Aneurysms:

- Classification of aneurysms: A. size(small<6mm, medium7-10 mm, large 11-24 mm and giant> 25 mm) B. Shape (saccular and fusiform) C. Etiology (atherosclerotic, dissecting, traumatic, mycotic)

- Genetics of intracranial aneurysms: The etiology and pathogenesis of intracranial aneurysms remain unclear. There is evidence supporting acquired factors such as HTN and smoking. There is also evidence supporting the role of genetic factors A. Association of aneurysms with heritable connective tissue disorders B. Familial occurrence of intracranial aneurysm.
- Heritable connective tissue disorders associated with aneurysms:

1. Autosomal dominant polycystic kidney disease: 1/1000, autosome dominant with complete penetrance and variable expressivity, 20% spontaneous mutations, 85% due to mutations in PKD1 gene on chromosome 16, 15% due to PKD2 mutations on chromosome 4, characterised by multiple cysts in the kidneys, liver, pancreas, spleen, seminal vesicles. Aneurysms are present in 25%. Other neurosurgical manifestations include carotid and vertebral dissections, arachnoid cysts, spinal meningeal diverticula, intracranial dolichoectasia. Screening patients with ADPKD remains controversial, but should be strongly considered in those with family history of aneurysms.
2. Ehler-Danlos syndrome type 4: 1/50000-500000 , autosome dominant, 50% spontaneous mutations, mutations of the gene encoding for type III collagen on chromosome 2, characterised by facial features(lobless ears, thin nose , large eyes), fragile transparent skin, joint hypermobility usually mild . Neurosurgical manifestations include aneurysms, spontaneous dissections and rupture of large and medium sized arteries, carotico-cavernous fistulas. Screening for asymptomatic aneurysms is not recommended because treatment options are limited and treatment is associated with high MM.
3. Alpha 1 antirypsin deficiency: antitrypsin inhibits proteases that degrade extracellular matrix collagen and elastin. Characterised by emphysema, thin skin (cutis laxa), increased incidence of aneurysms, carotid dissections. Screening for asymptomatic patients is not advocated.
4. NF1: 1/3000, autosome dominant, mutations of NF1 gene on chromosome 17 which encodes for neurofibromin (tumour suppressor gene product and may play a role in connective tissue regulation) , characterised by café-au-lait spots, lisch nodules, fibromas, optic gliomas, sphenoidal and long bone fibrodysplasia (look tumour section). Cerebrovascular manifestations include Moyamoya syndrome, aneurysm and fistula formation. Screening is not advocated.
5. Marfan's syndrome: 1/10000, mutations of the gene encoding fibrillin protein. (Important to maintain the integrity of elastin). Mitral and aortic insufficiency is the most common cause of death in children, carotid dissection in adults, characterised by skeletal manifestations, tall stature, arachnodactyly, lens subluxation. Neurosurgical manifestations include ectasia and tortuosity of

carotid and vertebral arteries which renders endovascular treatment difficult. Increased frequency of aneurysms and dissection.

6. Pseudoxanthoma elasticum: disorder affecting elastic fibres of the skin, ocular and cardiovascular system. Increased incidence of aneurysms

- 7-20% of patients with aneurysms have first or second degree relative with aneurysm which is more than expected from the 1% prevalence of aneurysms in the population. The inheritance pattern is unknown with some pedigrees supporting autosome dominant pattern while others support autosome recessive pattern. Familial aneurysms rupture at earlier age; rupture at smaller size, more incidence of de novo aneurysms and an increased proportion have multiple aneurysms. Screening for asymptomatic aneurysms is indicated in those with two or more first or second degree relatives with aneurysms. The yield is 10%. There is no particular age at which screening should be started (> 30 years). To detect de novo aneurysms repeat screening 5 years later is advised. Patients diagnosed with familial aneurysms should be referred to medical genecist and be assessed for the above mentioned syndromes (NF1, Ehler-Danlos etc..).

The natural history of saccular aneurysms:

- Natural history of any disease is defined as the outcome of the disease in the absence of intervention. The natural history of intact and ruptured aneurysms is different. Knowledge of the natural history is very important.
- The prevalence of aneurysms from autopsy and radiological studies (the number of people affected in a population divided by the number of people in that population at a particular time) ranges 0.2-9% and the most frequently quoted prevalence of 5% reported by Heros and Sekhar must be interpreted with caution (selection bias).
- The incidence of SAH is 10-11/100000 population per year with higher incidence in the Finnish and Japanese.
- The natural history of UIA is not perfectly defined. The annual risk of haemorrhage in most published studies is 1-2% (Winn-1%, Juvula-1.3%, Rinkel-.3%).
- **ISHUA** study (international study of UIA) is the largest Multicenter study. It has two arms. This is a landmark study that has influenced the thinking about the natural history of UIA

1. Retrospective arm which looked at the natural history of UIA. In this arm 1449 patients divided into 2 groups: Group 1-had no prior history of SAH and Group 2- had history of SAH from other aneurysm. The annual risk of haemorrhage for group 1 was 0.05% for aneurysms<10mm, 1%for those larger than 10mm and 6% for giant aneurysms (.25mm). In the second group the risk was 0.5% for those <10mm and 1% for those >10mm. In this study in addition to **size**, **location** was related to haemorrhage risk, with higher risk of haemorrhage for posterior circulation aneurysms.
2. In the prospective component the morbidity and mortality related to treatment were evaluated.

The **strengths** of this study include its **Multicenter** design which minimizes referral and treatment bias and its **size** which provides strong statistical power. The **most significant criticism** is related to the possibility of **selection bias** (all patients were selected for observation or surgery after consultation with a neurosurgeon, this may have created a subgroup of patients with less risk of rupture by removing the patients with high risk from the group 'those who had surgery". Removal of these high risk patients skewed downward the risk of rupture. The **retrospective nature** of this study might have introduced unrecognised selection bias and another concern is the **inclusion of patients with intracavernous aneurysms in the study population** (although investigators after excluding intracavernous aneurysms from the analysis concluded that the risk of haemorrhage was 0.066% for small and 1.38% for large aneurysms). Winn et al demonstrated that the rupture rate of 0.05%-0.066% is in conflict with the prevalence rate of UIA and the incidence of aneurysmal SAH (30000 SAH per year in North America). Using the figures provided by ISHUA study, one would expect that the prevalence of UIA in USA will be 16% for those older than 30 years, which is not supported by the prevalence of UIA from MRI/MRA studies.

- **Unruptured intracranial aneurysms: natural history, clinical outcome and risks of surgical and endovascular treatment: Weibers et al (Mayo clinic) 2003**.

1. Data from the **prospective arm of ISUIA study**: 4060 patients. **Group1** –no history of SAH (**1692**) and **group 2** had clipping of ruptured aneurysm (**1917**) and coiling (**451**).Average follow up 4.1 years (5 years or more in 50%). The aims of this study were to assess the natural history of unruptured aneurysms and to assess the risk of surgical or endovascular intervention.

