Renal Hypertension

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Hypertension

- Most common chronic illness in UK
- Affects 37% of adult population
- Increasing incidence with age affecting over 50% in >60years

Hypertension - why worry?

- Stroke

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  - Reduces risk of by 35-40%

Hypertension - why worry?

- Stroke

- Cardiovascular disease

A blood pressure below 140/90 mmHg could prevent 25% coronary heart disease events,

optimal control to below 130/80 mmHg could prevent even more events, 40% percent.

Hypertension - why worry?

- Stroke
- Cardiovascular disease
- Heart Failure

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- Left Ventricular Hypertrophy

Hypertension - why worry?

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Even where the impact on an individual's risk may seem modest, effects on a population basis are profound.

Definitions and the right cuff

British Hypertension Society Definitions

OPTIMAL BP…….120/80 or less

NORMAL BP…….120-129/80-84

HIGH NORMAL…….130-139/85-89

HYPTENSION

Mild or Grade 1……140-159/90-99

Moderate or Grade 2……160-179/100-109

Severe or Grade 3……>180/>110

Isolated Systolic Hypertension……>140/<90

Use the right cuff & measure it properly

- Standardise the environment as much as possible:
  - relaxed temperate setting, with the patient seated
  - arm out-stretched, in line with mid-sternum, and supported.
- Correctly wrap a cuff containing an appropriately sized bladder around the upper arm and connect to a manometer. Cuffs should be marked to indicate the range of permissible arm circumferences.
- Palpate the brachial pulse in the antecubital fossa of that arm.
- Rapidly inflate the cuff to 20 mmHg above the point where the brachial pulse disappears.
- Deflate the cuff and note the pressure at which the pulse re-appears: the approximate systolic pressure.
- Re-inflate the cuff to 20 mmHg above the point at which the brachial pulse disappears.
- Using one hand, place the stethoscope over the brachial artery ensuring complete skin contact with no clothing in between.
- Slowly deflate the cuff at 2–3 mmHg per second listening for Korotkoff sounds.

White Coats

- Occurs 1/4-1/5 of patients

- Use Ambulatory blood pressure monitoring to exclude.
- Average BP >135/85

	Low	Normal	Borderline	Mild	Moderate	Severe
Daytime						
SBP ,	100	100 – 135	136 – 140	141 – 155	156 – 170 .	>170
DBP ,	65	65 – 85	86 – 90	91 – 100	101 – 110 .	>110
Night-time						
SBP ,	90	91 – 120	121 – 125	126 – 135	136 – 150 .	>150
DBP ,	50	51 – 70	71 – 75	76 – 85	86 – 100 .	>100

Primary or Secondary

- Primary is also called essential - the vast majority
  - characterised by
  - increased in peripheral vascular resistance.
  - Familial
  - More common in the overweight.
  - Increased salt intake
  - high alcohol intake also increases blood pressure.
  - black population.
- Secondary in a small but important number of cases.

Secondary Considerations

Hypertension with a discernable cause.

Consider when hypertension presents in

young

refractory to standard therapy,

severe (SBP>180mmHg, DBP>110mmHg),

with end-organ damage at diagnosis.

Approaching a hypertensive patient

- History -
  - presence of precipitating/aggravating factors
  - natural course of the blood pressure
  - Determine overall cardiovascular risk status

- Examination
  - extent of target organ damage
  - Clues for secondary HTN
  - Urinanalysis
- Perform a limited number of investigations aware of the clinical clues suggesting secondary hypertension.

Secondary causes

- Renal
  - Intrinsic Renal disease eg Glomerulonephritis
  - Chronic kidney disease
- *Renovascular Disease*
- Vascular Disease
  - Coarctation
- Endocrine causes
  - Cushings disease
  - Conn's syndrome/ Hyperaldosteronism
  - Phaeochromocytoma
- Drugs
  - Alcohol
  - Nasal decongestant
  - Cocaine
  - OCP
  - NSAIDS
  - Corticosteroids
- Sleep Apnoea

Secondary Hypertension – who to suspect?

- Young
- Severe Hypertension
- Acute rise in BP readings when previously stable
- Difficult to treat
- Creatinine rise after starting ACE-I or ARB
- USS shows unequal sized kidneys
- Sudden onset pulmonary oedema
- Renal Bruit auscultated

Renal Hypertension – who to suspect?

