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Giant Intracranial Aneurysms – Surgical Treatment, Accessory Techniques and Outcome

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1. Introduction

There are various intracranial aneurysms: saccular, fusiform, dissecting or mycotic. Saccular aneurysms are the most common type and account for up to 98% of all intracranial aneurysms (Yasargil, 1984). If the widest diameter of the aneurysm is equal to or exceeding 25 millimetres (mm), the aneurysm is defined by convention as giant (GIA). The etiology of GIAs is similar to smaller ones (Lemole, 2000), theories about the development of all saccular aneurysms include congenital and acquired artery defects. GIA's and other aneurysms are etiologically divided into "sidewall" and "bifurcation" aneurysms (LeRoux, 2003). In flow-related phenomena, constant enlargement of a small aneurysm in the distal part of the neck results in GIA formation. However, de novo development of GIA has also been described (Barth, 1994). The histology of GIA wall is different from smaller aneurysms: GIAs often lack a muscular layer as well as elastic laminae layers show degeneration. The incidence of intraluminal thrombosis significantly increases with the lumen size of aneurysms; in GIAs this phenomena may occur in approximately 60% of cases (LeRoux, 2003). Krings publication (Krings, 2005) was a breakthrough in large aneurysms formation knowledge; he proved that the GIA development in the internal carotid artery (ICA) and vertebral artery (VA) differ from those in other locations. Repeated subadventitial haemorrhages from vasa vasorum are a predominant factor in GIA aneurysm pathogenesis. Therefore, GIA formation can be considered as a "proliferative disease of the vessel wall induced by extravascular activity". Historically GIA rupture is known as devastating due to higher amount of extravasated blood. In contrast, recent papers indicate that rupture of some smaller aneurysms leads to more extensive SAH. The study ISUIA (Kassell, 1990) proved that the risk of rupture of GIA can reach 40% in five-year follow-up, while treatment of unruptured intracranial aneurysm carries relatively low mortality that does not exceed 2% (Molyneux, 2005). Therefore, treatment is warranted for most patients suffering from GIAs. There are two treatment modalities that can be offered to patients afflicted with GIA

pathology: endovascular or surgical. In general endovascular treatment is less invasive and has fewer complications than surgery, and therefore is preferable. Surprisingly, no randomized comparison study of these two methods in GIA treatment have been published. However, the outcome measurement and analysis may be difficult to conduct a trial in GIAs; these aneurysms constitute a heterogeneous group and they are treated using different methods in different institutions. Furthermore, there is not enough observational data in the literature discussing results of treatment and their pertinence to quality of life in patients with GIAs in comparison to smaller ones. Additionally, radiographic results assessed several years post-operatively have not been reported sufficiently. Probably it is due to the unique peculiarity of GIAs as these require extensive comprehension of the treatment strategies to achieve better results. The current study is not only aimed at describing available methods, but to compare the prognosis after treatment of GIAs versus smaller aneurysms. A new neurovascular surgeon should be accustomed to all surgical techniques for GIAs. All of the treatment possibilities, technical issues and their clinical implications are to be learned meticulously and considered preoperatively.

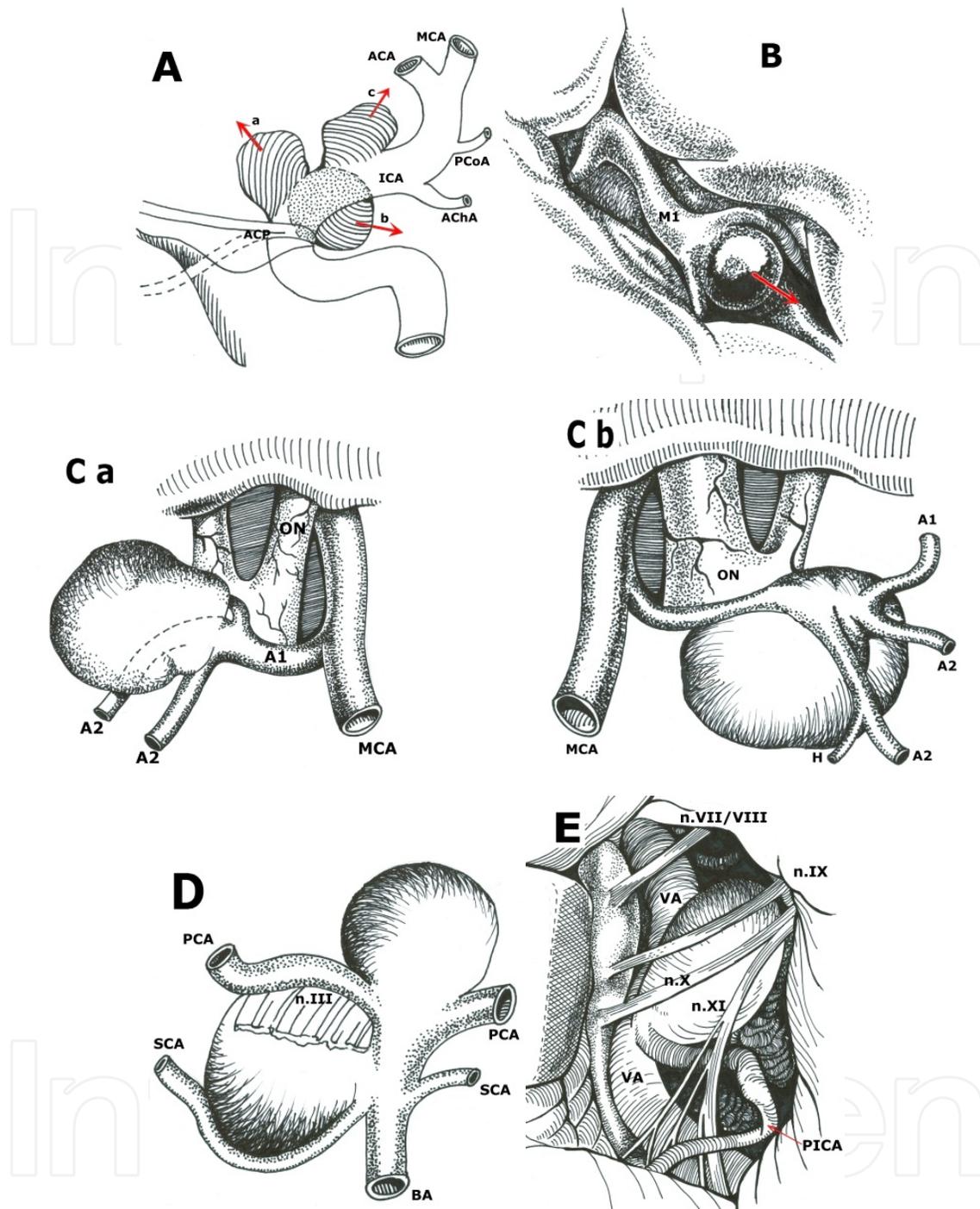
2. Epidemiology and clinical presentation

Approximately 2% to 5% of all intracranial aneurysms are classified as giant. Epidemiological studies have demonstrated increased incidence of GIAs in elderly, most cases present in the fifth to seventh decades of life (Anson, 1995). These lesions are slightly more common in females. In the paediatric population approximately 5 to 10% of all aneurysms exceed 25mm. Up to 40% of GIAs are found in posterior cerebral circulation while 80% to 90% of smaller aneurysms are located in anterior cerebral vasculature. ICA is the predominant localization. In general 40% of GIAs are seen in the carotid artery, 25% in the anterior (ACA) and middle (MCA) cerebral arteries, and 30% per cent in the vertebrobasilar (VB) arteries (Fig. 1).

The ratio of giant aneurysms to all other intracranial aneurysms is six to one in the posterior circulation, which is statistically higher than in anterior circulation. The cause of somewhat different distribution of GIAs from that of smaller aneurysms is unknown. Krings theory about the role of repeated subadventitial haemorrhages in giant VA and ICA aneurysm formation partially explains referral patterns. Above all, adduced aneurysm distribution is only based on clinical publications referring to hospitalized patients, although population based studies have not been performed (Table 1).

	Peerless n=635	Kodama n=1023	Weir n=573	Karavel n=309	Sharma n=181	own series n=128
ICA	34%	51%	39%	42%	84%	61%
V-B	56%	27%	25%	33%	7%	9%
ACA	3%	8%	16%	10%	2%	12%
MCA	16%	13%	12%	15%	7%	18%

Table 1. The distribution of GIAs based on large series studies (Peerless, 1990, as cited in Youmans, 1990; Kodama, 1982; Weir, 1987; Karavel, 1988; Sharma, 2008) and own material.



Abbreviations: ACA - anterior cerebral artery; A1-first segment of ACA; A2 - second segment of ACA; ACP - anterior clinoid process; AChA - anterior choroidal artery; H - artery of Heubner; BA - basilar artery; MCA - middle cerebral artery; M1 - first segment of MCA; n.VII/VIII - complex of facial and vestibulocochlear cranial nerves; n.IX - glossopharyngeal cranial nerve; n.X – vagus cranial nerve; n.XI - accessory cranial nerve; ON – optic nerve; PCA - posterior cerebral artery; PCoA - posterior communicating artery; PICA - posterior inferior cerebellar artery; SCA - superior cerebellar artery; VA - vertebral artery;

Figure 1. The location of selected GIAs and their projection. (A) Three variations of ophthalmic segment of ICA aneurysms and their growth projection: a - supraclinoid, b - carotid cave, c - dorsal wall blood blister-like). (B) MCA bifurcation growth direction. (C) ACoA (two variations: a - anterior, b - posterior) (D) BA bifurcation and SCA. (E) PICA.

Both smaller aneurysms and GIAs can present as either mass effect or subarachnoid haemorrhage (SAH). Unruptured smaller aneurysms are rarely symptomatic and therefore are found accidentally in computed tomography (CT) or magnetic resonance imaging (MRI) due to unspecific symptoms or after head trauma. On the contrary, almost two-thirds of GIAs are diagnosed before rupture. The location of the GIA determines its symptoms due to the size and direct contact with neural structures. Coexisting retro-orbital pain, diplopia, ptosis, trigeminal pain and mild headache are characteristic manifestation of GIAs from cavernous portion of the ICA. Visual loss and Horner's syndrome are rare findings even in large or giant aneurysms. The observational study of 20 non-treated cases demonstrated that cranial neuropathies may improve due to compression-induced cranial nerve ischemia resolution (Linskey, 1990).

If the aneurysm erodes surrounding bones massive epistaxis can lead to sudden death before admission. The history of patients with ruptured cavernous GIAs, derived from our own records, confirm that cavernous aneurysms are the last resort diagnosis for chronic haemorrhage from the nose.

Medially directed GIAs of paraclinoid segment and may present visual loss or hypopituitarism (Cawley, 1998). Retro-orbital pain, ophthalmic nerve paresis and headaches are sporadic however clinically relevant. Smaller aneurysms of the paraclinoid segment tend to remain asymptomatic for years. Asymmetrical visual field defects and visual loss are pathognomonic signs of GIAs in the ophthalmic segment of ICA, but are rarely observed in smaller aneurysms. Superior hypophyseal artery aneurysms may expand superomedially remaining ophthalmic aneurysms. Inferomedial aneurysms are called 'carotid cave aneurysms' and if they meet giant classification, produce superior bitemporal hemianopsia or hypopituitarism, generally indicative of pituitary tumours. Supraclinoid segment of ICA include aneurysms at posterior communicating artery (PCoA), anterior choroidal artery (AChA) and ICA bifurcation. Approximately one fourth of all aneurysms arise from ICA at the PCoA origin. Third-nerve palsy is usually complete and is observed in both GIAs and smaller PCoA aneurysms. However, expanding giant PCoA aneurysm may also produce 'thunderclap headaches'. No specific presentation is associated with smaller AChA and ICA bifurcation aneurysms. Nevertheless, GIAs arising from AChA origin may occasionally present third-nerve palsy and GIA's in the ICA bifurcation may cause visual deficits, epilepsy, and dementia, hemiparesis or aphasia. Posterior and anterior ICA wall GIAs are rarely symptomatic before rupture as those lesions usually have friable neck and thin wall. ACoA aneurysms may have different dome projection (Out of place). Mental disturbances, visual deficit, monocular blindness are common in GIAs, however, they are very sporadic symptoms in patients with smaller aneurysm of ACoA origin. Giant basilar artery (BA) bifurcation aneurysms and superior cerebellar artery (SCA) typically cause oculomotor palsy, Weber's syndrome, ataxia, hydrocephalus, gait disturbances or dementia. Other ocular findings observed, including Parinaud's syndrome supranuclear gaze palsy and internuclear ophthalmoplegia. If oriented anteriorly, GIAs in BA bifurcation may mimic sellar lesions by compressing the optic apparatus and causing visual disturbances. Anterior inferior cerebellar artery (AICA), BA trunk, inferior junction of vertebral artery (VA) and

posterior inferior cerebellar artery (PICA) GIAs may manifest themselves by hydrocephalus or symptoms referable to brain stem or cranial nerve compression (mimicking cerebellopontine angle tumours). In contrast, smaller aneurysms of the earlier mentioned locations often remain asymptomatic prior to rupture. Unlike ACoA, ICA and posterior circulation aneurysms, GIAs in MCA often reach large sizes before producing pressure-related symptoms. Moreover, focal neurologic deficits before rupture are indicative but not diagnostic in GIAs of MCA; in which, transient ischemic attacks (TIA), epilepsy, dysphasia, hemiparesis and occasionally bruits can be observed. Distally located GIAs, including posterior cerebral artery (PCA), anterior cerebral artery (ACA) and MCA, are diagnosed before rupture by epilepsy or brain stem compression symptoms.

