NEUROVASCULAR EMERGENCIES: MEDICAL AND SURGICAL MANAGEMENT OF STROKE

by JAFRI MALIN DATOK HJ. ABDULLAH MD, Phd,
Diplomate Neurosurgery (Belgium) FICS,
Neurosurgical Division,
Hospital University Sciences Malaysia,
16150 Kubang Kerian,
Kelantan, Malaysia.

E-mail: amiruljafni@yahoo.com

Introduction

In most states in Malaysia, strokes or better known as cerebrovascular accidents (CVA) are the 3rd cause of mortality and the main cause of disability. Due to the improvement of the treatment of ischeamic heart disease over ischaemic or haemorraghic brain disease over the last 10 years, more and more patients can benefit from non surgical and surgical intervention. The nineties have been called the decade of the brain in developed countries where brain attack has been treated as aggresively as heart attacks. Thus it is now the time for all developing countries to follow the same pathway.

Next to heart disease and cancer, cerebrovascular disease is the most frequent cause of death in the western world and now in Malaysia. And at least one-half of all neurologic patients in general has some type of cerebrovascular disease.

The term cerebrocvascular disease denotes any abnormality of the brain resulting from a pathologic process of blood vessels, be they arteries, arterioles, capillaries, veins, or sinuses. The pathologic change in the vessels takes the form of occlusion by thrombus or embolus, or of rupture, and the resulting abnormalities in the brain are of two types: ischemia, with and without infarction, and hemorrhage. Rarer forms of cerebrovascular disease are those due to altered permeability of the vascular wall and increased viscosity or other changes in the quality of blood. The latter changes underlie the strokes that complicate diseases such as sickle-cell anemia and polycythemia and account for the headache, brain edema, and convulsions of hypertensive encephalopathy. There are many more types of cerebrovascular disease; these are listed in Table1, and the relative frequency of the main types is indicated in Table 2

The stroke syndrome

The distinctive mode of presentation of cerebral vascular disease is the stroke, defined as any sudden or acute nonconvulsion focal neurologic deficit. In its most severe form the patient becomes hemiplegic or falls senseless, an event so dramatic that it is given its own name-apoplexy, cerebrovascular accident, stroke, or shock. If death does not follow within hours or days, there is nearly always some degree of recovery of

function. This temporal profile of neurologic events, whether condensed into several hours or days, is diagnostic. Variations in the temporal profile reflect the type of vascular lesion. Embolic strokes characteristically begin with absolute suddenness, and they may at times recede rapidly or they may last. Thrombotic strokes may have a similarly abrupt onset, but often they evolve somewhat more slowly, over a period of minutes to hours or even days. Cerebral hemorrhage from its onset causes a deficit that is steadily progressive for hours or longer.

The major neurovascular thrombotic and embolic syndromes their symptoms and signs, and the corresponding cerebral structures that are involved.

Table ONE. Types of Cerebral Vascular Disease

1.	**Atherosclerotic thrombosis**
2.	**Transient ischemic attacks**
3.	**Embolism**
4	**Primary (hypertensive) intracerebral hemorrhage**
5	**Ruptured or unruptured saccular aneurysm or AVM**
6	**Arteritis**
	a. Meningovascular syphilis, arteritis secondary to pyogenic and tuberculous meningitis, rare infectivr types (typhus, schistosomiasis, malaria, trichinosis, mucormycosis, etc.)
	b. Connective tissue disease (polyarteritis nodosa, lupus erythematosus), nectrozing arteritis, Wegener arteritis, temporal arteritis, Takayasu disease, granulomatous of giant cell arteritis of the aorta, and giant cell granulomatous angiitis of cerebral arteries.
7	**Cerebral thrombophlebitis: secondary to infection of ear, paranasal sinus face, etc.;with meningitis and subdural empyema; phlebothrombosis with debilitating postpartum, postoperative states; prolonged immobility, cardiac failure, hematologic disease (polycythema, sickle-cell disease); and of undetermined cause**
8	**hematologic disorders: polycythemia, sickle-cell disease, thrombotic thrombocytopenic purpura, thrombocytosis, etc.**
9.	**Trauma and dissection of carotid and vertebral arteries**
10	**Dissecting aortic aneurysm**
11	**Systemic hypotension with arterial stenoses: "simple faint," acute blood loss, myocardial infarction. Strokes-Adams syndrome, traumatid and surgical shock, sensitive carotid sinus, severe postural hypotension**
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13.	**Neurologic migraine with persistent deficit**
14.	**With tentorial, foramen, magnum, and subfalcial herniations**
15	**Miscellaneous types: fibromuscular dysplasia, excessive x-irradiation, unexplained middle cerebral artery territory infarction in closed headinjury, pressure of unruptured saccular aneurysm, complication of oral contraceptives**
16	**Undetermined caused in children and young adults: moyamoya; multiple, progressive intracranial arterial occlusions (Taveras)**

Table Two. Major Types of Cerebrovascular Disease and Their Frequency

	CVA in alive patient	CVA resulting in death
Atherosclerotic thrombosis	32%	12%
Lacunes	18%	18.5%
Embolism	32%	32%
Hypertensive hemorrhage	11%	15.5%
Ruptured aneurysms and vascular malformations	7%	4.5%
Indeterminate	-	9.5%
Others	-	8%

Atherosclerotic-thrombotic infarction

The large intracranial arteries, like the aorta and coronary arteries, are predisposed to atherosclerotic changes. Favored sites are the common and internal carotid, the vertebral and basilar, and the stems of the major cerebral arteries. Factors enhancing this atheromatous process are hypertension, diabetes mellitus, and hyperlipidemia, both genetic and dietary.

More than one half of patients who develop a thrombotic stroke have one and more brief warning episodes, called transient ischemic attacks (TIAs), the diagnosis and treatment of which may prevent an oncoming stroke. The thrombotic stroke, whether or no it is preceded by warning attacks, develops in one of the following ways: Most often there is an abrupt onset of the neurologic deficit, evolving over a few minutes to a few hours; or there may be a stuttering onset and intermittent progression over several hours of a day or longer; or symptoms may regress for hours and then advance again. More perplexing still is the rare stroke in which the deficit advances in a series of steps over a period of weeks. Often the onset is during sleep; the patient awakens paralyzed.

