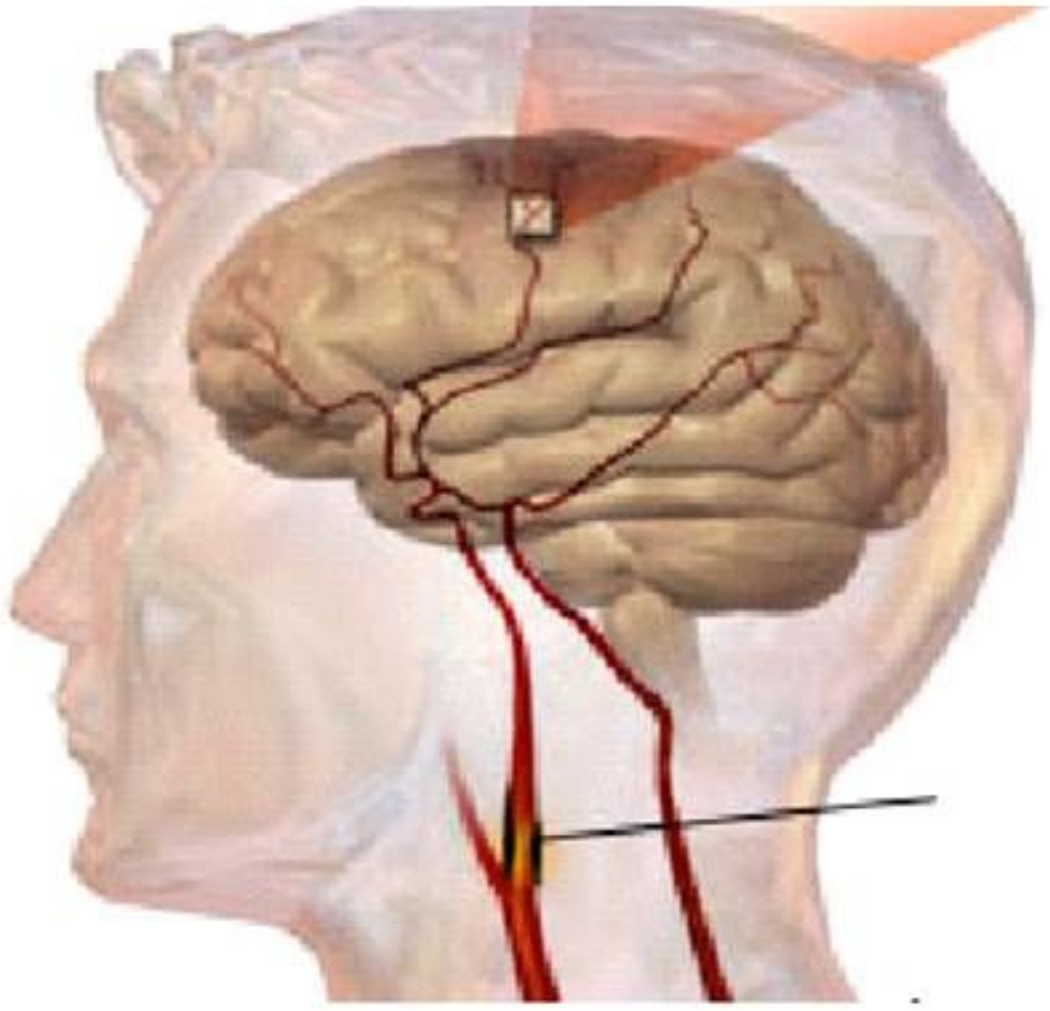

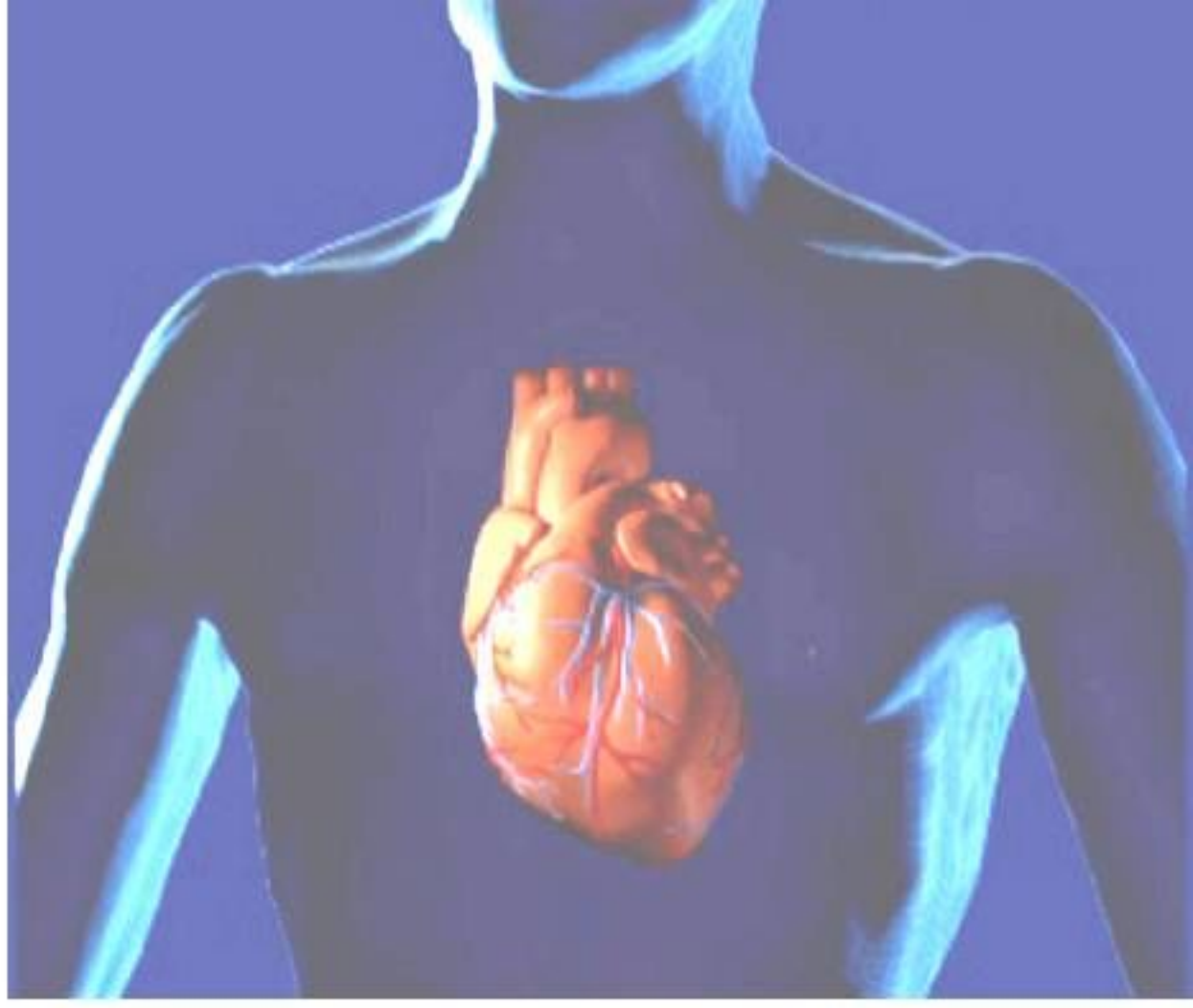

TRANSIENT ISCHEMIC ATTACKS OF HEART

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NATIONAL INSTITUTE OF NEUROLOGICAL DISORDERS AND STROKE

TIAs are well recognized entity in cerebrovascular circulation

"Sudden focal neurological deficit that clears completely within 24 hrs"

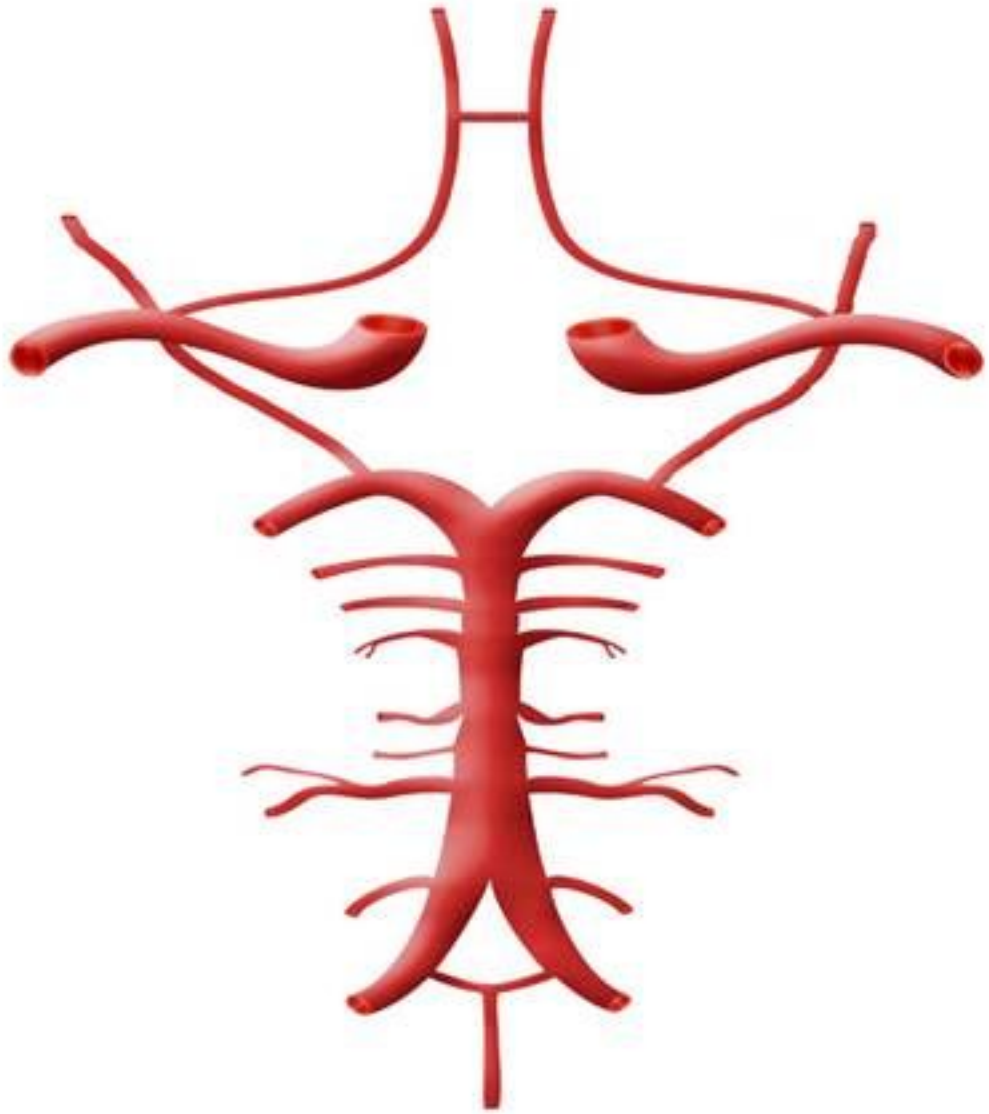
It's estimated millions of episodes occur every day in elderly population

