Pekka Jartti

COMPUTED TOMOGRAPHY IN SUBARACHNOID HAEMORRHAGE

STUDIES ON ANEURYSM LOCALIZATION, HYDROCEPHALUS AND EARLY REBLEEDING
PEKKA JARTTI

COMPUTED TOMOGRAPHY IN SUBARACHNOID HAEMORRHAGE
Studies on aneurysm localization, hydrocephalus and early rebleeding

Academic dissertation to be presented with the assent of the Faculty of Medicine of the University of Oulu for public defence in Auditorium 7 of Oulu University Hospital, on 15 October 2010, at 12 noon
Subarachnoid haemorrhage (SAH) is a life-threatened disease with poor outcome. It is usually caused by an intracranial aneurysm (IA) rupture and rapid diagnosis and treatment are of great importance. Computed tomography (CT) is a reliable method to detect the blood in the subarachnoid (SA) spaces. Digital subtraction angiography (DSA) offers dynamic and morphological information of a ruptured IA. The treatment options for excluding an aneurysm from the main circulation are neurosurgical clipping and endovascular procedures.

The purpose of the present study was to evaluate the risk factors of acute hydrocephalus (HC) and the reliability to localize the ruptured aneurysm based on non-contrast CT. The aim was also to compare the effect of neurosurgical and endovascular treatment on the development of chronic HC, and evaluate the incidence and the risk factors of early rebleeding (< 30 days) after coiling.

The data of 180 operated patients with a ruptured IA were checked. Two neuroradiologists separately located the IAs based on non-contrast CT. The analyses of blood amount and distribution was a reliable method for estimating the location of ruptured middle cerebral artery (MCA) aneurysms and anterior communicate artery (ACoA) aneurysms. Intracerebral haemorrhage (ICH) was a predictor for detecting the precise site. The results confirmed that intraventricular haemorrhage (IVH) was the most consistent single risk factor for the development of acute HC. Haemorrhage in the basal region and the large total blood amount in the SA spaces were strong predictors.

The effect of early treatment modality for ruptured IAs on the development of chronic HC with 102 clipped and 107 coiled patients was compared. The treatment method used was not significantly associated with the occurrence of chronic HC or the need for shunt operation.

The incidence and risk factors of early rebleeding after coiling were investigated in 194 consecutive acutely (within 3 days) coiled patients with ruptured IAs. The incidence of early rehaemorrhage was 3.6%. The presence of ICH at admission and poor clinical condition were significant predictors for rebleeding. An early rehaemorrhage appeared as an enlargement of the ICH in all of these patients.

In conclusion, the non-contrast CT is a reliable method to detect the location of ruptured IA in patients with MCA and ACoA aneurysms. The risk factor for the development of acute HC is IVH. Other predictors are the total SA blood amount and blood in the basal regions. The treatment method used for acutely ruptured IA has no significant effect on the occurrence of chronic HC. The incidence of early rebleeding after coiling is low. The risk factors of rebleeding are the presence of ICH and poor clinical condition. Rehaemorrhage appears often as an enlargement of the ICH.

Keywords: aneurysm rerupture, computed tomography, endovascular treatment, intracranial aneurysm, subarachnoid haemorrhage
To Airi,
Jaakko and Olli-Pekka
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Pekka Jartti
Abbreviations

3D    three-dimensional
3DRA   three-dimensional rotational angiography
3DTOF   three-dimensional time of flight angiography
ACA   anterior cerebral artery
ACoA   anterior communicating artery
BA    basilar artery
CMI    Cella media index
CSF    cerebrospinal fluid
CT    computed tomography
CTA    computed tomography angiography
DCI    delayed cerebral ischemia
DSA    digital subtraction angiography
EVD    external ventricular drainage
GDC    Guglielmi detachable coil
GOS    Glasgow Outcome Scale
H&H    Hunt and Hess grading scale
HC    hydrocephalus
HU    Hounsfield unit
IA    intracranial aneurysm
ICA    internal carotid artery
ICH    intracerebral haemorrhage
ICHvol    volume of intracerebral haemorrhage
ISAT    International Subarachnoid Aneurysm Trial
IVH    intraventricular haemorrhage
IVHsc    sum score of intraventricular blood
MCA    middle cerebral artery
MDCT    multi-detector computed tomography
MDCTA   multi-detector computed tomography angiography
MRA    magnetic resonance angiography
MRI    magnetic resonance imaging
PCoA   posterior communicating artery
PICA   posterior inferior cerebellar artery
SA    subarachnoid
SAH    subarachnoid haemorrhage
SAHsc    sum score of blood in subarachnoid spaces
TBsc    total blood score
wIII    the width of the third ventricle
List of original publications

This thesis is based on the following articles, which are referred to in the text by their Roman numerals.


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1 Introduction

The incidence of subarachnoid haemorrhage (SAH) has remained stable over the last 30 years, at around 6/100 000 patient year, but in Finland and Japan the incidence is much greater, up to 20/100 000 (van Gijn & Rinkel 2001). Race and gender have a marked influence on the occurrence of SAH. Black people have 2.1 times higher risk than whites, and women have 1.6 times higher risk than men (Broderick et al. 1992, Linn et al. 1996). Genetic factors are related to the presence of SAH, and 5–20% of patients have a positive family history (Schievink 1997a). The important risk factors of SAH are smoking, hypertension, and heavy drinking (Feigin et al. 2005).

The main cause of non-traumatic SAH is the rupture of an intracranial aneurysm (IA). Without risk factors for SAH, IA is found in 2% among adults with an annual risk of rupture 0.7% (Rinkel et al. 1998). The case-fatality rate is about 50% overall, 10% die prior to arrival at hospital and up to 30% die during the first day (Pakarinen 1967, Hop et al. 1997, Stegmayr et al. 2004, Trojanowski 2008). A sudden, explosive headache is a classical symptom of SAH. The focal signs develop usually at the same time as a headache together with loss of consciousness. The aneurysmal SAH is often associated with intraventricular haemorrhage (IVH). Intracerebral haemorrhage (ICH) occurs in 20–30% of SAH patients, together or occasionally without subarachnoid (SA) blood (van der Jagt et al. 1999).

Non-contrast computed tomography (CT) is the primary method to detect SAH. Negative CT with negative lumbar puncture makes it possible to rule out SAH (Perry et al. 2008). Digital subtraction angiography (DSA) is still the gold standard for detection of ruptured IAs, offering both morphological and dynamic information. Three-dimensional rotational angiography (3DRA) is used for depicting more details of the aneurysm. Multi-detector computed tomography angiography (MDCTA) is nowadays an alternative to DSA as a first imaging modality in localizing the aneurysm (Velthuis et al. 1999, Westerlaan et al. 2007). Magnetic resonance angiography (MRA) with 3D time of flight sequence (3DTOF) is a non-invasive method without radiation for searching for IAs (Kähärä et al. 1999).

Both microsurgical ligation and endovascular embolization are accepted methods for the treatment of IAs. During the last decades endovascular embolization has gained more popularity at the expense of surgical clipping. The aim of the treatment is to exclude the aneurysm from the main circulation as early as possible and prevent rerupture. Rehaemorrhage occurs usually within a few days after the initial haemorrhage and is often hazardous, causing death or disability. Other
common complications are delayed cerebral ischemia (DCI) and hydrocephalus (HC). An aggressive approach to treat HC with external ventricular drainage (EVD) is often necessary.

The purpose of this thesis was, first, to analyse the value of blood amount and distribution on CT in SAH patients in the localization of a ruptured IA and in the development of acute HC. Second, the influence of the treatment method, neurosurgical clipping or endovascular coiling, on the development of chronic HC was compared. Finally, the aim was to evaluate the incidence and risk factors for early rebleeding after coiling of ruptured IAs.
2 Review of the literature

2.1 Subarachnoid haemorrhage

2.1.1 Etiology, incidence and epidemiology

Sir William Gowers described the correlation of blood on the surface of the brain due to ruptured aneurysm and sudden death as far back as 1875 (Gowers 1875). The entity was SAH and it takes place when blood leaks out into the SA space, which is the area on the surface of the brain between the layers of the arachnoid membrane and pia mater (Fig. 1). The rupture of IA is the cause of non-traumatic SAH in over 80% of cases (Kassell et al. 1990a, Velthuis et al. 1998, van Gijn et al. 2001). Other rarer conditions leading to SAH are perimesencephalic haemorrhage, vascular malformations, dural arteriovenous fistulas, arterial dissections, spinal haemorrhage, bleeding disorders and alcohol abuse (van Gijn et al. 2001).

In Finland SAH accounts for 7 to 11% of all strokes (Sarti et al. 1991, Sivenius et al. 2004), but world-widely SAH comprise 1 to 7% of strokes (Feigin et al. 2003). The incidence of SAH is 6–8/100 000 person-years in most western populations (Linn et al. 1996, Anderson et al. 2000, van Gijn et al. 2001, Molyneux et al. 2002, van Gijn et al. 2007). In Finland and Japan the incidence is higher and is reported to be around 20/100 000 (Sarti et al. 1991, Linn et al. 1996, van Gijn et al. 2001). The frequency of SAH increases almost linearly with increased age (Fogelholm 1981, Anderson et al. 2000, Juvela et al. 2000, van Gijn et al. 2007). In adults over 30 years the incidence is estimated to be 30–40/100 000 and the peak incidence is 40 to 65 years (Sarti et al. 1991, Juvela 2004). SAH occurs more often in women than men, especially in adults over 40 years (Linn et al. 1996). This may be related to hormonal aspects (Kongable et al. 1996).
2.1.2 Risk factors

Feigin et al. (2005) presented an overview of longitudinal and case-control studies of risk factors for SAH published in English from 1966 to 2005. In this meta-analysis smoking, hypertension, and heavy drinking were the only important risk factors. Non-white ethnicity was a minor risk factor. The same major factors have been published in the Finnish series (Juvela et al. 1993, Juvela et al. 2000).

Evidence has been found that genetic factors are related to the presence of SAH. Between 5 and 20% of patients with SAH have a positive family history (Schievink 1997a). First-degree relatives have a 3 to 7-fold increased risk of suffering SAH (Bromberg et al. 1995, Schievink et al. 1995, Wang et al. 1995, Gaist et al. 2000). The familial cases seem to occur at an earlier age than sporadic ones (Lozano & Leblanc 1987). Ronkainen et al. (1993, 1997) has confirmed this familial predisposition also in the Finnish population. In some hereditary diseases such as polycystic kidney disease, Ehlers-Danlos syndrome type IV and neurofibromatosis, the existence of SAH is slightly increased (Schievink 1997a).
2.1.3 Pathophysiology

After the onset of aneurysmal SAH the blood spread into the SA space, often associated with IVH. This pre-existing leads to cisternal and intraventricular blood clots, decreased cerebrospinal fluid (CSF) outflow, HC and elevated intracranial pressure (Solenski et al. 1995, Gonzales et al. 2007). The long-existing volume loading of the SA space reduces cerebral compliance, and global ischemia may occur (Trojanowski 2008). Within four hours after SAH the blood-brain barrier (BBB) disruption occurs with generalized brain oedema (Trojanowski 1982).

Delayed arterial vasospasm is the most common and very hazardous complication of SAH. It typically occurs from 4 to 14 days after the initial haemorrhage, with a peak incidence at 7 days (Phillips et al. 1980, Kassell et al. 1985, Bracard & Schmitt 2008). Vasospasm is related to the blood amount and blood clot resolution in the SA space, but its pathophysiology remains partly unclear. The situation is aggravated by complex molecular mechanisms and inflammation reactions (Dumont et al. 2003). It is supposed that vasospasm is related to modifications of the vascular wall, including smooth muscle cell proliferation, sub-endothelial fibrosis and hyperplasia of the intimae together with smooth muscle contraction (Smith et al. 1985). Not all patients with DCI have macrovascular arterial narrowing. Hypotension, hypovolemia, inflammatory and endothelium-related processes lead to changes in coagulation cascades and microthrombosis (Chang et al. 2000, Vergouwen et al. 2008).

ICH occurs in 20–30% of SAH patients, together with SA blood or occasionally without it (van Gijn & van Dongen 1982, van der Jagt et al. 1999). The existence of non-traumatic acute subdural haemorrhage associated with aneurysm rupture is uncommon, but some cases have been described (O’Sullivan et al. 1994, Inamasu et al. 2002).

2.1.4 Clinical course and outcome

A sudden, severe and persistent headache is usually the first symptom of SAH, later together with vomiting, neurological focal symptoms and loss of consciousness. Over 10% of patients suffering SAH die prior to arrival at hospital, 25–30% die within 24 hours, and the case-fatality rate is about 50–60% (Pakarinen 1967, Hop et al. 1997, Stegmayr et al. 2004, Trojanowski 2008). The Hunt and Hess grading scale (H&H) is in common use for evaluating a patient’s neurological condition at admission (Table 1) (Hunt & Hess 1968).
Table 1. The Hunt and Hess grading scale for non-traumatic subarachnoid haemorrhage patients (Hunt & Hess 1968).

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Unruptured aneurysm</td>
</tr>
<tr>
<td>1</td>
<td>Asymptomatic, mild headache, slight nuchal rigidity</td>
</tr>
<tr>
<td>2</td>
<td>Moderate to severe headache, nuchal rigidity, no neurologic deficit other than cranial nerve palsy</td>
</tr>
<tr>
<td>3</td>
<td>Drowsiness, confusion, or mild local deficit</td>
</tr>
<tr>
<td>4</td>
<td>Stupor, moderate to severe hemiparesis</td>
</tr>
<tr>
<td>5</td>
<td>Coma, decerebrate posturing</td>
</tr>
</tbody>
</table>

A variety of factors are related to the outcome in SAH patients. The three most important variables are clinical condition on admission, age, and the amount of SA blood on the initial CT, together with subsequent events (Hijdra et al. 1988, Kassell et al. 1990b, Fogelholm et al. 1993, Niskanen et al. 1993, Solenski et al. 1995). Acute HC with large amounts of blood in the ventricles is associated with a poor clinical condition. The outcome of patients with ICH is worse than in patients with purely SA blood (Hauerberg & Eskesen 1994). In addition to the primary bleeding and DCI, rebleeding is a major course of death and disability (Vermeij et al. 1998, Hop et al. 1999). During hospitalization 40–79% of patients suffer at least one medical complication, such as myocardial injury or pulmonary and metabolic complication (Solenski et al. 1995, Wartenberg et al. 2006). Approximately one-third of the SAH patients remain dependent (Hop et al. 1997). Only 19% of independent patients had no significant reduction in life quality four months after the attack (Hop et al. 1998). Although functional outcome improves between 4 months and 18 months, only a small minority of patients with SAH have finally a good outcome (Hop et al. 2001).

The three ubiquitously used clinical scales related to outcome are the Glasgow Outcome Scale (GOS) (Jennet & Bond 1975), the grading scale of the World Federation of Neurosurgical Societies Scale (WFNS) (Report 1988) and the Rankin Outcome Scale (Rankin 1957). These grading scales are presented in Tables 2, 3 and 4. More recently a comprehensive grading system approaching the consideration of various factors on outcome for both surgical and endovascular management has been proposed (Ogilvy & Carter 1998, Ogilvy et al. 2006).
Table 2. Glasgow Outcome Scale for estimating outcome after treatment (Jennet & Bond 1975).

