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Management of chronic cough in adults

Cough is one of the most common causes of presentation to GPs. Although not life threatening, chronic cough causes severe impairment of a patient's quality of life. Algorithm-based approaches to investigation and empirical treatment for the four common causes of chronic cough should resolve this condition in more than 90% of cases.

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Cough is one of the most frequent causes of visits to GPs. Worldwide hundreds of millions of dollars are spent on over-the-counter medications aimed at cough suppression. The physical consequences of cough are diverse and include chest pain, retching, sleep disruption, headache, facial flushing, stress incontinence, syncope and abdominal herniation. Psychological morbidity is also significant, with many individuals describing long lasting anxiety about possible threatening underlying diseases and also depression as cough chronicity extends. Functional impairment is substantial as chronic cough can lead to disabling embarrassment, social withdrawal and occupational limitation. Regardless of the cause, chronic cough, while not life threatening, substantially impairs quality of life and creates major direct and indirect health care costs.

Definitions of cough

Cough is a reflex and volitional protective mechanism of the airways causing air to be expelled from the lungs at a high velocity, enabling fluid and particulates to be dislodged and expelled from upper and lower airways. Many conditions, however, can accentuate the cough reflex and lead to nonfunctional persistence of this symptom.

Acute cough

Acute cough, lasting up to three weeks, is usually self-limiting and caused by viral upper respiratory tract infections (e.g. rhinosinusitis or the common cold) and lower respiratory tract infections. Treatment is supportive and antibiotics are generally not required unless purulent secretions are evident. Other common causes include an exacerbation of existing conditions such as asthma,

IN SUMMARY

- **Cough is one of the most common causes of presentation to GPs. Chronic cough causes major physical and psychological morbidity.**
- **Chronic cough may be assessed qualitatively and semiquantitatively using cough counters and cough sensitivity testing.**
- **In most cases, chronic cough is caused by asthma, gastro-oesophageal reflux disease (GORD), postnasal drip syndrome or chronic bronchitis.**
- **Appropriate investigation and empirical treatment should control cough in the majority of individuals. If cause-specific treatment fails then a trial of antitussive therapy is indicated to suppress the cough.**
- **If algorithm-directed investigation and treatment of chronic cough is unsuccessful then referral to a cough clinic may be appropriate.**

Table 1. Causes of chronic cough

Common causes

Asthma

Gastro-oesophageal reflux disease

Postnasal drip syndrome
- sinusitis
- rhinitis
- nasal polyposis

Chronic bronchitis (e.g. COPD)

Postinfectious cough

Less common causes

Extrathoracic conditions
- dental/gum disease

Extratracheal conditions
- goitre
- mediastinal mass
- vascular ring

Tracheobronchial conditions
- smoking
- bronchiectasis
- recurrent/chronic infection (e.g. tuberculosis, whooping cough, HIV infection)
- neurological
- follicular bronchiolitis
- tracheomalacia

Endobronchial conditions
- aspiration
- foreign body
- microlithiasis

Parenchymal conditions
- cryptogenic fibrosing alveolitis
- alveolitis/interstitial lung disease
- sarcoidosis
- chronic haemosiderosis

Pulmonary vascular conditions
- pulmonary embolism

Drugs (e.g. ACE inhibitors, inhaled agents)

Environmental/occupational exposure

Pleural effusion

Left ventricular failure

Neuromuscular conditions

Psychogenic conditions

chronic obstructive pulmonary disease (COPD), bronchiectasis, postnasal drip syndrome (PNDS) and aspiration, or an exposure to an occupational or environmental agent such as an allergen. If cough onset is acute and severe, pulmonary diseases associated with the pulmonary vasculature, including pulmonary embolism and pulmonary venous congestion should also be considered.

