Wednesday, 8 November 2017

- **Topic 1: Stroke in Sub-Saharan Africa**
- 9:45 – 10:15: Epidemiology and Classification *A. Gallo Diop, Senegal*
- 10:15 – 10:45: Clinical presentations, signs and symptoms, including videos *P. Sandercock,*
- *10:45 – 11:00: coffee break*
- 11:00 – 11:30: State of the art in diagnostic work-up and therapeutic management *D. Leys, France tbc*
- 11:30 – 12:00 Diagnostic and therapeutic work-up in urban and rural SSA – what can be done, what must be done *Yomi Ogun,Nigeria*
- 12:00 – 12:15: Discussion

- A Lecture delivered on the 8th of November 2017 at the
  - REGIONAL TRAINING COURSE
- on
- Diagnostic and Therapeutic work-up in urban / rural sSA –
- what can be done, what must be done

- By
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- LASUCOM / LASUTH IKEJA NIGERIA

- Venue:. OAGADOUGU , BURKINA FASO

Lecture Overview

Need to know

Diagnostic WORK- UP

Therapeutic WORK-UP
urban / rural SSA –

what can be done ?
what must be done ?

Consequences / Impact on health related Quality of Life

Way forward / Roadmap

Holistic approach

- Africa: CONTINENT : 52
- WHO: 47
- WORLD BANK : 2
- UNICEF : 3

- African > ***1 B***: 1/7th of world popn.
- X7 morbidity/mortality
- Peculiarities in sSA
- Risk factors
- Epidemiological transition

- Diagnostic WORK- UP
- Therapeutic work-up
-
- Way forward

AFRICA

802984AI (R02109) 6-03

If A Single Teacher Can't Teach Us All The Subjects, Then..
How Could You Expect A Single Student To Learn All Subjects??

Introductn: Brain Attack / Stroke

Acute neuro-vascular syndrome

666 (Revelation 13/18)

(1 in 6 individuals dev stroke in lifetime.**;**

q 6s, one patient dev stroke q 1 min (? 2s)

q 6s, 1 death recorded in its favor;

6M death worldwide

Significant economic, social, public health and medical problem

loss of productivity and burden on families, caregivers and society

- Most common cause of acquired disability (DALYs)
- Second leading cause of *preventable* deaths in adults
-
- ***Preventable***: but preventive efforts are still far from optimal .
- ***Treatable***: evidence-based Rx available, but not fully used in an region, especially low resource areas

Brain Attack / Stroke

- ***Peculiarities in sSA***

- Changing global and African epidemiology of stroke

- WHO (2001): death from stroke and disability adjusted life years due to stroke > 7 times higher in low- and middle-income countries (LMIC) than in high income countries (HIC).

- Deaths in developing(LMIC): ***85.5% of strok deaths worldwide***
- 87% of global stroke mortality in 2005 (a > 1.5% cf with 2001)

DALYs) : Number of disability-adjusted life years –
(yrs of life lost + yrs lived with disability),

EXTRACRANIAL / INTRACRANIAL

EXTRACRANIAL

- endothelial plaque / plaque rupture
- Common in whites
- Common in males
- Associated with hyperlipidaemia
- Smoking
- Assoc PVD and CAD
- > CRP (? Genetics)

INTRACRANIAL

- Insitu thrombus
- Smaller vessels
- Common in blacks and Hispanics
- Commoner in females
- Associated with hypertension
- Associated with D.M / Metabolic Syndrome.

Extracranial / Intracranial (BP= PR X SV)

Extracranial

- > peripheral resistance
- Haemodynamic probl. >.
- Artery to artery embolism
- >TIA (< stroke)
- Less progressive
- clopidrogel useful
- Statin useful

Intracranial

- Hypervolaemia
- Haemodynamic problem
- Perforator occlusion
- Circulating endothelial particles (CEP)
- >strokes (<TIA
- More progressive
- Aspirin + cilostazole
- Statins

STROKE IN AFRICA

- Recent incidence indicate that whereas stroke incidence < 12% in HIC, it > by 12% in LMIC over the ***last decade***.

- Exploding, neglected burden of ***NCD***s: HBP, DM, dyslipidaemia, which often culminate in S

- The burgeoning/ > incidence driven by ***epidemiological transition: an ageing popn, popn growth, rapid urbanisation and accompanying lifestyle changes.***

STROKE IN AFRICA

- Peculiarities of S in people of African ancestry: ***Enhanced predisposition,***
- ***different pattern of subtypes,***
- ***worse severity // poorer outcome / younger***
- INTERSTROKE study: *Afric*: CI 66% / ICH 34%

HIC: CI 91% , ICH 9%

CI subtypes: small vessel (27%), cardio-embolism (25%), large vessel (14%), others (20%) and undetermined (14%).

STROKE IN AFRICA

- African Americans:
- higher predisposition; worse severity
- poorer outcomes cf to Caucasian Americans.
- multi-ethnic South London Stroke Registry: black stroke survivors:
- worse cognitive outcome cf to other racial groups. ?
- ?S-E differences,
- disparities in healthcare-seeking practices
- differential access to healthcare services,
- influence of underlying differences in genetic factors
- interactions bt genes / environmental factors: ***cultural practices and health-seeking Behaviour*** / distinct potential impact of genomics.

CVD and major risk factors

Non-modifiable Risk Factors
- Age
- Sex
- Genes

Behavioural Risk Factors
- Tobacco
- Diet
- Alcohol
- Physical Activity

Genes
Socio-economic, Cultural & Environmental Conditions

Intermediate Risk Factors
- Hypertension
- Blood lipids
- Obesity / Overweight
- Glucose Intolerance

HIV

NEW:
OBSTUCTIVE SLEEP APNEA,
ELECTRONIC SMOKING,
> LIPOPROTEIN

Atherosclerosis

Endpoints
- Stroke
- Kidney diseas
- Ischaemic heart disease

Retinopathy

Stroke in the Young

- 15-45yrs; (40% Haemorrhagic)
- Migraine ;
- Heroine, cocaine, methamphetamine, oral contraceptives ;
- prothrombotic state; Coagulopathies vasculitis / CTD; Haemoglobinopathy; HIV
- *Mitral valve prolapse ; Patent foramen ovale;*
- Arteritic (Takayasu ,kawasaki)
- Moyamoya syndrome
- Cardiac: - congenital cardiac lesions atrial ventricular and pulmonary vascular shunting,
- Tetralogy of Fallot's; Cardiomyopathies
- Dissection and traumatic vascular injuries
- Cerebral venous thrombosis:Infectn of the endothelium

New Risk factors – MELAS CADASIL / CARASIL

- Mitochondrial Encephalomyopathy with Lactic Acidosis and Stroke-like episodes;
- **Cerebral Autosomal Dominant / Recessive Arteropathy with Subcortical Infarcts and Leucoencephalopathy**
- ***CARASIL***: + Teen alopecia, spondylosis ; mutation of TGF (< signalling)
- **HYPERHOMOCYSTEINAEMIA cystathionine synthase deficiency tendency to venous / arterial thrombosis ; Folate deficiency predispose to > homocyst. Final common pathway for MI / CI**

Stroke and HIV

> risk for CI than ICH.

CI (stroke-like syndrome: stroke mimics) **:**
non-bacterial thrombotic endocarditis
Cryptoccocoma;Tuberculoma; Toxoplasma abscess;Kaposisarcoma;cerebral lymphoma

Intra cerebral haemorrhage:
Thrombocytopenia/Thromboasthenia (Thrombocytopathy), Primary CNS lymphoma; metastatic kaposi sarcoma.

Pathobiology**:– : vasculitis - HIV associated vasculopathy,**
> deposition of circulating Ig complexes;
direct toxic effect of virus on the vascular endothelium.
Protein S deficiency
Anti-phospholipids antibodies, including anticardiolipin antibodies - high frequency .

Aetiology / Risk factors - ICH

Cerebral Amyloid angiopathy (CAA)
elderly non- hypertensive with recurrent lobar hemorrh.
Neoplasm.

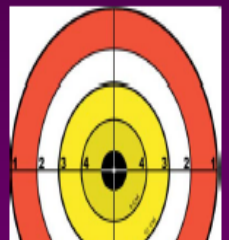
- **Coagulopathy-Anticoagulant / Thrombolytics**
- **systemic dx**
- **A.V. Malformation / Aneurysm**
- **Drugs (Cocaine/Amphetamine)**
- **memantime;**
- **Atorvastatins; celecoxib**
- **12% of Nig. < 40 years – risk factor^0**
- Age, male sex, hypocholesterolaemia / Low Triglyceride
- Alcohol abuse,(SAH: Linear; ICH: U; CI: J)
- ilicit drugs, iatrogenic
- Diet high in salt and saturated fat intake
- Diet low in K+, polyunsaturated fatty acids and fish oil.
- Physical inactivity , stressful life events; Low SE class
- The association is strong, dose related and consistent

Mechanism of Stroke

Stroke Prevention: Mechanism-Specific Considerations

What is the cause of the initial cerebrovascular event?

Large vessel athero	Cardioembolism	Small vessel dz
Carotid endarterectomy Antiplatelet agent	Anticoagulation	Antiplatelet agent



PATHOPHYSIOLOGY/genesis

C.I - Occlusion in absence of adequate collateral circulation

ie: blockage of capillaries/arterioles

- **Collaterals (Leptomeningeal / circle of Willis);**
- **relevant with proximal occlusion.**
- **Irrelevant with lacunar infarcts b/c end arteries**

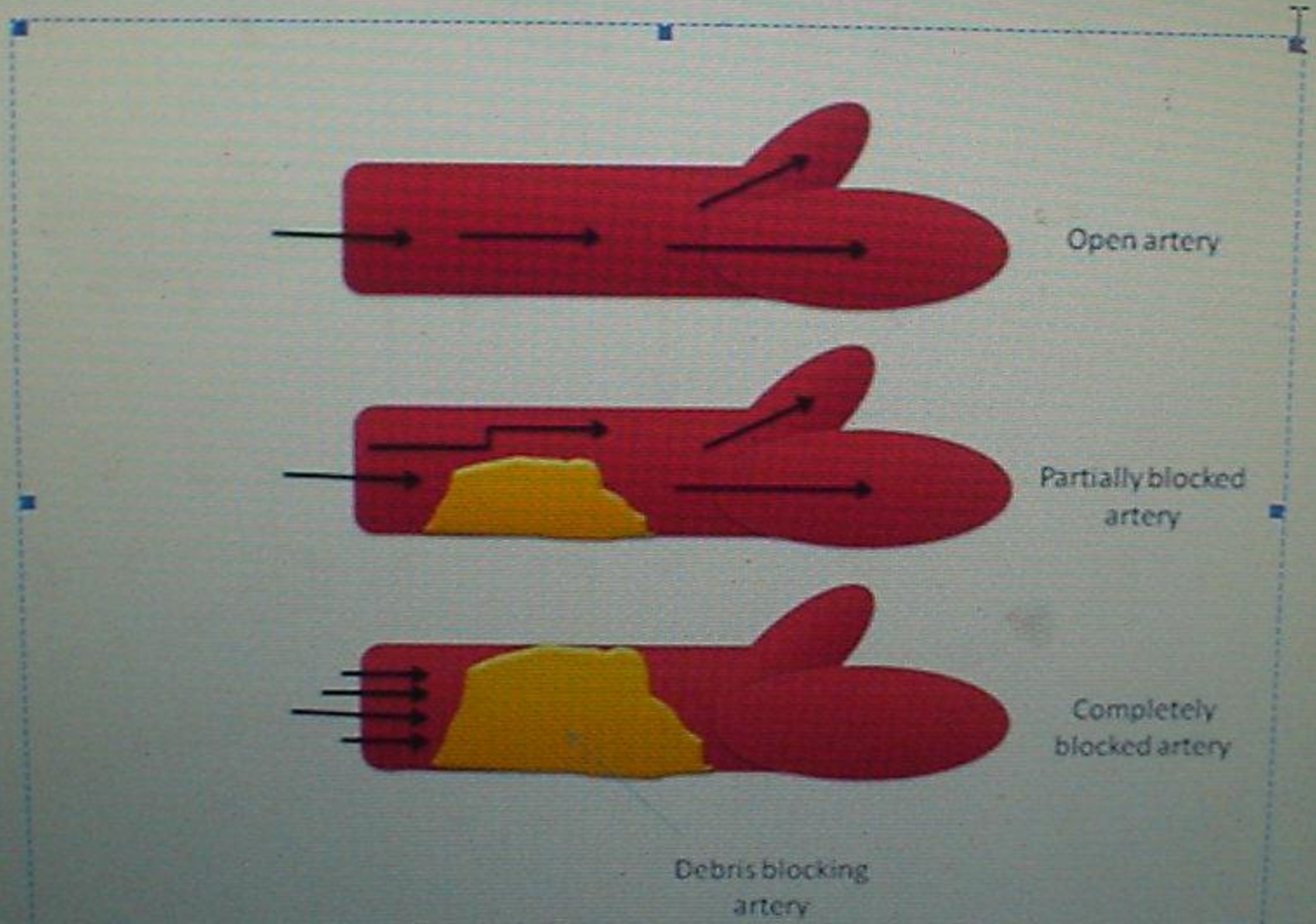
- **Thrombosis / Embolism / Vasospasm**
- **HBP: Lipohyalinosis – Charcot–Bouchard aneurysm**
- microatheroma

- **Haastrup : Penumbra (Apoptosis); Umbra (Necrosis)**
- **Penumbra = Ishaemia minus infarction**

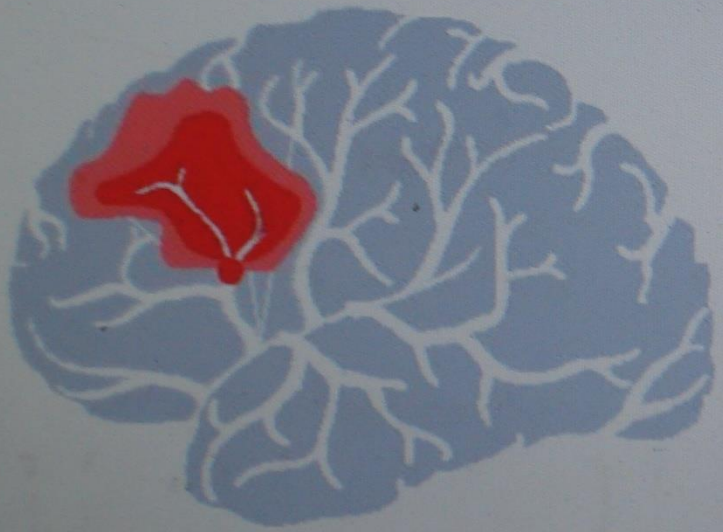
Images of stroke

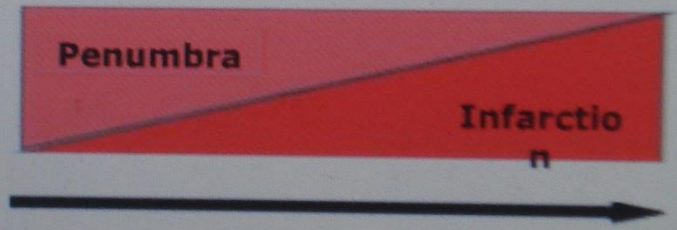
Image 1:

If the artery becomes blocked fully, there is no blood flow to the brain. See image below.



