Heart Failure

Version 2 Final

Document control

Version history

Version	Date	Comments
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2a		Initial Draft

Changes since last version

1. Introduction

Definition

Heart failure is a clinical condition which occurs when an abnormality of cardiac function causes failure of the heart to pump blood at a rate sufficient for metabolic requirements under normal filling pressure**.** [1] In practical terms the definition is restricted to those patients whose condition is sufficiently severe to cause signs or symptoms.

Classification

Clinically heart failure may be in one of three categories:-

LEFT sided failure
RIGHT sided failure
BIVENTRICULAR (or congestive cardiac) failure

The consequences of heart failure can be divided into those arising from inadequate cardiac output (forward failure) and those arising from increased filling pressure with fluid retention causing either pulmonary or systemic venous congestion (backward failure).

Description

Heart failure manifests itself clinically with breathlessness, effort intolerance, fluid retention, malaise and poor survival. [1]

Heart failure may arise as a consequence of a myocardial, valvular, pericardial, endocardial or a conduction problem (or some combination of these).

In contrast with chronic heart failure, the term acute heart failure is often used to mean acute (cardiogenic) dyspnoea characterised by signs of pulmonary congestion including pulmonary oedema.

Associated physiological changes in many organ systems occur and cause biochemical, metabolic and functional impairments.

Aetiology

The underlying diagnosis and aetiology must be sought in patients presenting with heart failure syndrome.

A syndrome is a constellation of symptoms and signs and is not a single disease. This is the only way in which optimum treatment can be provided i.e. the treatment varies depending on whether the underlying cause is myocardial dysfunction, valve disease or some other aetiology.

The commonest cause of heart failure is myocardial dysfunction which is commonly systolic, i.e. there is reduced left ventricular contraction. Around two thirds of these cases result from coronary heart disease (CHD) and there is often a past history of myocardial infarction (MI).

The remainder have a non-ischaemic cardiomyopathy, which may have an identifiable cause (e.g. hypertension, thyroid disease, valvular disease, alcohol excess, or myocarditis) or may have no known cause (e.g. idiopathic dilated cardiomyopathy). [2]

Prevalence

Both the incidence and prevalence of heart failure increase with age. Studies of heart failure in the USA and Europe have found that under 65 years of age, the annual incidence is 1:1000 for men and 0.4:1000 for women.

Over 65 years of age, the annual incidence is 11:1000 for men and 5:1000 for women.

Under 65 years of age, the prevalence of heart failure is 1:1000 for men and 1:1000 for women and over age 65 years the prevalence is 40:1000 for men and 30:1000 for women.

The prevalence of asymptomatic left ventricular systolic dysfunction (LVSD) is 3% in the general population. The mean age of people with asymptomatic LVSD is lower than that for symptomatic individuals. Both heart failure and asymptomatic LVSD are more common in men.

Systolic heart failure is caused when the heart does not pump out enough blood.
The prevalence of diastolic heart failure (caused when the heart does not fully relax, so it does not fill properly with blood) in the community is unknown.

The prevalence of heart failure with preserved systolic function in people in hospital with clinical heart failure varies from 13 – 74%. Fewer than 15% of people with heart failure under 65 years of age have normal systolic function, whereas in people over 65 years of age normal function is maintained in about 40%

The ageing of the population in Western society has meant that heart failure is becoming increasingly common.

Recent improvements in the management of myocardial infarction and coronary artery disease, with an increased survival rate, have led to the development of more heart failure in later life. [1]

2. Diagnosis

The presentation of heart failure is variable. It depends on the severity of the disease and the associated clinical disorders.

Diagnosis may be difficult in the early stages when symptoms are non-specific.

The important points in the diagnosis are:

- High index of suspicion
- Thorough history taking
- Directed clinical examination.

History and Symptoms

The main presenting symptoms are:

- Reduced exercise capacity
- Dyspnoea (wheeze, orthopnoea, paroxysmal nocturnal dyspnoea (PND)
- Reduced appetite
- Weight loss.

The clinical symptoms occur as a result of the effects of the impaired ventricular function on the various organs. There is an increase in pulmonary pressure with an accumulation of fluid leading to an increase in the work of breathing.