2. The 5 year cumulative rupture rates for (ICA,ACA,A-com and MCA) were < 7mm (0), 7-12 mm (2.6%), 13-24 mm (14.5% and >25mm (40%).
3. The 5 year cumulative rupture rates for (posterior circulation and p-com aneurysms) were <7mm (2.5%), 7-12mm (14.5%), 13-24mm (18.5%) and >25 mm 50%.
4. This data indicates that aneurysm **size** and **location** are significant factors in determining the risk of future rupture
5. The **total mortality and morbidity** in the surgical group was 12% in group1 and 10% in group 2. Factors associated with increased risk were age>50, large size, posterior circulation aneurysms and CVD.
6. The total mortality morbidity rates for endovascular treated patients were 9.1% (group1) and 9.5 %(group 2). The M and M were less dependent on age in this group
7. The risk of surgical or endovascular treatment equalled or exceeded the 5 year cumulative rate of rupture

The lowest risk natural history group were asymptomatic patients with aneurysm < 7mm in the anterior circulation. The lowest surgical risk patients were < 50 years with aneurysm < 24 mm in the anterior circulation (5-6% at

- The risk of rupture of UIA may be affected by the following factors:

1. **Size**: ISHUA study 0.05-0.066%-0.5% for those < 10mm, 1% for those >10mm and 6% for giant aneurysms.
2. **Location** : higher risk of rupture for basilar bifurcation, p-com and A-com aneurysms
3. **Multiplicity**: 15-20% of aneurysms are multiple. The risk of rupture is higher if the aneurysm is part of multiple aneurysms. The Japanese data 6.5% for multiple aneurysms Vs 1.9% for single lesions.
4. **History** of SAH from another aneurysm (ISUA-0.05% VS. 0.5% for aneurysms < 10 mm.
5. **Smoking**: strong association between smoking and aneurysmal rupture (Juvula et al
6. Aneurysm growth: controversial data in literature regarding whether growth of aneurysm on serial imaging increases the risk of rupture
7. Symptomatic aneurysms: headaches, cranial nerve palsies are symptoms of aneurysms other than SAH. Some studies suggested increased risk of haemorrhage from these aneurysms, others did not find a relation between symptomatic aneurysms and the risk of rupture
8. Age; most literature supports the concept of increasing age being associated with increased risk of rupture except in the very elderly.
9. Hypertension: most data are unclear about whether hypertension increases the risk of haemorrhage from UIA.

10. Gender: women may have an increased risk of rupture, but the data are inconclusive.

- Based on the available data treatment of UIA is indicated in patients with history of SAH, with symptomatic aneurysms, with aneurysms> 10mm, with aneurysms 6-10mm in young people and for aneurysms <6mm which show growth on serial Neuroimaging.
- The risk of rebleeding after aneurysm rupture is 4% in the first day and 1-1-2% per day in the following 4 weeks, approximately 25% at 2 weeks and 50% at 6 weeks. 1/3 of patients with aneurysmal SAH die before reaching the hospital and 50-70% die from the second haemorrhage.
- Early surgery (in the first 3 days) eradicates the risk of rebleeding, facilitates the use of haemodynamic therapy for vasospasm and early mobilisation. International cooperative study on timing of surgery (prospective non randomised study) found that early surgery significantly improved the outcome.(70% of patients undergoing surgery 0-3 days had good outcome , while 62% of those undergoing surgery after 14 days had good outcome). **20-40%** of patients with SAH are grade 4 and 5. The outcome in this group is poor with mortality of 50% in the first 48 hours and 30-40% poor clinical outcome. **There are two schools in managing high grade SAH. The first school resuscitate patients and clip or coil the aneurysm if they improve to the point of localising pain. The other**

school treat all patients aggressively with EVD, fluids, and early clipping or coiling followed by aggressive treatment of the vasospasm. Patients with high grades are at higher risk of rebleeding and vasospasm and aggressive treatment may provide the patients with the best chance of recovery. On the other hand it may result in larger number of patients in the vegetative state. Delayed treatment may be appropriate for giant complex aneurysms that require further evaluation of CBF and collateral flow in case bypass graft of hypothermic cardiac arrest are necessary.

- ICH increases the mortality from SAH. Early evacuation of the ICH with clipping of the aneurysm may be associated with improved outcome. CTA will detect 90% of aneurysms larger than 5 mm.
- Acute hydrocephalus develops in 20% of patients with SAH. The incidence is higher in patients with IVH, in patients with large SAH and in poor grade patients. Hydrocephalus can be due to obstruction of the aqueduct, outlet of 3d ventricle, basal cisterns or arachnoid villi, EVD should be inserted and CSF drained to ICP of 25 mmHg. EVD drainage to lower pressure increases the risk of aneurysm rupture (alters transmural pressure). Attempts to remove the casted blood through urokinase infusion does not appear to improve the outcome. Chronic hydrocephalus develops in 10-20% and is due to sclerosis of arachnoid villi.
- Paediatric aneurysms are rare. Posterior circulation, giant, traumatic and infectious aneurysms are more common than in adults. In theatre avoid hypothermia and rupture (low blood volume 80ml/kg)
- Infectious aneurysms account for 2-6% of all aneurysms an often associated with bacterial endocarditis or immunological compromise. Streptococcus and staphylococcus are the most common organisms, rarely can be due to fungal infection (Candida and aspergillosis). Usually located on distal branches of MCA. These aneurysms should be treated with antibiotics for 6 weeks and serial angiography. 30-50% resolves with conservative treatment. Surgery is indicated if the aneurysm ruptures or is associated with an abscess or fails to resolve with conservative treatment. Surgeon should be prepared for aneurysm excision and ligation of the parent artery with or without bypass grafting.
- Traumatic aneurysms account for 1% of all aneurysms. They are false aneurysms and most commonly complicate penetrating injuries and localised to MCA territory. Aneurysms complicating base of skull fractures are localised to ICA .Usually develop 2-3 weeks after injury. They don't have a neck and treatment is by excision, ligation of parent artery or trapping with or without bypass.
- Giant aneurysms account for 5% of all aneurysms. Untreated, the 2 year mortality is 60-100%. Treatment goals are exclusion of the aneurysm from circulation and relief of mass effect. Procedure related mortality is 5-15%. Surgical options include clipping, trapping, proximal vessel ligation with or without bypass and aneurysmorrhaphy. These cases should be treated at tertiary referral centres by experienced neurovascular surgeons and in institution where hypothermic cardiac arrest can be done. Technical considerations include wide exposure (skull base approaches including tranlabyrinthine, transpetrosal etc...), proximal and distal control and clip reconstruction. Orbit zygomatic approach is suitable for anterior circulation and upper 2/5 of basilar artery, temporal approaches for middle

1/5 of basilar artery and the far lateral approach for lower 2/5 of basilar artery.

- What would you do if you find a residual aneurysm on postoperative angiogram?. Residual aneurysms can thrombose or enlarge. The risk of haemorrhage is 0.5%. Small dog ear residual aneurysms can be observed with serial angiograms, CTA or MRA particularly if the surgeon knows that he could not improve on the position of the clip. Hypertension should be treated and patient should be strongly advised to stop smoking. Broad based residual aneurysms have a high chance of enlargement and should be repaired.
- **When should patients with SAH be allowed to die without intervention?**

This question often arises when an elderly patient presents with WFNS grade 4 and 5 SAH which is believed to be associated with poor prognosis (50% mortality and 30-40% poor outcome "international cooperative study on timing of aneurysm clipping". There are two approaches to such patients

1. **The Universal approach**: all patients with high grade SAH regardless the ages are treated aggressively with early (within 24 hours) or ultra early (within 6hours) clipping or coiling. The rationale behind this approach is that the risk of rebleeding is higher in patients with high grade and early occlusion of the aneurysm allows aggressive treatment of vasospasm. The concern with this approach is the possibility of creating a group of vegetative, dependent patients. This approach is requiring operating after hours in suboptimal conditions and with unexperienced staff.

There is a growing body of published evidence that provides a strong rationale for the aggressive surgical or endovascular management of these patients[1, 2, 3]. Laidlaw et al reported their experience with of 132 patients with grade 4, 5 SAH (20% > 70 years old) who were clipped within 12 hours of presentation. At 3 months 40% were independent, 45% died and 15% were dependent. In this series elderly patients had similar outcome to younger ones. (In this series the grading of the patients was done on admission before the resuscitation and insertion of EVD which may result in including patients who are not truly high grade).

2. **Traditional selective approach**: Patients with high grade SAH are resuscitated (ventilation, fluids) and EVD is inserted for 24-48 hours after that sedation is stopped and patients are assessed neurologically. If they improve to grade 3 (best motor response 4), they undergo angiography and clipping or coiling. This approach subjects patients with high grade to the risk of rebleeding with associated mortality and morbidity. On the other hand it can potentially decrease the number of dependent vegetative patients.