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>Good recovery</td>
</tr>
<tr>
<td>4</td>
<td>Moderately disabled (independent life)</td>
</tr>
<tr>
<td>3</td>
<td>Severely disabled (conscious, but dependent on others)</td>
</tr>
<tr>
<td>2</td>
<td>Vegetative state</td>
</tr>
<tr>
<td>1</td>
<td>Death</td>
</tr>
</tbody>
</table>

Table 3. World Federation of Neurosurgical Societies Scale (WFNS) (Report 1988).

<table>
<thead>
<tr>
<th>Grade</th>
<th>GCS*</th>
<th>Motor deficit</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>15</td>
<td>No, No SAH</td>
</tr>
<tr>
<td>1</td>
<td>15</td>
<td>No</td>
</tr>
<tr>
<td>2</td>
<td>13–14</td>
<td>No</td>
</tr>
<tr>
<td>3</td>
<td>13–14</td>
<td>Yes</td>
</tr>
<tr>
<td>4</td>
<td>7–12</td>
<td>Yes</td>
</tr>
<tr>
<td>5</td>
<td>3–6</td>
<td>Yes or no</td>
</tr>
</tbody>
</table>

* Glasgow Coma Scale (Teasdale & Jennett 1976)

Table 4. Rankin Outcome Scale for estimating prognosis (Rankin 1957).

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>No significant disability, gets over daily routines</td>
</tr>
<tr>
<td>2</td>
<td>Slight disability, unable to get over some daily routines</td>
</tr>
<tr>
<td>3</td>
<td>Moderate disability, needs help in daily routines, able to walk without help</td>
</tr>
<tr>
<td>4</td>
<td>Severe disability, bedridden and incontinent, needs constant care</td>
</tr>
</tbody>
</table>

2.1.5 Radiological diagnostic methods

Computed tomography

Imaging of the brain parenchyma was the first application of CT being used since the early 1970s (Ambrose & Hounsfield 1973). CT has been available in Finland from 1978 (Suoranta 2006). Today it remains one of the most widely performed radiological examinations. CT has a good availability and it is a fast and easily repeatable method. Density measurements of CT voxel data, Hounsfield units (HU), help to analyse different tissue compartments.
CT has a great accuracy to detect SAH and to reveal additional parenchymal or other haemorrhage (Schievink 1997b, van Gijn et al. 2001). On CT images fresh blood in the SA space and in the ventricles is seen as a high-density (white) area (Fig. 2). Blood typically has a HU 60–75 differing from a HU of normal brain parenchyma (HU 35–45) presenting a good contrast to surrounding tissues. CT is also sensitive to reveal the important consequences of SAH such as HC, ischemic lesions and rehaemorrhage. SAH patients are exposed to a high total radiation dose after different and multiple examinations. The radiation dose given to patients should be minimized considering the necessity of examination and optimizing the imaging acquisition parameters. With a modern and fast CT the movement artefacts are only a minor problem. In the follow-up imaging clips and coils may produce streak artefacts degrading image quality.

The ability to detect haemorrhage on CT depends on the interval after onset, the amount of blood, the resolution of the scanner and the skills of the radiologist (van Gijn et al. 2007). The false negative CT increases with elapsing time. In up to 95% of SAH patients, blood will be present in the SA space on the first day (van der Wee et al. 1995, Boesiger & Schiber 2005). Sidman et al. (1996) investigated third-generation CT scanners for SAH and found 100% sensitivity, when CT was performed at or before 12 hours after symptoms. The sensitivity decreased to 82% after 12 hours of symptom duration. After five days of the onset blood is detected on CT in fewer than 60% of cases (Kassell et al. 1990a). In a report with fifth generation scanners no patients who had a negative CT were found to have a SAH (Boesiger et al. 2005). They concluded that modern CT scanners are probably more sensitive than earlier scanners. Normal CT diagnosis should be followed by lumbar puncture to exclude SA blood, and the combination of a negative CT result and a negative lumbar puncture is sufficient to rule out SAH (Perry et al. 2008).

False-positive CT findings simulating SAH have been described in patients with comatose and cerebral oedema. In these rare situations the hyperdense material in the SA space represents blood in congested subarachnoid vessels (Opeskin & Silberstein 1998, Chute & Smialek 2002). The frontobasal or temporal contusion haemorrhage after trauma may be difficult to distinguish from aneurysmal haemorrhage (Rinkel et al. 1993, Sakas et al. 1995).
Magnetic resonance imaging

Magnetic resonance imaging (MRI) with fluid attenuated inversion recovery (FLAIR) or proton density (PD) sequences has proved to be sensitive in the diagnosis of SAH (Noguchi et al. 1995, Mikami et al. 1996, Wiesmann et al. 2002, Fiebach et al. 2004). However, in general practice MRI is considered to be of little value depending on costs, availability and also difficulties with restless patients. Later, in the subacute phase from a few days to 50, MRI is a good method to detect SAH and may give more information of haemorrhage, especially in patients with suspected subacute SAH and normal CT (Ogawa 1995, Nogushi et al. 1997).

2.1.6 Grading scales of blood amount and distribution

Traditionally all grading scales for estimating the amount of SA blood detected on non-contrast CT after SAH are based on axial slices and on the width of SA blood layers (Fisher et al. 1980, Gurusinghe & Richardson 1983, Ljungren et al. 1984, Mohsen et al. 1984). The grading scales have mostly used to predict symptomatic vasospasm. Graeb et al. (1982) described a semiquantitative scale for the blood volume of IVH. A final score was the sum of lateral ventricles (1–4 points each) and the third and fourth ventricle scores (1–2 points each), and the maximum score of 12 points. Some modified and newer grading scales also take into account the existence of IVH or ICH (Hijdra et al. 1990, Claassen et al. 2001, Frontera et al. 2006).
Fisher grading scale

Fisher et al. (1980) presented a grading scale for estimating the amount of blood in SAH patients to predict vasospasm (Table 5). They assumed that localized blood in the SA space in sufficient amount is strongly related to DCI and outcome. The Fisher grading scale is a well-established method and world-wide used (Kassell et al. 1990a, Hernesniemi et al. 1993, Niskanen et al. 1993).

Table 5. Fisher grading scale for estimating blood amount on CT after SAH (Fisher et al. 1980).

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>No blood</td>
</tr>
<tr>
<td>2</td>
<td>Diffuse SAH or vertical layers &lt; 1 mm thick</td>
</tr>
<tr>
<td>3</td>
<td>Localized clot or vertical layer ≥ 1 mm thick</td>
</tr>
<tr>
<td>4</td>
<td>IVH or ICH with or without SAH</td>
</tr>
</tbody>
</table>

The Fisher grading scale has received criticism, because it does not differentiate between ICH and IVH. Claassen et al. (2001) proposed a new revisited grading scale based on the Fisher scale that accounts for the independent predictive value of SA and ventricular blood for DCI. Woertgen et al. (2003) concluded in their highly selected series of ruptured IA that the Claassen grading scale is predictive for DCI, but it gives no additional information compared to the Fisher scale. Recently another modified Fisher grading scale has proved to be more accurate to predict DCI than the original Fisher scale (Frontera et al. 2006).

Hijdra grading scale

Hijdra et al. (1990) described a new method to grade the amount of blood in the SA space and ventricles on CT. They divided SA space into 10 different basal cisterns and fissures (Fig. 3). Each cistern, fissure and ventricle was graded 0–3 depending on the amount of blood (Fig. 4).

In a recent report by van Norden et al. (2006) the Hijdra grading scale was considered superior to the Fisher grading scale in evaluating the amount of extravasated blood. The Hijdra scale has a good interobserver agreement, and its usability in research dealing with prognostic factors is excellent (Hirashima et al. 2003, van Norden et al. 2006). Brouwers et al. (1993) presented that the total amount of SA blood using the Hijdra grading scale has independent predictive power for DCI.
the occurrence of DCI. On the other hand, van der Jagt et al. (2000) criticized this method showing a considerable interobserver variability for predicting DCI. The method has some drawbacks for anatomical and technical reasons. The small amount of SAH in the SA space lying next to the skull base may be inaccurate because the slice thickness of CT causes a partial volume effect. The SA space varies in size between patients of different ages, and the absolute quantity of haemorrhage may hence be difficult to estimate. Also, the calcified falx in the interhemispheric fissure may cause difficulties in grading the SAH.

![Diagram of subarachnoid space](image)

**Fig. 3.** Division of subarachnoid space into fissures and basal cisterns by Hijdra (Hijdra et al. 1990). A = interhemispheric fissure, B = lateral sylvian fissure, C = basal sylvian fissure, D = suprasellar cistern. E = ambient cistern, F = quadrigeminal cistern.

![CT images](image)

**Fig. 4.** An example of grading blood amount on CT slices by Hijdra (Hijdra et al. 1990). Scores of cisterns and fissures: interhemispheric fissure 2; lateral sylvian fissure right 3, left 1; basal sylvian fissure right 3, left 2; supracellar cistern 3; ambient cistern right 3, left 2; quadrigeminal cistern right 3, left 1. (Total score 23). Scores of ventricles: lateral ventricles right 0, left 1; third ventricle 2; fourth ventricle 1. (Total score 4).
2.2 Hydrocephalus after subarachnoid haemorrhage

Bagley (1928) was the first to describe HC following aneurysmal SAH. This complication is now a well-recognized sequela of SAH (van Gijn et al. 1985, Graff-Radford et al. 1989, Vale et al. 1997). Vale et al. (1997) divided HC into three stages: acute (0–3 days after SAH), subacute (4–13 days after SAH) and chronic (≥ 14 days after SAH). The practical division is acute and chronic HC, where acute HC appears immediately or soon after SAH and chronic HC after two weeks from the attack.

2.2.1 Determination of hydrocephalus

HC is easily identified with visual impression observing ventricular size, rounding of the frontal horns, periventricular lucencies, and obliteration of the cerebral sulci (Gunasekara & Richardson 1977). Sheehan et al. (1999) confirmed radiographic evidence of HC by observing enlarged temporal horns (≥ 2 mm wide), and sylvian fissure, interhemispheric fissure, and cerebral sulci not visible on CT images (Fig. 5).

![Fig. 5. Hydrocephalus after SAH on CT image. Sylvian fissure, interhemispheric fissure and cerebri sulci are not visible, rounded frontal and enlarged temporal horns.](image)
Many different methods to calculate ventricular size and to determine HC on CT images have been used. Galera and Greitz (1970) calculated a ventriculocranial index (bifrontal index) (width of the ventricles on axial CT between parallel portions of the medial caudate nuclei divided by the width of the brain at the same level). Vassilouthis and Richardson (1979) calculated ventricular size and expressed it as a ratio of the width of the lateral ventricles at the level of the foramen Monroe behind the caudate nucleus and the transverse inner diameter of the skull at the same level. Van Gijn et al. (1985) presented measurements of HC as the width of the frontal horns at the level of the caudate nuclei divided by the diameter of the brain at the same level, and called it the bicaudate index (Fig. 6). Vermeij et al. (1994) evaluated ventricular dilatation using the bicaudate index exceeding 95th percentile for each age excluding the effect of brain atrophy on the values.

**Fig. 6.** The bicaudate index = A/B. A = width of the ventricles at the level of caudate nuclei. B = diameter of the brain at the same level (van Gijn et al. 1985).

The Cella media index (CMI), the width of both cellae media (central part of the lateral ventricle) in relation to the width of the outer layer of the skull at the same level, has also been used to calculate ventricular dilatation in cases of HC and atrophy (Meese et al. 1980, Soininen et al. 1982) (Fig. 7). The different variations of CMI, like inversed CMI, values related to inner skull diameter and the use of MRI images in calculation have also been presented (Grumme et al. 1998, Bendel et al. 2009a, Mondorf et al. 2009). The enlargement of the third ventricle reflects
the disturbance of CSF circulation at an early stage being one possible sign to use in the determination of HC (Widenka et al. 2000, Bakker et al. 2007).

Fig. 7. The Cella media index A/B. A = width of both cellae media, B = width of the outer layer of the skull (Meese et al. 1980).

2.2.2 Acute hydrocephalus


The SA blood and cellular exudates block the foramina of the 4th ventricle, basal cisterns and arachnoidal villi (Kolluri & Sengupta 1984). The fibrosis and occlusion of arachnoid granules leads to a dysfunction in CSF absorption (Kibler et al. 1961, Graff-Radford et al. 1989, Vale et al. 1997, Widenka et al. 2000). The development of acute ventricular enlargement is often associated with a large amount of ventricular and cisternal blood or haemorrhage in the basal regions.
In the absence of ventricular blood, the blood in the ambient cisterns significantly increases the risk of acute HC (Hasan & Tanghe 1992). The rupture of internal carotid and posterior circulation artery aneurysms is prone to cause acute HC (Graff-Radford et al. 1989, Hasan & Tanghe 1992, Mehta et al. 1996). It has been reported that the rate of HC is lower in patients with middle cerebral artery (MCA) aneurysms compared to aneurysms close to the mid-line (Kibler et al. 1961, Graff-Radford et al. 1989, Tapaninaho et al. 1993, Pietilä et al. 1995, Schmieder et al. 1999). On the contrary, Lin et al. (1999) found no correlation with the anatomical location of ruptured IA and acute HC. The relationship between the increasing age and acute HC has been documented (Black et al. 1986, Graff-Radford et al. 1989), but controversial reports with no effect of age have also been published (Mehta et al. 1996, Lin et al. 1999). The size of IA has no influence on acute HC (Lin et al. 1999). Acute HC does not necessarily lead to chronic HC, and in most cases, the acute phase resolves by itself (Kolluri & Sengupta 1984, Sheehan et al. 1999).

EVD placement is a safe and effective procedure for management of acute HC, when performed by an experienced neurosurgical team. Immediate drainage of CSF removes haemorrhagic fluid, reduces cerebral oedema and subsequent meningeal thickening (Kolluri & Sengupta 1984). Infection is the most common complication after EVD, but the definition of EVD infection is not uniform, leading to a wide range of infection rates reported (Roitberg et al. 2001). EVD should be replaced or removed anytime there is a positive culture.

**2.2.3 Chronic hydrocephalus**

Chronic HC appears when CSF absorption is permanently reduced or CSF flow is permanently impeded (Schutz et al. 1980, Sheehan et al. 1999). It is assumed that chronic HC is due not only to disturbance of CSF absorption by arachnoid granules (major CSF pathway), but also to disturbance of the minor pathway within the parenchyma (Hirashima et al. 2003). The incidence of chronic HC is 10 to 48% depending on the criteria used for the diagnosis (Galera & Greitz 1970, Yasargil et al. 1973, Kolluri & Sengupta 1984, Tapaninaho et al. 1993, Vermeij et al. 1994, Vale et al. 1997). Many factors are associated with the occurrence of chronic HC following SAH. These factors include the patient’s clinical condition on admission, acute HC, IVH, the amount and distribution of blood in the SA spaces (Vale et al. 1997, Schmieder et al. 1999, Sheehan et al. 1999, Widenka et al. 2000). The influence of the initial
clinical condition on chronic HC is obvious and these patients frequently require more shunting (Sundt et al. 1982, Spallone & Gagliardi 1983, Pietilä et al. 1995). Studies have indicated that chronic HC is more frequent following the rupture of posterior circulation and midline aneurysms (Galera & Greitz 1970, Yasargil et al. 1973, Pietilä et al. 1995, Sethi et al. 2000). The rupture of these aneurysms is often accompanied with IVH, which predisposes to the occurrence of chronic HC together with a large amount of blood in the basal cisterns.