Subacute cough

Subacute cough lasts three to eight weeks and is most frequently postinfectious in nature and antibiotics are rarely required. In this group, viral infection is most common, although bacterial infection (e.g. *Mycoplasma* spp.) may supervene and occasional isolates of *Bordetella pertussis* are detected. Treatment is usually directed at PNDS and upper airway irritability, using nasal decongestants and antihistamines, and consideration is given to a trial of inhaled ipratropium. For more severe symptoms a trial of inhaled corticosteroids may be considered. Whooping cough may be suggested if paroxysms of coughing, posttussive vomiting, and/or an inspiratory whooping sound are present. Although whooping cough is not usually life threatening in adults, prompt treatment of *Bordetella* with azithromycin, clarithromycin or erythromycin may prevent spread to susceptible children.

Chronic cough

Chronic cough exists when cough persists for longer than eight weeks. Although most respiratory conditions may present with chronic cough (Table 1), prospective studies have suggested that about 90% of cases may be attributed to one of the four most common causes, namely asthma, PNDS, gastro-oesophageal reflux disease (GORD) and chronic bronchitis. In approximately 10 to 20% of cases, two or more contributing factors may coexist, which may require independent treatment and/or investigation.

Pathophysiology

No single pathological mechanism has yet been identified to explain chronic cough. There continues to be a debate over the precise nature of mechanoreceptors and chemoreceptors, which stimulate the afferent limb of the cough reflex. It is clear, however, that these receptors are widely distributed through the upper and lower aerodigestive tracts. In many patients with chronic cough, upper airway hyper-reactivity with variable upper airway obstruction is evident, whereas in patients with GORD, significant impairment of laryngopharyngeal mechanosensitivity has been identified.

The wide variety of causes of chronic cough suggests that the mechanisms inciting cough are multiple, but there is some evidence to suggest a common final pathway.

Neurophysiological studies have demonstrated that proinflammatory mediators, such as bradykinin, increase discharge frequency in the sensory nerves (autonomic and somatosensory C-fibres), which forms the afferent limb of the cough reflex. At the peripheral nervous system level, histological studies of epithelium in patients with chronic cough have demonstrated both an increase in density of neural tissue and increased expression of the cough receptor, transient receptor potential vanilloid-1 (TRPV-1). At the central nervous system level, plastic changes in intrinsic and synaptic excitability at the brain stem, spinal or ganglionic level may be mechanisms contributing to increased cough reflex sensitivity. Both features may help explain the increased cough sensitivity to nonspecific stimuli that is a hallmark of chronic cough.

Inflammatory patterns in chronic cough appear disparate. In nonasthmatic chronic cough, neutrophils and neutrophil chemotactic factors (such as interleukin-8 and tumour necrosis factor-α) predominate, yet a subgroup of patients with chronic cough and nonasthmatic

eosinophilic bronchitis (eosinophilic airway infiltration in the absence of asthma) is also recognised in the cough clinic. Importantly, the eosinophil predominant pattern of airway inflammation is likely to be responsive to a trial of inhaled corticosteroids, but not the neutrophil predominant pattern.

Measurement of cough

Beyond a patient's description of cough, several tools are available to create semi-quantitative assessments of severity. An example of such a tool is the Leicester Cough Questionnaire, a 19-item questionnaire that assesses chronic cough-related impairment of quality of life. This tool is being progressively validated and has the capacity to measure changes in quality of life longitudinally, describing treatment responses both in clinical and research practice. Tussometers or cough counters have been used in a variety of research settings but technical limitations have limited their practical use.

Conversely, challenge testing similar to the bronchoprovocation test used in asthma has been more reliably used in clinical practice. Capsaicin, a chilli-derived noxious stimulus, triggers activation of C-fibres in the upper airways after inhalation. By inhaling progressively more concentrated solutions of capsaicin (or citric acid), thresholds of cough sensitivity, defined as the concentration needed to cause two (C2) and five coughs (C5), may be established. Patients with chronic cough invariably have an increased sensitivity to capsaicin. Capsaicin cough sensitivity improves after the specific treatment of cough precipitants, such as proton pump inhibitor (PPI) therapy in GORD-induced cough, and inhaled corticosteroid therapy in asthma-induced cough.

