Urgent Management of TIA

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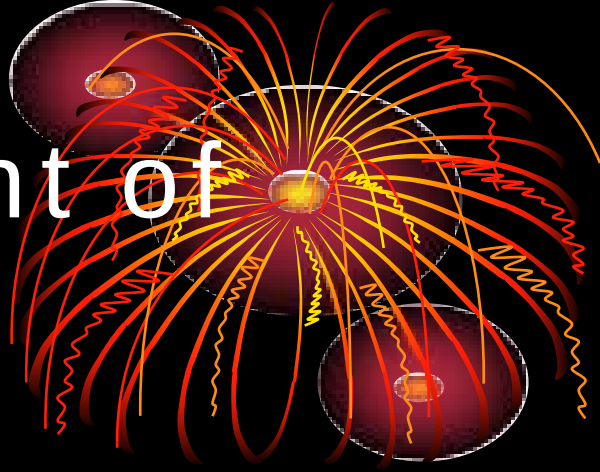
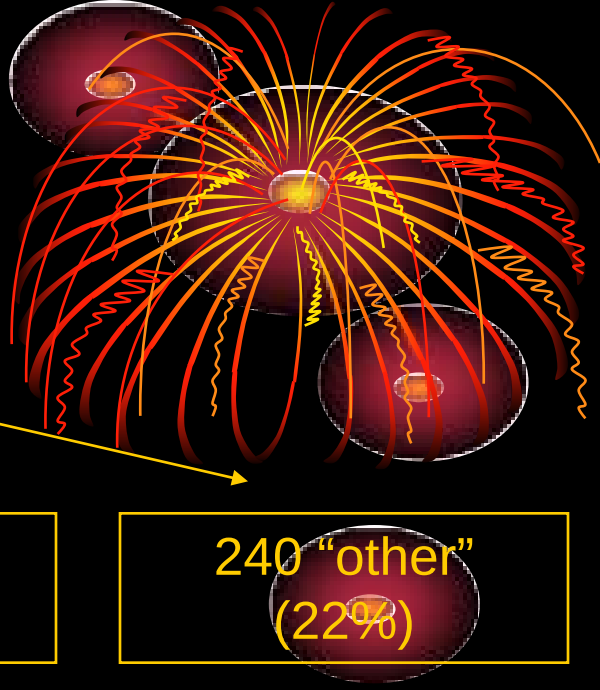
Urgent management of TIA & minor stroke



- Is urgent management effective?
- Evidence for urgent management
- Is urgent management safe?
- Questions