- Creatinine rise after starting ACE-I or ARB
- USS shows unequal sized kidneys
- Sudden onset pulmonary oedema
- Renal Bruit on auscultation
- Low Potassium values
- Severe salt and water retention

Renal Hypertension – causes

- Atherosclerosis
  - Often associated with widespread artheroma but can be isolated
  - Often associated with peripheral vascular disease

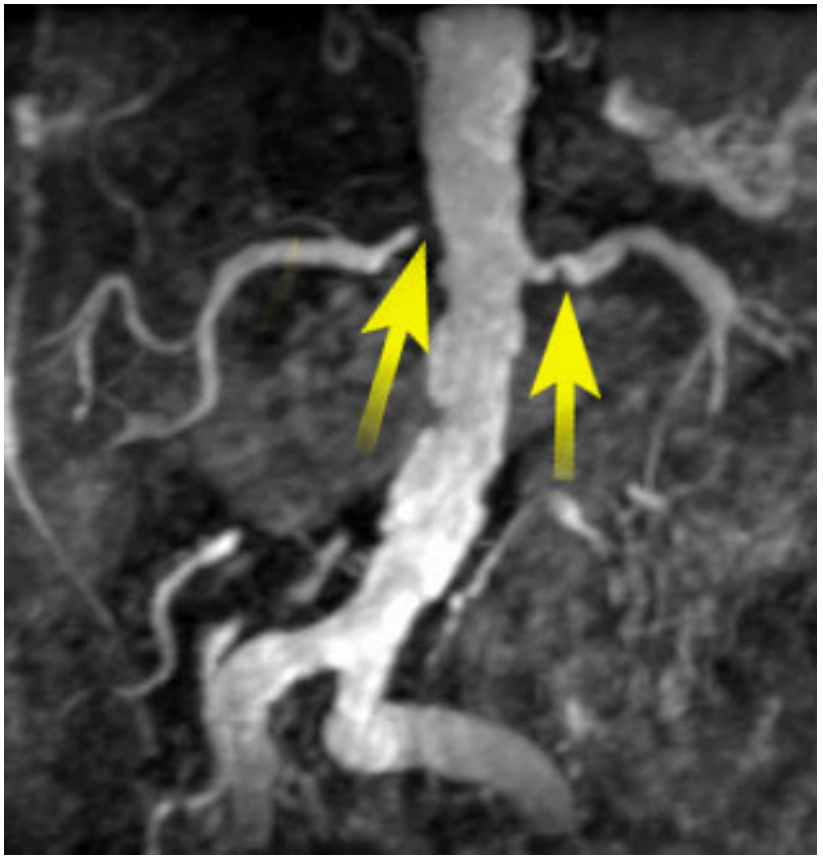
- Fibromuscular dysplasia
  - Affects young women