Clinical evaluation and investigations

Initial evaluation of patients with chronic cough aims to identify factors known to be associated with the condition including smoking, occupational irritant exposure, medications that induce cough (e.g. ACE inhibitors) and structural lung disease (e.g. COPD, bronchiectasis and interstitial lung disease). Although cough characteristics are not highly specific, a few points are worth noting and include the following:

- sputum production suggests the presence of chronic bronchitis, bronchiectasis, respiratory tract infection and/or sinusitis
- patients frequently deny sputum production and often this is only confirmed when they are prompted about early morning throat clearing
- diurnal variation in cough with

ACE inhibitors and cough[1]

- The mechanisms of ACE inhibitor-induced cough remain unclear. Possible protussive mediators include bradykinin and substance P, which are degraded by ACE and therefore accumulate in the upper airway or lungs when the enzyme is inhibited.
- The onset of cough may occur within a few hours of the first ACE inhibitor dose or months after the initiation of therapy.
- The incidence of ACE inhibitor-induced cough has been reported to be in the range of 5 to 35% among patients who have been treated with these agents.
- The incidence of cough associated with angiotensin-receptor blockers appears to be similar to that of ACE inhibitors.
- Resolution typically occurs within one to four weeks after cessation of ACE inhibitor therapy, but in some individuals cough may linger for up to three months.
- The only consistently effective treatment for ACE inhibitor-induced cough is the cessation of the medication causing the cough.
- In the minority of patients, cough will not recur after the reintroduction of ACE inhibitor therapy.

nocturnal or early morning worsening may suggest asthma
- worsening at night or when lying in bed may suggest GORD or PNDS as inciting factors
- GORD may induce cough while a person is speaking on the phone due to diaphragmatic flattening during phonation and increased intra-abdominal pressure (most people sit while talking on the phone)
- smoking history, ACE inhibitor use (see box on this page), inhaled and/or recreational drug use, and a history of exposure to dusts and gases should also be elicited.

Examination findings that should be sought include sinus tenderness, rhinitis or polyposis with nasal secretions, dental and periodontal disease, and anatomical abnormality of the oropharynx and uvula. The neck should be palpated for goitre or masses indicative of intrathoracic disease. During chest examination, the presence of hyperinflation, wheeze or coarse crepitations may suggest diagnoses of asthma, COPD or bronchiectasis.

Examination of the upper airway by rhinoscopy (nasendoscopy) is a simple and underutilised element of the physical examination that confidently identifies rhinitis and nasal polyposis. In some cases, a specialist ear, nose and throat review involving a detailed endoscopic examination may identify local lesions and features, such as reflux-related cord irritation. A sinus CT scan may confirm chronic sinusitis, anatomical variants and nasal polyposis, whereas normal sinus x-rays have a high negative predictive value. A peak flow diary, spirometry with measurement of bronchodilator responsiveness and, if necessary, bronchoprovocation testing may help identify asthma; negative bronchoprovocation results have high negative predictive value for asthma as a cause of chronic cough. Radiological evaluation with a chest radiograph, and if necessary high resolution CT scan, can define major structural pathologies such as bronchiectasis and emphysema and rarer entities such as pulmonary fibrosis.

A flexible approach to investigating GORD is necessary. Empirical treatment, oesophageal manometry, oesophageal pH monitoring, gastroscopy, laryngoscopy or bronchoscopy can help confirm the diagnosis. The gold standard for the confirmation of GORD as a precipitant of cough is the beneficial response to reflux-directed therapy, although given the possibility of spontaneous resolution of cough this standard remains flawed.

