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Contents

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FOUNDATION YEARS JOURNAL 2010

Volume 4, Issue 7

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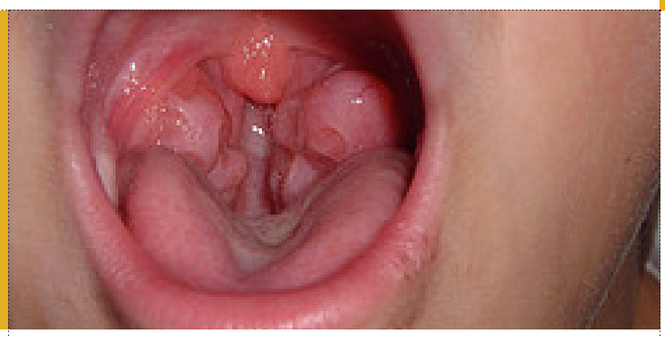
TONSILS AND ADENOIDS: SLEEP-RELATED BREATHING DISORDERS IN CHILDREN

M Buckingham, AC Leong and DA Bowdler

Tonsils and adenoids: Sleep-related Breathing Disorders in Children. Good Clinical Care.



Introduction

Sleep-related breathing disorders (SBD) in children are common, ranging from snoring, which is a relatively benign and common condition, to obstructive sleep apnoea (OSA) at the other end of the spectrum, typically characterised by oxygen desaturation, reduced oronasal air flow and paradoxical movement of the chest and abdomen (see Figure 1)[1]. In the UK, 12% of 4-5 year old children snore on a regular basis and the typical affected child who suffers from SBD is aged 2-5 years old[2]. Up to 3% of children experience episodes of intermittent complete upper airway obstruction or OSA, which is the most extreme form of SBD[3]. The purpose of this article is to highlight SBD in children to FY2 trainees as a common but serious condition which presents to general practitioners (GP), paediatricians and otolaryngologists, with a view to guiding trainees through its diagnosis and management.

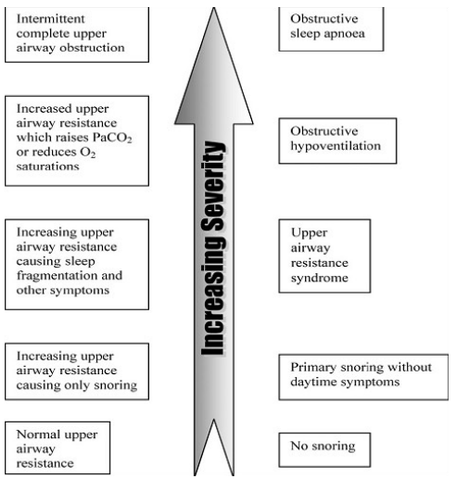


Figure 1: Continuum of upper airway resistance severity in paediatric sleep-disordered breathing[3].

OSA may have severe consequences for the child's development, such as neurocognitive impairment, behavioural problems and failure to thrive, and if left untreated, may lead to pulmonary hypertension (cor pulmonale) with symptoms of heart failure[4]. The neurodevelopmental sequelae include marked deficit in attention span, an executive function which is crucial in planning and decision making, phonological processing, visual attention and general conceptual ability. This is particularly worrying because it occurs at a crucial age for the development of reading skills[5].

Case example

Child B, an 8 year old who was under regular follow-up with the paediatricians and her GP for asthma and poor growth (9th centile), was referred to ENT due to severe nasal congestion and snoring in spite of good asthma control. Her mother was concerned about her loud snoring at night and episodes of apnoea lasting up to 10 seconds each. B was grumpy during the daytime and difficult to rouse in the morning. B also had a poor appetite and was underweight.

At the ENT clinic, she was breathing persistently and loudly through her mouth. She also had bilateral mucopurulent nasal discharge, which was chronic. Examination revealed grade 4 non-inflamed tonsils while rhinoscopy showed hypertrophy of the inferior turbinates. A course of antihistamines, antibiotics and intranasal steroids failed to alleviate her symptoms. One month later at the follow-up appointment, her mother expressed great concern about her worsening snoring, apnoeic episodes and deteriorating school performance. She was diagnosed with sleep-disordered breathing with an element of OSA. She subsequently underwent adenotonsillectomy and was discharged home the following day with paracetamol and ibuprofen for seven days.

When reviewed three months post-operatively, B's upper airway obstructive symptoms of snoring and apnoeic spells had completely resolved. She also no longer suffered chronic purulent rhinorrhoea. Her appetite had improved and she had gained 3kg in weight. Her mother was also pleased at B's improved behaviour, remarking that B seemed "like a new person".

Causes of SDB

The most common cause of SBD in children is enlarged tonsils and adenoids. This may occur against a background of subtle abnormalities of upper airway control of muscle tone and central neural drive[6]. Most children with OSA do not have significant apnoea while awake but suffer mixed central and obstructive apnoea when asleep. Normally, during sleep, chemoreceptors sense changing levels of CO_2 and O_2, leading to a compensatory increase in respiratory muscle activity. In OSA, there is a defect in the central ventilatory drive response to hypercarbia, hypoxaemia and reduced airway muscle tone, possibly due to habituation or mechanical damage. This more extreme form of SBD is more prevalent in male, black or Hispanic children and those with a history of prematurity[7].

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Other causes for SBD may include mid-face hypoplasia, e.g. craniosynostosis syndromes of Crouzon and Apert, mandibular hypoplasia, e.g. Pierre-Robin syndrome, micrognathia and macroglossia, e.g. Down's syndrome[6].

The adenoids and tonsils are part of the lymphoid tissues that circle the pharynx, which are known as Waldeyer's ring (Figure 2). It grows throughout childhood until the age of eleven years and then decreases in size. It plays an important role in the child's immunity, particularly in the generation of B cells[6].

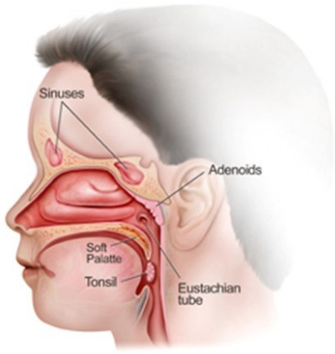

Figure 2: Anatomy of the adenoids and tonsils.

Controversial issues

Currently, there are no officially endorsed diagnostic criteria for childhood sleep-disordered breathing, unlike adult sleep-disordered breathing. In view of this, the American Academy of Pediatrics and the Royal College of Surgeons have recently issued diagnostic and management recommendations regarding surgical treatment for SBD in children[3,4] (Appendix 1).

The main issues raised concern:

1) The lack of a definitive tool for diagnosis.
2) The lack of unambiguous tertiary referral guidelines for general practitioners and ENT specialists at district general hospitals.
3) The ability to detect children at risk of post-operative complications

Presentation

Referral to the ENT team in suspected cases of sleep-disordered breathing is usually initiated by the GP. At present, there are no clear guidelines for referral to a specialist in the case of suspected SBD. Child B displayed symptoms of snoring with apnoeic episodes at night, tiredness in the morning and grumpiness during the day. She had a poor appetite and had difficulty putting on weight. Her difficulties at school intensified her mother's concerns, which prompted further investigation and a specialist referral.

Differential diagnoses to exclude:

- Laryngomalacia (which presents with stridor rather than stertor, typically exacerbated by crying or feeding and tends to present in infants or very young children; stridor is a higher pitched sound).
- Parasomnias (which include sleepwalking, sleep terrors and REM sleep; these can be diagnosed on polysomnography).
- Epileptic fits (which may be diagnosed on EEG).

Clinical Assessment and Investigations

A good history of the child's sleep and daytime symptoms is crucial in making a diagnosis. It is important to establish how much the carer is aware of the child's sleeping problems (Table 1). Other symptoms suggestive of significant childhood SBD include snoring, disturbed sleep and enuresis. Neurocognitive impairment, behavioural problems and failure to thrive may be associated with developmental delay, attention disorders and poor school performance. Physical examination while awake is important to evaluate airway structure and exacerbating factors, and is summarised in Table 1. Findings are often normal, even in severe OSA syndrome.

History	
Sleep history	1. Onset 2. Duration 3. Awakening difficulties 4. Enuresis 5. Movements 6. Discontinuity 7. Environment
Snoring	1. Onset 2. Frequency 3. Proportion of the night spent snoring 4. Loudness and pitch 5. Position when snoring 6. Increased respiratory effort or not when snoring
Daytime	1. Hyperactivity 2. School concerns 3. Mouth-breathing
Drug history	Any medication that may explain any of the above symptoms
Past medical or surgical history	1. Previous airway surgery 2. Previous intubation 3. Thyroid or metabolic problems
Family history	1. Snoring 2. Obesity 3. OSA
Examination	
General	1. Height 2. Weight 3. Growth curve 4. Attention span 5. Hyperactivity
ENT	1. Oropharynx 2. Tonsil and adenoid enlargement 3. Nasal patency 4. Signs of infection 5. Neck masses
Cardiovascular	1. Cor pulmonale (loud S2, gallop rhythm) 2. Signs of heart failure
Other	1. Craniofacial features 2. Neuromuscular weakness 3. Cerebral palsy

Table 1: Proposed history and examination of the child with suspected SBD.

TONSILS AND ADENOIDS: SLEEP-RELATED BREATHING DISORDERS IN CHILDREN

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It remains controversial as to which investigations are most helpful in reaching a clinical diagnosis. In the United States, polysomnography is the gold standard, consisting of simultaneous recordings of multiple physiological variables during sleep. It is an expensive test done in very few specialised centres in the UK. In younger children, it is even more difficult to reach a clear diagnosis using polysomnography alone because of the lack of universal criteria to define SBD and the difference between age groups.

Overnight pulse oximetry is commonly used to measure the severity of the SBD. Saturations of <80% are associated with OSA syndrome, but negative pulse oximetry does not exclude the diagnosis of OSA[8]. Sleep nasendoscopy, snoring audiotapes/ videotapes and quality of life questionnaires may also assist the clinician in making a diagnosis[7].

Treatment

Adenoidectomy and tonsillectomy are the most common procedures used to treat SBD and are often done simultaneously. Possible complications are summarised in Table 2. Other surgical treatment may include maxillar/ mandibular surgery and rarely, tracheostomy[9]. Medical treatments include corticosteroids such as intranasal fluticasone as adjunct therapy and continuous positive airway pressure (CPAP) for persistent cases[10,11].

- Reactionary and secondary bleeding (0-3.5% and 0-30% respectively) [7]
- Infection
- Postoperative respiratory compromise (16-27%) [8]
- Rarer complications: dental injury, nasopharyngeal scarring and stenosis, lingual nerve palsy, Eustachian tube injury.

Table 2: Complications of adenotonsillectomy

The most worrying complication after adenotonsillectomy is post-operative respiratory compromise, which may occur due to upper airway oedema, pooling of oropharyngeal secretions and pulmonary oedema. The incidence of post-operative respiratory complications for adenotonsillectomy in children with OSA is as high as 16-27%, compared to 0-1.3% in the general paediatric population[12,13]. The main difficulty lies in identifying the subset of patients with OSA who have a potentially high risk of post-operative respiratory complications. As a result, the Royal College of Surgeons recently recommended guidelines to identify these patients who would be more appropriate candidates for adenotonsillectomy at a tertiary referral centre with access to a paediatric intensive care unit with appropriate staff and facilities (see Table 3).

- Age < 2
- Weight < 15 kg
- Failure to thrive (weight < 5th centile)
- Obesity (BMI > 2.5 standard deviations)
- Severe cerebral palsy
- Hypotonia or neuromuscular disorders
- Significant craniofacial anomalies
- Mucopolysaccharidosis and syndromes associated with difficult airway
- Significant co-morbidity such as congenital heart disease, chronic lung disease
- ECG or echocardiographic abnormality
- Severe OSA on polysomniography

Table 3: Children at risk of postoperative respiratory complications after adenotonsillectomy[3].

Conclusion

Sleep-disordered breathing in children is a common condition with potentially severe consequences for the child's development. It is important that general practitioners and paediatricians are aware of its symptoms and signs to maintain a high index of suspicion. The diagnosis is mainly clinical and should involve multidisciplinary input from the child's family, their school teacher, GP, speech therapist and paediatrician. Polysomnography is usually reserved for children with underlying structural and neuromotor abnormalities and syndromes, or children with OSA persisting after adenotonsillectomy. Children with severe OSA or significant co-morbidities may require surgical management at a tertiary referral centre with access to intensive care facilities.

Multiple Choice Questions

True/False?

1. Sleep-related breathing disorders typically present with stridor at night.
2. Children who have undergone adenotonsillectomy have a reduced immune response to infection.
3. Adenotonsillectomy is only used in the treatment of sleep-related breathing disorders.
4. A positive polysomnograph is required for the diagnosis of obstructive sleep apnoea.
5. When a child is suspected of suffering from a sleep-related breathing disorder it is important to refer promptly to an ENT specialist.

1. False

Stridor is described as a high pitch sound that may be inspiratory or biphasic. It is typically due to an obstruction of the larynx and should always be treated as a potential clinical emergency. Stertor describes a lower pitch sound that is heard in SBD and obstruction of the oropharynx and may commonly be described as "snoring".

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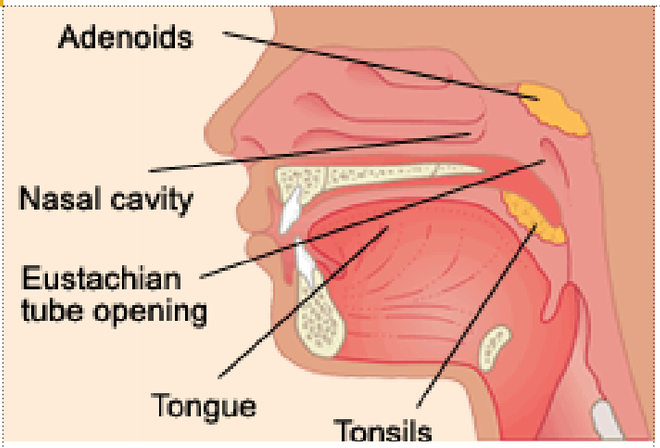

2. False

Adenoids or pharyngeal tonsils, palatine and lingual tonsils form part of Waldeyer's tonsillar ring. They have similar histology and contain B and T-cell lymphocytes. They are involved in inducing a secretory immune response and regulating immunoglobulin production. This system is independent from the lymphatic drainage and allows direct exposure to antigens that enter via the oral cavity. These tissues are most active during the ages of 4 to 10 and involute after puberty. Recent studies have showed that immunoglobulins, complements and lymphocyte count were not compromised in children a few weeks post-adenotonsillectomy[14, 15].

3. False

Tonsillectomy is commonly used as a surgical treatment for recurrent episodes of acute tonsillitis and is often coupled with adenoidectomy in children. The adenoids are easily seen during the procedure and may be removed if large. Adenoidectomy is often performed in children suffering from recurrent secretory otitis media as the adenoids may be a source of infection for the middle ear.

4. False

Polysomnography remains the gold standard in investigating SBD in adults in the United States. In children, there are no definitive diagnostic criteria in different age groups for the OSA spectrum. In the UK, access to sleep study units is also difficult and expensive. Diagnosis is mainly based on a good history and clinical findings (see Table 1).

5. True

Once a diagnosis of SBD, in particular OSA, is suspected, it is important to refer the child to an ENT specialist promptly. These disorders typically affect the child at a crucial time in their neurological development and delay in treatment may lead to severe consequences.

Single best answers

1. You are a general practitioner seeing a 4-year-old girl who was brought in to see you by her mother. The mother is concerned about the fact that her daughter is hyperactive at school and very irritable in the morning.

Do you:

a) Agree with the mother that this probably just a phase that her child is going through and that as long as she is not ill no further consultation is necessary.

b) Refer the child to a psychiatrist who will assess her with regards to treating her for attention-deficit hyperactivity disorder.

c) Refer the child to an ENT specialist in order to book her on the next available list for an adenotonsillectomy.

d) Book the child for some nocturnal sleep studies and routine blood tests.

e) Ask the mother for a further history of her daughters symptoms and in particular if she or someone else in the family have noticed any snoring symptoms or any concerns that they might have with her sleeping habits.

2. You are a FY2 doctor working on a night shift in an ENT service when you are asked to come down to paediatric A&E to review a 5 year old child who has had an adenotonsillectomy 5 days ago and is bleeding form his throat.

Do you:

a) Tell the nurse in charge to admit the child overnight and that you will see the child on the morning ward round.

b) Tell the nurse in charge to send the child home and re-attend in the morning when your full team will be present.

c) Go to see the child in A&E by which time he is no longer bleeding. His observations are normal and he is apyrexial so you send him home.

d) Go to see the child in A&E by which time he is no longer bleeding. You assess the child to make sure that he is stable and you ensure that routine bloods have been taken and that the child is kept nil by mouth. You re-assure the mother that this is a recognised complication from the operation and that he will need to stay in overnight for observation.

e) Get IV access and prepare the child for theatre. You call your registrar and the anaesthetist urgently to inform them of the situation.

Answer: 1. e).

The presentation of this child has raised some concerns and in particular, one would want to exclude a sleep-related breathing disorder. A good history from the mother and whoever might be with the child at night time but also during the day is vital to establishing a possible diagnosis of SBD. Table 1 lists signs and symptoms that may help the GP to confirm any suspicion of such a diagnosis. A referral to an ENT specialist would then be appropriate before booking any sleep studies.

Answer: 2. d).

Post-tonsillectomy secondary bleed is a well known complication of the operation and must be treated seriously. It is very important to keep the child in overnight for further observation and keep him ready for possible surgery. He must be kept nil-by-mouth and started on IV replacement fluids after IV access and some basic blood tests have been sent to the lab. Most post-op bleeds are due to infection so antibiotic cover should be initiated according to local hospital protocol. Once the child has been admitted to a children's surgical ward the registrar should be informed of his admission and treatment.

TONSILS AND ADENOIDS: SLEEP-RELATED BREATHING DISORDERS IN CHILDREN

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1. **All children should be screened for snoring.**
2. **Complex high-risk patients should be referred to a specialist.**
3. **Patients with cardiorespiratory failure cannot await elective evaluation.**
4. **Diagnostic evaluation is useful in discriminating between primary snoring and OSAS, the gold standard being polysomnography.**
5. **Adenotonsillectomy is the first line of treatment for most children, and continuous positive airway pressure is an option for those who are not candidates for surgery or do not respond to surgery.**
6. **High-risk patients should be monitored as inpatients postoperatively.**
7. **Patients should be reevaluated postoperatively to determine whether additional treatment is required.**
8. **Most children can be managed safely closer to home at a DGH. Complex high-risk patients should be referred to a tertiary centre with PICU facilities.**

Appendix 1 - Royal College of Surgeons recommendations

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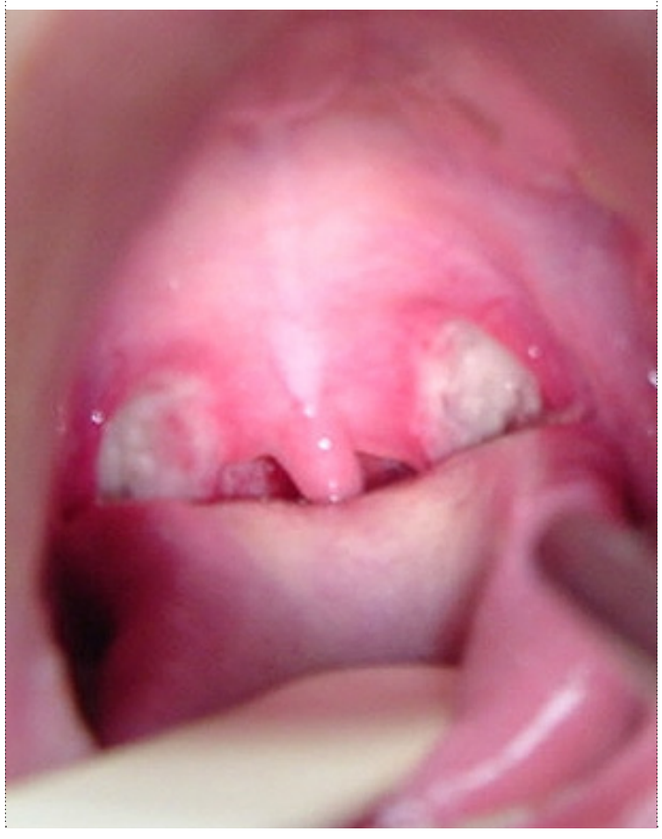
Authors and Correspondence

M Buckingham
ENT FY2 doctor at University Hospital Lewisham
Dept of Otolaryngology

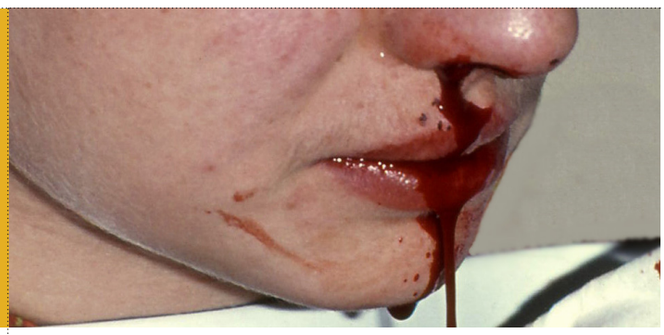
AC Leong
ENT SpR at University Hospital Lewisham Dept of Otolaryngology

DA Bowdler
Consultant ENT Surgeon Dept of Otolaryngology
University Hospital Lewisham



EPISTAXIS

James Thambyrajah, Annabelle C. Leong and David Bowdler



Epistaxis.
Good Clinical Care.

Introduction

Epistaxis is the most common ENT emergency, affecting 60% of the population in their lifetime, with 6% presenting to hospital[1]. It often presents to other specialties, including A&E, medical and paediatric disciplines. The objective of the article is to guide foundation trainees in the acute assessment and management of epistaxis.

Case Example

A 77-year old retired porter presented to A&E with profuse left-sided epistaxis. His past medical history included hypertension and a cerebrovascular accident four years ago. He was on aspirin, bisoprolol and candesartan. Full blood count and clotting profile was normal. Upon examination of the nose, the bleeding vessel could not be visualised. As the epistaxis failed to settle with simple pressure applied to the nose, a Rapid Rhino® nasal pack was inserted into the left nasal passage, which managed to control the bleeding. He was then admitted as an inpatient on the ENT ward where the pack remained inserted for 24 hours without further bleeding. The patient's aspirin dose was omitted for that day. The pack was removed the following day and repeat examination revealed a small blood clot in the anterior portion of the nose. The underlying vessel was cauterised with silver nitrate and the patient discharged home with a two-week course of Naseptin® ointment to be applied twice a day to both nasal passages.