Investigating renovascular disease

- Gold Standard Investigation
  - Intraarterial Digital Substraction Angiography

Investigating renovascular disease

- Magnetic Resonance Angiography



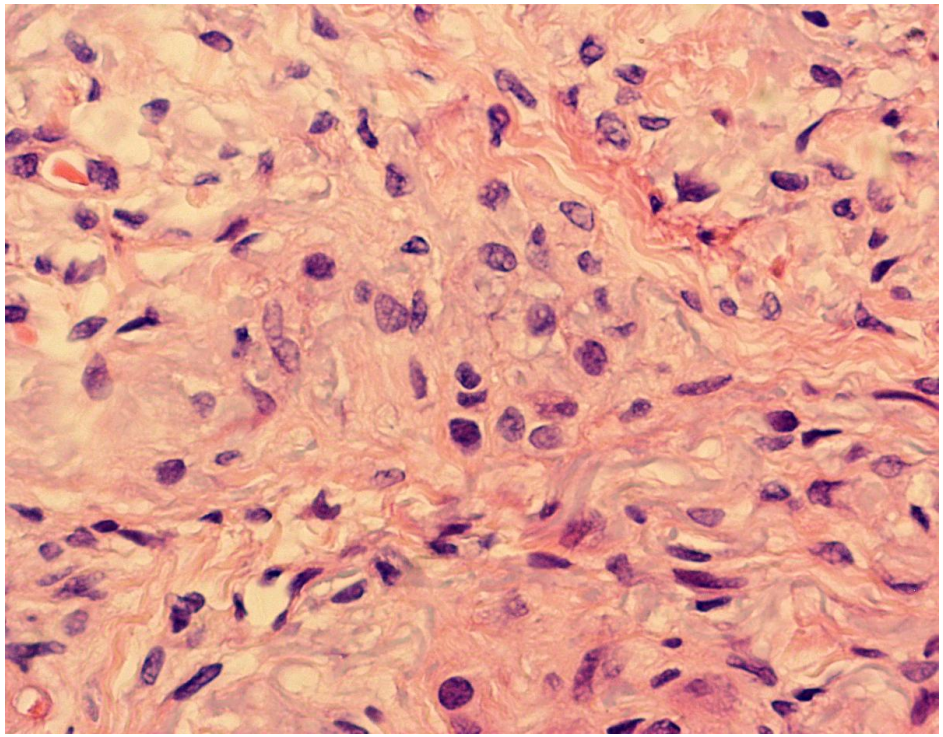
Investigating renovascular disease

- Magnetic Resonance Angiography
- Very sensitive and specific test (100% and 96% respectively) in some studies.
- Probably not that good but very useful in atheromatous renovascular disease

- Claustophobia
- Can’t see Fibromuscular Dysplasia very well
- Gadolinium toxicity in Renal Failure

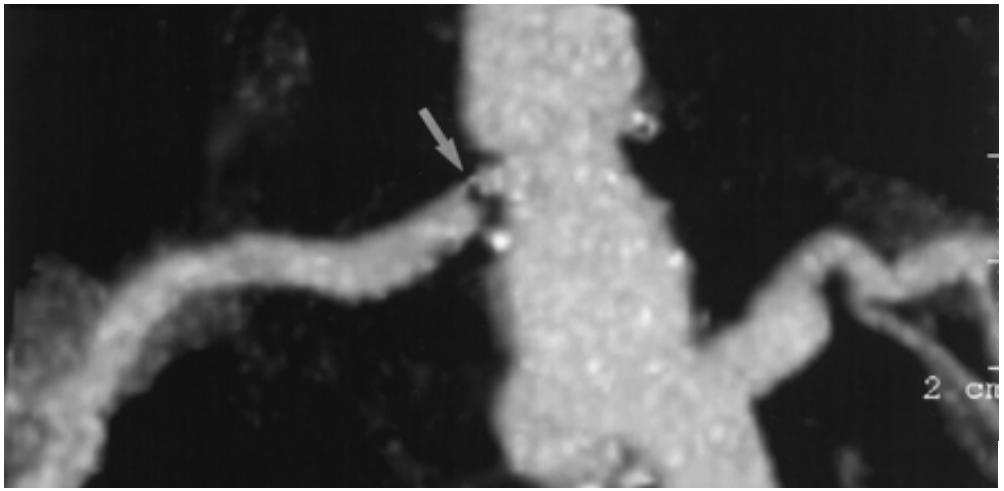
MR Angiography -

- Gadolinium toxicity
- Nephrogenic Fibrosing Dermopathy or
  Nephrogenic Systemic Fibrosis when GFR <60



CT Angiography -

- Less good than MRA but offers
- 93% sensitivity and 81% specificity
- Very good at picking up fibromuscular hyperplasia



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Other imaging techniques

Doppler Ultrasound

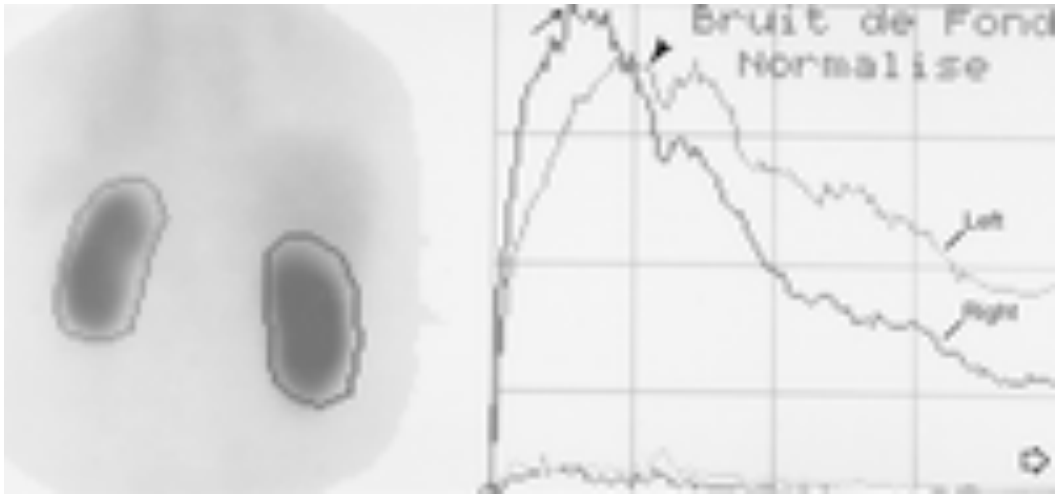
- Peak systolic velocity increases across a stenosed blood vessel.
- Compare systolic velocities in aorta and renal artery

