed tomography
CTA..... Computed tomography angiography
DSA ..... Digital subtraction angiography
GOS..... Glasgow outcome scale
H&H ..... Hunt and Hess grade
ICA..... Internal carotid artery
ICH ..... Intracerebral hemorrhage
ISUIA .... International Study of Unruptured Intracranial Aneurysms
IVH ..... Intraventricular hemorrhage
MCA ..... Middle cerebral artery
MRA ..... Magnetic resonance angiography
MRI..... Magnetic resonance imaging
NA..... Not available
OA..... Occipital artery
OAG ..... Occipital artery graft
OR..... Odds ratio
PCA..... Posterior cerebral artery
PCKD..... Polycystic kidney disease
PEG ..... Percutaneous endoscopic gastrostomy
PComA .. Posterior communicating artery
PICA ..... Posterior inferior cerebellar artery
RAG ..... Radial artery graft
SAH ..... Subarachnoid hemorrhage
SCA..... Superior cerebellar artery
STA ..... Superficial temporal artery
UCAS..... Unruptured Cerebral Aneurysm Study of Japan
VA..... Vertebral artery
VBJ ..... Vertebro-basilar junction

Abstract

Objective: Aneurysms of the vertebral artery (VA) and its branch posterior inferior cerebellar artery (PICA) are rare, comprising only about 1 to 3% of all intracranial aneurysms. The series published thus far on these lesions are small. We aim to describe the special anatomical and morphological features of these aneurysms compared to aneurysms in other locations, and to describe the variety of symptoms they cause. We describe their treatment and analyze the outcome. Additionally, we describe their anatomy imaged with computed tomography angiography.

Patients and methods: We reviewed retrospectively 9709 consecutive patients with intracranial aneurysms treated in the Department of Neurosurgery at Helsinki University Central Hospital, Finland, between 1934 and 2011. The study population included 268 patients with 284 VA or PICA aneurysms or both. Follow-up data came from the Population Registry Centre (dates of death), Statistics Finland (causes of death), from written questionnaires to patients still alive, medical records of the Department of Neurosurgery, and for those deceased, medical records from all public health services.

Results: Among all the aneurysm patients, 5.1% had an aneurysm in the VA or PICA. Most aneurysms, 51%, were located at the VA–PICA junction. The proportion of fusiform aneurysms was 28%. Compared to patients with ruptured aneurysms at other locations, patients with a ruptured VA or PICA aneurysm were older and had a higher Fisher grade. Ruptured distal PICA aneurysms also re-bled more regularly. Compared to other ruptured aneurysms, ruptured VA and PICA aneurysms were smaller and more often fusiform. At least one VA or PICA aneurysm was treated in 209 (78%) patients. The most common technique for aneurysm occlusion was clipping, used in 107 aneurysms. Total occlusion of the aneurysm was achieved among saccular aneurysms in 90%, and among fusiform aneurysms in 61%. Within one year of aneurysm diagnosis, 26% of the patients were dead. Among those who survived a minimum one year and in whom the VA or PICA aneurysm received active treat-

ment; those returning to an independent or their previous stage of life amounted to 92%.

Conclusion: In treatment of VA and PICA aneurysms, their special anatomical and morphological features are challenge. Despite this, and often severe hemorrhage, most patients surviving the initial stage make a good recovery. ❦

Abstract

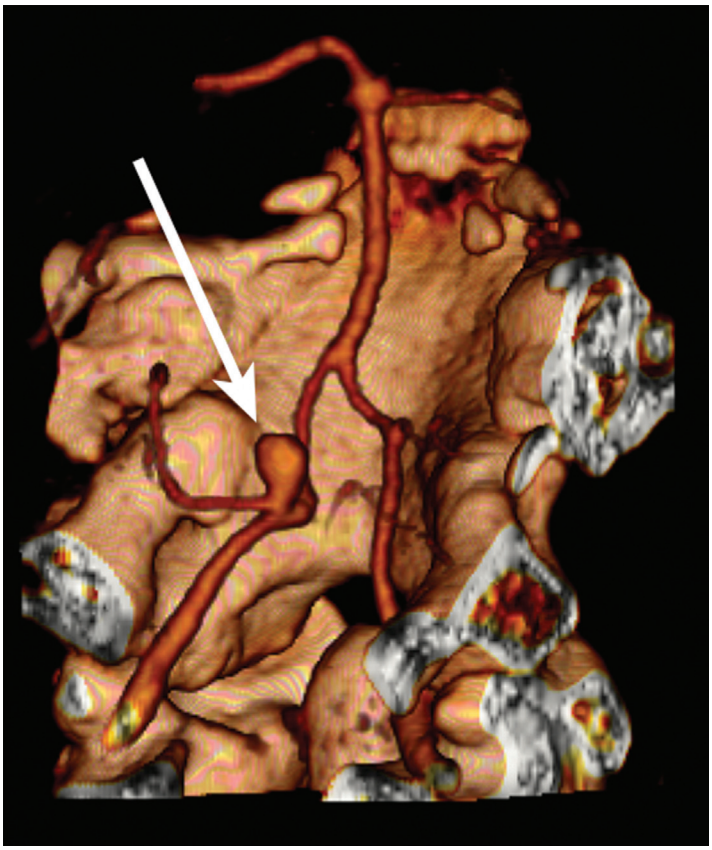
Introduction

Intracranial aneurysms are acquired dilatations of cerebral arteries. Mostly they are saccular, pouch- like, and located at the branching point of an artery close to the skull base. Their prevalence in general population is 2 to 3% (232, 235).

Almost all unruptured aneurysms are asymptomatic. When ruptured, they cause a subarachnoid hemorrhage (SAH), which manifests as sudden severe headache, nausea, and often also unconsciousness. The overall mortality from this disease is high, around 40% (158, 220). For previously diagnosed unruptured aneurysms, the annual rupture rate is around 1% (96). Risk factors for aneurysm rupture include smoking, female gender, and aneurysm size over 7 mm (111). Diagnosis of bleeding is made by CT or lumbal puncture; diagnosis of the aneurysm mostly by CTA or DSA.

The posterior circulation consists of vertebral arteries, the basilar artery, and their branches. The vertebral arteries are paired and originate from the subclavian arteries. They ascend to traverse the skull through the foramen magnum, and join each other close to the pontomedullary junction to form the basilar artery. The posterior circulation comprises around 10% of all intracranial aneurysms (252). Compared to aneurysms in the anterior circulation, risk for rupture is also higher (244). Microsurgically, they are more difficult to access, and nowadays endovascular treatment is the preference in most centers.

Figure 1
A typical PICA aneurysm



Special features of aneurysms arising from the vertebral artery, or from its branch posterior inferior cerebellar artery, are a high proportion of fusiform and dissecting aneurysms, and many times a close relationship to cranial nerves and to the brainstem. The course of the vertebral artery can be tortuous, and additionally, the course of PICA is the most variable among cerebral arteries. Figure 1.1 shows a typical PICA aneurysm.

In addition to the series of Drake, Peerless, and Hernesniemi in 1966, other series on VA aneurysms are small. In this retrospective study, we present 268 patients with 288 vertebral artery and PICA aneurysms. We aim to report their incidence, features compared to aneurysms at other locations, treatment, and outcome. Most aneurysms in this series were treated by microsurgery.

Review of the literature

Cerebral artery aneurysms and subarachnoid hemorrhage

Incidence and prevalence

Overall prevalence of aneurysms in the general population is 2 to 3% (192, 232, 235). Overall, the incidence of aneurysmal subarachnoid hemorrhage is around 9/100 000 per year (43). The mean age of SAH patients has increased from 52 in the 1970s to 62 in 2000 (158).

Diagnostics

Subarachnoid hemorrhage is practically always detectable on CT for a minimum of six hours after aneurysm rupture (16, 181). If needed, the diagnosis can be verified by lumbar puncture, which was also the method of diagnosis before the era of the CT scans. In aneurysm diagnostics, DSA has been the gold standard until recently but is now challenged by CTA (28, 238). Compared to DSA, CTA is less invasive, faster, cheaper, and has better availability; it makes reconstruction images is easier, and it visualizes better the bony structures in addition to the blood vessels (172, 233).

Risk factors

The reported risk factors for aneurysm formation and growth are female gender and smoking (97). Risk factors for SAH include female gender, current smoking, aneurysm size over 7 mm, hypertension, excessive alcohol intake, and patient age inversely (52, 96, 111). Aneurysm growth during follow-up is associated with rupture (97).

Morphology and etiology

Morphologically, aneurysms are mostly divided into two categories: saccular and fusiform (non-saccular). By far most intracranial aneurysms are saccular. i.e. sac-like pouches mostly located in the bifurcation of an artery. Fusiform aneurysms are spindle-shaped dilatations of an artery without clearly identifiable necks; their exact proportion is not known but has been estimated as less than 1% (12).

Additionally, some authors further add to the categorization dissecting, dolichoectatic, serpentine, and atherosclerotic aneurysms. Dissecting aneurysms are morphologically mostly fusiform; a dissection of the vessel wall has led to its outward bulging. Dolichoectasia is dilatation, elongation, and tortuosity

of an artery, and when such an aneurysm contains thrombosis, it is called a serpentine aneurysm. Atherosclerotic aneurysms are morphologically fusiform, but show atherosclerosis in their walls.

All these terms overlap. The rare saccular-shaped aneurysms located outside branching points of an artery are occasionally classified as a separate group (148). To distinguish a non-saccular aneurysm from a normal vessel, Flemming et al., based on radiographic data, proposed dilatations greater than 1.5 times the normal vessel lumen to be called aneurysms (57). Lately, Sacho et al. used the same definition for a fusiform aneurysm (200). To differentiate small fusiform aneurysms from giant dolichoectatic ones, Flemming et al. further classified the non-saccular aneurysms into fusiform, dolichoectatic, and transitional groups: fusiform aneurysms they defined as dilatation of a portion of an artery, dolichoectatic as uniform dilatation of the whole artery or several arteries, and transitional aneurysms as dilatation of one or several arteries with dilatation superimposed on one segment (56). Additionally, fusiform aneurysms are occasionally divided into dissecting (acute) and chronic subgroups, with dissecting aneurysms showing a classical "pearl-and-string" sign or double lumen in angiography and the chronic showing only a dilatation of the vessel (156).

Histology

Compared to the normal wall of a cerebral artery, the wall of a saccular aneurysm lacks elastic lamina and carries different degrees of degenerative changes and inflammatory reactions (58, 106). The unruptured aneurysms' wall have myointimal hyperplasia and an organized thrombi, whereas the wall matrix of a ruptured aneurysm is decellularized and degenerated, there is ongoing inflammation, and lipid accumulation is visible throughout the vessel wall (59, 60). The degeneration may be related to impaired function of the endothelium and high oxidative stress, partly caused by intraluminal thrombosis (60).

Similarly, fusiform aneurysms carry a defect in elastic lamina; in dissecting aneurysms, the breakage is believed to be acute, leading to intramural hemorrhage (49). In the chronic stage of fusiform aneurysms, beside disruption of the elastic lamina and intramural hemorrhage, neovascularization occurs in the thickened intima and in the intramural thrombus formed (156). The newly formed vessels also cause repetitive new hemorrhages within the vessel wall (156).

Genetics

A few, rare genetically inheritable diseases such as autosomal dominant polycystic kidney disease may carry a higher incidence of intracranial aneurysms than in a normal population (27). In general, studies on the genetics of patients with intracranial aneurysms, show risk loci to be the same as in cardiovascular diseases (62), and SAH is primarily of non-genetic origin (113).

Treatment

Acute treatment of aneurysmal SAH consists of aneurysm occlusion to prevent rebleeding, prevention of delayed cerebral ischemia, standard medical management, and treatment of possible complications like infections and hydrocephalus.

If the aneurysm is not occluded, a rebleeding occurs within the first 24 hours in about 12% of all patients; most bleedings occurring within six hours after the first ictus (219). After this, the risk remains at 1 to 2% per day for next two weeks. After a month, the risk decreases, but it remains about 3% per year. Without aneurysm occlusion, mortality in SAH within a week is 40 to 45%, within a month 50 to 60%, within a year 65%, and within 5 years 65 to 70 % (171). Because of the risk for rebleeding, an-

eurysm treatment is recommended within first the 24 hours and at the latest within 72 hours after the first symptoms (41, 165, 220). Antifibrinolytic drugs (tranexamic acid) reduce risk for rebleeding before aneurysm treatment, but have no influence on poor outcome or mortality (75, 193).

The aneurysm is occluded either by microsurgical or endovascular means. Microneurosurgery consists mainly of clipping of the aneurysm, but in rare cases involves trapping or proximal occlusion with or without bypass. Endovascular surgery consists mainly of coil embolization of the aneurysm; in more complicated aneurysms, recent development has led to use of balloon- or stent-assisted embolization and flow-diverters.

Of patients surviving the initial hemorrhage, cerebral ischemia occurs in about 30%, mostly between days 4 and 10 (194). Calcium antagonist (nimodipine) reduces both occurrence of the secondary ischemia and risk for poor outcome (198). One recent study on preventive use of simvastatin showed no benefit (108).

The figures on incidence of acute and chronic hydrocephalus after subarachnoid hemorrhage are variable: shunt-dependent hydrocephalus occurs in roughly 10% of SAH patients (42). In a recent report, 33% of the patients later needed a shunt, with the predictive factors being poor clinical grade on admission, severe SAH, large ventricular size before the aneurysm occlusion, and large amount of CSF drained within the first week after bleeding (50).

Outcome

Despite treatment, the overall mortality of the patients with aneurysmal SAH is high, around 40% (158, 220). In a review of 2 424 patients with outcome assessment ranging from 1 to 12 months after SAH, 55% of the patients were independent, and 19% needed help in their daily activities (158). Compared to the 1970s, in 1997, Hop et al. found a 15% decrease in case-fatality despite the higher mean age of SAH patients more recently. Additionally, over the years, the proportion of patients who remain independent has increased by 1.5% per year (77).

In addition, those surviving beyond one year after the bleeding have still excess mortality, mainly due to the higher risk of death especially in vascular events (83, 112, 243). A risk of recurrent SAH is within the first 10 years is 3.2%, which is 22 times higher than the expected risk of SAH in general population (242). The likelihood of de novo aneurysm formation is 0.8% per year (97).

Preventive treatment

Less than one-third of all unruptured aneurysms ever rupture, and identification of those at risk is currently impossible. Because of the poor prognosis of aneurysmal SAH patients, prophylactic treatment of unruptured aneurysms is considered in each case. The ongoing discussion conserns which aneurysms and in which patients require treatment, and whether the aneurysm should be treated by microsurgical or endovascular means. Discussion is mainly focused on the International Study of Unruptured Intracranial Aneurysms (ISUIA) published in 2003 in the Lancet (244). This study includes 4 060 patients either being followed up or treated by surgery or endovascular procedures. Regardless of the high number of patients recruited, it has received much criticism for selection bias, short follow-up, including posterior communicating artery aneurysms in the posterior circulation, and other matters.

More recently, two other larger studies have influenced this field: The Unruptured Cerebral Aneurysm Study of Japan (UCAS) on the natural history of unruptured aneurysms, with 6 697 aneurysms studied with a follow-up of 11 600 aneurysm-years, and development of the PHASES score, a score aimed to predict aneurysm's rupture risk.

(63, 229) PHASES is based on systematic review of six earlier studies, an analysis including 8 283 patients with a follow-up of 29 166 person-years (63). The study on the natural history of aneurysms with the most extensive follow-up (median 21 years) and risk-factor analysis comes from Finland (96, 111).

Beside occlusion of the aneurysm, essential in preventive treatment are cessation of smoking and treatment of hypertension (111).

Posterior circulation aneurysms

Definition

Posterior circulation aneurysms consist of aneurysms located in the posterior cerebral artery, basilar artery, superior cerebellar artery (SCA), anterior inferior cerebellar artery (AICA), vertebral artery, and the posterior inferior cerebellar artery (PICA).

Incidence

In the series of Yasargil, posterior circulation aneurysms accounted for 10% (252). In a widely cited study from 1966, among SAH patients with one aneurysm only, posterior circulation aneurysms accounted for 5% (137). In a recent series on saccular aneurysms, those located in the posterior circulation accounted for 9% (83).

Location

By far the most common location for the posterior circulation aneurysm is the basilar bifurcation (47, 82, 204, 233). In the series of Drake et al., basilar artery aneurysms counted 51% (47).

Special features compared to aneurysms in the anterior circulation

Classically, non-saccular aneurysms have been thought to occur more frequently in the posterior circulation (12). In the book of Drake et al., of the posterior circulation aneurysms, non-saccular aneurysms accounted for 225 (10%) (47).

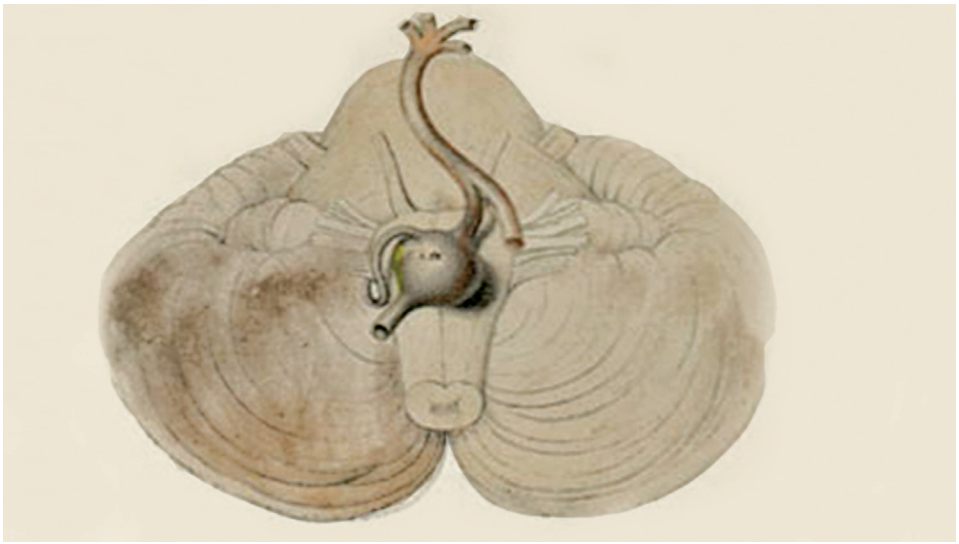
Compared to anterior circulation aneurysms, one theory is that posterior circulation aneurysms have a poorer natural course. In ISUIA, the rupture rates for posterior circulation aneurysms ranged from 2.5% of those < 7 mm to 50% of giant aneurysms, compared to 0% to 40% for the same size-groups in the anterior circulation (244). That study, however, grouped PComA aneurysms as part of the posterior circulation aneurysms. In the UCAS study, with the exception of large aneurysms, those located in the posterior circulation were not more prone to rupture than were aneurysms in the anterior circulation (229). Both the UCAS and ISUIA studies struggle, however, with rather short median follow-up times. Besides their possibly worse natural course, in SAH patients, location of a ruptured aneurysm in the posterior circulation has been associated with poorer prognosis (195, 209).

Anatomically, posterior circulation aneurysms are located closer to the brainstem and most of the cranial nerves, which challenges their surgical treatment. When anterior circulation aneurysms are treated via pterional or lateral supraorbital craniotomy, or in the case of a pericallosal aneurysm, via an interhemispheric approach (38, 71, 127, 128), the complex anatomy of posterior circulation aneurysms demand a much wider range of craniotomies (226). Approaches used include far-lateral (73), suboccipital (47), pterional (252), subtemporal (47), transpetrosal (103) approaches and their variations. Treatment of unruptured posterior circulation aneurysms is also more prone to complications: based on ISUIA, in non-giant anterior circulation aneurysms, mortality and morbidity is 1% and 2%, in non-giant posterior circulation aneurysms 3% and 13%, in giant anterior circulation aneurysms 7% and 27%, and in giant posterior circulation aneurysms 10% and 38% (244).

