

1 **British Thoracic Society Clinical Statement on the Assessment and**
2 **Management of Cough in Children**

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34 Summary of clinical practice points

35 Definition

- 36 • Cough is both a symptom and a clinical sign and should be assessed systematically using
37 history, examination and, where possible, direct or recorded observation of its characteristics.
- 38 • **Acute cough** is responsible for 25% of paediatric primary care appointments
- 39 • **Chronic cough** has an adverse on the quality of life of the child affected and their family

40 Classification

- 41 • **Classification by duration is fundamental:**
 - 42 ○ *Acute cough* (<4 weeks) is usually caused by a recent respiratory tract infection (RTI)
43 and is self-limiting.
 - 44 ○ *Chronic cough* (>4 weeks) is more likely to be associated with an underlying
45 respiratory disorder, though clinical context and red flag features are more important
46 than duration alone.
- 47 • **Classification by aetiology improves diagnostic precision:**
 - 48 ○ *Specific cough* is attributable to a recognisable cause (e.g. asthma, cystic fibrosis,
49 protracted bacterial bronchitis) based on identification of specific cough traits.
 - 50 ○ *Non-specific cough* has no identifiable specific cough traits.
- 51 • **Classification by acoustic quality provides diagnostic clues:**
 - 52 ○ A *wet cough* suggests airway secretions often associated with infection.
 - 53 ○ A *dry cough* suggests airway irritation/inflammation without excess secretions.
 - 54 ○ Distinctive sounds (e.g. barking, brassy, paroxysmal, honking) may indicate specific
55 underlying disorders.

56 Clinical assessment

- 57 • A history, including environmental exposures, and clinical examination are key to
58 establishing the underlying cause.
- 59 • Key aspects within history and examination (Table 2 and 3) help to identify possible
60 diagnosis

61 Investigations and treatment principles (Figure 2 and 3)

- 62 • Although **acute cough** is most commonly caused a recent RTI, other causes such as inhaled
63 foreign body need to be considered.
- 64 • In the absence of specific cough traits suggesting a non-viral cause, **acute cough** should
65 be managed with reassurance and watchful waiting.
- 66 • In children with **chronic dry cough** and no specific cough traits, reduce exposure to
67 airborne irritants and review after 4–8 weeks. Inhaled corticosteroids should only be used
68 if there is objective evidence of asthma.
- 69 • In children with **chronic wet cough** and no specific cough traits, obtain sputum culture (if
70 feasible) and initiate a 2–6 week course of antibiotics. Resolution of cough by two weeks
71 supports a diagnosis of protracted bacterial bronchitis (PBB); if there is no improvement,
72 further investigation for alternative causes of chronic wet cough is required.
- 73 • If specific cough traits are present, management should follow condition specific guidance

74 Indications for referral

- 75 • Urgent referral in the presence of red flags (Box 2 and 3)
- 76 • Diagnosis uncertainty, limited access to diagnostic tools
- 77 • Treatment failure
- 78 • Parent/carer or healthcare professional concern

79

80 **Key message**

81 Assessment of cough in children depends on careful clinical evaluation to identify the underlying
82 cause. The vast majority of those with an acute cough only require reassurance. In contrast those
83 with chronic cough are more likely to require investigation and treatment.

84

85 **Introduction**

86 Cough is a vital physiological reflex that aids the clearance of secretions and foreign material from the
87 airways and helps prevent aspiration. It is therefore normal for children to cough. However, cough can
88 also be a symptom of a serious or even life-threatening illness. Good clinical skills are required to
89 determine whether an individual child needs investigation, or reassurance (the hardest diagnosis being
90 'normal child'). The differential diagnosis of cough encompasses virtually the whole of paediatric
91 respiratory medicine and missing a diagnosis can have catastrophic long-term consequences. For
92 example, untreated or misdiagnosed chronic wet cough can lead to bronchiectasis[1–3] meaning the
93 progression to permanent lung damage could potentially have been prevented with earlier
94 treatment.[4] The previous BTS guidelines[5] have been cited nearly 600 times but were published
95 almost 20 years ago so there is an urgent need for an update that includes the latest evidence. The
96 definitions used in the 2008 document no longer align with other major international guidelines.[6–8]
97 Moreover, the long-term significance of chronic wet cough has been increasingly appreciated, as has
98 the knowledge that isolated dry cough is not a form of asthma and will likely improve over time.

99

100 **Scope**

101 This clinical statement provides guidance and information for healthcare professionals and trainees
102 involved in the care of children presenting with acute or chronic cough across all healthcare settings.
103 It outlines the pathophysiology of cough, offers clear definitions of acute and chronic cough, and
104 provides recommendations for appropriate investigations and management strategies. In addition, it
105 identifies clinical features that should prompt referral to secondary or tertiary care. The treatments of
106 the specific respiratory disorders causing chronic cough (see Table 4) are beyond the scope of this
107 clinical statement, and should be determined in line with established, disease specific guidelines.

108

109 **Methodology**

110 The Clinical Statement Group (CSG) was jointly chaired by AG-L and FJG. Membership included
111 paediatric respiratory consultants, a general paediatric consultant with respiratory interest, a general
112 practitioner with respiratory interest, a paediatric respiratory specialist nurse, an advanced
113 nurse/paramedic practitioner, a specialist respiratory physiotherapist, a paediatric respiratory trainee,
114 and two parents of children affected by recurrent or chronic cough. The overall content reflects the
115 scope approved by the BTS Standards of Care Committee (SOCC) and used the methodology described
116 in the clinical statement production manual.[9] Following initial discussions, the clinical statement was
117 divided into sections and allocated to subgroups who drafted the content. The individual sections, and
118 later the document combined by FJG, were reviewed and edited by CSG members before being
119 submitted to the BTS SOCC for review and approval and subsequently posted on the BTS website for
120 public consultation and peer review.

121

122 **Rationale for a separate statement on management of acute and chronic cough in children, distinct**
123 **from the adult statement**

124 The causes of cough in children differ significantly from those in adults, highlighting the need for
125 separate statements .[5] Using a paediatric-specific, structured approach to the assessment of children
126 with chronic cough improves clinical outcomes by promoting early diagnosis and treatment, as well as
127 the timely referral of those requiring specialist input.[10,11] A number of key adult diagnoses have
128 little or no relevance in paediatric practice:

- 129 • Cough hypersensitivity syndrome

130 Although some conditions increase cough receptor sensitivity in children, if the underlying condition
131 is treated, the cough resolves. This suggests cough hypersensitivity in children is a consequence of an
132 underlying condition rather than a disease itself.[12] The possibility that cough hypersensitivity
133 syndrome develops in adulthood is supported by a lack of difference in the prevalence of Arnold's ear-
134 cough reflex (initiation of cough via auricular branch of the vagus nerve when the external auditory
135 meatus is mechanically simulated) between children with chronic cough and healthy controls. In adults
136 it is more than ten times higher in those with chronic cough.[13]

- 137 • Gastroesophageal reflux (GOR)

138 The relationship between GOR and chronic cough in children is complex. When investigating children
139 with chronic cough, GOR can be identified in up to a quarter of individuals[14] but the temporal
140 relationship between GOR and cough is usually not causal[15] and is not responsive to treatment
141 trials.[16] Children with chronic cough should therefore only be started on acid-suppressive therapy if
142 they have clinical features of GOR such as recurrent regurgitation/dystonic neck posturing in infants,
143 or heartburn/epigastric pain in older children.[17] The treatment should be discontinued if ineffective.

