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Pneumonia or just a cough?

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It is the middle of a cold, winter's night. You are on standby in the ambulance with the heating turned up to max, fleece zipped all the way up to your chin and you are hoping you will be sent back to station for a break any minute now; when the radios rings. Control: "I know it's approaching your break but we have an elderly female who is short of breath, do you mind?" You know this in not really a question so, as your crewmate moans, you politely tell control: "of course we don't mind!"

The truth is, all you have been to tonight are patients with shortness of breath; exacerbation of COPD and asthma. You don't mind, but your crewmate has a habit of listening to the chest, describing every lung sound as 'noisy' and suggesting nebulisation. You're not even sure he has heard of the various lung sounds. Fortunately, you are attending to this 78 year old female who coherently tells you that she has a 2 day history of increasing difficulty in breathing and feeling generally unwell. She is concerned that her breathing has worsened.

Observations

Respiration rate – 22/min
Pulse rate – 88bpm (regular)
Temperature – 38.5
SpO2 – 95% (air)
HGT – 5.8mmol/l
BP – 110/74mmHg

She coughs and splutters as you get out your stethoscope to auscultate, and you notice that the sputum is green in colour. Your crewmate puts the pulseoximeter probe on and takes a temperature, waving the thermometer in front of your eyes.

You auscultate in all lung fields and notice crackles in the right upper lobe, quite distinctive. You relay that information to your crewmate who, rather predictably, suggests nebulising because "it can't do any harm". You are confident it's a chest infection - but what now? Does she require hospital treatment? Does she need antibiotics? You are worried about leaving her at home, especially at night, in case she deteriorates. Here are some key differences between a viral chest infection and pneumonia, and some evidence-based information that can help to guide management.

Pathophysiology of Pneumonia

Gould and Dyer (2011) describe many differing methods for the classification of pneumonia: based on anatomical location, epidemiologic data or causative agent, indeed, a cursory

glance over the current literature reveals classifications such as ventilator, chemical, idiopathic and fungal pneumonia.

NICE guidelines refer to 2 main types of pneumonia: hospital acquired - which NICE suggest is an onset within 48 hours of admission (NICE 2016a) and community acquired pneumonia (CAP), which is more pertinent to the field of paramedic practice.

In both cases, the pathophysiological processes involved are the same and in this study we will consider this process, based on the anatomical location and structures involved: Lobar pneumonia, bronchopneumonia and primary atypical pneumonia.

In pneumonia the infection is usually caused by inhaled bacteria, where the normal defence mechanisms of the lungs become overwhelmed (Huether and McCance 2017), although on rare occasions bacteria can be spread to the lung tissue via blood; often in the case of infections elsewhere or in cases of intravenous (IV) drug abuse (Huether and McCance 2017). The most common pathogen in CAP is strepoccocus pneumoniae - although Morgan and Glossop (2016) note that in 36% of cases no causative organism is identified.

In lobar pneumonia, an inhaled pathogen will manifest within the nasopharynx - often without symptoms before passing down into the lower respiratory tract. Here, inflammation and vascular congestion will form in the walls of the alveoli- causing exudate to pool in the alveoli- interfering with gaseous exchange (Gould and Dyer 2011). After this, there is an accumulation of red blood cells (RBCs), fibrin and neutrophils - which lead to a solid mass and consolidation within a lobe of the lung. Blood flow can be impaired through the affected lobe and often the local pleura will become inflamed.

Bronchopneumonia presents as a slower onset - with a more widespread pattern of infection- with many alveoli across different lobes affected. Existing secretions within the alveoli are affected by pathogens from the bronchi and this inflammatory exudate interferes with gaseous exchange as for lobar pneumonia (Gould and Dyer 2011). Bed bound or immobilised patients are at increased risk of bronchopneumonia as these patients are often unable to adequately expel existing secretions through coughing.

In primary atypical pneumonia (PAP) the causative organism is often viral- a key difference from the other presentations. In PAP the interstitial space around the alveoli becomes inflamed - with very little or no exudate within the alveolar sacs themselves - this means there is often an absence of productive cough (Gould and Dyer 2011).

The onset of PAP is non-specific with vague symptoms and this, coupled with the absence of productive cough makes diagnosis pre-hospitally problematic and it is likely that signs and symptoms can be mistaken for acute bronchitis.

Acute Bronchitis is caused by a virus leading to inflammation and mucous production in the bronchi and often the trachea. It is commonly caused by the influenza A and B virus, but additional pathogens such as heat, smoke and chemical irritants have been known to lead to an episode of acute bronchitis (Schumann 2013). As acute bronchitis does not affect the

alveoli or gaseous exchange it tends to be a less severe infection and is often self-limiting (BMJ 2016).

