CHAPTER 18

Mouth Breathing

Dr. Hossam Makki

A 6-year-old boy is brought by his mother to the family physician with a chief complaint of mouth breathing and snoring for the past year. Recently, his snoring has become more severe, and his mother has noticed that her son stops breathing for a few seconds once or twice during his sleep.

The mother also mentions that he is a restless sleeper, and it is difficult to wake him up in the morning. In addition, he has attention difficulties in the school. His medical history shows that he experienced two URTIs in the last year. The mother has asthma.

On examination, his tonsils are hypertrophic (grade IV), but his nose and ears are normal. An X-ray is taken of the nasopharynx (Figure 18.1).

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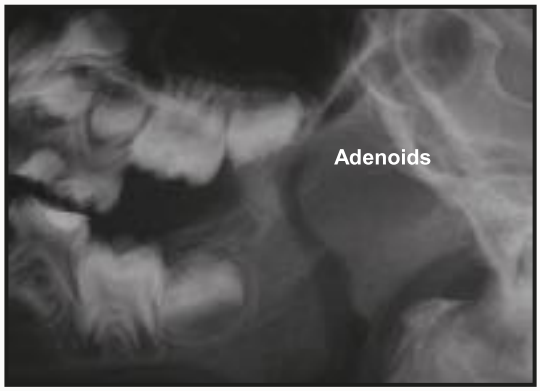

Figure 18.1: *X-ray of the nasopharynx with enlarged adenoids.*

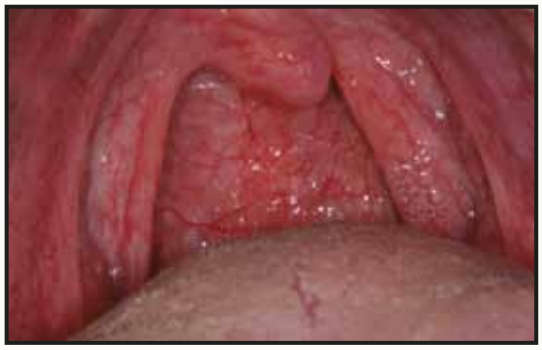

Figure 18.2: *Tonsils grade I.*

Q1. What do you mean by grade IV tonsils?

Tonsils are graded according to their size and the degree of obstruction of the oropharynx (Figures 18.2 and 18.3).

Grade 0: tonsils are entirely in the tonsillar fossae.

Grade 1: tonsils occupy 25% or less of the space between the anterior pillars.

Grade 2: tonsils occupy 25-50% of the space between the anterior pillars.

Grade 3: tonsils occupy 50-75% of the space between the anterior pillars.

Grade 4: tonsils occupy >75% of the space between the anterior pillars.

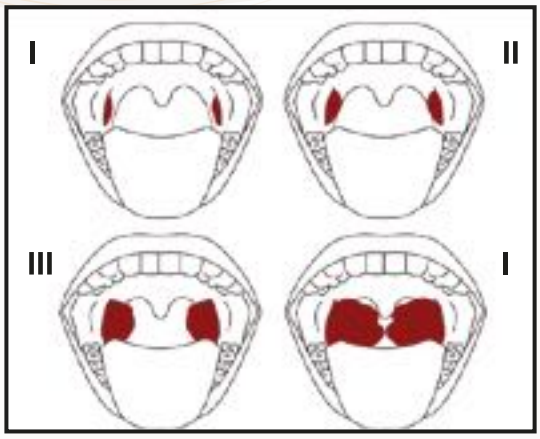

Figure 18.3: Grading of tonsil enlargement.

Q2. Where are the palatine tonsils located?

The palatine tonsils, also referred to as the faucial tonsils, lie between the anterior and posterior tonsillar pillars on the lateral walls of the oropharynx.

Q3. What are the anatomical relationships of the palatine tonsils?

The palatine tonsils are masses of lymphoid tissue on the sides of the oropharynx. Anteriorly, the tonsils are related to the palatoglossus muscle (anterior pillar), and posteriorly, they are related to the palatopharyngeus muscle (posterior pillar). The tonsils have a capsule that separates the tonsils laterally from the superior constrictor muscle.

Q4. What are the blood, nerve and lymphatic supplies of the tonsils?

Their main blood supply comes from the facial artery. The tonsils also receive blood from the ascending pharyngeal the lingual and the maxillary branches of the external carotid artery (Figure 18.4). Venous blood drains through a peritonsillar plexus to the lingual and pharyngeal veins, which drain into the internal jugular vein. The tonsils do not possess afferent lymphatics. The efferent lymphatics drain directly into the upper deep cervical chain, including the jugulodigastric and the retropharyngeal lymph nodes. The tonsils are innervated by the tonsillar branches of the maxillary nerve and the glossopharyngeal nerve.

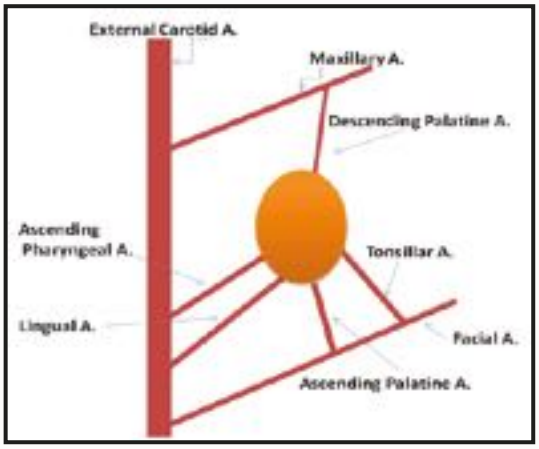

Figure 18.4: The rich blood supply of the tonsils.

Q5. Are there other tonsils?

Yes, the other tonsils (Figure 18.5) are the following:

A. The pharyngeal tonsils (adenoids) lie on the roof and posterior wall of the nasopharynx, facing the choana.

B. The tubal tonsils lie laterally to the pharyngeal tonsils and surround the openings of the Eustachian tubes.

C. The lingual tonsil lies at the base of the tongue (posterior 1/3).

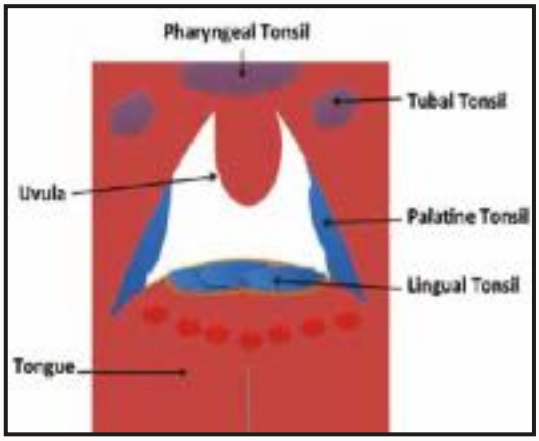

Figure 18.5: *Other tonsils.*

Q6. What is Waldeyer's ring (Figure 18.6)?

Waldeyer's ring is an interrupted ring of lymphoid tissue that encircles the back of the nose and oropharynx.

The ring consists of the following:

A. The pharyngeal tonsil (adenoids) in the roof of the nasopharynx

B. The tubal tonsils around the Eustachian tubes

C. The palatine tonsils on the sides of the oropharynx

D. The lingual tonsil at the base of the tongue

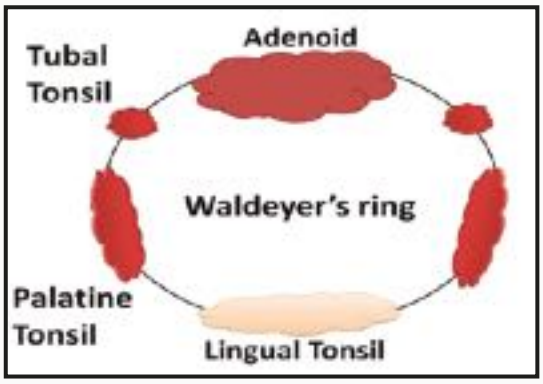

Figure 18.6: *Waldeyer's ring.*

Q7. What is the function of Waldeyer's ring?

Due to its strategic anatomic position at the entrance of the respiratory and alimentary tracts, Waldeyer's ring is continuously exposed to inspired air and ingested food antigens. These lymphoid tissues are composed primarily of B and T lymphocytes. Antigen exposure produces secretory antibodies, interferon and lymphokines. Hyperplasia is thought to result from B-cell proliferation in response to high doses of antigen exposure from the air and food to form a barrier against infectious agents that enter through the nose or mouth.

Q8. What are the causes of snoring and mouth breathing in this child?

Hypertrophic tonsils and/or the adenoids are the most common causes in children. Other causes include the following:

- Nasal obstruction due to nasal septal deviation, nasal polyps, choanal stenosis or nasal tumors
- Cerebral palsy
- Neuromuscular weakness
- Down syndrome

Q9. Is it normal for children to have enlarged tonsils and adenoids?