History of treatment

"I know of no successful outcome from oper-

Figure 2
The first known image of a VA aneurysm from 1829.



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ative attack upon an aneurysm of the posterior cranial fossa, but for those upon the vertebral and posterior inferior cerebellar arteries, which afford good exposure, cures will certainly come in time" (Walter Dandy 1944) (37)

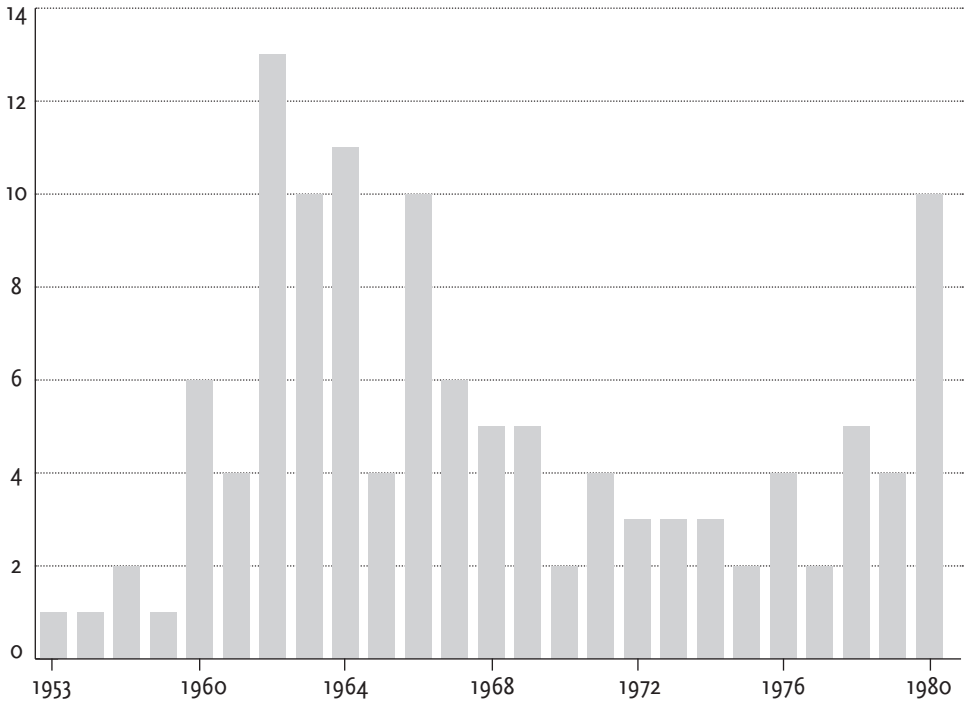
Worldwide

The first posterior circulation aneurysm (basilar, autopsy finding) was descried by Blackall in 1813. The first illustration preserved of a VA aneurysm is the one by Cruvilhier in 1829 (Fig. 2.21).

The first VA angiography, done via injection to a surgically exposed subclavian artery, was reported by Moniz in 1933 (149, 150). Hugo Krauenbühl was the first to make the first posterior circulation aneurysm diagnosis in an angiography in 1941; he used the same method as Moniz (114). For many years, vertebral angiographies were done through a direct puncture in the neck. After development of brachial and femoral cannulation, vertebral angiographies become more routine. Nowadays, the complication rate has been less than 0.5% (53).

The first operations for posterior circulation aneurysms took place for presumed tumors; the first was likely done by Cushing in 1915. The first ligation of the cervical VA due to an intracranial aneurysm was by Dandy in 1928 (37). The first successful direct treatment was reported by Schwartz on November 6th, 1946: he trapped a ruptured aneurysm of a an unnamed branch of the basilar artery (210). The next successful surgery took place on January 23rd, 1947: Rizzoli et al. trapped and excised a PICA aneurysm (189). The first reported operation after aneurysm diagnosis by angiography was in 1956 by DeSaussure et al. (44), when they trapped a PICA aneurysm. Drake, for his part, operated on his first posterior circulation aneurysm in 1959; the patient with a ruptured mid-basilar aneurysm clipped

Figure 2.21
Patients with posterior circulation aneurysms diagnosed in Helsinki before 1980



via a subtemporal approach made a good recovery (47). According to Drake et al., before 1960, 25 patients underwent surgery for a ruptured basilar or vertebral artery aneurysm; among them 10 were treated with proximal vertebral artery occlusion. Among these 25 patients, 4 died, but 16 suffered no adverse effect from the surgery (47).

Besides exovascular techniques, development of endovascular treatment is an important part of the history of treating cerebral aneurysms. Especially it involves posterior circulation aneurysms, as most are nowadays treated by endovascular means. In 1964, Luessenhop and Velasquez occluded a carotid aneurysm by endovascularly placing a silicone balloon into its neck (138); this method was later developed by Serbinenko (213). Besides balloons, aneurysms were filled with an iron-particle suspension (Alksne, in 1969) and with isobytyl-2-cyanoacrylate (Sheptak, 1977), but the operations were done via a craniotomy or a burr hole (8). Additionally, Mullan et al. in 1965 described an electrically induced thrombosis of an aneurysm: they punctured an aneurysm though a craniotomy or a burr hole, positioning a wire in the aneurysm and inducing a current into it in order to produce thrombosis (153). Unfortunately, among the six aneurysms with angiographic follow-up, one aneurysm showed 10% filling at two weeks; the others were filling a minimum of 40%. In their report in 1974, they used the method also for basilar aneurysms (154). In 1990, Dowd et. al. reported endovascular embolization of a PICA aneurysm with platinum coils (45). Finally,

in 1991, Guglielmi reported his initial experience with Guglielmi detachable coils. His invention allowed separation of the coil from the guide wire with an electric current at will, which helps to control the coiling. The technique was further developed by Moret, who introduced a "balloon-remodeling technique" for wide-necked aneurysms reported in 1997 (151). Also in 1997, Higashida et al. published on treatment of a fusiform aneurysm of the basilar artery with coiling through a stent (74). Later, in 2008, Fiorella et al. published their first experience in using a pipeline embolization device (55).

Helsinki

Before 1980, 119 patients were diagnosed with a posterior circulation aneurysm (Figure 2.21). The first diagnosis was made in 1953: a ruptured basilar bifurcation aneurysm was diagnosed at autopsy in a 57-year-old woman. The first posterior circulation aneurysm diagnosis based on angiography took place in 1958, when a PCA aneurysm was diagnosed in the carotid angiography; this patient refused surgery. In vertebral angiography, a posterior circulation aneurysm was found for the first time in 1960; the aneurysm was located in the basilar artery. This aneurysm was considered inoperable, and the patient died from a re-bleeding. Figure 2.22 shows a 1960 VA angiography.

Before 1980, only 16 surgeries for posterior circulation aneurysm were performed in Helsinki. The first operation for a posterior circulation aneurysm was done in July 1961, when Tapio Törmä clipped a ruptured basilar bifurcation aneurysm. Unfortunately the patient did not wake up from the surgery. In September 1961, Professor Gunnar af Björkesten wrapped a ruptured basilar bifurcation aneurysm. Postoperatively, this patient had hemiparesis, cranial nerve deficits, and memory deficits, but finally he recovered to an independent state.

The first attempt to embolize an aneurysm was done in Helsinki in 1991. The next year, came an attempt to embolize a posterior circulation aneurysm. The first posterior circulation aneurysm was succesfully embolized in in 1994. The first balloon-occlusion of the ICA took place the same year as the first aneurysm embolization, 1991. The same year, an ICA aneurysm was filled with a balloon. The VA was occluded for the first time with a balloon in 1997, and with coils in 2004.

Additionally, in 1976, ophthalmic aneurysm occlusion was attempted by filling it with some kind of filaments through a craniotomy.

The vertebral artery and the posterior inferior cerebellar artery

Anatomy

Vertebral artery

The paired vertebral arteries are the first and largest arteries originating from the subclavian arteries. Occasionally they can arise di-

Figure 2.22
Example of vertebral angiography from 1960

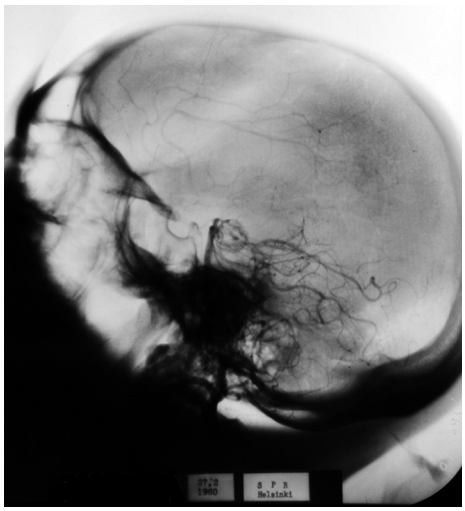
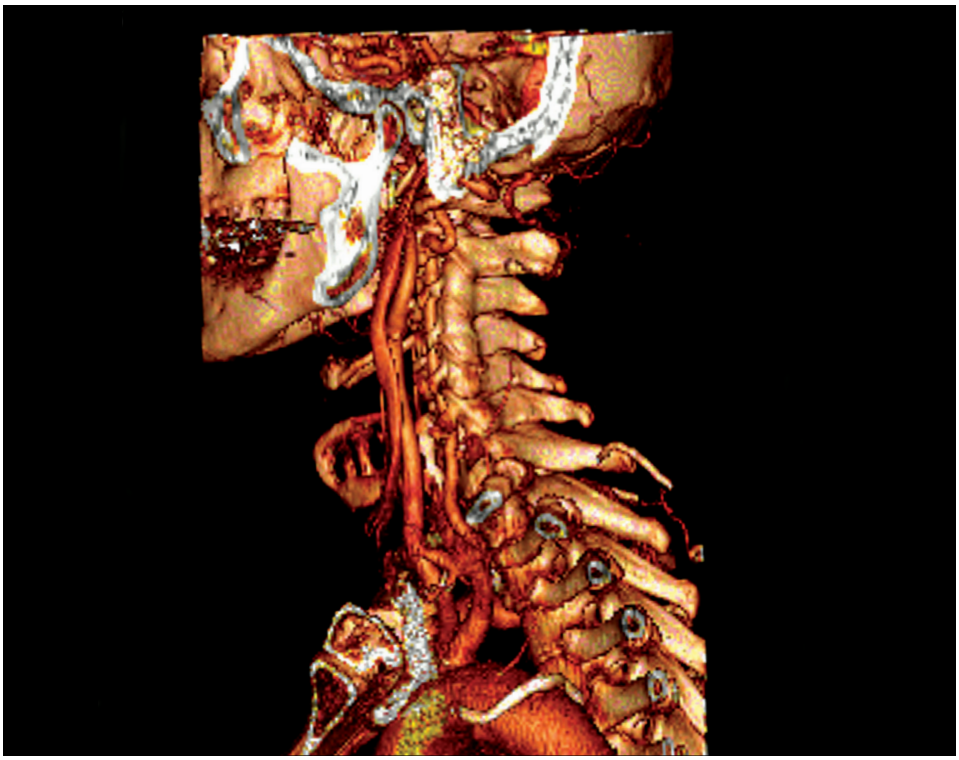


Figure 2.31
Extracranial VA



rectly from the aortic arch. Each vertebral artery is subdivided into four segments: the first segment (V1) ascends to the C6 transverse process, the second segment (V2) extends to the foramen of the transverse process of the atlas, the third segment (V3) up to the passage of the artery through the dura, the fourth segment (V4) is intradural.

The first VA segment (V1) ascends behind the internal jugular veins between the musculus longus colli and musculus scalenius anterior. This segment is often affected by atherosclerotic wall transformation leading to consecutive narrowing of inner vessel diameter.

The second VA segment (V2) runs in front of the cervical nerve roots through the foramen transversarium of the upper six vertebrae to the axis. From the V2 segment originate the cervicospinal branches for nutrition of the spinal cord, nerve roots, and the vertebral bodies. In the narrowed corridor of the V2, an external effect on the vertebral artery is possible because of congenital anomalies, a narrowed foramen transversarium, degeneration, osteophytes, ligamentous hypertrophy, herniated discs, or hypermobility of a cervical motion segment. Cervical luxation fractures can result in a traumatic vertebral artery dissection or occlusion. The close relation of the vertebral artery to the cervical nerve roots exposes the vertebral artery also to iatrogenic injuries.

The third VA segment (V3) is closely related to the craniocervical junction and the foramen magnum. After traversing the foramen of C3 to reach the C2 tranverse process, the vertebral artery ascends laterally. A vertically oriented segment traverses the C1 transverse process, after which a horizontal portion travels in a groove of the superior surface of the posterior arch of the atlas. Then V3 turns obliquely upward to reach the dura. The V3 segment is surrounded by the venous plexus, which is well perfused by anastomoses between the deep cervical and epidural veins. The dural entrance lies just at the inferior-lateral side of the foramen magnum. A thick dura forms a tunnel around 5 mm long around the vertebral artery. This tunnel also harbors the first cervical nerve, lying on the caudal surface of the VA, and the posterior spinal artery lying posterior to the VA. Occasionally the dural tunnel is partially or even totally surrounded by bone. During surgery, the V3 segment is important to obtain extra-cranial proximal control of the ipsilateral vertebral artery. The segment can be affected by injuries to the craniocervical junction. To protect the VA from iatrogenic injuries, its identification by intraoperative Doppler ultrasound is very helpful. Anastomoses with the occipital, ascending pharyngeal, and cervical arteries occur in the V3 segment. Commonly, the posterior spinal artery arises from the V3 segment beside V4 segment. In variations, an extra-dural origin of the PICA is also possible.

The intracranial VA segment (V4) is subdivided into the lateral and anterior medullary segments, the division being at the preolivary sulcus. The anterior medullary segment lies on the clivus and terminates when the vertebral arteries join each other to form the basilar artery usually near the pontomedullary junction. Intracranially, the vertebral artery gives rise to the PICA and anterior spinal artery.

In most cases, the left vertebral artery is larger than the right, and a hypoplastic vertebral artery is more often found on the right side (104). Instead of terminating at the basilar artery, an atretic vertebral artery terminates at the PICA, occipital artery, or spinal artery. Among the primitive arteries, the proatlantal artery terminates at the vertebral artery.

Posterior inferior cerebellar artery

The PICA usually originates from the intracranial segment (V4) of the vertebral artery. However, an extra-cranial origin from the V3 segment is also possible.

There exists, as well, separate classifications for segments of the PICA, the most commonly used anatomy-oriented classification of Rhoton, and the more surgically oriented classification of Drake (47, 136). Rhoton divides the PICA into five segments: an anterior medullary segment (1) extending up to the most prominent point of the inferior olive. This segment may also be absent. A lateral medullary segment (2) continues up to the rootlets of CNs IX to XI. A tonsillomedullary segment (3) extends until the mid-portion of the PICA's ascent along the medial surface of the cerebellar tonsil. A telovelotonsillar segment (4) terminates when the PICA exits to the suboccipital surface of the cerebellum. The cortical segment (5) designates PICA's terminal branches.

Drake divides the PICA into two segments: a proximal segment extending about one centimeter from its origin at the VA and a distal segment.

The PICA shows several anatomical variations. It may be absent or bihemispheric, supplying both hemispheres or with a bilateral vermian supply only, or its origin can be duplicate. The PICA can also originate from the hypoglossal, proatlantal, or posterior meningeal artery.

A number of variations and anomalies of the PICA exist: some of them are listed in Table 2.31.

Table 2.31
Some reported anatomical PICA variations

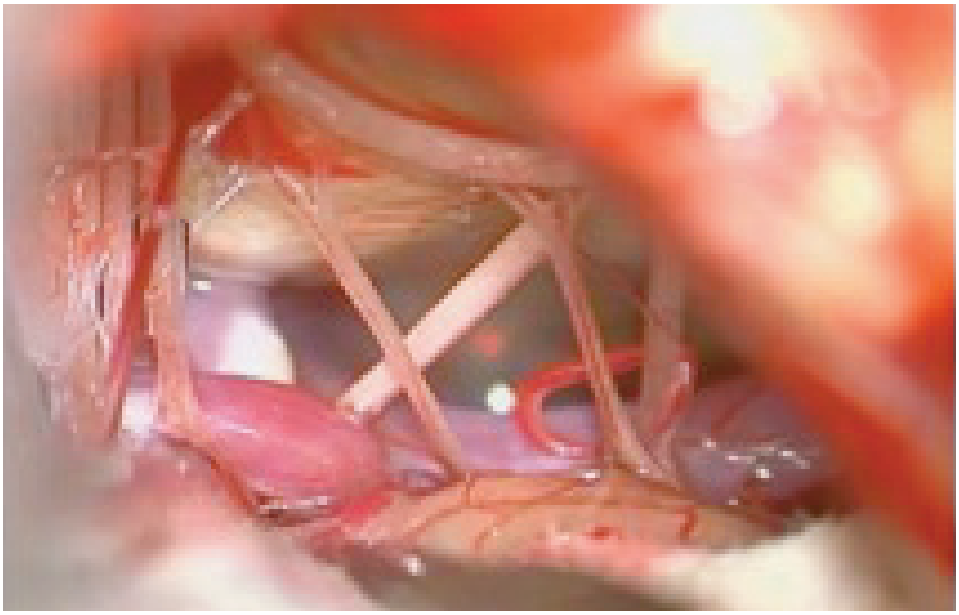
Series (year)	Variation
Lesley (2008) (130)	PICA fenestration
Kumar (2012) (118)	
Trivelato (2011) (170)	Double origin of PICA
Pasco (2002) (176)	
Fine (1999) (54)	Extracranial-extradural origin
Uchino (2011) (230)	PICA supplied by branch of ascending pharyngeal artery
Okuno (1988) (166)	Posterior meningeal artery rising form PICA
Katsuno (2012) (101)	Extracranial origin, anomalous with V_3
Carlson (2012) (22)	Bihemispheric PICA
Manabe (1991) (143)	PICA originating from the ICA
Perot (2011) (180)	Persistent trigeminal artery terminating in PICA
Ali (2008) (7)	
Raphaeli (2009) (185)	PICA supplied by trigeminal artery arising from a cavernous ICA
Andoh (2001) (10)	Persistent hypoglossal artery ending in PICA
Kuruvilla (2011) (120)	Middle meningeal artery originating from PICA
Cho (2011) (30)	Double origin with fenestration

Cranial nerves

The cranial nerves most frequently encountered during surgery on vertebral artery aneurysms are CN IX, (glossopahryngeal), X (vagus), and XI (accessory). They leave the medulla as a group of rootlets through the postolivary sulcus, with the posterior olive ventrally and inferior cerebellar peduncle dorsally. The cranial root forms CN IX, and three to five caudal rootlets the CN X and XI. The nerves travel through the cerebellomedullary cistern. After sending off a tympanic branch, they exit the skull through the jugular foramen.

In large VA aneurysms, CN VII (facial) and VIII (vestibulocochlear) can also be encountered, and if the aneurysm lies caudally, also CN XII (hypoglossal). Both CN VII and VIII merge from the pontomedullary junction, CN VII above CN VIII. They travel through the cerebellopontine cistern to enter the internal acustic meatus. CN XII

Figure. 2.32
VA, PICA, and cranial nerves



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emerges from the medulla in the preolivary sulcus, i.e. with the pyramid anteriorly and inferior olive posteriorly. The nerve consists of 10 to 15 small rootlets, which converge into two bundles to enter the hypoglossal canal.

Controversies in nomenclature

The present literature displays a few controversies regarding nomenclature related to VA and PICA aneurysms. First, the definition of the PICA is diverse: it is defined either as an artery originating from the VA to supply the cerebellum (136, 252), or as an artery originating from the VA or the basilar artery to supply the posterior inferior part of the cerebellum (251). Due to high variation in the area supplied by the PICA, we defined as PICA the vessel originating from the VA to supply the cerebellum.