The oedematous swelling may also cause a non-asthmatic bronchial constriction mimicking asthma.

Increasing pulmonary hypertension may eventually cause gross dyspnoea, lung crackles and pink frothy sputum in acute failure.

In chronic failure, the patient remains dyspnoeic but with less well marked lung changes.

Heart failure can cause expiratory airflow reduction, especially at night. This may cause confusion in differentiating heart failure from asthma.

Informal Observations

In chronic heart failure the patient will tend to walk slowly, with signs of breathlessness and fatigue. The observed tachypnoea with the use of accessory muscles of respiration and gasping gives an indication of the severity of heart failure and disability.

These observations will be noticeable on climbing on and off the examination couch, dressing and undressing, and walking up stairs. At more severe degrees of disablement they will be apparent on walking on the flat and ultimately even at rest.

Clinical Signs

The important signs on examination are:

- Abnormal blood pressure
- Forceful apex beat
- Abnormal pulse
- Respiratory crackles
- Oedema
- Abnormal heart sounds
- Raised JVP
- Hepatomegaly/ascites
- Pleural effusion
- Parasternal heave.

A similar picture can develop in conditions with volume or pressure overload such as:

- Severe anaemia
- Overtransfusion
- Arteriovenous malformation
- Shunts
- Prosthetic valve dysfunction

Investigations

Basic early investigations are necessary to differentiate heart failure from other conditions and to provide prognostic information. Urinalysis, serum urea and creatinine tests may help to determine if there is kidney failure since symptoms of kidney failure are similar to those of CHF (chronic heart failure).

Chest X-ray may indicate signs of CHF such as cardiomegaly, pulmonary congestion or pleural effusion and also non-cardiac indications such as lung tumours which account for breathlessness.
Other basic investigations should include:-
Full blood count
Fasting blood glucose
Serum urea and electrolytes
Thyroid function.

Further Investigations

Following clinical examination and basic investigation a decision should be made as to whether the patient should undergo echocardiogram. To help make this decision the patient should undergo either:-
Electrocardiogram (ECG)
or
Brain natriuretic peptide (BNP) test
or both.

ECG

The ECG is used firstly as a screening test to assess the likelihood of CHF and the need for subsequent echocardiography to confirm or refute a diagnosis
It is unusual for a patient with Chronic Heart Disease to have a normal ECG. The ECG changes reported in CHD are non-specific and common in elderly patients. CHD is a disease of the heart caused by decreased blood flow to the heart muscle which may result in myocardial infarction, or in angina but not necessarily with failure.

Electrocardiograph abnormalities seen in CHD may include:-
Pathological Q waves
Left bundle branch block
Left ventricular hypertrophy (LVH)
Atrial fibrillation
Non-specific ST and/or T wave changes

In CHF there has been described association of peripheral (o)edema (PERED), sometimes even imperceptible on physical examination, of patients with CHF, and attenuation (ATTEN) of the amplitude of P-waves, QRS-complexes, and T-waves.
Electrocardiography is also useful once the diagnosis of CHF has been confirmed as it may help to determine the cause (e.g. Q waves indicate previous MI, LVH is seen in hypertension and aortic valve disease) and it is important to exclude atrial fibrillation.

B-Type Natriuretic Peptide

Brain natriuretic peptide and N terminal-pro-BNP (NT-proBNP) are peptide hormones produced in the heart by breakdown of a precursor protein (pro-BNP). BNP promotes natriuresis (the process of excretion of sodium in the urine via action of the kidneys), diuresis, vasodilatation and muscle relaxation. NT-proBNPis inactive.

Plasma BNP and NT-proBNP tend to rise in patients with heart failure and the concentrations tend to rise with New York Heart Association (NYHA) classification. (see Appendix C)

Echocardiography

Echocardiography is a safe and relatively inexpensive investigation which is very helpful in diagnosing heart failure and determining the cause.
It may reveal previously undiagnosed valve disease.
It provides a semi-quantitative assessment of left ventricular systolic and diastolic function, valve disorders can usually be accurately delineated, and pulmonary artery systolic pressure can be estimated.
As it may not be feasible or cost effective to refer all patients with suspected heart failure for echocardiography, screening with either ECG and/or BNP is desirable. Brain natriuretic peptide testing has the practical advantage of being a simple blood test.