This hypertrophy is considered normal or physiological. The tonsils and adenoids normally increase in size rapidly after birth through adolescence because the peak immunological activity is observed between the ages of 3 and 10. After this peak in immunological activity, they regress in size.

Q10. Why does the physiological hypertrophy of the tonsils and adenoids become pathological hypertrophy in some children, causing mouth breathing, snoring and even obstructive sleep apnea?

The causes of adenotonsillar hypertrophy are as follows:

- Chronic adenotonsillitis
- Allergy
- Reflux esophagitis has been claimed recently to be one of the main causes (more research is needed to support this claim)

Q11. In addition to mouth breathing and snoring, what are the other symptoms of adenotonsillar hypertrophy?

- Difficulty with feeding
- Frequent awakenings or restlessness and frequent nightmares
- Excessive daytime sleepiness
- Difficulty waking up in the morning
- Secondary enuresis
- Hyperactivity and/or behavioral problems
- Poor school performance, which might be incorrectly diagnosed as ADHD (attention deficit hyperactive disorder)

Q12. What signs might you see in such a child?

- Adenoid facies with a flattened nasolabial fold, protruding upper incisors and an open mouth (Figure 18.7)
- Failure to thrive
- Otitis media with effusion and chronic ear infection due to Eustachian tube blockage
- Maldevelopment of the upper jaw, dental malocclusion and a high arched palate (Figure 18.8)
- Possible alveolar hypoventilation, pulmonary hypertension, cor pulmonale, and heart failure in severe cases

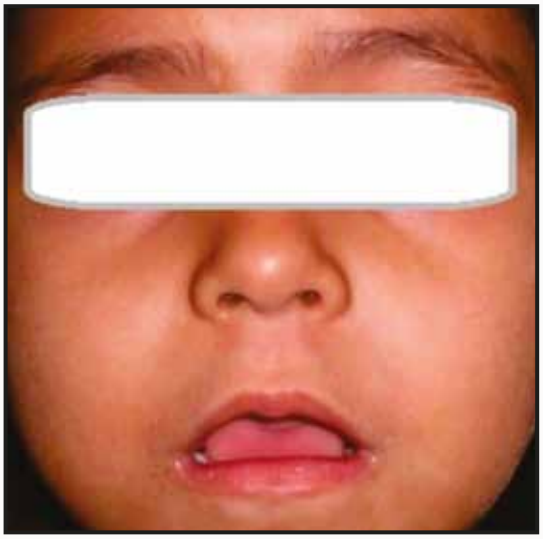

Figure 18.7: *Adenoid face*

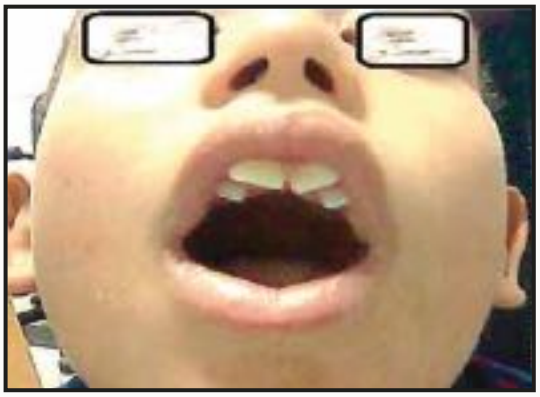

Figure 18.8: *Dental malocclusion*

Q13. Now that you have diagnosed the child as having symptomatic (pathological) adenotonsillar hypertrophy, what is the treatment?

The gold standard treatment is adenotonsillectomy (Figures 18.9,1 8.10, 18.11 & 18.12)

However, some children with allergic rhinitis may benefit from a nasal steroid spray.

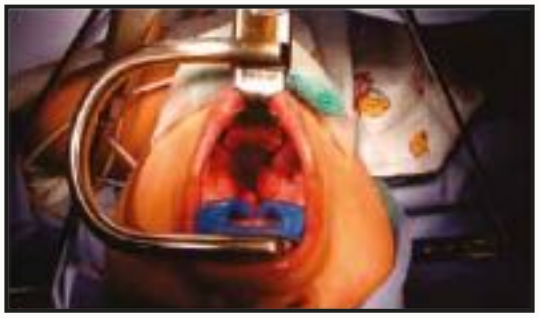

Figure 18.9: *Intraoperative positioning for adenotonsillectomy*

Figure 18.10: *A mirror is used to visualise the nasopharynx during adenoidectomy*

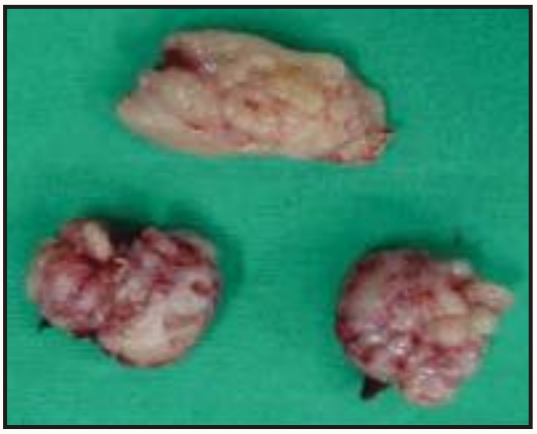

Figure 18.11: *The two tonsils and adenoids at the top after removal.*

Figure 18.12: *A curette is the classic tool used to remove the adenoids.*

Q14. If a child undergoes an adenotonsillectomy, is his immune status going to be affected? The tonsils and adenoids are part of the immune system, right?

With chronic or recurrent tonsillitis, they become more harmful than useful. There would appear to be a therapeutic advantage to removing recurrently or chronically diseased tonsils.

However, some studies have demonstrated minor alterations in Ig concentrations in the serum and adjacent tissues following tonsillectomy. There are no studies to date that have demonstrated a significant clinical impact of tonsillectomy on the immune system too.

CHAPTER 19

Sore Throat

Dr. Mohammed Hodan

A 36-year-old male presents to the ENT clinic complaining of a sore throat for 4 months that is not associated with fever and has not responded to three different antibiotics given by different doctors. He also complains of a dry tongue and lips in the early morning. He uses a steroid nasal spray for allergic rhinitis. The patient said he did not have other symptoms such as dysphagia, weight loss or a neck mass. He underwent tonsillectomy 2 years ago. He has smoked 15 cigarettes a day for 15 years as well as Shisha (Water-pipe tobacco) on the weekends.

On physical examination, there is a tonsillectomy scar, congestion in the pharynx, a hairy tongue and poor oral hygiene. A nasal examination shows a deviated nasal septum and bilateral inferior turbinate hypertrophy. The remaining head and neck examinations are negative.

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Q1. What is sore throat?

Sore throat is a symptom that presents as pain, itching or irritation in the back of the throat. It can be associated with other symptoms such as fever, odynophagia, dysphagia, cough, dyspnea, neck mass and heart burn.

Q2. What is the difference between dysphagia and odynophagia?

Dysphagia refers to difficulty in swallowing, while odynophagia refers to painful swallowing.

Q3. Anatomically, what is meant by "throat"?

The throat includes the anatomical structures of the pharynx, larynx, tonsils, tongue base and soft palate.

Q4. What is the pharynx?

The pharynx is a musculo-membranous tube that extends from the base of the skull to the sixth cervical vertebra posteriorly and the cricoid cartilage of the larynx anteriorly and is connected to the esophagus at the bottom. The pharynx is divided into three parts: the nasopharynx, the oropharynx and the hypopharynx.

Q5. What are the possible causes of the sore throat in this case presentation?

The 4-month duration of the symptoms without fever excludes the possibility of infection. The absence of a neck mass, weight loss, dysphagia and the fluctuating nature of the disease decrease the possibility of a tumor. The patient has nasal obstruction that might cause a sore throat due to mouth breathing. Some patients may undergo tonsillectomy as a result of an incorrect diagnosis of the underlying cause, which could have been the case with this patient. The patient is a smoker with poor oral hygiene, which could also explain the chronic nature of his illness.

Q6. How do we prepare properly for a throat examination?

The patient should be sitting comfortably. Good lighting should be used. A headlight is ideal because both hands can be used, which is especially important in children. An otoscope light is not appropriate (minimum light and only one free hand). Tongue depressors (Figure 19.1), gloves and a local anesthetic spray should be available.

Figure 19.1: *A tongue depressor.*

Q7. How should we examine the throat?

The sequence learned in medical school usually stays with individuals throughout their careers. It is important to establish a sequence to perform the procedure in the same manner for each oral exam. Often "quick" assessments are not adequate to determine subtle abnormalities. Throat examination starts by examining the lips, teeth, gums and tongue. The patient should open his mouth wide. Use two tongue depressors with both hands to visualize the cheeks, gums and teeth on both sides and compare them with each other (Figure 19.2). The mucosa should be bright pink, smooth, glistening, uniform, moist and odorless.