The other controversy involves categorization of PICA aneurysms. A "PICA aneurysm" typically refers to an aneurysm either at the VA–PICA junction or more distally in the PICA. The term "proximal PICA aneurysm" is used either for an aneurysm at the VA–PICA junction only (47), or also for those arising from the anterior medullary segment of the PICA (24), or any medullary segment of the PICA (133, 177). Aneurysms at the PICA outside VA–PICA junction are also called "true PICA aneurysms" (258).

Aneurysms

Incidence

Probably due to their rarity, reports on incidence of VA and PICA aneurysms in a non-selected series are scarce. In the series of Yamaura et al., the prevalence of VA and PICA aneurysms among posterior circulation aneurysms was 32% (250). In a cooperative

study from Japan, among ruptured saccular aneurysms, those located in the VA or PICA accounted for 1.9%; among posterior circulation aneurysms they accounted for 39% (253). In the vertebrobasilar aneurysm series of Drake et al., VA and PICA aneurysms accounted for 12.5% (47). In the Cooperative study from 1966, among SAH patients with a single aneurysm only, those locating in the VA or PICA numbered 1.3% (137). In a Finnish study on saccular aneurysms, the incidence of VA and PICA aneurysms was 2.4% (82).

Mere PICA aneurysms are believed to comprise 0.5 to 3%. Among all treated aneurysms, Peluso et al. reported 2.8% to be located in the PICA (177). In 2013, Bacigaluppi et al. reported in 621 patients the incidence of PICA aneurysms as being 3.7% (15). In the series from Kuopio, Finland, at the junction of PICA were located 1.9% of aneurysms (82). The incidence of distal PICA aneurysms is low, 0.56% of all aneurysms according to a report by Ishikawa et al. in 1990 (91) and 0.86% in a report by Tokimura et al. in 2011 (227).

Location

Most VA and PICA aneurysms are located at the junction of these vessels (47, 82, 250). In the series of Drake et al., they accounted for 69% (47). Altogether in their series, the number of aneurysms located in the proximal VA was 8 (3%), the VA–PICA junction 168 (69%), distal VA 41 (17%), and distal PICA 26 (11%). Bertalanffy et al. in 1988 reported their experience with the VA and distal PICA aneurysms: in the proximal VA were located 5 (21%), VA–PICA junction 7 (29%), distal VA 3 (13%), and in the distal PICA 9 (38%). In this series, distribution of the PICA aneurysm is exceptional, with distal PICA aneurysms outnumbering aneurysms at the origin of PICA. However, three patients with distal PICA aneurysms also had a posterior fossa AVM, which could at least in part explain the high number of distal PICA aneurysms.

In previous series, location of distal PICA aneurysms have been mainly reported according to the classification of Rhoton (15, 78, 91, 92, 131, 167, 227). The reported distribution of aneurysms has been slightly variable: Horiuchi et al. in their series of 27 distal PICA aneurysms reported the most common location to be the telovelotonsillar segment (30% of aneurysms), followed by the lateral medullary segment (26%) (78). Tokimura et al. found 9 (30%) aneurysms in the lateral medullary segment, and as the next common location, 7 (23%) aneurysms in the tonsillomedullary segment (227). Lewis et al. reported the most common location of distal PICA aneurysm to be the cortical segment; they included in their series of 20 patients 6 with an AVM (131). In the series of Orakcioglu, the two most common locations, with an equal distribution of 33%, were the anterior medullary segment and telovelotonsillar segment (167). With the exception of the report of Lewis et al., the larger series report medullary segments as being the most common location for a distal PICA aneurysm (78, 167, 190, 227).

Morphology and etiology

In the VA, saccular aneurysms, located at the junction of the PICA, are by far most common. The proportion of fusiform aneurysm ranges from 13 to 26%, and dissecting 7 to 28% (11, 19, 47, 250). Dolichoectasias many times reach from the vertebral artery to involve also the basilar artery (12, 57, 187, 245); the incidence of those located in the vertebral artery alone is unknown. Serpentine aneurysms are mostly in case-reports (169, 224, 234). In the distal PICA alone, fusiform aneurysms account for 7 to 41% and dissecting 0 to 41% (78, 131, 167, 227).

Table 2.32
Larger surgical series conserning VA and distal PICA aneurysms

Series (Year)	Year	No. of aneurysms	Notes
Series (Year)	2008	6	Dissecting distal PICA aneurysms only
Horiuchi (78)	2007	24	Distal PICA aneurysms only
Al-khayat (3)	2005	52	Study on lower cranial nerve palsies
Liew (133)	2004	13	Study on lower cranial nerve palsies
D'Ambrosio (35)	2004	20	
Nussbaum (161)	2003	7	Fusiform distal PICA aneurysms only
Lewis (131)	2002	20	Distal PICA aneurysms only
Matsushima (145)	2001	8	
Horowitz (79)	1998	38	
Bertalanffy (19)	1998	27	
Sano (207)	1997	45	Dissecting aneurysms
Andoh (11)	1992	38	
Yamaura (249)	1990	24	Dissecting aneurysms
Ishikawa (90)	1990	12	Distal PICA aneurysms only
Yamaura (250)	1988	86	
Gacs (61)	1983	16	

Symptoms

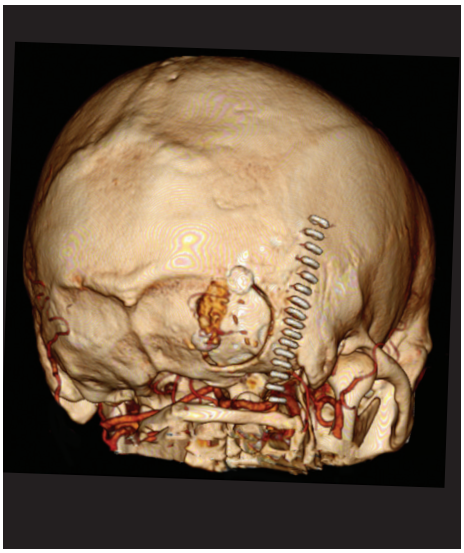
As with all intracranial aneurysms, most VA and PICA aneurysms are found as a result of subarachnoid hemorrhage (11, 250). Yet the close location of the cranial nerves and the brainstem occasionally causes the aneurysm to manifest as a cranial nerve deficit or as symptoms caused by brainstem compression (12, 19, 47, 139, 179). Symptoms caused by mass effect include ataxia (47, 64, 79, 85, 131, 224, 237, 239), sensory loss or paresthesias (47, 183), hemi-, para-, or tetraparesis (2, 12, 13, 47, 133, 224, 254), lateral medullary syndrome (86), bulbar palsy (1, 17, 47, 64, 85, 133, 144, 237), gaze palsy (47, 129, 177, 183, 240), nystagmus (19, 84, 216, 224, 240), impaired hearing (47, 129, 237, 254), facial nerve palsy or hemifacial spasm (17, 47, 110, 208, 231, 237), or trigeminal symptoms (12, 47, 152). These symptoms are occasionally also caused by compression of the hemorrhage upon the neural structures (47, 139). Additionally, a giant aneurysm can cause obstructive hydrocephalus (47).

Besides SAH and symptoms caused by compression, VA dissecting aneurysms are associated with cerebellar and brainstem ischemia (12, 87, 98, 99, 140, 188, 250).

Table 2.33
Some reported approaches to VA and distal PICA aneurysms

Approach	Reference
Suboccipital	47, 68, 80
Midline suboccipital subtonsillar	70
Midline (medial) suboccipital	72, 125, 196, 203
Paramedian suboccipital	252
Combined lateral and medial suboccipital	20
Suboccipital transcondylar	103
Lateral suboccipital	11, 19, 72, 203, 250
Lateral suboccipital and partial condylectomy without laminectomy	255
Far-lateral	73, 115, 122
Far-lateral suboccipital	26, 35, 164
Far-lateral supracondylar	241
Far-lateral paracondylar	241
Far-lateral with partial condylar resection	100
Far-lateral approach with resection of occipital condyle	157
Far-lateral transcondylar	19, 40, 46, 145, 241
Far-lateral transcondylar transtubercular	218
Transcondylar fossa (supracondylar transjugular tubercle)	145
Extreme lateral transcondylar	14, 191
Extreme-lateral inferior transtubercular	39, 202
Extreme lateral, transcondylar, transjugular	211
Contralateral far-lateral	21
Presigmoid transpetrosal	103
Retromastoid	68
Subtemporal	121
Pterional	121
Transfacial transclival	26
Minimally invasive supracondylar (MIST)	197

Figure 2.33
Lateral sucboccipital craniotomy



Treatment

In VA and PICA aneurysms, the challenges caused by location, variable morphology, and etiology have led to a wide range of techniques for their occlusion. This can be seen also in the studies published after 2000, with methods including clipping (35, 204), wrapping (204), surgical proximal occlusion or trapping in possible combination with a bypass (35, 87, 204), selective coiling (177), internal trapping (119), stent-assisted coiling (31), or mere stenting or use of a flow-diverter (25, 256), proximal occlusion with coils or balloon (177), and endovascular vessel occlusion combined with bypass surgery (65).

Microneurosurgery

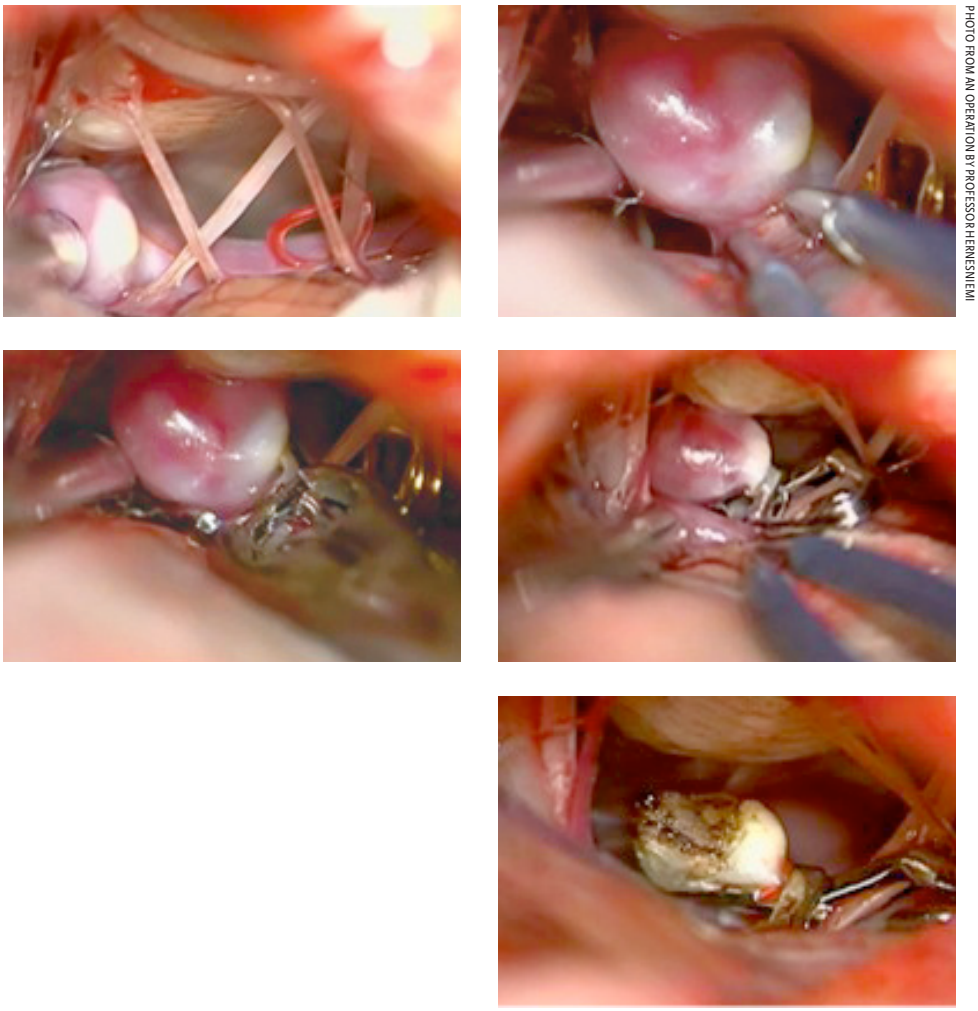
In Table 2.32, see surgical series on VA and PICA aneurysms. Surgical treatment is mainly challenged by location of the cranial nerves many times in close proximity to the aneurysm. Another challenge is the complete and stable occlusion of fusiform (dissecting) aneurysms.

As one approach, most surgeons favor the far-lateral approach described especially by Heros (72) (Table 2.33). Its extensions have been divided according to their relation to the occipital condyle, i.e. transcondylar, paracondylar, and supracondylar (231). Another common approach is the lateral suboccipital (Figure 2.33) or the retrosigmoid approach, which consists of less bony opening than with the far-lateral approach. The suboccipital approach was favored by Drake et al. (47).

In our department, Professor Juha Hernesniemi uses a small retrosigmoid craniotomy for most VA and PICA aneurysms. The prerequisite for this approach is location of the lesion a minimal 10 mm above the foramen magnum. Aneurysms located below this line require a more lateral approach. Instead of the classic far-lateral approach, the choice is an approach "lateral enough." In this, the foramen magnum is opened, but in most cases C1 is left intact. When needed, removing part of C1 or minimal drilling of the occipital condyle extends the approach. Drilling the whole condyle as in the transcondylar approach, drilling of the jugular process as in the paracondylar approach, or drilling of the jugular tubercle as in the supracondylar approach have been unnecessary. For aneurysms in the cortical branches of the PICA close to the midline, the approach is a median or paramedian suboccipital. The small craniotomies require good neuroanesthesia and spinal drainage to gain maximal space for dissection.

Even if the primary method used for surgical aneurysm occlusion is clipping, especially in fusiform (dissecting) or giant aneurysms clipping may be impossible. When treated surgically, these cases may require VA or PICA occlusion. A non-dominant VA distal to the PICA can in most cases be occluded safely. It is assumed that distal PI-

Figure 2.34
Clipping of a PICA aneurysm



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CA can also be occluded safely distal to the medullary segments, as no perforators to the brainstem typically arise after this point (136). This, however, has turned out to be somewhat unreliable (92). When in doubt as to the safety of occluding VA or PICA, some authors advocate using a balloon occlusion test, while others limit its use to the anterior circulation (215, 217, 223). If the patient fails the balloon occlusion test, or otherwise needs flow augmentation, the most commonly used are or OA–PICA bypasses (9, 34, 123, 129, 161, 212). The other possibilities include re-anastomosis of the PICA (64, 123, 161), (re-)implantation of the PICA to the VA (17, 123, 162, 163) or to the AICA (51, 126), AICA–PICA side-to-side anastomosis (51) VA–RAG / OAG / vein graft–VA (51, 116, 123), CCA–RAG / vein graft–PICA (32, 212), PICA–STA–VA bypasses (64–66).

Endovascular treatment

Endovascular treatment has gained popularity for VA and also in PICA aneurysms (Table 2.33). With the advantage of no cranial nerve manipulation, endovascular treatment still is, however, challenged by attempts to keep the PICA open, and to achieve complete occlusion of the aneurysm. The small caliber and tortuous course of the PICA challenge treatment of distal PICA aneurysms in particular. In recent years, the number of papers on endovascular treatment of VA and distal PICA aneurysms, dealing with the challenges and advantages of endovascular treatment has been growing (24, 33, 225, 228). Besides selective embolization, internal trapping has been useful, especially in distal PICA (15, 24, 92, 134, 168) and dissecting VA aneurysms (48, 105, 124, 178, 222). To avoid a vessel occlusion, use of stent-assisted coiling (95, 107, 155, 186, 201), mere stenting (95, 175, 186, 201, 256, 257), or lately also flow diverters has evolved (6, 25, 182, 184, 199, 236).

Outcome

Radiological

In the surgical series of Drake et al. (221 aneurysms), total occlusion of the VA and distal PICA aneurysms was achieved in 86% and a neck remnant was found in only 4% (47). That series also included 59 aneurysms treated with Hunterian ligation and 7 aneurysms treated with wrapping. In the more recent series of Sanai et al., among 59 aneurysms, all but one clipped PICA aneurysm and three wrapped aneurysms were totally occluded, giving a total occlusion rate of 93% (204).

In a recent endovascular series of 76 proximal and distal PICA aneurysms, complete occlusion was achieved in 63% (24). In a series of Endo et al. on internal trapping of 38 ruptured VA dissecting aneurysms, after primary total occlusion 5 (21%) aneurysms recanalized (48). In a series of Lv et al. on 22 dissecting aneurysms of VA and PICA treated with various endovascular methods, 6 (27%) aneurysms were totally occluded (140).

Clinical

In good-grade patients, the outcome of treatment is generally favorable. In the series of Drake et al., among the 181 good-grade (Botterel classification grades 0 to 2) patients with non-giant aneurysms, only 2 poor outcomes and 4 deaths occurred (47). In series of Yamaura et al. on VA aneurysms, GOS 4 to 5 recoveries occurred in 64 (94%) of surgically treated patients (250). In their recent surgical series, GOS 4 to 5 occurred in 42 (76%) (205), 47 (90%) (3), and 14 (94%) patients (35). Morbidity from surgery is many times related to cranial nerve deficit: up to 48% of patients suffer from lower cranial nerve palsies (3, 19, 79, 250). In a study on 52 patients with such postoperative palsies, within 6 months 76% recovered (3). Even better outcomes have been reported: in the series of Yamaura, only one patient of eight with lower cranial nerve palsies was left with a hoarse voice (250).

Figure 2.33
Recent series on VA and PICA aneurysms

	No. of patients	Treatment
Chen et al. (2013) (29)	8	Endovascular
Dabus et al. (2013) (36)	9	Endovascular
Ishihara et al. (2013) (89)	9	Endovascular
Trivelato et. al. (2013) (228)	14	Endovascular
Chalouhi et al. (2013) (24)	76	Endovascular
Wu et al. (2013) (246)	15	Endovascular
Cho et al. (2013) (31)	7	Endovascular
Bacigaluppi et al. 2013 (15)	23	Endovascular and surgical
Endo et al. (48)	38	Endovascular
Suma et al. (2013) (225)	10	Endovascular
Ioannides et al. (2013) (88)	10	Endovascular
Lin et al. (2012) (135)	9	Endovascular and surgical
Su et al. (2011) (222)	12	Endovascular and surgical
Hong et al. (2011) (76)	20	Endovascular and surgical
Kim et al. (2011) (105)	111	Endovascular
Shin et al. (2011) (214)	7	Endovascular
Tokimura et al. (2011) (227)	28	Endovascular and surgical
Lv et al. (2010) (139)	72	Endovascular
Sadato et al. (2010) (201)	26	Endovascular

In their series including both endovascular and surgical treatment of the aneurysms, Hong et al. reported a GOS 5 to 4 outcome in 65% of patients with ruptured PICA aneurysms (76). In a series on distal PICA aneurysms, of 24 patients with a ruptured aneurysm, 19 (79%) recovered to GOS 4 or 5 (227).

In one recent endovascular series, among patients with ruptured VA dissection, a modified Rankin Score of 0 to 2 was reported in 61% (23 patients) (48). Among proximal and distal PICA aneurysms in SAH patients, GOS 4 to 5 recovery was noted in 77% (24). In another study on proximal PICA aneurysms, GOS 4 to 5 occurred in 68% (177). In a study on patients with lower cranial nerve palsy caused by a PICA aneurysm, the palsy recovered totally in 6 months in 9 (75%) (139).