Fibreoptic bronchoscopy is not generally required but is indicated in the investigation of chronic cough if there are clinical, chest x-ray or CT abnormalities that raise suspicion of infection, local or obstructive lesion, foreign body or infiltrate. Bronchial mucosal biopsy may help identify eosinophilic bronchitis, and bronchial lavage allows microbiological sampling for chronic infective processes such as mycobacterial or fungal infection.

Treating the common causes of chronic cough

In general, response of chronic cough to appropriate treatment is successful in approximately 90% of cases; however, treatment may be required at a higher dose or for a longer duration than usually indicated. In the practical setting, clinically guided empirical treatment is the first line therapy for the major precipitants. Treatment should be directed firstly at PNDS, then asthma and then GORD (Table 2). Despite being such a common phenomenon, there are few areas in which treatment is confidently evidence-based. (In a recent review four of 12 recommendations were based on expert opinion and eight recommendations were made on the basis of a low level of evidence.)

Postnasal drip syndrome

The symptoms and signs of PNDS-induced cough (also known as upper airway cough syndrome) may be non-specific, and a firm diagnosis will not always be achieved by medical history and physical examination alone. Symptoms suggestive of PNDS include a sensation of a liquid dripping down the throat, a tickle in the throat, hoarseness and constant throat clearing, in addition to typical nasal symptoms of blockage

and discharge. A recent history of upper respiratory illness is often present.

The differential diagnosis of PNDS is broad and includes allergic and non-allergic rhinitis, postinfectious rhinitis, bacterial sinusitis, allergic fungal sinusitis, rhinitis due to anatomic abnormalities and physical or chemical irritants, occupational rhinitis, rhinitis medicamentosa, rhinitis of pregnancy and nasal polyposis. The mechanism by which these diverse causes induce cough is uncertain, although it seems likely that stimulation of the afferent limb of the cough reflex in the hypopharynx or larynx is involved.

Treatment is directed at the specific cause. If it is not possible to advance a diagnosis more specific than PNDS then a trial of a first generation antihistamine and a decongestant should be undertaken; a treatment response is often evident within one to two weeks.

Asthma

Asthma symptoms are frequently evident in individuals with chronic cough, although this diagnosis may not be made until formal lung function with spirometry and bronchoprovocation testing have been performed. The presence of small volumes of sputum is not uncommon in asthma, although this finding not infrequently leads away from a diagnosis of asthma towards a diagnosis of chronic bronchitis and hence reluctance to trial inhaled corticosteroids. If asthma is suspected as a cause of cough then an inhaled corticosteroid with a short-acting β-agonist should be trialled. Responses may take up to eight weeks of treatment and oral corticosteroids may be required.

GORD

GORD has been implicated in up to 41% of adults with chronic cough. However, as both of these conditions are so common it is difficult to distinguish between their coexistence and a clear cause and effect relationship. Nevertheless, GORD has been mechanistically implicated in cough both directly and indirectly.

Achieving certainty that GORD contributes to cough can be difficult because there are multiple potential links between GORD and chronic cough. For example, GORD has been shown to induce cough by direct acid stimulation of the lower oesophagus via a tracheobronchial reflex, by irritant activity at the level of the larynx and pharynx, and by direct microaspiration into the lung itself. Additionally, the oesophageal refluxate may incite cough by a mechanism independent of acid – that is, alkali or volume mediated oesophageal stimulation.

Another difficulty in achieving certainty that GORD contributes to cough is the

variability of markers of cough between individuals. At the clinical level, GORD-induced cough is often silent, occurring in the absence of 'typical' gastrointestinal reflux symptoms such as retrosternal discomfort, dyspepsia or acid brash. Similarly, investigation of cough may provide false-negative (silent) results. The demonstration of gastric acid or pepsin in the airways might be strongly supportive of GORD and aspiration as causative of cough, yet conversely the failure to identify gastric acid in the distal oesophagus by pH monitoring may be diagnostically unhelpful due to the effect of PPI therapy or an inability to exclude nonacid (volume or alkaline) reflux.