Concept of the Ischemic Penumbra





PenumbraInfarct core

1 hour 2 hours 3-4 hours 4-6 hours

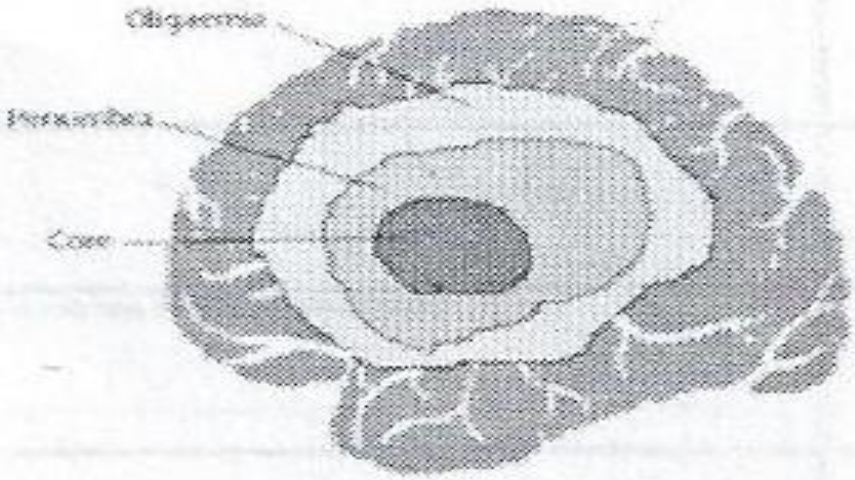
Time is Neurone / Brain

The Concept of Physiologic Time

Core: Tissue that will inevitably die and is beyond salvage

Penumbra: Total area of hypoperfusion

Benign Oligemia: Tissue that will most likely survive even without reperfusion



"Imaging of Acute Stroke." Muir et al. Lancet 2006

Pathophysiology

- **global disruption of brain metabolic process→ Energy substrate delivery:O_2 / glucose / blood flow**

Stroke is flow; Everything about stroke is flow

- ***Haemorrheology*** **: rbc concentration/ aggregation /deformability; platelet aggregation/ adhesiveness; fibrinogen level /Plasma/whole blood viscosity**

progression and extent of ischemic injury is influenced by: Health of systemic circulation;

Status of collateral circulation; age; co-existing metabolic abn. (hyperglycaemia) Premorbid medicatn + confounding factors. The rate of onset and duration,-

- **Hematological factors,- Temperature & glucose metabolism.**

0_2→3.5ml/100g/min; glucose→5mg/100g/m

- **2% body wt: 20% blood vol; 20%Oxygen**
- **Autoregulation: (> O2 extraction if it fails)**

Pathophysiology

- **blood flow → 55ml/100g/min**
- **75ml/100g → grey; 30ml/100g → white**

<25ml – EEG diffusely slowed (met.enceph

<15ml → e- activity ceases

functional threshold: **fn ceases (penumbra)**

(apoptosis – caspace)

reversal of ischaemia: Therapeutic window

- **2-3 hrs (animal); 5-6 hrs (primates)**
- **<10ml → irreversible :** ***morphological threshold:*** **cell death ensue necrosis (Umbra): necrosis - lysosomal protease)**
- **CPP = MAP – ICP;**
- **CPP = 70-100; MAP=100-130; ICP= 30-60**

Table 1 :PHASES OF CONTEMPORARY MANAGEMENT OF STROKE

Phases	Period from onset	Activities	Prefered location
1Acute (emergency) care: Hyperacute: 4.5hrs Acute : 48hrs	1st-7th day	a)Assessment b)Early supportive care	Stroke Unit Hospital
2 Early sub-acute(supportive) care	2nd-4th week	a)prevention and treatment of complications	Hospital
3 Late sub-acute(maintanance) care	2nd-6th month	a)Rehabilitation b)Psychological support c)Prevent recurrence	Hospital/Community
4.Long-term (chronic) care	7th month onwards	a)Rehabilitation b)Psychological support c)Social support d)Prevent recurrence	Community

If you do have a stroke, act ***fast***

- Do not hesitate.
- Do not ignore this type of symptoms as they may herald a big stroke which could become permanent and devastating.
- Most people do not present to the doctor in time and this delay could be very costly.
- , Do not go to bed and hope it will go away by the time you wake up. You may not wake up.
- Act **FAST** if you think someone may be suffering a STROKE:
- **S**=SMILE, **T**= TALK, **R** = RAISE ARM; ***OK***

ACT FAST at the First Sign of STROKE

www.strokemn.org

Minnesota Stroke Association

Chapter of the National Stroke Association

Mobile stroke unit

“Brain attack!"

- describes the acute presentation of stroke emphasising the need for urgent action.
- Concept is very important to sensitize physicians and the public to the need for rapid mobilization and Rx.
- Requires educational programs directed at the general public, general practitioners, and primary and emergency department physicians,
- Teach recognition of symptoms and the importance of ***Rx with the same urgency as for heart attack and acute trauma***.

Brain Attack /Time is Neurone

- The longer the delay b4 active / effective Rx, the more brain tissue is lost by the patient.
- *TIME LOST = BRAIN LOST*
- acute intervention to restore blood to the brain must take place within a few hours. TIME is "NEURONE"
- Damaged brain and the surrounding at risk brain need to be protected as soon as possible. ***Time is "Brain"***
- *> 80% present after 24 hrs (Ogun et al WAJM 2000*

Time is Neurone / Time is brain

- 200 Billion Neurone in the brain
- 2 million neurone die / minute.
- **Time is brain is the key concept**
- **200 billion neurons in the brain**
- **2 million neurons lost per minute**
- **Age 3.6 years per hour of hypoxia**

Lost / hr : 830 billion synapses *(14 billion synapses /min);* Lost / hr:

714 km of myelinated fibres *(12 km fibres / min)*

TIME IS BRAIN;
TIME IS NEURONE

- TIME IS BRAIN;

- TIME IS NEURONE

Road map = 6 R

- Recognition
- Reaction - ambulance services
- Response - emergency dept
- Reveal - CT scan
- Rx
- Rehabilitation

Road Map

Timeline of Care – NINDS Recommendations

- ED physician sees patient within **10 minutes**
- Stroke physician notified within **15 minutes**
- CT scan is completed within **25 minutes**
- CT interpretation is obtained within **45 minutes**
- IV rtPA should be initiated within **60 minutes**
- *Mobilize for IA therapy as rapidly as possibly*

Treatment

Aims to

- **a. Protect ischemic brain tissue from necrosis**
  - attempt to reverse/limit the degree of brain dysfn

- **b. Rx underlying disease process if possible**

- **c. Prevent and treat complications**

- **d. Rehabilitate the disabled patient**
  physio / occupational /speech / swallow Rx

- **e. Prevent reoccurrence**

Treatment

- Thrombolytic therapy
- Early antithrombotic treatment
- Treatment of elevated intracranial pressure
- Prevention and management of complications

- No 2 strokes are alike / same for 2 individuals
- Need to individualize Rx
- Response not the same for 2 individuals

Treatment

- **Traditional: '*wait and see*'**
- **Now: '*Watch and intervene as appropriate*'**
- ***Stroke unit*: multidisciplinary committed professional staff: Physician: neurosurgeon; physiotherapist; occupational / Speech (largo paedics) swallow therapist; nursing staff and social worker**

< mortality; < morbidity in survivors (> 30%)

< need for institutional care; < dependency

- **need for:**
- **improved case identification (Recognition)**
- **immediate transfer of patients and (Reaction)**
- **mobilisation of health personnel (Response)**

Stroke Unit

- ***All stroke pts: irrespective of gender, age, stroke type, subtype and severity,***
- dedicated / geographically defined part of hosp/ specialisd staff wt coordinated. multidisciplinary expert approach to Rx/ care; Keep > = 24 hrs
- *Assessment and diagnosis (*exclude mimics)
- *early assessment of nursing and therapy* needs
- *Early mobilisation, prevention of complications,*
- *Rx of* hypoxia, >glycaemia, pyrexia and dehydration.
- 6H:Hydration, Hypoxia, Hyperglycaemia, Hypogycaemia, Hypertension, Hyperpyrexia
- *Ongoing rehabilitation; Coordinated multidisciplinary*

Stroke Unit

- ***diagnostic tests to df types of stroke, assess*** underlying cause of CI, prognosis, rule out other brain diseases or stroke mimics, identify concurrent diseases or complications
- *Assessment of neurological and vital functions para*llels Rx of acutely life-threatening conditions.
- Hx on risk factors for arteriosclerosis/cardiac dx
- Initial o/e: breathing and pulmonary function
- BP / HR; Targeted neurological examination
- Observation of early signs of dysphagia
- arterial oxygen saturation; clinical chemistry, coagulation ; haematology
- A) Standard stroke Unit
- B) Comprehensive stroke unit: acute management; secondary prevention; early mobilisation; rehabilitation; nursing care; speech therapist (Logopaedics);swallow Rxist

Assessment - Investigations

- **1. confirm clinical diagnosis if in doubt**
- **2. establish any treatable cause**
- **3. establish baseline**

  ? Improvement / deterioration
- **4. determine any risk factors which might be manipulated to prevent recurrence.**
- **5. predict likelihood of immediate complications**

. 6. Exclude other possibilities.

(age; potential for recovery; recurrence; clinical suspicion. R/o other diagnosis)

Assessment

- ***All pts: Brain Imaging: CT or MRI***
- Chest X-ray; ECG; Echocardiography;
- Cbc; platelet count, PT or INR, PTT; CRP/ESR electrolytes, glucose; LFT / Renal fn; FLP
- correlation between lacunar stroke and HB, None between Hb and non-lacunar; leucocytosis is associated with poor prognosis
- **Urinalysis -** Microalbuminuria predicts haemor transformation in CI– endothelial dysfunctn
- ***In selected patients: Duplex / Doppler ultrasound TCD,*** MRA or CTA
- Diffusion and perfusion MR or perfusion CT
- Pulse oximetry and arterial blood gas analysis
- Lumbar puncture; EEG ;Toxicology screen

Stroke bio-markers

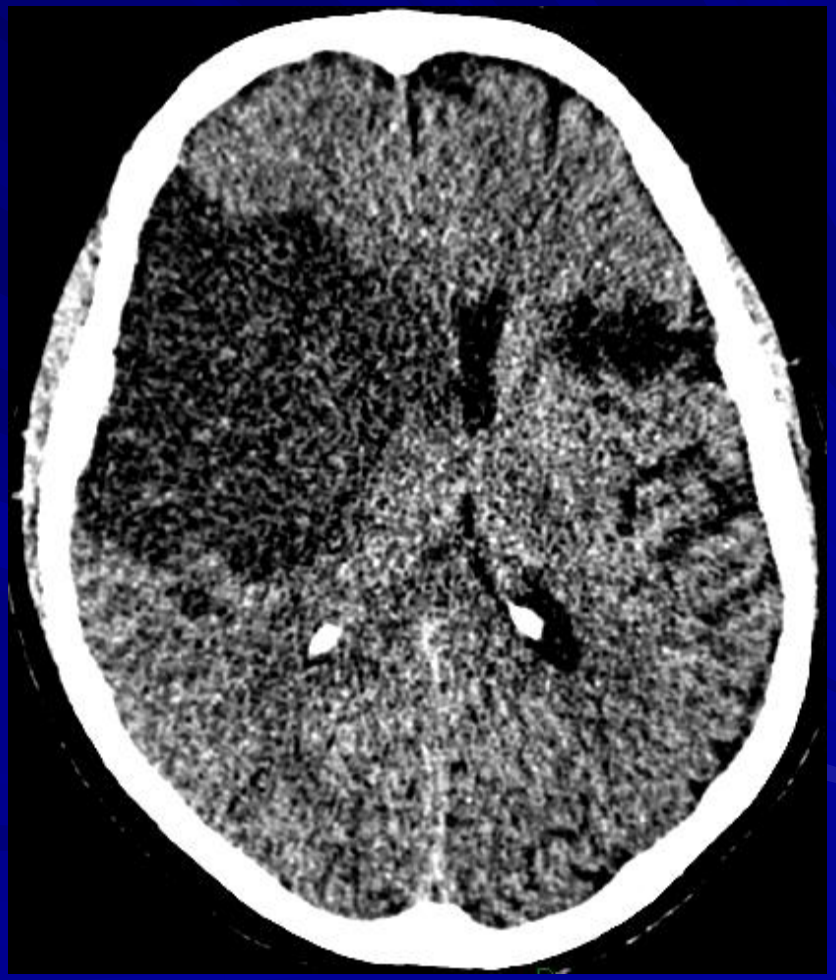
- Cf cardiac specific Troponin, CPK, LDH)
- Serum S 100 β - CI (Astroglial protein)
- Serum Glial fibrillary acidic protein (GFAP) - ICH

H –fatty acid binding protein (H-FABP)
Apo lipoprotein CI (Apo CI) - CI
Apo lipoprotein C III (Apo C III) - CI
Serum Amyloid A (SAA)
Antithrombin III (AT-III) fragment

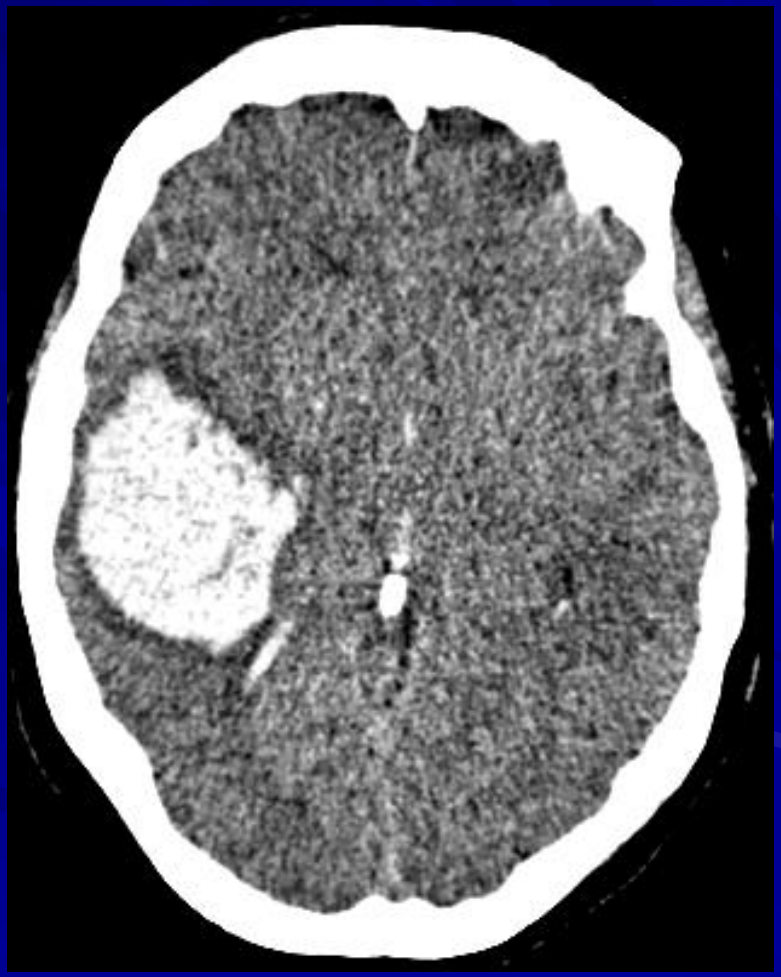
?? N A A

- Commonly measured markers include S100 calcium binding protein B or S100B, glial fibrillary acidic protein, brain natriuretic peptide, and matrix metalloproteinase.
- None of these substances are routinely measured by hospital laboratories in the time frame needed to make acute care decisions but are a focus of clinical research.