SOS TIA: results



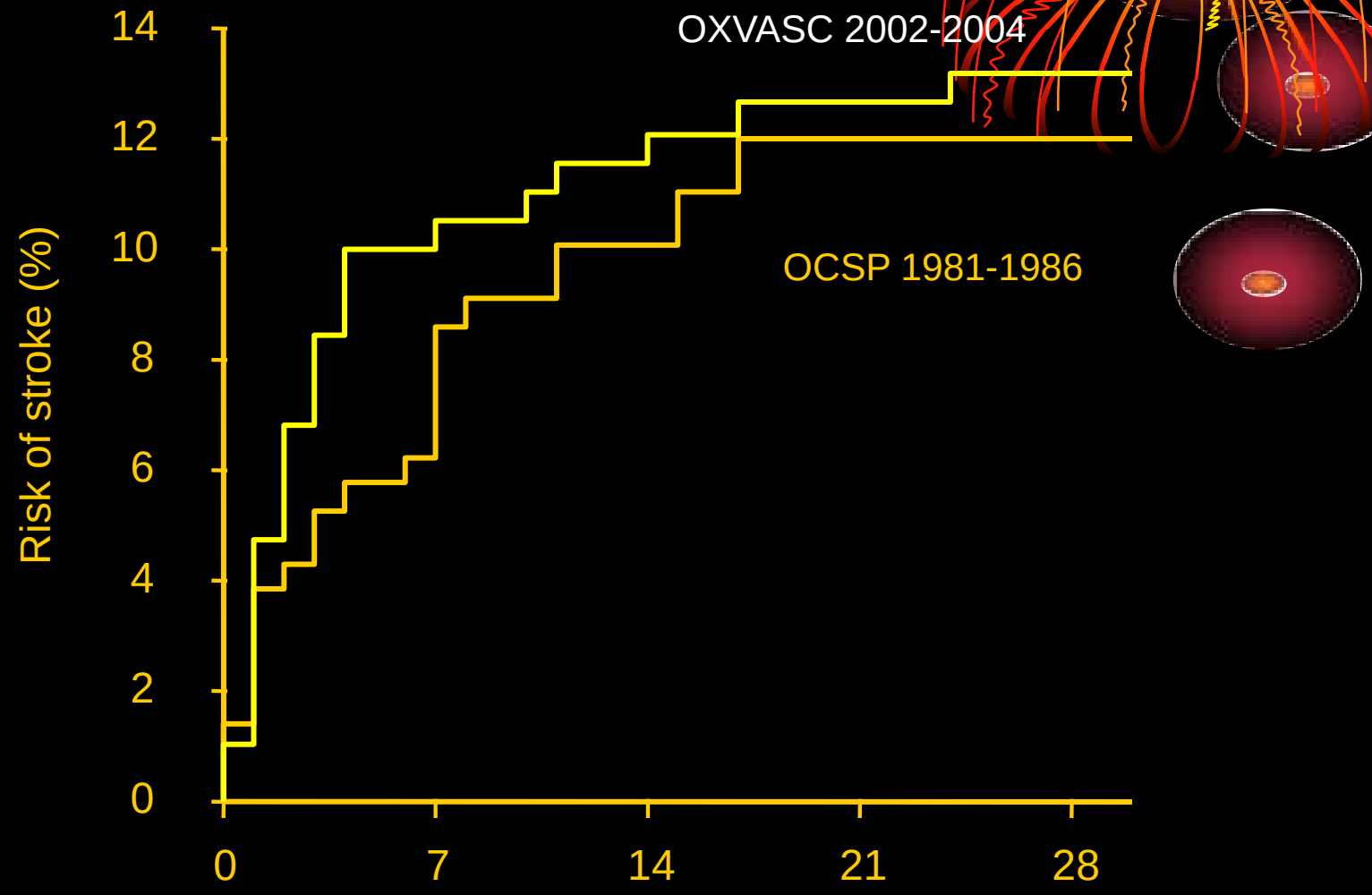
1085 patients

- 643 definite TIAs (59%)
- 144 possible TIAs (13%)
- 58 minor stroke (5%)
- 240 “other” (22%)

- Mean age 62 years old, 51% male
- 53% of patients seen on same day as TIA
- 26% admitted

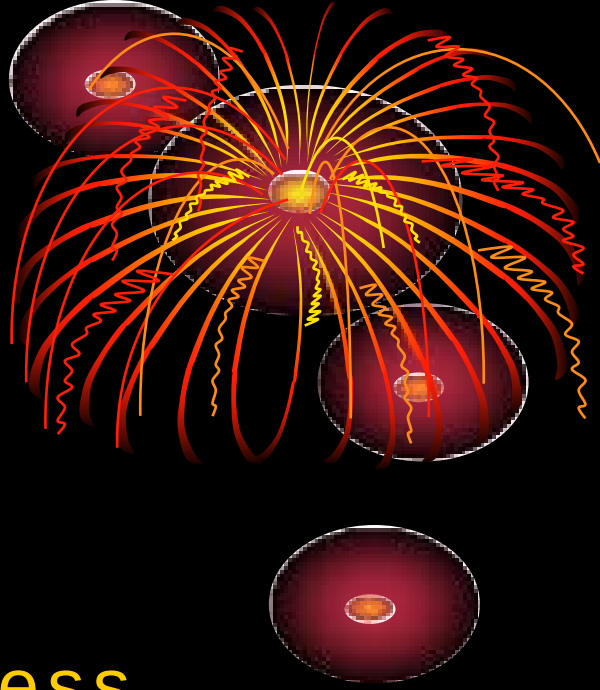
- 13 strokes at 90 days
- 90-day stroke rate- 1·2% (95% CI 0·7–2·1) vs 6.0% predicted by ABCD² score

Cumulative risk of stroke after TIA: OXVASC & OCSP



Lancet 2005; 366: 29-36

ABCD score



A: age = 1 in **≥ 60 yrs**

B: BP = 1 for > 90 D.