To answer the question

Whether one choses the first or second approach at least resuscitation and insertion of EVD should be done in almost all patients(intervention) and only in exceptional cases when patient presents with GCS of 3 and fixed dilated pupils and the scan shows large ICH with midline shift, particularly in elderly patient one could consider doing nothing.

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Aneurysms of proximal ICA:

- Aneurysms of cav. ICA, Clinoidal ICA and ophthalmic segment are grouped together because they have much in common. These aneurysms are more common in females F:M 9:1, often multiple 30-50% and often large > 1cm 50% for Clinoidal and 75% for ophthalmic segment.
- Patients should be investigated by CT scan (SAH, calcification, ACP erosions), MRI (thrombosis and relation to soft tissue), cerebral angiogram including ECA to assess STA (potential fro EC-IC bypass, balloon test occlusion with hypotensive challenge and assessment of CBF using xenon CT, SPECT or PET. Intolerance to balloon occlusion and or drop in BF< 35ml/100gr/min are indications for bypass graft using saphenous vein (high flow) or STA
- Cavernous segment aneurysms account for 5% of intracranial aneurysms and 15% of ICA aneurysms. The risk of SAH is very low except for large aneurysms eroding the dura. They may be diagnosed incidentally. If large they can cause CS syndrome +pain and numbness of the face and retroorbital pain) and CC fistula if they rupture. Rarely can they erode into SA space or into sphenoid sinus and present with SAH or epistaxis .Asymptomatic regardless the size or mildly symptomatic in elderly can be treated conservatively. Progressive ophthalmoplegia, intractable pain, SAH and epistaxis require treatment. For aneurysms at or distal to anterior genu endovascular treatment and direct clipping are valid options. Large complex aneurysms and those of proximal CS segment require indirect treatment (trapping or proximal ligation with /without bypass). Surgical access to these aneurysms is through Parkinson's triangle or anteromedial triangle and requires exploration of ICA in the neck for proximal control and drilling of ACP forming the roof of the anterior part of the sinus and division of falciform lig and dural ring.
- Clinoidal aneurysms are rare. There are 2 variants

1. Anteriolateral extends superiolaterally and can erode ACP.. Can be mistaken fro O aneurysm (proximal to OA, double density on angio).
2. Medial variant extends medially under the chiasm and can cause hypopituitarism. Occassionally can erode the sphenoid sinus and cause epistaxis. It can be mistaken for superior hypophyseal aneurysm 9narrow neck, proximal to OA). Both aneurysms have low risk of SAH unless they are > 1 cm and erode the dura.

Small asymptomatic aneurysms are observed. Symptomatic regardless the size should be treated. Coiling or clipping. (Cervical ICA exposure for proximal

control, drilling of ACP (avoid extradural drilling in case of anteriolateral variant because the aneurysm tends to erode the ACP increasing the risk of iatrogenic rupture, in placing the clip avoid injuring the OA and SHA. Giant aneurysms require temporary clipping with barbiturate EEG burst suppression and hypothermia, suction decompression, thrombectomy and rarely hypothermic circulatory arrest)

- Ophthalmic segment aneurysms: Account for 10% of intracranial aneurysms. 10% are bilateral. 50% part of multiple aneurysms and 75% >1 cm. More common in females. Can present with SAH, visual symptoms, headaches and incidentally. There are 3 types of aneurysms arising in the ophthalmic segment.

1. Ophthalmic artery aneurysms arising from the dorsomedial surface of ICA just distal to the origin of OA and project superiomediallly. They push the optic nerve superiorly against the falciparum lig causing initially monocular loss of ipsilateral inferior nasal fields and eventually loss of vision on ipsilateral eye
2. Superior hypophyseal artery aneurysms: arise from the inferomedial surface of ICA. There are 2 subtypes A. Parasellar: extend medially into the carotid cave (present in 70% of anatomic specimens and located posteromedial to dural ring and bounded by sphenoid bone (carotid sulcus medially and ICA laterally. Three variants has been described (slit, pocket and mesh). These aneurysms can mimic pituitary adenoma both clinically and radiologically

B. Suprasellar: extend superiorly into the suprasellar cistern.

3. Dorsal type: arises from ophthalmic segment 2-4 mm distal to OA.. These aneurysms allow exposure without drilling the ACP and proximal control can be achieved by temporal clipping of supraclinoid ICA.

Small asymptomatic aneurysms can be observed. Symptomatic and large aneurysms should be treated either end vascularly or by clipping or trapping with /without bypass (the distal occlusion point should be proximal to p-com.).

- Complications peculiar to this group of aneurysms include 1. ICA stenosis and thrombosis due to clip kinking the parent artery (intraoperative angiogram, Doppler)

2. Injury to optic nerve /chiasm by retraction or clip
3. III, IV, VI paresis usually transient (in case of Clinoidal and cavernous aneurysm.

- Giant calcified aneurysms may require temporary clipping/trapping with barbiturate EEG burst suppression, circulatory arrest, thrombectomy, suction deflation , booster clips, reconstruction of ICA , fenestrated clips etc and should be treated at centres and by surgeons experienced in using these techniques.
- If you get examination case with complex giant aneurysm: Although I am aware of the available techniques and strategies in managing such aneurysm, I have no experience in clipping complex aneurysms If II work at a centre where an experienced vascular surgeon works I will ask him to give me a hand

and do it with me otherwise I will refer the patient to a centre and surgeon capable of dealing with complex aneurysms.

Aneurysms of intracranial ICA:

- Posterior communicating artery aneurysms account for 35% of intracranial aneurysms. F: M 2:1. They present with SAH, 3-d nerve palsy 30%, seizures from mesial temporal lobe irritation (laterally projecting), intracerebral haemorrhage (uncus) and TIA's. The aneurysm fundus can project inferiorly and cause compression of the 3-d nerve or laterally into the temporal lobe. During clipping one should avoid retraction on temporal lobe. Clipping can be achieved using straight or fenestrated clip with blades parallel to ICA. One should assess the vertebral angiogram preoperatively to exclude fetal type P-com. One should visualise anterior choroidal, p-com and the thalamic perforators before clipping which is facilitated by sylvian fissure dissection.

- Anterior choroidal aneurysms are rare and difficult to differentiate from P-com aneurysms. In 30 % of cases the anterior choroidal artery is double. The key to safe clipping is the identification and preservation of this artery. They can present with SAH in the ambient and suprasellar cisterns similar to P-COM aneurysms.

- Carotid bifurcation aneurysms account for 15% of all intracranial aneurysms. They can project 1. Superiorly and then the position for surgical clipping is similar to A-com aneurysms (60 degrees tilt) and one should avoid initial frontal retraction until the neck is visualised or 2. Posteriorly and then the position will be similar to MCA aneurysm (45 degrees tilt) and one should avoid initial temporal retraction. These aneurysms are located under the anterior perforated substance and can be draped by the perforators (recurrent artery of Heubner, medial lenticulostriate (MCA) and ACA. ICA and A – choroidal branches)posterior and medial to the neck and these should be identified and protected. Wide splitting of the fissure is very important. Giant aneurysms may require temporary clipping and hypothermia, circulatory arrest, thrombendartectomy and occasionally trapping with/without bypass.