Patients often fail to recognise atypical GORD-related symptoms, which may include burning in the throat, morning hoarseness, redness/throat irritation, difficulty swallowing/lump in throat, choking episodes, constant throat clearing, burning chest pain or tightness when

Table 2. Treating the common causes of chronic cough

Condition	Treatment
Asthma	Short-acting β-agonist (e.g. salbutamol 100 μg two puffs as required or terbutaline 500 μg one puff as required) Inhaled corticosteroids (e.g. fluticasone 250 μg one puff twice daily or budesonide 400 μg twice daily) Long-acting β-agonist (e.g. salmeterol 50 μg twice daily or eformoterol 6 μg twice daily) Oral corticosteroid (e.g. prednisolone 50 mg for one week)
Postnasal drip syndrome	
Allergic rhinitis	Nasal corticosteroids (e.g. fluticasone 100 μg twice daily until cough is controlled then 50 μg once daily) Antihistamine (e.g. loratadine 10 mg once daily or cetirizine 10 mg once daily) Decongestant (e.g. pseudoephedrine 30 mg three times daily)
Nonatopic rhinitis	Nasal corticosteroid Anticholinergic (e.g. ipratropium two to four sprays into each nostril two to three times daily) Decongestant
Vasomotor rhinitis	Anticholinergic Decongestant
Polyposis	Nasal/systemic corticosteroid
Sinusitis	Antibiotic (e.g. doxycycline 100 mg twice daily for two weeks with or without extension) Nasal douche (sinus rinse/FESS) Irritant/allergen avoidance
Gastro-oesophageal reflux disease	Conservative measures: • elevate the head of the bed • do not eat for two to three hours before going to bed • eat smaller, more frequent meals • eliminate caffeine, alcohol, carbonated beverages, chocolate, mint, fatty foods and nicotine • take a liquid antacid after meals and as required for symptoms • avoid bending, straining, constipation or tight clothes • lose weight, if indicated Medications: • proton pump inhibitor (e.g. omeprazole 20 mg twice daily) • H_2-receptor antagonist (e.g. ranitidine 150 mg twice daily) • prokinetic agent (e.g. domperidone 10 mg three times daily)
Nonasthmatic eosinophilic bronchitis	Smoking cessation Short-acting β-agonist (e.g. salbutamol 100 μg two puffs as required or terbutaline 500 μg once as required) Long-acting anticholinergic Inhaled corticosteroid (e.g. fluticasone 250 μg one puff twice daily or budesonide 400 μg twice daily)

performing Valsalva-like manoeuvres such as peak flow or gym exercises, night time or early morning awakening or even recurrent pneumonia/bronchitis-type symptoms.

PPI treatment is the most effective treatment for GORD and has been shown to control symptoms and heal oesophagitis in about 80% of cases. Treatment failure, however, is recognised if not well defined. Among the treatment responders, 86% will achieve symptom control within four weeks and 99% by eight weeks.[2] The failure of PPI treatment warrants some attention as a variety of reversible factors may be identified. Adherence is the key and not infrequently related to patients' disbelief that GORD or even silent GORD could be related to chronic cough. Dose timing is important as PPIs are most effective if given 30 minutes before meals and are significantly less effective if given more than 60 minutes before meals or at bedtime. PPI failure may also be more frequent in patients with nonerosive reflux disease or hiatus hernia or those with genetic polymorphism for CYP2C19 expression responsible for the hepatic metabolism of PPIs, although the clinical significance of these factors is yet to be confirmed.

Multichannel intraluminal impedance, a novel measure of oesophageal function, with 24-hour pH monitoring, may help further characterise nonacid reflux and potentially help identify patients with GORD who are likely to respond to surgical approaches such as laparoscopic fundoplication.

