Cardiology

Objectives: To undertake a structured approach to the history, examination and investigation of patients presenting with symptoms that may be due to a cardiological cause. To be able to interpret the results of investigations such as ECG, chest x-ray and cardiac marker testing. See below for specific conditions.

Specific paediatric objectives: To have the knowledge and skills to be able to assess and initiate management of babies and children presenting to the Emergency department with cardiological disorders. To understand the life-threatening nature of some of these conditions and when to ask for the help of a cardiologist or those with more specialised expertise. To know the indications for cardiological investigations including ECGs at all ages and echocardiography.

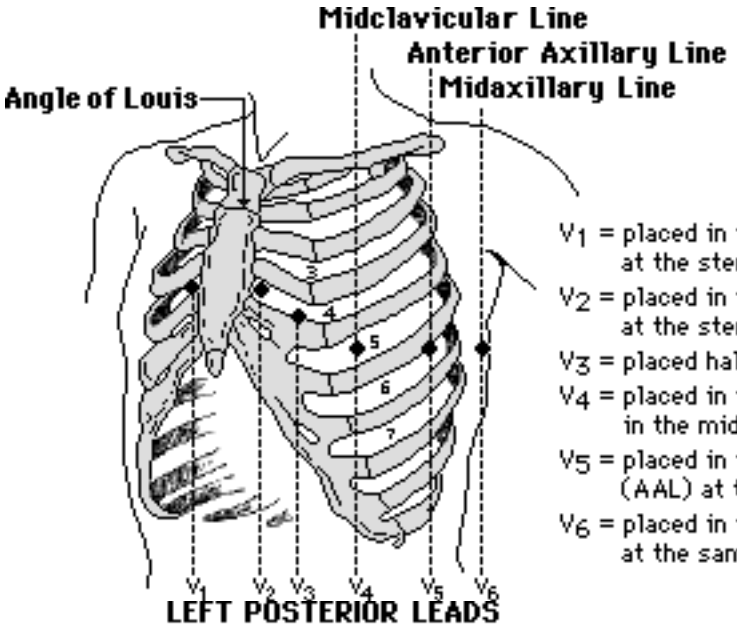
Problem	Knowledge	Skills / Attitudes	Learning	Assessment
Chest pain	Causes (cardiac/vascular, respiratory gastrointestinal, locomotor, psychological, trauma/ musculoskeletal, other)	Appropriate monitoring, treatment and investigation and be familiar with local guidelines for the management of patients with chest pain of possible cardiac origin and pulmonary embolism. To be able to risk stratify patients with chest pain and to be able to follow appropriate departmental pathways.	LP LT GT PS LS SL ODA ODB	OC MC CBD AUD ME FCEM MCEM

Acute coronary syndromes	Understand stable and unstable angina and myocardial infarction. (ACS) Pathophysiology of STEMI/ non STEMI. Recognise ECG changes related to ACS, including right ventricular infarct and posterior infarct. Indications, contraindications and complications of thrombolysis. Adjunctive treatments. Indications for interventional cardiology. Causes of ST elevation in the absence of myocardial infarction. Management of left ventricular failure in the setting of myocardial infarction. Management of cardiogenic shock Pharmacology of cardiac drugs.	Recognise the need for urgent assessment and prompt treatment with thrombolysis when indicated. To be able to obtain assent for thrombolysis. To identify and treat complications such as arrhythmias, pulmonary oedema and hypotension.	LP LT GT PS LS ODA ODB	OC MC CBD AUD ME FCEM MCEM
Patients presenting with syncope.	Causes (cardiac, neurological, endocrine and others) To be able to risk stratify. Appropriate diagnostic testing of patients with syncope.	To be able to identify those patients that require admission, those that need out patient follow up and those that can be safely discharged. To work with support services closely e.g. Syncope Clinics etc.	LP LT GT PS ODA	OC MC CBD ME FCEM MCEM

