

Ischemic Stroke, Arterio-Occlusive Diseases and Cerebral Venous Sinus Thrombosis (CVST)

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Abstract: There is an excess of 13.7 million strokes per year globally. Globally, there are more than eighty million people currently alive who have had a stroke. Its management, cost and morbidity have a huge social and economic impact on families, societies and countries. Stroke has important predisposing factors that need to be addressed for stroke prevention. Time is a very important factor in the management of acute stroke to avoid mortality and morbidity. An early diagnosis and initiation of appropriate management, including IV thrombolysis, mechanical thrombectomy and emergency surgical interventions (decompression or revascularization), are utterly precious for reasonable outcomes. This chapter will discuss the pathophysiology, presentation, imaging, principles of medical and surgical management, and prevention of ischemic stroke. This chapter will also outline a short discussion of atherosclerotic cerebrovascular disease, cerebral arterial dissection and cerebral venous sinus thrombosis (CVST).

Abbreviations

AAD	atlanto-axial dislocation	ABG	arterial blood gas
ACZ	acetazolamide	ACA	anterior cerebral artery
ADC	apparent deficient co-efficient	AICA	anterior inferior cerebellar artery
ATA	anterior temporal artery	AVM	arterio-venous malformation
BA	basilar artery	BHS	bow hunter's stroke
BP	blood pressure	CAD	cerebral arterial dissection
CBC	complete blood count	CBF	Cerebral blood flow
CBV	cerebral blood volume	CCA	common carotid artery
CCU	coronary care unit	CEA	carotid endarterectomy
CHF	chronic heart failure	CRV	cerebrovascular reserve
CT	computed tomography	CTA	computed tomographic angiogram
CTV	computed tomographic venography	CSF	cerebrospinal fluid
CXR	chest X-ray	CVA	cerebrovascular accident
CVST	cerebral vein and dural sinus thrombosis	DSA	digital subtraction angiogram
DTI	diffusion tensor imaging	DW	diffusion weighted
ECA	external carotid artery	ECG	Electrocardiogram
EC-IC	extracranial-intracranial	EEG	electro encephalogram
ER	emergency room	EVD	external ventricular drainage
FMD	fibro muscular dysplasia	FND	focal neurological deficit
fMRI	functional magnetic resonance imaging	GI	Gastrointestinal
HTN	hypertension	IA	Intraarterial
ICA	internal carotid artery	ICH	intracerebral hemorrhage
ICP	intra cranial pressure	IIH	idiopathic intracranial hypertension
IV	intra venous	LP	lumbar puncture
MCA	middle cerebral artery	MI	myocardial infarction
MRI	magnetic resonance imaging	MRA	magnetic resonance angiogram
MRV	magnetic resonance venography	OA	occipital artery
OEF	oxygen extraction fraction	ONSF	optic nerve sheath fenestration
OP	opening pressure	PAN	polyarteritis nodosa
PCA	posterior cerebral artery	PET	positron emission tomography
PICA	posterior inferior cerebellar artery	PT	prothrombin time
PTT	partial thromboplastin time	PW	perfusion weighted
RIND	reversible ischemic neurological deficit	rtPA	recombinant tissue plasminogen activator
SAH	subarachnoid hemorrhage	SCA	superior cerebellar artery
SPECT	single-photon emission computed tomography	SSS	superior sagittal sinus
TCD	transcranial Doppler	TIA	transient ischemic attack
tPA	tissue plasminogen activator	TS	transverse sinus

U/A urine analysis
 VBI vertebrobasilar insufficiency

VA vertebral artery

1. Ischemic Stroke

There is an excess of 13.7 million strokes per annum globally. The yearly incidence rate is 185/100,000 population. The global prevalence rate is 1083/100,000. Globally, there is an excess of eighty million people currently alive who have had a stroke (GBD 2016 Stroke Collaborators 2019).

1.1. Cerebral Blood Flow and Its Relation to Ischemia

Cerebral blood flow (CBF) at rest is 45–60 mL blood/minute/100 mL of brain tissue.

CBF < 20 mL/min/100 gm of brain tissue results in cerebral ischemia.

CBF < 16–18 mL/min/100 gm of brain tissue causes a flat EEG.

CBF < 15 mL/min/100 gm of brain tissue causes physiologic paralysis.

CBF < 10 mL/min/100 gm of brain tissue leads to infarction (Greenberg 2010).

1.2. Suddenly Developed Focal Neurological Deficit

Patients presenting to the hospital emergency department with sudden development of a new focal neuro-deficit (Greenberg 2010; Lindsay et al. 2011):

- Neoplasm, epilepsy or psychogenic—5%;
- Neurovascular (stroke)—95%;
 - Ischemic infarct—85%;
 - Unknown cause—41%;
 - Lacunar infarct—21%;
 - Cardiogenic embolus—16%;
 - Large artery lesion—11%;
 - Tandem arterial pathology—10%;

Hemorrhagic stroke—15%;

- Intracerebral hemorrhage (ICH)—11%. “Hypertensive” hemorrhage, amyloid angiopathy;
- SAH (aneurysmal, AVM)—5%;

Venous infarction—a small proportion of strokes.

1.3. Risk Factors for Stroke

Risk factors are shown in Table 1.

Table 1. Major and other risk factors.

Major Factors	Other Factors
Hypertension	Age
Hypertension—Major risk factor for brain infarction and hemorrhage.	Sex (male > female)
Cardiac disease	Race
Cardiac failure and cardiomegaly, and arrhythmias, valvular diseases and patent foramen ovale.	Hereditary
Diabetes mellitus	Sedentary lifestyle
Smoking	Diet and environment
Hyperlipidemia	Polycythemia
	Oral contraceptives
	Heavy alcohol consumption

Source: Authors’ compilation based on data from Greenberg (2010).

1.4. Pathophysiology of TIA and Infarction

When the CBV is compromised (by arterial diseases), i.e., below the 20 mL/min/100 gm brain tissue and a drop-in blood pressure or perfusion pressure (such as arrhythmia, thrombus or embolus) leading the CBV below 15 mL/min/100 gm of brain tissue, it can lead to a TIA. If the CBV improves, the TIA recovers, and if the CBV does not improve and instead decreases under the 10 mL/min/100 gm of brain tissue, a cerebral infarction ensues.

1.5. Cerebrovascular Reserve (CRV) and Reactivity

The CRV may be evaluated with a xenon-enhanced CT, perfusion CT, TCD, SPECT or an MRI with perfusion images. The reaction of the CBF to a vasodilator challenge with 1 gm of IV acetazolamide (ACZ) is classified in Table 2.

Table 2. Types of CRVs and reactivity.

Type	Description
Type I	Normal, baseline CBF with 30–60% rise after ACZ challenge
Type II	Reduced baseline CBF with a response (blunted) of <10% rise or <10 mL/100 g/min absolute rise following ACZ challenge
Type III	Reduced baseline CBF with a paradoxical fall in regional CBF after ACZ challenge, alluding a steal phenomenon in areas with the most dilated vessels at the baseline

Source: Authors' compilation based on data from Greenberg (2010).

1.6. Penumbra and Treatment Rationale of Cerebral Infarction

With the total lack of blood flow to cerebral neurons, neuronal death occurs within 2 to 3 min. However, in most infarctions, there is a recoverable penumbra (tissue at risk) that survives for a period of time due to poor collateral flow perfusion. If a local cerebral edema from the lesion progresses, these collaterals are compromised and the ischemic penumbra progresses to infarction if flow is not restored. The prevention of this secondary damage drives the management of stroke and has prompted the evolution of dedicated primary stroke centers that offer proper and timely triage and treatment for all potential stroke patients. The current standard of care requires the administration of IV tPA (tissue plasminogen activator) to all eligible patients. In centers with advanced capabilities (comprehensive stroke centers), other management options are also offered (Greenberg 2010; Lindsay et al. 2011).

1.7. Transient Ischemic Attacks (TIA)

TIA's are episodes of focal neuro-deficit due to poor blood circulation to the brain. The attacks come on suddenly, last for 24 h or less, and leave no lingering neuro-deficiency. These assaults could be a sign of an impending cerebral infarction. Migraine, partial seizures, hypoglycemia, syncope and hyperventilation are all examples of transient neurological malfunction. After a TIA, 5% of patients suffer cerebral infarction within one week and 12% within three months. A TIA warning occurs in about 10% of people who have a stroke (Greenberg 2010; Lindsay et al. 2011).

1.8. Clinical Presentation of an Ischemic Stroke

Patients may present with (TIA's) or features of infarctions. Clinical features depend on the artery that is affected by the disease process (Lindsay et al. 2011).

1.8.1. Clinical Pictures of a TIA

Ninety percent (90%) of TIA's are anterior-circulation (ICA territory) TIA's that include hemiplegia, hemisensory disturbance, dysphasia and mono-ocular blindness (amaurosis fugax). Seven percent (7%) are posterior-circulation (vertebrobasilar territory) TIA's that include unconsciousness, bilateral sensory and motor disturbances, binocular blindness, diplopia, vertigo, tinnitus and dysarthria. Three percent (3%) are indistinguishable between anterior- and posterior-circulation TIA's.

1.8.2. Clinical Pictures of an Infarction

Large Vessel Occlusion—Internal Carotid Artery (ICA) Occlusion

Increasing lumen constriction and thrombosis, or a repeated emboli may manifest in a 'stuttering' manner. The severity of the deficiency varies. An asymptomatic ICA occlusion is possible, or a catastrophic infarction may occur. The initial prodromal symptoms include amaurosis fugax, and a transient hemi-motor or hemisensory disturbance. In the worst-case scenario, the symptoms may be a dwindling of level of consciousness; contralateral

homonymous hemianopia, hemiplegia, hemisensory disturbances and gaze palsy; or global aphasia (in the case of the dominant hemisphere). There is an absence of ICA pulsation at the angle of the jaw.

Large Vessel Occlusion—Anterior Cerebral Artery (ACA) Occlusion

A thrombus or embolus can obstruct the ACA. The clinical symptoms vary depending on the location of the blockage (most commonly in relation to the anterior communicating artery) and anatomical variance; for example, an enlargement of the anterior communicating artery might cause both anterior cerebral arteries to emerge from one side. A pre-communicating ACA blockage is well tolerated and may be asymptomatic. In most cases, a distal ACA blockage causes paralysis and cortical sensory loss in the contralateral lower limb, as well as incontinence. Cerebral paraplegia with the paralysis of both lower limbs, cortical sensory loss and incontinence can result from a proximal ACA occlusion when both ACAs originate from one side or an azygos ACA occlusion. The grasping reflex, snout and palmo-mental reflex may be present. An altered level of consciousness and akinetic mutism may be present in the case of an infarction of both sides of the frontal lobe.

Large Vessel Occlusion—Middle Cerebral Artery (MCA) Occlusion

The MCA produces deep branches (lenticulostriate perforating arteries) that supply the anterior limb of the internal capsule and a portion of the basal nuclei. It then travels to the insula of the Sylvian fissure on the lateral surface of the cerebral hemisphere. It produces cortical branches here such as the temporal, frontal and parietal cortical branches.

Clinical features: An embolus or thrombus can obstruct the MCA. The clinical signs and symptoms vary depending on the blockage site and whether the dominant or nondominant hemisphere is affected. When specific cortical branches are occluded, the clinical symptoms are less severe. For example, the involvement of the parietal branches alone can cause Wernicke's dysphasia without limb paresis or sensory problems. Tiny infarcts may be caused by the MCA's deep branches (perforating arteries) (lacunar infarct).

Occlusion at the insula: Again, the symptoms depend on which branch or branches are impacted. If all branches are compromised, there will be contralateral hemiplegia (leg largely spared), contralateral hemianesthesia and hemianopia, aphasia (in the case of the dominant hemisphere), neglect of the contralateral side and clothing difficulties (in the case of the non-dominant hemisphere).

Large Vessel Occlusion—Vertebral Artery (VA) Occlusion

The VA arises from the subclavian artery bilaterally and passes through the foramina transversarium of the cervical vertebrae. It enters the cerebral cavity through the foramen magnum after piercing the dura and arachnoid tissue. It joins with its companion at the pons' lower border to produce the basilar artery. Before creating the basilar artery, the VA and its branches are distributed on the medulla and inferior surface of the cerebellum.

Clinical features: When the VA is blocked down in the neck, anastomotic channels compensate well. When one of the vertebral arteries is hypoplastic, the blockage of the other is analogous to the occlusion of the basilar artery. The flow of the vertebral artery is solely responsible for the posterior inferior cerebellar artery (PICA). As a result, the blockage of the vertebral artery can cause PICA syndrome. The vertebral artery's close proximity to the cervical spine is crucial.

Injury to the intervertebral foramina or the atlanto-axial joints as a result of subluxation can cause intimal damage, thrombus development and embolization in rare cases. Intermittent vertebrobasilar insufficiency can be caused by the compression of the vertebral artery during neck extension.

Posterior Inferior Cerebellar Artery Syndrome (PICA/Lateral Medullary Syndrome)

Cerebellar features include dysarthria, ipsilateral limbs, vertigo, ataxia and nystagmus; and lateral medullary features include ipsilateral Horner's syndrome, and ipsilateral pain and temperature sensation loss in face, ipsilateral laryngeal and pharyngeal paralysis, and contralateral pain and temperature sensation loss in the trunk and limbs.

Large Vessel Occlusion—Basilar Artery (BA) Occlusion

From the medulla to above, the BA nourishes the brain stem, finally dividing into posterior cerebral arteries. Posterior cerebral arteries, long circumflex branches and paramedian branches are the three types of branches of BA.

