16.2 Clinical presentation of heart disease 3276 1

16.2 Clinical presentation of heart disease 3276 16.2.1 Chest pain, breathlessness, and fatigue 3276 Jeremy Dwight

16.2 Clinical presentation of heart disease CONTENTS 16.2.1 Chest pain, breathlessness, and fatigue 3276 Jeremy Dwight 16.2.2 Syncope and palpitation 3284 K. Rajappan, A.C. Rankin, A.D. McGavigan, and S.M. Cobbe 16.2.1 Chest pain, breathlessness,
and fatigue Jeremy Dwight ESSENTIALS Chest pain, breathlessness, and fatigue are common diagnostic challenges, with a broad differential diagnosis that includes several life-threatening pathologies. Chest pain The most reliable discriminating feature for angina, as opposed to other causes of chest pain, is its constricting nature, a fixed and pre- dictable relationship to exertion, and that is relieved, within a few minutes, by rest or glyceryl trinitrate. The pain in acute coronary syndromes is similar to exertional angina, but usually more severe and usually reaches maximal intensity over the course of a few min- utes: pain reaching its maximum intensity instantaneously suggests an alternative cause. Specific clues in history and physical examination are critical for diagnosis of aortic dissection and pericarditis. Breathlessness and fatigue Most patients find it impossible to distinguish between cardiac and pulmonary causes of dyspnoea. In the diagnosis of left ventricular failure the most helpful features in the history are exertional breath- lessness, orthopnoea, paroxysmal nocturnal dyspnoea, or a history of myocardial infarction. A displaced apex on palpation is helpful and relatively specific; a third heart sound has a high specificity but low sensitivity; basal inspiratory crackles are suggestive of pulmonary oedema but have low

sensitivity and specificity. Other considerations The cardiovascular history routinely includes assessment of risk factors and those aspects of the patient's past medical history that make cardiovascular disease more likely. The presence of numerous risk factors may, on occasion, prompt the physician to proceed to further investigation even in the face of a relatively unconvincing history. Most diagnoses are made on the basis of patient history, and the physician is always compelled to return to the initial history and examination to put the findings of any investigations into context and to plan therapy appropriate for the individual patient. Introduction The symptoms of chest pain, breathlessness, and fatigue present a frequent diagnostic challenge in the outpatient and acute medical departments, as well as the emergency department. They have a broad differential diagnosis that includes several life-threatening pathologies. As with all clinical presentations, the initial presenting symptom will prompt a differential diagnosis that the physician must narrow down, using a thorough history, to one or two possibilities. The onset, nature, and precipitating causes of symptoms need to be ac- curately defined, with carefully directed questions used to assess their relevance. The process involves a partnership between the pa- tient and their doctor and is enhanced by explaining the reasoning behind the questions asked and their relevance to making a diag- nosis. In this way history-taking is a useful opportunity to assist the patient to a better understanding of their symptoms and to improve their compliance with any management plan. The cardiovascular history routinely includes assessment of risk factors such as age, occupation, diabetes, hypertension, smoking, hypercholesterolaemia, drugs (both therapeutic and recreational), and a family history. It should also record those aspects of the patient's past medical history that make cardiovascular disease more likely, such as stroke, transient ischaemic attack, claudica- tion, vascular surgery, renal disease, or connective tissue disease. The presence of numerous risk factors may, on occasion, prompt the

16.2.1 Chest pain, breathlessness, and fatigue 3277 physician to proceed to further investigation even in the face of a relatively unconvincing history. Armed with a differential diagnosis obtained from the history, the physical examination is directed to identifying further supporting evidence. In isolation, however, there are surprisingly few examin- ation findings that will provide a definitive diagnosis. The cardiologist has a large armamentarium of diagnostic tools available to assist in making a diagnosis—ECG, echocardiog- raphy, coronary angiography, MRI, and so on. These may appear to threaten to displace history-taking with the allure of high-definition images and impressive software. However, most diagnoses are made on the basis of patient history, and the physician is always compelled to return to the initial history and examination to put the findings of any investigations into context and to plan therapy appropriate for the individual patient.
Chest pain Chest pain accounts for up to 20% of all medical consultations and is one of the commonest presentations to the emergency depart- ment. In the community setting musculoskeletal or gastrointestinal causes are most common, whereas cardiac causes are more frequent in the emergency department (Table 16.2.1.1). The circumstances of chest pain Chest pain on exertion: Angina pectoris They who are afflicted with it are seized while they are walking (more especially if it be uphill and soon after eating) with a painful and most disagreeable sensation of the breast, which seems as if it would extinguish life, if it were to increase or continue, but the moment they stand still, all this uneasiness vanishes. (Heberden, 1768) Unfortunately for the physician, the descriptors used by patients with angina are highly variable and include burning, heaviness, tightness, pressure, squeezing, aching, and strangling. Patients may not describe pain and it is preferable to ask for symptoms of discomfort in the chest. Most patients with angina recognize the pain as being worrying or serious. The location of the discomfort