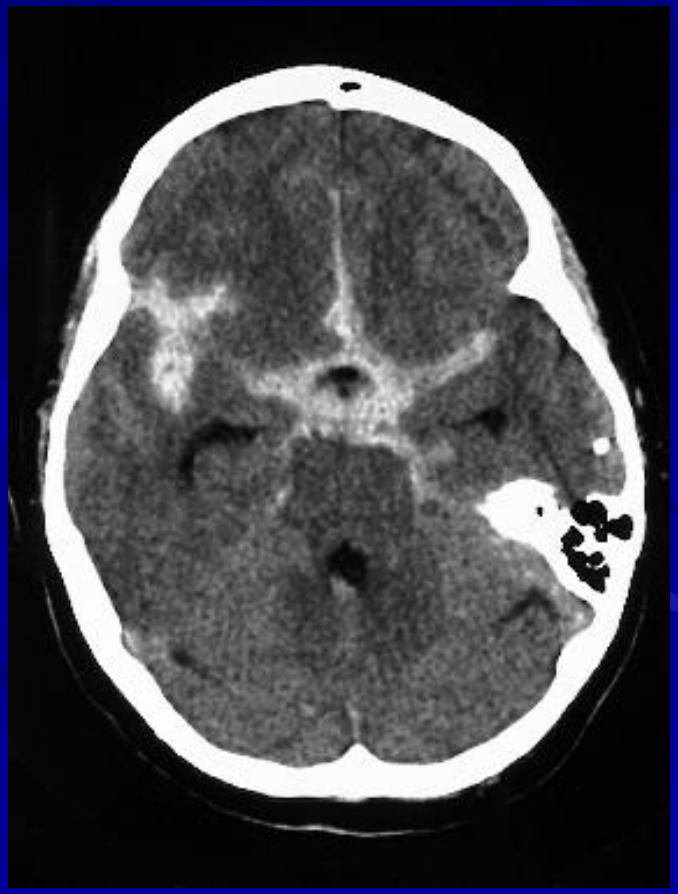
Ischaemic

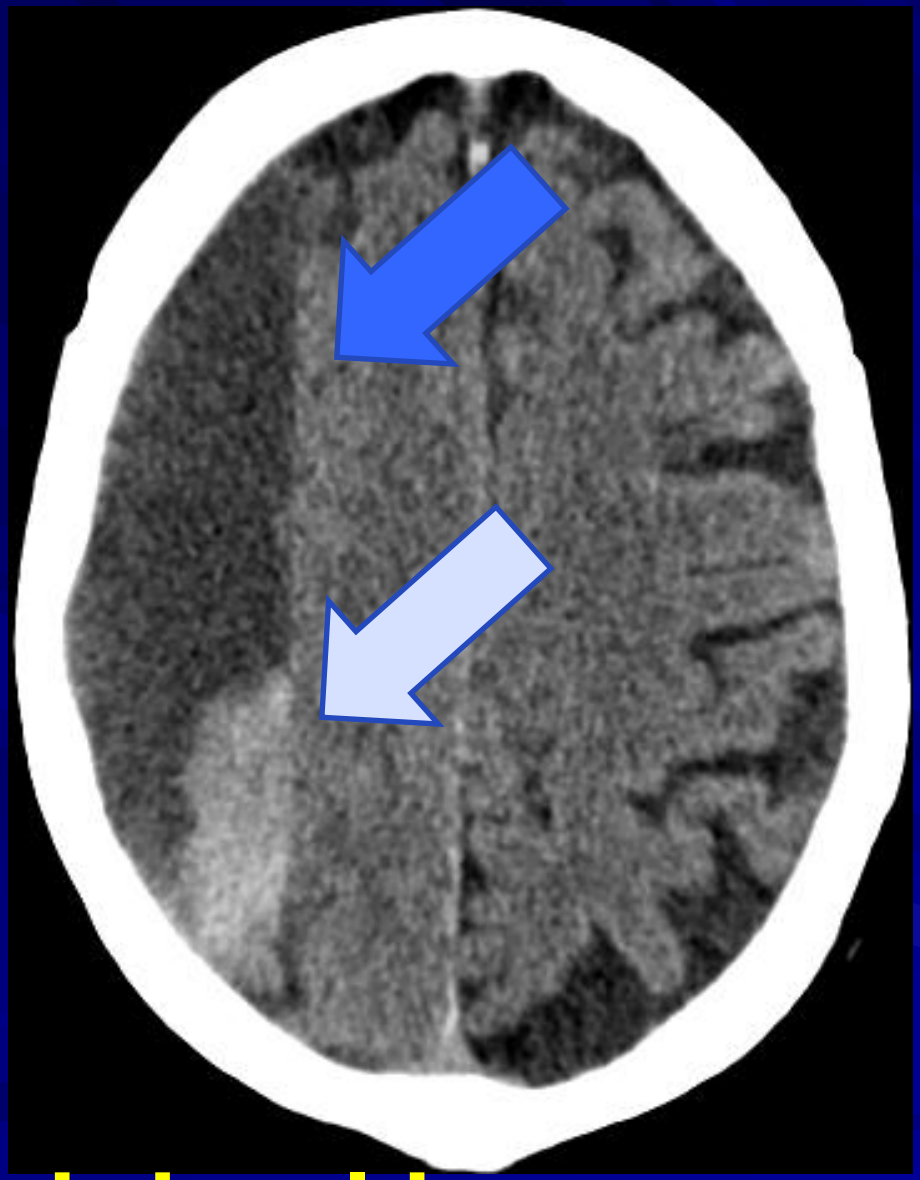


Haemorrhagic stroke

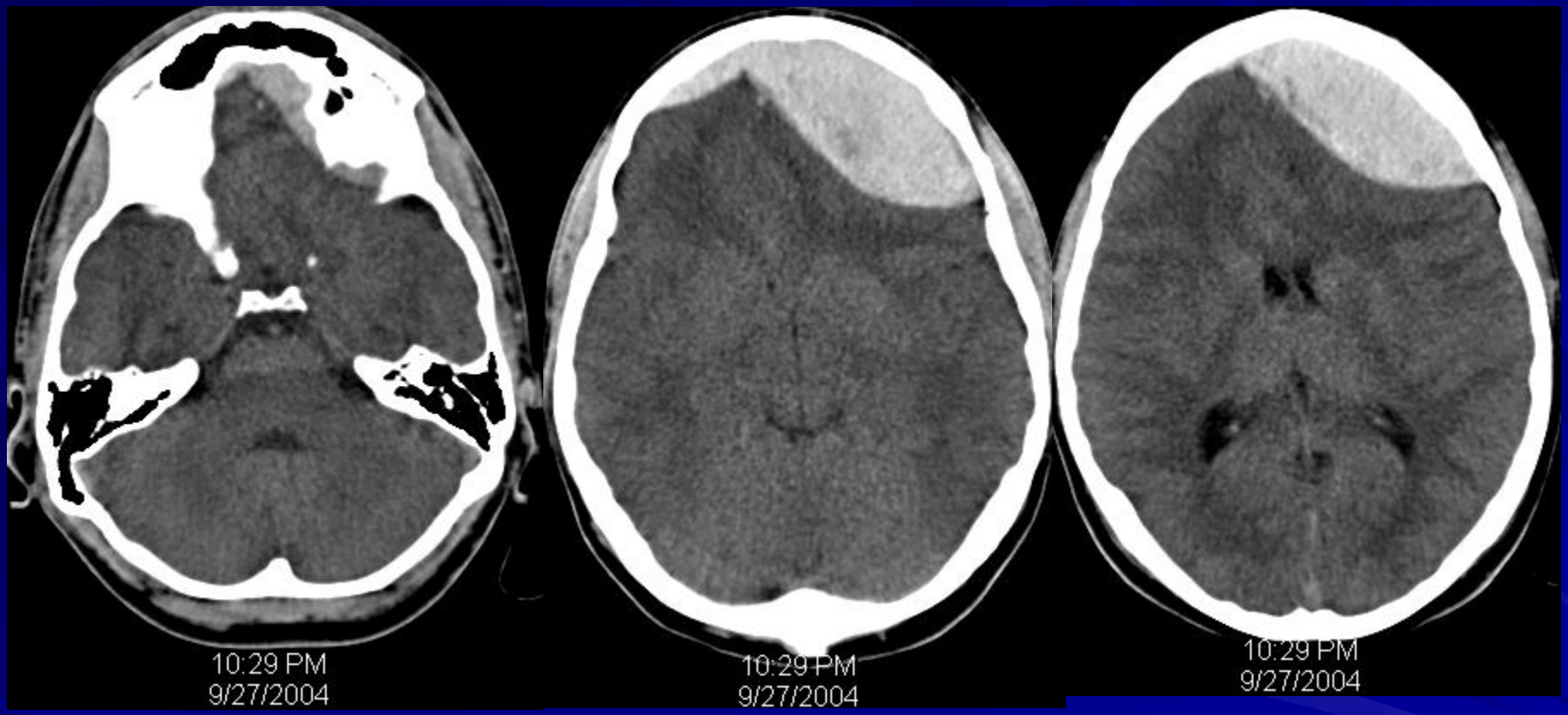


Subarachnoid Haemorrhage

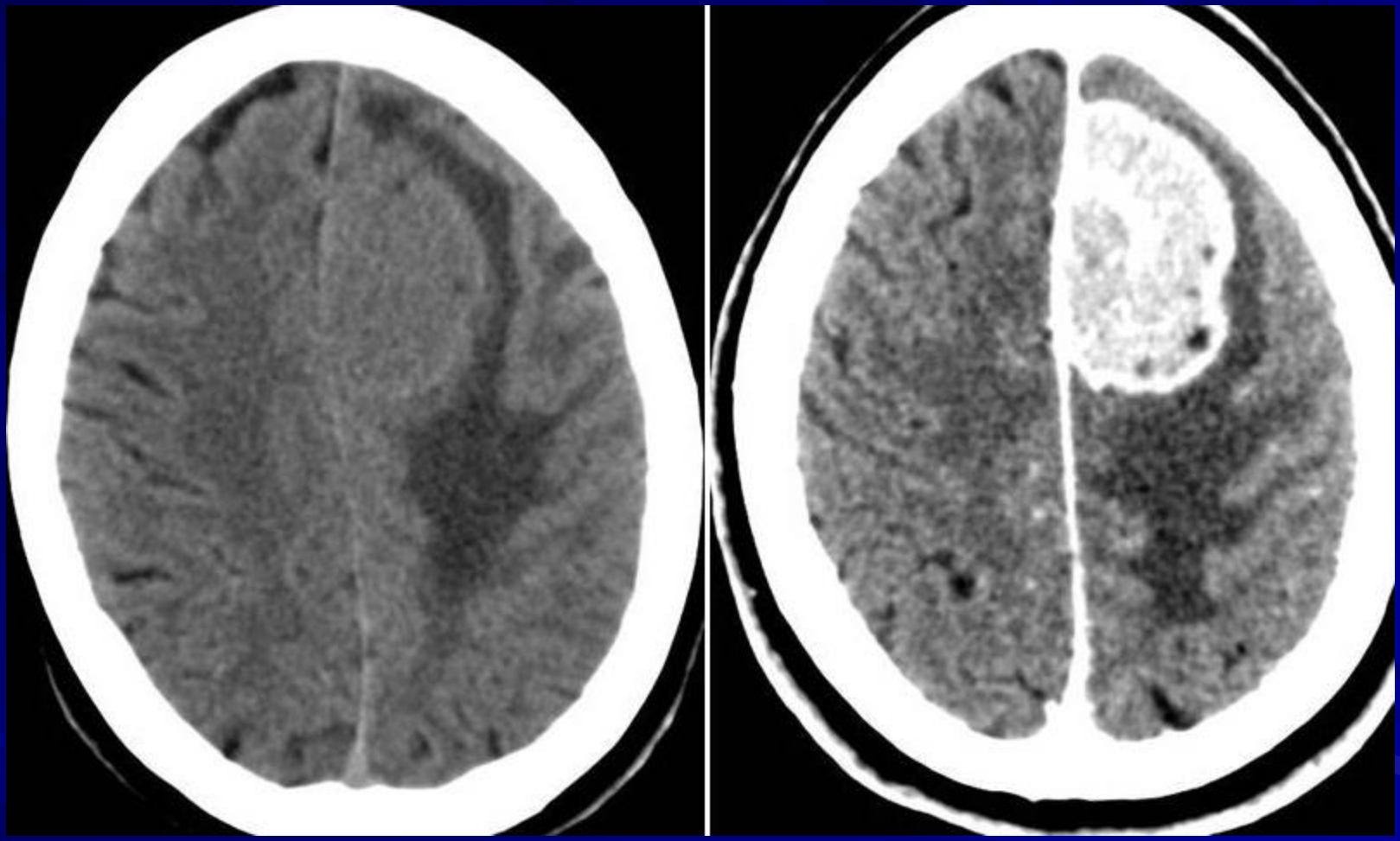




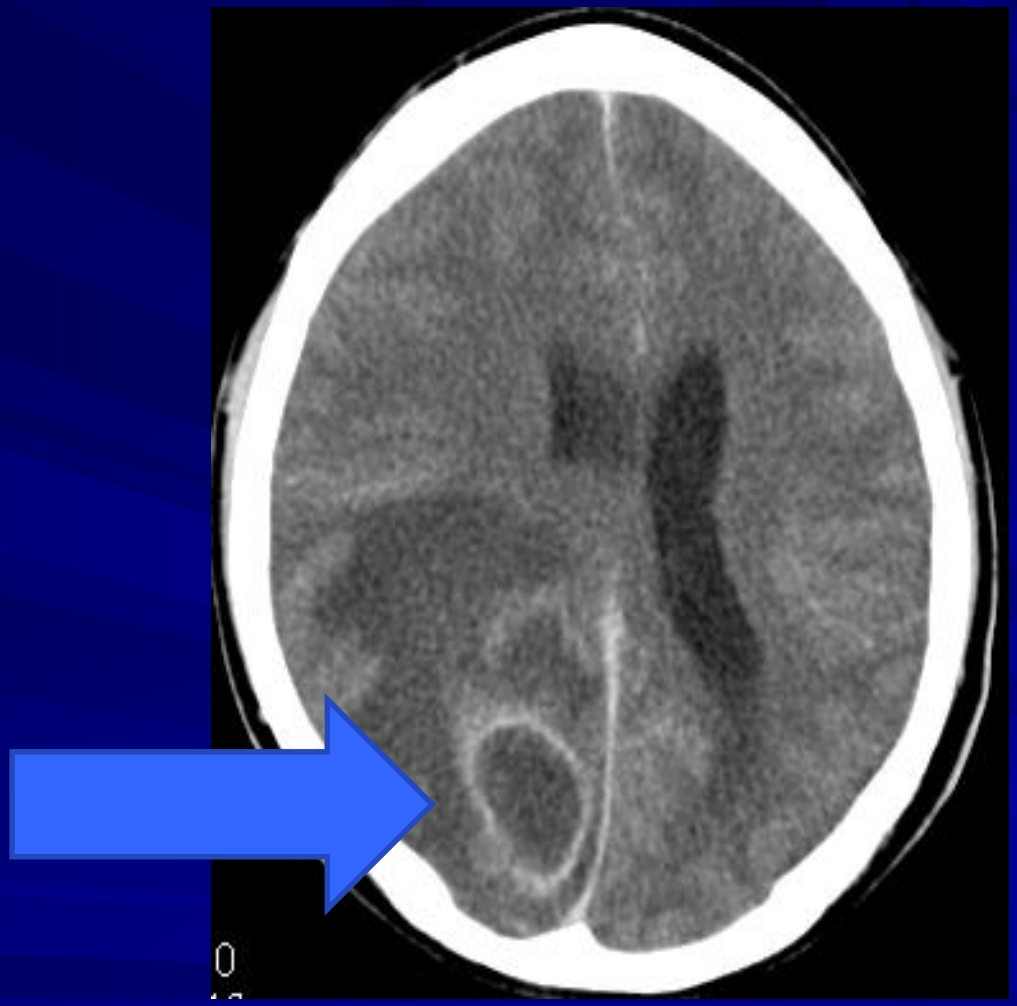
Subdural hematoma



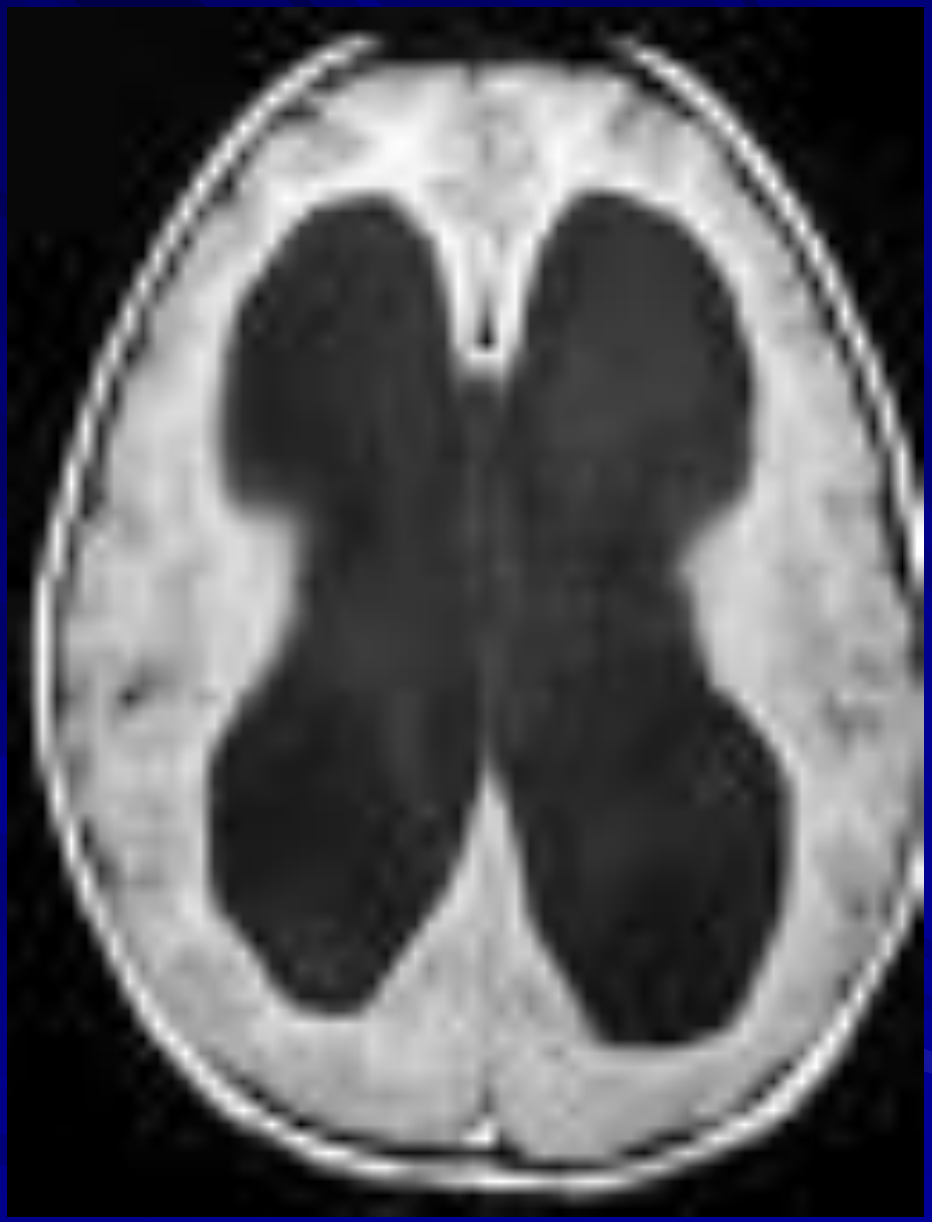
Trauma and extradural hematomas



Brain tumours



Brain abscess and infections



Hydrocephalus

- Treatment

AHA Classes/Levels of Evidence

- Classes indicate what we should generally do
  - I: Should do it
  - II: Consider it
    - IIa - per most experts; IIb - conflicted
  - III: Don't do it

- Levels indicate how sure we are about doing it
  - A: 2 randomized trials
  - B: 1 randomized trial or nonrandomized studies
  - C: Expert opinion or case series

Stroke RX Summary

5 EBM: Minimal / Essential /Advance Service of care

1) STROKE UNIT proved by EBM– 90%

- *2) Anti-platelet agents (proved by EBM – 80%)*
- *3) Thrombolysis (proved by EBM - 30%)*
- *Anti-coagulation (limited efficacy)*
- *Neuroprotection (ABDF / NeuroAID ? proved by EBM): citicoline ; cerebrolysin.*

- *Knives for Stroke treatment: (surgical treatment)*
- *4) Endovascular Thromebectomy (EBM 50%)*
- *Large Vessel proximal occlusion*
- *5) Decompressive craniectomy: EBM -50%*
- *Malignant MCA occlusion (unacceptable complications)*
- *Carotid endarterectomy (limited indications)*
- *EC/IC bypass surgery (it works, but does not help)*
- *Clamp/Coil/Gluing/ Flow Diverter: aneurysm (limited to SAH*

Summary: acute stroke

Use treatments supported by evidence:

- stroke unit care,
- aspirin,
- i.v. thrombolysis < 4.5 hrs
- BP lowering for acute ICH
- Intermittent pneumatic compression to prevent DVT

No evidence to support routine use of

- Aggressive BP lowering in acute ischaemic stroke
- iv heparin
- Intensive glucose control with insulin

Begin secondary prevention early

- All stroke: Blood pressure lowering
- Ischaemic stroke + antiplatelet + statin,
- POLYPILL (STATIN + Folate + Aspirin + antihypert)

Comprehensive stroke unit = dedicated area (beds) in acute hospital

Assessment & monitoring

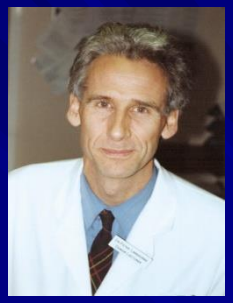
Acute management

