4 SHORTNESS OF BREATH

M Woollard, I Greaves

Emerg Med J 2004;**21**:341–350. doi: 10.1136/emj.2004.014878

Shortness of breath is the chief complaint for about 8% of 999 calls to the ambulance service, and is the third most common type of emergency call. It can also be an important symptom in patients with a wide range of conditions. Reference should therefore be made to other relevant articles—particularly that discussing chest pain. The conditions covered in this paper include asthma, chronic obstructive pulmonary disease, acute pulmonary oedema, and chest infections. The objectives for this paper are listed in box 1.

Box 1 Article objectives

- To consider the causes of breathlessness
- To describe the recognition of primary survey positive patients and treatment of immediately life threatening problems
- To describe the recognition and treatment of primary survey negative patients requiring immediate hospital admission
- To describe the findings and treatment of primary survey negative patients suggesting delayed admission, treatment and referral, or treatment and discharge may be appropriate
- To consider a list of differential diagnoses.

The common causes of shortness of breath are asthma, chronic obstructive pulmonary disease, and pulmonary oedema but there are many other conditions that can pose diagnostic problems (box 2).

Box 2 Causes of breathlessness

Very common
- Asthma
- Chronic obstructive pulmonary disease
- Pulmonary oedema attributable to left ventricular failure

Common
- Pneumonia
- Pneumothorax
- Pulmonary embolus
- Pleural effusion
- Pregnancy

Rare
- Metabolic acidosis
- Aspirin poisoning
- Renal failure

PRIMARY SURVEY POSITIVE PATIENTS

See end of article for authors' affiliations

Correspondence to: Mr M Woollard, The James Cook University Hospital, Department of Academic Emergency Medicine, Education Centre, Marton Road, Middlesbrough TS4 3BW, UK; Malcolm.woollard@ukgateway.net

Recognition

Patients with a life threatening respiratory emergency will present in either respiratory failure or respiratory distress. Patients with respiratory distress are still able to compensate for the effects of their illness, and urgent treatment may prevent their further deterioration. They present with signs and symptoms indicating increased work of breathing but findings suggesting systemic effects of hypoxia or hypercapnia will be limited or absent. Conversely, patients with respiratory failure may have limited evidence of increased work of breathing as they become too exhausted to compensate. The systemic effects of hypoxia and hypercapnia will be particularly evident in this group and immediate treatment will be required to prevent cardiac arrest. The key findings of primary survey positive patients with shortness of breath are presented in box 3.

Box 3 Recognition of the primary survey positive patient with shortness of breath

Increased work of breathing
- Stridor associated with other key findings
- Use of accessory muscles
- Need to sit upright
- Tracheal tug
- Intercostal recession
- Expiratory wheeze associated with other key findings
- Cessation of expiratory wheeze without improvement in condition
- Inability to speak in whole sentences

Systemic effects of inadequate respiration
- Respiratory rate<10 or >29
- Weak respiratory effort
- Decreased, asymmetrical, or absent breath sounds
- Oxygen saturation<92% on air or <95% on high concentration oxygen
- PEFR<33% of normal
- Hypercapnia (measured with end tidal CO_2 monitor)
- Tachycardia (⩾120) or bradycardia (late and ominous finding)
- Arrhythmias
- Pallor and/or cyanosis (particularly central cyanosis)
- Cool clammy skin
- Falling blood pressure (late and ominous finding)
- Changed mental status—confusion, feeling of impending doom, combativeness
- Falling level of consciousness
- Exhaustion (+/−muscular chest pain)

⚠ Pitfall

Cessation of wheeze in a patient with severe asthma may be misinterpreted as an improvement in the patients condition

⚠ Tip

Cyanosis may be detected in patients with increased skin pigmentation by examining the inside of the mouth and eyelids

Treatment

If it is not possible to obtain an airway, if the patient's condition is deteriorating rapidly, or they show signs of significant respiratory failure (in particular failure to maintain Spo_2 of 95% on high concentration oxygen) consider immediate transportation to a hospital with appropriate facilities. Important treatment points for primary survey positive patients are listed in box 4.

PRIMARY SURVEY NEGATIVE PATIENTS WITH NEED FOR HOSPITAL ATTENDANCE

Primary survey negative patients with the findings listed in box 5 who do not respond to prehospital treatment will require hospital admission.

SECONDARY SURVEY

The SOAPC system should be used to undertake a secondary survey (see article 2 of this series). In primary survey positive patients, a secondary survey may not be completed in the prehospital phase of treatment as the focus must be on treatment of life threatening problems. For primary survey negative patients requiring hospital care the secondary survey may be undertaken during transportation. For the remaining patient population a secondary survey may be undertaken at the point of contact and will contribute to the decision to admit, treat and refer, or treat and leave.