Many reports have stated that older people are more likely to develop chronic HC (Tapaninaho et al. 1993, Vermeij et al. 1994, Lin et al. 1999, Yoshioka et al. 2000). On the other hand, Hirashima et al. (2003) reported that age was unrelated to the incidence of chronic HC after SAH.

### 2.3 Intracranial aneurysms

#### 2.3.1 Prevalence

The prevalence of IAs varies between 0.2 and 8.1% depending on different study methods (Bannerman et al. 1970, Jellinger 1979, Byrne & Guglielmi 1998, Ronkainen et al. 1998). According to a large autopsy study by Bannerman et al. (1970) the prevalence of ruptured IA was 0.34%. Rinkel et al. (1998) examined the risk of rupture of saccular IAs and presented a meta-analysis of studies between 1955 and 1996. They concluded that without risk factors for SAH, aneurysms are found in 2% among adults with an annual risk of rupture of 0.7%.

#### 2.3.2 Classification

A different classification has been used depending on aneurysm morphology, etiology, location, size or clinical presentation. Based on morphology, saccular IAs account for 98% of all aneurysms (Yasargil 1984a). Saccular aneurysms are berry-like outpouchings of the vessel wall having an irregular shape with a neck opening. The wall structure of an aneurysm is disorganized with no defined layers (Kumar et al. 1999). In non-saccular aneurysms an enlargement of the entire vessel circumference takes place, and the vessel dilatation is often called a fusiform aneurysm (Fig. 8). Flemming et al. (2004) classified radiographically non-saccular aneurysms as fusiform, dolichoectatic, and transitional. Based on etiology, aneurysms can be divided as false or traumatic aneurysms, dissecting aneurysms, flow-related aneurysms and infectious aneurysms (Isokangas 2006, Krings et al. 2008).
According to a new approach presented by Krings et al. (2008), an etiological classification may be based on alterations of the vessel wall, either due to luminal or abluminal factors. Saccular aneurysms are typical example of luminal aneurysmal vasculopathies. Structural vessel wall diseases and dissections belong to abluminal aneurysmal vasculopathies. The role of the vasa vasorum (arterial network within the adventitia) in the development of aneurysm growth is not known in detail. The recurrent bleeding from the vasa vasorum can result in an increase in the size of an aneurysm and in further proliferation of new vessels. The increased size in a giant aneurysm is not due to intraluminal factors but to the apposition of new layers of thrombus at the periphery (Krings et al. 2008). The walls of ruptured saccular IAs have also shown inflammatory reactions including macrophage infiltration, which may trigger the rupture of IA (Frösen et al. 2006).

Fig. 8. A. Saccular aneurysm. B. Saccular wide-necked aneurysm C. Fusiform aneurysm.

2.3.3 Locations of saccular aneurysms

Saccular IAs are thought to develop from defects in the muscular layer of arteries. Alterations in the internal elastic membrane of arteries weaken vessel walls and render them less resistant to changes in intraluminal pressure (Selman et al. 2000). The blood flow is most turbulent and shear forces against the arterial wall are greatest at sites of bifurcation. It may explain why the typical locations of cerebral aneurysms are at the bifurcations of major arteries. Most saccular aneurysms arise from first- or second-order branches of the circle of Willis at the cerebral basal region.

Approximately 85% of all IAs arise in the anterior (carotid) circulation. The typical locations are the anterior communicating artery (ACoA) in 30–35% of cases, MCA in 20–33% of cases and internal cerebral artery (ICA) in 24–40% of cases. The minority of aneurysms are situated in the posterior (vertebro-basilar) circulation (4–12%), and the rest in miscellaneous locations (Weir & MacDonald 1996). Based on some Scandinavian reports the distribution of IAs is different in Finland and Norway and the predominance of MCA has been found (Pakarinen 1967, Fogelholm 1981, Tapaninaho et al. 1993, Rinne et al. 1994, Pedersen et al. 2001).
Rinne et al. (1994) analysed all the reports with more than 300 SAH patients (altogether 31 866 patients). Based on their analysis 16% harboured IAs in multiple locations, but also higher frequencies (33–45%) have been published (Nehls et al. 1985, Wilson & Jaspan 1989). Multiple IAs have been detected in up to one-third of patients in a Finnish series (af Björkesten & Halonen 1965, Rinne & Hernesniemi 1995). With the regular use of DSA an increasing number of patients with multiple aneurysms have been found.

### 2.3.4 Localization of ruptured intracranial aneurysms

**Computed tomography**

Although non-contrast CT is sensitive to detect SAH, the precise detection of the site of a ruptured IA is not always possible. The distribution of haemorrhage, based on the initial CT, predicts the location of an aneurysm in over 80% of cases (Hillman 1993, Latchaw et al. 1997, Tryfonidis 2007). The prediction of the site of an aneurysm has been reported with no contradictory results in cases of both single aneurysms and multiple aneurysms (Kendall et al. 1976, Nehls et al. 1985, Lee et al. 1996, Hino et al. 2000). It could help the radiologist to focus the DSA to the region of interest in the brain, which may shorten the angiographic procedure.

The localization of the ruptured IA on CT images would be of particular significance in the presence of multiple aneurysms. In such patients the diagnostic value of DSA for detecting the target aneurysm is diminished (Liliequist et al. 1977, Vajda et al. 1986, Hino et al. 2000). It has been reported that postoperative rebleeding is usually due to a misinterpretation of the ruptured IA in the presence of multiple aneurysms (Lee et al. 1996, Hino et al. 2000).

SAH with a parenchymal haemorrhage has proposed to be a predictor of the location of a ruptured IA (Nehls et al. 1985, Hillman 1993, van der Jagt et al. 1999). Van der Jagt et al (1999) concluded that the location of the ruptured aneurysm without parenchymal haemorrhage can only be determined by CT, when the bleeding originates from an anterior cerebral artery (ACA) or an ACoA aneurysm. ICH in MCA aneurysms may also be helpful in localization.

**Computed tomography angiography**

After introduction of helical CT with a continuously rotating gantry during the movement of patient, 3D post-processing reconstruction images were available
The problems with resolution of 3D images were diminished later when multi-detector computed tomography (MDCT) scanners became general (Klingenbeck-Regn et al. 1999). MDCTA was a significant improvement in detecting anatomic information of IAs (Kato et al. 2002).

Nowadays CTA is an alternative to DSA as a first imaging modality in localizing the aneurysm (Velthuis et al. 1999, Westerlaan et al. 2007). CTA can be performed immediately after routine non-contrast CT. It is an effective, fast, less invasive and easily applied method without patient motion artefacts. The important advantage of CTA is the possibility to reconstruct angiography images in various angles and planes. The sensitivity to detect a ruptured aneurysm with CTA is 85–98% (Alberico et al. 1995, Hope et al. 1996, Pedersen et al. 2001). Sixty-four-row MDCT is in common use and it has tremendously improved the accuracy, and the detection rate of ruptured IA is nearly 100% (Kangasniemi et al. 2004, Lubicz et al. 2007, Nijjar et al. 2007). McKinney et al. (2008) concluded in their study that even 64-row MDCTA may occasionally miss aneurysms less than 3-4 mm in size. In SAH patients with no blood on CT or perimesencephalic haemorrhage the negative MDCTA findings may be reliable to rule out IAs (Agid et al. 2010).

For the acute management of ruptured IA there are disadvantages of CTA, such as the need for contrast media and long post-processing. If DSA with endovascular treatment is followed soon after CTA, the large amount of contrast media may lead to overloading. The limitation with CTA is the difficulty to detect small aneurysms, aneurysms close to bone structures and, in rare cases, partially thrombosed aneurysms (Alberico et al. 1995, Hope et al. 1996, Kangasniemi et al. 2004). Some promising new techniques, such as dual-energy bone removal and matched mask bone elimination, have been described to help to identify the aneurysms located near cranial bones (Romijn et al. 2008, Watanabe et al. 2009).

Digital subtraction angiography

Selective DSA offers both dynamic and morphological information of the intracranial circulation and it is accepted as the gold standard in diagnosis and pre-treatment characterization of aneurysms (van Gijn et al. 2001). It has a high sensitivity and specificity to detect aneurysms with false negative results from 5 to 10% (Tatter et al. 1995). DSA with 3DRA has increased the accuracy to determine aneurysm morphology and its environment (Tanoue et al. 2000). McKinney et al. (2008) concluded in their report, that the combination of DSA with 3DRA is the most sensitive technique to evaluate an aneurysm smaller than 4 mm. On the other
hand, DSA is an expensive, time-consuming and invasive modality including the risk of complications in 1–2% and permanent neurological deficit in up to 0.5% (Cloft et al. 1999, Dawkins et al. 2007, Willinsky et al. 2009).

*Magnetic resonance angiography*

MRA with 3DTOF is a non-invasive and safe modality without radiation for searching for IAs. The post processing of 3DTOF- images provides maximum intensity projection (MIP) reconstructions (Kähärä et al. 1999). MRA is especially useful in identifying the anatomy of thrombosed aneurysms, and it may significantly improve pre-treatment depiction of vascular anatomy and adjacent structures of such aneurysms (Nagasawa et al. 1998). This modality is less suitable in the acute stage with restless patients because of motion artefacts. White et al. (2000) compared MRA to CTA in a meta-analysis and found both methods equal in accuracy to depict aneurysms (about 90%). In another study, the sensitivity of MRA for detecting all aneurysms was 70–97% compared to DSA (Wardlaw & White 2000). In cases of small aneurysms (3 mm or less) the sensitivity of MRA decreases markedly from 94 to 38% (Wardlaw & White 2000).

Contrast-enhanced MRA is a promising technique for detecting recently ruptured IAs (Unlu et al. 2005). Even more information may be achieved with a modern technique with first-pass bolus contrast enhanced MRA and high-spatial resolution (Isoda et al. 2007).

2.4 **Treatment methods of ruptured intracranial aneurysms**

The aim of the treatment of ruptured IA is to exclude the aneurysm sac and neck from the main circulation and prevent rerupture. Earlier the only modality to reach this goal was neurosurgical treatment, but during the last two decades an endovascular approach has evolved and became used world-wide.

2.4.1 **Neurosurgical treatment**

The neurosurgical approach by clipping of the base of the aneurysm is a confirmed effective method to treat IAs, and it is still successfully used as a primary method in many institutes and in special cases. The principle of neurosurgery is to expose the affected artery and ruptured IA along the skull base and to clip the aneurysm neck. The use of microsurgery techniques was first introduced by Yasargil (Yasargil & Fox

Early surgical care, less than 72 hours after the rupture, is widely accepted as a treatment policy, but the published series show partly contradictory opinions of the benefit of early operation (Vapalahti et al. 1984, Kassell et al. 1990ab, Haley et al. 1992, Fogelholm et al. 1993, Ross et al. 2002). In many cases early operation does not affect the surgical outcome, and one important factor related to outcome is the amount and severity of the haemorrhage (Orbo et al. 2008). In good-grade patients without associated illnesses the usefulness of early surgery has been proved (Öhman et al. 1989, Hernesniemi et al. 1993, Osawa et al. 2001, van der Jagt et al. 2009). Patients with massive ICH caused by the rupture of the aneurysm should be operated as a matter of urgency.

The neurosurgical results of anterior circulation aneurysms are superior compared to posterior circulation aneurysms. In particular MCA aneurysms are usually favourable for neurosurgical treatment because of their anatomical and hemodynamic features (Dashti et al. 2007). The large or giant aneurysms (over 25 mm) are often more suitable for neurosurgical ligation than endovascular treatment, and in such cases it is usually possible to keep the main artery branches open in surgery. The larger the aneurysm the poorer the outcome after treatment (Koivisto et al. 2008).

After clipping the complete occlusion rate has proved to be 82–95% (Sindou et al. 1998, Thornton et al. 2000, Kivisaari et al. 2004, Molyneux et al. 2005). In the study of Kivisaari et al. (2004) angiography after surgery for ruptured saccular IAs revealed surprisingly unplanned major vessel occlusion in 6% and incomplete occlusion in 14% of aneurysms. The conclusion was that the patients should undergo postoperative DSA even when surgeons are competent and have experience. The procedural morbidity and mortality varies between 2.9–6.9% and 1.7–4.0%, respectively (Hernesniemi et al. 1993, Vanninen et al. 1999, Fridriksson et al. 2002).

2.4.2 Endovascular treatment

Endovascular treatment of ruptured IAs is an effective and safe method and has proven to be a good alternative to neurosurgery (Vanninen et al. 1999, Koivisto et al. 2000, Molyneux et al. 2005). The popularity of endovascular treatment has become even more common together with the progressive improvement of the technique and devices and with the results of a large international trial (ISAT) (Molyneux et al. 2005, Molyneux 2006, Molyneux et al. 2009).
History and development

Endovascular techniques were developed originally instead of surgery in patients with complex or surgically difficult IAs. The first results of endovascular parent artery occlusion by detachable balloons were described by Serbinenko (1974). Thereafter the detachable balloons were used to fill the aneurysm and preserve the parent artery. These techniques had advantages compared to surgical ligation such as the analysis of collateral flow and the possibility of test occlusion, but they also included technical problems and complications (Higashida et al. 1989, Higashida et al. 1991). The experimental study of Graves et al. (1990) in dogs presented treatment of carotid artery aneurysms with platinum coils. In a report of Dowd et al. (1990) three patients underwent platinum coil embolization with so-called free coils. The problem was that free coils tended to migrate into the parent artery leading to complications.

Guglielmi published two reports describing the use of electrically detachable platinum coils, known as Guglielmi detachable coils (GDC) for the treatment of IAs (Guglielmi et al. 1991a, Guglielmi et al. 1991b). The GDC system includes a delivery guide-wire and a tip with a platinum coil which is deployed inside the aneurysm sac through a micro-catheter. The detachment of the coils and the thrombus formation is achieved with the help of electric current. The platinum coils are very soft and they adapt to the size and shape of the aneurysm with minimal intra-luminal pressure. Since 1996, endovascular coiling became used worldwide in patients with ruptured IAs (Malisch et al. 1997). Later, soft coils, 2D and 3D shaped coils, coils with different detachment technologies (mechanical and hydraulic), biologically active coils, radioactive coils and coated coils has became available (Cekirge et al. 1996, Reidy & Qureshi 1996, Cloft et al. 2000, Murayama et al. 2003, Raymond et al. 2003a, Cloft & Kallmes 2004, Sugiu et al. 2004, Lubicz et al. 2005).

Techniques

The standard coil technique with platinum detachable coils is the procedure mostly in use to treat ruptured IAs (Guglielmi et al. 1991a, Guglielmi et al. 1991b). The diagnostic DSA is performed prior to the coiling. The placement of flexible microcatheters into the intracranial circulation is by way of a femoral puncture and angiographic technique. 3DRA with post processing images is necessary to determine the details of an aneurysm and its environment (Anxionnat et al. 2001).
The patients have heparinization per-procedurally and in cases of coil protrusion into a parent artery or verified wide-necked aneurysms low molecular heparin or acetylsalicylic acid is continued for a few days after the procedure. During the coiling the activating clotting time (ACT) is monitored, and the level is targeted to 200–250 sec.