Clinical features: Diplopia, visual field loss, occasional memory disturbance and a slew of other brain stem symptoms such as vertigo, ataxia, paresis and paresthesia are all frequent prodromal symptoms. Following BA occlusion, complete basilar syndrome develops, which includes the loss of awareness or coma, bilateral motor and sensory deficits, cerebellar symptoms and cranial nerve indications that indicate the amount of blockage. The clinical picture, on the other hand, is diverse and may be asymptomatic. Occlusion of the top of the basilar artery causes infarction of the lateral midbrain, and thalamic, occipital and medial temporal lobes. Visual loss, pupillary abnormalities, gaze palsies, reduced conscious level and behavioral disorders are all symptoms of hemiballismus.

'Locked-in' syndrome and lacunar infarction are caused by obstruction of the paramedian perforating artery.

Large Vessel Occlusion—Posterior Cerebral Artery (PCA) Occlusion

PCAs are the basilar artery's terminal branches. Midbrain structures, the choroid plexus and the posterior thalamus are all served by small perforating branches. The posterior temporal artery supplies the undersurface of the temporal lobe, while the parieto-occipital and calcarine arteries nourish the occipital and visual cortices.

Clinical features: Perforating branches and structures are affected by the proximal blockage of PCA by a thrombus or embolism.

The midbrain syndrome includes third nerve palsy with contralateral hemiplegia (Weber's syndrome), thalamic syndromes such as chorea or hemiballismus with hemisensory impairment, and obstruction of cortical vasculature, which result in visual field loss (homonymous hemianopia) but macular vision sparing. Color and object naming may be affected by posterior cerebral infarction in the dominant hemisphere.

BA Branch Occlusion Syndrome

Superior cerebellar artery (SCA) syndrome: cerebellar features, including gait disturbances and limb ataxia; and lateral midbrain features, including ipsilateral Horner's syndrome and hemisensory loss (pain and temperature sensation loss, including in the face).

Anterior inferior cerebellar artery (AICA) syndrome: cerebellar feature, including limb ataxia; and lateral pontine features, including ipsilateral Horner's syndrome and ipsilateral pain and temperature sensation loss in the face; ipsilateral facial weakness; ipsilateral lateral gaze palsy; and contralateral pain and temperature sensation loss in the trunk and limbs.

Lacunar Infarcts

Different perforators from anterior and posterior circulation can produce different lacunar stroke syndromes.

Anterior circulation lacunar stroke—pure sensory syndrome, pure motor syndrome, etc.

Posterior circulation lacunar stroke—dysarthria/clumsy hand syndrome, ataxic hemiparesis, etc.

1.9. Evaluation and Investigations

1.9.1. History—Key Components to Consider

- Time when the patient was last observed to be normal;
- Current deficit/s and clinical presentation;
- Stroke scale (such as the NIH) score should be assessed and recorded;
- Causes for not starting IV tPA (if any) must be written (Greenberg 2010; Lindsay et al. 2011; Awad 2005).

1.9.2. Investigations

1. CT scan of brain, perfusion CT with a CTA of the brain and neck vessels from the arch of aorta;
2. MRI of the brain as per the ischemic stroke protocol (routine images with DW, ADC, PW images, DTI and tractography, MRA and MRV of the head and neck vessels and fMRI of the brain);
3. Carotid Doppler;
4. Cerebral DSA;
5. CXR P/A view, ECG and echocardiogram;
6. Routine hematological tests (Greenberg 2010; Lindsay et al. 2011; Awad 2005).

1.9.3. Computed Tomography (CT) Scan, CT Angiogram and Perfusion CT

CT Scan

On presentation with the signs and symptoms of a potential stroke, a brain CT scan without contrast should be carried out urgently to exclude hemorrhage (intra- parenchymal or SAH), early signs of ischemia, hematoma, old infarcts or injuries, and other pathologies (e.g., tumor).

Hyperacute (<6 h after stroke): Early signs of an infarction involving large areas of the MCA territory correlate with poor outcomes. Early findings may include the following:

1. Hyperdense artery sign (Figure 1): low sensitivity, but helpful if present;
2. Focal low attenuation within the gray matter;
3. Loss of the gray–white matter interface;
4. Attenuation of the lentiform nucleus;
5. Mass effect
 - A. Early: effacement (obscuration) of the cerebral sulci (often subtle);
 - B. Late: midline shift in large territory infarction;
6. Absence of the insular ribbon (hypodensity involving the insular area);
7. Enhancement: occurs in only 33%, where stroke becomes isodense (called the “masking” effect) or hyperdense with the normal brain, and, rarely, may be the only sign of an infarction.



Figure 1. Axial CT scan of the brain showing a right MCA infarct with the ‘hyperdense MCA’ sign (arrow marked). Source: Figure by authors.

24 h: Most strokes can be identified as a low density by this time.

1–2 weeks: Strokes are starkly demarcated.

3 weeks: Stroke area approaches CSF density.

In 5–10%, there may be a short window (approximately day 7–10) where the stroke turns isodense, known as the “fogging effect”. A IV contrast will generally visualize these.

Mass effect: common between days 1 and 25. Then, atrophy is usually seen by week 5 (2 weeks at the earliest). Serial CT scans of the brain have demonstrated that the midline shift increases following ischemic stroke and reaches its highest 2 to 4 days after the event.

Calcifications: Only 1–2% of strokes calcify. Thus, in an adult person, calcifications almost rule out a stroke.

Hyperdense artery sign (Figure 1): The cerebral vessel (usually the MCA) appears as a high density on an unenhanced CT, pointing out an intra-arterial thrombus or embolus. It is found in 12–34% of patients within 24 h of stroke. Sensitivity for an MCA occlusion is low, but specificity is high (although it may also be seen with a carotid dissection).

Contrast enhancement:

1. Many infarcts take up contrast by day 6, most by day 10, and some will contrast enhance up to 5 weeks;
2. Rule of the 2s: 2% contrast enhance at 2 days, 2% contrast enhance at 2 months;
3. Gyral contrast enhancement (“ribbon” enhancement) is frequent, commonly seen by 1 week, and a differential includes inflammatory infiltrating lesions such as lymphoma, neuro-sarcoidosis, etc.;
4. There should not be contrast enhancement at the same time when there is a mass effect (Greenberg 2010; Marks et al. 1999; Tomandl et al. 2003; Lyden et al. 1994; Sims et al. 2005).

CTA

CTA is useful for assessing the location and extent of vascular occlusion in acute ischemic stroke. The findings can direct treatment toward endovascular or microsurgical options when a proximal or significant large vessel occlusion is seen.

Perfusion CT

A perfusion CT identifies salvageable penumbra as a region of mismatch between the CBF and CBV. An infarcted core has a decreased CBF within a region of a decreased CBV (CBF/CBV match). A decreased CBV without a decrease in the CBF (CBF/CBV mismatch) represents a potentially salvageable penumbra.

1.9.4. Magnetic Resonance Imaging (MRI), MR Angiogram (MRA) and MR Perfusion

MRI

With newer, faster acquisition times, MRIs (Figures 2 and 3A,B) are increasingly being utilized in the hyper-acute setting, at times replacing CTs for an initial evaluation. They are more sensitive than CTs (especially DWI-MRIs, particularly within the first 24 h after stroke), especially for imaging the brainstem or a cerebellar infarction.

Contrast MRI: (not used often) several enhancement patterns can be seen:

1. Intravascular contrast enhancement occurs in 75% of 1–3-day-old cortical infarcts, and is thought to be caused by a slow flow and vasodilation (thus, it is not seen with a complete occlusion). It is possible that certain parts of the brain are at a risk of an infarction.
2. Dural enhancement is found in 35% of 1–3-day-old cortical strokes.
3. Parenchymal enhancement has shown as a cortical or subcortical gyral ribbon enhancement in the past. It may not be noticeable for the first 1–2 days, but by the end of the week, it reaches 100% (Greenberg 2010; Lindsay et al. 2011; Awad 2005; Barber et al. 1998).

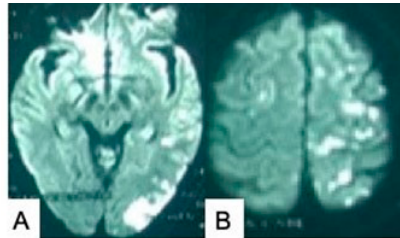


Figure 2. (A,B) MRI of brain axial DW images showing “strings of beads” as watershed infarcts under ischemic conditions. Source: Figure by authors.

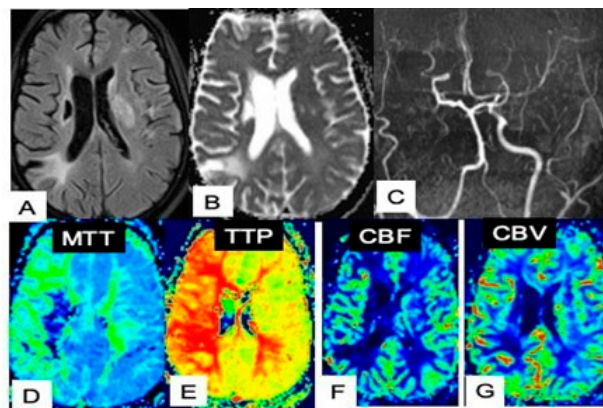


Figure 3. MRI of the brain. (A) Axial FLAIR and (B) axial T2W image showing multiple infarcts and ischemic zones in both hemispheres. (C) MRA of the brain showing an occlusion of the right ICA and scarcity of the right MCA vessels. (D–G) Perfusion MRI showing ischemic “penumbra”. Source: Figure by authors.

MRA

An MRI (Figure 3C) is useful for assessing the site, as well as the extent of a vascular occlusion in acute ischemic stroke, like CTA.

The findings can direct treatment toward endovascular or microsurgical options when a proximal or significant large vessel occlusion is seen.

MRI Perfusion

An MRI perfusion (Figure 3D–G) is akin to a CT perfusion, where areas of a matched DWI and PWI abnormality are thought to represent the infarcted tissue. PWI abnormalities that do not have a DWI correlation are thought to represent a potentially salvageable penumbra.

1.9.5. Emergency Cerebral Digital Subtraction Angiography (DSA)

Digital subtraction angiography is rarely needed and is usually carried out with therapeutic intervention. A DSA should be avoided in unstable patients with a severe disabling neuro-deficit. Indications include the following:

1. An early stroke in the carotid circulation combined with a history of amaurosis fugax, bruit or retinal emboli, etc., could indicate a growing carotid stenosis, thrombogenic ulcerated plaque or carotid dissection;
2. If the diagnosis is still up in the air (e.g., aneurysm, vasculitis);
3. In the face of a growing stenosis, the quick recovery of neuro-deficits suggests a carotid TIA.

The findings include the following:

1. Cut off sign: the vessel abruptly ends at the point of impediment;
2. String sign (Figure 4): a narrow strand of contrast in an artery with a stenosis of high severity;
3. "Luxury perfusion": reactive hyperemia is a well-known brain tissue reaction to damage (Greenberg 2010; Lindsay et al. 2011; Awad 2005).



Figure 4. Cerebral DSA right carotid injection showing a high-grade ICA stenosis (string sign is arrow-marked). Source: Figure by authors.

1.10. Management

1.10.1. Aims of Treatment and General Management

Aims of general management and treatment are shown in Table 3.

Table 3. The aims of treatment and general management are shown in the boxes.

Aim of Treatment	General Management
<ul style="list-style-type: none"> • Reopening of blocked vessels • Preclusion of progression of the present event • Avoidance of immediate complication • Prevention of the development of subsequent events and complication/s • Rehabilitation of the patient 	<ol style="list-style-type: none"> 1. Management of airways and oxygenation 2. Maintenance of hydration 3. Maintenance of blood sugar 4. Treatment of hypertension if BP > 185/110 mm of Hg

Source: Authors' compilation based on data from Greenberg (2010), Lindsay et al. (2011) and Awad (2005).

1.10.2. Specific Management

Within 4.5 h of initiation of the event:

- IV thrombolysis (with tPA);
- Failures to respond to IV thrombolysis;
 - (i) intraarterial tPA or
 - (ii) mechanical embolectomy/clot disruption.

4.5–6 h after onset:

- Intra-arterial tPA/rtPA or
- Mechanical embolectomy/clot disruption.

6–9 h after onset:

- Check perfusion with CT perfusion or MRI-DWI and PWI
- Mechanical embolectomy;
- Balloon angioplasty and stenting likely works by buttresses the clot.

A higher efficacy is noticed in the failure of other available options.

(These times are more applicable to anterior circulation strokes. Posterior circulation occlusions may be treated more aggressively, e.g., IA tPA has been used up to 12 h).

Contra-Indication of Thrombolysis

The contra-indications of thrombolysis include an uncertain onset, spontaneous improvement, brain injury or previous stroke in the previous 3 months, GI surgery in the preceding 21 days, BP > 180/110, on anticoagulant, seizure and hypodensity on CT.

Complications of Thrombolysis

The complications of thrombolysis include intracranial hemorrhage (ICH) following IV tPA.

There is a chance of an increased risk of symptomatic intracerebral hemorrhage with the thrombolysis (6.4–8.8%).

Except in the rare case of a big hematoma, ICH has no bearing on the outcome.

Management of Post-Thrombolysis ICH

1. Discontinuation of tPA infusion and obtaining STAT head CT;
2. Lab investigations: PT, APTT, fibrinogen and platelet count, as well as type and cross;
3. Preparation to inject 6–8 units of cryoprecipitate-containing factor VIII;
4. Preparation to administer 6–8 units of platelets.

