Neurosurgical Vascular Pathology

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Cerebral Aneurysms

Etiologies of SAH:

- Trauma (the most common cause)
- Spontaneous SAH:
  - Ruptured intracranial aneurisms
  - Cerebral AVMs
  - Rarely due to tumor
  - Cerebral artery dissection
  - Coagulation disorders

Miscellaneous facts about SAH

- Peak age for aneurismal SAH is 55-60 years
- 30 % of aneurismal SAH occurs during sleep
- 50 % of patients with aneurisms have warning symptoms

Risk factors for SAH

- Hypertension
- Cigarette smoking
- Oral contraceptives
- Alcohol consumption

Symptoms

- HA (the worst headache of my life)

- Vomiting
- Syncope
- Neck pain
- Photophobia
- Meningismus (nuchal rigidity)

Hunt and Hess scale of SAH

Grade 0 - Unruptured aneurysm

Grade 1 - Asymptomatic or minimal headache and slight nuchal rigidity

Grade 1A - No acute meningeal or brain reaction but with fixed neurologic deficit

Grade 2 - Moderate-to-severe headache, nuchal rigidity, no neurologic deficit other than cranial nerve palsy

Grade 3 - Drowsiness, confusion, or mild focal deficit

Grade 4 - Stupor, moderate-to-severe hemiparesis, possible early decerebrate rigidity, and vegetative disturbances

Grade 5 - Deep coma, decerebrate rigidity, and moribund appearance

Evaluation

- Non-contrast high resolution CT
- If CT is negative: LP
- Cerebral angiography
  - The gold standard for evaluation of cerebral aneurisms
  - Demonstrates sourse
  - Shows if radiographic vasospasm is present (clinical vasospasm never occurs < 3 days following SAH)

Vasospasm

= a delayed focal ischemic neurologic deficit following SAH, characterized by decreased LOC with focal neurologic deficit (speech or motor)

- Onset: almost never before day 3 post-SAH
- Maximal frequency of onset during days 6-8
- Clinical SAH almost always resolved by day 12, radiographic – 3-4 weeks

Treatment

- Early surgery: removing the risk of rebreeding, direct removal of blood clot
- Direct pharmacological arterial dilatation (Ca channel blockers)
- Direct mechanical arterial dilatation: catheter directed balloon
- Indirect arterial dilatation: hyperdynamic therapy
- Improvement of the rheologic properties of blood

  hyperdynamic therapy = 3H therapy
  - hypervolemia
  - hypertension
  - hemodilution

INTRODUCTION TO ANEURISMS

Aneurysm is an abnormal local dilatation in the wall of a blood vessel, usually an artery, due to a defect, disease, or injury.

The 3 major types of true intracranial aneurysms are decribed:

- saccular
- fusiform
- dissecting

Incidence

In one series of patients undergoing coronary angiography, incidental intracranial aneurysms were found in 5.6% of cases, and another series found aneurysms in 1% of patients undergoing 4-vessel cerebral angiography.

CAUSES AND CLASSIFICATION OF INTRACRANIAL ANEURYSMS

The common causes of intracranial aneurysm are

- hemodynamic induced/degenerative vascular injury
- atherosclerosis (typically leads to fusiform aneurysms)
- underlying vasculopathy (eg, fibromuscular dysplasia)

Uncommon causes include trauma, infection, drugs, and neoplasms (primary or metastatic).

Etiology

Most intracranial aneurysms probably result from hemodynamically induced degenerative vascular injury.

Classification of intracranial aneurysms

A. Morphology
   1. Saccular
   2. Dissecting
   3. Fusiform

B. Size
   1. <10 mm
   2. 11–24 mm
   3. >25 mm (giant)

C. Location

1. Anterior circulation arteries
    a. Internal carotid artery
    b. Middle cerebral artery
    c. Anterior cerebral artery
2. Posterior circulation arteries
    a. Vertebral artery
    b. Basilar artery
    c. Posterior cerebral artery

Most-common sites of saccular aneurysms

- Each aneurysm arises from the branching site of a large artery.
- Most are located on or near the circle of Willis.
- More than 90% are located at one of the following five sites

- the internal carotid artery at the level of the posterior communicating artery
- the junction of the anterior cerebral and anterior communicating arteries
- the proximal bifurcation of the middle cerebral artery
- the junction of the posterior cerebral and basilar arteries
- the bifurcation of the carotid artery into the anterior cerebral and middle cerebral arteries.