The advised protocol for assessing the need for bypass graft:

- Balloon test occlusion for 30 min with hypotensive challenge. If the patient tolerate the test assessment of CBF using xenon CT or SPECT. If CBF drops below 35ml/100g/min or patient fails the balloon occlusion test, bypass graft should be performed before ICA ligation.
- Types of bypass:

Anterior communicating artery segments (Rhoton):

1. A1-from ICA bifurcation to A-com passes medially and anteriorly above chiasm (70%) and optic nerve (30% of cases). A1 diameter is 2.6 mm and length 13 mm on average. One A1 is hypoplastic in 10% of normal brains and in 26-85% of patients with A-com aneurysms. Duplication of A1 is found in 2% with the accessory artery originating from ICA. The A1 gives on average 8 perforators (medial thalamostriate arteries). Most of the perforators arise from the superior (54%) and posterior (32%) surfaces of A1. 41% terminate in the anterior perforating substance. The remaining end in hypothalamus, basal frontal lobe, optic chiasm. In 14% of cases it gives origin to artery of Heubner A-com artery can be double in 30% and triple in 10%. This can be explained by embryology as a-com develops from multiple vascular channels connecting the primitive ACA at 44 days of intrauterine life. A-com gives 3 perforators from its superior and posterior surface to optic chiasm and hypothalamus.

2. A2 –from a-com to the junction of the rostrum with genu of corpus callosum. It ascends anterior to lamina terminalis. It gives the following perforators
    a. The most important perforator is recurrent artery of Heubner (1mm diameter, 23 mm long). It originates from A1-14%, A2-78% and a-com-8%.The majority arise within 4 mm of a-com. It courses anterior to A1 in 60% and superior to it in 40%. It supplies the caudate nucleus, anterior limb of internal capsule, putamen and anterior portion of globus pallidus. Injury to this artery can result in hemiparesis involving mainly the arm and face.
    b. Orbito frontal: runs across rectus gyrus and olfactory tract.
    c. Frontopolar: courses anteriorly along the medial surface of the frontal lobe. It can arise from A3.
    d. 5 basal perforators to optic chiasm, anterior hypothalamus, fornix and anterior-inferior portion of striatum.

3. A3-from A2 curves along the genu and ends at the point where the artery turns posteriorly into callosal cistern. It gives origin to callosomarginal (absent in 18%.
4. A4-runs in the callosal cistern to the level of coronal suture
5. A5-in the callosal cistern beyond the coronal suture. A4 and A5 segments receive collaterals from MCA and posterior pericallosal artery from PCA.

A2-A5 is collectively called distal ACA. They give two groups of perforators
    a. Central or basal to CC, SP and anterior hypothalamus (5,3,3,3)
    b. Cortical (orbit frontal, Frontopolar, anterior, middle and posterior internal frontal arteries, paracentral artery and superior and inferior parietal arteries.

ACA and A –com aneurysms:

- These account for 39% of all intracranial aneurysms (international cooperative study on timing of clipping 1983).The most common presentation is SAH. The bleeding pattern is characteristic with blood located in interhemispheric fissure or rectus gyrus. These aneurysms have the highest false –ve rate on cerebral

angiograms (balanced flow from both A1). Carotid cross compression should be done routinely to fill the A-COM complex.

- These aneurysms can project

1. superiorly into the interhemispheric fissure (most common): little risk of rupture during frontal retraction , high risk of hypothalamic and infundibular perforators injury which are closely related to the posterior wall of the neck
2. Inferiorly (the least common). The fundus is adherent to optic apparatus , high risk of rupture if traction is applied to frontal lobe , less risk of perforator injury
3. Anteriorly: intermediate risk of rupture due to frontal retraction. Away from the direction of the perforators. (The easiest to clip)
4. Posterior (the most difficult to clip). Closely related to hypothalamic perforators which can run on the superior or inferior wall of the neck.

- Surgical tips : position supine with head tilted to the apposite site 45-60%, head elevated and neck extended so malar eminence is the highest point. Infiltrate the pins site by local anaesthetic and ask the anaesthetist to give a bolus of analgesics to avoid hypertensive induced premature rupture. Abs, ACs, Brain relaxation (mannitol 0.5g-1g/kg, moderate hyperventilation to bring PCOS -30-35, CSF drainage), pterional craniotomy with drilling of sphenoid wings or orbitozygomatic osteotomy. Avoid frontal lobe retraction in inferiorly and anteriorly directed aneurysms, open carotid cistern, dissect on the superior surface of ICA to the bifurcation, Proximal control by exposing both A1(difficult in inferiorly directed aneurysms). Subpial rectus gyrus resection, before clipping identify (A1x2, A2 X2, FO X2, FP X2, Heubner A X2, A-com and perforators. Dissection should be along the **anterior and inferior surface of the A1** to avoid the perforators. **The length of the straight** clip should be **1.5 times the diameter** (½ the circumference of the aneurysm = r x 3.14. Use temporary clipping as needed with moderate hypothermia and barbiturate induced EEG burst suppression. After clipping puncture the aneurysm to confirm complete clipping.
- Peculiar complications of A-com aneurysms are

1. Electrolyte disturbance: hyponatriemia-40%, hypernatremia-6%.
2. Cognitive dysfunction (Acom A syndrome): poor STM, personality changes and confabulation.

- Distal ACA aneurysms are aneurysms distal to A-com segment (A2-A5).They account for **5% of intracranial aneurysms**. The majority are located in the region of the **genu of CC (81**%). The most common location is the origin of **callosomarginal** and the second most common location is the origin of **Frontopo**lar. 37% are associated with other aneurysms and **67% of ruptured aneurysms are < 5mm in diameter**. The distal ACA is a common site for infectious (5%) and traumatic aneurysms (34%). (MCA -39% and 36% respectively). 80% present with SAH, 20% incidental. The haematoma is located in interhemispheric fissure. Some reported cases of parafalcine convexity SDH. The majority aneurysms are **approached through right parasagittal precoronal frontal craniotomy and interhemispheric approach**. Those arising from proximally can be approached through pterional approach. A4, A5 segment aneurysm are approached through

interhemispheric approach with more posterior craniotomy. Technically clipping of these aneurysms can be difficult because the callosal and interhemispheric cisterns are narrow, the cingulate gyri can be stuck together mimicking CC, it may be difficult to ascertain preoperatively which pericallosal artery gives origin to the aneurysm, the dome can buried in cingulate gyrus and retraction fro exposure can cause premature rupture, difficult to apply temporary clip because of the limited space. The average reported **perioperative mortality 7%** (0%-14%) and the major morbidity 9.7% (0%-17%)

- Results of clipping of A –com aneurysms.

1. Before 1970 the mortality was high (36%-internmational cooperative study of intracranial aneurysms and SAH-1958-1965)
2. Yasargil series: operative mortality 5.9% (selective group mostly low grade)
3. International study of intracranial aneurysms and the timing for surgery (1983). Surgical mortality 16.8% and overall management mortality 30% fro all grades

MCA aneurysms:

- 20-40% of all aneurysms. 15% occur along M1 segment, 80% at the bifurcation AND 4% DISTAL (M2-M4). 10% of MCA aneurysms are giant and 10% of giant aneurysms are MCA aneurysms. 50% of traumatic aneurysms are within MCA (M2-M4) territory and 36% of mycotic aneurysms are MCA aneurysms. Multiple in 39%, bilateral in 20% and mirror image in 10%.
- They present with SAH, ICH (41% comparing to 11% of other aneurysms. Hence the high percentage of high grade SAH), seizures (mesial temporal lobe pressure and distal embolisation and cortical infarctions. For grade 1,2 early operation is associated with better outcome.
- They can be approached through :

1. Lateral transsylvian approach (Yasargil): for unruptured and ruptured small uncomplicated aneurysms of the bifurcation particularly if M1 is long. Dissection starts 3cm lateral to the sphenoid wing. Disadvantages –aneurysm is exposed before proximal control
2. Medial transsylvian approach: for M1 aneurysms and complex large aneurysms .It allows early proximal control and CSF drainage from optic and carotid cisterns. Disadvantage –brain retraction.
3. Superior temporal gyrus approach (Heros): through superior temporal gyrus: advantage _drainage of ICH and minimal retraction. Disadvantage- aneurysm is exposed before proximal control and possibly increased risk of epilepsy.
4. Combination of 1 and 2.