Emergency external ventricular drain (EVD) placement or an ICH evacuation, which is needed rarely (Greenberg 2010; Lindsay et al. 2011; Awad 2005; Paciaroni et al. 2008).

1.10.3. Management of Patients Not Undergoing Antithrombotic Therapy

The following recommendations for initial treatment should be continued 48 h following the last neuro deterioration:

1. Frequent vitals and neuro-status checks.
2. Bed rest and nothing except oral and IV fluids.
3. Laboratory investigations:
 - (i) Routine: CBC + platelet count, electrolytes, PT/PTT, U/A, ECG, CXR, ABG;
 - (ii) At 24 h: CBC, platelet count, cardiac profile, lipid profile, ECG.
4. Oxygen (O₂) inhalations as needed.
5. Nursing care:
 - (i) Indwelling urinary Foley catheter if consciousness is impaired or indicated;
 - (ii) Accurate input–output chart maintenance;
 - (iii) Control of blood sugar and maintenance of normoglycemia;
 - (iv) Adequate hydration and avoidance of overhydration.

6. Treatment of CHF and arrhythmias. Patients with myocardial ischemia and neurological deficit should be admitted to the CCU.
7. Blood pressure (BP) containment:
 - (i) For patients presenting with an HTN: baseline BP must be taken into account. If the patient has a known case of hypertension, the treatment endpoints for the HTN have lower limits at a systolic BP of 180–185 mmHg and diastolic BP of 105–110 mmHg. If the patient has no prior history of an HTN, the treatment endpoints for an HTN have lower limits at a systolic BP of 160–170 mmHg and diastolic BP of 95–105 mmHg.
 - (ii) For patients presenting with hypotension (DBP < 70 or SBP < 110):
 - a. Administration of IV fluids;
 - b. Vasopressors if fluid ineffective or contraindicated.
8. Medications:
Aspirin 300 mg daily for 14 days or Clopidogrel where aspirin is intolerant.
9. Transfer-to-stroke unit:
Multidisciplinary care in a stroke unit has been shown to enhance the outcome of stroke patients.
10. Assessment of swallowing capacity:
After a stroke, aspiration pneumonia is a common consequence. Swallowing should be assessed and a nasogastric tube for fluids and food used if necessary to reduce this risk.
 - (i) Swallowing is dangerous;
 - (ii) Early mobilization as soon as feasible and patients should be assisted in sitting up and mobilizing.
11. Special situations
 - (i) Decompressive hemicraniectomy
A limited number of young individuals (under 70 years old) with big middle cerebral artery strokes develop significant cytotoxic cerebral edemas that are resistant to medical treatment after 24–72 h. Surgical decompression can save a patient’s life and allow them to recuperate within a reasonable amount of time.
 - (ii) Other neurosurgical procedures
When an edema causes compression of the posterior fossa and concomitant hydrocephalus, patients with massive cerebellar infarcts can decline 24–48 h following their stroke. Decompression of the posterior fossa can save a patient’s life, and many patients recover fully.

1.10.4. Preclusion of Further Stroke

The identification of risk factors (Table 4), as well as their amendment to minimize the risk of further stroke forms an essential and standard step in long-term treatment.

Table 4. The strategies for prevention (utilized for the treatment of a TIA).

Prevention of Further Stroke	
(i)	Controlling of hypertension.
(ii)	Stopping of tobacco smoking.
(iii)	Correction of hyper-lipidaemia.
(iv)	Give antiplatelet drugs (Clopidogrel or aspirin) to decrease the rate of reinfarction.
(v)	Removal or treatment of embolic source (long-term use of anticoagulation in atrial fibrillation). Stop anticoagulation in disabling a stroke for 14 days as a risk of hemorrhage more than the benefits.
(vi)	Treatment of vascular inflammatory or inflammatory diseases.
(vii)	Avoidance of prothrombogenic drugs (i.e., oral contraceptives).

Source: Authors’ compilation based on data from Greenberg (2010) and Lindsay et al. (2011).

1.11. Cerebellar Infarction

Cerebellar infarction is seldom seen. Cerebellar infarcts may be categorized as involving the PICA distribution (cerebellar tonsil and/or inferior vermis), superior cerebellar artery distribution (superior hemisphere or superior vermis) or other indeterminate patterns. After developing the signs of brainstem compression, 80% of patients die usually within hours to days. In the majority of cases, the onset is abrupt. The first 12 h following onset are marked by a lack of improvement. Dizziness or vertigo, nausea/vomiting, loss of balance

(frequently accompanied by a fall and inability to get up), headache, truncal and appendicular ataxia, nystagmus and dysarthria are common early symptoms. Later findings include features of a raised ICP for the development of triventriculomegaly and brainstem compression. Clinical findings generally increase between 12 and 96 h following the onset (Greenberg 2010; Lindsay et al. 2011; Chen et al. 1992; Vahedi et al. 2007).

1.11.1. Surgical Indications

If any of the following indications appear and medicinal treatment is ineffective, surgical decompression should be performed as soon as possible. If no intervention is made, the findings proceed in the following order: 1. abducent nerve palsy; 2. ipsilateral gaze loss (compression of VI nucleus and lateral gaze center); 3. peripheral facial nerve paresis (compression of facial colliculus); 4. disorientation and somnolence (perhaps owing to developing hydrocephalus); 5. Babinski sign; 6. hemiparesis; 7. lethargy; 8. tiny but responsive pupils; 9. coma; 10. posturing flaccidity; and 11. ataxic respirations.

The results of a CT scan may be normal in the early stages. Compression or obliteration of the basal cisterns or the fourth ventricle may be modest signs of a tight posterior fossa. MRIs (including DWI) are more sensitive for ischemia, especially in the posterior fossa (Greenberg 2010; Lindsay et al. 2011; Chen et al. 1992; Vahedi et al. 2007).

Suboccipital Craniectomy for a Cerebellar Infarction

Unlike the situation with supratentorial masses causing herniation, patients with a deep unconsciousness from direct brainstem compression who are operated upon without delay can make a good recovery. The operation of choice is a suboccipital decompression to include an enlargement of the foramen magnum with removal of an infarcted cerebellum. (*It is very important to identify a lateral medullary syndrome (LMS) that is not accompanied by a change in the sensorium. There is no place for surgical decompression in the LMS since it indicates primary brainstem ischemia and not compression.*)

1.12. Malignant Middle Cerebral Artery (MCA) Territory Infarction

A very different syndrome happens in up to 10% of stroke patients which can cause mortality of up to 80%. Patients often present with findings of severe hemispheric stroke (hemiplegia and deviated eye, as well as head deviation). Most develop drowsiness shortly after admission. There is a continuation of deterioration during the first 2 days; transtentorial herniation usually occurs within 2–4 days of stroke. Mortalities are often accompanied by severe drowsiness, dense hemiplegia, age > 45–50 years, early parenchymal hypodensity involving > 50% of the MCA distribution on CT scan, midline shift > 8–10 mm, early sulcus effacement, as well as a hyperdense artery sign in the MCA. Aggressive surgical therapies in these patients may reduce morbidity and mortality. Treatment options include the following: 1. usual conventional measures to control ICP (mortality is very high); and 2. hemicraniectomy (decompressive craniectomy) with or without the removal of an infarcted brain, especially temporal brain (Greenberg 2010; Lindsay et al. 2011; Vahedi et al. 2007; Gage et al. 2001).

1.12.1. Hemicraniectomy for Malignant MCA Territory Infarction

A hemicraniectomy may decrease mortality to as low as 32–37%, with a surprising decrease in hemiplegia and dominant-side strokes, with only mild-to-moderate aphasia (better outcomes occur with early intervention, especially if surgery is accomplished before any changes linked to herniation occur). The indication guidelines are as follows: 1. age < 70 years; 2. more seriously considered on the nondominant side (commonly right); and 3. clinical and CT evidence of acute, complete ICA/MCA infarcts and direct signs of inevitable or complete severe hemispheric brain swelling (Greenberg 2010).

1.13. TIA and Minor Infarction—Management

The goal of management to avoid later cerebral infarction includes the establishment of a diagnosis, as well as exclusion of other pathologies causing transient neurological symptoms such as migraine and the correction of predisposing factors. Examination of patients is vital for extracranial and neck vascular disease. Hence, the palpation of carotids and upper limb pulses, along with auscultation of the neck for bruits should be routine. The measuring of blood pressure in both arms and examination of the heart are invaluable (Greenberg 2010; Lindsay et al. 2011; Awad 2005; Brott et al. 2016; Sardar et al. 2017; Naylor 2018; Xu et al. 2017; White et al. 2019; Cai and Peng 2017; Pirau and Lui 2020).

1.13.1. Medical Treatment

- (i) Controlling hypertension;
- (ii) Stopping smoking;
- (iii) Correction of lipid abnormality;
- (iv) Prescription of antiplatelets (aspirin or Clopidogrel) to decrease the reinfarction;
- (v) Removal or treatment of the embolic source.

1.13.2. Surgical and Other Interventional Treatments

Carotid stenosis (Figures 4–6)—High-quality surgical studies have shown that individuals with a carotid stenosis greater than 70% (but not occlusion) and a TIA or minor stroke in carotid territory have a lower risk of a second stroke if they have a carotid endarterectomy. This advantage is contingent on the procedure being performed by a skilled surgeon with a low risk of complications. With higher grades of stenosis and in patients with a hemispheric TIA, the risk of stroke, and consequently, the benefit of surgery is the greatest (as opposed to amaurosis fugax). For patients with lower degrees of stenosis, the risk of consequences outweighs the benefit. In individuals with a stenosis of more than 70%, carotid angioplasty with stenting is an option instead of carotid endarterectomy; however, recent studies have indicated a greater risk of stroke, and perioperative mortality and morbidity are a little higher than CEA, though this was not statistically significant. Other surgical techniques such as the superficial temporal-to-middle cerebral artery (STA–MCA) bypass have no benefit in carotid stenosis.

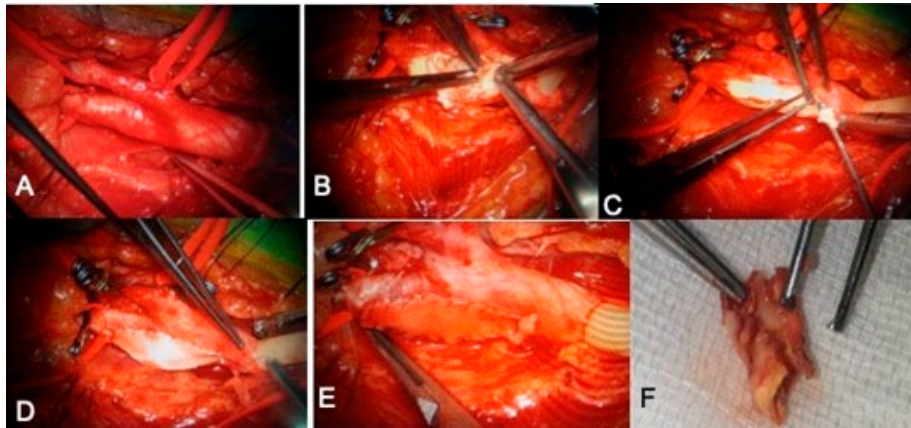


Figure 5. (A–E) Preoperative sequential pictures of the right ICA CEA of the patient from Figure 4. (F) Atherosclerotic plaque after a CEA. Source: Figure by authors.

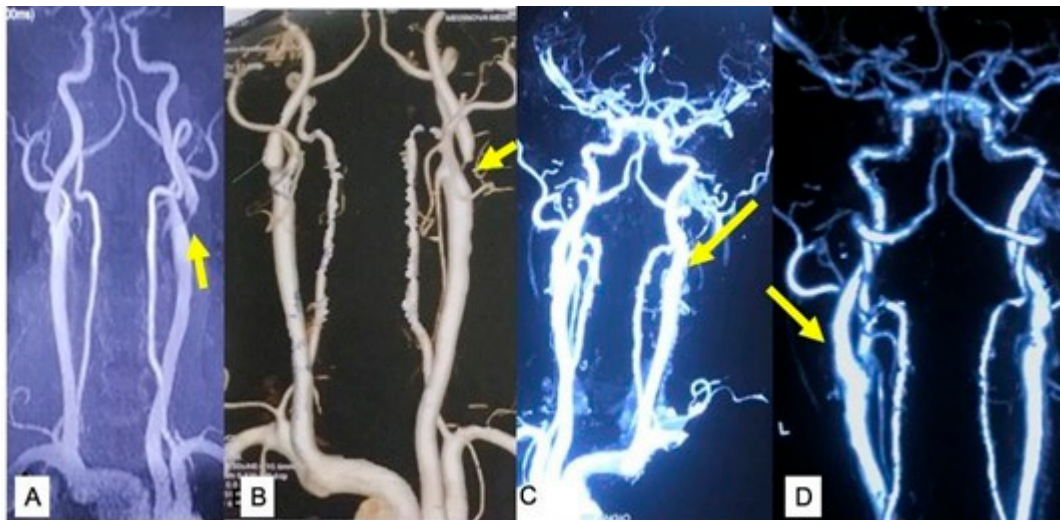


Figure 6. (A) Preoperative MRA showing a high-grade ICA stenosis on the left side. (B) Preoperative CTA of the same patient showing the same ICA stenosis. (C,D) Postoperative CTA on the 1st POD after a CEA of the same patient showing relief of left ICA stenosis. Source: Figure by authors.