Most-common sites of saccular aneurysms

Other aneurysm sites on the carotid artery are at the origins of the ophthalmic, superior hypophyseal, and anterior choroidal arteries. Other sites on the vertebral and basilar arteries include the sites of origin of the anteroinferior cerebellar, posteroinferior cerebellar, and the superior cerebellar arteries and the junction of the basilar and vertebral arteries. A.C.A., *anterior cerebral artery;* A.Ch.A., *anterior choroidal artery;* A.Co.A., *anterior communicating artery;* A.I.C.A., *anteroinferior cerebellar artery;* B.A., *basilar artery;* C.A., *internal carotid artery;* M.C.A., *middle cerebral artery;* Op.A., *ophthalmic artery;* P.C.A., *posterior cerebral artery;* P.Co.A., *posterior communicating artery;* P.I.C.A., *posteroinferior cerebellar artery;* S.C.A., *superior cerebellar artery;* S.Hypo. A., *superior hypophyseal artery;* V.A., *vertebral artery.*

D. Etiology

1. Saccular
    a. Idiopathic, acquired due to hemodynamic stress
    b. Acquired hemodynamic related to increased flow (e.g., arteriovenous malformation, arterial hypoplasia)
    c. Polycystic kidney disease
    d. Fibromuscular dysplasia
    e. Coarctation of the aorta
    f. Familial
    g. Trauma
    h. Infectious
    i. Neoplastic
    j. Radiation
2. Fusiform
    a. Atherosclerosis
    b. Structural
    c. Hemodynamic (e.g., coarctation of the aorta)
    d. Genetic (possibly associated with Marfan syndrome, pseudoxanthoma elasticum)
    e. Infectious
    f. Radiation
    g. Other angiopathies (e.g., giant cell arteritis)
3. Dissecting

Pathology

A normal artery wall consists of 3 layers:

- the intima, which is the innermost endothelial layer;
- the media, which consists of smooth muscle;
- adventitia, the outermost layer, which consists of connective tissue

The aneurysmal sac itself is usually composed only of intima and adventitia.

Location

Aneurysms commonly arise at the bifurcations of major arteries. Most saccular aneurysms arise on the circle of Willis or the middle cerebral artery (MCA) bifurcation.

Multiplicity

- Intracranial aneurysms are multiple in 15-20% of all cases . About 75% of patients with multiple intracranial aneurysms have 2 aneurysms, 15% have 3, and 10% have more than 3

- Multiple aneurysms can be bilaterally symmetric (ie, mirror aneurysms) or asymmetrically located on different vessels. More than one aneurysm can be present on the same artery.

Flow dynamics and aneurysm growth

- The apex of vessel bifurcations is the site of maximum hemodynamic stress in a vascular network.
- These augmented hemodynamic stresses probably cause the initiation and subsequent progression of most saccular aneurysms.
- Thrombosis and rupture are also explained by intra-aneurysmal hemodynamic stresses.

Age at presentation

- Aneurysms typically become symptomatic in people aged 40-60 years

Clinical presentation

Most aneurysms do not cause symptoms until they rupture; when they rupture, they are associated with significant morbidity and mortality.

- SAH
- Intracerebral hemorrhage
- Intraventricular hemorrhage
- Subdural blood

Other Symptoms and Signs

depending on the site and size of the aneurysm

- hemiparesis
- dysphasia
- cranial nerve palsies

  third nerve palsy secondary to posterior communicating artery aneurysm.

Carotid–ophthalmic artery aneurysms may produce unilateral visual loss or visual field defects.

Aneurysms may produce focal neurologic deficits due to mass effect from the aneurysm, seizures, focal ischemia due to embolism from the aneurysm.

Natural history

- Ruptured aneurysms that are not operated on have a very high risk of rebleeding after the initial hemorrhage has occurred.
- The risk is estimated at 20-50% in the first 2 weeks, and such rebleeding carries a mortality rate of nearly 85%.

Clinical outcome

Ruptured aneurysms have their highest rebleeding rate within the first day; if untreated, at least 50% rebleed during the 6 months after the initial hemorrhage.

Treatment

- Although cases have been reported with spontaneous resolution, direct treatment is usually recommended.
- Perform balloon trapping or balloon embolization on ICA aneurysms at the skull base.
- Treat peripheral lesions surgically with clipping of the aneurysm neck, excision of the aneurysm, or wrapping, if no other method is feasible.