Physiological control	Early mobilisation	Manage complications	Skilled nursing

Multidisciplinary rehabilitation

Discharge planning

Nurse



Doctor

Physiotherapy

Occupational Therapy

Speech therapy

Co-ordinated multidisciplinary care
(multidisciplinary team meetings 1x per week)

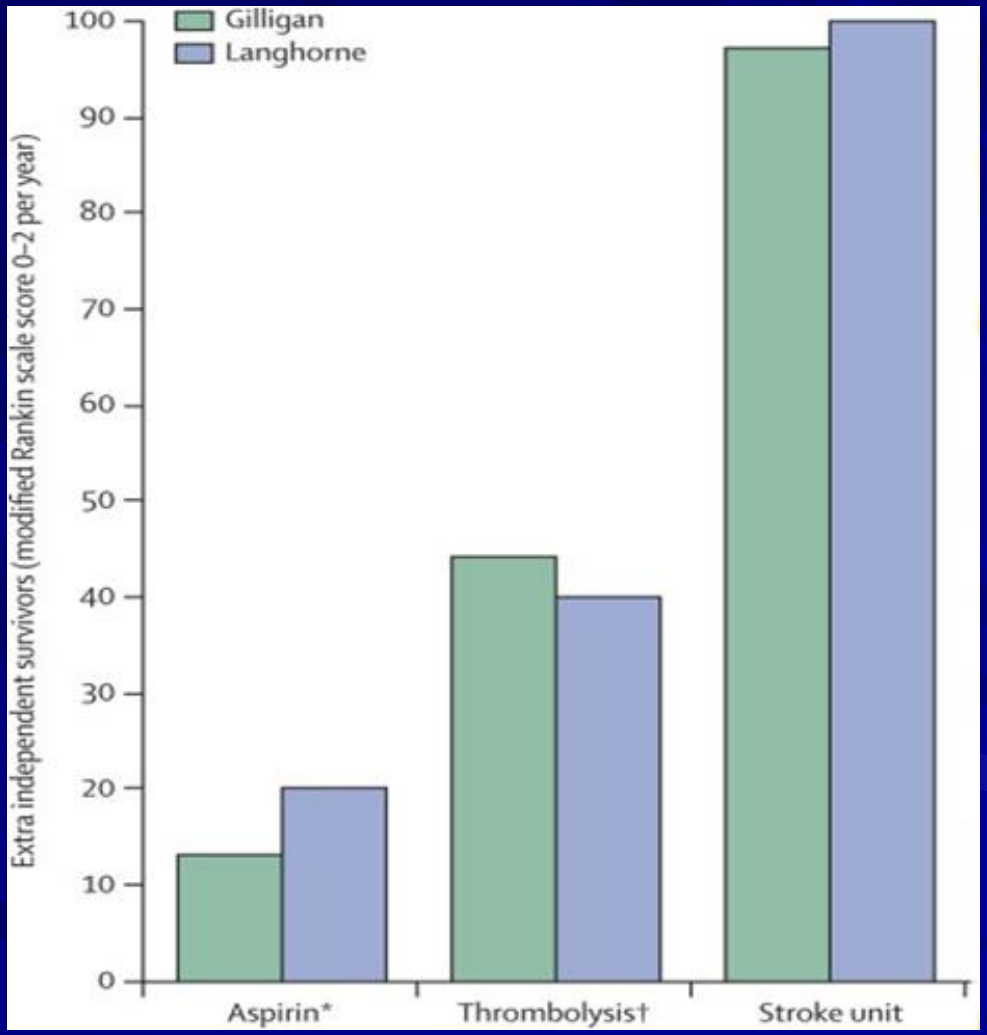
SUTC Cochrane Library (2007)

Beneficial effects of stroke unit care appear to be :

- Independent of patient age, sex, severity, stroke type
- Independent of parent specialty (Neurology, Geriatric Medicine, Internal Medicine)
- Demonstrated in all regions studied (including low- and middle-income countries)
- Not dependent on high technology (benefit even where no access to CT scanning)
- A result of a reduction of stroke complications

Good quality stroke unit care does not require ‘high technology’: stroke unit St Petersburg, Russia

Potential population effect of stroke interventions in a population of one million



Langhorne. Lancet Neurology 2012; 11: 341-8

Acute ischaemic stroke: i.v. thrombolysis

- rt-PA should only be administered
  - by personnel trained in its use,
  - with minimal ‘door-to-needle’ time
  - in a centre able to monitor patients adequately
- 3% risk of fatal intracranial haemorrhage
- ‘Time is brain’, i.v. rt-PA is most effective < 3hrs , but benefit up to 4.5 hours of onset
- IST-3 trial showed treatment benefits:
  - people > 80 years,
  - severe stroke
  - Less long-term disability and better quality of life

Other antithrombotic agents in stroke Rx and prevention

- Unfractionated heparin
- Low molecular weight heparin (not beneficial)
- Heparinoids
- Warfarins
- Ancrod – < circulating fibrinogen - beneficial

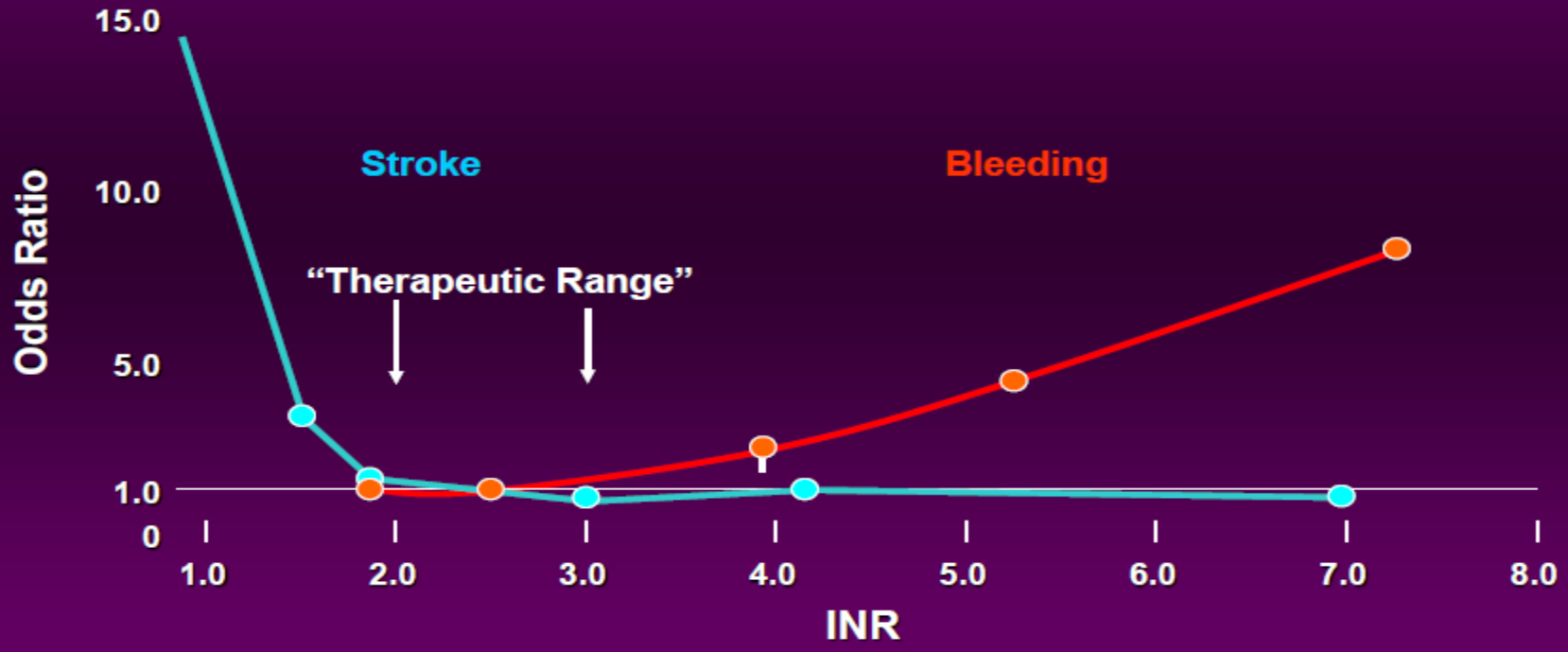
- Other reperfusion strategy: rtPA non responders
- Epifibatide (EP): glycoprotein IIb /IIIa receptor antagnst
- combine with rtPA : Argatroban = rtPA + EP

Combine IV (4.5hrs) / IA rtPA (6 hrs)

- Lacks scientific validation. ? risk of intracranial haemorr

Warfarin

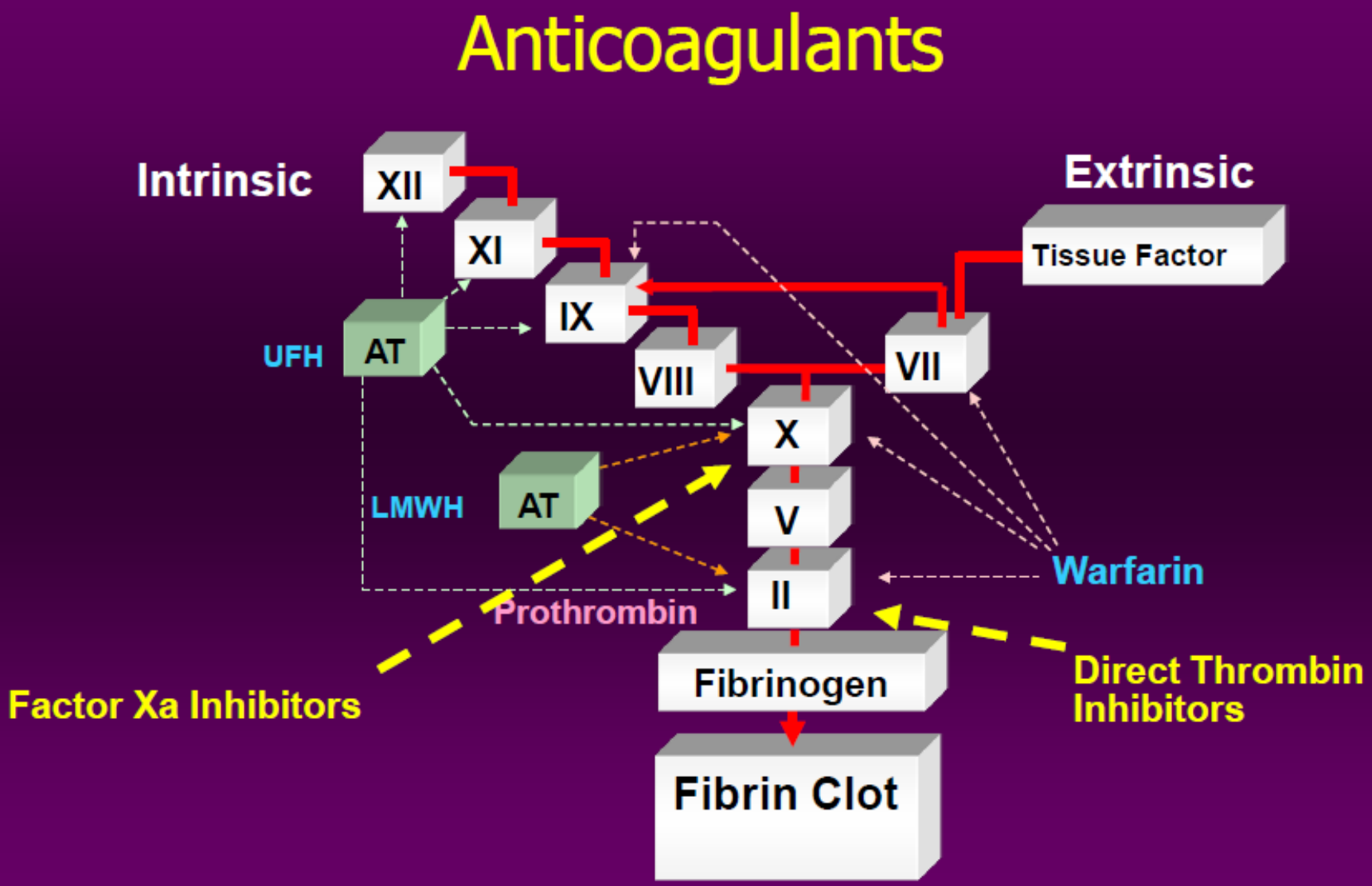
Therapeutic Range for Warfarin Balancing Safety and Efficacy



Hylek EM, et al. *Ann Intern Med.* 1994;120:897-902.
Hylek EM, et al. *N Engl J Med.* 2003;349:1019-1026.