C: C/F = 2 for focal weakness

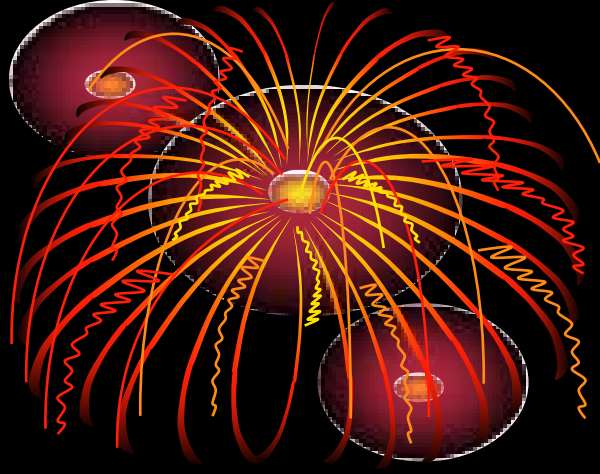
1 for speech problem

D: Symptom duration = 1 for io-59 min

2 for **≥ 60 min**

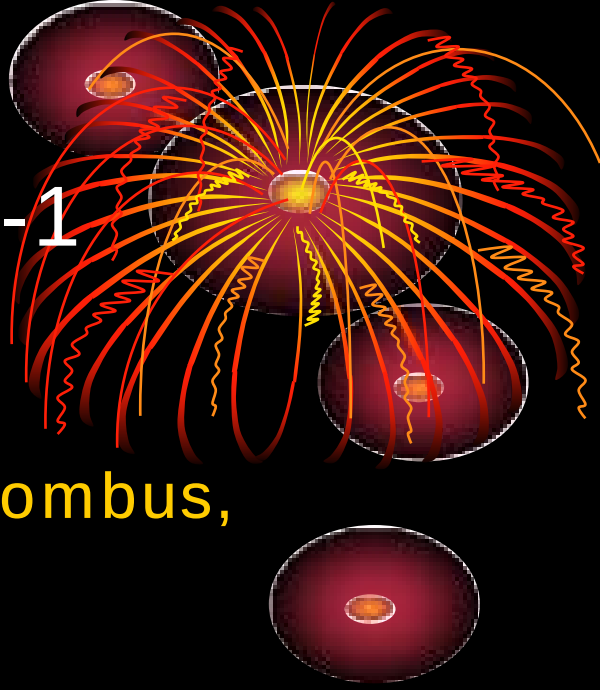
* Score 6 high risk : <4=0% >4=35%

Definition of Stroke



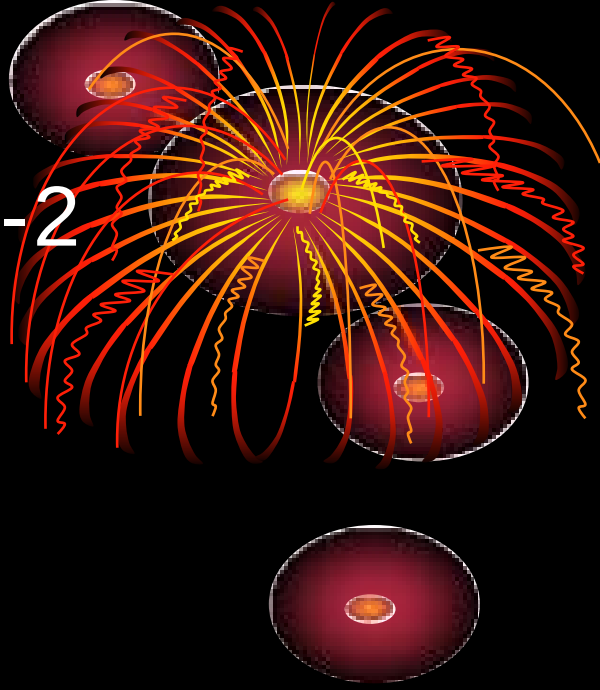
- Stroke (Brain attack) is a syndrome characterized by rapidly developing clinical symptom/or sign of focal loss(sometimes global) of cerebral function in which symptom lasts >24 hours or leads to death where no apparent cause other than vascular origin
- TIA:Same symptoms but lasing for <24 hours.

Etiological classification-1



- ISCHAEMIC
  - **Cardioembolic**:AF,mural thrombus, paradoxical embolism from PFO,endocarditis
  - **Atherothromboembolic**: carotid atheroma, vertebral atheroma, cerebral a occlusion, carotid dissection
  - **Small vessel disease**: hypertensive vasculopathy, diabetic vasculopathy, vasculitis

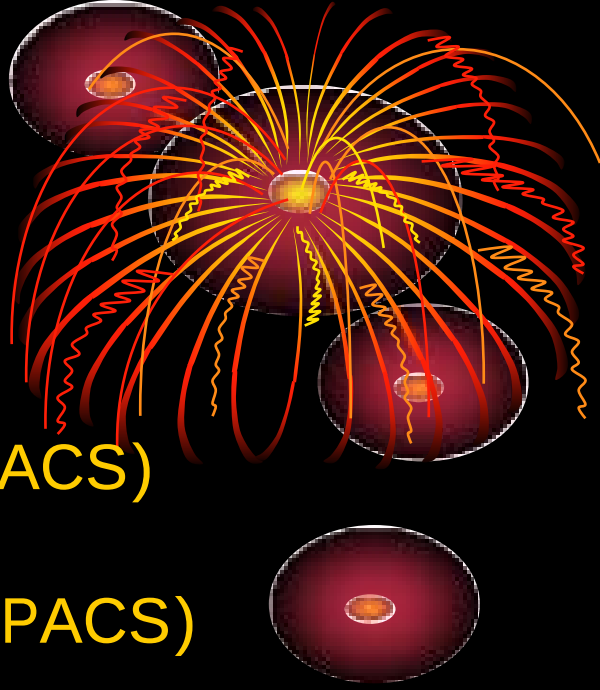
Etiological classification-2



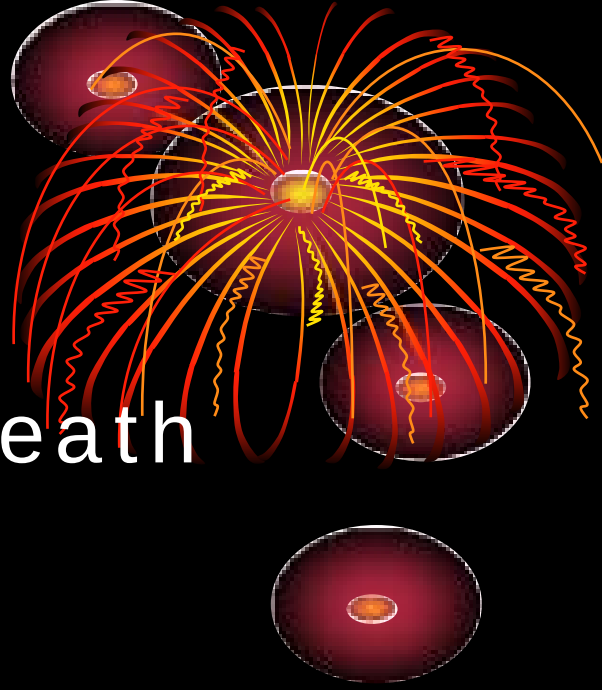
- **Others**
  venous sinus thrombosis
  Unknown

- HAEMORRHAGIC
  Subarachnoid: AVM,Aneurysm
  Parenchymal haemorrhage:
  hypertensive, amyloid

Clinical classification



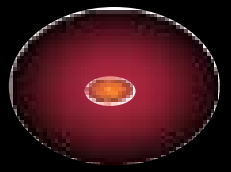
- Total anterior circulation stroke(TACS)
- Partial anterior circulation stroke(PACS)
- Lacunar stroke
- Posterior circulation stroke(POCS)



Major Pathways of cell death

- Excitotoxicity
- Oxidative stress
- Apoptosis

How neurone die after stroke



- Not all cell die immediately
- Cell death evolve slowly in heterogenous area- penumbra *Sharp,* 2000.
- Penumbra is electrically silent
- Metabolically active to sustain membrane potential
- Precise timing & mechanism ?