is usually retrosternal and may radiate to the arms, neck, and jaw (Fig. 16.2.1.1). Less commonly, the pain may be felt in the back and upper abdomen. The most reliable discriminating feature for angina as opposed to other causes of chest pain is a fixed and predictable relationship to exertion that is relieved within a few minutes by rest or glyceryl tri- nitrate (nitroglycerin). The discomfort characteristically occurs when walking up an incline and compels the patient to stop. In some cases, the characteristic symptoms occur at the start of exertion and then ease, which is termed ‘walk-through angina’. Surprisingly, patients may still be able to perform substantial anaerobic exercise without limitation. Angina is often worse in cold weather, in a cold wind, or after eating. Occasionally the pain is only present at the start of the day, when the patient is shaving or brushing their teeth. Symptoms of chest discomfort occurring after rather than during exertion, or which are present continuously throughout the day, are not due to angina. Taking a careful history of the time course of relief with rest and glyceryl trinitrate is important. Many patients mistakenly report a response to glyceryl trinitrate when their pain has taken more than 15 min to resolve, but a response to glyceryl trinitrate is only helpful diagnostically when it occurs within a few minutes. Oesophageal spasm also responds to glyceryl trinitrate and may produce similar discomfort, but the pain is not related to exertion and is nearly always associated with symptoms of reflux. The three key clinical features of anginal pain are that it is (1) a constricting discomfort in the front of the chest, neck, shoulders, jaw, or arms; (2) precipitated by exertion; (3) relieved by rest or GTN within about 5 min. These features are used to identify patients with typical angina (all three features), atyp- ical angina (two features), or noncardiac pain (one or none of these features). In the United Kingdom this classification has been incorp- orated into National Institute for Health and Care Excellence (NICE) guidelines for management of recent onset chest pain. Chest pain at rest Chest pain due to ischaemia that occurs at rest has a broader dif- ferential diagnosis. The important life-threatening differential diag- noses are myocardial infarction, aortic dissection, and pulmonary embolism. Rest pain due to angina without infarction is usually accompanied by a history of exertional angina, but there are a few exceptions. Arrhythmias (e.g. paroxysmal atrial fibrillation) may precipitate angina at rest and a history of palpitations should be sought in those with unpredictable symptoms. Emotional stress may also precipitate an attack. An important example of this is Takotsubo cardiomyopathy, where chest pain is accompanied by a character- istic pattern of left ventricular damage in the absence of significant coronary disease. Nocturnal angina may be precipitated by night- mares or the onset of pulmonary oedema, but a history of exertional angina is nearly always present. Where nocturnal chest pain is pre- sent in the absence of exertional symptoms, a history of acid reflux (relief on sitting up or with antacids, and discomfort on drinking hot fluids) should be sought. Reflux symptoms are common and may coexist with angina, and the patient may find it impossible to differ- entiate between the two. Table 16.2.1.1 Cardiovascular causes of chest pain and differential diagnoses Frequency as cause of chest pain Cardiovascular Noncardiovascular Common Angina Oesophageal reflux Acute coronary syndromes Pleurisy Pericarditis Musculoskeletal, including osteochondritis Pulmonary embolism Syndrome X Uncommon Valvular heart disease Pneumothorax Pulmonary hypertension Herpes zoster Aortic dissection Peptic ulcer disease Myocarditis Pulmonary or mediastinal tumours Takotsubo cardiomyopathy Mediastinitis

 Particular causes of chest pain Acute coronary syndromes The term ‘acute coronary syndrome’ encompasses myocardial in- farction and unstable angina, conditions which are usually caused by a common pathology—the rupture or erosion of an atheromatous plaque. Because of the need for rapid assessment and treatment, the ECG is often