FUSIFORM ANEURYSMS

Pathology

- These lesions are exaggerated arterial ectasias that occur because of a severe and unusual form of atherosclerosis.
- Damage to the media results in arterial stretching and elongation that may extend over a considerable length.

Clinical presentation

- Fusiform aneurysms usually occur in older patients.
- The vertebrobasilar system is commonly affected.
- Fusiform aneurysms may thrombose, producing brainstem infarction.
- They can also compress the adjacent brain or cause cranial nerve palsies.

Imaging

These aneurysms typically do not have an identifiable neck.

- MRI is helpful in delineating the relationship between vessels and adjacent structures such as the brainstem and cranial nerves.

DISSECTING ANEURYSMS

Pathology

- In arterial dissections, blood accumulates within the vessel wall through a tear in the intima and internal elastic lamina.
- The consequences of this intramural hemorrhage vary.
  - If blood dissects subintimally, it causes luminal narrowing or even occlusion.
  - If the intramural hematoma extends into the subadventitial plane, a saclike outpouching may be formed.

Etiology

- Dissecting aneurysms may arise spontaneously.
- More commonly, trauma or an underlying vasculopathy is implicated.

Location

- Most dissecting aneurysms that involve the craniocerebral vessels affect the extracranial segments; intracranial dissections are rare and usually occur only with severe head trauma.

Imaging

- Dissecting aneurysms are elongated, ovoid, or saccular contrast collections that extend beyond the vessel lumen.
- MR studies delineate an intravascular or perivascular hematoma associated with dissections.