The symptomatology of GIAs in all locations is typically characteristic. On the contrary, most of patients with smaller aneurysms do not present with indicative symptoms, but tend to be diagnosed after experiencing SAH. Based on Laplace's law, tension on the wall is higher in large or giant aneurysms than smaller ones. Drawing from the conclusion of the above law: the rupture of GIA causes a more severe haemorrhage than in smaller aneurysms. That is convincing theory; however, it was contradicted by other observational studies. Some authors (Roos, 2000; Russell, 2003; Taylor, 2004) find in contrast to Laplace's law that rupture of some smaller aneurysms (ACoA and ICA at the PCoA origin) lead to more extensive SAH. Therefore, the size of an aneurysm may not be regarded as a single prognostic factor in patients with SAH due to ruptured GIA. Based on ISUIA study (Kassell, 1990), unruptured GIAs' are related to high annual risk of rupture. Five-year cumulative rupture rates for patients who did not have a history of SAH for aneurysms less than 7 mm, 7-12 mm, 13-24 mm and 25 mm or greater reached 2.5%, 14.5%, 18.4% and 50% respectively. Moreover, the probability of SAH was also higher for BA bifurcation and ICA at the PCoA origin than for other locations. Others proved that the shape of GIA, neck to dome ratio and other factors should be considered in risk of rupture estimation (Patel, 2010). Untreated GIAs have a poor natural history, as the mortality rate can attain 100% within 2 years (Peerless, 1990, as cited in Youmans, 1990). High SAH rate in patients with unruptured GIAs justifies treatment in most cases. The potential consequences of the aneurysm rupture are devastating and usually have a sudden onset. Two-third of all patients after rupture will die or suffer from mental or physical deficits in the near future. Even when treated, unfavourable results usually exceed 30%. The incidence of SAH increases with age as well as related to some genetic diseases.

3. Treatment

Endovascular and surgical techniques are the options considered in treatment of GIAs. Although endovascular treatment has a short history of 50 years, the beginning of surgery for GIAs is dated at 19th century and. Extracranial internal carotid artery (ICA) ligation was the first attempt of excluding GIA from circulatory system in 1885 (Youmans, 1990). In 1911 Harvey Cushing, despite scepticism to abovementioned method, used alloy clip for extracranial ICA ligation. Dandy was a pioneer in aneurysm treatment. In 1936, he ligated ICA proximally and distally to the aneurysm (nowadays called trapping method) and one

year later he used Cushing's clip to obliterate the aneurysm neck. Dandy's work diffused a new era of clipping the aneurysms neck. Herbert Olivecrona modified the silver clip by adding winged blades, than Schwartz introduced miniaturized spring forceps as clips. However, Mayfield brought significant innovation by the use of an applicator and various types of detachable stainless steel clips. Followed by Mayfield, whose further development was merely a minor modification on Mayfield's work. Sundt developed Teflon-lined, encircling clip-graft, used for emergent reconstruction of a torn GIA. Sugita created very long clips, which are used for securing GIAs, and developed bayonet applicators and clips. Nowadays the optimized preoperative and intraoperative clip selection seems to play the most important role in correct GIA clipping approaches, as clipping still remains the "gold standard" among microsurgical methods. The features of clips intended for GIAs differ from those used for smaller aneurysms. The blades of standard aneurysm clips are usually longer and some clips (T-bar or J-shaped) are produced specifically for large or giant aneurysms (Fig. 2).



Figure 2. Left combined photo: contemporary permanent clips used for a GIA securing. Clips come in a variety of sizes and curve (straight, fenestrated, curved, bent, angled, bayonet, deflected, J-shaped, T-bar, et cetera). Right graphics: historical clips used in both smaller aneurysms and GIAs.

In the sixties and seventies, an enormous number of surgical approach improvements were observed. Yasargil described pterional craniotomy, which is still a routine approach to most of supratentorial GIAs. Temporary by-pass or hypothermia, bipolar coagulation, floating operating microscope and pharmacological neuroprotection were supportive methods, which led to significant GIA mortality reduction. At the end of 20th century, sweeping improvements of microsurgical techniques, stemming from technological development and microsurgical training, were ceased by the novel endovascular approach. The introduction of Guglielmi Detachable Coils (GDC) in 1991 began the modern era of neurovascular

surgery and soon revolutionized aneurysm treatment. Initially GBC devices were approved for aneurysms and patients not amenable for surgery, although they have been used successfully to occlude all types of GIAs in patients with any neurovascular aneurysm condition. However, complete occlusion with only GDC remains insufficient in treatment of GIAs with particularly broad necks, thus balloon-assisted GDC placement or stent intervention was introduced to remedy this problem. For the last decade microsurgical occlusion of GIAs is constantly being displaced, as current endovascular techniques are regarded as having a lower risk for the patient. It is quite unusual in Evidence Based Medicine that rapidly developing endovascular techniques quickly (smoothly doesn't work here) became the standard for securing GIAs, when the neurosurgical discipline has developed a multitude of different approaches and advancements in the treatment of aneurysms. Despite the above dispute, GIAs are the most challenging lesions for both experienced neurosurgeons and experienced neuroradiologists.

Since the first attempt at excluding giant ICA aneurysms from circulatory system in 1885, many surgical occlusion and accessory techniques have been developed and elaborated. Although the introduction of Guglielmi Detachable Coils changed aneurysm treatment, soon limitations of its inability to occlude giant and widenecked aneurysms required further exploration. Constantly developing endovascular techniques are regarded as having lower risks for patients than open surgery and still seem to be unsatisfactory in terms of durability in aneurysm occlusion. The balloon-assisted remodelling and stent-assisted techniques partially solved the problem of neck remnants. Therefore microsurgery and the combination of microsurgical and endovascular method will still be up-to-date for years.

3.1. Treatment considerations

There are two main considerations regarding GIAs:

- Should we treat or observe unruptured GIAs ?
- Should we clip or insert endovascular coils to treat GIAs ?

Since 2003, when the ISUIA study was published, a justification for any treatment for GIA unanimously has been found. Up to 40% risk of rupture was observed in five-year observation. However, Wermer's publication (Wermer, 2007) complicated treatment decision making. As a result of the previously mentioned meta-analysis, the size, site and type of aneurysm should be considered when deciding whether to treat an unruptured aneurysm. Other factors like age, gender and population are also important risk factors of rupture estimation. Those multifactorial results are convincing as the material comprised sixty SAH cases among observed untreated GIAs. Before patient treatment, a pooled analysis of individual data is needed to identify the independent risk of rupture and possible risk of therapy complications. The presence of some factors significantly affects GIAs' treatment and the outcome. The number of people older than 65 years is still growing. Comorbid diseases that are often related to the elderly population include: cardiac disease, hypertension, atherosclerosis, carotid disease and multiple aneurysms. Conservative approaches to GIAs can be applied to older patients with comorbidities, although it is

controversial. The risk of surgery in older patients is greater than in younger patients in part because of comorbid disease. Some studies (LeRoux, 2003) suggest that not only old age, but the patient's clinical condition should determine treatment decision. However, no randomized trial has compared treatment with conservative management in elderly patients.

To date, there is no consensus in treatment modality for ruptured, unruptured, and furthermore giant aneurysms. Probably the unique peculiarity of GIAs requires extensive comprehension of the treatment strategies, suggesting that individual approach is preferred. In year 2005, ISAT study (Molyneux, 2005) was a landmark in choosing treatment modality. 2143 patients with ruptured aneurysms took part in the multicentre trial and were randomly assigned to neurosurgical clipping or endovascular coiling. Despite of the fact that rebleeding was lower, one-year survival rate and epilepsy rate was higher in the clipped group. The overall short-term conclusions of ISAT study engendered controversy on several fronts, as the results somewhat favoured endovascular coiling. The awaited long-term follow-up of ISAT patients was published in *Lancet* in 2009 (Molyneux, 2009). The results confirmed early observations: the risk of death at 5 years was significantly lower in the coiling group than in the clipping group. However, the insight revision of ISAT study revealed basic inconsistencies. The main remark refers to intent-to-treat analysis conception. Only 30% of screened patients with ruptured aneurysms were included to the study and randomized. If we exclude patients who died before treatment, there is the same mortality rate in neurosurgical and endovascular group (Bakker, 2010). However, the main problem is that the results of ISAT study were wrongly adjusted to the unruptured aneurysms by neuroradiologists, though the management and prognosis of ruptured and unruptured aneurysms differ fundamentally. Additionally, the results of above trial are absolutely inconclusive in term of GIAs. There were 155 patients with aneurysms exceeding 11 mm whereas GIAs were not distinguished. The mortality rates in endovascular coiling and microsurgical clipping groups were similar for patients with large aneurysms. The question about the ideal treatment for specific GIA characteristics remained unanswered. Fraser (Fraser, 2011) opposed handling GIAs the same way as other aneurysms, and suggested that case-based aneurysm treatment should be applied for GIAs. Indeed, revising the literature neither retrospective nor prospective randomized trials comparing endovascular and microsurgical approach regarding GIAs' exists. Lack of published comparisons stems from diversity of GIAs as one is amenable to endovascular therapy and another for surgery. Such lesions often demand a combined endovascular and microsurgical approach. The full armamentarium should be available to the cerebrovascular team to facilitate a comprehensive treatment method for these lesions. Maximizing efficacy and minimizing risk should always be a goal of effective approach for GIAs. Tabulated comparisons of these two methods, based on other publications, elaborate the present controversy in GIA treatment (Table 2). Mortality and rehemorrhage rates are similar, but complete occlusion and retreatment rates are higher in endovascular therapy studies. However, the assessment is valid only when meta-analysis would be performed. Listed series rarely exceed a hundred patients, comprises both patients with ruptured and unruptured GIAs and where different therapy strategies were applied in different publications. It seems impossible to provide one

and ultimately the best treatment modality or to perform randomized trials for patients suffering from GIAs.

Author	Year of publication	No of patients	Mortality (%)	Retreatment (%)	Rehemorrhage (%)	Complete occlusion (%)
Endovascular						
Gruber	1999	28	UNK	82	4	61
Sluzewski	2003	29	21	55	7	17
Jahromi	2008	39	29	54	5	36
Shi	2009	9	11	0	0	100
Lylyk	2009	8	UNK	0	0	67
<i>Summary</i>		8-39	11-29	0-82	0-7	17-100
Neurosurgical						
Lawton	2002	28	14	0	0	100
Jafar	2002	29	3	0	0	100
Hauck	2008	62	15	0	3	90
Sharma	2008	181	9	0	0	90
Sano	2010	109	22	0	0	100
Sughrue	2010	140	13	1	1	84
Sloniewski	2011	75	13	UNK	0	UNK
<i>Summary</i>		28-181	3-22	0-1	1-3	84-100

Abbreviations: UNK – unknown

Table 2. The comparison of treatment results: endovascular versus neurosurgical therapy in GIAs. Data derived from various authors (Gruber, 1999; Sluzewski, 2003; Jahromi, 2008; Shi, 2009; Lylyk, 2009; Lawton, 2002; Jafar, 2002; Hauck, 2008; Sharma, 2008; Sano, 2010; Sughrue, 2010; Szmuda & Sloniewski, 2011).

The current results for the endovascular treatment of GIAs with parent vessel preservation are not encouraging and are not as favourable as those for smaller aneurysms. However, most GIAs are amenable to endovascular coiling alone, balloon-assisted or stent-assisted coil embolization. Vessel reconstruction, especially in fusiform aneurysms, can be achieved by flow diverting stents. Nevertheless endovascular therapy is a treatment of choice in the majority of GIAs in most centres. A continuous development of techniques and devices can supersede surgery of GIAs in the future.