used to triage patients with chest pain on admission to the emergency department. Where there are classic features of ST elevation infarction, treatment is commenced with thrombolysis or angioplasty after a brief confirmatory history (see Chapter 16.13.4). However, patients with ST elevation represent only a small fraction of those presenting with chest pain, and those without ST elevation present the greater diagnostic challenge. Some will simply have dys- pepsia or musculoskeletal pain, whereas those at the other end of the spectrum will be at imminent risk of myocardial infarction. The history has two important roles: first to establish whether the pain is cardiac, and secondly to contribute to the risk stratification process that determines the nature and time course subsequent therapy and investigation. The character of pain in acute coronary syndromes is similar to exertional angina, but usually more severe. It usually reaches max- imal intensity over the course of a few minutes. Pain reaching its maximum intensity instantaneously suggests an alternative cause, in particular, aortic dissection. The patient should be asked to de- scribe exactly what they were doing at the onset of the pain: sudden onset during a specific movement will suggest a musculoskeletal origin. The classical description of the pain of myocardial infarction is of a heavy, crushing, or constricting pain. In comparison to angina the duration of pain in myocardial infarction is longer (>15 min), and with increasing duration myocardial infarction is more likely, but the pain rarely lasts more than a few hours. Infarction is more likely to be associated with systemic symptoms (breathlessness, sweating, nausea, and vomiting) and does not respond to glyceryl trinitrate. About one-half of patients will have a history suggestive of worsening exertional angina, or short-lived episodes of chest pain at rest before presentation. The pain of an acute coronary syndrome usually discourages the patient from attempting any exertion and does not improve with exercise. Although the history alone cannot definitively rule out myocardial infarction, it can be used to assess the probability of this condition (Box 16.2.1.1). During the examination, the patient should be asked to map out the distribution of the pain. Pain radiation to both arms is sug- gestive of acute coronary syndrome. Highly localized pain of less than a few centimetres in distribution is unlikely to ischaemic in origin. Tenderness on palpation of the chest wall or pain exacer- bated by rotation of the thorax or passive movements of the arms or neck suggest musculoskeletal pain but does not infallibly rule out cardiac ischaemia. Components of the history, the ECG, and markers of myocardial damage are used in non-ST elevation acute coronary syndromes to determine the risk of subsequent events in the TIMI (Thrombolysis in Myocardial Infarction) risk score (Table 16.2.1.2) and a scoring system based on the GRACE (Global Registry of Acute Coronary Events) registry. Great emphasis has been placed on the use of troponin estimation in determining the risk of subsequent events in these patients and this is undoubtedly a useful tool. However, in the absence of definitive ECG changes or troponin rise, the patient may still score 5 on the TIMI risk score from the history alone, giving RETROSTERNAL INTERSCAPULAR Myocardial ischaemic pain Pericardial pain Oesophageal pain Aortic dissection Mediastinal lesions Pulmonary embolization SHOULDER Myocardial ischaemic pain Pericarditis Subdiaphragmatic abscess Diaphragmatic pleurisy Cervical spine disease Acute musculoskeletal pain Thoracic outlet syndrome ARMS Myocardial ischaemic pain Cervical/dorsal spine pain Thoracic outlet syndrome LEFT LOWER ANTERIOR CHEST Intercostal neuralgia Pulmonary embolization Myositis Pneumonia/pleurisy Splenic infarction Splenic flexure syndrome Subdiaphragmatic abscess Precordial catch syndrome Injuries EPIGASTRIC Myocardial ischaemic pain Pericardial pain Oesophageal pain Duodenal/gastric pain Pancreatic pain Gallbladder pain Distention of the liver Diaphragmatic pleurisy Pneumonia Myocardial ischaemic pain Musculoskeletal pain Gallbladder pain Pancreatic pain RIGHT LOWER ANTERIOR CHEST Gallbladder pain Distention of the liver Subdiaphragmatic abscess Pneumonia/pleurisy Gastric or duodenal penetrating ulcer Pulmonary

embolization Acute myositis Injuries Fig. 16.2.1.1 Differential diagnosis of chest pain according to location and radiation. Serious intrathoracic or subdiaphragmatic diseases are usually associated with pains that begin in the central or left anterior chest, left shoulder or upper arm, the interscapular region, or the epigastrium. The scheme is not all inclusive (e.g. intercostal neuralgia occurs in locations other than the left lower anterior chest area). From Miller AJ (1988). Diagnosis of chest pain. New York, Raven Press (LWW), p. 175.

