ANAESTHESIA IN UPPER RESPIRATORY OBSTRUCTION

BY

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SUMMARY

The causes of upper respiratory obstruction are reviewed. The symptoms and signs are graded according to severity. It is stressed that cyanosis indicates very severe obstruction, and that stridor at rest is a very informative local sign of severe obstruction. In severe obstruction respiratory depressant drugs and atropine are contraindicated while helium may be helpful. When general anaesthesia is necessary in severely obstructed patients, the obstruction may first be bypassed by nasotracheal or orotracheal intubation, tracheostomy or bronchoscopy. Each of these can be performed with local anaesthesia. The value of blind nasal intubation in the conscious patient cannot be over-stressed. When tracheostomy is indicated it should be done early, as an elective procedure.

Maintenance of a clear airway is a fundamental principle of anaesthesia. Obstruction of the upper respiratory tract, occurring anywhere from the lips or nostrils to the carina, is a danger to life. The degree of obstruction is likely to be increased by anaesthesia. This problem has engaged the attention of anaesthetists since the early days of anaesthesia, as shown by the following references.

Sansom (1866) was one of the first to emphasize the dangers of chloroform in cases requiring tracheostomy or laryngotomy, and in 1912 Sir Frederick Hewitt wrote: “There are perhaps no cases demanding greater care and experience on the part of the anaesthetist than those in which extensive cellulitis of the submaxillary or cervical regions is present.” The dangers of general anaesthesia in abscesses related to the upper respiratory tract were stressed by Layton (1913). Thirty-one cases of Ludwig’s angina were reviewed by Williams (1940). Five of these required emergency tracheostomy during anaesthesia, and one patient died while this was being performed. Bennett (1943) reported on anaesthesia in six cases of submandibular abscess. Death occurred in one patient during the induction of anaesthesia and another required emergency tracheostomy because of failure to intubate.

In a series of 1,000 deaths associated with anaesthesia, Edwards et al. (1956), reported that 463 were reasonably certain due to anaesthesia and, of these, six were associated with pre-operative respiratory obstruction. Of the latter, three cases had inflammatory oedema of the glottis, one had carcinoma of the tongue, one had an obstructive lesion of the larynx and the other thyrotoxicosis with deviation of the trachea. Boyan and Howland (1959) reviewed 839 patients who underwent total laryngectomy and 190 who underwent partial laryngectomy. Of this total, twenty-seven patients required emergency tracheostomy because of intubation difficulties.

In this paper, the causes and effects of upper respiratory obstruction are reviewed, the assessment and anaesthetic management discussed and some illustrative cases described.

THE CAUSES OF UPPER RESPIRATORY OBSTRUCTION

For the purpose of this paper the upper respiratory tract is divided into five regions: the mouth, nose, pharynx, larynx, and trachea.

Any lesion of the upper respiratory tract, above the level of the posterior part of the tongue, may obstruct the oral or nasal airway, but rarely both. The pre-anaesthetic obstruction of the alternative airway increases the likelihood of serious obstruction during anaesthesia.

Oral Obstruction.

Congenital abnormalities limiting the oral airway include microstomia, macroglossia and hypoplasia of the mandible with prolapase of the tongue. Tumours of the tongue and jaws encroach upon
The oral cavity. Inflammatory conditions of the jaws and teeth obstruct by virtue of the bulk of the inflammatory reaction and may also cause trismus.

Ludwig's angina is an inflammatory condition of the submaxillary space, which obstructs the airway, by pushing up the tongue, by trismus and by oedema of the tissues around the glottis. Trauma in the mouth leads to swelling and the occurrence of bleeding further threatens the airway. Burns and scalds inevitably cause oedema and later scarring. Hypersensitivity phenomena such as angioneurotic oedema and insect stings may cause gross swelling of the tongue or lips.

Neurological disorders which may give rise to oral obstruction include bilateral hypoglossal nerve palsy causing prolapse of the tongue and tetanus with trismus. The opening into the mouth may be scarred into a small aperture or obstructed by a plastic procedure such as the creation of an Abbé flap. Conditions which affect the oral airway, by limiting the movement of the jaw, include temporo-mandibular arthritis and the wiring together of the jaws which is often practised in the treatment of the fractured mandible.

Nasal Obstruction.

Congenital absence of the nose has been recorded and the nasal airway may be obstructed from birth by post-choanal atresia or by marked deviation of the septum. Neonatal nasal obstruction is relatively serious as it interferes with feeding and the neonate does not readily adopt the oral airway. Polyps and other neoplasms, such as haemangioblastoma, fibromata and dentigerous cysts, may limit the nasal airway. The common cold is a familiar cause of blocked nose and the nostrils may be obstructed by other infections. Hypertrophy of the adenoids is the commonest cause of chronic nasal obstruction in childhood.

Fracture of the nasal bones leads to much swelling and narrowing of the nasal passages. Hay fever and other forms of allergic rhinitis lead to difficulty in nasal breathing. Children commonly insert foreign bodies such as beads and beans into the nostrils.

Pharyngeal Obstruction.

The pharynx may be congenitally obstructed by cystic hygroma, branchial cysts, or by aberrant thyroid tissue and cysts at the base of the tongue. Carcinoma of the pharynx is not uncommon. Abscesses may arise in the peritonsillar and retro-
pharyngeal regions and the resulting oedema may involve the glottis. These infections are much less common since the introduction of antibiotics.