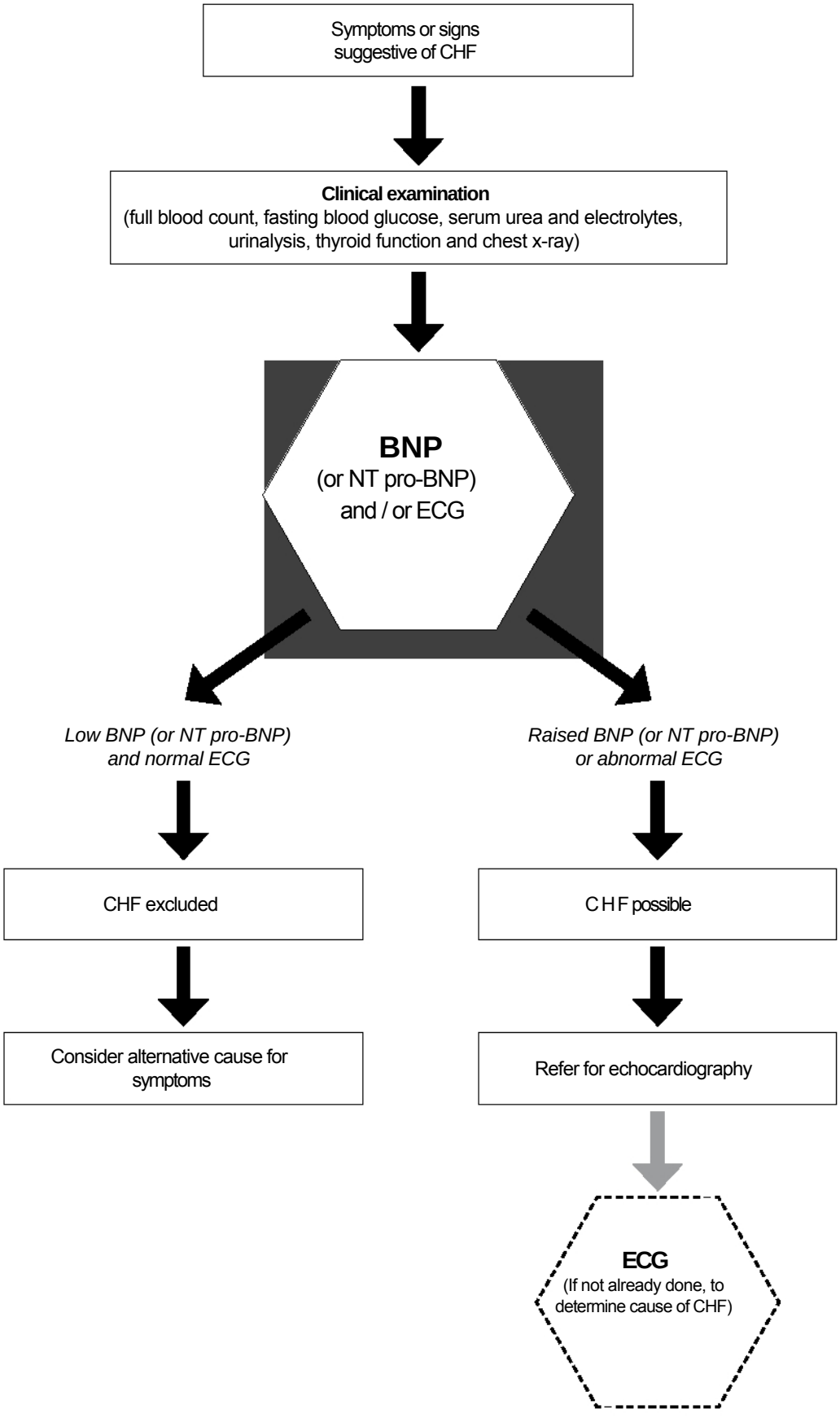
See diagram below. [2]

Chest X-ray

This is still considered important in the assessment of left heart failure. The rise in pulmonary venous pressure first shows as a distension of the upper pulmonary lobe veins.

Subsequent changes include the development of Kerley B lines.