1.13.3. Carotid Occlusion

For a carotid occlusion, the surgical options are an EC–IC bypass, CEA with an opening of the ICA and endovascular carotid stenting with a wire-guided catheter-assisted reopening of the ICA. All have definite indications with pros and cons.

1.13.4. MCA or ACA Stenosis

For an MCA or ACA stenosis, an EC–IC bypass is employed in selective cases.

1.13.5. Vertebrobasilar Stenosis

For a vertebrobasilar stenosis, a vertebral artery endarterectomy, VA reimplantation in CCA/ICA/ECA or OA–VA or OA–PICA bypass are employed. The role of these interventions in vertebrobasilar stenosis is not yet established.

2. Atherosclerotic Cerebrovascular Disease

2.1. Carotid Artery

2.1.1. Presentation

The majority (80%) of carotid atherothrombotic strokes occur without warning symptoms. The prevalence of an asymptomatic bruit increases with age (2.3–8.2% in ages 45–75 years). The accuracy of a bruit in predicting an ICA stenosis is 50–83% and sensitivity is as low as 24%. Symptomatic carotid disease may present as a TIA, RIND (reversible ischemic neurological deficit) or CVA, with findings such as amaurosis fugax or monocular blindness (retinal insufficiency or infarction), contralateral motor or sensory TIA (arm and face worse than legs) or language deficits if the dominant hemisphere is involved (MCA symptoms) (Greenberg 2010; Kistler and Furie 2000; Sonecha et al. 2006; Nighoghossian et al. 2005).

2.1.2. Assessment Options

Cerebral DSA

The “gold standard” test for a cerebral DSA is a catheter angiogram (Figures 4 and 6). It cannot be justified as a screening test because it is invasive, costly and risky. Also, unlike duplex Doppler and MRA, it does not provide any information about the thickness of the plaque. It is usually carried out when a simultaneous endovascular intervention is planned if needed.

Duplex Doppler Ultrasound

B-mode image evaluates the artery in a cross-sectional plane, and it is noninvasive. It performs poorly with a “string sign”. Its sensitivity is 88% and specificity 76% (Greenberg 2010; Lindsay et al. 2011; Awad 2005; Kaufmann et al. 2007; Heiserman et al. 1996; Koelemay et al. 2004).

Magnetic Resonance Angiography (MRA)

It is noninvasive and can be carried out at the time as an MRI with ischemic stroke protocol in TIA/stroke cases (Figure 6A). It obviates the need for a DSA in some risky symptomatic cases of a carotid stenosis. Sometimes, it overestimates the degree of a stenosis. An MRA has 91% sensitivity and 88% specificity for extracranial carotid disease. It can detect a thrombus or dissection. An MRA is less operator-dependent than Doppler, but is costlier as well as more time-consuming. An MRA is more difficult to perform if the patient is critical and in contraindicated cases. A high-resolution MRI may also detect vulnerable plaques.

Computed Tomography Angiography (CTA)

The results of a CTA (Figure 6B–D) are comparable to that of an MRA. A CTA can be performed within minutes and can display high-resolution images of all vessels from the arch of the aorta through the intracranial/extracranial vessels, including the surrounding soft tissues. In a meta-analysis, the sensitivity and specificity for the identification of a 70–99% stenosis were 85% and 93%, respectively. A CTA may help detect vulnerable plaques. Another potential advantage is the ability to get CT–perfusion images at the same time.

Choice of Imaging Test/Management Decisions

Doppler, CTA or MRA are usually acceptable initial screening tests. In patients having an abnormal screening test, a common protocol is to go for a second confirmatory noninvasive investigation to reassess the carotid bifurcation before the intervention. If noninvasive investigations are not concordant, a DSA should be carried out before the intervention (Greenberg 2010; Lindsay et al. 2011; Awad 2005; Kaufmann et al. 2007; Heiserman et al. 1996; Koelemay et al. 2004).

2.1.3. Treatment

The treatment alternatives are primarily the following:

1. Medical management with antiplatelet therapy and a lipid-lowering agent;
2. Carotid endarterectomy is a time-tested and gold standard treatment;
3. Endovascular carotid angioplasty and stenting, where perioperative mortality and morbidity are little more than the CEA and the long-term results are yet to be established (Greenberg 2010; Lindsay et al. 2011; Awad 2005; Brott et al. 2016; Sardar et al. 2017; Naylor 2018; Cremonesi et al. 2006).

Asymptomatic Carotid Artery Stenosis

Due to the increased frequency of carotid screening, more asymptomatic cases are being diagnosed. In these cases, the chance of a stroke is 2%/year. Large, randomized trials have revealed that moderate surgical benefits are significantly superior than medical management for asymptomatic stenosis >60%. The patient's age, gender and comorbidities (and therefore life expectancy), as well as perioperative complication rate are the factors to consider in the selection of treatment options.

Practice Guideline 33-1: Asymptomatic Carotid Stenosis

For patients with a neurosurgical risk < 3% and life expectancy > 6 years:

In asymptomatic stenosis >60%, carotid endarterectomy (CEA) should be carried out, and a unilateral CEA is also indicated for asymptomatic stenosis > 50% when an atherosclerotic plaque is large, deep, complex or a cavitated ulcer.

An ipsilateral CEA is recommended for a stenosis > 75% with a contralateral ICA stenosis 75–100%, even in patients with a 3–5% surgical risk.

Once again, endovascular angioplasty with stenting is an option (popular option) in these cases with less favorable (minutely) results than a CEA.

Carotid stenting should be carried out with enough procedural quality levels and should be considered instead of a CEA in the presence of the following:

1. Severe cardiovascular comorbidities (such as heart failure) and severe pulmonary disease.
2. Specific situations:
 - (i) Laryngeal nerve palsy on the contralateral side;
 - (ii) Radio-therapy to the neck;
 - (iii) Previously performed CEA with recurrent restenosis;
 - (iv) High cervical internal carotid/below the level of a clavicle common carotid stenosis;
 - (v) Severe tandem stenosis (Greenberg 2010; Lindsay et al. 2011; Awad 2005; Brott et al. 2016; Sardar et al. 2017; Naylor 2018; Cremonesi et al. 2006).

2.1.4. Totally Occluded Internal Carotid Artery

Introduction

About 10–15% of patients presenting with an ICA territory infarct or TIAs are seen to have a total internal carotid artery occlusion (Figures 7–9). The prevention of a second stroke in symptomatic individuals with a complete carotid blockage is still a difficult task. Following a stroke, the overall rate of a subsequent stroke is 7% per year for all strokes, and 5.9% for ischemic stroke ipsilateral to the blocked carotid artery. These risks persist in spite of treatment with antiplatelets and anticoagulants. The prevalence of an asymptomatic carotid occlusion is unknown, and the incidence of an ipsilateral stroke in never-symptomatic carotid stenosis is negligible.

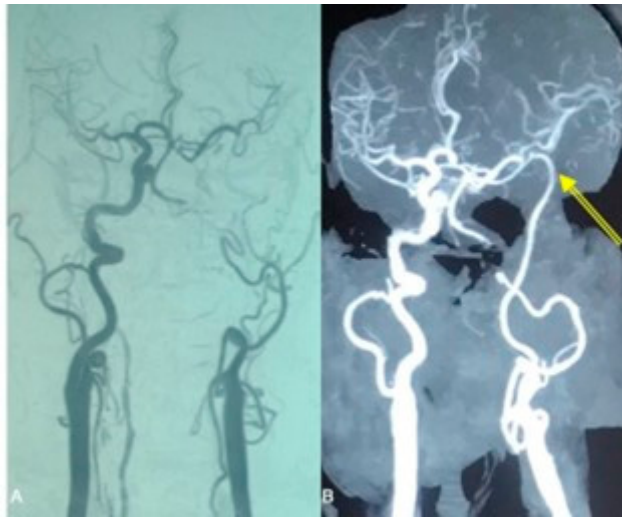


Figure 7. (A) CTA showing a chronic occlusion of the left ICA and both VA with presented with a “Crescendo TIA”. (B) CTA after an urgent left-sided intermediate flow EC-IC bypass (CCA-RAG-MCA) in the same patient. Source: Figure by authors.

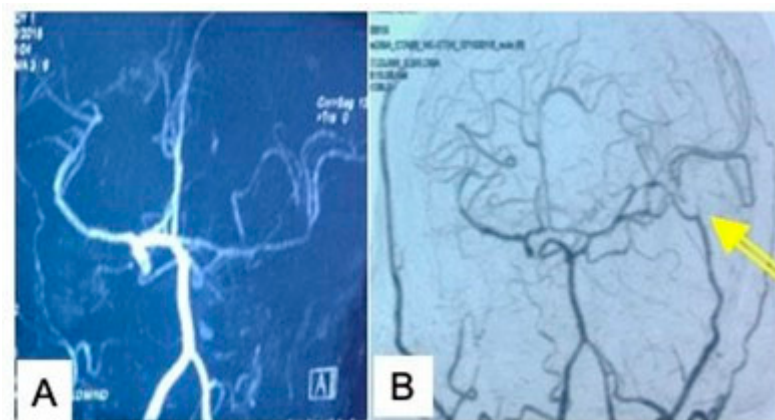


Figure 8. (A) Preoperative MRA of the brain showing a bilateral ICA occlusion presented with a recurrent TIA and recurrent strokes. (B) Postoperative CTA after a left STA-MCA bypass (arrow-marked). Source: Figure by authors.

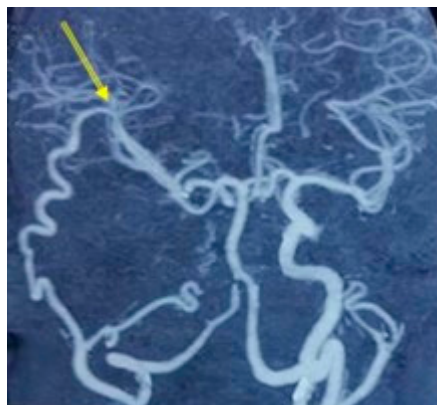


Figure 9. Post-STA-MCA bypass CTA in the case of a right ICA occlusion showing a “robust STA-MCA bypass”. Source: Figure by authors.

Presentation

Three types of CVA can occur with an acute carotid artery occlusion:

1. Stump emboli can produce cortical infarcts. Emboli usually go up the external carotid, then through an ECA-ICA anastomosis into the ICA to the embolic infarct.
2. Whole-hemisphere infarction.
3. A watershed infarct hemiparetic TIA (53%), dysphasic TIA (34%), furred neuro-deficit (21%), crescendo TIAs (21%), amaurosis fugax (17%) and an acute hemiplegia (6%) can occur in symptomatic patients. Patients may have the so called "slow carotid stroke" of a carotid occlusion, which is a stuttering progressive stroke. An MRI may show the so called "string of pearls" sign (small areas of intra-parenchymal increased density on DWI) of watershed-type infarcts. Of patients with an acute ICA occlusion with profound neurological deficit, 2–12% make reasonably good recovery, 40–69% have severe neuro-deficit and 16–55% die by the time of follow-up.

Treatment and Surgery

Options include the following:

- Endarterectomy;
- Fogarty balloon catheter embolectomy (utilizing a no. 2 French catheter with a 0.2 mL balloon gently passed 10–12 cm up the ICA from a small arteriotomy made distal to the atheromatous plaque);
- Extracranial-intracranial (EC-IC) bypass (Figures 7–9);
- Endovascular thrombolysis and stenting (although the results of case reports appear promising, randomized controlled trials on cervical carotid thrombolysis and/or stenting are lacking);
- Hybrid techniques for the re-opening of the ICA.

The patency restoration rate is inversely related to the duration of the occlusion. A chronically occluded ICA has a poor patency rate and little gain from re-opening. The retrograde filling of the ICA to petrous or cavernous segment from an ECA anastomosis or contralateral ICA is a good sign of operability.

Operating pearls:

- Emergency operations for an acute neuro-deficit associated with the total occlusion should not be performed after about 2 hrs;
- Extremely poor neuro status (lethargy/coma) is a contraindication to surgery;
- Patients without a persistent neuro-deficit should be operated on as soon as possible;
- If the patient has recurrent TIAs (despite maximal medical therapy) following a recent carotid occlusion and no definite infarct on MRI, an EC-IC bypass surgery should be considered (Greenberg 2010; Xu et al. 2017; White et al. 2019; Powers et al. 2000; Sugg et al. 2005; Powers et al. 1987; Hafner and Tew 1981).

2.2. Vertebrobasilar Insufficiency (VBI)

2.2.1. Introduction

VBI may be suspected in a patient with transient episodes of "dizziness" (vertigo without apparent cause) that is initiated by positional changes. The estimated stroke rates are 22–35% over 5 years, or 4.5–7% per year. The risk of infarction after the first VBI-TIA has been estimated to be 22% for the first year.

Depending on the severity of the disorder, VBI can cause a variety of symptoms. Some symptoms may just last a few minutes, while others may last a lifetime. Loss of vision in one or both eyes, diplopia, nausea and vomiting, dizziness or vertigo, slurred speech, numbness or tingling in the hands or feet, changes in mental status (including confusion or loss of consciousness), sudden and severe weakness throughout the body also known as a drop attack, loss of balance and coordination, difficulty swallowing and weakness in a part of the body are all common symptoms of VBI. As with a TIA, the symptoms may come and go.

2.2.2. Clinical Presentation

The diagnostic criteria for VBI are shown in Table 5.

