CHEST PAIN and TROPONIN
HYPERTENSION

Waitemata District Health Board
Te Wai Awhina

AUCKLAND HEART GROUP

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OCT 2011

Patient 1

75 year old male patient
Retired
Very active- enjoys kite fishing
mows 3 family members lawns

PM/SH:
Benign prostatic hypertrophy

Medication:
Doxazosin 2mg/d

Family history:
Older brother who died from an acute MI at age 78 years.

Acute Problem

Episode of chest pain while mowing his lawn 2 weeks ago

- Central
- Heavy
- Radiated to his jaw and throat
- Came on gradually and he needed to stop mowing
- Resolved with rest

Opening of the RWC

- Walked up a steep ramp- had to stop due to similar symptoms
- Settled after 5 min
- Able to watch the fireworks

3am-woke with similar pain

- Got up and made a cup of tea
- Lasted 20 min
- Went back to sleep
- Decided he would see his GP in the morning

Examination

- Well
- Excited $\rightarrow$ Tickets to the Australia vs Italy game at North Harbour

- BP=135/80mmHg
- PR=70bpm

- JVP-not raised
- No carotid bruits

- CVS-normal S1 and S2, no murmurs
- Chest-no failure
- Peripheries- no PVD, no peripheral oedema

ECG

Rx:
Dx:

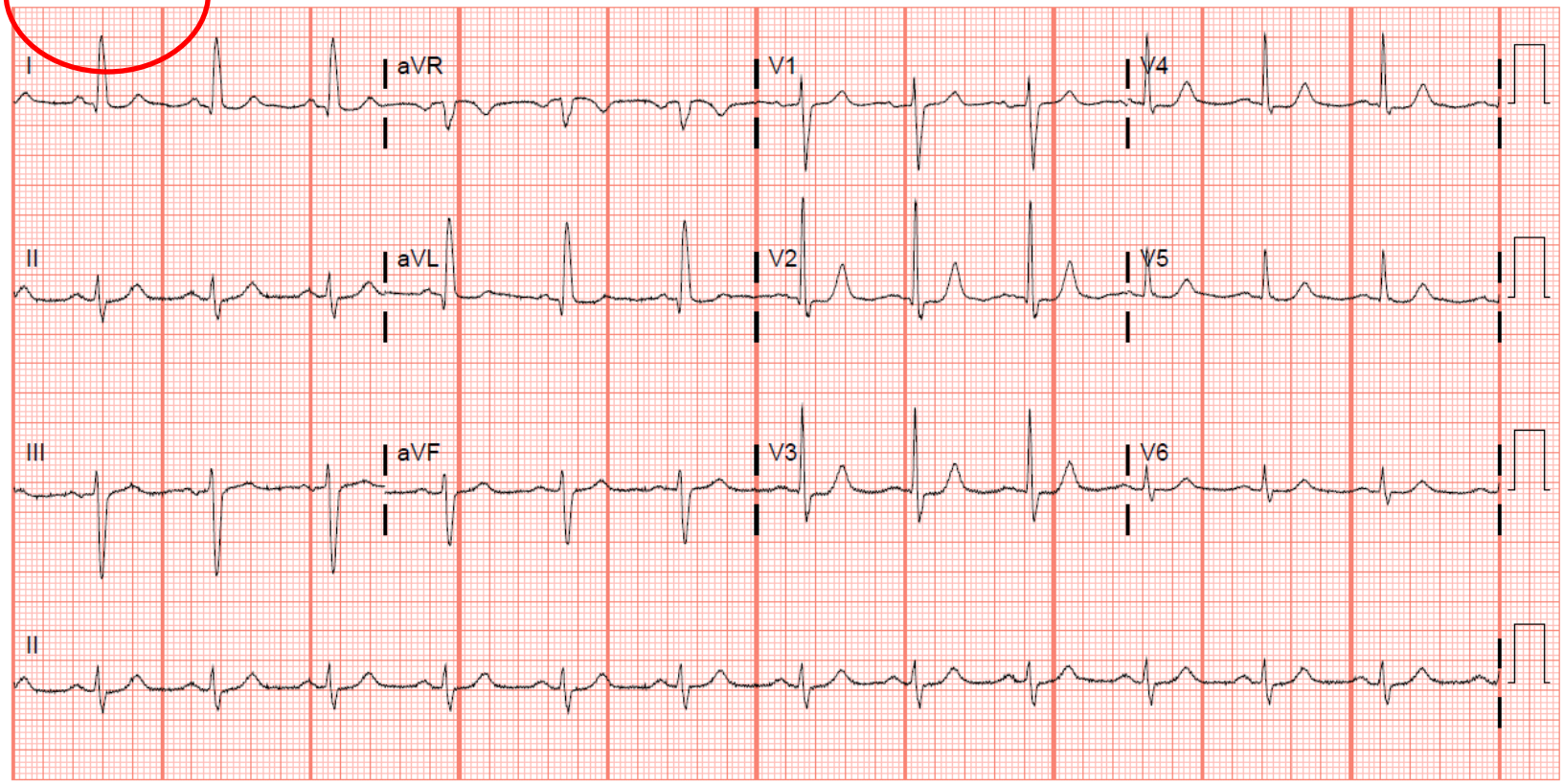
Room CCU 4
Tech

Rate	76
PR	172
QRSD	104
QT	412
QTc	463
--AXIS--	
P	32
QRS	-34
T	64

Req MD:
Field1:
Field2:
Field3:
Field4:

- ABNORMAL ECG -

Confirmed By: 31/08/2011 20:43:52



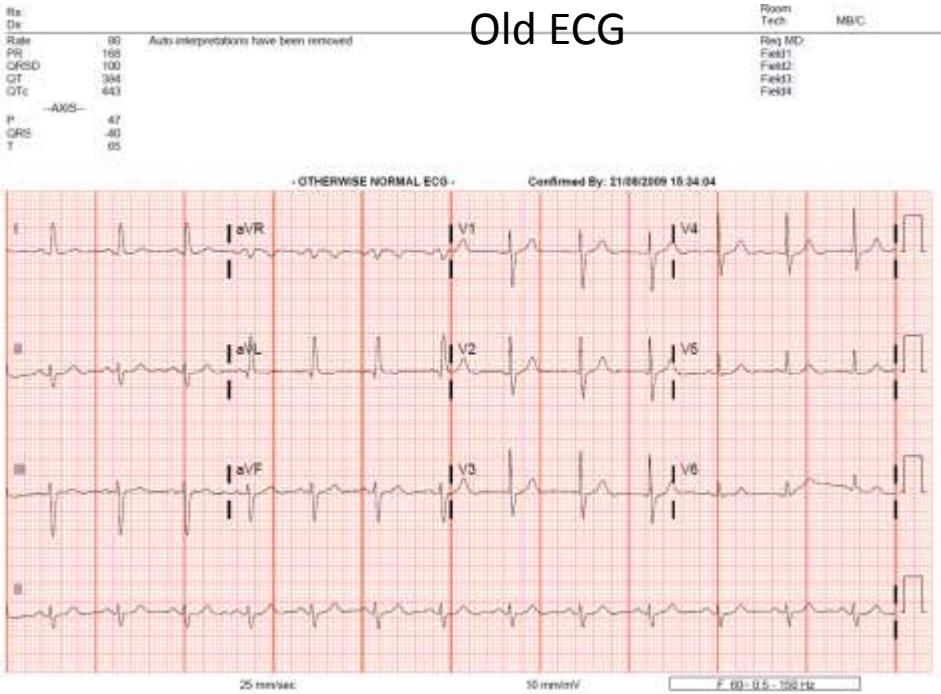
Management options

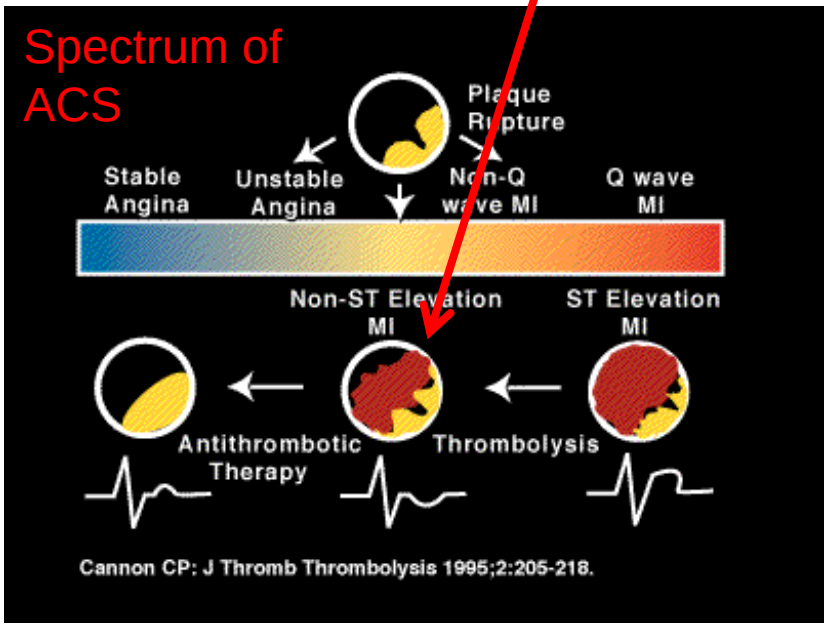
1. Start aspirin, B-Blocker, GTN spray
   Private referral to be seen urgently next week

   Tickets to the Australia vs Italy game

2. Admit to hospital- possible acute coronary syndrome

Old ECG





Non-ST elevation MI

Ruptured fibrous cap

Final Diagnosis

- New onset angina
- Prolonged rest angina
- Probably got ST depression on ECG

Acute Coronary Syndrome

Unstable angina

NSTEMI

Admitted

Mildly raised troponin

Clexane, aspirin, Clopidogrel, statin, B-Blocker

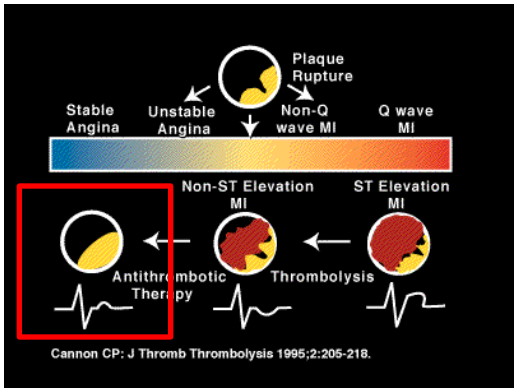
In-patient CABG

Summary One

Angina for outpatient investigation:

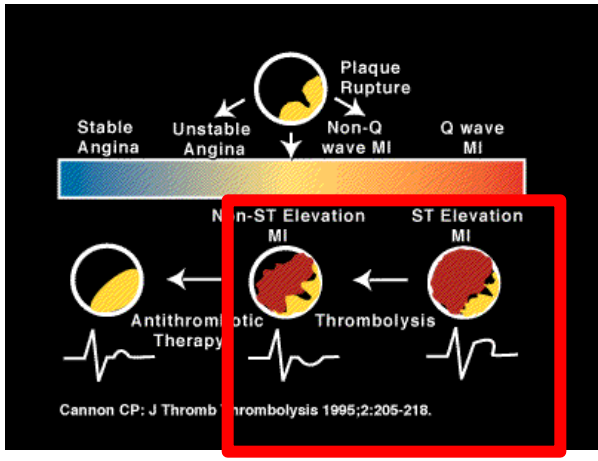
- Angina occuring predictably on exertion
- Never at rest
- Not progressive, no new ECG changes

→ Start aspirin, B-Blocker, GTN spray and refer cardiology



Angina requiring in-patient Evaluation:

- New-onset angina- occurring at a low threshold-class III
- Crescendo or increasing angina- more frequent, of longer duration or lower threshold of onset i.e. ↑ to class III
- Rest angina – prolonged
- Angina with new ECG changes