In the balloon remodelling technique a non-detachable balloon is inflated temporarily in a parent vessel in front of the neck during each coil placement (Moret et al. 1994, Moret et al. 1997). In the stent remodelling technique a stent is placed into a parent vessel and coils are inserted through the stent wall into the aneurysm sac (Kessler et al. 2005). Liquid embolization with ethylene vinyl alcohol copolymer (Onyx) is seldom used, but is a proper method in large or giant broad-necked aneurysms (Cekirge et al. 2006). In peripheral aneurysms, especially fusiform and dissecting aneurysms, the occlusion of a parent artery with coils or liquid material is a method of choice (Isokangas 2006). The use of a covered stent excluding the aneurysm sac without employing any material may be feasible with some complex IAs situated especially in proximal regions (Saatci et al. 2004).

**Indications, results and outcome**

Indications for endovascular treatment of ruptured IAs are expanding and predictive selection criteria are becoming clearer. A list of absolute and relative indications is presented in Table 6 (Byrne 2000).

**Table 6. Indications for endovascular treatment of ruptured IAs (Byrne 2000).**

<table>
<thead>
<tr>
<th>Indications</th>
<th>Aneurysm criteria</th>
<th>Patient criteria</th>
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<tbody>
<tr>
<td><strong>Absolute</strong></td>
<td>Posterior fossa aneurysms</td>
<td>Extreme old age</td>
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<td></td>
<td>Surgically inaccessible aneurysms</td>
<td>Poor clinical grade</td>
</tr>
<tr>
<td></td>
<td>Distal aneurysms</td>
<td>Medical contra-indication to craniotomy</td>
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<tr>
<td></td>
<td>Failed surgical clipping</td>
<td>Religious objection to blood transfusion</td>
</tr>
<tr>
<td><strong>Relative</strong></td>
<td>Angiographic vasospasm</td>
<td>Patient choice</td>
</tr>
<tr>
<td></td>
<td>Multiple aneurysms</td>
<td>Professional drivers/pilots</td>
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<td></td>
<td>Flow aneurysms</td>
<td>Randomised trials</td>
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<tr>
<td></td>
<td>Prior to ICH evacuation</td>
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</tr>
<tr>
<td></td>
<td>Infectious aneurysms</td>
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</table>

The most common type of complication is thromboembolic events. In a review of 23 studies using GDC for treatment of IAs by Qureshi et al. (2000) the overall rate of thromboembolic complications was 8.2%. In a study of Willinsky et al. (2009) dealing with ruptured IAs thromboembolic complications occurred in 5.8%. They are mostly asymptomatic and transient in nature, such as transient ischemic attacks. The catheterization or placement of coils may lead to aneurysm perforation. A recently ruptured IA carries a higher risk for this complication, but the perforation is mostly dependent on the coiling technique (Tummala et al. 2001, Ng et al. 2002). The successful treatment after iatrogenic aneurysm rupture include reverse anticoagulation therapy and continued coiling procedure aiming to achieve complete occlusion (Vinuela et al. 1997, Tummala et al. 2001, Spelle et al. 2006). The rapid and proper treatment of aneurysm perforation results in a good clinical outcome (Spelle et al. 2006).


The clinical outcome after endovascular treatment of a ruptured IA is highly dependent on the neurological state of the patient. The studies with follow-up clinical outcomes present GOS 4–5 in 57–81%, GOS 2–3 in 5–30% and GOS 1 in 5–23% of the patients (Byrne et al. 1999, Raymond & Roy 1997, Vanninen et al. 1999, Friedman et al. 2003, Molyneux et al. 2005, Norbäck et al. 2005). The overall results are comparable to neurosurgery. The formation of recurrent aneurysm is higher up to two years after the treatment, but usually it has no effect on clinical outcome (Hayakawa et al. 2000, Raymond et al. 2003b). The risk of epilepsy is lower in patients allocated to endovascular coiling than after neurosurgery (Ukkola et al. 2001). Endovascular coiling is more likely to result in independent survival at 1 year than neurosurgical clipping. This survival benefit continues for at least 7 years (Molyneux et al. 2005).
2.4.3 Comparative studies of chronic hydrocephalus after neurosurgical and endovascular treatment

There are discrepancies between reports of the incidence of chronic HC after neurosurgical and endovascular treatment of ruptured IAs (Gruber et al. 1999, Vanninen et al. 1999, Sethi et al. 2000, Taha et al. 2006, Varelas et al. 2006). A few publications have suggested that chronic HC is more likely to develop after endovascular coiling (Dorai et al. 2003, Varelas et al. 2006). Auer and Mokry reported (1990), that the evacuation of cisternal and subarachnoidal clots and the use of CSF drainage reduce the risk of HC. This statement is based on the opinion that, in open surgery, the removal of clots diminishes the disturbance of CSF circulation by reducing the formation of leptomeningeal fibrosis and blocks in the minor pathways. On the contrary, some authors have reported that the treatment method used does not affect the risk of chronic HC (Gruber et al. 1999, Sethi et al. 2000, Taha et al. 2006). According to a study by Dehdashti et al. (2004) no significant difference in shunt-dependent HC was seen between the treatment groups after controlling for covariates. Vanninen et al. (1999) found that more patients with HC needed shunt operation in the surgical treatment group. They assumed that the inflammation caused by surgery may lead to aseptic arachnoiditis and disturbance of CSF flow.

2.5 Rebleeding of intracranial aneurysms

Rebleeding after ruptured IAs occurs usually soon after the initial haemorrhage, within three days of ictus and most often during the first few hours (Brilstra et al. 2000, Ohkuma et al. 2001, Naidech et al. 2005). In the first few hours up to 15% of patients have sudden worsening of the clinical condition suggesting rebleeding (Ohkuma et al. 2001). The total risk of rebleeding without intervention during the first four weeks after the first day of rupture has been 35–40% (Hijdra et al. 1987, Brilstra et al. 2002). In untreated IAs the risk of rebleeding decreases in the course of time from the level of 1–2% a day to the level of 3% a year (Winn et al. 1977). Rebleeding is often disabling or fatal, and the mortality rate is over 50% (Byrne et al. 1999, Holmin et al. 2008, Willinsky et al. 2009).
2.5.1 Rebleeding after neurosurgical treatment

Rebleeding is possible even after a successful neurosurgical procedure. The movement of the clip or an unobserved neck remnant may lead to the growth of an aneurysm and rehaemorrhage (Lin et al. 1989). The incidence of rebleeding after neurosurgery has been reported 1.2% over 4 years and 2.2–3.2% over 10 years (Tsutsumi et al. 1998, Molyneux et al. 2005, Wermer et al. 2005). In the ISAT trial the incidence of rebleeding from a primarily completely clipped aneurysm was 0.5%, 11 out of 13 rebleedings occurred during the first year, and 7 of them (54%) were fatal (Molyneux et al. 2002, Molyneux et al. 2005). In a large study containing 727 completely clipped aneurysms Asgari et al. (2003) found the incidence of rerupture 0.4%. Fridriksson et al. (2002) presented 5 rebleedings (1.4%) among 355 clipped aneurysms with six months follow-up. Van der Schaaf et al. (2005) identified three randomised trials dealing with aneurysms of anterior circulation, and 14 (1.2%) of 1137 patients allocated for surgical clipping had an episode of rebleeding up to one year after treatment.

Wermer et al. (2005) presented a cohort of 752 patients after successful clipping with long-term clinical follow-up (mean 8 years). The patients were interviewed about new attacks of SAH. In 18 patients (2.4%) a new SAH was found, and 10 of them the attack was fatal. Tsutsumi et al. (2001) reported 2.9% aneurysm re-growths after clipping on long-term follow-up angiography. David et al. (1999) found a late rebleeding risk of 1.9% per year after a mean follow-up period of 4.4 years.

2.5.2 Rebleeding after endovascular treatment

Early rebleeding

The rate of early rehaemorrhage (within 30 days) after endovascular treatment is low. The reports have shown the incidence up to 1.9%, depending on the method criteria used (Vanninen et al. 1999, Molyneux et al. 2005, Sluzewski et al. 2005a, Willinsky et al. 2009). In the ISAT trial the authors observed an incidence of 1.5% (Molyneux et al. 2005). In a study with 431 consecutive coiled patients Sluzewski et al. (2005a) presented an incidence of 1.4%. Willinsky et al. (2009) recently documented 6 patients with early rebleeding (1.6%) among 377 coiled patients, 5 in the first 48 hours.

The presence of an ICH at admission may increase the risk of rehaemorrhage. Sluzewski et al. (2005a) reported five out of six early rebleeding cases with an adjacent ICH. The reopening of thrombosed pseudoaneurysm in the ICH may
cause early rehaemorrhage (Nomura et al. 2000, Mori et al. 2004, Tanoue et al. 2004, Sluzewski et al. 2005a). This theory explains why rehaemorrhage may occur as an enlargement of ICH (Fig. 9). The poor clinical condition of the patient has risen to be a risk factor of early rebleeding (Sluzewski et al. 2005a). However, it is difficult to present poor clinical condition as an independent risk factor, because it is strongly dependent on ICH, IVH and HC.

![Fig. 9. Early rehaemorrhage after coiling. 60 year old man with ruptured ACoA aneurysm. A. CT image before coiling. A small ICH between the frontal horns of the lateral ventricles and mild ventricular enlargement. B. CT image at the same level after coiling with incomplete aneurysm occlusion. ICH has enlarged and HC has progressed.](image)

Some reports have proved that a sub-totally packed aneurysm increases the risk of rebleeding (Vinuela et al. 1997, Molyneux et al. 2005, Johnston et al. 2008). In the CARAT study the initial occlusion grade < 70% was a strong predictor for rehaemorrhage (Johnston et al. 2008). Rehaemorrhage may also occur after complete occlusion. This rate is less than 0.8% (Asgari et al. 2003, Molyneux et al. 2005, Willinsky et al. 2009). Coil compaction, recanalization, coil migration, and recurrent aneurysm formation are possible events in the early phase (Bavinzski et al. 1999, Nguyen et al. 2007). Sluzewski et al. (2005a) observed that aneurysm size less than 6 mm is an independent risk factor of rebleeding. In other reports an increasing effect of large aneurysm size has been raised (Naidech et al. 2005, Pleizier et al. 2006). An example of a patient with ICH suffering early rebleeding and residual aneurysm after coiling is presented in Fig. 9.
The insertion of EVD to manage HC before treatment of a ruptured IA is presented as a risk factor of rebleeding (Hasan et al. 1989, Bogdahn et al. 1992, Paré et al. 1992, Kawai et al. 1997, Fountas et al. 2006). EVD may reduce intracranial pressure and cause a ventricular shift, and raise aneurysmal transmural pressure (Paré et al. 1992). Insertion of a catheter may lead to fibrinolytic activity and lysis of the aneurysm clot (Hasan et al. 1991). Controversially, in a study of McIver et al. (2002) no evidence was found that preoperative EVD is associated with an increased risk of aneurysm rebleeding. Patients receiving additional thrombolytic therapy during the coiling procedure or prolonged heparinization are at a greater risk of rebleeding. The ISAT study (Molyneux et al. 2002) presented five out of 20 early rebleeding patients with intravenous thrombolysis.

The outcome of the patients after early rehaemorrhage is usually poor. In two recent studies nearly all of these patients died (Sluzewski et al. 2005a, Willinsky et al. 2009).

Late and overall rebleeding

The relative risk of late rebleeding (> 30 days after treatment) is higher after endovascular than neurosurgical treatment. The overall incidence of rebleeding is 2.6–3.3% over 3.5–4.2 years (Kremer et al. 2002, Molyneux et al. 2005, Aikawa et al. 2007). In a recent report Willinsky et al. (2009) had a mean follow-up of 22 months and an incidence of rehaemorrhage of 2.1% (8/377). Raymond et al. (2003b) presented an incidence of 1.1% (3/271) and Byrne et al. (1999) 1.3% (4/317). In a long-term follow-up study rebleeding after coiling of ruptured IAs was infrequent with an incidence of 1.3% (5/392) and annual late rebleeding rate is as low as 0.35% (Sluzewski 2005b). The longest reported interval between endovascular treatment and rebleeding is almost 10 years (Willinsky et al. 2009).

Risk factors for the occurrence of late rebleeding have been found to be a large aneurysm size, an initial incomplete aneurysm occlusion, and an incomplete aneurysm occlusion at 6-month follow-up angiography (Sluzewski et al. 2003, Sluzewski et al. 2005b, Aikawa et al. 2007). It is controversial if a complete aneurysm occlusion at 6 months opens later. In two studies by Sluzewski et al. (2003, 2005b) late reopening of an aneurysm was not observed. In a later study none of 248 aneurysms that were completely occluded rebled over a very long period. However, Raymond et al. (2003b) found, that a considerable proportion of aneurysms reopen after 6 months. In a study by Sluzewski et al. (2005b) mortality of late rebleeding was 0.76% (3/393), and the annual mortality rate from late rebleeding was 0.19%.
3 Purpose of the study

The main purposes of this study were:

1. to evaluate the reliability to localize the ruptured IA with the help of blood amount and distribution on non-contrast CT
2. to evaluate the risk factors of acute HC after SAH
3. to compare the occurrence of chronic HC after neurosurgical and endovascular treatment of ruptured IAs
4. to evaluate the risk factors of early rebleeding after coiling of ruptured IAs
4 Materials and methods

4.1 Patients

The study populations consisted of three patient groups. All the patients suffered SAH caused by a ruptured IA, and patients were either neurosurgically clipped or endovascularly coiled in Oulu University Hospital. The clinical and radiological data was collected retrospectively with the permission required by reviewing imaging studies and the medical records. The basic characteristics of the patients and aneurysms in studies I–IV are summarized in Table 7.

Table 7. The basic characteristics of the patients and aneurysms in the studies I–IV.

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Study I–II (n = 180)</th>
<th>Study III (n = 209)</th>
<th>Study IV (n = 194)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Surgical group</td>
<td>Endovascular group</td>
<td></td>
</tr>
<tr>
<td>Male / Female</td>
<td>100 / 80</td>
<td>48 / 54</td>
<td>89 / 105</td>
</tr>
<tr>
<td>Age years (mean (range))</td>
<td>47 (21–74)</td>
<td>52 (21–82)</td>
<td>54 (14–82)</td>
</tr>
<tr>
<td>Aneurysm location</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anterior circulation</td>
<td>173</td>
<td>100</td>
<td>88</td>
</tr>
<tr>
<td>ICA*</td>
<td>24</td>
<td>7</td>
<td>30</td>
</tr>
<tr>
<td>ACA**</td>
<td>64</td>
<td>35</td>
<td>44</td>
</tr>
<tr>
<td>MCA</td>
<td>85</td>
<td>58</td>
<td>14</td>
</tr>
<tr>
<td>Posterior circulation</td>
<td>7</td>
<td>2</td>
<td>19</td>
</tr>
<tr>
<td>BA</td>
<td>3</td>
<td>–</td>
<td>12</td>
</tr>
<tr>
<td>Other</td>
<td>4</td>
<td>2</td>
<td>7</td>
</tr>
<tr>
<td>Aneurysm size</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Small (1–9 mm)</td>
<td>134</td>
<td>79</td>
<td>85</td>
</tr>
<tr>
<td>Large (10–25 mm)</td>
<td>46</td>
<td>23</td>
<td>22</td>
</tr>
<tr>
<td>Giant (&gt; 25 mm)</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Multiple aneurysms</td>
<td>26</td>
<td>30</td>
<td>23</td>
</tr>
<tr>
<td>H&amp;H***</td>
<td>I–II NE</td>
<td>61</td>
<td>48</td>
</tr>
<tr>
<td></td>
<td>III NE</td>
<td>23</td>
<td>30</td>
</tr>
<tr>
<td></td>
<td>IV–V NE</td>
<td>18</td>
<td>29</td>
</tr>
</tbody>
</table>

NE = not examined
* including posterior communicating artery, ** including anterior communicating artery, ***on admission (III), prior to treatment (IV)
4.1.1 Study I and II

The study population was collected from 548 patients operated in 1988–1999. The definitive data consisted of 180 neurosurgically clipped patients. The inclusion criteria were: a) the DSA and CT examinations were performed in Oulu University Hospital, b) non-contrast CT was performed within 24 hours after SAH c) selective DSA was performed within 48 hours after SAH. All the other patients were excluded.