Ask the patient to stick his tongue out and move it up, down, left and right. In addition to the movement, assess the mucosal color, moisture and papillae. The palate is easy to examine, so do not forget to look at it.

Some clinicians tend to begin the throat exam in the oropharynx. The examination should end at the back of the throat. In some cases, with cooperation, you may not need to use a tongue depressor to see the back of the throat. Ask the patient to say "aah" to depress the tongue for a full view.

Figure 19.2: *Using two tongue depressors make it easier to visualise the cheeks, gums or gingival and teeth . (Courtesy of Dr. Ameen Al-Herabi)*

Q8. How can you overcome difficulties when examining the uvula, tonsils and posterior oropharynx?

Some patients have a large tongue and small throat or a difficult gag reflex. In these cases, you need to counsel your patient properly to gain their cooperation. The back of the throat should be the last part of the examination. Use a local anesthetic such as lidocaine spray to reduce the throat gagging. Ask the patient to take a deep breath, hold it and extend his or her neck. This movement may add a few millimeters of throat exposure, which may be all you need. A fiber-optic flexible nasal scope (Figure 19.3) can help to visualize the uvula, tonsils and posterior oropharynx.

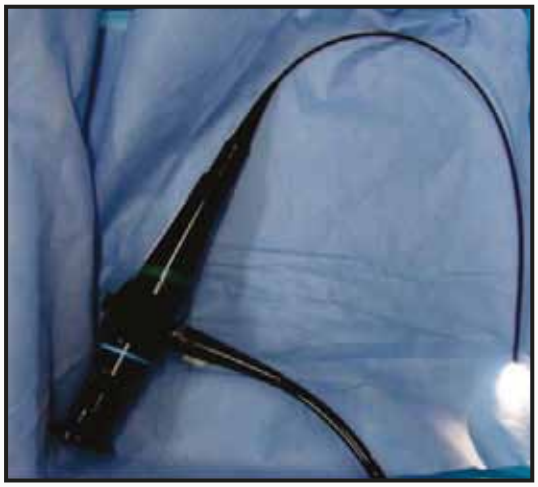

Figure 19.3: *Fibre-optic scopes are available in all ENT clinics in the present day.*

Q9. How do you examine a child's throat?

Children are usually afraid of tongue depressors but several techniques can make examination easier. For example, give the child a clean tongue depressor to play with, let him examine his parent's mouth or a puppet's mouth first. I tell the children three times "Do not bite my finger please, please" to help alleviate the child's fear. Performing the examination in front of a mirror is a great aid in enlisting the child's cooperation. The last option is to perform the examination during episodes of crying. If the child resists opening his mouth, pinching both nostrils gently will force the child to breathe through his mouth, and then you can examine his throat.

Q10. How should you complete your examination?

The larynx, hypopharynx and the neck should be examined to complete the throat exam. The larynx and hypopharynx can be examined using a mirror, fiber-optic flexible scope or rigid telescope to look at the epiglottis, aryepiglottic folds, vocal folds, arytenoids and pyriform fossae.

Q11. What are the common lip lesions?

The doctor should note the presence of cracks or fissures of the lips (cheilitis). Angular stomatitis is characterized by fissuring at the angles of the lips and may indicate deficiencies of the vitamins riboflavin or niacin. Vesicular eruptions on the lip are sometimes present with herpes simplex virus infection.

Q12. What is the rule of palpation during a mouth exam?

Each suspicious area should be visually inspected and palpated against the bones of the jaw or between the clinician's fingers. Always try to press against a structure that is firm. This palpation is very helpful in diagnosing salivary gland stones and tonsilloliths and feeling the deep tissue for the possibility of a submucosal tumor. Examine the patient with a gloved hand for protection (Figure 19.4). The mouth may also be the site of a primary syphilitic chancre, which appears as a firm nodule that ulcerates and crusts. Milking the salivary gland can bring a stone out of the duct.

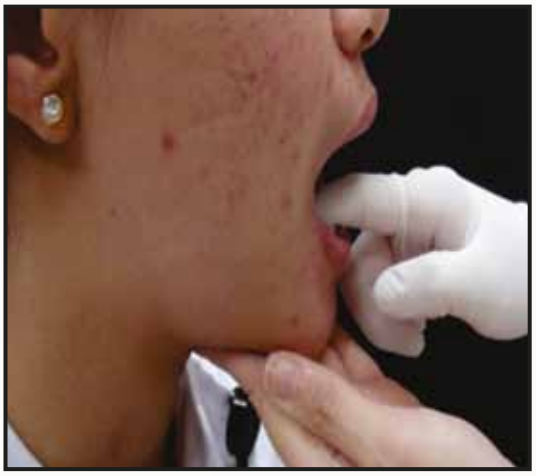

Figure 19.4: *A manual examination using the fingers can help diagnose sub-mucosal lesions.*

CHAPTER 20

Bad Breath

Dr. Saad Saleh

A 12-year-old boy presents to the otolaryngology clinic to be assessed for bad breath odor. His parents report that he had had a few tonsillar infections before his symptoms started approximately 1 year prior. His symptoms have been stable but persistent despite trials of multiple mouthwashes and several courses of antibiotics prescribed by his pediatrician. He also has a chronic sore throat and odynophagia with decreased oral intake. His parents said there wasn't any history of sleep disturbance or laryngopharyngeal reflux symptoms. He is otherwise healthy with no other medical issues. His family history is unremarkable.

Physical examination reveals a healthy boy in no apparent distress. An oropharyngeal examination shows grade II tonsils with wide crypts containing a tonsillolith (Figure 20.1). The oropharyngeal mucosa is erythematous, and he has healthy dentition. No cervical lymphadenopathy is detected. The remainder of the head and neck examination is unremarkable.

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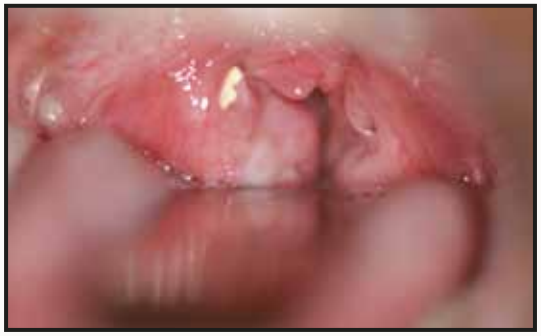

Figure 20.1: *Tonsillolith observed in the right tonsil.*

Q1. What is the medical terminology for foul-smelling breath?

Halitosis.

Q2. What is the most likely diagnosis in the case illustrated above?

Chronic tonsillitis: symptoms that result from chronic inflammation of the palatine tonsils lasting more than 4 weeks. The symptoms include sore throat, odynophagia, otalgia, halitosis and/or persistently tender cervical lymph nodes.

Q3. What are the other causes of halitosis?

- Periodontal disease/poor oral hygiene
- Chronic sinusitis/nasal foreign body
- Upper aerodigestive tract malignancy
- Laryngopharyngeal reflux disease
- Lower respiratory infections, e.g., lung abscess
- Tobacco use
- Dietary causes (e.g., garlic)
- Systemic causes: diabetic ketoacidosis, hepatic failure, etc.

Q4. What are tonsilloliths?

They are calculi that are produced within the tonsillar crypts. Tonsilloliths contain bacteria that produce fetid substances as a result of protein degradation, leading to their foul smell.

Q5. How can you clinically classify tonsillitis?

- Acute tonsillitis: acute symptoms of fever, sore throat, odynophagia and malaise with clinical evidence of inflamed tonsils covered by exudate
- Recurrent acute tonsillitis
- Chronic tonsillitis

Q6. What common pathogens cause tonsillitis?

A. Viral causes: rhinovirus, adenovirus, influenza virus, parainfluenza virus, Epstein-Barr virus (EBV), herpes simplex virus and cytomegalovirus

B. Bacterial causes:

- Group A β-hemolytic streptococci (GABHS) and other streptococcal species
- *Staphylococcus aureus, Haemophilus* species (in recurrent tonsillitis)
- Other bacteria: *Neisseria gonorrhoeae*.
- *Corynebacterium diphtheria* and *Bacteroides* species

C. Other pathogens: *Candida* species, *Treponema pallidum* (syphilis)

Q7. What tests are needed in the above-mentioned case?

Normally, no tests are needed because the diagnosis of chronic tonsillitis is clinical. In cases of sleep-disordered breathing issues, a sleep study (polysomnogram) and/or endoscopic examination/imaging of the nasopharynx to assess the size of the adenoids can be considered in select cases.

Q8. What tests can be considered in cases of acute tonsillitis?

- Throat swab culture and/or rapid streptococcal antigen testing (Figure 20.2)
- Monospot test and viral serology
- Complete blood count
- Imaging (lateral neck X-ray or CT scan) if suppurative complications are suspected

Figure 20.2: *Swab for cultures*

Q9. Which antibiotic is recommended to treat tonsillitis caused by GABHS (strep throat)?