Diagnostic algorithm for patients with suspected chronic heart failure



3. Management

Lifestyle Changes

The mainstays of treatment are listed in Appendix B and include:

- General life style advice
- Treatment of any underlying cause(s)
- Drug therapy.

General Measures

Advice should be given as to:

- Influenza and pneumococcal immunisation,
- Abstinence from smoking,
- Moderation of alcohol intake,
- Avoidance of harmful drugs.
- Restriction of salt intake may be useful in some patients

Adequate patient education is important, and the patient should be made aware of the necessity of any required lifestyle modifications and of the need for any regular medication.

Exercise training

This can improve functional capacity by 15 - 20% and reduce cardiac events. It is advised in those with stabilised mild to moderate heart failure. [4] [7]

Concurrent Illness

Any associated disease (e.g. hypertension, hypothyroidism, coronary artery disease, and cardiomyopathy) which may be contributing to the heart failure should be treated.
(see Appendix A)

Mood Disorders

Depression is common in patients with chronic heart failure and is associated with an increased risk of mortality in some but not all studies and may be related to morbidity and rehospitalisation.

Screening in heart failure may help to identify patients who are at poorer prognostic risk. If antidepressant medication is to be used then a tricyclic antidepressant should not be used in patients with chronic heart failure.

Drug Therapy

Drug therapy, and in particular balanced vasodilatation, leads to the greatest clinical benefit.

Angiotensin Converting Enzyme Inhibitors

ACE inhibitors (such as enalapril or ramipril): These improve function and reduce mortality by 23% and hospital admissions by 35%. [2] They are used as first line treatment in patients with a reduced left ventricular (LV) ejection fraction. If used in asymptomatic LV dysfunction, fewer develop heart failure. [4] [11] Side effects of cough, the development of allergy, renal impairment or hyperkalaemia may limit their use.

Beta Blockers

β-blockers: Combining β-Blockers with ACE inhibitors can reduce hospital admissions and mortality. Careful titration of the dose is needed to avoid bradycardia and hypotension.
Bisoprolol and Carvedilol have been shown to reduce mortality. Treatment should be initiated under specialist supervision
There is consistent evidence for positive benefits from beta blockers in patients with heart failure, with a risk of mortality from cardiovascular causes reduced by 29%, mortality due to pump failure reduced by 36% and all cause mortality reduced by 23%. [2]

Angiotensin Receptor Blockers

Angiotensin II type1 receptor blockers (ARBs) block the biological effect of angiotensin II, mimicking the effect of ACE inhibitors.
Unlike ACE inhibitors they do not produce cough as a side effect and should be used in patients who cannot tolerate an ACE inhibitor. Candesartan is the drug of choice.

Aldosterone Antagonists

Aldosterone produces many adverse extra-renal effects, for example on vascular function and myocardial fibrosis.

The RALES trial demonstrated that adding the aldosterone antagonist Spironolactone to an ACE inhibitor reduced all cause mortality by 30% and cardiac mortality by 31%. The frequency of hospitalisation for worsening heart failure was 35% lower in patients receiving Spironolactone

Diuretics/ Loop Diuretics/ Metolazone

Diuretics: These are essential for the treatment of fluid overload and can cause a rapid improvement in symptoms and exercise tolerance. Both thiazides (such as bendroflumethiazide) and loop diuretics (such as furosemide) are used, often with an ACE inhibitor. Side effects such as gout or urinary retention in subacute prostatism may become a problem.

Digoxin

A Cochrane review has shown a 64% improvement in symptoms and a 23% reduction in hospitalisation for patients receiving Digoxin. Digoxin did not improve survival.

Evidence of benefit must be weighed against the possibility of an increase in sudden death due to toxicity associated with Digoxin.

In patients with heart failure and atrial fibrillation a beta-blocker is preferred for control of ventricular rate, though digoxin may be used initially while the beta-blocker is being introduced.

If excessive bradycardia occurs with both drugs then digoxin should be withdrawn. [2]

Summary of the use of major drug classes in the treatment of Heart Failure [2]

Class	Prescribe
NYHA I	**ACE inhibitor** **Beta blocker**
NYHAII-III	**ACE inhibitor** **Beta blocker** **Candesartan (initiation requires specialist advice)**
NYHA III-IV	**ACE inhibitor** **Beta blocker** **Spironolactone (initiation requires specialist advice)**

Other Measures

Bed rest for a short period is a common treatment for acute failure. However it is preferable that the patient be treated in the sitting position rather than lying prone.