Patients presenting in heart failure.	Causes, precipitating factors and prognosis. Knowledge of which drugs to use, contraindications and side effects. Non-invasive ventilation. Understand pathophysiology of cardiac failure.	Initiate investigations to identify the cause. Initiate treatment including non-invasive ventilation. To be able to identify those who require invasive ventilation.	LP LT GT PS LS SL ODA	OC MC CBD ME FCEM MCEM
Arrhythmias	ECG recognition of narrow and broad complex tachycardias and bradycardias. Indications, contraindication and side effects of anti-arrhythmic drugs. Knowledge of ALS guidelines for management of arrhythmias. Recognition of complex arrhythmias, eg Wolff-Parkinson-White in AF Indications for pacing.	To recognise and correctly identify arrhythmias. Ability to perform carotid sinus massage. Explain the valsalva manoeuvre. Perform DC cardioversion. Manage arrhythmias according to Resuscitation Council Guidelines. Use of external pacing equipment. To be able to manage those patients haemodynamically compromised	LP LT GT PS LS SL ODA	OC MC CBD ME FCEM MCEM
Severe haemodynamic compromise	Cardiogenic shock, secondary to myocardial infarction, massive PE, aortic dissection, valve rupture etc. Emergency imaging including echocardiogram and CT. Role of thrombolysis / angioplasty / surgery. Use of inotropes.	Recognise the need for rapid assessment. Initiate investigation and treatment. Liaise with appropriate in-patient teams and co-ordinate investigation.	LP LT GT PS LS ODA	OC CBD ME FCEM MCEM

Other topics.	Endocarditis		LP	OC
	Implantable cardiac devices		LT	DOPS
	External and internal emergent cardiac pacing		GT	ME
			PS	FCEM
	Hypertensive emergencies		ODA	MCEM
	Disorders of the myocardium and pericardium		ODB	
	Valve disorders			
	Cardiac transplantation			
	Congenital abnormalities as they present in adults			
	Indications for exercise ECG testing			
Syncope in children	Understand the common causes of syncope	Be able to form a differential diagnosis for syncope	LP	OC
			LT	MC
		Be able to recognise those patients who need immediate treatment, investigations and admission and those who can be managed as outpatients	GT	FCEM
		Cardiology		

CURE.pdf



V_1 = placed in the 4th right intercostal space at the sternal border.

V_2 = placed in the 4th left intercostal space at the sternal border.

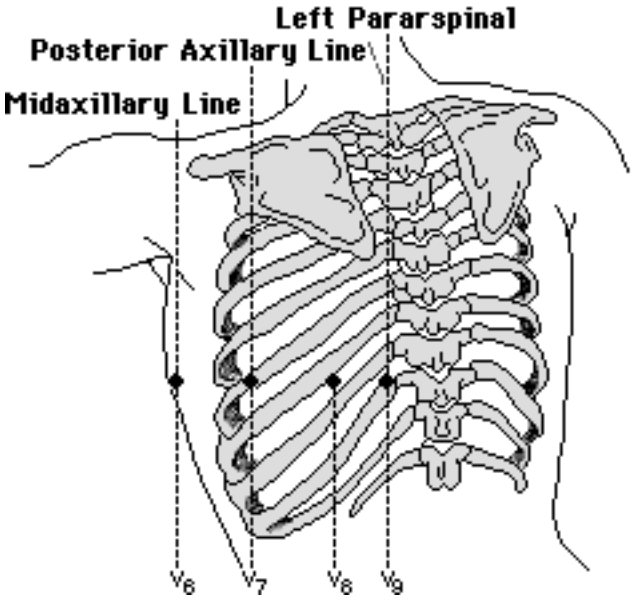
V_3 = placed halfway between V_2 and V_3.

V_4 = placed in the 5th left intercostal space in the midclavicular line (MCL).