Nonasthmatic eosinophilic bronchitis

It has been suggested that nonasthmatic eosinophilic bronchitis accounts for as many as 12% of cases seen in the tertiary referral cough clinic. These patients present with cough and airway eosinophilia and usually respond within four weeks to appropriate inhaled corticosteroid therapy. Eosinophilic bronchitis may be diagnosed if eosinophils comprise more than 2.5% of spontaneous/induced sputum cells, although the finding is not pathognomonic and may be found in patients with asthma, cough variant asthma, atopic cough and COPD. The key distinction of eosinophilic bronchitis from asthma is the demonstration of normal lung physiology with no evidence of airflow obstruction or bronchial hyper-responsiveness. Each of these conditions responds well to inhaled corticosteroid therapy.

Antitussive treatment for cough suppression

Although the principal approach to the treatment of chronic cough is the identification and appropriate treatment of the cause of the cough, in some individuals distressing cough persists and cough suppression becomes the important objective. If cough is associated with significant secretions and mucus, the suppression of cough may have a negative impact and strategies aimed at improving mucus clearance need to be incorporated.

As yet few convincing and satisfactory trials of antitussives have been published. The most compelling data come not from clinical trials investigating established cough but from models of induced cough in healthy controls or in acute cough associated with upper respiratory tract infection. Codeine, however, has been compared with dextromethorphan in a double-blind, crossover trial using both an objective and a subjective assessment of efficacy in 16 patients with chronic, stable cough. Both preparations, at a dose of 20 mg, were similarly effective in reducing the frequency of cough. Dextromethorphan, however, lowered cough intensity to a greater degree than codeine and was considered the better antitussive by most patients.[3]

More recently, a trial comparing low dose, slow-release morphine sulphate (5 mg twice daily) with placebo has suggested some benefit in cough scores in individuals with chronic cough. After a four-week treatment period, however, two-thirds of patients continuing into an open-label extension phase sought dose increases to 10 mg twice daily, suggesting that cough suppression was suboptimal in these patients. In addition, 40% complained of constipation and 25% described drowsiness, which may potentially limit the usefulness of this treatment.[4]