Lee et al. (2010) (124)	25	Endovascular
Nourbakhsh et al. (2010) (159)	15	Endovascular and surgical
Lv et al. (2010) (140)	22	Endovascular
Lv et al. (2010) (141)	24	Endovascular
Jeon et al. (2009) (94)	15	Endovascular
He et al. (2009) (69)	6	Endovascular
Isokangas et al. (2008) (92)	12	Endovascular
Li et al. 2008 (132)	5	Endovascular and surgical
Cellerini et al. (2008) (23)	11	Endovascular
Peluso et al. (2008) (177)	46	Endovascular
Kudo et al. (2007) (117)	9	Endovascular
Mericle et al. (2006) (146)	31	Endovascular
Maimon et al. (2006) (142)	6	Endovascular
Orakciolgu et al. (2005) (167)	16	Endovascular and surgical
Albuquerque et al. (2005) (4)	23	Endovascular
Al-khayat et al. (2005) (3)	52	Surgical
Sandalcioglu et al. (2005) (206)	28	Endovascular and surgical

Aims of the study

1. To describe the anatomy of PICA aneurysms as diagnosed by CTA (Study I)

2. To clarify the special features of intracranial VA and distal PICA aneurysms (Studies II and III)

3. To describe the treatment of VA and distal PICA aneurysms, and analyze the outcome of the treatment (Studies II and III)

Patients and methods

Patients

We reviewed retrospectively 9 709 consecutive patients with intracranial aneurysms treated in the Department of Neurosurgery at Helsinki University Central Hospital, Finland, between 1934 and 2011. We excluded, to ensure a population-based material, those referred from abroad. We also excluded all vertebro-basilar junction aneurysms or aneurysms extending to the basilar artery.The study population thus included 268 patients with 284 VA or PICA aneurysms or with both.

Study I (Seventy aneurysms of the posterior inferior cerebellar artery: anatomical features and value of computed tomography angiography in microneurosurgery) included 70 patients with VA–PICA junction aneurysms imaged by CTA.

Study II (Intracranial vertebral artery aneurysms: clinical features and outcome of 190 patients) included 190 patients with 193 VA aneurysms, including aneurysms at VA–PICA junction.

Study III (Distal posterior inferior cerebellar artery aneurysms: Clinical features and outcome of 80 patients) included 80 patients with 91 aneurysms in distal PICA.

Aneurysms were identified either by CTA, DSA, conventional angiography, or by MRA, or at autopsy, or they were merely surgical findings. CT, lumbar puncture, or autopsy diagnosed SAH. A suspected incidental aneurysm was considered earlier ruptured if a clear surgical finding of a former rupture was evident (visualized rupture site in the aneurysm with hemosiderin next to it, with no other source of hemorrhage). In cases of multiple aneurysms, the site of rupture was decided based on CT findings, size and irregularity of the aneurysm, or findings in surgery or at an autopsy or both. In cases of AVM and associated aneurysm, the rupture site was diagnosed based on findings in CT, surgery, and possibly at autopsy.

Patients' neurological status was assessed on the Hunt and Hess scale (H&H) without the correction for general disease (81).

We compared patients with a ruptured VA and PICA aneurysm to those with a ruptured aneurysm elsewhere (age, gender, aneurysm size and morphology, number of aneurysms, Fisher grade, occurrence of IVH or ICH, rebleeding rate, and H&H). We used for comparison all other patients

treated for ruptured intracranial artery aneurysm in the Department of Neurosurgery, Helsinki, Finland, from 1980 to 2009. The patients treated before the era of CT scanning and microsurgery, i.e. before 1980, were excluded from this part of the study.

Data and analysis

Radiological data

The radiological images were re-reviewed by a neurosurgeon-radiologist (Riku Kivisaari). Digital archiving started in 1999, and the images were reviewed with AGFA Impax (version 5.3, Agfa, Mortsel, Belgium). Imaging studies from 1989 onwards were available for review. In cases in which images were unavailable or their quality was poor (n = 44, 16%), the data came from radiological and surgical reports.

The first CT-scanner came to Töölö Hospital in 1980. Since acquiring the CTA program, the examinations were preformed with a 4-slice scanner until 2007 (GE Lightsoeed QX/I; GE Medical Systems, Milwaukee, WI, USA) and later with a 32-slice scanner (GE LightSpeedPro 32) or 64-slice scanner (GE LightSpeed VCT Advantage).

SAH was diagnosed either by CT (192 patients) or lumbal puncture (31 patients). In an additional three patients the aneurysm was incidentally found, but during surgery hemosiderin around the aneurysm was a sign of earlier bleeding.

For aneurysm diagnostics, conventional cut film angiography was used in our clinic until 1991. After this, DSA was the only method until 1995 when CTA was introduced. Since 2000, CTA has been the primary aneurysm-diagnostic method for SAH patients, and is also always performed before aneurysm surgery. Nowadays, we perform DSA if endovascular surgery is considered, if CTA remains negative, or if a suspicion arises of a small aneurysm, or in case of a need to study flow dynamics. We use MRA for screening, in patients with iodine allergy, and MRI together with MRA when study (partially) thrombosed aneurysms. Additionally, patients are also admitted to our clinic with their aneurysms diagnosed by MRA.

Table 4.21
Imaging methods used for aneurysm diagnostics

Angiography	No. of aneurysms
DSA only	116
CTA only	74
MRA only	2
DSA and CTA	55
DSA and MRA	5
DSA, CTA and MRA	9
CTA and MRA	11

CTA, computed tomography angiography; DSA, digital substraction angiography; MRA, magnetic resonance angiography

Table 4.21 shows the different imaging methods used for VA and PICA aneurysm diagnostics. One patient had no preoperative angiography, as the aneurysm was initially wrongly diagnosed as recurrent hemangioblastoma. Five aneurysms were intraoperative findings: preoperatively, DSA was performed in three, CTA in one, and both DSA and CTA in one case. Two of these aneurysms were thrombosed, and three were 2-mm aneurysms, found during an operation performed for another VA or PICA aneurysm. At autopsy 14 aneurysms were diagnosed: 11 had no kind of angiography, in 3 aneurysms DSA had been negative.

In each aneurysm we measured the maximal width and length of the sac, and the neck diameter (Fig 4.21). Maximal diameter of both saccular and fusiform aneurysms was considered the largest measurement in any direction.

For the anatomical study based on CTA (Study I), all the CTAs were reanalyzed. We measured the relationship of the aneurysm to the skull base (distances from foramen magnum, midline, closest bony structure, clivus, and relation to hypoglossal canal and jugular tubercle). Additionally, we measured the size of the jugular tubercle in three dimensions. We recorded anatomical variations in the VA, cerebellar arteries, basilar artery, and also possible persistent fetal anastomoses, additional aneurysms, AVM, and AVF. We measured size of the PICA at its origin; if the PICA could not be visualized in CTA, it was considered hypoplastic. The VA we considered hypoplastic, if its diameter was equal to or less than 2 mm at the level of the foramen magnum (173). If no VA was visible distal to PICA, it was considered atretic.

Follow-up data

We followed up the patients until death or the end of 2011. The Population Registry Centre, comprising all Finnish residents, provided the vital status of the patients on December 31, 2011, as well as possible date of death. The causes of death came from Statistics Finland (http://www.stat.fi/index_en.html). To those still alive we sent a written questionnaire concerning their present health status. For those who did not respond, we obtained the follow-up data only from the department of neurosurgery. For those deceased, we used medical records from all public health services.

Statistical analysis

We performed the data analysis by IBM SPSS Statistics, version 20.0.0 for Mac (a commercial statistical software). To compare groups, we used Pearson's x^2 or Fisher's exact test for categorial variables, and the Mann-Whitney U-test for continuous variables. We considered probability value < 0.05 statistically significant. We analyzed the risk factors for death at one year after treatment of ruptured VA or PICA aneurysm. In univariate analysis we included age, gender, Fisher grade, size and morphology of the aneurysm, possible re-bleeding, shunt-dependent hydrocephalus, and H&H grade in good-grade (H&H 1 to 3) and poor-grade (H&H 4 and 5) grade groups, as well as decade of treatment. To calculate odds ratios (ORs) and 95% confidence intervals (CIs) of independent factors associated with one-year case fatality, we used unconditional binary logistic regression analysis. To a stepwise forward elimination procedure, the selected variables were added on the basis of their probability values.

Figure 4.21

Measurement of saccular (left side) and fusiform (right side) aneurysms. L = length, W = width, N = neck

Results

Incidence of vertebral and posterior inferior cerebellar artery aneurysms

After 2000, all aneurysm patients treated in our clinic have been investigated with cerebral CTA or MCA, or in rare cases only autopsied. Among these patients, those diagnosed with a VA or PICA aneurysm or both accounted for 5.1%. Of those with aneurysmal SAH, the rupture site was in the VA or PICA in 3.9%. Of all aneurysms, 3.7% were located in the VA or PICA.

Anatomy of aneurysms at junction of the vertebral artery and the posterior inferior cerebellar artery

Patients and aneurysms

In 70 patients the saccular aneurysm at the VA–PICA junction was diagnosed by CTA, each had one PICA aneurysm (Table 5.21). Multiple aneurysms were diagnosed in 32 patients, with 17 patients suffering from SAH from an aneurysm at another location. Two PICA aneurysms were diagnosed due to mass effect. The most common associated aneurysm was that of MCA (Table 5.22). No patient had multiple aneurysms in the VA or PICA; one had an occipital AVM.

PICA aneurysms

Location

Of the 68 postPICA aneurysms and 2 prePICA aneurysms (Figure 5.21), most (61%)

Table 5.21.
Patient characteristics (n = 70)

Gender	**No. (%)**
Female	47 (67)
Male	23 (33)
Age	**median (range)**
All	59.5 (29 – 85)
Presentation	**No. (%)**
SAH from PICA aneurysm	42 (60)
SAH from another aneurysm	11 (16)
Incidental	13 (19)
Screening	1 (1)
Mass lesion	2 (3)
Total number of aneurysms per patient	**No. (%)**
1	38 (54)
2	10 (14)
3	17 (3)
4	2 (3)
5	1 (1)
6	1 (1)
7	1 (1)

PICA, posterior inferior cerebellar artery; SAH, subarachnoid hemorrhage

Table 5.22.
Associated vascular lesions

	No. of patients	No. of lesions
Aneurysm		
ICA	11	15
MCA	23	34
ACA	10	10
BA	5	8
AVM	1	1

ACA, anterior cerebral artery; AVM, arteriovenous malformation; BA, basilar artery; ICA, internal carotid artery; MCA, middle cerebral artery

Table 5.23
Distance from PICA aneurysm to adjacent anatomical structures

	Median (m)	Range (mm)
Distance from the foramen magnum	16.5	-1.0 – 31.0[a]
Distance from the midline	6.0	-7.5 – 14.0[b]
Distance from the clivus	11.0	3.0 – 33.0
Distance from the bone	4.0	1.0 – 15.0

a Negative figure located below the foramen magnum.
b Negative figure located on the contralateral site of the skull compared to parent artery origin.

were on the left side. Most were within 10 mm from the midline, at a distance ranging from 8 mm contralateral to 14 mm to ipsilateral of the side of the parent artery (Table 5.23). On the contralateral side of the skull were four aneurysms (Figure 5.22). In relation to the foramen magnum, one aneurysm was located below it (Fig. 5.23), within 10 mm above it were 15 (22%), from 11 to 20 mm were 23 (47%) and over 21 mm above the foramen were 20 (29 %). Table 5.24 presents location of aneurysms in relation to different skull base structures. To the skull base were attached 11 (16%) aneurysms.

Morphology

Maximum median diameter of aneurysms was 5 mm (Table 5.25). Small aneurysms, with a maximal size of 6 mm, numbered 48 (69%). Only one aneurysm was giant. Calcifications were present in 10 (14%). Five aneurysms (7%) were partially thrombosed: two were medium-sized (7–14 mm), two large (15-24 mm), and one giant (> 24 mm).

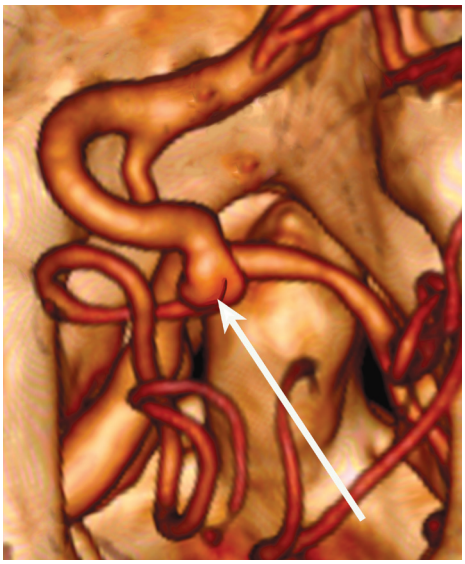
Variations of VA and PICA

The left VA was larger than the right (p = 0.002); the median diameter of the left VA was 2.8 mm (range 0-4.8) and of the right VA 2.1 mm (range 0-4.1). Both hypoplastic and atretic VAs were more frequent on the right side, as was the hypoplastic PICA (Table 5.26). Hypoplastic VA on both sides was diagnosed in two patients; an additional two patients had the right VA thrombosed. Of the aneurysms, 89% were located on the dominant VA. One patient had an aneurysm on the side of a hypoplastic PICA. Two PICAs were of extracranial origin.

Jugular tubercle

Of the 70 aneurysms, the level of the jugular tubercle was the location of 39 (56%).

Figure 5.21
PrePICA (left) and postPICA (right) aneurysms



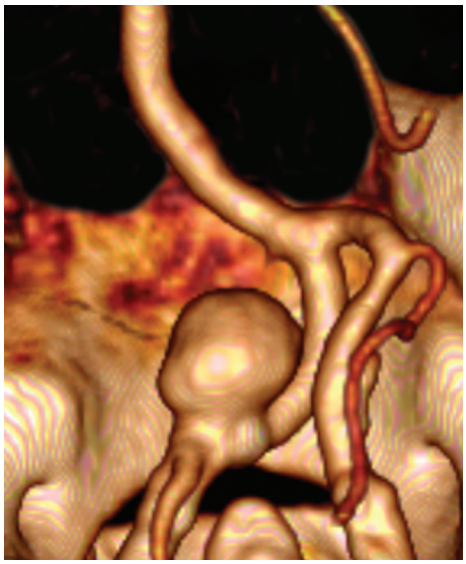


Table 5.24.
Aneurysm in relation to skull base

	No. of aneurysms (%)
Extracranial	1 (1)
Below the hypoglossal canal	5 (7)
At the hypoglossal canal	10 (14)
At the jugular tubercle	39 (56)
Above the jugular tubercle	15 (21)

Figure 5.22
Left PICA aneurysm on the right side of the posterior fossa as compared to origin of the parent artery

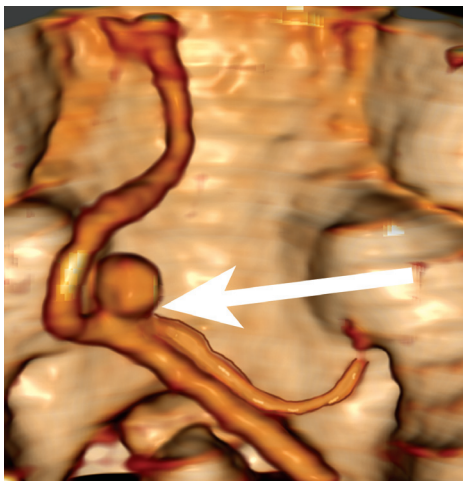


Figure 5.23
Aneurysm located extracranially

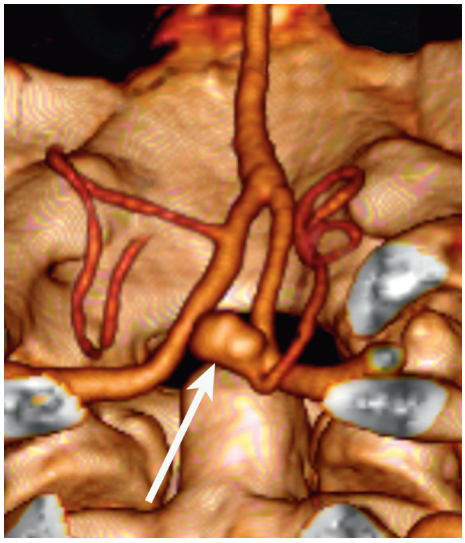


Figure 5.24
A left jugular tubercle

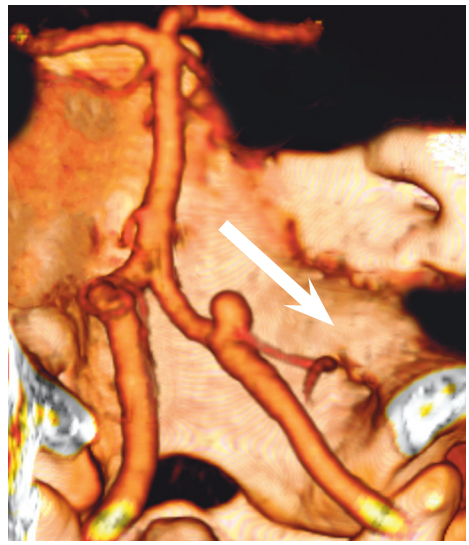


Table 5.25.
Sizes of VA – PICA junction aneurysms

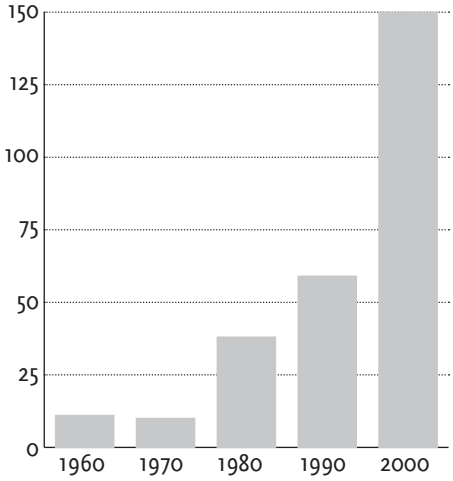
	Ruptured	Unruptured	Total
Number of aneurysms	42	28	70
Measurements	median (range)	median (range)	median (range)
Maximal size (mm)	6 (2 – 20)	4 (2 – 27)	5 (2 – 27)
Length (mm)	6 (1 – 20)	4 (1 – 27)	5 (1 – 27)
Width (mm)	4 (2 – 13)	3 (2 – 20)	4 (2 – 20)
Neck (mm)	3 (2 – 6)	3 (1 – 8)	3 (1 – 8)
Neck-to-width	0.7 (0.3 – 1.0)	0.8 (0.2 – 1.0)	0.7 (0.2 – 1.0)
Size distribution	No.	No.	No.
< 7 mm	28	20	48
7 – 14 mm	13	5	18
15 – 24 mm	1	2	3
> 24 mm	-	1	1

Table 5.26
Variation in the VA and PICA

	Right; No.(%)	Left; No.(%)
VA		
Hypoplasia	31 (44)	14 (20)
Atresia	5 (7)	2 (3)
PICA		
Hypoplasia	11 (16)	5 (7)
Extracranial origin	2 (3)	-

PICA, posterior inferior cerebellar artery;
VA, vertebral artery

Figure 5.31
Decade of admission



The right jugular tubercle was slightly longer (p = 0.04), with its mean diameter on the right side 18.4, and on the left side 17.6 mm. Figure 5.24 demonstrates the location of the jugular tubercle. There existed no difference in mean width or thickness between the tubercles (on the right side 11.2 mm and 6.1 mm, and on the left 10.9 mm and 6.3 mm). No association appeared between size of the tubercle and side of the aneurysm.

Overview of patients with vertebral artery or posterior inferior cerebellar artery aneurysms

Patients and aneurysms

The 268 patients had altogether 284 VA and PICA aneurysms. The first VA aneurysm was diagnosed in 1961. After 1990 78% of the patients were admitted, and after 2000, 56% (Figure 5.31). Among all intracranial aneurysm patients admitted after 2000 – the era of routine use of CTA and MRA in our department – a VA or PICA aneurysm was diagnosed in 4.2%. Among all aneurysms VA and PICA aneurysms accounted for 3.2% and among ruptured aneurysms 5.3%.

Table 5.31 summarizes patient characteristics, revealing a slight female predominance (59% vs. 41%), and age at diagnosis as 40 to 70 (Figure 5.32), with no age difference between the genders. The most common reason for aneurysm diagnosis was SAH in 224 (84%). Of those with an unruptured aneurysm, 12 (4%) were symptomatic.