IMAGING OF INTRACRANIAL ANEURYSMS

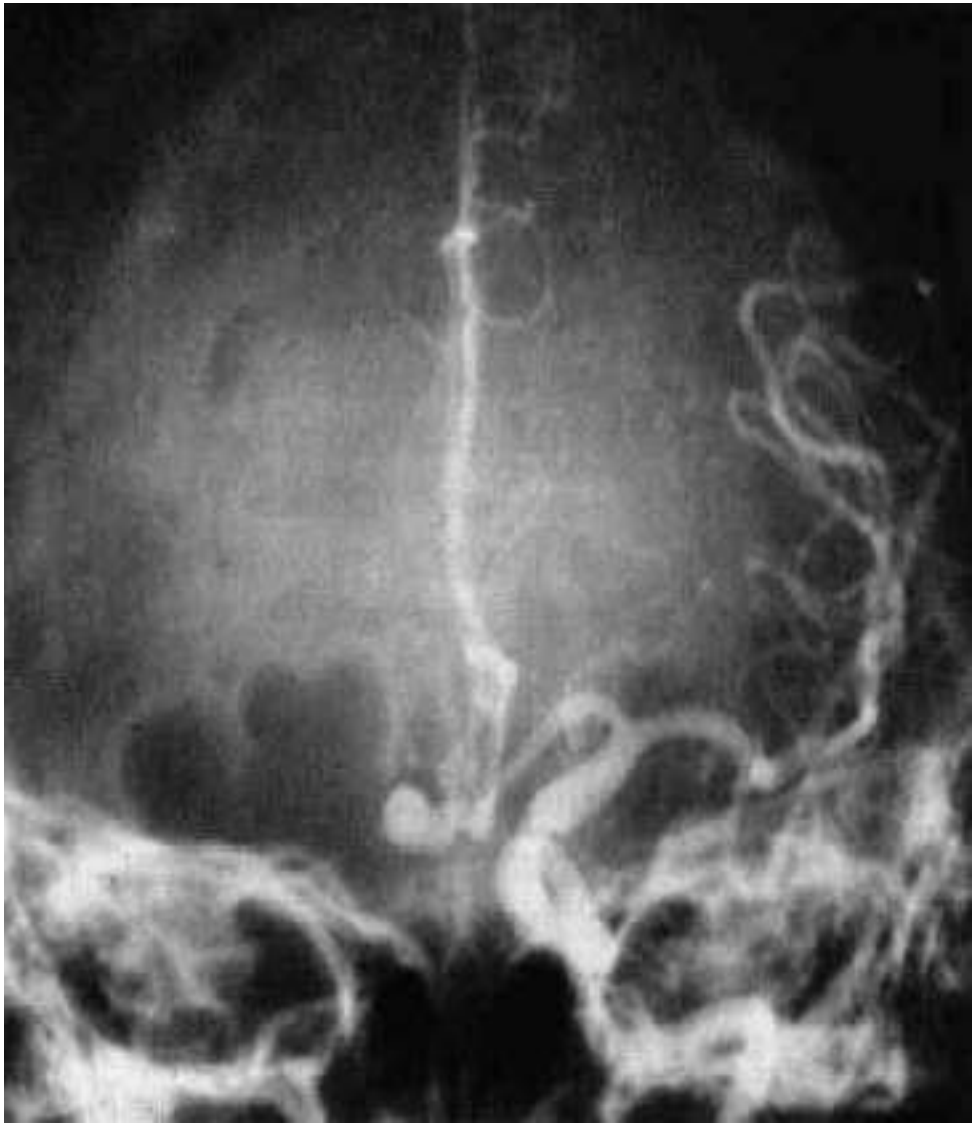
Angiography

The role of diagnostic cerebral angiography in patients with nontraumatic SAH is

- to identify the presence of any aneurysms,
- to delineate the relationship of an aneurysm to its parent vessel and adjacent penetrating branches
- to define the potential for collateral circulation to the brain, and to assess for vasospasm.

Angiography

A large aneurysm of the anterior communicating artery



CT

Computed tomography scanning is important to diagnose

- intracerebral and intraventricular hemorrhage
- hydrocephalus
- low-density areas
- calcification
- the aneurysm itself

  located somewhat excentrically in the suprasellar subarachnoid space or sylvian fissure

Magnetic resonance imaging

- Aneurysm appearance on MRI is highly variable and may be quite complex.
- The signal depends on the presence, direction, and rate of flow, as well as the presence of clot, fibrosis, and calcification within the aneurysm itself.

Treatment

Surgical clipping

The goal of surgical treatment is usually to place a clip across the neck of the aneurysm to exclude the aneurysm from the circulation without occluding normal vessels.

Choice of surgical technique

Whether to obliterate an aneurysm surgically through a **craniotomy and clipping** or to use **endovascular methods** is a decision made by the neurosurgeon and the endovascular radiologists as a team.

Choice of surgical technique

Younger patients tend to undergo surgical clipping because coiling has a high recurrence rate. Posterior fossa aneurysms (especially the basilar artery tip) tend to be treated using the coil procedure. In most major aneurysm centers, most cases are still obliterated by surgical clipping, but coiling is being used more frequently

Early versus late surgery

Controversy exists between so-called early surgery (generally, but not precisely defined as 48-96 h post-SAH) and late surgery (usually >10-14 d post-SAH).

Early surgery is advocated for the following reasons:

- If successful, surgery virtually eliminates the risk of rebleeding,
- Early surgery facilitates treatment of vasospasm, by allowing induction of arterial hypertension and volume expansion without danger of aneurysmal rupture.
- Surgery allows lavage to remove potentially vasospasmogenic agents from contact with vessels.
- Although the operative mortality rate is higher (this finding is controversial), the overall patient mortality rate is lower.

Arguments against early surgery and in favor of late surgery include the following:

- Inflammation and brain edema are most severe immediately following SAH. Such inflammation makes brain retraction more difficult.
- The presence of solid clot makes surgery more difficult.
- The risk of intraoperative rupture is higher with early surgery.
- The incidence of vasospasm may be increased following early surgery from mechanic trauma to vessels.

Factors favoring the choice of early surgery include the following:

- Good medical condition of patient
- Good neurologic condition of patient (Hunt and Hess grade <3)
- Large amounts of subarachnoid blood, increasing the likelihood and severity of subsequent vasospasm (clipping the aneurysm facilitates treatment of vasospasm)
- Conditions that complicate management in the face of unclipped aneurysm (eg, unstable blood pressure, frequent and/or intractable seizures)
- Large clot with mass effect associated with SAH
- Early rebleeding, especially multiple rebleeds
- Indications of imminent rebleeding (eg, development of third nerve palsy with posterior communicating artery aneurysm, increase in aneurysm size on repeat angiography)

Factors favoring delayed surgery (10-14 d post-SAH) include the following:

- Poor medical condition of patient
- Poor neurologic condition of patient, ie, Hunt and Hess grade >4
- Aneurysms difficult to clip because of large size or difficult location necessitating a lax brain during surgery
- Significant cerebral edema observed on CT scan

AVMs

Key features

- Dilated arteries and veins with dysplastic vessels and no capillary bed and no intervening neural parenchyma
- Medium-to-high pressure and high-flow
- Usually presents with hemorrhage, less often with seizures
- Congenital lesions with a lifelong risk of bleeding
- Demonstrable on angiography, MRI, CT

Description

An abnormal collection of blood vessels wherein arterial blood flows directly into draining veins without the normal interposed capillary beds, appear as a tangle of vessels, veins containing oxygenated blood.