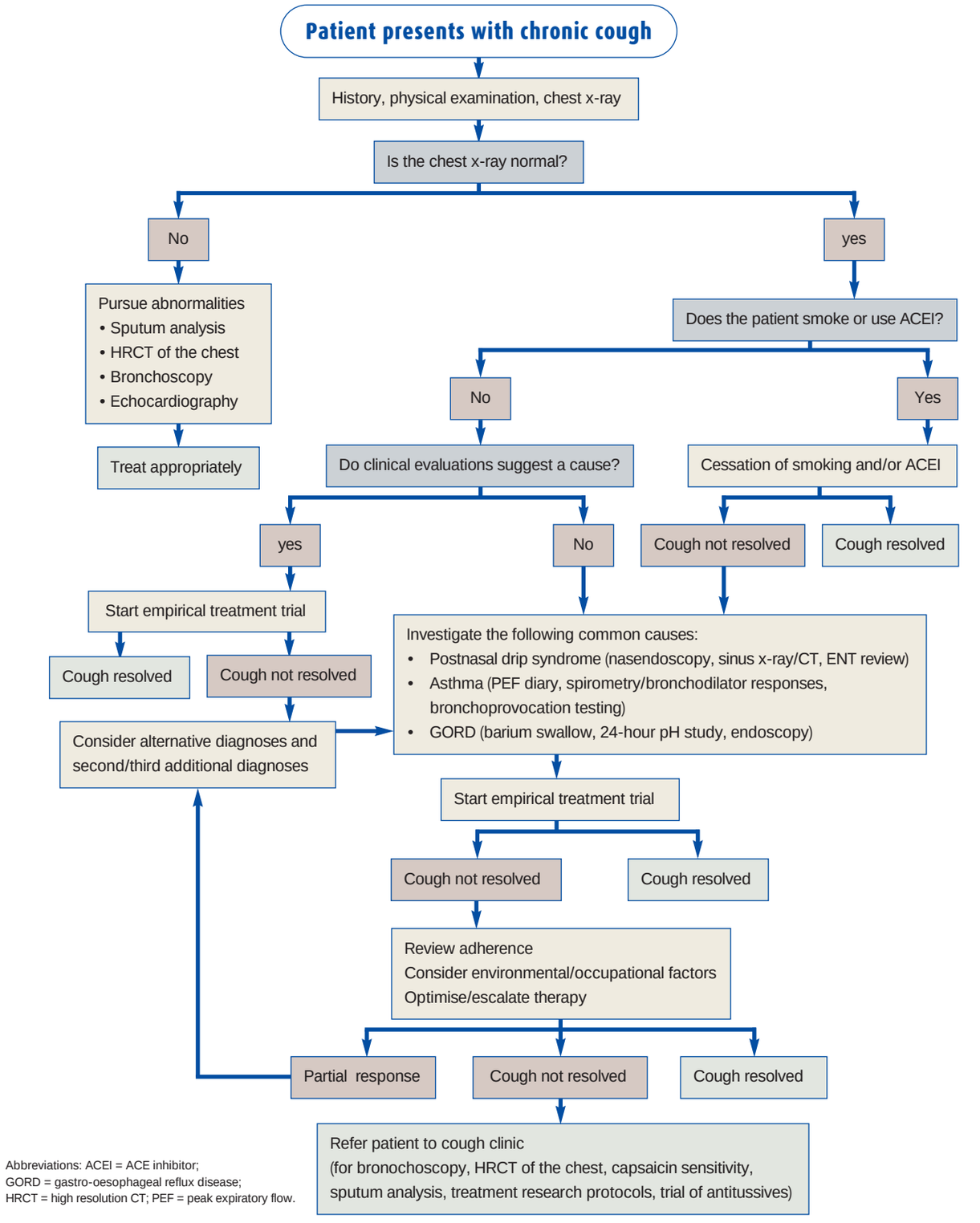
Codeine has recently been shown to be no more effective than placebo in individuals with chronic bronchitis, suggesting that sputum production may impact on opiate responsiveness. Both this and the study mentioned above suggest that groups of responders and nonresponders will be identified through empirical trials.

Common over-the-counter antitussives tend to include an opiate analgesic (e.g. dextromethorphan, codeine or pholcodine) either alone or in combination with one or several other agents including antihistamines (e.g. brompheniramine, chlorpheniramine), decongestants (e.g. pseudoephedrine), mucolytic expectorants (e.g. guaifenesin, bromhexine), oral antiseptics (e.g. cetylpyridinium chloride), local anaesthetics (e.g. benzocaine) and paracetamol. If PNDS is suspected, a combination of an opiate analgesic, antihistamine and decongestant may be helpful. At night it might also be helpful to select a formulation containing a slightly sedating antihistamine, such as doxylamine or chlorpheniramine, and avoid decongestants such as pseudoephedrine that can cause some CNS stimulation (pseudoephedrine is relatively contraindicated in patients with severe hypertension or ischaemic heart disease and with concurrent monoamine oxidase inhibitor use).

New and more effective treatments for cough suppression are sorely needed and avenues of current investigation include neurokinin and bradykinin receptor antagonists, novel opiates with more acceptable side effect profiles and ion channel modulators acting at transient receptor

continued

Diagnostic and management algorithm for chronic cough



Abbreviations: ACEI = ACE inhibitor; GORD = gastro-oesophageal reflux disease; HRCT = high resolution CT; PEF = peak expiratory flow.

potential channels, potassium channels and chloride channels.

Management algorithms

A variety of algorithms have been developed to address chronic cough with success rates of over 90% given appropriate therapy (one approach is given in the algorithm on page 43). If readily identifiable factors are evident there should be a realistic expectation that the cough should be largely resolved or diminished by appropriate treatment of the single or multiple factors contributing to it. If a preliminary examination fails to suggest a specific cause for cough and a chest x-ray is within the normal limits then PNDS, asthma and GORD should be considered as these few diagnoses account for up to 90% of causes of chronic cough.