4.1.2 Study III

The study population covers patients from two series: 399 neurosurgically clipped patients from February 1999 to December 2005 and 494 endovascularly coiled patients from December 1993 to December 2005. The DSA and CT examinations were undertaken in Oulu University Hospital. Exclusion criteria were: a) incidental unruptured aneurysms, b) patients treated with parent artery occlusion, c) aneurysms treated both neurosurgically and endovascularly, d) unsuccessful treatment attempts, e) conservatively treated patients, f) missing data. The definitive data include 102 clipped and 107 coiled patients.

Table 8. The differences of patient’s gender, age, clinical condition, aneurysm size or multiplicity between the treatment modalities (III).

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Surgical n = 102</th>
<th>Endovascular n = 107</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td>48</td>
<td>44</td>
<td>0.4061</td>
</tr>
<tr>
<td>Age, years</td>
<td>52</td>
<td>53</td>
<td>0.6033</td>
</tr>
<tr>
<td>Multiple aneurysms</td>
<td>30</td>
<td>23</td>
<td>0.2061</td>
</tr>
<tr>
<td>Size</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Small</td>
<td>79</td>
<td>85</td>
<td>0.7401</td>
</tr>
<tr>
<td>Large (≥ 10mm)</td>
<td>23</td>
<td>22</td>
<td></td>
</tr>
<tr>
<td>H&amp;H*</td>
<td></td>
<td></td>
<td>0.0852</td>
</tr>
<tr>
<td>I-II</td>
<td>61</td>
<td>48</td>
<td></td>
</tr>
<tr>
<td>III</td>
<td>23</td>
<td>30</td>
<td></td>
</tr>
<tr>
<td>IV-V</td>
<td>18</td>
<td>29</td>
<td></td>
</tr>
<tr>
<td>Time to (days; median, range):</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>2.0 (0-23)</td>
<td>1.0 (0-22)</td>
<td>0.0394</td>
</tr>
<tr>
<td>Follow-up CT</td>
<td>74 (39-273)</td>
<td>68 (14-1975)</td>
<td>0.0884</td>
</tr>
</tbody>
</table>

1Fisher’s exact test. 2Chi-square test. 3Student’s t-test. 4Mann-Whitney U-test
*on admission
The H&H was used to grade clinical condition on admission. As regards the patient’s gender, age or clinical condition on admission or aneurysm size or multiplicity, there were no statistically significant differences between the groups. The interval from the onset of bleeding to the treatment was 2.0 d (range 0–23 d) before clipping and 1.0 d (range 0–22 d) before coiling (p = 0.039). The differences between the treatment groups are shown in Table 8.

4.1.3 Study IV

The study population consisted of consecutive endovascularly coiled patients from January 2000 to December 2007. In all the patients SAH was confirmed by CT or, in four cases where the CT scan was negative for SAH, by lumbar puncture. The aneurysm(s) was verified by 4-vessel DSA in all of the patients and coiling was performed in the acute stage within 3 days from the onset of the SAH. In four patients with multiple aneurysms, the ruptured aneurysm could not be determined by imaging and two aneurysms were treated at the initial session. These four patients with 8 aneurysms were excluded from the analysis. Altogether 194 patients and aneurysms were included. The H&H was used to grade clinical condition at the time of coiling.

4.2 Imaging methods

4.2.1 Computed tomography (I–IV)

Studies I–II. Non-enhanced cranial CT with 4 or 5 mm axial slice thickness in the posterior fossa and 8–10 mm slice thickness in the suprasellar region was performed as the first examination within 24 hours after the onset of the symptoms of SAH.

Study III. The follow-up non-enhanced CT was performed two weeks or later after the treatment.

Study IV. Early rebleeding was defined by CT images revealing increased amount of blood compared to initial images.

4.2.2 Digital subtraction angiography (I–IV) and 3D rotational angiography (IV)

Studies I–IV. The patients underwent 4-vessel selective DSA (Angiontron Siemens, Polytron DSA/Neurostar Siemens, Polytron Plus DSA/Bicor HS CF Siemens,
Biplane Neurostar Siemens, Erlangen, Germany; Integris Allura Philips, Best, the Netherlands) within 48 hours (I–III) and 72 hours (IV) of the onset of SAH.

Study IV. 3DRA (Integris Allura Philips, Best, the Netherlands) with post-processing images was available and used from July 2003 to determine the details of the aneurysm and its environment.

4.3 Analysis of images

4.3.1 Aneurysm and haemorrhage (I–IV)

Study I–II. The location of the ruptured IA on non-contrast CT was predicted independently by two neuroradiologists. The amount of blood in the ventricles, cisterns and fissures were determined by two experienced neuroradiologists using the grading scale described by Hijdra et al. (1990). The observers used the grading scale to support the subjective estimation of the precise location. All the patients underwent an operation in which the site of the ruptured aneurysm was determined. In cases of disagreement, consensus after discussion was used as the result. The amount of extravasated blood was graded in each cistern and ventricles on both sides: 0 = no blood, 1 = small amount of blood, 2 = moderately filled with blood, 3 = completely filled with blood. The sum score for ten cisterns and fissures (= SAHsc) ranged from 0–30. The sum score for four ventricles (= IVHsc) ranged from 0–12.

Study II. SAHsc were categorized into subgroups: a) 0.0–15.5 and b) 16.0–30. The IVHsc were categorized into subgroups: a) 0.0–0.5 b) 1.0–12.0. A mean value of the two observers was used.

Study III. The SAHsc were categorized into the subgroups: a) grade 1 (0–10 scores), b) grade 2 (11–20 scores) c) grade 3 (21–30 scores). The IVHsc were categorized into the subgroups: a) grade 1 (0 score), b) grade 2 (1–4 scores) c) grade 3 (5–12 scores).

Study I–IV. The location and volume of ICH (ICHvol) and the type of haemorrhages were registered. The localization and maximum diameter of the ruptured aneurysm were determined. The initial occlusion grade at the end of the procedure was classified by an experienced interventional radiologist using a modified Raymond scale as a complete occlusion, a neck remnant (dog ear or residual neck), or a residual aneurysm (Raymond et al. 2003b) (IV).
4.3.2 Hydrocephalus (II–IV)

Acute HC was identified by observing ventricular size, rounded frontal horns and diminished cerebral sulci. The wIII and CMI were calculated on CT images. The values of CMI above 0.24 and wIII above 7 mm were considered as pathological (II). Chronic HC was considered to be present based on visual impression in the follow-up CT and/or on the need for EVD or permanent shunt (III). The assessment of EVD for treatment and as an indicator of HC was checked from clinical data (IV).

4.3.3 Early rebleeding (IV)

Study IV. Early rebleeding was defined as appearing within 30 days after coiling with worsening of the patient’s clinical condition and diagnosed by CT images revealing an increased amount of blood compared to initial images. The onset of rebleeding was established according to the time of worsening of the patient’s clinical condition. The timing of coiling was calculated as days from an initial haemorrhage. Delay of rebleeding was calculated as days after coiling. The outcome of the patients suffering rehaemorrhage was verified retrospectively from patients’ files using the GOS (Jennet & Bond 1975).

4.4 Treatment methods

The patients’ selection between neurosurgical and endovascular treatment was decided by a multidisciplinary team consisting of neurosurgeon(s) and neurointerventional radiologist(s). Standard SAH management practices were used to prevent HC, vasospasm and rebleeding. The operation to assess EVD or permanent shunt was performed by a neurosurgeon when necessary to treat HC.

Study I–III. The neurosurgical aneurysm operations were performed under general anaesthesia by a team of neurosurgeons using a standard microsurgical method for clipping of the aneurysm neck. During operations the blood clots were rinsed out from basal cisterns without chemical agents.

Studies III–IV. The coiling procedures were performed by a neuroradiological interventional team. All the treatments were performed under general anaesthesia and the patients were heparinised during the procedure (activating clotting time was monitored to target the level 200–250 seconds). In cases of coil protrusion into a parent artery or verified wide-necked aneurysms, low molecular heparin or acetylsalicylic acid was continued for a few days after the procedure. The techniques
used were the standard coil technique with detachable platinum coils, the balloon remodelling technique and the stent remodelling technique (Guglielmi et al. 1991a, Guglielmi et al. 1991b, Moret et al. 1997, Kessler et al. 2005).

4.5. Statistical methods

The SPSS software was used in the statistical analysis (SPSS, version 9.0.1; version 13.0; SPSS, Chicago Ill, USA). The statistical level of significance was set at p < 0.05.

Study I. The reliability values (κ- values) of each neuroradiologist for each vessel were calculated separately by comparing the neuroradiologist’s review of the CT images to the operative finding, which was considered “the gold standard”. The κ- values of the neuroradiologists were compared. A κ- value for each vessel, concerning the level of agreement between the neuroradiologists was also calculated. The criteria judging κ- coefficients was: A κ- value of 0.81–1.00 indicates almost perfect reliability, 0.61–0.80 substantial, 0.41–0.60 moderate, 0.21–0.40 fair, 0.00–0.20 slight and less than 0.00 poor reliability (Landis & Koch 1977). The statistical significance was calculated as p-values. χ²-test was used to ascertain if SAH with an ICH is a good predictor of the site of a ruptured aneurysm.

Study II. The analysis was made by using a logistic regression model to evaluate the value of the factors predicting acute HC, and the probability of acute HC could be presented with model formulae. The examiners’ observations of SAHsc, IVHsc and ICHVol were combined before the estimation. Analysis of variance was used to assess the distribution of CMI and wIII, and χ²-test to assess the existence of acute HC in the categories of the factors of interest.

Study III. Fisher’s exact test was used to compare the differences in sex, aneurysm size and multiplicity between the treatment groups. The influence of age was calculated with Student’s t-test. The effect of H&H grades and the differences in SAHsc and IVHsc were analysed using χ²- test. The rates of occurrence of ICH in the neurosurgical and endovascular subgroups were compared with Mann-Whitney U- test.

Study IV. The risk factors for early rebleeding were analysed using logistic regression analysis with early rebleeding as a dependent variable and the possible risk factors separately as independent variables (univariate analyses). The odds ratios (OR) with 95% confidence intervals (95% CI) were presented, as well as the frequency distributions of risk factors. The significance of the predictors which did not suit the logistic model was defined separately using a χ²- test.
5 Results

5.1 Localization of ruptured aneurysm on non-contrast CT (I)

The numbers of correctly located aneurysms with respect to the gold standard (DSA) by both neuroradiologists are presented in Table 9. In 45/56 patients with ACoA aneurysm, the correct diagnoses were identical for both observers. In 48/51 patients with right MCA aneurysms the correctly located aneurysms were the same. In the group of 34 left MCA aneurysms in 30 patients the correctly located ruptured aneurysm was the same for both. All BA aneurysms were correctly located by both evaluators. IAs in other vessels were seldom located correctly.

Table 9. The numbers of correctly located aneurysms with respect to DSA by both neuroradiologists.

<table>
<thead>
<tr>
<th>Location</th>
<th>DSA</th>
<th>Nr1</th>
<th>Nr2</th>
<th>Nr1+Nr2</th>
</tr>
</thead>
<tbody>
<tr>
<td>MCA dex</td>
<td>51</td>
<td>49</td>
<td>49</td>
<td>48</td>
</tr>
<tr>
<td>MCA sin</td>
<td>34</td>
<td>30</td>
<td>32</td>
<td>30</td>
</tr>
<tr>
<td>ACA dex</td>
<td>5</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>ACA sin</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>PCA sin</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>ACoA</td>
<td>56</td>
<td>52</td>
<td>47</td>
<td>45</td>
</tr>
<tr>
<td>PCoA dex</td>
<td>5</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>PCoA sin</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>BA</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>PICA dex</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>PICA sin</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>ICA dex</td>
<td>12</td>
<td>6</td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>ICA sin</td>
<td>5</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Overall</td>
<td>180</td>
<td>142</td>
<td>139</td>
<td>129</td>
</tr>
</tbody>
</table>

Nr1 = neuroradiologist 1, Nr2 = neuroradiologist 2
Nr1+Nr2 = correctly located IA were identical for both
The κ-values of both observers for each ruptured vessel are presented in Table 10. For right and left MCA and ACoA aneurysms the κ-values between the two neuroradiologists are alike. For right ICA aneurysms the κ-value of Nr1 is 0.613 and that of Nr2 is 0.247. This was the largest difference in the κ-values between the two. For the rest of the vessels the sample size was very small. ICH was a good predictor for evaluating the location of the ruptured aneurysm, with a statistical significance of p = 0.003 (data not presented).

Table 10. Reliability values (κ-values) of the neuroradiologists for each ruptured aneurysm.

<table>
<thead>
<tr>
<th>Location</th>
<th>n</th>
<th>Nr1*</th>
<th>Nr2*</th>
<th>Nr1 vs Nr2**</th>
</tr>
</thead>
<tbody>
<tr>
<td>MCA dex</td>
<td>51</td>
<td>0.893</td>
<td>0.831</td>
<td>0.911</td>
</tr>
<tr>
<td>MCA sin</td>
<td>34</td>
<td>0.861</td>
<td>0.861</td>
<td>0.877</td>
</tr>
<tr>
<td>ACA dex</td>
<td>5</td>
<td>0.203</td>
<td>0.203</td>
<td>0.489</td>
</tr>
<tr>
<td>ACA sin</td>
<td>3</td>
<td>0.008</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>PCA dex</td>
<td>0</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>PCA sin</td>
<td>1</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>ACoA</td>
<td>56</td>
<td>0.733</td>
<td>0.720</td>
<td>0.736</td>
</tr>
<tr>
<td>PCoA dex</td>
<td>5</td>
<td>0.327</td>
<td>0.274</td>
<td>0.664</td>
</tr>
<tr>
<td>PCoA sin</td>
<td>2</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>BA</td>
<td>3</td>
<td>0.590</td>
<td>0.487</td>
<td>0.739</td>
</tr>
<tr>
<td>PICA dex</td>
<td>1</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>PICA sin</td>
<td>2</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>ICA dex</td>
<td>12</td>
<td>0.613</td>
<td>0.247</td>
<td>0.386</td>
</tr>
<tr>
<td>ICA sin</td>
<td>5</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Overall</td>
<td>180</td>
<td>0.704</td>
<td>0.655</td>
<td>0.780</td>
</tr>
</tbody>
</table>

* κ-values of Nr1 and Nr2 concern the level of agreement between an individual observer and the gold standard
** κ-values of Nr1 vs Nr2 concern the level of agreement between the observers
(not all κ-values were calculated, either because the location was constant or because the number of ruptured IAs was too small)

5.2 Risk factors of acute hydrocephalus after subarachnoid haemorrhage (II)

Cranial CT showed acute HC on admission in 62% of the patients when the ventricle and the temporal horn size were analysed by visual impression. Twenty-three
percent of the calculated values of CMI and 25% of the wIII were pathological. The SAHsc was statistically significant (p = 0.005) in the logistic regression model indicating that having blood distributed into several regions increases the risk of acute HC. The IVHsc and the ICHvol to predict acute HC were also statistically significant (p < 0.001 and p = 0.009, respectively). The detailed statistics of the factors of the logistic regression model predicting the occurrence of acute HC are given in Table 11.