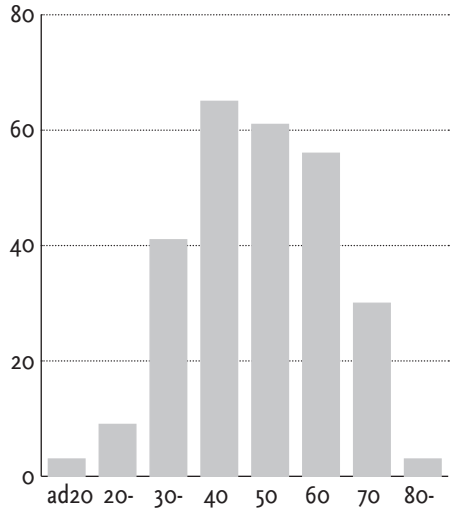
The 268 patients harbored altogether 445 aneurysms. A total of 92 (34%) patients had multiple aneurysms; several VA or PICA aneurysms were diagnosed in 12 (4%) (Table 5.32), among whom 3 had also additional aneurysm(s) elsewhere. The most common associated aneurysms were that of the MCA (Table 5.33). Patients with aneurysm(s) in the anterior circulation accounted for 75 (28%). Besides VA or PICA aneurysms, aneurysms elsewhere in the posterior circulation occurred in 21 (8%). Nine patients had additional aneurysms diagnosed later (Table

Table 5.31.
Characteristics of the whole patient population (n = 268)

Gender	No. (%)
Male	111 (41)
Female	157 (59)
Age at primary diagnosis	**median (range)**
All	52 (16 – 85)
Presentation	**No. (%)**
SAH from VA or PICA aneurysm	194 (74)
SAH from another aneurysm	29 (11)
Mass effect	9 (3)
Ischemia	3 (1)
Ruptured AVM/AVF	1 (0,3)
Incidental	32 (12)

AVF, arteriovenous fistula; AVM, arteriovenous malformation; PICA, posterior inferior cerebellar artery; SAH, subarachnoid hemorrhage; VA, vertebral artery

Figure 5.32
Patients according to age groups



5.34), among them definite de-novo aneurysms numbering four. Time from the first aneurysm diagnosis to the second ranged from 3 to 38 years, with a mean of 15 years. Four-vessel angiography was primarily done for 221 (83%) patients, and at some point 227 (85%).

Among the 268 VA and PICA aneurysms, most were saccular, but the percentage of fusiform aneurysms was rather high, 28% (Table 5.35). Both saccular and fusiform VA and/or PICA aneurysms were diagnosed in three patients. Of the fusiform aneurysms, two were traumatic. Most saccular aneurysms were located at the junction of the VA and PICA. Fusiform aneurysms were distributed more evenly, the most common locations being the distal PICA and distal VA (Figures 5.33 and 5.34). Aneurysms sized less than 7 mm accounted for 196 (68%). Giant aneurysms were rare, diagnosed in only six patients.

Treatment

Of all the patients, 225 (84%) had at least one aneurysm treated, 209 (78%) at least one VA or PICA aneurysm treated. In two patients, not all VA and PICA aneurysms were treated. The most common reason for conservative treatment was poor condi-

Table 5.32.
Patients with multiple VA or PICA aneurysms (n = 12)

	No. of patients
Multiple distal PICA aneurysms only	7
2 aneurysms	5
3	-
4	1
5	1
2 VA aneurysms	3
1 VA and 1 distal PICA aneurysm	2

PICA, posterior inferior cerebellar artery; VA, vertebral artery

Table 5.33.
Associated aneurysms (patients n = 85, aneurysms n = 161)

	No. of aneurysms (% of all associated aneurysms	**No. of patients** (% of all patients)
MCA	76 (40)	53 (20)
ACA	24 (13)	22 (9)
ICA	34 (18)	26 (10)
BA	19 (10)	15 (6)
VBJ	3 (2)	3 (1)
PCA	5 (3)	5 (2)

ACA, anterior cerebral artery; BA, basilar artery; ICA, internal carotid artery; MCA, middle cerebral artery; PCA, posterior cerebral artery; VBJ, vertebrobasilar junction

Figure 5.33
Locations of saccular aneurysms (n = 202)

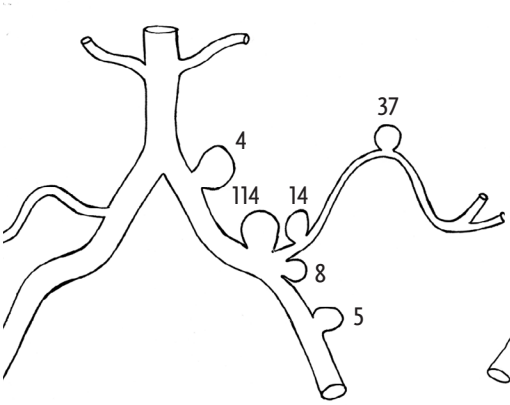


Figure 5.34
Locations of fusiform aneurysms (n = 81)

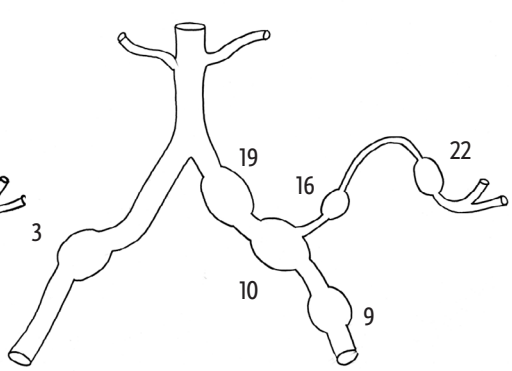


Table 5.34.
Additional aneurysm(s) found later

Age at primary diagnosis	Gender	Reason for primary diagnosis	Reason for angiography later on	Interval between diagnosis (years)	Aneurysms diagnosed first; Location (morphology)	Aneurysms diagnosed later; Location (morphology)	Total no. of aneurysms	No. of certain denovo aneurysms
28	F	SAH	Screening, PCKD	22	PComA (sac)	MCA (sac), PICA (sac)	3	1
29	F	SAH	SAH	50	AcomA (sac), ICA (sac)	PICA (sac)	3	-
32	M	SAH	SAH	10	MCA (sac)	proximal VA (sac)	2	-
35	F	SAH	Screening		PComA	distal PICA (sac)	2	-
35	M	SAH	Suspected aneurysm in MRI	8	VA[a] (fus)	VA[a] (fus)	2	1
36	M	SAH	SAH	16	AcomA (sac), MCA (sac)	PICA (sac)	3	-
44	M	SAH	SAH	6	PICA (sac)	AcomA (sac)	2	1
44	F	SAH	SAH	13	PICA (sac)	PcomA (sac)	2	1
64	F	SAH	SAH	2	MCA (sac), PComA (sac)	PICA (sac), proximal VA	4	1

[a] Exact location not available
AComA, anterior communicating artery; F, female; fus, fusiform; ICA, internal carotid artery; M, male; MCA, middle cerebral artery; MRI, magnetic resonance imaging; PCKD, polycystic kidney disease; PComA, posterior communicating artery; PICA, posterior inferior cerebellar artery; sac, saccular; SAH, subarachnoid hemorrhage; VA, vertebral artery

Table 5.35.
Characteristics of VA and PICA aneurysms

	Ruptured; No. (%)	Unruptured; No. (%)	Total; No. (%)
No of aneurysms	200	84	284
Location			
Proximal VA	9 (5)	5 (6)	14 (5)
VA, no PICA	1 (<1)	2 (2)	3 (1)
PICA	103 (52)	51 (61)	164 (58)
prePICA	6 (3)	2 (2)	8 (3)
postPICA	79 (40)	35 (42)	114 (40)
fusiform, including origin of PICA	6 (3)	4 (5)	10 (4)
Exact location NA	12 (6)	2 (2)	14 (5)
distal PICA	68 (34)	23 (27)	91 (32)
distal VA	15 (8)	8 (10)	23 (8)
VA, exact location NA	4 (2)	3 (4)	7 (2)
Morphology			
Saccular	145 (73)	57 (68)	202 (71)
Fusiform	54 (27)	27 (32)	81 (29)
NA	1 (<1)	-	1 (<1)
Maximal size			
<7 mm	104 (52)	51 (61)	155 (55)
7–14 mm	83 (42)	13 (15)	96 (34)
15–24 mm	7 (4)	10 (12)	17 (6)
>24 mm	-	6 (7)	6 (2)
NA	6 (3)	4 (5)	10 (4)

NA, not available; PICA, posterior inferior cerebellar artery; VA, vertebral artery

tion after SAH (n = 24) (Table 5.36). Most of those treated conservatively because of an anticipated technical difficulty in surgery were diagnosed in the early part of the series.

Among actively treated VA and PICA aneurysms, surgically treated accounted for 198, endovascularly 14, and those treated by both methods 7 (Figures 5.35 to 5.37). Among the 156 saccular aneurysms, 145 (93%) were treated surgically, 5 (3%) by embolization, and 5 (3%) by combined means. Active treatment of fusiform aneurysms was more variable, with those surgically treated numbering 53 (82%), endovascularly 9 (14%), and by both means 2 (3%). Mere clipping was done to 30 (47%) fusiform aneurysms. Endovascular treatment was more common in fusiform than saccular aneurysms (four versus nine patients). The reason for multimodality treatment was failed embolization in two aneurysms, bleeding of a residive aneurysm after clipping in another two, incomplete occlusion, aneurysm perforation and failed embolization, and failed proximal occlusion in one aneurysm each.

Table 5.36.
Reasons for conservative treatment of a VA or PICA aneurysm (patients 59)

	No.
Poor grade after SAH	25
Technical reason	8
Old age / severe general disease	6
The wrong aneurysm interpreted as a ruptured one	3
Reason not specified	3
Rebleeding and death after a negative angiography	3
Small aneurysm	3
Spontaneous thrombosis of the aneurysm and vessel	2
Refused surgery	2
Followed up	1
AVM-related (flow-related) aneurysm	1
Aneurysm missed in angiography	1
Unruptured aneurysm	1

AVM, arteriovenous malformation;
SAH, subarachnoid hemorrhage

Outcome

Angiographic

Postoperative angiography after treatment of a VA or PICA aneurysm was performed on 185 (84%) aneurysms in 182 patients. The angiographic method was catheter angiography in 106 aneurysms (57%), CTA in 74 (40%), and both in 5 (3%) aneurysms. Among these, total occlusion was achieved for saccular aneurysms in 106 (91%), among those clipped 97 (90%); and for fusiform in 33 (63%), among those clipped 22 (79%). The best surgical results occurred in saccular distal PICA aneurysms, with 94% of the aneurysms totally occluded.

After surgery, six aneurysms re-ruptured, and additionally one previously unruptured giant aneurysm ruptured after proximal oc-

Figure 5.35.
Surgical treatment of VA and PICA aneurysms (saccular/fusiform)

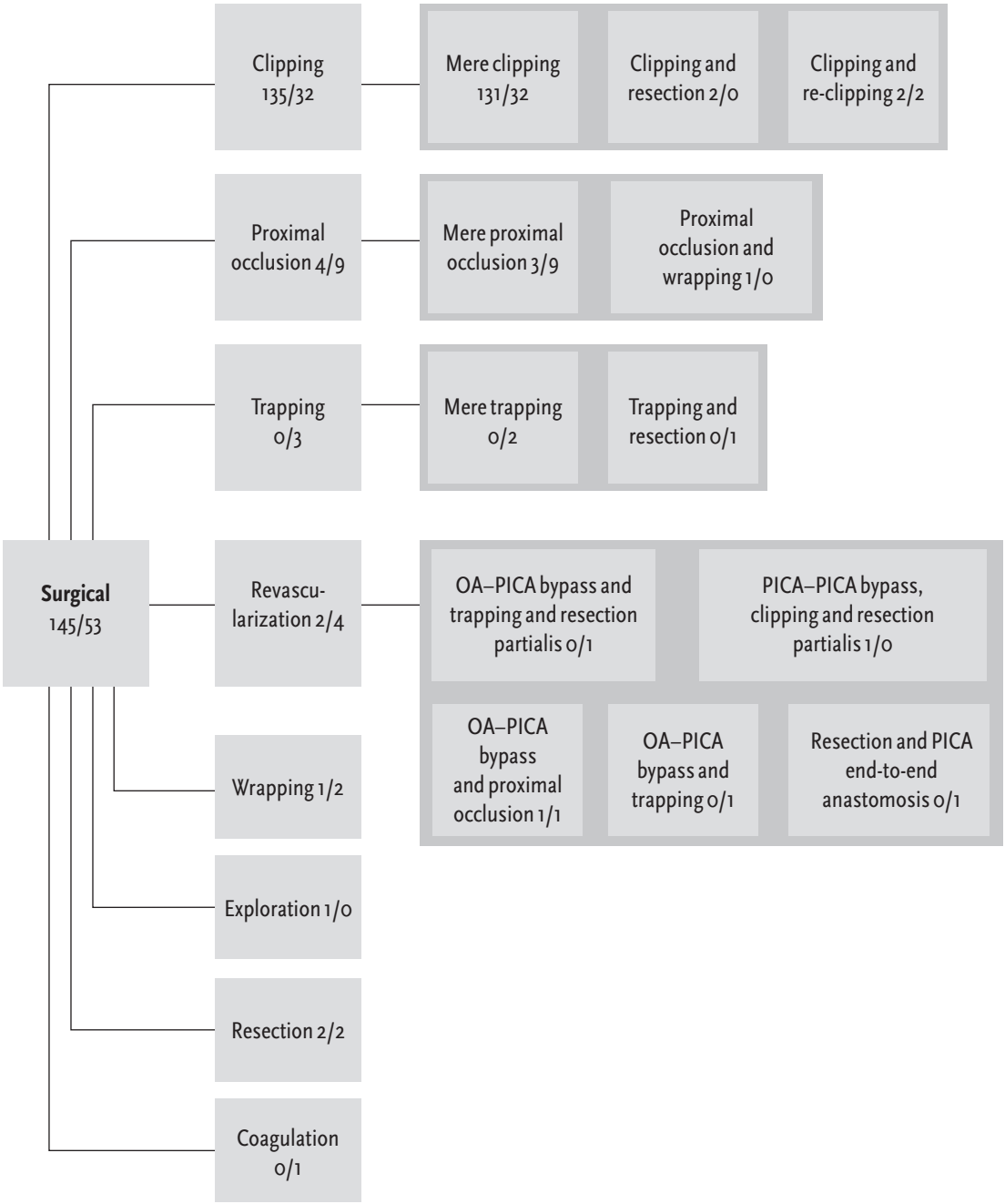


Figure 5.36
Endovascular treatment of VA and PICA aneurysms (saccular / fusiform)

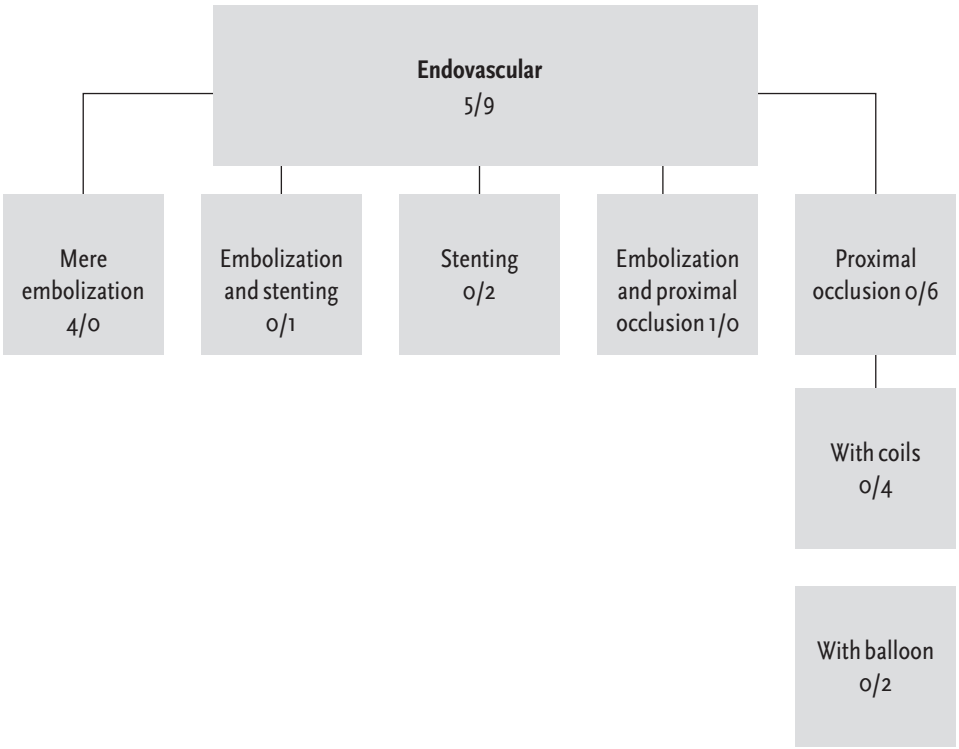


Figure 5.37
Multimodality treatment of VA and PICA aneurysms (saccular / fusiform)

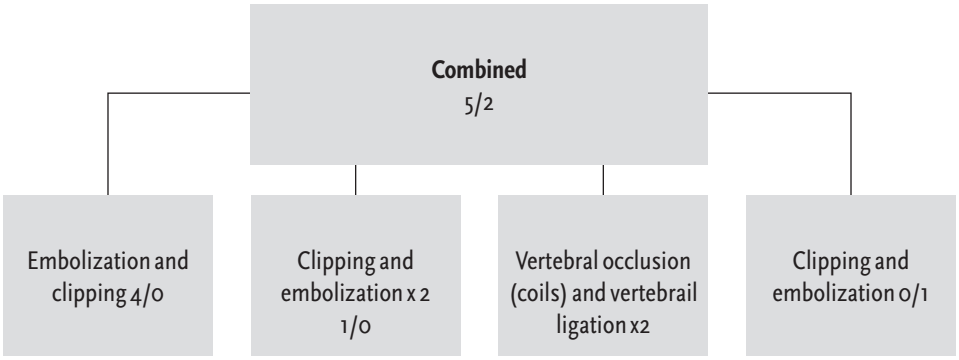


Table 5.37.
Patients/aneurysms with aneurysm rupture after treatment

Aneurysm location	Morphology / earlier ruptured	Primary occlusion	Time from treatment to rupture	Treatment pre/post (year of first treatment)	Final GOS
Distal PICA	Fusiform / ruptured	Total	14 days	clipping (2005) / coiling	3
VA–PICA	Saccular / ruptured	Total	3 years	clipping (2005) / coiling x 2	5
Distal VA	Fusiform / ruptured	Failed	17 days	proximal occlusion (1998) / -	1
VA–PICA	Fusiform / ruptured	Partial	9 days	clipping (2008) / -	1
VA–PICA	Saccular / ruptured	No angiography	13 years	wrapping (1966) / -	1
VA–PICA	Saccular / ruptured	No angiography	0 days	wrapping (1966) / proximal occlusion	1
VA	Giant, saccular / unruptured	No angiography	2 days	Crutchfield / -	1
Proximal VA	Fusiform / ruptured	Partial	6 days	stenting / -	1

GOS, Glasgow outcome scale, PICA, posterior inferior cerebellar artery, VA, vertebral artery

clusion (Table 5.37). One dissecting aneurysm ruptured after stenting. Of the eight patients suffering from aneurysm rupture after treatment, only two survived.

Clinical

Among patients with a minimal theoretical follow-up of one year, within one year 70 (26%) died. The most common cause was severe SAH (Table 5.38). Later, eight patients died for aneurysm-related reasons: three in a rupture of a initially undiagnosed aneurysm (two VA and one AComA aneurysm), one with IVH from shunt removal, one with rupture of a previously conservatively treated incidental aneurysm, one for re-rupture after the patient refused surgery, one from re-bleeding after aneurysm wrapping, one after hemiparesis caused by mass effect of a residive VA aneurysm, and one

of pneumonia after early removal of tracheostomy.