16.2.1 Chest pain, breathlessness, and fatigue 3279 a risk of 25% of major cardiovascular adverse events in the next 14 days. For further discussion, see Chapter 16.13.4. There are no specific findings on cardiovascular examination in acute coronary syndromes. In the context of severe coronary disease the patient may present with the clinical features of left ventricular failure (see 'Particular causes of breathlessness') or cardiogenic shock. Features of increased sympathetic tone, pallor, tachycardia, and sweating are often present in infarction, but are also features of all causes of severe chest pain. A pansystolic murmur may indicate the development of a ventricular septal defect or papillary muscle rupture and severe mitral regurgitation, complications which are usually associated with haemodynamic compromise and left ven- tricular failure. The presence of peripheral vascular disease increases the prob- ability of coexistent coronary disease and the patient should be examined for carotid, femoral, and renal bruits and an abdominal aortic aneurysm. The foot pulses should also be assessed. The presence of neck and/or chest wall tenderness will point to alternative diagnoses such as cervical spondylopathy, costochondritis, or nerve entrapment. Hypochondrial tenderness suggests a gastrointestinal cause (e.g. peptic ulcer disease, pancrea- titis, or gallstones). Coronary spasm, Prinzmetal's angina, syndrome X, atypical angina Patients with unpredictable angina due to the occurrence of cor- onary spasm, either in the context of coronary disease or with normal coronary arteries, have been described. The diagnosis should only be considered in the patient with a classical description of ischaemic chest pain that usually responds rapidly to glyceryl trinitrate, preferably in the context of ECG changes (ST elevation in the case of Prinzmetal's angina). Cocaine abuse is a frequent cause of this presentation to the emergency department. Syndrome X, as its name suggests, is poorly understood. This label (whether it can properly be called a diagnosis is debatable) is often attached to patients with cardiac-sounding chest pain and a normal angiogram. This finding is more common in women. The pain often has features atypical of angina. It is often of submammary location or radiation, and precipitating factors are highly variable. This diag- nosis should only be considered after other causes of chest pain have been carefully excluded, since it may expose the patient to a lifetime of inappropriate treatment and anxiety. The term 'atypical chest pain' is meaningless (especially for the patient) and is best avoided. There are, however, many patients for whom a confident diagnosis cannot be made. Serious pathology can be excluded and the patient reassured that they have an excel- lent prognosis. It is better to leave the diagnosis at 'chest pain-type symptom' than to inappropriately label the patient as having 'atyp- ical angina' or syndrome X. Aortic dissection Aortic dissection is a rare but important cause of chest pain: up to one-half of all patients with an untreated proximal aortic dissec- tion die within 48 h. The pain of aortic dissection is very sudden in onset, is usually described as tearing or ripping, and the pa- tient may report that it migrates from the front to the back of the chest. There should be a particularly high index of suspicion when chest pain is associated with neurological features such as hemi- plegia or paraplegia due to involvement of the carotid vessels and spinal arteries, but these are present in less than 20% of cases. Risk factors in the history include hypertension, Marfan syndrome, a bicuspid aortic valve, previous aortic valve replacement, cocaine usage, and the third trimester of

pregnancy. Of the clinical fea- tures (see Box 16.2.1.2) aortic pain (as described earlier), loss of Box 16.2.1.1 Risk stratification for acute myocardial infarction and acute coronary syndrome according to components of the chest pain history Low risk: • Pain that is pleuritic, positional, or reproducible with palpation, or is described as stabbing Probably low risk: • Pain not related to exertion or that occurs in a small inframammary area of the chest Probably high risk: • Pain described as pressure, is similar to that of a prior myocardial infarction or worse than prior anginal pain, or is accompanied by nausea, vomiting, or diaphoresis High risk: • Pain that radiates to one or both shoulders or arms or is related to exertion Table 16.2.1.2 TIMI risk score for non-ST elevation acute coronary syndromes Clinical feature Points Age ≥65 years 1 At least three risk factors for coronary diseasea 1 Prior demonstration of significant coronary artery stenosis 1 ST deviation on ECG 1 Severe anginal symptoms (e.g. ≥2 anginal events in the last 24 h) 1 Use of aspirin in previous 7 days 1 Elevated cardiac markers (e.g. troponin) 1 a Family history, hypertension, hypercholesterolaemia, diabetes, current smoking. From Antman EM et al. (2000). The TIMI risk score for unstable angina/non-ST elevation MI: a method for prognostication and therapeutic decision making. JAMA, 284, 835–42. Box 16.2.1.2 Clinical features associated with aortic dissection • Sudden onset tearing, ripping chest pain that migrates to the back • Loss of peripheral pulses • Blood pressure difference more than 20 mm Hg between arms • Hemiparesis • Paraparesis • Diastolic murmur • Pleural effusion (usually left-sided) • Hoarseness • Horner’s syndrome • Bilateral testicular tenderness • Pulsatile sternoclavicular joint • Superior vena cava obstruction • Pulsus paradoxus (with pericardial tamponade)