Patient 2

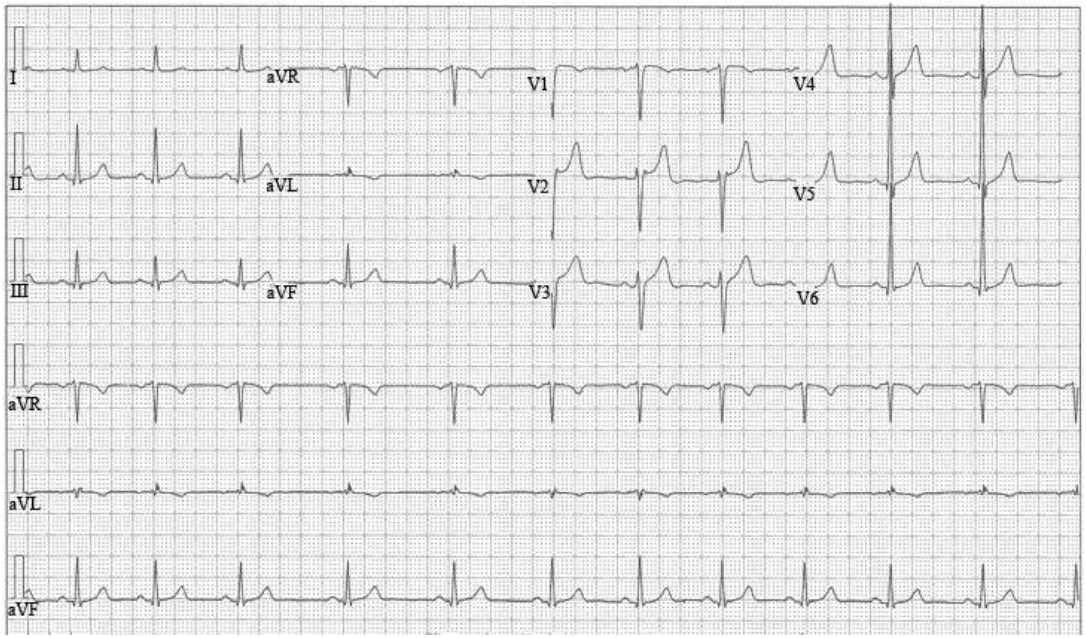
48 year old Moari lady
Presents to her GP complaining of breathlessness and vague chest tightness.
Symptoms mostly at rest and last for days- NOT TYPICAL ANGINA

Smoker, no asthma
Hypertensive-POORLY CONTROLLED.
BMI 32, NOT DIABETIC
No past hx of IHD, no family hx of IHD

Examination: BP 180/100mmHg, Pr 75bpm, JVP-N
S1 S2, S4; Chest-no crackles, no wheeze

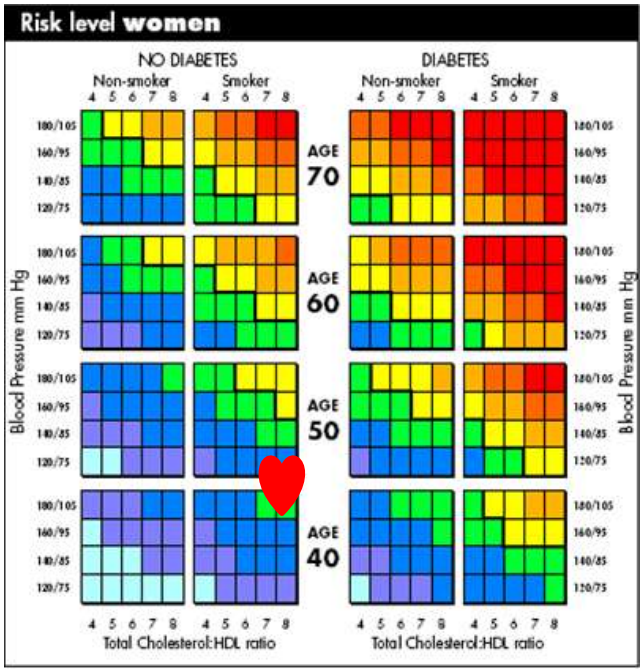
Meds:
Inhibace 0.5mg/d

ECG- ↑ LV voltages
no new ischaemic changes



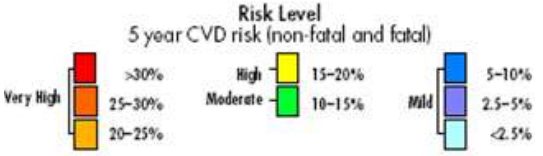
ASSESSMENT

- Breathlessness, probably 2° to poorly controlled hypertension
- Acute coronary syndrome- unlikely
- Multiple risk factors-?? Coronary disease



TC-6.2 mmol/l
HDL-0.65 mmol/l
LDL- 5.1 mmol/l
TG- 3.0 mmol/l
Ratio- 9.5

Maori



Management and Investigations

Inhibace 2.5mg/d $\rightarrow\uparrow$ Inhibace Plus
Plan to add Felodipine – target BP 130/80mmHg.
Lipitor-40mg/d

Bloods- FBC, U+E, BNP, troponin I

Pt forgot about bloods

2 weeks later found the slip- **Trop I 236 (N<40),** nt-BNP-normal

GP called pt- states she feels perfect-much better on Inhibace Plus

GP calls on-call Med Reg to admit pt-?NSTEMI

Reg refuses to take pt and suggests repeating troponin

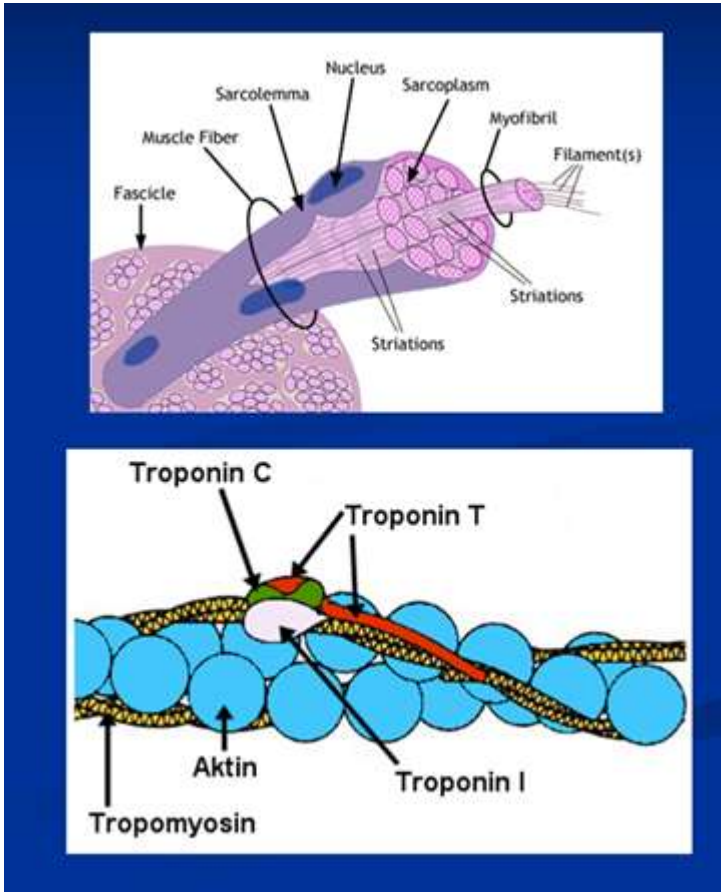
GP- then calls me $\rightarrow$ admission to cardiology ward

hs Troponin

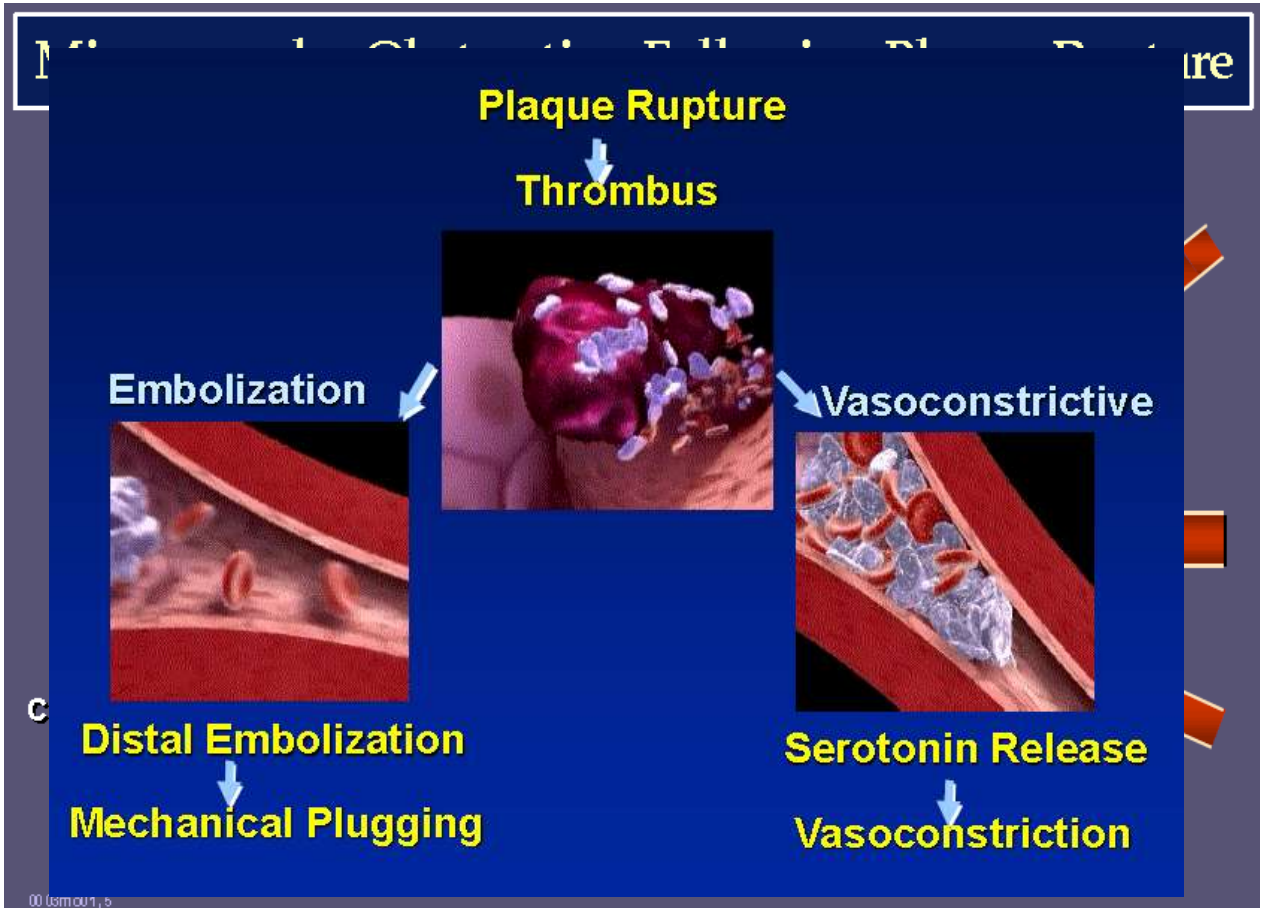
New more sensitive and reliable assays-10 x more sensitive.