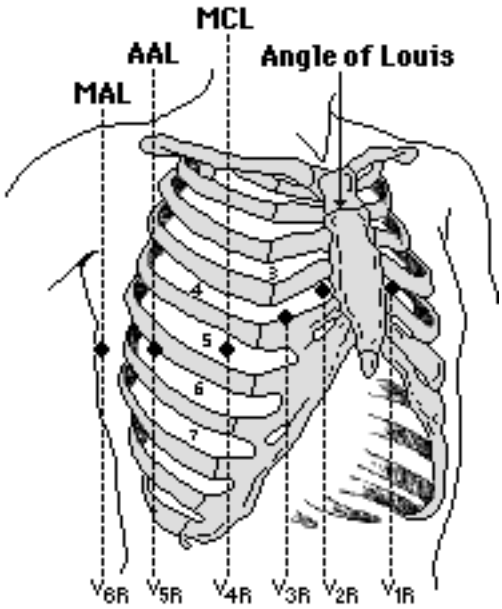
V_5 = placed in the anterior axillary line (AAL) at the same *horizontal* plane as V_4.

V_6 = placed in the midaxillary line (MAL) at the same *horizontal* plane as V_4.

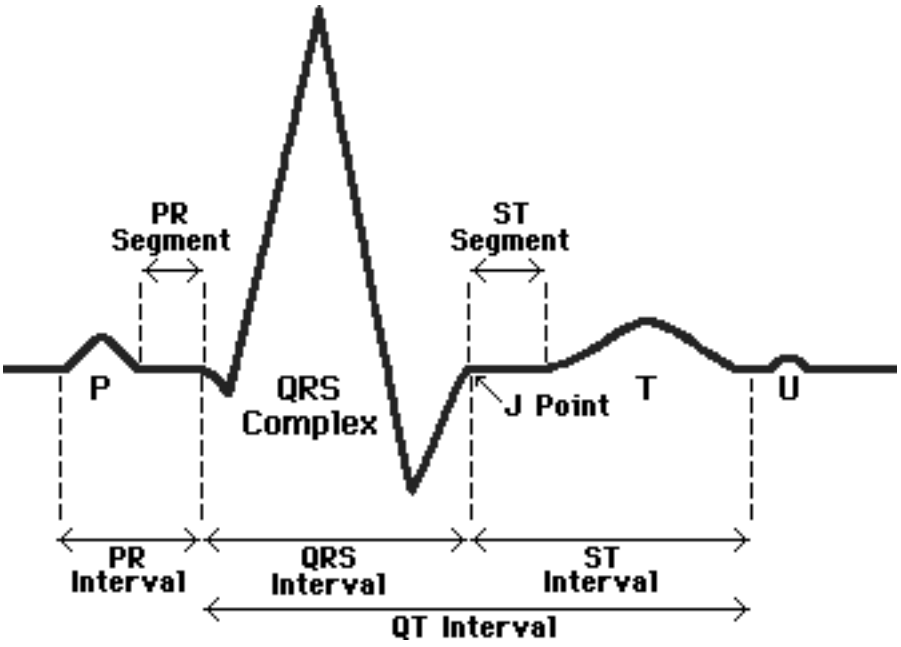
LEFT POSTERIOR LEADS



RIGHT PRECORDIAL LEADS



PR	0.12-0.22
QRS	0.08-0.11
QT	0.35-.42
QRS	0.08-0.11


PR
Segment
ST
Segment
P
QRS
Complex
J Point
T
U
PR
Interval
QRS
Interval
ST
Interval
QT Interval

The exercise ECG

Historical background

The present-day use of the exercise stress electrocardiogram in the diagnosis of coronary heart disease (in the form of the graded-exercise stress test—GXT) has evolved as a result of numerous observations and developments.

