A Clinical Guide to Referring Cardiac Conditions in Primary Care

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www.hertslondoncardiology.co.uk

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Primary Care Cardiology Referrals

	Chest pain	Palpitations Family Hx of SCD	Heart failure/SOB FH of cardiomyopathies	Syncope	Hypertension	Murmurs
Primary Care Investigations	Blood tests Risk assessment ECG CxR ?Echo (open access)	Blood tests ECG 24- Holter Event recorder Smart phone devices ?Echo (open access)	Blood tests NT-pro BNP ECG CxR Spirometry ?Echo (open access)	Blood tests ECG 24- Holter Event recorder Smart phone devices ?Echo (open access)	Blood tests Renin-aldosterone Urinary catecholamines. ECG 24- ABPM Ultrasound CT ?Echo (open access)	Blood tests ECG ?Echo (open access)
Secondary or Tertiary Care Investigations	Cardiac CT Exercise testing Echo Stress echo Myocardial perf. scan Calcium scoring Coronary angio CTPA Cardiac MRI Genetic testing	Echo Exercise testing Implantable loop recorder Electrophysiology testing Cardiac MRI Ajmaline/flecainide tests Genetic testing	Echo Stress echo Myocardial perf. scan Cardiac CT Coronary angio Cardiac MRI Genetic testing	Echo Tilt table test Exercise testing Implantable loop recorder Electrophysiology CT MRI EEG	Echo CT MRA kidneys/adrenals Assessment for non pharmacological intervention	Echo Cardiac CT MRI Cardiac Cath.

A significant number of diagnoses can be made at this level with above investigations without further secondary testing

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Chest pain

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History

- Location. Where, Focal vs Diffuse.
- Character. Dull/Sharp/Stabbing/Burning/Squeezing/crushing/Tearing/Heavy
- Radiation.
- Timeframe. When did it start? Previous events
- Onset/Offset. Gradual/Sudden
- Severity. Scales e.g. 9/10.
- Aggravating factors. Exertion, Emotion, Respiration, Coughing, Eating, Posture
- Relieving factors. Rest, Sitting up, Antacids
- Associated symptoms. Nausea/Vomiting, Sweating, SOB, pre-syncope, Confusion, Neurological symptoms.

Examination ± ECG

Refer or not to refer

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Causes of chest discomfort

Skin – shingles

Muscular
- Postural/movement
- Cough

Skeletal
- Bone pain from metastases/fractures

Pleural
- Infection
- Malignancy

Oesophageal
- Spasm
- Reflux/ulcer/tear
- Malignancy
- Infection etc

Pulmonary
- Embolism
- Pneumonia
- Asthma
- Malignancy
- Pneumothorax

Pericardium/Myocardium

Vascular
- Aortic dissection

Sub-diaphragmatic
- Cholelithiasis/Cholecystitis/panrceatis
- Median arcuate ligament syndrome

Neural
- Thoracic disk
- Cervical disk

Anxiety

Myocardial ischaemia/infarction

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Chest Pain

Diagnostic features of angina

Aggravating factors:	predictable level of exercise, emotional stress, exercise plus heavy meal, cold weather
Relieving factors:	GTN, cessation of activity
Duration:	less than 15 minutes
Location:	retrosternal, infrequently epigastric or infrascapular
Radiation:	bilaterally across the chest, one or both arms, shoulders, back epigastrium, neck and lower jaw
Description:	heaviness, tightness, pressure, constriction, dull and deep, indigestion

Other features which make a diagnosis of stable angina unlikely are when the chest pain is:
- continuous or very prolonged and/or
- unrelated to activity and/or
- brought on by breathing in and/or
- associated with symptoms such as dizziness, palpitations, tingling or difficulty swallowing.

Consider causes of chest pain other than angina (such as gastrointestinal or musculoskeletal pain).

NICE Clinical Guideline

Primary Care

Blood tests
Risk assessment
ECG
CxR
?Echo (open access)

Secondary Care

Cardiac CT
Exercise testing
Echo
Stress echo
Myocardial perf. scan
Calcium scoring
Coronary angio
CTPA
Cardiac MRI
Genetic testing

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NICE Guidelines Stable Angina 2016

Clinical diagnosis of angina.

Take a history
- Age and sex
- Characteristics of chest pain
  - Constricting discomfort in the front of the chest, neck, shoulders, jaw or arms
  - Precipitated by physical exertion
  - Relieved by rest or GTN within about 5 minutes
- Associated symptoms
- Cardiovascular history
- Cardiovascular risk factors

Physical examination
- Identify risk factors for CVD
- Identify signs of other CVD
- Identify non-coronary causes of angina (eg severe aortic stenosis)
- Exclude other causes of chest pain.