Reliably detect changes in concentration of the marker above the 99th percentile for a normal population

- Regulatory protein of the thin actin filaments
- Structural and cytosolic pool
- Early release is thought to be from the cytosol
- Highly specific and sensitive marker for myocardial injury



IMPLICATIONS OF A RAISED TROPONIN:

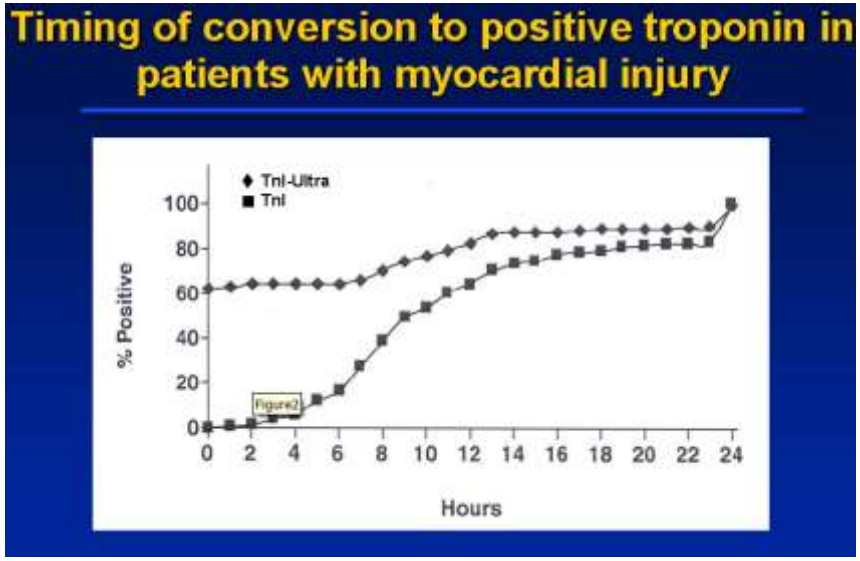


↑ troponin in a patient with chest pain or SOB → MI (NSTEMI)

Patients do better with invasive mx- coronary angiography and revascularisation.

hs Troponin

New assay- POSITIVE EARLIER



Assay is very sensitive and BUT **not specific for acute MI**

Conditions that strain the myocardium- can cause a rise in troponin e.g. rapid AF, congestive heart failure, poorly controlled hypertension, sepsis.

We need a new approach.....we need to go back to the definition of acute MI.

Universal Definition of Acute Myocardial Infarction

The ESC-ACC-AHA-WHF Criteria

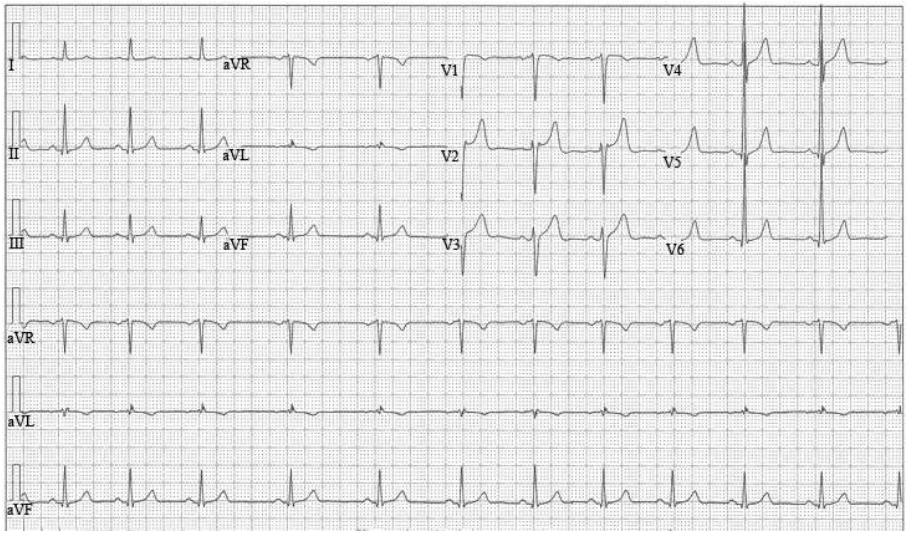
Detection of *rise and/or fall* of cardiac biomarkers (preferably troponin) with *at least one value above the 99th percentile* of the upper reference limit together with evidence of ischemia with at least one of the following

- Symptoms of ischemia
- ECG changes of new ischemia (new ST-T changes or new LBBB)
- Development of pathologic Q waves
- Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality

Back to patient…

She looked well, wasn't SOB, no CP
BP 160/100mmHg
4th heart sound, no heart failure

Trop I repeated- 210, 236...3 mildly elevated levels



So by definition- no rise and fall of troponin, no symptoms of infarction, no new ischaemic ECG changes.....therefore probably not infarction.....BUT

She is young but has lots of CV Risk factors
Could the SOB be a chest pain equivalent AND....
ECG is abnormal
Therefore needs to be admitted and sorted out

Management....

CORONARY ANGIOGRAPHY:
normal

Intensified antihypertensive therapy

needs to loose weight, restrict salt....get rid of the junk food

compliance.....

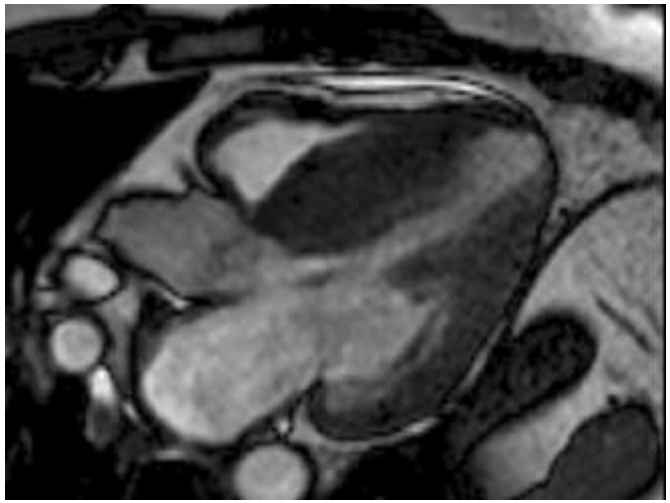
Meds: ADDED in a dihydropyridine Ca blocker- amlodipine 5mg/d

Inhibace plus 1 daily
Amlodipine 5mg/d
BP-target 130/80

What about the raised troponin

Contrast Cardiac MRI- ? SCAR, myocarditis etc.

Massive LVH ....probably from hypertension
BUT...no scar or prior MI



- **LVH → occult subendocardial ischemia → CYTOSOLIC AND STRUCTURAL TROPONIN RELEASE**
- **↑ muscle mass → ↑oxygen demand, decreased flow reserve (due to remodelled coronary microcirculation)**

Left ventricular hypertrophy

- 74 consecutive patients without myocardial ischemia $\rightarrow$ routine echocardiography.
- 7of 25 patients in the tertile with the greatest LV mass $\rightarrow \uparrow$cTnI.
- 1 patient in the intermediate range, and none of patients in the lowest tertile had an elevated troponin level

SUMMARY 2

CLINICAL SITUATIONS ASSOCIATED WITH A RAISED TROPONIN

1. ACS (typical rise and fall, absolute trop levels usually higher)- Type I MI

2. Transient elevation of troponin from intermittent myocardial strain
   e.g. Rapid AF, Pulmonary embolus, sepsis, acute heart failure- Type II MI

3. Persistent elevated hs troponin (low grade) e.g. LVH (poorly controlled hypertension, aortic stenosis, congestive heart failure

Marathon runners- >50% have minor elevation, 10% significantly raised levels
Younger age and marathon inexperience were associated with higher levels.
No association with conventional risk factors and marathon duration.

Troponin elevation in the absence of CAD

ADVERSE PROGNOSIS

e.g. Acute Heart failure with raised trop worse than HF without raised trop.

MARKER OF DISEASE SEVERITY

- Valvular heart disease,
- Indicator of hypertension control
- Prognosis in heart failure

DIFFICULT HYPERTENSION

Classification of Hypertension- JNC VII

BP Classification	Systolic BP, mm Hg*		Diastolic BP, mm Hg*
Normal	<120	and	<80
Prehypertension	120-139	or	80-89
Stage 1 hypertension	140-159	or	90-99
Stage 2 hypertension	≥160	or	≥100

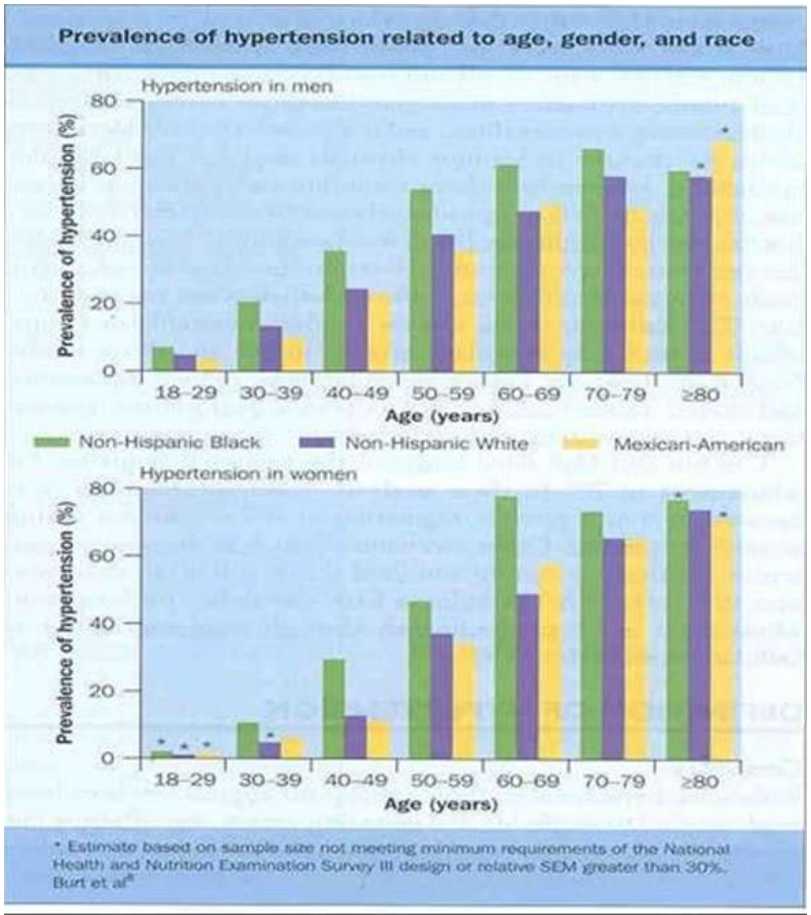
2 x risk of developing true hypertension

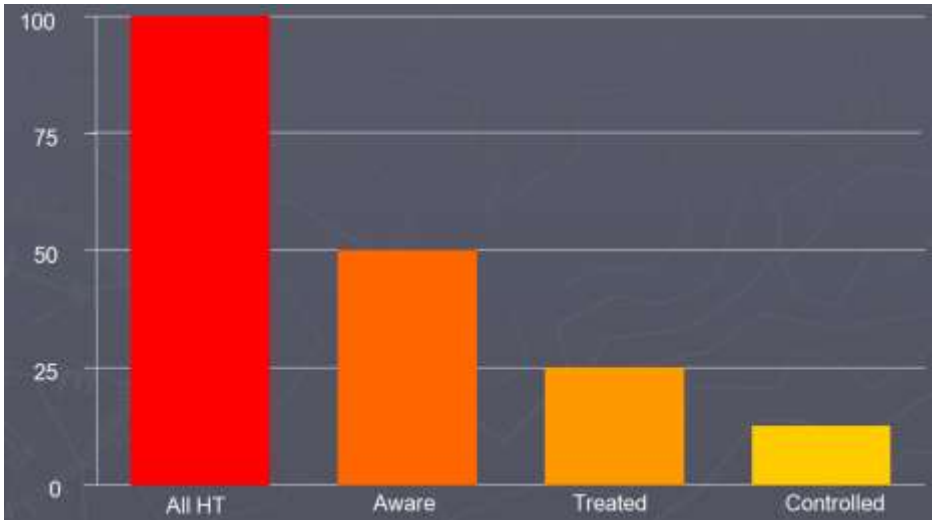
BP MEASUREMENTS
- measured seated
- mean of 2 or BP recordings
- at least 2 visits