3.2. Microsurgical techniques

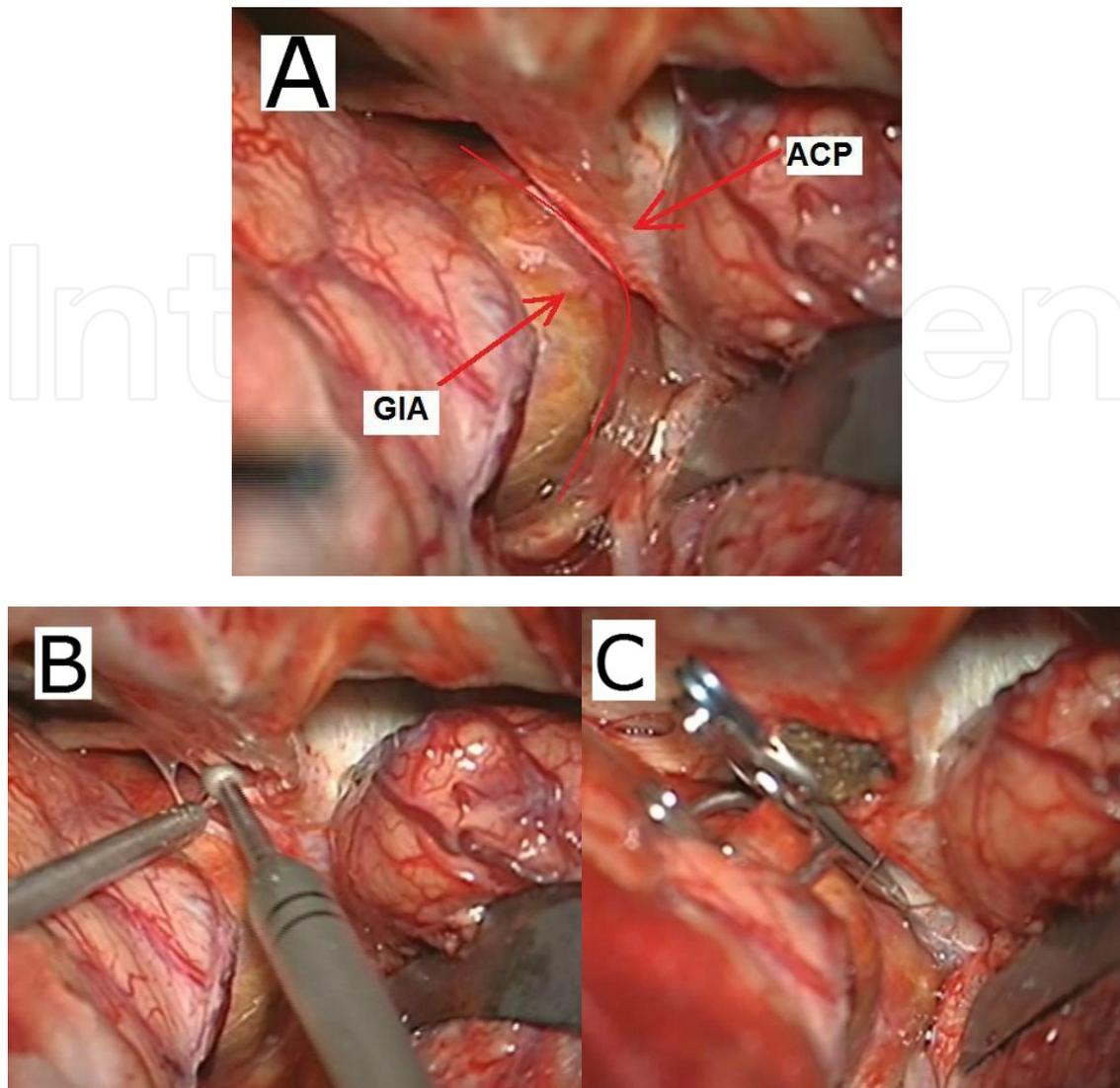
Large neck-to-dome ratio and limited surgical access are the main challenging therapeutic characteristics of GIAs. A part of the parent vessel proximally and distally to GIA, associated perforators, adjacent vessels and neural structures should be identified before GIA securing. These actions are imperative to reduce the consequences of intraoperative GIA rupture. Additionally, skull base surgery can cause severe complications which should be considered preoperatively. The aim of every craniotomy or craniectomy is an enhanced exposure achieved by removing additional bone and therefore minimizing cerebral retraction. Aneurysm location dictates the appropriate approach (Fig. 3).



Figure 3. The skull base approaches to GIAs (marked in colours) recommended by authors: orange – orbitozygomatic; green – pterional; red – modification of pterional approach to aneurysms of BA bifurcation; pink – far lateral; blue – retrosigmoid.

Pterional or orbitozygomatic craniotomy is dedicated for most GIAs: ICA, ACoA, MCA and BA bifurcation. Pterional craniotomy is preferred as it is a routine approach in neurosurgery. Additional opening of the superior orbital fissure should always precede dura incisure. Extended exposure for proximal ICA GIAs is reached by extradural or most often intradural anterior clinoid process removal (Fig. 4). This manoeuvre effectively increases the angle of view, although it can rarely cause postoperative cerebrospinal fluid leakage. The anterior clinoid process assessment in preoperative CT is strongly recommended. When deemed necessary, optic strut drilling is performed.

BA bifurcation can also be approached by a modification of pterional approach, which is commonly used in our institution (Krisht, 2005; Sloniewski, 2008). We do not use extent bone by drilling the whole zygomatic arch but we remove only its upper part. The anterior clinoid process is occasionally removed while the lateral part of the orbit and zygomatic notch widening (by drilling its superior aspect) should be performed. These techniques increase the angle of view at about approximately 10° comparing to the classical pterional craniotomy (Sloniewski, 2008). Midbasilar, SCA, AICA, VA and PICA GIAs can be secured by either of petrosal, extended retrosigmoid and far-lateral craniectomies. We always propose the use of limited a far-lateral craniectomy with opening of the foramen magnum and without posterior C1 arch removal. In our opinion the visualization of cerebellar tonsils



Abbreviations: ACP - anterior clinoid process.

Figure 4. Intradural anterior clinoid process removal. (A) ACP is in direct contact with GIA's neck at ophthalmic ICA origin, what prevents safe clipping. (B) ACP removal by the use of high speed drill with small diamond burr. (C) The hole that remained after ACP removal is filled with wax and haemostatic material (Surgicel®). Drilled ACP gives a space for prudent aneurysm neck clipping.

(via C1 arch osteotomy) is not essential while operating most GIAs. Petrosal or transclival approaches are associated with higher complication rates and therefore discontinued for GIA treatment in our institution. A possible cerebrospinal fluid leakage, meningitis, massive intraoperative bleeding outweigh extended exposure. Retrosigmoid approach is not originally intended for posterior circulation GIAs. This approach can be applied occasionally for some GIAs at the PICA or inferior junction of VA origin when the neck of the aneurysm is located higher than normal. Using a retrosigmoid craniectomy for GIA surgery should be supported by accessory and temporary endovascular balloon occlusion.

A variety of microsurgical occlusion techniques are available for vascular neurosurgeons: aneurysm neck clipping, aneurysmectomy, trapping of parent vessel, wrapping aneurysm

dome or extra- to intracranial by-pass. Typically, GIAs with well-defined neck are the most feasible for clipping. Vascular clips and microsurgical skills used in GIAs securing are different from those used in smaller aneurysms. A neurosurgeon should prepare before the surgery and be equipped with a complete selection of aneurysm clips: small and large, straight, angled, bayonet, fenestrated, Sugita and Sundt. One clip usually cannot bring the aneurysm walls together thus several clips or tandem angled fenestrated clips are placed in wide-necked or fusiform aneurysms (Fig. 5). These techniques are used to reconstruct the lumen of the afflicted parent vessel. Aneurysm clips have their limitations, whereas the most important in GIA surgery is weak closing forces. Placing several clips or stacking one on the top of another can prevent clip slippage. Intraluminal thromboses located at the aneurysm's neck, quite often in GIAs, need to be evacuated before definite clipping, which in a sense complicates the procedure.

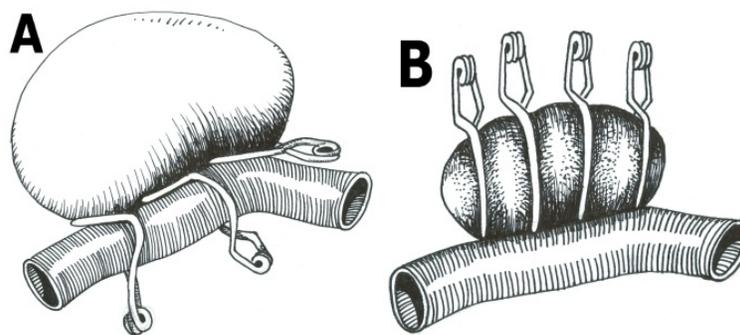


Figure 5. Schematic drawings of clipping techniques used in GIAs: (A) Tandem of fenestrated angled clips. (B) Several straight clips placement.

However, in two different situations - when a clip cannot embrace a GIA's broad neck and a standard clip slips from the aneurysm, we first place a fenestrated clip to form the neck. Then it is easier to stack the second clip, usually a straight or bayonet clip. All of the above microsurgical manoeuvres can lead to aneurysm rupture by puncturing the wall by the tip of a blade. Massive bleeding is a devastating event that results in altered clip positioning, differing from the positioning originally intended. In emergent situations, when other techniques are not feasible, parent artery sacrifice (trapping) can save a patient's life, although is regarded as a complication of aneurysm surgery. Aneurysmectomy, followed by clipping, theoretically resolves the compression of GIA on the neural structures. However, the studies of coiled GIAs revealed that neuropathies were caused by the pulsation of the aneurysm (Gonzalez, 2006). Therefore the aneurysmectomy or thrombosis evacuation may be abandoned. Aneurysm dome incision produces massive bleeding if the aneurysm neck is incompletely clipped.

Wrapping is used as a sole method of securing GIAs or combined with clipping (clip-wrapping technique). The treatment of GIAs should not be aimed at wrapping, although long-term findings based on 63 cases indicated that it is safe and durable method (Deshmukh, 2006). In our opinion, preventing rehaemorrhage from a GIA before further by-pass or coiling is the goal of wrapping. Various materials can be used, including cotton, muscle, gauze, Teflon, adhesives (fibrin glue and sealant) or collagen-impregnated Dacron

fabric. We prefer to use cotton because it causes an intermediate inflammatory response (Herrera, 1999). The previous results suggested that wrapping ruptured aneurysms is less effective than clipping in preventing rehaemorrhage or regrowth (Minakawa, 1987; Todd, 1989). The contemporary papers showed that wrapping of unclippable aneurysms (mostly GIAs) may be protective. Furthermore, the risk of complications due to wrapping is low.

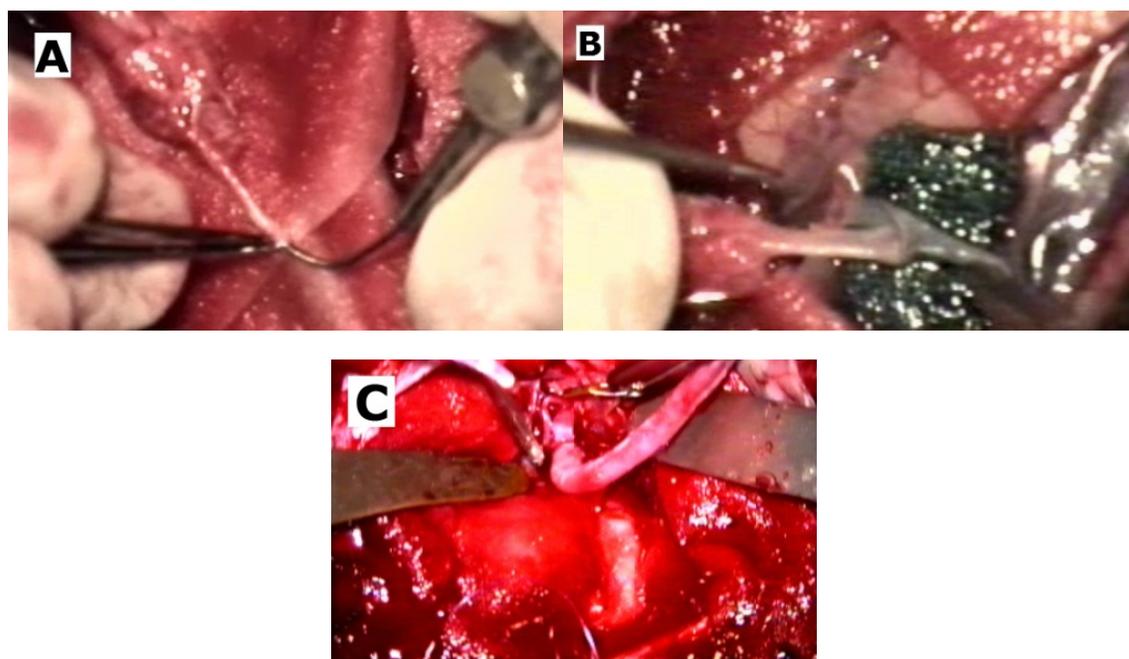


Figure 6. Extracranial to intracranial bypass. (A) Saphenous vein graft filled with saline and heparin. (B) The graft was anastomosed to the M4 segment, as part of an ICA to MCA bypass. (C) Temporary clips are being opened and the vein graft is filled with circulating blood.