Routine investigation
- Blood tests to rule–out conditions exacerbating angina eg anaemia
- 12 lead ECG – changes consistent with coronary disease
  - Pathological Q waves
  - Left bundle branch block
  - Regional ST/T change

Clinical diagnosis driven mainly by the characteristics of chest pain

Non–anginal pain *≤1 characteristic* | Atypical angina *2 characteristics* | Typical angina *All 3 characteristics*

Adam Timmis, and Carl A Roobottom Heart 2017;103:982-986

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History taking:

Care should be taken to ensure the Clinician's interpretation equates to the patient's description

Northern England: sharp means severity rather than character

Ethnic groups/language barrier

Cardiac chest pain: sharp, dull, burning, discomfort, heavy, tight, "just pain", "like fire"

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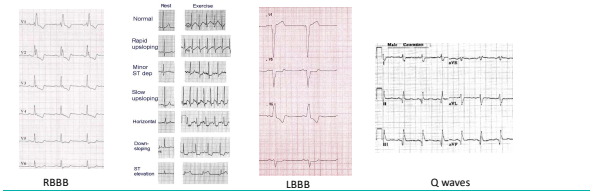
ECG

A number of changes on a resting 12-lead ECG are consistent with CAD and may indicate ischaemia or previous infarction.

These include:

- pathological Q waves
- LBBB
- ST-segment and T wave abnormalities (for example, flattening or inversion).



RBBB | LBBB | Q waves

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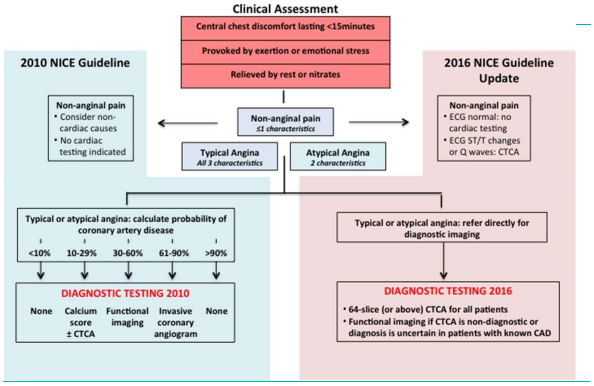
NICE National Institute for Health and Care Excellence — Update November 2016



Adam Timmis, and Carl A Roobottom Heart 2017;103:982-986

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Anatomical vs Functional testing

CTCA provides anatomical evidence of the presence and severity of epicardial CAD, while the other tests provide 'functional' information about stress-induced perfusion defects or wall motion abnormalities as evidence of ischaemia caused by obstructive epicardial coronary disease.

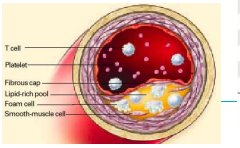

Table 1 Ischaemia testing for diagnosis of CAD: sensitivity and specificity data from meta-analytic reviews

Imaging modality and first author	Year of study	Sensitivity (%)	Specificity (%)
Exercise ECG			
Gianrossi *et al*[18]	1989	68	77
Kwok *et al*[19]	1999	61	70
SPECT			
Fleischmann *et al*[20]	1998	87	64
Heijenbrok-Kal *et al*[21]	2007	88	73
Jaarsma *et al*[22]	2012	88	61
McArdle *et al*[23]	2012	85	85
Stress echo			
Fleischmann *et al*[20]	1998	85	77
Geleijnse *et al*[24]	2007	72	88
Heijenbrok-Kal *et al*[21]	2007	83	84
CMR perfusion			
Nandalur *et al*[25]	2007	91	81
Jaarsma *et al*[22]	2012	89	76
Desai and Jha[26]	2013	89	85
CTCA			
Mowatt *et al*[27]	2008	99	89
von Ballmoos *et al*[28]	2011	100	89
Vorre *et al*[29]	2013	94	91

CAD, coronary artery disease; CTCA, CT coronary angiography; CMR, cardiac magnetic resonance; CT coronary angiography; SPECT, single-photon emission computed tomography.