Epidemiology

Size of problem:

- 20% of adults in Western societies have hypertension
- Accounts for 1 in 6 deaths
- 95% is essential hypertension





Cruickshank et al. J Hypertens 2001

Benefits of adequately treating Hypertension (JNC VII)

Trials → 35-40% mean reduction in stroke
20-25% reduction in MI
50% reduction in heart failure

Pt with BP 159/95 mmHg (stage 1)
on treatment for 10 yrs
12mmHg ↓
prevent 1 death for every 11 pts treated

CASE

GP Referral

63 year old female patient, retired secretary, married

- Known coronary artery disease and peripheral vascular disease
- CV Risks: longstanding mild hypertension
  treated dyslipidemia
  ex-smoker

ACUTE PROBLEM:
- acceleration of hypertension x 6months
- poor control despite 3 agents
- more breathless- struggling to finish 18 holes of golf

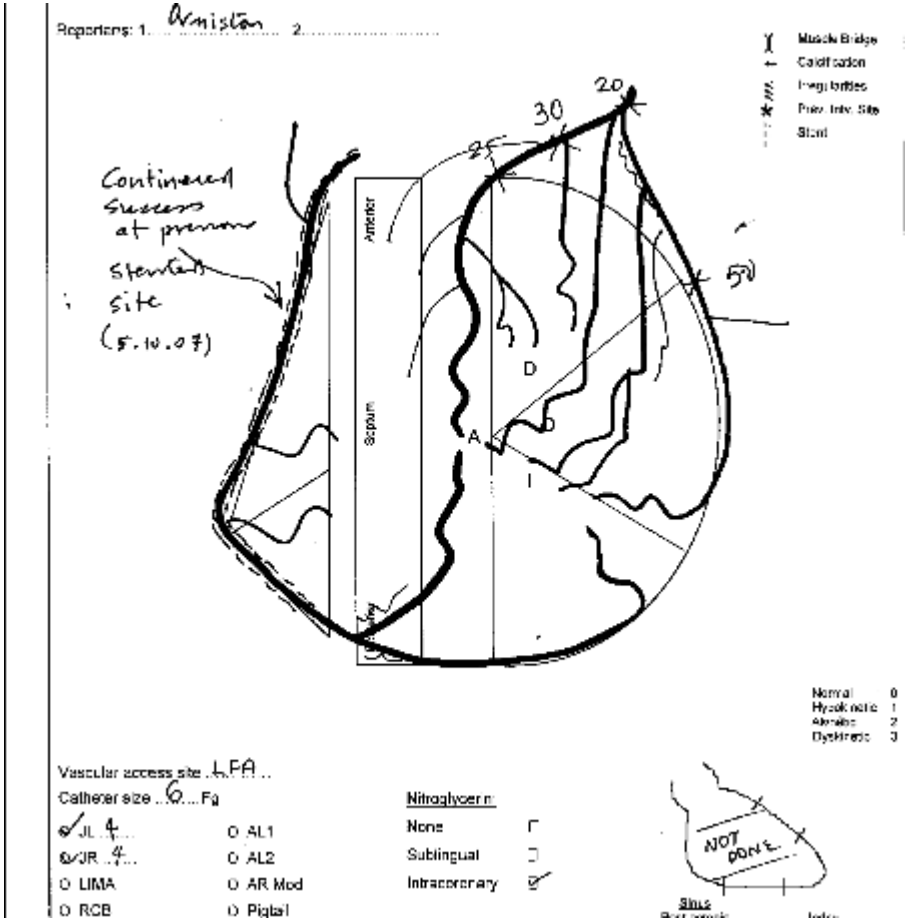
Medical History

Stable Coronary Artery Disease at present:

- Unstable angina- PCI (2000)
- NSTEMI-RCA PCI (2002)
- NSTEMI – severe proximal RCA-DES (2006)

Peripheral Vascular Disease:

PCI L iliac artery 2002



Medication

Cilazapril 5mg/d
Metoprolol 23.75mg/d
Amlodipine 5mg/d
Lipitor 80mg/d- TC- 4.8mmol/l, HDL- 0.9 mmol/l, LDL- 2.8 mmol/l, TG-2.4 mmol/l

Compliance

- prefer not to be on medication; occasionally forgets
- Resveritrol, Ω 3 oil, and glucosamine- takes religiously

History of Hypertension

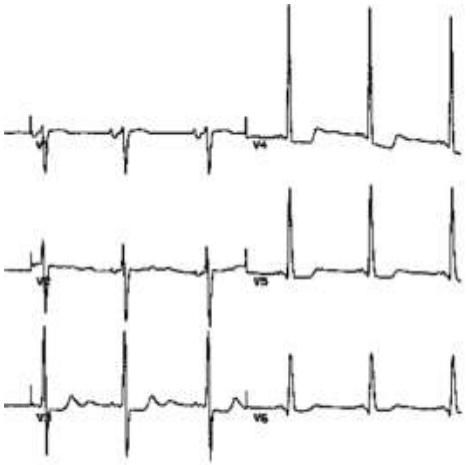
- First diagnosed at age 40 years
- Mother was a bad hypertensive- RIP stroke at 65 yrs
- Normal BP control through 3 pregnancies in her late 20's
- Enjoys salty snacks after golf- 19th hole
- Alcohol- wine every night with evening meal- 1-2 glasses
  1 or 2 drinks after golf
  No liquorice liquor
- Exercise- 2 rounds of golf per week
- Uses Voltaren 75mg before golf → bad back
- No HRT
- No hx of malignant phase ht, renal failure or heart failure

STRESS ??
- work, financial, leaky home, son divorce etc...

Examination

- Appeared a little anxious
- BMI-30
- BP-190/110mmHg; PR-90 bpm
- Fundi- arteriolar narrowing
- Heart sounds- S4, S1, S2 no CHF
- Abdomen- peri-umbilical bruit
  bruits over both femoral arteries

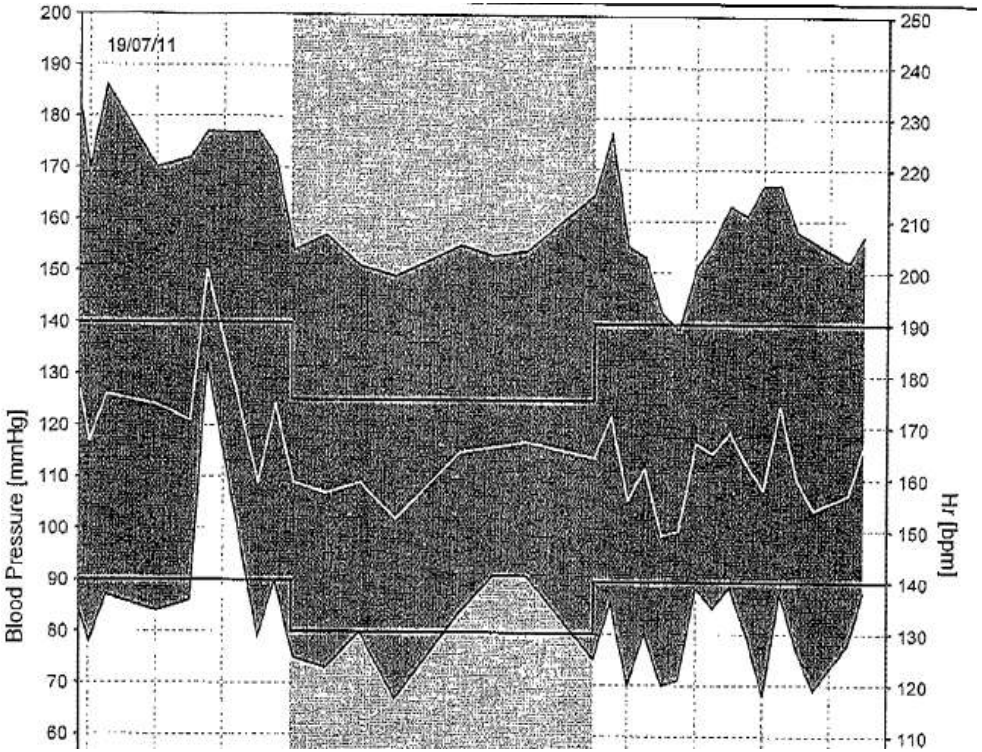
ECG- SR (p-mitrale), inferior Q waves,
↑LV voltages + lateral strain pattern



Creatinine-63umol/l, K+ 4.8mmol/l
Fasting glucose-5.8mmol/l
Urine-+proteinuria
Thyroid functions-normal

Result-24 hour ambulatory BP monitoring

- 45/48 –interpretable measurements >95%
- 24 hour mean-168/93 mmHg (normal <135/85mmHg)
- ‘non-dipper’- minimal drop (6%) in nocturnal BP measurements



Assessment

Significant hypertension- possibly resistant
- 3 anti-hypertensive agents- no diuretic, low doses of meds
- ?compliance; NSAID; salty snacks; ↑BMI

End organ involvement-LVH, CAD, PVD, proteinuria

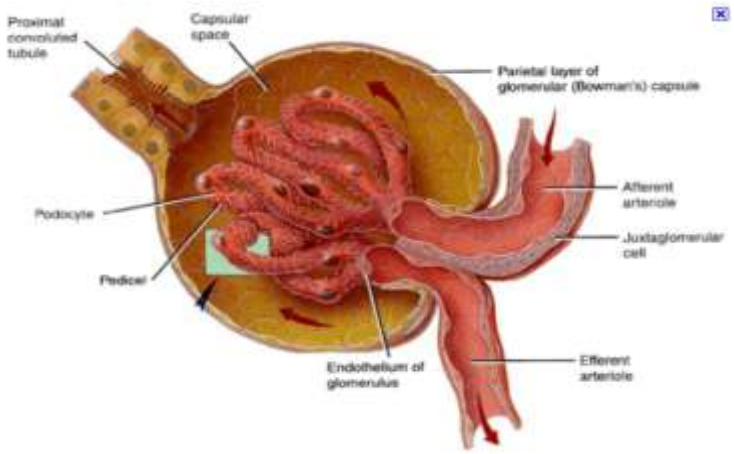
Acceleration of hypertension x 6months
- ? Secondary-RAS
- Loss of nocturnal dipping
- Para-umbilical bruit

Vasculopath- stable coronary disease
peripheral vascular disease

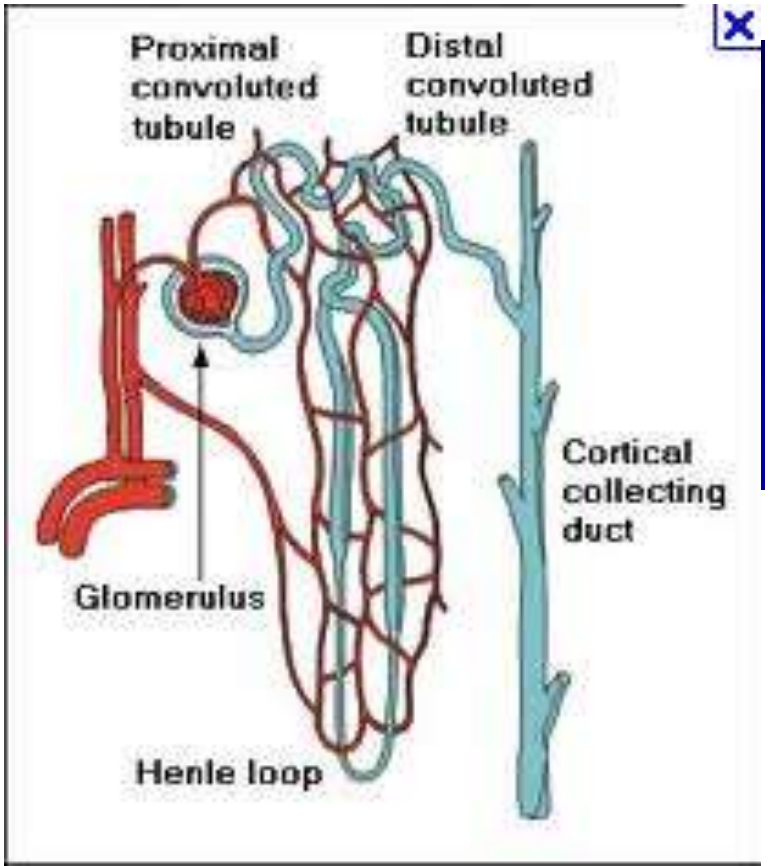
Diuretics and Hypertension

VOLUME EXPANSION:

- Vasodilators (ACE and Amlodipine)-lead to fluid retention OR volume expansion due to efferent arteriolar dilatation.