In 1908, Einthoven observed S–T depression after exercise but did not comment on it. In 1918, Blousfield recorded S–T-segment depression in leads I, II, and III during spontaneous angina. Feil and Siegel, in 1928, exercised patients known to have angina and observed S–T-segment and T-wave changes. Master and Oppenheimer, in 1929, developed an exercise test to assess 'circulatory efficiency' (using pulse and blood pressure) but did not use the ECG. In 1931, Wood and Wolferth described S–T changes associated with exercise, but felt that the test was too dangerous to use in patients with coronary disease. In 1932, Goldhammer and Scherf reported S–T depression in 75 per cent of patients with angina—a figure indicating a remarkably similar false-negative rate to that of current-day studies. In 1941, Master and Jaffe suggested that the ECG recorded before and after exercise could be used to detect 'coronary insufficiency'. Paul Wood and colleagues, in 1950, at the National Heart Hospital in London, described their experience of a test in which the patients had to run up 84 steps adjacent to the laboratory. They showed an 88 per cent reliability (compared with 39 per cent in the Master's test) and emphasized that the amount of work required should be adjusted to the patient's physical capacity.

The era of modern, stress testing began in 1956 when Bruce reported his findings and established guidelines for a standardized GXT procedure. Subsequently, the application of Bayesian techniques of analysis; the addition of nuclear techniques (myocardial scintigraphy and cardiac blood pool analysis) and echocardiographic stress testing; and the use on non-exercise stress

techniques (using dipyridamole, dobutamine, and adenosine) have all brought greater sophistication and applicability to cardiac stress testing.

This section will be confined to the use of the exercise stress ECG in the assessment of the heart and circulation and, in particular, to the role of the GXT in the detection and assessment of ischaemic heart disease.

Current usage

Although the exercise ECG may be used for several purposes, its commonest uses are in the diagnosis and assessment of ischaemic heart disease (IHD). In this respect, however, it is extremely important at the outset to recognize that the test has a significant false-negative rate, even in populations with an appreciable prevalence of IHD, and that the false-negative rate may be unacceptably high in populations with a low prevalence. The test is therefore of very limited value in screening low-risk, asymptomatic subjects. Most subjects who have undergone exercise stress testing as a screening procedure and who subsequently experience sudden cardiac death are found in retrospect to have had a normal exercise test result. A meta-analysis of 147 consecutive studies involving a total of 24 074 patients who had undergone both exercise stress testing and coronary angiography revealed sensitivities ranging from 23 to 100 per cent (mean 68) and specificities ranging from 17 to 100 per cent (mean 77). In patients with multivessel coronary disease the sensitivities ranged from 40 to 100 per cent (mean 81) and the specificities from 17 to 100 per cent (mean 66). For patients with single-vessel disease a positive GXT is most likely for lesions in the left anterior descending artery. Patients with lesions in the circumflex artery are least likely to give a positive result, while those with lesions in the right coronary artery occupy an intermediate position.

Exercise electrocardiography is also used in the estimation of prognosis in patients with known IHD, for

risk stratification following myocardial infarction, for screening of professionals in high-risk situations (e.g. pilots and professional athletes), and in the assessment of some cardiovascular symptoms (e.g. palpitations, tachyarrhythmias, and syncope) when these are exercise related. The database for the evaluation of the usefulness of the technique in these situations is less well established than is the case in relation to its use in the assessment of IHD.

Exercise testing in females

The specificity of exercise testing is less in women than in men. It seems likely that this is, in part at least, related to their lower prevalence of IHD. However, biological differences might be relevant. It has been suggested that oestrogens (with certain chemical structural similarities to digitalis) contribute to S–T-segment depression, but it has also been pointed out that women secrete more catecholamines during exercise than men. Both of these postulated mechanisms have been thought possibly to act via coronary vasoconstriction.

Risks

High-level exercise carries a cardiovascular mortality risk, and a maximal-exercise stress ECG is, basically, supervised high-level exercise. Inevitably, therefore, a GXT carries a risk, but multiple studies have shown the risk to be remarkably low. In 1971 a survey of 73 medical centres summarized the risks in relation to approximately 170 000 stress tests. A total of 16 deaths were reported (mortality rate 0.01 per cent), and 0.04 per cent required admission within 24 h because of arrhythmia or prolonged chest pain.

