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.

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 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**

**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.
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 SpO2 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 SOACP 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 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 CO2 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

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” SpO2)
- Assist ventilations if respiratory rate <10 or >29, titrated to SpO2
- Nebulised β2 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 β2 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 β2 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)
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.

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.

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 CO₂ 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 breathes in and out of an open mouth. Listen for the sounds of bronchial breathing, wheeze, or crackles. Look in the mouth to examine for tonsillar and pharyngeal inflammation. White frothy sputum, which may also be tinged with pink, suggests pulmonary oedema.

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

### Box 7 Pertinent features of the respiratory examination

<table>
<thead>
<tr>
<th>General</th>
<th>Discoloured sputum</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Consider examination of cardiovascular, ENT, and other systems</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Feel (palpate)</th>
<th>Chest wall tenderness</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tactile vocal fremitus</td>
<td>Percussion note</td>
</tr>
<tr>
<td>Crepitus</td>
<td>Surgical emphysema</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Look (inspect)</th>
<th>Skin colour</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jugular venous pressure</td>
<td>Tracheal deviation</td>
</tr>
<tr>
<td>Breathing through pursed lips</td>
<td>Use of accessory muscles</td>
</tr>
<tr>
<td>Hand tremor</td>
<td>Symmetry of chest wall movement</td>
</tr>
<tr>
<td>Hyperinflation or fixed expansion</td>
<td>Scars from previous surgery</td>
</tr>
<tr>
<td>Chest wall deformity</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Listen (auscultate)</th>
<th>Bronchial breathing, wheeze, or crackles</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vocal resonance</td>
<td>Pleural rub</td>
</tr>
</tbody>
</table>

### 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 do so 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 panic-stricken), early treatment with β₂ 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 β₂ agonists, oral corticosteroids, and early review (tables 1 and 2).³

### 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

<table>
<thead>
<tr>
<th>Subjective assessment</th>
<th>Objective examination</th>
</tr>
</thead>
<tbody>
<tr>
<td>History of:</td>
<td>General findings include wheeze and increasing dyspnoea</td>
</tr>
<tr>
<td>Episodic wheezing</td>
<td>Moderate acute asthma:</td>
</tr>
<tr>
<td>Nocturnal cough</td>
<td>PEFR &gt; 50% normal (table 2)</td>
</tr>
<tr>
<td>Previous diagnosis</td>
<td>Normal speech</td>
</tr>
<tr>
<td>Previous episodes</td>
<td>Respiration &lt; 25</td>
</tr>
<tr>
<td>requiring intervention</td>
<td>Pulse &lt; 110</td>
</tr>
<tr>
<td>additional to</td>
<td>Severe acute asthma:</td>
</tr>
<tr>
<td>maintenance drug therapy</td>
<td>PEFR 33–50% of normal (table 2)</td>
</tr>
<tr>
<td>Increasing dyspnoea</td>
<td>Cannot complete sentences</td>
</tr>
<tr>
<td>and wheeze</td>
<td>Respiration &lt; 25</td>
</tr>
<tr>
<td>Decreasing wheeze in</td>
<td>Pulse &lt; 110</td>
</tr>
<tr>
<td>the absence of</td>
<td>Life threatening acute asthma:</td>
</tr>
<tr>
<td>recovery</td>
<td>PEFR &lt; 33% of normal (table 2)</td>
</tr>
<tr>
<td></td>
<td>SpO₂ &lt; 92% on air/ &lt; 95% on oxygen</td>
</tr>
<tr>
<td></td>
<td>Silent chest (no wheeze)</td>
</tr>
<tr>
<td></td>
<td>Cyanosis</td>
</tr>
<tr>
<td></td>
<td>Feeble respiratory effort</td>
</tr>
<tr>
<td></td>
<td>Bradycardia or arrhythmia</td>
</tr>
<tr>
<td></td>
<td>Hypotension</td>
</tr>
<tr>
<td></td>
<td>Exhaustion, changed mental status, or falling GCS</td>
</tr>
</tbody>
</table>

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

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

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

<table>
<thead>
<tr>
<th>Subjective assessment</th>
<th>Objective examination</th>
</tr>
</thead>
<tbody>
<tr>
<td>History of:</td>
<td>Hyper-inflated chest</td>
</tr>
<tr>
<td>Previous diagnosis</td>
<td>Hyper-inflated chest</td>
</tr>
<tr>
<td>with recent exacerbation</td>
<td>Hyper-inflated chest</td>
</tr>
<tr>
<td>Increasing wheeze and chest tightness</td>
<td>Decreased SpO₂ and cyanosis (bronchitis and late stage emphysema)</td>
</tr>
<tr>
<td>Increased sputum production and purulence</td>
<td>Normal end tidal CO₂ and SpO₂ and pink colour (emphysema)</td>
</tr>
<tr>
<td>New peripheral oedema</td>
<td>Increasing dyspnoea on exertion</td>
</tr>
<tr>
<td>Episodic deterioration in condition, possibly with hospital admission</td>
<td>Use of accessory muscles of respiration</td>
</tr>
<tr>
<td>Smoking (common)</td>
<td>Possible productive cough</td>
</tr>
<tr>
<td>Family history</td>
<td>Crepitations or wheezes may be present</td>
</tr>
<tr>
<td>Reducing mobility</td>
<td>Cor pulmonale (right heart failure, for example, ankle swelling) is a sign of late stage COPD</td>
</tr>
<tr>
<td>Increasing limitations in activities of daily living</td>
<td>Increasing pulse and respiratory rates indicate an exacerbation of COPD</td>
</tr>
<tr>
<td>Occupational exposure to dust, etc</td>
<td>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)</td>
</tr>
<tr>
<td>Asthma may also be present</td>
<td>Medications such as β₂ agonists (inhaler or nebuliser), corticosteroids, and antibiotics may be evident</td>
</tr>
</tbody>
</table>