Children may swallow corrosive fluids which cause acute oedema and later scarring. Occasionally after thyroidectomy or parathyroidectomy extensive scarring develops in the tissues around the glottis, causing intubation difficulties during anaesthesia.

Paralysis of the pharyngeal musculature is seen in myasthenia gravis, pseudobulbar palsy, in lesions of the vagi and in progressive bulbar paralysis. The last-named may result from motor neurone disease, poliomyelitis, arteriosclerosis, tumours, disseminated sclerosis or syringomyelia. Reduction in the efficiency of swallowing and laryngeal reflexes makes likely the aspiration of foreign material into the tracheobronchial tree.

Angioneurotic oedema may cause massive swelling of the uvula and posterior pharyngeal wall. Fish and meat bones commonly lodge in the mucosa of the pharynx and may result in much swelling. Leathard (1960) has drawn attention to the massive extravasations of blood which may develop in the tongue, floor of the mouth, and around the pharynx and larynx in haemophilia and other haemorrhagic diatheses. The maximum oropharyngeal airway is obtained by extension of the head in the midline. Conditions which prevent this are potential causes of respiratory obstruction. They include rigid fixation of the neck as a result of osteoarthritis, rheumatoid spondylitis, kyphoscoliosis and torticollis. Causes resulting from medical treatment include surgical fusion of the cervical vertebrae and the encasing of the head and neck in plaster of Paris.

Lipoid storage disorders such as Niemann-Pick disease, Gaucher’s disease and lipochondrodystrophy may lead to gross lymphoid hyperplasia in the pharynx (Bougas and Smith, 1958).

**Laryngeal Obstruction.**

Eckenhoff (1951) has described the features of the infant larynx which are of interest to the anaesthetist. The organ is funnel-shaped with its narrowest diameter at the cricoid ring. In congenital stenosis the cricoid cartilage is the site of obstruction. The infant larynx may be the site of congenital webs which bridge the anterior parts of the vocal cords. Laryngoceles and laryngeal cysts may also be present from birth.

Congenital laryngeal stridor, or laryngomalacia, is a transient form of stridor occurring in infants and associated with an abnormal flaccidity of the tissues of the larynx. With each inspiration the epiglottis is folded and drawn into the glottis. There may be, in addition, laxity of the mucosa of the aryepiglottic folds. This condition may be encountered by the anaesthetist attempting intubation in the infant. The abnormality improves with growth and increasing rigidity of the laryngeal cartilages and usually disappears in the second year of life.

Carcinoma of the larynx is one of the commonest causes of upper respiratory tract obstruction of concern to the anaesthetist. Laryngeal polyps and papillomata occur. The latter may be multiple and recurrent in juveniles, necessitating repeated surgery. Cysts of the epiglottis are usually small but may become grossly enlarged (Norris, 1957).

Infections of the larynx may compromise the airway by causing oedema. Acute laryngitis is a common affliction but usually causes negligible respiratory obstruction in the adult. Laryngotracheobronchitis occurring in infants is a serious disease and respiratory obstruction results from oedema and laryngeal spasm. Epiglottitis is a disease characterized by enormous enlargement of the epiglottis and septicemia with Haemophilus influenzae as the causal organism in most cases. It is rapidly progressive and tracheostomy is usually required.

Laryngeal diphtheria, tuberculosis and syphilis

<table>
<thead>
<tr>
<th><strong>Table IV</strong> Causes of laryngeal obstruction.</th>
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<tbody>
<tr>
<td><strong>Congenital</strong></td>
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<tr>
<td><strong>Neoplastic</strong></td>
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<td><strong>Inflammatory</strong></td>
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<td><strong>Traumatic</strong></td>
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<td><strong>Neurological</strong></td>
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<tr>
<td><strong>Allergy</strong></td>
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<td><strong>Foreign bodies</strong></td>
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are now uncommon. Unusual inflammatory lesions causing laryngeal obstruction include cricoarytenoid arthritis, perichondritis, acute thyroiditis and leprosy.

Injuries to the larynx may result from external or internal violence. In the latter case endotracheal tubes, laryngoscopes and bronchoscopes are possible agents. Trauma to the laryngeal cartilages may lead to stenosis and laryngotomy is no exception.

Bilateral midline abductor paralysis of the larynx, whether due to recurrent laryngeal nerve damage or central causes, leads to respiratory obstruction in the conscious patient. Other forms of laryngeal paralysis may not cause respiratory difficulties in the conscious patient but predispose to laryngeal obstruction in the anaesthetized. Laryngeal spasm is a complication with which anaesthetists are familiar. Fink (1956) has discussed the aetiology and treatment. Other neurological causes of laryngeal obstruction are laryngismus stridulus and laryngeal tetany.

Glottic oedema may result from the inhalation of hot or irritant gases and from insect stings and hypersensitivity phenomena, such as angioneurotic oedema and food and drug allergies. The larynx may also be the site of foreign body obstruction.

Tracheal Obstruction.
The trachea may be the site of congenital stenosis or webs. It may be compressed by anomalies of the aortic arch (Kirklin and Clagett, 1950).

Primary malignant disease of the trachea is uncommon but secondary invasion may occur from carcinoma of the thyroid or upper oesophagus. Papilloma of the trachea is uncommon but may be associated with multiple papillomata throughout larynx, trachea and bronchi (Stein and Volk, 1959).