Other imaging techniques

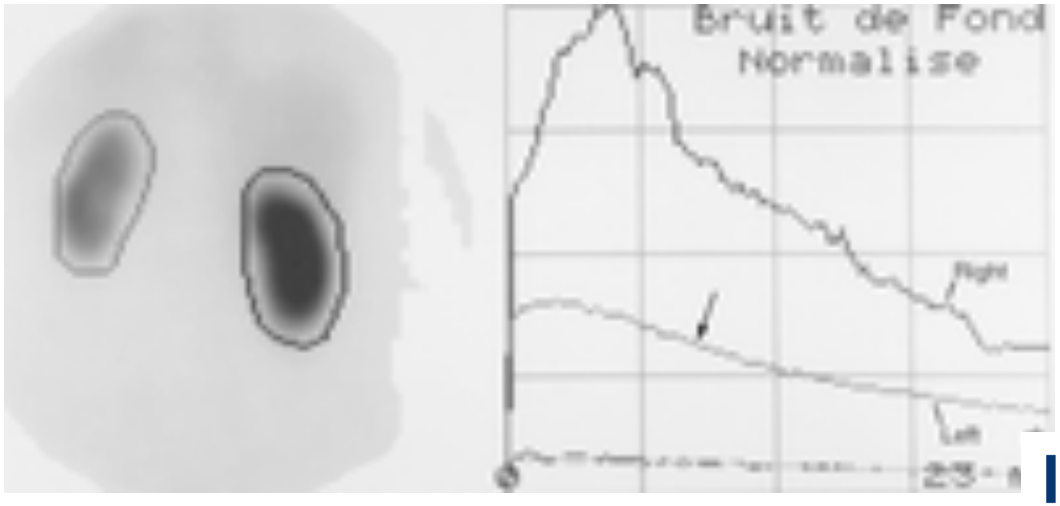
Captopril Renogram

- Using a maker of glomerular filtration such as DTPA or MAG3 see renogram.
- Compare images before and after captopril
- In renal artery stenosis see fall in renogram in stenosed kidney.

Captopril Angiogram



Before Captopril



After Captopril

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What are the down stream effects of renovascular diease?

- Progression of atheromatous plaques

- Reduction in renal blood flow leads to glomerular changes and reduction in renal size with time
  - Ischaemic atrophy or ischaemic glomerulopathy

Treatments

- What's on offer?

Treatments

- What's on offer?
  - Life style changes
  - Drug therapy – avoid agents that influence renal blood flow – ACE-i/ARB/Aldosterone Antagonists (eg spironolactone)
  - Angioplasty alone
  - Angioplasty with stenting

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Treatment - non drug

- Life style modifications
  - weight loss,
  - regular exercise,
  - avoidance of excess alcohol and a
  - diet low in salt.
  - smoking
  - caffeine

Treatment - salt

- When sodium intake rises above 6g/day, essential hypertension is likely to develop, with a subsequent increase in the risk of cardiovascular events.

- Restricting dietary sodium to 6g/day in hypertensive patients lowers systolic pressure by 6.3mmHg in the over 45s, which will have an enormous impact on population morbidity and mortality.

Drug treatment for primary hypertension

- Control BP to target values
- In uncomplicated patients 140/90

Use ACEi/ARB whereever possible but also target treatment

– Eg Ca channel antagonists in Afro-Caribbeans
– Alpha blockers (doxazocin) if urinary symptoms
– Beta-blockers (atenolol) if cardiac disease

NICE treatment

- Young (<55yrs) non-black patients should be given an ACE inhibitor or angiotensin receptor blocker as a first line drug
- Older (>55yrs) or black patients should be given a calcium channel blocker or thiazide diuretic as a first line drug. With ACE-I or ARB as third line.
- Use an alpha blocker as forth line.
- Add further diuretic therapy or a beta blocker if needed

Drug treatment for renal hypertension

- Control BP to target values
  - lower targets compared with eessential hypertension
  - Use ACEi, ARB and aldosterone antagonists when you can
  - (125/75 if proteinuria or 130/85 without)

What should get intervention?