Box 4 Treatment for primary survey positive patients

Treatment before transportation
- Secure the airway (in moribund patients it may be necessary to escalate rapidly through manual methods, simple adjuncts, intubation, and cricothyroidotomy until airway secured)
- High concentration oxygen via non-rebreathing mask (consider titrating concentration to a COPD patient's "normal" Spo_2[1])
- Assist ventilations if respiratory rate<10 or >29, titrated to Spo_2
- Nebulised β_2 agonist in the presence of wheeze (for example, salbutamol 5 mg initially)
- Nebulised anticholinergic in the presence of asthma or COPD (for example, ipratropium bromide 0.5 mg, may be mixed with salbutamol)
- IM adrenaline in the presence of anaphylaxis (see shock article)
- Decompress tension pneumothorax
- Consider MI/acute coronary syndrome: if present consider nitrates, aspirin, morphine and consider thrombolysis and heparinisation (see chest pain article)

Treatment during transportation

In addition to the above, consider:
- Further nebulised β_2 bronchodilators (no maximum dose for salbutamol)
- IV fluids (asthma and anaphylaxis)
- Intravenous or oral corticosteroids (asthma and anaphylaxis)
- Antihistamines (anaphylaxis)

⚠ Tip

If the patient is unable to tolerate a nebuliser, administer 10–25 puffs of β_2 agonist (for example, salbutamol 1.0 to 2.5 mg) from the patients' own inhaler via a large volume spacer, which can be improvised if necessary (fig 1)

Figure 1 Improvised large volume spacer using plastic soft drinks bottle.

Box 5 Diagnostic criteria for primary survey negative patients requiring hospital admission

Findings (not reversed by initial treatment) suggesting need for hospital admission

- Inspiratory or expiratory noises (stridor or wheeze) audible without the aid of a stethoscope
- Cannot speak in whole sentences
- Respiration ⩾25 breath/min
- Pulse ⩾110 beat/min
- Supplemental oxygen required to maintain Sp_{O_2} at 95% or above (or at "usual" level of Sp_{O_2} for COPD patients)
- PEFR<50% of normal
- Inability to rule out MI or acute coronary syndrome
- Lack of carer support for those patients unable to look after themselves

Subjective assessment

Confirm that the chief complaint is shortness of breath. Remember that this may be a symptom of conditions affecting systems other than the chest (for example, hypovolaemia attributable to bleeding). Determine if this is a new problem or an exacerbation of a chronic condition. Ask what precipitated the problem and what, if anything, makes the patient feel more or less breathless. Ask about associated symptoms, such as chest pain, cough and sputum production, palpitations, fever and malaise, and leg pain or swelling. Has the patient been using inhalers or nebulisers more than normal? Have they recently sought other medical assistance?

Inquire about previous similar episodes. If this has occurred before, find out what treatment led to its resolution. Has the patient been hospitalised previously for this condition? What is their general previous medical history? What medications are they currently taking, and why? Is there a family history of respiratory illness or heart disease?

Finally, investigate the patient's social circumstances. Is there evidence of self neglect? If the patient is not capable of caring for themselves, is there adequate carer support from family, friends, or health and social services? Does the patient smoke? Is there evidence of drug or alcohol misuse that may make the patient susceptible to infection?

Objective examination

Vital signs

The vital signs that should be recorded in a patient with shortness of breath are listed in box 6.

Box 6 Vital signs for assessing shortness of breath

- Respiratory rate and effort
- Sp_{O_2}
- Peak expiratory flow rate (PEFR) (fig 2)
- Pulse rate
- Blood pressure
- Orientation and Glasgow coma score
- Temperature

Social context

In addition to the clinical assessment, it is important to consider the patient's ability to care for themselves or whether suitable support mechanisms are available. If these are absent, can they be arranged? Can the patient perform the normal activities of daily living—feeding and washing themselves and using the toilet—either with or without support? The time of day and day of the week may also influence the decision about whether to admit or refer the patient, as this may dictate how quickly a patient could be seen by their own GP or reviewed by the emergency care practitioner.

⚠ Pitfall

Rrepeated "practice" attempts to measure maximum PEFR can worsen bronchospasm. Limit measurement to best of three forced exhalations

Figure 2 Peak flow meter in use.

General examination

Look for signs of the "unwell" patient (see article two in the series). A detailed examination of the respiratory system is mandatory for patients with shortness of breath. Remember, however, that myocardial infarction, acute coronary syndromes, and congestive cardiac failure can also result in respiratory distress, as may endocrine and neurological problems (for example Kussmaul's and Cheyne-Stokes respiration in hyperglycaemia and raised intracranial pressure respectively). If a respiratory problem cannot be readily identified as the cause of the patient's symptoms, undertake an examination of the other systems.