<table>
<thead>
<tr>
<th>Causes of tracheal obstruction.</th>
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<tbody>
<tr>
<td>Congenital</td>
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<tr>
<td>Stenosis; webs; vascular rings.</td>
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<td>Neoplastic</td>
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<tr>
<td>Mediastinal tumours; carcinoma of thyroid or oesophagus.</td>
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<td>Inflammatory</td>
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<td>Tracheitis.</td>
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<td>Traumatic</td>
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<td>Post-tracheostomy stenosis.</td>
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<td>Foreign body</td>
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<td>Rare.</td>
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<td>Other causes</td>
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<td>Aneurysm of aortic arch; collapse of trachea.</td>
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Injury involving tracheal cartilages may lead to stenosis. High tracheostomy is more likely to be followed by stenosis than a low tracheal incision. Occasionally, collapse of the tracheal cartilages follows the relief of chronic compression of the trachea. Foreign bodies small enough to pass through the glottis often lodge in the main bronchi rather than the trachea. Smaller foreign bodies may move up the trachea with expiration and down with inspiration.

Allergic responses causing bronchospasm often involve the trachea in a similar process. Many of the lesions which have been discussed here require surgical intervention in the form of tracheostomy. Any lesion of the upper respiratory tract will, however, complicate anaesthesia for surgery unrelated to the respiratory system.

SYMPTOMS AND SIGNS OF UPPER RESPIRATORY TRACT OBSTRUCTION

The symptoms and signs of obstruction of the upper respiratory tract depend on the site and severity of the obstruction. Local effects are produced by the lesion and in severe obstruction the general manifestations of asphyxia are present.

The symptoms and signs of respiratory obstruction may be graded according to severity as shown in table VI (modified from Forbes, 1961).

<table>
<thead>
<tr>
<th>Stage I (Mild or potential obstruction)</th>
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<tr>
<td>Hoarseness; cough; stridor on moderate exertion; oral obstruction alone; nasal obstruction alone.</td>
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<th>Stage II (Moderate obstruction)</th>
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<tr>
<td>Stridor on slight exertion; rib retraction on inspiration; accessory muscles of respiration in use; alae nasi dilating on inspiration; indrawing of soft tissues of neck; jaw and trachea tugged down on inspiration; dyspnoea.</td>
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<th>Stage III (Severe obstruction)</th>
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<tr>
<td>Stridor at rest; apprehension; restlessness; sleeplessness; sweating; pallor; increase of pulse rate and blood pressure; exaggerated excursion of neck veins.</td>
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<th>Stage IV (Very severe obstruction)</th>
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<td>Slowed respiration; hypotension; cyanosis; impairment of consciousness.</td>
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General symptoms and signs.
Asphyxia is a combination of hypoxia and carbon dioxide retention. Its effects on the central nervous system produce apprehension in the adult and restlessness in the child.

Moderate obstruction necessitates the use of the
accessory muscles of respiration in an effort to maintain normal ventilation. Only the scalene and sternomastoid muscles show significant activity as accessory muscles of respiration in man (Campbell, 1958). These muscles are under voluntary control so that sleeplessness results from severe obstruction. Observation of the patient and palpation of the neck will quickly reveal whether the accessory muscles are in use. Their action is often accompanied by dilatation of the alae nasi on inspiration.

Dyspnoea has been defined by Meakins (1934) as "consciousness of the necessity for increased respiratory effort". It is frequently caused by mechanical difficulties in ventilating the lungs. One measurable facet of dyspnoea is the work of breathing. Upper respiratory tract obstruction implies narrowing of the airway and hence increased resistance to airflow; the work of breathing is increased and this leads to dyspnoea. This increase in the work of breathing results in greater oxygen consumption and energy expenditure. A vicious circle is set up. The mechanical efficiency of the respiratory muscles is low (5-10 per cent) so that much of this energy is dissipated as heat; sweating is a common accompaniment of respiratory obstruction.

When the airway is inadequate, the exaggerated actions of the respiratory muscles produce a variety of effects. The muscular efforts to expand the chest increase the negative intrathoracic pressure normally present during inspiration and the intrathoracic pressure becomes increasingly positive during expiration. These pressure changes are reflected in the jugular veins which may be seen exaggeratedly filling and emptying. The soft tissues between the ribs, the supraclavicular and suprasternal areas, may be indrawn during inspiration and pushed out during expiration. The chest expansion remains small in spite of these efforts.

The diaphragm acts very forcibly in these situations and may draw in the lower ribs during inspiration. This may be particularly noticeable in the flexible rib cage of the child. The jerk of the diaphragm in its efforts to descend may be transmitted via the fibrous pericardium and the deep fascia of the neck to the trachea and even the lower jaw—the familiar tracheal tug.

In acute and severe obstruction, hypoxia dominates the situation and respiratory failure is quickly followed by circulatory failure. In chronic obstruction the effects of carbon dioxide retention may overshadow those of hypoxia. This results in increases in pulse rate and blood pressure. Severe and prolonged obstruction will result in circulatory failure.

Cyanosis is an unreliable index of the oxygen saturation of the blood. The average physician will recognize it when the arterial oxygen saturation has fallen to within 75-85 per cent. Well known are the difficulties of assessing cyanosis in the presence of anaemia, regional slowing of bloodflow and pigmentation of the skin. In the absence of other complicating factors, cyanosis is a late sign of upper respiratory tract obstruction and indicates very severe restriction of the airway.