- 144 • Post-nasal drip (PND)

145 It is unclear if PND causes cough in children. Furthermore, the criteria for the diagnosis of PND in
146 children are unclear with uncertainty about whether it is a symptom or a clinical sign. Furthermore,
147 cough in the presence of posterior pharyngeal secretions may be caused by co-existing airways
148 pathology.[18]

- 149 • Cough variant asthma

150 Although cough is common in children with asthma, this is almost always associated with wheeze and
151 the vast majority of children with isolated cough do not have asthma.[19–22]

152

153 **The burden of cough on the child, their family and the healthcare system**

154 Acute cough is responsible for around a quarter of primary care consultations in children.[23] These
155 consultations are almost twice as common in those aged 0-4 years than 5-14 years, and slightly more
156 prevalent in boys than girls (1.2 to 1).[24] The annual cost to the United Kingdom (UK) National Health
157 Service (NHS) of acute cough in pre-school children in 2008 was estimated as £31M[25], based on
158 inflation this suggests a cost in 2025 of £52M. A child with acute cough caused by a respiratory tract
159 infection (RTI) is likely to have disturbed sleep, reduced oral intake and disturbance of play or other
160 daily activities.[26] In a prospective study based in primary care, 52% of children with acute cough
161 were absent from school/childcare for at least one day, resulting in time off work for 28% of mothers
162 and 20% of fathers.[26] Chronic cough is also extremely burdensome for the child and their family. It
163 affects all aspect of the child's life including their ability to sleep, play and attend school/nursery.[27]
164 Parents repeatedly seek medical consultation with 80% of affected children having ≥5 doctor visits and
165 53% >10 doctor visits in the previous 12 months.[28] The parents of children with chronic cough have
166 high levels of emotional distress caused by stress which improves when the child ceases coughing.[28]

167 **Definition and Pathophysiology**

168 Cough is a complex, protective airway reflex that functions to clear the respiratory tract of mucus,
169 pathogens, and foreign matter.[29] While it serves an essential biological function, persistent or
170 abnormal cough can signal disease, particularly in children.[30] The cough reflex arc is initiated by
171 mechanical or chemical activation of cough receptors in the pharynx, larynx and proximal airways. This

172 produces a signal which is sent to the 'cough centre' in the medulla oblongata via afferent pathways in
173 the vagus nerve. The brainstem then generates an efferent signal which travels via the vagus, phrenic
174 and motor nerves to expiratory musculature to produce the cough.[29,31] The cough centre in the
175 medulla oblongata can also be activated by a signal from the cerebral cortex allowing the voluntary
176 initiation of cough.

177 The mechanical event of cough has three phases:[32]

- 178 1. Inspiratory Phase: A rapid inhalation takes place through a wide-open glottis. The size of the
179 inhalation varies in relation to the stimulus and receptor type but can be up to vital capacity.
- 180 2. Compressive Phase: Glottis is closed and abdominal and respiratory muscles contract to
181 increase the intrathoracic and intraabdominal pressure.
- 182 3. Expiratory Phase: Rapid glottis opening produces an explosive flow of air from the lungs
183 removing airway secretions/debris and producing the characteristic cough sound.

184 Box 1: What is and isn't normal?

- Preschool children may have >10 URTIs per year causing acute cough.[33]
- A 'normal' cough resolves spontaneously within four weeks and is not associated with red flag features (e.g., weight loss, clubbing, haemoptysis, persistent wet cough).
- A chronic, unremitting cough, or cough with red flag symptoms, is not normal and warrants further evaluation.[34]

185

186 **Classification of cough**

187 Cough is a symptom but also an important clinical sign. Clinicians should determine the duration of
188 the cough from the history and listen to the cough to categorise its acoustic quality. If the child does
189 not cough during the consultation, parents should be requested to record the child's cough on a smart
190 phone or recording device, if they have access to one.[35]

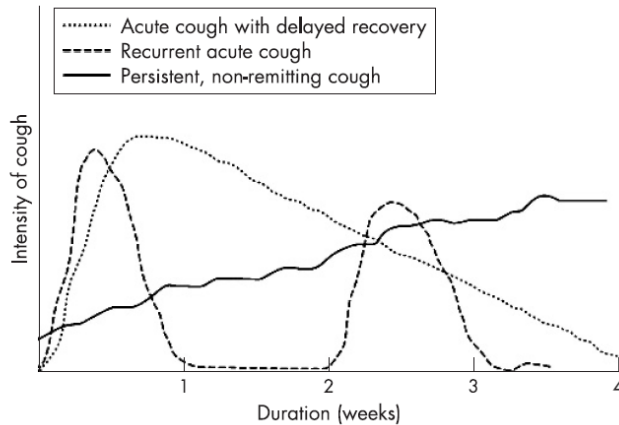
191 Classification by duration

192 The duration of cough is fundamental to its clinical assessment.

- 193 • **Acute Cough:** Lasts <4 weeks in children.
194 A four-week cutoff is used as 80-90% of coughs in children associated with a recent RTI will
195 resolve by this time point. Most such cases do not require further investigation or
196 treatment.[36–38] Recurrent severe acute cough may warrant further investigation.
- 197 • **Chronic Cough:** Persisting, non-remitting cough lasting >4 weeks.
198 The likelihood of a serious underlying respiratory disorder increases when the duration of a
199 daily cough exceeds four weeks. When such children are assessed by a respiratory specialist,
200 almost half are diagnosed with protracted bacterial bronchitis and a third with a new chronic
201 lung disease such as asthma or bronchiectasis.[36] It is important to distinguish chronic cough
202 from recurrent acute cough which can be difficult if the time between episodes is short. See
203 Figure 1.

204 It is important to note the four-week cut-off is an arbitrary guide as to who needs investigation and
205 there will be exceptions. For example, a child who has only been coughing for two weeks but is
206 systemically unwell or has red flag features will need immediate investigation / treatment, whereas a
207 child who has been coughing for six-weeks but the intensity is improving will only need a period of
208 watchful waiting while their post-infective cough resolves.

209 Figure 1: Cough classification by duration according to the patterns of intensity vary over time[39]



210

211 Classification based on aetiology (by underlying cause)

212 Aetiological classification improves diagnostic precision and management:

- 213 • **Specific Cough:** Attributable to a recognisable cause based on 'specific cough traits' identified
- 214 in the history or examination. Causes include asthma, cystic fibrosis (CF), pertussis, primary
- 215 ciliary dyskinesia (PCD) and recurrent / chronic pulmonary aspiration (R/CPA). See Tables 4 and
- 216 5.
- 217 • **Non-specific Cough:** No identifiable 'specific cough traits'.

218 Classification of cough by acoustic quality

219 The acoustic quality of a cough can suggest the location of the causative pathology within the

220 respiratory tract and/or the underlying cause. See Table 1. A key point when differentiating between

221 coughs, is whether it is wet or dry.[40,41] A wet cough implies excess airways secretions which can be

222 caused by either increased production or impaired mucociliary clearance. Both parental and clinician

223 report of wet cough correlates well with the volume of secretions seen at bronchoscopy.[42] It is

224 important to remember a wet cough in a young child is unlikely to be productive, as they usually

225 swallow, rather than expectorate sputum. Not all coughs are easily classified as wet or dry. In chronic

226 infections such as pertussis, the cough changes between wet and dry during the different stages of the

227 illness. In R/CPA the cough associated with episodes of aspiration is usually dry but becomes wet

228 during a lower respiratory tract infection (LRTI). If a cough is variable i.e. sometimes wet and

229 sometimes dry, it is considered wet.[43] Other acoustic qualities include a barking, croupy cough

230 suggesting upper airway obstruction, a brassy cough suggesting airway malacia, paroxysmal cough

231 with or without a whoop suggesting pertussis, a honking cough suggesting somatic cough disorder and

232 a repetitive, throat-clearing cough suggesting tic cough.[44] See Table 1.

233 Table 1: The acoustic quality of a cough and associated significance

Acoustic Quality	Potential Significance
Wet	LRTI, PBB, CSLD, bronchiectasis.
Dry / non-productive	Viral URTI, post-infective cough, asthma*, inhaled irritants.
Barking or croupy,	Croup, inhaled foreign body, bacterial tracheitis
Brassy	Laryngo-tracheomalacia,
Paroxysmal or spasmodic	Pertussis, pertussis-like illness, inhaled foreign body.
Honking	Somatic cough syndrome.
Repetitive / throat-clearing	Tic cough.

234 *Asthma is unlikely to be the cause of chronic cough if not associated with wheeze.

235 CSLD: chronic suppurative lung disease, LRTI: lower respiratory tract infection, PBB: protracted bacterial bronchitis, URTI:

236 upper respiratory tract infection.

237 **Clinical assessment of the coughing child**

238 A thorough history and physical examination are crucial first steps in the evaluation of a child with
 239 cough. The findings will guide the clinician as to what, if any, investigations are needed.

240 History

241 The first aspect of the history is to determine when the cough started and how the intensity has
 242 changed since that time. This will enable the differentiation between acute cough, recurrent acute
 243 cough and chronic cough.[23] See Figure 1. For acute cough, the history is also used to identify the
 244 likely cause and red flags highlighting those requiring urgent investigation, treatment, referral or
 245 admission. For chronic cough, the history is focused on trying to identify specific cough traits
 246 suggestive of a specific disorder. Key aspects for a cough history are detailed in Table 2.