⚠ Tip

Elderly patients are likely to have multiple pathologies, so undertake a general systems examination

⚠ Tip

Although shortness of breath can result from problems in many systems a useful clue is to note if there is any increase in effort of breathing. This invariably means the problem has a respiratory basis.

For details of the respiratory examination, refer to boxes 3, 5, 6, and 7 of this article and article 2 of this series. Note if the patient has excessive production of sputum. What colour is this? Yellow, green, or brown sputum indicates a chest infection. White frothy sputum, which may also be tinged with pink, suggests pulmonary oedema.

Look at the patient to determine their colour, and for signs of raised jugular venous pressure. Is the patient breathing through pursed lips, or using accessory muscles, perhaps suggesting COPD? Are there signs of CO_2 retention (tremor of the hands, facial flushing, falling conscious level)? Palpate the trachea to check that it is in the midline. Examine the chest and observe chest expansion. Is this the same on both sides? Is there evidence of hyperinflation? Are scars present from surgery? Is there evidence of chest wall deformity?

Feel the chest to confirm equality of movement, and check for chest wall crepitus and surgical emphysema. Is there evidence of chest wall tenderness or pain? Is any pain positional, or worsened on inspiration (as, for example, in pleurisy)? Feel for tactile vocal fremitus (see the journal web site http://www.emjonline.com/supplemental).

Listen to the chest. Percuss the anterior and posterior chest wall bilaterally at the top, middle, and bottom of the back. Is the percussion note normal, dull, or hyper-resonant? Auscultate the chest at the same locations and in the axillae while the patient breaths in and out of an open mouth. Listen for the sounds of bronchial breathing, wheeze, or crackles. Listen for vocal resonance (see journal web site http://www.emjonline/supplemental) and pleural rubs.

⚠ Tip

If it is uncertain if a percussion note is dull or normal, compare with the result of percussing over the liver (lower ribs on the right). The percussion note will sound dull as the liver is a solid organ.

⚠ Tip

Tactile vocal fremitus and vocal resonance are increased in consolidation and decreased in pleural effusion and pneumothorax.

If the adult patient complains of symptoms of a respiratory tract infection, undertake an ENT examination. Look in the mouth to examine for tonsullar and pharyngeal inflammation, and feel for enlargement of the lymph nodes in the neck.

⚠ Pitfall

Do not attempt to examine the upper airway of a child with respiratory distress associated with stridor or drooling. These findings may be indicative of epiglottitis and attempts to examine the mouth and throat may provoke complete airway obstruction.

In all patients with sudden onset of shortness of breath and in the absence of other findings strongly suggestive of a respiratory problem, undertake an examination of the cardiovascular system (see articles two and three of this series).

The pertinent features of the respiratory examination are summarised in box 7.

Box 7 Pertinent features of the respiratory examination

General
- Discoloured sputum
- Consider examination of cardiovascular, ENT, and other systems

Feel (palpate)
- Chest wall tenderness
- Tactile vocal fremitus
- Percussion note
- Crepitus
- Surgical emphysema

Look (inspect)
- Skin colour
- Jugular venous pressure
- Tracheal deviation
- Breathing through pursed lips
- Use of accessory muscles
- Hand tremor
- Symmetry of chest wall movement
- Hyperinflation or fixed expansion
- Scars from previous surgery
- Chest wall deformity

Listen (auscultate)
- Bronchial breathing, wheeze, or crackles
- Vocal resonance
- Pleural rub

ANALYSIS (DIFFERENTIAL DIAGNOSIS)

Diagnosis is often straightforward with a typical history and findings. For example, the patient presenting with wheeze and tachypnoea may state that they have asthma. The skill is in determining the *severity* of the condition. Few patients die as a result of the misdiagnosis of asthma but significant numbers die because professionals or patients underestimate the severity of an episode. Differential diagnosis can also be very difficult, the classic situation being in distinguishing between an exacerbation of COPD and cardiogenic pulmonary oedema. This may be made simpler by the use of b-naturetic peptide (BNP) estimations. This has recently been made available as a near-patient test and may become increasingly common in the out of hospital setting.