↓02 & glucose

↓

↓ ATP

↓

Ionic gradient loss

↓

Glutamate release & re-uptake impaired

Binds to receptor → promotes Ca^{++} entry & release

↓

Ca++dependent synthases impaired

↓

Degrade Cyto-skeletal & enzymatic protein

Generate NO, $ONOO^-$

Mitochondrial function fail

↓

Reactive Oº release

↓

Attack protein, lipid, nucleic acid

↓

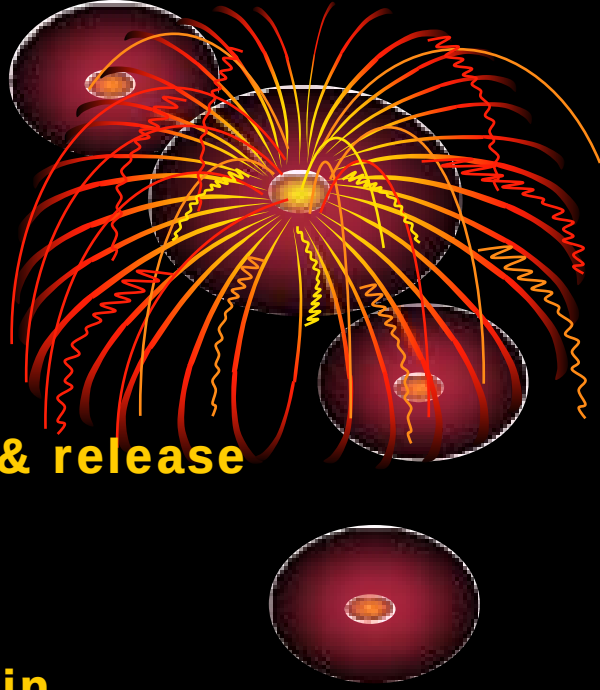
Release families of executioner molecule (capsases, AIF)

↓

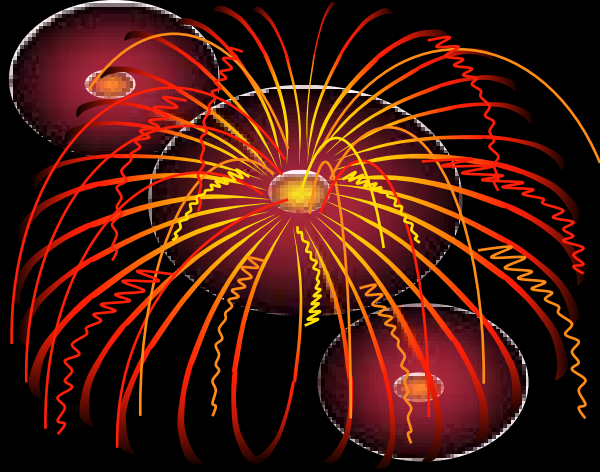
Dismantle cytoplasm & nucleus

↓

Promote apoptosis



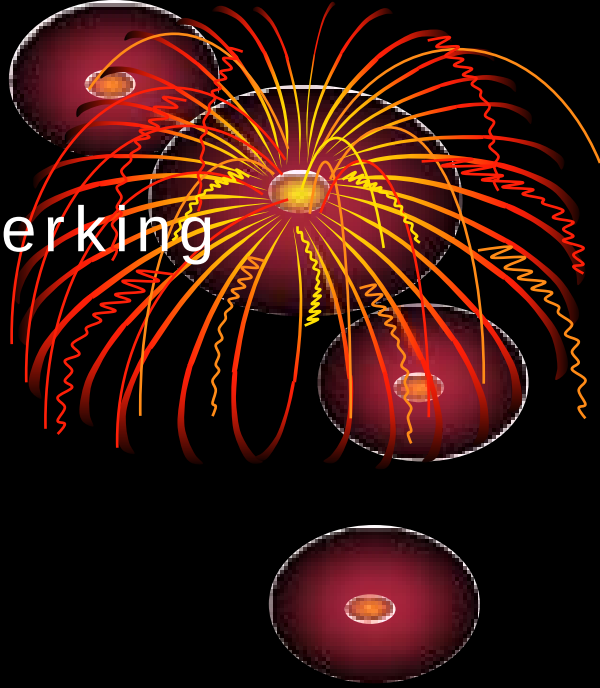
History taking



- Sudden numbness/weakness of face,arm,leg sp. One side
- Sudden confusion, speech/understanding
- Sudden deterioration of vision-one/both
- Sudden difficulty in walking/dizziness/balance/co-ordination
- Sudden severe headache with no cause