Diagnosis

In hospital, a diagnosis of pneumonia is made via the use of a chest x-ray and the presence of C-reactive protein within blood test results (NICE 2016a, Searle-Barnes and Phillips 2016). In the absence of these tests in pre-hospital care, assessment relies on good history taking and respiratory system examination. Once the diagnosis of a chest infection seems likely then the assessment is focussed upon differentiating between viral causes (acute bronchitis) and bacterial causes (pneumonia).

History

Cough

Both acute bronchitis and pneumonia may present with an acute cough. Bronchitis mainly affects the bronchi and often results in increased production of mucous. This means that a cough in bronchitis may or may not be productive. Pneumonia affects the alveoli, causing a localised inflammatory response. The result of the inflammatory response means that sputum produced may be green or yellow (as a result of increased white blood cells locally) or a rusty colour as a result of increased red blood cells locally (McCance and Huether 2006). In distinguishing between acute bronchitis and pneumonia many clinicians have traditionally relied on the presence of coloured sputum being more indicative of pneumonia. However, it has since been argued that using colour of sputum to differentiate between viral and bacterial aetiologies is not a reliable predictor (Altiner et al. 2009). It is suggested that sputum colour is used a part of a larger clinical picture in differentiating between acute bronchitis and pneumonia. This is complicated by the incidence of primary atypical pneumonia which is a viral infection that often presents without a cough.

Respiratory Exam

The main positive finding that will help in the diagnosis of pneumonia is the presence of focal chest signs on auscultation and percussion.

Auscultation

Secretions and exudate within the alveoli cause the alveoli to ‘pop’ open on inspiration. This ‘popping’ sound causes coarse crackles to be heard on auscultation. Some of the coarse crackles may also be explained by the flow of air through secretions (Bickley 2009).

In lobar pneumonia, coarse crackles may be limited to a single lobe of the lungs. It is important to auscultate all lobes to reduce the risk of not recognising lobar pneumonia. For example, pneumonia isolated to the lower lobe of either lung is likely to only be heard when auscultating on the patient's back.

In bronchopneumonia coarse crackles may be more widespread. A wheeze may be heard on auscultation in acute bronchitis but the presence of coarse crackles is highly suggestive of a bacterial aetiology (Bickley 2009).

Percussion

Percussion is the process of producing an audible sound on the chest wall. It helps the clinician to establish if the underlying tissues are filled with air, fluid or solid. Under normal circumstances one would expect the lungs to be filled with air, and the resulting sound on percussion would be described as resonant. You can try this on yourself. If the underlying tissue is filled with fluid or a solid then the percussion sound would be dull, or hypo-resonant.

In pneumonia you would expect to hear dullness on percussion over the affected area, and over the same area that you auscultated coarse crackles. This is because the exudate and secretions mean that much of the alveoli in the affected area are filled with fluid rather than air. When this consolidates then the alveoli become almost solid and the percussion sound becomes even duller. This is in contrast to acute bronchitis where there is no consolidation so the sound heard on percussion is resonant/normal. It is important when percussing to compare both sides of the lungs. This helps the clinician to distinguish the different sounds heard on percussion.

Vocal Fremitus

Vocal fremitus is the process of auscultating the chest whilst the patient transmits a voice sound. The premise of vocal fremitus is that sounds are transmitted more easily through a solid/fluid than through air. There are two ways to assess this: bronchophony and egophony (Bickley 2009)

Bronchophony – Ask the patient to say “ninety-nine” whilst auscultating in all lung fields. If the chest is clear then this should be muffled and indistinct on auscultation. If there is consolidation in the alveoli then the sound will be transmitted more easily than through air and “ninety-nine” will be heard clearly.

Egophony – ask the patient to say the letter “e” whilst auscultating in all lung fields. If the chest is clear then the “e” will sound muffled. If there is consolidation than it will be heard like the letter “a”.

Tactile Fremitus

Using the same premise as above, that vibrations are transmitted more easily through solid/fluid than air, tactile fremitus can be used to assess for exudate and consolidation. The practitioner places the ulna aspect of both hands on the patient’s chest, over each lung field. The patient says “ninety-nine” and if consolidation then the clinicians will feel increased vibration over the affected lobe/s. (Bickley 2009).

The presence of these three positive findings (i.e. bronchophony, egophony and increased fremitus) indicate that there is exudate or consolidation in the alveoli and thus adds weight to the diagnosis of pneumonia, rather than bronchitis. NICE (2016b) support the use of assessing fremitus in helping to discriminate between bacterial and viral chest infections. Table 1 summarises the assessment of a chest infection to help discriminate between a viral and bacterial aetiology.