Some GIAs' have features that do not permit direct clipping or endovascular obliteration. Incorporation of parent vessels, giant dome, arteriosclerosis or dense calcification of the aneurysm dome and neck, or fusiform shape may prevent successful obliteration. Excluding GIA from the circulation by Hunterian ligation and trapping (parent artery sacrifice) without bypass are not recommended techniques nowadays, as approximately 30% of patients have insufficient collateral flow (Barnett, 1994). The balloon occlusion test (BTO) is useful method for proper qualification of an individual for trapping or extracranial to intracranial bypass surgery with positive predictive value of 98% (vanRooij, 2005). However, BTO has several variations, technical nuances and interpretations (Lesley, 2009). The adjunct of xenon¹³³ cerebral blood flow measurement, single-photon-emission in computer tomography and transcranial Doppler ultrasonography increased the sensitivity of BTO (Fraser, 2011). Bypass is an alternative method of securing from further rupture. Since the first superficial temporal artery (STA) to MCA by-pass, the revascularisation methods have significantly developed. Radial artery or saphenous veins are used as a graft material. The anatomic location of a particular GIA dictates the endpoint of the by-pass. VA, petrous segment of ICA or external carotid artery (ECA) to MCA or PCA connections were made by various authors and described (LeRoux, 2003). Contemporarily the ICA to MCA high-flow bypasses are the most common. In addition microvascular skills are required and

should be maintained by constant training. When performing bypass surgery, even by skilful neurosurgeons, the temporary occlusion of the proximal major brain artery can result in brain ischemia. To solve the problem of temporary occlusion of the main brain arteries Tulleken developed the Excimer laser-assisted nonocclusive technique (ELANA) (Tulleken, 1995). The above method constructs an anastomosis without the need for temporary occlusion of brain arteries. For other bypasses a balloon occlusion test (BTO) or Xenon computer tomography are useful methods for proper qualification of individuals for extracranial to intracranial bypass surgery or parent artery sacrifice.

3.2. Accessory techniques

In many GIA cases, direct clipping is impossible. Therefore accessory techniques have been refined for years to provide adequate alternatives to patients with such presentations. Temporary occlusion by vessel clipping, endovascular balloon occlusion, temporary extracranial to intracranial by-pass, or retrograde suction decompression comprise some of the safest accessory techniques facilitating microsurgical exclusion of an aneurysm from circulation.

Temporary occlusion is a valuable method, which is used by most neurosurgeons in most clipped GIAs. Safety of this method varies according to the vessel occluded and the respective time of occlusion. To date, there are no time-limits for arterial occlusion. High tolerable occlusion times (without infarction observed in postoperative CT) of 60 minutes for ICA, 35 minutes for MCA and 19 minutes for BA were observed (LeRoux, 2003). Others used sophisticated techniques and proved that even brief episodes of cerebral vessel occlusion produced changes in the brain signals (Jiang, 2009). We use no more than three minutes of arterial occlusion and four to five minutes of reperfusion. However, poor clinical condition of patients with ruptured aneurysms and advanced age are significant risk factors for stroke related to temporary artery occlusion. Intermittent episodes of occlusion and reperfusion are controversial and therefore not recommended. The application of intraoperative monitoring (electroencephalography or somatosensory evoked potentials) during temporary clipping reduces the risk of ischaemic complications, although complicates the whole procedure. Instead of a surgical temporary clip, endovascular balloon introduction may be used for temporary occlusion. The efficacy of both methods is similar. The use of endovascular balloon does not carry surgery-related complications, though both methods of temporary obstruction increase the risk of ischemic deficit by local endothelial cell damage (MacDonald, 1994).

The role of STA to MCA bypass in excluding GIAs is regarded as historical. However, temporary low-flow bypass can be applied in some individuals when a prolonged clipping is regarded preoperatively. On the contrary to temporary occlusion of parent vessels, a circulatory may be superseded by low-flow shunt, which is not limited by time required for GIA securing. Neck clipping of a GIA with accessory temporary occlusion of the parent artery is a superior treatment to accessory by-pass, although it is inevitably associated with the risk of cerebral ischemia. Hongo proposed a 'double insurance bypass' of both the STA and radial artery to different portions of the MCA (Hongo, 2002). The STA is anastomosed

to the distal cortical branch of the MCA and is responsible for the blood flow to the distal territory while the radial artery is sutured to M2 or M3 and secures the ICA territory during temporary occlusion of the ICA during clipping a GIA. The results of this safe accessory technique were encouraging (Hongo, 2002; Ishikawa 2005), however, not confirmed by other authors.

Circulatory arrest and deep hypothermia are abandoned in most institutions nowadays. If neurosurgeons were to explore the abovementioned method, remarkable discussion concerning thorough technique learning, potential risk of complication and alternatives would be required. Hypothermia and cardiac arrest are still relatively high-risk procedures related to high rates of mortality and morbidity. The complications include hemodilution, coagulopathies, fibrinolysis, impairment of platelets as well as postoperative haematomas. Circulatory arrest should not exceed 30 minutes because of the increased occurrence of significant hypothermia-induced coagulopathy. Limited time is an additional factor in securing GIAs in cardiac arrest.

Retrograde suction decompression is a simple and effective method used in paraclinoid as well as distal portion of ICA GIAs. A method consists of retrograde suction of blood from closed circulatory resulting in deflation of the aneurysm. Followed by surgical exposure of the ECA, superior thyroid artery is dissected and then catheterised. After temporary clipping of the ECA proximally to an introduced catheter and the ICA distal to the aneurysm, manual syringe suction is performed (Fig. 7). The dome of the aneurysm collapses facilitating the aneurysm neck preparation and its clipping. The drawback of that method is the development of thromboses within the lumen of a GIA, which is relatively common complication. A variety of modifications has been published including novel employment of endovascular embolectomy device for retrograde suction (Hoh, 2007).

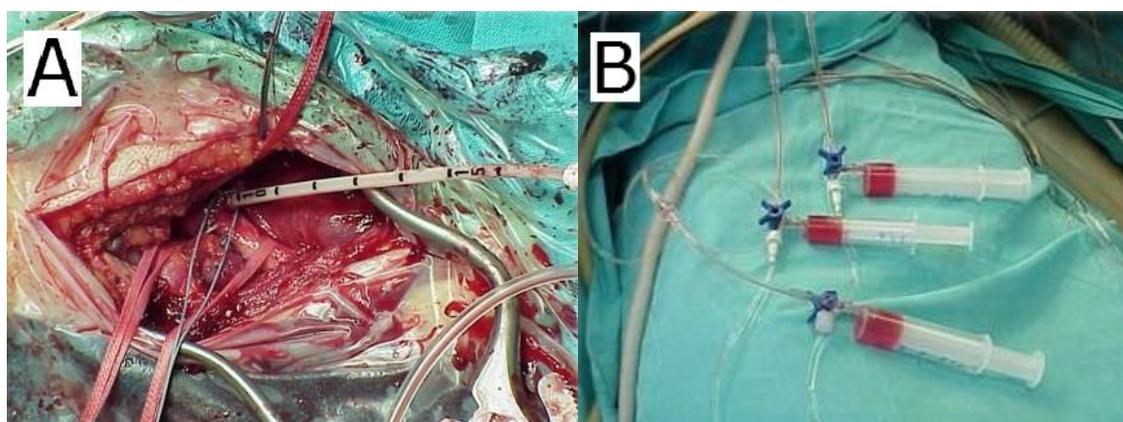


Figure 7. Retrograde suction. (A) The superior thyroid artery is catheterised by means of common central venous catheter. The external carotid artery is temporarily closed at the moment of suction. (B) We use three syringes: two for retrograde suction and one filled with heparin for flushing purposes.

Several monitoring methods test vascular patency and proper aneurysm occlusion: intraoperative fluorescence, Doppler ultrasonography examination or intraoperative angiography. The last one is supposed to be the most beneficial over others, though is

invasive and thus related to increased complication rate. A routine use of intraoperative angiography in all operated aneurysms is debatable. In literature, necessary intraoperative angiography was performed in about 6% of cases of altered aneurysm clip position (Klopfenstein, 2004). Some aneurysms, including GIAs, are more susceptible to incomplete clipping and therefore may require intraoperative evaluation with angiography. The authors of retrospective analysis in postoperative angiography following aneurysm clipping concluded that the routine intraoperative angiography is recommended in treatment of GIAs (Kivisaari, 2004). In large and giant aneurysms the incomplete occlusion rate exceeded 50% and these patients required further complementary endovascular therapy or surgical revision.

Intraoperative fluorescence is obtained by the addition of near infrared imaging to surgical microscopes and high resolution videoangiography. When administered intravenously, the dye reacts in plasma in approximately 4 minutes. Then the fluorescence (indocyanine green) is induced by near infrared and recorded by a camera (Snyder, 2011). Intraoperative fluorescence angiography is helpful in performing 'Matas test' during clipping ACoA GIA (Murai, 2011) or ensuring the patency of the parent artery and perforators. However, in 5% of cases the image quality is poor (Raabe, 2005). The limitations of fluorescence angiography refer to GIAs affected by calcifications, thrombosed and those with thick walls (Snyder, 2011).

Colour Doppler and micro-Doppler ultrasonography are reliable and simple methods to verify the correct placement of the clip in aneurysm surgery. Micro-Doppler can detect incomplete exclusion of the aneurysm, stenosis of a parent vessel or occlusion of the parent or adjacent arteries and therefore is used routinely in GIAs. The confirmation of proper blood flow confirmed in ultrasonography allows an addition of another clip to a GIAs neck and afterwards in case of stenosis the clip can be removed. Comparing to other intraoperative vascular patency methods, the cost efficiency of micro-Doppler is favourable (Kapsalski, 2005).

Gruber compared the intraoperative monitoring and vascular imaging methods (Gruber, 2011). He concluded that these methods rather complement than compete. None of them are reliable when used as a single method.

3.4. Complications

Open surgery of GIAs results in more complications than any endovascular securing method (Gobin, 1996; Johnston, 1999). Many of the surgically treated GIAs referencing adverse events are dated prior to the introduction of Guglielmi detachable coils, microsurgery and neuroanaesthesiology development. Issues regarding complication rates of endovascular and surgical methods are indications for performing randomised trials in GIAs.

General and procedure-related adverse events are distinguished. General ones derive from aneurysm rupture, anaesthesia and imperfection of postoperative care.

Procedure	Specific complications of method
Craniotomy	
Pterional	Impaired memory or cognition (due to brain damage), facial nerve paresis, temporal muscle atrophy, cerebrospinal fluid leakage.
ACP removal	GIA or ICA wall damage, cerebrospinal fluid leakage through paranasal sinuses.
Orbitozygomatic	Severe orbital swelling, changes in visual activity, cerebrospinal fluid leakage through paranasal sinuses.
Retrosigmoid	VA injury, cranial nerves deficits, nasal cerebrospinal fluid leakage through mastoid cells, injury of venous sinuses.
Far-lateral	VA injury, cranial nerves deficits, nasal cerebrospinal fluid leakage through mastoid cells, injury of venous sinuses.
Securing methods used in GIAs	
Clipping	Intraoperative aneurysm rupture or cerebral injury, brain swelling due to spatulas use, major vessel stenosis or occlusion, clip slippage, aneurysm residue.
Wrapping	Major vessel stenosis or occlusion, recurrent haemorrhage, vasospasm, arachnoiditis, granuloma in the region of wrapping that can cause cranial nerves neuropathies.
Trapping	Cerebral ischemia in the region of occluded parent artery, thrombotic occlusion of perforators.
By-pass	Cerebral ischemia due to bypass insufficiency, thrombotic occlusion of perforators, heparin-induced haemorrhages.
Accessory techniques	
Temporary vascular occlusion	Cerebral ischemia in the region of temporarily occluded parent artery, endothelial damages.
Retrograde suction	Cerebral ischemia in the region of temporarily occluded parent artery, thrombotic occlusion of arterial branches, endothelial damages.
Deep hypothermia and cardiac arrest	Thrombophlebitis, cardiac arrhythmia, new neurologic deficits occurrence, temperature instability, delayed awakening, coagulopathies, interstitial fluid sequestration.
Temporary bypass	Cerebral ischemia in the region of occluded parent artery, thrombotic occlusion of perforators, heparin-induced haemorrhages.
Intraoperative angiography	Femoral artery thrombosis or pseudoaneurysm, thrombotic occlusion of cerebral arteries, groin hematoma, aortic dissection.
Fluorescence angiography	Possible vasovagal reactions, contraindicated in patients with a history of iodine allergy.
Doppler ultrasonography	None.