Timmis A, Roobottom CA. Heart 2017;103:982–986

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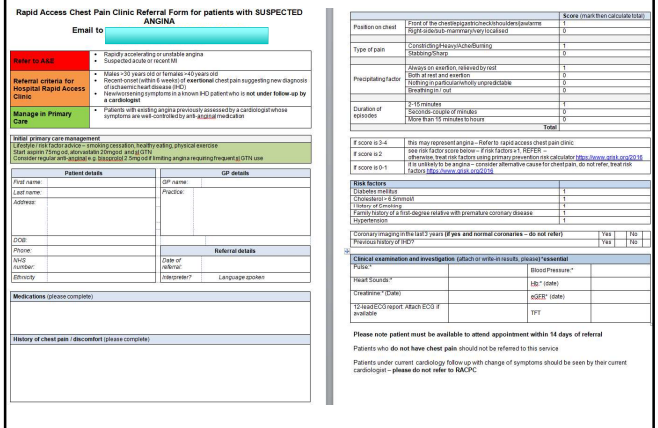

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Lister Hospital Rapid Access Chest Pain Clinic
Summary of clinic attendance

70 yr. Female

Date of Clinic 23/8/2013 Referred 9/8/2013 For the attention of [redacted]

Patient Details	[redacted]
Presenting Symptoms	Recurrent chest pain at rest but normally when very stressed not on exertion. Normally described as a tightness that radiates down the left arm lasted all day and at times on and off for the 3 days. Has had this for many years seen [redacted] but never investigated or treatment started, can have short lived pains every day especially when stressed. No chest pain on exertion only mild SOB Daughter and son in law doctors at UCLH
Blood Results	Na 142 U 4.9 Creat 62 Chol 6.0 HDl 1.53 Trig 1.04 LDL 4.0 Gluc 4.6
PMH	Glaucoma Arthritis Never smoked
Family History	Mother – RIP heart attack 69 mother had angina prior to that Grandmather- MI 66yrs
Medication	Eye drops
Examination	Blood Pressure 112/69mmhg Heart sounds normal
Investigations	ECG – NSR rate 52bpm nil changes
Diagnosis	Atypical chest pain
Risk Factors	**Prediction of CHD in a patient presenting with non-acute chest pain:** Chest pain categorised as: atypical cp Probability of significant CHD (ACC/AHA): 64% Probability of significant CHD (Duke): 27%
Change of medication	**Aspirin 75mg od**
Management Plan	Diet, lifestyle & risk factors discussed. Leaflets given.
Follow up	CTCA

Dr A ghuran
Cardiologist

[redacted]
Cardiology Specialist Nurse ext 4489

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Palpitations

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Diagnostic pathway – Primary Care

- History
- Examination
- Resting ECG
- ECG with symptoms
- Investigations (primary care) - Blood tests, ECG, 24- Holter, Event recorder, Smart phone devices, Echo (open access)

Secondary Care
Echo
Exercise testing
Implantable loop recorder
Electrophysiology testing
Cardiac MRI
Ajmaline/flecainide tests
Genetic testing

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History

- Frequency
- Onset / offset characteristics
- Perceived rate - slow, fast, very fast/rapid
- Characteristics - regular or irregular
- Duration
- Associated symptoms- SOB, sweating, dizziness, hot, pre-syncope, syncope
- Aggravating / relieving factors

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History – RED FLAGS

- Alarm features (Referral indicated)

Exercise induced
Associated syncope
Chest pain
Family history of sudden cardiac death
Underlying structural heart disease

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History

- Drug history including OTC medicines

Decongestants (ephedrine)
Alcohol
Caffeine
Cardio-active drugs (QT interval ?)
Recreational drugs
Performance enhancing drugs

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Clinical history and findings with palpitations, and suggested diagnosis

History and findings	Suggested diagnosis
Missed beats, skipped beats, pounding, butterflies in the chest	Ectopy (supraventricular and ventricular ectopics)
Unable to catch breath, need to take a breath, single pounding sensations, coughing, fullness in the head, butterflies in the chest, heart is going to burst out of chest	Ventricular ectopics
Rapid, regular pounding in neck	Supraventricular tachycardia (SVT) / atrial arrhythmias
Palpitations worse at night	Ectopy, runs of SVE's, atrial fibrillation (AF)
Palpitations associated with exercise	SVT, VT,IHD
Positional palpitations	SVT/PAF
Heat intolerance, tremor, goitre	Hyperthyroidism
Palpitations since childhood	SVT
Rapid irregular rhythm, mixture of fast and slow beats	AF
Palpitations terminated with deep breathing, cold drinks, Valsalva, coughing	SVT
General anxiety	Panic attacks

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Thank you very much for referring this pleasant 71-year-old lady. On 6th May 2015, whilst playing badminton, she suddenly noticed that her heart was racing and did not settle. She felt lightheaded and felt that she could not breathe easily. There were no associated pre-syncopal or syncopal symptoms, chest pain or tightness. She described her palpitations as fast and irregular. She managed to drive home and her blood pressure machine recorded a systolic blood pressure of 100 mmHg and a pulse of 142 beats per minute. I believe there were some error messages initially trying to record her pulse rate (this is not unusual in the setting of atrial tachyarrhythmias). Her blood pressure is normally around 130/60. Her symptoms lasted for approximately 3 hours and gradually resolved. She has experienced no further subsequent symptoms or previous symptoms prior to this episode. She plays badminton twice a week and is quite active.