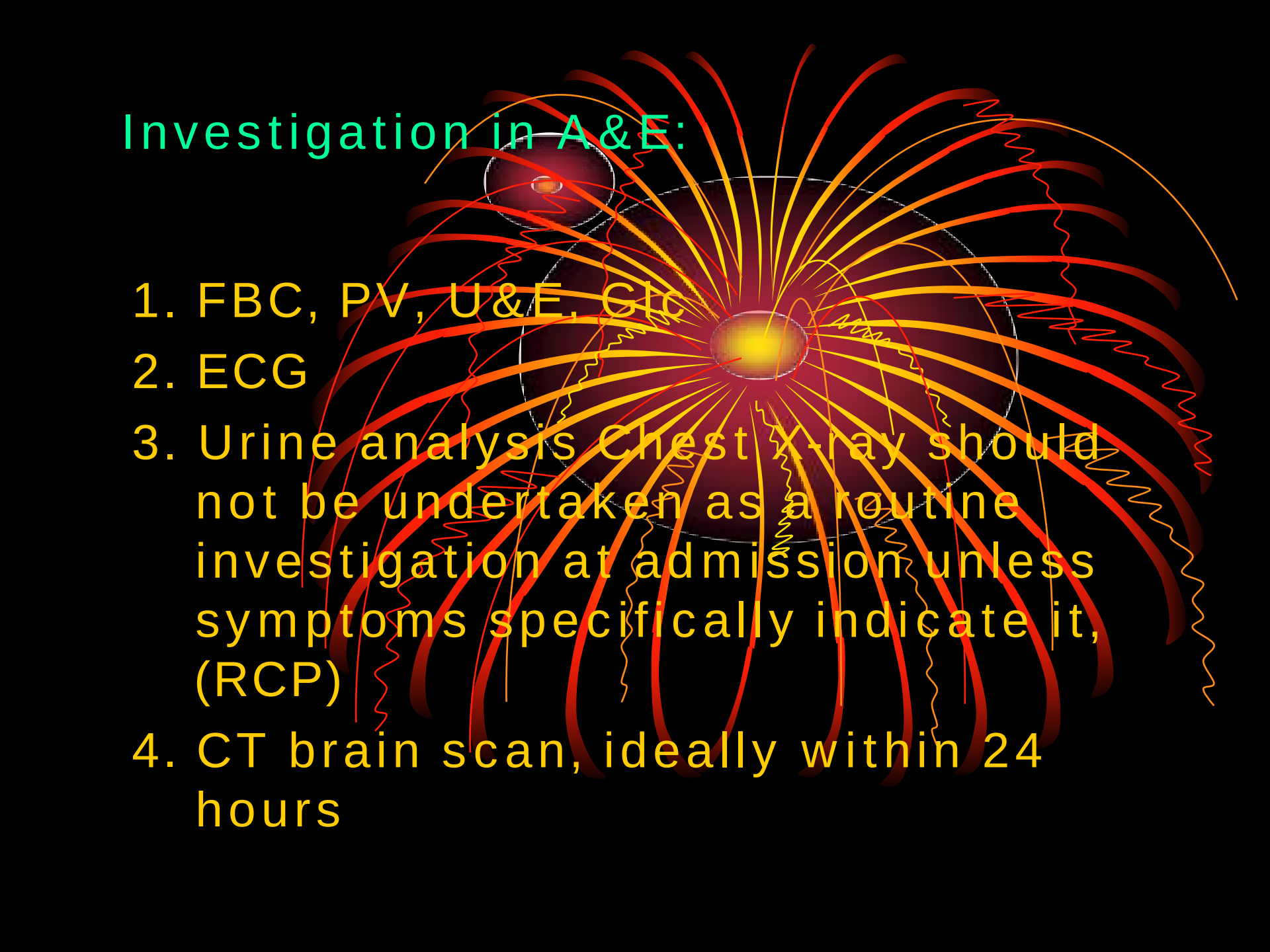
Clinical Examination:

(best done using Leeds Stroke Clerking proforma)



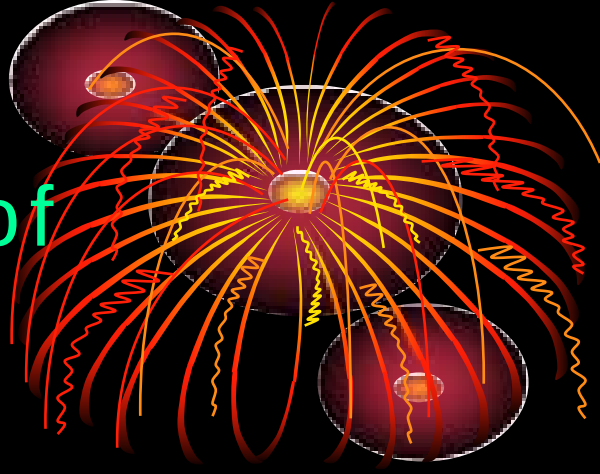
1. General examination
2. GCS
3. Communication difficulty
4. Examine cranial nerves
5. Assess for swallowing difficulties using test swallow
6. Examine limbs for tone, power, sensation, refleses

Investigation in A&E:



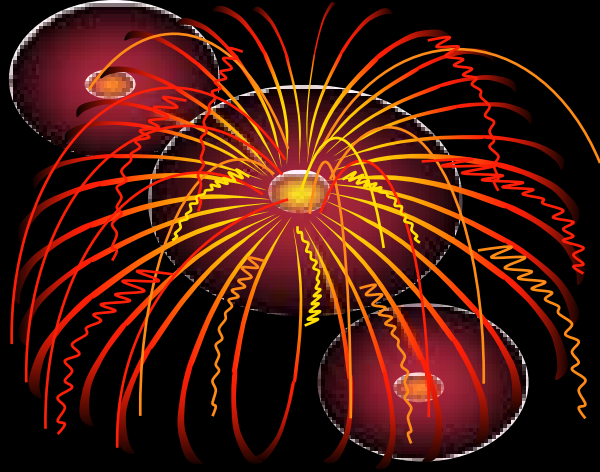
1. FBC, PV, U&E, GIC
2. ECG
3. Urine analysis Chest X-ray should not be undertaken as a routine investigation at admission unless symptoms specifically indicate it, (RCP)
4. CT brain scan, ideally within 24 hours

Brain imaging should be undertaken as a matter of urgency if:



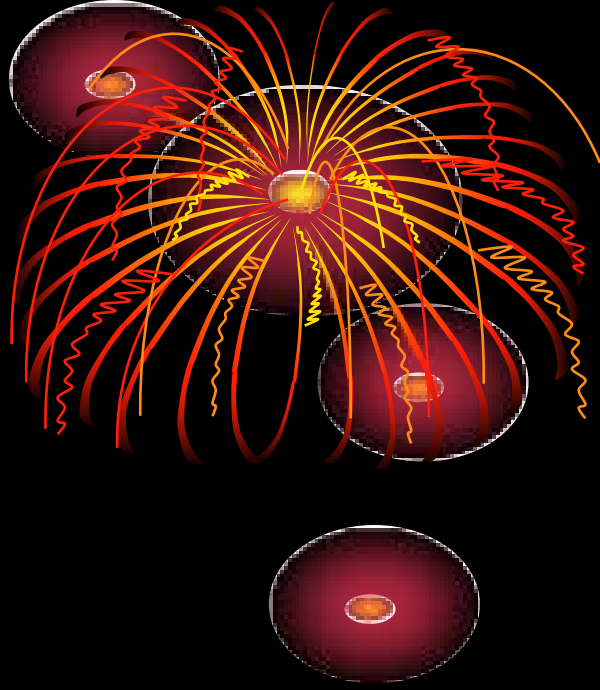
- There is a clinical deterioration in the patient's condition;
- Subarachnoid haemorrhage is suspected;
- Hydrocephalus secondary to intracerebral haemorrhage is suspected;
- Trauma is suspected;
- The patient is on anticoagulant treatment, or has a known bleeding tendency;
- The diagnosis is in doubt because of other unusual features.

Treatment



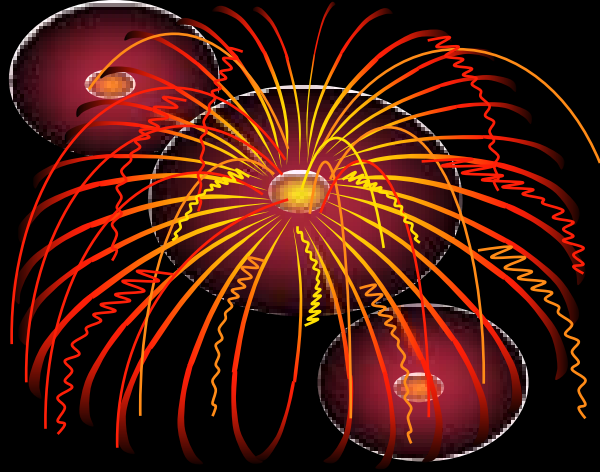
- Premedical care
- NINDS recommendations for trrombolysis
- Door to doctor 10mins
- Access to neurology 15min
- Door to CT 25min
- Door to CT interpretation 45mins
- Door to treatment 60mins
- Admission to monitor bed 3hours

Guideline for TIA

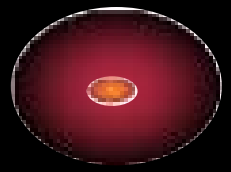


- Timing
- Hospitalization
- Laboratory test
- Echocardiography
- Brain imaging
- Carotid duplex
- Anti coagulation/anti platelet
- CEA

General measures

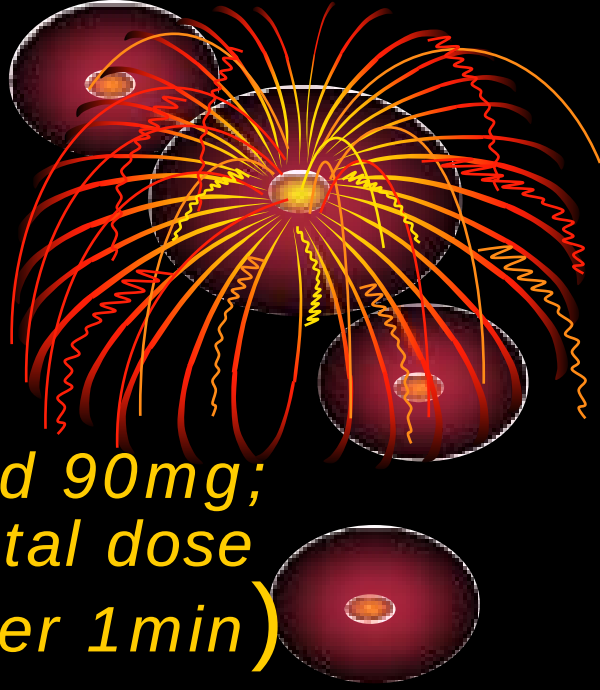


- DIET
  - NPO until swallowing is assessed
  - If risk of aspiration tube or PEG