Table 3. The characteristic of specific complications related to craniotomies, securing methods and accessory techniques used in GIAs.

The course of treatment following SAH is different than methods used in patients with unruptured GIAs. The consequences of aneurysm rupture include: hypovolemia, hyponatremia, hydrocephalus, cardiac problems, seizures, rebleeding from unsecured aneurysms, symptomatic cerebral ischemia secondary to cerebral vasospasm, coma and death.

However, the main complication of SAH from a neurosurgical point of view is cerebral vasospasm. It is defined as self-limited narrowing of a cerebral vasculature and is observed angiographically with or without clinical manifestation. Angiographic vasospasm refers up to 97% of patients, while neurological signs are observed only in 33% (Dorsch, 1994). Cerebral vasospasm is responsible for about 10% of deaths and 10% of permanent disability after SAH (Dorsch, 1994). These high rates of unfavourable outcomes followed by cerebral vasospasm underestimate the role of postoperative management secondary to SAH and underline the remaining challenges (LeRoux, 2003). Rebleeding from previously secured GIAs occurs rarely in the postoperative period, however, is related to high mortality.

The complications after general anaesthesia in GIAs and smaller aneurysms are similar. Theoretically, the prolonged surgery of GIAs may result in higher rate of adverse events. SAH increases the risk of pulmonary complications, including pneumonia, pulmonary emboli or oedema, adult respiratory distress syndrome (Solenski, 1995) and cardiac arrhythmia (up to 5%). Hypovolemia, hypokalaemia and hypotension generally are iatrogenic consequences of inappropriate management after SAH.

Procedure-related complications are divided into groups of procedures: craniotomy, aneurysm securing and accessory techniques (Table 3). Brain contusion is the most serious, while surgical wound infection is the most common consequence of craniotomy.

Amongst GIA securing methods a bleed from a ruptured neck or dome of the aneurysm without any vascular control is the most dangerous intraoperative failure. All of the accessory techniques are low risk procedures, in contrast to deep hypothermia.

However, the number of complications can differ significantly among authors.

LeRoux indicates that complication rate can reach 100%, depending on accepted criteria applied by investigator (LeRoux, 2003).

4. Outcome

Surgical mortality of GIAs is estimated on average rate at 10% and may range from 4 to 21% (Sharma, 2008). Short-term outcome of ruptured GIAs achieved in a multicentre study was worse than smaller ones (Kassell, 1990). The study from our institution (Szmuda & Sloniewski, 2011) did not coincide with the stereotype of unfavourable treatment results in GIAs. Mortality rate, short and long-term outcome after the operation of giant and smaller ICA aneurysms were similar. Our results proved that size of an aneurysm is not a prognostic factor, but there are other more prominent variables to explore when determining mortality. A thorough multivariate analysis should be a tool used in prognosis evaluation. Moreover, the outcomes of ruptured and unruptured GIAs differ and therefore should be analysed separately. However, the quantification of outcomes in treated aneurysms is an elusive problem. Beyond dispute mortality is the most important endpoint in GIA studies, followed by clinical condition at discharge, functional status, and all aspects with regards to the quality of life several years following the operation. The radiological outcome of secured GIAs should run parallel to both the physical and mental assessment of

treated patients. The results of surgery based on older publications do not reflect the current practice in the treatment of GIAs (Sughrue, 2010). The concern about the best treatment method in that group is challenging, whereas evidence based proofs are ambiguous.

Recent studies emphasize economic evaluations. Cost-effective analyses, comparing endovascular and microsurgical methods, should be translated into GIAs.

4.1. Radiological outcome

Postoperative subtracted angiography or occasionally computer tomography angiography after successful by-pass or occlusion of a GIA can reveal initial complications. Cerebral vasospasm, clip slippage or critical stenosis in some cases may result in postoperative management alteration or a second operation. Clip slippage after successful occlusion occurs in 0.2% of cases (Asgari, 2003) due to inadequate closing forces of clips, which are intended for use in smaller aneurysms, are used in the treatment of GIAs. If clip displacement is observed intraoperatively it can be replaced safely once again and additionally strengthened by the positioning of a second forcing clip. Even application of several aneurysm clips may be insufficient. In our series, clip displacement in further control angiography was noted in one patient of 128 operated GIAs (0.8%). The adhesions around complex of clips prevented their safe reposition during the revision (Fig. 8). However, a slipped clip may lead to fatal intracerebral haemorrhage (Wester, 2009).

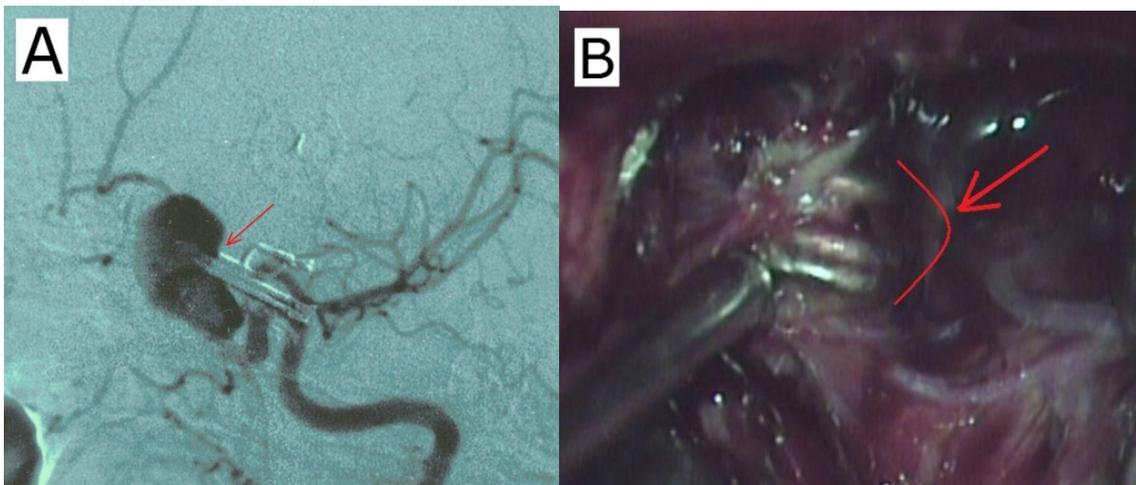


Figure 8. Slipped complex of two straight clips and one fenestrated bayonet clip from ICA GIA. (A) The blades of the fenestrated clip are totally out of the aneurysm's dome. Probably the summary closing force of applied clips proved to be insufficient. (B) Four months after initial surgery the adhesion of the clips located outside the Sylvian fissure, prevented their safe reposition.

The evaluation methods measuring the visual degree of an aneurysm occlusion in postoperative angiography may vary among studies (Gonzalez, 2006). Modified Raymond classification is commonly used following endovascular coiling (Raymond, 1997). The application of the above scale to surgical occlusion assessment is erroneous as a dog-ear remnant is a characteristic finding after coiling. The occlusion of GIAs can be classified using postoperative angiography as incomplete – more than 5% of remaining aneurysm

lumen, minimal contrasting aneurysm residue – small neck remnant and as complete – no remnant (Sughrue, 2010). Complete occlusion refers to the majority of clipped GIAs in various studies (see Table 2), although in one study (Kivisaari, 2004) is lower (57%). In our institution postoperative assessment of the duration of occlusion was not assessed in every case, therefore it is impossible to compare our results with other reported works (Szmuda & Sloniewski, 2011). In term of occlusion rate endovascular therapy methods seems to be inferior to surgery; postprocedural incomplete occlusion after coiling can be as low as 17% (Sluzewski, 2003). However, a part of contrasting neck is quite often observed in GIAs (Kivisaari, 2004). Supplementary stent or coil embolization of the aneurysm residue could be offered after angiographic assessment (Fig. 9). The significant limitation of supplementary endovascular therapy is that during the acute phase of subarachnoid haemorrhage stenting is not recommended, due to the need of anticoagulant therapy and should be postponed (Gonzalez, 2006).

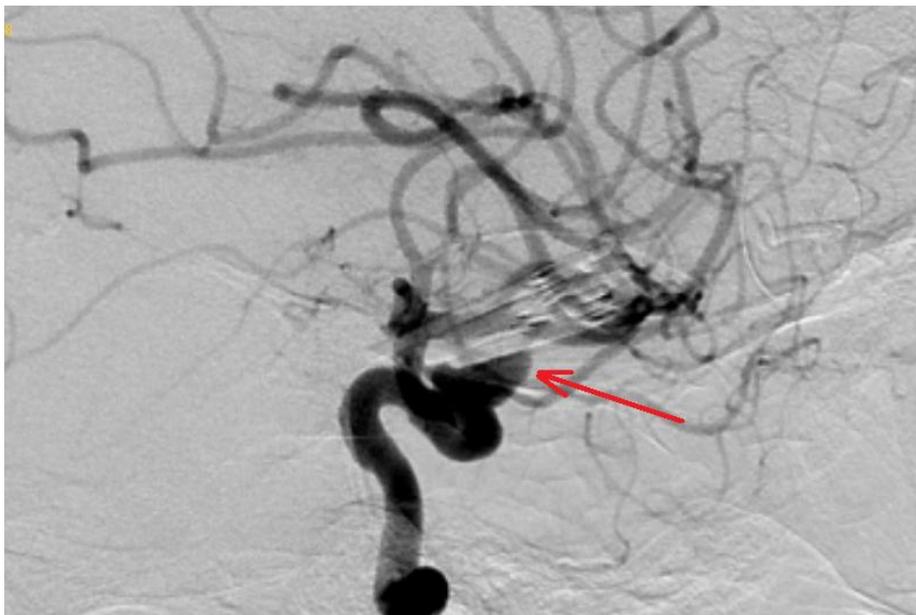


Figure 9. Four long straight clips were applied in unruptured paraclinoid GIA. Residual aneurysm neck was observed in postoperative DSA. The patient required a supplementary coiling of an incompletely occluded GIA.

4.2. Clinical outcome analysis

We analysed the clinical outcome following treatment of single artery ICA GIAs in our institution from 1997 to 2006. In 2011 two papers concerning our treatment results were published (Szmuda & Sloniewski, 2011, 2011) and one another is in press. ICA saccular aneurysms were assessed; the retrospective analysis of series consisted of 78 GIAs and 250 smaller aneurysms. Both groups comprised ruptured and unruptured aneurysms. All patients suffering from GIAs of ICA origin were offered surgery and all underwent surgical treatment by our senior author (PS). Therefore the analysis reflected a single-surgeon experience measured by means of clinical treatment results. The general outcomes of GIA surgery were published in *Acta Neurochirurgica* (Szmuda & Sloniewski, 2011). There were

no significant differences between GIAs and smaller ICA aneurysms with respect to mortality, unfavourable outcomes rates as well as quality of life. Moreover, the treatment results were similar in separate comparisons of size aneurysm groups among ruptured and unruptured ones. Mortality of presented GIAs' as well as unfavourable outcome rates were comparable to other published works. Postoperative death rate for GIAs depends on group characteristic and ruptured to unruptured aneurysm ratio. Mortality rate may vary from 4 to 21%, with an average of 10% (Sharma, 2008), while in our study was 12.8% (Table 4).

	GIAs (n; %)	Smaller aneurysms (n; %)	<i>p</i>
All ICA aneurysms	78	250	
Mortality rate	10; 12.8%	30; 12.0%	0.84
Unfavourable short-term outcome rate (GOS score equal or lower than 3)	22; 61.1%	119; 57.8%	0.26
Low quality of life rate (total SF-36 score equal or lower than 50)	21; 41.2%	70; 45.1%	0.62
Ruptured ICA aneurysms	36	206	
Mortality rate	6; 16.7%	30; 14.6%	0.74
Unfavourable short-term outcome rate (GOS score equal or lower than 3)	14; 38.9%	87; 42.2%	0.78
Low quality of life rate (total SF-36 score equal or lower than 50)	6; 27.3%	56; 45.2%	0.12
Unruptured ICA aneurysms	42	44	
Mortality rate	4; 9.5%	0; 0.0%	0.06
Unfavourable short-term outcome rate (GOS score equal or lower than 3)	6; 14.3%	3; 6.8%	0.26
Low quality of life rate (total SF-36 score equal or lower than 50)	16; 54.8%	15; 48.3%	0.19

Table 4. The comparison of general treatment results between giant and smaller ICA aneurysms based on own series (Szmuda & Sloniewski, 2011).