In 2011, after six immunization injections, prior to flying to South Africa, she woke the following morning with shortness of breath, and subsequently had a 24-hour tape and echocardiogram at the Hammersmith Hospital. These investigations, we believe

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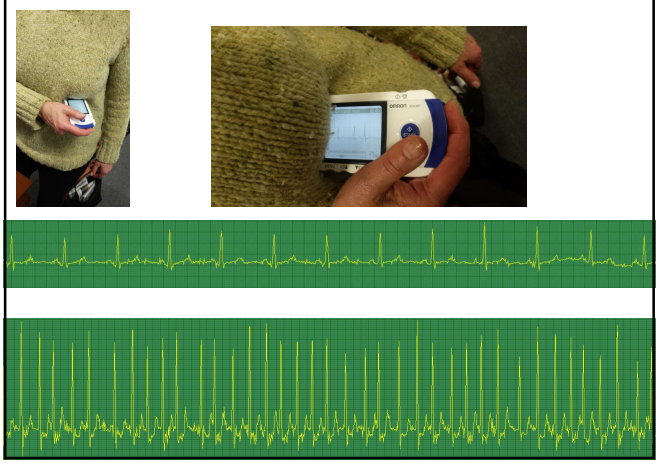

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Heart Failure and SOBE

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Heart Failure: CLINICAL PRESENTATION

Heart failure is a clinical syndrome caused by the heart working ineffectively as a pump to support the circulation. It is caused by structural or functional abnormalities of the heart.

Symptoms characterised by fluid retention

- SOBE
- Exercise effort intolerance
- Fatigue
- PND/orthoponea
- Leg/ankle oedema
- Hepatomegaly
- Ascites

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Primary Care Investigations

- BNP/ NT-pro BNP
- Perform an ECG (LVSD very unlikely if ECG normal, problems with confidence of interpretation in primary care, must be entirely normal or else loses reliability)
- Chest X-ray
- Blood tests (electrolytes, urea and creatinine, eGFR, thyroid function tests, liver function tests, fasting lipids, fasting glucose, full blood count), urinalysis, and peak flow or spirometry.
- Cardiomyopathy screen: above + B12, Ferritin, ANA, CK, ACE
- Imaging: echocardiography (open access)

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BNP/NT-pro BNP as a screening test for heart failure

- Marker of structural heart disease rather than systolic dysfunction.
- Age related increase
- BNP/NT-pro BNP is also raised in hypertension, AF, Valvular heart disease, diastolic dysfunction, acute coronary syndromes, stable angina, renal failure, cor-pulmonale, and PE's.
- Highly sensitive test for HF, stable for up to 72hours, 'bedside' testing available if desired, relatively inexpensive
- Low BNP/NT-pro BNP effectively rules out heart failure or LVSD, elevated BNP/NT-pro BNP indicates need for an echo/cardiac assessment

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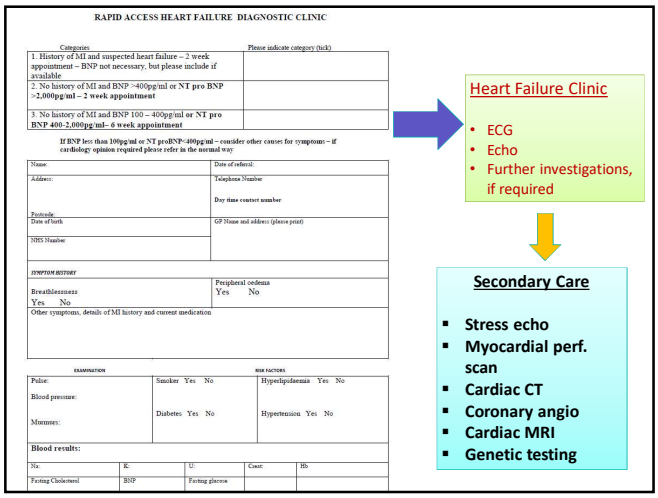
RAPID ACCESS HEART FAILURE DIAGNOSTIC CLINIC

Categories	Please indicate category (tick)
1. History of MI and suspected heart failure – 2 week appointment – BNP not necessary, but please include if available	
2. No history of MI and BNP >400pg/ml or NT pro BNP >2,000pg/ml – 2 week appointment	
3. No history of MI and BNP 100 – 400pg/ml or NT pro BNP 400-2,000pg/ml– 6 week appointment	