Local signs.

The local signs of respiratory obstruction indicate the site of the lesion and may point to its severity. The patient may be able to localize accurately the site of his airway obstruction. Palpation and auscultation of the neck may reveal the level of obstruction.

It is a simple matter to test the patency of the nostrils by their alternate occlusion. The oral cavity is easily inspected, as is the oropharynx. This should be done routinely before any anaesthetic procedure.

Difficulty in swallowing obviously suggests a pharyngeal or oesophageal lesion, which may compromise the airway.

The quality of the voice may provide useful information. Patency of the nasal passages is necessary for resonance. Lesions in the mouth cause defects in articulation, while pharyngeal affections may cause a moist guttural voice. Hoarseness directs attention to the larynx. Lesions below the level of the vocal cords may be associated with a weak but clear voice.

The croupy cough of the child with laryngitis is a familiar sound. Other afflictions of the upper respiratory tract commonly produce a reflex non-productive cough. Bilateral adductor paralysis of the larynx is evidenced by a non-explosive type of cough which has been described as bovine. Tracheal lesions may produce a rough, barking cough, worse at night. Classically, aneurysm of the aortic arch produces a brassy cough.

Stridor is the most important localizing physical sign indicating serious obstruction in either larynx,
Stridor is a harsh noise produced by respiratory air passing through an obstructed main air passage. Airflow through the normal upper respiratory tract is mainly laminar but severe obstruction causes turbulence and stridor is produced by the vibration of soft tissues in a turbulent airflow. The vibrating surface may be the vocal cords or the mucosa of the larynx, trachea or bronchus.

There is a certain critical velocity below which airflow is laminar. Above this, flow becomes turbulent (Dean and Visscher, 1941). A patient with mild obstruction may therefore have no stridor at rest, but stridor may result from exercise, dyspnoea, apprehension or pain because of the increased airflow through the obstruction. It has been said that the patient with stridor going upstairs may need tracheostomy, one with stridor walking on the flat will need tracheostomy and the patient with stridor at rest does need tracheostomy (Watkin-Thomas, 1953).

Stridor is best listened for by having the patient breathe through the half-opened mouth. It may be mainly inspiratory or expiratory and this gives a clue to the site of its origin (Wilson, 1952). Obstruction at or above the vocal cords produces predominantly inspiratory stridor. This may be due to the relative lack of support of the soft tissues above the glottis and their tendency to be drawn into the airway by the negative intrathoracic pressure. Bronchial obstruction commonly gives rise to expiratory stridor; this may be correlated with the decrease in bronchial diameter during expiration and an increase in diameter during inspiration. Severe tracheal obstruction and lesions of the larynx below the cords usually produce inspiratory and expiratory stridor.

Stridor is often the presenting symptom of laryngeal disease in infants. Early lesions of the adult larynx are more likely to cause hoarseness.

Investigations.

Indirect examination of the glottis provides information of great value to the anaesthetist. The factors of particular interest are the site and extent of the lesion, its consistency, and whether it bleeds easily, the degree of obstruction and the movement of the vocal cords.

Routine chest films often reveal tracheal deviation and compression likely to lead to intubation difficulties. Radiological examination of the larynx may be useful. The epiglottis, pharynx, pyriform fossae and aryepiglottic folds can often be seen in a lateral view. Anteroposterior tomograms may reveal the extent of lesions below the cords (Sheehan et al., 1960, and figure 1).

Upper respiratory obstruction usually results in prolongation of inspiration and expiration, and reduction of tidal volume. Vital capacity usually remains within normal limits. Timed vital capacity and maximum breathing capacity are progressively reduced with increasing airway obstruction. Residual volume is often increased.

Consideration of the signs, symptoms and investigations dealt with here should enable the anaesthetist to assess the severity of upper respiratory obstruction in most cases.

PREPARATION FOR ANAESTHESIA

Premedication of patients with upper respiratory obstruction merits careful attention. Atropine stimulates the cortex, raises oxygen requirements and increases the viscosity of secretions so that they are more likely to clog a diminished airway. The drug is therefore contraindicated in moderate or severe obstruction.

There is disagreement on the place of sedation in these cases. There is no doubt that the use of powerful respiratory depressants is dangerous in the presence of severe obstruction. In mild or moderate obstruction, however, there are those who believe that effective sedation leads to lessening of anxiety and apprehension and reduction in oxygen requirements. The antagonists of this view regard anxiety and apprehension as signs of hypoxia and contraindications to sedation. The writer believes that the opium alkaloids and the synthetic narcotic analgesics should not be used in the presence of respiratory obstruction, but that the phenothiazine derivatives in carefully assessed doses will often produce effective sedation without serious respiratory depression.

In severe respiratory obstruction when airflow has become turbulent, the factors determining the resistance to flow are the dimensions of the remaining airway and the density of the flowing gases. A mixture of 80 per cent helium with 20 per cent oxygen has a density of 0.33 compared with 1.0 for air. The resistance to flow with the helium and oxygen mixture is therefore approximately one-third of that for air.
In mild respiratory obstruction, where, with the patient at rest, airflow is predominantly laminar, the viscosity of the flowing gases is the relevant factor. As a mixture of 80 per cent helium and 20 per cent oxygen has a viscosity of 1.11 compared with 1.0 for air, helium offers no advantage in mild respiratory obstruction.