Penicillin. In cases of penicillin allergy, a macrolide or a first-generation cephalosporin can be chosen.

Q10. What are the possible non-suppurative complications of GABHS tonsillitis?

- Scarlet fever
- Rheumatic fever
- Post-streptococcal glomerulonephritis

Q11. What is scarlet fever?

A condition caused by the release of exotoxins produced by GABHS. Manifestations of scarlet fever include an erythematous rash, severe lymphadenopathy, fever, strawberry tongue, yellowish exudate on the tonsil and arthralgia.

Q12. What are the possible suppurative complications of tonsillitis?

- Peritonsillar abscess (quinsy)
- Parapharyngeal abscess
- Retropharyngeal abscess
- Neck abscess
- Septic thrombosis of the internal jugular vein (Lemierre's syndrome), which may lead to metastatic abscesses

Q13. What are the signs of developing suppurative complications of tonsillitis?

- Trismus
- Deviation of the uvula with asymmetry of the oropharynx
- Neck stiffness or torticollis
- Stridor and respiratory distress

Q14. How do you manage a patient with chronic tonsillitis?

Management options include the following:

- Treating the underlying cause e.g. laryngopharyngeal reflux and stop smoking
- Tonsillectomy
- Administering a 3-6-week course of antibiotics
- Employing a mouthwash or rinse

CHAPTER 21

Snoring

Dr. Ali Ismaili Swaid

A 4-year-old boy presents with a history of snoring, mouth breathing and hyponasal speech for the prior year. There is no history of recurrent sore throat, recurrent otitis media, hearing loss, sleep apnea or allergic symptoms.

Physical examination reveals an underweight child with grade I tonsils, bilateral nasal discharge and grade IV adenoid hypertrophy visualized using a flexible naso-endoscope. There is no septal deviation, bifid uvula or submucous cleft palate.

* * * * *

Q1. What are the adenoids?

The adenoids are lymphoid tissue situated at the junction of the roof and posterior wall of the nasopharynx (Figure 21.1). The adenoids are composed of vertical ridges of lymphoid tissue separated by deep clefts and covered by a ciliated columnar epithelium. They have few crypts and no capsule.

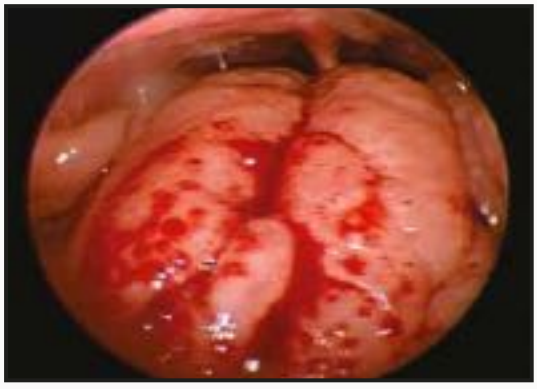

Figure 21.1: *Endoscope image of the nasopharynx showing enlarged adenoids. A suction catheter is used to lift the palate and give a wide view, but the procedure can cause some trauma to the mucosa.*

Q2. What is the function of the adenoids?

The adenoids are predominantly B-cell lymphoid structures that are probably involved in inducing secretory immunity.

Q3. Is it normal to have adenoids?

Yes, the adenoid tissue is present at birth, enlarges during childhood and regresses during puberty. Due to repeated infections, the adenoid tissue may undergo hyperplasia.

Q4. What are the main complaints associated with adenoid hypertrophy?

- Nasal obstruction, which causes snoring and mouth breathing
- Hyponasal speech
- Nasal and/or postnasal discharge

- Eustachian tube dysfunction and consequently recurrent acute otitis media or chronic otitis media
- May contribute to chronic rhinosinusitis in young children

Q5. How can you diagnose adenoid hypertrophy?

The diagnosis of adenoid hypertrophy is mainly based on the history and an assessment of nasal patency using a flexible naso-endoscope. A lateral soft tissue radiograph of the nasopharynx can be performed in uncooperative patients. However, the radiograph may be misleading because the apparent size of the airway can vary with the phase of respiration. In a cooperative child, a flexible scope (Figure 21.2) is useful to confirm the diagnosis and exclude other differential diagnoses such as inferior turbinate enlargement, which may mimic adenoid hypertrophy symptoms or be present simultaneously. Inferior turbinate enlargement should be treated medically or surgically at the same time; otherwise, the symptoms may not resolve completely (Figures 21.3 and 21.4).

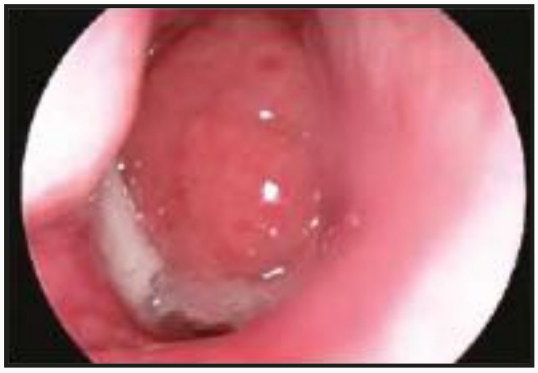

Figure 21.2: *Flexible nasoendoscope images showing enlarged adenoids.*

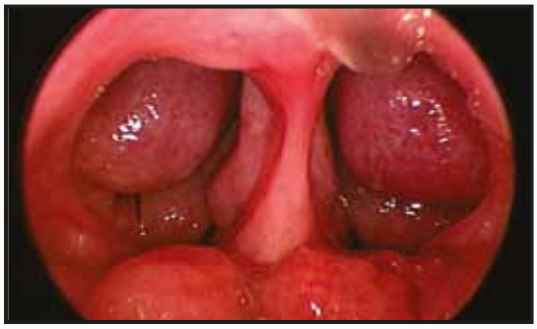

Figure 21.3: *An enlarged inferior turbinate posteriorly can block the choana and cause symptoms similar to enlarged adenoids. Note the small adenoids.*

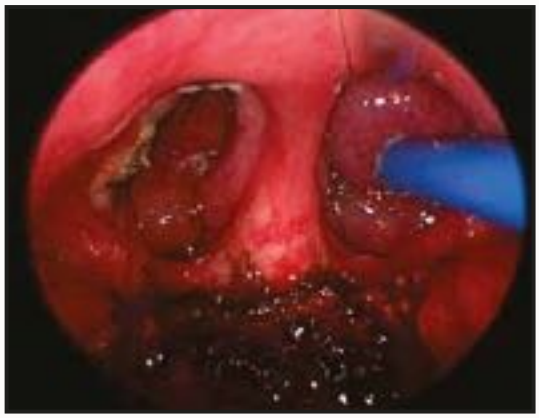

Figure 21.4: *Suction cautery can shrink the inferior turbinate under direct visualisation.*

Q6. What are the complications of long-standing nasal obstruction due to adenoid hypertrophy?

- Obstructive sleep apnea
- Pulmonary hypertension
- Cor pulmonale
- Adenoid face

Q7. What is the adenoid face?

The child has a flattened mid-face with a dull expression, an open mouth, prominent crowded upper teeth and a high-arched palate.

Q8. How can we treat a child with adenoid hypertrophy?

Adenoidectomy is the standard treatment for adenoid enlargement. However, if symptoms are mild and referable to infection and/or allergy, the following conservative treatment may be advocated:

- Antibiotic against β-lactamase-producing organisms
- Anti-allergy medications
- A 6-8-week course of intranasal steroids

Q9. What are the indications for adenoidectomy?

Adenoid hypertrophy resulting in or associated with the following:

- Sleep disturbances or sleep apnea
- Cor pulmonale
- Failure to thrive
- Nasal obstruction associated with orofacial abnormalities
- Swallowing and speech abnormalities
- Recurrent acute otitis media or chronic otitis media
- Suspected neoplasm

Q10. What are the contraindications for adenoidectomy?

- Cleft palate
- Velopharyngeal insufficiency
- Bleeding tendency
- Contraindications of anesthesia

Q11. What are the complications of adenoidectomy?

- Hemorrhage is usually observed in the immediate post-operative period, but it is rare with suction cautery (Figures 21.5 and 21.6).
- Injury to the Eustachian tube opening may lead to otitis media.
- Injury to the pharyngeal musculature and vertebrae is usually due to hyperextension of the neck when operating on patients with Down syndrome because 10-20% of such patients have atlanto-axial instability.
- Velopharyngeal insufficiency can be avoided by inspecting and palpating the palate prior to adenoidectomy to exclude submucosal cleft palate.
- Nasopharyngeal stenosis can occur due to scarring.
- Recurrence is usually due to regrowth of adenoid tissue that was left behind.