AVMs

- Pial
- Subcortical
- paraventricular

Presentation

- Headache
- Hemorrhage
- Seizures
- Mass effect
- Ischemia: by steal

Evaluation

- CT
- Angiography
- MRI
- MRA

AVMs GRADING

TABLE 1. THE SPETZLER–MARTIN SCALE FOR EVALUATING THE RISK OF SURGERY IN PATIENTS WITH ARTERIOVENOUS MALFORMATIONS.*

CHARACTERISTIC	NO. OF POINTS ASSIGNED
Size of lesion	
Small (maximal diameter, <3 cm)	1
Medium (maximal diameter, 3–6 cm)	2
Large (maximal diameter, >6 cm)	3
Location	
Noneloquent site	0
Sensorimotor, language, or visual cortex; hypothalamus or thalamus; internal capsule; brain stem; cerebellar peduncles; or cerebellar nuclei	1
Pattern of venous drainage	
Superficial only	0
Any deep	1

*A score of 4 or 5 is associated with the highest risk of persistent neurologic deficits after surgery. Data are from Spetzler and Martin.[60]

AVMs TREATMENT OPTION

1. **Microsurgical removal**
2. **Embolization**
3. **Radiosurgery**
   - Gamma knife
   - Linear Accelerator
4. **Combined**
5. **Conservative-abstinence**

AVMs SURGERY STEP BY STEP

- large incision
- large bone flap
- slack brain
- little or no retraction of the brain
- gentle handling of brain and vessels
- proximal control
- extremely careful closure