section 16 Cardiovascular disorders 3280 peripheral pulses, blood pressure difference between the two arms (>20 mm Hg), and mediastinal widening on the chest radiograph are the most helpful. In the absence of these features the incidence of aortic dissection is less than 5%. The absolute level of blood pressure in unhelpful in discriminating aortic dissection from other causes of chest pain.
Pericarditis Pericarditis occurs most commonly following a myocardial infarc- tion or viral infection. The patient may describe a preceding viral illness with fever and cough. The pain is usually sharp and precor- dial. The onset is often sudden. It is characteristically worse on in- spiration and relieved by sitting up and leaning forward, and it can be accompanied by classic pleuritic pain.
A less typical description occurs when a pericardial effusion has developed and the pain arises from pericardial distension, when the pain may be a dull retro- sternal ache or pressure. Radiation of pericarditic pain occurs to all those areas associated with myocardial infarction, but radiation to the trapezius ridges is pathognomonic of the diagnosis. The patient is usually well and not compromised haemodynamic- ally (except where there is pericardial tamponade). Clinical examin- ation may initially be normal. A pericardial friction rub heard over the sternum may be positional and appear and disappear within hours. Repeated examination may be helpful, including auscultation of the patient lying flat in expiration. The ECG finding of concave ST elevation in multiple lead is helpful, but ECG findings are equivocal or normal in 40–50% of cases.
Breathlessness and fatigue Breathlessness (or dyspnoea, derived from Greek words meaning painful or difficult breathing) is the endpoint of a variety of path- ologies and is mediated by a series of neural pathways, the sensory inputs of which originate in the lungs, chest wall, and peripheral and sensory chemoreceptors (see Fig. 16.2.1.2). Patients may describe the sensation of breathlessness as tightness, wheeze, ‘inability to get enough air’, sighing, choking, or suffocating. Heart failure, asthma, and chronic obstructive airways disease account for about three- quarters of hospital admissions with breathlessness in industrialized nations. Symptom clusters have been described for these patholo- gies, but most patients find it impossible to distinguish between car-

diac and pulmonary causes of dyspnoea. The time course of the illness is an important aid to the diagnosis in patients with dyspnoea but must be interpreted in the context of the patient's day-to-day activities. Even when the disease progresses gradually the patient may report a recent onset of symptoms be- cause they have (often subconsciously) adapted their lifestyle over the course of many months. This is particularly true of patients with chronic heart failure. Efferent signals Motor cortex Effort? Sensory cortex Brain stem Air hunger Chemoreceptors Upper airway Ventilatory muscles Chest wall Chest tightness Upper airway Afferent signals Effort Fig. 16.2.1.2 Efferent and afferent signals that contribute to the sensation of dyspnoea. The sense of respiratory effort is believed to arise from a signal transmitted from the motor cortex to the sensory cortex coincidently with the outgoing motor command to the ventilatory muscles. The arrow from the brainstem to the sensory cortex indicates that the motor output of the brainstem may also contribute to the sense of effort. The sense of air hunger is believed to arise, in part, from increased respiratory activity within the brainstem, and the sensation of chest tightness probably results from stimulation of vagal-irritant receptors. Although afferent information from airway, lung, and chest wall receptors most likely passes through the brainstem before reaching the sensory cortex, the dashed lines indicate uncertainty about whether some afferents bypass the brainstem and project directly to the sensory cortex. From Manning HL, Schwartzstein RM (1995). Pathophysiology of dyspnea. New England Journal of Medicine, 333, 1547–53. http://content.nejm.org/cgi/content/extract/333/23/1547.

16.2.1 Chest pain, breathlessness, and fatigue 3281 Until relatively recently, symptoms of fatigue and breathlessness in heart failure have been assumed to be due purely to a combin- ation of poor cardiac output and pulmonary congestion. However, in patients with heart failure the correlation between symptoms and left ventricular ejection fraction is very poor. Changes in skeletal and respiratory muscle function appear to contribute significantly to symptoms, a hypothesis that is supported by the response ob- served to exercise training programmes in patients with chronic heart failure, and which may account for part of the considerable variability in disability in patients with similar haemodynamic and echocardiographic findings. Because of the contribution of fa- tigue, it is more helpful to ask about a change in exercise tolerance in patients with suspected heart failure, since this may correlate more closely with the underlying pathology. The New York Heart Association (NYHA) classification is used to classify the extent of disability (Table 16.2.1.3). The time course of onset of breathlessness can be particularly useful in determining the underlying pathology (Table 16.2.1.4). Breathlessness of dramatic onset (over minutes) is suggestive of pul- monary embolism, pulmonary oedema, upper airway obstruction, or a pneumothorax. Chronic dyspnoea presents in the context of worsening breathlessness over a period of months or years is typical of chronic obstructive airways disease, interstitial lung disease, or anaemia, but may also be a feature of heart failure. Acute or chronic dyspnoea indicates an exacerbation of breathlessness in a patient with established disease. Chronic obstructive airways disease, asthma, and heart failure are common in the population of industrialized countries and most elderly patients presenting to the emergency department with breathing difficulties will have a prior history of pulmonary or car- diac disease. However, it is important not to automatically attribute any deterioration in symptoms as being due to progression of their underlying disease process. Alternative causes should be considered, and this situation is often a major diagnostic challenge. A common example is a sudden deterioration in the patient with long-standing well-controlled heart failure, which should prompt consideration of further pathology such as a silent myocardial infarction, pulmonary embolism, or arrhythmia. Breathlessness at rest occurs in pulmonary embolism or pul- monary