If BNP less than 100pg/ml or NT proBNP<400pg/ml – consider other causes for symptoms – if cardiology opinion required please refer in the normal way

Name:	Date of referral:
Address: Postcode:	Telephone Number Day time contact number
Date of birth	GP Name and address (please print)
NHS Number	
SYMPTOM HISTORY	
Breathlessness Yes No	Peripheral oedema Yes No
Other symptoms, details of MI history and current medication	

EXAMINATION		RISK FACTORS
Pulse: Blood pressure: Murmurs:	Smoker Yes No Diabetes Yes No	Hyperlipidaemia Yes No Hypertension Yes No

Blood results:

Na:	K:	U:	Creat:	Hb
Fasting Cholesterol	BNP	Fasting glucose		

Heart Failure Clinic

- ECG
- Echo
- Further investigations, if required

Secondary Care

- Stress echo
- Myocardial perf. scan
- Cardiac CT
- Coronary angio
- Cardiac MRI
- Genetic testing

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Syncope

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Syncope

History

- Complete Description
  - From patient and observers, mobile phones-videos
- Onset
- Associated Symptoms
- Posture
- Duration of Attacks
- Sequelae

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Neurally-mediated syncope

- Provocation, Prodromal, Postural
(Vasovagal, post-micturition, cough, swallow, defecation, blood drawing, post prandial)

- Absence of cardiac disease
- Long history of syncope
- After sudden unexpected unpleasant sight, sound, smell or pain
- Prolonged standing or crowded, hot places
- Nausea, vomiting, tunnel vision, tinnitus, yawning
- During or in the absorptive state after a meal
- With head rotation, pressure on carotid sinus (as in tumours, shaving, tight collars)
- After exertion

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Syncope caused by orthostatic hypotension

- After standing up
- Temporal relationship with start of medication leading to hypotension or changes of dosage
- Prolonged standing especially in crowded, hot places
- Presence of autonomic neuropathy – Diabetes Mellitus or parkinsonism
- After exertion

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Cardiac syncope

- Presence of severe structural heart disease
- During exertion, or supine
- Preceded by palpitation or accompanied by chest pain
- Family history of sudden death

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SYNCOPE RED FLAGS - suggest early / urgent referral

Syncope with:

- No warning
- With Exercise/ exertion.
- Palpitations (sequence of events important i.e. palpitations & then dizziness = more likely cardiac)
- Chest pain/ SOB
- Being supine
- Cardiac history
- Signs of heart failure
- Abnormal ECG
- Prolonged LOC, post recovery confusion for longer than a minute or so.
- FH of sudden death even neonatal deaths,? Cot deaths
- Frequent recurrence, severe injury or driving involvement e.g. PSV,HGV drivers.

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Syncope versus Epilepsy

	Epileptic seizure	Neurocardiogenic syncope
Symptoms pre event	Aura (déjà vu, jamais vu), chewing, lip smacking, abnormal stereotypical behaviour	Situational, nausea, vomiting, abdominal discomfort, dizziness, sweating, blurred vision. Improvement lying down
Findings during LOC	Tonic-clonic movement, 1-2min., rhythmic, hemilateral clonic movements	Myoclonic jerks~80%, <15-30 sec.,
	Blue	Pallor
Tongue biting	Common (side)	Uncommon/rare (tip)
Incontinence	Common	Common
Symptoms after the event	Prolonged confusion > 10min., aching muscles	Short duration (<30sec), nausea and vomiting

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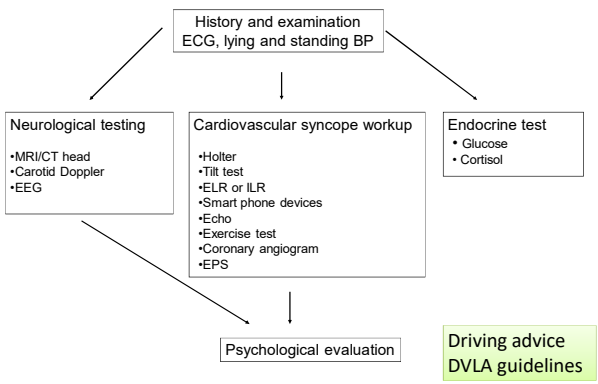
Syncope



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Dear

Re:

Diagnoses:

1. Neurocardiogenic syncope. Tilt test in 2005 at St Mary's Hospital reported by Professor Sutton and stated that although it was technically negative in that syncope was not induced, there was classical oscillation of blood pressure strongly suggesting a vasovagal diagnosis. A repeat tilt test on 11th February 2010 at the Brompton and Harefield NHS Trust was negative despite sublingual GTN
2. Dual-chamber pacemaker December 2007 for bradycardia (20 beats per minute) and pauses demonstrated on a Reveal device
3. Previous migrainous headaches and vertigo
4. Hiatus hernia
5. Mild asthma
6. Normal coronary angiogram and good LV systolic function January 2006
7. Functional diarrhoea
8. Reviewed by Consultant Neurologist for recurrent syncope in 2007 who felt that an epileptic cause was very unlikely and more likely to be hypotensive related syncope

came for a cardiology review today. I greeted her in the waiting room and as we were walking towards the clinic room while she was trailing behind me she suddenly collapsed to the floor. As I turned around she was lying on the floor, motionless with a palpable pulse with a reasonable volume. Her eyes were flickering and her colour remained normal. 20 seconds later she completely recovered without any sequelae or ongoing symptoms. By the time we got a blood pressure machine her blood pressure was 116/47 with a pulse of 66 beats per minute. She immediately got up and walked into the clinic room. We had our consultation as if nothing had happened.

I must confess given the sequence of events today, I was not convinced that there was any cardiac component to account for her syncopal episode and I wonder whether there may be an underlying emotional element. You may wish to explore this in further details.

Although she certainly does have a history of vasovagal syncope, I do not think the sequence of events today is likely to be cardiac in origin and for that reason I have not arranged any further follow-up appointments. Should you have any further queries or like to discuss her case in more detail, please do not hesitate to contact me.

Yours sincerely,

Dictated and verified by Doctor but not signed

Dr Azad Ghuran MB ChB, MRCP, MD
Consultant Cardiologist

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Hypertension

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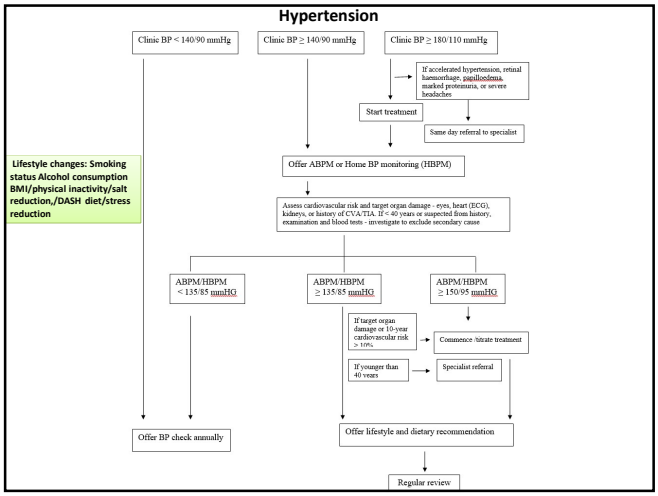

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BP Recommendation

Classification of Blood Pressure (Office BP*)

Recommendations	Class	Level
It is recommended that BP be classified as optimal, normal, high-normal, or grades 1–3 hypertension, according to office blood pressure.	I	C

Category	Systolic (mmHg)		Diastolic (mmHg)
Optimal	< 120	and	< 80
Normal	120–129	and/or	80-84
High normal	130–139	and/or	85-89
Grade 1 hypertension	140–159	and/or	90-99
Grade 2 hypertension	160–179	and/or	100-109
Grade 3 hypertension	≥ 180	and/or	≥ 110
Isolated systolic hypertension	≥ 140	and	< 90

*Conventional office BP rather than unattended office BP

www.escardio.org/guidelines

Williams B, Mancia G et al. Eur Heart J (2018); doi:10.1093/eurheartj/ehy339
Williams B, Mancia G et al. J Hypertens (2018); doi:10.1097/HJH0000000000001940

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Definition of hypertension according to office*, ambulatory, and home blood pressure levels

Category	SBP (mmHg)		DBP (mmHg)
Office BP*	≥ 140	and/or	≥ 90
Ambulatory BP			
Daytime (or awake) mean	≥ 135	and/or	≥ 85
Night-time (or asleep) mean	≥ 120	and/or	≥ 70
24-h mean	≥ 130	and/or	≥ 80
Home BP mean	≥ 135	and/or	≥ 85

*Conventional office BP rather than unattended office BP.