The pattern of the neurologic deficit is determined by the site of arterial occlusion and the available anastomotic arrangements.

Ancillary Examinations

Noninvasive blood flow procedures, such as carotid Doppler studies, may reveal a stenotic or occluded artery. This can be verified by angiography, a procedure that carries a small risk of worsening the neurologic deficit. Digital subtraction angiography (DSA), preferably by the arterial route, more safety but less clearly visualizes the aorta and its main cranial branches. All these methods will probably be replaced by MR angiography. One can see by these several techniques both stenotic segments or occlusion of arteries and sometimes mural thrombi that may become embolic (artery-to-artery embolism).

Fig. ONE. Diagram of the left cerebral hemisphere, lateral aspect, showing the branches and distribution of the middle cerebral artery and the principle regions of cerebral localization. Following is a list of the clinical manifestations of infarction in the territory of this artery and the corresponding regions of cerebral damage

Signs and symptoms	Structures involved
Paralysis of the contralateral face, arm, and leg	**Somatic motor area for face and arm and the fibers descending from the leg area to enter the corona radiata**
Sensory impairment over the contralateral face, arm, and leg (pinprick, cotton touch, vibration, position, two-point discrimination, stereognosis, tactile localization, barognosis, cutaneographia)	**Somatic sensory area for face and arm and thalamoparietal projections**
Motor speech disorder	**Broca's area and frontal operculum of the dominant hemisphere**
"Central" aphasia, word deafness, anomia, jargon speech, alexia, agraphia, acalculia, finger agnosia, right-left confusion (the last four compose the Gerstmann syndrome)	**Central language area and parieto-occipital cortex of the dominant hemisphere**
Apractagnosia (amorphosynthesis), anosognosia, hemiasomatognosia, unilateral neglect, agnosia for the left half of external space, "dressing apraxia," distortion of visual coordinates, inaccurate localization in the half field, impaired ability to judge distance, upside-down reading, visual illusions; inattention and confusion usually associated	**Usually nondominant parietal lobe. Loss of topographic memory is usually due to a nondominant lesion, occasionally to a dominant one**
Homonymous hemianopia (often superior homonymous quadrantanopia)	**Optic radiation deep to second temporal convolution**
Paralysis of conjugate gaze to the opposite side	**Frontal contraversive field or fibers projecting therefrom**
Avoidance reaction of opposite limbs	**Parietal lobe**

FIG. ONE . (CONTINUED)

Signs and symptoms	Structures involved
Miscellaneous:	
Ataxia of contralateral limbs (s)	**Parietal lobe**
So-called Bruns ataxia or apraxia of gait	**Frontal lobes (bilateral)**
Unilateral neglect of space and body parts	**Parietal lobe , more often right**
Agitated delirium	**Right temporal**
Loss or impairment of optokinetic nystagmus	**Supramarginal or angular gyrus**
Limb-kinetic apraxia	**Premotor or parietal cortical damage**
Mirror movements	**Precise location of responsible lesions not known**
Cheyne-Stokes respiration, contralateral hyperhidrosis, mydriasis (occasionally)	**Precise location of responsible lesions not known**
Pure motor hemiplegia	**Upper portion of the posterior limb of the internal capsule and the adjacent corona radiata.**

Fig. Two. Diagram of a cerebral hemisphere, medial aspect, showing the branches and distribution of the anterior cerebral artery and the principal regions of crebral localization. Following is a list of the clinical manifestations of infarction in the territory of this artery and the coreesponding regions of cerebral damage.

Signs and symptoms	Structures involved
Paralysis of opposite foot and leg	Motor leg area
Paresis of opposite arm	Involvement of arm area of cortex or fibers descending therefrom to corona radiata
"Cortical" sensory loss over toes, foot, and leg	Sensory area for foot and leg
Urinary incontinence	Posteromedial part of superior frontal gyrus (bilateral)
Contralateral grasp reflex	Premotor and supplementary motor areas
Abulia (akinetic mutism), slowness, delay, lack of spontaneity, whispering, motor inaction, reflex distraction to sights and sounds	Uncertain localization-probably deep medial-orbital (usually bilateral)
Impairment of gait and stance (gait "apraxia")	Inferomedial frontal-striatal
Mental impairment (preseveration and amnesia	Localization unknown
Miscellaneous	
Dyspraxia of left limbs	Corpus callosum
Cerebral paraplegia	Motor leg area bilaterally (due to bilateral occlusion of anterior cerebral arteries)

What to do in the Emergency Room?

When a patient presents him or herself to the Emergency Room with the abrupt onset of a focal cerebral deficit; 5% are seizure, tumor, or psychogenic, 95% are vascular (i.e. stroke) of which I5% are hemorrhage such as intracerebral haemorraghe, subarchnoid haemorraghe or subdural hemorraghe. 85% of these diagnosed strokes are ischemic infarct and early cerebral angiography or computer spiral 3 Dimension angiogram of the brain has show that arterial occlusion can be demonstrated in 80% of these, regardless of subtype of infarcts. The ischeamic infarcts are there after clasified into various groups according to their anatomopathology causes:

i) 41% unknown cause (this may decrease with the use of early

angiography)

ii) 21% lacune (small artery or arteriole cerebrovascular lesion)

iii) 16% cardiogenic embolus

iv) 11% large artery cerebrovascular lesion

v) 11% tandem arterial pathology

A recent study showed that atherosclerotic plaques in the aortic arch more than 4mm thick are a risk factor for the recurrent CVAs and other vascular events (myocardial infarcts, peripheral embolism, and death from vascular causes)

Table 3. Outcome in CVAs presenting to most major hospital in South East Asia

Outcome	%
Died	35
Home but Disabled	40
Improved	20
Normal	5

Emergency CAT SCAN of the Brain

One should strive to obtain a CT as an emergency procedure. If this is not possible, a CT should be obtained at least within 24 hrs for all cases. Hospitals with Computed Tomogram Spiral Three Dimension Scan facilities can at the same time do a CT SCAN angiogram to determind the type and vessel location which is effected.