247 Environmental exposures

248 When evaluating a coughing child, clinicians should routinely assess environmental exposures due to
 249 their important role in the onset and persistence of respiratory symptoms. Key exposures include
 250 direct and second-hand tobacco smoke, e-cigarette vapour, indoor dampness/mould, household
 251 allergens, and outdoor or indoor air pollution.[45] Children are particularly susceptible to these factors
 252 due to their developing lungs, higher respiratory rates, and increased time spent in the home
 253 environment. Identifying and addressing such exposures is critical, as targeted mitigation can reduce
 254 symptom burden, prevent recurrent presentations, and support long-term respiratory health.[45]

255

256 Table 2: Key aspects of the history and their clinical significance.

	Key aspects of history	Clinical Significance
Age of onset	As neonate	Laryngeal cleft, H-type TOF, primary ciliary dyskinesia, cystic fibrosis
Type of onset	Sudden or associated with choking episode Preceded by coryzal illness	Inhaled foreign body Post-infective cough
Pattern of intensity	Gradually improving Acute episodes with periods of remission Worse at night or seasonal Absent when sleeping Suggestibility and distractibility	URTI / post-infective cough Recurrent acute cough Asthma Tic cough / somatic cough disorder Tic cough
Acoustic quality	Wet / dry / barking / croupy / brassy / paroxysmal / honking / throat clearing	See Table 1
Triggering factors	Cold air / exercise Feeding	Asthma* Laryngeal cleft, TOF, R/CPA, unsafe swallow
Alleviating factors	Bronchodilators Antibiotics	Asthma* Protracted bacterial bronchitis / bronchiectasis**
Associated symptoms	Fever, runny / blocked nose / sore throat Poor feeding, lethargy, or irritability Wheeze Failure to thrive / steatorrhoea Night sweats / weight loss / haemoptysis Chronic otitis media and/or rhinosinusitis	URTI Systemic infection Asthma* / bronchiolitis / bronchiolitis obliterans Cystic fibrosis Tuberculosis Primary ciliary dyskinesia
Past medical & surgical history	Severe, persistent, unusual or recurrent (SPUR) infections Rectal prolapse Neurodisability Previous TOF repair Neonatal respiratory distress	Immunodeficiency Cystic fibrosis R/CPA Swallowing incoordination causing R/CPA Primary ciliary dyskinesia
Medications	ACE inhibitors	May cause chronic cough
Immunisation status	Pertussis / influenza / pneumococcus / BCG	Increased risk if unvaccinated
Environmental exposures	Tobacco smoke / e-cigarette vapor	Worsen asthma, chronic cough, LRTIs

	Key aspects of history	Clinical Significance
	Ambient or household air pollution Allergens (pets / pollen / HDM / mould)	Worsen asthma, chronic cough, LRTIs Asthma*
Social & family history	Recent travel or tuberculosis exposure	Tuberculosis

257 *Asthma is unlikely to be the cause of chronic cough if not associated with wheeze.

258 ** Bronchiectasis is a lung condition with multiple potential underlying causes and improvement with antibiotics reflects
259 treatment of bronchiectasis-related infection rather than resolution of the underlying disorder

260 ACE: Angiotensin-converting enzyme, LRTI: HDM: House dust mite, Lower respiratory tract infection, R/CPA: recurrent or
261 chronic pulmonary aspiration, TOF: Tracheoesophageal fistula, URTI: Upper respiratory tract infection,

262 Examination

263 All children with cough should have a full systemic examination, including assessment of their general
264 health, nutritional status (including height and weight centiles / z scores) and examination of their
265 upper and lower respiratory system.[46] In a child with acute cough, the aim of the examination is to
266 identify the unwell and/or conditions requiring investigation or treatment.[47] For the child with
267 chronic cough, the aim of the examination is to identify cough specific pointers which suggest an
268 underlying respiratory disorder and to identify those who need to be referred to secondary or tertiary
269 care. Key examination findings and their clinical significance are shown in Table 3.

270 Table 3: Key examination findings and their clinical significance

	Finding	Clinical Significance
General Appearance and observations	Lethargy, confusion, poor perfusion Elevated respiratory rate or heart rate Grunting Stridor, barking cough Pyrexia Nutritional status (height & weight percentiles / z scores)	Signs of severe illness / sepsis[48] Key signs of the 'unwell child' Sign of severe illness Upper airway obstruction, inhaled foreign body Infective cause Failure to thrive: cystic fibrosis, immunodeficiency, interstitial lung disease
Inspection	Digital clubbing Nasal flaring, tracheal tug, I/C or S/C recession Harrison sulci	CSLD, bronchiectasis, interstitial lung disease Signs of respiratory distress Chronic / recurrent respiratory distress
Palpation	Tracheal deviation Subcutaneous emphysema Asymmetrical chest expansion	Atelectasis, effusion, thoracic mass Air leak Pneumothorax, atelectasis, consolidation, effusion
Percussion	Dull Stony dull Hyperresonant	Consolidation, atelectasis Pleural effusion Pneumothorax, acute severe asthma
Auscultation	Decreased breath sounds Bilateral wheeze, polyphonic wheeze Unilateral wheeze, monophonic wheeze Coarse crackles Fine crackles	Consolidation, atelectasis, pneumothorax, effusion Asthma, bronchiolitis, bronchiolitis obliterans Inhaled foreign body, obstruction of main bronchi Bronchiolitis, LRTI Interstitial lung disease
Upper respiratory tract	Drizzling or muffled voice Mucopurulent nasal discharge Nasal polyps Tonsillar exudates Chronic otitis media and/or rhinosinusitis	Epiglottitis, bacterial tracheitis URTI, acute or chronic rhinosinusitis Cystic fibrosis Tonsillitis Associated with primary ciliary dyskinesia
Extra-pulmonary	Conjunctivitis, coryza, rash Subconjunctival haemorrhages, petechiae Recurrent or severe infections Poor feeding, tachycardia, tachypnoea, sweating, oedema, hepatomegaly Dextrocardia	URTI Intense coughing (pertussis) Immunodeficiency Cardiac failure Associated with primary ciliary dyskinesia

271 CSLD: Chronic suppurative lung disease, I/C: intercostal, LRTI: Lower respiratory tract infection, S/C: Subcostal, URTI: Upper
272 respiratory tract infection,

273 Investigations

274 *Radiological*

- 275 • Chest X-ray (CXR):

276 Most children with acute cough do not need a CXR. This includes those with uncomplicated community
277 acquired pneumonia which is a clinical diagnosis.[49] A CXR should only be performed on a child with
278 acute cough in the following circumstances:

- 279 1. Signs or symptoms suggesting inhaled foreign body.[50]
280 Perform inspiratory and expiratory CXRs if the child is able to cooperate or bilateral
281 decubitus views if not. A normal CXR does not exclude a foreign body.
282 2. Signs or symptoms suggesting pleural effusion / empyema.[51]
283 If effusion confirmed, size and appropriateness for drainage to be informed by chest
284 ultrasound scan.
285 3. Sign or symptoms suggesting a pneumothorax.[52]
286 4. A history of haemoptysis.[53]

287 To aid the diagnosis of LRTI, tuberculosis, pulmonary oedema and malignancy.

288 In contrast to acute cough, all children with a chronic cough should have a CXR.[54]

- 289 • High resolution computerised tomography (HRCT):

290 Bronchiectasis is confirmed using HRCT images.[55] These are usually obtained using a multidetector
291 CT (MDCT) scanner which acquires a high-resolution, volume dataset of the whole thorax in a single
292 breath hold.[56] These data can then be reformatted into multiple thin axial slices (0.5mm) or 3D
293 reconstructions. Due to the associated radiation dose,[57] the timing of the HRCT is a balance between
294 making an early diagnosis, obtaining the best quality scan and limiting the need for a repeat. HRCT is
295 also used to diagnose a wide range of other respiratory disorders including bronchiolitis obliterans
296 (aided by expiratory images), childhood interstitial lung disease (chILD) and congenital lung
297 abnormalities (may require contrast enhancement). See Chronic Cough Section. HRCT should only be
298 performed in secondary or tertiary centres with paediatric radiographers and radiologists, to minimise
299 radiation dose while maximising image quality.