oedema, and with a pneumothorax. Exertional dyspnoea occurs in left ventricular failure and chronic obstructive airways disease. Psychogenic breathlessness is frequently present at rest and is associated with sighing, features of hyperventilation such as perioral or peripheral paraesthesiae, and chest tightness. The pres- ence of breathlessness at rest but not on exertion strongly suggests a functional origin. Particular causes of breathlessness Left ventricular failure The incidence of left ventricular failure in the community is 1–2%. It is important to attempt to identify the cause during the initial as- sessment. A history of ischaemic or valvular heart disease, alcohol abuse, smoking, diabetes, hypertension, and a family history are important. Patients with left ventricular failure commonly present to the out- patient clinic, but may present for the first time to the emergency Table 16.2.1.3 New York Heart Association classification of breathlessness according to severity Class I No limitation—ordinary physical activity does not cause undue fatigue, dyspnoea, or palpitation Class II Slight limitation of physical activity—comfortable at rest, but ordinary physical activity results in fatigue, dyspnoea,
or palpitation Class III Marked limitation of physical activity—comfortable at rest, but less than normal activity produces symptoms Class IV Inability to carry out any physical activity without discomfort Table 16.2.1.4 Conditions causing breathlessness classified by the rate of onset Acute Acute on chronic Chronic Asthma Infective exacerbation of COPD COPD Myocardial infarction Decompensated chronic heart failure Cardiac failure PE PE complicating congestive cardiac failure or COPD Anaemia Cardiogenic pulmonary oedema (secondary to ischaemia, valvular disease, arrhythmias) Pneumothorax complicating COPD or asthma Pulmonary vascular disease (PE, pulmonary hypertension) Pneumonia Atrial fibrillation/flutter complicating COPD or cardiac failure Parenchymal lung disease, e.g. UIP, sarcoid Noncardiogenic pulmonary oedema Chordal rupture in chronic nonrheumatic mitral regurgitation Pleural disease, e.g. effusion, asbestosis Pulmonary haemorrhage Chest wall disease, e.g. kyphosis, ankylosing spondylitis Spontaneous pneumothorax Neuromuscular disorders, e.g. muscular dystrophy, polio, myasthenia gravis Chest trauma Malignancy Upper airway obstruction Obesity/deconditioning Hyperventilation syndrome Sleep apnoea Silent myocardial ischaemia COPD, chronic obstructive pulmonary disease; PE, pulmonary embolism; UIP, usual interstitial pneumonia.

section 16 Cardiovascular disorders 3282 department. An acute presentation is more likely when there has been a rapid rise in the left atrial pressure generating pulmonary oedema. In severe cases this is associated with haemoptysis in the form of frothy pink sputum. This type of presentation occurs with myocardial infarction, mitral valve papillary muscle or chordal rupture, malignant hypertension, tachyarrhythmias, and endocar- ditis with major valve destruction. Where a rise in left atrial pres- sure occurs over a longer time course, sustained elevated left atrial pressures are compensated for by increased lymphatic drainage and structural changes in the pulmonary capillary and alveolar base- ment membrane and patients more commonly present with fatigue, exertional breathlessness, and orthopnoea. Prolonged increases in left atrial pressure are associated with pulmonary hypertension and the associated clinical features of right ventricular enlargement, tricuspid regurgitation, and a loud pulmonary second sound. This type of presentation is more frequently a feature of patients with an idiopathic, ischaemic, hypertensive, or alcoholic cardiomyopathy. Clinical findings that help in assessing impaired left ven- tricular function or elevated left atrial filling pressures are shown in Table 16.2.1.5. The most helpful features in the history are exertional breath- lessness, orthopnoea, paroxysmal nocturnal dyspnoea, or a his- tory of myocardial infarction. Breathlessness that is worse on lying flat and relieved promptly on sitting up is characteristic for orthopnoea. Patients with chronic obstructive airways disease may also