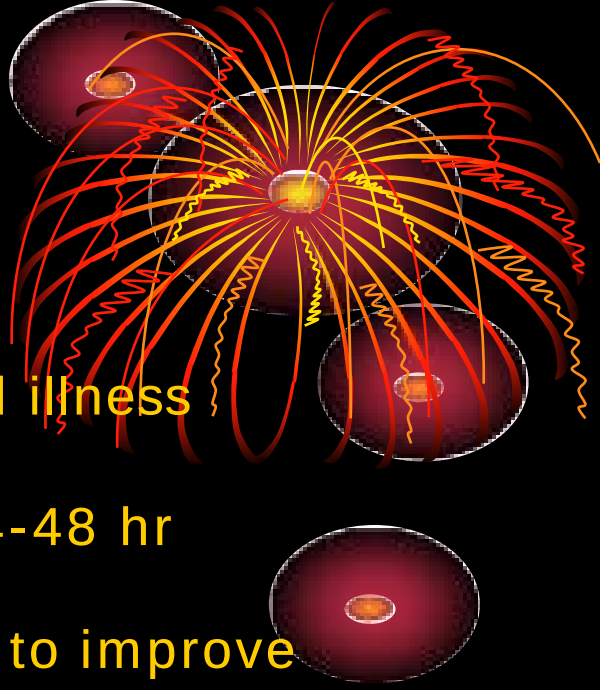
- ACTIVITY
  - activity is tailored to the severity of stroke
  - physical therapy should be started within hours
  - early mobilisation

Medicines In Stroke



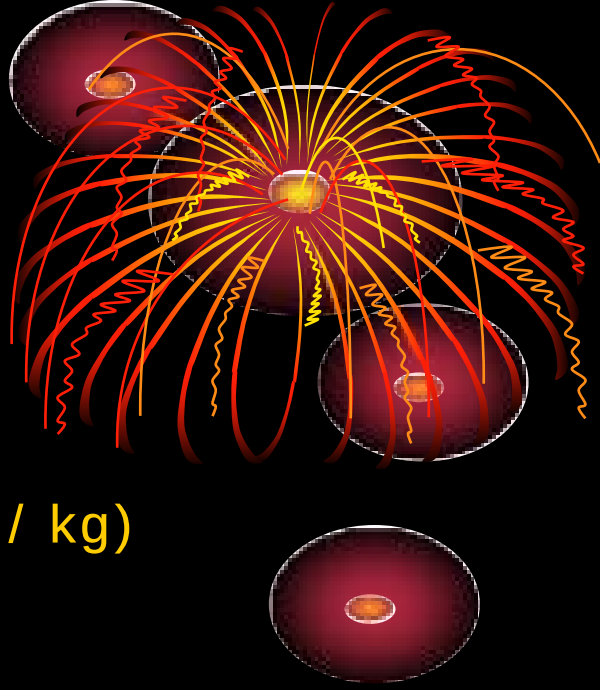
- T-PA *(0.9mg/kg iv; not to exceed 90mg; infuse over 60min with 10% of total dose administered as initial iv bolus over 1min)*
- Antihypertensive Labetalol,Enalapril,Nicardipine,Sodium nitropruside
- Antiplatelet Aspirin,
- Anticonvulsant Diazepam,Lorazepam,
- Antipyretic Paracetamol

Further inpatient care



- Care is tailored to severity and comorbid illness
- Observe pts condition 25% worsen in 24-48 hr
- facilitate medical and surgical measures to improve outcome eg carotid stenosis, cadioembolic source
- Prevent subacute complication eg LMW heparin, stockings,fluid,electrolyte etc
- Initiate therapy to prevent recurrence
- Begin effort to restore function through rehab
- Treat comorbid condition ziz, DM, Hyperlipidemia,cardiac arrhythmia, hypertension

New therapies



Desmoteplase – Favorable outcome

In Rx 22.0% placebo 13.3% (62.5mg / kg)