- Complex large aneurysms may require temporary clipping with barbiturate EEG burst suppression (**10-15 min intermittent clipping**), thrombendartectomy and vessel reconstruction, trapping with EC-IC bypass. Mycotic and traumatic aneurysms are hard to clip because of the fragile wall and often require excision with bypass grafting. During dissection of MCA aneurysms always look for and protect the lenticulostriate perforators, injury of which can result in severe neurological deficit (internal capsule, basal ganglia and thalamus).

- Mycotic and traumatic aneurysms

Surgical approaches to posterior circulation aneurysms:

- 15% of intracranial aneurysms. Most commonly occur at basilar apex followed by the origin of SCA followed by PICA aneurysms.
- Approaches to basilar tip, PCA and SCA and distal basilar trunk:

1. Subtemporal (Drake): advantages- good visualisation of the important posterior perforators. Disadvantages-excessive retraction of temporal lobe, poor visualisation of contralateral PCA, SCA and 3-d nerve, ipsilateral 3-d nerve in the middle of the field. Division of the tent provides wider exposure of lower lying bifurcation, but increases the risk of 4^{th} cranial nerve injury. Approach is usually from the right side to avoid injuring the dominant temporal lobe except in cases of left 3-d palsy and right hemiparesis. Protection of vein of Labbe is of paramount importance. Difficult exposure in high grade SAH and some surgeons resect inferior temporal and parahippocampal gyri.
2. Pterional transsylvian (Yasargil): advantages- good visualisation of ipsilateral and contralateral PCA, SCA, and third nerve. Disadvantages- poor visualisation of posterior perforators. Difficult to clip posteriorly and anteriorly pointing aneurysms and poor visualisation of low basilar apex (>1cm from the dorsum sellae). Wide dissection of sylvian fissure to avoid kinking of M1. Division of the veins draining temporal pole into sphenoparietal sinus. The P-com artery is followed down to P1/P2 junction. Dissection should be a long the inferior surface of P-com and P1 to prevent injury to anterior and posterior thalamoperforators.
3. Orbitozygomatic approach: provides better trajectory and angle of exposure for high or low basilar bifurcation (in 90% of cases basilar bifurcation is within 1 cm of dorsum sellae), decreases the degree of temporal lobe retraction.
4. Extended orbitozygomatic: similar to the above, but the craniotomy involves taking temporal bone. This approach is a combination of 2 and 3 and provides good visualisation of ipsilateral and contralateral PCA,SCA and 3-d nerves as well good visualisation of posterior perforators and good angle to look at low and high bifurcations and is the approach of choice for these aneurysms
5. PAVEL : combination of transsylvian and subtemporal approaches

- For revascularisation the recipient artery is SCA or PCA. The donor artery can be EC (STA-low flow or ECA-high flow) or IC PCA-SCA

Exposure of low lying aneurysms can be enhanced by **drilling the posterior clinoid, retraction of the tent, incision of the tentorium (a. transtentorial retrocaverous behind the entry of 4^{th} nerve or B. transtentorial transcavernous between 3-d and 4^{th} nerves) or by medial petrosectromy (drilling the triangle of Kawase)**

- Approaches to basilar trunk aneurysms:

1. Presigmoid retrolabyrinthine approach: mastoidectomy and drilling of the petrous bone with preservation of semilunar canals and facial nerve gives good access to CP angle and may be suitable for small aneurysms and in

conjunction with subtemporal approach as part of combined approach. The exposure can be increased by incising the sigmoid sinus.

2. Translabyrinthine approach: all semicircular canals are drilled away and the facial nerve skeletonised (loss of hearing). CPA, anterolateral brainstem and inferior clivus are better visualised.
3. Transcochlear approach: the IAM and cochlea are drilled and 7th nerve retracted. Gives good exposure of anterolateral brainstem, clivus and basilar trunk.
4. Combined supra and infratemporal approach: Transcochlear approach with division of sigmoid sinus and opening posterior fossa and middle fossa dura and incision of the tentorium provides excellent exposure of anterior brain stem, inferior clivus and basilar trunk.
5. Transoral approach carries high risk of CSF leak and meningitis and is replaced by the above approaches.

- For revascularisation the recipient artery is **AICA**. The donor can be EC (OA-low flow or ECA-high flow) or IA (PICA-AICA)
- Approaches to vertebral trunk , vertebrobasilar junction and proximal trunk:

1. **Midline suboccipital**: for distal PICA and bilateral proximal vertebral artery aneurysms. Rarely used
2. **Far lateral approach**: involves taking C1 lamina, posterolateral foramen magnum and the posterior -1/3-2/3 of occipital condyle and lateral suboccipital craniotomy. The anterior resection of occipital condyle is defined by the hypoglossal canal and the condylar emissary vein.. It provides wide exposure of the vertebral trunk and is the most commonly used approach for vertebral artery aneurysms.
3. **Extended far lateral approach or ELITE** (extended lateral inferior transtubercular exposure: The approach is similar to the far lateral except that the suboccipital craniotomy is extended to the transverse sigmoid junction and the sigmoid sinus is skeletonised to the jugular bulb. This involves lateral suboccipital craniotomy, limited mastoidectomy to skeletonise SS to the jugular bulb. Dura is opened along the SS. It provides trajectory along the vertebral artery
4. **Combined –combined approach**: combination of extended far lateral, Transcochlear and subtemporal; approaches with incision of the incisura. It provides extensive view of the posterior fossa, all cranial nerves and vessels from midbrain to upper cervical spine and usually is used for large petroclival tumours.

- For revascularisation the recipient artery is PICA. The donor artery can be EC (OA-low flow or VA –high flow) or IC (PICA-PICA).
- The majority of dissecting aneurysms are fusiform and unamenable for clipping. One option is proximal occlusion (Hunterian ligation either surgical with/without bypass depending on balloon test occlusion and the status of collateral circulation. For revascularisation options for posterior circulation look table on page1988. (Youmans)
- For more details look Youman's 1971—2007.

Vertebral artery, PICA and vertebro-basilar aneurysms:

- Posterior circulation aneurysms account for 15% of all intracranial aneurysms. 25% of those occur at vertebral artery /PICA region. The most common presentation is SAH 80% (**high incidence of IVH 75-95% and hydrocephalus 40-90%**). Other presentations include with mass symptoms (lower cranial nerves and brain stem compression, and TIAs or infarctions from embolisation. **Many aneurysms in this location are fusiform and due to dissection (28% in Yamaura's series)**
- Investigations include CT scan, **CTA(sensitive in detecting aneurysms larger than 3 mm, provides three D visualisation of the aneurysm** , calcification in the neck and intramural thrombus), MRI and 4 vessel angiogram (looking for relation to other arteries, perforators, presence of absence of contralateral PICA and size of P-com artery. For fusiform and giant aneurysms balloon test occlusion should be done as well as imaging of ECA (for revascularisation procedures).
- Surgical approaches to PICA aneurysms: A. anterior and lateral medullary segments-far lateral transcondylar B. tonsilomedullary segment-combined far lateral and suboccipital craniotomy C. Telovelotonsillar and cortical segment-median suboccipital
- Treatment options include :

1. Surgical clipping for small, large saccular aneurysms through lateral or far lateral suboccipital approach or ELITE approach as above. Complications include lower cranial nerve injury 11%, Wallenberg syndrome 25%, and 6th nerve injury 4% (Yamaura's series of 90 patients). Transoral or transfacial transclival approach is associated with 50% incidence of CSF leak and meningitis. Clip is applied through the window between IX and X superiorly XI inferiorly and the medulla medially or through the window between VII and VIII superiorly and IX, X inferiorly
2. Endovascular: GDC, stenting and Balloon occlusion for fusiform and complex giant aneurysms. GDC coiling is associated with 70-80% total obliteration rate for small<10 mm aneurysms, 50% obliteration rate for giant aneurysms (>25 mm) and 35% total occlusion for large aneurysms (10-25 mm). The long term radiological and clinical outcome fro GDC embolisation is not known. Coil compaction can develop inn28% of coiled aneurysms and in larger proportion of large and giant aneurysms.(prospective Multicenter study involving 402 patients).
3. Proximal occlusion surgical or endovascular for some fusiform and complex giant aneurysms. This should be a last option and should be preceded by balloon test occlusion. Mortality 9-24%, high risk of complications (rebleeding, thromboembolism and enlargement of the aneurysm).