For the clinical diagnosis of VBI, two or more of the following criteria are needed:

- Sensory or motor symptoms or both, happening bilaterally at the same time;
- Diplopia: due to ischemia of the upper brainstem (midbrain) near the ocular nuclei;
- Dysarthria: due to ischemia of the lower brainstem;
- Homonymous hemianopsia: as a result of ischemia of the occipital cortex (this is binocular, where amaurosis fugax which is monocular).

Table 5. Diagnostic criteria for VBI.

“The 5 Ds of VBI” Diagnostic Criteria
Drop attack
Diplopia
Dysarthria
Defect (visual)
Dizziness

Source: Authors’ compilation based on data from Greenberg (2010).

2.2.3. Etiology

Atheromatous and stenotic lesions occur most frequently at the VA origin which usually causes VBI. Other atheromatous lesions on the BA or PCA can cause symptoms related to VBI. VBI symptoms may be due to hemodynamic insufficiency (perhaps the most common etiology), including subclavian steal syndrome where reversed flow in the VA due to a proximal stenosis of the subclavian artery and sometime stenosis of both VAs or one VA where the other is hypofunctional.

VBI may sometimes be due to squeezing of the VA at the level of C1–C2 with head turning (bow hunter’s syndrome) or anterior atlantoaxial subluxation (e.g., in rheumatoid arthritis) with rotatory atlantoaxial subluxation. Embolism from the ulceration of a plaque or cardiac origin can also cause a vertebrobasilar infarction.

2.2.4. Investigations

An MRI (MRA) is part of the ischemic stroke protocol, CT (with perfusion CT) and CTA, or a six-vessel cranial-selective DSA.

2.2.5. Treatment

Surgical treatments are the main way of management. The options are as follows:

- Vertebral endarterectomy should be carried out in
 - (i) Bilaterally substantial VA stenosis, defined as a stenosis of more than 60% in both arteries;
 - (ii) If the contralateral is hypoplastic, the dominant vertebral artery (VA) has a greater-than-60% stenosis, terminating in the posterior inferior cerebellar artery (PICA), or is obstructed;
 - (iii) Symptomatic embolism thought to be caused by a spinal lesion;
- Transposition of the VA to the ICA, CCA or ECA (with or without a saphenous vein patch graft), or to the thyrocervical trunk or subclavian artery;
- Bypass grafting (for example, occipital artery to the PICA);
- For a C1-2 posterior reduction, arthrodesis with stabilization may prevent a potentially life-threatening CVA in the cases of os odontoideum, AAD or bow hunter’s syndrome.

Anticoagulation is the mainstay of medical treatment. Alternatives to anticoagulants include antiplatelet drugs. The efficacy of either drugs remains unproven. Secondary prevention, like all types of ischemia events, necessitates a multimodal approach that includes blood pressure control, quitting smoking, stringent blood sugar control, statin use and lifestyle changes such as diet and exercise (Greenberg 2010; Lindsay et al. 2011; Awad 2005; Schaller 2008; Kuether et al. 1997; Pirau and Lui 2020; Caplan 2003).

2.2.6. Bow Hunter’s Stroke (BHS)

BHS Hemodynamic

Here, VBI (TIA to infarct) is induced by an intermittent VA occlusion resulting from head rotation. It may also occur with forced (e.g., chiropractic neck manipulation) or voluntary head rotation. An occlusion usually involves the VA contralateral to the direction of rotation, and usually occurs at the C1–C2 junction (due to the immobility of the VA at this location). However, other sites can also be involved. A VA occlusion does not produce clinical symptoms in most individuals due to collateral supply through the contralateral VA and/or the circle of Willis. A symptomatic occlusion usually involves the dominant VA, however, may also occur with a non-dominant VA. Most cases of a BHS occur in patients with an isolated posterior circulation (incompetent posterior communicating arteries).

Contributing Factors to a BHS

- 1 External VA compression
 - (i) Spondylotic bone spurs: particularly in the foramen transversarium;
 - (ii) Tumors;
 - (iii) Fibrous bands (e.g., proximal to entrance of the VA into the C6 foramen transversarium);
 - (iv) Infectious processes;
 - (v) Trauma.
- 2 Tethering of the VA
 - (i) At the transverse foramina of C1 and C2;
 - (ii) Along the sulcus arteriosus proximal to where the VA enters the dura;
 - (iii) Defect in the odontoid process;
 - (iv) Atherosclerotic vascular disease.

Diagnosis

A dynamic cerebral DSA is the investigation of choice, but significant consequences can be precipitated during a DSA in patients with a BHS. The involved VA shows loss of flow as the head is rotated from the neutral position to the contralateral side. Carotid injections demonstrate patency of the posterior communicating artery, as well as the presence of any persistent fetal anastomoses.

CT angiogram (CTA): The same precautions are needed as with a dynamic DSA. A CTA is not the initial diagnostic study of choice. If the dynamic DSA is negative, a CTA is not needed. If the dynamic DSA is positive, a CTA with a CT scan of the cervical spine with the craniovertebral junction may be helpful to demonstrate the arterial relationship to the bony anatomy.

Treatment

Conservative: conservative treatment includes anticoagulation with the cervical collar to remind the patient not to turn their head. Surgical treatment is the definitive treatment.

For VA compression at C1-2:

- (i) C1-2 fusion and fixation after reduction;
- (ii) VA decompression: C1 "hemilaminectomy" via a posterior approach.

For compression at other sites: elimination of the source of compression where possible (e.g., sectioning of an offending fibrous band, removal of osteophytic spurs) (Greenberg 2010; Schaller 2008; Pirau and Lui 2020; Lemole et al. 2002).

3. Cerebral Arterial Dissections (CADs)

3.1. Introduction

When intraluminal blood penetrates the layers of the vessel wall, a cerebral arterial dissection occurs. In the young population, a cranial-cervical dissection is responsible for 15–20% of strokes (Rajpal and Naik 2018; Anson and Crowell 1991). In CAD, hemorrhage occurs in the medial layer of an artery which may be spontaneous or post-traumatic, and may be intracranial or extracranial. It usually presents with ipsilateral pain, as well as features of an infarction or subarachnoid hemorrhage (SAH).

3.2. Pathophysiology

A pathological trans-intimal extravasation of blood from the true lumen into the vessel wall either dissects the internal elastic membrane from the intima, causing a narrowing of the true lumen leading to ischemia or infarction, or it may dissect into the sub-adventitial plane, producing an adventitial outpouching from the vessel wall (pseudoaneurysm). Rupture through the vessel wall producing an SAH occurs occasionally.

Subintimal dissections are more common with intracranial dissections, whereas extracranial vessels usually dissect either at the media or between media and adventitia.

CADs primarily affect middle-aged patients with an average age of 45 years (average age of traumatic dissections is slightly lower). CADs are more frequent in men; however, the incidence is unknown, as it often causes mild and transient symptoms. Some internal carotid artery cases (though considered spontaneous) may

actually be due to trivial trauma, including violent coughing, nose blowing and simple neck turning. This usually occurs in young women.

The VA was the commonest intracranial site for dissection. VA dissections are less frequent than carotid dissections. Extracranial lesions outnumber intracranial ones. Traumatic dissections frequently occur where the VA crosses bony prominences, e.g., at the C1–2 junction or where it enters the foramen transversarium (usually at C6). Spontaneous dissections tend to be intracranial and commonly occur on the dominant VA.

Dissecting aneurysms of the VA tends to be fusiform and may be amenable to clipping. Basilar artery dissections tend to present with a brainstem infarction and the prognosis is generally regarded as poor (Greenberg 2010; Lindsay et al. 2011; Rajpal and Naik 2018; Yamaura 1994; CA VAT AS Investigators 2001).

3.3. Etiology of “Spontaneous” Dissections

Etiologies of spontaneous dissection are shown in Table 6.

Table 6. Etiologies of a spontaneous dissection.

Common	Others
Fibromuscular dysplasia (FMD): found in 15% cases	Ehlers–Danlos syndrome
Cystic medial necrosis (or degeneration)	Takayasu’s disease
Saccular aneurysm	Medial degeneration
Marfan syndrome	Syphilitic arteritis
Atherosclerosis	Polyarteritis nodosa (PAN)
Strenuous physical exercise	Moyamoya disease
Autosomal dominant polycystic kidney disease	Allergic arteritis
Homocystinuria	Migraine

Source: Authors’ compilation based on data from Greenberg (2010), Lindsay et al. (2011), Anson and Crowell (1991) and Halbach et al. (1993).

3.4. Clinical Presentation

The most frequent presentation in patients under 30 years is usually due to an internal carotid (anterior circulation) dissection without an SAH but can present with an SAH (Figure 10). In patients > 30 years, a vertebrobasilar artery (VBA) dissection with an SAH is the most common. Headaches are commonly severe and usually predate neurologic deficits by days or weeks.

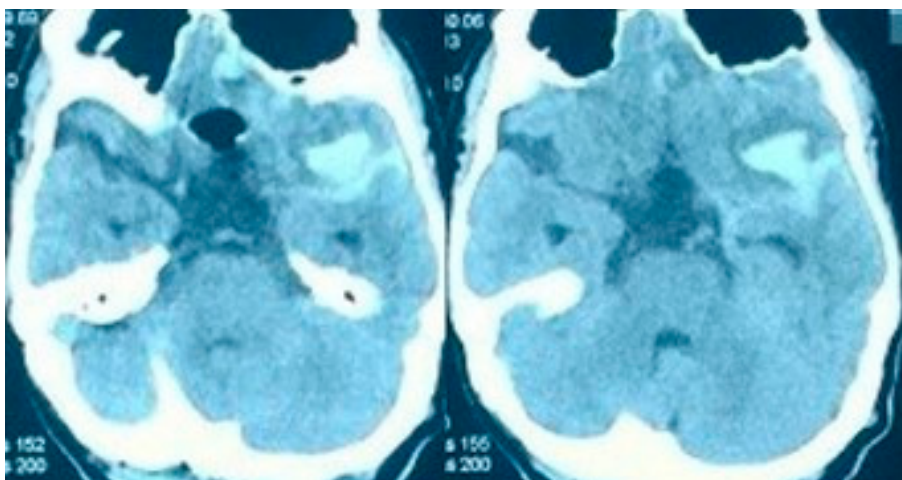


Figure 10. Axial CT scan showing a left Sylvian SAH (the patient presented with features of an SAH with left hemiparesis and aphasia). Source: Figure by authors.

3.4.1. Internal Carotid Dissection

In a spontaneous dissection, the most common initial symptom is an ipsilateral headache. It may also produce a sudden onset of severe pain over the carotid artery (carotidynia). For incomplete Homer's syndrome (oculosympathetic palsy), ptosis and miosis without anhidrosis may occur. A bruit may be heard either by the examiner or by the patient. It may be a cause of infantile and childhood hemiplegia and hemiparesis. Post-traumatic ICA dissections are much more common than spontaneous ones, and are managed like of arterial dissections with the management of other injuries.

3.4.2. Vertebrobasilar System Artery Dissection

In spontaneous extradural dissections, neck pain and severe headache are common, along with TIAs or stroke (usually lateral medullary syndrome or cerebellar infarction, especially in patients with an occlusion of the third or fourth portion of the VA. A VA dissection may be bilateral). Dissecting aneurysms may present with an altered level of consciousness, and may cause an SAH. Rebleeding occurs in 24–30% of these cases presenting with an SAH, making these lesions risky with a very high mortality. Traumatic extradural dissections or pseudoaneurysms may have a similar presentation but can also produce massive external hemorrhage or neck hematomas (Greenberg 2010; Yamaura 1994; Halbach et al. 1993; Welling et al. 1983; Pozzati et al. 1994).

3.5. Evaluation

A CT is very useful for evaluating the brain for an infarction (perfusion CT). Dissections can sometimes be visualized directly. A CTA identifies the CAD with 99% accuracy (Figures 10–12).

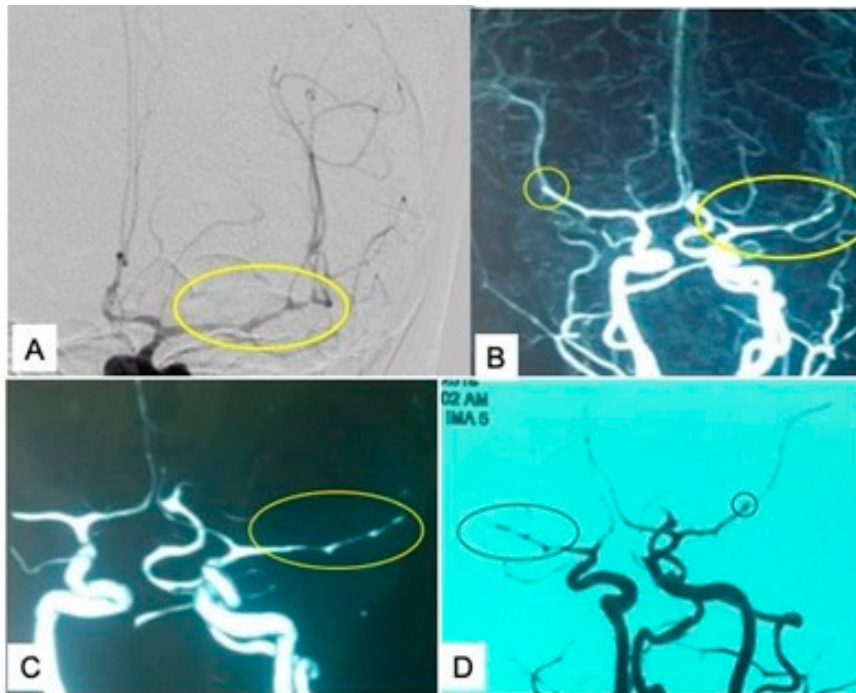


Figure 11. DSA and CTA of the patient from Figure 10. (A) Cerebral DSA of the left CCA injection showing a beaded appearance of the left MCA. (B–D) CTA of the brain showing beaded appearance of the left MCA, suggesting an MCA dissection. The patient (27-year-old female) also had a right MCA aneurysm (incidental) and dorsal scoliosis. Source: Figure by authors.