MICRONEUROSURGERY OF CAVERNOUS MALFORMATIONS

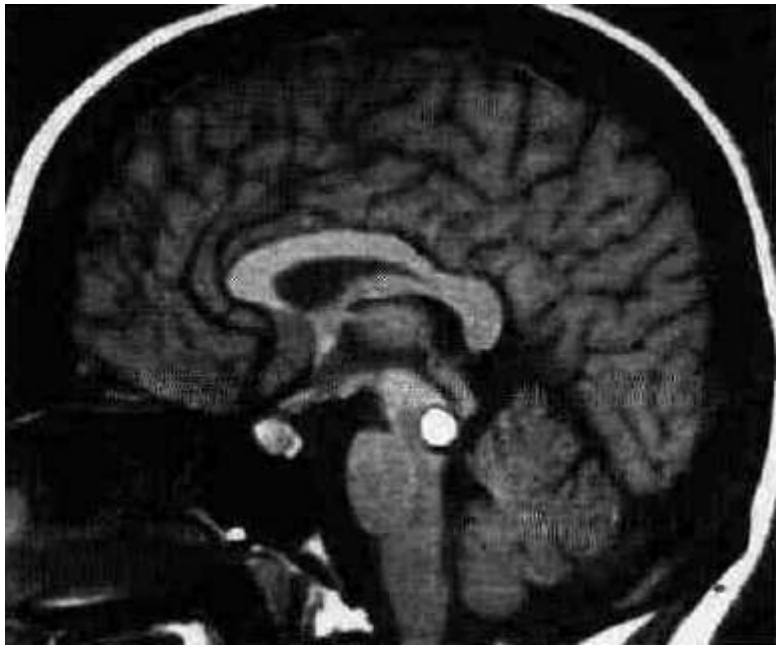
Key features

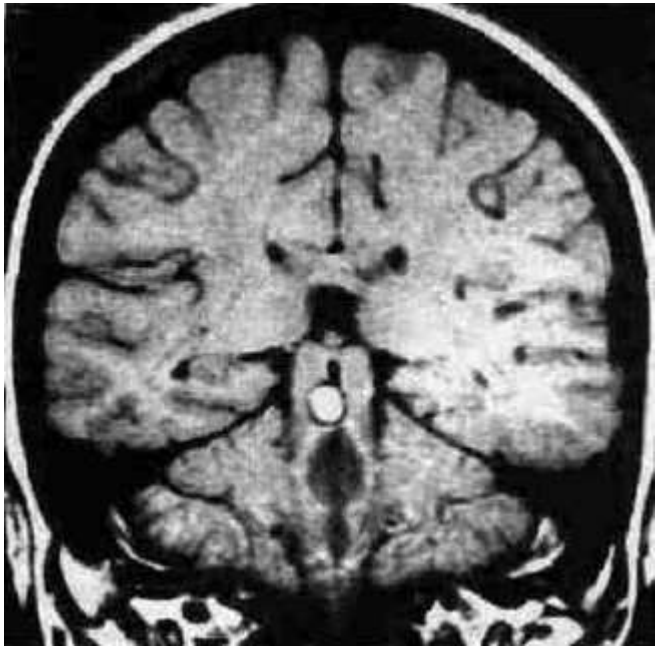
- Represent the venous drainage of the area
- A tuft of veins that converge into an enlarged central trunk that drains to venous system
- No abnormal arteries are found
- There is normal parenchyma between the vessels
- Classically produce a distinct caput medusae on the angiogram
- Low-flow and low-pressure

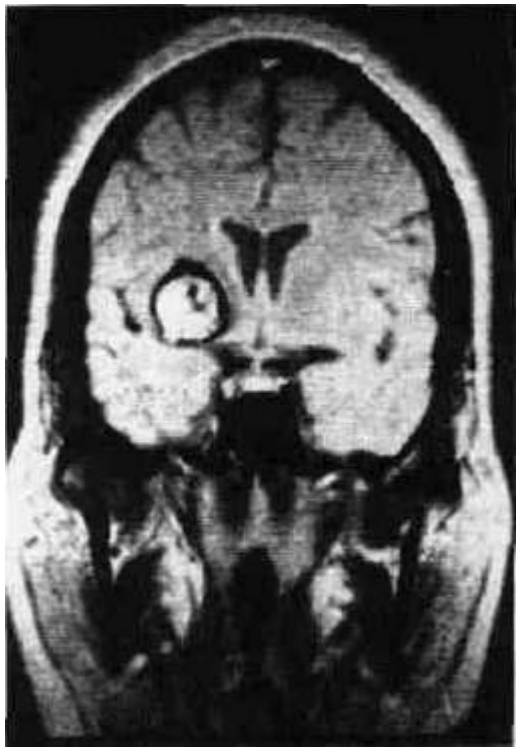
Key features

- Most clinically silent, rarely seizures or hemmorhage may occur
- MRI is the unique method of diagnosis
- In general, these should not be treated as they are venous drainage of the brain in that area
- Surgery is indicated only for bleedeng or seizures

CAVERNOUS MALFORMATIONS







Carotid-cavernous fistula

Direct high-flow shunts between the internal carotid artery and cavernous sinus

- Result of severe head trauma resulting in basilar fractures
- Spontaneous: ruptured cavernous internal carotid aneurysms

Presentation

- Orbital and/or retro-orbital pain
- Chemosis (arteriolization of conjunctiva)
- Pulsatile proptosis
- Occular and/or cranial bruit
- Deterioration of visual acuity
- Diplopia
- Ophtalmoplegia

Treatment

Detachable Balloon Embolization

- Transarterial through internal carotid
- Transarterial through external carotid
- Transvenous

Spontaneous Intracerebral Hemorrhage

Etiologies

- Hypertension
- Vascular anomalies (aneurism, AVM)
- Arteriopathies
- Brain tumor
- Coagulation disorders

Clinical

- Severe headache
- Vomiting
- Alterations of LOC
- Contralateral hemiparesis
- Contralateral hemisensory loss

Evaluation

CT

Treatment

Indications for surgery must be individualized based on

- the patient's neurologic condition
- size and location of hematoma
- patient's age
- patient's expressed wishes and the family's wishes concerning "heroic" measures in the face of catastrophic illness.

Surgery is no better than medical management, and both have little to offer

Indications for surgery

- Symptomatic lesions
- Marked mass effect, edema, midline shift
- Moderate volume

  too little for surgery < 10-50 cc < poor outcome
- Rapid deterioration
- Favorable location (lobar, non-dominant hemisphere)