Most of ICA GIAs (n=57; 73%) were clipped; the rest of the aneurysms were excluded from circulation via parent vessel occlusion with extracranial to intracranial bypass (n=15; 19%) or without graft surgery (n=2; 3%). ECA to distal segment (M3 or M4) of MCA bypass was the most common (n=10), while ICA to MCA or low-flow (STA to MCA) bypasses were performed occasionally (n=5). In one individual the aneurysm was wrapped and in three patients a GIA was not secured at all. The operative methods were analysed regarding mortality, short and long term outcome. There were no statistically significant differences observed between these results, although two of three patients that GIA was not secured intraoperatively died and one of two patients after trapping experienced permanent disability. The outcome of these two patients with unsecured GIA from our series is a reflection of a poor natural history of untreated lesions reported by Peerless (Peerless, 1990,

as cited in Youmans, 1990). Mortality rates were 68% and 85% in two and five years of follow-up respectively. ICA occlusion due to ruptured GIA without performing by-pass (trapping) is a permissible surgical method in case of intraoperative bleeding. A permanent neurological deficit in patients from our series with occluded ICA is a consequence of both SAH and rescue clipping. In two individuals low-flow bypasses were found to be insufficient as one patient died due to cerebral ischemia. (Table 5)

GIA securing method	Mortality rate % (n)	Unfavourable short-term outcome (%)	Unfavourable long-term outcome (%)
Clipping	10.7% (6/57)	18.0% (9/50)	40.5% (15/37)
High-flow by-pass	10.0% (1/10)	0.0% (0/12)	33.3% (4/12)
Low-flow bypass	20.0% (1/5)	0.0% (0/4)	25.0% (1/4)
Trapping	0.0% (0/2)	50.0% (1/2)	100.0% (1/1)
Wrapping	0.0% (0/1)	0.0% (0/1)	0.0% (0/1)
Not secured	66.6% (2/3)	no FU	no FU

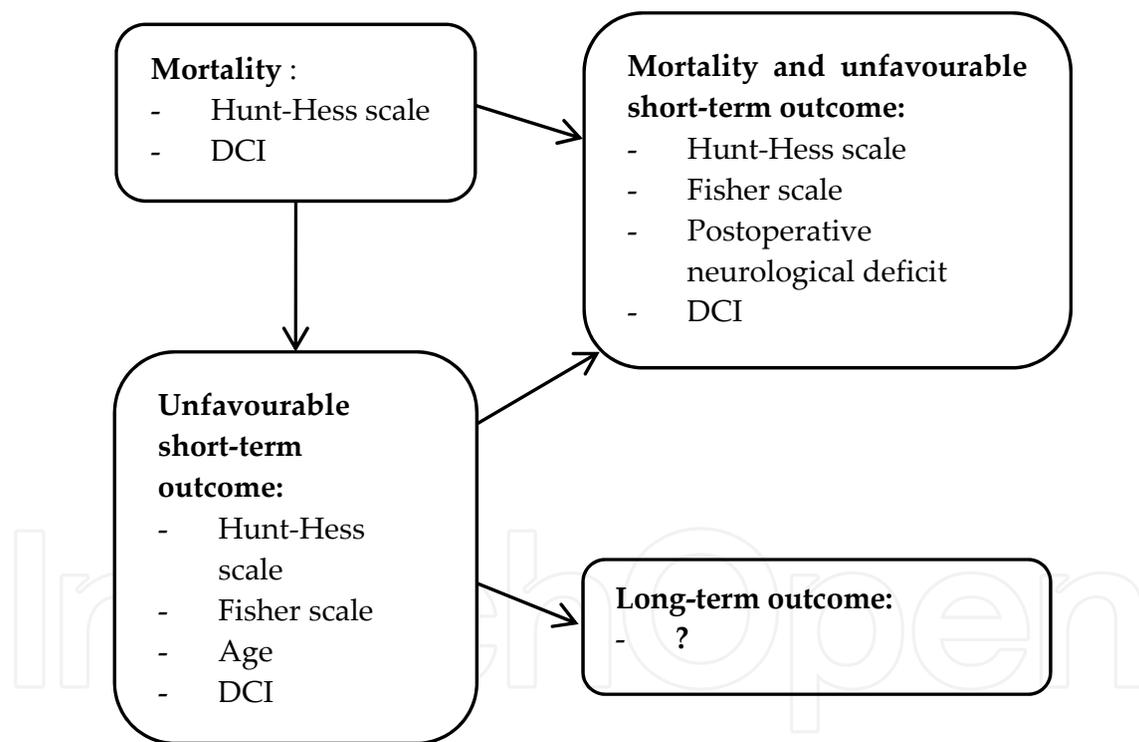
Abbreviations: no FU – no follow-up

Table 5. Characteristic of outcomes in ICA GIAs by treatment methods, derived from own series (Szmuda & Sloniewski, 2011).

A variety of accessory techniques were used in eight cases from the series. Temporary low-flow bypass (n=1), retrograde suction (n=4), temporary balloon occlusion (n=1) and deep hypothermic circulatory arrest (n=2) were undoubtedly beneficial in clipping. Both patients with GIA secured under cardiac arrest survived, did not experience any method-related complication and were discharged home with favourable outcome. However, abovementioned excellent outcome of used hypothermia refer to small group of GIAs at the ICA origin, the complication rate in patients operated on due to other indications was increased. The application of deep hypothermic cardiac arrest is contemporarily limited in our institution to individuals when simultaneous cardiocirculatory approach is needed. In our opinion, retrograde suction is a powerful tool in paraclinoid GIAs among accessory techniques. The simplicity and low complication rate are its two main advantages.

In our series the giant size of ICA aneurysms was not related to mortality and short and long-term outcome. However, the analysis of clinical outcomes in ruptured aneurysms should include other factors directly related to SAH. The summary of various analyses led to the creation of an accepted neurosurgical doctrine, in which, the triad of factors: age, clinical status on admission and vasospasm affect mortality after surgery in ruptured intracranial aneurysms (Salary, 2007; Roos, 2000; Taylor, 2004). Kassell also introduced the size of the aneurysm is an independent factor of a worse outcome (Kassell, 1990). Moreover,

an aneurysm occurrence at the posterior circulation resulted in a higher rate of poor treatment results. Concluding from Kassell's study the analysis of factors that might influence the outcome following SAH should comprise the patients with aneurysms derived from a selected artery, for instance ICA. Multivariate analyses of outcome in ruptured ICA aneurysms was published (Szmuda & Sloniewski, 2011). As a result of these analyses various factors appeared significant, although different for mortality and short-term outcome (Fig. 10). Moreover, when mortality and unfavourable short-term outcome were analysed together there were also discrepancies. Clinical state at admission (based on Hunt-Hess scale) and delayed cerebral ischemia (as a resolution of cerebral vasospasm) affected all outcome measurements. SAH intensity (Fisher scale) influenced short-term outcome as well as a combined mortality and unfavourable short-term outcome. However, older age was prevalent in determining clinical state on discharge, although was not related with mortality. Geriatric populations are considered to be more sensitive to surgery due to comorbidities affecting the course of treatment. No significant factors connected with long-term quality of life were found.

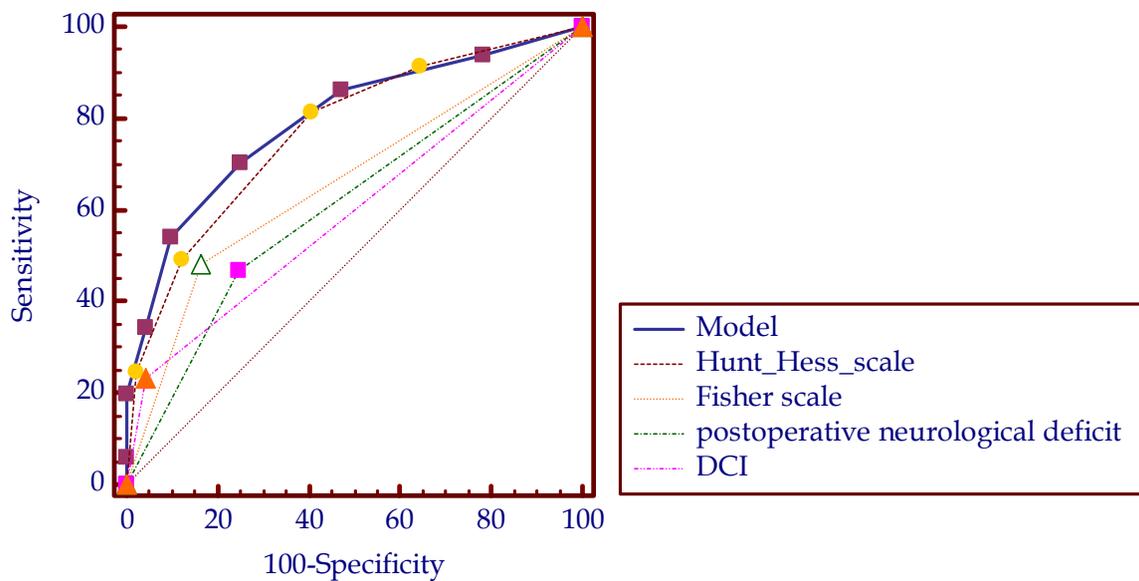


Abbreviations: DCI - delayed cerebral ischemia.

Figure 10. Diagram presenting factors determining mortality, short and long-term outcome in ruptured ICA aneurysms.

Fourth or fifth grade in Hunt-Hess scale found to be dominant factor in determining mortality and unfavourable short-term outcome in ruptured ICA aneurysms based on a statistical tool called receiver operating characteristic (ROC). Followed by poor clinical state,

a massive bleed assessed by the Fisher scale, postoperative neurological deficit occurrence and delayed cerebral ischemia were consecutively responsible for worse outcome (Fig.11).



Abbreviations: DCI – delayed cerebral ischemia.

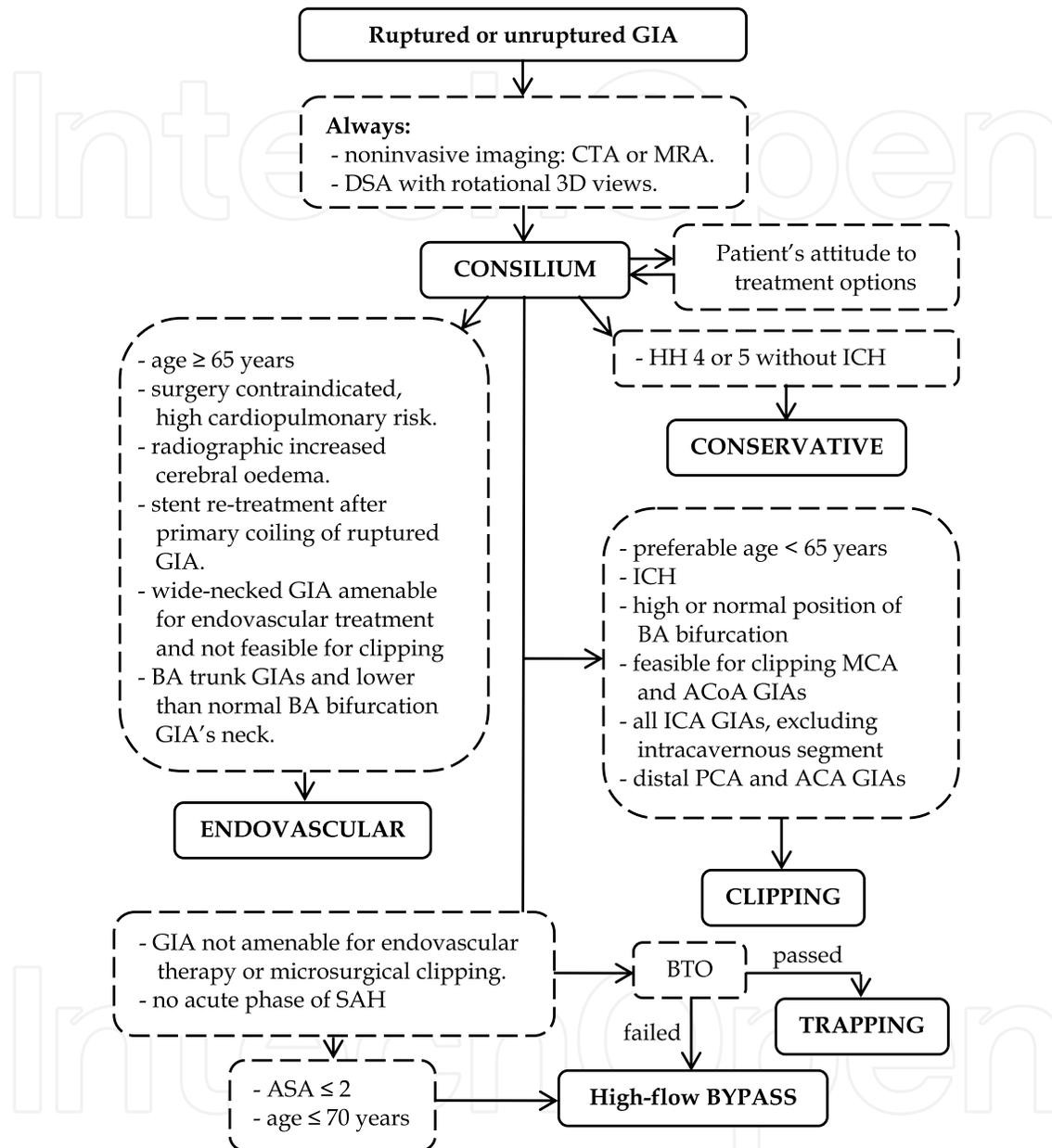
Figure 11. Receiver operating characteristic curve (ROC curve) presenting consecutive factors (according to importance) responsible for combined postoperative mortality and unfavourable short-term outcome in ruptured ICA aneurysms.