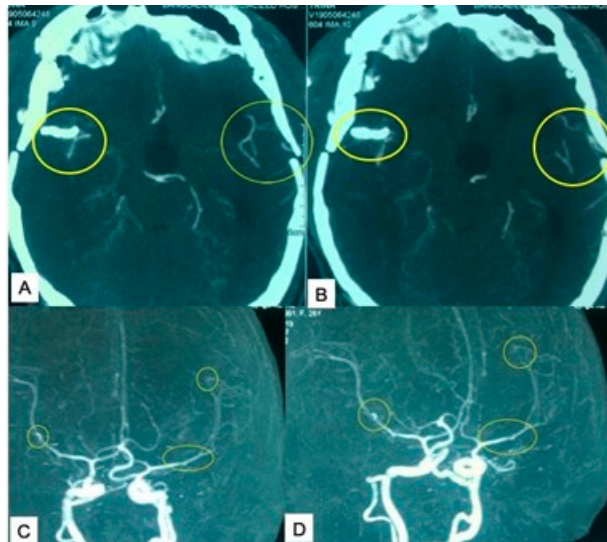


Figure 12. The patient from Figure 10 underwent a left STA–MCA bypass and microsurgical clipping of the right MCA aneurysm in the same sitting. (A–D) Postoperative CTA on the first POD. Source: Figure by authors.

The definitive diagnostic study is a cerebral DSA. However, diagnosis is challenging and may be delayed or misinterpreted as a saccular aneurysm or vasospasm. Angiographic findings in CAD are shown in Table 7.

Table 7. Angiography (DSA, CTA, MRA) findings.

(i)	Luminal stenosis (“string sign”)	(vi)	“Double lumen sign”: true vessel lumen and an intramural pseudo lumen is the only pathognomonic sign
(ii)	Fusiform dilation with distal or proximal slandering (string and pearl sign)	(vii)	Wavy “ripple” appearance
(iii)	Occlusion of artery	(viii)	Severe kinking (frequently bilateral); VBA may show dolichoectasia
(iv)	Intimal flap		
(v)	Proximal beading (“string of beads” configuration, indicative of an FMD) (Figures 10–12)		

A finding of arterial dissections is that they often alter configuration on repeat angiography due to resolve or worsen.

Source: Authors’ compilation based on data from Greenberg (2010), Lindsay et al. (2011), Rajpal and Naik (2018), Eastman et al. (2006) and Kitanaka et al. (1994).

MRI: On T1 weighted magnetic resonance imaging (MRI), an intramural hematoma appears as a region of elevated signal intensity, whereas contrast MRI shows a thick ring-like or railroad-like enhancement corresponding to the twofold lumen (Crescent sign). MRA can also show the details of CAD (Greenberg 2010; Rajpal and Naik 2018; Eastman et al. 2006; Kitanaka et al. 1994).

3.6. Treatment

- Extracranial dissections are usually treated medically (anticoagulation).
- Whereas intracranial dissections with SAH are treated surgically (Figures 10–12).
- But treatment should be individualized as case by case.

The clinical presentation, collateral circulation, access-related problems for both surgical and endovascular techniques, the presence of any leptomeningeal anastomoses, and contralateral flow all influence the treatment. After a rebleed, there is a substantial risk of morbidity and mortality, especially in dissecting aneurysms, hence this condition necessitates intensive surgical or endovascular therapy. Heparin, followed by warfarin or antiplatelet medicine, can be used to treat patients with ischemic intracranial dissection. Stenting and balloon dilatation or surgical bypass followed by occlusion or trapping of the affected section are options for patients with acute or recurrent strokes who do not respond to pharmacological treatment. The role of surgical treatment in patients with fusiform dilated aneurysms is clip reconstruction of the vessel, wrapping of the aneurysm, and clipping or trapping of the parent artery and revascularization, especially if there are insufficient leptomeningeal anastomoses or collaterals. Endovascular therapy options include proximal blockage of the parent artery with materials, coils

or detachable balloons, proximal and distal trapping of the diseased arterial, or a surgical distal bypass followed by a parental vessel occlusion. The parent vasculature can be preserved using stent-assisted coiling, or the vessel can be remodeled utilizing flow diverters with or without coiling (but with a lack of long-term results, with periprocedural complications) (Greenberg 2010; Rajpal and Naik 2018; Zhang et al. 2016; Uhl et al. 2003; Grigoryan et al. 2016; Gory et al. 2017; Sugita et al. 1981; Anxionnat et al. 2003; Aymard et al. 1991; Kurata et al. 2001; Peluso et al. 2008; Lylyk et al. 2009; Ogata et al. 2017; Ramgren et al. 2005; Kühn et al. 2015).

3.7. Outcome

Based on an evaluation of 260 cases, a mortality of 26% was found. Of the cases, 70% had a favorable outcome (based on the Glasgow Outcome scale) and 5% were poor. Mortality was higher for ICA lesions (49%) than VBA lesions (22%). Mortality was 24% in the SAH group and 29% in non-SAH cases (Yamaura 1994).

4. Cerebrovascular Bypasses

4.1. Introduction

Cerebrovascular bypasses are highly specialized, skill-requiring and assiduous armamentariums for the correction/treatment of many cerebrovascular lesions and some cases of skull base lesions. An extracranial-intracranial (EC-IC) bypass was first introduced and pioneered by Donaghy and Yasargil in 1967.

4.2. Classification

Cerebrovascular bypass can be classified in many ways.

Types of cerebrovascular bypasses:

- EC-IC bypass (Figures 7–9):

Low flow (blood flow 20–40 mL/min)

- STA-MCA (superficial temporal artery and middle temporal artery) (Figures 8 and 9);
- STA-PCA (superficial temporal artery–posterior cerebral artery);
- STA-SCA (superficial temporal artery–superior cerebellar artery);
- OC-PICA (occipital artery–posterior inferior cerebellar artery);
- OC-PCA (occipital artery–posterior cerebral artery);
- OC-VA bypass.

Intermediate flow (blood flow 50–80 mL/min) (Figure 7)

- CCA/ICA/ECA-RA (radial artery) graft-MCA/PCA/SCA bypass;
- IMA (internal maxillary artery)-MCA bypass.

High flow

- CCA/ICA/ECA-GSV (great saphenous vein) graft-MCA/PCA/SCA

- IC-IC (intracranial-intracranial) bypass.

Side-to-side bypass

- MCA-MCA (upper trunk and lower trunk) bypass;
- ACA-ACA (anterior cerebral artery–anterior cerebral artery) bypass;
- PICA-PICA bypass;
- PCA-SCA bypass.

End-to-side bypass

- ATA (anterior temporal artery)-PCA bypass;
- ATA-ACA bypass;
- ATA-MCA(M2) bypass.

Reimplantation

- M2 to M2.

End-to-end re-anastomosis

- A3-A3;

- M2–M2.

4.3. Indications

- Symptomatic cerebral ischemic conditions (CBV and CBF mismatch on perfusion images or increase O₂ extraction fraction on PET):
ICA occlusion;
ICA stenosis;
MCA/ACA stenosis or occlusion;
Vertebrobasilar insufficiency (VBI).
- For management of a complex giant or fusiform aneurysm.
- ICA/VA dissection.
- Moyamoya disease and moyamoya syndrome.
- Traumatic arterial injury.
- Skull base tumor where a radical excision is performed (cavernous sinus malignant tumor/fungal mass excision with ICA).

4.4. EC–IC Bypass for Cerebrovascular Ischemia

After the introduction of an EC–IC bypass (Figures 7–9), it became popular rapidly, but after the failure of an EV–IC trial in 1985, it plummeted suddenly. In spite of a graft patency rate of 96%, surgical patients failed to show any superiority over the medical management group. An extensive evaluation showed the failure of the study’s inclusion criteria to distinguish between hemodynamic vs. thromboembolic causes of stroke.

4.4.1. Present Recommendation for EC–IC Bypass in Ischemia

Currently, imaging can identify flow-dependent ischemia. Xenon-CT, TCD, SPECT and MRI may be utilized in combination with an acetazolamide challenge test to investigate the cerebrovascular reserve and reactivity. As cerebral perfusion pressure decreases in severe atherosclerotic occlusive disease, cerebral vascular autoregulation fails to maintain an adequate CBF to keep up with metabolic demands. In this situation of “misery perfusion”, the oxygen extraction fraction (OEF) of available blood flow will increase. An increased OEF, as quantified by a PET, is an independent predictor of subsequent stroke. Patients with an abnormal response to acetazolamide challenge and/or with an elevated OEF are therefore potential candidates for cerebral revascularization (Greenberg 2010; Crowley et al. 2008; Garrett et al. 2008; Garrett et al. 2009; Kuroda et al. 2001; Lawton 2018).

5. Cerebral Vein and Dural Sinus Thrombosis (CVST)

5.1. Introduction

A CVST is a less frequent cause of stroke, with a yearly incidence of approximately 5/million, most commonly afflicting those in younger age groups and females. Its clinical presentation varies, and thus may delay the diagnosis (Al-Sulaiman 2019; Ferro et al. 2004; Boussier and Ferro 2007; Ferro and Canhão 2014).

Three types of CVST may produce cerebral venous infarctions:

- (i) Dural sinus thrombosis;
- (ii) Cortical venous thrombosis;
- (iii) Deep venous thrombosis.

5.2. Etiologies

Etiological condition are listed in Table 8.

Table 8. Many conditions have been associated with a CVST.

Common	Others
Infection, i.e., otitis media, sinusitis, meningitis	Cardiac disease (including CHF)
	Ulcerative colitis
Pregnancy and puerperium	Periarteritis nodosa Sickle cell trait
Oral contraceptives, dehydration, burn and cachexia (malignancy)	Trauma, including closed head injury
	Malignancy, including myeloproliferative disorders
Hypercoagulable state or thrombophilia (protein C, S, antithrombin III and plasminogen deficiency; anti-phospholipid antibodies; systemic lupus erythematosus)	Diabetes mellitus, especially with ketoacidosis
	Homocystinuria

Source: Authors' compilation based on data from Greenberg (2010), Al-Sulaiman (2019) and Dolan and Chowdry (1995).

5.3. Frequency of Involvement of Dural Sinuses and Other Veins

The superior sagittal sinus (SSS), left transverse sinus (TS) and superficial cortical veins are the most commonly involved. Multiple sinuses/veins involvement occur in 71% of cases. The cavernous sinus, straight sinus and deep venous system are rarely involved (Greenberg 2010).

5.4. Pathophysiology

A CVST reduces venous return from the brain tissue and reduces essential circulation to the brain.

This venous engorgement results in cerebral edema. The elevated venous pressure may also result in an infarction and/or hemorrhage. All these sequences may lead to a raised ICP. Hence, the clinical features may be due to a raised ICP, and the focal deficit/s may be due to edema and/or hemorrhage. A brain infarction in this situation is known as a venous infarction (Greenberg 2010; Al-Sulaiman 2019).

5.5. Clinical Features

There are no pathognomonic findings. Many signs and symptoms are due to an elevated ICP. They may present as a syndrome clinically indistinguishable from idiopathic intracranial hypertension (IIH). The anterior 1/3 of the SSS may have a blockage often without symptoms. In the posterior 2/3 occlusion, a venous infarction is more likely to evolve. The middle portion of the SSS occlusion usually causes hypertonia ranging from spastic hemi- or quadri-paresis. A posterior SSS occlusion can cause visual field cuts or cortical blindness, or a massive venous infarct with edema and death. Thrombosis of the TS may occur without symptoms, unless the opposite TS is hypoplastic or aplastic where the clinical presentation is akin to posterior SSS thrombosis. An isolated SSS blockage will not result in a cranial nerve deficit, except visual impairment and sixth nerve palsy from an elevated ICP. An occlusion of the jugular bulb may press the nerves in the jugular foramen, resulting in hoarseness, dysphonia, dysphagia and dyspnea.

A CVST should be diagnosed based on clinical evidence and supported imaging investigations, and should always be investigated in patients who have the following symptoms:

- Symptoms of FNDs in the absence of recognized vascular risk factors;
- New onset of an atypical headache;
- Intracranial hypertension;
- Neuroimaging evidence of hemorrhagic infarctions, particularly if the infarctions are many and do not follow arterial vascular regions (Greenberg 2010; Al-Sulaiman 2019; Kalbag 1984; Stam 2005).

5.6. Diagnosis

5.6.1. CT and CT Venogram (CTV)

May be normal in 10–20% of cases of CVST. The findings include the following:

- (i) Hyperdense sinuses and veins (the cord sign which is pathognomonic);
- (ii) Intraparenchymal petechial “flame” hemorrhages, seen in 20% of cases;
- (iii) Small/slit ventricles are seen in 50% of cases;

- (iv) Thrombosis of the SSS may produce a triangular-shaped high density within the sinus;
- (v) White matter edema;
- (vi) All of the above changes occurring bilaterally;
- (vii) Enhancement of the dura around the sinus, intense tentorial enhancement and gyral enhancement on contrast CT;
- (viii) CTV shows occluded sinus/veins with evidence of redirected venous flow (Greenberg 2010; Al-Sulaiman 2019; Stam 2005; Perkin 1995).