Outcome

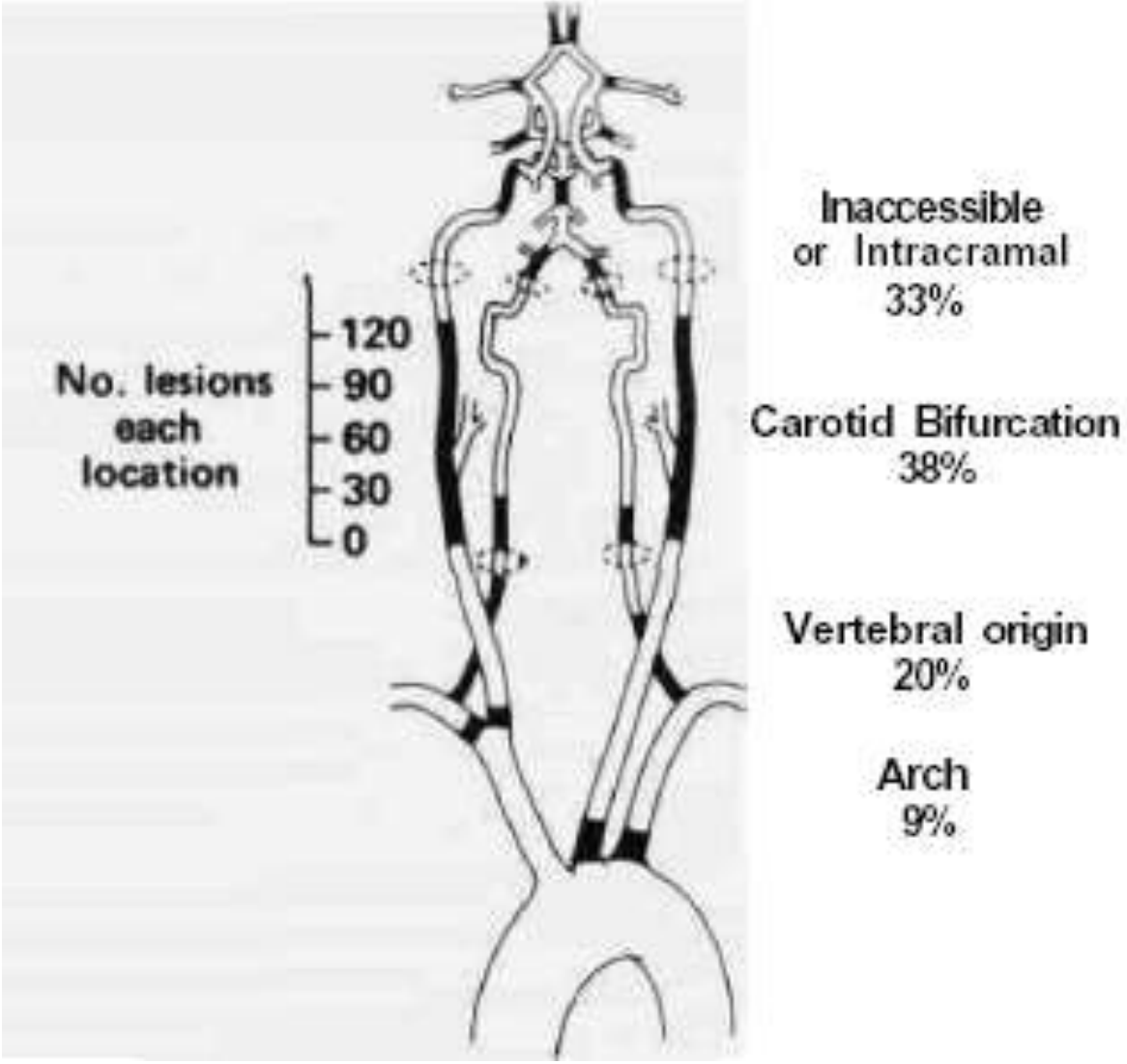
30-day mortality rate is 44%

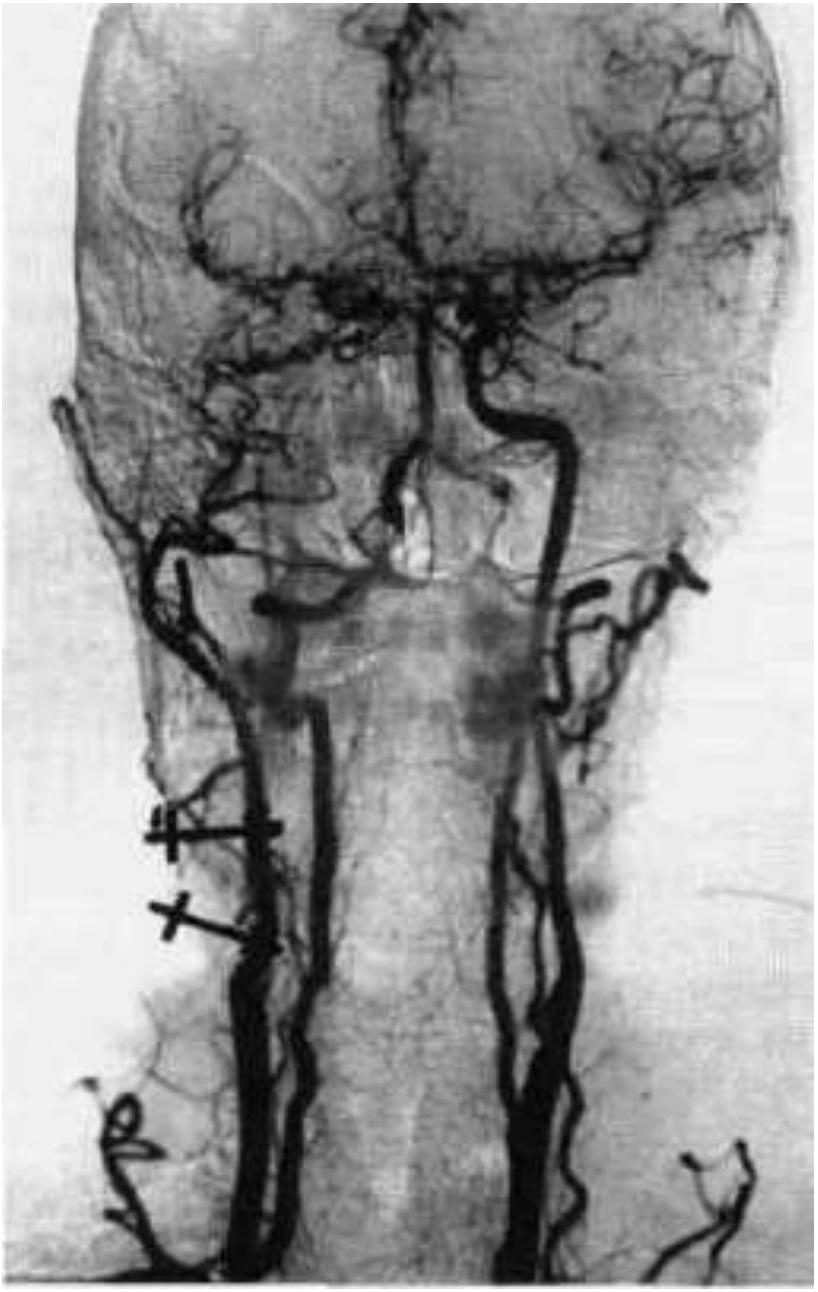
Surgical treatment of stroke

Classification

1. Transient ischemic attack (TIA)
2. Prolonged reversible ischemic neurological deficit (PRIND or RIND)
3. Stroke in evolution
4. Complete stroke

Location and incidence of significant atherosclerotic lesions





Arch aortogram shows nonfilling of the right ICA

Surgical management

- Decompressive craniectomy for progressive neurologic deterioration due to cerebral edema
- Carotid endarterectomy for high grade carotid stenosis
- Extra-intracranial shunting

Endarterectomy

In patients who have a high-grade stenosis of the internal carotid artery, endarterectomy restores normal flow through the carotid system, and removes the source of emboli.

CAROTID ENDARTERECTOMY

Indications

The clinical syndromes to which carotid endarterectomy may be applied are

- transient ischemic attacks,
- small infarcts with mild residual neurological deficits,
- the asymptomatic carotid bruit,
- acute neurological deficit that is progressing or fluctuating.

EXTRACRANIAL-INTRACRANIAL BYPASS SURGERY

- The extracranial-intracranial bypass (ECIC) is a surgical procedure that involves the construction of an end-to-side anastomosis between a branch of the external carotid and the middle cerebral artery.
- The superficial temporal artery is more commonly used, the occipital artery being used less commonly.

Indications

1. Focal TIAs with signs and symptoms related to an inaccessible internal carotid artery or middle cerebral artery (MCA) stenosis or occlusion.
2. Slow strokes, in which there is progressive neurologic dysfunction often mimicking symptoms of a spaceoccupying lesion.
3. Progressive dementia: multiple occlusions may cause hypoperfusion with dementia.
5. Acute stroke.