Another important issue is how the cost-effectiveness of GIAs therapy compares with smaller aneurysms. The undoubted increased cost of GIA therapy in comparison with smaller aneurysms is related to more complex operative procedures, increased time of surgery and hospital stay. There is a lack of publications regarding such comparisons, however, the economic analysis of unruptured aneurysms proved that treatment is cost-effective if addressed to large aneurysms and GIAs (Johnston & Gress, 1999). These lesions produce symptoms by compressing neural structures and have a high risk of rupture. Therefore a symptomatic patient harbouring unruptured GIA may potentially benefit more quality-adjusted life-years (QALY) (Qureshi, 2007).

5. Evidence-based paradigms for treatment:

GIAs can be secured effectively by neurosurgical or endovascular therapy, though there is a subset of factors (size, morphology, location, segment of artery, related anatomy, comorbidities as well as timing of surgery) which complicate treatment decision. Understanding the ability of variety techniques to the cerebrovascular team facilitates a comprehensive method for treating these lesions, maximizing efficacy and minimizing risk. In 2011 Fraser from Cornell University (New York) created a paradigm for approaching all aneurysms at the institution using currently accessible technology. We reported single-surgeon's experience of ICA GIAs treatment. Based on literature (Fraser, 2011; Cantore, 2008; Kai, 2007, Sharma, 2008), American Society of Anaesthesiologists as well as senior

author (PS) reflections, we propose the detailed model of treatment methods to decision making processes in GIAs, also indicating the possible alternatives. However, it should be pointed that a paradigm is a proposal referring to current technology in our institution.



Abbreviations: ASA - American Society of Anaesthesiologists scale; BTO - balloon occlusion test; CTA - computer tomography angiography; HH - Hunt-Hess scale; ICH - intracranial haematoma; MRA - magnetic resonance angiography.

Figure 12. Treatment algorithm for GIAs evaluation and treatment in our institution.

Despite patient's previous radiograms, a meticulous preoperative diagnosis is to be complemented by means of cerebral rotational, three dimensional subtracted angiography (3D DSA) and computer tomography angiography (CTA) or optionally by magnetic resonance angiography (MRA). 3D DSA enables a visualisation of a detailed GIA's

anatomical features and originating perforators, though its ability to demonstrate calcification or thrombosis is limited (Hoit, 2006). CTA accomplishes above limitations and moreover shows surrounding bony structures. In posterior fossa or ICA GIAs MRA can visualise adhering neural structures, although is performed occasionally in our institution. Conservative approach is preferred in individuals in fourth or fifth Hunt-Hess grade, excepting those with intracerebral haemorrhage. Conscious and informed patient's attitude to proposed GIA's treatment method is an important factor in making a decision. Endovascular therapy is approached to older individuals with high cardiopulmonary risk and when surgery is contraindicated. For ruptured GIAs an increased radiographic cerebral oedema may prevent direct clipping. Wide-necked GIAs not feasible for clipping should be secured by endovascular methods. GIAs originating at BA trunk or BA bifurcation with the neck located lower than normal are also offered endovascular treatment. A preferable group of patients for direct neck clipping are those younger than 65 years old. All GIAs amenable for clipping in neurosurgeon's opinion should be secured in this manner. In our institution distal PCA or ACA GIAs are excluded from a circulation by clipping technique. However, the most controversy refers to GIAs that are not suitable for both endovascular therapy and microsurgical clipping. In this case an endovascular therapy transforms these lesions into a chronic disease with a relapsing clinical course by further retreatments and repeated risk exposure (Sughrue, 2010). Flow-diverting stents potentially offer a meaningful benefit over surgery, although the outcome has not been sufficiently confirmed. Nonetheless, if endovascular therapy or direct clipping are not amenable bypass or parent artery sacrifice (trapping) is recommended, though bypass is not allowed in acute phase of SAH. Proper qualification to one of above surgical method is validated in balloon occlusion test (BTO). However this test is not meticulous enough, therefore the decision of treatment method can be supplemented by Xenon computer tomography or single-photon emission computed tomography (SPECT). Patients younger than 70 years old with equal or lower than grade II in American Society of Anaesthesiologists scale are qualifying for high-flow bypass without BTO, which is in accordance with contemporary literature (Cantore, 2008).

The contemporary experience with GIAs is limited to retrospective analysis of selected group of ICA GIAs (Szmuda & Sloniewski, 2011). Nonetheless, it demonstrates that experienced neurosurgeon (senior author - PS) can achieve excellent results using a single surgery, definitive and durable therapy.

6. Conclusions/perspectives

General unsatisfactory outcomes of GIAs do not warrant risky microsurgical or endovascular interventions. The more accustomed the neurovascular surgeon is the more difficult is the selection of the appropriate method for securing GIAs. However, in experienced hands the outcomes after treatment of giant and smaller aneurysms do not differ. In elderly populations, the efficacy must be weighed against the natural history of the GIA by considering expected remaining lifetime.

Endovascular embolization competes with open microsurgery in the field of cerebral aneurysms. Prospective and randomized trials (CURES, ATENA and STAT) are intent-to-treat analyses, therefore not dedicated for GIAs. The promising outcomes achieved by endovascular therapy for small aneurysms nevertheless remain unconfirmed for GIAs. Application of these results to GIAs is misleading. To date, the knowledge is based on small published series.

Forced by the completion of both treatment options, continuous development of neither endovascular nor microsurgical methods is being observed. Hopefully for patient's benefit!

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7. References

- Anson, JA. (1995). Epidemiology and natural history, In: *Giant Intracranial Aneurysms*, Awad IA, Barrow DL, pp. (23–33), American Association of Neurological Surgeons, Park Ridge.
- Asgari, S., Wanke, I., Schoch, B., Stolke, D. (2003). Recurrent hemorrhage after initially complete occlusion of intracranial aneurysms. *Neurosurg Rev*, 26, 4, October 2003, (269–274), 0344-5607.
- Bakker, N.A. (2010). International subarachnoid aneurysm trial 2009: Endovascular coiling of ruptured intracranial aneurysms has no significant advantage over neurosurgical clipping. *Neurosurgery*, 66, 5, May 2010, pp. (961-962), 0148-396X.
- Barnett, D.W., Barrow, D.L., Joseph, G.J. (1994). Combined extracranial-intracranial bypass and intraoperative balloon occlusion for the treatment of intracavernous and proximal carotid artery aneurysms. *Neurosurgery*, 35, pp. (92–97), 0148-396X.
- Barth, A., de Tribolet, N., Barth, A. (1994). Growth of small saccular aneurysms to giant aneurysms: presentation of three cases. *Surgical Neurology*, 41, pp. (277–280), .
- Cantore, G., Santoro, A., Guidetti, G., Delfinis, C.P., Colonnese, C., Passacantilli, E. (2008). Surgical treatment of giant intracranial aneurysms: Current viewpoint. *Neurosurgery*, 63, 4., October 2008, pp. (279-289), 0148-396X.
- Cawley, C.M., Zipfel, G.J., Day, A.L. (1998). Surgical treatment of paraclinoid and ophthalmic aneurysms. *Neurosurg Clin North Am*, 9, pp. (765-783), .

- Deshmukh, V.R., Kakarla, U.K., Figueiredo, E.G., Zabramski, J.M., Spetzler, R.F. (2006). Long-term clinical and angiographic follow-up of unclippable wrapped intracranial aneurysms. *Neurosurgery*, 58, 3, March 2006, pp. (434-442), 0148-396X.
- Dorsch, N.W., King, M.T. (1994). A review of cerebral vasospasm in aneurysmal subarachnoid haemorrhage Part I: Incidence and effects. *Journal of Clinical Neuroscience*, 1, 1, January 1994, pp. (19-26), .
- Fraser, J.F., Stieg, P.E. (2011) Surgical bypass for intracranial aneurysms: Navigating around a changing paradigm. *World Neurosurgery*, 75, 3-4, March 2011, pp. (414-417), .
- Gobin, Y.P., Vinuela, F., Gurian, J.H., et al. (1996). Treatment of large and giant fusiform intracranial aneurysms with Guglielmi detachable coils. *J Neurosurg*, 1996, 84, 1, pp. (55-62), .
- Gonzalez, N.R. et al. (2006). Challenges in the endovascular treatment of giant intracranial aneurysms. *Neurosurgery*, 59, 5, November 2006, pp. (S3-113), 0148-396X.
- Gruber, A., Killer, M., Bavinzski, G., Richling, B. (1999). Clinical and angiographic results of endosaccular coiling treatment of giant and very large intracranial aneurysms: a 7-year, single-center experience. *Neurosurgery*, 1999, 45, 4, pp. (793-803), 0148-396X.
- Gruber, A., Dorfer, C. , Standhardt, H., Bavinzski, G., Knosp, E. (2011). Prospective comparison of intraoperative vascular monitoring technologies during cerebral aneurysm surgery. *Neurosurgery*, 68, 3, March 2011, pp. (657-673), 0148-396X.
- Hauck, E.F., Wohlfeld, B., Welch, B.G., White, J.A., Samson, D. (2008). Clipping of very large or giant unruptured intracranial aneurysms in the anterior circulation: an outcome study. *J Neurosurg*, 109, 6, 2008, pp. (1012-1018), .
- Herrera, O., Kawamura, S., Yasui, N., Yoshida, Y. (1999). Histological changes in the rat common carotid artery induced by aneurysmal wrapping and coating materials. *Neurol Med Chir (Tokyo)*, , 39, 1999, pp. (134-139), .
- Hoh, D.J., Larsen, D.W., Elder, J.B., Kim, P.E., Giannotta, S.L., Liu, C.Y. (2008). Novel use of an endovascular embolectomy device for retrograde suction decompression-assisted clip ligation of a large paraclinoid aneurysm: technical case report. *Neurosurgery*, 62, 5 Suppl 2, May 2008, pp. (412-413), 0148-396X.
- Hoit, D.A., Malek, A.M. (2006). Fusion of three-dimensional calcium rendering with rotational angiography to guide the treatment of a giant intracranial aneurysm: Technical case report. *Neurosurgery*, 58, Suppl 1, February 2006, pp. (173-174), 0148-396X.
- Hongo, K. , Horiuchi, T., Nitta, J., Tanaka, Y., Tada, T., Kobayashi, S., Barrow, D.L., Sekhar, L.N., Rak, R., Miyamoto, S., Hashimoto, N., Yamaura, A. (2003). Double-insurance bypass for internal carotid artery aneurysm surgery. *Neurosurgery*, 52, 3, March 2003, pp. (597-602), 0148-396X.
- Jafar, J.J., Russell, S.M., Woo, H.H. (2002). Treatment of giant intracranial aneurysms with saphenous vein extracranial-to-intracranial bypass grafting: indications, operative technique, and results in 29 patients. *Neurosurgery*. 51, 1, 2002, pp. (138-144), 0148-396X.
- Jahromi, B.S., Mocco, J., Bang, J.A., et al. (2008). Clinical and angiographic outcome after endovascular management of giant intracranial aneurysms. *Neurosurgery*, 63, 4, pp. (662-674), 0148-396X.