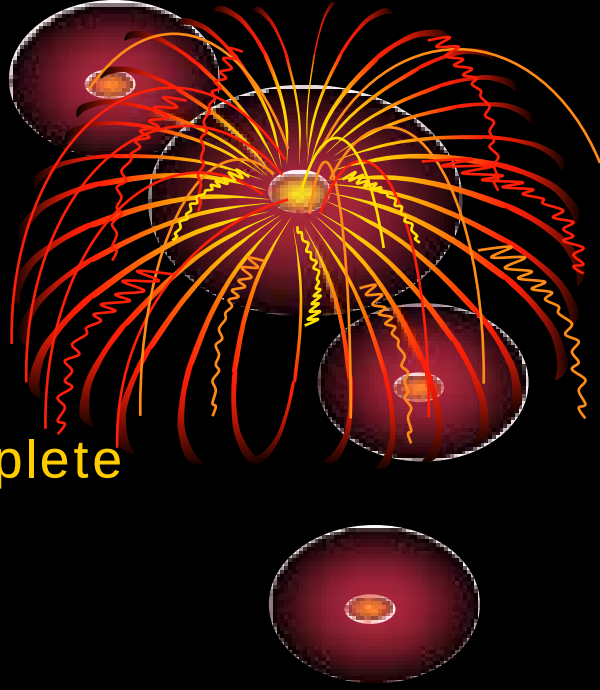
46.7% (90mg/kg) = P= .053

60% (125mg/kg) P= 0.009

Reduction of perfusion lesion volume in MRA was 71.4%

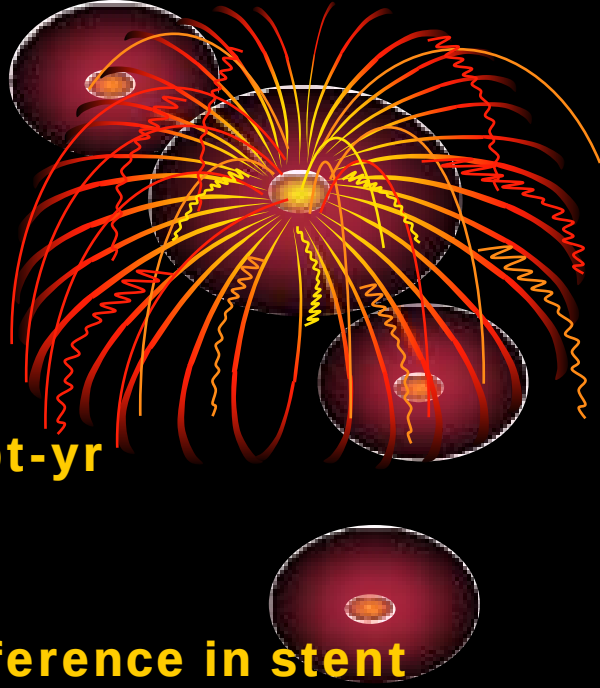
Ximelagatrans- a thrombin inhibitor is better than warferin because it does not need regular INR check-up. SPORTIF, 2005

New Rx



- Mechanical clot removal
- The primary endpoint of partial or complete recanalization rate was
  53.5% in MERCI
  18% in PROACT placebo
- Endarterectomy >70% in symptomatic
- In Asymptomatic – less clear
  ACAST trial 3120 pt. Over 60% stenosis
- Risk of stroke in CEA in 30 days 3.1% vs 0.8%
- 5 yr risk of non-preoperative stroke was 3.1% & 11% ($p < .0001$)
- Carotid stenting / angioplasty (SAPHIRE) equivalent to CEA

Secondary Prevention



- **Carotid revasularisation :**
  - **Carotid endarterectomy**
    ECST & NASCET- 5893 pt 33000 pt-yr
    - Benefit greater aged > 75 yrs
  - **Carotid stent**
    SAPPHIRE : 3 yrs – absolute difference in stent & CEA were 7.9%
    - Yadav 2004
- **Cholesterol**
- Relationship is less understood

  Incidence increase in highest total cholesterol : HDL ratio

HPS : 4.3 yrs after stroke – Simvastatin

After 4 yrs 20% risk reduction in vascular events.

Statin- in addition to lipid lowering actiona) Uprgrade NO synthase b) inhibit iNO c) attenuate inflamatory cytokines in cerebral ischaemia d0modify endothelial function, Arc Neurology 2005

Target level 100mgdl(2.6mmol/L) Start 3.4mg/dl

Secondary Prevention (contd.)

- Small increase (2-6/ 1000) in hemorrhage is offset by reduction in vascular events 51/1000.

- Homocysteine –considered as a risk factor-independent?

  VISP (VITOPS) –B vitamins help in reduction

- Antiplatelet

  CURE / MATCH / CAPRIE

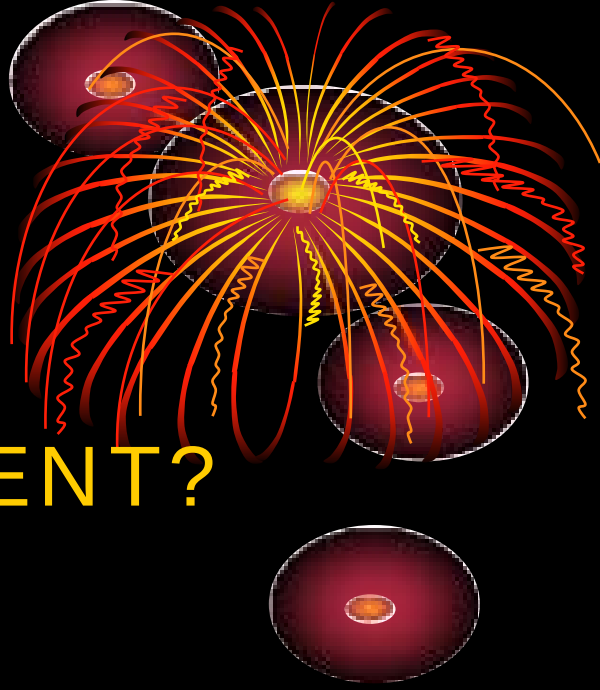
  - Clopidogrel + Aspirin reduced RR of vascular event by 20% vs Aspirin alone.

  Xime lagatrans-a thrombin inhibitor is better than warferin because it does not need regular INR check, JAMA 2005

CONCLUSION

- NOT WARNING- CONSIDER TIA AS STROKE
- CONSIDER FOR ACUTE TREATMENT
- WORK UP FOR SECONDARY PREVENTION

DILEMA IN TIA TREATMENT



- WHAT IS THE TREATMENT?
- INTERVENE
- WATCH
- TREATMENT FOR SECONDARY PREVENTION