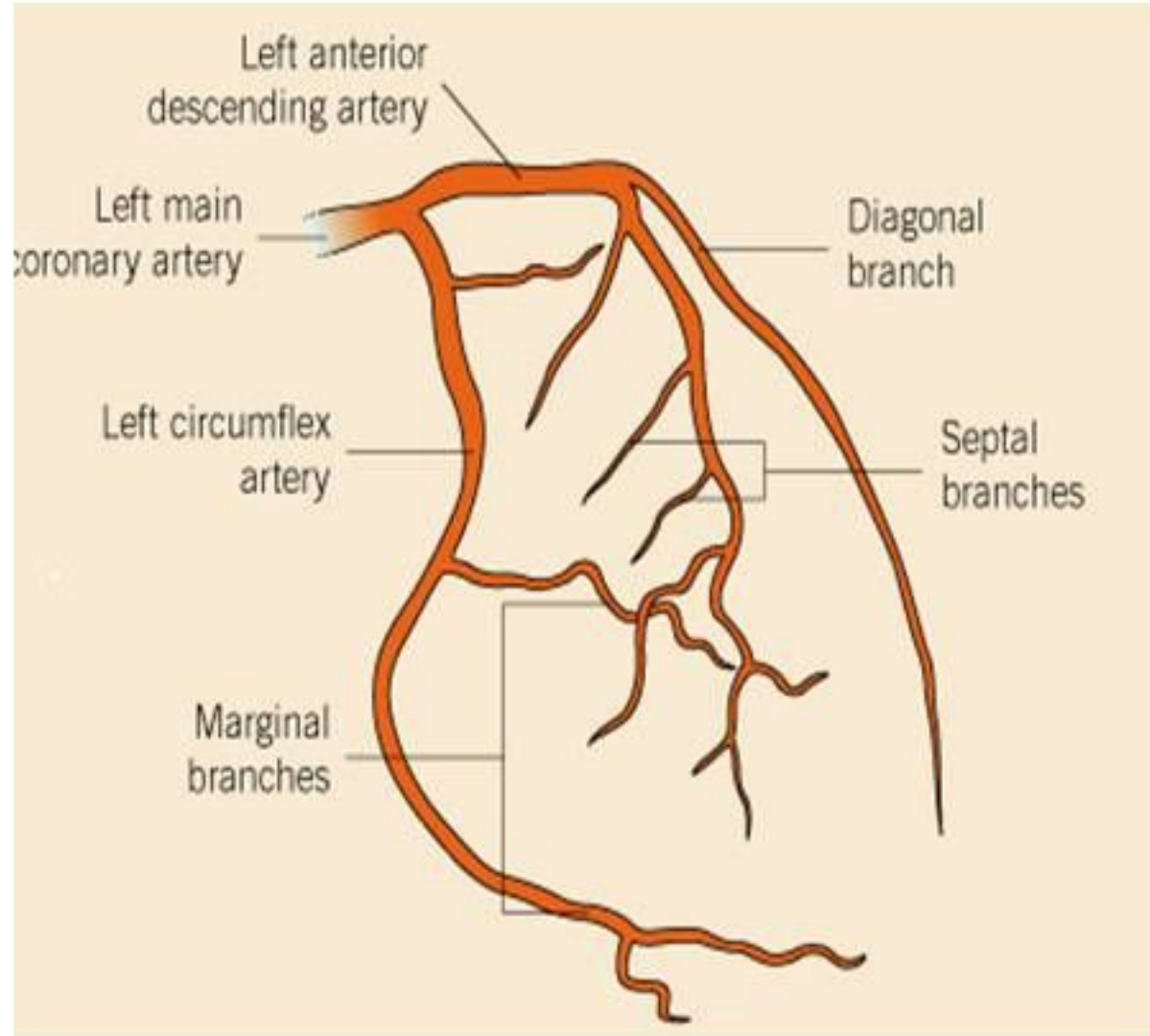
Many of them go un recognised

There is an established link between TIA and heart .It predict a ACS in the near future

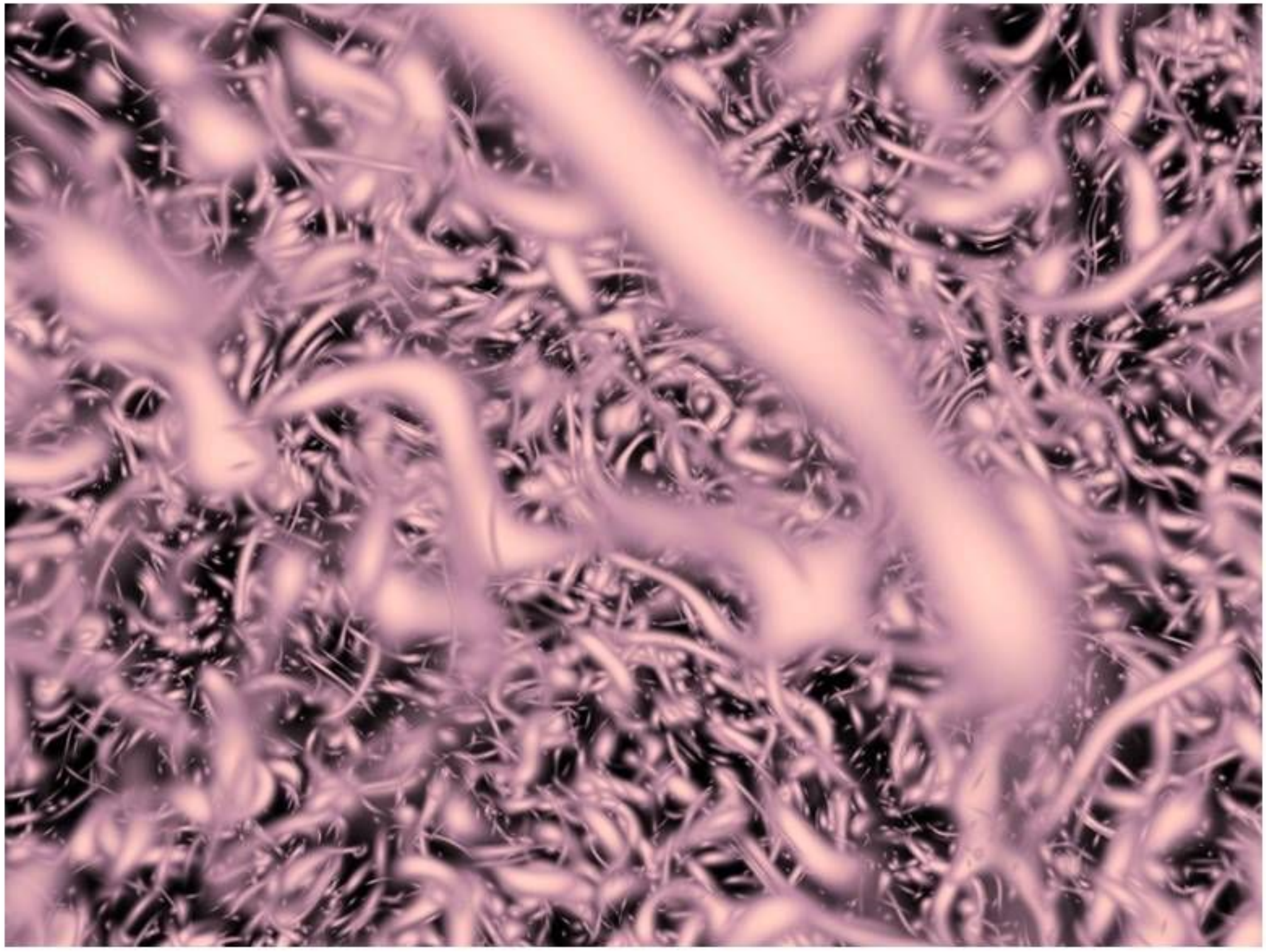
Many patients with TIA succumb to cardiac events