Breathing a mixture of helium and oxygen may improve oxygenation in patients with severe respiratory obstruction and the inhalation of such a mixture should be a useful preliminary to anaesthesia.

ANAESTHETIC MANAGEMENT

Oral obstruction.

When the opening into the mouth or the oral cavity is obstructed, the nose can often be utilized as the alternate pathway. Nasotracheal intubation is indicated and it has to be decided whether to intubate with the patient awake or whether it is justifiable to induce anaesthesia before intubation.

Blind nasal intubation under local anaesthesia is not a difficult technique to master and this is the procedure of choice; 5 or 10 per cent cocaine sprayed into the nostril provides good local anaesthesia and shrinks the mucosa, increasing the airway and reducing the likelihood of epistaxis. Care must be taken not to exceed a total dosage of 3 mg/kg. A selection of tubes should be available; it is often easier to introduce one with a long bevel.

If there are signs of moderate or severe obstruction, the mouth cannot be opened sufficiently to use a laryngoscope and it is not possible to perform blind nasal intubation under local anaesthesia, tracheostomy should be performed.

When the oral airway is restricted but there is otherwise no evidence of upper respiratory obstruction, the anaesthetist may be tempted to try intubation with the patient asleep. It is difficult
to suck out the pharynx if the oral airway is insufficient to admit the sucker and the treatment of laryngeal spasm is seriously hindered by the absence of a good oral airway. For these reasons blind nasal intubation under local anaesthesia is indicated and if this fails tracheostomy must be seriously considered. General anaesthesia should precede intubation only when the oral airway is sufficient to admit the use of laryngoscope and suction.

CASE 1.

An 18-year-old girl was involved in a traffic accident. She was conscious when seen but had sustained concussion and multiple superficial lacerations of the face. There was an extensive laceration of the chin exposing the mandible. The lips were cut and the mouth and nostrils were full of blood. She had eaten a full meal several hours before the accident. Blood pressure was 140/80 mm Hg and pulse rate 100/min. The mouth could be opened about 2 cm only but there were no other signs of respiratory obstruction.

For premedication atropine 0.5 mg was given intravenously. After sucking out blood, the right nostril was sprayed with 4 per cent lignocaine. A No. 6 plain rubber nasotracheal tube was carefully advanced and lignocaine sprayed through the tube as it approached the larynx. The pharynx was kept empty by suction with a rubber catheter through the mouth. The trachea was intubated without difficulty and general anaesthesia induced with a mixture of nitrous oxide, oxygen and halothane 1 per cent. A throat pack was inserted when the pharyngeal reflexes were sufficiently subdued.

Emergency tracheostomy may be difficult to perform quickly in these patients. Oedema in the neck and displacement may make it difficult to find the trachea. If obstruction is obviously severe elective tracheostomy should be done. In less serious cases nasotracheal intubation should be attempted under local anaesthesia. If this fails after one or two gentle attempts, tracheostomy must be performed.

When major surgery is performed in the mouth, as for malignant disease of the jaws and tongue, even if the anaesthetist has successfully passed a nasotracheal tube, it is wise to perform tracheostomy at the end of surgery in order to retain control of the airway.

Where there has been severe trauma to the upper airway as may result from gunshot wounds or motor accidents, tracheostomy should be carried out before anaesthesia, otherwise oedema and haemorrhage will almost always compromise the airway and may take several days to subside.

Trismus is commonly seen in association with dental infections. Usually the mouth can be opened only slightly and this causes potential rather than actual respiratory obstruction.

CASE 2.

A girl of 19 attended the dental surgery of a casualty department, having had a left lower molar extracted 7 days previously. There was considerable
swelling and redness overlying the angle of the left mandible and the inflammation extended down into the neck on the left side. The mouth could not be opened more than 1 cm without severe pain. There were no other signs of respiratory obstruction.

The patient was admitted for drainage of the abscess. Premedication consisted of promethazine 25 mg and atropine 0.5 mg. The right nostril was sprayed with 10 per cent cocaine and blind nasal intubation was performed, using a No. 7 plain rubber tube, on the conscious patient without difficulty. General anaesthesia was induced with thiopentone 300 mg and continued with a mixture of nitrous oxide, oxygen and halothane. The jaw relaxed with the onset of unconsciousness and a throat pack was inserted. The abscess was drained into the mouth and a small rubber drain stitched into the cavity. The larynx was inspected with a Macintosh laryngoscope and was normal in appearance. There was, however, reddening and oedema of the mucosa of the left pharyngeal wall. The nasotracheal tube was replaced and left in position until consciousness returned.

Comment. This patient's trismus disappeared with the onset of unconsciousness and obviously anaesthesia could have been induced with thiopentone and a relaxant without difficulty. Nevertheless the appearance of the pharynx illustrates how a dental infection can spread downwards towards the glottis.

Macintosh and Bannister (1943) have advised the gradual introduction of a boxwood wedge to overcome trismus and Goldman (1959) has suggested the infiltration of the masseter muscles over the masseter muscle and a small rubber drain stitched into the cavity. The larynx was inspected with a Macintosh laryngoscope and was normal in appearance. There was, however, reddening and oedema of the mucosa of the left pharyngeal wall. The nasotracheal tube was replaced and left in position until consciousness returned.