In cases of severe, intractable failure, surgical intervention such as cardiomyoplasty or cardiac transplantation may be considered. [9]

Prognosis

The prognosis of heart failure is poor, with 5 year mortality ranging from 26% – 75%. Up to 16% of people are readmitted with heart failure within 6 months of first admission.

In the USA, heart failure is the leading cause of hospital admission among people over 65 years of age.

In people with heart failure, a new myocardial infarction increases the risk of death by nearly eight fold.

(Relative risk 7.8; [95% Confidence interval 6.9 to 8.8]).
About a third of all deaths in people with heart failure are preceded by a major ischaemic event. Sudden death, mainly caused by ventricular arrhythmia, is responsible for 25 – 50% of all deaths, and is the most common cause of death in people with heart failure.

This is being constantly improved with increasing use of implanted intracardiac defibrillators (AICDs).

The presence of asymptomatic LVSD increases an individual's risk of having a cardiovascular event.

One large prevention trial found that the risk of heart failure, admission for heart failure, and death, increased linearly as the ejection fraction fell.
Relative risk for heart failure was 1.20 (95% confidence interval 1.13 to 1.26).

Relative risk for hospital admission was 1.28 (95% confidence interval 1.18 to 1.38).

For each 5% reduction in ejection fraction the relative risk for mortality was1.20 (95% confidence interval of 1.13 to 1.29).

The annual mortality for people with diastolic heart failure varies in observational studies (1.3% – 17.5%).

The reasons for this variation appear to include age, the presence of coronary artery disease, and variation in the partition value used to define abnormal ventricular systolic function.

The annual mortality for left ventricular diastolic dysfunction is lower than that found in people with systolic dysfunction. [1]

4. Main Disabling Effects

General Considerations

The actual disability due to chronic heart failure is very variable, and each patient must be assessed individually.

In the early stages, there is very little impairment of daily function. The decrease in exercise tolerance in severe heart failure can be very limiting.

Functional capacity can be improved by alteration in general and lifestyle factors and most notably by drug treatment.

One of the most marked changes in the skeletal system is a substantial reduction in total skeletal muscle bulk, and subtle changes may be functionally important in heart failure.

Patients frequently complain that muscle fatigue is a major limitation to their daily performance. Some patients with chronic failure may benefit from exercise training. In a patient with stable chronic heart failure and no evidence of exercise induced ventricular arrhythmia, regular exercise should be encouraged. [5]

For those in employment, heavy industry where the employee is required to climb stairs or carry heavy weights (e.g. 20 kg) may be impossible, but lighter or sedentary work may be well within their capabilities (see Appendix D).

An ability to reach Stage 4 of the Bruce Protocol (see Appendix E) is judged to place an individual at low risk of sudden cardiac events. This is reflected in the DVLA guidelines which allow vocational driving at this stage. These guidelines are now being applied more widely to other occupations where there may be a significant risk attached to a sudden cardiovascular collapse.

Assessing the Claimant

Clinical examination findings in mild to moderate heart failure do not correlate well with functional ability and the assessment is best made from the evidence of:

1. The History of Activities of Daily Living (Typical Day) taking variation into account.

2. Informal Observation of the claimant's activities at examination.

3. Medication taken (see Appendix B.) and attendance at Cardiac Clinic.

Claimants with well treated mild and moderate failure are likely to retain the ability to self care, and although exercise tolerance may be reduced should be able to walk to local shops or round a supermarket.

Severe heart failure can cause appreciable functional impairment. It is likely that in these cases the claimant would be under active specialist care and be on maximum doses of medication. Significant abnormalities would be present on clinical examination.

The drugs used in the treatment of heart failure usually have few side effects, but can occasionally have a functional effect e.g. causing postural hypotension or polyuria with diuretics.