More emergent CT should be obtained:

1. if anticoagulation therapy (e.g. heparin) is suspected to be a cause of the CVA.

2. if intracerebral haemorraghe is suspected (e.g. if level of consciousness unusually depressed according to Glasgow Coma Scale)

3. if surgical intervention may save the patient or prevent further deterioration of neurological deficits.

Table 4.

Note: General findings of CAT scan with CVAs.
NB: these principles do not apply to small lacunar infarcts.

First 12-24 hrs after pale infarcts: CT is normal in 25-30% at this time. Most (60-75% show subtle mass effect (effaces sulci) or density changes.

Most CVAs can be seen as areas of low density by 48 hrs.

IN 5-10 % there may be a short window (at around day 7-10) where CVA becomes isodense, called "fogging effect". IV contrast will usually demonstrate these.

CVAs are sharply demarcated by 1-2 wks, and approach CSF density in 3 wks.

Mass effect: common from day 1 to25. Then atropy usually seen by 5 wks (earliest 2 wks). Serial CT scans have shown that midline shift increases after ischemic CVA and reaches a maximun 2-4 days after the insult.

Hyperdense artery sign: high density in the configuration of a cerebral vessel on CT, usually the middle cerebral artery indicating intra-arterial clot (thrombus or embolus). Seen in 12% of 50 patients scanned within 24 hrs of CVA, and in 34% of 23 very early CTs done to rule out hemorrhage. May also be seen with carotid dissection.

Calcifications: in adults, only 1-2% of CVA calcify. Therefore, in an adult, calcifications almost rule-out a CVA.

Enchancement on CT with IV contrast in CVA:
1. **many enhance by day 6, most by day 10, some will enhance up to 5 wks**
2. **rule of 2's: 2% enhance at 2 days, 2% enhance at 2 months**
3. **gyral enhancement: called "ribbon" enhancement. Is common. It is usually seen by 1 week (grey matter enhances > white)**
4. **as a rule of thumb: there should not be enhancement at the same time as there is mass effect.**

MRI

In patients able to cooperate, MRI is more sensitive than CT (especially between 8 and 24 hrs post-ictus), especially in brain stem or cerebellar infarction. Four types of MRI enhancement patterns have been described:

1. intravascular enhancement: occurs in around 75% of 1-3 day-old cortical infarcts, and is probably due to sluggish flow and vasodilatation (thus, it is not seen with complete occlusion). May indicate areas of brain at risk of infarction

2. meningeal enhancement:especially involving the dura. Seen in 35% of carotid CVAs 1-3 days old (not seen in deep cerebral or brain stem CVAs). No angiographic nor CT equivalent

3. transitional enhancement: above two types of enhancement coexist with early evidence of BBB breakdown; usually seen after 3-6 days

4. parenchymal enhancement; classically appears as a cortical or subcortical gyral ribbon enhancement. It may not be apparent for the first 1-2 days, and gradually approaches 100% by 1 week. Enhancement may eliminate "fogging effect" (as on CT) which may obscur some CVAs at ≈ 2 weeks on unenhanced T2W1

Emergency Cerebral Angiography or three Dimension CT SCAN Angiogram of the cerebral and Aortic Arch vessels.

Is done if

1. early CVA in carotid distribution = history of amaurosis fugax or bruit or retinal emboli, et. suggesting increasing carotid stenosis, thrombogenic ulcerated plaque, or carotid dessection

2. if diagnosis still questionable (e.g. aneurysm, vasculitis)

3. with rapid recovery, suggesting carotid TIA in face of increasing stenosis

4. <u>AVOID</u> angio if unstable or if severe disabling neuro deficit. (Three Dimension CT SCAN Angiogram is safer)

Findings:

1. cutoff sign: vessel ends absruptly at the point of obstruction

2. string sign: narrow strand of contrasr in a vessel with high grade stenosis

3. "luxury perfusion": reactive hyperemia is a recognized response of cerebral tissue to injury (trauma, infarction, epileptogenic focus). Luxury perfusion is blood flow in excess of demand due to abolition of cerebral blood flow autoregulation due to acidosis. The exact nature of the perfusion is not known (i.e. it is capillary or arteriole). On angiography it shows up as accelerated circulation adjacent to the infarct with a stain or blush and early venous drainage.

Table 5.

NIH stroke Scale (NIHSS) (Score used to asses stroke patients, used by most hospitals)

1. a). Level of consciousness (LOC)

    0. **alert: keenly responsive**

    1. **not alert; but arousable by minor stimulation to obey, answer or respon**

    2. **not alert, requires repeated stimulation to attend, or is obtunded and requires strong painful stimulation to make movement (not stereotyped)**

    3. **Comatose: responds only with reflex motor (postruring) or autonomic effects, or totally unresponsive, flaccid and areflexic**

b). LOC questions

patients is asked the month and their age.

    0. **answers both questions correctly (no credit for being close)**

    1. **answer 1 question correctly, or cannot answer because of : ET tube, orotracheal trauma, severe dysarthria, languafe barrier, or other problem not secondary to aphasia**

    2. **answers neither question correctly, or is: aphasic, stuporous, or does not comprehend the questions**

c). Level of consciousness commands.

Patient is asked to open and close the eyes, and then go trip and release the non-paretic hand. Substitute another 1-step command if both hands cannot be used. Credit is given for an unequivocal attempt even in it cannor be completed due to weakness. If there is no response to commands, demonstrate (pantomime) the task. Record only first attempt.

    0. **performs both tasks correctly**

    1. **performs one tasks correctly**

    2. **performs neither task correctly**

Table 5. (Continued).