5.6.2. MRI and MR Venogram (MRV)

An MRI (Figures 13 and 14) is excellent for a diagnosis and follow-up. It demonstrates the absence of venous flow and thrombus; it also demonstrates cerebral parenchymal changes. It can differentiate an occluded sinus from congenital aplasia. An MRI shows cerebral edema and non-acute hemorrhagic changes brilliantly at different stages. An MRV shows occluded sinus/veins, with evidence of redirected venous flow, but tends to overestimate the degree of the occlusion (Greenberg 2010; Al-Sulaiman 2019; Stam 2005; Perkin 1995).

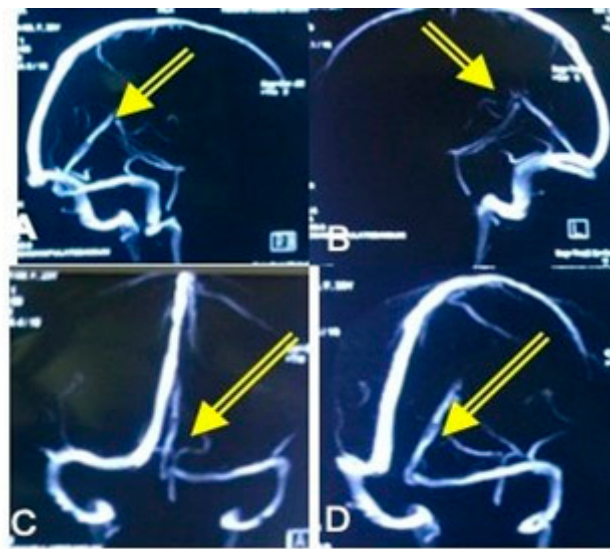


Figure 13. (A–D) Normal MRV of the brain where the deep venous system (through the straight sinus) is drained into the right transverse sinus (TS), and the SSS into the left TS. Source: Figure by authors.

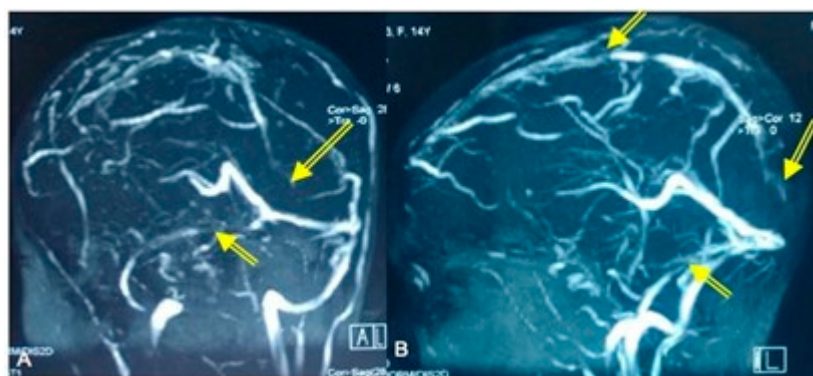


Figure 14. MRV of the brain (A,B) showing a CVST, especially the SSS and left TS (arrow-marked), where the deep venous drainage system is intact. Source: Figure by authors.

5.6.3. Cerebral DSA

A DSA is better to demonstrate the non-filling of segments of sinuses/veins or filling defects, residual flow and long circulation time, and can find out the areas of reversal of venous flow. Sometimes, a DSA can show a thrombus.

A DSA is often used as a complementary or therapeutic intervention.

5.6.4. Lumbar Puncture (LP)

An OP is usually increased. The CSF may be bloody or xanthochromic.

5.6.5. Hematological Tests

Hematological tests are used to find out predisposing factors when the cause is not known.

An evaluation for thrombophilia and hypercoagulability includes the following:

- Proteins C and S, as well as antithrombin III levels, antiphospholipid antibodies and lupus anticoagulants;
- CBC, factor II level, paroxysmal nocturnal hemoglobinuria panel, serum homocysteine level and leukocyte alkaline phosphatase (Greenberg 2010; Al-Sulaiman 2019; Stam 2005; Perkin 1995).

5.6.6. Ultrasound of the Head in Neonates

An ultrasound can be used in diagnosis of SSS thrombosis in neonates.

5.6.7. Detection of Underlying Disorders

During presentation, laboratory work-up is difficult, as the acute and active process will cause a lot of abnormalities in the coagulation system. The best time to work with these patients up is 3 months after the patient recovers from the acute stage of the disease.

5.7. Prognosis

Prognostic factors are listed in Table 9. Mortality: approximately 30% (range: 5–70%).

Table 9. Poor prognostic factors in CVST.

Poor Prognosticator
Coma
Rapid neurologic deterioration/focal signs
Extremes of age
Male sex
Large hemorrhage
Venous infarct
Deep venous system thrombosis

Source: Authors' compilation based on data from Greenberg (2010).

5.8. Treatment

- Treatment approach should be aggressive as the recovery rate is better than arterial stroke, and mortality and morbidity are very high without appropriate treatment;
- Again, management is intricated as anticoagulation increases the risk of an already increased hemorrhagic infarct, and available measures that lower the ICP may increase blood viscosity and coagulability.

Specific treatment:

- (i) Treatment of underlying cause/s when identified (e.g., antibiotics for infection).
- (ii) Early systemic heparin therapy—it reduces mortality and morbidity even with the evidence of intracerebral hemorrhage where there is a risk of increasing the size of the hemorrhage.
- (iii) Avoidance of steroids and control of blood pressure.
- (iv) Control seizures via anticonvulsants.
- (v) Maintenance of hydration.
- (vi) Monitoring the ICP—if the patient continues to deteriorate, then ventriculostomy or lumbar CSF drainage (continuous or intermittent).

Measures to lower the ICP:

- a. Ease of venous drainage by elevation of the head;
- b. Hyperventilation;
- c. Drain CSF (LP or ventriculostomy);
- d. Pentobarbital;
- e. Use hyperosmotic and/or loop diuretics last, as they can cause dehydration, hyperosmolarity and hypercoagulability.

- (vii) Thrombolytic treatment—systemically or directly into the thrombosed sinus, usually followed with heparin.
- (viii) When the patient is deteriorating in spite of the above measures:
 - Decompressive craniectomy (\pm decompressive lobectomy) decreases the ICP, but may not allure the outcome;
 - Direct “attack” on thrombosed sinus—sinotomy and sinuplasty with removal of the thrombus.
- (ix) Endovascular neurosurgery—success rate is much less with a chronic occlusion.
- (x) Visual loss with papilledema may be managed with optic nerve sheath fenestration (ONSF).
- (xi) Long-term anticoagulants after resolution of the acute stage with heparin and/or warfarin (3–6 months).

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References

- Al-Sulaiman, Abdulla. 2019. Clinical aspects, diagnosis and management of cerebral vein and dural sinus thrombosis: A literature review. *Saudi Journal of Medicine & Medical Sciences* 7: 137–45.
- Anson, Jhon, and Robert M. Crowell. 1991. Cervicocranial arterial dissection. *Neurosurgery* 29: 89–86. [CrossRef]
- Anxionnat, René, João Ferreira de Melo Neto, Serge Bracard, Jean Christophe Lacour, Catherine Pinelli, Thierry Civit, and Luc Picard. 2003. Treatment of Hemorrhagic Intracranial Dissections. *Neurosurgery* 53: 289–300; discussion 300–1. [CrossRef] [PubMed]
- Awad, Issam A. 2005. Cerebrovascular occlusive disease. In *Principles of Neurosurgery*. Edited by Setti S. Rengachary and Richard G. Ellenbogen. New York: Elsevier Mosby, pp. 197–213.
- Aymard, Armand, Y. Pierre Gobin, Jonathan E. Hodes, Siegfried Bien, Daniel Rufenacht, Daniel Reizine, Bernard George, and Jean J. Merland. 1991. Endovascular occlusion of vertebral arteries in the treatment of unclippable vertebrobasilar aneurysms. *Journal of Neurosurgery* 74: 393–98. [CrossRef] [PubMed]
- Barber, P. Alan, D. G. Darby, P. M. Desmond, Q. Yang, R. P. Gerraty, D. Jolley, G. A. Donnan, B. M. Tress, and Stephen M. Davis. 1998. Prediction of stroke outcome with echoplanar perfusion- and diffusion-weighted MRI. *Neurology* 51: 418–26. [CrossRef]
- Bousser, Marie-Germaine, and Jose M. Ferro. 2007. Cerebral venous thrombosis: An update. *Lancet Neurology* 6: 162–70. [CrossRef]
- Brott, Thomas G., George Howard, Gary S. Roubin, James F. Meschia, Ariane Mackey, William Brooks, Wesley S. Moore, Michael D. Hill, Vito A. Mantese, Wayne M. Clark, and et al. 2016. Long-Term Results of Stenting versus Endarterectomy for Carotid-Artery Stenosis. *The New England Journal of Medicine* 374: 1021–31. [CrossRef] [PubMed]
- CA VAT AS Investigators. 2001. Endovascular versus surgical treatment in patients with carotid stenosis in the carotid and vertebral artery transluminal angioplasty study (CA VATAS): A randomized trial. *The Lancet* 357: 1729–37. [CrossRef]
- Cai, Bin, and Bin Peng. 2017. Intracranial artery stenosis: Current status of evaluation and treatment in China. *Chronic Diseases and Translational Medicine* 3: 197–206. [CrossRef]
- Caplan, Louis R. 2003. Atherosclerotic vertebral artery disease in the neck. *Current Treatment Options in Cardiovascular Medicine* 5: 251–56. [CrossRef]
- Chen, Han-Jung, Tao-Chen Lee, and Chi-Peng Wei. 1992. Treatment of cerebellar infarction by decompressive suboccipital. *Stroke* 23: 957–61. [CrossRef] [PubMed]
- Cremonesi, Alberto, Carlo Setacci, Angelo Bignamini, Leonardo Bolognese, Francesco Briganti, Germano Di Sciascio, Domenico Inzitari, Gaetano Lanza, Luciano Lupattelli, Salvatore Mangiafico, and et al. 2006. Carotid Artery Stenting. *Stroke* 37: 2400–9. [CrossRef] [PubMed]
- Crowley, R. Webster, Ricky Medel, and Aaron S. Dumont. 2008. Evolution of cerebral revascularization techniques. *Neurosurgical Focus* 24: E3. [CrossRef] [PubMed]
- Dolan, Robert W., and Khalid Chowdry. 1995. Diagnosis and treatment of intracranial complications of paranasal sinus infections. *Journal of Oral and Maxillofacial Surgery* 53: 1080–87. [CrossRef]