- Percutaneous Angioplasty and stenting
- Difficult to control BP
- Flash Pulmonary oedema
- Malignant Hypertension

Percutaneous Angioplasty and stenting

- BUT an RCT (ASTRAL) did not show a benefit of intervention.
- 806 pts in UK & Australasia
- Controlled BP, gave statin and antiplatelet agents and randomised to intervention vs none

Percutaneous Angioplasty and stenting

- 12 month follow up point no difference in
  - renal function (creatinine increased by 18 mmol/l in both groups),
  - Systolic (decrease by 5 mmHg in both) or diastolic blood pressure,
  - combined renal events &
  - combined cardiovascular events (including death)
- At 4 years
- slight benefit in favour of revascularization for
  - creatinine (10 mmol/l difference),
  - systolic blood pressure,
  - combined renal (17% vs. 23%) and cardiovascular events (34% vs. 41%), but the data was underpowered to be conclusive at this stage.

Percutaneous Angioplasty and stenting

- My practice

- Offer stenting to allow maximal use of ACE-I and ARB to offer nephroprotection

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Surgery

- Offer if need complex revascularisation

Fibromuscular dysplasia

- Noninflammatory, nonatherosclerotic disorder that leads to arterial stenosis.
- Can affect any arterial bed
- Causes hypertension, transient ischemic attack, and stroke.

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Fibromuscular dysplasia

- Management
  - Control the BP with ACE-i or ARB (discontinuing the ACE inhibitor or ARB if hyperkalaemia cannot be controlled, or if there is progressive increase in the plasma creatinine
  - Offer intervention with angioplasty if
    - Can't control BP
    - Evidence for loss of renal mass from ischaemic atrophy

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Hypertension in renal disease

- Controlling blood pressure is the essential in renal failure
- Use of ACE-I, ARB and now direct renin inhibitors are all able to slow decline in GFR
- Targets are low
  - Proteinuria low: ACR<70 or PCR<100 - Target blood pressure <140/90 (NICE suggests 130-139/90)
  - Proteinuria high: ACR>70 or PCR>100: - Target blood pressure <130/80 (NICE suggests 120-129/80)

Further reading

Renal Association Website www.renal.org

British Hypertension Society www.bhsoc.org

NICE guidelines for Hypertension www.nice.org.uk

ASTRAL study www.astral.bham.ac.uk

Mann and Pickering, TG. Detection of renovascular hypertension. State of the art: 1992. Ann Intern Med 1992; 117:845.

Gandhi, et al. The pathogenesis of acute pulmonary edema associated with hypertension. N Engl J Med 2001; 344:17.

Safian, RD, Textor, SC. Renal-artery stenosis. N Engl J Med 2001; 344:431.

Slovut, D, Olin, JW. Fibromuscular dysplasia. N Engl J Med 2004; 350:1862-71

Balk, et al. Effectiveness of management strategies for renal artery stenosis: a systematic review. Ann Intern Med 2006; 145:901.

Treatment - ABC or D?

- Historical treatment was with a thiazide diuretic- [ALLHAT trial] then a beta -blocker or calcium channel antagonist

- But thiazides tend to have an adverse impact upon glucose and lipid profiles.

- Is there a specific reasons for an alternative agents.
  - ACE-I/ARBs improve outcomes in heart failure
  - ACE-I/ARBs slow the progression of proteinuria and renal failure.
  - Alpha-blockers, may be preferred in older men with symptoms of prostatism but avoided in patients with heart failure.
  - Calcium channel blockers are particular efficacious in black patients.

- ASCOT found that the combination of amlodipine and perindopril was able to reduce all major cardiovascular events, all-cause mortality and new-onset diabetes in hypertensives compared with atenolol and a thiazide diuretic.

Targets and goals

- Uncomplicated patients 140/90
- Diabetes <130/80
- Renal Patients <130/80 (<125/75 with proteinuria)
- In the elderly with isolated systolic hypertension, do not lower DBP < 65 - associated with an increased risk of stroke.