Coronary vs cerebral circulation





Both share a rich micro circulation



TIAs

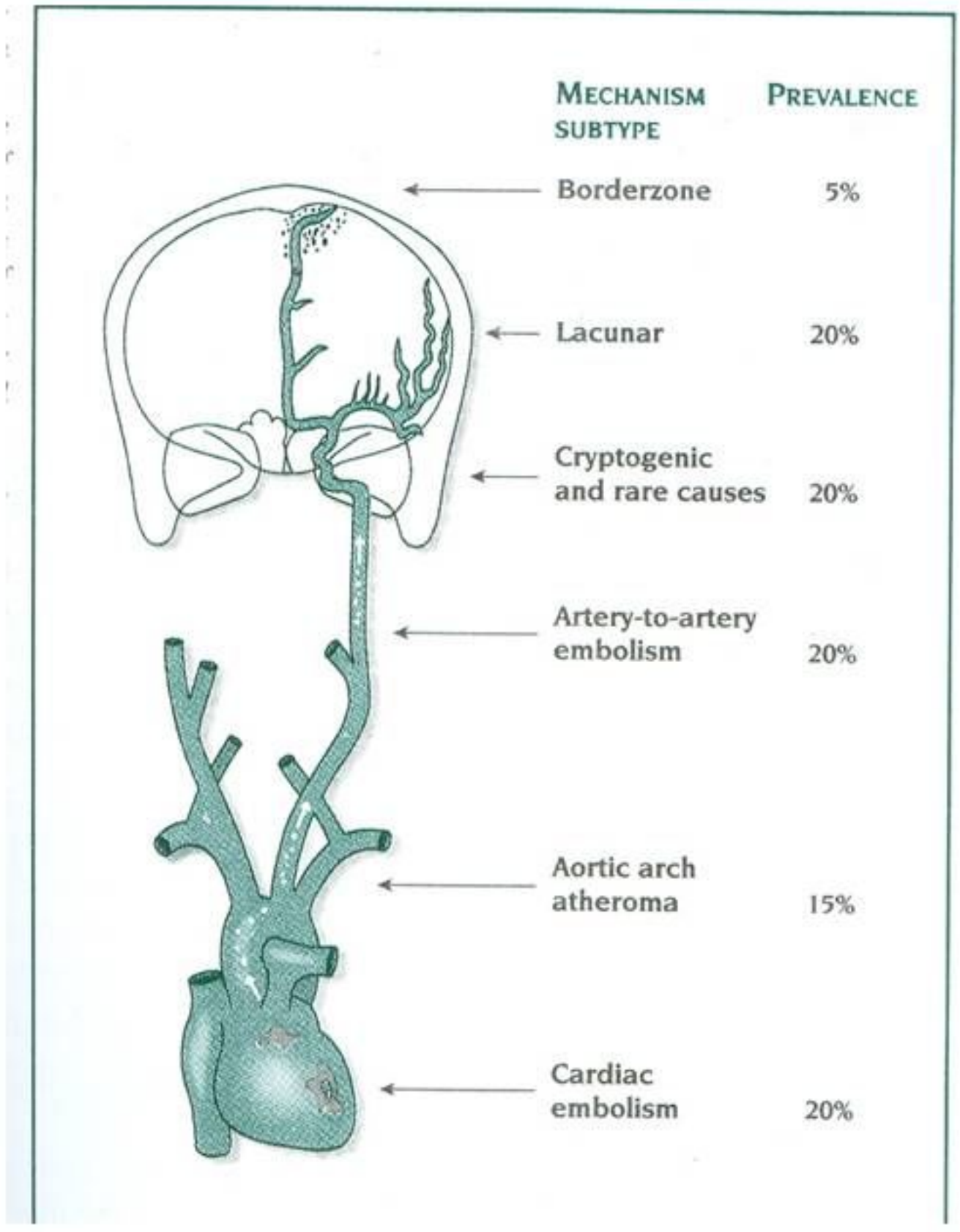
Two major forms recognized

Embolic TIA

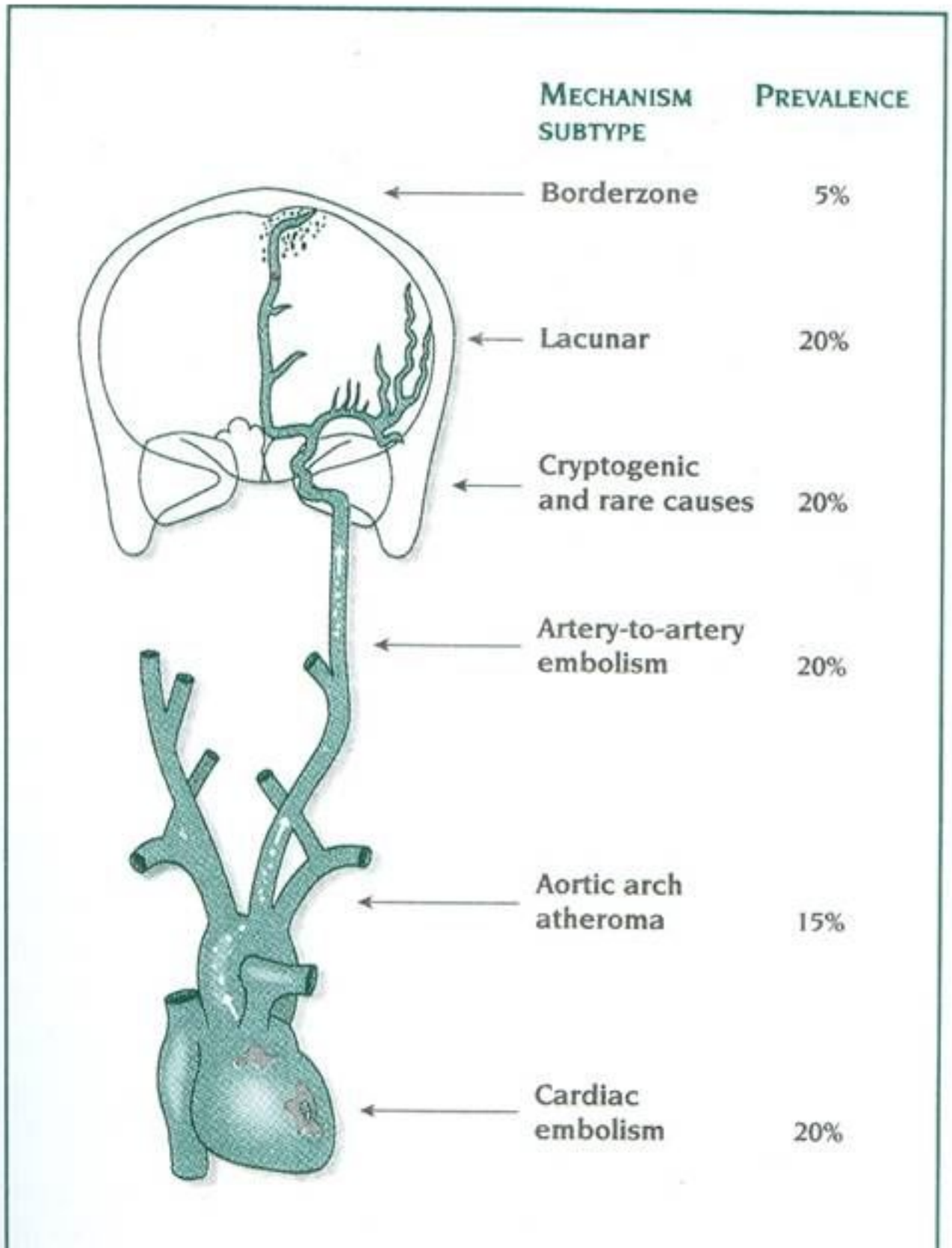
Low flow TIA

(When cerebral auto regulation is challenged)

Cerebral emboli sources

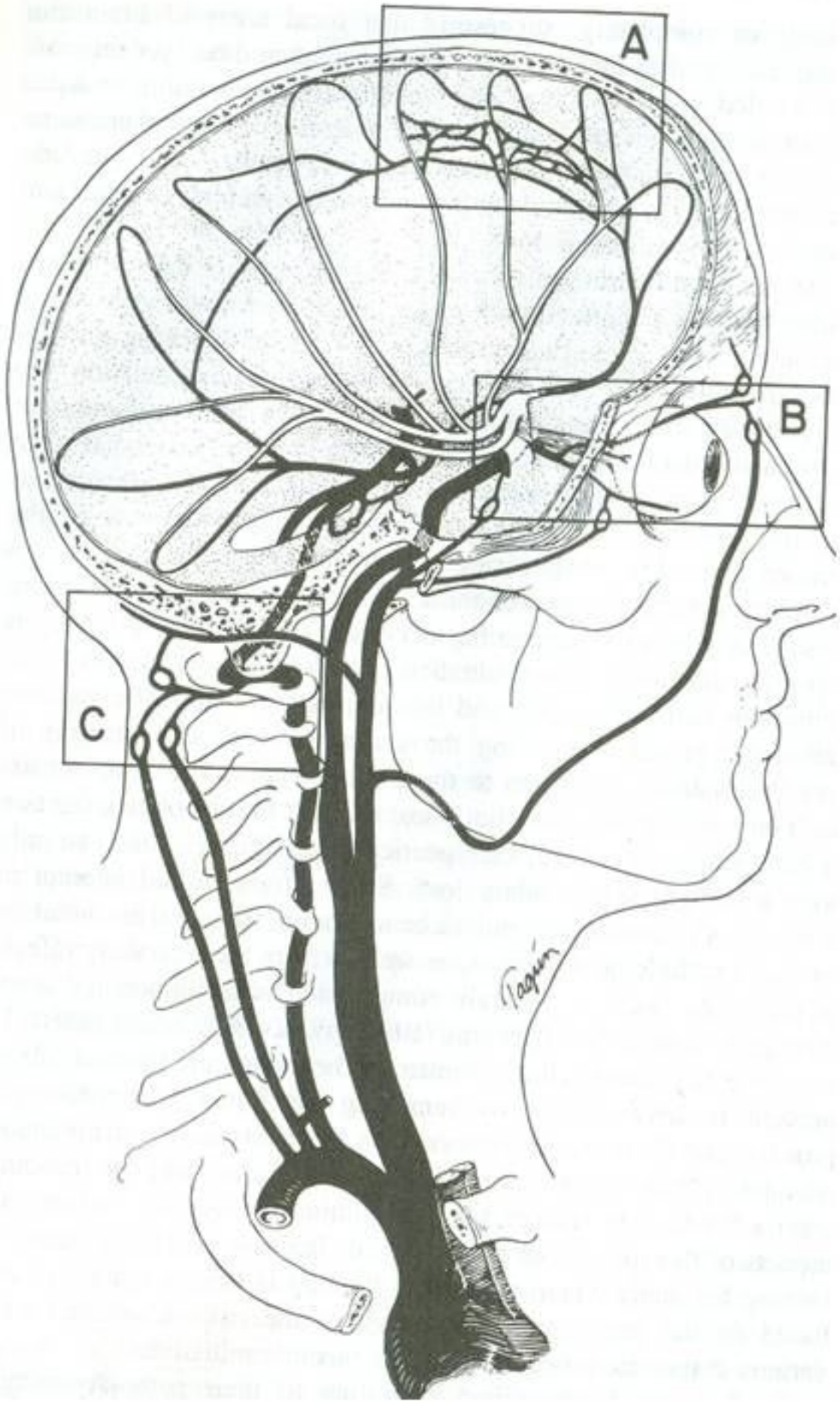


Why TIAs are more common in cerebral circulation ?



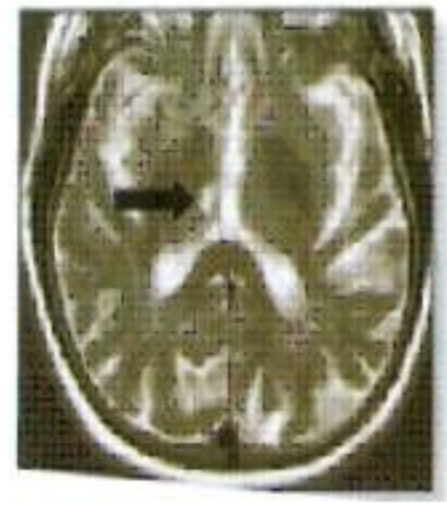
The coronary circulation is the first "port of call" for blood after leaving the heart.