300 *Microbiological*

- 301 • Viral molecular testing

302 The vast majority of children with a cough caused by a viral URTI or LRTI do not need viral molecular
303 testing. However, if the child is unwell enough to require admission, this should be performed for the
304 purposes of infection control, including SARS-CoV-2, influenza A&B, and respiratory syncytial virus
305 (RSV).[58]

- 306 • Sputum microscopy, culture and sensitivity (MC&S)

307 Obtaining sputum from a child with a wet cough is difficult as most will swallow it rather than
308 expectorating. In children with PBB, identifying the causative pathogen promotes antibiotic
309 stewardship by enabling a narrow spectrum antibiotic to be prescribed.[59] In secondary and tertiary
310 settings, obtaining a lower airway sample for MC&S, such as induced sputum, should be attempted
311 prior to prescribing a prolonged antibiotic course.[60] Routinely obtaining a sputum sample
312 (spontaneously expectorated or induced) is not recommended in children with acute LRTI.[61]

- 313 • Bordetella Pertussis

314 In a child with suspected whooping cough, early confirmation of the diagnosis helps control outbreaks,
315 limit complications and enable notification to UK Health Security Agency (UKHSA). The optimal
316 diagnostic test depends on the duration of cough:[62]

- 317 ○ <21 days: Culture and/or PCR of a nasopharyngeal swab or aspirate.
318 ○ >21 days: Serological testing of blood, or saliva for anti-pertussis toxin IgG

319 *Bronchoscopy*

- 320 • Rigid bronchoscopy

321 Rigid bronchoscopy is the gold-standard for foreign body removal[63] and for the identification of
322 structural airway abnormalities such as laryngeal cleft and H-type tracheoesophageal fistula (TOF).[64]

323 • Flexible bronchoscopy:
324 Flexible bronchoscopy with bronchoalveolar lavage (FB-BAL) is an important investigation in children
325 with chronic cough to obtain lower airway microbiological samples and diagnose airway
326 abnormalities.[65] The potential advantages of FB-BAL need to be balanced against the burden for the
327 child and their family.[66] In PBB, FB-BAL should be reserved for those with recurrent relapses and may
328 not be necessary if an induced sputum is obtained in this patients' group.[60] When undertaking FB-
329 BAL to obtain lower airway samples, sampling 6-lobes (including lingula) increases microbiological
330 yield.[59,67,68] Various biochemical (pepsin or bile acids) and cytological (lipid-laden macrophages)
331 BAL markers of aspiration have been investigated but none are used routinely.[69] Flexible
332 bronchoscopy is also increasing being used for foreign body removal and has been shown to be safe
333 and efficient, with a shorter procedure time and fewer complications.[70,71]

334 *Impedance/pH study*

335 Investigations for acidic and non-acidic reflux are not commonly necessary when investigating children
336 with cough but may be used in selected patients. Combined oesophageal multichannel intraluminal
337 impedance and pH (MII-pH) monitoring is the investigation of choice for diagnosing GOR and non-acid
338 reflux (NAR). As reflux is common, its detection does not necessarily mean it is the cause of the cough.

339 *Swallowing assessment*

340 In children with suspected R/CPA, a videofluoroscopic swallowing study (VFSS) can be used to evaluate
341 if a child can swallow safely.[72] This is normally requested after assessment by a speech and language
342 therapist. Flexible endoscopic evaluation of swallow (FEES) is an alternative method to assess
343 swallow.[73]

344 *Asthma investigations*

345 In a child with a clinical suspicion of asthma, the investigations should be undertaken as per the
346 BTS/NICE/SIGN Joint Guideline on Asthma.[74] Objective tests to confirm the diagnosis in those ≥ 5
347 years include FeNO, spirometry with bronchodilator reversibility, total IgE, skin-prick tests and blood
348 eosinophil count. In those < 5 years old, if there is strong clinical suspicion of asthma, an 8-week trial
349 of inhaled corticosteroid can be used.

350 *CF investigations*

351 Most children with CF in the UK are now identified by newborn screening. Quantitative pilocarpine
352 iontophoresis with sweat chloride analysis and CFTR genetics are used to confirm the diagnosis and
353 test older children with clinical suspicion of CF.[75,76] CF is diagnosed in those with a sweat chloride
354 ≥ 60 mmol/L and those with an intermediate sweat chloride (30-59mmol/L) but two disease causing
355 CFTR variants. CF can also be diagnosed by nasal transepithelial voltage measurements in individuals
356 with high clinical suspicion of CF but a normal (< 30 mmol/L) sweat chloride.[77]

357 *PCD investigations*

358 There is no single, gold-standard diagnostic test for PCD. Tests used to aid diagnosis include: ciliary
359 beat pattern and frequency (measured using digital high-speed video), ciliary ultrastructure assessed
360 using electron microscopy, ciliopathy mutation screening, and immunofluorescence[78,79] Nasal nitric
361 oxide (nNO) concentrations are low in individuals with PCD[80], this together with symptom based
362 scoring systems[81,82] can be used as a gateway to further testing

363 When to refer

364 Most children with cough present to primary care and are managed appropriately without the need
365 for referral. Although referral pathways may show geographical variation and depend on the level of
366 expertise of the healthcare professionals involved, the principles of referral from primary to secondary
367 or secondary to tertiary care are:

- 368 1. When the diagnosis is in doubt, and the practitioner does not have the facilities to perform
369 further testing.

- 370 2. When the progress of the disease is not as anticipated, or the treatment is not working.
 371 3. When any party (professional or family) remains concerned.
 372 4. If a child with a known diagnosis (e.g. cystic fibrosis) presents to a setting such as primary care
 373 in which otherwise unsupported management is inappropriate.
 374 Specific guidance on when to refer children with acute and chronic cough is included in the relevant
 375 sections below.

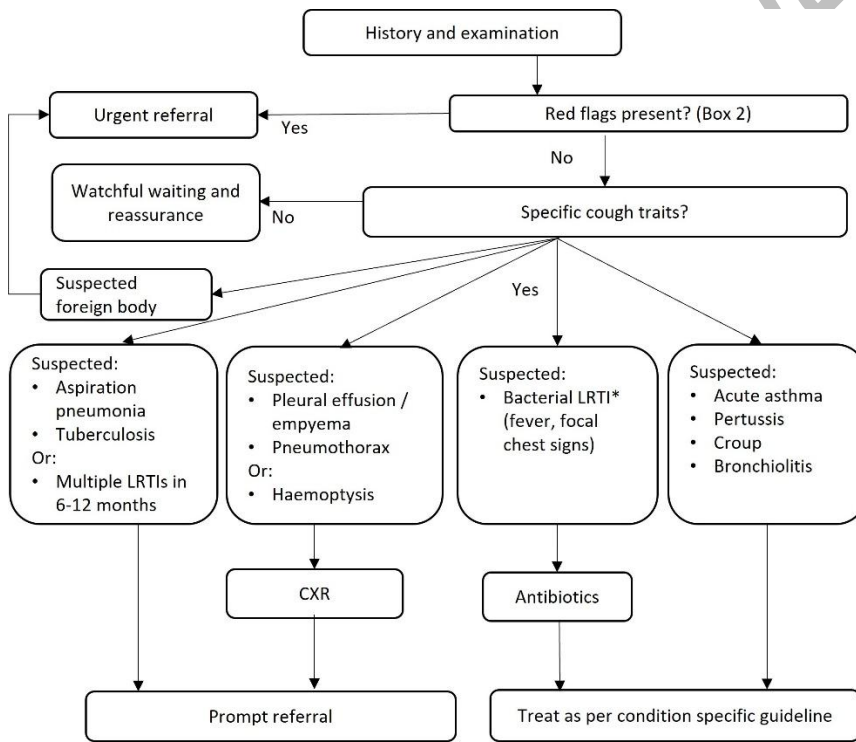
376

377 **Acute Cough**

378 Structured Assessment

379 The majority of cases of acute cough are self-limiting but it can be a feature of a more significant
 380 condition requiring investigation and treatment such as inhaled foreign body or bacterial LRTI.[83]
 381 Occasionally, acute cough is a presenting symptom of a serious underlying disorder such as CF,
 382 immunodeficiency or PCD.[83] The structured assessment of the child with acute cough begins with a
 383 detailed history and systemic examination. The first aim is to identify children who require referral or
 384 admission as they are acutely unwell. The second aim is to identify those with specific cough traits
 385 suggesting a cause requiring investigation, treatment, referral or admission (see Table 4).