The following week, the patient was re-admitted with left-sided epistaxis which could not be controlled with Rapid Rhino® or Merocel® packing. A Foley catheter was thus inserted into the left nasal passage and its balloon inflated to provide a tamponade effect to the back of the nose. A Rapid Rhino® was then inserted anterior to the Foley packing and the patient was given adequate analgesia. In spite of these measures, the patient continued to bleed. After discussion with the ENT and anaesthetic consultants, he was transferred to theatre where he underwent an examination of his nose under general anaesthesia to identify the bleeding point. The bleeding was found to be originating from the sphenopalatine artery, which was ligated with the use of bipolar diathermy. The patient was monitored overnight on the ENT ward and was discharged home the following day. At outpatient follow-up four weeks later, the patient no longer suffered from epistaxis.

Anatomy

Epistaxis is classified on the basis of the primary bleeding site as anterior or posterior. More than 90% of epistaxis originates from Kiesselbach's vascular plexus at Little's area, which is found at the anterior part of the nasal septum. Little's area is a vascular anastomosis of branches of the internal and external carotid arteries. The internal carotid artery supplies the nose via its anterior and posterior ethmoid branches, while the external carotid artery contributes with its sphenopalatine and facial branches.

In 10% of epistaxis cases, bleeding occurs from a more posterior source from the lateral nasal wall, septum or nasal floor[2]. Branches of the sphenopalatine artery via the internal maxillary branch of the external carotid artery are thought to be responsible for most cases of "posterior epistaxis", with some contribution from the branches of the anterior and posterior ethmoid arteries3. (See Figure 1) .

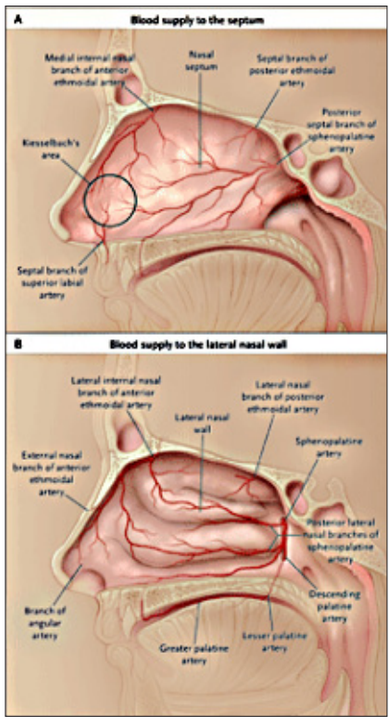


Figure 1: Blood Supply to the Nasal Septum and Lateral Nasal Wall.

EPISTAXIS

James Thambyrajah, Annabelle C. Leong and David Bowdler

Aetiology Of Epistaxis

Most cases of epistaxis do not have an easily identifiable cause and are termed idiopathic. Recognised causes of epistaxis are summarised in Table 1. Hypertension as a direct causative factor in epistaxis remains unproven, although patients with epistaxis commonly present with an elevated blood pressure and epistaxis is more commonly seen in hypertensive patients.

Local Causes	Systemic Causes
Local trauma – nose picking, blunt trauma	Drug-related – anticoagulant drugs (warfarin, heparin), antiplatelet drugs (aspirin, clopidogrel, NSAIDs), topical corticosteroids
Local inflammation or infection – vestibulitis, rhinitis	Vascular abnormalities – Hereditary haemorrhagic telangiectasia, arteriovenous malformations
Foreign bodies	Inherited coagulopathies – haemophilia, von Willebrand's disease)
Neoplasm (benign/malignant)	Acquired coagulopathies – liver disease, renal failure, chronic alcohol intake, leukaemia
Iatrogenic – nasogastric tube insertion, nasotracheal intubation	
Septal perforation	

Table 1: Causes Of Epistaxis.

Acute Management

Resuscitation of the patient with epistaxis should always take precedence over obtaining a lengthy medical history (Figure 2).

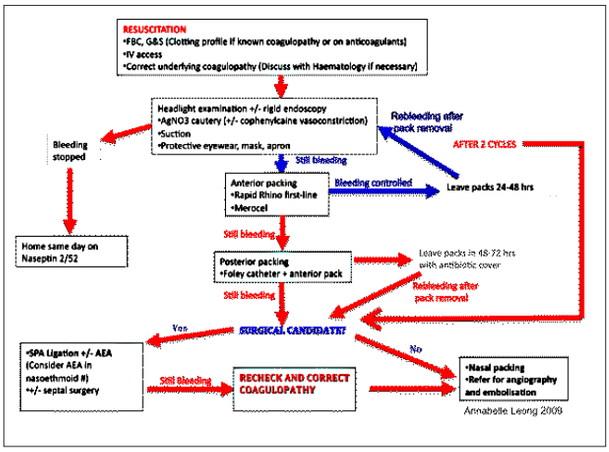


Figure 2: Suggested algorithm for the acute management of primary or idiopathic epistaxis in adults (AEA = Anterior Ethmoid Artery; SPA = Sphenopalatine Artery).

1. Assess:

a. Airway b. Breathing c. Circulation

2. Send blood samples off for a full blood count, clotting and group and save.

3. Commence fluid resuscitation if patient is displaying signs of hypovolaemia e.g. pallor, sweatiness, tachycardia (bradycardia if severe), hypotension.

History

When the patient has been stabilised, enquire about:

- Duration, severity of the haemorrhage and side of initial bleeding.
- Previous epistaxis.
- Relevant past medical history, e.g. hypertension, hepatic or other systemic disease.
- Family history of recurrent epistaxis, easy bruising, or prolonged bleeding after minor surgical procedures which may suggest an underlying inherited coagulopathy, e.g. von Willebrand's disease.
- Use of medications e.g. antiplatelet drugs such as aspirin and clopidogrel, non-steroidal anti-inflammatory drugs (NSAIDs), warfarin, heparin and dipyridamole should be documented, as they not only predispose to epistaxis but may make treatment more difficult[4].

Physical Examination

1. Ensure universal health and safety precautions before approaching the patient:

a. Wash your hands.
b. Wear gloves.
c. Wear apron.
d. Wear facemask.
e. Put on protective eyewear.

2. With the patient seated, use a good headlight to examine the nose for the bleeding source, with the help of a nasal speculum inserted into the nasal passage and suction. Enlist the help of a nurse if available. Rigid nasoendoscopy may be used at this point if required, to examine the nose more closely, but is not essential.

3. Cautery with silver nitrate sticks is attempted if a bleeding point is visualised. It is helpful to cauterise around the bleeding vessel first before going on to cauterise the actual bleeding point itself.

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Patients On Anticoagulation Therapy

• Those on antiplatelet therapy should stop aspirin or clopidogrel without the need for routine platelet function tests. In very severe epistaxis cases resistant to control with conservative and surgical measures, platelet transfusions may be required[4].

• The management of epistaxis patients on warfarin depends on 2 factors:
i) the extent of over-anticoagulation and
ii) the therapeutic indication for taking warfarin.

• Those on warfarin with INR in the therapeutic range may continue their normal dose warfarin.

• Patients who are warfarinised and considered "high risk" for future thromboembolic events, e.g. mechanical heart valves and recurrent deep vein thromboses or pulmonary emboli, need to be managed to maintain their INR in the therapeutic range. If their INR is <4, warfarin may need to be stopped with close monitoring of the INR at the lower end of the therapeutic range. If the INR is 4-8, then warfarin should be stopped and fresh frozen plasma (FFP) may be necessary for reversal of anticoagulation. If INR >8, then Beriplex (plasma-derived concentrate of clotting factors II, VII, IX and X or prothrombin complex) may be required[4].

• "Low risk" warfarinised patients may stop their warfarin and do not require maintaining anticoagulation in the acute stage.

Anterior Epistaxis

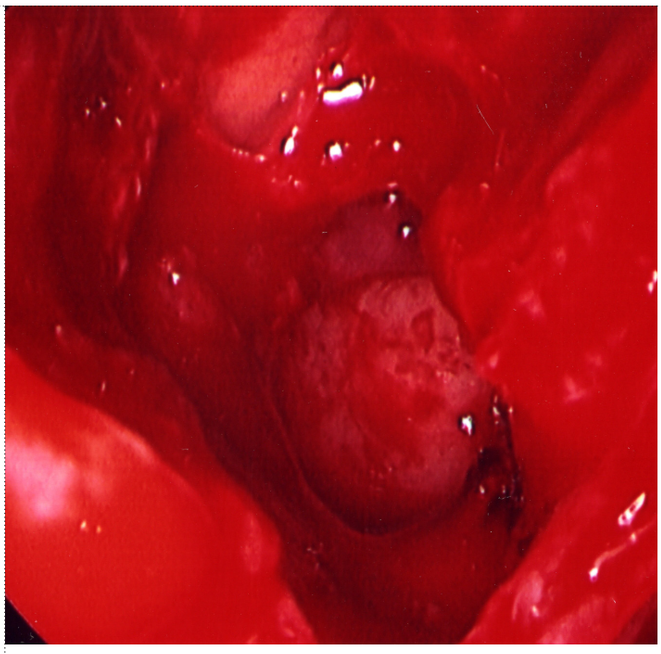
Packing of the nose may be undertaken with:

• Merocel® – Ensure the merocel is lubricated and insert it along the floor of the nasal cavity until it is all the way in. Warn the patient that this may be uncomfortable.

• Rapid Rhino® – Immerse in sterile water for 10 seconds. Insert along the floor of the nasal cavity, parallel to the hard palate. Using a 20ml syringe, inflate the balloon with air (maximum volume 10ml). Secure the balloon to the patient's cheek with tape.

• Ribbon gauze impregnated with bismuth iodoform paraffin paste (BIPP). BIPP has antiseptic properties and is commonly used in the UK as a packing material for the nose5. It is applied using a Tilley's nasal forceps in tight layers along the floor of the nasal cavity in decreasing lengths until the entire nasal cavity is filled. Local anaesthetic such as co-phenylcaine (combination of phenylephrine and noradrenaline) may be sprayed topically prior to packing the nose.

Nasal packs remain inserted for 24–48 hours and are effective at controlling most cases of epistaxis.



Posterior Epistaxis

• Posterior bleeding is difficult to control with anterior packing alone. A posterior source is suggested by failure to visualize an anterior source, haemorrhage from both nares and by visualization of blood draining in the posterior pharynx.

• The most common form of posterior packing is with a Foley catheter, usually size 12 or 14 male – Pass it along the floor of the nasal cavity and follow it down until it is seen just behind the soft palate in the oropharynx. Inflate the balloon with 5–8ml of water and then pull on the catheter until the balloon is felt to exert a tamponade effect on the back of the nose (posterior choanae).

• The catheter is clipped using a plastic 'umbilical' clip. Ensure that sufficient gauze padding is placed between the clip and nose to avoid pressure necrosis of the nasal alar skin.

• Anterior packing may then be inserted in front of the Foley catheter.

Posterior packing usually remains in situ for at least 48 hours. Antibiotic cover for nasal packing may be considered but the timing is controversial, ranging anywhere from immediately after pack insertion to after 24 or 48 hours of packing. If bleeding persists or recurs in spite of this, then surgical management should be considered.

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Surgical Options

When the above conservative measures fail to control the bleeding, surgical intervention is indicated.

- Correction of septal deviation by performing a septoplasty may not only facilitate access for packing, but may itself lead to disruption of the blood supply to the septum.
- Surgical ligation of the responsible artery may also be undertaken. If the source of bleeding is thought to originate posteriorly, then endoscopic ligation of the sphenopalatine artery is performed, either by ligation with surgical clips or electrocautery with specially-designed bipolar diathermy forceps. Failure to use diathermy to ligate the sphenopalatine artery has been associated with post-operative recurrence of bleeding, possibly explained by the variable anatomy of the sphenopalatine arterial branches which may not all have been clipped6. Anterior epistaxis recalcitrant to nasal packing may warrant ligation of the anterior ethmoidal artery, usually via an external approach through a small incision at the medial canthus (the area between the eye and nasal bridge). The artery is ligated with a metal clip or with bipolar diathermy. In cases of intractable epistaxis with no defined source of bleeding, both the anterior ethmoid and sphenopalatine arteries may be ligated[3].

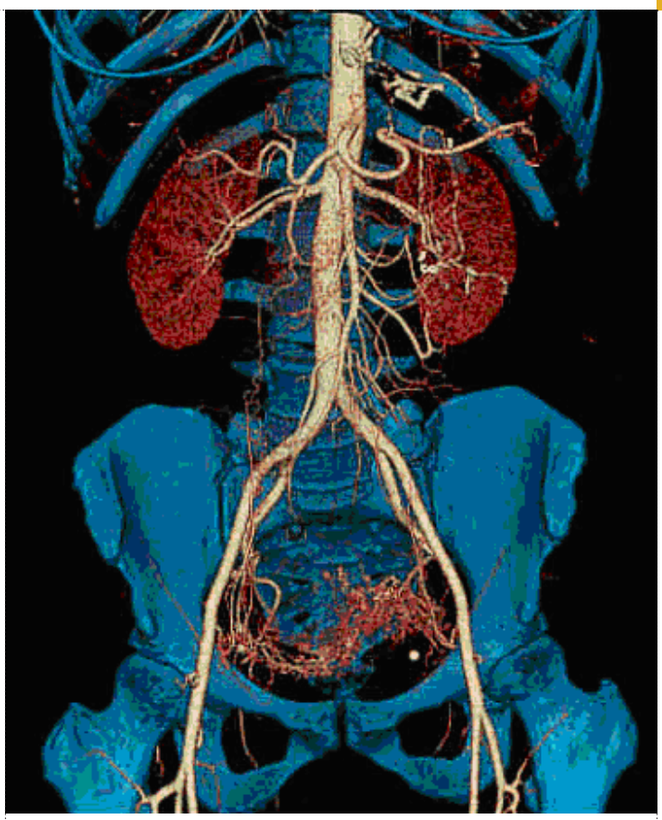
Arterial Embolisation

Arterial embolisation is indicated when the patient is not fit for general anaesthesia or when surgical ligation has failed to control the bleeding. Interventional radiologists may perform embolisation of the ipsilateral internal maxillary and facial arteries under local anaesthesia through a transfemoral approach. The patient must be actively bleeding in order for the bleeding source to be identified clearly. Embolisation has similar success rates to surgical ligation but it is associated with more serious complications, such as:

- Stroke.
- Facial Paralysis.
- Blindness.
- Skin necrosis.

Summary

Epistaxis is a common acute condition which is usually idiopathic. It is important to resuscitate the patient first and foremost before proceeding to localise the bleeding point. Once identified, cautery of the responsible vessel may be attempted. If the bleeding point is not visualised, nasal packing is undertaken which will be effective at controlling epistaxis in most cases. If the above measures fail, then the nose should be examined under general anaesthesia to ligate the bleeding vessel and/or pack the nose appropriately. Arterial embolisation under radiological guidance may be considered in patients who are not fit to undergo a general anaesthetic. Most importantly, correction of any underlying coagulopathy underpins the management of epistaxis and discussion with haematology colleagues may be necessary.



Multiple Choice Questions (Select One Correct Answer)

1. Regarding the anatomy of the nose:

a) 50 % of nose bleeds originate in Kiesselbach's area.
b) Little's Area is found in the posterior part of the septum.
c) Little's area is a vascular anastomosis of the branches of the internal and external carotid arteries.
d) The external carotid artery supplies the nose via its anterior and posterior ethmoidal arteries.
e) The internal carotid artery supplies the nose via the facial and sphenopalatine branches.

2. Regarding acute management of epistaxis:

a) Patient with an acute episode of epistaxis should be packed with a Rapid Rhino® straight away.
b) Silver nitrate cautery sticks are used to cauterise a bleeding point.
c) Merocel® packs should be not lubricated first before placement.
d) Rapid Rhino® packs should be left in for at least 4 days.
e) Posterior packing of the nose is never done using a Foley catheter.

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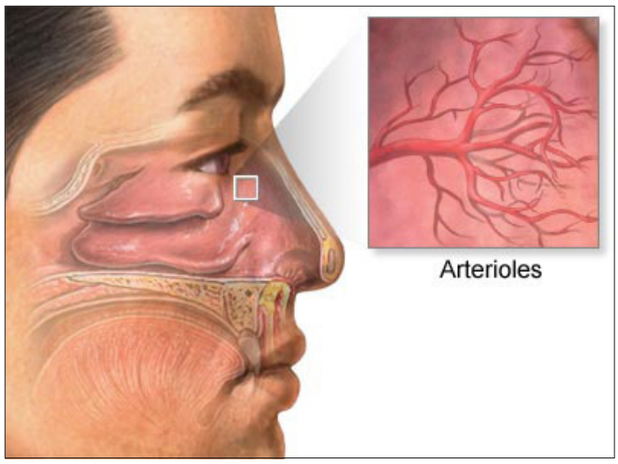

Answers

1. c) – 90% of epistaxis occurs in Kiesselbach's area. Little's area is located in the anterior part of the septum and is an anastomosis of the branches of the internal and external carotid arteries. The internal carotid artery supplies the nose via the anterior and posterior ethmoid arteries while the external carotid artery supplies the nose via the facial and sphenopalatine branches.

2. b) – Patients who present with an acute episode of epistaxis should be assessed to see if they require resuscitation before proceeding to take a clinical history and examine the nose for the bleeding source, which may be cauterised with silver nitrate as first line therapy. If bleeding persists, anterior nasal packing is performed, using Merocel® or Rapid Rhino® packs. Merocels® are usually lubricated with water or saline before insertion, while Rapid Rhino® packs are soaked in water for 30s before placing along the floor of the nasal cavity. Rapid Rhinos® are usually not left inserted for more than 2 days due to the risk of infection. The timing of antibiotic cover is controversial, ranging anywhere from immediately after pack insertion to after 24 or 48 hours of nasal packing. Posterior packing is commonly done using a Foley catheter. The balloon is inflated with water and the catheter is yanked away from the patient so that the balloon tamponades the back of the nose.

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Authors

James Thambyrajah (FY2)
Dept of Otolaryngology
University Hospital Lewisham
Lewisham High Street
London SE13 6LH

Annabelle C. Leong (SpR)
Dept of Otolaryngology
University Hospital Lewisham
Lewisham High Street
London SE13 6LH

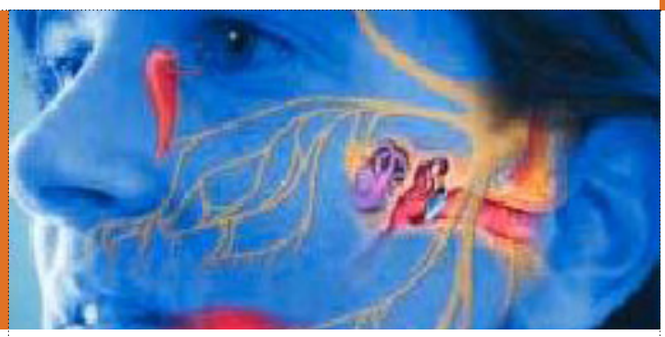
David Bowdler (Consultant)
Dept of Otolaryngology
University Hospital Lewisham
Lewisham High Street
London SE13 6LH

Correspondence

James Thambyrajah (FY2)
Dept of Otolaryngology
University Hospital Lewisham
Lewisham High Street
London SE13 6LH
email: jthambyrajah@hotmail.com

FACIAL PALSY

E. Naomi Smith and Ian Bottrill



Facial Palsy.
Case Based Discussion.

Abstract

This case-based discussion focusses on a patient who presented with a left-sided facial palsy. The assessment, differential diagnoses and treatment of the patient's facial palsy will be discussed, together with an overview of the relevant anatomy.

Case History

A previously fit and well 51-year-old man (Mr. FC) presented to the Emergency department with a unilateral facial weakness (Figure 1), which was present on waking. His forehead muscles were involved. He had no associated hearing or balance symptoms. There were no other neurological symptoms. The patient expressed concern that he has had a stroke.

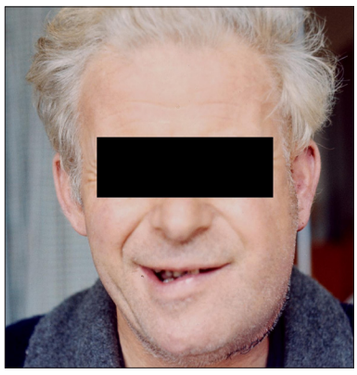


Figure 1

- **What are the possible causes of Mr. FC's facial weakness?**
- **What would you do next?**
- **A detailed History and Examination are Required**

Focussed History

• Was the onset of the weakness rapid or gradual? This is important, as a slowly progressive weakness is most suggestive of a tumour, while a sudden onset facial palsy, developing over hours, is typical of a Bell's palsy[1].

• Is the weakness associated with any unilateral ear symptoms, such as otalgia, otorrhoea or unilateral hearing loss? These symptoms may indicate active middle ear disease, such as acute otitis media, cholesteatoma or malignant otitis externa[2].

• Is there an associated symptom of severe stabbing facial pain? This would raise suspicion of Ramsay-Hunt syndrome[3]. Vesicles in the ear canal, on the eardrum or pinna may or may not be present depending on the time of presentation.

• Is there any history of trauma or surgery? Blunt trauma can cause temporal bone fracture, resulting in traumatic facial paralysis[4]. Iatrogenic trauma to the facial nerve from surgery, in particular, surgery of the middle ear or parotid gland, must also be considered[4]. Facial palsy may occur as a delayed phenomenon following middle ear surgery and present a few days after the operation.

• Is there altered taste and/or altered secretion of the lacrimal or salivary glands? These symptoms may or may not accompany a Bell's palsy but are important in the management of the patient.

• Has the patient had previous episodes of facial palsy? Recurrence of facial palsy has been noted to occur[13]. Is there a history of diabetes? Is the patient pregnant? Bell's palsy is more common in these populations[1].

• If hearing loss is present, is it associated with tinnitus or vestibular disturbance? This may indicate the presence of an acoustic neuroma[5]. However, an acoustic neuroma is a rare tumour and virtually never presents with facial weakness.

Focussed Examination

• Is the facial weakness due to an upper or lower motor neuron lesion? Sparing of the upper facial muscles imply an upper motor neuron lesion[3]. Forehead involvement is highly suggestive of a lower motor neuron lesion[2].