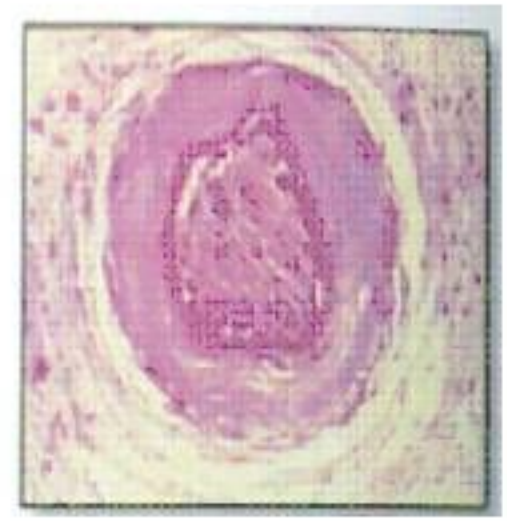
while cerebral blood flow must traverse many atherosclerosis prone arteries



Low flow TIA ,

lacunar & water shed Infarcts





TIA

Symptoms are referred to arterial territory

Carotid,MCA,

The duration,, frequency tells us the mechanism. *It can be transient motor or sensory deficits*

Repetitive hand ,arm weakness indicate focal ischemic TIA

While single episode of facial weakness or transient loss of vision indicate embolic TIA

Posterior circulation TIAs

Often low flow related than embolic

Characterized by dizziness, diplopia or dysarthria

The cerebral & coronary circulation

What's different ?

	Cerebral circulation	Coronary circulation
Blood flow	750ml/mt	250ml/mt
% CO	14%	5%
O2 consumption	19%	12%
Resistance (Cvr)	10RU	6.4RU
Collaterals	developed	Poor
Auto regulation	Prominent	Less prominent
Auto regulation	Neural control	Metabolism dependent

The cerebral & coronary circulation

What's common ?

Atherosclerosis is a diffuse disease of medium sized vessels

It ***does not differentiate*** between coronary arteries from carotid or cerebral vessels

Both have complex neural, hormonal, and metabolic control which is vulnerable under extreme physiological situations

Clinical effects of atherosclerosis in Heart & Brain

Vascular events in brain

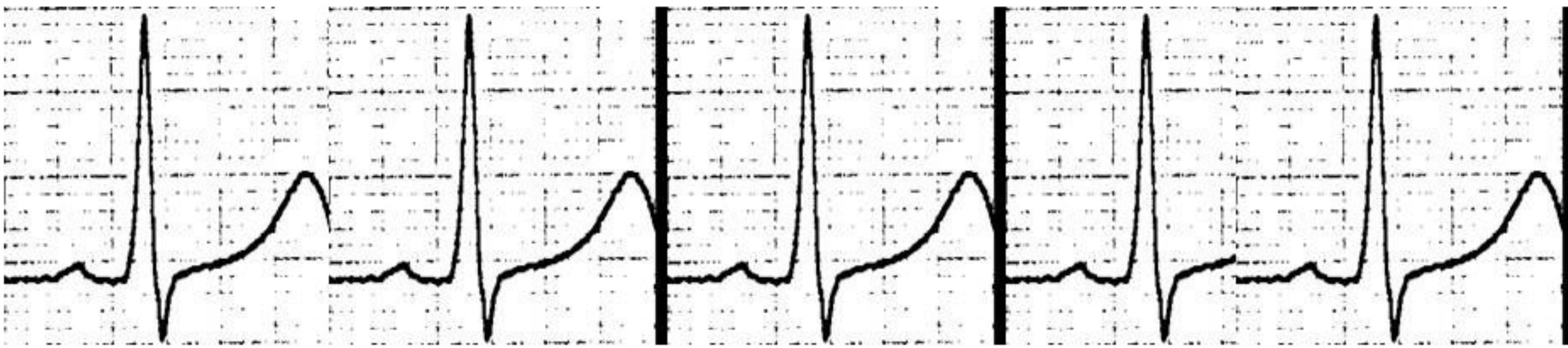
TIA –RIND-Stroke in evolution – completed stroke

Vascular events in heart

Angina –Unstable angina-NSTEMI-STEMI

Genesis of the concept

In the CCU it's not uncommon to see patients with typical angina with transient ST segment shifts.



TIA s are called warning shots in cerebral circulation

No warning shots for heart ?

or

Are we deaf to it?

Aim of the study

We sought to analyse whether there could be reversible ischemic events in the coronary circulation.

The study population

Patients who were admitted in CCU with history of transient ECG changes suggestive of ischemia with or with out angina

Retrospective –Observational study

14 patients

Mainly presented as Unstable angina(**one following CABG**)

5 Patients were referred from post operative ward

(Non cardiac surgery with episodes of prolonged hypotension)

Male female ratio was 1:1.5,

Mean age 42(Range 29-68).

Clinical

Typical rest angina

Atypical angina like pain during emotional stress

Post cabg anginal pains

ECG

Transient ST shifts

Runs of VPDs, NSVT

Episodes of Bradycardias

Transient AV blocks

Clinical profile

Diabetes and SHT was detected in 30%.

None had previously documented CAD. (except one post CABG)

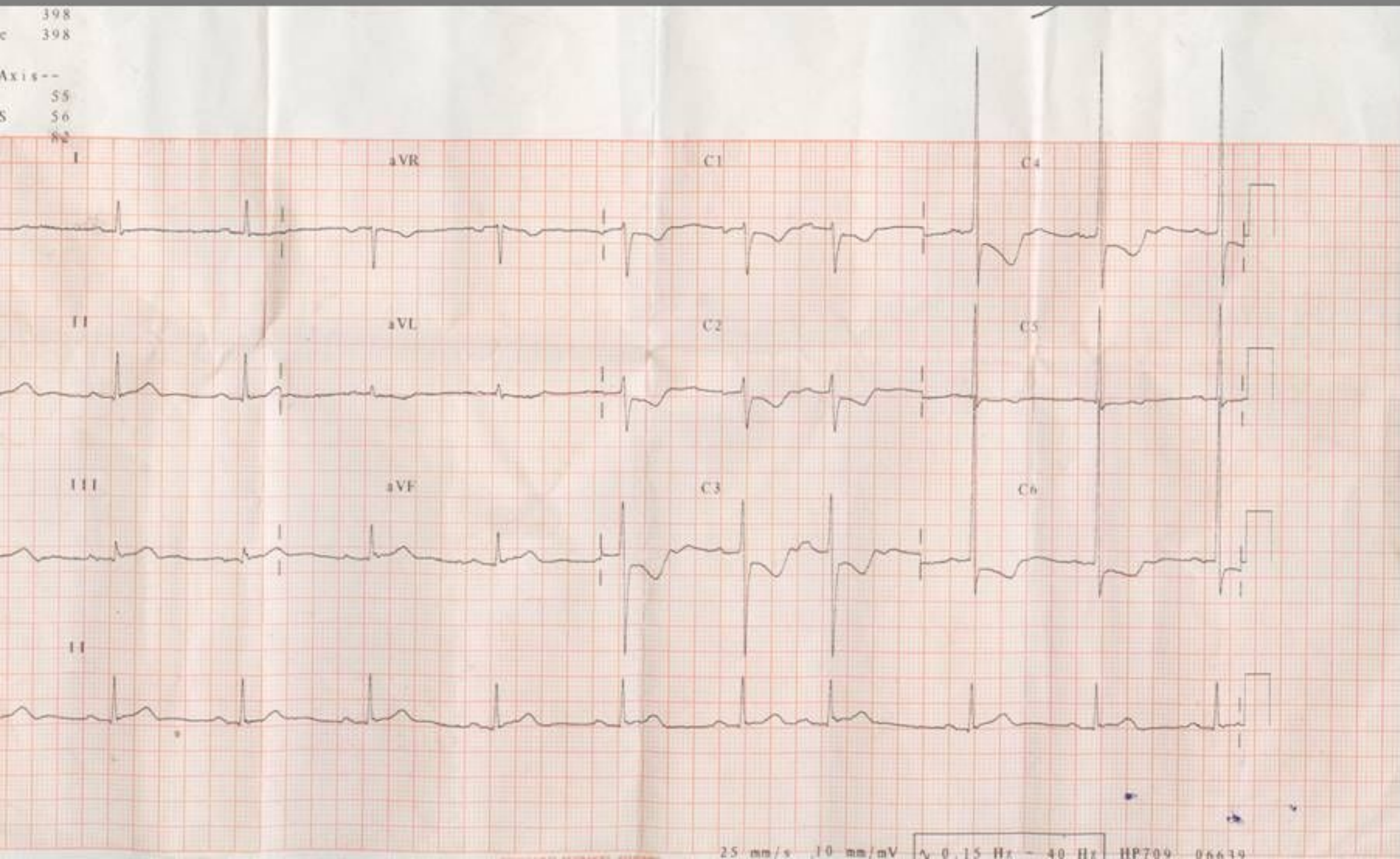
All presented with new onset ST dep or T inv >1mm at least transiently