We Need Better Arrows

Anti coagulants

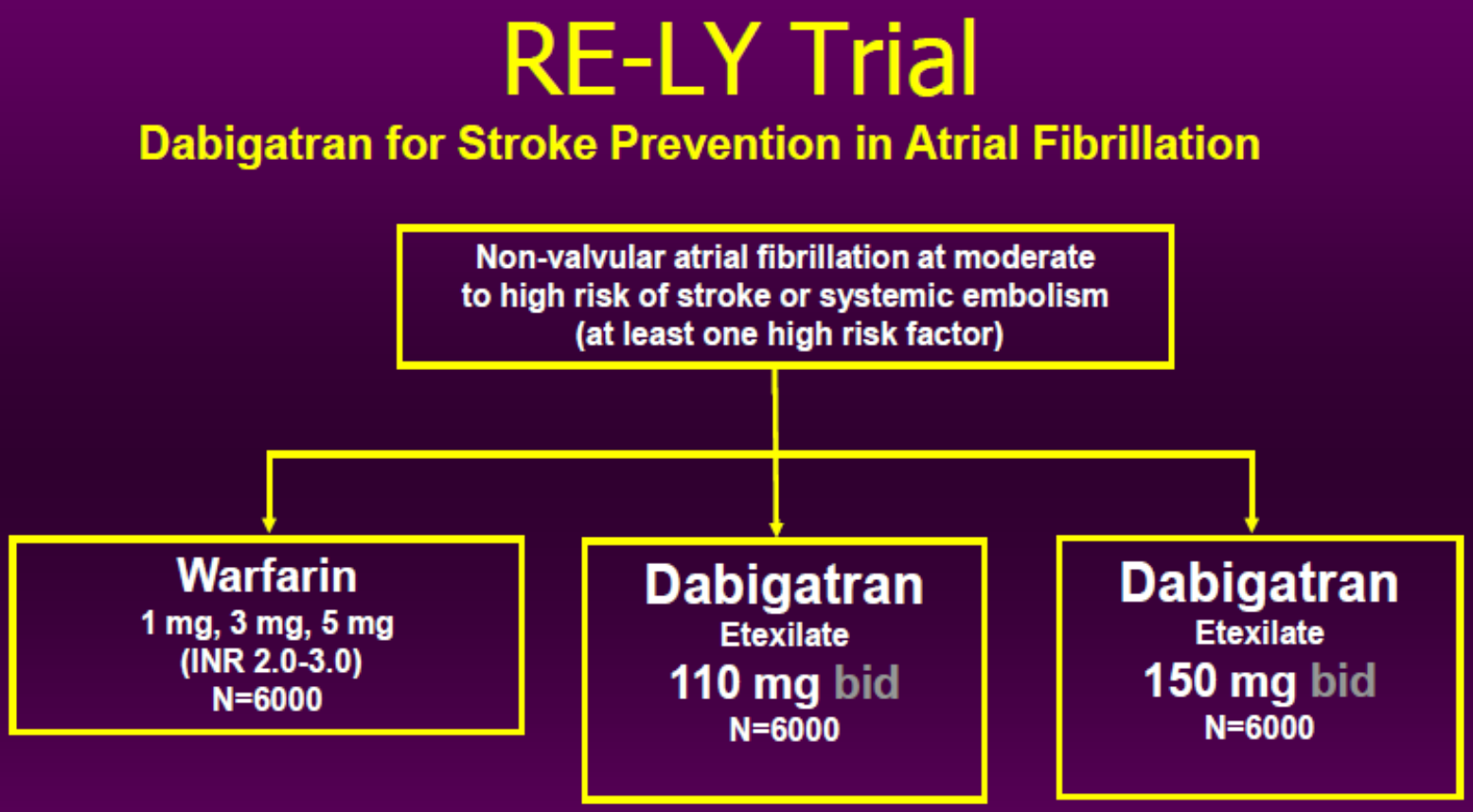


Dabigatran

DTI: Dabigatran

- Competitive direct thrombin inhibitor
- Prodrug dabigatran etexilate converted completely to active dabigatran
- Orally active, peaks in plasma 2 hours post-dose
- Half life: 14 – 17 hours
- Few and minor drug interactions
  - Proton pump inhibitors reduce absorption
  - P-glycoprotein inhibitors and inducers
- No routine blood monitoring
- No major dietary restrictions
- Eliminated predominantly by kidneys

RE-LY Trial


RE-LY Trial
Dabigatran for Stroke Prevention in Atrial Fibrillation
Non-valvular atrial fibrillation at moderate to high risk of stroke or systemic embolism (at least one high risk factor)
Warfarin
1 mg, 3 mg, 5 mg
(INR 2.0-3.0)
N=6000
Dabigatran
Etexilate
110 mg bid
N=6000
Dabigatran
Etexilate
150 mg bid
N=6000
Primary objective: Noninferiority to warfarin
Primary end point: Stroke + systemic embolism
Minimum 1 year of follow-up, maximum of 3 years and mean of 2 years of follow-up

Dabigatran

Dabigatran

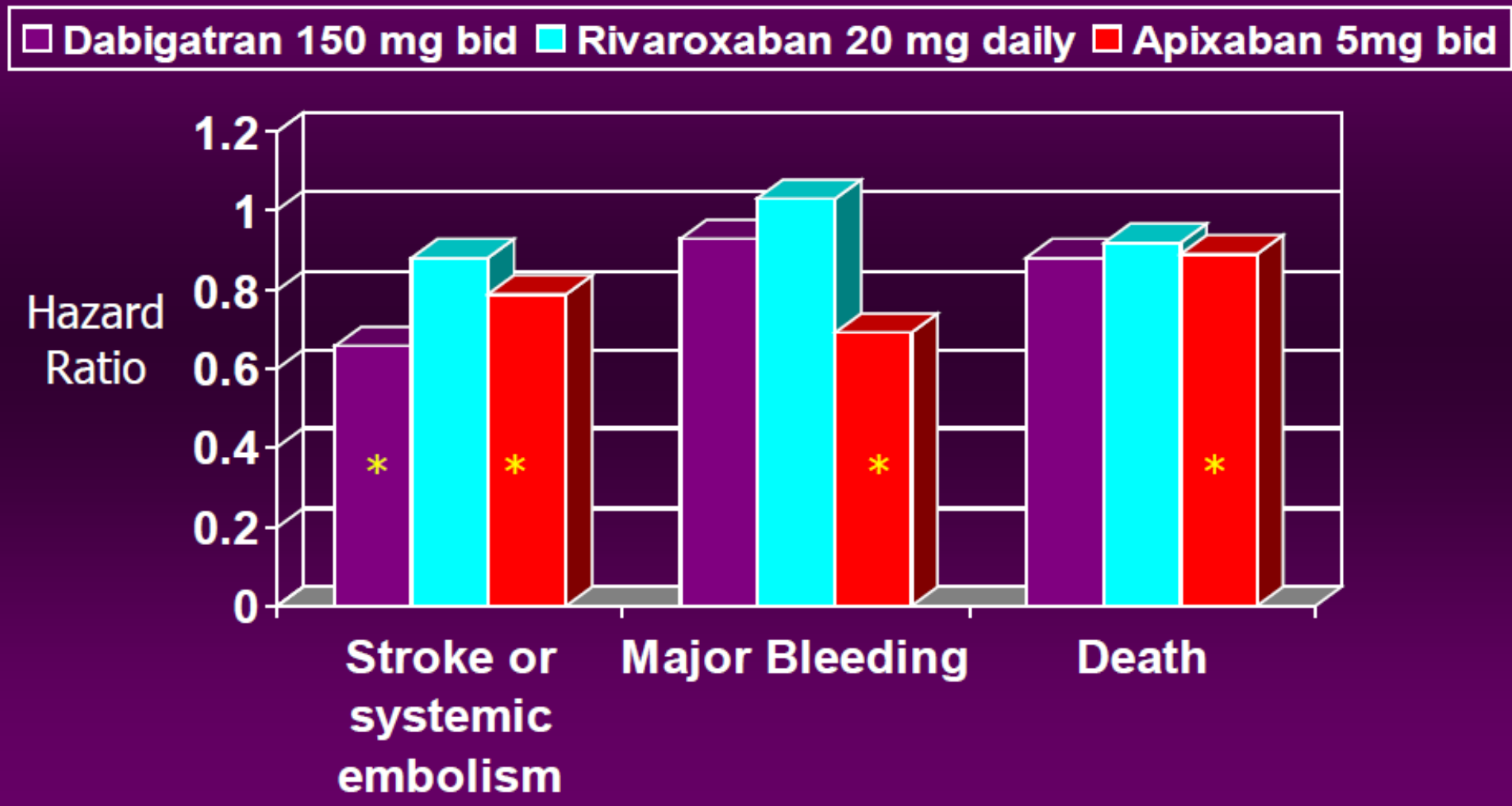
- Dabigatran 150 mg bid
  - reduces the risk of stroke and systemic embolism by ~25% compared to warfarin
  - risk of major bleeding is similar to warfarin
- Dabigatran 110 mg bid
  - about the same efficacy as warfarin
  - risk of major bleeding about 20% lower than warfarin

Factor Xa inhibitor: Rivaroxiban, Apixaban

Factor Xa inhibitors

- Reversible inhibitor of factor Xa
- Orally bioavailable
- Rapid onset
- Factor Xa may be a better target than thrombin
  - Has few effects beyond coagulation (compared with thrombin)
  - Thrombin inhibitors associated with rebound thrombin generation – no evidence with FXa inhibitors

New Anticoagulants Side-by-Side



Caution

- New weapons may be sharp and it takes time to learn to use them!

- No reliable way to monitor new drugs
- Less interaction with patients
- Thrombolysis?
- Indications beyond AFib?

? risk of intracranial haemorrhage

- Rx: Prothrombin Complex Concentrate - Yes
- Fresh Frozen Plasma : No
- Idarucizumab for NOAC - Dabigatran
- Andexanet / Aripazine for Factor X inhibitors.
- In addition, Rx: iv Vit K 10mg bc half life of all above = 2-3 hrs.
- Platelet infusion: harmful.

- Recommence Antiplatelets / Anticoagulants:
- 1 3 6 12 (+/- 2) days Principles
- depending on severity

ACCP 2012: Antithrombotic & Thrombolytic Therapy for Ischemic Stroke (Lansberg et al)-2

5. Antiplatelet Therapy for Secondary Prevention of Noncardioembolic Stroke or TIA:

A. Long-term treatment with *1 of the following over no antiplatelet therapy* (Grade 1A), *oral anticoagulants* (Grade 1B), *combination clopidogrel + aspirin* (Grade 1B) *or trifusal* (Grade 2B):

(1) Aspirin (75-100 mg/day)

(2) Clopidogrel (75mg/day)

(3) Aspirin/extended-release dipyridamole (25mg/200mg bid) or

(4) Cilostazol (100mg bid) [NEW RECOMMENDATION]

6. Suggest clopidogrel or aspirin/extended-release dipyridamole over aspirin (Grade 2B) or cilostazol (Grade 2C)

Source: Chest 2012; 141 (Executive Summary): (2) Suppl 6S-47S

How to give aspirin immediately.
Ask: can the patient swallow safely?

Swallow safely? YES

YES !