The risks are greater when the test is conducted soon after an ischaemic event. Even in this situation, however, the test is still remarkably safe. A survey of 151 941 tests undertaken within 4 weeks of acute myocardial infarction revealed a mortality rate of 0.03 per cent and a 0.09 per cent rate of non-fatal reinfarction or (successfully resuscitated) cardiac arrest.

Contraindications

Exercise stress testing is contraindicated to some extent whenever the pre-existing clinical state indicates a significantly increased risk of mortality or morbidity. In some situations the additional risk is so great as to constitute an absolute contraindication. In other situations the presenting clinical state indicates the need for more vigilant supervision than usual. Exercise, whilst not 'contraindicated', is of limited or negligible value in situations where abnormalities of the resting ECG make interpretation of the exercising record difficult or impossible.

Absolute contraindications

These include:

acute ischaemic syndromes: unstable angina, suspected acute myocardial infarction, known acute myocardial infarction within 5 days; known left main-stem stenosis; acute myocarditis; acute pericarditis; severe aortic stenosis; severe congestive cardiac failure; recent acute pulmonary oedema; current acute systemic illness; absence of trained supervisory staff or of resuscitation equipment; failure of the patient to understand the procedure or to give informed consent

Situations requiring intensive supervision

These include:

known severe coronary disease; known moderate or mild aortic stenosis; severe or moderate systemic hypertension; severe or moderate pulmonary hypertension; severe impairment of ventricular function; known history of ventricular tachycardia; known history of supraventricular tachycardia; existing second- or third-degree atrioventricular block; hypertrophic cardiomyopathy; severe congestive cardiomyopathy; known hypokalaemia.

Situations where interpretation of the exercising record is difficult or impossible

Abnormalities of the resting ECG that preclude effective interpretation of the exercising record include:

left bundle-branch block; ventricular pre-excitation; currently paced ventricular rhythm; widespread S–T,T changes; widespread QS complexes (especially across the precordial leads).

Procedures

Lead positioning

During exercise it is not possible to maintain adequate physical and electrical stability in relation to limb lead connections at their usual (for the standard 12-lead ECG) location. Instead, the 'limb' lead electrodes are positioned on the torso: with the right and left arm connections situated at the most lateral aspects of the respective infraclavicular fossa, and the right and left leg electrodes positioned halfway between the respective anterior iliac crest and the rib margin. This Mason–Likar modification of the standard 12-lead ECG results in a rightward shift of the axis, which is more marked in the standing than in the recumbent position. This rightward shift (typically giving an axis of +90° to +120°) sometimes results in the appearance of new q waves in aVL (but it should be noted that, whenever the mean frontal plane QRS axis is +90° or more positive, aVL becomes a 'cavity' lead and the finding of a q wave in a cavity lead is not abnormal).

Exercise protocols

Various exercise modalities can be used, including static or dynamic exercise, arm or leg exercise, and bicycle ergometry or treadmill procedures, but the commonest procedure by far is dynamic treadmill exercise. The most popular protocol is the Bruce protocol. This has a starting walking speed of 1.7 mph (1 km/h) at a 10 per cent slope, giving an oxygen consumption of about four metabolic equivalents, which in general use has proved very satisfactory. One major advantage of the Bruce protocol is that large diagnostic and prognostic databases exist for this test.

Exercise endpoints

Exercise is continued until one of the following endpoints is reached: subject wishes to stop (chest pain, dyspnoea, fatigue, leg weakness, light headedness, exhaustion, claudication); target endpoint is reached (target heart rate or exercise level); operator terminates the procedure: early or severe (>2 mm) S–T depression, S–T elevation, ventricular

tachycardia, second- or
third-degree heart block, fall in heart rate (20 beats/min or more), fall in blood pressure (20 mmHg or
more), perceived patient distress, failure of monitoring equipment.

