

## **DIAGNOSIS AND TREATMENT OF REFLUX COUGH**

Reflux cough is by far the commonest cause of undiagnosed or treatment resistant cough. I think the most important thing is to be confident of the diagnosis. Over the last ten years it has become increasingly obvious to me that there is a very characteristic history in patients with reflux cough, which can all be explained with the knowledge of the pathophysiology of airway reflux. Visit [issc.info](http://issc.info) and do the questionnaire. A score of over 13 indicates the diagnosis of reflux cough.

Almost all reflux is due to transient openings of the lower oesophageal sphincter (LOS). The LOS closes while we are asleep, increasing pressure by about 5-10 mmH<sub>2</sub>O. Reflux cough then is rare at night. When it does occur it indicates a very lax LOS. In complete contrast to asthma the reflux cougher wakes without coughing. It's when they get up, usually on reaching the bathroom or going down the stairs that they start coughing. This is the LOS opening physiologically to allow gas trapped in the stomach overnight to escape. Other patients start coughing with food, particularly breakfast. There are two times when this occurs, firstly during the act of swallowing, usually dry foods such as toast or biscuits. What happens is the pharyngeal lower oesophageal sphincter reflex causes LOS opening and the patient coughs just as they are about to swallow. Secondly, classic reflux occurs post prandially. However, reflux cough occurs earlier than heartburn, since the stomach acid has been neutralised by the food. The key is to remember that reflux cough is not the same as heartburn and is a much less closely acid related. It may even be a gaseous mist which is inhaled and this does not show up on any conventional investigations.

Another set of symptoms, which are often dismissed as non specific, which in reality are highly specific, is cough on phonation. Typically the patient will be speaking on the telephone (raised intra abdominal pressure through sitting) and right in the middle of a sentence the patient will start coughing and frequently have to hand the receiver over to someone else. This is the diaphragm, which normally holds the LOS shut, being used for phonation and allowing reflux waves to slip past. A similar phenomenon occurs on singing and laughing. Frequently the reflux wave hits the larynx and leads to aphonia or a change in the quality of the voice.

It is important to realise that the reflux frequently goes all the way to the larynx and indeed the American ENT surgeons call it laryngopharyngeal reflux. It gives rise to sore throat, a characteristic tickle and the desire to clear one's throat; patients will be swallowing repeatedly. Often there is a funny taste in the mouth and I am sure that some of the nasal symptoms people complain of is reflux going up the back of the nose. It can even go up the Eustachian tubes and a paper in the Lancet described pepsin being found in the middle ears of a considerable number of children who were having grommets inserted.

The response of the body to this airway reflux is varied. It can just cause a cough but if inhaled can give rise to bronchitis or pneumonia. The patient is labelled as having repeated chest infections when in fact they are have recurrent aspiration with chemical injury to the airway. In about 20% of patients there is an allergic type reaction and late onset asthma or COPD is diagnosed. The patient is wheezy and there is some response to inhalers, but the cause is reflux and aspiration. These patients have rapid onset attacks precipitated by the factors listed above.

Having made a firm clinical diagnosis of reflux cough then courage and persistence is needed to treat it. Firstly, work by Don Castell in North Carolina shows that conventional acid suppression, whilst working very well for heartburn, does not cause anything like sufficient acid suppression for reflux cough. PPIs have a very short half life in the body and if one is to achieve total acid suppression one needs to give them twice daily with meals, breakfast and tea, and 300 mg of Ranitidine at night to block the histamine led nocturnal acid secretion. I would normally try this regime for two months. However a lack of response is common and DOES NOT MEAN IT'S NOT REFLUX. PPIs only block the acid and often it is the non acidic mist which is irritating the upper airway. If PPIs fail then Gaviscon Advance works quite well in some patients.

Next we would try a month's worth of Metoclopramide 10 mg tds or Domperidone 10 mg tds. Baclofen at the dose of 5mg tds can tighten up the LOS and inhibit transient opening. A further treatment strategy is the use of a low dose macrolide (erythromycin or azithromycin). Here they act as agonists of the hormone motilin improving oesophageal motility. These are our first line treatments aimed at the reflux. An alternative strategy in isolated cough is cough suppression. We have shown morphine in the form of MST 5-10 mg bd to be highly effective in about 50% of intractable coughers. This point if there is continued treatment failure I would consider surgery in the form of laproscopic fundoplication. Our experience is a favourable result in two thirds of patients. I would ask for oesophageal manometry and pH monitoring as a preliminary investigation since a completely amotile oesophagus is a contraindication. As stated above the absence of acid does not preclude a successful procedure.

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