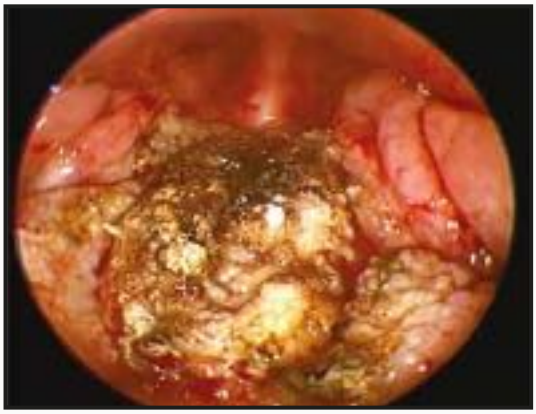

Figure 21.5: *A patent nasopharynx after removal of the adenoids and cauterising their bed.*

Figure 21.6: *Hand piece for suction cautery*

CHAPTER 22

My Child Has Recurrent Tonsillitis!

Dr. Nasser A. Faqeeh

A 4-year-old boy presents to the clinic complaining of recurrent attacks of sore throat associated with high-grade fever. His mother indicates that he has had more than three visits this year to his family physician for the same reason. She mentions multiple uses of different antibiotics to treat her son.

Physical examination shows that the child is generally thin and has multiple cervical lymphadenopathies. His throat examination indicates cryptic tonsils and erythema of the anterior pillars. His other tests are within normal limits.

* * * * *

Q1. What is the most likely diagnosis?

Recurrent acute tonsillitis.

Q2. What are the tonsils?

The literature refers to "the tonsils" as the two lymphoid tissue structures located in the tonsillar fossae of the oropharynx, which are located between the posterior tonsillar pillar (palatopharyngeus muscle) and the anterior tonsillar pillar (palatoglossus muscle). They are also called the palatine tonsils. (Figure 22.1)

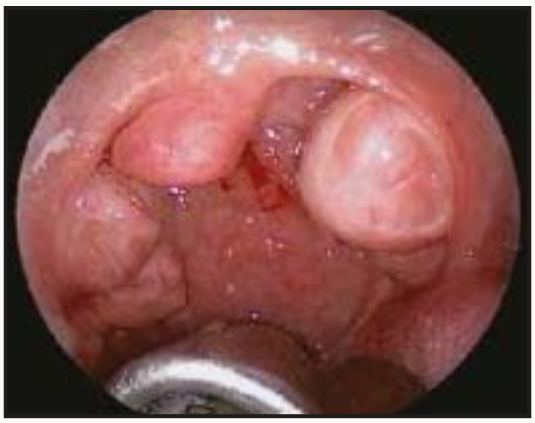

Figure 22.1: *Chronic tonsillitis. The figure demonstrates the crypts and the asymmetry between the two tonsils.*

Q3. What is the blood supply of the tonsils?

The tonsils have a rich blood supply, which is important because bleeding is a common and dangerous complication of tonsillectomy. The vessels responsible for the arterial blood supply are (Figure 18.4) the following:

- The tonsillar (the largest) branch of the facial artery
- The tonsillar branch of the dorsal lingual artery
- The ascending palatine branch of the facial artery
- The ascending pharyngeal artery
- The lesser palatine artery

The venous blood drains through the peritonsillar plexus into the lingual and the pharyngeal veins, which drain into the internal jugular veins. Lymph drains into the upper deep neck nodes, especially into the jugulodigastric lymph node. These lymph nodes commonly become enlarged due to tonsil infections and neoplasms.

Q4. What is the nerve supply of the tonsils?

The tonsillar region is innervated by the glossopharyngeal nerve. This innervation is the cause of referred otalgia in association with tonsillitis and post-tonsillectomy pain.

Q5. What is the clinical presentation of tonsillitis?

Acute tonsillitis:

The patient may present with one or more of the following symptoms: fever, chills, sore throat, poor appetite, malaise, joint pain and headache.

Upon physical examination, erythema of the tonsils with or without purulent exudate on the surface of the tonsils and cervical lymphadenopathy may be present.

Tonsillitis may also present acutely as a peritonsillar abscess (quinsy), which is a complication of acute infection. In addition to high fever, sore throat and muffled voice, the patient may also present with trismus and upper airway obstruction (Figure 22.2).

Chronic or persistent tonsillitis:

The typical presentation is recurrent or persistent sore throat, malaise, joint pain and halitosis (bad odor of the breath). Physical examination reveals tonsils with an irregular cryptic surface, erythema of the anterior pillars and cervical lymphadenopathy. The tonsils are usually symmetrical in size.

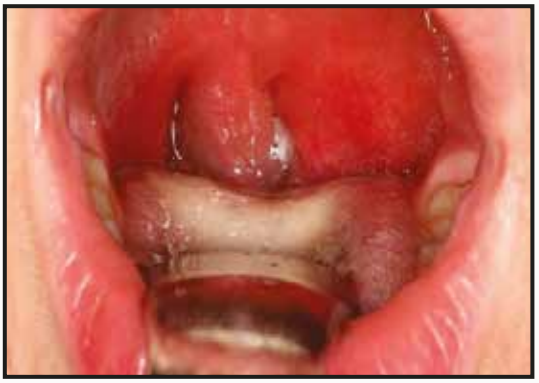

Figure 22.2: *A patient with a peritonsillar abscess.*

Q6. What are the common indications for tonsillectomy?

1. Enlarged tonsils that result in obstruction to breathing and/or feeding, e.g., obstructive sleep apnea
2. Suspicion of tonsillar tumor
3. Recurrent acute tonsillitis, which is

defined as three or more episodes of infection in one calendar year or 7 episodes in 2 years. The American Academy of Pediatrics recommends cut-off values of seven infections in one year, five infections per year for 2 years or three infections per year for 3 years. Other factors that strengthen the need for tonsillectomy include severe infectious attacks, recurrent febrile convulsions, an immunocompromised child suffering from recurrent attacks of fever, diabetes mellitus and/or cystic fibrosis.

4. Chronic tonsillitis, which is defined as a persistent sore throat and halitosis in association with chronic cryptitis and micro-abscesses of the tonsillar parenchyma.

Q7. The mother is concerned about her child's immune system after tonsillectomy.

Removal of the palatine tonsils has no proven major influential role in compromising the human body's general immune system. Clinical considerations still form the actual basis for surgery.

Q8. What is the microbiology of acute tonsillitis?

- Bacterial infection: Group A beta-Hemolytic streptococcus GABHS, which is the organism responsible for rheumatic heart disease and acute glomerulonephritis; *Groups B, C* and *G Streptococcus* species; *H. influenza*; *Moraxella catarrhalis*; *Staphylococcus aureus*; and *Bacteroides fragillis*.
- Viral infections: *rhinovirus*, *adenovirus, respiratory syncytial virus, influenza A* and *B, parainfluenza* viruses, *coxsackievirus, CMV* and *Epstein-Barr virus (EBV).*
- *Candida* infection (candidiasis) particularly occurs in immunocompromised patients.

Q9. What are the pre-operative requirements for a child undergoing tonsillectomy?

- Surgical consent signed by the child's guardian.
- Any history of hematological disease, bronchial asthma or other medical illnesses.
- Family medical history and previous anesthesia complications should also be identified.
- The child's guardian should be informed about the expected time of the surgery, so the child can fast for six hours prior to the surgery.
- Children younger than 6 years old should receive intravenous fluids while fasting and waiting for surgery.

Q10: How is tonsillectomy performed?

Tonsillectomy is performed in the operating room. The patient is positioned in a supine position with the neck extended.

A mouth gag is used to expose the oropharynx, and the tonsils are dissected out (Figures 22.3 and 22.4). Many of the methods used to perform tonsillectomy are discussed in the literature, including traditional cold dissection, electrocautery, and laser cutting. The method remains the surgeon's preference based on the way the surgeon was trained.

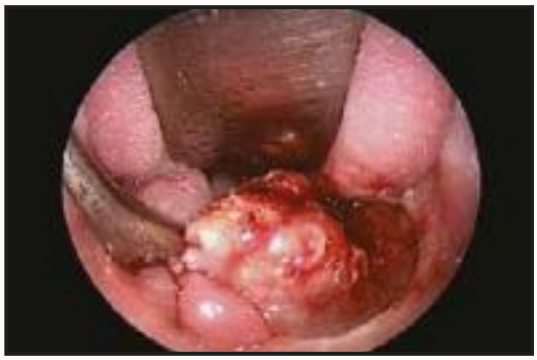

Figure 22.3: *Tonsillectomy performed using electrocautery.*

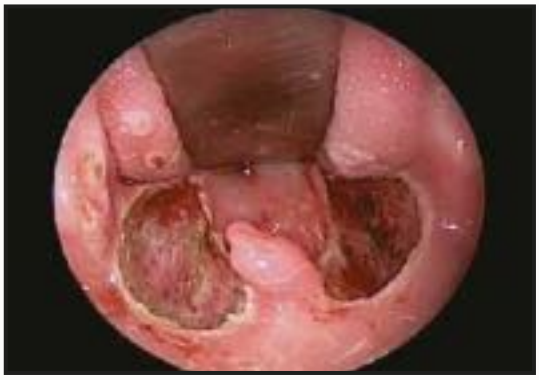

Figure 22.4: *Tonsillectomy procedure accomplished.*

Q11: What complications may follow tonsillectomy?