Chronic bronchitis is defined as a productive cough on most days for 3 months of the year for >= 2 years.
inability to expectorate, or CO₂ retention are the main respiratory failure in a normal person. Signs of exhaustion, against the SPO2 (controlled oxygen therapy—see the North-west Oxygen Group guidelines). If the episode is not severe hospital admission may be avoided (table 3). 3

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

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

<table>
<thead>
<tr>
<th>Subjective assessment</th>
<th>Objective examination</th>
</tr>
</thead>
<tbody>
<tr>
<td>History of:</td>
<td></td>
</tr>
<tr>
<td>Pre-existing heart disease (undiagnosed chest pain, angina, myocardial infarction, aortic or mitral valve disease, tachyarrhythmias)</td>
<td>Severe dyspnoea increased by recumbent positioning.</td>
</tr>
<tr>
<td>Increasing dyspnoea</td>
<td>May be worse at night.</td>
</tr>
<tr>
<td>Increasing exercise intolerance</td>
<td>Cough producing white frothy sputum, sometimes tinged pink (this may be copious = frank pulmonary oedema)</td>
</tr>
<tr>
<td>Rheumatic fever</td>
<td>Crackles over affected area</td>
</tr>
<tr>
<td>Lack of compliance to prescribed medications</td>
<td>Raised JVP</td>
</tr>
<tr>
<td>Failure to cope with normal activities of daily living</td>
<td>Third heart sound (requires practice to differentiate!)</td>
</tr>
<tr>
<td>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</td>
<td>Mitral murmur (requires practice to differentiate)</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>Subjective assessment</th>
<th>Objective examination</th>
</tr>
</thead>
<tbody>
<tr>
<td>History of:</td>
<td></td>
</tr>
<tr>
<td>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</td>
<td>Increasing dyspnoea</td>
</tr>
<tr>
<td>Contact with person with pneumonia or recent hospital admission (less than two weeks previously)</td>
<td>Dry cough becoming productive (green purulent sputum)</td>
</tr>
<tr>
<td>Increasing breathlessness</td>
<td>Fever</td>
</tr>
<tr>
<td>Upper/lower respiratory tract infection</td>
<td>Pleuritic chest pain (worse on inspiration, possibly positional, may be severe)</td>
</tr>
<tr>
<td>General malaise</td>
<td>Consolidation:</td>
</tr>
<tr>
<td>There may be evidence of failure to cope with normal activities of daily living</td>
<td>Reduced chest wall expansion on side of consolidation</td>
</tr>
</tbody>
</table>

**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).

**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 β₂ 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

<table>
<thead>
<tr>
<th>Condition</th>
<th>Key findings</th>
</tr>
</thead>
</table>
| 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 pneumothorax 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  
Haemoptysis 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  
Puerperium  
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

<table>
<thead>
<tr>
<th>Moderate acute asthma</th>
<th>Severe acute asthma (or no response to treatment in moderate asthma)</th>
<th>Life threatening acute asthma</th>
</tr>
</thead>
</table>
| Protect and maintain airway as necessary | 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 |
| Position for comfort (usually sitting upright) | 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* |
| Salbutamol 5 mg via oxygen driven nebuliser | If PEFR ~50–75% of normal, give prednisolone 40–50 mg orally  
Consider referring to GP or specialist nurse for delayed follow up if patient requires further support or review of treatment |
| If PEFR ~50–75% of normal, give prednisolone 40–50 mg orally | 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

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 (FEV₁), and lifestyle management advice (for example, smoking cessation, weight control, exercise).

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

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

Exclude:
- Stridor associated with other key findings
- Use of accessory muscles
- Need to sit upright
- Tracheal tug
- Inter-costoal recession
- Expiratory wheeze associated with other key findings
- Cessation of expiratory wheeze without improvement in condition
- Inability to speak in whole sentences
- 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 CO2 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)

Hospitalise urgently

Yes

No

Primary survey negative patient requiring hospital admission?

After initial treatment, exclude:
- Inspiratory or expiratory noises (stridor or wheeze) audible without the aid of a stethoscope
- Cannot speak whole sentences
- Respiration > 25 breaths per minute
- Pulse > 100 beats per minute
- Supplemental oxygen required to maintain SpO2 at 95% above (or at 'usual' level of SpO2 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

Hospitalise immediately

Yes

No

Evidence from secondary survey of need for admission?

During secondary survey, exclude:
- Pneumothorax
- Pleural effusion
- Lung collapse
- Previous history of near fatal episode (asthma and COPD)
- Acute pulmonary oedema
- Pulmonary embolism
- No response to treatment

Hospitalise

No

Yes

Consider treat and discharge

Refer to GP for follow up assessment

Figure 3 Disposition flow chart (shortness of breath).
Box 10 Treatment of pneumonia

1 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

2 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 SpO₂ above 95%
   - Consider intravenous crystalloids in the presence of dehydration

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Further reading

4 Shortness of breath

M Woollard and I Greaves

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