Associated with or without angina

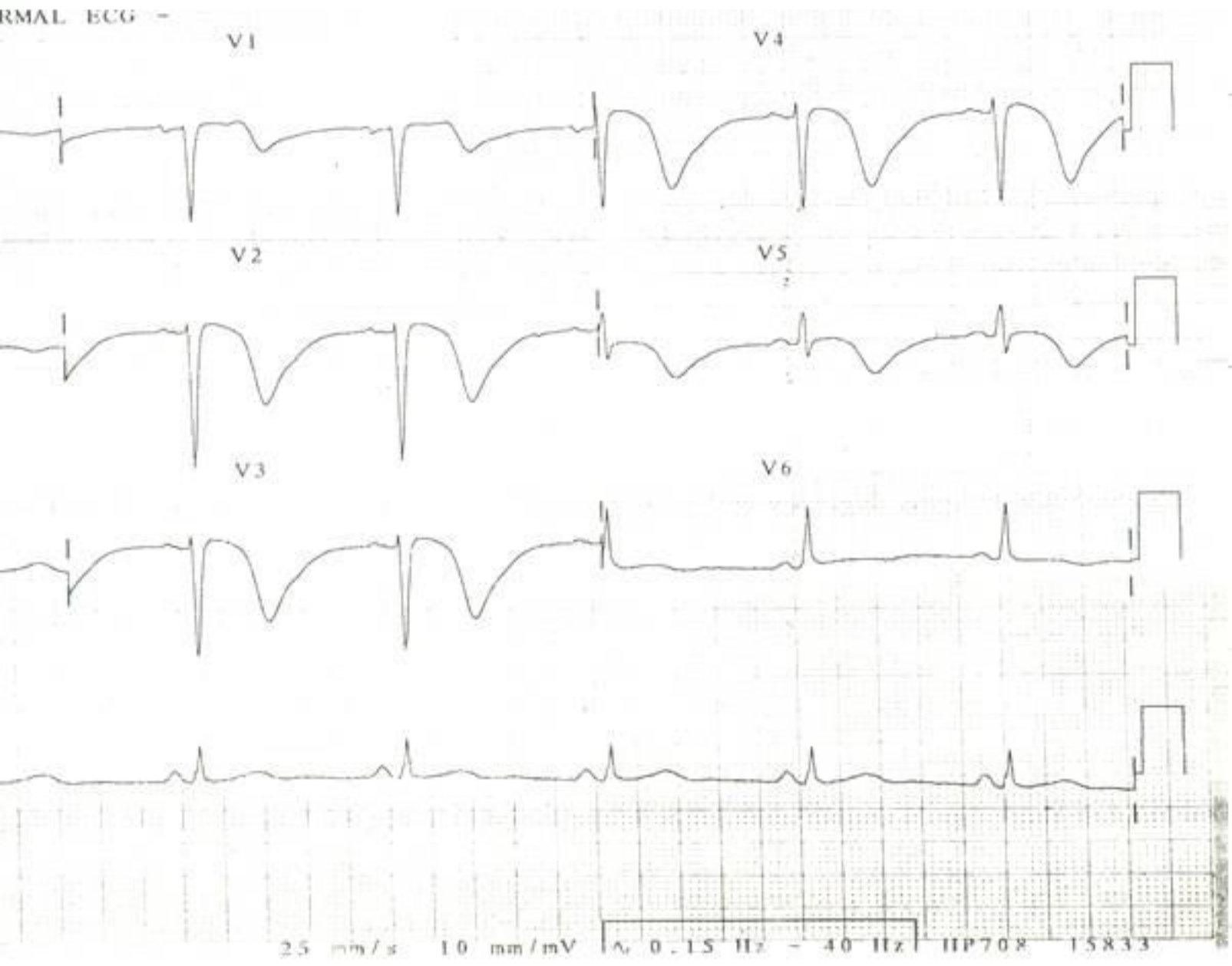
Cardiac enzymes were normal in all except minor elevation of CPK. Troponin not done

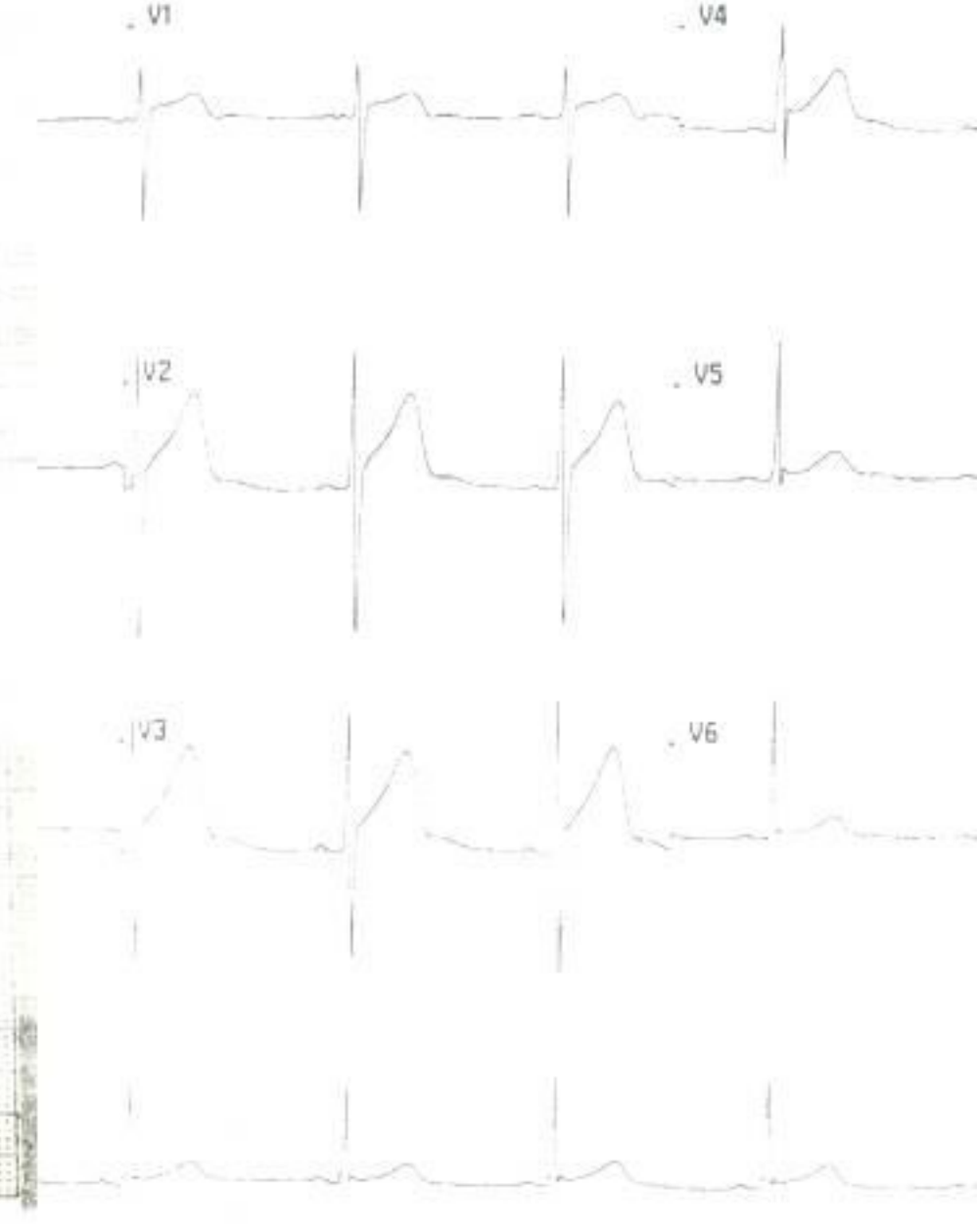
Electrolytes were normal.

ECG Presentations



RMAL ECG -


V1
V4
V2
V5
V3
V6
25 mm/s 10 mm/mV 0.15 Hz - 40 Hz HP708 15833


V1
V4
V2
V5
V3
V6

Echo cardiogram

2 patients showed RWMA

Performed mean time of 30hrs(12-72h)

Global LV function was normal

No other structural valve or myocardial disease were detected

TMT

Was done in 7 patients

3 were reported positive

coronary angiogram

Revealed normal CAG in 12.

Two patients showed non flow limiting *discrete eccentric irregular* lesions in proximal LAD.

Spontaneous *spasm of RCA* was seen in 2 patients .

Provocative tests were not done.

3 patients showed positive stress test.

Management

Heparin

IV NTG,

Beta blockers,

Aspirin as indicated.

All responded well with complete cessation of angina and reversal of ECG changes or both.

Typical scenario of a patient

56y M

Post operative setting

No obvious coronary risk factors

Cardiology fitness cleared

Episodes of hypotension

ECG

Silent non specific ST dep , Runs of VPD

Settles down

Echo normal study , CAG normal , TMT normal

How do you refer to this presentation

Chronology of Symptoms and ECG changes

Mean duration of angina was 12mts(5-60mts)

The mean duration of ST depression 20mts(5mts-2hrs)

All patients were totally symptom free by 24h

Duration of CCU stay 6hrs-36h

Follow up

All patients were followed up for 6 months. Two patients developed similar recurrent ischemic events and one developed AMI.

How to call these events ?

Unstable angina?

NSTEMI?

Episodes of silent ischemia?

Therapeutic implication

We are compelled to make a diagnosis of Unstable angina
Every time there is transient ECG changes in ECG and associated with Typical or atypical angina

And in the process a patients receive a full protocol of IV anticoagulation (Heparin or LMWH)

As it's likely many of the episodes unrelated to thrombus and are pure Mechanical events in the coronary circulation

Economic issue pertinent

We believe,

These ischemic episodes may be equivalent to the TIA's of the cerebral circulation occurring in coronary micro or macro circulation

What is the evidence for our belief ?

Currently the evidence is only sparse

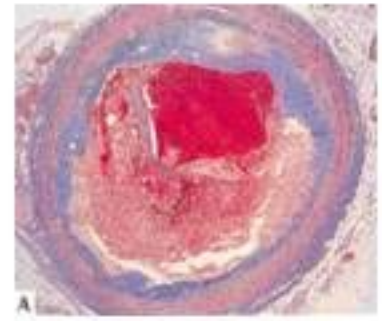
Clinical recognition is largely speculative

But pathogenetic concepts cannot be proved by randomised evidence

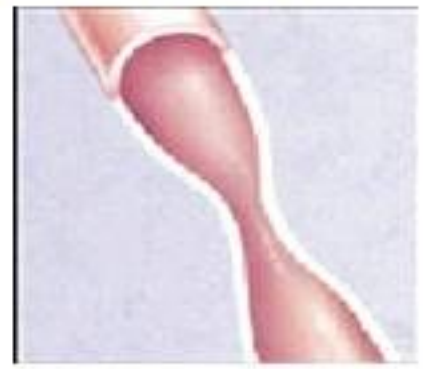
Our study has provided a question

But we are obliged to give some explanation for these events which are real

Is it a transient thrombus ?

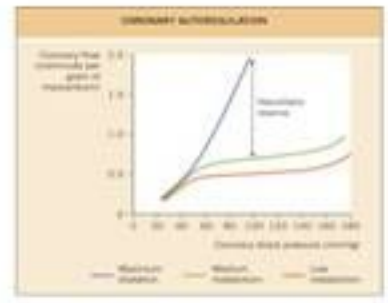


Is it a spasm?



Is it a embolus?

Is it simple coronary hypotension ?



Ischemic response of heart & brain

Heart

Has mechanical & electrical function

Brain

Has no mechanical function but rich cognitive function

Unlike brain heart cannot feel the ischemia through cognition.