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Table 1 (adapted from NICE 2016b and Bickley 2009)

Assessment	**Acute bronchitis**	**Community acquired pneumonia**
Cough	May or may not have sputum. Sputum less likely to be purulent.	Usually have a productive cough producing yellow, green or rusty coloured sputum.
Auscultation	Wheeze often present, but otherwise chest clear	Coarse crackles heard over affected areas of the lungs
Percussion	Normal/resonant	Dullness/hypo-resonance heard over affected areas
Vocal Fremitus	Negative bronchophony and egophony	Positive bronchophony – "ninety-nine" heard clearly on auscultation Positive egophony – "e" heard as "a" on auscultation
Tactile Fremitus	Vibration equal over all areas of the lung	Increased vibration felt over affected area.

How do I manage pneumonia?

Once pneumonia has been diagnosed, treatment will be guided by the severity of the illness. Pneumonia is a disease that ranges from mild to life-threatening, therefore some patients with pneumonia will be suitable to be treated in the community, whereas others will need to be admitted to hospital (BTS 2009). There are a number of tools that have been suggested to assess the patient's mortality risk, the most common and well validated of these for out-of-hospital use is CRB-65 (McNally et al 2010). CRB-65 has been found to be a good 30 day predictor of mortality which helps to guide the decision to manage the patient in hospital or in the community (McNally et al 2010, Bauer et al 2006). This tool can only be used for adults (over 18 years old) and should not be used to assess patients who are immunocompromised or are receiving chemotherapy. Table 2 shows the CRB-65 tool which looks at 4 clinical predictors. The patient scores one point for each feature that is present:

- New onset of confusion
- Respiratory rate over 30
- Systolic blood pressure under 90mmHg or diastolic under 60mmHg
- Aged over 65

If the patient scores 0 then they are safe to be treated in the community. If they patient scores 1-2 then consider hospital admission, taking into account the full clinical picture (this includes oxygen saturation and social circumstances (BTS 2009)). If the patient scores more

than 2, then they are considered a high risk of 30 day mortality and should be admitted to hospital to be managed (BTS 2009). See table 2 for full CRB-65 scoring.

Table 2

CRB65 Severity Score
Score 1 point for each feature present:

Confusion – new onset of acute confusion
Respiration – rate above 30
Blood Pressure – systolic below 90, or diastolic below 60
Age over **65**

Score
0 – safe to be treated at home
1-2 – consider hospital admission using clinical judgement
3+ - admit to hospital

(NICE 2016c)

In the community patients with pneumonia should be treated with oral antibiotics, amoxicillin being the first choice. For those patients with sensitivity to penicillin doxycycline or clarithromycin are acceptable alternatives. Patients may be able to be referred to Specialist Paramedics, Nurse Practitioners or General Practitioners for the supply of antibiotics. Remember that antibiotics are not effective against viral infections, such as acute bronchitis, and only serve to increase the risk of community antimicrobial resistance.

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CPD Questions (correct answers highlighted in yellow – in manuscript only)

1. Which of the following is NOT a feature of CRB65?
    a) Confusion
    b) Respiratory rate over 30
    c) Blood pressure over 90 systolic or 60 diastolic
    d) Age over 65

2. What score does the case study patient score on CRB65?
    a) 0
    b) 1
    c) 2
    d) 3

3. Why might Lobar pneumonia result in a productive cough which is 'rusty' in colour?
    a) The presence of red blood cells in exudate
    b) Reduced blood flow through the affected lobe
    c) Vascular congestion in the alveoli
    d) Irritation in the pulmonary system

4. Which of the following antibiotics is considered the first choice for the treatment of pneumonia?
    a) Clarithromycin
    b) Erythromycin
    c) Doxycycline
    d) Amoxicillin

5. Which of the following chest sounds is the best indicator of pneumonia?
    a) Wheeze
    b) Fine crackles
    c) Coarse crackles
    d) Pleuritic rub

6. CRB-65 can be applied to which of the following patient groups?
    a) Adults only
    b) Children over 12 and adults
    c) All patients
    d) Only if the patient has got pre-existing respiratory conditions

7. Which anatomical structure is most commonly affected by pneumonia?
    a) Trachea
    b) Main bronchus
    c) Bronchioles
    d) Alveoli

8. Which of the following is the most common causative organism in community acquired pneumonia?

a) Staphylococcus aureus
b) Streptoccocus Pneumoniae
c) Streptococcus pyogenes
d) Helicobacter pylori

9. In PAP, there is often an absence of a productive cough. Why?

a) The mucous membranes are not affected
b) The interstitial space is affected, not the alveoli
c) There is always a productive cough
d) There is never a productive cough

10. When assessing tactile fremitus, you would expect to feel increased vibrations in the affected area in patients with pneumonia. Why is this?

a) Vibrations travel more easily through solid matter
b) The lung is hyper resonant in that area
c) There is increased blood flow to that area of lung tissue
d) There is turbulent air flow in the bronchi

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