• Ask the patient to raise their eyebrows; are wrinkles present on one side, or both? If the lesion is above the level of the brainstem nucleus, bilateral cortical representation of the forehead muscles allows their function to remain relatively preserved[6]. Weakness of the lower facial muscles will be present on the contralateral side of the lesion.

• In contrast, with lower motor neuron lesions, all muscles of facial expression will be affected on the same side as the lesion[6]. This will include the loss of wrinkling and decreased muscle strength in the forehead.

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• A thorough examination if the ear is essential. Middle ear disease, such as otitis media, cholesteatoma or herpetic infection, must be excluded[3] (Figure 2 – An Attic Cholesteatoma). If any auditory symptoms are present, further assessment for conductive and sensory-neural deafness must be made[2].

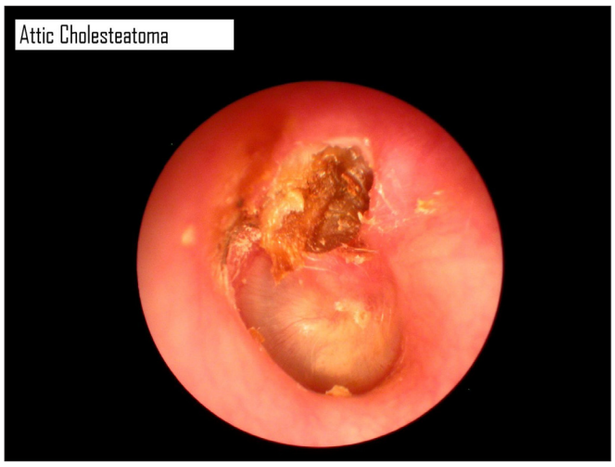

Figure 2: An Attic Cholesteatoma.

• Palpate the parotid gland. The presence of a parotid mass could indicate an invasive tumour, such as an adenoid cystic carcinoma. It is also important to look intraorally when assessing the parotid gland to assess if a deep lobe tumour is present[2, 3].

• The degree of facial weakness must be assessed and documented[3]. The "House Brackmann" grading system has been devised for this purpose. It is worth noting that this scale is subjective and observer dependent. The key feature is assessment of eye closure. If eye closure is incomplete, it is vital that good eye care is given. The scale is also useful in monitoring improvement[2].

• The eye is at risk of abrasion in facial palsy due to poor eye closure and compromised lacrimal gland function[7]. As well as assessment of eye closure, the eye itself must be assessed for signs of corneal ulceration, infection or exposure keratitis[2, 3, 7]. Eye assessment may involve obtaining an ophthalmology opinion.

Discussion

Following comprehensive assessment, Mr. FC was diagnosed with Bell's palsy. Bell's palsy is the most common type of lower motor neuron facial paralysis[4]. It is strictly a diagnosis of exclusion[2, 4]. Mr. FC had no "red flag" (worrying) symptoms.

Symptoms & Signs to Raise Suspicion of Malignancy	Additional Red Flag Symptoms and Signs
Parotid Mass (Note – the most common missed diagnosis is parotid malignancy)	Herpetic Vesicles and Facial Pain (Ramsay-Hunt syndrome)
Gradual onset facial weakness	Unilateral Ear Pain and Discharge
Facial weakness which does not resolve after several weeks	Unilateral Hearing loss Tinnitus and Vertigo

Table 1: Red Flag Symptoms in Facial Palsy.

Pathophysiology of Bell's Palsy

There is increasing evidence that Bell's palsy is caused by viral infection[2]. It has been hypothesised that the facial paralysis seen in Bell's palsy is due to swelling and thus compression of the facial nerve in an inflammatory response to viral infection[1]. The suspected virus is the herpes simplex virus type 1 [2]. To explore this further, a revision of the facial functions and anatomy is provided below:

Functions of the Facial Nerve – the facial nerve contains:

• Motor fibres, which innervate the facial muscles, the digastric muscle and the stapedius muscle of the middle ear.

• Sensory fibres for taste in the anterior two-thirds of the tongue, as well as somatic afferents from the external auditory canal and pinna.

• Parasympathetic fibres to the lacrimal, sub-mandibular and sublingual salivary glands.

Anatomy of the Facial Nerve

• The facial nerve consists of two roots, one motor root and one mixed sensory and a parasympathetic root, the nervus intermedius.

• The nerve arises at the pontomedullary junction and runs laterally to enter the internal acoustic meatus. It then travels through the internal auditory canal of the temporal bone. This canal consists of three consecutive segments, the narrowest of which is the labyrinthine segment. The nerve is particularly vulnerable to compression at this site.

• The greater superficial petrosal nerve innervates the lacrimal and palatine glands. The facial nerve then supplies a branch to the stapedius muscle. More distally, the chorda tympani branches to supply fibres to the sublingual and sub-mandibular glands, as well as afferent fibres for taste.

• The facial nerve finally passes through the stylomastoid foramen and travels through the parotid gland, where the nerve branches to supply the muscles of facial expression.

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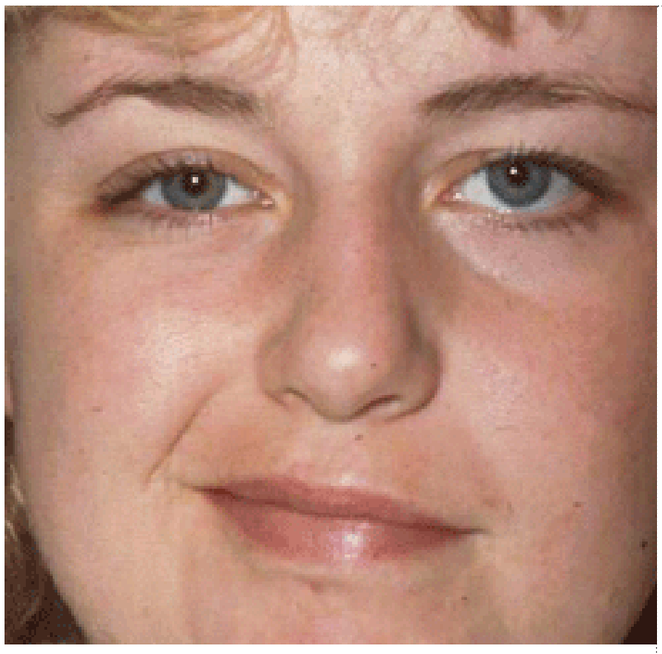

Management of Bell's palsy

Treatment of Bell's palsy remains controversial. It can be divided into the following categories:

• Medical Treatment – The use of oral corticosteroids and antiviral drugs has been fiercely debated.

Cochrane review summarises four trials, which show no significant benefit of steroid treatment over placebo[2]. However, two separate meta-analyses provide evidence supporting the use of corticosteroid treatment over placebo, both showing a higher rate of complete recovery associated with the steroid treatment[8,9].

There is some evidence in favour of using combined steroid and antiviral treatment[10]. However, many studies, including two recent double-blind, placebo-controlled multi-centre trials, showed no improvement in outcome of Bell's palsy with antiviral treatment[11,12].

The current recommendation for treatment is early use of corticosteroids with a regimen of prednisone (up to 80mg daily) for one week[1].

• Surgical Decompression – this is also a controversial form of treatment. Surgical decompression of the facial nerve is not currently a recommended treatment in the UK[13].

• Eye Care – A potentially devastating complication of Bell's palsy is damage to the eye. Regular ophthalmic drops and ointments, as well as taping the eye shut at night are some of the important protective measures used in eye care[7].

• If any red flag symptoms are present, an Ear Nose and Throat (ENT) referral should be made.

Mr FC was discharged on a course of oral steroids and given eye care information. A follow-up appointment in the outpatient clinic was made to monitor symptoms, eye care and provide psychological support. The prognosis of Bell's palsy is excellent, with the majority of cases resolving completely[3, 13]. Residual facial weakness or synkinesis only persist in a minority of cases, requiring referral to either an ENT or a plastic surgery department with particular experience in managing facial palsy[3].

Self Assessment - Best of Five Questions

1. Which of the following best describe the composition of the facial nerve?
a) Motor fibres only.
b) Sensory fibres only.
c) Mixed motor and sensory fibres only.
d) Mixed motor, sensory and parasympathetic fibres only.
e) Sensory and parasympathetic fibres only.

2. Which of the following is NOT a function of the facial nerve?
a) Salivation.
b) Lacrimation.
c) Facial movement.
d) Maintenance of tension on the stapes bone.
e) Taste.
f) Contraction of the tensor tympani muscle.

3. The facial nerve is particularly vulnerable to compression at which site?
a) At the cerebellopontine angle.
b) At its point of entry into the internal auditory meatus.
c) As it runs through the labyrinthine segment of the internal auditory canal.
d) As it emerges through the stylomastoid foramen.
e) As it traverses the parotid gland.

4. Bell's palsy is suspected to be due to an inflammatory response to which virus?
a) Herpes Zoster.
b) Herpes Simplex Virus Type 1.
c) Human Papillomavirus.
d) Influenza Virus.
e) Epstein Barr virus.

5. A gradual-onset unilateral facial paralysis is most suggestive of what?
a) A Tumour.
b) Ramsay-Hunt Syndrome.
c) Otitis Media.
d) An upper motor neuron lesion.
e) Melkersson Rosenthal Syndrome.

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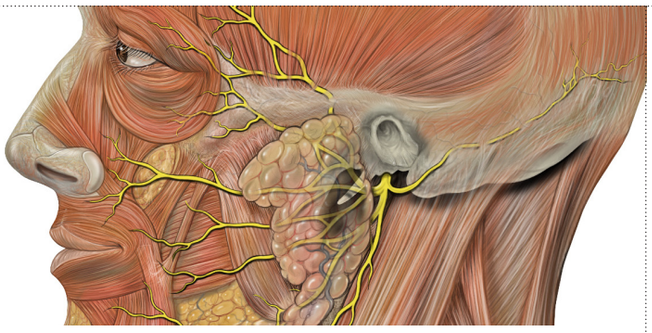

Answers

1. d. The facial nerve is a mixed nerve containing motor, sensory and parasympathetic fibres. Other cranial nerves, which contain mixed motor and sensory fibres include, the trigeminal nerve, the glossopharyngeal nerve and the vagus nerve.

2. f. Innervation of the tensor tympani muscle is a function of the trigeminal nerve. Contraction of the tensor tympani muscle while chewing allows dampening down of the noise produced by this action.

3. c. The facial nerve is most vulnerable to compression as it runs through the narrowest section of the internal auditory canal, which is the labyrinthine segment. Diameter at this site is approximately 0.68mm[1]. Recall, evidence suggests that the facial paralysis seen in Bell's palsy caused by swelling and thus compression of the facial nerve as an inflammatory response to viral infection.

4. b) The Herpes Simplex Virus type 1 (HSV-1). Research supports the concept that Bell's palsy is caused by swelling of the facial nerve due to an inflammatory response to viral infection. DNA fragments of the HSV-1 have been recovered from patients with Bell's palsy during surgical decompression, and independently at post mortem of patients demonstrating Bell's palsy shortly before death[2].

5. a) A tumour is implied by a slowly progressing palsy. Melkersson–Rosenthal syndrome characterized by recurring facial paralysis, while sparing of the forehead muscles is more suggestive of an upper motor neuron lesion.

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Authors

Dr. E. Naomi Smith
Foundation Year 2 in Cardiology
John Radcliffe Hospital
Oxford

Mr. Ian Bottrill
ENT Consultant
Stoke Mandeville Hospital
Buckinghamshire Hospitals NHS Trust

Correspondence

Dr. E. Naomi Smith
Foundation Year 2 in Cardiology
John Radcliffe Hospital
Oxford
email: elizabeth.smith@doctors.org.uk

IMPROVING HANDOVER OF SURGICAL PATIENTS AT WEEKENDS – A SIMPLE SOLUTION

Elizabeth Thorne, Kate Outerside, Nicholas Greaves, Deborah Nicol and Paul Murphy

Improving handover of surgical patients at weekends – a simple solution. Good Clinical Care.

Introduction

The introduction of the European Working Time Directive has resulted in a shift-based working environment with a greater requirement for handovers. It has been shown that these are times when errors commonly occur, resulting in difficulties for the on call doctor and risking patient safety. We propose the introduction of Weekend Handover Stickers (WHS) for every patient describing basic but essential information that aid weekend teams who are often unfamiliar with the patient.

Methods

All surgical inpatients at a district general hospital qualified for the audit. Their notes were assessed for the presence of various pieces of information on 2 occasions; before and after the introduction of weekend handover stickers. Doctors using the new system completed a questionnaire to establish their satisfaction with the new format.

Results

Eighty-five case notes were reviewed before the introduction and 63 afterwards. Weekend handover stickers (WHS) increased the frequency with which information, including diagnosis (↑62%), operation performed (↑21%), feeding status (↑25%), active problems (↑11%), weekend plan (↑67%) and a clear summary (↑53%) were included in the notes. 100% of the doctors asked felt the WHS were useful, improved the efficiency of weekend ward rounds and felt that their introduction was of long-term benefit.

Conclusion

This study has shown that weekend handover stickers are a simple, cheap and effective way to improve patient safety while concurrently increasing the ease of weekend ward rounds and patient assessment. The stickers have met with approval from the doctors using them and they have now been rolled out across the trust where the trial took place.

Background

The GMC guidance for good medical practice states that "you must be satisfied that, when you are off duty, suitable arrangements have been made for your patients' medical care. These arrangements should include effective hand-over procedures, involving clear communication with healthcare colleagues"[1] The European working time directive (EWTD) has led to the introduction of shift working on surgical wards with up to 3 handover sessions per 24 hours. Each hand over session may lead to a dilution of information and error. This may result in doctors reviewing patients previously unknown to them, with no documented clinical summary or future management plan. This is a common problem observed during weekends.

We suggest a simple, effective solution – introduction of a weekend handover sticker (WHS).

Methods

This work was performed in the department of general and colorectal surgery in a medium-sized district general hospital of 450 beds and was registered with the local hospital audit department.

Junior doctors were consulted on how best to manage the weekend handover, to streamline the process and produce the most information in the least time. It was decided that the junior doctors on Friday afternoon would produce a clinical summary for all their patients with the "weekend plan of management" (WPM). This was to be clearly placed in the clinical notes on a large fluorescent sticker.

WHS were introduced on surgical wards. These had a standard format with spaces to complete details of:

- **Summary.**
- **Diagnosis.**
- **Operation.**
- **Eating and drinking.**
- **Active problems.**
- **Plan for the weekend.**

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All doctors working the weekend shifts also anonymously completed a questionnaire before and after the introduction of the WPM comparing patient information and treatment plans. Furthermore they were asked to evaluate the impact of WHS.

Information obtainable from inpatient case notes were reviewed on two occasions over a month long period. First, before and then again, 4 weeks after the introduction of WHS.

Results

Eighty-five case notes were reviewed before the introduction and 63 afterwards. The percentage of case notes containing adequate information is shown in Table 1:

	Before	After
Summary	28%	81%
Diagnosis	20%	82%
Operation	38%	59%
E&D	54%	79%
Active problems	67%	78%
Weekend plan	14%	81%
Sticker used	-	88%

Table 1: The percentage of notes containing adequate information required for handover before and after the introduction of WHS.

All doctors (from house officer to consultant) working for a general surgery firm at this district general hospital were then asked to confidentially evaluate the WHS to assess whether this was a sensible and viable way to assist in the care of patients at weekends.

Question	Yes
Did you use the WPM	88%
Was the WPM useful	100%
Did the WPM increase efficiency of hand over/ward rounds	100%
Did the WPM aid you when asked to review patients	88%
Is this change of long term benefit	100%

Table2: The views of doctors regarding the WPM.

Conclusions

Good handover of patients is a GMC requirement and vital part of clinical care. With the continuing increase of shift working patterns, we must find ways to ensure that information is readily available to clinicians looking after our patients out of hours. At present, documented weekend handover is often not standardised, with incomplete details. We therefore propose that the introduction of WHS can improve the quality of handover information and optimise patient safety. It should become a compulsory and integral part of out of hours care. We have proved that it is feasible and effective, meeting with widespread approval from juniors and seniors alike since its introduction.

References

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Author

Dr Kate E Outterside MBChB, BSc Hons
University Hospital
Coventry and Warwickshire NHS Trust

Mr Nicholas Greaves MBChB, BSc(hons), MRCS
University Hospitals
Coventry and Warwickshire NHS Trust (Walsgrave site)
Clifford Bridge Road
Coventry
CV2 2DX

Corresponding Author

Dr Elizabeth Thorne MBChB, BSc Hons
University Hospital
Coventry and Warwickshire NHS Trust
Email: e.thorne@doctors.org.uk

Dr Paul Murphy FRCS PhD
South Warwick Hospital

Mrs Deborah Nicol MBChB MRCS MD
South Warwick Hospital

ASSESSMENT AND MANAGEMENT OF ADULT ACUTE UPPER AIRWAY OBSTRUCTION

G A Powell, A L Powell, O G J Booth, A K Arya and A C Swift

Assessment and Management of Adult Acute Upper Airway Obstruction. Case Based Discussion.

Abstract

A patient presented with acute onset shortness of breath and stridor due to a large tumour of the larynx. The initial assessment, investigation and treatment of patients presenting with acute upper airway obstruction is discussed. Systematic, clear guidance is presented for the physician involved in the management of this often frightening, emergency clinical presentation. Further, specialist ENT interventions relevant to the case report are then discussed in context.

Case History

A 71-year-old retired building surveyor presented to the A&E department as an emergency with acute onset shortness of breath and "noisy breathing". He had no other medical history, but had smoked 20 cigarettes per day for 40 years.

On examination, he had inspiratory stridor and was tachypnoeic; oxygen saturation was 97% on room air; he was tachycardic and normotensive. He had a hard, non-mobile mass, 6cm x 5cm, in levels 2 and 3 on the left side of the neck. The oropharynx was normal and the trachea was central.

He was initially treated with high flow oxygen using a non-rebreather mask and referred for urgent ENT review. Flexible nasendoscopy showed a supraglottic laryngeal tumour that was obstructing the airway. Regular intravenous dexamethasone was administered and his airway gradually improved.

Two days later, he underwent microlaryngoscopy and biopsy under general anaesthesia and the tumour was debulked with the aid of a CO_2 laser (Figure 1).

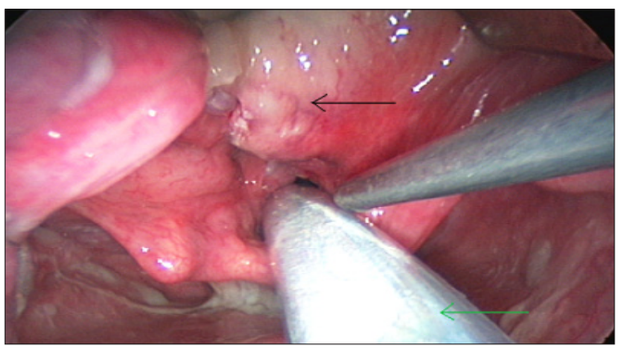
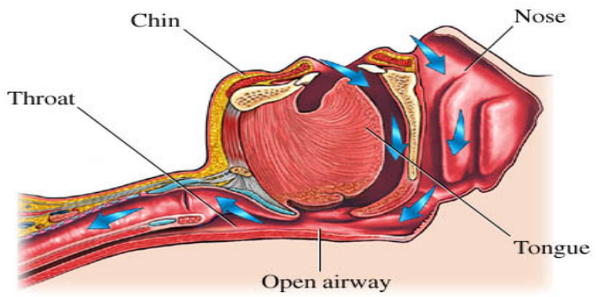


Figure 1: Photograph showing endoscopic view of larynx taken at laryngoscopy. The green arrow denotes the endotracheal tube entering the larynx at the level of the vocal cords and the black arrow denotes the supraglottic tumour.



The patient was discharged 3 days after admission. The histology was reported as squamous cell carcinoma and the patient subsequently underwent total laryngectomy with selective neck dissection from levels 2 to 4.

Discussion

This sequence of events demonstrates a not uncommon presentation of acute airway obstruction secondary to a tumour. As in this emergency scenario, assessment, consideration of differential diagnoses, initial investigations and treatment should occur promptly in a clear logical sequence.

An immediate, focused primary medical assessment should include management of the Airway, Breathing and Circulation as outlined by the Resuscitation Council[1]. Disability should then be addressed.

Airway

Severe, life-threatening airway obstruction should be recognised immediately and a senior anaesthetist and ENT surgeon should be summoned.

- Oxygen at 15 litres (L) per minute.
- Inspection including examination of the face, oral cavity, pharynx and neck.
- Listen for airway sounds including stridor and stertor.
- Feel for movement of air at the mouth.
- Remove obstructing foreign bodies.
- Airway opening manoeuvres.

Breathing

- Respiratory rate.
- Oxygen saturation/cyanosis.
- Ability to complete full sentences.

Circulation

- Pulse rate.
- Blood pressure.

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Disability

Conscious level should be ascertained rapidly using the A V P U (Alert, Voice, Pain, Unresponsive) categorisation[1].

This rapid primary medical assessment will identify airway obstruction and define severity. Adverse signs indicating severity include:

- Respiratory rate less than 8 or more than 22 breaths per minute.
- Inability to complete full sentences.
- Fall in oxygen saturation to less than 92%.
- Pulse rate less than 50 or more than 110 beats per minute.
- Systolic blood pressure lower than 90 millimetres of mercury.
- Reduction in conscious level.

In this case history, inspiratory stridor was recognised. Stridor, a sound produced by the glottis should be distinguished from stertor, a nasal sound originating in the pharynx. Stridor may be further divided into inspiratory, caused by the supraglottis, biphasic, at the level of the glottis and expiratory, occurring in the trachea. Although fully alert, the patient was tachypnoeic and tachycardic, therefore demonstrating adverse signs demanding the rapid involvement of an ENT specialist. At this stage, further specialist assessment and management was undertaken.

ENT Specialist Assessment

History and Examination

A comprehensive history of the presenting complaint includes the onset, progression, severity and aggravating, and relieving factors. Associated symptoms, such as dysphonia and dysphagia, in addition to odynophagia, referred otalgia and risk factors particularly smoking and alcohol history should be ascertained. Relevant past medical history should be sought, including ENT complaints and cardiorespiratory co-morbidities.

Examination of the patient includes the head and neck, respiratory and cardiovascular systems. Flexible nasendoscopy represents an essential diagnostic tool allowing direct visualisation of the larynx, leading to the provisional diagnosis of supraglottic laryngeal tumour.