Among those 168 who survived and whose VA and PICA aneurysms were actively treated, 155 (92%) returned to an independent or previous stage of life; 12 (7%) remained dependent on help, three were unable to live at home. One foreign patient was transferred to her home country in poor condition and was lost to follow-up. As mentioned, later, eight of these patients died of aneurysm-related causes.

5.4 Patients with ruptured vertebral or posterior inferior cerebellar artery aneurysms

Patients and aneurysms

Of the 268 patients, those primarily presenting with a ruptured VA or PICA aneurysm accounted for 194, giving an incidence of 5.3% among all ruptured aneurysms. Multiple aneurysms were diagnosed on admission in 47 (24%) patients (Table 5.41); multiple VA or PICA aneurysms were diagnosed in 8 (4%). Anterior circulation aneurysm(s) occurred in 37 (19%) and an additional posterior circulation aneurysm in 8 (4%) patients. Later, two patients were diagnosed with one de -novo aneurysm each, one in the PComA and the other in the proximal VA.

In addition, four patients later suffered a second SAH caused by a previously undiagnosed VA or PICA aneurysm (Table 5.42), and one conservatively treated incidental fusiform VA aneurysm ruptured 3.5 years after diagnosis.

Table 5.43 presents patient characteristics. Women numbered 112 (58%). Median age at diagnosis was 51, with most admitted at age 40 to 49 (Figure 4.41). No difference emerged in age between men and women ($p = 0.27$). Preoperatively good grade (Hunt&Hess grades 1-3) patients numbered 126 (65%) and poor grade (Hunt&Hess grades 3-5) 68 (35%). Among patients with CT, Fisher grade 1 was true of 6 (4%), grade 2 in 12 (7%), grade 3 in 24 (14%), and grade 4 in 128 (75%). Figure 5.42 shows a typical hemorrhage caused by a ruptured PICA aneurysm.

Among the 194 ruptured VA and PICA aneurysms, those saccular accounted for 140 and fusiform 53 (Table 5.44). Morphology of one ruptured aneurysm was unavailable. Figures 5.43 and 5.44 show locations

Table 5.38.
Causes of death within one year after aneurysm diagnosis in 70 patients

	No. of patients
Related to VA aneurysm	
Severe SAH	35
Per / post-treatment bleeding	5
Unruptured aneurysm initially operated on unintentionally	3
Brain infarction	
Spasm	3
Vessel occlusion	2
Pneumonia	6
Pulmonary embolism	1
Myocardial infarction / insufficiency	3
Trauma	2
Unrelated to VA aneurysm	
Severe SAH	6
Cancer	3
Cardiac infarction	1

SAH, subarachnoid hemorrhage;
VA, vertebral artery

Table 5.41.
Associated aneurysms

	No. of aneurysms (% all 108 associated aneurysms)	**No. of patients** (% of all 194 patients)
MCA	31 (48)	26 (13)
ICA	16 (25)	12 (6)
ACA	7 (11)	6 (3)
BA	5 (8)	4 (2)
PCA	4 (6)	4 (2)

Abbreviations as in Table 5.33

Figure 5.41
Age groups at diagnosis

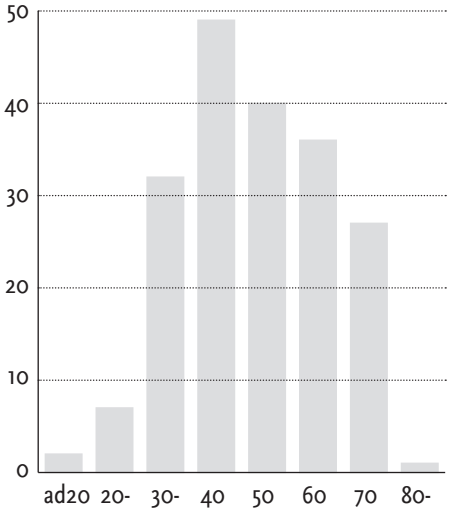


Table 5.42.
Patients with a VA/PICA aneurysm rupture after rupture of another aneurysm

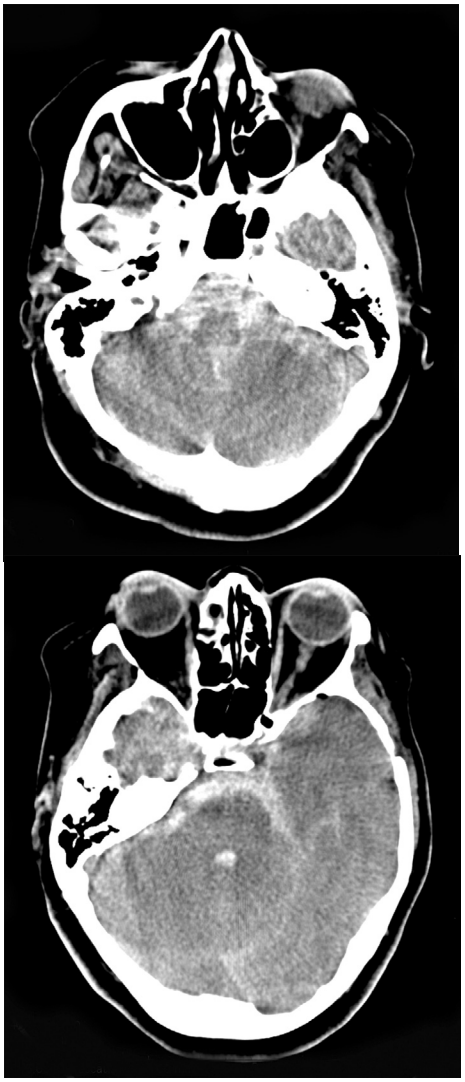
Age at primary diagnosis	Gender	The first ruptured aneurysm	Interval between diagnosis (years)	The second ruptured aneurysm	GOS after the first SAH	GOS after the second SAH
29	F	AComA (sac)	50	PICA (sac)	5	1
32	M	MCA (sac)	10	proximal VA (sac)	5	1
36	M	AComA (sac)	16	PICA (sac)	5	3
64	F	MCA (sac)	2	PICA (sac)[a]	5	4

[a] Definite de-novo aneurysm
AComA, anterior communicating artery; F, female; GOS, Glasgow outcome scale; M, male; MCA, middle cerebral artery; PICA, posterior inferior cerebellar artery; SAH, subarachnoid hemorrhage; VA, vertebral artery

Table 5.43.
Characteristics of those with a ruptured VA or PICA aneurysm

Age	**median (range)**
All	51 (16 – 82)
Male	53 (16 – 82)
Female	51 (17 – 79)
Gender	**No. (%)**
Male	82 (42)
Female	112 (58)
Hunt&Hess grade	
1	33 (17)
2	49 (25)
3	44 (23)
4	34 (18)
5	34 (18)
No. of aneurysms	
1	147 (76)
2	35 (18)
3	7 (4)
4	1 (1)
5	1 (1)
6	2 (1)
7	1 (1)

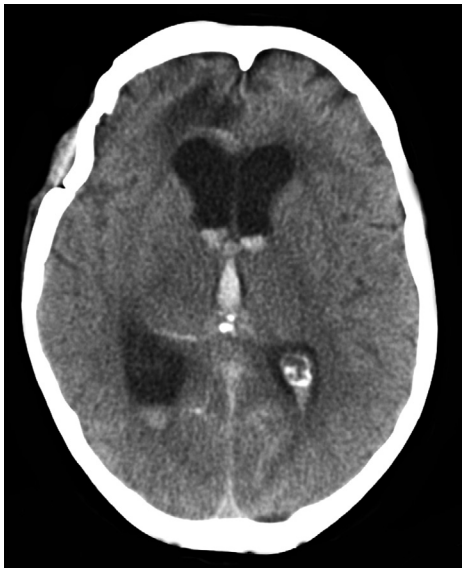
Figure 5.42 a and b.
Typical hemorrhage pattern after rupture of a PICA aneurysm



of the aneurysms in saccular and fusiform groups, showing more men in the fusiform aneurysm group (39% of all men versus 19% of women, p = 0.003). No age difference appeared between patients with saccular and fusiform aneurysms (p = 0.80). Saccular aneurysms were located more often on the left side (67% versus 33%, p = 0.013) and were smaller than those fusiform (p = 0.01).

Comparing patients with a ruptured VA or PICA aneurysm to those with another ruptured aneurysm, the patients were older, and their Fisher grade was higher, due to frequent IVH (Table 5.45). They also experienced re-bleeding more often before treatment (p = 0.001); in further analysis, this was true for distal PICA aneurysms (p < 0.001), but not for VA aneurysms (p = 0.10). Among the patients with a ruptured distal PICA aneurysm, the first rebleeding occurred within 3 hours in 10 (42%), in 3 to 12 hours in 4 (17%), in 12 to 24 hours in 2 (8%), and after 24 hours in 9 (38%). Compared to other ruptured aneurysms, VA and PICA aneurysms were smaller and more often fusiform.

Figure 5.42c
Typical hemorrhage pattern after rupture of a PICA aneurysm



Treatment and angiographic outcome

Of the 31 patients treated conservatively, 15 were of H&H grade 5. Due to an anticipated technical difficulty in surgery six were treated conservatively: four in 1970s, and one in both the 1980s and the 1990s. One fusiform (traumatic) aneurysm and its parent vessel thrombosed spontaneously. The aneurysm was missed in angiography in one patient; in three others, angiography was negative. Vertebral angiographies were not done initially in three patients, and an anterior circulation aneurysm was incorrectly considered to be the ruptured. One patient refused treatment.

Saccular aneurysms

Actively treated ruptured saccular VA and PICA aneurysms numbered 118. Among them, 111 (94%) were treated surgically, and endovascularly 4 (3%) (Figures 5.45 and 5.46). Treated with both methods were 3 (3%): one was clipped after partial embolization, the other after coil extravasation and incomplete occlusion, and one was initially clipped but re-ruptured and was subsequently coiled twice.

In the 1960s, two aneurysms were wrapped. One re-bled the same day, and was treated with proximal occlusion. The other re-ruptured 13 years later, and the patient died. Additionally, in the early part of the series, three aneurysms were treated with proximal occlusion. Later, one patient was treated with partial VA occlusion and OA–PICA bypass. One cortical partially thrombosed distal PICA aneurysm was resected.

Postoperative angiography was performed for 97 (82%) patients; among those with clipped aneurysms for 88 (85%). All

Fig 5.43
Locations of ruptured saccular aneurysms

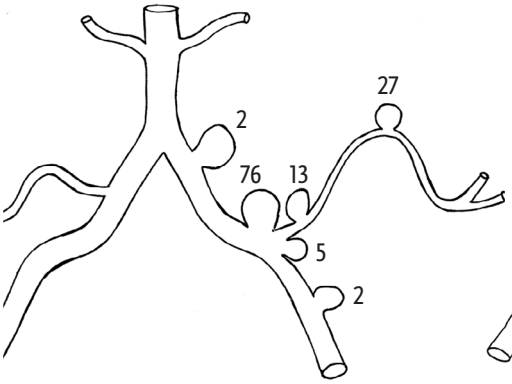
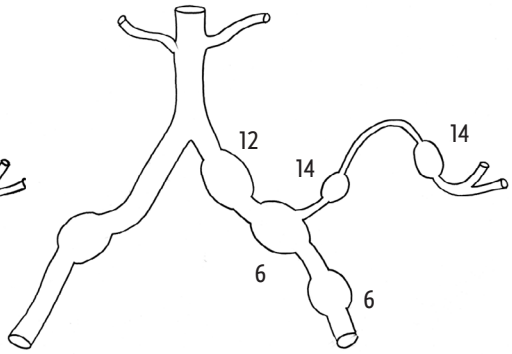


Fig. 5.44
Locations of ruptured fusiform aneurysms



the patients with no postoperative angiography done were admitted before 1998. The angiography method was DSA in 60 (62%), CTA in 36 (37%), and both in 1 (1%) aneurysms. Of all the aneurysms treated, total occlusion was achieved in 91 (78%), in clipped aneurysms in 76 (86%) of cases. After 2000, among clipped aneurysms, total occlusion was achieved in 45 (94%). Three aneurysms re-ruptured after treatment (see Table 5.37).

Fusiform aneurysms

Actively treated fusiform aneurysms numbered 45. Among them, those treated surgically were 39 (87%), and endovascularly 5 (11%), and with both methods, one. One fusiform (dissecting) distal PICA aneurysm re-ruptured after total clipping initially; the residive was coiled together with the PICA.

Postoperative angiography was performed for 39 (87%): 28 (72%) conventional angiography or DSA and 11 (28%) CTA. A total occlusion was verified in 24 (65%), and among the clipped in 17 (73%). Four aneurysms re-ruptured after treatment: one after stenting, one after proximal occlusion, and two after clipping (see Table 5.37).

Clinical outcome

Follow-up time

Mean follow-up time was 7.9 years (median 4, range 0 – 42). Total follow-up time was 1 535 patient years.

Mortality

Among the 60 (31%) who died within one year, most died from severe bleeding (Table 5.46). From postoperative re-bleeding died four (7% of deaths), and from difficulties in correct diagnosis of a ruptured aneurysm seven (12%).

Risk factors for death at one year after active treatment of the ruptured aneurysm were in univariate analysis male gender ($p < 0.001$), old age ($p < 0.001$), and poor H&H grade ($p < 0.001$). Fisher grade did not reach statistical significance ($p = 0.50$), nor did aneurysm size ($p = 0.21$), morphology ($p = 0.09$), rebleeding before treatment ($p = 0.53$), shunt-dependent hydrocephalus ($p = 0.49$) or decade of aneurysm treatment

Table 5.44.
Characteristics of ruptured VA and PICA aneurysms in fusiform and saccular groups [a]

	Saccular; No. (%)	Fusiform; No. (%)	Total; No. (%)
Number of aneurysms	140	53	193
Location			
Proximal VA	2 (1)	6 (11)	8 (4)
VA–PICA junction	93 (66)	6 (11)	99 (51)
prePICA	5 (4)	-	5 (3)
postPICA	76 (54)	-	76 (39)
fusiform, including origin of PICA	-	6 (11)	6 (3)
exact location not available	12 (9)	-	12 (6)
Distal PICA	40 (29)	28 (53)	68 (35)
proximal	13 (9)	14 (26)	27 (14)
distal	27 (19)	14 (26)	41 (21)
Distal VA	2 (1)	12 (23)	14 (7)
VA, no PICA	-	1 (2)	1 (1)
NA	3 (2)	-	4 (2)
Side			
Right	46 (33)	29 (54)	74 (38)
Left	94 (67)	25 (47)	119 (62)
Maximal size			
< 7 mm	77 (55)	22 (42)	99 (51)
7 – 14 mm	56 (40)	25 (47)	81 (42)
15 – 24 mm	2 (1)	5 (9)	7 (4)
> 24 mm	-	-	-
NA	5 (4)	1 (2)	6 (3)

[a] Morphology of one aneurysm not available
NA, not available; PICA, posterior inferior cerebellar artery; VA, vertebral artery

Table 5.45.
Patients with a ruptured VA or PICA aneurysm compared to patients with other ruptured aneurysms

	VA or PICA	Other	p value
Number of aneurysms	167	4329	
Age at diagnosis (years)	median (range)	median (range)	
All	52 (27 – 79)	49 (1 – 92)	0.01
Male	53 (29 – 78)	47 (1 – 88)	
Female	51 (27 – 79)	52 (12 – 92)	
Gender	No. (%)	No. (%)	
Male	70 (42)	1919 (44)	0.58
Female	97 (58)	2410 (56)	
Number of aneurysms			
Single	123 (74)	3042 (70)	0.23
Multiple	44 (26)	1287 (30)	
Aneurysm morphology			
Saccular	118 (71)	4214 (97)	< 0.001
Fusiform	49 (29)	93 (2)	
NA	-	22 (1)	
Maximal size			
< 7 mm	84 (50)	1363 (31)	< 0.001
7 – 14 mm	70 (42)	1967 (45)	
15 – 24 mm	7 (4)	384 (9)	
> 24 mm	-	60 (1)	
NA	6 (4)	555 (13)	

Fisher grade			
1	6 (4)	237 (6)	< 0.001
1	11 (7)	394 (9)	
3	24 (14)	1256 (29)	
4	119 (71)	1687 (39)	
NA	-	755 (13)	
IVH			
No	46 (28)	2483 (57)	< 0.001
Yes	117 (70)	1039 (24)	
NA	2 (1)	807 (19)	
ICH			
No	153 (92)	2354 (54)	< 0.001
Yes	5 (3)	1221 (28)	
NA	9 (5)	754 (17)	
Hunt&Hess grade			
1	23 (14)	809 (19)	0.23
2	44 (26)	1211 (28)	
3	40 (24)	867 (20)	
4	29 (17)	757 (18)	
5	31 (19)	565 (13)	
NA	-	120 (3)	
Rebleeding before treatment			
No	110 (66)	3318 (77)	0.001[a]
Yes	55 (33)	922 (21)	
NA	2 (1)	89 (2)	

[a] No difference between groups in time from rupture to occlusion of aneurysm (p = 0.52)
ICH, intracerebral hemorrhage; IVH, intraventricular hemorrhage; NA, not available;

Figure 5.45.
Treatment of ruptured saccular aneurysms
• Including one string ligation
• patient with wrapping+prox occl. entered twice