1. Early post-operative bleeding may occur in 1-2% of cases.
2. Late post-operative bleeding usually occurs between the seventh and tenth post-operative days in patients who have poor control of post-operative pain and poor oral intake, resulting in granulation tissue formation and eventual bleeding.
3. Infection may rarely occur in some patients, and it is usually manifested by fever.
4. Pain is a common subjective complaint noted more often in children younger than 3 years of age and in patients older than 12 years of age.
5. Referred otalgia may occur.
6. Aspiration is a rare complication that may result after operating on a patient with a full stomach.
7. Velopharyngeal sphincter insufficiency may occur.
8. Velopharyngeal sphincter stenosis may occur.

Q12. A child had a tonsillectomy and was taken to the family doctor 3 days after surgery for poor oral intake. The family doctor diagnosed a fungal infection of the throat and administered an antifungal medication after taking a swab for culture. The child's mother came to see you. What is your intervention?

Most tonsillar beds after surgery become covered by a white exudate that is mistaken by inexperienced physicians for a fungal infection. The mother should be reassured.

Q13. The child's mother also noticed a bad smell and wonders whether gauze was left inside the throat of her child during surgery?

Bad breath may be present after adenoidectomy and/or tonsillectomy procedure; it is more common due to dehydration, infection and the use of cautery.

CHAPTER 23

Noisy Breathing

Dr. Haifaa S. Alnasser

A 4-week-old newborn male is brought to your office with a 1-week history of "noisy breathing." The parents notice that the noisy breathing worsens when the newborn is lying down or crying. The noisy breathing is most noticeable during inhalation. There is no history of fever, coughing, runny nose, changes in crying, apnea or feeding difficulties. He has been gaining weight appropriately. The prenatal course was uneventful, and he was delivered at 38 weeks' gestation by spontaneous vaginal delivery without complications. The family history is unremarkable.

Exam: T 37.0°C, P 120 beats/min, RR 48 breaths/min and oxygen saturation 98% on room air. He is alert, active and exhibits no acute distress. His weight and height are within the fiftieth percentile. His anterior fontanel is soft and flat. His eyes and ears are normal. His lips are moist and pink. There is an audible inspiratory stridor noted in the supine position, which does improve with extension of his neck. No nasal flaring is visible, and his nares appear patent. There are no intercostals retractions or pectus abnormalities. His lungs are clear to auscultation once the stridor clears with airway repositioning. His heart rate is regular without murmurs. His color, perfusion, capillary refill and peripheral pulses are good.

* * * * *

Q1. What examination is missing?

A flexible fiber-optic laryngoscopy should be performed to complete the examination.

Q2. What is stridor?

Stridor is an abnormal, high-pitched sound produced by turbulent airflow through a partially obstructed airway at the level of the supraglottis, glottis, subglottis or trachea.

Q3. What are the types of stridor?

1. Inspiratory stridor: high pitched, usually caused by supraglottic obstruction
2. Bi-phasic stridor: intermediate pitched, caused by glottis, subglottic or extrathoracic, tracheal obstruction
3. Expiratory stridor: usually associated with retraction of the sternum, intercostal spaces and suprasternal tissue and caused by intrathoracic tracheal obstruction

Q4. What is the most likely diagnosis?

Laryngomalacia.

Q5. What is the most common cause of stridor in infants?

Laryngomalacia.

Q6. How was the diagnosis confirmed, and how he was followed up?

Flexible fiber-optic laryngoscopy was performed to confirm the diagnosis and to exclude other possibilities. His status was monitored clinically. He continued to feed well and gain weight appropriately. All of his symptoms resolved by 18 months of age.

Q7. The worried parents ask for more information about laryngomalacia.

Laryngomalacia (soft larynx) is a condition in which the soft, immature tissue of the upper larynx collapses inward during inhalation, causing partial airway obstruction. It is the most common congenital anomaly of the larynx and represents 60% of all congenital laryngeal anomalies. Males are affected twice as frequently as females. Laryngomalacia is generally a self-limiting disorder. However, severe cases of laryngomalacia can lead to failure to thrive and life-threatening dyspnea.

Q8. What is the etiology of laryngomalacia?

The exact etiology is unclear. However, theories include maldevelopment of the cartilaginous structures of the airway and immature neuromuscular control.

Q9. What are the symptoms of laryngomalacia?

Symptoms are typically absent at birth and arise at 2 to 4 weeks of age.

1. Common symptoms include:
   - Inspiratory stridor, which is worsened in the supine position and with agitation
   - Feeding difficulties.
2. Rare symptoms include chest deformities, obstructive apnea and failure to thrive.

Q10: What are the findings upon laryngoscopy?

Flexible laryngoscopy is performed while the patient is awake. Laryngoscopy typically reveals an elongated and laterally

extended epiglottis (omega-shaped) that falls posteriorly on itself during inspiration (Figure 23.1). Visualization also reveals inward collapse of the aryepiglottic folds (cuneiform cartilages) during inspiration and bulky arytenoids that prolapse during inspiration.

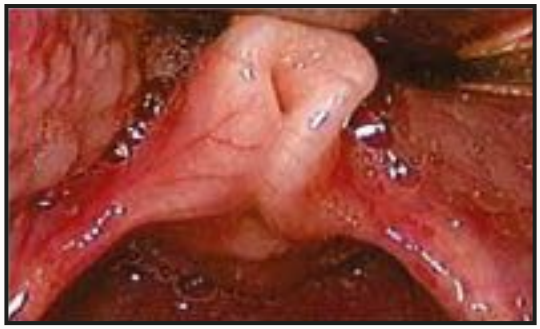

Figure 23.1: *Omega-shape epiglottis during rigid laryngoscope examination.*

Q11. When is the indication of performing a rigid laryngobronchoscopy?

Rigid laryngoscopy (Figure 23.2) and bronchoscopy are performed in certain circumstances:

- If you suspect other congenital laryngeal (e.g., laryngeal cleft) or tracheal anomalies.
- If the child is not improving with time, fails to thrive or has choking symptoms.

Figure 23.2: *A rigid laryngoscope with suspension.*

Q12. What are the other causes of stridor?

Vocal fold paralysis, which is the second most common congenital anomaly of the larynx and accounts for approximately 20% of laryngeal lesions (Figure 23.3)

- Congenital subglottic stenosis (SGS), which is the third-most common congenital anomaly of the larynx and accounts for approximately 15% of all cases
- Tracheomalacia, in which the trachea lacks firmness, thus causing the anterior and posterior walls to come together during respiration and decreasing the tracheal lumen
- Laryngeal webs: the most common are vocal fold webs (Figure 23.4)
- Laryngeal cysts: the most common is a supraglottic cyst
- Hemangiomas: the most common of which are subglottic
- Papillomata of the larynx (Figure 23.5)
- Laryngo-tracheo-esophageal clefts and fistulae
- Vascular abnormalities: double aortic arch, innominate artery compression and aberrant right subclavian vein
- Bronchogenic cyst
- Tracheal stenosis

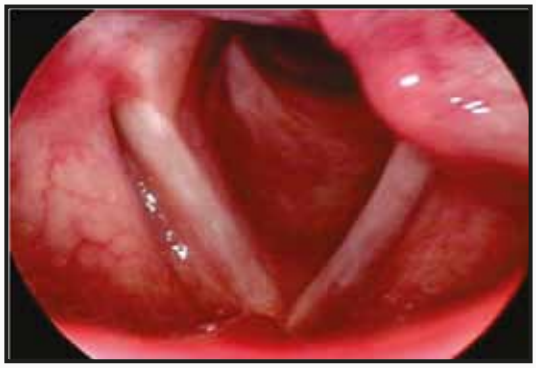

Figure 23.3: *Left vocal cord paralysis*

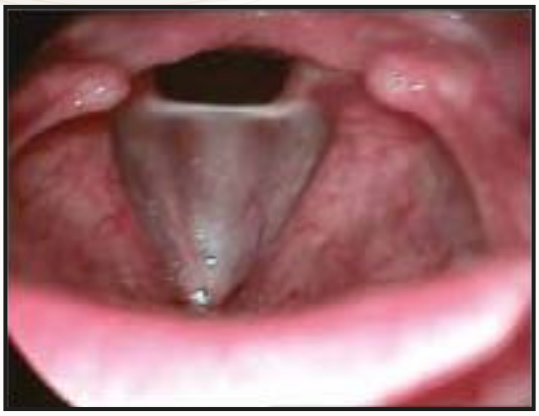

Figure 23.4: *Vocal folds web*

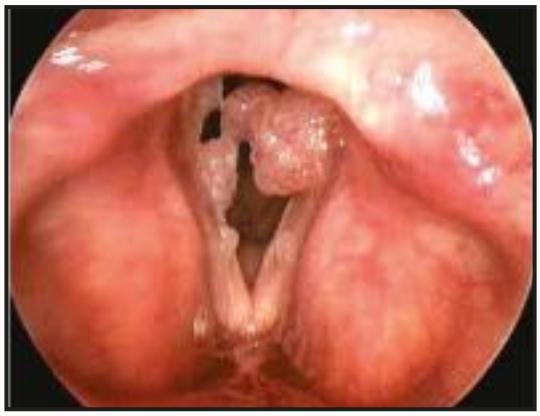

Figure 23.5: *Laryngeal papillomatosis*

Q13. What are the management options?