- Jiang, Y., Bu, B., Xu, B.-N., Sun, Z.-H., Jiang, J.-L., Zhou, D.-B., Yu, X.-G. (2009). Intraoperative neuroelectrophysiological monitoring of cerebral blood supply after parent-artery occlusion during intracranial aneurysm surgery. *Chinese Journal of Cerebrovascular Diseases*, 6, 11, November 2009, pp. (577-580).
- Johnston, S.C., Dudley, R.A., Gress, D.R., Ono, L. (1999). Surgical and endovascular treatment of unruptured cerebral aneurysms at university hospitals. *Neurology*, 52, 1999, pp. (1799–1805).
- Johnston, S.C., Gress, D.R., Kahn, J.G. (1999). Which unruptured cerebral aneurysms should be treated? A cost-utility analysis. *Neurology*, 1999, 52, pp. (1806–1815).
- Kai, Y., Hamada, J., Morioka, M., Yano, S., Mizuno, T., Kuroda, J., Todaka, T., Takeshima, H., Kuratsu, J. (2007). Treatment strategy for giant aneurysms in the cavernous portion of the internal carotid artery. *Surgical Neurology*, 67, 2, February 2007, pp. (148-155).
- Kapsalaki, E.Z., Lee, G.P., Robinson III, J.S., Grigorian, A.A., Fountas, K.N. (2008). The role of intraoperative micro-Doppler ultrasound in verifying proper clip placement in intracranial aneurysm surgery. *Journal of Clinical Neuroscience*, 15, 2, February 2008, pp. (153-157).
- Kassell, N.F., Torner, J.C., Jane, J.A., Haley, E.C. Jr, Adams, H.P. (1990). The International Cooperative Study on the Timing of Aneurysm Surgery. Part 2. Surgical results. *J Neurosurg*, 73, 1, pp. (37–47).
- Keravel, Y. (1988). *Giant Intracranial Aneurysms*, (1ed), Springer-Verlag, 3540181318, Berlin.
- Kivisaari, R.P., Porras, M., Ohman, J., Siironen, J., Ishii, K., Hernesniemi, J. (2004). Routine cerebral angiography after surgery for saccular aneurysms: Is it worth it? *Neurosurgery*, 55, 5, November 2004, pp. (1015-1022), 0148-396X.
- Klopfenstein, J.D., Spetzler, R.F., Kim, L.J., Feiz-Erfan, I., Han, P.P., Zabramski, J.M., Porter, R.W., Albuquerque, F.C., McDougall, C.G., Fiorella, D.J. (2004). Comparison of routine and selective use of intraoperative angiography during aneurysm surgery: A prospective assessment. *Journal of Neurosurgery*, 100, 2, February 2004, pp. (230-235), .
- Kodama, N., Suzuki, J. (1982). Surgical treatment of giant aneurysms. *Neurosurg Rev*, 5, 1982, pp. (155), .
- Krings, T., Piske, R.L., Lasjaunias, P.L. (2005). Intracranial arterial aneurysm vasculopathies: targeting the outer vessel wall. *Neuroradiology*, 47, pp. (931–937).
- Krisht, A.F., Kadri, P.A.S. (2005). Surgical clipping of complex basilar apex aneurysms: A strategy for successful outcome using the pretemporal transzygomatic transcavernous approach. *Neurosurgery*, 56, 4 Suppl., April 2005, pp. (261-272), 0148-396X.
- Lawton, M.T. (2002). Basilar apex aneurysms: surgical results and perspectives from an initial experience. *Neurosurgery*, 50, 1, 2002, pp. (1-8), 0148-396X.
- Lemole, G.M., Henn, J., Spetzler, R.F., Riina, H.A. (2000). Surgical management of giant aneurysms. *Operative Techniques in Neurosurgery*, 3, 4, 2000, pp. (239-254), .
- LeRoux, PD. (2003). *Management of Cerebral Aneurysms*, (1ed), W.B. Saunders, 0721687547, Pennsylvania.
- Lesley, W.S., Rangaswamy, R. (2009). Balloon test occlusion and endosurgical parent artery sacrifice for the evaluation and management of complex intracranial aneurysmal disease. *Journal of NeuroInterventional Surgery*, 1, 2, December 2009, pp. (112-120).

- Linskey, M.E., Sekhar, L.N., Hirsch Jr., W., Yonas, H., Horton, J.A. (1990). Aneurysms of the intracavernous carotid artery: Clinical presentation, radiographic features, and pathogenesis. *Neurosurgery*, 26, 1, 1990, pp. (71-79), 0148-396X.
- Lshikawa, T., Kamiyama, H., Kobayashi, N., Tanikawa, R., Takizawa, K., Kazumata, K. Experience from "double-insurance bypass." Surgical results and additional techniques to achieve complex aneurysm surgery in a safer manner. *Surgical Neurology*, 63, 5, May 2005, pp. (485-490).
- Lylyk, P., Miranda, C., Ceratto, R., et al. (2009). Curative endovascular reconstruction of cerebral aneurysms with the pipeline embolization device: the Buenos Aires experience. *Neurosurgery*, 64, 4, 2009, pp. (632-642), 0148-396X.
- MacDonald, J.D., Gyorke, A., Jacobs, J.M., Mohammad, S.F., Sunderland, P.M., Reichman, M.V., Mayberg, M.R., Selman, W.R. (1994). Acute phase vascular endothelial injury: A comparison of temporary arterial occlusion using an endovascular occlusive balloon catheter versus a temporary aneurysm clip in a pig model. *Neurosurgery*, 34, 5, 1994, pp. (876-881), 0148-396X.
- Minakawa, T., Koike, T., Fujii, Y., Ishii, R., Tanaka, R., Arai, H. (1987). Long term results of ruptured aneurysms treated by coating. *Neurosurgery*, 21, 1987, pp. (660-663), 0148-396X.
- Molyneux, A., Kerr, R., Stratton, I., et al. (2005). International Subarachnoid Aneurysm Trial (ISAT) of neurosurgical clipping versus endovascular coiling in 2143 patients with ruptured intracranial aneurysms: A randomised trial. *Lancet*, 366, 2005, pp. (809-817).
- Molyneux, A.J., Kerr, R.S., Birks, J., Ramzi, N., Yarnold, J., Sneade, M., Rischmiller, J. (2009). Risk of recurrent subarachnoid haemorrhage, death, or dependence and standardised mortality ratios after clipping or coiling of an intracranial aneurysm in the International Subarachnoid Aneurysm Trial (ISAT): long-term follow-up. *The Lancet Neurology*, 8, 5, May 2009, pp. (427-433).
- Murai, Y., Adachi, K., Takagi, R., Koketsu, K., Matano, F., Teramoto, A. (2011). Intraoperative Matas test using microscope-integrated intraoperative indocyanine green videoangiography with temporary unilateral occlusion of the A1 segment of the anterior cerebral artery. *World Neurosurg*, 76, 477, 2011, pp. (E7-477.E10).
- Patel, U. (2010). The development of a calculator to predict the risk of rupture of unruptured intracranial aneurysms@neurisk. *World Neurosurgery*, 73, 4, April 2010, pp. (231-233).
- Qureshi, A.I., Janardhan, V., Hanel, R.A., Lanzino. Comparison of endovascular and surgical treatments for intracranial aneurysms: an evidence-based review. *Lancet Neurology*, 6, 9, September 2007, pp. (816-825).
- Raabe, A., Nakaji, P., Beck, J., Kim, L.J., Hsu, F.P., Kamerman, J.D., Seifert, V., Spetzler, R.F. (2005). Prospective evaluation of surgical microscope-integrated intraoperative nearinfrared indocyanine green videoangiography during aneurysm surgery. *J Neurosurg*, 103, 2005, pp. (982-989).
- Raymond, J, Roy, D. (1997). Safety and efficacy of endovascular treatment of acutely ruptured aneurysms. *Neurosurgery*, 41, 1997, pp. (1235-1246), 0148-396X.

- Roos, E.J., Rinkel, G.J., Velthuis, B.K., et al. (2000). The relation between aneurysm size and outcome in patients with subarachnoid hemorrhage. *Neurology*, 54, 2000, pp. (2334-2336).
- Russell, S.M., Lin, K., Hahn, S.A., et al. (2003). Smaller cerebral aneurysms producing more extensive subarachnoid hemorrhage following rupture: a radiological investigation and discussion of theoretical determinants. *J Neurosurg*, 99, 2003, pp. (248-253).
- Salary, M., Quigley, M.R., Wilberger, J.E. (2007). Relation among aneurysm size, amount of subarachnoid blood, and clinical outcome. *J Neurosurg*, 107, 2007, pp. (13-17).
- Sano, H. (2010). Treatment of complex intracranial aneurysms of anterior circulation using multiple clips. *Acta Neurochir Suppl*, 107, 2010, pp. (27-31).
- Sharma, B.S., Gupta, A., Ahmad, F.U., Suri, A., Mehta, V.S. (2008). Surgical management of giant intracranial aneurysms. *Clin Neurol Neurosurg*, 110, 7, 2008, pp. (674–681).
- Shi, Z.S., Ziegler, J., Duckwiler, G.R., et al. (2009). Management of giant middle cerebral artery aneurysms with incorporated branches: partial endovascular coiling or combined extracranial-intracranial bypass—a team approach. *Neurosurgery*, 65, 6 Suppl., 2009, pp. (121-129).
- Sloniewski, P., Dzierzanowski, J., Szmuda, T. (2008). Modyfikacja dojścia pterionalnego do patologii dolu międzykonarowego – technika, morfometria i wyniki leczenia. *Neurol Neurochir Pol*, 42, 2 Suppl., 2008, pp. (160–168).
- Sluzewski, M., Menovsky, T., van Rooij, W.J., Wijnalda, D. (2003). Coiling of very large or giant cerebral aneurysms: long-term clinical and serial angiographic results. *Am J Neuroradiol*, 24, 2, 2003, pp. (257-262).
- Snyder, L.A., Spetzler, R.F. (2011). Current indications for indocyanine green angiography. *World Neurosurgery*, 76, 5, November 2011, pp. (405-406).
- Solenski, N.J., Haley Jr., E.C., Kassell, N.F., Kongable, G., Germanson, T., Truskowski, L., Torner, J.C., Spetzler, R.F., Selman, W.R., Warf, B., Barnett, G.H., Solomon, R.A., Friedman, A.H., Campbell, R.L., Horner, T., Nauta, H.J., Heros, R.C., Muizelaar, J.P., Mohr, G., et al. (1995). Medical complications of aneurysmal subarachnoid hemorrhage: A report of the multicenter, cooperative aneurysm study. *Critical Care Medicine*, 23, 6, 1995, pp. (1007-1017).
- Sughrue, M.E., Saloner, D., Rayz, V.L., Lawton, M.T. (2011). Giant intracranial aneurysms: Evolution of management in a contemporary surgical series. *Neurosurgery*, 69, 6, December 2011, pp. (1261-1270), 0148-396X.
- Szmuda, T., Sloniewski, P. (2011). Early and long-term outcome of surgically treated giant internal carotid artery aneurysms—comparison with smaller aneurysms. *Acta Neurochir*, 153, 2011, pp. (1611–1619).
- Szmuda, T., Sloniewski, P., Dzierzanowski, J., Rut, M. (2011). Predictors of postoperative mortality in ruptured aneurysms of internal carotid artery. *Neurologia i Neurochirurgia Polska*, 45, 6, 2011, pp. (543-555).
- Taylor, C.L., Steele, D., Kopitnik, T.A., Jr, et al. (2004). Outcome after subarachnoid hemorrhage from a very small aneurysm: a case control series. *J Neurosurg*, 100, 2004, pp. (623-625).
- Todd, N.V., Tocher, J.L., Jones, P.A., Miller, J.D. (1989). Outcome following aneurysm

- wrapping: A 10-year follow-up review of clipped and wrapped aneurysms. *J Neurosurg*, 70, 1989, pp. (841–846).
- Tulleken, C.A., Verdaasdonk, R.M. (1995). First clinical experience with Excimer assisted high flow bypass surgery of the brain. *Acta Neurochir (Wien)*, 134, 1995, pp. (66–70).
- van Rooij, W.J., Sluzewski, M., Slob, M.J., Rinkel, G.J. (2005). Predictive value of angiographic testing for tolerance to therapeutic occlusion of the carotid artery. *Am J Neuroradiol*, 26, 2005, 175-178.
- Weir, B. (1987). *Aneurysms Affecting the Nervous System*, (1ed), Williams & Wilkins, 0683089250, Baltimore.
- Wermer, M.J.H., Van Der Schaaf, I.C., Algra, A., Rinkel, G.J.E. (2007). Risk of rupture of unruptured intracranial aneurysms in relation to patient and aneurysm characteristics: An updated meta-analysis. *Stroke*, 38, 4, April 2007, pp. (1404-1410).
- Wester, K. (2009). Lessons learned by personal failures in aneurysm surgery: what went wrong, and why? *Acta Neurochir*, 151, 2009, pp. (1013–1024).
- Yasargil, MG. (1984). *Microneurosurgery vol 2*, (1ed), Georg Thieme Verlag Stuttgart, 9783136449011, New York.
- Youmans, JR. (1990). *Neurological Surgery*, (3ed), W.B. Saunders, 0721696554, Philadelphia.