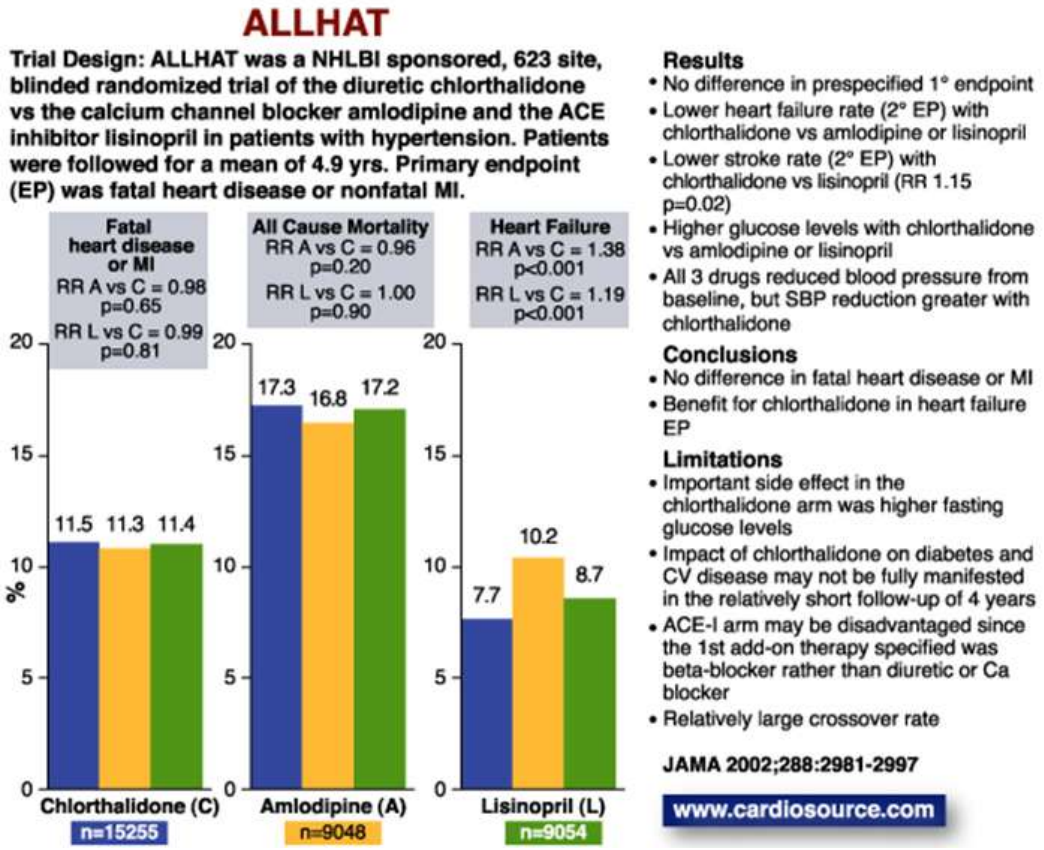


Add low dose diuretic e.g. Inhibace plus



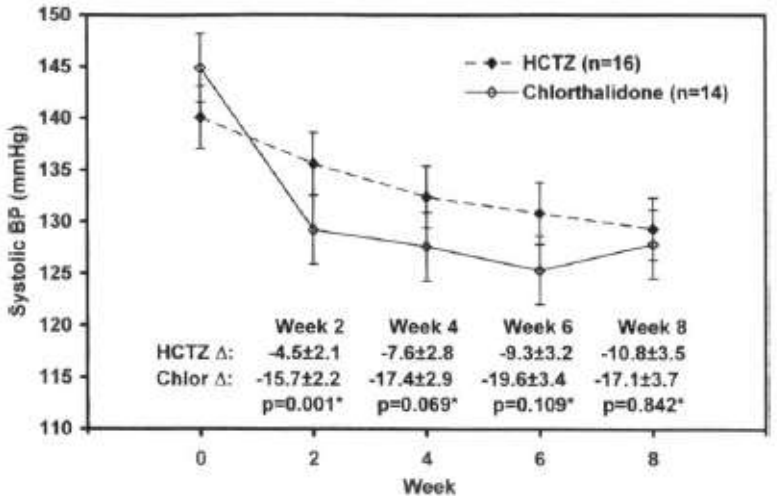
FAVORITE HYPERTENSION TRIALS

Diuretics and Hypertension



Study began 1994 x 8yrs
40 000 pts
Aim: ? Best drug to start Rx.
Results:
SBP↓-greatest with chlorthalidone
Less CHF with chlorthalidone

JNC VII- start with diuretic

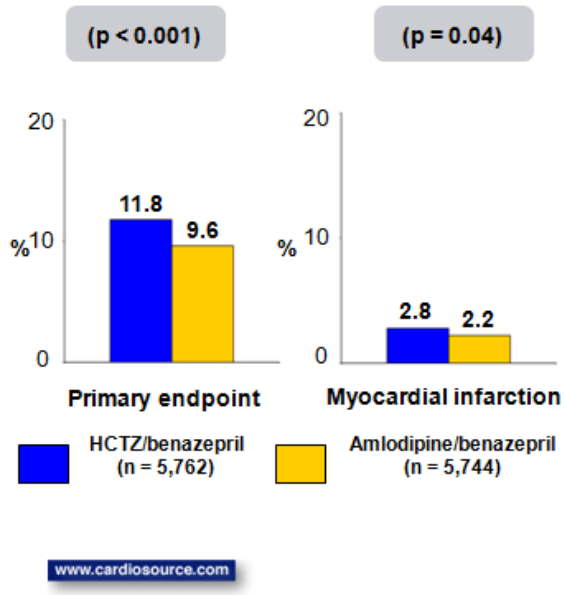


Hypertension 2006

Combination Therapy

ACCOMPLISH

Trial design: Patients with hypertension were randomized to fixed dose amlodipine / benazepril or hydrochlorothiazide (HCTZ) / benazepril. Patients were followed for 5 years. Trial was terminated early.



Results

- Primary endpoint (CV mortality, stroke, MI, revascularization, unstable angina, resuscitation from death 9.6% in amlodipine/benazepril arm, compared with 11.8% in HCTZ/benazepril arm (p < 0.001)
- MI was reduced with amlodipine/benazepril arm (p = 0.04); CV mortality and stroke, similar
- Adverse events were similar

Conclusions

- Fixed dose combination of amlodipine/benazepril better than HCTZ/benazepril in reduction in blood pressure as well as CV endpoints in patients with high-risk hypertension
- Important study—may require revision of current JNC-7 guidelines

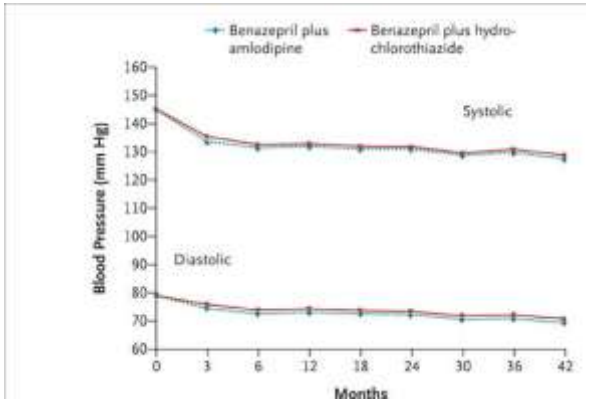
Jamerson K, et al. N Engl J Med 2008;359:2417-28

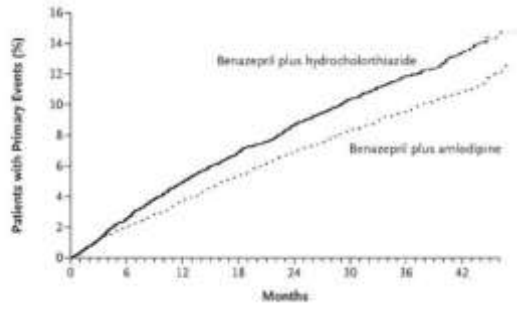
Aim: Optimal combination Rx for ht

- Compared the effects of two combinations AML/ACE or HCTZ/ACE on major fatal and nonfatal cardiovascular events.
- 11 400 men and women 55 years or older who had SBP ≥160 mm Hg

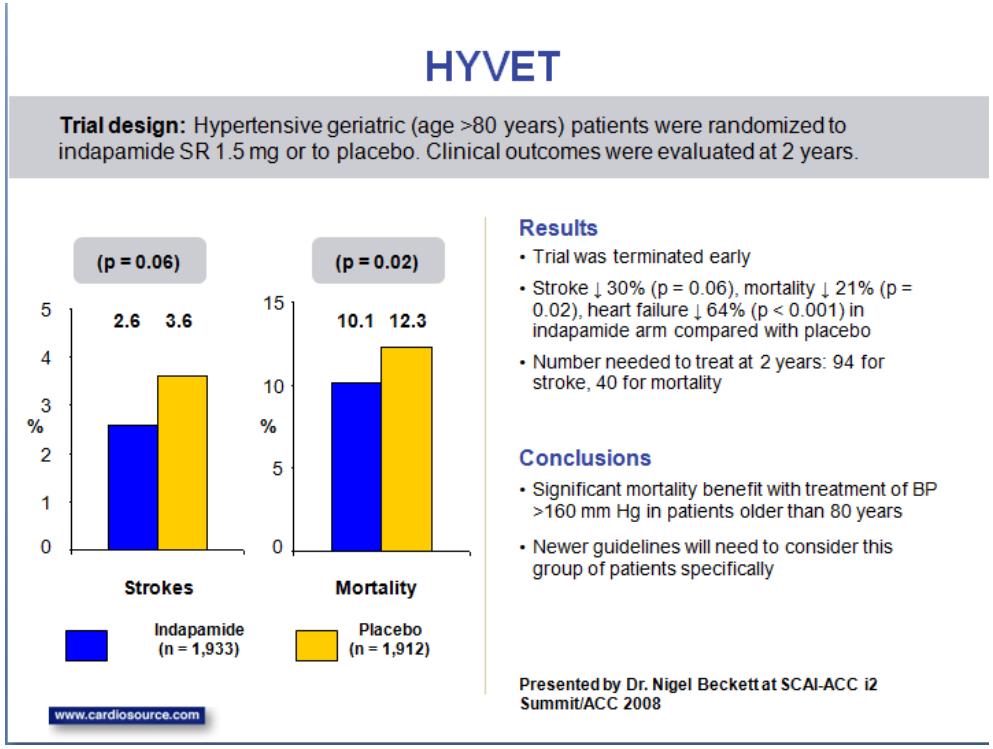
RESULTS:

- 20% ↓ in morbidity and mortality in AML/ACE group.
- ?JNC-7 revision





HYVET



Elderly pts are prone to postural hypotension
Monitor/measure BP's in the standing position