2. Test only horizontal eye movement. Use motion to attract attention of aphasic patients.

    0.   normal

    1.   partial gaze palsy (gaze abnormal in one or both eyes, but forced deviation or total gaze paresis are not present) has an isolated cranial nerve III, IV or VI paresis

    2.   forced deviation or total gaze paresis not overcome by occulocephalic (Doll's eyes) maneuver (do not do caloric testing)

3. Visual

    Visual fields (upper and lower quadrants) are tested by confrontation. May be acored as normal if patient looks at side or finger movement. Use occular threat where consciousness or comprehension limits testing. Then test with double sided stimultaneous stimulation (DSSS).

    0.   no visual loss

    1.   partial hemianopia (clear cut asymmetry), or extinction to DSSS

    2.   complete hemianopia

    3.   bilateral hemianopia (blind, including cortical blindness)

4. Facial palsy

    Ask patient (for pantomime) to show their teeth, or raise eyebrows and close eyes. Use painful stimulus and grade grimace response in poorly responsive or non-comprehending patients.

    0.   normal symmetrical movement

    1.   minor paralysis (flattened nasolabial fold, asymmetry on smiling)

    2.   partial paralysis (total or near total paralysis of lower face)

    3.   complete paralysis of one or both sides (absent facial movement in upper and lower face)

Table 5 (Continued)

5. Motor Arm (5a=left, 5b=right)

Instruct patient to hold the arms outstretched, palms down (at 90^0 if sitting, or 45^0 if supine). If consciousness or comprehension impaired, cue patient by actively lifting arms into position while verball instructing patient to maintain position.

- 0. no drift (holds arm at 90^0 or 45^0 for full seconds)
- 1. drift (holds limbs at 90^0 or 45^0 position, but drifts before full 10 seconds but does not hit bed or other suport)
- 2. some effort against gravity (cannot get to or hold initial position, drifts down to bed)
- 3. no effort against gravity, limb falls
- 4. no movement
- 9. amputation or joint fusion: explain

6. Motor leg (6a=left, 6b=right)

While supine, instruct patient to maintain the non-paretic leg at 30^0. If consciousness or comprehension impaired, cue patient by actively lifting leg into position & verbally instruct patient to maintain position. Then repeat in paretic leg.

- 0. no drift (holds leg at 30^0 full 5 seconds)
- 1. drift (leg falls before 5 seconds, but does not hit bed)
- 2. some effort against gravity (leg falls to bed by 5 seconds)
- 3. no effort against gravity (leg falls to bed immediately)
- 4. no movement
- 9. amputation or joint fusion : explain

Table 5 (Continued)

7. Limb ataxia

(Looking for unilateral cerebellar lesion). Finger-nose-finger and heel-knee-shin tests are performed on both sides. Ataxia is scored only of clearly out of proportion to weakness. Ataxia is abscent in the patient who cannot comprehend or is paralyzed.

- 0.   absent
- 1.   present in one limb
- 2.   present in two limbs
- 9.   amputation of joint fusion :explain

8. Sensory

Test with pin. When consciousness or comprehension impaired, score sensation normal unless decifit clearly recognized (e.g. clear-cut asymmetry of grimace or withdrawal). Only hemisensory losses attributed to stroke are counted as abnormal.

- 0.   normal, no sensory loss
- 1.   mild to moderate sensory loss (pin-prick dull or less sharp on the affected side, or loss of superficial pain to pinprick but patient aware of being touched)
- 2.   severe to total (patient unaware of being touched in the face, arm and leg)

9. Best language

In additional to judging comprehension of commands in the preceeding neurologic exam, the patient is asked to describe a standard picture, to name common items, and to read and interpret the standard text in the box below. The intubated patient should be asked to write.

Table 5. (Continued)

> **taukah tuan**
> **tiada sifat angkuh**
> **saya balik ke rumah**
> **dekat dengan meja makan**
> **mereka mendengar beliau bercakap di dalam**
> **radio semalam**

   0.   **normal, no aphasia**

   1.   **mild to moderate sensory loss (some loss of fluency, word findiong errors, naming errors, paraphasias and/or impairment of communication by either comprehension or expression disability)**

   2.   **severe aphasia (great need for inference, questioning and guessing by listener; limited range of information can be exchanged)**

   3.   **mute or global aphasia (no usable speech or auditory comprehension)**

10. Dysarthria

Patient may be graded based on information already gleaned during evaluation. If patient is thought to be normal, have them read (or repeat) the standard text shown in this box.

> **MAK**
> **BAGUS**
> **LIMA PULUH-LIMA PULUH**
> **TERIMA KASIH**
> **REFORMASI**
> **SEPAK TAKRAW**
> **ULAT BULU**

   0.   **Normal speech**

   1.   **mild to moderate (slurs some words, can be understood with some difficulty)**

   2.   **severe (unintelligible slurred speech in the absence of, or out of**

proportion to any dysphasia, or is mute/anarthric)

9.   **intubated or other physical barrier**

Table 5. (Continued)

11. Extinction and inattention (formerly neglect)

     Sufficient information to edentify neglect may already be gleaned during evaluation. If the patient has severe visual loss preventing visual DSSS, and the cutaneous stimuli are normal, the score is normal. Scored as abnormal only if present.

0.   **normal, no sensory loss**

1.   **visual, tactile, auditory. spatial or personal inattention or extinction to DSSS in one of the sensory modalities**

2.   **profound hemi-inattention or hemi-inattention to > 1 modality. Does not recognize own hand or orients to only one side of space**

A. Distal motor function (not part of NIHSS) (a =left arm, b=right)

     Patients hand is help up at the forearm by the examiner, and is asked to extend the fingers as much as possible. If patient cannot do so, the examiner does it for them. Do not repeat the command.

0.   **normal (no finger flexion after 5 seconds)**

1.   **at least some extension after 5 seconds (any finger movement is scored)**

2.   **no voluntary extension after 5 seconds)**

Management of RIND, Tia, or CVA

Thrombolytic therapy

Plasminogen activators catalyze the conversion of plasminogen to the fibringnolytic compound plasmin. Available agents include streptokinase and recombinant tissue plasminogen activator (rt-PA) which is FDA approved for the IV treatment of acute ischemic CVA. Streptokinase has been associated with a worse outcome at 3 months (even when given within 3 hours of onset of CVA the outcome was no better than placebo). Activase converts more fibrin-bound and less circulating plasminogen to plasmin then dose streptokinase.