Comment. This case illustrates a very severe trismus unresponsive to almost all measures and emphasizes the value of blind nasal intubation.

Nasal obstruction.

In the adult, complete obstruction of the nose presents few problems if the oral airway is available.

CASE 4.

A 47-year-old man required surgery because of obstruction of the nose and recurrent sore throats. He had undergone guillotine tonsillectomy at the age of 7. Inspection of the oropharynx revealed bilateral tonsil remnants. The posterior pillars of the fauces were adherent to the posterior pharyngeal wall and the only nasopharyngeal airway was a chink behind the uvula and a hole through the soft palate on the right side. There were no other signs of upper respiratory tract obstruction.

After premedication with papaveretum 20 mg and hyoscine 0.4 mg, anaesthesia was induced with thiopentone 400 mg followed by succinylcholinum 40 mg. A No. 10 cuffed armoured latex orotracheal tube was inserted without difficulty and anaesthesia continued with nitrous oxide, oxygen and halothane 1.5 per cent. The tonsil remnants were removed; both posterior pillars of the fauces were mobilized and their raw edges sutured. A rubber tube was tied into the naso- oropharyngeal airway to maintain its patency. After pharyngeal suction the armoured orotracheal tube was removed and the patient breathed freely through his mouth.

Comment. In spite of almost complete nasopharyngeal obstruction pre-operatively and the presence of a foreign body in the nasopharynx postoperatively, this patient had no problems with his airway because of the availability of the mouth as an alternative pathway.

Nasal obstruction presents difficulties in the infant as it has great difficulty in adapting to the oral airway. With complete nasal blockage infants may have severe respiratory obstruction with aspiration of feeds and oral secretions; tracheostomy may become necessary even when anaesthesia is not intended.

When surgery is to be carried out for relief of postchoanal atresia in infancy, it is often possible to intubate the child before the induction of anaesthesia. In the older child with this condition or with enlarged tonsils or adenoids, once anaesthesia is deep enough to permit the insertion of an oropharyngeal airway, the problems are resolved. This can usually be achieved with a suitable inhalational sequence. Occasionally the tonsils and adenoids are so hypertrophied that tracheal intubation is the only way of achieving a satisfactory airway.
Pharyngeal obstruction.

Cystic lesions of the pharynx and larynx may often be aspirated under topical anaesthesia, before general anaesthesia is induced in the usual way. Peritonsillar and retropharyngeal abscesses present the hazards of inhalation of pus and they may easily be ruptured by the instrumentation necessary to perform intubation. For this reason, these lesions should be dealt with under local anaesthesia without regard to the degree of respiratory obstruction.

It is helpful to rinse out the mouth with bicarbonate solution to dissolve excess mucus. After spraying the mucosa with 10 per cent cocaine solution the site of the proposed incision may be rubbed with a 10 per cent cocaine stick until blanching occurs. Incision is then carried out with the patient in a "head down" position.

Laryngeal obstruction.

Tracheostomy should precede all but the most minor surgical procedures on the infant larynx. An airway which is just adequate before surgery is likely to be further impaired by oedema in the postoperative period. If the accessory muscles of respiration are in use, or there are other evidences of moderate to severe obstruction tracheostomy should be done under local anaesthesia, prior to surgery. In the absence of these signs, the surgeon may be assisted by the passage of an endotracheal tube or bronchoscope before tracheostomy. An inhalational anaesthetic technique is indicated, and relaxants are best avoided. If there is the slightest difficulty with intubation, the attempt should be abandoned and tracheostomy performed without delay. Infants, up to the age of 4 weeks, may usually be intubated orally without anaesthesia.

Laryngo-tracheobronchitis in infancy necessitates tracheostomy when signs of severe respiratory obstruction develop. Seward and Fraser (1961) have discussed the merits of general and local anaesthesia in this particular situation, and have expressed a preference for the former, employing an inhalational technique and orotracheal intubation with every facility available for performing emergency tracheostomy. The writer favours careful sedation with a phenothiazine derivative such as trimiprazine (2 mg/stone; 0.3 mg/kg), followed by tracheostomy under local anaesthesia.

Small papillomata and polyps of the larynx usually cause little obstruction to respiration and anaesthesia for laryngoscopy and removal of these lesions can usually be safely conducted by a combination of local and general anaesthesia. In this sort of situation, however, sudden difficulties may arise, necessitating emergency tracheostomy and the anaesthetist and surgeon must always be prepared for this.

CASE 5.

A woman of 66 years was admitted for direct laryngoscopy. Twelve years previously small nodules had been removed from the interarytenoid region and a small papilloma from the left cord. She had been hoarse for 3 years before this time. Other complaints were shortness of breath and occasional attacks of paroxysmal nocturnal dyspnoea. She slept propped up on three pillows. The blood pressure was 160/120 mm Hg. There was no stridor and the accessory muscles of respiration were not in use.

At indirect laryngoscopy it was not possible to get a good view, but both cords appeared to be moving and no gross lesion was seen.

After premedication with pethidine 50 mg and atropine 0.5 mg anaesthesia was induced with thiopentone 200 mg and gallamine 40 mg followed by inhalation of nitrous oxide, oxygen and halothane. After a few breaths of this mixture, complete respiratory obstruction occurred and the patient became very cyanosed. A laryngoscope was easily inserted into the oropharynx. The vocal cords could not be seen, being totally obscured by voluminous false cords which were drawn into the glottis leaving a small diamond-shaped chink in their centre. A No. 2 plain tube was the largest that could be pushed through this opening and the surgeon was advised to perform immediate tracheostomy. This was promptly done and the cyanosis relieved by oxygen inflation through the tracheostomy. Direct laryngoscopy then revealed essentially normal appearances.