A full description of all potential diagnoses is beyond the scope of this discussion but the most common causes are presented in Table 1.

Infective/ Inflammatory	supraglottitis, laryngo-tracheitis, anaphylaxis
Neoplastic	Oral cavity, tongue base, pharyngeal, laryngeal
Trauma	External insult, noxious inhalation
Miscellaneous	Inhaled foreign body, blood, vomitus, laryngeal spasm

Table 1: Adult Acute Upper Airway Obstruction: Differential Diagnoses.

Investigations

Basic investigations should include blood tests (full blood count, urea and electrolytes, C-reactive protein, arterial blood gas) and chest radiograph.

Initial Management

Oxygen

All patients should receive 15 L/min oxygen via a non-rebreather mask, continuous pulse oximetry and regular basic observations including pulse rate, respiratory rate and blood pressure.

Topical Adrenaline and Systemic Steroids

Both nebulised adrenaline and intravenous steroids are useful in conditions where laryngeal oedema may be an exacerbating factor. Topical adrenaline causes local vasoconstriction and intravenous steroids decrease the synthesis of pro-inflammatory mediators and reduce subsequent inflammatory oedema[2]. Nebulised adrenaline, although not administered in this case, would be an acceptable addition to the initial management.

Heliox

Heliox is a combination of 21% oxygen and 79% helium[3]. The substitution of nitrogen for helium results in a gas that has a lower viscosity than air. Airflow is therefore enhanced and oxygen saturation can be maintained with reduced respiratory effort.

Antibiotics

Intravenous antibiotics should be administered promptly if an infective cause, such as epiglottitis/supraglottitis, is suspected.

Further Management

Endotracheal intubation may be indicated if a patient's airway remains compromised despite medical therapy. Traditional orotracheal intubation involves direct visualisation of the glottis using a laryngoscope. Rapid Sequence Induction (RSI), a method of quickly attainting a state of anaesthesia, involves the rapid administration of agents without an assessment of effect and may be used prior to intubation in this emergency scenario.

Fibreoptic bronchoscope guided endotracheal intubation is a useful technique considered when the glottis cannot be visualised or when there are anticipated anatomical abnormalities, commonly found in patients presenting with airway obstruction. Importantly, this method can be performed with the patient awake, allowing the patient to ventilate throughout. Administration of topical local anaesthetic, sedation and an antisialogogue are necessary prior to the procedure.

If the obstruction is unlikely to be relieved in the acute phase and a prolonged period of intubation would be necessary, a temporary tracheotomy may be created allowing further investigation and definitive management. In an emergency situation this may be performed under local anaesthetic. When endotracheal intubation fails and the patient is demonstrating adverse clinical signs a cricothyroidotomy may have to be performed.

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Following resolution of the acute episode, direct examination of the upper airway under anaesthetic, computed tomography and/or magnetic resonance imaging may be necessary where malignancy is suspected. In addition, imaging of the thorax is essential to exclude synchronous pulmonary tumours, present in up to 3% of head and neck cancer patients[5]. In this case, the patient was diagnosed with laryngeal carcinoma staged in accordance with the Tumour, Node, Metastasis classification[4] as stage III (T3N1M0). Endoscopic debulking was necessary prior to discharge with subsequent total laryngectomy and bilateral selective neck dissections.

Single Best Answer Self-Test Questions

1. From the options shown below, choose the most appropriate initial investigation for a patient with acute airway compromise.

a) Direct laryngoscopy under anaesthetic.
b) Flexible nasendoscopy.
c) MRI of neck.
d) Plain radiograph of cervical spine.
e) Ultrasound of neck.

2. A 34-year-old woman with stridor and tachypnoea is able to complete full sentences and has an oxygen saturation of 92% on air. She had a sore throat over the previous 2 days, but noticed her breathing had become much worse over the last few hours. From the options shown below choose the most appropriate person to call in this situation.

a) Cardiac arrest team.
b) Critical care consultant.
c) CST1 trainee.
d) ENT specialist registrar.
e) FY1.

Answers

1. b)

- In the acute setting, airway compromise can prove to be fatal within minutes if not rapidly identified.
- Flexible nasendoscopy is a readily accessible and simple investigation that can be used to rapidly visualise the patient's airway directly, providing definitive confirmation of obstruction within seconds.
- The main potential problem with flexible nasendoscopy is lack of availability.
- Flexible nasendoscopy, when performed skilfully, can provide a rapid accurate assessment of the airway and a diagnosis without exacerbating airway obstruction.

2. d)

- Airway compromise is associated with rapid deterioration in clinical condition. The likely diagnosis in this case is acute epiglottis and so the input of a senior ENT surgeon should be sought as quickly as possible.
- The FY1 would be discouraged from attempting to manage this type of situation alone, as the patient may deteriorate rapidly and requires expert input from a senior experienced in airway management.
- The opinion of an anaesthetist is also important in this situation, and should be considered early in the assessment process.

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Authors and Correspondence

G A Powell
Foundation Year 1 Doctor
Aintree University Hospital NHS Foundation Trust
Liverpool, L9 7AL

O G J Booth
Foundation Year 1 Doctor
Aintree University Hospital NHS Foundation Trust
Liverpool, L9 7AL

A L Powell
Medical Student (4th year)
University of Liverpool
Liverpool, L69 3BX

A K Arya
Specialist Registrar Otolaryngology
Head & Neck Surgery
Mersey Deanery

A C Swift
Consultant in Otolaryngology
Head & Neck Surgery and Honorary Senior Lecturer
Department of Otolaryngology, Head and Neck Surgery
Aintree University Hospital NHS Foundation Trust
Liverpool
L9 7AL

MANAGEMENT OF AN OTOLOGICAL EMERGENCY

G L Noblet, N L Whitham, S C Day, A K Arya, E Osman and A C Swift

Management Of An Otological Emergency. Case Based Discussion.

Abstract

A 25-year-old man with a long history of otitis media with effusion presented with a right-sided facial nerve palsy and ipsilateral facial swelling. Examination revealed complete facial nerve palsy and erythema and swelling over the right side of his face. Otoscopy showed a tympanic perforation and an attic polyp. A working diagnosis of cholesteatoma was made and confirmed by CT. He was treated with intravenous and topical antibiotics and systemic steroids. The cholesteatoma was removed by urgent modified radical mastoidectomy and the facial nerve was decompressed.

The presentation, investigation and management of this patient highlight serious complications arising from cholesteatoma and the importance and potential success of swift diagnosis and treatment. Points for consideration raised by this case include facial nerve paralysis, acute and chronic otitis media and cholesteatoma.

Case History

A 25-year-old asthmatic man was admitted with right-sided facial swelling and weakness. He explained that four days prior to admission, he had attended a local walk-in centre because he was experiencing sharp pain and otorrhoea from his right ear after his father had shouted loudly at him. A perforated tympanic membrane was diagnosed. He was commenced on erythromycin for 7 days. Four days later his pain had worsened, the discharge had increased, and he developed facial swelling and weakness.

He had suffered with recurrent ear problems as a child and had grommets inserted at the age of 9 years. He was unemployed, smoked 30 cigarettes a day and drank 20–30 units of alcohol a week.

On examination he was apyrexial and had a complete lower motor neurone right-sided facial weakness and erythematous swelling over the right side of the face and external ear. Otoscopy showed swelling of the ear canal and an attic defect with an attic polyp in the superior part of the ear drum.

A working diagnosis of facial nerve palsy secondary to mastoiditis or cholesteatoma was made. Tazocin, a broad spectrum intravenous antibiotic, was prescribed as well as ciprofloxacin eardrops and dexamethasone orally to reduce local inflammation (AHT1). His audiogram showed a mild conductive hearing loss.

A CT scan was requested and found features consistent with a chronic otitis media and erosive changes consistent with cholesteatoma.

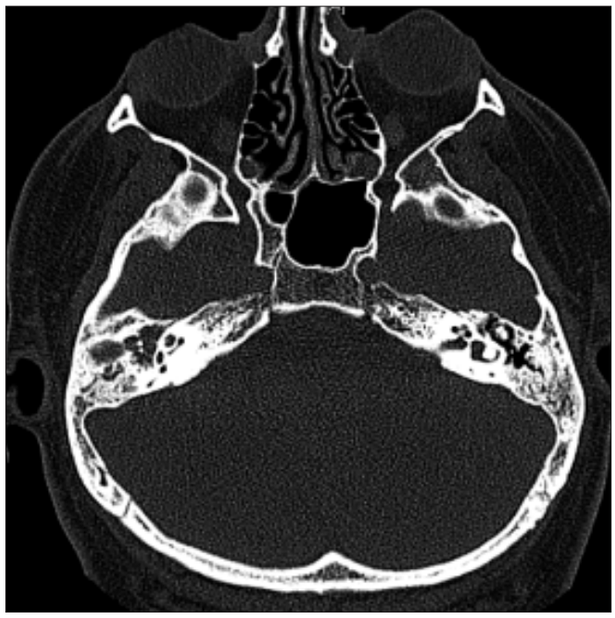

Figure 1: Axial CT scan of the temporal bones demonstrating soft tissue in the right middle ear. Note the patent mastoid air cells on the left (normal) ear. The soft tissue was identified as cholesteatoma at surgery.

The patient was taken to theatre for right modified radical mastoidectomy and facial nerve decompression. A cholesteatoma was found in the mastoid air cell system. This was impinging on the facial nerve. The cholesteatoma was removed and middle ear cavity completely cleared of all disease. His stapes was intact, but the incus and malleus head had to be removed to facilitate clearance of all the disease (AHT2).

The patient was discharged the following day with a 7 day course of cefaclor, hypromellose eye drops, lacrilube and analgesics. He was advised to tape the eye closed at night. When seen 3 weeks later the facial nerve was fully intact and the mastoid cavity was healing well.

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Discussion

This case highlights rare but potentially serious complications of acute or chronic otitis media. Other intracranial and extracranial complications are listed in Table 1.

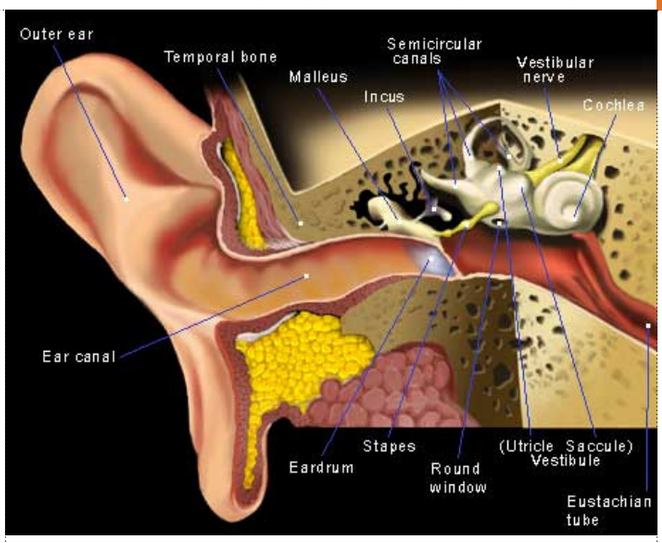
Intracranial	Extracranial
Meningitis (most common)	Mastoiditis (most common)
Brain abscess	Facial nerve paralysis
Extradural abscess	Labyrinthitis
Sigmoid sinus thrombophlebitis	Gradenigo's syndrome (triad of Otitic hydrocephalus abducens nerve paralysis, trigeminal nerve distribution pain, acute suppurative otitis media),
Otitic hydrocephalus	Subperiosteal abscess
	Bezold's abscess (laterocervical abscess)

Table 1: Complications of Otitis Media.

Cholesteatoma is a destructive disease that may require surgical treatment. Although its name suggests otherwise, cholesteatoma is not a tumour but is defined as a keratinizing squamous cell cyst occurring in the middle ear. Squamous epithelium becomes trapped within the temporal bone, middle ear or mastoid and may migrate into the surrounding bone containing it causing complications, which can be life threatening.

Cholesteatoma may be congenital (arising from trapped squamous epithelium within the temporal bone during embryogenesis) or acquired (arising as the result of tympanic membrane retraction or injury), such as perforation resulting from acute otitis media, trauma or surgery. This patient displayed the typical features of an acquired cholesteatoma. The disease often presents with a continuous or recurrent painless mucopurulent otorrhoea. Other significant features include marked hearing loss, facial nerve paralysis, dizziness, vertigo, earache and headaches. Examination typically shows granulation tissue in the middle ear and attic perforation, sometimes associated with an aural polyp.

Initial treatment includes regular cleaning and antibiotic ear drops to control infection and slow growth. However, as in this case surgical treatment is usually required for large or complicated cholesteatoma.



Complications from such surgery include

- facial nerve paralysis,
- sensorineural hearing loss,
- alteration in taste on the anterior ipsilateral tongue and, balance disturbance.

This patient's main presenting complaint was that of a facial nerve palsy, and this coupled with his otological symptoms warranted immediate ENT referral. Facial nerve palsy can be caused by many different conditions, but the most common cause is idiopathic (Bell's palsy, see Table 2). As such Bell's palsy is a diagnosis of exclusion and a thorough search of any likely aetiological factors must be undertaken before this diagnosis is made.

Causes of facial nerve palsy
Idiopathic (Bell's palsy)
Otitis media
Cholesteatoma
Ramsay-Hunt syndrome
Lyme disease
Trauma
Stroke
Guillain-Barré syndrome
Multiple sclerosis
Tumours (intracranial; posterior fossa; parotid gland)
Vasculitides
Sarcoidosis

Table 2: Causes of facial nerve palsy.

MANAGEMENT OF AN OTOLOGICAL EMERGENCY

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Facial nerve palsy is graded according to the House-Brackmann (HB) classification[1] (see Table 3). Grades above HB grade IV imply incomplete eye closure meaning that the cornea is unprotected. This patient's HB grade was VI and so adequate protection of the eye, such as by using artificial tears and taping the eye at night, was advised. In severe cases an ophthalmology review should be considered.

Grade	Definition
I	Normal symmetrical function in all areas
II	Slight weakness noticeable only on close inspection Complete eye closure with minimal effort Slight asymmetry of smile with maximal effort Synkinesis barely noticeable, contracture or spasm absent
III	Obvious weakness, but not disfiguring May not be able to lift eyebrow Complete eye closure and strong but asymmetrical mouth movement with maximal effort Obvious, but not disfiguring synkinesis, mass movement or spasm
IV	Obvious disfiguring weakness Inability to lift brow Incomplete eye closure and asymmetry of mouth with maximal effort Severe synkinesis, mass movement, spasm
V	Motion barely perceptible Incomplete eye closure, slight movement at corner of mouth Synkinesis, contracture and spasm usually absent
VI	No movement, loss of tone, no synkinesis, contracture or spasm

Table 3: House–Brackmann facial nerve grading system[1].

Test Yourself

1. What is the most common extracranial complication of otitis media?

a) Sigmoid sinus thrombophlebitis.
b) Mastoiditis.
c) Ramsay-Hunt syndrome.
d) Labyrinthitis.
e) Facial nerve palsy.

2. A patient presents with a visibly disfiguring facial weakness, incomplete eye closure, asymmetrical mouth and inability to raise their eyebrow. Which House-Brackmann grade would they be?

a) Grade I
b) Grade II
c) Grade III
d) Grade IV
e) Grade V

Answers

1. b) Mastoiditis.

Mastoiditis is the most common complication of otitis media (see Table 1). Mastoiditis can be diagnosed clinically by the observation of a red fluctuant swelling behind the ear, often causing the ear to protrude. Treatment may involve the use of antibiotics or surgical drainage in severe or non-refractory cases.

2. d. Grade IV

The House-Brackmann (HB) facial nerve grading system is used to assess the severity of facial nerve weakness. Normal function and symmetry is awarded a grade I HB while complete nerve palsy is a grade VI HB. (See Table 3). Facial nerve palsy may be caused by many disease processes (See Table 2).

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Authors

G L Noblet
Foundation Year 1 Doctor
Aintree University
Hospital NHS Foundation Trust, Liverpool, L9 7AL

N L Whitham
Foundation Year 1 Doctor
Aintree University, Hospital NHS Foundation Trust, Liverpool, L9 7AL

S C Day
Foundation Year 1 Doctor
Aintree University, Hospital NHS Foundation Trust, Liverpool, L9 7AL

A K Arya
Specialist Registrar, Mersey Deanery

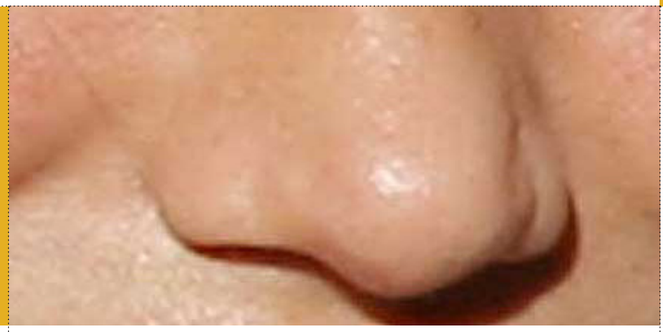
E Osman
Consultant in Otolaryngology, Head & Neck Surgery
and Honorary Senior Lecturer
Department of Otolaryngology, Head and Neck Surgery
Aintree University Hospital NHS Foundation Trust, Liverpool, L9 7AL

A C Swift
Consultant in Otolaryngology
Head & Neck Surgery and Honorary Senior Lecturer
Department of Otolaryngology, Head and Neck Surgery
Aintree University Hospital NHS Foundation Trust, Liverpool, L9 7AL

A REVIEW OF OLFACTORY TESTS TO ASSESS ANOSMIA

Megan Lamb and Shadaba Ahmed

A Review of Olfactory Tests to Assess Anosmia. Good Clinical Care.



Abstract

Anosmia, the total inability to smell, has often been regarded as an insignificant medical issue. However, it can be a sign of serious pathology and have multiple implications on a patient's quality of life. Many olfactory tests are available in the UK, however, there is much debate surrounding which is the best for use in regular clinical practice. Currently, little literature exists on the subject of olfactory testing, especially with reference to the UK, and to date only one test has been validated in the UK. This review is aimed at establishing the most suitable and reliable test for regular clinical practice. A literature search was completed and relevant articles were reviewed. In conclusion, the main reason no single universally accepted test exists is due to the fact that patients from different countries are not familiar with the same smells and the great variation in test reliability. Not all tests have been reviewed; therefore, there may be other more reliable, more valid tests commercially available. However, after critical review the UPSIT proved the most reliable test and probably the most suitable for use in the UK.

Introduction

Classically olfaction and anosmia, the total inability to smell, have often been seen as insignificant medical issues. However, anosmia can have serious implications on a patient's quality of life. When people lose their sense of smell, food has less flavour leading to malnutrition, because of loss of appetite, or obesity, because people overeat trying to gain pleasure from tasteless food. It may affect a patient's occupation, for example, a chef must be able to smell the food he cooks. Olfaction is necessary for a person to avoid danger; food that smells bad is often contaminated and mercaptan, a foul smelling substance, is added to natural gas so leaks can be identified. Many diseases present early with anosmia, for example, Alzheimer's disease. Timely detection enables treatment to be started early; possibly improving prognosis. For some diseases, such as intracranial pathology, anosmia may be the only symptom and, therefore, essential to detect[1]. Many olfactory tests are currently available in the UK. This report is aimed at establishing which may be the most suitable and reliable for regular clinical practice.

Methods

The following search terms were entered into the meta-search engine Metalib: "anosmia", "nasal anatomy", "olfaction", "olfactory anatomy", "olfactory disorder", "olfactory test" and "psychophysical test". Metalib searches several databases: Medline, PsychINFO, PsychArticles, Web of Science, Academic Search Complete, EMBASE: Excerpta Medica (Ovid), and CINAHL. Relevant articles were reviewed.

Anatomy of Olfaction

Olfactory Epithelium

The olfactory epithelium is situated in the nasal cavity; the region covering the cribriform plate and sections of the superior turbinate, middle turbinate and septum. It is an area of about 5cm^2 consisting of a layer of olfactory receptor cells scattered amongst supporting cells, sitting upon a layer of basal stem cells[2].

The olfactory receptor cells are first order bipolar neurons. From their apical pole they extend a single dendrite to the epithelial surface where it expands to a knob covered by 5–20 immotile cilia[3]. From its basal pole an unmyelinated axon extends through a foramen in the cribriform plate. The axons run in bundles forming the olfactory nerves which extend to the paired olfactory bulbs. The supporting cells are columnar epithelial cells with microvilli.

Covering the epithelium is a layer of mucous secreted by supporting cells and Bowman's Glands[2]. The mucous provides the optimum environment for odour detection and contains soluble odourant binding proteins, believed to contribute to odourant concentration and removal, but not directly to odour detection[3].

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Olfactory Bulb

The olfactory bulbs lay on the cribriform plates either side of the crista galli. Each can be divided into four layers (superficial to deep): the glomerular layer, the external plexiform layer, the mitral cell body layer and the granular layer[2]. Once the axons of the olfactory receptor cells pass into the olfactory bulb they synapse with second order neurons in glomeruli, forming the glomerular layer. There are three different second order neurons which the olfactory receptors may synapse with: mitral relay neurons, tufted relay neurons and periglomerular neurons. In each glomerulus about 25,000 olfactory receptors synapse with the primary dendrites of around 25 mitral or tufted relay neurons[2]. The cell bodies of mitral cells comprise the mitral cell body layer. The axons originating in the mitral and tufted cells leave the olfactory bulb and continue as the lateral olfactory tract to the anterior olfactory nucleus and the olfactory tubercle[4]. Some mitral cell axons extend towards the piriform cortex, the amygdala and the entorhinal cortex[3,4]. Secondary dendrites of mitral and tufted cells extend to the external plexiform layer where they synapse with granule cell interneurons. The cell bodies of granule cells form the granular layer. Periglomerular and granule cells make connections between the glomeruli; the former inhibit lateral glomeruli but allow excitation of a specific mitral cell whereas the latter inhibit mitral cells[5].

Physiology of Olfaction

Odourous molecules bind to specific receptors on the olfactory cilia. There is a huge array of receptors enabling a wide variety of odours to be identified. The receptors are G protein-coupled receptors which transduce signals by interactions between GTP-binding proteins. Once an odourant binds to a receptor, the G protein it is linked to activates adenylate cyclase. This results in an increase in the production of cyclic adenosine monophosphate (cAMP) from ATP. The increase in cAMP causes cyclic nucleotide gated channels to open allowing the entry of sodium (Na+) and calcium (Ca2+) into the neuron[3,4]. The inflow of sodium and calcium causes depolarisation and a graded potential which may cause an action potential at the cell body and transmission of a signal to the olfactory bulb – olfactory transduction.