1. Conservative management

   Because the condition is usually self-limiting, most patients respond to conservative treatment:

   - Reassuring the parents of the favorable prognosis
   - Adjusting positions more frequently when the baby is supine or agitated
   - Undergoing frequent evaluation by the pediatrician to assess growth, feeding and breathing

2. Surgical management

   Surgery is rarely necessary but indicated if the following are present:

   - Life-threatening airway obstruction
   - Inability to feed orally
   - Cor pulmonale
   - Failure to thrive

Q14. What are the surgical procedures for laryngomalacia?

- Supraglottoplasty (Figure 23.6)
- Epiglottoplasty
- Tracheostomy

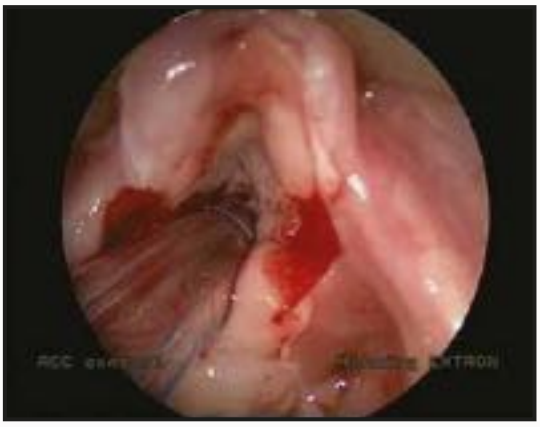

Figure 23.6: *Division of aryepiglottic folds using a CO_2 laser (supraglottoplasty). (Courtesy of Dr. Basel AlSabah)*

Take-home message

Congenital airway anomalies must be considered when evaluating stridor in infancy. The goal is to distinguish life-threatening conditions from self-limited conditions. With a thorough history, physical examination and fiber-optic laryngoscopy, a proper diagnosis can be established.

CHAPTER 24

Difficulty of Breathing

Prof. Ahmed Al Ammar

An 8-year-old child was involved in a traffic accident 3 months ago. The child lost consciousness and was intubated for 3 weeks. After extubation, the child was breathing well but gradually started to develop noisy and difficult breathing. The child was diagnosed and treated as a case of bronchial asthma. However, medical treatment failed to resolve his symptoms completely.

On presentation in the clinic, the child is found to have bi-phasic stridor and is in mild distress using accessory muscles with mild suprasternal recession. A flexible fiber-optic examination reveals a normal supraglottic region with normal vocal folds mobility. A lateral soft-tissue radiography (HKV) suggests a narrowing of the sub-glottic region (Figure 24.1). Rigid bronchoscopy (Figure 24.2) is performed, which confirms the diagnosis of subglottic stenosis (SGS) (Figure 24.3).

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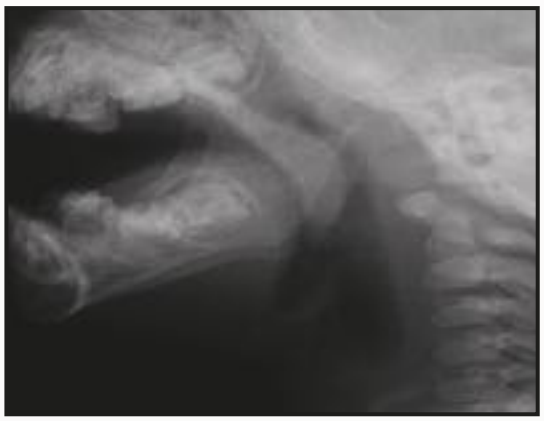

Figure 24.1: *Lateral soft-tissue X-ray.*

Figure 24.2: *Bronchoscopy*

Q1. What are the criteria for confirming a diagnosis of congenital SGS?

The diagnosis of congenital SGS can be made when the luminal diameter is

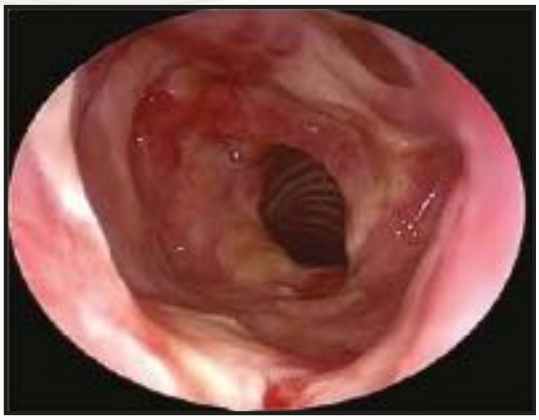

Figure 24.3: *Laryngobronchoscopy revealing SGS*

less than 4 mm at birth in a full-term infant or 3 mm in a premature infant.

Q2. Why is the subglottic region more susceptible to trauma?

Several factors confer increased vulnerability of the subglottic area to trauma:

- Its narrow diameter because it is framed by the cricoid cartilage
- Inextensibility and the frailty of the lining tissue
- Poor micro-vascularization

Q3. How do you classify SGS?

The most common grading system was proposed by Cotton et al.:

- Grade I: narrowing of up to 50% of the lumen
- Grade II: narrowing from 51% to 70% of the lumen
- Grade III: narrowing from 71% to 99% of the lumen
- Grade IV: no detectable lumen

Q4. What is the importance of radiological evaluation for SGS?

Radiologic assessment is very helpful in managing all cases of SGS. Radiologic assessment is the initial investigation to be performed for cases without respiratory distress. Simple lateral soft-tissue radiography can help with the diagnosis and localization of the site of narrowing. The CT scans and MRIs may help to define the status of the cartilaginous framework and assess the extent of the narrowed segment. The CT scans and MRIs may also rule out the possibility of a mass compressing the region from outside the lumen.

Patients with swallowing problems need to be further evaluated, e.g., with a functional endoscopic evaluation of swallowing (FEES). The evaluation will help to rule out aspiration, which can be a distressing post-operative situation.

Q5. How do you secure the airway in an emergency situation?

A high level of coordination between the anesthesiologist and the surgeon is required to safely secure the airway in emergency situations. It is important to discuss the best method for securing the airway before the patient is under anesthesia. Blind attempts at intubation should be avoided because they can traumatize the small airway and result in edema, which can cause complete obstruction of the compromised airway.

In many occasions, a tracheotomy under local anesthesia may be the best way to secure the airway in a severely distressed patient can traumatise the small airway and result in oedema, which can cause complete obstruction of the compromised airway.

At times, a tracheotomy under local anaesthesia may be the best way to secure the airway in severely distressed patients.

Q6. What are the most common medical conditions that can affect the outcome of surgical interventions for SGS?

Important factors need to be considered before performing a definitive surgical intervention for SGS. Gastroesophageal reflux is a common morbidity that can affect healing after surgical intervention. Commonly, pH monitoring must be performed for all patients before surgical intervention. Eosinophilic esophagitis is a rare situation that has been reported as a possible reason for failure. Similarly, patients need to be screened for possible infection with methicillin-resistant *Staphylococcus aureus* (MRSA) at the site of the tracheotomy.

Q7. What are the treatment options?

The management of acquired pediatric SGS remains a considerable challenge for otolaryngologists. The surgical options of tracheotomy, cricoid split, laryngotracheal reconstruction (LTR) and crico-tracheal resection (CTR) are well established. Cricoid split may be considered an option for mild cases in infants. The open techniques of LTR and CTR are considered for more severe stenosis. They often require prolonged intubations and lengthy intensive care unit admissions and carry significant risks for serious complications. Open reconstructions often necessitate adjuvant procedures for ultimate success. The use of LTR (Figure 24.4) may involve using anterior or posterior grafts or both with the consequence of using an endolaryngeal stent for a variable period in the case of a staged procedure. The LTR may be a good choice if the stenosis involves the glottic region. However, CTR (Figure 24.5) is more favorable than LTR in cases with loss of the cartilaginous framework.

More recently, otolaryngologists have revisited less invasive endoscopic procedures as both primary and adjuvant treatments for pediatric SGS. Endoscopic dilation has historically been performed with metal dilators or endotracheal tubes, which generate significant shearing force across the stenotic segment.