Postoperatively the patient was mildly hypotensive and complained of chest pain. Electrocardiographic examination 2 days later showed unequivocal evidence of anterolateral myocardial infarction.

Comment. This case illustrates the onset of severe and unexpected difficulties during the induction of anaesthesia. The situation was salvaged by the prompt intervention of the surgeon. The myocardium, which had given pre-operative evidence of its weakness, suffered permanent damage from the hypoxic episode.

The rapidly recurring multiple papillomata of childhood often necessitate tracheostomy to permit repeated excisions.

In carcinoma of the larynx or invasion of the larynx by other carcinomata, tracheostomy is almost inevitable. Careful assessment of the degree of obstruction will help to decide the timing of this procedure. When laryngectomy is projected, the anaesthetist should acquaint himself with the site and nature of the lesion, and assess the degree
ANAESTHESIA IN UPPER RESPIRATORY OBSTRUCTION

of obstruction. If there is stridor at rest tracheostomy should not be delayed and should be performed under local anaesthesia.

Case 6.

A 62-year-old man was admitted with respiratory obstruction. Several months previously he had received deep X-ray therapy for a carcinomatous ulcer of the pyriform fossa. He was sitting upright in a chair, in a state of great anxiety. He was hoarse, had a bovine cough and was able to swallow fluids only. He complained that he had not slept for a week.

Pulse rate was 84/min, respiration rate 30 b.p.m. The skin was pale, dry and slightly cyanosed, with goose-pimples. The arteriolar capillary refill time was prolonged. There was inspiratory and expiratory stridor, the former predominating. The accessory muscles of respiration and the alae nasi were working. The trachea was tugged downwards in inspiration and there was an exaggerated filling and emptying of the neck veins. The intercostal tissues and supraclavicular areas were indrawn during inspiration.

After premedication with promazine 25 mg the respiration rate fell to 20 b.p.m. Tracheostomy was then performed under local anaesthesia. The patient subsequently underwent total laryngectomy.

Comment. Clearly this patient was suffering from very severe respiratory obstruction, which necessitated tracheostomy without delay. The surgical treatment of his disease could then be planned unhurriedly.

Local anaesthesia for tracheostomy should be limited to the incision and a small injection into the lumen of the trachea, before opening it. More widespread infiltration may decrease the efficiency of the sternomastoid muscles, or occasionally diffuse into the recurrent laryngeal nerves, thereby increasing obstruction.

When the laryngeal growth is friable, attempts at intubation are likely to cause bleeding and the possible avulsion of pieces of the tumour. These may lodge in the bronchi.

Case 7.

A 67-year-old man was admitted with respiratory obstruction. He complained of noisy breathing, shortness of breath, hoarseness and haemoptysis. The night before admission he had been walking about in his room trying to get his breath. He had a history of post-traumatic epilepsy and was having daily doses of phenytoin and phenobarbitone. He was irrational and unco-operative.

The respiration rate was 16 b.p.m. and pulse rate 80/min. There was no cyanosis. Stridor was present at rest in inspiration and expiration. The accessory muscles of respiration were not in use and the alae nasi were not moving. At indirect laryngoscopy, a mass was seen below the left cord.

After premedication with atropine 0.5 mg, the mouth and pharynx were sprayed with 4 per cent cocaine. The patient became violently unco-operative when attempts were made to use a laryngoscope. The nasal septum was grossly deviated to the right, so the left nostril was sprayed with 4 per cent cocaine and blind nasotracheal intubation carried out through this nostril with a No. 6 rubber tube. As soon as the tube was in place, general anaesthesia was induced with thiopentone 500 mg and continued with nitrous oxide, oxygen and halothane. A suction catheter was passed down the nasotracheal tube and a small quantity of blood aspirated.

Tracheostomy was performed uneventfully and direct laryngoscopy revealed a growth below the left vocal cord, occupying the left lateral and anterior walls of the larynx.

Comment. Tracheostomy under local anaesthesia was not possible in this violently unco-operative patient. Neither was orotracheal intubation under local anaesthesia. Nasotracheal intubation can be achieved with less co-operation but in this case led to some bleeding from the growth.

If stridor is not present and the growth is not friable endotracheal intubation under general anaesthesia is usually without difficulties. A selection of tubes and gum elastic catheters should always be available and induction carried out in the presence of a surgeon, equipped and ready to perform tracheostomy.

If stridor is present, but there are no other signs of severe respiratory obstruction, an attempt may be made to pass an orotracheal tube under local anaesthesia. If the anaesthetist is not experienced in this manoeuvre or any difficulty is encountered, tracheostomy must be performed. As soon as either an endotracheal tube or tracheostomy tube is in place, general anaesthesia may be induced.

Elective surgery is contraindicated for a patient with an acute infection of the larynx. Should emergency surgery be necessary, pre-operative tracheostomy must be considered. Orotracheal or nasotracheal intubation in these circumstances is contraindicated because of the likelihood of spreading infection and the near certainty of increasing oedema following removal of the tube. If minor surgery is intended and there is no evidence of serious respiratory obstruction, an inhalational sequence without intubation is indicated. Thiopentone must be used with great caution in the presence of lesions affecting the larynx. It may predispose to laryngeal spasm.