Tissue Plasmonigen Activator

A randomized double-blink NINDS study of 624 patients with an ischemic stroke having a clearly defined time of onset and a CT Scan prior to drug administration, found that patients receiving alteplase as described under Rx initiated within 3 hours of the onset of symptoms had improved neurologic outcome at 3 months (these patients were 30% more likely to have minimal or no disability). No significant reduction of deficit was demonstrable at 24 hours. Although, there was an increased risk of intracerebral hemorrhage (ICH) (6.4%) vs. 0.6% with placebo), mortality was still lower in the rt-PA group at 3 mos (17% vs. 21%). In one study , ICH was more common in the presence of a hypodensity on CT, but ICH did not influence outcome except in the rare instance when a massive hematoma occurred. Exclunosionary criteria for rt-PA:

1. intracerebral haemorrhage: on admitting CT, or history of prior ICH
2. serious head trauma within past 3 months
3. SBP > 185 mm Hg, or DBP > 110 mm Hg
4. rapidly improving or minor symptoms
5. history of GI or urinary tract hemorrhage within past 21 days

6. arterial puncture at non-compressible site within past 7 days.

7. patients on anticoagulants, or those receiving heparin within the past 48 hours

8. PT > 15 seconds or platelet count < 100,000/mm^3

9. seizure at onset of CVA

10. symptoms suggestive of SAH

11. major surgery within last 14 days

12. another CVA within past 3 months

blood glucose > 400 mg% or < 50 mg%

A large placebo-controlled European trial using 1.1 mg/kg rt-PA given within 6 hours of hemispheric ischemic found no benefit when analyzed according to intention to treat. This supports the theory that the benefit to risk ratio may be lower with later initiation of treatment, higher doses, and/or inadequate control of BP.

Rx alteplase (Activaseυ): choice of two regimens to give a total of 0.9 mg/kg rt-PA(up to a maximun of 90 mg total, including any bolus)initiated < 3 hours from onset of deficit. Regimen 1) (NINDS protocol): 0.9 mg/kg IV bolus over 1-2 mins, followed by 0.81 mg/kg constant infusion over 60 minutes. Regimen 2): 0.9 mg/kg over 1 hour. The NINDS protocol required that no anticoagulants nor antiplatelat drugs be given for 24 hours after treatment, and BP was maintained as illustrated in table 27-3. Some clinicians prefer starting heparin acutely after rt-PA, however the NINDS investigators highly recommed getting a non-contrast CT first since there was a significant incidence of subclinical intracerebral hemorrhages.

Table 6.

Management of HTN after administration of rt-PA for acute CVA*

BP† (mm Hg)	Intervention
SBP 180-230 and/or DBP 110-120	labetalol 10 mg IV over 1-2 mins, repeat or double dose q 10-20 mins up to 150 mg. If labetalol contraindicated, use nifedipine 10 mg SL
SBP > 230 and/or DBP 121-140	as above, but if labetalol contraidicated or ineffective, use nitroprusside
DBP > 140	start nitroprusside 0.5-10 µg/kg/min

* this scheme is not intended for use outside of NINDS/rt-PA study

† SBP= systolic blood pressure, DBP = diastolic BP

Management of patients not undergoing thrombolytic therapy

These guidelines are for transient ischaemic attacks, RIND, or CVA, but not SAH nor intracerebral hemorrhage (ICH). The following guidelines for initial management should be maintained 48 hours after last neuro deterioration.

1. frequency vital signs with cranial checks (q 1 hrs 12 hrs, then q 2 hrs)
2. activity: bed rest
3. labs
   A. Routine: Full Blood Count + platelet count, electrolytes, PT/PTT, Blood urea, Electro cardiogram, Chest-X-ray, Arterial Blood Gases
   B. "Special: (when appropriate): TPHA /VDRL (to rule-out temporal arteritis), hepatic profile, lipid profile, Electrocardiogram (ECG)
4. O_2 at 2L per nasal cannula; repeat ABG on 2 L O_2
5. Monitor cardiac rhythm x 24 hrs (literature quotes 5-10 % prevelence of ECG changes, and 2-3% acute Mis in patients with CVAs)
6. diet: nothing by mouth
7. Nursing care
   A. indwelling Foley (urinary) catheter if consciousness impaired of if unable to use urinary or bedpan; intermittent catheterization q 4-6 hrs PRN no void if Foley not used.
   B. accurate input and output ; notify doctor for urine output < 20 cc/hr for 2 hrs by Foley, or < 160 cc in 8 hrs if no Foley catheter
8. IV fluids: NS or 1/2 NS at 75-125 cc/hr for most patients (to eliminate dehydration of present
   A. avoid glucose: hyperglycemia may extend ischemic zone (penumbra). Although hyperglycemia may be a stress response and may not be neurotoxic, recommendations are to strive for normoglycemia.