describe orthopnoea, but this is usually present only in the setting of severe disease and chronic breathlessness at rest. Paroxysmal nocturnal dyspnoea is due to the development of interstitial oedema and typically occurs 2–4 h after the onset of sleep. The patient usually stands up or sits on the side of the bed and symptoms resolve over the course of 10–15 min. This is usu- ally a frightening and memorable experience for the patient, and to avoid these symptoms they will sleep propped up on pillows or, in severe cases, in a chair. However, a history of paroxysmal noc- turnal dyspnoea or orthopnoea is only present in 20% of patients with heart failure and its absence does not exclude the diagnosis. Ankle oedema is supportive of a diagnosis of heart failure, but de- pendent oedema is often present in older people and in patients with chronic obstructive airways disease, and the astute physician should avoid the common mistake of assuming that 'ankle oedema means cardiac failure means diuretic prescription'. The clinical examination findings are used to support a sus- pected diagnosis of heart failure, but they are not always helpful. Tachycardia, cyanosis, and an elevated jugular venous pressure are features of heart failure, but they are also features of the major dif- ferential diagnoses, pulmonary embolism, and chronic obstructive airways disease. Although jugular venous pressure correlates with left atrial pressure it may be misleading in the presence of isolated right ventricular dysfunction, tricuspid regurgitation, and pul- monary hypertension. A displaced apex on palpation is helpful and relatively specific. Basal inspiratory crackles (rales) are suggestive of pulmonary oedema but can be present in fibrotic lung disease infection and chronic airways disease and have a sensitivity and specificity as low as 13% and 35%, respectively. The third sound is a low-pitched sound heard in mid-diastole, best with the bell of the stethoscope placed lightly over the apex. It can be confused with a split second sound but is later in diastole and has a much longer duration. It has a high specificity (90–97%) but low sensitivity (31– 51%) for detecting left ventricular dysfunction. Fever and purulent sputum usually point to a diagnosis of an in- fective exacerbation of chronic bronchitis or chest infection. In older people, however, a chest infection may precipitate decompensation of heart failure. Left ventricular failure is highly unlikely in the presence of a genu- inely normal ECG. Evidence of a previous myocardial infarction on the ECG, in particular the presence of Q waves in the anterior chest leads is highly predictive of left ventricular dysfunction. The most useful finding on chest radiography is cardiomegaly, but heart size may be normal, particularly in diastolic heart failure. Changes of pulmonary venous distension, pulmonary oedema, and pleural effusion are more common in acute presentations, but are frequently absent in patients presenting with chronic breathlessness. Following clinical assessment, including ECG and chest radiog- raphy, there may still be considerable uncertainty about the diagnosis of the cause of breathlessness, particularly in patients presenting to the emergency department. Measurement of blood brain natriuretic peptide (BNP) may assist in a more rapid and accurate diagnosis in this circumstance, a level below 100 pg/ml (>300 pg/ml for NT- proBNP) making the diagnosis of left ventricular failure highly un- likely and alternative diagnoses should be considered. High levels (>500 pg/ml) are strongly suggestive of heart failure. Intermediate levels are more difficult to interpret as there are certain confounding factors for BNP measurement (Table 16.2.1.6) As with troponin, BNP levels (see Chapter 16.5.3) must be inter- preted in the context of the history, clinical findings, and other inves- tigations. Scoring systems have been devised using BNP and other clinical and investigation findings in acute dyspnoea (Fig. 16.2.1.3). Given the relatively poor predictive value of the clinical history and physical signs in the diagnosis of left ventricular failure, open access to echocardiography may appear superior to clinical assess- ment. However, there are important arguments for careful clin- ical assessment. Firstly, echocardiography is not always available in the emergency setting. Secondly, cardiac and noncardiac causes of dyspnoea, particularly chronic obstructive pulmonary

disease (COPD), often coexist, and where there is dual pathology, deciding which treatment to escalate is more dependent on the appropriate interpretation of the symptoms, clinical signs, and chest radio- graphic findings than echocardiographic parameters. Thirdly, heart failure is frequently present in the presence of apparently preserved systolic function on echocardiography.
Table 16.2.1.5 Helpful and relatively specific clinical findings for predicting heart failure in patients presenting with dyspnoea History Examination Orthopnoea Elevated jugular venous pressure Paroxysmal nocturnal dyspnoea Cardiomegaly Recent onset peripheral oedema Third or fourth heart sound Prior history of heart failure Basal crepitations Previous myocardial infarction Positive hepatojugular reflux Peripheral oedema beyond mid-calf Source data from Badgett RG, Lucey CT, Mulrow CD (1997). Can the clinical examination diagnose left-sided heart failure in adults? JAMA, 277, 1712–19.