Table 11. The factors of the logistic model predicting the occurrence of acute hydrocephalus.

<table>
<thead>
<tr>
<th>Factors</th>
<th>OR</th>
<th>95% CI</th>
<th>Change in -2 log likelihood (df)</th>
<th>p *</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAHsc</td>
<td>7.74</td>
<td>(1) 0.005</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SAHsc ≤ 15.1</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>16.0 ≤ SAHsc</td>
<td>2.77</td>
<td>1.34–5.71</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IVHsc</td>
<td>24.61</td>
<td>(1) &lt;0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IVHscore ≤ 0.5</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.0 ≤ IVHscore</td>
<td>6.85</td>
<td>2.98–15.72</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ICHvol</td>
<td>9.40</td>
<td>(2) 0.009</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ICH vol ≤ 3</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 ≤ ICH vol ≤ 30</td>
<td>2.72</td>
<td>1.09–6.79</td>
<td></td>
<td></td>
</tr>
<tr>
<td>31 ≤ ICH vol</td>
<td>8.62</td>
<td>0.98–76.06</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Model predicted the occurrence of acute hydrocephalus correctly in 86.6% of positive cases and 52.9% of negative cases.

1 Control group.

OR = odds ratio, 95% CI = 95% confidence interval for the odds ratio.

* Significance of removing the predictor from the model.

The influence of factors to CMI was statistically significant in IVHsc (p = 0.003), ICHvol (p < 0.001) and the presence of IVH with other haemorrhage (p < 0.001). The p-values for SAHsc, IVHsc and ICHvol from the χ²-test to influence on occurrence of HC are all statistically significant. The incidence of HC among the patients with ventricular blood was much higher than that among the patients without ventricular blood. There was dissimilarity in the occurrence of HC in the different region combinations (p = 0.010). There were large variations between
the effects of the different types of haemorrhage on the occurrence of acute HC (p < 0.001). The detailed data of the effect of risk factors on acute HC and wIII related with blood amount and distribution into different regions are presented in Table 12.

Table 12. The influence of factors related with blood by mean and its 95% confidence interval on calculated CMI, wIII, and by proportions of occurrence on acute HC.

<table>
<thead>
<tr>
<th>Factors</th>
<th>CMI * 100</th>
<th>wIII (mm)</th>
<th>Occurrence of acute HC</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n  Mean 95% CI</td>
<td>p  Mean 95% CI</td>
<td>p  n % p</td>
</tr>
<tr>
<td>TBsc</td>
<td>0.005</td>
<td>0.001</td>
<td>0.001</td>
</tr>
<tr>
<td>0.0–15.5</td>
<td>71 18.4 16.8–20.1</td>
<td>4.2 3.4–5.0</td>
<td>33 46.5</td>
</tr>
<tr>
<td>16.0–30.0</td>
<td>109 21.1 20.1–22.0</td>
<td>5.9 5.3–6.5</td>
<td>79 72.5</td>
</tr>
<tr>
<td>IVHsc</td>
<td>0.003</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>0.0–0.5</td>
<td>108 18.9 17.8–20.1</td>
<td>4.2 3.7–4.7</td>
<td>49 45.4</td>
</tr>
<tr>
<td>1.0–12.0</td>
<td>72 21.6 20.3–23.0</td>
<td>6.8 6.0–7.7</td>
<td>63 87.5</td>
</tr>
<tr>
<td>ICHvol (ml)</td>
<td>&lt;0.001</td>
<td>0.066</td>
<td>0.019</td>
</tr>
<tr>
<td>0.0–3.0</td>
<td>132 21.0 20.0–21.9</td>
<td>5.5 4.9–6.0</td>
<td>75 56.8</td>
</tr>
<tr>
<td>4.0–30.0</td>
<td>35 18.8 16.8–20.9</td>
<td>5.1 3.6–6.7</td>
<td>25 71.4</td>
</tr>
<tr>
<td>31.0–80.0</td>
<td>13 13.4  8.9–17.9</td>
<td>3.2 1.3–5.0</td>
<td>12 92.3</td>
</tr>
<tr>
<td>Blood distribution</td>
<td>0.338</td>
<td>0.049</td>
<td>0.010</td>
</tr>
<tr>
<td>Anterior</td>
<td>7 19.0 14.6–23.4</td>
<td>3.6 0.9–6.2</td>
<td>2 28.6</td>
</tr>
<tr>
<td>Lateral</td>
<td>9 18.9 14.9–22.9</td>
<td>5.3 2.8–7.8</td>
<td>5 55.6</td>
</tr>
<tr>
<td>Ant.+Lat.</td>
<td>30 18.8 16.1–21.5</td>
<td>4.1 2.8–5.4</td>
<td>12 40.0</td>
</tr>
<tr>
<td>Ant.+Lat.+Basal</td>
<td>113 20.8 19.7–21.8</td>
<td>5.8 5.2–6.5</td>
<td>80 70.8</td>
</tr>
<tr>
<td>Differing opinions</td>
<td>21 18.6 15.4–21.7</td>
<td>4.1 2.7–5.5</td>
<td>13 61.9</td>
</tr>
<tr>
<td>Type of haemorrhage</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>SAH</td>
<td>70 20.1 18.9–21.3</td>
<td>4.5 3.9–5.1</td>
<td>31 44.3</td>
</tr>
<tr>
<td>SAH+IVH</td>
<td>46 23.4 22.1–24.7</td>
<td>7.2 6.2–8.2</td>
<td>39 84.8</td>
</tr>
<tr>
<td>SAH+ICH</td>
<td>36 16.5 14.1–18.9</td>
<td>3.3 2.3–4.2</td>
<td>17 47.2</td>
</tr>
<tr>
<td>SAH+ICH+IVH</td>
<td>24 18.5 15.6–21.4</td>
<td>6.2 4.4–8.1</td>
<td>23 95.8</td>
</tr>
<tr>
<td>Differing opinions</td>
<td>4 21.3 17.1–25.4</td>
<td>7.0 0.1–13.9</td>
<td>2 50.0</td>
</tr>
<tr>
<td>Total</td>
<td>180</td>
<td>112</td>
<td>62.2</td>
</tr>
</tbody>
</table>

p- values for CMI and wIII are from analysis of variance and for acute HC from $\chi^2$- test.
The age or gender of the patient or the size and location of the aneurysm had no significant effect on the occurrence of acute HC. The values of wIII increased along with the patient’s age, a similar but not significant tendency was seen with CMI. There was no such relation between HC and age. The values of both CMI and wIII were higher among males than among females, but again, no corresponding difference was seen in the occurrence of HC. The statistics for these factors are presented in Table 13.

**Table 13. The influence of patient’s gender and age, location and size of aneurysm on occurrence of acute HC and values of CMI and wIII.**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>CMI *100</th>
<th>Occurrence of acute HC</th>
<th>wIII (mm)</th>
<th>n</th>
<th>Mean</th>
<th>95% CI</th>
<th>p</th>
<th>Mean</th>
<th>95% CI</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient’s sex</td>
<td>0.112</td>
<td>0.643</td>
<td>0.001</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>100</td>
<td>20.7 19.3–22.0</td>
<td>6.0</td>
<td></td>
<td>5.2–6.7</td>
<td>64</td>
<td>64.0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>80</td>
<td>19.2 18.1–20.4</td>
<td>4.3</td>
<td></td>
<td>3.7–5.0</td>
<td>48</td>
<td>60.0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Patient’s age (y)</td>
<td>0.068</td>
<td>0.272</td>
<td>0.003</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>21–39</td>
<td>43</td>
<td>18.3 16.6–20.1</td>
<td>3.8</td>
<td></td>
<td>2.9–4.7</td>
<td>24</td>
<td>55.8</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>40–49</td>
<td>66</td>
<td>20.1 18.3–21.9</td>
<td>5.3</td>
<td></td>
<td>4.5–6.1</td>
<td>46</td>
<td>69.7</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>50–</td>
<td>71</td>
<td>21.0 19.9–22.2</td>
<td>6.1</td>
<td></td>
<td>5.2–6.9</td>
<td>42</td>
<td>59.2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Location</td>
<td>&lt;0.001</td>
<td>0.296</td>
<td>0.029</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AC</td>
<td>65</td>
<td>22.2 21.0–23.3</td>
<td>6.0</td>
<td></td>
<td>5.1–6.9</td>
<td>37</td>
<td>56.9</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MCA</td>
<td>85</td>
<td>18.3 16.8–19.8</td>
<td>4.5</td>
<td></td>
<td>3.8–5.2</td>
<td>54</td>
<td>63.5</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ICA</td>
<td>17</td>
<td>18.9 16.0–21.7</td>
<td>4.9</td>
<td></td>
<td>3.6–6.3</td>
<td>10</td>
<td>58.8</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PC</td>
<td>13</td>
<td>22.1 19.9–24.3</td>
<td>6.4</td>
<td></td>
<td>4.5–8.3</td>
<td>11</td>
<td>84.6</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Size (mm)</td>
<td>0.008</td>
<td>0.991</td>
<td>0.106</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0–4</td>
<td>55</td>
<td>20.2 18.6–21.7</td>
<td>5.3</td>
<td></td>
<td>4.3–6.3</td>
<td>34</td>
<td>61.8</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5–9</td>
<td>79</td>
<td>21.2 20.0–22.5</td>
<td>5.7</td>
<td></td>
<td>4.9–6.4</td>
<td>49</td>
<td>62.0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10–</td>
<td>46</td>
<td>17.8 15.8–19.8</td>
<td>4.4</td>
<td></td>
<td>3.4–5.3</td>
<td>29</td>
<td>63.0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>180</td>
<td>112 62.2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

p- values for CMI and wIII are from analysis of variance and for acute HC from χ² -test.

AC = anterior cerebral artery + anterior communicating artery

PC = posterior circulation + posterior communicating artery
5.3 Chronic hydrocephalus after treatment of ruptured intracranial aneurysms (III)

Table 14 shows the amount and types of haemorrhage in both treatment groups. The severity of the haemorrhage had an influence on the choice of the treatment method. Most of the patients with SAH and IVH grade 3 were coiled, and most of the patients with grade 1 were clipped. In the surgical group 37% and in the endovascular group 29% of the patients had ICH, but this difference was not statistically significant. Nevertheless, the patients with a large volume of ICH were mostly scheduled for neurosurgical clipping.

Table 14. Types of haemorrhage and amount of blood in the two treatment groups.

<table>
<thead>
<tr>
<th>Haemorrhages</th>
<th>Surgical n = 102</th>
<th>Endovascular n = 107</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAH sc</td>
<td></td>
<td></td>
<td>0.0871</td>
</tr>
<tr>
<td>grade 1 (0–10)</td>
<td>31 (30%)</td>
<td>22 (21%)</td>
<td></td>
</tr>
<tr>
<td>grade 2 (11–20)</td>
<td>29 (28%)</td>
<td>25 (23%)</td>
<td></td>
</tr>
<tr>
<td>grade 3 (21–30)</td>
<td>42 (41%)</td>
<td>60 (56%)</td>
<td></td>
</tr>
<tr>
<td>IVH sc</td>
<td></td>
<td></td>
<td>0.1671</td>
</tr>
<tr>
<td>grade 1 (0)</td>
<td>43 (42%)</td>
<td>33 (31%)</td>
<td></td>
</tr>
<tr>
<td>grade 2 (1–4)</td>
<td>33 (32%)</td>
<td>36 (34%)</td>
<td></td>
</tr>
<tr>
<td>grade 3 (5–12)</td>
<td>26 (25%)</td>
<td>38 (35%)</td>
<td></td>
</tr>
<tr>
<td>ICH</td>
<td></td>
<td></td>
<td>0.3582</td>
</tr>
<tr>
<td>volume (ml)*</td>
<td>30 (1–161)</td>
<td>8 (1–59)</td>
<td>0.0042</td>
</tr>
</tbody>
</table>

1Chi-square test. 2Fisher’s exact test. 3Mann-Whitney U-test.
*Median (range) within those with ICH.

Table 15 shows the number and proportions of subjects with acute and chronic HC and shunt dependence and the mean values of wIII and CMI. There were no significant differences between the treatment groups in the development of chronic HC (p = 0.570) or the need for a shunt operation (p = 0.881). Thirty patients with clipped aneurysms (29%) and 33 patients with coiled aneurysms (31%) needed a permanent or temporary shunt. Acute HC was seen in 51% in the surgical and in 65% in the endovascular group. The overall incidence of chronic HC was 37% (35% after clipping, 39% after coiling). The mean age of the patients without chronic HC was 50 years (SD ± 11.0), and with HC 56 years (SD ± 12.8) (p = 0.001).
Table 15. Numbers and percentages of subjects with acute and chronic HC and shunt dependence and the mean ± SD of wIII and CMI.

<table>
<thead>
<tr>
<th>Factors</th>
<th>Surgical n = 102</th>
<th>Endovascular n = 107</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute HC</td>
<td>52 (51%)</td>
<td>70 (65%)</td>
<td>0.051</td>
</tr>
<tr>
<td>Third ventricle</td>
<td>5.9 ± 3.4</td>
<td>7.9 ± 4.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Cella media index</td>
<td>0.23 ± 0.05</td>
<td>0.24 ± 0.04</td>
<td>0.049</td>
</tr>
<tr>
<td>Chronic HC</td>
<td>36 (35%)</td>
<td>42 (39%)</td>
<td>0.570</td>
</tr>
<tr>
<td>Shunt</td>
<td>30 (29%)</td>
<td>33 (31%)</td>
<td>0.881</td>
</tr>
<tr>
<td>Third ventricle</td>
<td>6.8 ± 3.8</td>
<td>7.6 ± 4.1</td>
<td>0.162</td>
</tr>
<tr>
<td>Cella media index</td>
<td>0.25 ± 0.05</td>
<td>0.25 ± 0.05</td>
<td>0.519</td>
</tr>
</tbody>
</table>

Fisher’s exact test. Student’s t-test.

5.4 Early rebleeding after coiling of ruptured intracranial aneurysms (IV)

The complete occlusion after coiling of ruptured IAs was achieved in 31% of cases. The neck remnant and residual aneurysm was found in 51% and 18%, respectively. The incidence of early rebleeding after coiling of ruptured IAs was 3.6% (7 out of 194). Since all the patients with rebleeding had an ICH at admission and all of them belonged to H&H grade 3–5 prior to coiling, these two factors were not suitable for a logistic regression model, but in the chi-square they were found to be predictors for early rehaemorrhage, p < 0.001, and p = 0.018 respectively. Univariate logistic regression analysis identified differences in residual aneurysm (OR 3.77; 95% CI 0.80–17.7), location of the MCA (OR 3.26; 95% CI 0.70–15.2) and large or giant aneurysm size (OR 1.80; 95% CI 0.39–8.31), but in these differences statistical significance was not achieved. The evaluated risk factors are shown in Table 16.