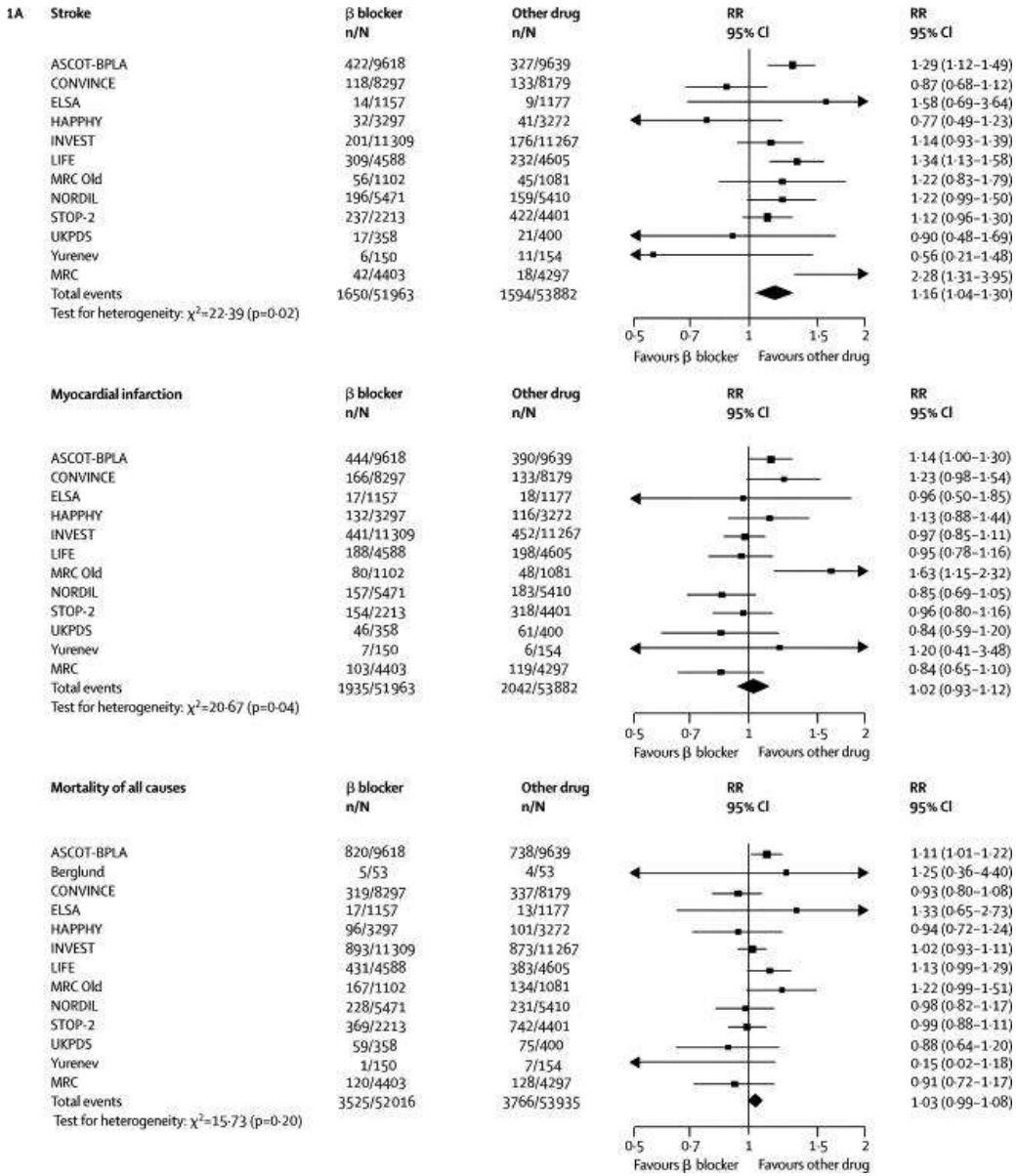
- **HYVET-** benefits and risks of reducing BP (SBP>160mmHg) in very elderly hypertensives. (aged 80 or more).
- BP lowering benefits could be offset by harm due to hypotension-related syncope.
- 3845 patients, international trial
  11 different countries
- Followed for 2 years

RESULT:

- Treatment of hypertension
  ↓Stroke 30%
  ↓ Mortality 21%
  ↓ Heart failure 64%

Should β blockers remain first choice in the treatment of primary hypertension? A meta-analysis

Lancet 2005; 366: 1545–53



1A

Stroke	β blocker n/N	Other drug n/N	RR 95% CI	RR 95% CI
ASCOT-BPLA	422/9618	327/9639		1·29 (1·12–1·49)
CONVINCE	118/8297	133/8179		0·87 (0·68–1·12)
ELSA	14/1157	9/1177		1·58 (0·69–3·64)
HAPPHY	32/3297	41/3272		0·77 (0·49–1·23)
INVEST	201/11309	176/11267		1·14 (0·93–1·39)
LIFE	309/4588	232/4605		1·34 (1·13–1·58)
MRC Old	56/1102	45/1081		1·22 (0·83–1·79)
NORDIL	196/5471	159/5410		1·22 (0·99–1·50)
STOP-2	237/2213	422/4401		1·12 (0·96–1·30)
UKPDS	17/358	21/400		0·90 (0·48–1·69)
Yurenev	6/150	11/154		0·56 (0·21–1·48)
MRC	42/4403	18/4297		2·28 (1·31–3·95)
Total events	1650/51963	1594/53882		1·16 (1·04–1·30)
Test for heterogeneity: χ^2=22·39 (p=0·02)				

0·5 0·7 1 1·5 2
Favours β blocker Favours other drug

Myocardial infarction	β blocker n/N	Other drug n/N	RR 95% CI	RR 95% CI
ASCOT-BPLA	444/9618	390/9639		1·14 (1·00–1·30)
CONVINCE	166/8297	133/8179		1·23 (0·98–1·54)
ELSA	17/1157	18/1177		0·96 (0·50–1·85)
HAPPHY	132/3297	116/3272		1·13 (0·88–1·44)
INVEST	441/11309	452/11267		0·97 (0·85–1·11)
LIFE	188/4588	198/4605		0·95 (0·78–1·16)
MRC Old	80/1102	48/1081		1·63 (1·15–2·32)
NORDIL	157/5471	183/5410		0·85 (0·69–1·05)
STOP-2	154/2213	318/4401		0·96 (0·80–1·16)
UKPDS	46/358	61/400		0·84 (0·59–1·20)
Yurenev	7/150	6/154		1·20 (0·41–3·48)
MRC	103/4403	119/4297		0·84 (0·65–1·10)
Total events	1935/51963	2042/53882		1·02 (0·93–1·12)
Test for heterogeneity: χ^2=20·67 (p=0·04)				

0·5 0·7 1 1·5 2
Favours β blocker Favours other drug

Mortality of all causes	β blocker n/N	Other drug n/N	RR 95% CI	RR 95% CI
ASCOT-BPLA	820/9618	738/9639		1·11 (1·01–1·22)
Berglund	5/53	4/53		1·25 (0·36–4·40)
CONVINCE	319/8297	337/8179		0·93 (0·80–1·08)
ELSA	17/1157	13/1177		1·33 (0·65–2·73)
HAPPHY	96/3297	101/3272		0·94 (0·72–1·24)
INVEST	893/11309	873/11267		1·02 (0·93–1·11)
LIFE	431/4588	383/4605		1·13 (0·99–1·29)
MRC Old	167/1102	134/1081		1·22 (0·99–1·51)
NORDIL	228/5471	231/5410		0·98 (0·82–1·17)
STOP-2	369/2213	742/4401		0·99 (0·88–1·11)
UKPDS	59/358	75/400		0·88 (0·64–1·20)
Yurenev	1/150	7/154		0·15 (0·02–1·18)
MRC	120/4403	128/4297		0·91 (0·72–1·17)
Total events	3525/52016	3766/53935		1·03 (0·99–1·08)
Test for heterogeneity: χ^2=15·73 (p=0·20)				

0·5 0·7 1 1·5 2
Favours β blocker Favours other drug

Beta Blockers

- B decreases BP but do not decrease mortality or MI compared to placebo
- B is slightly better than placebo at preventing stroke, but head to head comparisons with other anti-hypertensive agents they are much worse (16% raised risk)

Beta Blockers

The paper concludes that B should not be used as first line treatment for hypertension, unless the patient has coronary heart disease

Back to the Patient

Inhibace Plus 1 daily
Amlodipine 10 mg/d
Metoprolol 47.5 mg/d

Office BP 170/95mmHg

Home Monitoring:
Most levels elevated 150-160mmHg

Home Monitoring:
Encourage purchasing of unit
Keep a diary to bring to clinic- NEED AT LEAST 12 MEASUREMENTS
4 readings per week, at rest, sitting (2 morning readings, 2 evening readings).
Normal <135/85mmHg
Helps me adjust and uptitrate therapy
Engages the patient- encourages and motivates pts to remain compliant

Renal Ultrasound

Right kidney-10cm; good cortical thickness
Left kidney – 11cm; good cortical thickness

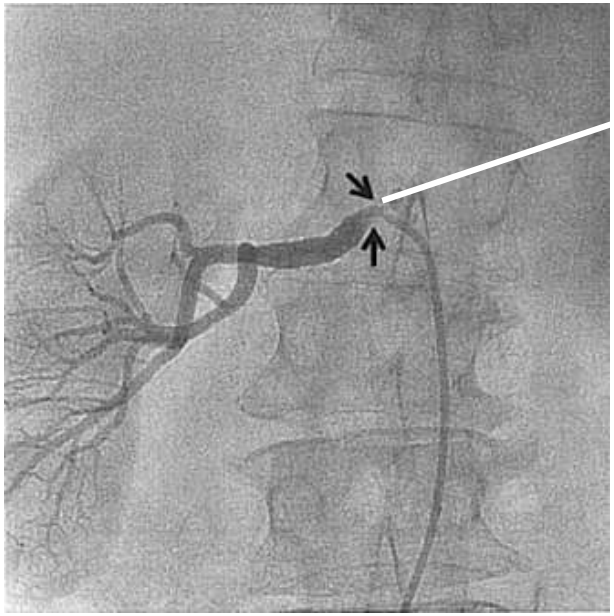
85% STENOSIS- ORIGIN OF RIGHT RENAL ARTERY
Resistive Index- 0.7

60% STENOSIS- ORIGIN OF LRA.

Serum Cr: 63umol/l
Urine: + proteinuria

Refractory hypertension- 4 agents
Probably has renal ischaemia, but still has good residual renal function.