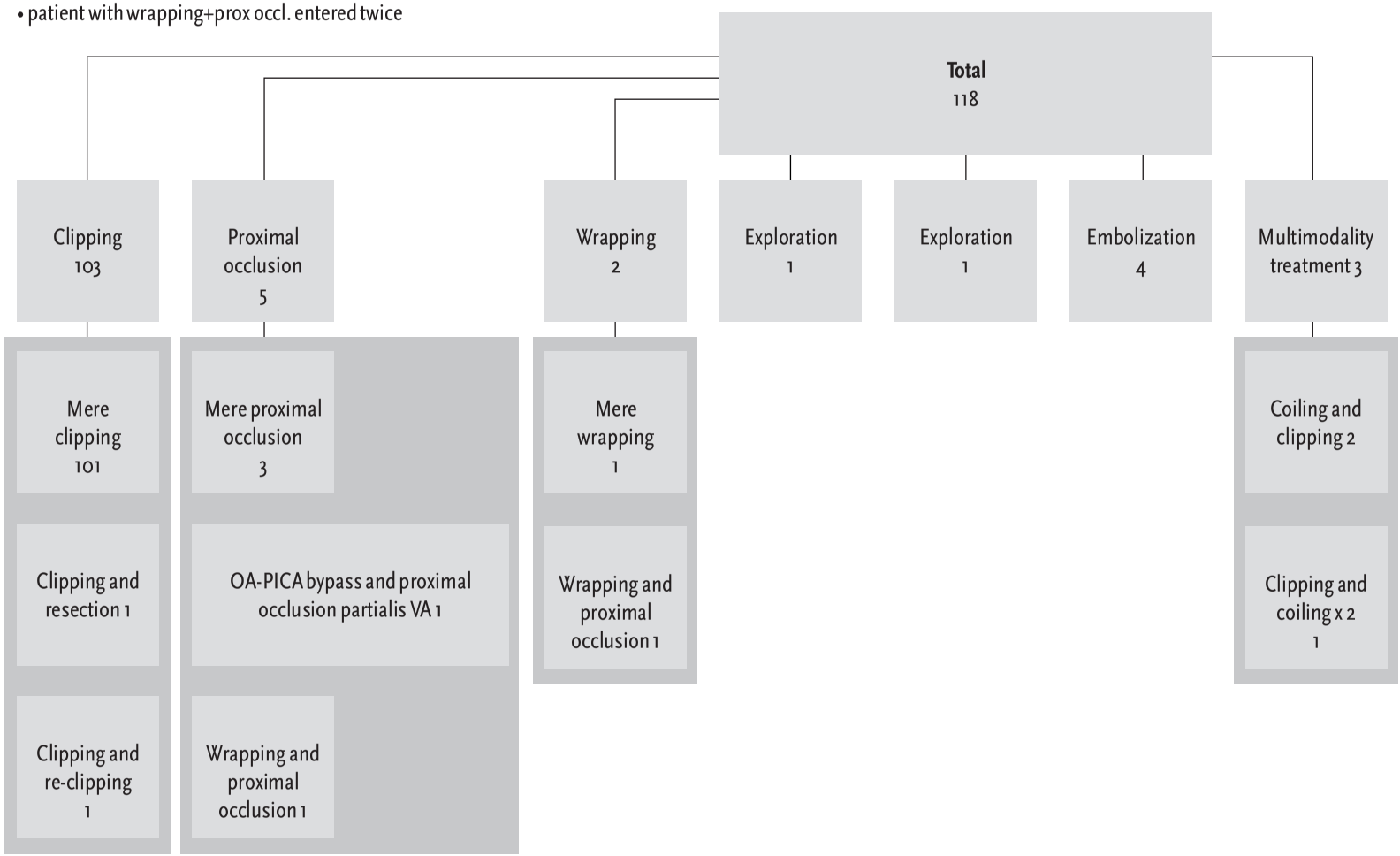


Figure 5.46
Treatment of ruptured fusiform aneurysms

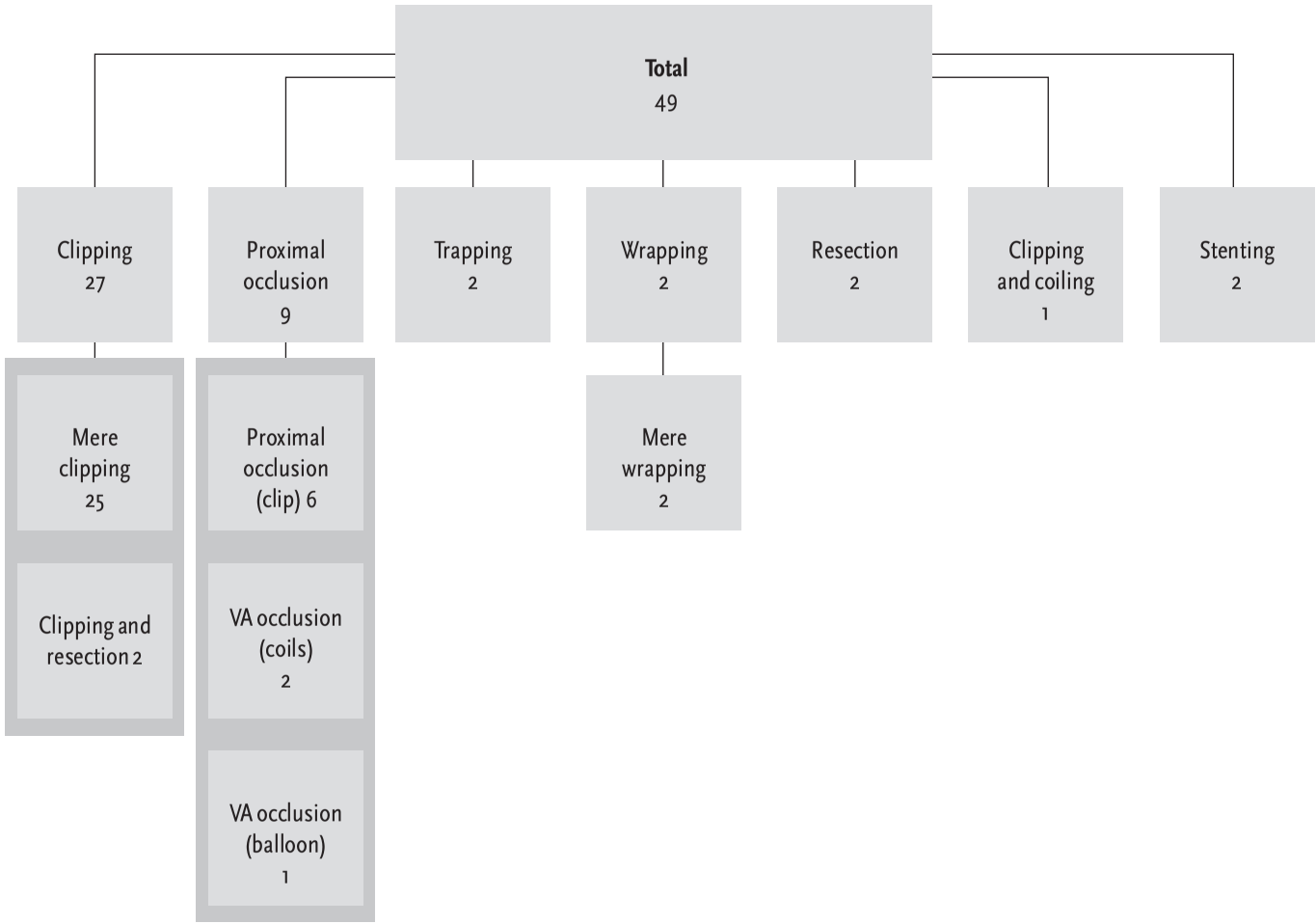


Table 5.46.
Causes of death within one year after diagnosis of a ruptured VA or PICA aneurysm (n = 60)

	No. of patients
Severe SAH	35
Per / post-treatment bleeding	4
Unruptured aneurysm treated first unintentionally	3
Brain infarction	
Spasm	3
Vessel occlusion	1
Pneumonia	6
Cardiac infarction / insufficiency	4
Trauma	2
Pulmonary embolism	1
Cancer	1

SAH, subarachnoid hemorrhage

Table 5.47.
Complications among actively treated patients

	No. (%)
Brain infarction	43[a] (29)
Aneurysm rebleeding	4 (2)
Postoperative hematoma	7 (4)
Shunt dependent hydrocephalus	38 (23)
Infection	
Meningitis	19 (12)
Wound infection	6 (4)
Sepsis	14 (9)
Suspected pneumonia	76 (47)
Urinary tract infection	46 (28)
Peritonitis	2 (1)
Cardiac infarction	4 (2)
Pulmonary embolism	2 (1)
Deep venous thrombosis	2 (1)
Gastrointestinal bleeding	3 (2)

[a] No CT, MRI or autopsy in 15 patients

(p = 0.24). In multivariate analysis, independent risk factors were male gender (OR 1.917, CI 2.417–9.148), old age (OR 0.065, CI 0.898–0.977), high H&H grade (OR 1.452, CI 1.567–11.637), and larger aneurysm size (OR 2.413, CI 1.126–110.710).

Complications

Complications among patients with a treated ruptured aneurysm are in Table 5.47. After treatment of the aneurysm, at least angiographic vasospasm was diagnosed in 44 (27%) patients. Infarction was diagnosed in 43 (26%); 15 (9%) had no a post-operative scan or autopsy. One patient required suboccipital craniectomy due to a cerebellar infarction; that patient returned to part-time work. One cerebellar infarction was resected but the patient did not recover. One patient underwent both infarction resection and decompressive craniectomy; he returned to independent life. Ventriculostomy was done for 51 (26%) patients; those who developed shunt-dependent hydrocephalus numbered 38 (20%).

Four patients suffered from re-bleeding of a treated aneurysm. One operation was interruptured due to aneurysm rupture when

Table 5.48.
Cranial nerve deficits

	Pre-operative, No. (%)	Post-operative; No. (%)	Total recovery	Partial recovery	No recovery	Dead
Laryngeal palsy	2 (1)	52 (32)	22	20	2	8
Diplopia	8 (5)	24 (16)	16	2	2	4
Facial nerve deficit	-	5 (3)	1	-	2	2
Trigeminal deficit	-	2 (1)	-	-	2	-
Hearing loss	-	1 (1)	1	-	-	-
Accessory nerve deficit	-	5 (3)	3	-	2	-
NA	53 (33)	14 (9)				

NA, not available

opening the dura. A postoperative hematoma was diagnosed in seven patients, four of these caused by ventriculostomy or shunt insertion. Three of the hematomas required evacuation.

Culture-positive meningitis was diagnosed in 19, and among them, 2 also had a wound infection. Mere wound infection was diagnosed in four patients. No lambeau removal was needed due to infection. Septicemia was diagnosed in 15; 8 of them also having meningitis. Pneumonia was at least suspected in 76 patients, and urinary tract infection in 46. Those diagnosed with any infection numbered 106 (65 %).

Additionally, the two patients with pulmonary embolism also had a deep venous thrombosis. Of the three patients with gastrointestinal bleeding, one had bowel perforation. Additionally, one patient suffered from peritonitis, but with no perforation evident.

Cranial nerve deficits

Among the 155 patients operated for a ruptured aneurysm, on admission a cranial nerve deficit was present in 10 (6%) patients who could be examined; due to their poor condition, 53 (33%) patients could not be examined properly (Table 5.48). After surgery on the aneurysm, 50 (32%) had a deficit; those not examined accounted for 14 (9%). Postoperatively, laryngeal palsy was most common, in 36% of patients examined. Tracheostomy was necessary for 33 (23%). Most patients recovered; two were left with a tracheostomy and PEG, and one died of pneumonia after early removal of the tracheostomy in a hospital elsewhere. Among those endovascularly treated, only one patient suffered from a cranial nerve deficit: it caused him diplopia.

Overall outcome

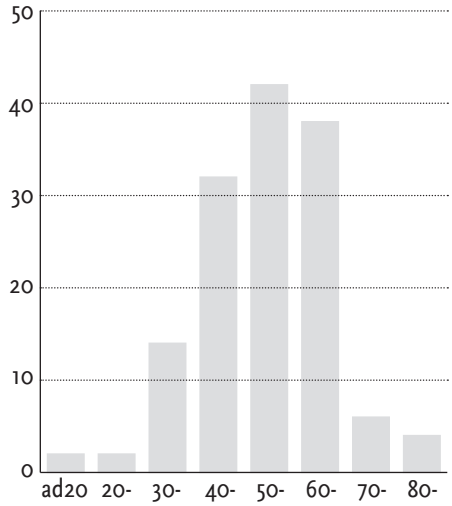
Within one year, 60 patients died. Among those who survived for more than one year of follow-up, those recovering to their former or an independent state of life numbered 123 (92%); 10 (9%) patients remained

Table 5.51.
The 69 patients with only unruptured VA and or PICA aneurysms

Gender	No. (%)
Male	27 (39)
Female	43 (61)
Age at primary diagnosis	**median (range)**
All	56 (16 – 85)
Decade of treatment	**No. (%)**
1960s	3 (4)
1970s	2 (3)
1980s	3 (4)
1990s	8 (12)
2000 -	54 (77)
Presentation	**No. (%)**
SAH from another aneurysm	25 (42)
Mass effect	9 (13)
Ischemia	3 (4)
Ruptured AVM/AVF	1 (1)
Incidental	32 (46)

Abbreviation as in Table 5.31

Fig 5.51.
Age distribution of the same 69 patients



dependent on help: one poor-grade patient died 13 months after bleeding, six returned home, three were unable to live at home. Additionally, one poor-grade patient was transferred to his home country in poor condition and was lost to follow-up.

Patients with unruptured vertebral artery or posterior inferior cerebellar artery aneurysms

Overview

Among the total of 268 patients, 70 (26%) were diagnosed with only unruptured VA or PICA aneurysm(s), or both. Most were incidental findings (n = 32), or diagnosed in addition to a ruptured aneurysm elsewhere (n = 25) (Table 5.51). Table 5.51 shows patient characteristics; additionally, age distribution is shown in Figure 5.51. After 2000, the number treated amounted to 77% of the patients.

Ruptured aneurysm in another location

Patients and aneurysms

We had 25 patients initially diagnosed with a ruptured aneurysm outside the VA and PICA; additionally, 4 patients were primarily diagnosed with a ruptured aneurysm else-

Table 5.52.
Patient characteristics of those 25 with a ruptured aneurysm outside the VA and PICA

Age	**median (range)**
All	52 (27 – 71)
Male	56 (44 – 63)
Female	51 (27 – 71)
Gender	**No. (%)**
Male	6 (25)
Female	19 (75)
Decade of admission	
1960s	1 (4)
1970s	1 (4)
1980s	2 (8)
1990s	3 (13)
2000 -	18 (71)
Hunt&Hess grade	
1	4 (4)
2	7 (25)
3	6 (25)
4	2 (8)
5	6 (25)
No. of aneurysms	
1	-
2	8 (33)
3	8 (33)
4	4 (17)
5	2 (8)
6	2 (4)
7	1 (4)

Table 5.53.
Locations of all the aneurysms

	No.
Location of the ruptured aneurysm	
MCA	8
BA	5
ICA	4
ACA	4
VBJ	2
Locations of other aneurysms	
MCA	19
ICA	7
BA	4
ACA	2
VBJ	-
VA	19
distal PICA	10

ACA, anterior cerebral artery; BA, basilar artery; ICA, internal carotid artery; MCA, middle cerebral artery; PICA, posterior inferior cerebellar artery; VA, vertebral artery; VBJ, vertebrobasilar junction

where, and later with SAH from a primarily undiagnosed aneurysm in the VA or PICA (see Table 5.34).

Patient characteristics are shown in Table 5.52. There was female predominance (19 versus 6), but no difference in age distribution emerged between the genders ($p = 0.45$). Good grade (H&H grades 1–3) accounted for 17 (67%).

The location of the ruptured aneurysm was the MCA in 8 (32%) patients, and in the posterior circulation in 7 (28%) patients (Table 5.53). All the ruptured aneurysms were saccular. One patient had five small saccular distal PICA aneurysms together with a posterior

Table 5.54.
VA and PICA aneurysms associated with ruptured aneurysms elsewhere

	No.
Locations of the VA / PICA aneurysm	
proximal VA	1
PICA	
prePICA	1
postPICA	15
distal PICA	10
exact location NA	2
distal VA	-
Morphology	
Saccular	25
Fusiform	3
Maximal size	
<7	27
7–14	2
15–24	-
>24	-

Abbreviations as in Table 5.44

Table 5.55.
Patients with AVMs

Age	Gender	Cause of bleeding	Total no. of aneurysms
44	F	AVM	2
69	F	Aneurysm (prePICA)	3
62	M	Aneurysm (distal PICA)	3
57	F	Aneurysm (BA)	6
63	F	Aneurysm (distal PICA)	1

AVM, arteriovenous malformation; BA, basilar artery; M, male; F, female; PICA, posterior inferior cerebellar artery

fossa AVM. Otherwise no multiple VA and/or distal PICA aneurysms were found. Two distal PICA aneurysms and the proximal VA aneurysm were fusiform (Table 5.54). No aneurysm was diagnosed over 15 mm of a size.

Treatment

The ruptured aneurysm was treated in 22 patients, and VA or PICA aneurysm(s) was treated in 12. In one patient with an AVM and multiple small distal PICA aneurysms, the two largest aneurysms were clipped; others disappeared after removal of the AVM. The most common reason for conservative treatment of the VA or PICA aneurysm was poor condition after SAH in nine patients.

An additional 11 aneurysms were clipped, after which one residual aneurysm was embolized twice.

Outcome

Of the 12 patients with actively treated VA or PICA aneurysm, all recovered to the previous or an independent state of life. Six returned to work.

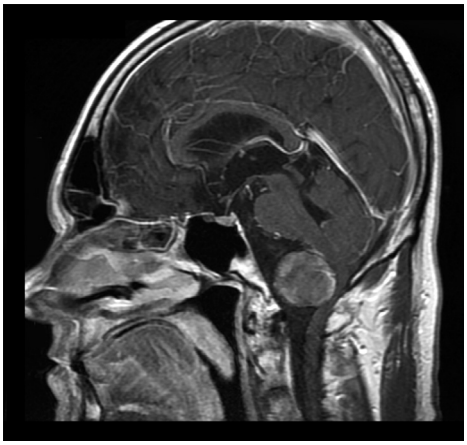
Postoperatively, three patients had a hoarse voice without swallowing difficulties. All recovered fully. One patient with a distal PICA aneurysm had a postoperative accessory palsy that did not resolve.

Table 5.56
Characteristics of those with mass effect caused by the aneurysm

Age	Gender	Location; morphology	Size (mm)	Symptom	Treatment	GOS	Occlusion	Year of treatment
16	F	NA saccular	50	Ataxia, dysphagia, dysartria, nystagmus	Proximal occlusion	1	No angio-graphy	1965
54	M	postPICA saccular	50	Oculomotor-, facial-, and accessory-nerve paresis, dysphagia, dysartria, dizziness	Clipping and resection	5	No angio-graphy	1984
55	F	distal VA fusiform	20	Trigeminal and abducens paresis, hearing loss	Conservative	NA	-	1994
42	M	NA saccular	34	Hemifacial spasm	Resection	5	Total	2001
73	F	distal PICA saccular	60	Dysmetria	Resection	3	Total	2004
51	M	distal VA fusiform	36	Tetraparesis	Trapping and partial resection	5	No angio-graphy	2008
81	F	postPICA saccular	20	Primarily hemifacial spasm: later hemiparesis, oculomotor paresis	Clipping and re-clip-pimg	1	Total	2006
58	M	distal PICA fusiform	44	Balance disturbance	OA–PICA bypass, trapping and resection	5	Total	2010
57	F	postPICA saccular	28	Hemiparesis	PICA–PICA bypass, clipping and resection	4	Total	2011

F, female; GOS, Glasgow outcome score; M, male; NA, not available; OA, occipital artery; PICA, posterior inferior cerebellar artery; VA, vertebral artery

Fig 5.52.
MRI of a patient with partially thrombosed giant VA aneurysm



Ruptured arteriovenous malformation

Among the five patients with AVM in the series, one presented with rupture of an AVM (Table 5.55). Along with extirpation of the ruptured AVM, the postPICA aneurysm was clipped, and the aneurysm in the tonsillomedullary segment of PICA was treated by proximal occlusion. Neither aneurysm was visible in the postoperative DSA. Postoperatively, the patient had a laryngeal palsy and diplopia; both symptoms resolved, and the patient returned to work.

Mass effect

Patient and aneurysm characteristics

Nine patients were diagnosed because of cranial nerve and brainstem compression caused by the aneurysm (Table 5.56). Patient age ranged from 16 to 81. Those originating from the VA accounted for seven (with exact location of the origin in the VA unknown in two), and those from the distal PICA two. Fusiform aneurysms accounted for three; each aneurysm was partially thrombosed. Figure 5.52 demonstrates a giant aneurysm with brainstem compression.

Table 5.57.
Characteristics of patients with incidental or screened aneurysms (n = 32)

Diagnosis	**No. (%)**
Incidental	28 (88)
Screening	4 (13)
Age	**median (range)**
All	58 (34 – 85)
Male	60 (34 – 85)
Female	56 (37 – 65)
Gender	**No. (%)**
Male	15 (47)
Female	17 (53)
Decade of admission	**No. (%)**
1970s	1 (3)
1970s	1 (3)
1980s	-
1990s	2 (6)
2000 –	28 (88)
No. of aneurysms	**No. (%)**
1	21 (66)
2	3 (9)
3	5 (16)
4	-
5	2 (6)
6	-
7	1 (3)

Treatment

The various treatment methods are in Table 5.56. One aneurysm was re-clipped; the patient suffered primarily from hemifacial spasm, and the aneurysm was clipped with a small neck remnant. Later, at the age of

Table 5.58.
Associated aneurysms in patients with incidental VA aneurysms

	No. of aneurysms	No. of patients
ICA	1	1
MCA	14	8
ACA	5	5
PCA	1	1
BA	5	3
VBJ	1	1

Abbreviation as in table 5.53

85, she developed a hemiparesis caused by a 27-mm residive aneurysm. Another complex VA aneurysm was treated conservatively because of assumed technical difficulties in the beginning of the 1990s.

Outcome
Two patients died: the 85-year-old with a hemiparesis from the residive aneurysm did not survive, and a young patient suffered from aneurysm rupture a few days after VA occlusion in 1965. The only patient who received conservative treatment is still alive. Among the rest, four patients returned to work, one to independent life, and one remained dependent on external help.