Assessment of the exercise electrocardiogram

As the heart rate increases with exercise, the PR, QRS, and QT intervals all reduce in normal subjects.

The P-wave amplitude increases and the atrial repolarization wave (the Ta wave) increases in amplitude.

Atrial repolarization wave

Sinus tachycardia is associated with an increase in the depth and duration of the Ta wave. This gives a
curved upsloping segment between the QRS complex and the T wave, often misconstrued as S–T-
segment depression, and a common cause of an incorrect conclusion that an exercise test is positive. A
Ta wave can be recognized when it is noted that back-extrapolation of a depressed S–T segment shows it
to be continuous with downsloping depression in front of the QRS complex (Fig. 29)

Standard criteria for a positive test

By definition, a positive test occurs when 1 mm (0.1 mV) of horizontal or downsloping S–T depression
occurs during exercise (usually at peak exercise) or in the early recovery period. Upsloping S–T
depression is less reliably predictive of the presence of coronary disease than flat or downsloping S–T
depression. Greater (than 1 mm) degrees of S–T depression are more reliably predictive of coronary
disease, as are S–T depression occurring earlier in the exercise period, more prolonged S–T depression,
and a more widespread (within the ECG recording leads) S–T change. Figure 30 shows an example of
significant (2 mm) S–T depression in the left precordial leads.

Sometimes the S–T depression is most marked or only occurs during the recovery period (Fig. 31).

An example of a negative stress test is shown in Fig. 29.

Interpretation of the test result

Positive or negative. Pre- and post-test probability. Bayesian analysis

The criterion for positivity of an exercise ECG is widely accepted as being 1

mm of flat or downsloping S–
T segment depression during or early after exercise. The interpretation of a positive result is more
problematical. Usually the question being asked is whether or not the test result indicates a high
probability that the patient has coronary heart disease. Bayesian analysis of this problem indicates the
enormous impact of the prevalence of coronary disease in the population group from which the subject is
drawn (the prior probability of the condition) in answering this question. In essence, Bayes's theorem
states the self-evident truth that interpretation of the future (probability of disease in the given subject) is
helped by a knowledge of past experience (prevalence of the disease in the population from which the
subject comes) as well as present observations (the test result).
Bayesian analysis expresses the probability that a subject with a positive exercise test result does
actually have coronary heart disease, in terms of the sensitivity and specificity of the test and the
prevalence of the disease, as follows:
Probability = [prevalence × sensitivity]/[prevalence × sensitivity + (1 – prevalence) (1 – specificity)].
If one inserts reasonable (on the basis of published results of exercise testing) values for the sensitivity
(say 0.8, i.e. 80 per cent) and specificity (say 0.9, i.e. 90 per cent) into this equation and then looks at the
impact of variations in prevalence on the predictive value of a positive test, then the values shown in
Table 2 are obtained. Clearly the false-positive rate is very high in low-prevalence populations (the
healthy population) and this limits the value of exercise testing as a screening procedure in
asymptomatic, presumptively healthy groups.
The likelihood that a subject with a positive stress-test result has coronary artery disease (the 'post-test or
posterior probability') is therefore dependent on the prevalence of the disease in the population from
which the subject is derived (the 'pretest or prior probability'). Equally, of course, the likelihood that a