Asthma

Table 1 summarises the pointers in history and examination in patients with asthma that help to gauge the severity of an episode. Patients with severe or life threatening asthma need calm reassurance (even if the healthcare provider is panicking internally), early treatment with β_2 agonists, oxygen, and immediate transfer to hospital. Patients with mild or moderate episodes who respond well to treatment may be suitable for home management with further inhaled β_2 agonists, oral corticosteroids, and early review (tables 1 and 2).[2]

COPD

Exacerbations of COPD are common. These can be triggered by a number of factors but a viral infection is the most frequent. Diagnosis is often simple but it is the assessment of

Table 1 Differential diagnosis of asthma

Subjective assessment	Objective examination
History of:	General findings include wheeze and increasing dyspnoea
Episodic wheezing	**Moderate acute asthma:**
Nocturnal cough	PEFR>50% normal (table 2)
Previous diagnosis	Normal speech
Previous episodes requiring intervention additional to maintenance drug therapy	Respirations<25
Increasing dyspnoea and wheeze	Pulse<110
Decreasing wheeze in the absence of recovery is a serious finding suggesting grossly inadequate ventilation	**Severe acute asthma:**
Precipitating factor, including infection, exercise, exposure to allergen, exposure to cold air	PEFR 33–50% of normal (table 2)
Other atopy (for example, eczema, hay fever)	Cannot complete sentences
History of previous hospital admission (particularly ICU)/need for ventilation is cause for significant concern, suggesting brittle asthma	Respirations≥25
Asthma medications may be evident (inhalers with β_2 agonists and corticosteroids are usual in adults; nebulisers and oral corticosteroids suggest more significant problems)	Pulse≥110
Patient or other household residents may smoke	**Life threatening acute asthma:**
Possible limitations in activities of daily living	PEFR<33% of normal (table 2)
	Sp_{O_2}<92% on air/<95% on oxygen
	Silent chest (no wheeze)
	Cyanosis
	Feeble respiratory effort
	Bradycardia or arrhythmia
	Hypotension
	Exhaustion, changed mental status, or falling GCS

Table 2 ''Personal best'' PEFR values with ranges for estimating severity of acute asthma episode

If patient's ''personal best'' peak flow meter reading is:	Normal variation above:	Moderate acute asthma	Severe acute asthma	Life threatening acute asthma less than:
100	80	50 to 80	33 to 50	33
125	100	63 to 100	41 to 63	41
150	120	75 to 120	50 to 75	50
175	140	88 to 140	58 to 88	58
200	160	100 to 160	66 to 100	66
225	180	113 to 180	74 to 113	74
250	200	125 to 200	83 to 125	83
275	220	138 to 220	91 to 138	91
300	240	150 to 240	99 to 150	99
325	260	163 to 260	107 to 163	107
350	280	175 to 280	116 to 175	116
375	300	188 to 300	124 to 188	124
400	320	200 to 320	132 to 200	132
425	340	213 to 340	140 to 213	140
450	360	225 to 360	149 to 225	149
475	380	238 to 380	157 to 238	157
500	400	250 to 400	165 to 250	165
525	420	263 to 420	173 to 263	173
550	440	275 to 440	182 to 275	182
575	460	288 to 460	190 to 288	190
600	480	300 to 480	198 to 300	198

Table 3 Differential diagnosis of chronic obstructive pulmonary disease (COPD)

Subjective assessment	Objective examination
History of:	Hyper-inflated chest
Previous diagnosis with recent exacerbation	Increased end tidal CO_2, decreased Sp_{O_2} and cyanosis (bronchitis and late stage emphysema)
Increasing wheeze and chest tightness	Normal end tidal CO_2 and Sp_{O_2} and pink colour (emphysema)
Increased sputum production and purulence	Increasing dyspnoea on exertion
New peripheral oedema	Use of accessory muscles of respiration
Episodic deterioration in condition, possibly with hospital admission	Possible productive cough
Smoking (common)	Crepitations or wheezes may be present
Family history	Cor pulmonale (right heart failure, for example, ankle swelling) is a sign of late stage COPD
Reducing mobility	Increasing pulse and respiratory rates indicate an exacerbation of COPD
Increasing limitations in activities of daily living	
Occupational exposure to dusts, etc	
Asthma may also be present	
Medications such as β_2 agonists (inhaler or nebuliser), corticosteroids, and antibiotics may be evident	
Chronic bronchitis is defined as a productive cough on most days for 3 months of the year for ≥2 years	

COPD includes chronic bronchitis (increased airway resistance attributable to narrowing of the airways) and emphysema (decreased outflow pressure attributable to loss of elasticity in lung tissues)

Table 4 Differential diagnosis of acute pulmonary oedema (left ventricular failure/LVF)

Subjective assessment	Objective examination
History of: Pre-existing heart disease (undiagnosed chest pain, angina, myocardial infarction, aortic or mitral valve disease, tachyarhythmias) Increasing dyspnoea Increasing exercise intolerance Rheumatic fever Lack of compliance to prescribed medications Failure to cope with normal activities of daily living Evidence of a wide range of drugs used to treat cardiac conditions may be found, including β blockers, calcium channel blockers, ACE inhibitors, nitrates, aspirin, diuretics and anti-arrhythmics	Severe dyspnoea increased by recumbent positioning. May be worse at night. Cough producing white frothy sputum, sometimes tinged pink (this may be copious = frank pulmonary oedema) Crackles over affected area Raised JVP Third heart sound (requires practice to differentiate!) Mitral murmur (requires practice to differentiate!) Possible arrhythmias Hypotension Chest pain may be present Dependent pitting oedema (generalised heart failure)