Aneurysms of the basilar trunk:

- In Drake's series of 1200 vertebrobasilar aneurysms **16% were in upper basilar** trunk, **8% lower trunk** and 7% VB junction. Most commonly they arise at the origin of AICA and project laterally, but can arise from the perforators and project posteriorly into the pons or anteriorly towards the clivus. SAH is the most common presentation (high incidence of IVH and hydrocephalus). Other presentations include cranial nerves injury (7^{th} and 8^{th}

for ruptured PICA aneurysms, lower cranial nerves for VB junction lesions and 3-d nerve for basilar apex aneurysms. Investigations are similar to vertebral aneurysms (**always obtain venous phase angiogram to assess the possibility of sigmoid sinus ligation).**

- Treatment options as above. Approaches as above: In addition for upper trunk one can use extended orbitozygomatic or subtemporal .Clip is applied through the space between 5th nerves superiorly and 7th and8th inferolaterally. For VB junction lesions one can also use the ELITE approach.(the muscular layers of the incision are three A. superficial(trapezius and sternocleidomastoid) B. Intermediate layer (splenius capitis, longissimus capitis and semispinalis capitis) C. Inner layer (superior and inferior oblique, rectus capitis posterior major and minor. The vertebral artery should be exposed in the suboccipital triangle (SO, IO, RCM).
- Complications :

1. Injury to the perforators
2. Cranial nerves injury
3. Temporal lobe contusion, injury to vein of Labbe for subtemporal approach

- Surgical series; poor outcome can be due to injury from the primary haemorrhage, technical complications (retraction, perforator injury) or to vasospasm.

1. Drake:1767 patients 84% had good outcome ,16% mortality or major morbidity
2. Sugita: 70% good outcome, 30% major morbidity or mortality.

Aneurysms of the basilar apex and PCA:

- **50%** of posterior circulation aneurysms occur at the basilar apex. The second most common location is SCA and the third location is V/PICA. The key to safe clipping is the preservation of thalamoperforating arteries (anterior from P-COM and posterior from P1 and Basilar arteries. These aneurysms can be approached through transsylvian approach (Yasargil), orbitozygomatic subtemporal approach (Drake), PAVEL approach (pterional approach via extended lateral craniotomy), and extended orbitozygomatic. The last 2 approaches combine the advantages of the 2 classical subtemporal and transsylvian approaches.
- Surgical series:

1. Drake: 545 patients, 87% good outcome 13% mortality and major morbidity.
2. Samson: 242 patients complete occlusion in 94%.85% good outcome
3. Spetzler review of published literature (957 patients): 82% good outcome, 5.1% mortality.
4. Rice (99 patients); 4% mortality

- **Aneurysms of PCA account** for 0.7%-2.2% of all intracranial aneurysms. In Rhoton's review of 118 published cases 15% were in P1, 16% P1-P2 junction, 20% proximal P2A, **36% distal P2 (P2p)** and 13% P3. 50% of these aneurysms are giant and many are fusiform (dissecting or atherosclerotic)
- P1 and P1-P2 junction aneurysms can be approached similar to basilar apex, P2 aneurysms can be approached subtemporally or trans temporal trans ventricular trans choroidal fissure, P3 aneurysms can be approached through occipital interhemispheric approach.

- Fusiform and many giant aneurysms are not amenable to clipping and can be treated with proximal occlusion. Balloon test occlusion can be used to assess the safety of occlusion. Revascularisation is very difficult and not needed in the majority of cases (Drake-9 cases of P1 and P2 aneurysms treated by Hunterian ligation, only 1 developed ischemic complication). Always look for the size of P-com artery.

Endovascular treatment of intracranial aneurysms:

- Endovascular treatment of intracranial aneurysms is divided into 2 groups;

1. Deconstructive: Involves occlusion of the parent vessel and the aneurysm using detachable balloons or coils with the aim of thrombosing the aneurysm. It is used to treat some complex, giant and fusiform aneurysms and some posttraumatic aneurysms. It should be preceded by balloon test occlusion (20 min with normal blood pressure, 20 min with reduced BP, with angiogram of the other vessels to assess the status of collaterals) and in some centres the CBF is assessed and the parent vessel is occluded only if CBF remains > 35 ml/100 g/min. This method should be used as last option. Ideally one should trap the aneurysm. This method is effective in treating cavernous aneurysms and OA aneurysms proximal to OA (100% thrombosis rate in some studies) . Also is effective in treating vertebral, VB junction and basilar trunk aneurysms. Less effective in ICA aneurysms distal to OA and basilar bifurcation aneurysms (distal filling). **Despite negative balloon test occlusion test 0-8% of patients develop early ischemic complications and up to 16% develop delayed ischemic complications**. In addition aneurysm can recanalise, enlarge and rupture. Closed clinical and radiological follow up is mandatory. It should be avoided early post SAH (vasospasm may reduce the collateral blood supply).

2. Reconstructive procedures: involves selective occlusion of the aneurysm. The most commonly used material is GDC. These are platinum spiral coils with circular memory (variable size and length, soft and standard, 2D, 3D some are made more thrombogenic by adding Dacron fibres to the coil). They are attached to guide wire and can be detached by electrolysis (applying small current 1mA). They were introduced in 1990 by Gugleimi (Italian neurosurgeon). They are effective in preventing rebleeding from ruptured aneurysms. The mechanism by which GDC embolisation works is not fully understood. Even with full radiological obliteration, there is blood flow into the aneurysm. It is thought that they act by changing the hemodynamics within the aneurysm leading to thrombosis. The thrombus undergoes organisation (neovascularisation and invasion by fibroblasts). Eventually the fibrous membrane that covers the aneurysm undergoes endothelisation and the aneurysm becomes excluded from the circulation. Although initially used to treat patients with high grades, poor medical conditions and difficult aneurysms unamenable to surgery, currently GDC embolisation is used as the treatment of first choice for ruptured and unruptured aneurysms in many centres around the world.

A. **ISAT** (International subarachnoid aneurysm trial): prospective, randomised Multicenter study. 2143 patients with SAH who were suitable for clipping and

coiling were randomised to one modality of treatment. The primary outcome was the proportion of patients with mR score of 3-6 (dependency or death) at 1 year after treatment. There was **6.9% absolute risk reduction** of death and dependency in the endovascular group at 1 year (23.7% Vs 30.6%). The study was terminated prematurely by the steering committee. The risk of rebleeding from coiled aneurysms was 2 /1276 and 0 in the surgical group Published in the Lancet, May2002. This study demonstrated unequivocally demonstrated the safety and efficacy of GDC embolisation of ruptured intracranial aneurysms.

B. The Finnish prospective randomised trial (Vanninen et al radiology 1999): 108 patients were randomised to endovascular treatment (52) and surgery (56). At 3 months there was no difference in the outcome between the 2 groups. 81% of endovascular group and 79% of surgical group had good or moderate recovery and there was no difference between the 2 groups when the neuropsychological outcome was assessed at 1 year.