386 Figure 2: Structured assessment for the child with acute cough



387

388 CXR: Chest X-ray, LRTI: Lower respiratory tract infection, CAP: Community acquired pneumonia.
 389 For Red Flags see Box 2.

390

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395 **Table 4: Causes of acute cough and associated ‘specific cough traits’**

DIAGNOSIS	COUGH NATURE	SPECIFIC COUGH TRAITS	
		History	Examination
Asthma exacerbation	Dry	Previous episodes, atopy	Wheeze, signs of respiratory distress
Aspiration pneumonia*	Wet +/- choking	Associated with feeding / meals, neurodisability	Normal / pyrexia / focal chest signs
Bronchiolitis	Variable	<1 year old, coryza, reduced feeding	Bilateral wheeze and crackles, signs of respiratory distress
CAP / LRTI (viral / bacterial / mixed)	Wet	Unwell, reduced appetite	Pyrexia, focal chest signs, signs of respiratory distress
Inhaled foreign body*	Choking / barking	Sudden onset, choking episode, relentless	Noisy breathing, unilateral chest signs
Laryngotracheobronchitis (croup)	Barking / croupy	Coryza, sore throat, hoarse voice	Pyrexia, stridor, signs of respiratory distress
Pertussis	Paroxysmal	Post-tussive vomiting, inspiratory whoop	Normal / sub-conjunctival haemorrhages
Tuberculosis*	Variable	TB contact / weight loss / night sweats / haemoptysis	Normal / focal chest signs/ weight loss
Viral URTI	Variable	Associated rhinorrhoea and sneezing	Normal / rhinitis / pharyngitis / lymphadenopathy

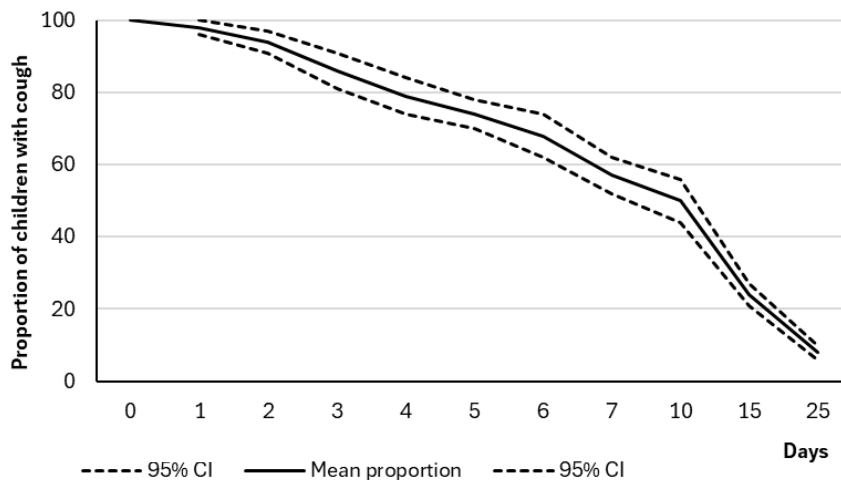
396 *Conditions likely to require referral to secondary/tertiary care for further investigation or management.
 397 CAP: community acquired pneumonia, LRTI: Lower respiratory tract infection, TB: tuberculosis, URTI: upper respiratory tract
 398 infection, ↑WOB: increased work of breathing.

399 **Management of acute cough**

400 *Watchful waiting and reassurance*

401 A systematic review of acute cough in preschool children (five studies with 866 participants) found
 402 cough resolution occurred in 50% by day 10 and in 90% by day 25.[37] See Figure 2. A subsequent
 403 cohort study of 766 children (median age 2.3 years) presenting to the emergency department with an
 404 acute respiratory infection and cough (duration <28 days), reported cough resolution in 64% of children
 405 by day 14 and 82% by day 28.[36] As such, the vast majority of children with acute cough do not
 406 require any investigations or treatment. It is important this information is imparted to the family in a
 407 thoughtful and understanding way, as they often have genuine concerns about the consequences of
 408 their child’s cough including fear of choking and long-term respiratory damage.[84]

410 **Figure 2: Proportion of children with persisting cough, 1-25 days after presenting with acute cough.**



411 Adapted from Thompson et al[37] which combines data from five studies[38,85–88] with a total of 866 preschool children.
 412 Duration of cough at presentation varied from 1 to 14 days.
 413

414 *Antibiotics for community acquired pneumonia (CAP)/LRTI*

415 A key aspect when managing children with acute cough is to avoid unnecessary prescribing of
416 antibiotics. When CAP/LRTI is suspected, antibiotics should be reserved for those with pyrexia and/or
417 focal chest signs as this increases the risk of complications and re-consultation.[89] The BTS[49] and
418 NICE[90] CAP guidelines both suggest oral amoxicillin as first-line treatment. The CAP-IT trial found 35-
419 50mg/kg/d oral amoxicillin for three days was non inferior to 70-90mg/kg/d for seven days in
420 preventing need for antibiotic re-treatment but children coughed for on average two days longer.[91]
421 A macrolide should be prescribed for pneumonia caused by an atypical pathogen (e.g. *Mycoplasma*
422 *pneumoniae*).

423 *Treatment of Pertussis*

424 If pertussis is confirmed or strongly suspected in a child coughing for <14 days who is not acutely
425 unwell, a macrolide should be prescribed (3-5 days azithromycin or 7 days clarithromycin).[92] Children
426 should stay off nursery/school until they have completed 48 hours of antibiotic treatment or for 14
427 days from onset of coughing if not treated. When recovered, any outstanding vaccinations should be
428 offered. For a child with pertussis who has been coughing for <14 days, close contacts should be
429 prescribed prophylaxis if they are vulnerable to severe complications or at increased risk of
430 transmitting to those that are vulnerable.[93]

431 *Over the counter (OTC) cough treatments*

432 Multiple systemic reviews have failed to show any benefit from the use of over the counter cough
433 treatments in children and report occasional instances of harm.[94–96] There is very weak evidence
434 that honey may improve cough symptoms although a systematic review concluded there was no strong
435 evidence for or against its use.[97]

436 When to refer

437 Although most cases of acute cough are self-limiting, it should not be forgotten that recurrent
438 especially severe acute cough may be the harbinger of an underlying important diagnosis. Any child
439 with acute cough who has red flags suggesting severe illness or an inhaled foreign body should be
440 referred urgently for immediate assessment. Children with aspiration pneumonia, tuberculosis or
441 multiple LRTIs (not URTIs) in the last 6-12 months should also be referred but the urgency of the
442 referral will vary depending on the clinical state of the child and the medical history. Red flags for
443 urgent referral of children with acute cough are shown in Box 2. There is no evidence base to guide
444 how many severe episodes should prompt investigation / referral but we empirically suggest a child
445 with ≥3 episodes in 12 months should be referred to a paediatrician with respiratory expertise for
446 further investigation.

447

448 Box 2: Red flags for urgent referral in children with acute cough

- Lethargy or confusion
- Poor perfusion (capillary refill ≥2 seconds)
- Poor oral intake (<75% of normal intake) or clinical dehydration
- Stridor
- Increased WOB (intercostal / subcostal recession, nasal flaring, grunting)
- Tachycardia
- SpO₂ ≤92%
- Possible inhaled foreign body: Sudden onset choking, unilateral chest signs
- Possible aspiration pneumonia: Sudden onset cough with feeds / meals or after vomiting

449 SpO₂: oxygen saturation, WOB: work of breathing.

450

451

452

453

454 **Causes of acute cough**

455 Viral URTI

456 Viral URTIs are the commonest cause of acute cough with most healthy children experiencing multiple
457 episodes each year.[34] Other symptoms include rhinorrhoea, sneezing, nasal congestion, sore throat
458 and malaise.[98] The commonest causative pathogens are rhinovirus, coronavirus, influenza virus,
459 parainfluenza virus and RSV. Although a quarter of children remain symptomatic after two weeks,[99]
460 the vast majority of URTIs are self-limiting and require no specific investigations or
461 treatment.[5,83,100–102]

462 CAP/LRTI

463 CAP is a clinical diagnosis based on the presence of persistent or recurrent fever >38.5°C, signs of
464 respiratory distress and focal chest signs.[49] In children hospitalised with CAP, a respiratory virus is
465 detected in 30-70%, atypical bacteria in 7-17% and pyogenic bacteria in 2-8%.[103] The commonest
466 causative viruses are RSV, influenza and rhinovirus and the commonest bacteria are *Streptococcus*
467 *pneumoniae*, *Haemophilus influenzae* and *Mycoplasma pneumoniae*. [104,105] It is not possible to
468 reliably differentiate between viral, bacterial or mixed CAP based on history, examination or point of
469 care tests.[44,84] For treatment see 'Antibiotics for community acquired pneumonia (CAP)/LRTI' on
470 P11.