subject with a negative stress-test result does not have coronary artery disease (the 'post-test probability')
is also dependent on the prevalence of the disease in the population from which the subject is derived
(the 'pretest probability'). This concept is shown graphically in Fig. 32.
Degree of abnormality of the test result
The degree of abnormality of the stress-test result also has a powerful bearing on the predictive value of
the result. Greater or lesser degrees of abnormality may be shown by:
the depth of the S–T depression; the time of onset of the S–T depression; the duration of the S–T
depression; the number of ECG leads showing significant S–T depression.
Only in respect of the depth of S–T depression, however, is there currently a large database of
information. The effect of varying degrees of S–T depression on the predictive value of a positive test is
shown in Fig. 33.
Confounding ECGs
Interpretation of the exercise ECG is dependent upon the assessment of the timing, duration, degree, and
distribution of S–T depression occurring during exercise. When the pre-exercise ECG shows significant
S–T-segment abnormalities (left bundle-branch block, ventricular pre-excitation, ventricular paced rhythm,
non-specific S–T-segment depression, etc.), interpretation of changes in the S–T segments occurring
during exercise is virtually impossible. In these situations the exercise stress ECG makes no useful
contribution to the diagnosis of or to the exclusion of significant coronary artery disease.
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Age > 65 OR 2.2 >75 3-5
weight <70kg
hypertension

The Myocardial Infarction National Audit Project (MINAP) collects data that enable clinicians to examine the management of patients with AMI within their hospitals in comparison to the standards in the NSF for CHD. This national audit includes collection of the following data that are relevant to this guidance:

- thrombolytic drug used
- reasons for non-administration of thrombolytic treatment
- reasons for delay in the administration of thrombolytic treatment
- location for the administration of treatment
- who made the initial decision for treatment.

NICE Recommends Clopidogrel/Gp 2b/3a for high risk NSTEMI

Sits on fence with regards thrombolytics no particular drug recommended.

Better outcomes v thrombolytics especially
cardiogenic shock
RV involvement
prev CABG
high risk thrombolysis from advanced age
> 4hours after onset

Pericarditis.pages Pericarditis.pdf

30 % Inf MIs
diagnosed inf MI plus V4R ST elevation
Clinically High JVP clear chest hhypotension
vital maintain LV preload

Complications associated with Right Ventricular Infarction

- Shock.
- 2nd or 3rd degree heart block [indicates a poor prognosis & occurs in as many as 48 percent of right ventricular infarctions].
- Atrial fibrillation [1/3 of RVIs}.
- Ventricular arrhythmias.
- Ventricular septal rupture [in patients with right ventricular infarction and transmural posterior septal infarction].
- Right ventricular thrombus formation and subsequent pulmonary embolism,
- Tricuspid regurgitation
- Pericarditis [due to the frequent transmural injury of the relatively thin-walled right ventricle].
- Right-to-left shunt through a patent foramen ovale [should be suspected in patients who have hypoxemia that is not responsive to the administration of oxygen].
-
- Treatment of Right Ventricular Infarction.
    - **Strategy:**
    - **1. Maintain Right Ventricular Preload**
    - Volume load – eg. iv Hartmann's / Saline / Gelo
    - ***Although volume loading increases RAP and PCWP, it does not increase cardiac output***
    - Avoid nitrates, diuretics, morphine boluses [these ¯ preload]
    - **Maintain atrioventricular synchrony:**
    - AV sequential pacing for complete heart block
    - Prompt cardioversion for atrial fibrillation
    - **2. Inotropic support**

- Dobutamine is the agent of choice, then adrenaline or noradrenaline, dopamine.

- ***Dobutamine increases cardiac output, stroke volume index and RVEF, consequently unloading the right ventricle.***

- **3. Reducing Right ventricular afterload**

- Intraaortic balloon counterpulsation

- Vasodilators [sodium nitroprusside]

- ***Caution: these may also ¯ LV preload and thus cardiac output.***

- **4. Reperfusion**

- Thrombolytic Agents

- Direct angioplasty

Causes
Collapse.pdf
Syncope review EMJ 2006.pdf

Table 1 Our emergency department's existing syncope guidelines based on the European Society of Cardiology,9,10 American College of Physicians6,7 and American College of Emergency Physicians guidelines8