The characteristics of the 7 patients with early rebleeding after coiling are presented in Table 17. Rehaemorrhage appeared as an enlargement of the initial ICH and it was detected within 48 hours after coiling in every patient (in five of them within 24 hours). In one patient additional new blood in the ventricles was noted. Three patients underwent the evacuation of ICH after rebleeding (patients 4, 6 and 7). In only one patient (patient 2) angiography was performed soon after rebleeding. It revealed a residual irregular shaped aneurysm as was detected after the initial coiling with a new finding of partial thromboses of the aneurysm. Rehaemorrhage after complete aneurysm occlusion occurred in two patients, patient 4 and 7. In patient 4 the follow-up angiographies after 1 month and 3 years did not reveal any recanalization or new aneurysm formation. In patient 7 the angiography also showed a totally packed aneurysm two weeks after coiling.

57
Table 16. The calculated possible risk factors of early rebleeding after coiling of a ruptured intracranial aneurysm by univariate logistic regression analysis.

<table>
<thead>
<tr>
<th>Factors</th>
<th>Early rebleeding</th>
<th>OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Females</td>
<td>3</td>
<td>2.9</td>
</tr>
<tr>
<td>Males</td>
<td>4</td>
<td>4.4</td>
</tr>
<tr>
<td>Hypertension</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
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<td>3.3</td>
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<tr>
<td>Yes</td>
<td>2</td>
<td>4.9</td>
</tr>
<tr>
<td>Multiple aneurysms</td>
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<tr>
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</tr>
<tr>
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<td>3</td>
<td>5.0</td>
</tr>
<tr>
<td>H&amp;H*</td>
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<td></td>
</tr>
<tr>
<td>Good (1–2)</td>
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<td>0.0</td>
</tr>
<tr>
<td>Poor (3–5)</td>
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<td>6.5</td>
</tr>
<tr>
<td>ICH</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
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<td>0.0</td>
</tr>
<tr>
<td>Yes</td>
<td>7</td>
<td>13.0</td>
</tr>
<tr>
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<tr>
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<tr>
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</tr>
<tr>
<td>Location</td>
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<tr>
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<td>2.9</td>
</tr>
<tr>
<td>Large or giant</td>
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<td>5.2</td>
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<tr>
<td>Initial result</td>
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<td></td>
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<tr>
<td>Complete or Remn</td>
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<td>2.5</td>
</tr>
<tr>
<td>Resid</td>
<td>3</td>
<td>8.8</td>
</tr>
</tbody>
</table>

Remn = neck remnant, Resid = residual aneurysm, OR = odds ratio, 95% CI = 95% confidence interval for the odds ratio
*prior to coiling
EVD was assessed in three patients (all after coiling), in one patient before rehaemorrhage on the same day as coiling (patient 6) and in two patients after rehaemorrhage (patients 4 and 5). A good outcome (GOS 5) was achieved in 2/7, moderate disability (GOS 4) in 2/7 patients, and only one patient died.

**Table 17. Characteristics of the 7 patients with early rebleeding after coiling of ruptured intracranial aneurysms.**

<table>
<thead>
<tr>
<th>Patient Number</th>
<th>1</th>
<th>2</th>
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<th>4</th>
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<th>6</th>
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<td>48</td>
<td>60</td>
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<td>M</td>
<td>M</td>
<td>M</td>
<td>F</td>
<td>M</td>
</tr>
<tr>
<td>Hypertension</td>
<td>−</td>
<td>+</td>
<td>−</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>−</td>
</tr>
<tr>
<td>Multiple aneurysms</td>
<td>+</td>
<td>−</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>+</td>
</tr>
<tr>
<td>H&amp;H*</td>
<td>4</td>
<td>3</td>
<td>5</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>ICH (initial) ml</td>
<td>4</td>
<td>30</td>
<td>37</td>
<td>10</td>
<td>7</td>
<td>25</td>
<td>15</td>
</tr>
<tr>
<td>ICH (rebleeding) ml</td>
<td>21</td>
<td>35</td>
<td>70</td>
<td>51**</td>
<td>15</td>
<td>55**</td>
<td>28**</td>
</tr>
<tr>
<td>IVH</td>
<td>−</td>
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<td>+</td>
<td>−</td>
<td>+</td>
<td>−</td>
<td>−</td>
</tr>
<tr>
<td>EVD</td>
<td>−</td>
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<td>−</td>
<td>+***</td>
<td>+***</td>
<td>+***</td>
<td>−</td>
</tr>
<tr>
<td>Aneurysm location</td>
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<td>ACoA</td>
<td>MCA</td>
<td>PCoA</td>
<td>ACoA</td>
<td>MCA</td>
<td>MCA</td>
</tr>
<tr>
<td>Aneurysm size (mm)</td>
<td>10 mm</td>
<td>6 mm</td>
<td>9 mm</td>
<td>7 mm</td>
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<td>10 mm</td>
<td>7 mm</td>
</tr>
<tr>
<td>Coiling delay (day)</td>
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<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Occlusion grade</td>
<td>Remn</td>
<td>Resid</td>
<td>Remn</td>
<td>Comp</td>
<td>Resid</td>
<td>Resid</td>
<td>Comp</td>
</tr>
<tr>
<td>Rebleed delay (hour)</td>
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<td>&lt; 48 h</td>
<td>&lt; 6 h</td>
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<td>&lt; 24 h</td>
<td>&lt; 22 h</td>
<td>&lt; 24 h</td>
</tr>
<tr>
<td>Rebleed type</td>
<td>ICH</td>
<td>ICH</td>
<td>ICH</td>
<td>ICH</td>
<td>ICH, IVH</td>
<td>ICH</td>
<td>ICH</td>
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<tr>
<td>Thrombolysis</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>+</td>
</tr>
<tr>
<td>Follow-up (month)</td>
<td>94.0</td>
<td>99.0</td>
<td>0.1</td>
<td>37.5</td>
<td>0.5</td>
<td>33.9</td>
<td>8.2</td>
</tr>
<tr>
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<td>5</td>
<td>1</td>
<td>4</td>
<td>3</td>
<td>4</td>
<td>3</td>
</tr>
</tbody>
</table>

AChoA = anterior choroidal artery
Comp = complete occlusion, Remn = neck remnant, Resid = residual aneurysm
*prior to coiling, ** evacuation of rehaemorrhage, ***after coiling
6 Discussion

IAs are not congenital, but develop in the course of life (Rinkel et al. 1998). Most of them will never rupture. SAH from an IA occurs at a fairly young age and is often fatal (van Gijn et al. 2007). The hazardous complications are rebleeding, HC and DCI. SAH accounts over 25% of all stroke-related years of potential life lost before the age of 65, and the loss of productive life years is similar to that for ICH or cerebral infarction (Johnston et al. 1998).

This study was performed in a retrospective manner using information from our data-base. The current status of the non-contrast CT in the diagnosis of SAH and in the localization of ruptured IA was identified. The influence of the amount and distribution of blood on acute HC and the effects of early surgical and endovascular treatment on the development of chronic HC were evaluated. The risk factors of early rebleeding after coiling of ruptured IA were analysed. The fact that the incidence of aneurysmal SAH is almost three times more common in Finland than in other western countries emphasizes the importance of the research concerning this disease in our country.

6.1. Study populations and distribution of aneurysms (I–IV)

In the study population I and II males were more frequently exposed to SAH than females. This is not in concordance with most international studies, which have usually shown a female preponderance (Vermeij et al. 1994, Mehta et al. 1996, Vale et al. 1997, Sethi et al. 2000, Pedersen et al. 2001). In some previous Finnish studies the preponderance of males has been presented evoking a discussion if this male preponderance is a Finnish phenomenon (Sarti et al. 1991, Tapaninaho et al. 1993, Hernesniemi et al. 1993, Rinne et al. 1995). In accordance with most studies in other countries the populations of the study III and IV consisted mostly of female patients.

Half of the patients with acute SAH are younger than 55 years, although the incidence increases with age (Anderson et al. 2000, van Gijn et al. 2007). The mean age of endovascularly treated patients in study III was 53 years and in study IV 54 years. This is in accordance with a meta-analysis of 89 studies of coiled ruptured aneurysms by Brilstra et al. (1999) presenting a mean age ranging from 46–57 years. The mean age of the surgically treated patients in studies I and II was 47 years, and in study III in the surgical group 52 years. As in most other series it was not possible to find considerable differences between the surgical and endovascular series related to age.
The distribution of IAs varies in different reports. Most reports indicate the higher proportion of IAs on the anterior circulation, as we did in every study (Vassilolthis & Richardson 1979, Hasan & Tanghe 1992, Pietilä et al. 1995, Lin et al. 1999, Schmieder et al. 1999). In study III nearly all of the operated IAs (100 out of 102) were located on the anterior circulation, predominantly in the MCA. The same distribution of IAs located mostly in MCA was noted in the study population I and II with surgically treated patients. Also Tapaninaho et al. (1993) presented a higher frequency of MCA aneurysms in the series of surgically treated Finnish patients. This is based on the fact that MCA aneurysms are proposed to be neurosurgically clipped. In the studies III and IV the predilection site of the coiled IAs was the ACoA aneurysm. In the present series the ruptured IAs in the posterior circulation were treated more often endovascularly than surgically. On the whole, the patient and treatment selection criteria in this and other published studies have a great influence on gender, age and distribution of the aneurysms.

6.2 Hijdra grading scale (I–III)

In the present study the quantity and distribution of the haemorrhage was estimated using the method of Hijdra (Hijdra et al. 1990). This method reflects the distribution of cisternal and ventricular blood well (Hirashima et al. 2003, van Norden et al. 2006). We had reliable interobserver agreement in estimating the distribution of blood and in locating the site of aneurysm on non-contrast CT in the common locations.

An advantage of the Hijdra scale is that it takes account of IVH independently. If a researcher records the presence, location and ICHvol separately, the overall impression of blood distribution is good. With modern CT equipment the drawbacks described with the Hijdra scale, such as its dependency on technical and anatomical factors, are not so considerable.

6.3 Localization of ruptured aneurysms on non-contrast CT (I)

There has been a progressive advancement in imaging techniques during the past decades, but non-contrast CT is still a very sensitive method for detection of aneurysmal SAH and other acute haemorrhage. With modern CT scanners, the false negative results have diminished, and the acute SAH can be ruled out with a negative CT result together with negative lumbar puncture (Perry et al. 2008).
The present study demonstrates the value of the quantity and distribution of SAH on CT for the prediction of the site of rupture of an aneurysm in selected patients. The aim to localize the aneurysm, which has bled, is of great importance, especially in the presence of multiple IAs. Even after DSA and in an operation it is occasionally difficult to decide, where the ruptured aneurysm exists. The strength of this study was that the precise location of an IA was confirmed in the operation. Another important topic was the use of $\kappa$-values in assessing the reliability of the results between two observers, which was previously only infrequently used.

In a recent study by Tryfonidis et al. (2007) the location of the aneurysm on CT images was correctly identified in 90% of cases. In the present study, the initial CT proved to be a reliable method for locating ruptured MCA and ACoA aneurysms and in these aneurysms the $\kappa$-values between the two neuroradiologists were alike. So, when the aneurysm is in the most usual locations, the distribution of blood is accurate in predicting the correct location. We found substantial, or almost perfect, reliability which differs from the conclusion of Latchaw et al. (1997) presenting that CT helps to predict the site of rupture in only a minority of cases.

The present study showed that in patients with a MCA and an ACoA aneurysm rupture most haemorrhages were usually in the “central” part and, not unexpectedly with a MCA aneurysm more haemorrhage was presented on the side of the rupture than contralaterally. With an ACoA aneurysm the amount of lateralised haemorrhage was, on average, equivalent. When a MCA aneurysm bleeds towards the central part of the brain or an ACoA aneurysm bleeds towards the lateral parts, there is a possibility to misinterpret the location of the aneurysm. It appears also that the location of other aneurysms than MCA or ACoA around the Circle of Willis cannot be predicted well because of the close location of these aneurysms in the basal parts of the brain. Usually the number of these aneurysms is too low to warrant reliable conclusions in this respect. In this study, the existence of ICH at admission was a predictor for evaluating the site of the ruptured aneurysm, which is in concordance to other reports (Nehls et al. 1985, Hillman 1993, van der Jagt et al. 1999). The publication by van der Jagt et al. (1999) concluded that the site of the ruptured IA without an ICH can only be determined by CT, when the source of bleeding is an ACA or an ACoA aneurysm.

The prediction of non-contrast CT is only an indication of the site of the ruptured aneurysm, helping to direct attention to the region of interest. DSA or MDCTA remain the methods to depict the exact location of an aneurysm.
6.4 Hydrocephalus (II,III)

6.4.1 Risk factors of acute hydrocephalus (II)

The incidence of acute disturbance of CSF circulation presented in the literature is highly dependent on the variable diagnostic criteria used, on the timing of the investigations and on the patient selection. It explains the large range of incidence of acute HC on admission. The fact that a longer time from the onset increases the existence of HC on admission is not usually taken account when estimating acute HC. In this data the incidence was high, but is conveniently in accordance with other reports (Vassilouthis & Richardson 1979, Spallone et al. 1983, Black 1986, Hasan et al. 1992, Vermeij et al. 1994, Vale et al. 1997, Lin et al. 1999, Schmieder et al. 1999). We noticed all the minor marks of HC with visual examination on CT, which increased the number of patients suffering HC.

In the present study the patients with IVH had a notable predisposition to develop acute HC. This is in concordance with many other reports and current pathophysiologic concepts that acute HC following SAH is an obstructive form of HC (van Gijn et al. 1985, Graff-Radford et al. 1989, Tapaninaho et al. 1993, Vermeij et al. 1994, Mehta et al. 1996, Vale et al. 1997, Lin et al. 1999). A large ICH was another predictive finding for HC, which is obviously caused by the mass effect.

In addition to IVH, haemorrhage in the basal cisterns and regions is prone to cause HC (Hasal & Tanghe 1992, Vermeij et al. 1994, Pietilä et al. 1995, Lin et al. 1999). Hasan & Tanghe (1992) reported that, in the absence of ventricular blood, blood in the ambient cisterns significantly increases the risk of acute HC. Schmieder et al. (1999) showed that shunt dependency is more likely after bleeding into the basal cisterns. Also in this report blood in the basal area was a determinant of ventricular enlargement. It was noticed that in cases with IVH there is often a considerable quantity of blood in other regions, and this total blood amount has an effect on the development of HC.

In the present study, the anatomical location of the aneurysm had no correlation with the development of acute HC. This is in concordance with the report by Lin et al. (1999). The carotid and posterior circulation artery aneurysms have, although, been reported to be more often associated with acute HC (Graff-Radford et al. 1989, Hasan & Tanghe 1992, Mehta et al. 1996). As in many other publications, the number of posterior fossa aneurysms in this data was too small to yield an accurate relationship between the site of aneurysm in this region and the development of HC. The frequencies of HC with both sides of MCA aneurysms and AcomA
aneurysms were almost equal in our series. It could not be confirmed that the rate of HC is lower in patients with MCA aneurysms compared to aneurysms close to the mid-line, in contrast to earlier reports (Kibler et al. 1961, Graff-Radford et al. 1989, Tapaninaho et al. 1993, Pietilä et al. 1995, Schmieder et al. 1999). A study by Lin et al. (1999) showed the predominance of acute HC in patients over 50 years, but we observed it most frequently among patients aged 40–49 years. Patients with acute HC tend to have high mortality rate and should be diagnosed early and treated aggressively, especially patients with high risk factors.