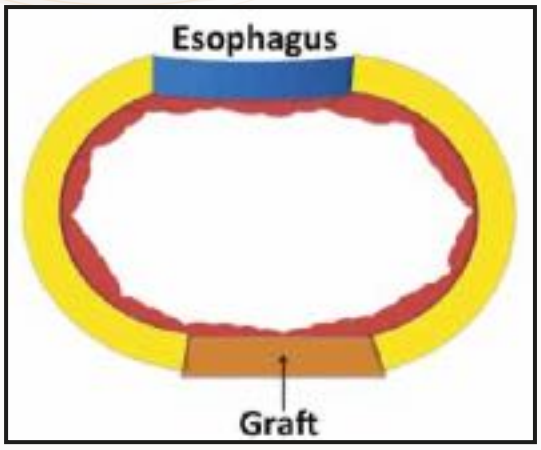

Figure 24.4: *An LTR with anterior and posterior grafts*

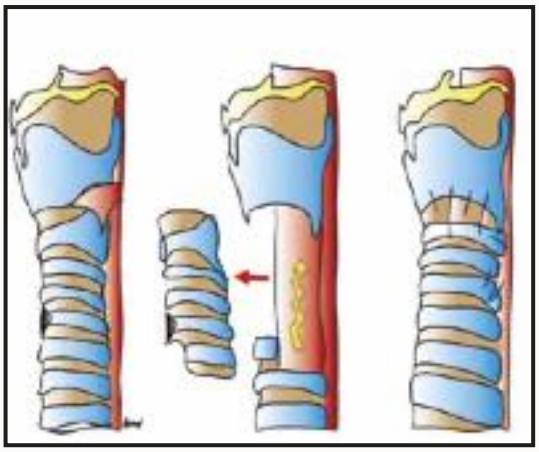

Figure 24.5: *The stages of CTR*

CHAPTER 25

Neonatal Respiratory Distress

Dr. Talal AlKhatib

A newborn girl is admitted to the neonatal intensive care unit (NICU) soon after delivery because of respiratory distress. The child was born at term via spontaneous vaginal delivery to a healthy 22-year-old mother. The baby weighed 3,520 grams and had Apgar scores of 6 at 1 minute after birth and of 8 at 5 minutes. Immediately after birth, it was noted that the girl had central cyanosis, nasal flaring, tracheal tugging and intercostal recessions. The cyanosis improves with crying, as does her oxygen saturation (SpO_2). Once the girl stops crying, the cyanosis and labored breathing recur, and her SpO_2 decreases to 80% on pulse oximetry. Chest auscultation is normal. A portable chest X-ray shows no abnormal findings. An attempt at passing a size 6 French nasal-suction catheter fails to progress more than 30 mm into the nose. An oral airway is inserted and appears to stop the cyclical cyanosis. A presumptive diagnosis of an upper airway obstruction is made, and an otolaryngologist is consulted. Examination of the oral cavity, oropharynx and anterior rhinoscopy reveals no abnormalities.

* * * * *

Q1. At what anatomical location is the upper airway obstruction occurring in this neonate?

Neonates are obligate nasal breathers. Cyclical cyanosis implies that mouth breathing relieves the obstruction. Therefore, the location of the obstruction would be above the oral cavity level, namely in the nose or the nasopharynx.

Q2. What are the differential diagnoses in this neonate?

- Choanal atresia (bilateral)
- Congenital nasal pyriform aperture stenosis (CNPAS)
- Nasolacrimal duct cyst (dacrocystocele)
- Congenital nasal masses (encephalocele, glioma, dermoid and hemangioma)

- Nasopharyngeal teratoma
- Arhinia (absent nose)
- Mid-facial hypoplasia

Q3. What is the most likely diagnosis?

Bilateral choanal atresia is the most likely diagnosis. Failure to pass a small suction catheter (size 6F) beyond the nasopharynx supports this diagnosis.

Q4. How would the otolaryngologist confirm the diagnosis?

The otolaryngologist can directly visualize the atretic plate by performing a nasal endoscopy.

Q5. What test would confirm the diagnosis?

A CT scan of the nose and paranasal sinuses (Figure 25.1) is diagnostic. The scan is also helpful in estimating the nature of the atretic plate(s) (i.e., bony vs. membranous) and the thicknesses of the vomer and the lateral pterygoid plates.

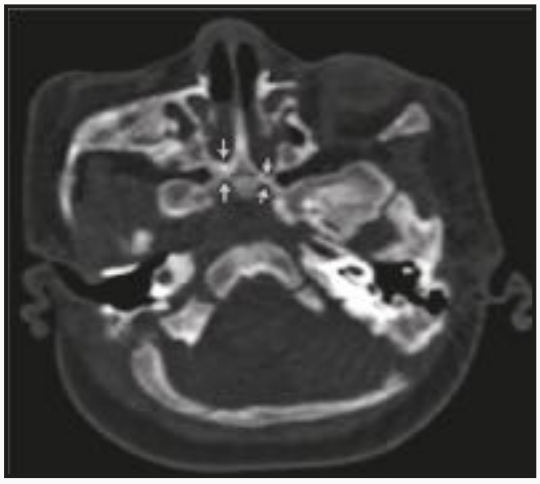

Figure 25.1: *Axial CT scan of the nose and paranasal sinuses showing bilateral choanal atresia (arrows). Note: both atretic plates are bony. The vomer is abnormally thick, and the right lateral pterygoid plate is medialized.*

Q6. What is the pathogenesis of choanal atresia?

- Persistence of the buccopharyngeal membrane.
- Failure of the nasobuccal membrane of Hochstetter to rupture at 6 weeks of gestation.

Q7. Can choanal atresia be unilateral?

Yes. Approximately 65-75% of atresias are unilateral. Unilateral atresia can be asymptomatic or present later in life with persistent tenacious nasal discharge. Therefore, management is not urgent and is usually delayed.

Q8. What is the management plan for bilateral choanal atresia?

- Establish an oropharyngeal or orotracheal airway.
- Start gastric feeding.
- Surgical repair (usually delayed several weeks until adequate facial growth has occurred).

Q9. How can choanal atresia be repaired?

- Endoscopic transnasal approach (preferred technique at present) (Figure 25.2)
- Transpalatal approach (higher risk of dental/facial growth abnormalities)
- Trans-septal approach (for unilateral atresia)

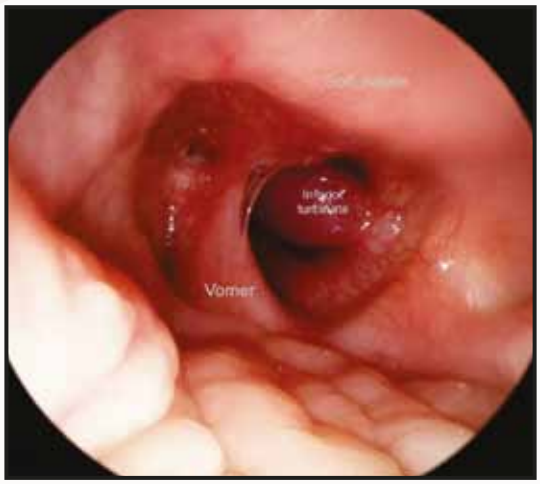

Figure 25.2: *A view of unilateral choanal atresia through a 120-degree angled nasopharyngoscope. Note the patient side showing the posterior end of the inferior turbinate.*

Q10. What syndromes are associated with choanal atresia?

- CHARGE syndrome (up to 75% association with bilateral cases)
- Crouzon syndrome
- Treacher Collins syndrome
- 22q11.2 deletion syndrome

Q11. What does the CHARGE acronym stand for?

- **C**oloboma
- **H**eart defects
- **A**tretic choana
- **R**etardation of growth
- **G**enitourinary disorders
- **E**ar abnormalities

Q12. What syndromes are associated with upper airway obstruction and at what level?

Syndrome	Location of upper airway obstruction
CHARGE	Bilateral choanal atresia
Crouzon and Apert	Mid-facial hypoplasia (maxilla)
Down (Trisomy 21)	Macro-glossia, SGS
Pierre Robin sequence	Micro-gnathia, glossoptosis
Treacher Collins	Mid-facial hypoplasia (zygoma, mandible)
Velo-cardio-facial (22q11.2 deletion)	Glottic web

Q13. What are the steps in managing upper airway obstruction in syndromic children?

- Insertion of oral or oropharyngeal airway
- Endotracheal intubation (orotracheal)
- Tracheotomy

Q14. The following infant (Figure 25.3) has a cleft palate and glossoptosis. What is the syndrome?

Pierre Robin sequence or syndrome

Figure 25.3: *Picture courtesy of Dr. Hasan Al-Ajmi, Montreal Children's Hospital, McGill University, Montreal Canada.*