Among those treated actively, all but one had a laryngeal palsy postoperatively, three requiring a tracheostomy. Two died, one had a permanently hoarse voice, the remaining five made a full recovery.

Table 5.59.
VA and PICA aneurysms associated with ruptured aneurysm elsewhere

	No.
Locations of the VA / PICA aneurysm	
proximal VA	2
PICA	
prePICA	-
postPICA	17
including origin of PICA	3
distal PICA	2
distal VA	5
VA, no PICA	2
exact location in VA NA	1
Morphology	
Saccular	21
Fusiform	11
Maximal size	
< 7 mm	15
7–14 mm	8
15–24 mm	6
> 24 mm	-

Abbreviations as in Table 5.44

Ischemia
Patient and aneurysm characteristics
Three patients presented with ischemia: two men and one woman, aged 40 to 60. Two aneurysms were located in distal VA and one in distal PICA. All were fusiform. One aneurysm and the VA were thrombosed at diagnosis and another partially thrombosed.

Treatment

The patient with the totally thrombosed VA and aneurysm was treated conservatively. The partially thrombosed aneurysm was treated with VA balloon occlusion, after which the VA and the aneurysm thrombosed totally. The proximal distal PICA aneurysm was trapped after OA–PICA bypass.

Outcome

All the patients returned to work. The patient treated with VA balloon occlusion already had a laryngeal palsy before the treatment; the palsy resolved partially during follow-up, as also did his facial nerve deficit.

Incidental aneurysms

Patient and aneurysm characteristics

In 28 patients the aneurysm was a mere incidental finding. Another four patients were diagnosed because of screening, which was performed on three suspected familial aneurysm carriers, and on one of whom also having PCKD.

The characteristics of these 32 patients are presented in Table 5.57. No statistical significance emerged for age by genders ($p = 0.19$). Nearly all (80%) patients were diagnosed after 2000. Multiple aneurysms were diagnosed in 11 (32%) patients (Table 5.58). Associated aneurysm(s) were found in the anterior circulation in all the 11 patients. An additional posterior circulation aneurysm outside VA and PICA was diagnosed in four patients. No patient had multiple VA or PICA aneurysms or both a VA and PICA aneurysm.

Most aneurysms were located at the junction of the VA and PICA, postPICA aneurysms accounting for 17 (53%). Half of the aneurysms were less than 7 mm in size, and a third were fusiform. (Table 5.59)

Table 5.60.
Treatment of incidental VA and PICA aneurysms

	Saccular	Fusiform	Total
Clipping	9	3	12
Conservative	9	2[a]	11
Proximal occlusion			
Clip	-	2	2
Coil	-	2	2
Coiling (attempted), clipping x 2		1	1
Stenting and coiling	-	1	1
Embolization and proximal occlusion with coils	-	1	1
OA–PICA bypass and partial occlusion of VA with a clip	-	1	1
Resection and PICA end-to-end anastomosis	-	1	1

[a] Later, one aneurysm was clipped after its rupture
OA, occipital artery; PICA, posterior inferior cerebellar artery; VA, vertebral artery

Treatment and angiographic results

Patients with an actively treated VA or PICA aneurysm numbered 22 (69%). Among the ten conservatively treated aneurysms, one is still followed up. Due to their old age, conservative treatment was the choice for three, and due to a small aneurysm for one; one patient was treated conservatively in the 1970s because of anticipated technical problems, and in the 1960s, unruptured aneurysms were not treated.

Table 5.60 shows treatment methods for the aneurysms. All the nine saccular aneurysms were clipped; eight were totally occluded; one had no postoperative angiography

Treatment of fusiform aneurysms was more diverse. Three patients were treated by endovascular means. The aneurysm treated by stenting and coiling resulted in near-total occlusion, and the one treated with internal coiling was totally occluded. An endovascular occlusion of the VA by a large fusiform aneurysm failed, and the VA was closed surgically; finally this aneurysm thrombosed. Among patients treated with proximal occlusion, this was the only aneurysm proven to be thrombosed.

One distal, large, partially thrombosed PICA aneurysm was resected, and the ends of the PICA sutured together. Another patient with a fusiform distal VA aneurysm was treated with an OA–PICA bypass, and the VA was occluded with a clip that was partially left open with the aid of absorbable string.

Outcome

Postoperatively, nine patients suffered from cranial nerve deficits. Six had a laryngeal palsy; among these, one needed a tracheostomy. One died, another was left with a hoarse voice and slight swallowing difficulties; all the others made a full recovery. Two patients had accessory nerve paresis: one recovered totally, and the other partially. One patient had, postoperatively, facial paresis and poor hearing; the facial paresis resolved but the hearing did not improve.

Fifteen patients returned to work and one to the previously independent life. A 65-year-old patient received help in housekeeping after the surgery. The patient with thrombosed PICA anastomosis died.

One conservatively treated incidental fusiform distal VA aneurysm ruptured three years after the diagnosis. The aneurysm was clipped, but the patient did not survive. ❦

Discussion

This retrospective series comprises 268 patients with 288 aneurysms in the VA and distal PICA. The patients were treated in Helsinki University Central Hospital, Finland, from 1961 to 2011. The present series describes characteristic features of the aneurysms, treatment, and outcome. This series is the largest reported up until now.

Incidence

Few reports have been involved VA and distal PICA aneurysms without significant selection bias. Yamaura et al. in 1988 reported VA aneurysms to comprise around a third of all posterior circulation aneurysms (250). Among all saccular aneurysms, Huttunen et al. reported those located in the VA and distal PICA to comprise 2.4% (82). In our series after the year 2000, among all aneurysm patients, 5.1% had a VA or distal PICA aneurysm; among ruptured aneurysms, those of the VA or distal PICA comprised 3.9%. Compared to earlier figures, ours are somewhat larger. The difference may be explained by a four-vessel angiography, MRA, or CTA for all our aneurysm patients, and in addition our material is non-selected, including also conservatively treated poor-grade patients. The shortcoming of our figures is the absence of data on those who died before reaching the neurosurgical unit.

Anatomic features

Location

As in the earlier reports on VA aneurysms (11, 47, 250), also in our series the most frequent aneurysm location was the VA–PICA junction. Aneurysms in the distal VA are more common than in the proximal VA: in the series of Drake et al., proximal VA aneurysms numbered only 4, and distal 41 (47). In our series the difference was not that distinct: in the proximal VA 14 aneurysms and in the distal

VA 23 aneurysms. Among distal PICA aneurysms, in all reports except one the majority of aneurysms were located in medullary segments (78, 131, 167, 190, 227). This was confirmed also by our series, with 74% located there, 17% in the telovelotonsillar segment, and 9% in the cortical segment.

Morphology

Many other series categorize the VA and distal PICA aneurysms as saccular, fusiform, and dissecting (11, 19, 47, 250). The number of non-saccular aneurysms in the VA is high, ranging from 13 to 48% (11, 47, 250), a wide range perhaps related to differing definitions for non-saccular aneurysms among series. Due to controversies over definitions, and lack of MRI in SAH patients leading to difficulties in diagnosing classical signs of dissection, we limited our categorization into saccular and fusiform. We classified as fusiform those aneurysms with a neck larger than the aneurysm's width; in our series the proportion of fusiform aneurysm in the VA was 22% and in the distal PICA 43%. Additionally, among saccular distal PICA aneurysms, classical saccular aneurysms at the branching point of an artery accounted for only 10%.

Anatomy of VA–PICA aneurysms in CTA

Even if DSA is still the gold standard for aneurysm imaging, compared to DSA, CTA is fast, cheap, non-invasive, more readily available, and superior in visualization of the skull base and calcifications (233). The literature provides, however, only one study on PICA aneurysms shown in CTA (248).

Anatomy of the VA and PICA is highly variable, with elongation on VA, tortuous course of both arteries, and variable location the PICA origin (136). As in earlier series (93, 104), in ours the left VA was larger than the right, with median diameter on the left side 2.8 mm and on the right 2.1 mm. Hypoplasia is also more common for the right VA, as it is also for the right PICA (93, 104), The reason for this asymmetry is not known.

Due to variations in anatomy of the VA and PICA, the location of a PICA aneurysm in the posterior fossa is highly variable. Several reports show a PICA aneurysm located extracranially or on the side of the skull contralateral to the origin of the parent artery (47, 221, 248); in our series, one aneurysm was located extracranially and four aneurysms were on the side contralateral to the parent artery. Distances of the aneurysm from the foramen magnum and midline are important for planning the site of the craniotomy and the extent of drilling the skull base (18, 115). Within our series, the distance of the aneurysm neck from the foramen magnum ranged from 1 mm below it to 31 mm above, and from the midline, 8 mm contralateral to 14 mm ipsilateral. Even if the size of jugular tubercles ranges within a few millimeters, noticing it is important in the tight space of the posterior fossa. For example, too much of lateral tilting of the head can cause an aneurysm to be hidden behind a large tubercle.

Additionally, patients with distal PICA aneurysms have a high incidence of different vascular variations, anomalies and lesions (167, 174, 227), as confirmed also by our series, where only 19% patients had no vascular deviation. The most common findings were multiple aneurysms (26%), hypoplastic VA (42%), and hypoplastic PICA (22%).

Treatment

Challenges related to treatment of VA and distal PICA aneurysms result from anatomical relationships of the aneurysm to the vasculature, the posterior cranial fossa, brainstem, and cranial nerves, as well as its morphology and etiology. Despite remarkable advances since the first successful direct occlusion of an aneurysm in the posterior circulation in 1946, treatment of dissecting VA aneurysms in particular still needs improvement.

Surgical treatment and radiological outcome

The complexity and variance of the lesions, as well as passionate search for the best solution, is reflected in the great variety of treatment methods and approaches. Techniques more recently reported include clipping (167, 227), trapping (35, 87, 204), wrapping (204), proximal occlusion (35, 87, 204) with or without different bypass procedures. In contrast to endovascular surgery, microsurgical occlusion of VA and distal PICA aneurysms meets challenges, especially the necessity of avoiding cranial nerve deficits. Regarding of the vasculature, for classical aneurysms at the VA–PICA junction, the PICA is seldom compromised. Conversely, for dissecting VA aneurysms, total and stable occlusion with preservation of flow is demanding. In proximal occlusion of fusiform and dissecting VA aneurysms, good outcomes are reported (47). Subsequent ruptures, however, are detectable, both by others (102, 109) and in our series. One cause of differing success may lie in the timing of treatment: re-rupture rate is highest within the first day after the first ictus, and decreases thereafter (147, 247). To our knowledge, no randomized trial covers the best treatment of dissecting VA. Based on the literature and our experience, at least consider trapping the whole diseased segment, with a bypass if needed.

Beside fusiform aneurysms, another challenge are giant aneurysms. Many times these patients are already invalidized by brainstem and cranial nerve deficits (47). Compared to endovascular surgery, microsurgery, if needed, has the advantage of debulking the mass of the aneurysm. Yet the challenge is operating in the posterior fossa made even tighter by the aneurysm. Another advantage of surgical treatment is the possibility of doing a bypass if blood flow is compromised. Several different bypass options exist, the most common being PICA–PICA and OA–PICA (9, 34, 123, 129, 161, 212).

In the series of Drake et al., 86% of the aneurysms became totally occluded, and 4% had a neck remnant (47). In a more recent series, total occlusion was achieved in 93% of aneurysms; in this surgical series, dissecting VA aneurysms were referred to endovascular surgery (204). In our series, total occlusion was achieved in 90% of clipped saccular and in 79% of clipped fusiform aneurysms, results consistent with those of other series.

Postoperative angiography after treatment of VA or PICA aneurysm was performed on 181 patients: of the investigations, 57% were by DSA/conventional angiography and 40% by CTA, with 3% imaged by both methods. Total occlusion was achieved in saccular aneurysms in 106 (91%) (among those clipped 97 (90%)), and in fusiform ones in 34 (63%) (among those clipped 22 (79%)). The best results were in saccular distal PICA aneurysms with all but one totally occluded. Even DSA was performed if in doubt about a neck remnant after CTA, some neck remnants may have been missed in cases without titanium clips used.

Endovascular treatment and radiological outcome

Although the present series has a strong surgical focus, within the last ten years most published series are purely endovascular (for the recent published series, see Table 2.33). Unfortunately, the emphasis on surgery in the present series makes comparison between surgical and endovascular treatment impossible.

Endovascular treatment escapes the risk of causing cranial nerve deficits. It is, however, challenged by preserving the flow in the PICA, as the PICA often courses inferiorly at a sharp angle when leaving the VA. In distal PICA aneurysms, aneurysms with well-defined necks can be embolized, but fusiform distal PICA aneurysms present a

challenge. In aneurysms distal to the choroidal point, occlusion of the artery can be considered (92). Using stents in a small-caliber artery is for the moment not recommended (5). Reducing the mass effect is uncertain in the use of endovascular techniques in complex aneurysms (86).

As treatment of ruptured dissecting VA aneurysms have been challenging by both endovascular and exovascular means, in the future, flow-diverters may become a treatment of choice. For the moment, however, series on their use in these lesions are scarce (25, 182); especially series on ultra-early treatment of the dissecting aneurysms would be essential.

Angiographic results in a recent study on endovascular PICA aneurysms showed complete occlusion in 63% (24). In a series on internal trapping of ruptured dissecting VA aneurysms, 21% of those initially totally occluded re-canalized, reminding us of the need for follow-up.

Choosing treatment modality and techniques

Even if treating cerebral aneurysms treatment is always planned in a case-by-case manner, this need is even more pronounced in aneurysms of the VA and PICA. For VA and proximal PICA aneurysms, no surgical treatment exist – and most likely also will never exist – that does not involve risk of laryngeal palsy. Surgery on most distal PICA aneurysms, and bypassing in the distal PICA will escape this risk. On the other hand, endovascular treatment still struggles with achieving total occlusion of the aneurysm, or knowledge of long-term significance of non-total occlusion. Both methods are, many times, suboptimal in occluding dissecting VA aneurysms: for the moment the safest treatment seems to be internal trapping with the risk of recanalization, or clip-trapping with the risk of laryngeal palsy; both may need to be combined with bypass. The effectiveness of flow-diverters in these aneurysms is still to be shown. In giant aneurysms, direct clipping together with resection or bypass if needed is an option, with a significant risk for laryngeal palsy. Mere coiling carries the risk of coil impaction, which may lead to problems in mass effect. In some cases, embolization or proximal occlusion may be combined with a bypass operation.

Outcome

The overall outcome of treating VA and distal PICA aneurysms is favorable, with 80 to 85% recovering at least to an independent stage of life (67, 78, 131, 167). In our series, mortality within one year of the diagnosis was high, with nearly a third deceased, mainly due to their severe initial bleeding. Those who survived, however, made a good recovery, with only 7% not regaining independence.

Additionally, we found a rather high rate, 9%, of recurrent SAH occurring later. Wermer et al. in 2005 calculated the incidence of new bleeding to be 3% within ten years (242). Our high number can in part result from suboptimal aneurysm occlusion, but additionally our series included five patients with rupture of primarily a undiagnosed (de-novo?) aneurysm.

Future perspectives

Etiology of VA and distal PICA aneurysms

What is specific in the VA and PICA that causes them to develop a high number of fusiform and dissecting aneurysms? In the distal PICA, a deficiency in embryological development is one proposal (78). In future, it would also be interesting to examine the general vasculopathy of these patients, and to compare the data to that of healthy individuals as well as to patients with saccular aneurysms. Optional, of course, is to find a way to prevent formation of aneurysms.

Optimal treatment of aneurysms would be total and permanent occlusion of the aneurysm with preservation of the flow together with low risk for a neurological deficit. With most saccular aneurysms, these aims can be met. For giant and fusiform aneurysms, the solution to many challenges maybe flow-diverters. What additionally would be needed, is a revascularization method with by-far easier and faster replacement of blood flow.

Conclusion

Findings of the present study are consistent with those of earlier. The VA and distal PICA aneurysms are rare, with an incidence of 5% of all aneurysms. The number of non-saccular aneurysms is high, comprising a third in the present series. Treatment of fusiform and dissecting aneurysms still requires optimization, with a high risk for laryngeal palsy in surgical treatment, and the challenge of gaining total occlusion in endovascular treatment. Despite many occasions of severe bleeding, most patients surviving the initial stage finally make a good recovery.

Acknowledgements

This study was carried out at the Department of Neurosurgery at Helsinki University Central Hospital between 2011 and 2015. I am grateful to all those working in Töölö Hospital for sharing their expertise and knowledge during this project and also in everyday life. I am also grateful for all the guidance and encouragement I have received in abundance.

I am deeply thankful to Professor **Juha Hernesniemi**. Besides coming up with the subject of this thesis and being its supervisor, his incomparable surgical skills gave meaning to this study. Thank you for the privilege and honor of being allowed to work in your department.

I owe my gratitude to **Mika Niemelä**, the other supervisor of this thesis. Thank you for sharing your great expertise, for your encouragement, and always being available when I needed help.

I am grateful to **Timo Kumpulainen** and **Topi Siniluoto**, the reviewers of this thesis, for your valuable comments that made the final result so much better.

I owe so much to **Riku Kivisaari**. No one understands the suffering and sandwiches and Biblical images I asked you to go through when checking thousands of images for the aneurysm database and this thesis. I admire you: certainly no other person would have been strong enough to accomplish what you did.

I am thankful to **Aki Laakso** for sharing your vast knowledge on making science and for answering my multiple questions during this project. You were irreplaceable.

For **Reza Dashti**, I am so grateful to you in so many ways. When I came to the clinic straight from the medical school, you introduced me to neurosurgery and showed me the way to walk. You have done so much more for me than what I deserved.

Definitely I did not create this thesis alone: I am more than thankful to **Päivi Koroknay-Pál**, **Jarno Satopää** and **Felix Göhre** for invaluable help in and friendship.

I am thankful to **Ali Harati**, **Romain Billon-Grand** and **Bruno Canato** for all the help in the beginning of this journey.

For **Behnam Rezai Jahromi**, **Hugo Andrade** and **Ferzat Hijazy**, besides all the practical help, I am more than grateful for sharing life and all the help during ups and downs in life and research. Surviving without you would have been one big hell.

I was privileged to share the experience of making thesis with **Elina Koskela** and **Ahmed Elsharkawy**. Elina, thank you for long phone calls and the possibility to talk about everything. Ahmed, thank you for sitting hours and weeks and finally years in the same room with me, thank you for letting me try to follow your example in honesty, working, and belief in goodness.

I am grateful to **Carolyn Brimley Norris**. Besides doing excellent language editing, you took time to sit for hours with me, answering all my questions, and you were always ready to help when needed. I owe you a lot. You can also be sure that I will not forget end-focus!

For **Eveliina Salminen**, **Virpi Tuukkanen**, **Heli Holmström**, **Outi Hämäläinen** and **Jessica Peltonen**, thank you for all the practical help but especially for letting me join the life of the "7th-floor-family" during the long and tedious years of making the aneurysm database.

I thank **Tomi Metsä-Heikkilä** for the beauty of this book.

There would be no life without friends and family. I thank you for your patience with me when I was drowning in my work. **Lotta**, thank you for friendship, and also for practical help together with **Rasmus Backholm** during a crises in this project. A big thank you for all my friends known in and through Kathos: **Sanna**, **Tanja**, **Hannu** and **Katja**, **Mirka** and **Miikka**, **Laura**, **Topi**, **Toni**, **Sami**, **Anna-Leena**, **Hanna-Kaisa**, **Ulla** and **Olli**, **Juha** and **Sofia**. Thank you also, **Anna** and **Sami** and your family! I also want to thank the Community of Grandchamp, Sisters of Saint Andrew, and the Taizé Community.

To my family, my **mother**, **father**, **Maija**, **Tuomo**, **Aino**, **Jaakko** and **Eero**, thank you for your love, being there, and your continuous support. Life is beautiful with you.

This thesis was financially supported by Finnish government funding for clinical research at Helsinki University Central Hospital (EVO), Maire Taponen foundation and the Emil Aaltonen Foundations. ❦

In Helsinki, March 2015

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