B. avoid overhydration in cases of intracerebral haemorraghe, congestive heart failure or SBP > 180. It had been suggested that an optimal haemotocrit for compromise between O_2 delivery and decreased viscosity was around 33% and that fluid management should strive for this, however the early promise of this theory has not been borne out

9. treat congestive heart failure and arrhythmias (check CXR & ECG). MI or myocardial ischemia may present with neuro deficit, these patients should be admitted to CCU

10. avoid diuretics unless volume overloaded

11. blood pressure (BP) management:

    A. for patients presenting with hypertension: management must take baseline BP into account

    B. for patients presenting with hypotension (SBP < 110 or DBP < 70):

        1. unless contraindicated (viz:ICH, cerebellar infarct, or decreased cardiac output) give 250 cc NS over 1 hr, then 500 cc over 4 hrs, then 500 cc over 8 hrs

        2. if fluid inffective or contraindicated: consider pressors

12. medications

    A. ASA 325 mg PO q d (unless hemorrhagic stroke proven or suspected

    B. stool softener

Hypertension in stroke patients

Hypertension may actually be needed to maintain CBF in the face of elevated ICP, and it usually resolves spontaneously. Therefore treat hypertension cautiously and slowly to avoid rapid reduction and overshooting the target. Avoid treating mild hypertension . Indications to treat hypertension emergently include:

1. acute ventricular failure (rare)

2. acute aortic disease (rare)

3. acute hypertensive renal failure (rare)

4. neurologic complications of hypertension

    A. hypertensive encephalopathy

    B. converting a massive pale (ischemic) infarct into a hemorrhagic infarct

    c. patients with ICH (some hypertension is needed to maintaon CBF)

Hypertension treatment algorithm (modified)

1. If DBP > 140 (malignant hypertension): ≈ 20-30 % reduction is desirable. Sodium nitroprusside IV drip or IV hydralazine are agents of choice; arterial-line monitor recommended; sympatholytics contraindicated (they reduce CBF)

2. SBP > 230 or DBP 120-140x20 mins; labetolol (unless contraindicated, start at 10 mg slow IVP over 2 mins, then double q 10 min (20, 40, 80, then 160 mg slow IVP) until controlled or total of 300 mg given. Maintenance: effective dose (from above) q 6-8 hrs PRN SBP > 180 or DBP >110

3. SBP 180-230 or DBP 105-120: defer emergency treatment unless there is evidence of LV failure or if readings persists x 60 mins

    A. unless contraindicated oral labetolol dosed as follows

        1. for SBP > 210 or DBP >110: 300 mg PO BID

        2. for SBP 180-210 or DBP 100-110: 200 mg PO BID

    B. if labetolol contraindicated:

        nifedipine start with 10 mg PO/SL, if still HTN after 1 hr, give 20 mg; then follow with 10-20 mg PO or SL q 6 hrs

    C. if monotherapy fails, or labetolol contraindicated, try either:

        hydralazine 10 mg slow intravenously QID q 6 hrs

       OR

        captopril 6.25 mg, 12.5 mg or 25 PO q 8 hrs

    4. SBP < 180 or DBP < 105: antihypertensive thrapy usually not indicated

Anticoagulations (heparin, etc.) and ASA.

Effectiveness is unproven in strokes and TIAs except with cardiogenic brain embolism. Anticoagulation may also be hazardous. Conversion rate of pale → haemorrhagic CVA is 2-5% (dog studies suggest the risk is increased only when HTN not well controlled). Conclusion : the risk of heparin therapy for acute focal cerebral ischemia exceeds any proven benefit; this treatment is not justified in most cases (especially when used just to placate the frustrated clinician). High-intensive warfarin therapy has proven helpful for the antiphospholipid antibody syndrome (APLAS).

For the rare indication for anticoagulation therapy:

1. first R/O bleed by CT before beginning therapy
2. ASA in all patients with non-hemorrhagic CVA where anticoagulants contraindicated(NB: angiography may be slightly more diffucult in patients on ASA)
3. anticoagulants (heparin/warfarin):
    A. indications (rare)
        1. probably effective for cardiogenic emboli
        2. shown ineffective for stroke in evoluation (neuro deficit that begins, recurs, fluctuates, or worsens while patients in hospital), crescendo TIA or completed stroke
        3. unproven, but generally used for carotid dissection
    B. Contraindicated with large cardiac embolism, large stroke (risk of hemorrhagic conversion), peptic-lcer disease that has bled in past 6 mos, uncontrolled severe hypertension

C. Start IV heparin and stimultaneous warfarin (maintain heparin during first ≈ 3 days of warfarin because of initial hypercoagulability

D. Stop warfarin after 6 months (benefits decline, risks rise)

Nootropics

Nootropics such as piracetam are given as a start dose on intravenously 12 gm bolus followed by 4 gm TDS intravenously and later 800mg TDS or QID. Literature studies have shown improvement of patients when started early.

Dexamethasone and steroids

Dexamethasone and steroids is used in these cases:

1. For steroid responsive vasculitis (e.g. giant cell arteritis, temporal arteritis
2. if cerebellar brain stem infarct/bleed with mass effect suspected
3. large volume cerebral haemotomes with mass effects

Mannitol

Mannitol is used:

1. for cerebral infarct/bleed, prior to surgery, or if mass effect
2. contraindicated im hypotension
3. does not work in hydrocephalus especially of the obstructive type.

Early findings

In most cases the onset is sudden, without premonitory symptoms. The first 12 hours after onset were characterized by lack of progression. Early findings are due to the intrinsic cerebellar lesion (ischemic infarction or hemorrhage:

1. Symptoms
   A. dizziness or vertigo
   B. nausea/vomiting

C. loss of balance, often with a fall and inability to get up

    D. headache (infrequent in one series)

2. Signs

    A. truncal and appendicular ataxia

    B. nystagmus

    C. dysarthria

Later findings

Patients with cerebellar infarction may subsequently develop increased pressure within the posterior fossa (due to cerebellar edema or mass effect from clot), with brain stem compression (particulary posterior pons). Clinical findings generally increase between 12-96 hrs following onset.

80% of patients developing signs of brain stem compression will die, usually within hours to days. Surgical decompression should probably be done as soon as any of the following signs develop if there is no response to medical therapy. Findings proceed in the approximate following sequence if there is no intervention:

1. abducens (VI) nerve palsy

2. loss of ipsilateral gaze (compression of IV nucleus and lateral gaze center)

3. peripheral facial nerve paresis (compression of facial colliculus)

4. confusion and somnolence (may be partly due to developing hydrocephalus)

5. Babinski signs

6. hemiparesis

7. lethargy

8. small but reactive pupils

9. coma

10. posturing → flaccidity

11. ataxic respirations

Imaging studies

CT scan may be normal very early in these patients. There may be subtle findings of a tight posterior fossa: compression or obliteration of basal cisterns or 4th ventricle.