16.2.1 Chest pain, breathlessness, and fatigue 3283 Airways disease The clinical features of heart failure and airways disease are often difficult to distinguish. Patients with lung disease tend to use the terms ‘chest tightness’ or ‘restriction’, whereas the patient with heart failure is more inclined to describe the sensation of ‘not being able to get enough air’. Patients are more likely to have COPD if they have a self-reported history of COPD, wheezing on examination (although this can be a feature of heart failure), a forced expiratory time of 9 s or more, and laryngeal descent. Clearly COPD is very unlikely in the absence of a smoking history and in patients under 45 years of age. Patients with COPD and left ventricular failure may suffer from a chronic cough, although in the case of heart failure this is usually a dry cough and more prominent at night. Fluid retention giving rise to an elevated jugular venous pressure and ankle oedema can occur in association with hypoxia, but only if saturations are persistently less than 93%. Ankle oedema may also be a feature of chronic CO_2 retention. Although often cited as a cause of the clinical features of right heart failure in COPD, true right ven- tricular failure is relatively uncommon, and the mechanism of fluid retention is complex. COPD and heart failure often coexist. The chest radiograph may be unhelpful and patients with em- physema and left ventricular failure may not have any radiological features of pulmonary congestion or oedema. In these situations, systolic heart failure can only be ruled out by echocardiography. Pulmonary embolism Pulmonary embolism is a common differential diagnosis in patients with breathlessness and should be considered in any presenting with breathlessness without clinical signs of left ventricular failure. The acute presenting symptoms are of breathlessness (usually of sudden onset), chest pain (classically pleuritic, but central with large pulmonary em- boli), and less commonly haemoptysis, cough, and syncope. The differ- ential diagnosis depends on the predominant presenting feature, such as pleuritic pain (chest infection with pleurisy, pericarditis), central chest pain (myocardial infarction), dyspnoea (COPD), or heart failure. Chronic pulmonary embolic disease and pulmonary hypertension present with exertional breathlessness, and patients may complain of central chest pain that is due to right ventricular subendocardial is- chaemia. The diagnosis of pulmonary embolism cannot easily be ex- cluded without investigation and the exclusion of an alternative, more likely, cause of breathlessness is crucial to the initial assessment. Most patients with acute pulmonary embolism are breathless or tachypnoeic (respiratory rate >20/min) and in the absence of these findings, haemoptysis and pleuritic chest pain are usually due to another cause. See Chapter 16.16.1 for further discussion of exam- ination findings and diagnostic strategy in patients with suspected pulmonary embolism. Dyspnoea with preserved left ventricular function Where breathlessness is present in the context of preserved left ven- tricular function, diastolic heart failure should be considered. This diagnosis can only be made in the context of an appropriate history and examination findings. Echocardiographic parameters of diastolic dysfunction (see Chapter 16.3.2) are common in the community setting,

but more than 50% of individuals with such an echocardio- graphic diagnosis are asymptomatic and the presence of diastolic dysfunction in a patient with breathlessness should not automatically lead to a diagnosis of the clinical syndrome of diastolic heart failure. COPD, ischaemic heart disease, and obesity are common in indi- viduals with diastolic dysfunction, and diastolic heart failure can be overdiagnosed. Hypertension, coronary disease, and left ventricular hypertrophy are important causes of diastolic dysfunction and in their absence diastolic heart failure is rare. Alternative causes for dyspnoea should be always be excluded, in particular, chronic thromboembolic disease, airways disease, sleep apnoea, and silent ischaemia. FURTHER READING Badgett RG, Lucey CR, Mulrow CD (1997). Can the clinical examin- ation diagnose left-sided heart failure in adults? JAMA, 277, 1712–19. Bugiardini R, Merz CNB (2005). Angina with normal coronary ar- teries. A changing philosophy. JAMA, 293, 477–84. Cayley WE (2005). Diagnosing the cause of chest pain. Am Fam Physician, 72, 2012–21. Table 16.2.1.6 Confounding factors in the interpretation of BNP measurements Increased BNP Decreased BNP Increasing age Obesity Female sex Cardioactive drugs Pulmonary disease ACE inhibitors Systemic hypertension Spironolactone Hyperthyroidism β-Blockers (long term) Cushing’s syndrome Diuretics Glucocorticoid usage Conn’s syndrome Hepatic cirrhosis with ascites Renal failure Paraneoplastic syndrome Subarachnoid haemorrhage < 3 0 20 40 60 80 100 % of patients with CHF 3-5 6-7 8-9 10-11 PRIDE acute CHF score 12-13 13-14 Derivation population Validation population Fig. 16.2.1.3 Scoring system to predict whether a patient presenting to the emergency department has congestive heart failure (CHF). The patient’s total score (maximum 14) is obtained by adding the points that they score for each clinical or investigation feature. Reprinted from Am J Heart, Vol 151(1), Baggish AL et al., A validated clinical and biochemical score for the diagnosis of acute heart failure: the Pro-BNP Investigation of Dyspnoea in the Emergency Department (PRIDE) acute heart failure score, pp. 48–54. Copyright (2006), with permission from Elsevier.

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