It sends ***subtle signals*** one has to recognize

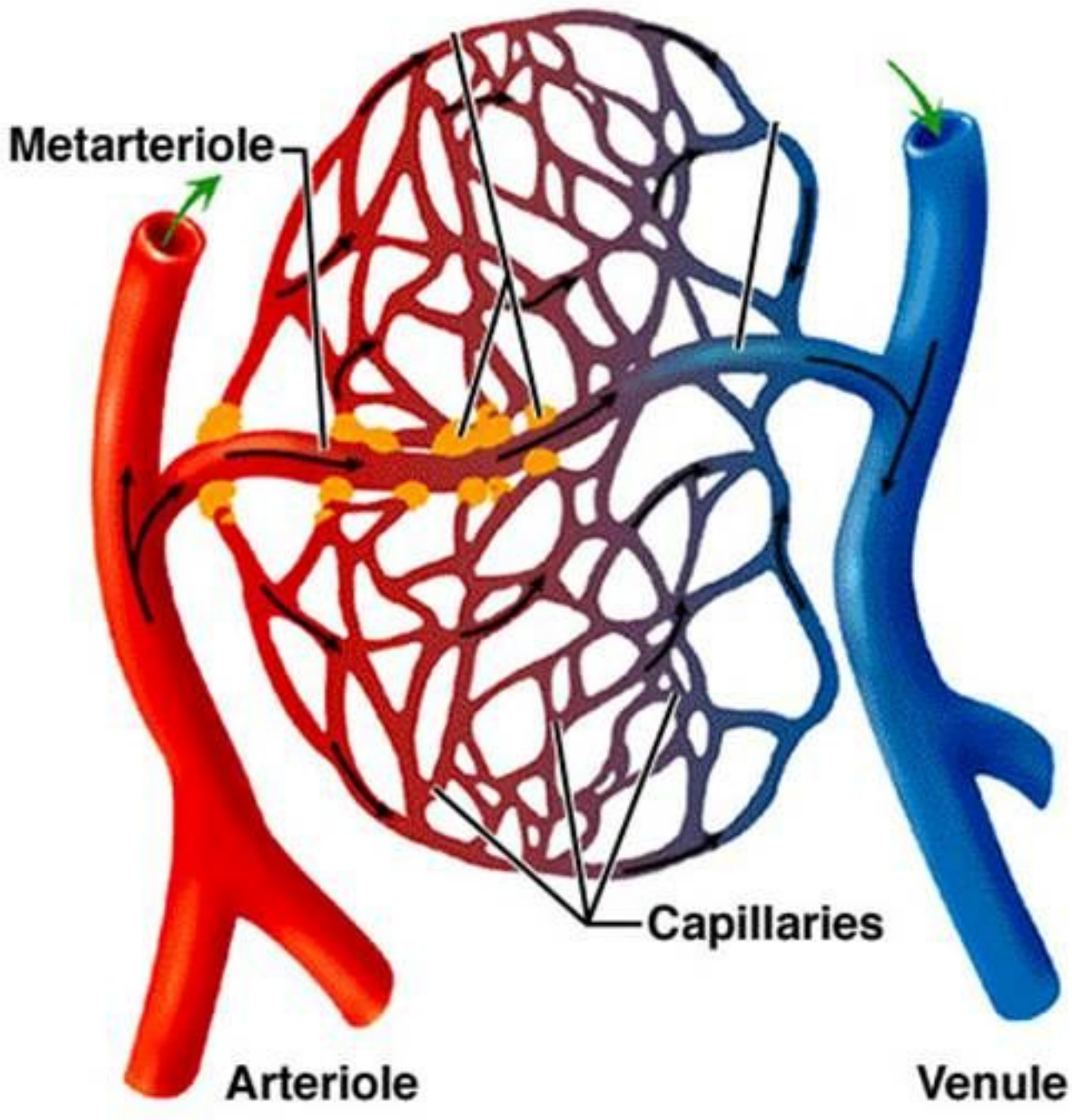
Proposed mechanisms of cardiac TIA s

Low flow TIA

&

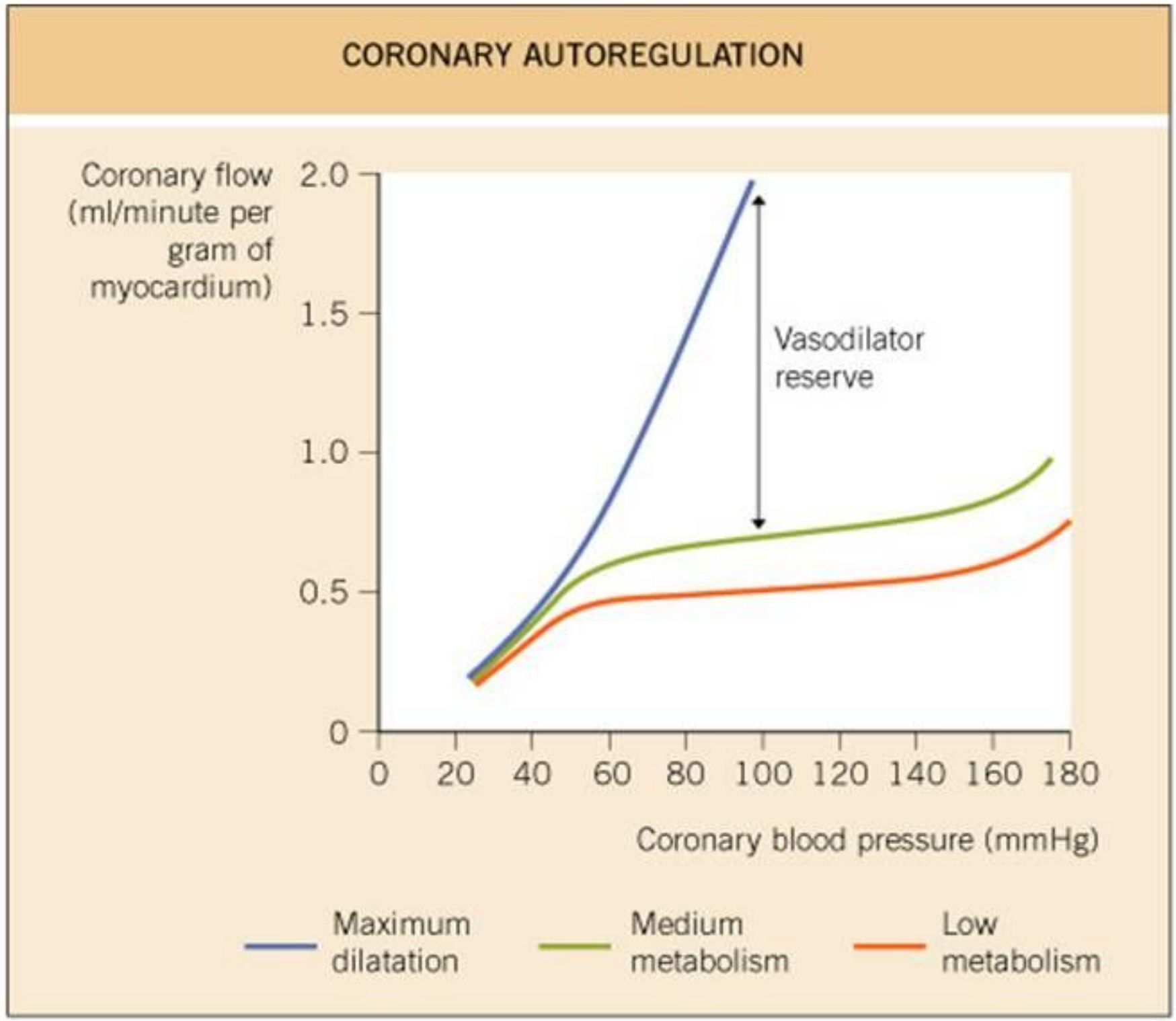
Auto-regulation failure

Mechanism of low flow TIA



	Coronary auto regulation
Pressure /flow mediated	<40mmhg with normal lumen
Neural	α,β receptors. NE vasodilation
Chemical	Hypoxia, H+, K, lactate, Adenosine

CORONARY AUTOREGULATION



Lessons from Anesthesia

Drummond JC.: The lower limit of autoregulation: Time to reverse our thinking?. *Anesthesiology 1997*

Drugs given by the anesthesiologist may affect coronary resistance

If blood pressure is low enough autoregulation fails and coronary flow becomes pressure-dependent.

This is *more pronounced in patients with CAD* and fixed lesion

Inadequate blood pressure compromise coronary blood flow to result in myocardial ischemia manifested in regional wall motion abnormalities, electrocardiographic changes and dysrhythmias

Embolic TIA s in heart?

Current Perspectives

Recognition of the Importance of Embolization in Atherosclerotic Vascular Disease

Eric J. Topol, MD Jay S. Yadav, MD

Disturbingly and unexpectedly high rate of arterial embolization in certain atherosclerotic conditions and to review the promise of newer therapeutics or devices to reduce the risk or ameliorate the sequelae of embolization