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Williams B, Mancia G et al. Eur Heart J (2018); doi:10.1093/eurheartj/ehy339
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2018 ESC-ESH Guidelines for the Management of Arterial Hypertension

Classification of hypertension stages according to BP levels, presence of CV risk factors, HMOD, or comorbidities

Hypertension disease staging	Other risk factors, HMOD, or disease	BP (mmHg) grading: High-normal SBP 130–139 DBP 85–89	Grade 1 SBP 140–159 DBP 90–99	Grade 2 SBP 160–179 DBP 100–109	Grade 3 SBP ≥ 180 DBP ≥ 110
Stage 1 (uncomplicated)	No other risk factors	Low risk	Low risk	Moderate risk	High risk
	1 or 2 risk factors	Low risk	Moderate risk	Moderate – high risk	High risk
	≥ 3 risk factors	Low –moderate risk	Moderate – high risk	High risk	High risk
Stage 2 (asymptomatic disease)	HMOD, CKD grade 3, or diabetes mellitus without organ damage	Moderate – high risk	High risk	High risk	High – very high risk
Stage 3 (established disease)	Established CVD, CKD grade ≥ 4, or diabetes mellitus with organ damage	Very high risk	Very high risk	Very high risk	Very high risk

ESC Congress Munich 2018

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Office Blood Pressure Treatment Target Ranges

Recommendations	Class	Level
It is recommended that the first objective of treatment should be to lower BP to < 140/90 mmHg in all patients, and provided that the treatment is well tolerated, treated BP values should be targeted to 130/80 mmHg or lower, in most patients.	I	A
In patients < 65 years receiving BP-lowering drugs, it is recommended that SBP should be lowered to a BP range of 120 to < 130 mmHg in most patients.[a]	I	A
In older patients (aged ≥ 65 years) receiving BP-lowering drugs:		
• It is recommended that SBP should be targeted to a BP range of 130 to < 140 mmHg.	I	A
• Close monitoring of adverse effects is recommended.	I	C
• These BP targets are recommended for patients at any level of CV risk and in patients with and without established CVD.	I	A

[a]Less evidence is available for this target in low–moderate-risk patients.

www.escardio.org/guidelines

Williams B, Mancia G et al. Eur Heart J (2018); doi:10.1093/eurheartj/ehy339
Williams B, Mancia G et al. J Hypertens (2018); doi:10.1097/HJH0000000000001940

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Hypertension

Investigation of patients with hypertension - baseline investigations

Blood tests - FBC, U&E, CREATININE, URIC ACID, LFT, gamma GT, Ca& P04, fasting GLUCOSE, fasting LIPIDS, TFT

ECG Presence of left ventricular hypertrophy

Urine tests Dip stick to test for CELLS, PROTEIN, BLOOD AND GLUCOSE

Ambulatory BP monitor / validated home BP monitor (BHS website)

Echocardiogram (open access) - Presence of left ventricular hypertrophy

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Hypertension

Patients requiring further investigation to exclude secondary causes

- Young age < 30 - 40yrs (particularly if end organ damage, CVD, renal disease of DM) and no risk factors
- Moderate/severe hypertension
- Presentation with hypertensive emergency
- Raised creatinine
- Blood, protein or cells in urine
- Low plasma K
- Variable hypertension
- Resistant hypertension - failure to respond to multiple antihypertensive drugs
- Large postural drop in blood pressure
- Sudden loss of BP control and non-dipping or reverse dipping on ABPM

Factors that can increase Blood Pressure

Medications

- NSAIDS
- Recreational drugs - Cocaine, Amphetamines
- Over the counter "cold" medication - phenylephrine
- Anabolic Steroids
- Oral Contraceptives

Excessive EtOH (>3-4 drinks/day)
High Salt Diet
Obesity
Sleep apnoea

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Hypertension

Criteria for requesting 24 hour urinary catecholamines excretion
- Clinical suspicion of phaeochromocytoma (headaches, palpitations and sweating)
- Moderate/severe hypertension
- Variable hypertension/postural hypotension
- Failure to respond to drug treatment

Criteria for renal investigations
- Clinical suspicion of renal disease
- Severe hypertension
- Young age <40yrs
- Raised creatinine
- Blood, protein or cells in urine
- Failure to respond to drug treatment

Which renal investigation?
- Renal U.S. if underlying renal disease suspected
- Renal CT angiogram, magnetic resonance angiography or invasive renal angiogram if renal artery stenosis is suspected.