Acute injuries of the larynx result in severe obstruction necessitating emergency tracheostomy. Patients with severe laryngeal stenosis coming to surgery will usually have a well-established tracheostomy and general anaesthesia may be conducted through this.

Occasionally a patient will present for general surgery with a history of previous laryngectomy or tracheostomy. The anaesthetist should be alert for
the scars of these procedures in the neck and aware of the possibility of stenosis. If stridor or any sign of serious obstruction is present, intubation should be carried out under local anaesthesia. The tube should be left in until the patient is well awake and then removed by the anaesthetist.

Bilateral midline abductor paralysis of the larynx requires tracheostomy under local anaesthesia. Other laryngeal palsies may cause obstruction during general anaesthesia. Patients with these lesions are best dealt with by intubation. This usually presents no difficulties under general anaesthesia. The tube should remain until the patient is awake.

A foreign body in the glottis is likely to produce signs of serious obstruction. In the adult it may be possible to perform laryngoscopy under topical anaesthesia and then treat the residual glottic oedema expectantly. In the child, topical anaesthesia may not be practicable and preliminary tracheostomy with local anaesthesia is the least dangerous approach to the problem.

**Tracheal obstruction.**

Obstructions in the upper part of the trachea may be assessed and managed in the same manner as similar lesions of the larynx.

When obstruction occurs in the lower part of the trachea, the situation is extremely serious, as it is then not possible to relieve the condition by tracheostomy. It is often possible to pass an endotracheal tube past the lesion under local anaesthesia. The selection of an appropriate endotracheal tube is very important. It must be long enough to get below the obstruction. A tube without a cuff is easier to pass and enables a larger size to be used. When there is deviation of the trachea, the direction of the bevel is important. It should be chosen so that the opening will not lie against the wall of the deviated trachea.

In cases of low intratracheal obstruction the only way to establish an airway may be the passage of a small bronchoscope past the lesion. It may even be necessary to core out a passage through the tumour aspirating the debris through the bronchoscope.

**Case 8.**

A boy of 16 years was admitted, having been in great respiratory distress for 12 hours. He had been treated in the past for carcinoma of the thyroid and the growth was ulcerating through the skin of the neck. His main complaints were difficulty in breathing, difficulty in swallowing, sleeplessness and a wheezing cough.

The blood pressure was 170/80 mm Hg, the pulse rate 108/min and the respiration rate 12 b.p.m. There was marked inspiratory and expiratory stridor at rest. The accessory muscles of respiration were acting strongly and the alae nasi were moving. Auscultation revealed slow air entry into the chest. Cyanosis was not present.

After premedication with promazine 50 mg the patient was given a mixture of 79 per cent helium and 21 per cent oxygen to breathe. The mouth and pharynx were sprayed with 4 per cent lignocaine and with the aid of a laryngoscope the vocal cords and pyriform fossae were sprayed. Lignocaine, 4 ml of 2 per cent, was then sprayed through the cords. After further inhalation of helium and oxygen a bronchoscope was passed into the trachea. Its wall was seen to be infiltrated with growth and the lumen was narrowed to a chink at the level of the sixth cervical vertebra. The bronchoscope was manoeuvred past this obstruction. General anaesthesia was then induced with thiopentone 150 mg, followed by inhalation of nitrous oxide, oxygen and halothane through the bronchoscope.

A low tracheostomy was performed. There was great difficulty in finding the trachea in the neck, even with the bronchoscope in situ, because of the mass of growth present. In making the tracheostomy opening, which involved coring out some of the growth, the right common carotid artery was inadvertently opened, it having become an anterior relation of the trachea. It was successfully sutured.

**Comment.** This case illustrates severe respiratory obstruction due to tracheal obstruction, and shows that general anaesthesia may be safely induced once the obstruction is bypassed. The difficulties encountered during tracheostomy emphasize the futility of depending on a quick emergency tracheostomy in this sort of situation.

**Case 9.**

A man of 44 years was admitted in the terminal stages of bronchial carcinoma. His main complaints were of a cough with bloody sputum and difficulty in breathing.

He was semiconscious and sitting bolt upright in a chair with his head thrown back in the midline. The blood pressure was 110/75 mm Hg, pulse rate 140/min and respiration rate 3-4 b.p.m. He had been having increasing doses of morphine and had had 30 mg by injection 3 hours previously. He was cyanosed, the accessory muscles of respiration were in use and stridor was present in inspiration and expiration, more marked in the latter. Auscultation revealed diminished breath sounds and very slow air entry into both lungs. Stridor was maximum at the level of the manubrium sterni.

It was felt that his condition might be improved temporarily by bronchoscopy. The mouth and pharynx were sprayed with 2 per cent lignocaine and with the aid of a laryngoscope 4 ml of 4 per cent lignocaine were squirted down the trachea. At bronchoscopy growth was seen blocking both bronchi at the tracheal bifurcation. A total of 2 ounces of growth was cored out and aspirated through the bronchoscope. His condition was greatly improved as a result; he became pink and the stridor largely disappeared.
Comment. This was a case of very severe obstruction of the lower trachea and main bronchi which could not be bypassed by tracheotomy. General anaesthesia has no place in this situation.

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References