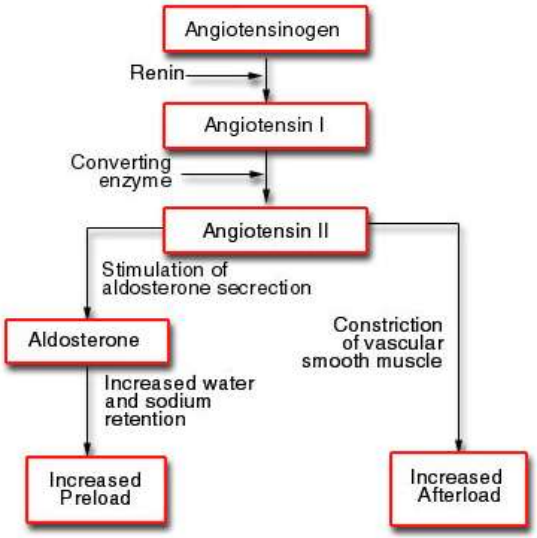
Renal Artery Stenosis



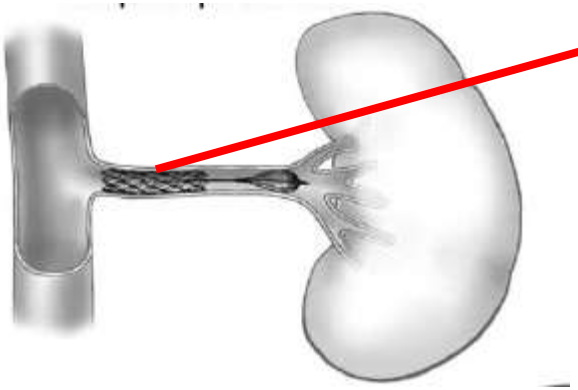
↓Renal perfusion

activation of juxta-glomerular cells

Release of renin



Renal Artery Stenosis



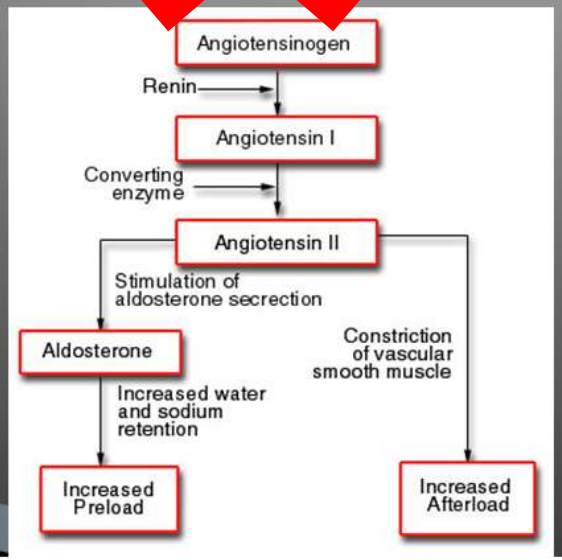
Improve renal perfusion

↓

activation of juxta-glomerular cells

↓

Release [illegible]



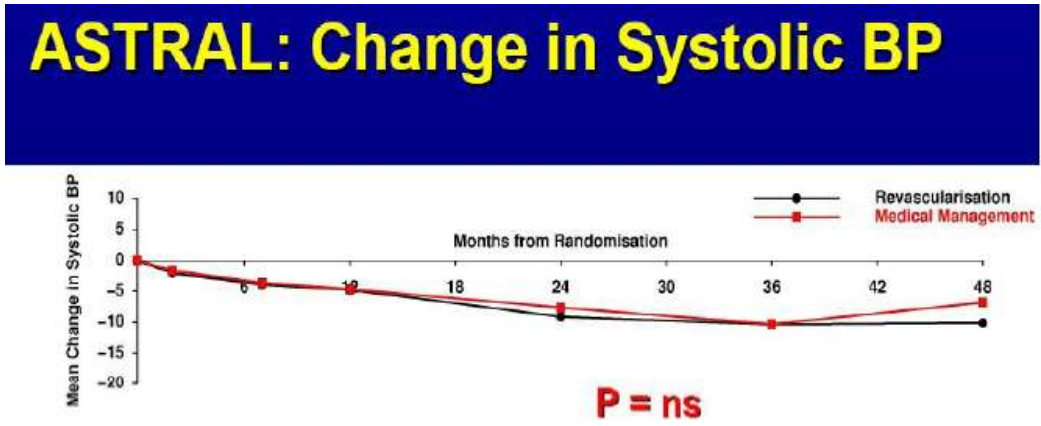
Will renal artery stenting

- Benefit the patient
- Improve BP control
- Prevent CV events

Risks vs. Benefits of renal artery stenting

As with any stent procedure, there is a possibility that complications may occur, including, but not limited to, the following:

- Air bubble(s) in your artery
- Allergic reactions
- Bleeding
- Blood clot(s)
- Bruising or leakage of blood at your groin area or catheter insertion site
- Death
- Heart attack
- Infection
- Injury or damage to your artery or wall of the artery requiring emergency surgery or potential loss of the kidney
- Migration of the stent from its original placement
- Restenosis or reoccurrence of the artery narrowing around or within the stent
- Renal failure



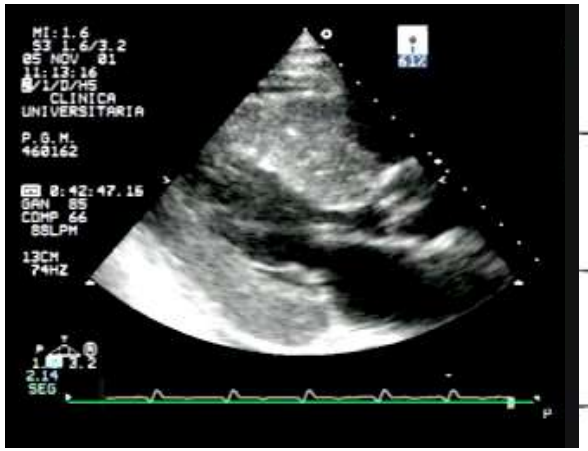
DATA ON RENAL ARTERY STENTING

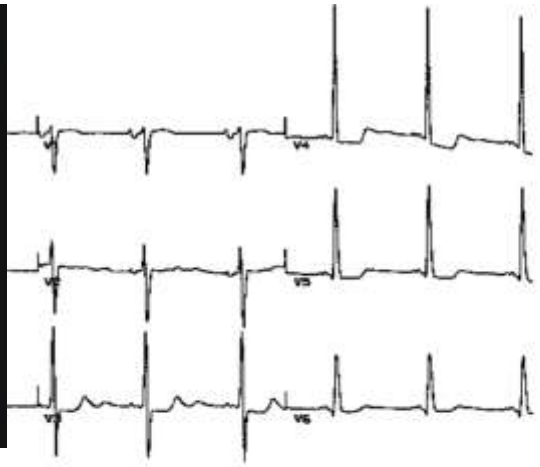
2 trials ASTRAL (806 PTS), STAR(140 pts)
-Endpoints-renal function, CV events, mortality
-No benefit of stenting plus medical therapy over medical therapy alone
-Limited by imprecise definitions of RAS, inclusion of pts with insignificant lesions, crossovers, multiple disease etc.

CORAL- well designed multi-centre study.
completion expected 2011
meantime revascularisation should be reserved for pts in whom aggressive medical therapy fails, trial participants.

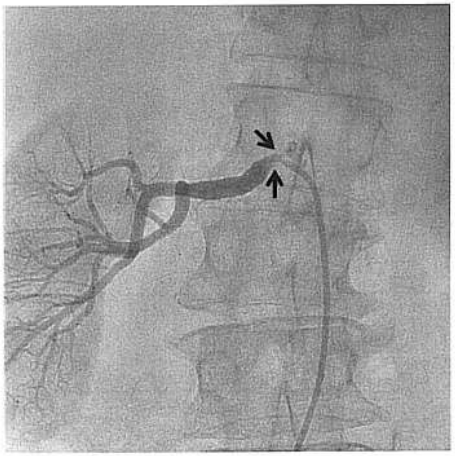
Persistent Hypertension + End Organ Damage + Ischaemic Viable Kidney

Urine: + proteinuria
Serum Cr- 64umol/l

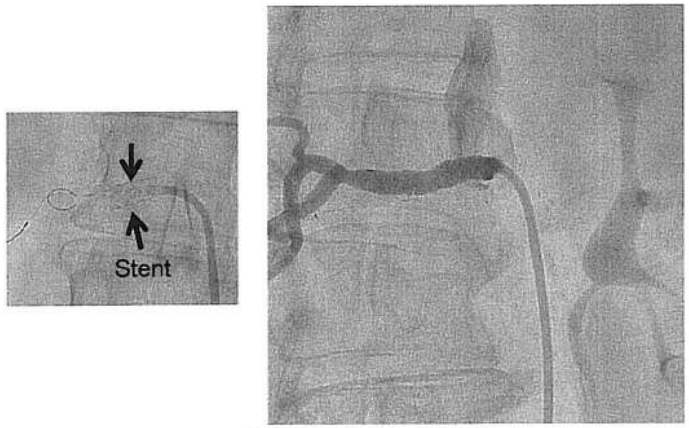




RRA STENTING



Right Renal Artery: Before Stent

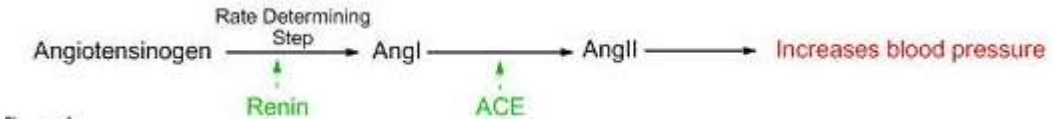


Right Renal Artery: After Stent

Respons e to BP therapy

On 4 medications plus renal artery stenting- 24 hr average of 154/88mmHg

Need Better Treatments



Direct Renin Inhibitor
e.g. Aliskiren

ACE Inhibitors

Angiotensin Receptor blockers
e.g. Losartan, candesartan

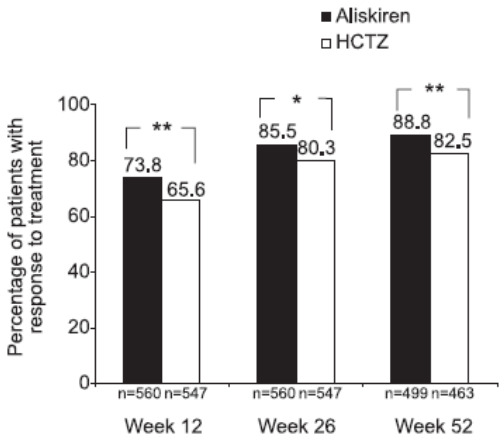
Long-Term Antihypertensive Efficacy and Safety of the Oral Direct Renin Inhibitor Aliskiren

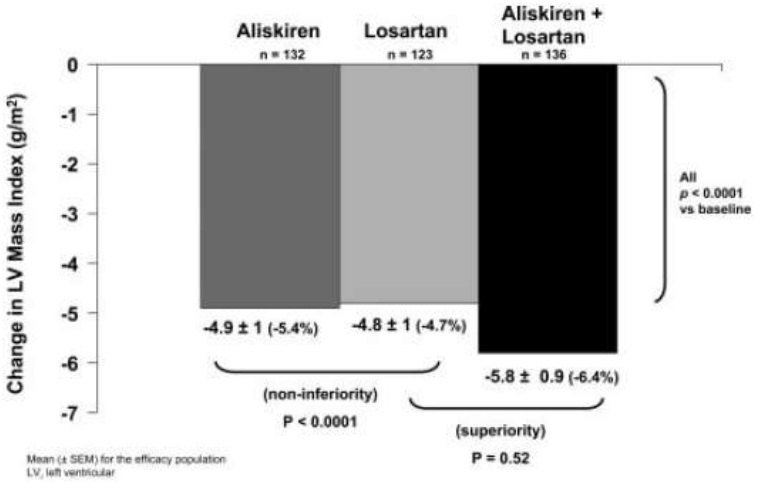
A 12-Month Randomized, Double-Blind Comparator Trial With Hydrochlorothiazide

(*Circulation*. 2009;119:417-425.)