Anosmia

Anosmia is the total loss of olfaction. Hyposmia is a partial loss of olfaction. Both occur naturally: 50% of people over the age of 65 and 75% of people over 80 have a reduced ability to smell[6]. There are many causes of anosmia, which can be classed into three major categories: conductive, sensory and neural[6]. Conductive causes are due to a blockage that prevents odourous molecules reaching the olfactory epithelium. Sensory causes occur when olfactory receptors have been damaged. Neural causes are due to damage to neural pathways. All olfactory disorders can be placed into one of these categories, but some straddle two. For example, rhinosinusitis can be a conductive and sensory cause: it causes a blockage and damages olfactory epithelium. Anosmia can either be intermittent or constant. Intermittent anosmia is usually due to conductive causes, whereas constant anosmia is usually due to sensory or neural causes. Around 22% of anosmias are idiopathic[1]. Smoking is a well-known cause of anosmia and occupational exposure to certain noxious chemicals may also cause anosmia[1]. Iatrogenic causes include medication, radiotherapy and previous nasal surgery[1,5]. Many diseases can cause anosmia (Table 1).

Neurological
Alzheimer's Disease
Down Syndrome
Epilepsy
Multiple Sclerosis
Parkinson's Disease
Congenital
Kallman's Syndrome
Choanal Atresia
Nutritional and Metabolic
Chronic Renal Failure
Liver Disease
Vitamin B12 Deficiency
Endocrine
Diabetes
Adrenal Cortex Insufficiency
Hypothyroidism
Cushing's Disease
Trauma
Head Injury
Laryngectomy
Inflammatory
Rhinosinusitis or nasal polyposis
Sarcoid
Wegener's Disease
Neoplastic
Olfactory Neuroblastomas
Anterior Skull Base Tumours
Degenerative Age
Infective
Acute Viral Hepatitis
HIV
Influenza-like
Other
Adenoid Hypertrophy
Familial
Psychiatric

Table 1: Medical Diseases causing Olfactory Dysfunction (reproduced from 1 with permission).

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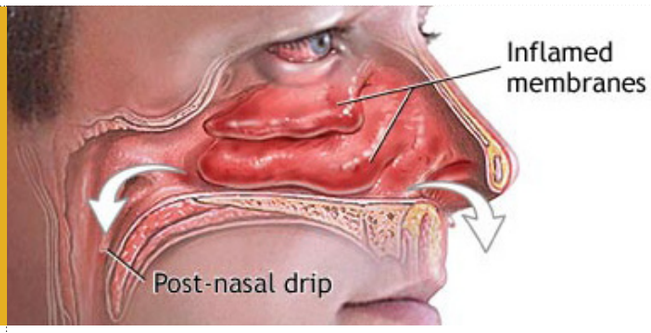
A Review of Olfactory Tests to Assess Anosmia. Good Clinical Care.



Diagnostic Testing

Olfactory tests are necessary to determine the extent of olfactory deficit[4] and to monitor the progression of any disease. However, they are unable to identify the actual cause of olfactory loss, therefore it is crucial that a thorough history and examination is undertaken in order to formulate a differential diagnosis[5]. Many olfactory tests are currently available commercially, but there is considerable variation in their reliability and no consensual agreement on which test is the gold standard, both in the UK and universally. The reliability and validity of tests are obviously important factors, but cost, patient preference and the complexity of the test should also be considered.

There are three main types of olfactory function test: psychophysical, electrophysiological and psychophysiological[7].

Psychophysical tests are used to assess a patient for:

1) odour identification – the ability to recognise different odours.
2) odour discrimination – the ability to differentiate an odour from others.
3) odour threshold – the ability to detect the odour at low concentrations[8].

Some psychophysical tests assess all three components of olfaction; others only assess one. However, it has been shown that testing for discrimination and threshold adds to the reliability of an identification test and provides a much more complete approach[9]. Odour memory, the ability to identify an odour from a selection after it has already been presented[4], can also be measured in some psychophysical tests. Electrophysiological tests work by assessing the brain and olfactory tract for electrical changes when a stimulus is presented to the patient[7]. Psychophysiological tests assess changes throughout the body, such as blood pressure and heart rate, which occur when a stimulus is present[7].

Sophisticated equipment is required to perform electrophysiological and psychophysiological tests. Psychophysical tests are quicker, easier and cheaper to perform and therefore are the most commonly used. However, problems exist in the application of odour identification tests. Across cultures different odours are familiar to different populations. Familiarity with an odour is essential for its identification[10]. A test developed in one country may not work in another because the population will not be able to recognise the odourants. This is the major factor preventing the creation of a universal test.

The reliability of the most popular and widely used olfactory tests were reviewed (Table 2). There is little literature available on the validity of the tests. Only the combined olfactory score has been validated in the UK even though it is not commercially available. From this literature review it was apparent that the most reliable test is the UPSIT. However, because the "Sniffin' Sticks" assesses more than one component of olfaction both them and the tests developed from them have been reviewed in more detail.

Test	Test-Retest Reliability Coefficients
University of Pennsylvania Smell Identification Test	0.90 - 0.95
Sniffin' Sticks	0.61 - 0.82
Sniffin' Sticks Screening Test	0.77
Cross-Cultural Smell Identification Test	0.71-0.73
Scandinavian Odour Identification Test	0.64 - 0.79
Smell Threshold Test	0.70 - 0.88
Sniff Magnitude Test	0.80

Table 2: The Test-Retest Reliability of Olfactory Tests (from [4,7,10,12,13,14,15]).

University of Pennsylvania Smell Identification Test (UPSIT).

The University of Pennsylvania Smell Identification Test, also known as The Smell Identification Test, uses a forced choice scratch and sniff method to identify 40 different suprathreshold odourants. Above each odourant are four possible answers. This forced choice technique reduces response bias[4] and enables the patient to dismiss unlikely smells if they do not recognise them. The test takes 10–15 minutes to perform.

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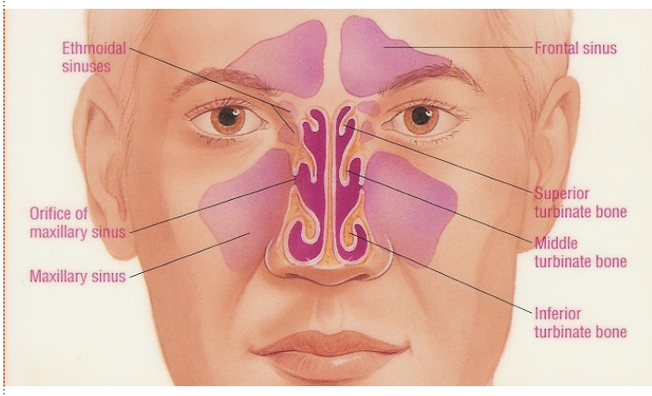
The patient's score is compared to age and gender related norms and then categorised as either: normosmia, mild microsmia, moderate microsmia, severe microsmia and anosmia[11] (someone who scores less than 10/40). The test can identify malingerers; anyone scoring less than 5/40.

Clove	Liquorice	Dill pickle	Pizza
Mint	Lilac	Motor oil	Strawberry
Peanut	Paint thinner	Rose	Smoke
Wintergreen	Root beer	Banana	Soap
Gasoline	Cherry	Bubble gum	Menthol
Grape	Natural Gas	Onion	Cedar
Leather	Chocolate	Pine	Cinnamon
Coconut	Peach	Turpentine	Watermelon
Orange	Gingerbread	Grass	Pineapple
Lemon	Lime	Cheddar cheese	Fruit punch

Table 3: Odourants used in the University of Pennsylvania Smell Identification Test[13].

The test-retest reliability of the UPSIT is high ranging between r=0.90[14] and 0.95[12,14]. In one study the sensitivity and specificity of the UPSIT was 91% and 88% respectively in differentiating between 32 male patients with Parkinson's disease and 128 male controls[7]. For the 28 females with Parkinson's disease and the 112 female controls in the same study the sensitivity was 79% and the specificity was 85%[7]. However, the UPSIT has not been validated in the UK.

From a questionnaire sent to consultants and associate specialist members of the British Association of Otolaryngologists and Head and Neck Surgeons it is the most commonly used test in the UK[1]. However, from all questionnaires sent out in the review only 45.1% were returned[1], so this may not be a true representation.



The UPSIT is available in English, Spanish, French, German and Japanese, however, the problems with "familiar odours" across cultures drove the creators to develop the Cross-cultural Smell Identification Test (CC-SIT), also known as the B-SIT. Based on the UPSIT and consisting of only 12 odourants, it takes 5 minutes to perform. However, evidence states the shorter the identification test, the lower the reliability[13]. The CC-SIT has a test-retest reliability, r=0.73[15]. However, this value was worked out using the Spearman-Brown formula, which relates test length to reliability, from the already known value for the UPSIT. Unpublished data from Doty et al. showed the CC-SIT has an empiric test-retest reliability, r=0.71[13]. These values are very similar; however, it would be useful if the test was validated in the UK. Malingerers cannot be identified because there are too few items in the test which makes the value ranges for malingerers and anosmics too close together. The smaller ranges for the categories, the inability to identify malingerers, as well as its lower reliability, all cause the test to have lower sensitivity than the UPSIT[15]. It is also quite expensive. Its advantage is that it is quick and easy to perform and can quantify olfactory loss, although not to the extent that the UPSIT can.

Sniffin' Sticks

In the Sniffin' Sticks test odourants are presented in felt tip pens, held 1–2cm from the patient's nostril. There are two types of Sniffin' sticks tests: a screening test and an extended test. The extended test measures odour identification, discrimination and threshold. To test for identification the patient is presented with 16 different suprathreshold odourants. All have a list of four possible answers to choose from. Testing for discrimination uses triplets of pens containing suprathreshold odourants; two smell the same and one smells different. The patient must identify which is different. Threshold testing uses different concentrations of an odourant in a multiple step staircase approach. The complexity of the test means it is difficult for a patient to self-administer, unlike the UPSIT. However, a study in Vienna found results were equally valid when the test was self-administered[11]. The results are related to age and gender norms and then classed as either normosmic, hyposmic or anosmic. The test is currently in use in Austria, Switzerland, Germany and Italy, but not routinely in the UK. However, it has been shown that the test can be applied to other cultures[17]. The reliability of the identification component for Sniffin' Sticks is 0.73 and for the threshold component the reliability ranges from 0.61–0.82 in different studies[7].

In one study among normosmic Italians, cloves was the only odourant individuals had difficulty identifying. However, because the test is multiple choice, several subjects correctly identified it: 23 out of 102[4]. However, this is only 22.5% of subjects; using random choice 25% of subjects should correctly identify the answer. This indicates multiple choice does reduce the risk of results being affected by unfamiliar odours.

The Sniffin' sticks screening test only assesses odour identification and uses 12 odourants, but is performed in the same manner. The test-retest reliability is 0.77[4], which deviates from the norm; that shorter tests are more unreliable. The extended test has a lower reliability.

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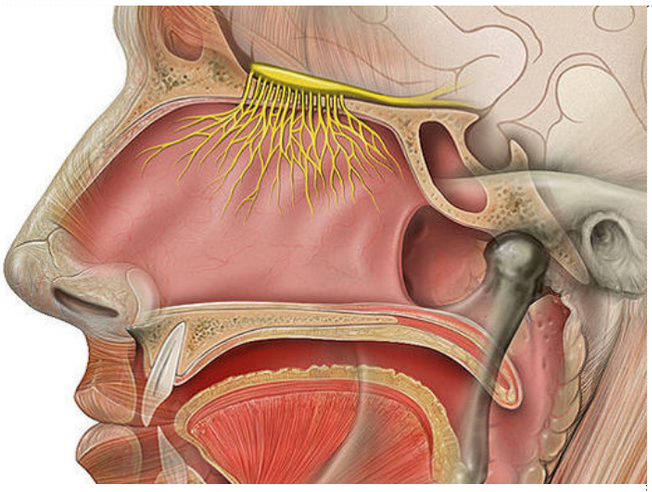

Conclusion

There is no single universally accepted olfactory test mainly due to the fact that patients in different countries are not familiar with the same smells.

Great variation is present in the reliability of the tests and as not all tests have been reviewed in this report there may be other more reliable, more valid tests commercially available. However, of all the tests reviewed, the UPSIT is the most reliable and most suitable for use in the UK. It is easy to understand that some odourants may be difficult to identify, for example would everyone know the difference between turpentine and paint thinner or exactly what pizza or bubblegum should smell like? It does overcome this somewhat by using forced choice and having completely different odourants in the multiple choice options to prevent confusion between unfamiliar smells. However, is this choice of odourants suitable for the UK? The UPSIT is only an odour identification test it would benefit from adding odour discrimination and threshold sections. Perhaps a test similar in structure to the Sniffin' sticks would be more appropriate.

Though the UPSIT is the most reliable test would it be more appropriate to see which test is the most reliable for each component of olfaction[13]? If odour threshold needed to be assessed would it be best to use the test which was the most reliable for assessing threshold? However, for ease and to increase reliability perhaps one test should incorporate odour identification, discrimination and threshold.

Not much literature is available on the validity of the tests. With the exception of the combined olfactory score, they all still need to be validated in the UK. Reliability and validity measures should be derived for all olfactory tests in the UK. Further research into the most appropriate test for the UK population should be considered with the intention of tailoring a test to the UK population. This could possibly be achieved by individually testing the reliability, validity and familiarity rating of each item in each test. This could indicate which odourants are most appropriate for use in the UK.

After thorough consideration the most appropriate test which is currently available for use in clinical practice is in fact the UPSIT.

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Authors

Megan Lamb
3rd Year Medical Student
Lancaster University

Shadaba Ahmed FRCS-ORL, HNS
ENT Consultant

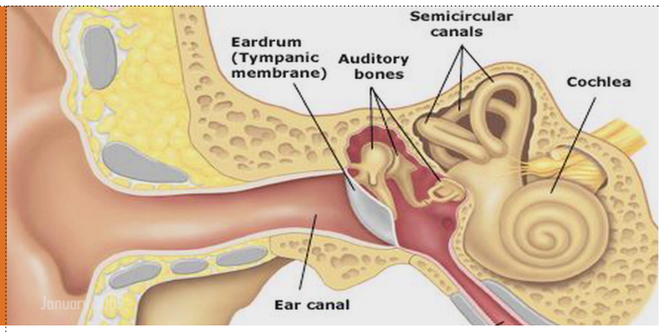
Correspondence

Megan Lamb
email: m.lamb@lancaster.ac.uk

ACUTE OTITIS MEDIA

Seema Yalamanchili, Rishi Talwar and Helen Caulfield

Acute Otitis Media.
Case Based Discussion.



Abstract

This case-based discussion centres on a 9-month-old boy presenting to A&E with acute otitis media and unilateral facial weakness. It outlines the clinical assessment, investigations and treatment of such a case and discusses the causes and complications relating to this diagnosis.

Case History

A 9-month-old infant presents to A&E with pyrexia and symptoms of an upper respiratory tract infection (URTI). He has been tugging at his ear and his mother has noticed the left side of his face is drooping.

Why should you consider Acute Otitis Media (AOM) in this case?

How would you make the diagnosis?

Acute otitis media (AOM) is an acute inflammatory process within the middle ear and one of the most common infections in children. It is often preceded by URTIs and can be complicated by facial nerve palsy. Though Bell's palsy (idiopathic LMN facial nerve palsy) is a differential, it is very rare in children and is only a diagnosis of exclusion. A complete history and examination must be completed to confirm middle ear disease and exclude other pathology along the course of the nerve.

What is the natural course of AOM?

Viral URTIs, most commonly caused by RSV, predispose to AOM by causing secretions and inflammation impairing Eustachian tube function. As middle ear air is absorbed, the resulting negative pressure draws in an effusion. This is a rich feeding medium for microbes, particularly bacteria, which are the most common cause of AOM. AOM can result in severe otalgia and pyrexia but often resolves spontaneously. If untreated, the tympanic membrane can rupture. This is associated with sudden otorrhoea and rapid relief of otalgia.[1]

What points should be clarified in the history?

Presenting Complaint

- When was the onset of symptoms? Are they worsening or improving?
- Did or does the child have nasal congestion? The Eustachian tube connects the nasopharynx to the middle ear, through which pathogens can migrate.
- Has the child been pulling at his ears? An infant cannot verbalise the site of pain.
- Has there been any otorrhoea?
- Is there any evidence of hearing difficulty?
- Is the facial nerve affected? Can the child close their eyes? Are they drooling?
- Is there any swelling or redness over the mastoid indicating acute mastoiditis?
- Any other concerning neurology, e.g. drowsiness or photophobia?

Past Medical History

- Have there been any recent coryzal illness symptoms or URTIs?
- Has the child had previous ear problems including operations, e.g. insertion of grommets?
- Are there any neuromuscular co-morbidities? These can also predispose to reflux from the nasopharynx via the Eustachian tube.
- Have they had chicken pox? Ramsay-Hunt syndrome is an important differential (*Herpes zoster* causing facial nerve palsy with vesicles in the ear canal and auricle).
- Does the child snore? Adenoid hypertrophy can cause Eustachian tube blockage and large adenoids may be covered in a biofilm that predisposes to the spread of infection.
- Consider perinatal history ask about prematurity, admission to the Special Care Baby Unit (SCBU) and breastfeeding.
- Enquire about eczema (in adults this may make you think of malignant otitis externa, especially if they are diabetic).

Drug History

- Have any antibiotics been used? The patient may have been undertreated or have a resistant strain.
- Are vaccinations up-to-date? (Pneumococcal vaccination in particular)
- Any allergies?

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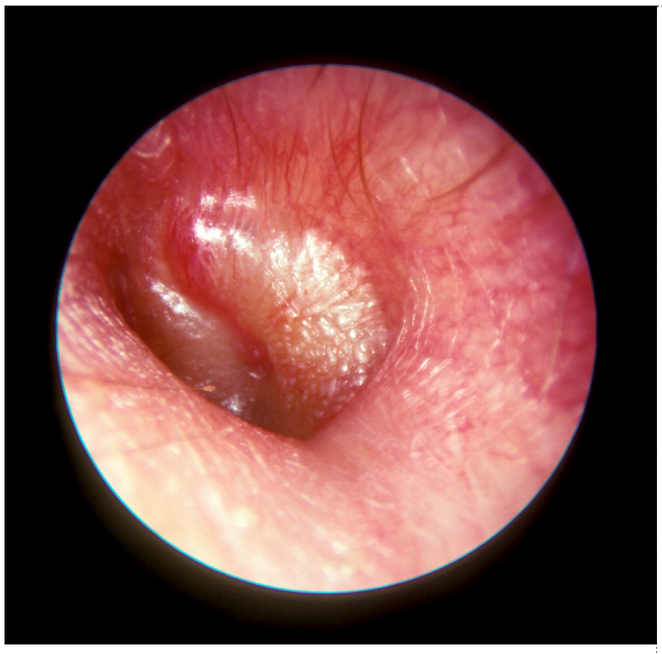

Social History

• Find out about the socio-economic status of the family. How many children are there at home? Is it overcrowded? Any damp housing concerns? Have any of the siblings experienced similar symptoms?

• In older children ask about allergy. Are there pets or smokers in the house? Allergy is a cause of rhinitis, which can predispose to Eustachian tube dysfunction.

The mother explained that the URTI symptoms commenced five days earlier. He had been irritable and tugging at his ear for at least a couple of days but the facial weakness had been present for only 24 hours. There was no otorrhoea. He had three URTIs over the last nine months. He was born at term and had not been admitted to SCBU. Otherwise his past medical history was unremarkable.

How would you approach the examination?

When examining a child it is important to establish good rapport with both the parents and patient[2]. The child needs to sit on the parent's lap with one of the parent's arms across the child's chest and arms and the other across the forehead.

Examination

• **Mastoid.** Look from behind for ear protrusion. Is it red, hot, swollen or fluctuant behind the ear? These are signs of acute mastoiditis.

• **Ear.** Are there any vesicles present? The ears need to be examined with an otoscope with an appropriate paediatric speculum. The pinna should be gently pulled downwards and backwards (upwards, outwards and backwards in adults). Is the ear canal inflamed? Is the tympanic membrane inflamed or bulging? Is there a perforation or discharge?

• **Nose.** Is there rhinorrhoea?

• **Mouth and oropharynx.** Is there tonsillar enlargement? Is there a cleft palate? This increases the likelihood of nasal regurgitation and Eustachian tube dysfunction.

• **Facial Nerve.** Can the child close his eyes? Is there any chemosis? This can be a sign of corneal ulceration due to inability to shut the eyelid. Is the forehead affected (as it should be in a lower motor neurone lesion)? Are there any scars or swelling in the face, e.g. the parotid?

• **Neck.** Check for lymphadenopathy. Note that post-auricular nodes can be mistaken for mastoiditis if tender and inflamed.

On examination the infant had bulging, hyperaemic tympanic membranes bilaterally. The left drum also appeared slightly yellow in colour. There was no perforation or otorrhoea. The child was drooling from the left side of his mouth and was unable to close his left eye but there was no chemosis. There was no evidence of a mastoid abscess.

What would be the next step in management?

The history and examination are highly suggestive of AOM complicated by a left lower motor neurone facial nerve palsy. Further investigations are required to establish the severity, characteristics and prognosis of the infection.

Investigations

Blood tests

• **Full Blood Count and C-reactive protein.** Leukocytosis suggests infection and neutrophilia would suggest a bacterial cause.

• **Urea and Electrolytes.** The child is febrile and possibly dehydrated so renal function should be assessed, especially if intravenous fluids are required.

• **Clotting screen** – should surgical intervention be needed.

Tympanometry (impedance audiometry)

Tympanometry is routinely used in the investigation of glue ear but can be used in acute suppurative otitis media to confirm the presence of fluid or pus in the middle ear objectively prior to surgical intervention. The middle ear fluid in otitis media generates a flat or skewed tympanogram. Ear canal volume can also be measured and will be increased in the case of perforation. This is particularly useful if a perforation cannot be directly visualised.