471 Inhaled foreign body

472 Inhaled foreign bodies are most prevalent in children aged 1-3 years, commonly impacting in the larynx
473 or right main bronchus.[63] This causes acute cough which classically starts abruptly with a choking
474 episode and may be associated with stridor or unilateral wheezing. Severe cases can be life
475 threatening. If the foreign body is not removed it can cause chronic cough, atelectasis, abscesses, and
476 cavities.

477 Pertussis (whooping cough)

478 Pertussis is a highly contagious infection caused by *Bordetella pertussis* that is implicated in up to 20%
479 of children coughing for >2 weeks.[106] The initial 'catarrhal' phase lasts 1-2 weeks causing mild URTI
480 symptoms. The 'paroxysmal' stage follows, characterised by bouts of coughing sometimes
481 accompanied by an inspiratory whoop (commoner in younger children).[107] This can lead to hypoxia,
482 apnoea and respiratory failure, as well as subconjunctival and intracranial haemorrhages.[108] A
483 gradual transition to the "convalescent" stage follows after 1 to 2 months, characterised by the cough
484 improving over several weeks.[109] Pertussis should be notified to the UKHSA Health Protection Team.
485 Details of testing for pertussis are on P8 and for treatment and prophylaxis of pertussis are on P10.

486 Bronchiolitis

487 Bronchiolitis is an acute viral LRTI that currently is most commonly caused by RSV, although this may
488 change in the future with maternal immunisation in pregnancy and the use of RSV monoclonal
489 antibody in high risk infants. It affects infants <1 year old and is the commonest reason for infants to
490 be admitted to hospital.[110] The usual presentation is with a short history of coryza, pyrexia, cough
491 and reduced feeding. Severely affected infants, particularly those <3 months old may have apnoeas.
492 Increased WOB, bilateral crackles and expiratory wheeze are the classical examination findings.
493 Treatment is supportive and includes ensuring adequate hydration and oxygenation.[110]

494

495 Aspiration pneumonia

496 Aspiration pneumonia refers to a LRTI caused by the inhalation of food, liquid, saliva or stomach
497 contents into the airway. Most children admitted to hospital with aspiration pneumonia have
498 underlying medical diagnoses such as neurodisability.[69,111]

499 Croup

500 Viral laryngotracheobronchitis (croup) is a common URTI in children. Although usually self-limiting, it
501 can be life-threatening. The infection causes subglottic, upper airway inflammation and obstruction

502 resulting in a hoarse voice, a barking/croupy cough, stridor and increased WOB. These symptoms are
 503 often preceded by a short history of cough, coryzal symptoms and pharyngitis. A single dose of oral
 504 dexamethasone is the mainstay of treatment although oral prednisolone and nebulised budesonide
 505 are also effective.[110]

506 Asthma exacerbation

507 Whilst exacerbations of asthma can be associated with an acute dry cough, the most common
 508 symptoms are expiratory wheeze and signs of respiratory distress. Isolated cough is rarely a sign of
 509 asthma. Triggers include URTI/LRTI, aeroallergens, environmental exposures, emotion, and non-
 510 adherence with preventive therapy. Treatment includes inhaled bronchodilators and
 511 corticosteroids.[113]

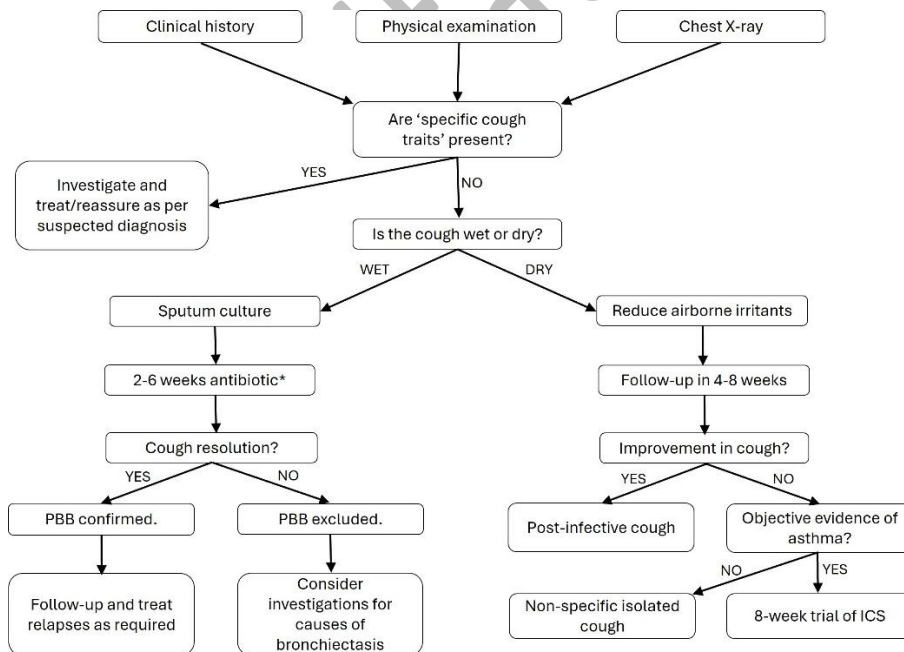
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513 **Chronic Cough**

514 Structured Assessment

515 The structured assessment of the child with chronic cough also starts with a detailed history, thorough
 516 examination and when possible, a chest radiograph. This allows identification of specific cough traits
 517 (see Table 5) indicating a specific respiratory diagnosis.[114] These diagnoses include the causes of
 518 bronchiectasis / chronic suppurative lung disease (CSLD), infections such as pertussis / tuberculosis
 519 and asthma. If suspected, these diagnoses should be confirmed with appropriate investigations and
 520 treatment. Some causes of chronic cough and their specific cough traits are shown in Table 4. A child
 521 with a chronic wet cough but no specific cough traits should be prescribed a course of an appropriate
 522 oral antibiotic to confirm or exclude protracted bacterial bronchitis (PBB).[115] If the cough does not
 523 resolve, investigations for the causes of bronchiectasis / chronic suppurative lung disease should be
 524 considered. For children with chronic dry cough but no specific cough traits, a period of observation
 525 will enable post-infective and non-specific isolated cough to be differentiated. See Figure 1.

526 Figure 3: A structured assessment for a child with chronic cough



527

528 If a cough is sometimes wet and sometimes dry, it is considered wet.

529 CXR: Chest x-ray, PBB: Protracted bacterial bronchitis. *A 4-6 week course should be used when prescribing in secondary /
 530 tertiary care with a review at 2 weeks, but a 2-week course may be appropriate for first episode, when prescribing in
 531 primary care or when sputum culture not possible.