- Eastman, Alexander L., David P. Chason, Carlos L. Perez, Amy L. McNulty, and Joseph P. Minei. 2006. Computed Tomographic Angiography for the Diagnosis of Blunt Cervical Vascular Injury: Is It Ready for Primetime? *The Journal of Trauma and Acute Care Surgery* 60: 925–29. [CrossRef] [PubMed]
- Ferro, José M., and Patrícia Canhão. 2014. Cerebral venous sinus thrombosis: Update on diagnosis and management. *Current Cardiology Reports* 16: 523. [CrossRef]
- Ferro, José M., Patrícia Canhão, Jan Stam, Marie-Germaine Bousser, and Fernando Barinagarrementeria. 2004. Prognosis of cerebral vein and dural sinus thrombosis: Results of the international study on cerebral vein and dural sinus thrombosis (ISCVT). *Stroke* 35: 664–70. [CrossRef]
- Gage, Brian F., Amy D. Waterman, William Shannon, Michael Boechler, Michael W. Rich, and Martha J. Radford. 2001. Validation of Clinical Classification Schemes for Predicting Stroke. *Jama-Journal of The American Medical Association* 285: 2864–70. [CrossRef]
- Garrett, Matthew C., Ricardo J. Komotar, Maxwell B. Merkow, Robert M. Starke, Marc L. Otten, and E. Sander Connolly. 2008. The Extracranial–Intracranial Bypass Trial: Implications for future investigations. *Neurosurgical Focus* 24: E4. [CrossRef]
- Garrett, Matthew C., Ricardo J. Komotar, Robert M. Starke, Maxwell B. Merkow, Marc L. Otten, Robert R. Sciacca, and E. Sander Connolly. 2009. The efficacy of direct extracranial–intracranial bypass in the treatment of symptomatic hemodynamic failure secondary to athero-occlusive disease: A systematic review. *Clinical Neurology and Neurosurgery* 111: 319–26. [CrossRef]
- GBD 2016 Stroke Collaborators. 2019. Global, regional, and national burden of stroke, 1990–2016: A systematic analysis for the Global Burden of Disease Study 2016. *The Lancet Neurology* 18: 439–58. [CrossRef] [PubMed]
- Gory, Benjamin, Michel Piotin, Diogo C. Haussen, Henrik Steglich-Arnholm, Markus Holtmannspötter, Julien Labreuche, Christian Taschner, Sebastian Eiden, Raul G. Nogueira, Papanagiotou Panagiotis, and et al. 2017. Thrombectomy in Acute Stroke with Tandem Occlusions from Dissection Versus Atherosclerotic Cause. *Stroke* 48: 3145–48. [CrossRef] [PubMed]
- Greenberg, Mark S. 2010. *Handbook of Neurosurgery*. New York: Thieme.
- Grigoryan, Mikayel, Diogo C. Haussen, Ameer E. Hassan, Andrey Lima, Jonathan Grossberg, Leticia C. Rebello, Wondwossen Tekle, Michael Frankel, and Raul G. Nogueira. 2016. Endovascular Treatment of Acute Ischemic Stroke Due to Tandem Occlusions: Large Multicenter Series and Systematic Review. *Cerebrovascular Diseases* 41: 306–12. [CrossRef]
- Hafner, Charles D., and J. M. Tew. 1981. Surgical management of the totally occluded internal carotid artery. *Surgery* 89: 710–17. [PubMed]
- Halbach, Van V., Randall T. Higashida, Christopher F. Dowd, Kenneth W. Fraser, Tony P. Smith, George P. Teitelbaum, Charles B. Wilson, and Grant B. Hieshima. 1993. Endovascular treatment of vertebral artery dissections and pseudoaneurysms. *Journal of Neurosurgery* 79: 183–91. [CrossRef]
- Heiserman, Joseph E., Joseph M. Zabramski, Burton P. Drayer, and Paul J. Keller. 1996. Clinical significance of the flow gap in carotid magnetic resonance angiography. *Journal of Neurosurgery* 85: 384–87. [CrossRef]
- Kalbag, Ramanand M. 1984. Cerebral Venous thrombosis. In *The Cerebral Venous System and Its Disorders*. Edited by Jhon P. Kapp and Henry H. Schmidek. Orlando: Grune and Stratton, pp. 505–36.
- Kaufmann, Timothy J., John Huston III, Jay N. Mandrekar, Cathy D. Schleck, Kent R. Thielen, and David F. Kallmes. 2007. Complications of Diagnostic Cerebral Angiography: Evaluation of 19826 Consecutive Patients. *Radiology* 243: 812–19. [CrossRef]
- Kistler, J. Philip, and Karen L. Furie. 2000. Carotid endarterectomy revisited. *The New England Journal of Medicine* 342: 1743–45. [CrossRef]
- Kitanaka, Chifumi, Jun-Ichi Tanaki, Masanori Kuwahara, Akira Teraoka, Tomio Sasaki, and Kintomo Takakura. 1994. Nonsurgical treatment of unruptured intracranial vertebral artery dissection with serial follow-up angiography. *Journal of Neurosurgery* 80: 667–74, Erratum in: *Journal of Neurosurgery* 1994, 80: 1132. [CrossRef]
- Koelmay, Mark J. W., Paul J. Nederkoorn, Johannes B. Reitsma, and Charles B. Majoie. 2004. Systematic Review of Computed Tomographic Angiography for Assessment of Carotid Artery Disease. *Stroke* 35: 2306–12. [CrossRef]
- Kuether, Todd A., Gary M. Nesbit, Wayne M. Clark, and Stanley L. Barnwell. 1997. Rotational Vertebral Artery Occlusion: A Mechanism of Vertebrobasilar Insufficiency. *Neurosurgery* 41: 427–33. [CrossRef] [PubMed]
- Kühn, Anna Luisa, Peter Kan, Francesco Massari, J. Diego Lozano, Samuel Y. Hou, Mary Howk, Matthew J. Gounis, Ajay K. Wakhloo, and Ajit S. Puri. 2015. Endovascular reconstruction of unruptured intradural vertebral artery dissecting aneurysms with the Pipeline embolization device. *Journal of NeuroInterventional Surgery* 8: 1048–51. [CrossRef] [PubMed]

- Kurata, Akira, Taketomo Ohmomo, Yoshio Miyasaka, Kiyotaka Fujii, Shinichi Kan, and Takao Kitahara. 2001. Coil Embolization for the Treatment of Ruptured Dissecting Vertebral Aneurysms. *American Journal of Neuroradiology* 22: 11–18. [PubMed]
- Kuroda, Satoshi, Kiyohiro Houkin, Hiroyasu Kamiyama, Kenji Mitsumori, Yoshinobu Iwasaki, and Hiroshi Abe. 2001. Long-Term Prognosis of Medically Treated Patients with Internal Carotid or Middle Cerebral Artery Occlusion. *Stroke* 32: 2110–16. [CrossRef]
- Lawton, Michael T. 2018. *Seven Bypasses-Tenets and Techniques for Revascularization*. New York and Stuttgart: Thieme Publishers.
- Lemole, G. Michael, Jeffrey S. Henn, Robert F. Spetzler, and Joseph M. Zabramski. 2002. Bow hunter's stroke. *BNI Quarterly* 17: 4–10.
- Lindsay, Kenneth W., Ian Bone, and Geraint Fuller. 2011. *Neurology and Neurosurgery Illustrated. General Approach to History and Examination*. London: C L Elsevier.
- Lyden, Patrick, T. Brott, B. Tilley, K. M. Welch, E. J. Mascha, S. Levine, E. C. Haley, J. Grotta, and J. Marler. 1994. Improved reliability of the NIH Stroke Scale using video training. NINDS. TPA. Stroke Study Group. *Stroke* 25: 2220–26. [CrossRef]
- Lylyk, Pedro, Carlos Miranda, Rosana Ceratto, Angel Ferrario, Esteban Scrivano, Hugh Ramirez Luna, Aaron L. Berez, Quang Tran, Peter K. Nelson, and David Fiorella. 2009. Curative endovascular reconstruction of cerebral aneurysms with the pipeline embolization device: The Buenos Aires experience. *Neurosurgery* 64: 632–43. [CrossRef]
- Marks, Michael P., Eric B. Holmgren, Allan J. Fox, Suresh Patel, Rudiger von Kummer, and Juergen Froehlich. 1999. Evaluation of Early Computed Tomographic Findings in Acute Ischemic Stroke. *Stroke* 30: 389–92. [CrossRef]
- Naylor, A. Ross. 2018. Endarterectomy versus stenting for stroke prevention. *Stroke and Vascular Neurology* 3: e000146. [CrossRef]
- Nighoghossian, Norbert, Laurent Derex, and Philippe Douek. 2005. The vulnerable carotid artery plaque: Current imaging method and new perspectives. *Stroke* 36: 2764–72. [CrossRef]
- Ogata, Atsushi, Shuji Sakata, Hiroaki Okamoto, and Tatsuya Abe. 2017. Ruptured dissecting aneurysm of the recurrent artery of Heubner: Consideration of pathological findings. *Neurology India* 65: 623–25. [CrossRef] [PubMed]
- Paciaroni, Maurizio, Giancarlo Agnelli, Francesco Corea, Walter Ageno, Andrea Alberti, Alessia Lanari, Valeria Caso, Sara Micheli, Luca Bertolani, Michele Venti, and et al. 2008. Early hemorrhagic transformation of brain infarction: Rate, predictive factors, and influence on clinical outcome: Results of a prospective multicenter study. *Stroke* 39: 2249–56. [CrossRef] [PubMed]
- Peluso, J. P. P., W. J. van Rooij, M. Sluzewski, G. N. Beute, and C. B. Majoie. 2008. Endovascular Treatment of Symptomatic Intradural Vertebral Dissecting Aneurysms. *American Journal of Neuroradiology* 29: 102–6. [CrossRef] [PubMed]
- Perkin, G. D. 1995. Cerebral venous thrombosis: Developments in imaging and treatment. *Journal of Neurology, Neurosurgery and Psychiatry* 59: 1–3. [CrossRef]
- Pirau, Letitia, and Forshing Lui. 2020. Vertebrobasilar Insufficiency. [Updated 5 February 2020]. In *StatPearls [Internet]*. Treasure Island: StatPearls Publishing. Available online: <https://www.ncbi.nlm.nih.gov/books/NBK482259/> (accessed on 31 July 2021).
- Powers, William J., C. P. Derdeyn, S. M. Fritsch, D. A. Carpenter, K. D. Yundt, T. O. Videen, and R. L. Grubb, Jr. 2000. Benign prognosis of never-symptomatic carotid occlusion. *Neurology* 54: 878–82. [CrossRef]
- Powers, William J., Gary A. Press, Robert L. Grubb, Jr., Mokhtar Gado, and Marcus E. Raichle. 1987. The effect of hemodynamically significant carotid artery disease on the hemodynamic status of the cerebral circulation. *Annals of Internal Medicine* 106: 27–34. [CrossRef]
- Pozzati, Eugenio, Alvaro Andreoli, Paolo Limoni, and Mario Casmiro. 1994. Dissecting aneurysms of the vertebrobasilar system: Study of 16 cases. *Surgical Neurology* 41: 119–24. [CrossRef]
- Rajpal, Girish, and Vikas Naik. 2018. Management of intracranial arterial dissection. *Neurology India* 66: 40–42.
- Ramgren, B., Mats Cronqvist, Bertil Romner, Lennart Brandt, Stig Holtås, and Elna-Marie Larsson. 2005. Vertebrobasilar dissection with subarachnoid hemorrhage: A retrospective study of 29 patients. *Neuroradiology* 47: 97–104. [CrossRef]

- Sardar, Partha, Saurav Chatterjee, Herbert D. Aronow, Amartya Kundu, Preethi Ramchand, Debabrata Mukherjee, Ramez Nairooz, William A. Gray, Christopher J. White, Michael R. Jaff, and et al. 2017. Carotid Artery Stenting Versus Endarterectomy for Stroke Prevention. *Journal of the American College of Cardiology* 69: 2266–75. [CrossRef]
- Schaller, Bernhard. 2008. Extracranial-intracranial bypass to reduce the risk of ischemic stroke in intracranial aneurysms of the anterior cerebral circulation: A systematic review. *Journal of Stroke and Cerebrovascular Diseases* 17: 287–98. [CrossRef] [PubMed]
- Sims, John R., Guy Rordorf, Eric E. Smith, Walter J. Koroshetz, Michael H. Lev, Ferdinando Buonanno, and Lee H. Schwamm. 2005. Arterial Occlusion Revealed by CT Angiography Predicts NIH Stroke Score and Acute Outcomes after IV tPA Treatment. *American Journal of Neuroradiology* 26: 246–51.
- Sonecha, T. N., K. T. Delis, and M. Y. Henein. 2006. Predictive value of asymptomatic cervical bruit for carotid artery disease in coronary artery surgery revisited. *International Journal of Cardiology* 107: 225–29. [CrossRef] [PubMed]
- Stam, Jan. 2005. Thrombosis of the Cerebral Veins and Sinuses. *New England Journal of Medicine* 352: 1791–98. [CrossRef]
- Sugg, Rebecca M., Marc D. Malkoff, Elizabeth A. Noser, Hashem M. Shaltoni, Raymond Weir, Edwin D. Cacayorin, and James C. Grotta. 2005. Endovascular Recanalization of Internal Carotid Artery Occlusion in Acute Ischemic Stroke. *American Journal of Neuroradiology* 26: 2591–94. [PubMed]
- Sugita, Kenichiro, Shigeaki Kobayashi, Toshiki Inoue, and Tatsuo Banno. 1981. New angled fenestrated clips for fusiform vertebral artery aneurysms. *Journal of Neurosurgery* 54: 346–50. [CrossRef]
- Tomandl, Bernd F., Ernst Klotz, Rene Handschu, Brigitte Stemper, Frank Reinhardt, Walter J. Huk, K.E. Eberhardt, and Suzanne Fateh-Moghadam. 2003. Comprehensive Imaging of Ischemic Stroke with Multisection CT. *RadioGraphics* 23: 565–92. [CrossRef]
- Uhl, E., R. Schmid-Elsaesser, and H. J. Steiger. 2003. Ruptured intracranial dissecting aneurysms: Management considerations with a focus on surgical and endovascular techniques to preserve arterial continuity. *Acta Neurochirurgica* 145: 1073–84. [CrossRef]
- Vahedi, Katayoun, Jeannette Hofmeijer, Eric Juettler, Eric Vicaut, Bernard George, Ale Algra, G. Johan Amelink, Peter Schmiedeck, Stefan Schwab, Peter M. Rothwell, and et al. 2007. Early decompressive surgery in malignant infarction of the middle cerebral artery: A pooled analysis of three randomised controlled trials. *The Lancet Neurology* 6: 215–22. [CrossRef]
- Welling, Richard E., Assad Taha, Tarun Goel, John Cranley, Raymond Krause, Charles Hafner, and John Tew. 1983. Extracranial carotid artery aneurysms. *Surgery* 93: 319–23.
- White, Timothy G., Hussam Abou-Al-Shaar, Jung Park, Jeffrey Katz, David J. Langer, and Amir R. Dehdashti. 2019. Cerebral revascularization after the Carotid Occlusion Surgery Study: What candidates remain, and can we do better? *Neurosurgical Focus* 46: E3. [CrossRef] [PubMed]
- Xu, Baofeng, Chao Li, Yunbao Guo, Kan Xu, Yi Yang, and Jinlu Yu. 2017. Current understanding of chronic total occlusion of the internal carotid artery (Review). *Biomedical Reports* 8: 117–25. [CrossRef] [PubMed]
- Yamaura, Akira. 1994. Nontraumatic intracranial arterial dissection: Natural history, diagnosis, and treatment. *Contemporary Neurosurgery* 16: 1. [CrossRef]
- Zhang, Yisen, Ming Lv, Conghai Zhao, Ying Zhang, Xinjian Yang, and Zhongxue Wu. 2016. Endovascular treatment of ruptured vertebrobasilar dissecting aneurysms: Review of 40 consecutive cases. *Neurology India* 64: 52–61. [CrossRef]

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