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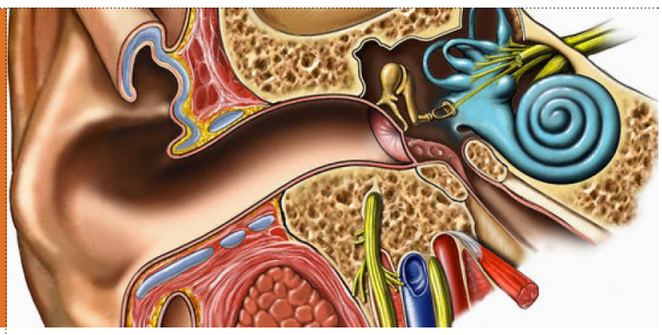

Imaging

In simple AOM imaging is not required. However, in the presence of complications, it may be extremely useful. In this context, imaging allows the visualisation of detailed anatomy and the extent to which infection has disseminated. Of particular concern is intracranial spread. It can also suggest other causes for the signs and symptoms, such as cholesteatoma. Computerised tomography (CT) is the modality of choice but in an infant the radiation can be avoided by using MRI (Magnetic Resonance Imaging).

Electroneuronography (ENG)

This is an objective technique to test the extent of damage to the nerve and gives an indication of prognosis. ENG is not routinely performed and is generally reserved for complicated cases.

In this child there was a neutrophilia and raised CRP. Tympanograms were flat bilaterally with no evidence of perforation. MRI showed increased signal in the middle ear but no spread of intracranial infection. ENG showed no neurophysiological evidence of axonal continuity in the left facial nerve.

How would you manage AOM medically?

Symptomatic

Analgesia should be given, e.g. paracetamol and ibuprofen, which also have both anti-pyretic and anti-inflammatory effects. Intravenous fluids should be given if necessary.

Antibiotics

In uncomplicated AOM the illness is likely to be self-limiting and there is little evidence supporting the efficacy of antibiotics. However, antibiotic administration has some benefit in children under two with bilateral AOM or AOM with otorrhoea[3].

Streptococcus pneumoniae is most commonly implicated (40%), followed by *Haemophilus influenzae* (30%) then *Moraxella catarrhalis* (10%)[4]. Amoxicillin is first line, then co-amoxiclav for beta-lactamase producing organisms and third generation cephalosporins are third line. Topical quinolone drops (ofloxacin) can be used where there is a perforation or patent grommets.

Steroids

Due to the nerve involvement a short course of prednisolone should be prescribed.

Decongestants +/- anti-histamines

These can be given for a week to reduce nasal congestion.

What further management would you consider?

This complicated case of AOM requires urgent review by ENT and Paediatrics. Complicated AOM failing to resolve with antibiotics warrants myringotomy to aid drainage from the middle ear. Swab any discharge to guide antibiotic therapy.

Failure of left eyelid closure leaves the eye at risk of corneal ulceration. This requires regular application of lubricating eye drops/ointment, taping the eye closed overnight and urgent ophthalmology review.

The child underwent myringotomy with bilateral grommet insertion. Over the next few days his temperature, leukocytosis, inflammatory markers and facial weakness improved. Post-operative otorrhoea resolved quickly.

At follow-up 2 months later, his ears were dry and grommets remained patent. At rest there was no facial asymmetry but moderate weakness was noted at the left nasolabial fold when smiling. He was able to maintain full eye closure when sleeping.

ACUTE OTITIS MEDIA

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Discussion

Acute otitis media may become recurrent. If present for 3 months it is reclassified as chronic. If an effusion is present for 3 months it is termed otitis media with effusion (OME) or glue ear. Otitis media may result in various complications (see Table 1).

Local	Regional
Tympanosclerosis	Mastoiditis
TM perforation	Meningitis
Facial nerve palsy	Petrositis (can result in Gradenigo's syndrome)
Hearing loss	Abscesses, e.g. cerebellar, temporal lobe, extradural, subdural
Chronic otitis media	Lateral sinus thromobosis
Ossicular erosion	Citelli's/Bezold's abscesses
Labyrinthitis	Otitic hydrocephalus

Table 1: Complications of Otitis Media.

Acute otitis media may be complicated by facial nerve paralysis for a number of reasons including damage from bacterial toxins or congenital dehiscence of the fallopian canal.

The horizontal part of the facial nerve runs though the middle ear cleft in the bony fallopian canal. In 5-10% of individuals it is dehiscent in this portion, i.e. there is no bony covering. In AOM the oedema caused by the inflammatory response contributes to paralysis of the nerve in this section.

Though this is a rare complication, this case highlights the importance of considering middle ear disease in such a presentation and managing the patient accordingly. Treatment of AOM complicated by facial nerve palsy is probably best treated with antibiotics, myringotomy and steroids. This usually results in resolution and no further surgical exploration is required.

Self-Assessment Best of Five Questions

1. Which of the following is the most common cause of AOM?

a) Streptococcus pneumoniae.
b) Haemophilus influenzae.
c) Moraxella catarrhalis.
d) Respiratory Syncytial Virus (RSV).
e) Pseudomonas aeruginosa.

2. Which of the following is first line treatment for simple AOM in a 5-year-old child?

a) *Oral Amoxicillin.*
b) *Analgesia.*
c) *Nothing.*
d) Gentamicin ear drops.
e) *Ofloxacin ear drops.*

Answers

1. a) The most common causative pathogen is *Streptococcus pneumoniae.* The impact of the introduction of the *Haemophilus influenzae* B vaccine is unclear as it targets the B serotype which is not the only serotype causing otitis media. RSV commonly causes the URTI that predisposes to AOM.

2. b) Antibiotics are generally not advocated in simple AOM. Analgesia should always be given. Gentamicin is ototoxic and the ear drops should be avoided in the presence of a perforated tympanic membrane.

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Authors

Dr. Seema Yalamanchili MA, MBBS
Foundation Year 1 Doctor
email: seema.yalamanchili@doctors.org.uk

Mr. Rishi Talwar BSc, MBBS, MRCS, DOHNS
Specialist Registrar in ENT
email: r.talwar@nhs.net

Mrs. Helen Caulfield MBBS, FRCS(ORL-HNS)
Consultant ENT Surgeon
email: Helen.Caulfield@royalfree.nhs.uk

Dr. Seema Yalamanchili and Mr. Rishi Talwar
are joint first authors

INVESTIGATING DEAFNESS

Kathryn Lucy Hage and Oliver J Corrado

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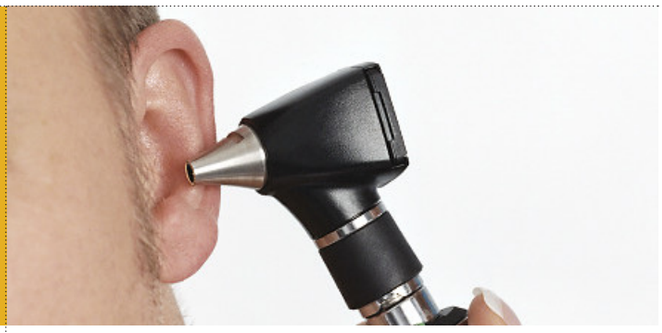

Abstract

This case-based discussion focuses on the investigation of a patient presenting with deafness in a Primary Care setting and highlights the most important steps of the assessment process with the intention of guiding Foundation Doctors in their patient management.

Case History

Mrs AB, a 30-year-old lady attends her GP surgery with symptoms of progressive hearing loss in her right ear.

What further information do you need from the patient?

Deafness and hearing loss can occur as a result of many different pathologies[1].

It is essential to take a thorough history as follows:

1. History of Presenting Complaint

- When did the hearing loss start?
- Was the onset sudden or gradual?
- Are the symptoms unilateral or bilateral?
- Is this the first episode or has it occurred before?
- Are there any associated symptoms, in particular:
  - pain
  - ear Discharge
  - tinnitus
  - loss of balance
  - cranial nerve involvement
  - nose or throat symptoms
  - systemic symptoms such as weight loss.

2. Past Medical History

- Prior or current history of trauma or head injury?
- Episodes of labyrinthitis, Meniere's Disease or tinnitus?
- Hearing problems as a child including ear infections?
- Prior ear surgery?

3. Drug History

Many medications are known to cause or exacerbate symptoms of hearing loss and tinnitus. Drugs which are commonly used and ototoxic include aspirin, some NSAIDs, aminoglycosides, loop diuretics, erythromycin and quinine[2].

4. Social History

- Is there any history of exposure to excessive noise, including that due to occupations or other daily activities?
- Any psychological impact as a result of symptoms?
  - If symptoms are severe it may be appropriate to utilise the type talk facility/ sign language interpreters or RNID services.

5. Family History

- Otosclerosis?
- Autoimmune disorders?
- Acoustic Neuroma or Neurofibromatosis?

On further questioning Mrs AB stated that she had frequently been bothered by "ringing and buzzing in her ears" (tinnitus) and found it particularly difficult to hear while eating and during quiet conversation. She had no previous history of ENT problems and was on no regular medication. She worked as a Classroom Assistant, but could not recall exposure to excessive noise levels. She had recently married and was trying for a baby. Mrs AB mentioned that her maternal grandmother became deaf in both ears before she was 50 years old and had bilateral hearing aids, she asks whether this could be connected. How would you proceed with the consultation?

Even a basic examination in Primary Care can provide useful supportive evidence. This should include:

1. A full ENT examination with particular attention to the examination of the ear:

- Always begin with the stronger ear and ask about the degree of any associated pain.
- Start by examining the external auditory meatus, including the pinna and check for any pre-auricular abnormalities (skin tags, scarring, etc).
- Pull the pinna upwards and backwards and insert the otoscope by visualising the tympanic membrane and following the handle of the malleus up to the lateral process[3].

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2. Neurological assessment including examination for facial nerve signs. Assessment of the vestibulocochlear nerve can be done as follows:

• Begin with a gross assessment of the patients hearing by asking the patient to close their eyes and whisper a number in to each ear at a distance of 60cm. The accuracy of this method can be improved by rubbing your finger over the tragus of the non-test ear. If the patient is accurate more than 50% of the time their hearing is better than 30dB.

• Tuning fork tests aim to determine between conductive/sensorineural or mixed pattern hearing loss and has strong significance when considering a possible aetiology.

Rinne test: Hold a 512Hz tuning fork first in front of the ear canal (sound 1) and then gently place it on to the mastoid process (sound 2). In a normal patient sound 1 is loudest as air conduction is greater than bone conduction (a positive test result). In a patient with a conductive hearing deficit (representing disease in the external or middle ear) sound 2 is loudest (a negative test result)[3].

Weber test: Hold a 512Hz tuning fork and place it in the midline of the forehead. In a normal patient the sound is heard equally on both sides. In a patient with conductive deafness the sound lateralises to the affected ear as bone conduction is preserved. In a patient with sensorineural involvement (disease of the inner ear and neural pathways) the sound is heard best in the unaffected ear[3].

On examination, Mrs AB was only able to hear sounds at 60cm when they were spoken to the right ear but could hear whispered sounds at 60cm in her left ear. The tympanic membrane appeared normal. Rinne's test was positive on the left but negative on the right, Weber's test lateralised to the right.

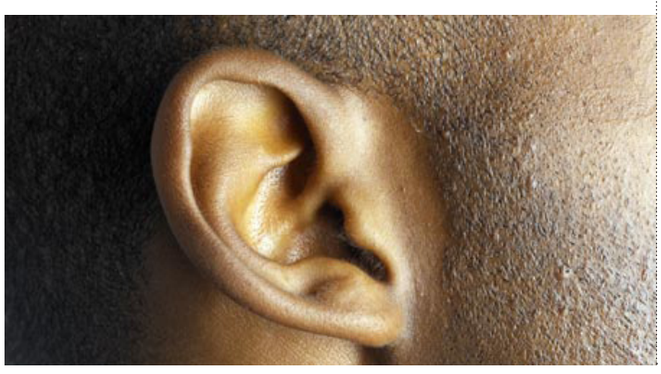
What pattern of deafness does this represent?

This represents a right-sided conductive hearing loss.

What would you do next?

In view of the above findings, in conjunction with her young age and positive family history, further investigation is warranted. This will in all likelihood necessitate referral to Secondary Care[4].

A useful adjunct to the final diagnosis is a pure tone audiogram, which can be organised directly by the referring GP or done on arrival at the ENT department.



Audiograms

Pure tone audiograms are obtained by testing each ear individually for air and bone conduction by playing sounds of varying frequencies through headphones. If a more sensitive approach is required, this can be done by playing words to the patient. The collected data is plotted for both ears as an audiogram (Figures 1,3). The results for Mrs AB (Figure 2) highlight a right sided conductive hearing loss.

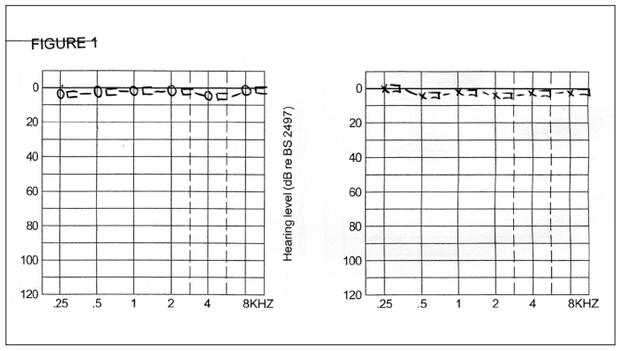


Figure 1

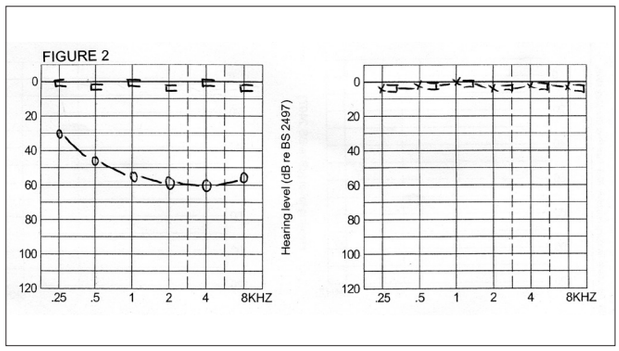


Figure 2

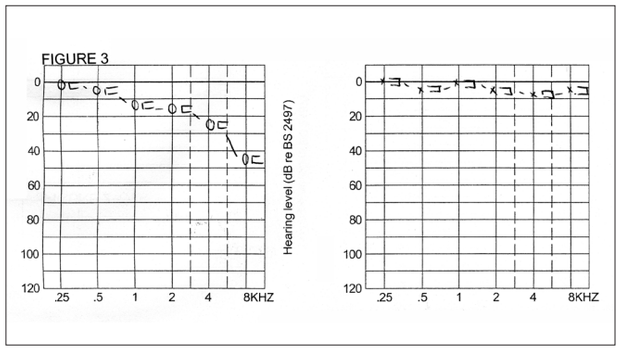


Figure 3

Impedance Audiometry

This is a more sophisticated test that records middle ear pressures by measuring tympanic membrane compliance in response to auditory stimuli. It has a specific role in diagnosing middle ear effusions and some types of sensorineural disease[3].

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Electric response audiometry (ERA)

Skin electrodes measure an evoked potential in the eighth nerve, brain stem or auditory cortex. This is the diagnostic test of choice in children up to 6 months of age[3].

Hearing Investigations in Children

As outlined above, ERA can be effective when treating infants. For those aged 6 to 18 months, a distraction test assessment is carried out by waving an object such as a rattle in front of each ear and observing if the child turns towards the rattle sound. From around 3 years of age, headphones can be used to make more sensitive assessments[3].

What do you think is the cause of the patient's symptoms?

Mrs AB's symptoms and investigations would support a provisional diagnosis of otosclerosis.

Discussion

The aetiology of deafness can be divided in to conductive, sensorineural and non-organic subgroups.

Conductive deafness

This is the most frequently encountered type of deafness, common causes include:

- Ear wax.
- Foreign bodies.
- Otitis media +/- effusion.
- Otitis externa.
- Otosclerosis.

Sensorineural deafness

Can be inherited or acquired. Inherited deafness has an incidence of 1-2 per 1000 live births and is predominantly transmitted in an autosomal recessive pattern. It is a feature of Alport syndrome (deafness and renal failure), Pendred's syndrome (deafness and hypothyroidism) and over a dozen rarer conditions.

Acquired sensorineural deafness can be caused by a wide range of conditions. It is frequently encountered in elderly patients due to the high incidence of presbyacusis. It presents with a progressive bilateral sensorineural deafness, which worsens with age and is the most common reason for hearing aid use. Rarer causes include:

- Neoplastic disease
  - Acoustic neuroma: slow growing tumour of the cerebello-pontine angle.
- Infections
  - Cytomegalovirus, rubella, syphilis, meningitis and herpes zoster
  - Acute labyrinthitis secondary to viral infection.
- Ototoxic medications
- Idiopathic
  - CVA.
- Autoimmune and metabolic diseases
  - Diabetes, hypothyroidism, Paget's disease, vasculitis, rheumatoid arthritis, systemic lupus erythematosis.

Any sudden onset of sensorineural hearing loss is a medical emergency. This includes deafness that has developed within 72 hours. These patients must be referred for immediate secondary care assessment[4].

It is important to remember that deafness may present due to a non-organic cause. These presentations frequently relate to compensation claims for potential occupational hearing loss. The most reliable diagnostic method is using brain stem audiometry as described above[4].

Mrs AB has a diagnosis of otosclerosis, which is a condition of disordered bone remodelling, involving the stapes bone. This leads to reduced movement of the stapes and consequently decreased sound production resulting in a conductive deafness. In time, the bone may become 'fixed' causing profound hearing loss. Occasionally the diseased bone may extend into the cochlea causing a sensorineural hearing loss[5].

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The disease commonly affects around 1-2% of people in the UK. It is twice as prevalent in women. There appears to be a strong genetic component, with around 60% of patients having a first degree relative with otosclerosis[4]. Mrs AB mentions she is trying for a baby, what do you need to tell her about her condition?

Otosclerosis commonly deteriorates during pregnancy. Many women often notice symptoms for the first time while they are pregnant[4]. Otosclerosis is likely to be jointly managed between primary and secondary care, but requires referral for initial diagnosis and exclusion of other diseases. This differentiation can involve more sensitive audiometry and possibly MRI, as it is important to exclude an acoustic neuroma in patients with unilateral hearing loss and tinnitus[4].

First line management is a trial of hearing aids, however these are often poorly tolerated, particularly in younger patients[5].

As the disease progresses, a stapedectomy is the operation of choice. This involves removal of the upper part of the stapes under microscope guidance. A small hole is then made through the footplate of the stapes and a prosthetic piston is inserted. This piston is attached to the incus permitting the transmission of sound waves. In over 90% of cases this produces full resolution of hearing, however complications include:

- Dizziness
  - Usually transient and can be reduced by rest and avoiding sudden head movements.
- Endotympanic leak
  - This risk is decreased by inserting a small piece of vein or tissue over the hole in the footplate.
- Facial nerve damage
  - If the facial nerve is abnormally positioned and overhanging the operation site, the operation may have to be abandoned.
- Unchanged hearing or further hearing loss
  - Occurs in 1-2% of patients.

Summary

Deafness is a frequently encountered symptom in General Practice that can present at any age[4]. It can be investigated using the methods mentioned above, with particular value gained from tuning fork testing and audiogram interpretation[1].

Deafness and hearing loss can be debilitating for patients. It is therefore important to consider the most holistic approach to investigations, with the utilisation of external agencies and support services in the assessment process.

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Useful Websites

1. Extensive resource containing health professionals section, patient information leaflets and links to local organisations.
http://www.rnid.org.uk/
2. Useful on-line translation guide including tutorials on basic sign language.
http://www.britishsignlanguage.com/
3. Explains how to set up a text-phone link with downloadable software.
http://www.textrelay.org

Authors

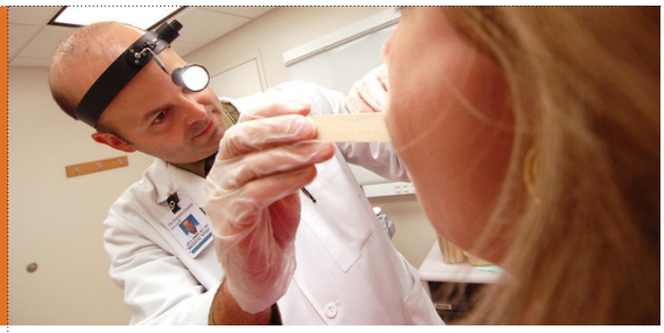
Dr Kathryn Lucy Hage MBChB
SHO Rheumatology

Dr Oliver J Corrado MBBS, FRCP (Lond)
Consultant Physician
The General Infirmary
Leeds

PHARYNGEAL POUCH/ZENKER DIVERTICULUM

Grace Dimbleby and Osama Hamarneh

Case-based discussion – pharyngeal pouch/zenker diverticulum. Case Based Discussion.



Abstract

This case based discussion focuses on the presentation and management options of a pharyngeal pouch in a 72-year-old woman.

Case History

Your patient is referred to the department of Otolaryngology with difficulty in swallowing and choking on food.

- What is the likely cause of her symptoms?
- What would you like to do next?

A detailed history from the patient

1) Presenting problem:

- When did the symptoms first start?
- Has she had any investigations previous to this presentation?
- Does she have a sensation of food sticking?
- Where does the food tend to stick? Is it painful to swallow (odynophagia)?
- Does she have any regurgitation of food?
- Has she had any weight loss?
- Does she suffer from symptoms of reflux, halitosis, or recurrent chest infections?
- Has she or a relative/friend noticed any changes in her voice?

2) Past medical history:

- Hiatus hernia.
- Oesophageal/gastric/duodenal ulceration.
- Achalasia.
- Previous surgery/radiotherapy for ear/nose/throat problems.

3) Drug History/Allergies:

- Is she taking any NSAIDs? – risk of ulceration/oesophagitis.
- Has she been prescribed a PPI? – masking symptoms of reflux.

4) Social History:

- Smoking.
- Alcohol consumption.
- Eating habits.
- Affect of symptoms on work/social life.

The patient reveals that she has had trouble swallowing food and liquids for the past three months. She describes this as food getting stuck in her "gullet". She has also complained of coughing and choking after meals. She is unaware of any reflux and does not report any weight loss. She has a past medical history of snoring managed conservatively. She also has a history of ischaemic heart disease for which she takes aspirin, has no known allergies and smokes 2-3 cigarettes per day. She is otherwise well.

What do you think is the cause of her symptoms?

The symptoms of dysphagia, food sticking, choking after meals and regurgitation, are a classic presentation of a pharyngeal pouch or Zenker's diverticulum[1,2]. Choking is specifically related to dysphagia occurring in the oropharyngeal phase of swallowing; although the aetiology of a pharyngeal pouch is undetermined, it is suggested that an abnormality of the cricopharyngeus muscle results in a uncoordinated swallowing mechanism resulting in the risk of choking and aspiration[2,3].