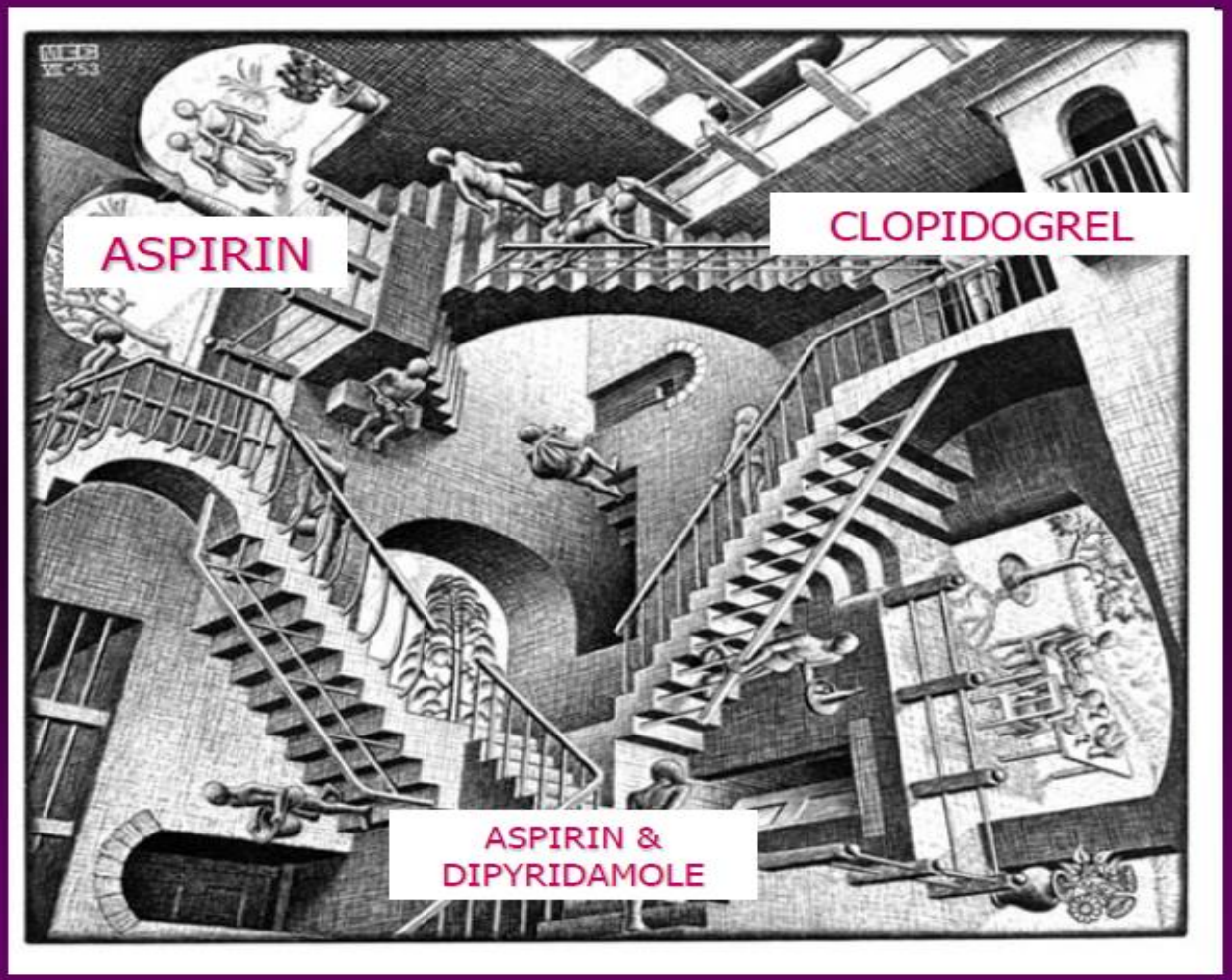
aspirin

aspirin

aspirin per rectum

Sandercock et al CDSR 2008

Antiplatelets



Anti platelets

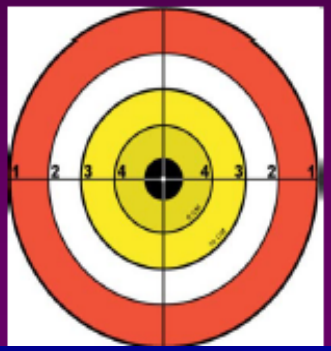
Antiplatelet Therapy for Secondary Stroke Prevention 2012

- Aspirin is still good
- Clopidogrel possible modest benefit over aspirin
- Clopidogrel conquered ASA/ER-Dipyridamole
  - Similar efficacy, but
  - Clopidogrel once-a-day and better tolerated
- ASA+clopidogrel is not recommended
  - Experimental for TIA
- Aspirin failures still uncertain
- Cilostazol, prasugrel, ticagrelor, thrombin receptor antagonists…?

Stroke Prevention: Mechanism-Specific Considerations

What is the cause of the initial cerebrovascular event?

Large vessel athero	Cardioembolism	Small vessel dz
Carotid endarterectomy Antiplatelet agent	Anticoagulation	Antiplatelet agent



A Fib

Look Harder for Occult AFib

- At least 20% of cryptogenic stroke pts have occult AF
  - Especially cortical or multiple strokes
  - Older patients, especially women
- Up to 90% AF episodes are asymptomatic
- AF yield increased with longer monitoring duration
  - >60% AF beyond 30d after initiating monitoring
  - Unknown optimal duration (CRYSTAL AF study)
- AF >6 hours: Doubles 1 yr stroke risk
- Short AF episodes likely predict longer episodes and increased stroke risk
- Treatment options for AFib expanding every day

Other treatment options

- Newer Anti-thrombotics
- Statins
- Neuroprotectants (Mg So4) – vasodilator)
- Stem cell Transplantation
- +IV cooling with iced saline via IVC
- USS clot lysis
- Endovascular Rx.
- Mechanical recanalisation

Neuroprotective agents (Neuroprotectants)

- Neurorestorative / Neuroregenerative / Neurogenesis
- Neuroprotective / Neuroproliferative /Neurotrophicity /REWIRING / NEUROPLASTICITY

Protect N from adverse milleu created by the biochemical changes triggered by ischaemia:

attenuate neuronal injury

- **free radical scavengers – Vit C ; E ; 21-aminosteroid ; antioxidants tirilazad**
- **inhibitors of excitatory A.A. (NMDA receptor blockers – MK – 801)** Glutamate antagonists
- **Caspace (apoptosis) inhibitors**
- **Lysosomal protease (necrosis) inhibitors**
- **Ca2+ antagonist – nimodipine**
- **Barbiturates; hypothermia; steroids (<met. Demand)**
- **Citicoline**

Neuroprotectants

- Cerebrolysin: a peptide with neurotrophic effect
- BDNF: (Brain Derived Neurotrophic Factor)
- one of the “Neurotrophin” family of growth factors that regulates neuronal survival and protects from glutamate induced damage.
- encourages proliferatn,differentiatn of new Neurons
- BDNF pathways are ***involved in cell survival, neuron – protection, brain plasticity and neurogenesis.***
- Neuronal cells require BDNF to regenerate

Naftidrofuryl : > efficiency of substrate use; < lactate level; >supply of ATP; lengthens the Rxic window period

NO (Nitric oxide) synthase inhibitors

neuronal / inducible → cytotoxic;

endothelial → vasodilate → protective

Repinotan - 5 HT1a Agonist - CI; Mg failed; Citicoline promising

BDNF / NeuroAID

- NeuroAiD: stimulates production of BDNF
- Promotes neurogenesis
- >neurites growth and synaptogenesis (neuronal connections)
- Provides a ***better post stroke recovery of Neurological function*** in pts with severe stroke.

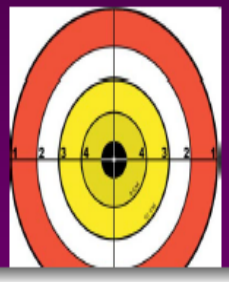
- FLUXETIN: enhances Motor Recovery

Mechanism of stroke

Stroke Prevention: Mechanism-Specific Considerations

What is the cause of the initial cerebrovascular event?

Large vessel athero	Cardioembolism	Small vessel dz
Carotid endarterectomy Antiplatelet agent	Anticoagulation	Antiplatelet agent



Surgical Rx:neurovascular intervn

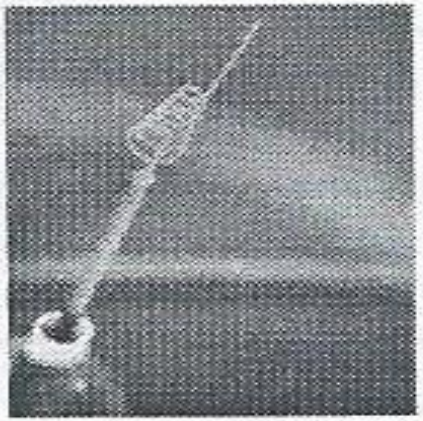
- Occlusive large Vessel dx:Artero-venous malformation
- Intra-Luminal thrombosis; Fibro muscular dysplasia
- Collateral channels of blood flow; Intra cranial Stenosis.; Artherosclerosis
- **Embolectomy:** Mechanical Embolus Removal in Cerebral Ischaemia (MERCI)
- Endovascular Cockscrew embolectomy
- **Vascular reconstruction:**BalloonAngioplasty/stenting Cf : Aorto-Femoral stenting of coronary
- **Thromboendarterectomy**
- Extracranial – Intracranial By Pass Surgery- ineffective
- **Hemicraniectomy (Malignant MCA occlussin)**
- **Devices**

Mechanical Embolectomy

Mechanical Embolectomy at <8 Hrs

AHA Class IIb, Level B Evidence

- Nonrandomized evidence
  - MERCI received FDA 510(K) clearance in 2004
  - Penumbra received FDA 510(K) clearance in 2007
  - Solitaire received FDA 510(k) clearance in 3/2012



Bose, 2008; Smith, 2005

Carotid end-arterectomy

- >70% CS : 18% risk of stroke
- **(12-17% (1st yr); 7% thereafter)**
- **(>C.O)–if >70% CS** ? Age
- < risk of stroke by 48%; lower benefit in females
- (more ICA affectation)
- < 70% CS : 7-8% risk

- **Balloon angioplasty + stenting—randomised trial in progress.**

CAS

Carotid Stenting

Choose Your Arrow wisely

- The more weapons you have against stroke, the better
- Choose the right arrow for your patient

- CEA still the better choice for most
- CAS worth considering
  - favorable anatomy
  - younger (<70 y) pts
  - high risk of MI
  - high risk of major CN injury

Neurosurgical treatment - ICH

- **CH: evacuate hematoma > 12 hrs.**

  60% mortality at 30 days

  (mortality not different from conservative)

  Minimally Invasive Surgery (MIS) – Endoscopic Surgery
- **CT guided stereotactic surg. + hemolysis**
- **conservative Mx if volume < 100 mls**

Stroke Rx .summary

5 EBM

1) STROKE UNIT proved by EBM– 90%

- *2) Anti-platelet agents (proved by EBM – 80%)*
- *3) Thrombolysis (proved by EBM - 30%)*
- *Anti-coagulation (limited efficacy)*
- *Neuroprotection (ABDF / NeuroAID ? proved by EBM): citicoline ; cerebrolysin.*

- *Knives for Stroke treatment: (surgical treatment)*
- *4) Endovascular Thromebectomy (EBM 50%)*
- *Large Vessel proximal occlusion*
- *5) Decompressive craniotomy: EBM -50%*
- *Malignant MCA occlusion (unacceptable complications)*
- *Carotid endarterectomy (limited indications)*
- *EC/IC bypass surgery (it works, but does not help)*
- *Clamp/Coil/Gluing/ Flow Diverter: aneurysm (limited to SAH*

Stroke treatment

- *Alternative and Complementary Treatment in China*
- *Herbal Medicine*
- *Ginkgo*
- *Ginseng*
- *Ligusticum*
- *Acupuncture*

Acupuncture Moxabustion / Cupping

- **Acupuncture** *is the insertion of very fine sterile needles into specific "acupuncture" points*
- *It has measurable effects on the autonomic nervous system: vascular, endocrine, neurotransmitters.*
- *Concepts include Yin and Yang theory, Qi and Blood, and Channels and Network vessels*
- *Acupuncture helps to rebalance proper flow of Qi and maintain homeostasis*
- **Moxabustion**: *process whereby moxa - a dried herb, usually the species mugwort - is burned, either directly on the skin, or just above the skin, over specific acupuncture points relative to a condition.*
- *When lit, moxa burns slowly and provides a penetrating heat that can enter the channels to influence qi and blood flow.*
- **Cupping**: *an ancient Chinese method of causing local congestion. A partial vacuum is created in cups placed on the skin either by means of heat or suction. This draws up the underlying tissues. When the cup is left in place on the skin for a few minutes, blood stasis is formed and localized healing takes place*

- 3) Treatment of Underlying dx process

Blood pressure

Hypertension and Stroke

The most important and treatable risk factor for all types of stroke : 60-92% of ICH (60 -90)

33-62% of CI (30-60)

19-30% of SAH (20-30)

undiagnosed or untreated in 60%

10%: asymptomatic; life-long Rx; adequately controlled

- > diastolic BP 10 mm Hg: > risk of first stroke > by > 50%
- < diastolic BP 6 mm Hg: < stroke risk by > 33%
- Antihypertensive therapy: < stroke risk by about 38%

Hypertension

- Variability of the BP is the key factor.
- Systolic and diastolic troughs. (>>> systolic)
- CcB: reduces variability the most
- BB: worsens variability esp non selective such as propranolol.
- ACEI: moderate effect

Antihypertensives

Weight of available evidence – Rx^0 x 10 D

Rx if MAP > 145 (SBP > 220; DBP>120)

"absence of evidence" is not "evidence of absence")

- **Aim: MAP = 130; DBP = 105; (185/105)**
- **Why high BP is required ???:**
- **loss of C. autoregulation; (> O2 extraction fraction)**
- **CPP = MAP – ICP**
- **keep collateral channels open**

Blood Pressure

Optimal BP Management for Nonreperfusion Candidates?

- Potential Strategies
  - Keep it the same?
    - Maintain home BP meds (COSSACS)
  - Lower it?
    - Start BP med (ACCESS, SCAST)
  - Raise it?
- Current AHA Guideline
  - Permissive hypertension (up to 220/120) (Class I, Level of Evidence C)

Indications for Rx of HBP

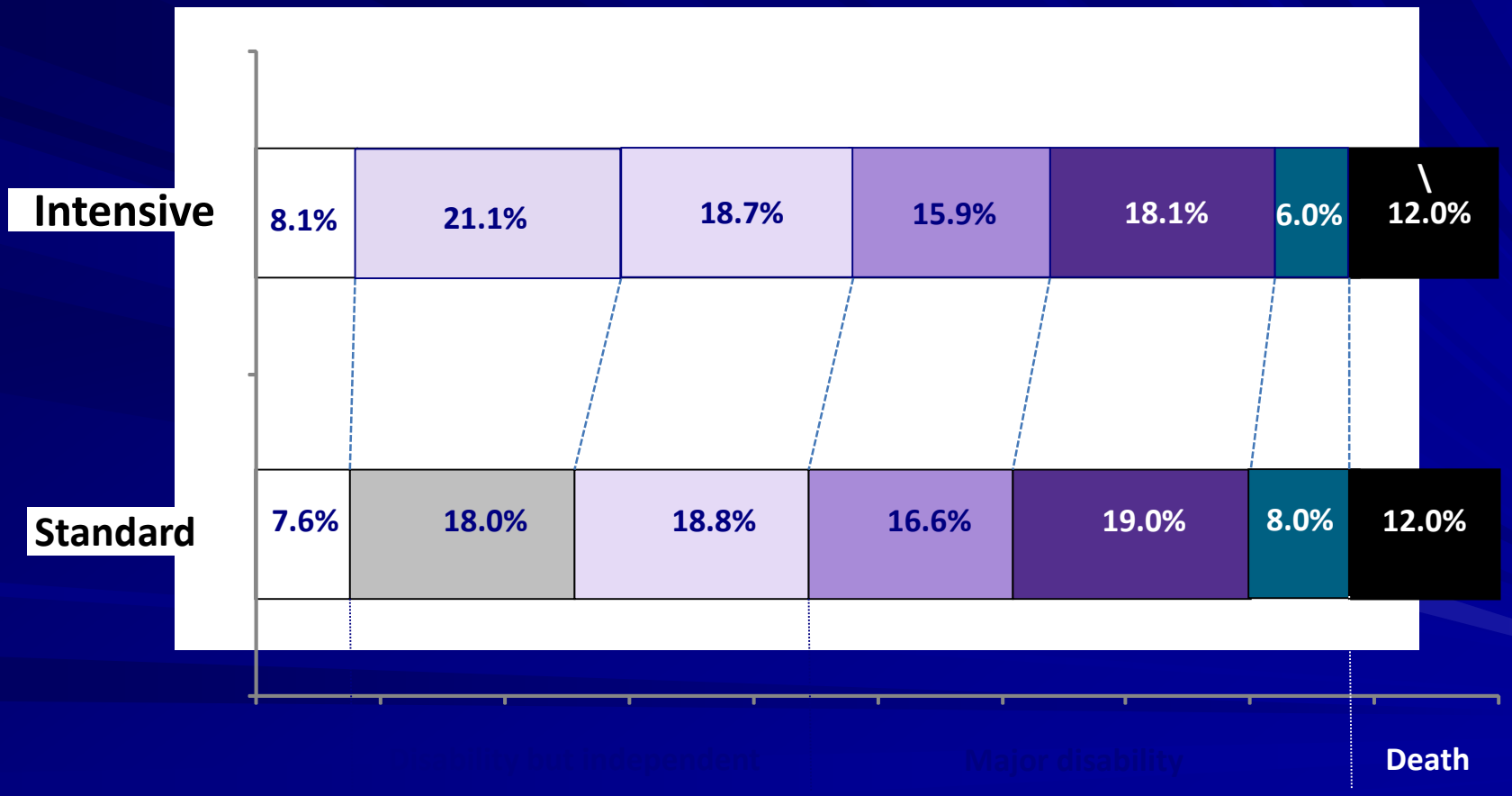
Extra-cerebral complications:

- **dissecting aortic aneurysm**
- **Ischemic heart dx / Myocardial Ischemia.**
- **Acute pulm. Oedema.**
- **Rapid decline in renal function.**

Antihypertensives of choice:

- **CcB: nifedipine; isradipine; nimodipine oral nimodipine : cerebrospp + cytoprotective:**
- ACEI
- BB worsens variability, next ACEI. Best is CaCB

INTERACT-2 trial. Rapid, intensive blood pressure lowering < 6hrs in ICH to target of <140 mmHg systolic vs target < 180 mm



Odds ratio 0.87 (95%CI 0.77 to 1.00); P=0.04

- Prevention and Rx of Complications

TABLE 2: ACUTE AND SUB-ACUTE COMPLICATIONS OF STROKE

	Neurological	Systemic
ACUTE (<7 days)	1.Cerebral oedema 2.Increased ICP 3.Hydrocephalous 4.Haemorrhagic transformation 5.Seizures 6.Transtentorial herniation	1.Hypoxia 2.Hypertension 3.Hyperglycemia 4.Aspiration 5.Cardiac arrythmias 6.Inappropriate ADH secretion
SUB-ACUTE (>7 DAYS)	1.Seizures 2. Depression	1.D.V.T & Pulmonary embolism 2.Bronchopneumonia 3.Urinary tract infections 4.Septicaemia 5.Decubitus ulcers 6.Joint stiffness 7. Post stroke weight loss

Elevated ICP / Cereb Oedema

Basic management

- Head elevation(Day 1: 0°; day 2: 45°; day 3: 90°; sit out of bed > 4 hrs from day 4)
- Pain relief and sedation; THAM-buffer
- Osmotic agents(glycerol,mannitol, hypertonic S)
- Ventilatory support: Controlled respiration using a ventilator Hyperventilation^{0}- ineffective
- Sedation: Barbiturates – ? high dose; coma
- Achieve normothermia. Hypothermia may < mo
- **Phosphodiesterase inhibitors: pentoxyfilline**
- paralysis

Cerebral Oedema - Rx

- **Diuretics – acetazolamide; frusemide**
- **Hypertonic / hyperosmolar agents: glucose; sucrose; urea; mannitol; sorbitol; glycerol; dextran; 2.34%; 23.4% Saline given as bolus over 30 mins**

  creates osmotic gradient (B = osmometer)
- **Glycerol improve mort; functional recovery^{o} subclinical hemolysis**
- **rebound > ICP^{o}**
- **Mannitol 0.25 – 0.5g/kg over 20 mins**
- **rpt 6 hrly max = 2g/kg**
- **Hypoosmolar agents^{0} 5% D in H_2O – inefficient - aggravates cerebral oedema**

Steroids

- **Counteract stress factor;**
- **<Cerebral Oedema;**
- **< 1CP;**
- **Strengthens the BBB;**
- **< cytokines: IL-1; TNF; prostaglandin**

dexamethasone^0 cytotoxic oedema^0
(vasogenic oedema+)
early death – 6 days vs 15 days

Steroids have been shown to rapidly enhance intraischemic CBF& reduce cerebral infarct size- upregulate eNOsynthase.

Hyperglycaemia

Hyperglycemia

- Hyperglycemia (>140 mg/dL) during the first 24 hours after stroke is associated with poor outcomes
- Higher serum glucose concentrations (possibly 140 to 185 mg/dL) probably should trigger administration of insulin (Class IIa, Level of Evidence C)

Hyperglycaemia & Ischemia

- > Glycolysis (lactate production); rather than oxidative phosphorylation.
- Metabolic acidosis – > > H+ / K+ - denatures enzymatic / structural proteins:- interferes with function (+ non-enzymatic glycation/glycosylation of proteins)
- AGEP (Advanced Glycation end product)– Toxic to endothelia + production of free radicals
- Haemorrhagic transformation incidence higher glucose – toxic to vasculature
- **hyperglycaemia – Rx as appropriate: insulin**
- BS>180mg%/10mmol; ensure HbA1c < 7%

Rx of Complications

- **DVT: 75% of hemiplegic leg**
- **Pul. Embolism: 9-13% of all stroke deaths;**
- **25% of late death (>1wk).**
- < Risk: good hydratn / v.early mobilizatn / IPC
- Frequent changes of the patient's position in bed q2-4hrs;pulmonary physical Rx(airway care
- **5000 units sc. Heparin 8/12 hrs (>48hrs)** Low-dose LMWH; Dabigatran / Rivaroxiban
- ***Aspiration pneumonitis: 51% dysphagia:*** ***w***ithhold oral feeding until intact swallowing, (**swallow test: 50mls of water):** NGT > 48hrs percutaneous enteral gastrostomy(PEG)>2wks

Rx of Complications

- ***Decubitus Pressure ulcer:*** support surfaces: Pp mattress frequent repositioning, optimizing nutritional status, and moisturizing sacral skin
- *Seizures: Rx PRN ; Agitation: Causal Rx must precede sedation or antipsychotic Rx*
- *Falls:* cognitive impairment, depression, poly pharmacy; sensory impairment; non-dominant
- improve bone strength and < fracture rates:
- Exercise, calcium supplements bisphosphonates. Rx Atrial Fibrillation PRN;
- Fever Rx (T>37.5):vasodilation : worsens C.O.
- catheterisation^{0}; Paul's tubing; condom
- Rehabilitation: PhysioRx: Early : Active /Passive

Mathew score/NIHSS – monitor progression Level of C; degree of weakness

Prognosis

Overall stroke–related case fatality = 20%35%

- **depends on**
- **1) location**

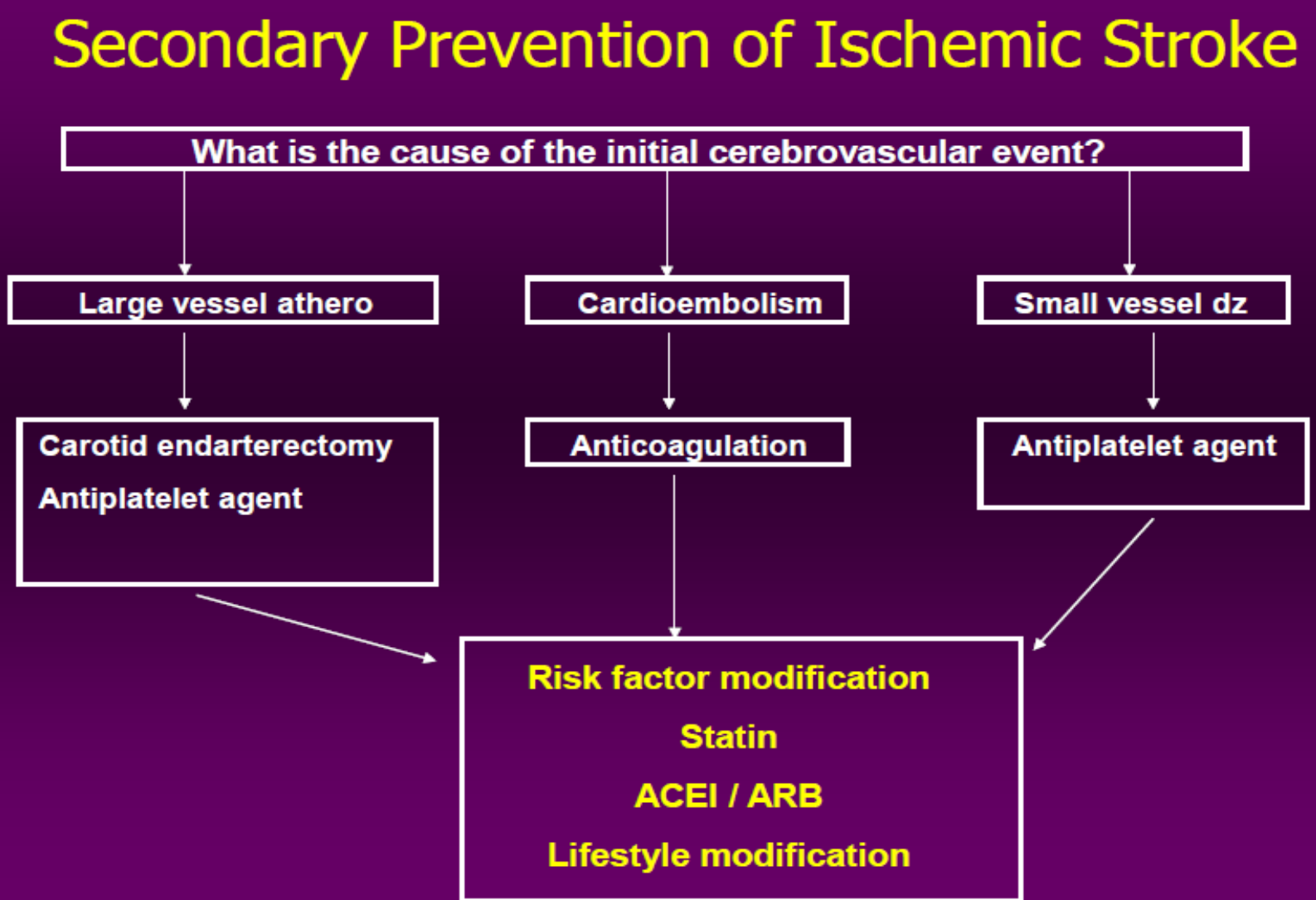
  post. fossae; + / - ventr. extention;

  deep location : poor prognosis
- **2. Size**
- **3. level of consciousness on admission**
- **4. later progression of neurologic signs and development of > ICP**

5. type of stroke ; clinical stroke syndrome.

(Died and Buried – ***The BAD)***

Stroke prevention



Primary prevention

- **Removal of risk factors**
- **HTN-control reduces risk by 40%**
- **Discourage smoking /**
- **Discourage alcohol (> Triglyceride / HDL)**
- **D.M control**
- **maintenance of normal weight**
- **regular exercise**
- **Life style changes/modification: dietary, cholesterol /hyperhomocystenaemia control**

AHA/ASA 2011 Recommendations for Lifestyle and Risk Factor Management in TIA or Ischemic Stroke

Factor	Recommendation	LOE
HTN	Initiate Rx beyond 24 hours; individualize Rx, consider ACEI/diuretics; BP reduction of 10/5 mm Hg	**Class I, LOE A** **Class IIa, LOE B**
Diabetes	Use existent guidelines for glycemic & BP targets; aim for hemoglobin A1c </= 7%	Class I, LOE B
Smoking	Discontinue smoking Counseling, NRT, and oral smoking-cessation medications	Class I, LOE A
Alcohol use	Eliminate heavy drinking or reduce Men </= 2 drinks/day and non-pregnant women 1 drink/day	Class I, LOE C Class IIb, LOE B
Obesity	Goal BMI 18.5 to 24.9 kg/m² and waist circumference women (<35 inches) and men (<40 inches)	No study shows weight reduction reduces stroke recurrence risk
Physical activity	If capable, at least 30 minutes of moderate-intensity to break a sweat or raise heart rate, 1-3x/wk	Class IIb, LOE C

ACEI = angiotensin converting enzyme inhibitor; BMI = body mass index; HTN=hypertension
NRT = nicotine replacement therapy; RX = treatment

Source: Furie KL et al. Stroke 2011; 42: 227-276

Mum told me not to touch it

Outline of activities for early supportive care

1. Ensure clear airway and good ventilation: Given oxygen if necessary but not routinely.

- **2. Nurse in slight head-up tilt (0 – 45 - 90^0) to improve venous drainage from the head region.**

Outline of activities

- **3. continuous monitoring of neurological deficit for deterioration, including the level of consciousness, which may herald impending herniation.**
- **4. Continuous cardiac monitoring, if indicated, particularly if risk factors for coronary heart disease are present.**

Outline of activities

- **5. Do not feed orally if patient is unconscious or drowsy. Swallowing test should be done in conscious patients before oral feeding and feed in the semi-recumbent position (45^0) – ensure correct consistency of food.**

Outline of activities

- **6. If hemiplegia is dense, commence DVT prophhylaxis**
- **7. Do not give hypotonic solution, eg 5% Dextrose in water, as it may worsen cerebral oedema. Monitor serum sodium.**
- **8. Do not treat hypertension except mean blood pressure is above 145mmHg, systolic BP is > 220mmHg or diastolic BP is > 120mmHG. Even then, use oral agents (captopril, calcium channel blockers).**

Outline of activities

- **9. Treat hyperglycaemia as usual**
- **10.Treat fever symptomatically, Search for source and treat appropriately.**
- **11.Avoid urinary catheterisation, as much as possible.**
- **12.Treat neurological complications.**

Consequences: Impact on health related quality of life

- Educational systems are falling apart.
- Funding for education is being cut to compensate for the rising costs of health care.

- Health care workers, Teachers, factory workers, business people and government officials are all dying in alarming proportion.
- Agricultural production is dwindling. Farmers and Farm laborers are dying. Farms do not have the workers they need to produce at high levels.

- ***No segment of the society is left untouched.***
- ***The dx has devastated not only families of those who are affected, but the political, social and economic structures of entire nations; and extends to generations unborn.***

Problems Perculiar to Africa

- ***POVERTY***
- Impact of S-E factors and race / ethnicity on incidence/outcomes
- ***Poor healthcare infrastructure***
  - Inadequate facilities for acute S and post-S rehabilitation
- ***Lack of government programs***
- High cost of S care: **out of pocket syndrome**
  - Exacerbated by inadequate health insurance schemes,
- Poor community awareness
- Enhanced genetic predisposition: incidence, severity, outcome.
- *Epidemiological transition*: ageing popn, popn growth, rapid urbanisation, life style changes; PAR, predominance of S mortality over IHD mortality and important geographical variations in stroke rates, with a clear north–south gradient

  > role of ID as a cause of S in Latin America/Caribbean cf devd

How can we improve stroke Outcome?

- The real challenge of stroke therapy at the outset of this millennium is ***how to translate basic pathophysiologic evidence of ischemic neuronal injury into novel neuroprotective therapies*** either independently or combined with **thrombolysis**

- The management of stroke is changing rapidly as new ideas appear for acute treatment, rehabilitation and secondary prevention

4 main targets areas;

- 1) Population strategy in stroke care,
- 2) The role of the physician in preventive care
- 3) Managing the acute stroke patient
- 4) The place of rehabilitation and prevention of recurrence.

1 Population strategy in stroke care

- ***Public awareness programs are important***
- delays in presentation caused mostly by lack of awareness .
- **All patients within the age range and with a high stroke risk should know the symptoms of stroke.**
- need to present early for evaluation, Rx, prevention of further attacks must be discussed at various levels
- Information on S should be made widely available to the public

introduced in schools, churches, mosques, plays on TV, in the theater and brought to national attention.

The population should be educated on lifestyle modification to prevent/manage cardiovascular disease

•SLYTER H. Guidelines for the management of patients with acute ischemic stroke. Stroke 1995;26(137-138).

2-The role of the physician in preventive care – strategy for risk factor control

- Stroke study grps / development of local guidelines .
- Physicians must identify patients at risk:
- ***Hx of risk factors, thorough clinical examination, simple tests: BP, pulse (ECG if concerned), cholesterol, C-reactive protein* and calculate the body mass index*** / Individual patient's risk of stroke.