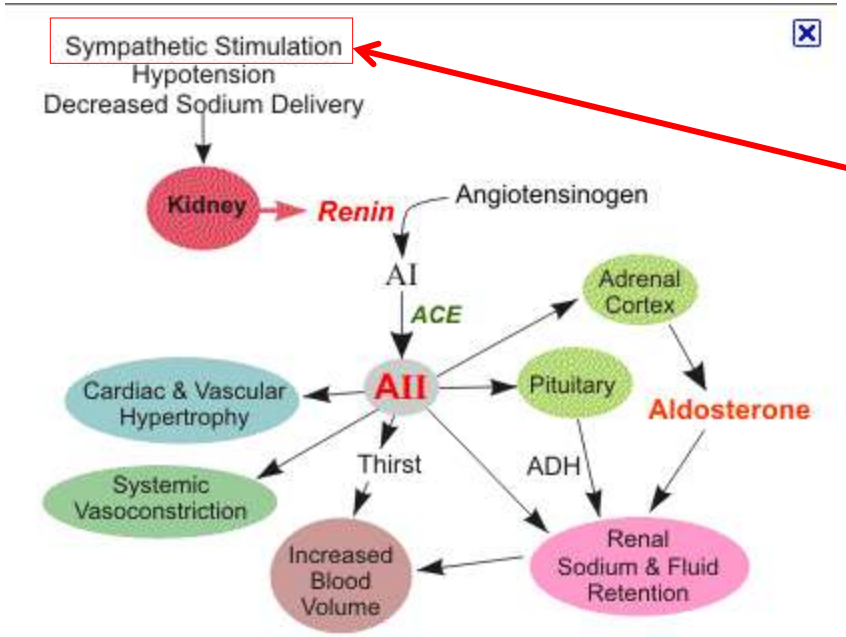
Effect of the Direct Renin Inhibitor Aliskiren, the Angiotensin Receptor Blocker Losartan, or Both on Left Ventricular Mass in Patients With Hypertension and Left Ventricular Hypertrophy

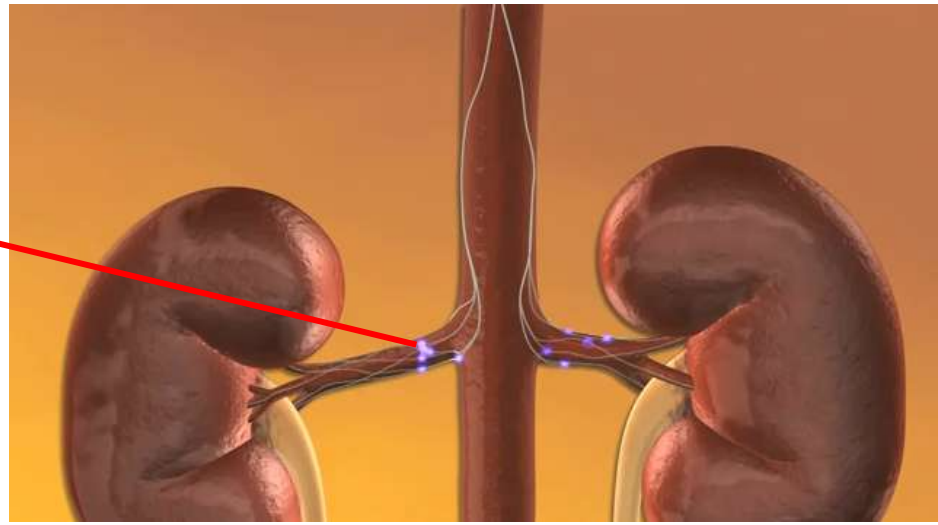
***Circulation.*2009;119:530-537**

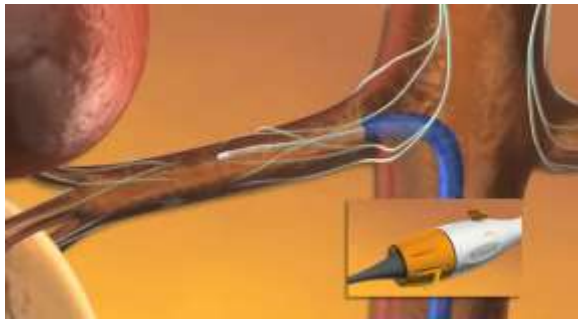




New Invasive Therapies to block the RAAS



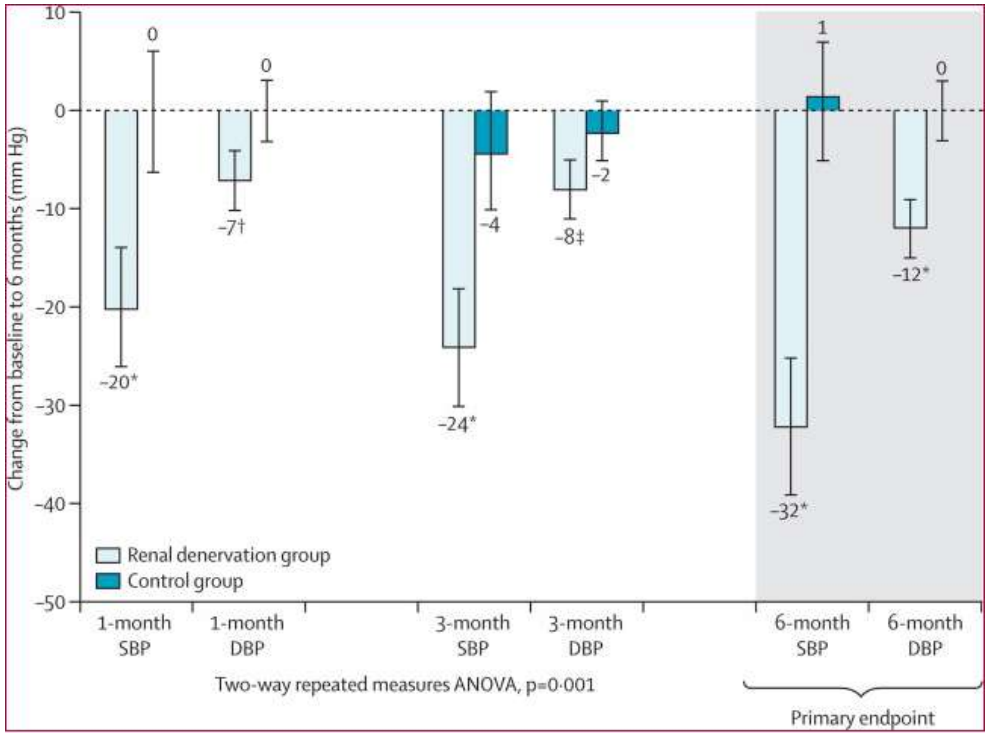




Renal sympathetic denervation in patients with treatment-resistant hypertension (The Symplicity HTN-2 Trial): a randomised controlled trial

Symplicity HTN-2 Investigators‡

	Renal denervation group (n=52)	Control group (n=54)
Baseline systolic blood pressure (mm Hg)	178 (18)	178 (16)
Baseline diastolic blood pressure (mm Hg)	97 (16)	98 (17)
Age (years)	58 (12)	58 (12)
Sex (female)	18 (35%)	27 (50%)
Race (white)	51 (98%)	52 (96%)
Body-mass index (kg/m²)	31 (5)	31 (5)
Type 2 diabetes	21 (40%)	15 (28%)
Coronary artery disease	10 (19%)	4 (7%)
Hypercholesterolaemia	27 (52%)	28 (52%)
eGFR* (mL/min per 1·73 m²)	77 (19)	86 (20)
eGFR* 45–60 mL/min per 1·73 m²	11 (21%)	6 (11%)
Serum creatinine (μmol/L)	91 (25)	78 (18)
Urine albumin-to-creatinine ratio (mg/g)†	128 (363)	109 (254)
Cystatin C (mg/L)‡	0·9 (0·2)	0·8 (0·2)
Heart rate (bpm)	75 (15)	71 (15)
Number of antihypertension medications	5·2 (1·5)	5·3 (1·8)
Patients on hypertension medication for more than 5 years	37 (71%)	42 (78%)
Patients on five or more medications	35 (67%)	31 (57%)
Patients receiving (drug class)		
ACE inhibitors/ARBs	50 (96%)	51 (94%)
Direct renin inhibitors	8 (15%)	10 (19%)
β blockers	43 (83%)	37 (69%)
Calcium-channel blockers	41 (79%)	45 (83%)
Diuretics	46 (89%)	49 (91%)
Aldosterone antagonist	9 (17%)	9 (17%)
Vasodilators	8 (15%)	9 (17%)
α-1 blockers	17 (33%)	10 (19%)
Centrally acting sympatholytics	27 (52%)	28 (52%)



Summary-Difficult Hypertension

Take a good history- compliance, diet (salt), stress, alcohol, NSAID's

Resistant hypertension- persistent ht despite good doses of 3 agents including a diuretic- ACE/ARB, Ca blocker, thiazide diuretic → refer

Need a good diuretic e.g. Chlorthalidone 25mg/d
eGFR <40mls/min Frusemide bd

Maximise doses of ACE/ARB, calcium blockers

If overweight- spironolactone 25mg/d

Look at heart rate- good indicator of sympathetic activity
B-Blocker
Diltiazem with the amlodipine

Summary-Difficult Hypertension cont.

Benefit in treating hypertension in the very elderly (HYVET)
BP Target are lower- SBP-150mmHg
Postural hypotension- monitor Rx in the standing position

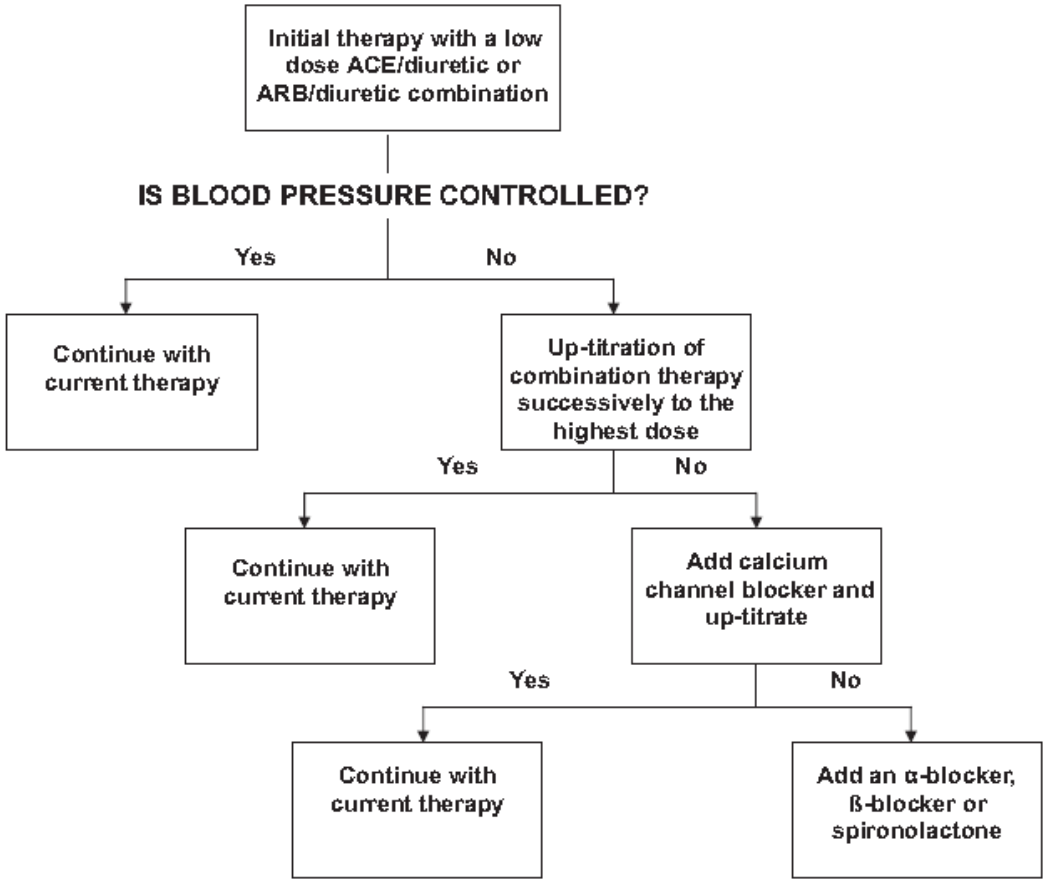
Promote home BP monitoring-engages the pt and improves compliance

24 hour BP monitoring in useful
Useful information on nocturnal BP-dippers, non-dippers and risers

A Simplified Approach to the Treatment of Uncomplicated Hypertension

A Cluster Randomized, Controlled Trial

(*Hypertension*. 2009;53:646-653.)



THANK YOU

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