Criteria for requesting plasma renin and aldosterone measurements

- clinical suspicion of 1° Hyperaldosteronism

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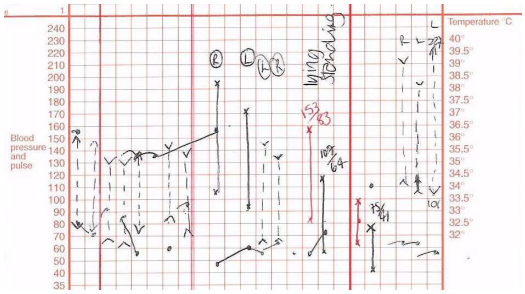
Phaeochromocytoma

52 yr. old female with episodic severe headache, photophobia, N&V, agitation, confusion, and aggression



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Hypertension

Symptoms	Possible cause
Low Potassium (excluding diuretic induced hypokalaemia) 50% of patients with Conn's do not have hypokalaemia. Low potassium brought on by a small dose of diuretic may be a clue.	Primary Hyperaldosteronism (Including Conn's) Secondary Hyperaldosteronism (e.g. Renal Artery Stenosis, renal artery fibromuscular dysplasia)
Cushingoid appearance, oligomenorrhorea, easy bruising	Cushing's Glucocorticoid treatment
Palpitations, sweats, postural hypotension, anxiety pale skin (pallor), blurred vision, weight loss, increased thirst and urination, constipation, abdominal pain, elevated glucose, red and white blood cells, psychiatric disturbances, and cardiomyopathy.	Phaeochromocytoma
Cardiac murmur without previous investigation Radiofemoral delay	Aortic coarctation
Resistant hypertension	Sleep apnoea, non-compliance

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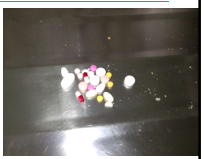
Re: [redacted] 50 yr. old female

Diagnoses:
1. Previous resistant hypertension due to non-compliance. Thoroughly investigated, has normal urinary catecholamines, plasma metanephrines, renal MRA, renal angiogram, renal aldosterone levels and immunology
2. CVA
3. COPD
4. Breast cancer
5. Coronary angiogram 26th July 2013 showed moderate disease in the mid LAD and diffuse mild plaque disease in the circumflex artery and diffuse plaque disease and moderate narrowing in the mid course of a dominant right coronary artery

I reviewed [redacted] today in clinic. We finally came to the cause of her resistant hypertension which no doubt was non-compliance with her medication. I am glad to say that since she has been changed to a dosette box her blood pressure is well controlled at 105/62 with a pulse 65 beats per minute. I have not arranged any further follow-up appointments but I will of course be happy to see her should the need arise.

Yours sincerely,

Dr Azad Ghuran MB ChB, MRCP, MD
Consultant Cardiologist



22 tablets found in the patient's bed side bin

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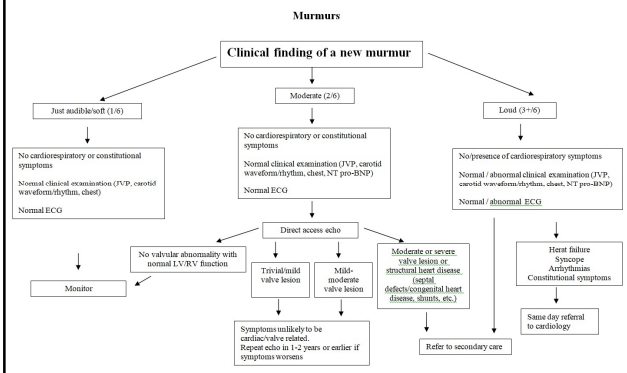
Murmurs



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Direct Echocardiogram Interpretation

Valve disease
- Mild mitral regurgitation: no further action required
- Isolated Tricuspid Regurgitation – treat with diuretics if symptomatic
- Mild aortic valve disease:
  - repeat echo and review symptoms after 5 years
- Moderate aortic or mitral valve disease:
  - without symptoms: repeat echo and review symptoms after 2 years, depending on patient age and comorbidities
  - with symptoms – consider cardiology referral
- Severe aortic or mitral valve disease: consider cardiology referral

Atrial fibrillation
- Valvular AF defined as AF with moderate-severe mitral stenosis

Dilated aortic Root and ascending aorta
- 40 mm: 2 yearly surveillance with echo (1 year if bicuspid Aortic Valve)
- 45 mm: consider cardiology referral

Heart Failure
Prescribe loop diuretic to treat symptoms
Reduced ejection fraction: prescribe and uptitrate ACE-I and B-blockers to help symptoms and prognosis
Consider cardiology referral for patients with:
- Severe heart failure (New York Heart Association [NYHA] class II-IV)
- Heart failure that does not respond to treatment in primary care or can no longer be managed in the home setting
- Heart failure resulting from severe valvular heart disease
- New left ventricular systolic impairment (ejection fraction of 45% or less)

Pulmonary Hypertension
- PA systolic pressure (PASP) > 50mmHg: consider referral to cardiology if abnormal left heart findings, otherwise consider referral to respiratory

Pericardial effusion
- Moderate: Consider cardiology referral
- Large effusion: consider referral for admission

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A Clinical Guide to Referring Cardiac Conditions in Primary Care

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