Psychological factors may sometimes be very important in the disability due to heart failure. An individual may have only mild physical symptoms, but be significantly restricted due to depression or fear of an untoward event. Similarly, the family may try to protect the patient and erroneously discourage any physical activity. [10]

In the IB-PCA, the functional areas first affected are Walking Up and Down Stairs, and then Walking.

Later the effort of mild exertion limits activities, such as dressing and undressing, washing, rising from sitting and walking even a few steps.

Eventually even minimal effort is not tolerated and there will be breathlessness at rest.

Exemption from the assessment should be considered if the limitation of effort tolerance is severe and progressive, causing significant limitation of normal daily activities such as self care tasks.

Appendix A - Causes of Heart Failure

Most Common Cause:

- Ischaemic Heart Disease

Other Causes:

- Hypertension
- Cardiomyopathy (familial, infective, toxic)
- Rheumatic heart disease.
- Valvular disorders
- Arrhythmias
- Endocarditis
- Endocrine disorders.

Rare Causes:

- Nutritional deficiencies (e.g. thiamine, iron)
- Infiltrative conditions (e.g. sarcoid, neoplasms)
- Myocardial fibrosis

Appendix B - Treatment of Chronic Heart Failure

General Measures.

- Achieve optimal weight
- No smoking
- Encourage exercise
- Discourage added salt
- Treat hypertension if appropriate
- Discourage excess alcohol
- Detect and treat associated risk factors (e.g. diabetes, myxoedema, hyperlipidaemia)

Mild Failure

- Thiazide loop diuretic
- ACE inhibitor
- Beta blocker for atrial fibrillation

Moderate Failure:

- Loop diuretic or combination of diuretics
- ACE inhibitor
- Beta blocker

Severe Failure:

- ACE inhibitor
- Beta blocker
- Spironolactone
- Digoxin
- Cardiomyoplasty
- Transplant

Appendix C - Functional Classification of Cardiac Diseases

New York Heart Association Functional Classification of Cardiac Disease

Grade 1 Ordinary activity does not cause symptoms of undue fatigue, palpitations, dyspnoea, or anginal pain.

Grade 2 Greater than ordinary physical activity results in symptoms. (Relevant for PCA)

Grade 3 Ordinary physical activity results in symptoms. (May be exemptible for PCA)

Grade 4 Symptoms at rest, and worse with any physical activity. (Exemptible for PCA. May be relevant for DLA/AA)

Appendix D - Haskell Work Classification

Type of Work	Activity
Very Heavy	Climb stairs
Medium	Carry 50 lbs. (22.5 kg)
Light	Carry 20 lbs. (9 kg)
Sedentary	Sit/carry 10 lbs. (4.5 kg)

This is a standard grading of work effort requirement.

The critique that it does not take into account the occasional increased episodic requirements for normal jobs is entirely reasonable. [13]

The classification can be linked to graded exercise tests (see Appendix E)

Appendix E- Bruce Protocols

Full (Standard) Bruce Protocol

Stage	Speed (mph)	Gradient (%)	Duration (min.)	Cumulative time (mins	METS
I	1.7	10	3	3	5
II	2.5	12	3	6	7
III	3.4	14	3	9	10
IV	4.2	16	3	12	13
V	5.0	18	3	15	16
VI	5.5	20	3	18	19
VII	6.0	22	3	21	22

This is a standard graded exercise test, and is a good measure of exercise tolerance in cardiac disease.

It may be too strenuous for the deconditioned subject, in which case the modified Bruce protocol can be used.

The energy used to accomplish a particular task can be calculated in METS (a measure of energy expenditure as a multiple of resting energy expenditure).

Haskell Work Classification (Appendix D)

Task Grade	Peak METs	Activity
Very Heavy	>6	Climb stairs
Medium	4-6	Carry 50 lbs (22.5 kg)
Light	2-4	Carry 20 lbs (9 kg)
Sedentary	<2	Sit. Carry 10 lbs (4.5 kg)

Modified Bruce Protocol

Stage	Speed (mph)	Gradient (%)	Duration (mins.)	Cumulative time(mins.)	METS
1	1.7	0	3	3	2
II	1.7	5	3	6	3
III	1.7	10	3	9	5
IV	2.5	12	3	12	7
V	3.4	14	3	15	10
VI	4.2	16	3	18	13
VII	5.0	18	3	21	16

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