MRI is more sensitive, but may be difficult to obtain quickly, and is very difficult in a ventilated patient.

Neurovasular Emergency Surgery for Stroke.

There are several specific areas where acute surgical intervention can be beneficial in stroke patients. Surgical reconstruction of the cervical carotid artery is indicated in patients with crescendo TIAs who are otherwise in good neurologic condition on an emergency basis, and is considered urgent in patients with greater than 70% stenosis who are neurologically symptomatic. In patients likewise in good neurologic condition, emergency reopening of an occluded carotid is an appropriate procedure, albert the circumstances rarely a rise. Surgical timing and availability of collateral circulation are the key considerations in consedering microsurgical embolectomy of intracranial vessels, and microsurgical exploration is likely to be performed in all cases of sons if nothing else. Decompressive procedures for both cerebral and cerebellar infarction are readily performed and may be helpful with well-defined criteria for patient salvage and avoidance of vegetative survivors.

A side from these procedures, most stroke surgery falls into the realm of subacute management, including most cases of cerebral revascularization and many cases of carotid artery reconstruction. Nonotheles, with the increasing awareness of "brain attack" as a medical emergency as severe as heart attack, it behooves the neurosurgical practitioner to be aware of the strict and well-defined indications for emergency surgical intervention in stroke patients, and to be conversant with the surgical techniques involved.

Decompression Procedures for Cerebral Infarction

There appears to be a small subgroup of patient following massive supratentorial cerebral infarction who qualify for a decompressive procedure of some kind to prevent uncal herniation and death. Like most other stroke surgery patients, they must be very carefully selected. The aim decompressive surgery following cerebral infarction is patients salvage during the acute period of brain swelling, and this approach is reasonable only if other factors are in place that would indicate a potential for significant neurologic recovery.

Published decompressive techniques for post-infarct brain swelling include hemicraniectomy with duraplsty, and the more invasive craniotomy with resection of infarcted tissue and/or uncal resection. A number of authors have described the technique of hemicraniectomy with dural opening and either application of a Silastic sheet for brain protection or placement of watertight Lyodura or pericranial grafts. Other groups have advocated resection of infarcted cerebral tissue and/or temporal lobectomy including removal of the herniated uncus. Both types of procedures appear to yield satisfactory outcome results.

No matter what the surgical method, it is clear that strict preoperative selection guidelines need to be followed. First, most reported cases are in the nondominant hemisphere, thereby satisfying thr criterion that the patient has a chance for a reasonable neurologic recovery. Second, as emphasized by Rengachary et al, it is recommended that conservative measures such as hyperventilation, infusion of mannitol, and administration of steroids be exhausted before consideration is given to surgical decompression. Whether barbiturate coma should be instituted prior to surgical treatment is an open question. Young et al emphasize strongly that it is imperative to perform the decompressive procedure priorto frank clinical deterioration from as herniation syndrome

if good surgical benefit is to be obtained. They and others recommen continuous intraranial pressure (ICP) monitoring to determine when medical treatments are no longer of significant benefit and surgical therapy needs to be instuted.

In conclusion, there is a small group of cerebral infarct patients, primarily with large nondominant infarcts in younger age groups, who may benefit from decompressive procedures for ICP. As in all forms of stroke surgery, patient selection and meticulous attention to management details are crucial to ensuring maximization of successful outcomes.

Decompressive procedures for cerebellar infarction

The possibility of surgical intervention to relieve posterior fossa swelling and brain stem compression in cases of cerebellar infarction were first described in 1956 by Fairburn et al and, in a separate report by Lindegren. Sporadic reports of clinical successes with various treatments for cerebellar infarction appeared in the literature prior to the advent of CT scanning, but the diagnostic capabilities prior to CT and magnetic resonance (MR) imaging were quite limited. Sypert and Alvord described extensively the pre-CT manifestations of cerebellar infarction, including the low percentage of correct diagnose. Until tomographic imaging was available, diagnosis rested upon both clinical grounds and the angiographic appearance of an a vascular posterior fossa mass with arterial occlusion. All too often in these patients the diagnosis was unfortunately made postmortem.

The treatment of cerebellar infarction has progressed remarkably with the availability of tomographic imaging. patients with early signs of posterior fossa compression can have immediate confirmatiom of cerebellar hemorhage, cerebellar infarction, or other spaceoccupying processes in the posterior fossa as well as the presence of associated hydrocephalus, and management schemes can be formulated accordingly.

Several treatment strategies have been proposed for cerebellar infarction with posterior fossa mass effect. Khan et al patients treated either conservatively or with ventricular drainage only, attributing their single poor result to brain stem infarction at the time of vertebral artery occlusion. Horwitz and Ludolph proposed a combined medical and surgical scheme in which patients were first intubated and received dexamethasone and mannitol. A ventriculostomy was then placed if the state of consciousness did not improve with medical treatment., and likewise, suboccipital craniectomy was proposed if the level of consciousness did not improve within a few hours ventriculostomy. The most recent references on this subject, however, stress that the surgical procedures proposed are relatively benign compared to the malignant natural history of the disease and the rather fulminant course that can occur with compression in the posterior fossa. Thus, both Chen at al and Heros recommend combined ventricular drainage and suboccipital decompression with resection of infarcted tissue as primary treatment. With this strategy, Chen et al reported functionally independent survival in 8 of 11 patients.

The surgical procedures are straightforward and within the province of any neurosurgeon. A simple suboccipital craniectomy is performed with opening of the dura and resection of infarcted tissue followed by meticulous hemostasis. As would be suspected from the patients for arterial occlusion, almost all such operations will be unilateral. The ventriculostomy is performed according to standard landmarks and long-term conversion to a shunt may be necessary based on the guidance of followup tomographic scans.

Several authors stress that even patients in poor clinical condition (i.e. severely depressed level of consciousness) can do well because of the physiologic mechanism of brain stem compression accounting for their deficits. It must be understood, however vertebral artery infarcts can also involve the brain stem primarily, and, of course, the prognosis would be expected to be much poorer in patients of this kind.