- Occlusion of coiled aneurysms depends on the size and geometry of the aneurysm. Total occlusion can be achieved in 70-80% of small aneurysms and in <50% of giant aneurysms.
- The fate of the residual aneurysm is variable with some undergoing thrombosis and others enlarging? Compaction of the coils and recurrence occurs in the majority of large coiled aneurysms (Gruber from Vienna).
- The GDC embolisation technology is rapidly evolving and what was considered uncoilable aneurysms because of wide neck is currently treatable with the use of 3D coils, balloon-remodelling technique and stent-coil technique
- GDC embolisation can be associated with the following complications :

1. Thromboembolic complications: up to 24% in some studies (from intraluminal clot, thrombosis induced by manipulation, prolapsing coil in the parent artery, dissection). Most prolapsed coils cause no trouble and are treated by anticoagulation for 48 hours followed by antiplatlets for few weeks, symptomatic can be treated by stenting the parent artery to push the coil against the vessel wall. For distal embolisation and occlusion, superselective thrombolysis is an option of the aneurysm has been packed with coils
2. Intraoperative rupture: 4-8% from the microcatheter, guide wire or coil. (Reverse heparin, continue coiling, occasionally balloon occlusion
3. Infection is very rare: mycotic aneurysms.

- GDC embolisation and surgical clipping are complementary and not competitive method of treating intracranial aneurysm. The decision to use either or should be made by neurosurgeon in close cooperation with interventional neuroradiologist. Some complex aneurysms ma require combination of both methods

Giant aneurysms:

- Aneurysms larger than 25 mm. 2-5% of intracranial aneurysms. 34-67% ICA, 10-40% ACA, MCA. 11-50% posterior circulation. **60% has intramural thrombus.**
- **1/3** of patients present with **SAH**, 2/3 with mass effect (anterior circulation-visual symptoms, dementia and posterior circulation with cranial nerves deficit

and bulbar palsy). **8%** with distal embolisation. Investigations should include 6-vessel angiogram, CT, CTA, MRI, MRA, balloon test occlusion and assessment of CBF (anticipating proximal occlusion with or without bypass).

- The annual risk of haemorrhage in ISUIA study was **6% per year**. Haemorrhage caries **50%** risk of mortality or severe deficit (Laplace's law-wall tension is proportional to (power 4 of the radius) and intraluminal pressure).
- Pathologically they can be1. **Saccular** (hemodynamic stress at bifurcation points) 2. **Fusiform** (atherosclerotic, arteriopathies and traumatic dissection).
- Management of aneurysms is indicated in the majority of cases because of the poor natural history. Exceptions are patients in poor grade and medically high risk patients. The principles of treatment are similar to small aneurysms.
- Anterior circulation aneurysms can be approached through pterional craniotomy with orbitozygomatic osteotomy (wide corridor, shallow field and less brain retraction). Proximal control for ICA aneurysms is in the neck, or petrous ICA in Glascock's triangle. EEG, electrophysiological monitoring, cerebral protection and hypothermic circulatory arrest are possible adjuncts. Posterior circulation aneurysms are approached through orbitozygomatic with drilling anterior and posterior clinoid for upper basilar artery, transpetrsonal approaches for middle basilar segment and far lateral approach for vertebral, VB junction and PICA aneurysms.(look above for approaches. For details look pp. 2084-2093)
- Endovascular treatment is an option, however complete obliteration rate is < 50% and the risk of coil compaction and recurrence is high.

Infectious Intracranial aneurysms "Mycotic aneurysms":

- Account for **5%** of intracranial aneurysms in adults and **10%** of ICA in children. The most common organism is **streptococcus** 25-44% followed by **staph. 14-18**%. In **5% multiple** organisms are responsible. **In 10%** no organism can be isolated. Rarely due to fungal infection (in immunocompromised patients). Aneurysms due to fungus are rare (40 reported cases). The most common fungus is **aspergillus** followed by phycomycetes and Candida albicans.
- **80%** of infections occur in patients with **SBE**. (20-40% of patients with SBE develop neurological deficit most commonly due to infarction and in **5%** due to aneurysms). Aneurysms secondary to SBE are due to infectious emboli. They are **multiple in 20%** of cases and located in MCA territory in 60%. On the other hand aneurysms that develop secondary to extravascular infection such as meningitis, cavernous thrombophlebitis, sinusitis and osteomyelitis tend to affect proximal vessels at the base of the skull and tend to be single and larger than those due to SBE. Fungal aneurysms also tend to be proximal.
- Natural history of these lesions is not known due to the rarity of the condition. Infectious aneurysms carry high **mortality** (**30%** for all), **80%** for ruptured ones and **90%** for fungal aneurysms. The large discrepancy between the incidence in clinical and autopsy series may indicate that many aneurysms remain asymptomatic
- Diagnosis require high index of suspicion. SAH or ICH associated with fever should initiate investigations to prove or exclude infectious aneurysms. This

includes (FBC, CRP,ESR, Blood cultures, cardiac echo looking for vegetations on the valve +ve in 90%, CT,CTA, MRI,MRA and cerebral angiogram which is the gold standard. **CTA and MRA are not sensitive in detecting small distal aneurysms** but can be used as non-invasive tests for follow up.

- Treatment :
    1. **Medical therapy** is the first line of treatment. Appropriate antibiotics should be given for at least 6 weeks with follow up angiograms or CTA, MRA if the lesion is visualised on these tests (initially every 7-10 days) then 6/52, 3 months, and 6 months because these lesions have unpredictable cycle of growth and resolution despite medical treatment**.30%** of aneurysms can disappear with Abs, another **19%** can significantly decrease in size. Even if the aneurysm persists partial healing with wall fibrosis renders surgery less difficult. Medical therapy includes cardiac medications. (Diuretics, antiarrhythmic etc...)
    2. **Cardiac consideration**: Valve replacement before treating the infectious aneurysms may be indicated if patient develops refractory cardiac failure, bacteremia despite antibiotics and continues to through emboli. **Bioprosthesis** should be used to avoid long term anticoagulation
    3. **Surgery**: Surgical options include clipping, excision with the parent artery (if small), and excision with end-end anastomosis MCA/MCA or STA/MCA bypass. Surgery for these lesions is difficult because the aneurysm and the parent artery are fragile. A course of Abs may result in partial healing by fibrosis and strengthening of the wall of the aneurysm. Indications for surgery are:

    A. In case of mass lesion such as haematoma or abscess
    B. Increase in the size of the aneurysm despite Abs
    C. Failure of resolution of the aneurysm after 6 weeks of Abs
    D. Rupture of aneurysm

The following factors should be considered: patient's general condition, multiplicity of the aneurysms, eloquence of the brain and the GCS.
These aneurysms tend to be small and buried in the sulci , hence stereotaxy is indicated to localise the lesions.

GDC embolisation has been used successfully recently (theoretical risk of persistent infection in the FB)

Traumatic aneurysms:

- Less than **1%** of all aneurysms. 30% mortality rate. Are caused by

1. Penetrating injuries particularly low velocity injuries .**10-12%** of patients with stab wound to the brain developed false aneurysms (South Africa where 6% of head injuries are penetrating stab wounds). **0.1-8%** incidence of false aneurysms following penetrating gunshot injuries (Lebanon, Iraq wars)
2. Closed head injury (base of skull fractures)
3. Iatrogenic: transphenoidal surgery, EVD, 3-d ventriculostomy, brain biopsy.

- Pathogenesis: Injury to the arterial wall (intima, media or adventia) can lead to the formation of :

1. False aneurysm: disruption of all layers of the wall with contained perivascular haematoma. False aneurysms develop distally. most commonly in MCA territory followed by pericallosal artery
2. Dissection: Intimal tear with the formation of a second lumen between intima and media or media and adventia.
3. Arterio-venous fistula: C-C or DAVF.