Source of embolus

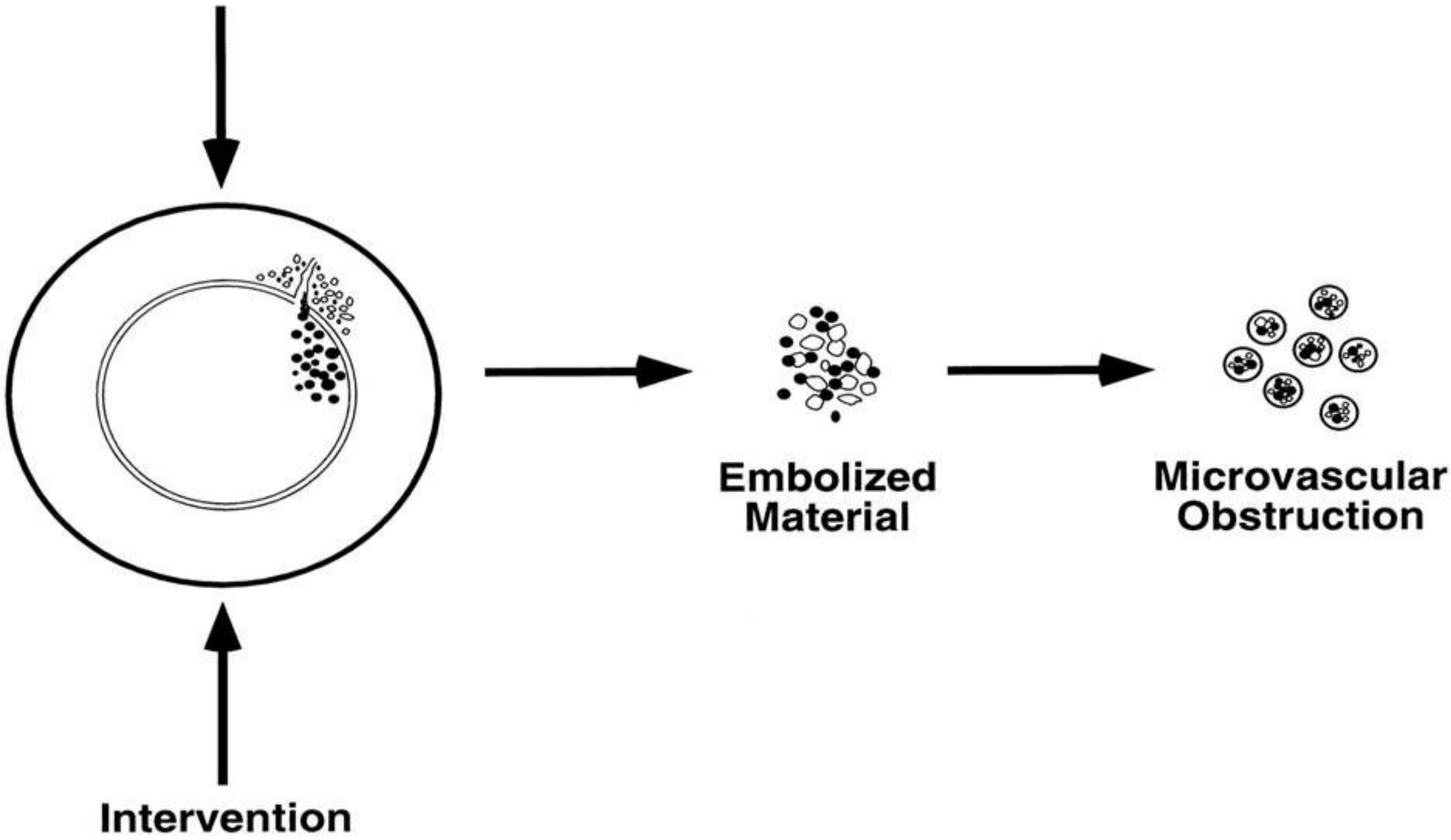
Platelet

Fibrin

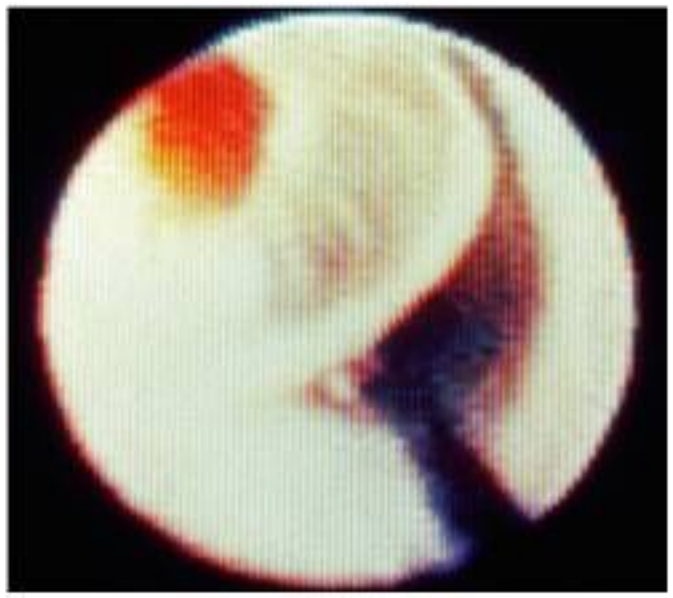
cholesterol

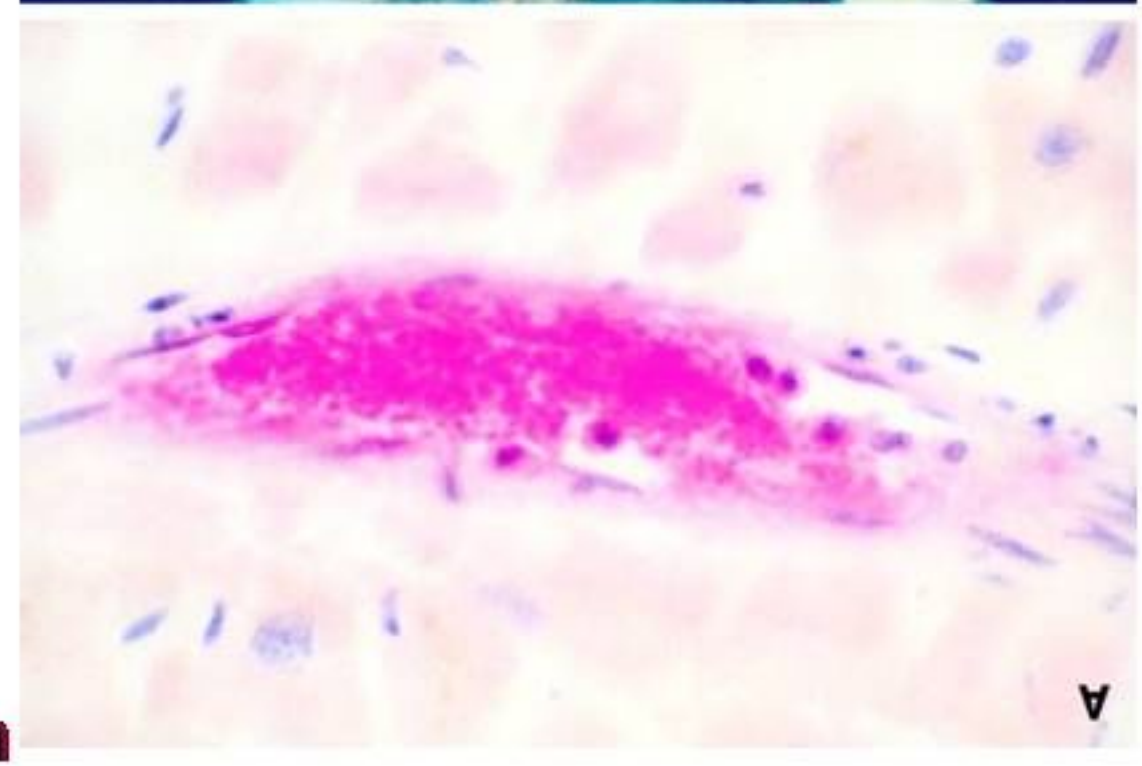
Atheromatous debri

Coronary embolism -proposed scheme



Coronary embolism - Histological documentation





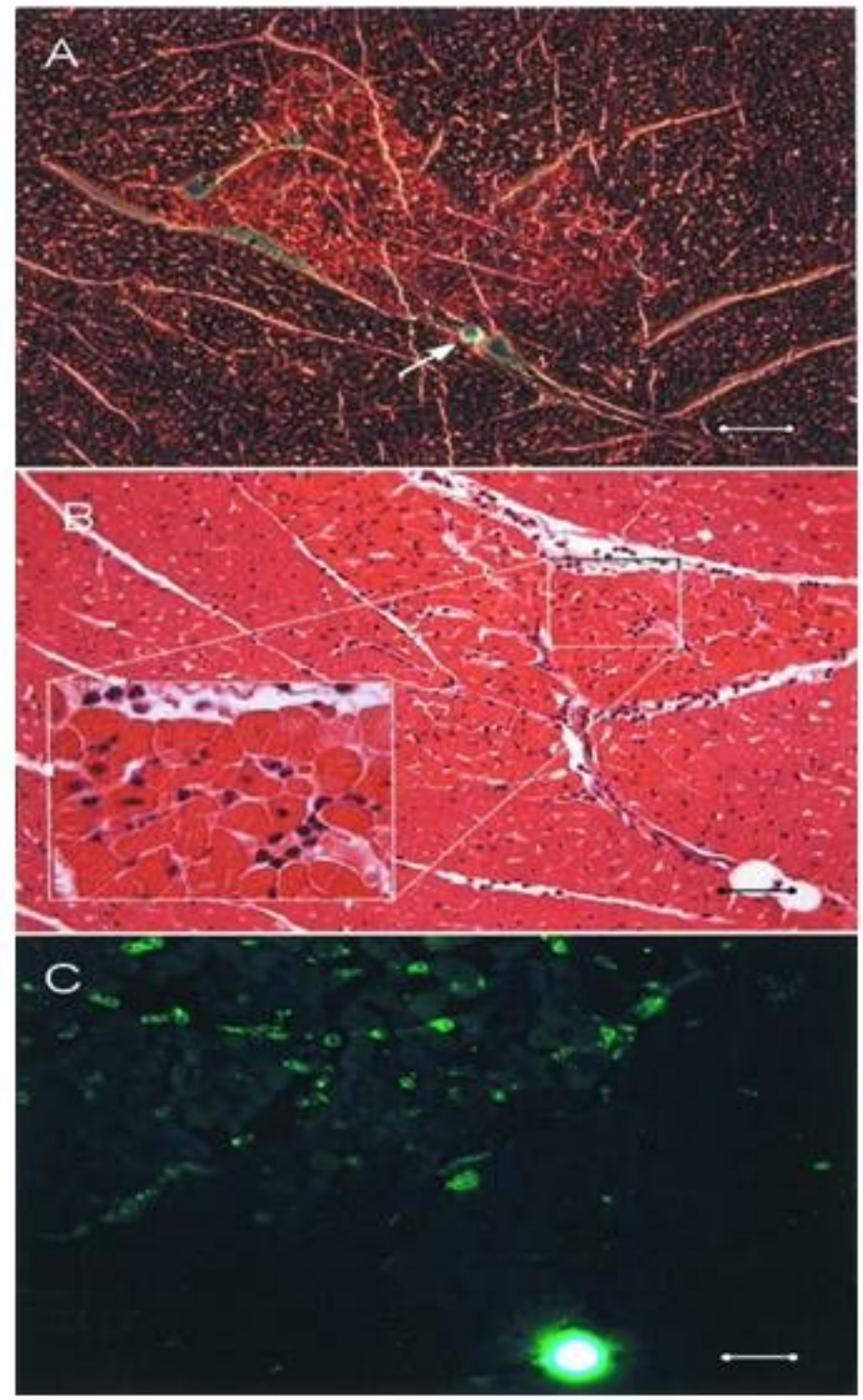
Embolus and micro vascular obstruction

Coronary embolism Effect on myocardium

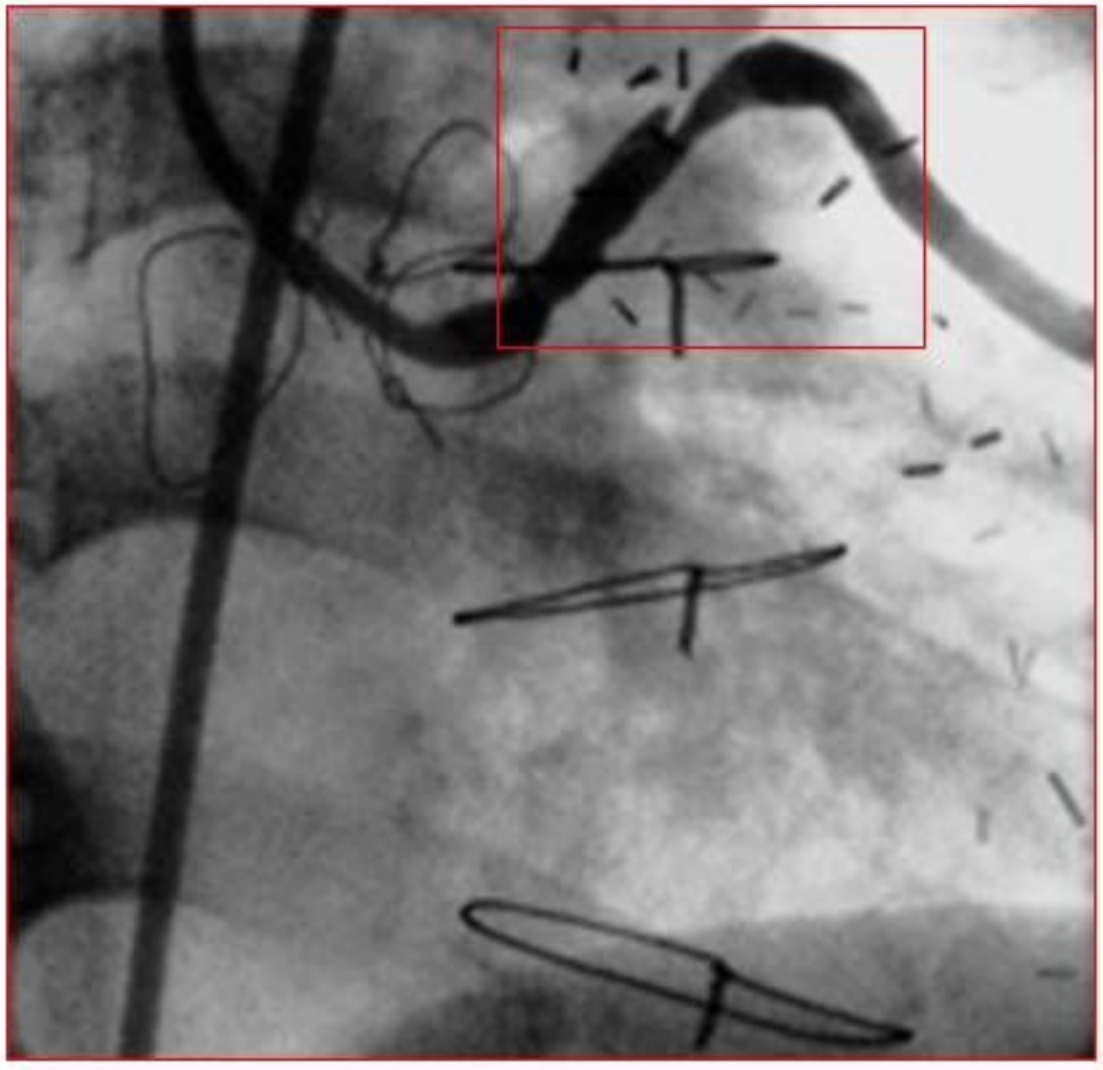
Documented by phase contrast microscopy In myocardial necrosis

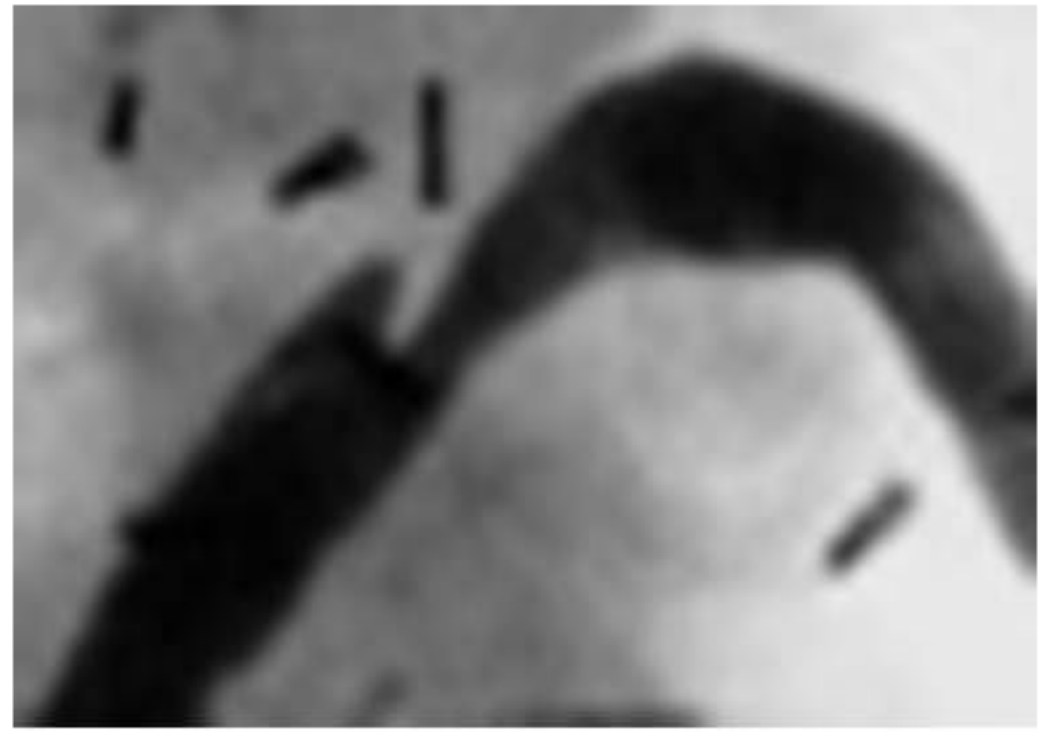
Infiltration of inflammatory cells

Fluorescence immunohistochemical staining of macrophages/monocytes



Emboli from degenerated venous graft