A single cooperative trial is in progress to evaluate differential treatments for cerebrellar infarction. The German-Austrian Space Occupying Cerebellar Infarction Study (GASCIS) will enter patients into three treatment arms on a prospective basis. First, awake patients will be treated conservatively with hyperventilation, osmotic diuresis, and

best medical care. Second, drowsy patients will be treated either conservatively or with ventricular drainage, and third, comatose patients will be treated with either surgical decompression alone or surgical decompression with concomitant ventricular drainage. As of this writing (1993), the study design calls for 180 patients with at least 2 years for patient entry before any analysis can be proposed.

In conclusion, the clinical syndrome of posterior circulation arterial occlusion and cerebellar infarction has been increasingly recognized since the advent of tomographic imaging. An aggressive surgical approach is warranted since brain stem compression is potentially reversible in many of these patients. At present, surgical decompression of the posterior fossa and resection of infarcted tissue with or without ventricular drainage for concomitant hydrocephalus are recommended with the unserstanding that patients with infarction of the brain stem may represent a subgroup more likely to have a poor outcome.

Rationale for subacute surgical management of stroke

A side from the emergency surgical indications previously discussed, there is a large population of stroke patients who will warrant subacute intervention for stroke prophylaxis. The aim of such procedures is not salvage, but rather to prevent the recurrence of stroke events from the original source, whether embolic or hemodynamically mediated. Some of these procedures (carotid endarterectomy, extracranial-intracranial [EC-IC] bypass) have undergone scientific scrutiny, whereas others (posterior circulation revascularization, stump syndromes) are rare enough that they may remain empirically indicated only.

Carotid endarterectomy

Carotid endarterectomy is one of the most frequently performed vascular operations. Several recent studies have provided some guidelines for performing carotid endarterectomy in selected patients with carotid artery territory TIAs or nondisabling strokes. NASCET has demonstrated that surgery is highly beneficial for symptomatic

patients with ipsilateral 70% to 99% ICA stenosis defined by angiography. The study is ongoing for symptomatic carotid artery stenosis between 30% to 69%. The European Carotid Surgery Trial (ECST) showed that surgery was benefial for symptomatic patients with angiographically defined ipsilateral 70% to 99% ICA stenosis. Carotid endarterectomy was not beneficial for symptomatic patients with less than 30% ICA stenosis. In the VA Cooperative Study, surgery was beneficial for symptomatic patients with angiographically defined ipsilateral ICA stenosis of greater than 50%. It is recommended that patients continue taking aspirin after carotid endarterectomy.

Prophylactic surgical management with carotid endarterectomy for patients with asymptomatic advanced ICA stenosis is controversial. Prospective, randomized trials comparing medical and surgical therapies in patients with asymptomatic ICA stenosis include the VA Cooperative Trial on Asymptomatic Carotid Stenosis, the Asymptomatic Carotid Atherosclerosis Study (ACAS), the Carotid Artery Stenosis with Asymptomatic Narrowing Operation Versus Aspirin (CASANOVA)study, and the Mayo Asymptomatic Carotid Endarterectomy Trial (MACE). The CASANOVA study did not show any benefit in morbidity and mortality of carotid endarterectomy for patients with asymptomatic ICA stenosis of less than 90%. No recommendations were made for patients with higher (greater than 90%) grades of stenosis, since such patients were excluded from this trial. The MACE study, which was published in 1992, was interrupted. If anything, findings from this trial point to the benefit of prophylactic aspirin in preventing myocardial infarction in patients having carotid endarterectomy. The VA trial randomized 444 male patients in 1987, and clinical followup of the medically and surgically treated patients has been completed. Findings from this trial showed that carotid endarterectomy reduced the overall incidence of ipsilateral neurologic events (TIAs included) for patients with asymptomtic ICA stenosis of 50% or more. However, this trial found no significant effect from carotid endarterectomy on the combined incidence of stroke and death.

Extracranial-intracranial anastomosis

The development and wide application of EC-IC bypass procedures (most commonly superficial temporal artery [STA] to MCA bypass) appeared to hold great promise in the treatment of ischemic cerebrovascular disease, and was well received by the neurovascular community. A large randomized trial, however, failed to demonstrate any significant difference between medical treatment and STA-MCA bypass, and has reduced the number of surgical bypass procedures dramatically. Following the release of the EC-IC bypass trial results, a number of criticisms and rebuttals have centered on the study design, particularly the question of large numbers of eligible patients being operated upon outside the trial and perhaps biasing the results against a favorable surgical outcome. Despite this level of debate, however, there appears to be little doubt that the bypass trial data will stand, and that the use of STA-MCA or other bypass procedures will now be limited to special clinical situations not addressed by the cooperative study. These would include moyamoya disease, preoperative preparation for elective carotid sacrifice, and, as suggested by Awad and Spetzler, cases of documented medical failure or well-studied hemodynamic compromise. The degree to which bypass surgery will gain acceptance on such cases remains to be determined.

Other reconstructive vascular procedures

Other procedures, such as posterior circulation bypass surgery, endarterectomy of the vertebral artery, creation of arterial anastomoses between blood vessels, correction of subclavian steal, transluminal angioplasty, or other intra-arterial procedures, have not been tested sufficiently according to the evidence available to make any specific recommendations. Further carefully designed studies will have to be performed to see if there are any subgroups of patients who might benefit from these surgical reconstructive procedures.

Future "emergency" management of stroke

With the publication of Borlongon CV et al from the University of South Florida College of Medicine (USA) and the use of HNT neurons and chromaffin cells in the stroke patients, we will see "startrek" and "tailor-made" management where transplantation may bedone as an emergency procedure by a neurosurgeon. Depending on the stage of the cerebral vascular accident, patients may be managed with combination of drugs and surgery according to certain standards and scores yet to be determined. As for any other disease, prevention is still better than cure. **Remember** : BRAIN ATTACK IS AS FATAL AS HEART ATTACK!!!!!