**6.4.2 Chronic hydrocephalus after treatment (III)**

The present report compared two widely accepted treatment methods for ruptured IAs, surgical clipping and endovascular coiling, with reference to the development of chronic HC. This type of HC is caused by impaired CSF absorption because of fibrosis and arachnoidal adhesions (Foltz & Ward 1956, Galera & Greitz 1970, Yasargil et al. 1973, Sethi et al. 2000).

In this study, the incidence of chronic HC and the number of patients needing a shunt for the management of HC is within the wide range reported in the literature (Pietilä et al. 1995, Vale et al. 1997, Schmieder et al. 1999, Sheehan et al. 1999, Widenka et al. 2000, Yoshioka et al. 2000, Dorai et al. 2003, Ohwaki et al. 2004). Older people were more likely to develop chronic HC supported by the findings of some other researches (Tapaninaho et al. 1993, Vermeij et al. 1994, Lin et al. 1999, Yoshioka et al. 2000). On the contrary, Hirashima et al. (2003) presented that age was unrelated to the incidence of chronic HC after SAH.

The results of the influence of treatment modality on the development of chronic HC are conflicting. Some publications have presented that chronic HC develops more likely after coiling (Ukkola et al. 2001, Dorai et al. 2003, Varelas et al. 2006). It has been suggested that the evacuation of cisternal and subarachnoidal clots reduce the formation of leptomeningeal fibrosis and blocks of the minor pathways and the risk of HC (Auer & Mokry 1990). Although the material of the endovascular group of this study was, in many respects, more prone to HC, no statistical differences between the two treatment modalities and the development of chronic HC were seen, and we could not confirm the benefit of early clot removal. This is accordance with the results by Dehdashti et al. (2004), who did not find significant difference in shunt-dependent HC between the treatment groups after controlling for covariates. Vanninen et al. (1999) had comparable surgical and endovascular groups with regards to the severity of bleeding, preoperative HC and
clinical grade. They found more patients with HC needed shunt operation in the surgical treatment group and assumed that the inflammation caused by surgery may lead to aseptic arachnoiditis and disturbance of CSF flow.

There is a difficulty to distinguish between brain atrophy and chronic HC. The resolution of brain oedema after the acute phase of SAH increases the ventricle size on the follow-up CT. To diminish false positive diagnoses of HC, consensus concerning ambiguous images is necessary with at least two experienced neuroradiologists. It is of great importance to find even mild forms of chronic HC, above all in elderly patients. The mild increase in the wIII in the surgical group, as was seen in this study, may represent mild hydrocephalus or, more probably, brain atrophy. Surgery as a treatment method causes damage to the brain parenchyma and may predispose to the progression of atrophy. The areas of reduced gray matter and parental artery territory lesions are more pronounced after surgical than endovascular treatment. It is recommended to follow-up these patients carefully and for more than six months based on their symptoms, and treated with a shunt operation if necessary (Bendel et al. 2008, Bendel et al. 2009b).

The drawback of this retrospective study was that volumetric MR imaging techniques were not used. This modern 3D MR image analysis may help to determine the cause of ventricular and sulcal enlargement. Bendel et al. (2009a) quantified CSF spaces after one year of patients recovered from SAH with volumetric MR imaging. They concluded that the diffuse enlargement of ventriculi and sulci indicate general atrophy rather than HC.

Comparisons between surgical and endovascular groups should be made with caution because of the wide heterogeneity in IAs, patients and study design. In many researches the imbalance in the material has caused difficulties in analysing the results. In the present study the treatment groups compared were not matched for aneurysm location. The majority of posterior circulation and ACoA aneurysms fell into the endovascular treatment group. Similarly, chronic HC may be more frequent following the rupture of posterior circulation and midline aneurysms (Galera & Greitz 1970, Yasargil et al. 1973, Sethi et al. 2000). In the series of Pietilä et al. (1995) none of the patients with MCA aneurysms needed a shunt. At the same time, the percentage of chronic HC was high in patients with ACoA aneurysms and highest with an aneurysm in the vertebrobasilar region. It is due to the fact that ACoA and posterior circulation aneurysms are often associated with IVH.
6.5  Risk factors of early rebleeding after coiling (IV)

Rebleeding after SAH occurs soon after the initial haemorrhage, and up to 14 percent of the patients may suffer rebleeding before admission (Ohkuma et al. 2001). Even with treatment in the acute stage a complete elimination of rehaemorrhage is not possible.

In the present report the incidence rate of early rehaemorrhage after coiling was 3.6%. It is approximately two times the rates found in other studies with the incidence of 1.4–1.9% (Vanninen et al. 1999, Molyneux et al. 2005, Sluzewski et al. 2005a, Willinsky et al. 2009). The differences may be caused by a different patient population and clinical condition at the time of coiling. In the current study the patients were treated at the acute stage within 3 days (mean delay 1.2 days), the percentage of the patients with poorer clinical condition, H&H grade 3–5 patients, was 57%. On the contrary, in the series of Sluzewski et al. (2005a) the mean delay of treatment was 9.1 days, and the percentage of H&H grade 3–5 patients was only 37%. In a report of Willinsky et al. (2009) and in ISAT study the majority of patients were also in a good condition.

ICH at admission was found in all seven coiled patients with rehaemorrhage, and it appeared to be a risk factor for early rebleeding. This is in accordance with the results of Sluzewski et al. (2005a), who documented adjacent ICH as an independent risk factor. They concluded that the presence of a thrombosed pseudoaneurysm in the ICH may cause early reopening and rehaemorrhage. Similar postulations have been published by other researchers (Nomura et al. 2000, Mori et al. 2004, Tanoue et al. 2004). The current study supports the results of Sluzewski et al. (2005a) who documented H&H 3–5 as a possible predictor for early rebleeding.

In the current study a statistically significant relationship between the occlusion grade of the ruptured IAs and early rebleeding was not found. However, the results suggest that the frequency of rebleeding among patients with residual aneurysm is larger. In concordance with this, some reports have shown that a sub-totally packed aneurysm increases the risk of rebleeding (Vinuela et al. 1997, Molyneux et al. 2005, Johnston et al. 2008). The presence of a residual aneurysm in three of our patients may be the cause of rebleeding. In the present study, two rehaemorrhages occurred after complete occlusion. The explanation of this may be recurrent aneurysm formation, coil compaction and recanalization, which are possible events in the early phase (Bavinzski et al. 1999, Nguyen et al. 2007). The environment of a ruptured aneurysm, like intracisternal arachnoid membranes, may also change the shape of aneurysms and increase the risk of rerupture (Valavanis 2008).
Sluzewski et al. (2005a) observed that small aneurysms less than 6 mm are an independent risk factor of rebleeding. Controversially, in this report the size of the aneurysms with rehaemorrhage was between 6 to 10 mm. The increasing effect of large aneurysms size to rebleeding has also been reported (Naidech et al. 2005, Pleizier et al. 2006). The location on ACoA has been a predilection site in two recent reports (Sluzewski et al. 2005, Willinsky et al. 2009). In the present study more rehaemorrhages with MCA aneurysms compared with other locations were found, but the difference was not statistically significant.

In this series the insertion of EVD was used more often than for every third patient to treat acute HC. The increased risk after EVD of early rehaemorrhage was not confirmed. None of the unprotected aneurysms rebled after the assessment of EVD, and the overall rebleeding rate in patients with EVD was not found to be increased. The same result is published by McIver et al. (2002). There is no ground for avoiding early aggressive treatment of HC, because acute HC tend to have high mortality rate. According to the present study, the practice of our institute includes the aggressive and successful strategy of the assessment of EVD.

Additional thrombolytic therapy during the coiling procedure or prolonged heparinization increases the risk of rebleeding (Molyneux et al. 2002). In the present series one patient suffered early rebleeding after receiving intravenous thrombolysis. We could not analyse the statistical significance of additional thrombolysis due to the lack of information on all the other drugs the patients received.

In this series the outcome of the patients after early rebleeding was usually good, despite the fact that all the patients belonged to the group of H&H 3–5. Only one patient died, and two patients had outcome GOS 5. The outcome after rebleeding presented in two other recent studies is much worse (Sluzewski et al. 2005a, Willinsky et al. 2009), in which nearly all of the patients died. The difference may be explained by careful patient selection and treatment strategy. In our institute, many patients who are not chosen for surgical treatment undergo coiling with subsequent evacuation of ICH. This strategy may lead to a good outcome, as Chung et al. (2009) recently reported. Another aspect to be considered is our aggressive treatment of HC with EVD (almost 36% of the patients), which may have an influence on better outcome.

The study IV contains a rather large material based on consecutive acutely coiled patients. A limitation is that this is a retrospective, selected, non-randomized series. Another main limitation is the difficulty to perform statistically relevant analysis with a small number of positive cases. Thirdly, it is not possible to determine the timing of a rehaemorrhage accurately, and it may occur prior to the coiling or per-
procedurally without observing it. Fourthly, the real incidence of rehaemorrhage is supposed to be larger because small amounts of new blood may remain clinically and radiographically undetected.

Should the practice be changed to prevent early rebleeding after coiling? The complete occlusion of an aneurysm in the early first session is the best protection against rebleeding. Early follow-up angiograms may show the new lumen of an aneurysm invisible on the initial images, especially in cases with adjacent ICH. However, daily angiograms are usually not possible due to the lack of resources. In some special cases the diagnostic use of MRA or CTA alternative, or together with DSA, may help to depict the morphology of an aneurysm. The reduction of routine anticoagulation may be possible in some cases with ICH. The patients with ICH and poor clinical grade should undergo close examination of clinical and radiological facts and the possibility of operation should be kept in mind.

6.6 Future prospects

Despite the progressive knowledge of medicine and the development of treatment modalities, SAH is still a life-threatened disease with a poor outcome. It depends mostly on the nature of the disease. The rapid diagnosis and treatment is of great importance. A modern CT is reliable enough for the diagnosis of aneurysmal SAH combined with a lumbar puncture, if necessary.

MDCTA in the detection of ruptured IA is increasingly used. CTA is a relatively safe, fast, accessible and reliable technique. It replaces DSA as a first-choice examination in many centres. The information of MDCTA in the acute phase depicting the ruptured aneurysm is often enough for the neurosurgeon, and sometimes there is no time to undergo DSA. If necessary, the indistinct areas can be examined more closely later. DSA is still considered as the gold standard for detecting the exact location, shape and vascular environment of IAs. The potential advantage of DSA is the possibility to embolize the ruptured IA during the same procedure. 3DRA improves diagnostic accuracy and offers detailed information on an aneurysm and surrounding vessels. MRA is a very important technique, above all in the follow-up of embolised aneurysms. In some cases the aneurysm wall, the accurate shape, pseudolumen or true lumen of a ruptured aneurysm may be better visible on MRA or MDCTA than on a conventional angiogram.

It is probable that the diagnostic use of MDCTA alternative to diagnostic DSA becomes more common in the future. The rapid technical evolution of MDCT scanners in vascular imaging has evolved from 4-detector row systems to 256-slice
and 320-detector row CT systems. The latest MDCTA scanner is faster, enables volumetric imaging and is more accurate even with a lowered contrast load and reduction of CT radiation dose. With a new advantage, dual-energy CT, the various tissue types can be automatically differentiated in one scan. It may be possible to detect calcifications better from contrast media in IAs. In the detection of IAs using dual-energy CTA high diagnostic accuracy compared with 3DRA has been seen (Zhang et al. 2010). These emerging technical advances and novel applications of CTA will continue to change the way we study intracranial vessels and IAs.

The use of CT perfusion after SAH in detection of vasospasms and in prediction of infarcts will increase. In acute HC after SAH perfusion CT has shown the reduction of cerebral blood flow (van Asch et al. 2010). A new approach is flat detector CT coupled to an angiography device providing an imaging technique for interventions to evaluate brain parenchyma, vasculature, cerebral blood volume and haemorrhages (Struffert et al. 2009, Kamran et al. 2010, Struffert et al. 2010). A number of portable CT scanners for clinical imaging have developed, some of them especially for neuroradiological head imaging. Rumboldt et al. (2009) concluded that the portable CT generates satisfactory clinical images at acceptable patient doses.

The use of endovascular technologies in the treatment of IAs continues to expand. Presumably GDC and other uncovered platinum coils will maintain their popularity as embolized material far into the future. The introduction of the balloon and stent remodelling techniques has enabled coiling of IAs that are difficult to treat surgically. Tähtinen et al. (2009) proved that the stent-assisted coil embolization was a feasible method for treatment of wide-necked IAs during acute SAH. The new covered coils have been examined to clarify if recanalisation of an aneurysm diminishes with them (Kallmes & Cloft 2007). The flow conditions within the parent artery and the aneurysm itself have long been considered as playing an important role in aneurysm growth and rupture (Jou et al. 2003, Cebral et al. 2005, Sforza et al. 2009). The studies have led to the development of stent-like flow modifying devices in aneurysm treatment. First results using these braided stents (like silk stent) of the newest generation are promising in preventing recanalisation. Since the mesh of these stents is very dense covering of side branches and perforators it has still proved to be safe (Wanke 2008). These flexible stents are useful even in large fusiform aneurysms.

While endovascular strategies to treat IAs may hold the key to future success, there will be always patients who need surgical treatment. Technical advances in the surgical management of aneurysms are limited and they have changed little over the
last decade. Neurosurgeons have referred aneurysms of the vertebrobasilar region, and partly of other regions, to radiologists, and the proportion of endovascularly treated patients continues to increase. This change has increased after the ISAT study, creating problems and leading to difficulties for neurosurgical trainees to achieve competence in clipping aneurysms in many centres. Some neurosurgeons have expressed criticism of the ISAT study. They remind that the size of samples of the ISAT trial does not exclude false conclusions. The series of the ISAT study are composed of a highly selected group of patients with mostly small anterior circulation aneurysm, representing less than 22% of the SAH population admitting to the recruiting centres (Lindsay 2003).

Although most aneurysms can be clipped or coiled, a subset of patients may require a combined approach. The use of cerebral endovascular neurosurgery has evolved rapidly in the past decade through advances in tools and techniques. Multimodality approaches are best used with complex aneurysms in which the conventional techniques with a single modality have failed. This combined therapy enables the endoluminal reconstruction of an aneurysm, and it will become more common in the future (Lawton et al. 2008). The application of endovascular techniques combined with the use of bioactive implants and gene or drug therapy in the treatment of aneurysms is still on the theoretical level (Ribourtout et al. 2004, Frösen et al. 2006).
7 Summary and conclusions

1. Analyses of blood amount and distribution on initial non-contrast CT after SAH appears to be a reliable method for estimating the location of ruptured MCA and ACoA aneurysms. Adjacent ICH is an excellent predictor for detecting the precise site of a ruptured aneurysm.

2. IVH is the most consistent single risk factor for the development of acute HC. Haemorrhage in the basal region and cisterns, and the total blood amount in SA spaces are other strong predictors. The results confirm that acute HC following aneurysmal SAH is an obstructive form of HC. Patients with large ICH are proposed to have an acute HC partly due to the mass effect of ICH.

3. The early treatment method, whether neurosurgical clipping or endovascular coiling, used for ruptured IAs is not significantly associated with the occurrence of chronic HC or the need for a shunt operation.

4. The incidence rate of early rebleeding after coiling of a ruptured saccular IA is low. Risk factors for post-procedural rehaemorrhage are the presence of ICH on the initial CT and H&H grade 3–5 prior to coiling. Rehaemorrhage appears on CT as enlargement of the initial ICH and not an increased amount of blood in the SA space.
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Original publications


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