Table 5 Differential diagnosis of shortness of breath with fever and malaise (pneumonia)

Subjective assessment	Objective examination
History of: Predisposing factors, such as influenza, smoking, suppressed cough reflex (for example, coma), pulmonary oedema, COPD, alcoholism, immunosuppression, long term administration of broad range antibiotics, general debility or immobility Contact with person with pneumonia or recent hospital admission (less than two weeks previously) Increasing breathlessness Upper/lower respiratory tract infection General malaise There may be evidence of failure to cope with normal activities of daily living	Increasing dyspnoea Dry cough becoming productive (green purulent sputum) Fever Pleuritic chest pain (worse on inspiration, possibly positional, may be severe) Consolidation: Reduced chest wall expansion on side of consolidation Dull to percussion over affected area Increased TVF and vocal resonance over affected area Crackles over affected area Wheeze Pleuritic rub

the severity of the condition that needs skill. The main differential diagnosis is of cardiogenic pulmonary oedema (LVF). A pneumothorax is an uncommon reason for a severe sudden exacerbation of COPD. Knowledge of the patient's normal pulmonary function is important. Some patients with COPD have a ''normal'' Po_2 that would indicate severe respiratory failure in a normal person. Signs of exhaustion, inability to expectorate, or CO_2 retention are the main worrying features indicating a severe episode.

Oxygen treatment in these patients should be titrated against the SPo_2 (controlled oxygen therapy—see the North-West Oxygen Group guidelines).[1] If the episode is not severe and the patient has adequate home support, then hospital admission may be avoided (table 3).[3]

Acute cardiogenic pulmonary oedema

The onset is often sudden and severe. The patient is older and usually has a history of ischaemic heart disease although this may be the first indication of heart problems. Acute MI is often a precipitating factor. Severe shortness of breath, white frothy sputum, tachypnoea, and tachycardia are common. Such patients need to be transported to hospital, sitting upright if possible. Immediate treatment consists of buccal nitrates (providing the blood pressure is not low), oxygen, and intravenous opioids (table 4).

Pneumonia

Fever, malaise, and purulent sputum suggest a diagnosis of pneumonia. The criteria for home treatment varies from country to country (table 5).[4]

CONDITIONS FOR EXCLUSION IF HOSPITAL ATTENDANCE IS NOT CONSIDERED APPROPRIATE

Box 5 lists the key findings that indicate the need for immediate hospital admission in primary survey negative patients. Table 6 describes additional findings determined from the secondary survey that will suggest the need for hospital admission. In asthma or COPD, failure to respond to the initial dose of a β_2 agonist (for example, nebulised salbutamol) is also an indication for considering hospitalisation, as is a history of a previous near fatal attack—regardless of the severity of the current episode. All patients with a first episode of pulmonary oedema or an acute exacerbation of a chronic problem should be admitted to hospital for further investigation and treatment.

Pneumothorax

Spontaneous pneumothorax is most common in tall, thin, fit young men (see table 6). It is an uncommon complication of asthma and COPD. There are some rarer causes but these will be very uncommon in the community setting. If a pneumothorax is suspected, the patient will need to be referred to hospital for a radiograph and further evaluation.

Pulmonary embolism

Half of all patients suffering for pulmonary embolism will develop this condition while in hospital or long term care. The remainder will have an unknown aetiology or will have been exposed to a known risk factor (see table 6). If a pulmonary embolism is suspected the patient will require

Table 6 Findings from secondary survey suggesting need for hospital admission

Condition	Key findings
Pleural effusion	History of cancer, cardiac failure, or renal failure Limited chest expansion on the affected side Dull percussion note over the affected area Reduced breath sounds, TVF, and vocal resonance over the affected area Possible crackles in the presence of LVF Possible pleuritic rub (infection) Tracheal shift away from the effusion (late sign)
Pneumothorax (most spontaneous pneumothoracies occur in tall, thin, fit young adults and are ideopathic)	Sudden onset of dyspnoea and pleuritic chest pain (early sign) Development of tension pnuemothorax may be identified by increasing dyspnoea, and: Reduced chest expansion on the affected side Hyper-inflated, fixed chest wall on the affected side Surgical emphysema (rare) Trachea deviated *away* from affected side Chest hyper-resonant to percussion Decreased or absent breath sounds on the affected side Raised JVP Deteriorating cardiovascular status (late sign)
Lung collapse (bronchial obstruction)	Dyspnoea Reduced chest expansion on affected side Tracheal deviation *towards* side of collapse Dull to percussion over non-inflated area Decreased TVF over affected area Breath sounds absent or decreased over affected area; increased bronchial breathing elsewhere
Pulmonary embolism (PE)	A Clinical features compatible with PE Dyspnoea *and/or* Tachypnoea (>20 breaths per minute) *and* Heamoptysis *and/or* Pleuritic chest pain B Major risk factors for PE Major abdominal or pelvic surgery Hip or knee replacement Postoperative intensive care Late pregnancy Caesarean section Pueperium Lower limb fracture Varicose veins Abdominal, pelvic, or metastatic malignancy Reduced mobility due to hospitalisation or institutional care Previous history of venous thromboembolism C The absence of another reasonable clinical explanation for the signs and symptoms If A, B, and C are all confirmed the likelihood of PE is high; If A and B *or* C are present the likelihood of PE is intermediate; If A is present but B *and* C are both absent the likelihood of PE is low, especially in cases of pleuritic chest pain or haemoptysis *not* accompanied by breathlessness