Psychogenic: globus (sensation of a mass in the throat), anxiety, depression
Malignancy: laryngeal/oesophageal/thyroid, mediastinal mass
Endocrine: Cushing's syndrome, hypo/hyperthyroid
Neurogenic: CVA, Parkinson's disease, multiple sclerosis, dementia, myasthenia gravis
Infective: viral/bacterial/fungal infections of the pharynx and upper oesophagus
Inflammatory: pharyngitis/laryngitis/tonsillitis, lymphadenitis
Iatrogenic: CNS depressants, antipyschotics, corticosteroids, head & neck surgery
Benign obstruction: strictures, tumours, foreign body, mediastinal mass, achalasia, oesophageal webs, oesophageal spasm, pharyngeal pouch, prominent cervical osteophytes

Table 1: Differential diagnosis of dysphagia[3].

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What would you do next?

Full Examination of the patient

1) ENT examination. For completeness:
• **Ear** – Examine the external auditory meatus, canal and the tympanic membrane via otoscopy
• **Nose** – Inspect for patency of vestibules, mucosa/septum/inferior and middle turbinates via anterior rhinoscopy

Focused examination:

• **Throat** – Inspect the lips and perioral region, mouth including buccal mucosa, gums, teeth, floor of mouth, bimanually palpate the floor of the mouth.
• **Neck** – Inspect for lumps/skin changes/scars, palpate for lumps/swellings/tenderness/swallow.

2) Neurological examination
• Cranial nerves – V and VII to XII.

3) Flexible nasendoscopy (FNE)
• To exclude any upper aerodigestive epithelial pathology and visualise the oropharynx, hypopharynx and larynx – may show pooling of saliva in the piriform fossae.

Your patient appears well, with no excessive weight loss. Her ENT examination was normal, all cranial nerves were intact and FNE showed no abnormalities.

What would you do next?

Investigations

1) Blood tests: (to exclude other medical conditions)
• Full blood count and CRP.
• Urea and electrolytes.
• Thyroid function tests.

2) Barium swallow
• Will demonstrate the position (commonly posterior, and just superior to the cricopharyngeus muscle) and size of a pharyngeal pouch – usually >2cm in diameter in symptomatic patients[1].

Blood tests were normal. The barium swallow showed a small posterior pouch, excessive reflux and oesophageal incoordination.

What would your management include?

• Explain to the patient the cause of her symptoms and reassure her that it can be managed effectively in the majority of cases.
• Discuss the options of treatment – depends on patient preference (extent of symptoms), size of pouch, and patient co-morbidities:
- Conservative – mainly asymptomatic patients/small pouches <1cm[4,5].
- Medical – trial of PPI and review; can relieve cricopharyngeal spasm and lessen symptoms of reflux.

Explain that these options do not reduce the risk of aspiration and pneumonia which carry a high morbidity and mortality.

• Surgical – includes the following options:
- Endoscopic stapling – direct pharyngoscopy followed by stapling of the pouch using a bivalve endoscope.
- Open (diverticulectomy with cricopharyngeal myotomy) – left neck incision made at the level of the cricoid cartilage – cricopharyngeal muscle divided and pouch excised, primarily closed with suction drain in place[1].

An endoscopic approach is minimally invasive therefore allows a shorter duration of anaesthetic, quicker resumption of oral feeding, reduced risk of infection and subsequently a shorter hospital stay[5]. However, it does carry the risk of oesophageal perforation. It is of limited use in those with reduced access due to dentition and/or jaw opening and patients with kyphosis. In these cases, an open approach may be necessary, which is not without its risks; recurrent laryngeal nerve palsy, surgical emphysema, mediastinitis, cutaneous fistula, and may require the use of nasogastric tube feeding post-operatively. A gastrografin swallow study may be performed to exclude a leak before oral feeding is commenced[6]. In both approaches it is important to communicate the risk of recurrence.

The patient reports that due to her coughing episodes following meals, she is reluctant to go out to eat with friends as she feels embarrassed. Following discussion of the above options, she is put on the list for endoscopic stapling of the pharyngeal pouch, and is also started on lanzoprazole in the meantime to help relieve symptoms.

Follow up and further management

Your patient is followed-up six weeks later and the majority of her symptoms have improved, although she still suffers from the occasional sensation of food sticking in her throat (globus sensation) but she is managing a normal diet. Despite the risk of recurrence/failure following endoscopic surgery, the majority of patients report complete relief of symptoms post-operatively and the patient satisfaction rate is high[6].

PHARYNGEAL POUCH/ZENKER DIVERTICULUM

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Discussion

A pharyngeal pouch or Zenker's diverticulum is rare and occurs more commonly in men over the age of seventy[2]. A pharyngeal pouch is a protrusion of the pharyngeal mucosa through a weakening between the cricopharyngeus and thyropharyngeus muscles at the top of the oesophagus[7]. It is hypothesised that this herniation occurs due to a poorly coordinated swallowing mechanism, which leads to increased pressure on the mucosa. Since the weakest portion of this area is located posteriorly, the herniation commonly occurs in this direction. It is unclear whether or not there is an anatomical predisposition to this condition.

The collection of food in the pouch, results in patients typically presenting with regurgitation of undigested food, halitosis, difficulty swallowing (dysphagia), chronic cough, recurrent chest infections and less commonly, changes in their voice. The patient may commonly report a history of hiatus hernia, peptic ulceration, oesophageal spasm or achalasia. Mild to moderate weight loss can occur. Due to the complications of aspiration and pneumonia, a pharyngeal pouch should be operated on in those who are fit for surgery[1].

The mortality rate of surgery has been recorded as 1.2%, and complications occurring in less than 10%. These figures are similar for both endoscopic and open repair[1]. The major advantages of the former approach are a faster recovery time, and the use for those who are not fit for open surgery and is therefore preferred by the majority of otolaryngologists[8,9]. Follow-up of patients is for review of symptom relief and wound repair, and long term follow-up is not routine. However, there is some controversy regarding this, as although extremely rare, there have been cases of squamous cell carcinomas developing in recurrent pharyngeal diverticulum. This may be an argument for long term follow-up or management with an open excision[10].

Self Assessment-Best of Five

1. An 82-year-old man comes to your clinic with a 6-month history of regurgitating food after meals and mild weight loss. He has a history of GORD for which he takes lanzoprazole, but is otherwise well. You suspect that he has a pharyngeal pouch. Which of the following is not a common sign/symptom of this condition?

a) Recurrent chest infections.
b) Halitosis.
c) Voice changes.
d) Neck swelling.
e) Dysphagia.

2. Which of the following is not a potential benefit of the endoscopic approach?

a) Reduced operative time.
b) Ability to manage the smallest sized pouches.
c) Earlier resumption of oral feeding.
d) Reduced hospital stay.
e) Allows for safe re-operation if necessary.

Answers

1. d) Very rarely, the pouch (a protrusion of oesophageal mucosa between the cricopharyngeus and the inferior pharyngeal constrictor muscles) reaches a size that can be felt as a neck swelling on external examination. The retention of food within the pouch, leads to symptoms of dysphagia, halitosis, regurgitation of food and the sensation of food sticking. Hoarseness of voice occurs due to pressure on the recurrent laryngeal nerve.

Due to the poor coordination of the swallowing process, thought to be the cause of the diverticulum, it is easy for food to be aspirated, leading to a chronic cough and recurrent chest infections.

2. b) The endoscopic approach is not suitable for pouches less than 3cm long since the stapler blade is too long for the common wall. The larger sized pouches can be managed by repeating the division and stapling process until the bottom of the pouch is reached.

The anaesthetic time is reduced from up to 90 minutes for the open approach to approximately 25 minutes when managed endoscopically[1]. The hospital stay is shorter as patients avoid the morbidity of an open excision.

In the case that a repeat operation is required, the endoscopic approach avoids the complication of a re-incision.

Figures

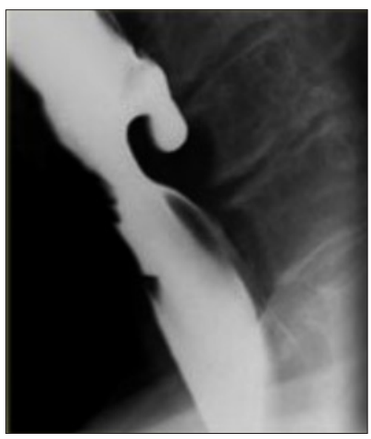

Figure 1: barium swallow demonstrating a posterior. References figure 1 = Emedicine Specialities. Zenker diverticulum. http://emedicine.medscape.com/article/194265-overview.

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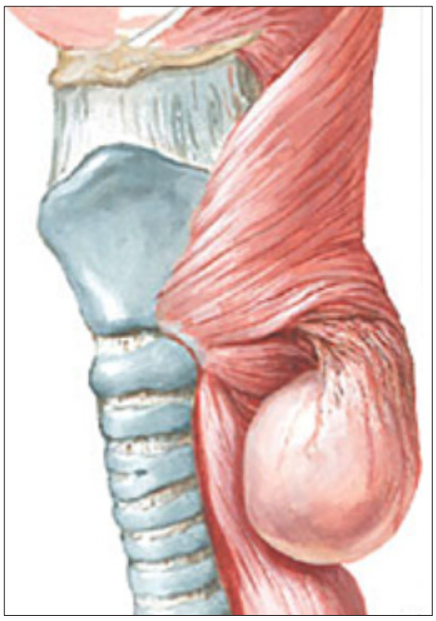


Figure 2: protrusion of oesophageal mucosa between the cricopharyngeus muscle and the thyropharyngeus muscles forming a pouch.

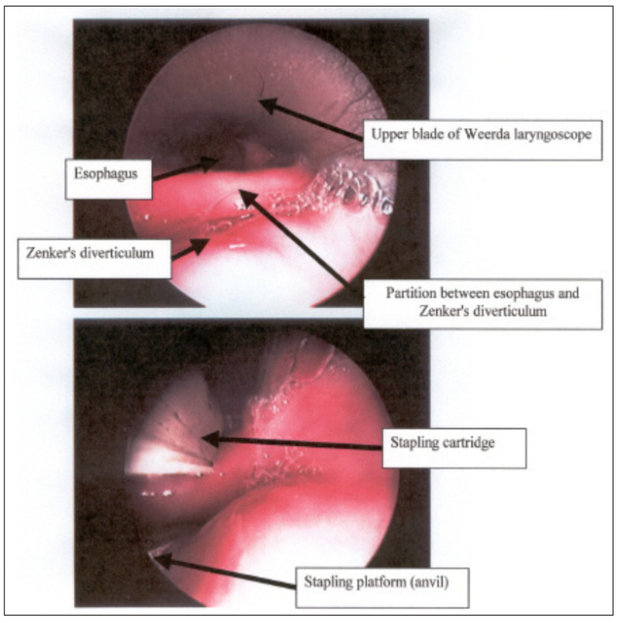
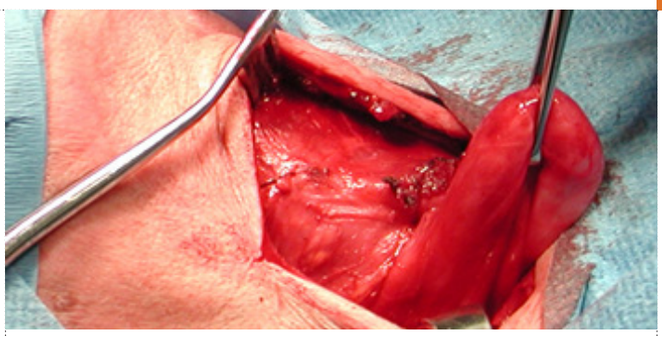


Figure 3: endoscopic view of the oesophagus and pharyngeal pouch. One jaw of the stapler is inserted into the pouch and one into the oesophagus. The stapler is then locked across the common septum and fired.



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Authors

Dr G Dimbleby MBChB
Foundation Year 1 ENT
Royal Wolverhampton Hospital
Wednesfield road
Wolverhampton, WV10 0QP
email: gdimbleby@doctors.org.uk

Dr Osama Hamarneh SpR ENT
New Cross Hospital
Wolverhampton
email: ohamarneh@gmail.com

ACUTE OTITIS MEDIA

M Wasik, V Anthony, S Berry and CN Marnane

Acute Otitis Media - Figure[1].
Case Based Discussion.

Abstract

This case-based discussion looks at the presentation of acute otitis media (AOM) in a young child. It will focus on the key points to look out for when taking a history and performing an examination, potential differential diagnoses and the management of the patient.

Case History

A 3-year-old boy is brought by his parents to see the family general practitioner. Over the last few days, they have noticed that he has not been his usual boisterous self. He has not been able to sleep well at night and has been feeling very tired during the day. His parents have also noticed that he is tugging at his right ear all the time.

What are the differential diagnoses of otalgia in this patient?

This presentation can be typical of a middle ear infection in a young child. The paucity of specific symptoms can make it difficult to establish a diagnosis. It is therefore vital to take a comprehensive history from the patient's parents and more importantly, to always examine the ears[2].

History

Presenting complaint

1) The most common symptom of AOM is otalgia. In a child who is unable to communicate this pain, it may manifest itself by constant rubbing or tugging of the infected ear.
2) Occasionally, there may also be otorrhoea (discharge from the ear), which is usually accompanied by an instant resolution of the ear pain. This occurs because the accumulation of exudate and pus behind the tympanic membrane results in a perforation, thus releasing the pressure exerted on the middle ear.
3) There may be associated nasal symptoms, such as obstruction and rhinorrhoea. At night when the child is sleeping, there may be noticeable snoring or episodes of apnoea.
4) Systemic symptoms are very common in middle ear infections. Look out for any fever, irritability, lethargy, anorexia or vomiting.

Past medical history

1) Prior to the onset of AOM, the child may have had an upper respiratory tract infection. Symptoms like a cough or rhinorrhoea a couple of weeks before the current presentation should be enquired about.
2) There are a number of environmental risk factors associated with AOM. These include passive smoking, exposure to other children, particularly at day care centres, bottle-feeding and low socio-economic status.

The parents inform you that there has been no discharge from the ear and no nasal symptoms. He attends a local nursery school three times a week. Three weeks ago, he had been sneezing and coughing, which they assumed he had picked up from the other children at the nursery. Neither parent smokes at home, and both have full-time jobs.

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Examination

A general examination should be performed to look for signs of systemic illness, specifically heart rate and temperature.

The diagnosis of AOM will be made using an otoscope and examining the tympanic membrane.

In AOM, the eardrum will be bulging into the external auditory canal. As a result, the normal anatomical landmarks will not be clearly visible (handle of malleus) and the normal light reflex will be lost. The eardrum itself will have changed colour to either red or yellow.

There may be a perforation in the tympanic membrane, and pus may be seen in the external auditory canal. A swab of the pus may be taken and sent for microscopy, culture and sensitivities.

Except for measuring the patient's temperature, which shows a fever of 39.0°C, a general examination of this patient is otherwise normal. With the aid of an otoscope, the tympanic membrane appears red and bulging, and the handle of malleus cannot be distinguished.

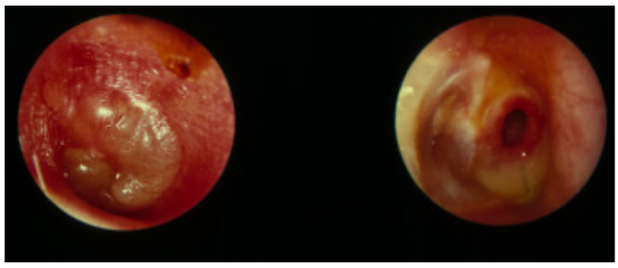

Figure 2: The appearance of tympanic membranes in patients with AOM. The left image shows an inflamed, bulging eardrum and the outline of the handle of malleus is lost. The right image shows a central perforation, which allows the egress of middle ear exudate.

Management of AOM

Symptom relief

The majority of patients with AOM will resolve spontaneously and no further treatment is required. Symptomatic relief of pain and fever can be achieved with paracetamol or ibuprofen.

Antibiotics

The use of antibiotics in AOM has been a controversial topic. While they may hasten the recovery of the patient, they may not always be required for resolution of the infection, and there is always the threat of antibiotic resistance in the foreseeable future.

Clinical recommendations from the Scottish Intercollegiate Guidelines Network[3] suggest a "wait and see" approach should be employed. Parents should be reassured that AOM does not require immediate antimicrobial therapy. If the patient's symptoms have not resolved within 48–72 hours after the first consultation, then a five day course of amoxicillin or erythromycin may be prescribed.

In certain circumstances, it may be appropriate to prescribe a course of antibiotics from the initial consultation. These groups of patients include children under the age of 18 months, those with otorrhoea, those with bilateral AOM, those with systemic signs (fever and vomiting) or those where the localised inflammation of the tympanic membrane is particularly severe.

If the infection persists beyond seven days despite initial antibiotic therapy, then a broad-spectrum antibiotic such as co-amoxiclav may be prescribed.

Due to the patient's fever, the GP prescribed a 5-day course of oral amoxicillin. He advised the parents to keep the ear dry. When the patient was due a bath or shower, a piece of cotton wool coated with Vaseline would provide an appropriate waterproof barrier. After a few days, the otalgia had settled.

Discussion

AOM refers to inflammation of the middle ear. The cause of infection in the majority of patients is viral, the most common culprits being respiratory syncytial virus, adenovirus and influenza A virus. *Streptococcus pneumoniae, Haemophilus influenzae* type B and *Moraxella catarrhalis* are the main bacterial causative agents of AOM.

It is most commonly seen in young children before the age of five. This is primarily due to the anatomy of the Eustachian tube, which allows any infection within the nasal cavity to tract up into the middle ear. It is more common during the winter months, when infections of the upper respiratory tract are especially prevalent.

The stages of acute otitis media are summarised in Figure 3. Eustachian tube obstruction due to an upper respiratory tract infection results in a build-up of negative pressure within the middle ear, causing retraction of the eardrum. Capillary dilatation and goblet cell hyperplasia induces the production of an inflammatory exudate. The accumulation of fluid within the middle ear increases the pressure resulting in a bulging tympanic membrane, and the patient experiences severe otalgia. The pressure on the eardrum may create a perforation within it and allow the fluid and pus to drain out of the ear, giving instant pain relief to the patient. In a minority of patients, the perforation and otorrhoea may become chronic – this is termed chronic suppurative otitis media.

ACUTE OTITIS MEDIA

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Stages Of Acute Otitis Media

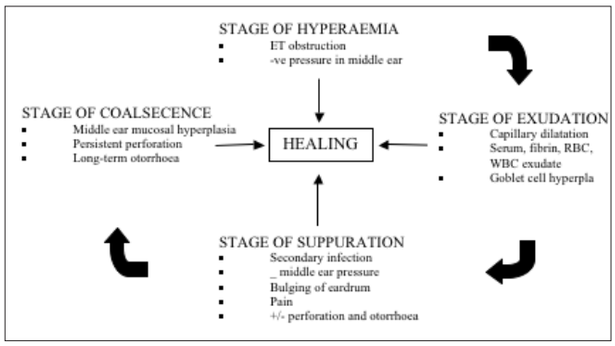


Figure 3: The progression of AOM from initial Eustachian tube (ET) obstruction, due to an upper respiratory tract infection, to the formation of an exudate, eventually accumulation of fluid behind the eardrum and finally, resolution or continued middle ear discharge and infection[4].

Complications of AOM include acute mastoiditis, labyrinthitis and facial paralysis. Further extension of the primary middle ear infection may result in intracranial complications, such as meningitis, brain abscesses and lateral sinus thrombosis.

A child who has suffered numerous episodes of AOM may require a referral to an ENT specialist. It is important to assess the impact these recurrent episodes have on the child's speech and language development. Further investigation, which includes anterior rhinoscopy (looking for abnormalities, anatomical or otherwise) and hearing tests (tympanometry and pure-tone audiometry) will allow the ENT surgeons to decide the most appropriate treatment.

There are a few modes of management that can be considered:

- A conservative approach where each episode of AOM is treated as it comes. The majority of children will eventually grow out of the problem.
- A 6-week course of amoxicillin taken once a day can be trialled.
- Surgical options include the insertion of grommet tubes to prevent the build-up of fluid behind the tympanic membrane. Removing the adenoids may be considered if they are enlarged or are contributing to Eustachian tube dysfunction.

Self-assessment Best of Five Questions

1. A 5-year-old boy complains of left otalgia for three days. On examination, he has a temperature of 38.5°C and is tachycardic at 120bpm. The left ear is more noticeably protruding than the right. He is very tender behind his ear. The external auditory canal is inflamed and oedematous with mucopus present. The tympanic membrane could not be visualised. What is the diagnosis?

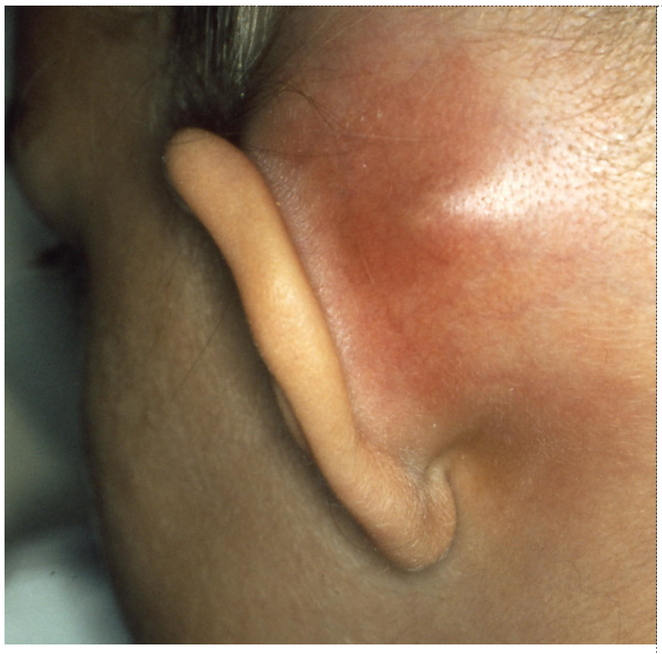
a) lateral sinus thrombosis
b) mastoiditis
c) chronic suppurative otitis media
d) meningitis
e) malignant otitis externa.

2. A4-year-old girl presents with a one day history of right otalgia. On examination, the tympanic membrane is red and bulging. She is allergic to penicillin. What would be the most appropriate initial management?

a) Simple analgesia only
b) Oral amoxicillin
c) Oral erythromycin
d) Oral co-amoxiclav
e) Intravenous co-amoxiclav.