- ***Secondary***: Polypill

- ***Tertiary P***: < morbidity/mortality, > outcome
- Stroke Unit:< mortality 30%; >functional outcom < disability / need for institutionalised care.
- ***Quatenary P***: Therapeutic options

*BLAKE GJ, RIDKER PM, KUNTZ KM. Projected life-expectancy gains with statin therapy

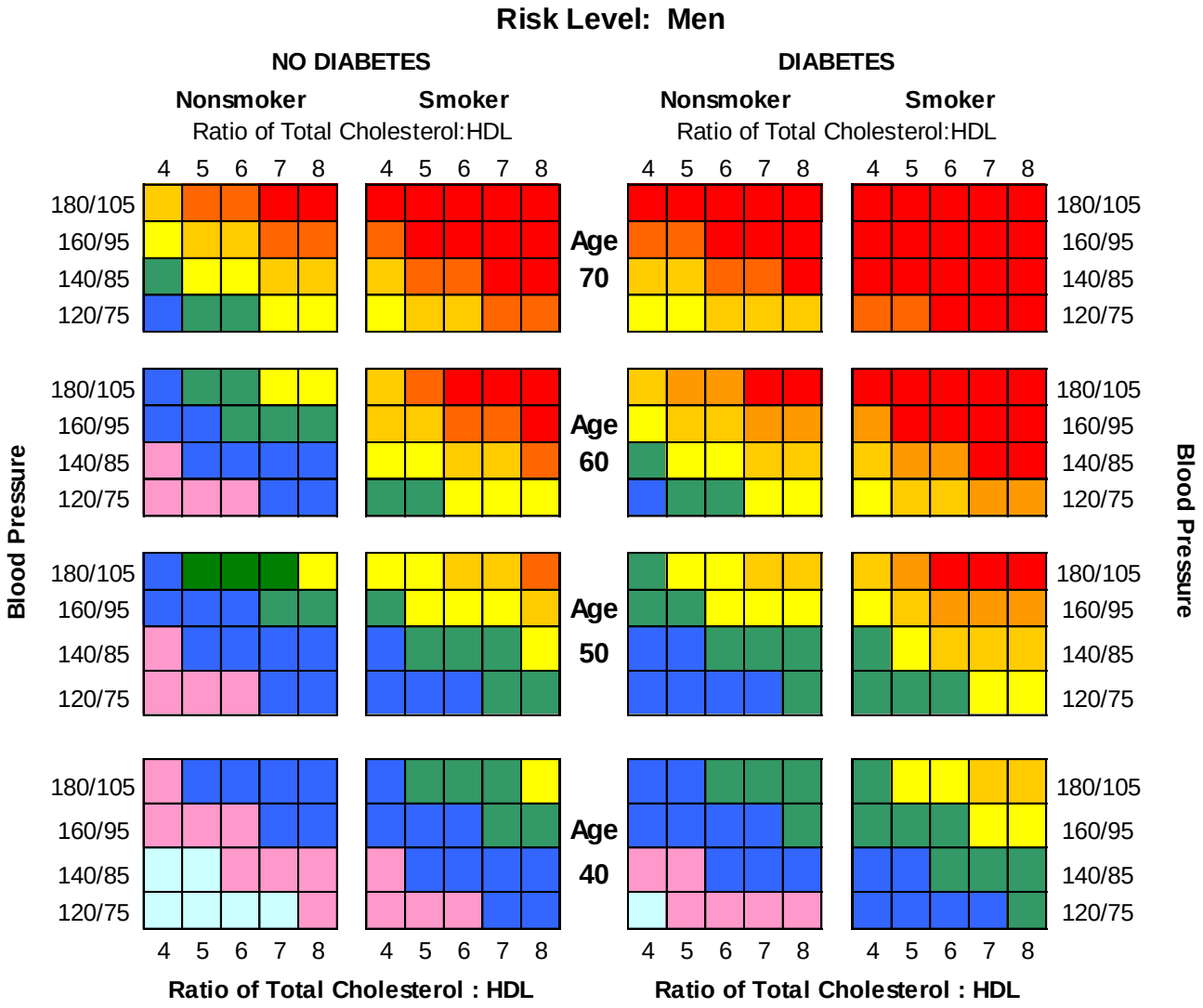
Stroke Risk Scorecard

Each box that applies to you equals 1 point. Total your score at the bottom of each column and compare with the stroke risk levels on the back.

National Stroke Association

Risk Factor	High Risk	Caution	Low Risk
Blood Pressure	> 140/90 or I don't know	120-139/80-89	<120/80
Cholesterol	>240 or I don't know	200-239	<200
Diabetes	Yes	Borderline	No
Smoking	I still smoke	I'm trying to quit	I am a non-smoker
Atrial Fibrillation	I have an irregular heartbeat	I don't know	My heartbeat is not irregular
Diet	I am overweight	I am slightly overweight	My weight is healthy
Exercise	I am a couch potato	I exercise sometimes	I exercise regularly
I have stroke in my family	Yes	Not sure	No
Score (each box=1)			

Assessing absolute CVD risk: the New Zealand guidelines



Present challenges

- Overall, Mx in sSAfrica **is sub-optimal.**
- Stroke units are not yet developed.
- Neuro-imaging centers are very few and assess limited by cost and distance.
- ***Most patients settle for IVF of hypertonic / isotonic infusion, medical decompression:steroid/ mannitol free radical scavengers, folate supplement, statins, anti-platelets PRN antihypertensives*** .
- A significant proportion is seen by non-Neurologist /GPs who inadvertently bring down BP and compromise cerebral perfusion with its attendant poorer prognosis.

- Multidisciplinary rehabilitation team Mx is difficult bc of dearth of paramedical staff, physio/occupational Rxist ; stroke nurses.

What do our patients deserve?

- Our patients deserve timely access to quality services appropriate to their needs.
- ***The most important strategy for stroke treatment is modification of risk factors and life style modification (excercise , diet, no smoking, modest alcohol (+/-)***
- The existing evidence strongly implies that good care of patients with stroke starts with ***organization of the entire stroke chain; from the prehospital scene, through the emergency room, to the stroke unit.***
- Without structured stroke services no pharmacological or intervening Rx is likely to improve the outcome. Thrombolysis apart, our patients deserve better care from the moment they have their first TIA.
- Patients with mild stroke should be managed in a specialist stroke/TIA clinic.
- managed on an acute stroke unit for stabilization, CT scanning and other investigation, and diagnosis, and then referred, if possible, to a specialist stroke rehabilitation unit.
- *Attention should be paid to risk factors to prevent recurrence. This is the ideal that requires modification for the situation in Afr*

We must not set our standards too low if we are to compete in the global village!

What's the big deal about Jacuzzis?

Way forward / Road Map

- Future projection based on current trends
- Incidence velocity / Risk factor prevalence / Relative risk
- PAR Population - Attributable Risks (PAR)

- ***SHAPING THE FUTURE***
- ***Health edu / awareness; Voluntary BP, BS, FLP, counseling / T***
- ***Life style modification in high risk individuals.***
- ***Capacity building: Health Care Workers be trained***
- ***Developing protocols and National guidelines***

- ***Organising substantial Rx network at the local / national levels***

- ***Procurement / distributn of drugs/PolyPill; ensure compliance:***

- ***Strong political will and leadership***

Way Forward

community based stroke prev and Rx programs (eg, national guidelines)

- urgent need for well-designed epidemiological studies and clinical trials to test various widely used, but unconventional Rxic interventions.
- quality and quantity of stroke care in developing countries varies widely: patients' location, S-E status, education, and cultural beliefs.
- Need for further research, appropriate acute care and rehabilitation strategies
- establish basic SU that can be later incorporated int comprehensive specialist stroke acute care and rehabilitation services.

Way forward

if 2% reduction per annum in stroke mortality (due to better mx),

- would result in 6.4 million fewer deaths from S bt 2005 and 2015,
- with most deaths averted and yrs of life gained in LMIC.
- experience of HIC has shown feasibility of such reductn
- advocate a wider use of ***early administration of aspirin*** for ischaemic stroke in LMIC.
- most important contribution to < Mortality is likely to come from
- ***primary prevention***: emphasis on the > major cardiovascular risk factors:
- HBP / DM common to stroke, heart disease, diabetes, and other chronic diseases ***likely*** > burden of S ***to epidemic proptns*** unless effective interventions put in place.

Challenge: implement a comprehensive nation wide approach to stroke prevention, mx, and Rehabilitation.

- ? methodological limitations and study design heterogeneity

- Incidence and outcomes show little geographical variation in the region and seem ***similar*** to those in developed countries: proportional > frequency of ICH and lacunar infarctions

STROKE IN AFRICA – WAY FORWARD

Rx wt BP-lowering drugs to all patients with stroke IF INDICATED,

- basic multidisciplinary acute SU in hospitals that provide emergency care for S
- Development of consensus statements and national stroke guidelines by recognised experts from the region to address local issues on the basis of the best available evidence.
- Effective strategies to improve awareness (including campaigns to remove stigma associated) and ***training of healthcare workers to be developed and implemented on a larger scale.***
- Further research is needed for affordable and potentially widely applicable primary / secondary prevention strategies: polypill containing aspirin, BP-lowering drugs, statin, and perhaps folic acid.
- More research: to assess indigenous medicines and cf various capacity-building strategies.
- Emphasis on effectiveness and efficiency of the interventions in the specific context of developing countries..
- Monitoring effects of interventions at the population level and
- obtaining comparable and reliable data on stroke incidence, prevalence, and risk factors in developing countries

STROKE IN AFRICA: ***Prevention***

- a priority on the health agenda in all countries.

emphasis on effective population-wide interventions to control / < exposure to leading risk factors: ***HBP smoking, high cholesterol, low fruit and vegetable intake, physical inactivity, and alcohol excess.***

- Population-wide efforts to ***< salt intake and tobacco use*** through multiple economic and educational ***policies*** and ***programmes*** suggested as cost-effective primary prevention interventions.
- Primary prevention measures complemented with proven secondary prevention measures,

Summary

- STROKE has ravaged the world for decades, leading to the deaths of millions of people and forcing millions more to Be DISABLED. If they have supportive measures available, that help keep them alive. It has been a dreadful scourge, one that has challenged science, medicine, interpersonal and international relations, philantropy and religion. While many strides have been made on all those fronts in dealing with Stroke, the push for prevention, and effective / efficacious treatment and better ways to ameliorate its effects, both physical and social, goes on ----------

summary

- Increasing incidence and Prevalence in developing world

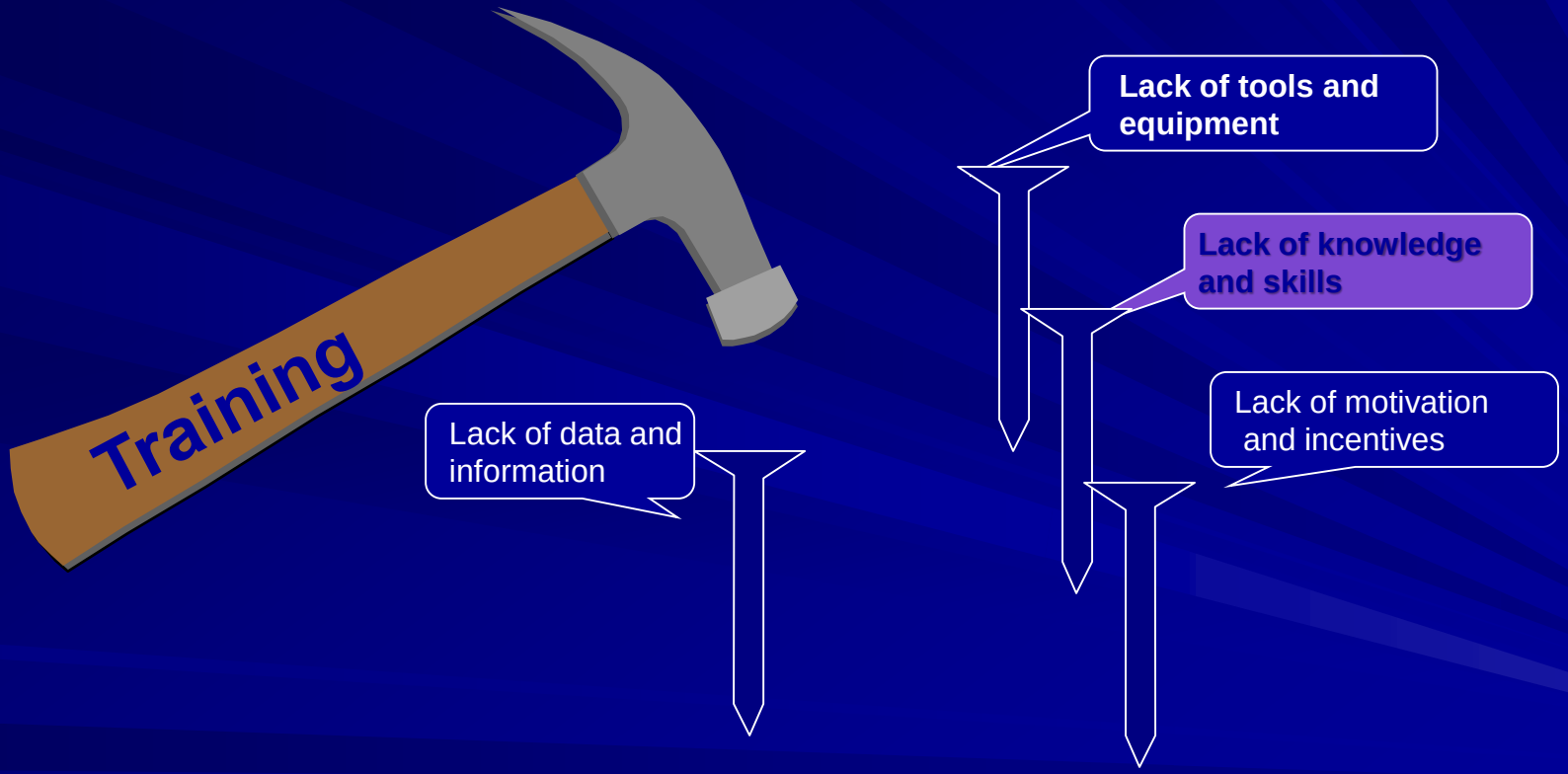
Positive note: Incidence dropping in devd world and response to effective Rx available

- Support from Donor community and Government – a MUST.
  more cost effective to fund prevention programs than to fund Rx.
- Prevention efforts will not be sufficient to quell the spread of this Monster.
- Prevention, care, support and treatment are same package
- Only through a comprehensive approach, combining applied stroke research with sustainable, affordable
- and context-specific evidence-based prevention and Mx strategies will it be possible to stem the
- Global S epidemic, improve outcomes, monitor burden, and save millions of lives around the world.

- stigma and discrimination are major challenges facing victims;
- ROLL BACK STROKE
- ***Achieving stroke – free generation by 2030 is feasible***, but requires innovative approaches to reach marginalised populations

- advancements have arisen on the horizon at a time, when the battle against Stroke is at a cross road.
- Glimmer of hope that we may finally achieve the ‘’three zeros’’.
- ***Zero new Strokes***
- ***Zero stigma/discrimination***
- ***Zero Stroke deaths***

“If all you have is a HAMMER… everything around looks like a NAIL.”



The Bad; The Ugly; The Good

- **The Bad: Died and Buried.**
- **The Ugly : Where we are presently**
- **The Good : Where we are going**
- **My dream - video clip**

(Died and Buried – ***The BAD)***

(Where we are presently)

THE UGLY

THE ROAD TO SUCCESS (THE GOOD)

CHALLENGES

- If you can’t fly, run
- If you can’t run, walk
- If you can’t walk. Crawl
- Whatever you do, please keep moving.

(Where we are going)

THE GOOD


National Institute of Neurological Sciences, Nigeria

Prayer

"*The young chick in the claws of the preying hawk says that she cries* ***so that the world may learn of her plight;***

It knows the hawk will not let go!"

(Igbo proverb)

Merciful God, may those who can change things listen to our plight.

(15419)

Where there is a will, there is a way

Strength through Unity

- Knowing is not enough
- We must apply
  - Willing is not enough
- We must do

(15419)

Yes, we can !
Yes, we can !
Yes, we can !
Yes.....

The Lecture is over - please wake-up and wake your neighbours

Thank You: Shukran

- ROLL
- BACK
- STROKE


Stroke