Table 7 Treatment of asthma[2]

Moderate acute asthma	Severe acute asthma (or no response to treatment in moderate asthma)	Life threatening acute asthma
Protect and maintain airway as necessary Position for comfort (usually sitting upright) Salbutamol 5 mg via oxygen driven nebuliser If PEFR>50–75% of normal, give prednisolone 40–50 mg orally Treat and leave if patient responds to treatment Arrange re-assessment, possibly by telephone, at a suitable time Consider referring to GP or specialist nurse for delayed follow up if patient requires further support or review of treatment	Oxygen via non-rebreathing mask* Repeat salbutamol 5 mg nebuliser at 5 to 15 min intervals until symptoms are controlled* Administer prednisolone 40–50 mg orally or hydrocortisone 100 mg IV *Consider* treat and leave if patient fully responds to treatment and has adequate carer support If discharged, arrange re-assessment, possibly by telephone, at a suitable time Refer to GP for immediate appointment	Oxygen via non-rebreathing mask* Start transportation to hospital Repeat salbutamol 5 mg nebuliser at 5 to 15 min intervals until symptoms are controlled* Administer prednisolone 40–50 mg orally or hydrocortisone 200 mg IV* Oxygen via non-rebreathing mask* Repeat salbutamol 5 mg nebuliser at 5–15 min intervals until symptoms are controlled* Ipratropium 0.5 mg via nebuliser (may be mixed with salbutamol) Consider intravenous crystalloids in the presence of dehydration

urgent transfer to hospital for possible heparinisation or thrombolysis.[5]

TREATMENT AND DISPOSAL (PLAN)

The initial out of hospital treatment of each of the four key conditions is given in table 7 and boxes 8 to 10. Interventions recommended in the JRCALC guidelines for paramedic use are asterisked.[6]

Pitfall

Tension pneumothorax is a rare complication of asthma. Monitor for its signs and perform needle thoracocentesis (decompression) if these are present

Tip

Check the inhaler technique of patients left at home.[7]

Pitfall

Tension pneumothorax is a rare complication of COPD. Monitor for its signs and perform needle thoracocentesis (decompression) if these are present

Pitfall

Rule out acute MI: if present consider opioids, nitrates, aspirin, heparin, and thrombolysis according to relevant guidelines

DISPOSITION FLOW CHART

Figure 3 describes the decision making process for patient disposition.

FOLLOW UP

Patients with an acute exacerbation of the conditions discussed in this paper but not requiring hospital admission should be advised to request further assistance if their condition deteriorates once the carer has left. Reassessment of the need for hospital admission is then mandatory.

All patients provided with home care should be referred for an appointment with their general practitioner within a suitable time frame for further assessment. This will include consideration of the patient's ongoing condition, their ability to use inhalers correctly, measurement of their respiratory function (FEV_1), and lifestyle management advice (for example, smoking cessation, weight control, exercise).

ACKNOWLEDGEMENTS

Thanks to Jim Wardrope, Peter Driscoll, and Colville Laird whose feedback resulted in valuable improvements to earlier drafts of this paper.

Contributions

Malcolm Woollard wrote the first draft of the paper. Malcolm Woollard and Ian Greaves edited all subsequent drafts.