- Clinical features and diagnosis: High index of suspicion is required. Patients with false aneurysms tend to **present in delayed fashion** with ICH, SAH, and SDH. **Angiography** is the gold standard and should be obtained in **all patients with penetrating injuries in the second week and if negative repeated 3 weeks later** or earlier if patient develops new haemorrhage.
- Treatment: Most of these aneurysms are not amenable to clipping. Treatment options include :

1. Excision of distal aneurysms (rarely associated with neurological morbidity)
2. Bypass grafting (STA/MCA bypass) + excision for proximal aneurysms. Depending on balloon test occlusion results and CBF measurements(xenon, SPECT, PET, Doppler u/s "decrease of MCA velocity to 60% of normal indicates the need for bypass")
3. Trapping +- bypass for ICA aneurysms
4. Endovascular coiling or occlusion of the parent vessel
5. Direct clipping (rarely possible)

Multimodality treatment of complex cerebrovascular lesions:

- **Multiple aneurysms**: **15-20%** of intracranial aneurysms are multiple. The following characteristics can help to identify the ruptured aneurysm (pattern of SAH, size, shape of the aneurysm, location, statistically more likely to rupture etc… however in some cases it is hard to decide which one has ruptured. The options for treating multiple aneurysms are:

1. Clip or coil the ruptured one and observe the others if they are small and the patient is elderly.
2. Clip all the aneurysms if that is possible through single craniotomy.
3. Coil all the aneurysms
4. Clip the bleeding and coil the others or coil the bleeding one and clip the others

- **Failed coiling**: The best option to treat incompletely obliterated or recurrent coiled aneurysms is to **recoil them if possible**. If coiling not possible the options are either observation or clipping. Clipping of coiled aneurysms is more difficult (less mobile and difficult visualisation around them).
- **Residual or recurrent neck after clipping** occurs in **4%** of clipped aneurysms. The options are observation, reclipping or coiling.
- **Patients with aneurysms and AVM. 10%** of patients with **AVM** have aneurysms. Most of them are flow related and located on a proximal vessel of the circle of Willis, on a feeding artery, intranidal or not flow related on a distant vessel. Treatment should be directed on the symptomatic lesion. Options include:

1. Clipping of the aneurysm and excision of the AVM through the same craniotomy. This is the ideal treatment if possible.
2. Coiling of the aneurysm and excision of the AVM (2 procedures)
3. For deep AVM (basal ganglia, thalamus and brain stem) .Coiling or clipping of the aneurysm followed by radiosurgery.

Although some authors report the disappearance of some flow related aneurysms after excision of the AVM, there is a risk of aneurysm enlargement and rupture due to the sudden increase in arterial resistance.

Revascularisation techniques for complex aneurysms and skull base tumours:

- Indications: **80-90%** of patients tolerate balloon test occlusion(BOT) of ICA. The indications for revascularisation procedures are variable among centres. Most neurosurgeons advocate selective approach (In patients who fail balloon test occlusion with hypotensive challenge or those with drop in CBF < 35ml/100g/min. Others argue for universal approach (revascularisation of all patients requiring proximal occlusion of major vessels to prevent delayed ischemia (16% of patients with negative BOT) and the development of De novo aneurysms on the collateral circulation

1. Unclipable giant and fusiform aneurysms, particularly with atherosclerotic calcified necks.
2. Base of skull tumours involving the carotid artery. The use of revascularisation procedures for base of skull tumours is controversial. In Spetzler's series of 300 base of skull tumours revascularisation was done in 10. In the majority of benign tumours (pituitary adenoma, craniopharyngioma, meningioma and chordoma) the ICA is encased rather than invaded by the tumour and residual tumours can be treated with radiosurgery.
3. In selected group of patients with ischemic symptoms secondary to hypoperfusion in the presence of significant stenosis of intracranial ICA or Basilar artery and absence of infarction.
4.

- Technique:

1. Preoperative angiogram , balloon test occlusion with and without hypotensive challenge , measurement of CBF to assess the need for the revascularisation and to characterise the anatomy of the donor and recipient artery
2. EEG and electrophysiological monitoring and brain protection(barbiturate, propofol, etomidate to produce burst suppression of EEG, mild hypothermia and hypertension)

- Types of revascularisation: for details look pp-2109

1. Type 1: saphenous vein graft between petrous and supraclinoid ICA: technically difficult, takes long time and carries high risk of complications. Flow through MCA is about 250 ml/min. Saphenous bypass flow is about 50-150 ml /min.

2. Type 2: EC-IC saphenous venous graft: between ICA and ECA proximally and MCA or PCA distally.
3. Type 3: scalp artery to intracranial artery: STA/MCA bypass, OA/AICA , STA/SCA. Blood flow through this type is 10-70ml/min and can only supplement and not replace the circulation.
4. Type4: Intracranial/intracranial bypass: PICA/PICA, PICA/AICA, pericallosal/pericallosal side to side anastomosis.

- Complications:

1. Early graft occlusion: **6%** in a series of 58 (kinking, thrombosis). If any doubt about the patency of the bypass get angiogram and revise.
2. Intraoperative rupture secondary to hemodynamic stress from increased flow (one should trap or proximally ligate the aneurysm as soon as possible either through the same procedure or soon later.
3. Ischemic deficit: prolonged temporary clamping (brain protection and hypothermia can reduce the risk).
4. Subdural, EDH.

- Outcome:

1. International cooperative EC-IC bypass study 1985 found postoperative patency rate of 663 STA/MCA bypass was 96 % and 91% at 1 year.
2. Regli et al found early patency rate of saphenous graft 88%, at 5 years 82% and at 13 years 73% (mean annual graft failure rate of 1-1.5% per year)
3. Excellent and good outcome was reported in 80% of patients having revascularisation procedures and trapping of the aneurysm ((Sundt, Sekhar).

Techniques of large calibre flow bypass

Balsam Darwish: 4th year research, Liverpool hospital.

- There are 3 types of Cerebrovascular bypass:

1. Low flow bypass such as STA/MCA, occipital/PCA: flow through these bypasses is about 15-25 ml/min.
2. Moderate flow bypass utilizing radial artery: flow 40-70ml/min.
3. High flow bypass using reversed saphenous vein graft : 70-140 ml/min

Normal flow through MCA is about 250ml/min and through PCA is about 200ml/min.

- Indications:

1. Giant complex aneurysms not amenable to clipping before trapping or proximal occlusion
2. Base of skull tumours involving ICA
3. In selected group of patients with ischemic symptoms secondary to hypoperfusion in the presence of significant stenosis of intracranial ICA or Basilar artery and absence of infarction.

- **Techniques**: Intraoperative EEG, SSEP and MEP monitoring, moderate hypothermia (33 degrees) and cerebral protection (barbiturates, propofol and etomidate) are very important to decrease the risk of ischemic complications. If there is doubt bout the potency of the graft, intraoperative angiogram should be obtained.

1. Petrous ICA/ supraclinoid ICA using saphenous venous graft: advantages (intracranial procedure, short segment of saphenous vein and potentially long term potency). Disadvantages (technically demanding procedure, prolonged temporary clamping time and increased risk of ischemic deficit).Spetzler et al.
2. Cervical ICA/ ECA-supraclinoid ICA/MCA: Advantages (technically easier operation, potentially less temporary clamping time). Disadvantages (long segment saphenous vein with potential risk of kinking and trauma, potentially less long term potency).
3. ECE/PCA: used for complex symptomatic basilar tip aneurysms (technically demanding.

ELANA (excimer laser-assisted non-occlusive anastomosis) allows connecting the graft to ICA without temporary clamping. After suturing the graft to adventitia of the intracranial ICA, the laser tip (2.2-2.8 mm in diameter) is inserted through a side hole in the graft and fixed to ICA wall by vacuum suction. The laser cuts a hole in the wall of ICA about 2.8 mm. The flow through this anastomosis was 140 ml/min in 90 patients.

- **Outcome**: Average mortality 5-9% (up to 20%). Functional decline and severe neurological morbidity 5-13% (up to 50% in some series). Postoperative graft potency 95-100%. Annual graft occlusion rate is about 1-1.5%.