532 **Table 5: Causes of chronic cough and associated specific cough traits**

DIAGNOSIS	COUGH NATURE	SPECIFIC COUGH TRAIT		DIAGNOSTIC TEST
		History	Examination	
Cystic fibrosis*	Wet	Steatorrhoea, recurrent LRTIs, nasal polyps, rectal prolapse, haemoptysis[116]	FTT, inspiratory crackles, finger clubbing	Sweat test CFTR genetics
Primary ciliary dyskinesia*	Wet	Neonatal onset, chronic sinusitis, otitis media with effusion, recurrent LRTIs[81,82]	Dextrocardia / situs inversus, inspiratory crackles, digital clubbing	nNO, ciliary structure and function, ciliopathy genetics
Inborn error of immunity / Primary immunodeficiency*	Wet	Severe, persistent, unusual or recurrent infections, chronic diarrhoea, parental consanguinity [117]	FTT	FBC, immunoglobulins, functional Ab's
Airway malacia	Dry	Brassy, barking or croupy cough. Worse with URTI / LRTI, exertion or feeding[118]	Stridor, signs of respiratory distress	Bronchoscopy
Asthma	Dry	Wheeze and breathlessness associated with allergies and family history[119]	Nil when well, wheeze with exacerbations	FeNO, eosinophil count, bronchodilator reversibility or serial PEFR
ChILD*	Dry	Shortness of breath, hypoxia[120]	Fine crackles, hypoxia, tachypnoea, signs of respiratory distress	HRCT chest, genetic tests
Prolonged post-infective cough	Dry	Viral URTI within 6-8 weeks, cough slowly improving[121]	Normal	Not needed
Somatic cough syndrome*	Dry	Bizarre honking or barking cough, settles when asleep, may increase when observed[122]	Normal	Not needed
Tic cough	Dry	Frequent throat-clearing cough, settles when asleep, may increase when observed[122]	Normal	Not needed
Non-acidic reflux	Variable	Chronic, variable cough, GOR symptoms non-responsive to PPI[123]	Often normal, feature of LRTI	MII-pH
Pertussis	Variable	Paroxysms of coughing, post-tussive vomiting, inspiratory 'whoop'[106]	Often normal, subconjunctival haemorrhage, hypoxia	Culture/PCR of per-nasal swab
Recurrent / chronic pulmonary aspiration*	Variable	Relentless cough with neonatal onset, choking / spluttering with feeds, previous thoracic surgery, recurrent LRTIs[43]	Often normal, features of LRTI	VFSS, FEES
Retained inhaled foreign body*	Variable	Acute onset with choking, relentless cough, recurrent LRTIs[43]	Stridor, unilateral respiratory signs	Inspiratory & expiratory CXR, bronchoscopy
Tuberculosis*	Variable	Weight loss, night sweats, haemoptysis[124]	Often normal, features of LRTI	CXR, culture/PCR of sputum, gastric lavage or BAL

533 *Conditions likely to require referral to secondary/tertiary care for further investigation or management.
 534 ChILD: childhood interstitial lung disease, FEES: fiberoptic endoscopic evaluation of swallowing, FTT: failure to thrive, GOR:
 535 gastro-oesophageal reflux, LRTI: lower respiratory tract infection, MII-pH: multichannel intraluminal impedance and pH
 536 monitoring, nNO: nasal nitric oxide, PCR: polymerase chain reaction, PEFT: peak expiratory flow rate, VFSS: videofluoroscopic
 537 swallow study.

538 **Treatment**

539 ***Chronic wet cough with no specific cough traits***

540 In a child with a chronic wet cough but no specific cough traits, a two-week antibiotic course is long
 541 enough to confirm PBB and extending beyond this duration does not increase the rate of cough
 542 resolution.[125] However, in children whose cough does resolve with two-weeks of antibiotic and
 543 therefore have PBB, extending the antibiotic duration to 4-6 weeks reduces the risk of PBB

544 relapse.[126,127] A two week course is therefore suggested for the first episode, or if sputum culture
545 is not possible. A 4-6 weeks course is recommended when prescribing in secondary/tertiary care.
546 Antibiotic choice should be guided by a respiratory sample from the lower airway. If this is not
547 available, co-amoxiclav is the first line choice.

548 Chronic dry cough with no specific cough traits

549 The commonest causes of chronic dry cough in children with no specific cough traits are post-infective
550 and non-specific isolated cough, both of which usually resolve over time without the need for
551 treatment. Identification and avoidance of cough triggers or exacerbating factors such as tobacco
552 smoke and e-cigarette vapor may expedite cough resolution.[128] Treatment with inhaled
553 corticosteroids should only be used if there is objective evidence of asthma.[74,119] In children with
554 non-specific isolated cough, management should include addressing child and parental stress and
555 concerns.[129]

556 Airway clearance

557 Airway clearance techniques (ACTs), including those that incorporate relevant devices, are a key
558 management approach for children with chronic wet cough of various origin [130] Similar to the adult
559 population[131], the approach uses the concept of treatable traits and although it lacks strong
560 evidence due to inherent challenges in relevant studies, most clinicians consider ACTs a vital part of
561 clinical care.[132] Having access to physiotherapists with relevant paediatric experience and being
562 taught ACTs were also identified as management priorities in a European Lung Foundation (ELF) survey
563 for paediatric bronchiectasis.[133] Importantly, there is no strong evidence in favour of a specific ACT
564 so patient or family preference should be considered as key to adherence. Implementing ACTs should
565 be tailored to the child's age and the underlying condition. The ACT needs to be taught by a trained
566 professional and adapted according to the child's response and clinical progression. Nebulised
567 hypertonic saline is the most widely used mucoactive agent. This stimulates coughing and reduces
568 secretion viscosity thereby aiding expectoration, but supportive evidence regarding outcomes is
569 scarce[134] and an RCT in adults with bronchiectasis questioned its effectiveness.[135]

570 When to refer

571 In addition to the general guidance regarding referrals given on P8, children with a chronic specific
572 cough such as those highlighted with asterisk in Table 4 are likely to require referral to secondary or
573 tertiary care for investigation or management. Red flags for urgent referral of children with chronic
574 cough are shown in Box 3.

575

576 Box 3: Red flags for urgent referral in children with chronic cough

- | |
|--|
| <ul style="list-style-type: none">• Symptoms from first days of life• Haemoptysis• Coughing or choking when eating/drinking• Chronic or recurrent otitis media• Chronic diarrhoea / steatorrhoea• Severe, persistent, unusual or recurrent infections• Weight loss / failure to thrive / faltering growth• Digital clubbing• Nasal polyps• Persistent crackles on auscultation• Persistent chest x-ray changes• Persisting fever• Signs of cardiac disease / failure |
|--|

577

578 Longterm outcomes for children with non-specific cough

579 Whilst most preschool children with chronic wet cough will become asymptomatic, some are still
580 coughing many years later. One study which prospectively followed-up 166 children diagnosed with
581 PBB, found the mean number of PBB episodes fell consistently over the subsequent five years as did
582 the percentage of children with recurrent PBB. However, 68% had ongoing/intermittent respiratory

583 symptoms at five years.[136] A second study obtained symptom questionnaires from 120 children
584 (median age of 6.8 years) who presented at <3 years of age with recurrent episodic or chronic wet
585 cough.[137] At follow-up, 57% of children were symptom free, 32% had mild symptoms and 11%
586 severe symptoms. Of those who had initially been diagnosed with PBB, 67% were symptom-free at
587 follow-up, 25% had mild symptoms and 8% had severe symptoms.[137] A third study performed a
588 clinical assessment and lung function on 62 children, 8 years after were diagnosed with PBB. At follow-
589 up 18% had a chronic wet cough in the last 12 months and 13% had an abnormal FEV₁. [138]
590 Bronchiectasis has been confirmed in 8-10% of children previously diagnosed with PBB.[136,139]

591

592 **Causes of Chronic Wet Cough**

593 Protracted bacterial bronchitis

594 PBB is a chronic endobronchial bacterial infection and the commonest cause of chronic wet cough in
595 young children from developed countries.[65] The diagnostic criteria are: 1) chronic wet cough; 2)
596 absence of specific cough traits suggesting another respiratory diagnosis; 3) cough resolution with an
597 appropriate oral antibiotic.[140] The identification of the causative pathogen from a lower airway
598 sample is not part of the diagnostic criteria but when available, promotes antibiotic stewardship.[141]
599 By definition, cough in PBB resolves with antibiotics but relapses are frequent and recurrent PBB
600 (>3/year) is associated with an increased likelihood of a future bronchiectasis diagnosis.[139,142] As
601 PBB is a diagnosis of exclusion, an alternative, specific cause such as bronchiectasis or
602 immunodeficiency should be considered particularly in recurrent PBB.[143] For treatment see '*Chronic
603 wet cough with no specific cough traits*' on P14-16.

604 Bronchiectasis and CSLD

605 Recurrent PBB and failure of a chronic wet cough to respond to four weeks of antibiotic are risk factors
606 for bronchiectasis.[139,144] The diagnosis of bronchiectasis in children is based on the presence of a
607 clinical syndrome (chronic or recurrent acute wet cough, sometimes with coarse crackles and digital
608 clubbing), confirmed by a broncho-arterial ratio [BAR] >0.80 on HRCT.[55] This is lower than the cutoff
609 used in adults (>1.0) as BAR increases with age.[145] The change from a normal to dilated airway
610 happens over time due to an ongoing or recurrent lung insults, such as infection or aspiration[143] and
611 the longer the insult is present, the more severe the bronchiectasis.[146] The development of
612 bronchiectasis therefore manifests as a clinical continuum with early features indistinguishable from
613 PBB, progressing to CSLD in which the clinical syndrome of bronchiectasis is present without airway
614 dilation, and eventually to bronchiectasis.[142] Interventions to remove or treat the insult, can halt or
615 reverse the progression towards fixed dilated airways.[147,148] Managing treatable insults such as
616 infection and aspiration is therefore key to preventing long term morbidity. When bronchiectasis is
617 confirmed, the potential causes (see Table 1) need to be excluded.