Box 8 Treatment of COPD[3]

- Protect and maintain airway as necessary*
- Position for comfort (usually sitting upright)*
- Salbutamol 5 mg via nebuliser*
- Ipratropium 0.5 mg via nebuliser (may be mixed with salbutamol)
- Re-assess: if patient's condition returns to their normal state, consider managing at home:
- Confirm appropriate technique when using inhalers
- Consider increasing dose of bronchodilator
- Consider oral corticosteroids if:
  - Previous recorded response to corticosteroid therapy
  - Dyspnoea is increasing despite prior increase in bronchodilator dose
- Consider antibiotics if two or more of the following are present:
  - Increasing dyspnoea
  - Increasing sputum volume
  - Development of purulent sputum
- Refer to GP for appointment for re-assessment within 24 hours,
- If no response to initial nebuliser, transport to hospital. On route:
  - Repeat salbutamol 5 mg nebuliser at 5 min intervals until symptoms are controlled*
  - Administer oxygen at 24 to 28% via Venturi mask initially
  - Monitor SpO_2 and adjust oxygen concentration to maintain at ''usual'' level for patient or at 90 to 92% if unknown (see North West Oxygen Group guidelines)
  - Consider supporting ventilation if SpO_2 cannot be maintained, patient becomes exhausted or respiratory rate or effort declines inappropriate

Box 9 Treatment of acute pulmonary oedema

All patients with an acute exacerbation of pulmonary oedema require hospitalisation

- Protect and maintain airway as necessary*
- Position for comfort (usually sitting upright)*
- Oxygen via non-rebreathing mask*
- Use continuous positive airway pressure ventilation (CPAP) if available: otherwise consider assisting ventilations with BVM if respiratory failure evident
- 400 μg glyceryl trinitrate spray if systolic BP>90 mm Hg*
- Consider recording 12 lead ECG
- Start transportation to hospital*
- Consider second dose of GTN if SBP>90 mm Hg*
- Give furosemide 40 mg IV*
- Give morphine 5–20 mg IV (monitor respirations and assist ventilation if respiratory depression becomes evident)
- Consider repeating furosemide 40 mg IV at 10 minute intervals to a maximum dose of 120 mg*
- Consider salbutamol 5 mg via nebuliser in the presence of wheeze*
- Consider further GTN 400 μg if SBP>90 mm Hg

Information on tactile vocal fremitus and vocal resonance is available on the journal web site (http://www.emjonline.com/supplemental).