Challenges in diagnosis and management

There are a number of challenges in diagnosing and managing patients with chronic cough. Chief amongst these is the recognition and communication of realistic therapeutic expectations. Cough should be recognised as an expected consequence of a variety of established lung diseases, including interstitial lung disease, bronchiectasis and COPD. In these circumstances the aims are to ameliorate cough by strategies that reduce inflammation, clear infection and reduce exposure to inhaled irritants such as cigarette smoke, and thereafter maximise the clearance of mucus and secretions from the airways.

However, features such as airway distortion, airway collapse, mucus hypersecretion and an inability to avoid inhaled irritants (continued smoking) may continue to promote cough and a patient may be resistant to further intervention. In these individuals, cough may persist despite our best efforts, and therefore the objectives may focus on minimising cough and enabling patients to deal best with exacerbations.

Role of the cough clinic

Although the majority of patients with chronic cough can be managed in primary care, a minority (5 to 10%) continue to have chronic cough despite an initial investigation and treatment. The cough clinic enables the co-ordination of initial investigations and also the addition of specialist investigations such as bronchoscopy and high resolution CT.

Studies comparing general respiratory clinics with specialist cough clinics have shown improved evaluation outcomes within the specialist clinics, perhaps related to a clearer definition of investigative and treatment protocols. Treatment algorithms in specialist clinics may also be extended to include newer investigational agents. Patients attending cough clinics who may have suffered from cough over a period of years should also benefit from experienced appraisal of the likely outcome of the cough assessment.

Conclusion

If a specific cause for chronic cough cannot be found in a patient and a chest x-ray is within the normal limits then PNDS, asthma and GORD should be considered as possible causes of cough. It is important to remember that in approximately 10 to 20% of cases two or more causes may contribute to cough and that these conditions may require specific treatment. Referral of patients to a cough clinic should be considered in patients who continue to have chronic cough despite initial investigation and treatment. Some useful online resources for chronic cough are listed in the box on this page. MT

Useful online resources

The Australian Lung Foundation
Adult chronic cough patient information sheet: www.lungnet.com.au/fact_sheets/chronic-cough-adults-health.html

British Thoracic Society
Cough guidelines: recommendations for the management of cough in adults: www.brit-thoracic.org.uk/guidelinescough

European Respiratory Society
The diagnosis and management of chronic cough: www.ersnet.org/ers/lr/browse/viewPDF.aspx?id_attach=9730

American College of Chest Physicians
Diagnosis and management of cough executive summary, evidence-based clinical practice guidelines: www.chestjournal.org/cgi/reprint/129/1_suppl/1S

International Society for the Study of Cough
www.issc.info

Cough Journal
Published by BioMed Central: www.coughjournal.com

References

1. Dicpinigaitis PV. Angiotensin-converting enzyme inhibitor-induced cough. ACCP evidence-based clinical practice guidelines. Chest 2006; (1 Suppl): 169S-173S.
2. Poe RH, Kallay MC. Chronic cough and gastroesophageal reflux disease: experience with specific therapy for diagnosis and treatment Chest 2003; 123: 679-684.
3. Matthys H, Bleicher B, Bleicher U. Dextromethorphan and codeine: objective assessment of antitussive activity in patients with chronic cough. J Int Med Res 1983; 11: 92-100.
4. Morice AH, Menon MS, Mulrennan SA, et al. Opiate therapy in chronic cough. Am J Respir Crit Care Med 2007; 175: 312-315.

DECLARATION OF INTEREST: Dr Stirling has served on advisory boards for Astra Zeneca and has received speaker's honoraria from Boehringer, Astra Zeneca and GlaxoSmithKline.