High risk (admit)	**Medium risk (consider discharge with early outpatient review)**
History findings	
Palpitations related to syncope	Age >60 years
Associated chest pain	No prodromal symptoms
Associated headache	Previous myocardial infarct
Related to exertion	Known history of valvular heart disease
Family history of sudden death at <60 years	Known angina/coronary artery disease
Previous history of VT/VF/cardiac arrest	Known history of congestive cardiac failure
Examination findings	
Systolic heart murmur heard	>20 mm Hg drop on standing
Signs of heart failure present	Diastolic heart murmur heard
Systolic BP <90 mm Hg	Ventricular pause >3 s on carotid sinus massage
Suspicion of pulmonary embolism	Trauma associated with collapse
AAA detected	
New neurological signs on examination	
Suspicion of CVA or SAH	
FOB present on PR	
Other suspicions of GI bleed	
ECG findings	
Mobitz type II heart block	Right bundle branch block
Wenkebach heart block	QRS duration >120ms
Bifascicular block	Old T wave/ST segment changes
Complete heart block	Frequent pre-excited QRC complexes
Sinus pause >3 s	Q waves unchanged from old ECG
New ST elevation ventricular tachycardia	Atrial fibrillation or flutter
Sinus bradycardia <50	PR >200 ms (first-degree heart block)
Sinoatrial block	
QTc >450 ms	**Low risk (consider discharge)**
NEW T wave/ST segment changes	None of the above characteristics
Brugadas (ST segment elevation V1–V3)	
Arrhythmogenic right ventricular dysplasia	

AAA, abdominal aortic aneurysm; BP, blood pressure; CVA, cerebrovascular accident; FOB, faecal occult blood; GI, gastrointestinal; PR, rectal examination; SAH, subarachnoid haemorrhage; VF, ventricular fibrillation; VT, ventricular tachycardia.

San Francisco Syncope Rule: CHESS

Congestive heart failure history of

Hematocrit less than 30 percent

Abnormal ECG (not sinus rhythm or new changes compared with the previous ECG)

fShortness of breath

Systolic blood pressure of less than 90 mm Hg at triage

TIMI RISK SCORE for UA/NSTEMI

HISTORICAL	POINTS
Age ≥ 65	1
≥ 3 CAD risk factors (FHx, HTN, ↑ chol, DM, active smoker)	1
Known CAD (stenosis ≥ 50%)	1
ASA use in past 7 days	1
PRESENTATION	
Recent (≤24H) severe angina	1
↑ cardiac markers	1
ST deviation ≥ 0.5 mm	1

RISK SCORE = Total Points (0 - 7)

RISK OF CARDIAC EVENTS (%) BY 14 DAYS IN TIMI 11B*

RISK SCORE	DEATH OR MI	DEATH, MI OR URGENT REVASC
0/1	3	5
2	3	8
3	5	13
4	7	20
5	12	26
6/7	19	41

*Entry criteria:UA or NSTEMII defined as ischemic pain at rest within past 24H, with evidence of CAD (ST segment deviation or +marker)

For more info go to www.timi.org

Antman et al *JAMA 2000*; 284: 835 - 842

TIMI RISK SCORE for STEMI

HISTORICAL	POINTS
Age ≥ 75	3
65-74	2
DM or HTN or angina	1
EXAM	
SBP < 100 mmHg	3
HR >100 bpm	2
Killip II-IV	2
Weight < 67 kg (150 lb)	1
PRESENTATION	
Anterior STE or LBBB	1
Time to Rx > 4 hrs	1

RISK SCORE = Total points (0 -14)

RISK SCORE	30-DAY MORTALITY IN InTIME II(%)*
0	0.8
1	1.6
2	2.2
3	4.4
4	7.3
5	12
6	16
7	23
8	27
>8	36

*Entry criteria: CP > 30 min, ST ↑, sx onset < 6hrs, fibrinolytic-eligible

For more info go to www.timi.org

Morrow et al. *Circulation* 2000; 102:2031-7