TIAs in cath lab ?

Transient ischemia and transient enzyme elevation reported after PCI, CABG

Now

Troponin levels are found elevated even following routine CAG

*Role of Distal protective devices

Conclusion

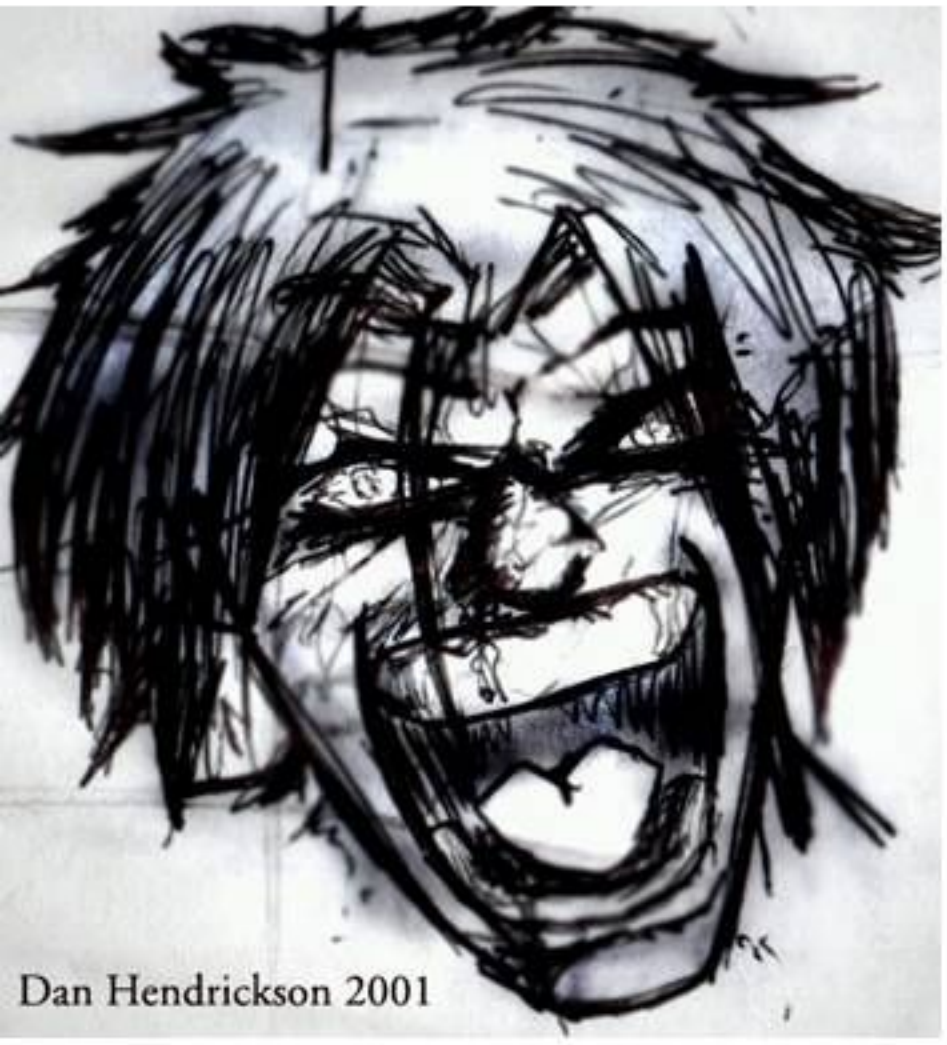
TIAs *may not be an not* exclusive phenomenon confining to cerebral circulation

Many of the episodes of clinical ACS/UA could be *TIA equivalents* of brain

Mostly occur in the stetting of CAD, Post PCI, or CABG

Rarely occur in normal heart especially peri-operative setting

These episodes likely to have *different therapeutic and economic* implications in the management of ACS



“A person with the new idea is a crank until it’s proven”

Thank you