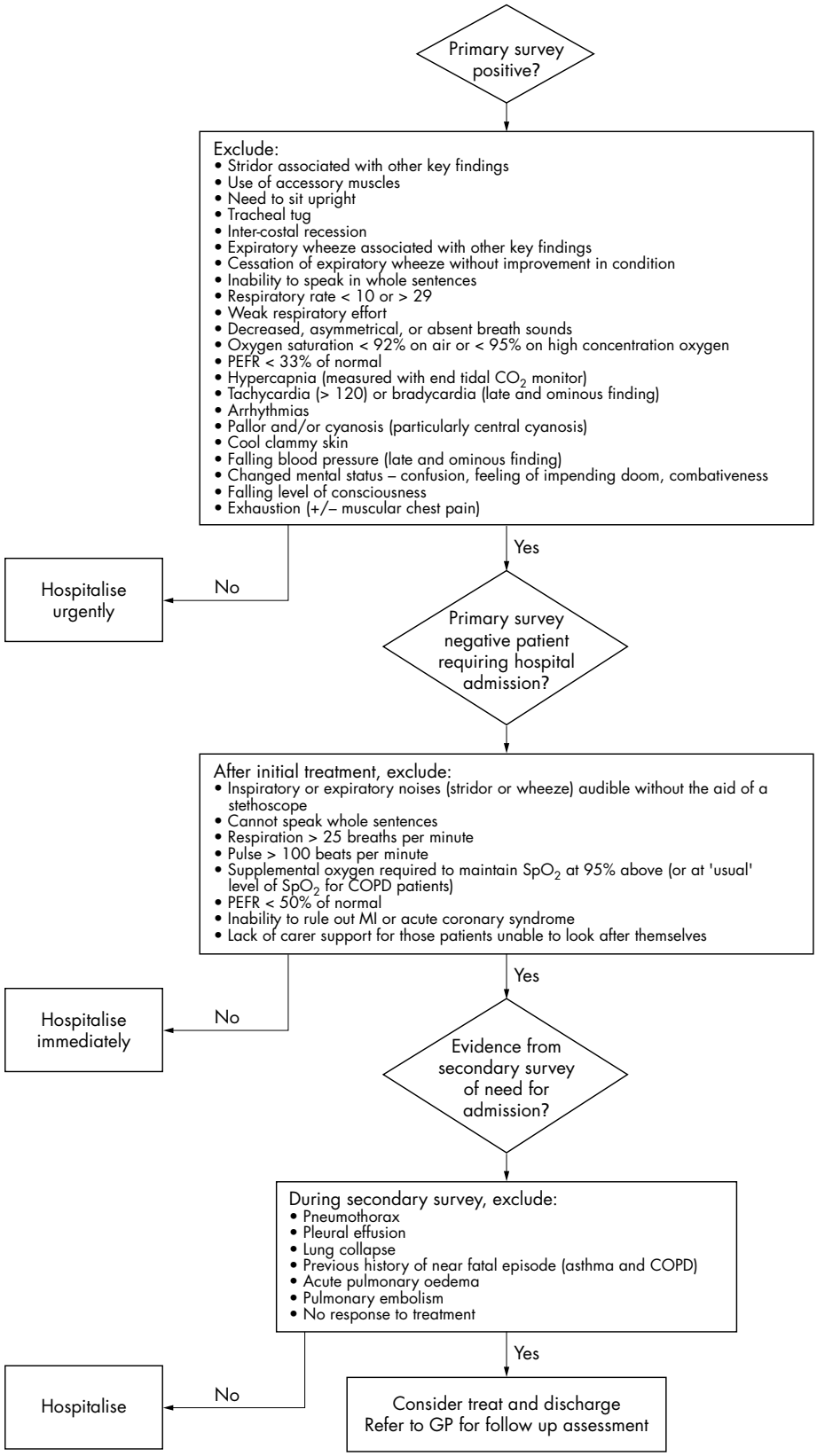

Figure 3 Disposition flow chart (shortness of breath).

Box 10 Treatment of pneumonia[4]

- If no evidence of respiratory failure or severe respiratory distress, and the patient has adequate carer support and can manage normal daily activities of living (see chest pain article):
  - Position for comfort (usually sitting upright)
  - Antibiotic therapy
  - Refer to GP for appointment for follow up within 24 hours
- In the absence of adequate carer support and if unable to manage daily tasks of living, or if tachycardia, tachypnoea or chest pain are present:
  - Consider hospital admission
  - Oxygen via non-rebreathing mask if required to maintain SpO_2 above 95%
  - Consider intravenous crystalloids in the presence of dehydration

Authors' affiliations
M Woollard, I Greaves, Pre-hospital Care Research Unit, Department of Academic Emergency Medicine, The James Cook University Hospital, Middlesbrough, UK

Further reading

Marsh J. Respiratory examination. In: *Crash course. History and examination.* London: Mosby International, 1999.
McGill University. Examples of normal and abnormal breath sounds can be found at the McGill University virtual stethoscope web pages. http://www.music.mcgill.ca/auscultation/the_lungs.html (accessed 29 Jan 2004).
O'Conner DJ, Jones BG. Pathology of the respiratory system. In: *Crash course. Pathology.* 2nd edn. London: Mosby International, 2002.
British Thoracic Society. The British Thoracic Society web site contains extensive information about respiratory illness and relevant guidelines. http://www.brit-thoracic.org.uk (accessed 29 Jan 2004).
Wardle T. Respiratory emergencies. In: Greaves I, Porter K, eds. *Pre-hospital medicine. The principles and practice of immediate care.* London: Arnold, 1999.

REFERENCES

1 **Murphy R**, Mackway-Jones K, Sammy I, *et al*. Emergency oxygen therapy for the breathless patient. Guidelines prepared by North West Oxygen Group. *J Accid Emerg Med* 2001;**18**:42–3.
2 **British Thoracic Society**. The BTS/SIGN British guideline on the management of asthma. *Thorax* 2003;**58**(suppl 1):i1–94.
3 **The British Thoracic Society Standards of Care Committee**. Guidelines on the management of COPD. *Thorax* 1997;**52**(suppl 5):S1–28.
4 **British Thoracic Society**. The British Thoracic Society. Guidelines for the management of community acquired pneumonia in adults. *Thorax* 2001;**56**(suppl 4):iv1–64.
5 **The British Thoracic Society Standards of Care Committee Pulmonary Embolism Guideline Development Group**. BTS Guidelines for the management of suspected acute pulmonary embolism, 2003. *Thorax* 2003;**58**:470–84.
6 **Joint Royal Colleges Ambulance Liaison Committee**. *Pre-hospital guidelines version 2.1.* London: JRCALC/Ambulance Service Association, 2002.
7 **Asthma Organisation**. National asthma campaign. How to use your inhaler. http://www.asthma.org.uk (accessed 29 Jan 2004).

Appendix

Tactile vocal fremitus (TVF) is assessed by placing a hand on the chest bilaterally in the anterior and posterior upper, middle, and lower zones and asking the patient to say ‘ninety-nine’. The resulting low-frequency sounds should normally be palpable, and transmission should be compared for symmetry and differences between the areas assessed.

Vocal resonance is assessed by asking the patient to whisper ‘one, two, three’ and listening with a stethoscope in the areas described for assessing TVF. Some resonance (although not the words themselves) should normally be detected and differences between areas should be noted as described above. Increased sound transmission, demonstrated by clear detection of words spoken by the patient and known as whispering pectoriloquy, is abnormal.

Both TVF and vocal resonance are used to assess for the presence of consolidation (increased resonance) or pneumothorax or pleural effusion (decreased resonance).