ACUTE OTITIS MEDIA

M Wasik, V Anthony, S Berry and CN Marnane



Answers

1. b) mastoiditis

This is a classical presentation of middle ear infection extending into the mastoid air cells. This patient requires prompt hospital admission and treatment with intravenous antibiotics (co-amoxiclav).

2) a) simple analgesia only

According to the SIGN guidelines, this patient does not warrant an initial oral antibiotic therapy. A prescription for oral erythromycin may be given to the parents, which is to be utilised if the patient's symptoms have persisted for two or more days after the first consultation.

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Authors

Martin Wasik MBBCh, BSc
Foundation Year 1 House Officer in ENT
Singleton Hospital
Sketty Lane
Sketty
Swansea
SA2 8QA

Victoria Anthony MBBCh
Foundation Year 1 House Officer in ENT
Singleton Hospital
Sketty Lane
Sketty
Swansea
SA2 8QA

Sandeep Berry MBBS, MS, DLO, MRCS, FRCS(ORL-HNS)
Specialist Registrar
Singleton Hospital
Sketty Lane
Sketty
Swansea
SA2 8QA

Conor Marnane MBBCh, FRCS (ORL-HNS)
Consultant Otolaryngologist
Singleton Hospital
Sketty Lane
Sketty
Swansea
SA2 8QA

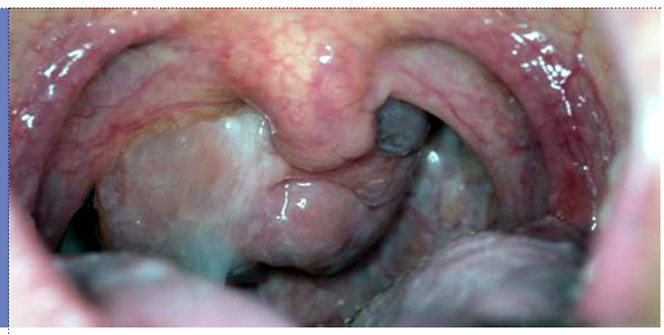
Correspondence

Martin Wasik MBBCh, BSc
Foundation Year 1 House Officer in ENT
Singleton Hospital
Sketty Lane
Sketty
Swansea
email: martin.wasik@"wales.nhs.uk

FLEXIBLE NASENDOSCOPY

Anna Melissa Harrison and Karl Frederick Braekkan Payne

Flexible Nasendoscopy.
Practical Procedures.



Abstract

We present a case history of a patient presenting with a sore throat and odynophagia, who underwent a flexible nasendoscopy to diagnose epiglottitis. The practical procedure of flexible nasendoscopy is reviewed; focusing on explaining the procedure and gaining consent, along with the indications, contraindications and complications of nasendoscopy.

Case History

A 32-year-old male presented via A&E, to the ENT department with a 24-hour history of a severe sore throat, odynophagia and total dysphagia. His past medical history was unremarkable, apart from not having received a Hib vaccine. The patient appeared unwell, toxic and drooling with a hot potato like voice. On examination the mouth was clear however nasendoscopy revealed a cherry red epiglottis. The patient was diagnosed with epiglottitis and admitted to HDU to receive full resuscitation and IV antibiotics, as per trust protocol. He recovered well and received serial daily nasendoscopy to monitor his progress, and was discharged 5 days later.

Discussion

Symptoms of adult epiglottitis include: sore throat (anterior neck specifically), odynophagia, dysphagia, a muffled voice and a preceding URTI.

The patient may appear unwell; with drooling, pyrexia and signs of airway obstruction (stridor) with respiratory distress.

Most often cultures will isolate *Haemophilus influenzae* as the causative organism, but other non-infective causes should be considered[1] (Table 1). For patients without signs of airway compromise, treatment involves intravenous antibiotics, usually a third generation cephalosporin, such as Ceftriaxone. Intubation or a tracheostomy will be required in the patient is unable to maintain an airway.

Causative Organisms	Non-infective Causes
Haemophilus influenzae	Thermal injury (smoking)
Haemophilus parainfluenzae	Caustic insult
Streptococcus pneumoniae	Foreign body ingestion
Group A Streptococcus	Chemo-radiotherapy

Table 1: Most common causes.

Flexible Nasendoscopy:

Indications/Contraindications

Flexible nasendoscopy is performed to allow visualisation of the nasal passage, nasopharynx, base of the tongue, supraglottic, glottis and hypopharynx. It is a quick and minimally invasive procedure, and is beneficial because of its ability to be performed in the clinic or ward treatment room environment by a foundation year doctor. Indications for this procedure in an emergency include: investigation of stridor or hoarseness, foreign body visualisation, investigation of epistaxis and visualising the upper airway in patients presenting with a neck abscess (Table 2).

Viewing upper aerodigestive tract: investigate stridor, hoarseness
Persistent adult glue ear: to rule out nasopharyngeal tumour
Foreign body: visualisation
Epistaxis: to visualise bleeding

Table 2: Indications for flexible nasendoscopy.

Contraindications to flexible nasendoscopy are few. However, recent nasal/laryngeal surgery, or a suspected base of skull fracture would warrant caution. It should also be mentioned that care should be taken with the adult epiglottitis patient. Irritation of the epiglottis with the tip of the scope or induction of gagging, may rapidly aggravate airway problems.

Explaining the procedure and gaining consent

Start as always by washing your hands and introducing yourself to the patient. Explain that a nasendoscope is a thin flexible endoscope that is passed along the base of the nose to the back and then down into the top of the throat. It allows visualisation of the throat and vocal cords. Explain that it can be an uncomfortable procedure, but should not cause any pain. Some people may sneeze, get watery eyes and occasionally gag. It is important to reassure patients that coughing/sneezing, etc. are all fine to do during the procedure even with the scope in place. If you choose to use a local anaesthetic spray, inform them that the spray will taste unpleasant and will cause an unusual sensation in the nose and throat. Take the time to answer any questions and alleviate any concerns they may have, and then give them a patient leaflet if this is available in your trust. Finally ask the patient if it they are happy to go ahead with the procedure and gain verbal informed consent.

FLEXIBLE NASENDOSCOPY

Anna Melissa Harrison and Karl Frederick Braekkan Payne

Procedure

Gather all your other equipment, including anaesthetic spray, lubricant jelly, gauze and alcohol wipes (Figure 1). Position the patient seated upright. You may choose to use one or two sprays of anaesthetic, usually lidocaine. Opinion is split regarding the benefit of using anaesthetic spray and using lubricant jelly or water as a lubricant in experienced hands[2]. However, from experience, this author routinely uses anaesthetic spray and lubricant jelly. Assemble the nasendoscope by plugging in the cord to a light source and turning on the power (Figure 2). Orientate yourself with the nasendoscope; it may be connected to a computer screen, or you will be required to physically view down the eyepiece. The tip of the nasendoscope can be manoeuvred up and down using the dial on the main body of the scope, use a pen grip to comfortably hold the scope while allowing you to operate the dial (Figure 3). Ask the patient to look straight ahead and position yourself in front of the patient, holding the end of the scope in your left hand, horizontal to the patients face. The tendency is to believe you need to advance the scope upwards, whereas the nasal floor actually runs horizontal posteriorly from the nostril. Touch the tip of the scope on the patient's tongue to prevent misting of the lens when inside the nasal cavity Figure 4). Place some aquagel on a piece of gauze and lubricate the end of the nasendoscope. Angle the tip of the scope slightly upwards to gain access into the nasal cavity (Figure 5), once in, angle the tip straight and advance it smoothly under direct vision along the floor of the nasal passage inferior to the inferior turbinate (Figure 6). Check for septal deviation, turbinate hypertrophy or the presence of nasal polyps and look for any mucosal changes, masses or other abnormalities. As you exit the nasal cavity moving into the nasopharynx (Figure 7), angle the tip of the scope downwards and advance to visualise the larynx observing the vocal cords (Figure 8). If you experience difficulty, or your view becomes blurred or obstructed by secretions, ask the patient to swallow and this should rectify the situation. Inspect the vocal cords for asymmetry, nodules or growths, and then ask the patient to say "ah" and "ee", observing for vocal cord palsies. Once completed straighten the scope and withdraw smoothly out of the nasal cavity. Place the scope on a tissue or in a paper bag and follow your trust protocol for scope cleaning and sterilisation.

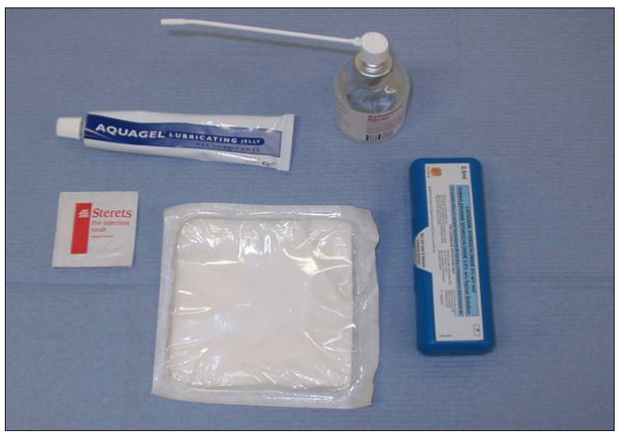


Figure 1: Equipment needed to perform nasendoscopy: aquagel lubricant, lidocaine spray, gauze pack, alcohol wipe.

Figure 2: Nasendoscope, with plug visible.

Figure 3: View of hand holding nasendoscope in pen grip, with index finger on dial.

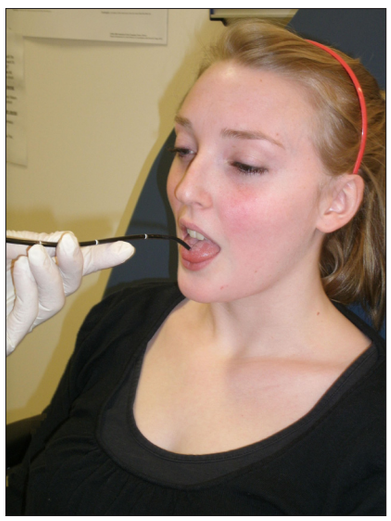


Figure 4: Touching the tip of the nasendoscope on the tongue, to prevent misting.

FLEXIBLE NASENDOSCOPY

Anna Melissa Harrison and Karl Frederick Braekkan Payne

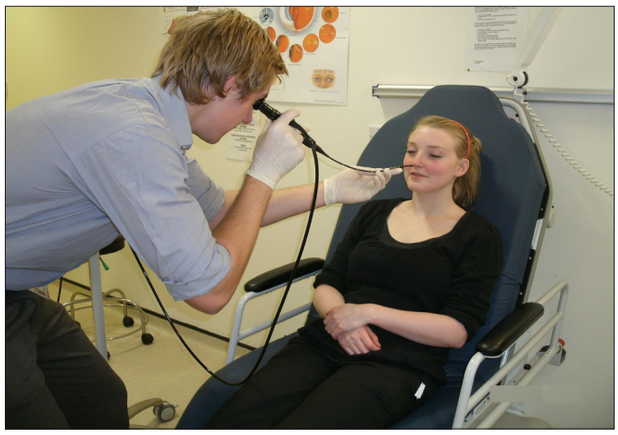


Figure 5: Positioning yourself in front of the patient, with the nasendoscope horizontal and the tip slightly upturned.

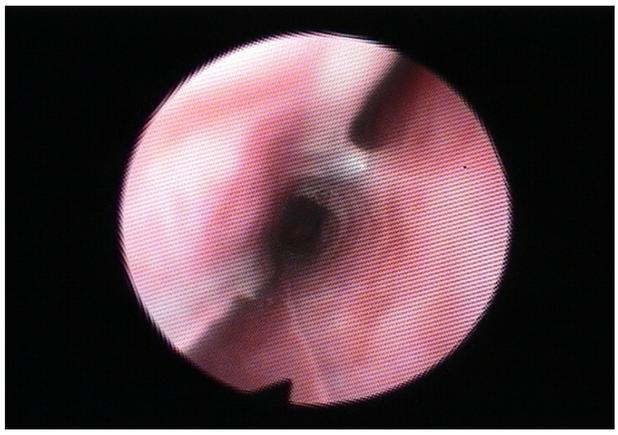


Figure 6: View inside nasal cavity: showing nasal septum (right), and turbinate (left).

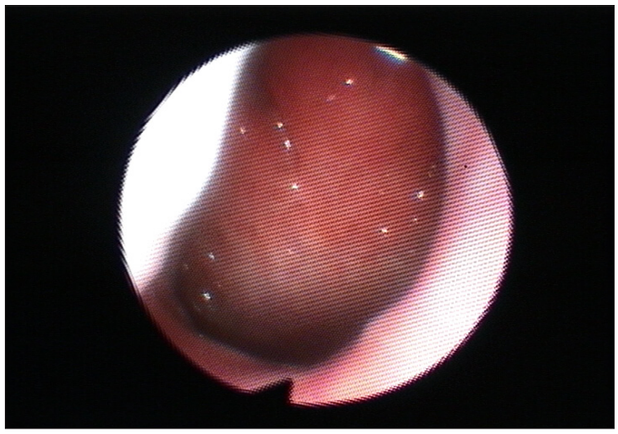


Figure 7: View out of nasal cavity, visualising posterior nasal space.

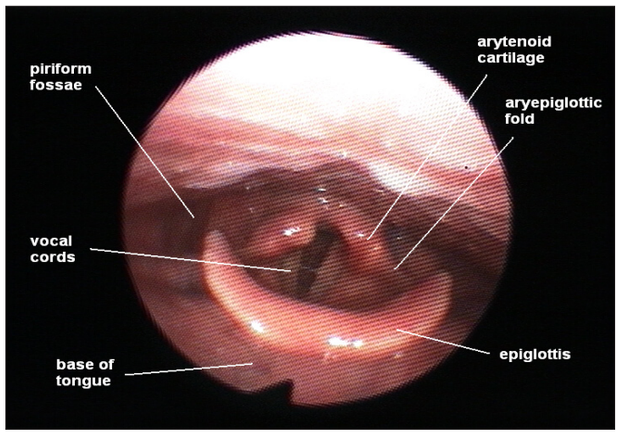


Figure 8: View of larynx, with annotations of major structures.

Documentation

Any procedure needs to be documented fully in the patient's case notes. Comment that the procedure was fully explained to the patient and consent gained. Document the type and strength of anaesthetic spray used. Describe the procedure, which side of the nose the scope was passed up, what was seen (even if normal anatomy) and how well it was tolerated. Areas to mention specifically are the nasal cavity, posterior nasal space, epiglottis, valleculae, piriform fossae, vocal cords and arytenoids[3].

Complications and their management

Nasendoscopy is a safe and well tolerated procedure. However, complications may include minor trauma to the nasal mucosa causing bleeding, gagging if the scope irritates the epiglottis or laryngospasm. After each use the scope must be cleaned to prevent cross contamination between patients, especially from MRSA, which is found in the nasopharynx of around 1.5% of the general public[4].

Conclusion

This article presented the case of an adult patient with acute epiglottis who required flexible nasendoscopy. It is of note that in an adult the symptoms of acute epiglottitis manifest less severely than in a child[5], and so nasendoscopy may be performed to make a diagnosis. In a child with suspected acute epiglottitis, senior help should be sought immediately, preferably an anaesthetist, and nasendoscopy should not be performed. Flexible nasendoscopy is used to visualise the nasal passage, nasopharynx, base of the tongue, supraglottic, glottis and hypopharynx, and is a simple and safe procedure with few complications.

FLEXIBLE NASENDOSCOPY

Anna Melissa Harrison and Karl Frederick Braekkan Payne

Nasendoscopy MCQ's

1. Which one of the following would not require a flexible nasendoscopy?
a) Adult glue ear.
b) Tonsillitis.
c) Stridor.
d) Otorrhoea.
e) Epistaxis.

2. Which one of the following structures would not be visible on a flexible nasendoscopy?
a) Piriform fossae
b) Base of tongue
c) Buccal sulcus
d) Aryepiglottic fold
e) Valleculae

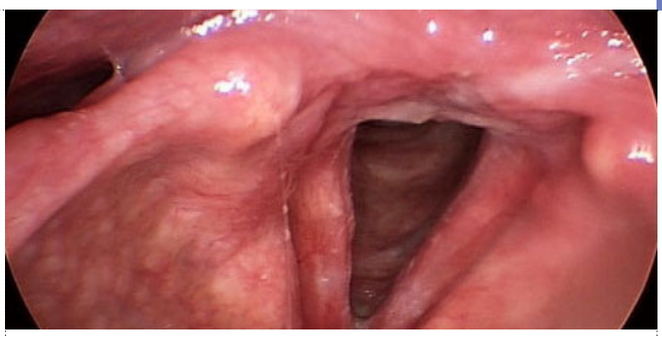
3. Which three nasal arteries form Kiesselbach's Plexus
a) Superior labial artery.
b) Dorsal nasal artery.
c) Anterior ethmoid artery.
d) Alar artery.
e) Posterior ethmoid artery.

4. Which one of the organisms below, is not an organism known to cause adult epiglottitis?
a) *Haemophilus influenzae*
b) *Neisseria meningitidis*
c) *Streptococcus pneumoniae*
d) Group A *Streptococcus*
e) *Chlamydia psittaci*

Answers

1. d) Persistent adult glue ear is uncommon, and you should think of performing flexible nasendoscopy to help rule out a secondary cause, i.e. a nasopharyngeal tumour. Patients with tonsillitis can be partially obstructed, and nasendoscopy may reveal excessive pooling of secretions. Patients with stridor will require nasendoscopy to visualise the aerodigestive tract, and assess for common causes of stridor, such as foreign bodies, tumours, infections or vocal cord palsies. Treatment of epistaxis in the acute phase will involve arrest of bleeding, most likely with an inflatable pack, such as a rapid rhino. However persistent bleeding may warrant nasendoscopy to try and identify the bleeding site, with the view to cautery.

Otorrhoea or otitis media, would require otoscopy and would not routinely warrant flexible nasendoscopy.



2. c) Flexible nasendoscopy gives you a view of the larynx. The piriform fossae, base of the tongue, aryepiglottic fold and valleculae all lie above the vocal cords, and are therefore visible from a superior view point when performing nasendoscopy. The buccal sulcus is a groove lateral to the teeth, and is not routinely viewed on nasendoscopy.

3. a), c) and e) Kiesselbach's plexus, or Little's Area, is the plexus of vessels in the mucosa of the septum anterior to the nasal valve. It is made up of the triad of arteries: anterior ethmoidal artery, posterior ethmoidal artery and superior labial artery. It is the most common site of bleeding in epistaxis due to the superficial and fragile nature of the vasculature in this area. The Dorsal nasal artery is a terminal branch of the ophthalmic artery, supplying the dorsum of the nose. The Alar artery is the terminal branch of the facial artery, supplying the external aspect of the nose.

4. e) Common isolates in adult epiglottitis include: *Haemophilus influenzae, Streptococcus Pneumoniae* and Group A *Streptococcus. Neisseria meningitidis* is an uncommon cause of adult epiglottitis. *Chlamydia psittaci* is not a known causative organism of adult epiglottitis.

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Authors

Anna Melissa Harrison
FY1 Doctor
Royal Derby Hospital

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FY1 Doctor
Royal Derby Hospital

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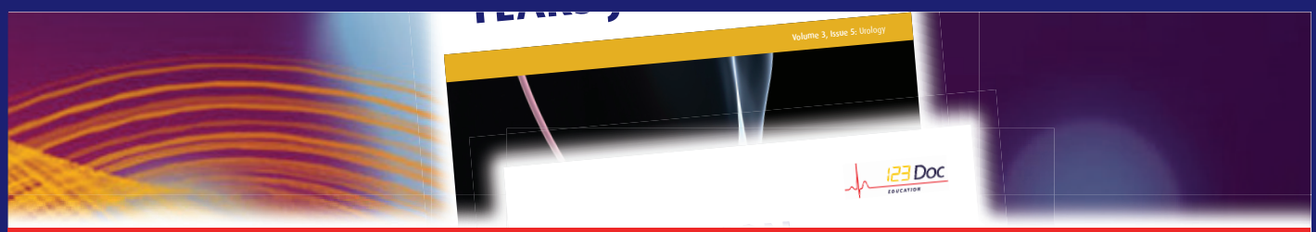

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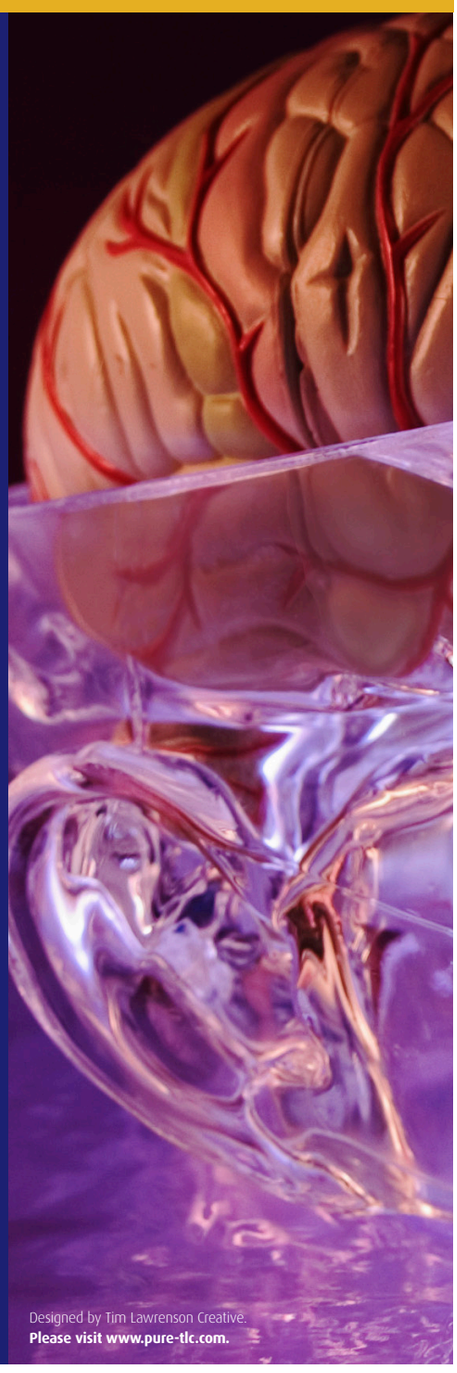

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