618

619 **Causes of Chronic Dry Cough**

620 Airway malacia

621 Airway malacia refers to the excessive collapsibility of the large airways due to laxity of the posterior
622 tracheal wall or weakness in the airway cartilage.[149] Congenital airway malacia (prevalence ≈1 in
623 2100 children) can occur in isolation or be associated with conditions such as tracheal-oesophageal
624 fistula or mucopolysaccharidoses.[150] Causes of acquired airway malacia include trauma, external
625 compression, prolonged positive pressure ventilation, and inflammation.[150] Chronic cough, typically
626 described as brassy or barking is present in up to 80%, wheeze or stridor may also be present.[151,152]
627 Diagnosis is based on >50% expiratory reduction in the cross-sectional airway diameter seen at
628 bronchoscopy.[118] End-inspiratory HRCT images paired with either end-expiratory or dynamic
629 expiratory HRCT images can also aid diagnosis.[118] The severity ranges from mild, only causing an

630 intermittent cough, to severe, with life-threatening episodes of respiratory arrest. In most children,
631 treatment is not needed as symptoms improve with age due to airway growth and strengthening.[150]
632 As severity increases, prophylactic antibiotics and chest physiotherapy may be used to prevent
633 recurrent LRTIs and positive end expiratory pressure delivered by non-invasive ventilation or
634 tracheostomy to prevent airway collapse. Aortopexy or airway stenting may be considered in the most
635 severe cases but are limited to specialist centres.[118]

636 Asthma

637 Although cough is common in children with asthma, this is usually associated with wheeze and the
638 vast majority of children with isolated cough do not have asthma.[19–21] Inhaled corticosteroids (ICS)
639 should only be prescribed to children aged 5-16 years with dry chronic cough if there is objective
640 evidence of asthma from FeNO, blood eosinophil count, bronchodilator reversibility or serial peak
641 expiratory flow measurements.[74]

642 Childhood Interstitial lung disease (chILD)

643 ChILD is a rare (4.4-8.2 cases per million per year) but likely under-estimated group of non-specific
644 disorders affecting the lung interstitium, alveoli, or small airways.[153–155] The underlying cause can
645 be genetic, inflammatory, environmental, related to systemic disease or unknown.[156] Neonatal
646 chILD classically presents with unexplained respiratory distress progressing to respiratory failure.[157]
647 Later clinical presentations develop insidiously or after a viral illness include dyspnoea, chronic dry
648 cough (up to 75% of patients) and failure to thrive.[158] Examination findings include hypoxia,
649 tachypnoea, increased WOB, fine crackles, and pulmonary hypertension.[159] The diagnosis is usually
650 based on HRCT or genetic testing, with lung biopsy reserved for inconclusive cases. Management is
651 challenging, with little evidence to inform clinical decisions. Corticosteroids are often tried, with
652 variable effectiveness. More targeted treatments like antifibrotic agents, JAK inhibitors, and gene or
653 cell-based therapies are emerging.[160] In the most severe cases, lung transplantation may be
654 considered.[161]

655 Prolonged post-infective cough

656 Post-infective cough is the commonest cause of chronic cough in children and refers to a persistent dry
657 cough which lingers after other symptoms of the causative RTI have resolved.[8,162] This happens in
658 10-20% of coughs caused by an acute RTI in children.[37,38] The cough reduces in frequency and
659 severity over time, so treatment is not required. However, it is important to monitor for 'cough specific
660 pointers' suggestive of an alternative diagnosis such as PBB, asthma or bronchiectasis.

661 Tic cough and somatic cough disorder

662 Tic cough (previously called habit cough) and somatic cough (previously called psychogenic cough) are
663 distinct entities but both are highly disruptive, crucially both are absent during sleep and occur in
664 children without underlying respiratory pathology.[122] Tic cough is a persistent, throat-clearing, dry
665 cough that classically develops after a LRTI. Its core features are suppressibility, distractibility,
666 suggestibility, variability, and the presence of a premonitory sensation.[163] Somatic cough syndrome
667 describes a loud, honking or barking cough that reflects the 'transfer of psychologic distress into a
668 physical symptom'. [122] In conditions, investigations are normal and should be avoided when possible.
669 Management is non-pharmacological and focuses on breaking the subconscious cycle with
670 reassurance and suggestion therapy.[164–166] Psychology input may be required in somatic cough
671 disorder.[122]

672 Non-specific, isolated cough

673 Children with a chronic dry cough, no specific cough traits, no improvement during a prolonged period
674 of observation and no objective evidence of asthma, should be labelled with non-specific isolated
675 cough. This is not a diagnosis in itself and may be caused by a specific but as yet unidentified cause.[5]
676 No medication has been shown to be beneficial for these children. Follow-up is needed, to identify
677 those who develop specific cough traits, suggesting a respiratory disorder.[30]

678 **Causes of Chronic Variable Cough**

679 Recurrent / Chronic Pulmonary Aspiration (R/CPA)

680 R/CPA is the repeated passage of food, drink, gastric refluxate or saliva into the subglottic airways.[167]
681 Chronic cough is the commonest symptom in children with R/CPA, whether or not they have
682 bronchiectasis. Aspiration is silent (no associated cough) in up to 28% of children with neurodisability
683 but may be suggested by wet/gurgly speech, rattly chest after feeds and recurrent LRTIs. Bronchiectasis
684 is highly prevalent, can develop rapidly but can also be reversed with early identification and aggressive
685 management of aspiration.[168] The cause of R/CPA can be structural, dynamic or GOR with aspiration.
686 Structural causes include congenital anomalies such as laryngeal cleft and 'H' type tracheoesophageal
687 fistula (TOF). The commonest dynamic cause is swallowing incoordination. This is commonest in
688 children with neurodisability but can also occur after TOF repair or tracheostomy insertion. In children
689 with neurodisability, R/CPA may be compounded by co-existent GOR +/- impaired laryngeal sensation
690 (ILS) which is a failure to recognize food, saliva, or gastroesophageal reflux in the larynx.[169] They are
691 also at risk of salivary aspiration causing aspiration symptoms that persist despite cessation of oral
692 feeding treatment / exclusion of GOR.

693

694 Tuberculosis

695 Tuberculosis (TB) should be considered in the differential diagnosis for child with chronic cough,
696 especially in areas where it is prevalent. The additional symptoms suggestive of active TB infection are
697 night sweats and weight loss. If suspected, pulmonary TB is confirmed by culture/PCR of early morning
698 gastric aspirate, sputum (spontaneously expectorated or induced) or BAL sample.[124] Tuberculin skin
699 test and interferon-gamma release assay (IGRA) do not differentiate between latent or active TB
700 infection.

701

702 Gastro-oesophageal reflux (GOR) and non-acidic reflux (NAR)

703 GOR alone should not be accepted as the cause of isolated chronic cough in otherwise healthy
704 children. In children with chronic cough and no overt regurgitation, international guidelines
705 recommend only investigating / treating GOR if one or more of the following are present: unexplained
706 feeding difficulties (refusing to feed, gagging or choking), distressed behaviour, faltering growth,
707 hoarseness or a single episode of pneumonia.[17,170] In children with neurodisability, the co-existence
708 of swallowing dysfunction and GOR can result in R/CPA.[171] In contrast to acid reflux, there is some
709 evidence that non-acid reflux (NAR), defined as reflux with pH >4.0 causes chronic cough, worsens
710 other respiratory diseases and causes pulmonary aspiration.[123,172]

711 **Summary**

712 The assessment of cough, whether acute or chronic, relies on clinical skills to identify the few children
713 who require investigation or treatment; the vast majority require only reassurance. The differential
714 diagnosis is very different from that in adults. Investigation pathways in those children in whom cough
715 portends a serious illness should be carefully focused. In particular, chronic wet cough of >4 weeks
716 duration may lead on to bronchiectasis and mandated aggressive investigation and management to
717 prevent chronic airflow obstruction and infection.

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