Congenital laryngomalacia

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There is at present, very little information on congenital laryngomalacia in the anaesthetic literature. The purpose of this article is to review this topic, which in 90% of patients represents a benign self-limiting condition, disappearing by the age of two to five years. However, if untreated, the remaining 10% of cases can prove fatal. This severe form presents as persistent sternal recession, feeding difficulties, and failure to thrive, progressing to apnoeic attacks, cor pulmonale and eventually death. The developmental and functional anatomy of the larynx will be included, with a discussion of the pathophysiology and history of the disorder. Its diagnosis and a résumé of the various treatment strategies, will be presented. The anaesthetic management is controversial as is the surgical technology. Our technique, for diagnosis or definitive repair, is based upon suspension laryngoscopy using topical local analgesia and spontaneous ventilation. Halothane is then administered by insufflation into the pharynx, using a #8 nasopharyngeal catheter, and suction is applied to the mouth. During the surgical repair, an endotracheal tube (ETT), may be inserted, at the discretion of the anaesthetist and surgeon. Finally, the role of the carbon dioxide CO_2 laser and its hazards will be introduced.

Jusqu'à présent, la littérature anesthésique donne très peu d'information sur la laryngomalacie congénitale. Le propos de cet article est de revoir ce sujet, qui dans 90% des cas est bénin et disparaît vers l'âge de 2 à 5 ans. Cependant, les 10% des cas restant peuvent devenir léthaux s'ils ne sont pas traités. Cette forme grave se manifeste par un enfoncement sternal persistant, des difficultés d'alimentation et un retard de croissance. Elle s'aggrave de crises apnéiques, de coeur pulmonaire et éventuellement il y a décès. L'anatomie fonctionnelle ainsi que le développement du larynx sont décrits avec une discussion de la physiopathologie et de l'histoire du désordre. On présente également le diagnostic et un résumé des différents traitements. Autant la démarche anesthésique que la technique chirurgicale sont controversées. Notre technique de diagnostic et de réparation définitive est basée sur une laryngoscopie en suspension réalisée sous analgésie locale et ventilation spontanée. L'halothane est ensuite administré par insufflation pharyngée à l'aide d'un cathéter nasopharyngé n° 8 et une succion est appliquée à la bouche. Au cours de la réparation chirurgicale, un tube endotrachéal peut être inséré à la discrétion de l'anesthésiste et du chirurgien. Finalement, le rôle du laser au CO_2 et ses risques sont évoqués.

Key words
ANATOMY: larynx;
ANAESTHESIA: paediatric;
ANAESTHETIC TECHNIQUES: insufflation;
EQUIPMENT: lasers;
SURGERY: laser, carbon dioxide: microinstrumentation.

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Congenital Laryngomalacia (CLM) is the most frequent cause of stridor in children. It is the most common congenital abnormality of the larynx, of unknown aetiology.[1] Other common intrinsic congenital anomalies causing airway compromise in infants include choanal atresia, mandibular hypoplasia, vocal cord paralysis and congenital subglottic stenosis.

Embryology of the larynx

The primary function of the larynx is to provide a protective sphincter at the entrance to the air passages; phonation is therefore of secondary importance.

The larynx starts to appear in the 4 mm embryo at four weeks. It develops at the cephalad end of the laryngotracheal groove from branchial arches III to VI. The laryngotracheal groove is a longitudinal, ventral outgrowth of the foregut. By five weeks the arytenoid swellings arise and grow cephalad producing a T-shaped glottis, reducing the laryngeal lumen to a slit. At six weeks the epiglottis is forming from branchial arches III and IV. By eight weeks all the major cartilages are present, and at nine weeks the laryngeal aditus enlarges and recanalizes. The false and true vocal cords appear at ten weeks and by 28 weeks the definitive configuration is present (Figure 1).

Anatomy of the larynx

The larynx extends from the superior tip of the epiglottis, which is narrower in infants and children than in adults, to the inferior border of the cricoid cartilage. The cricoid is the only complete cartilaginous ring in the respiratory

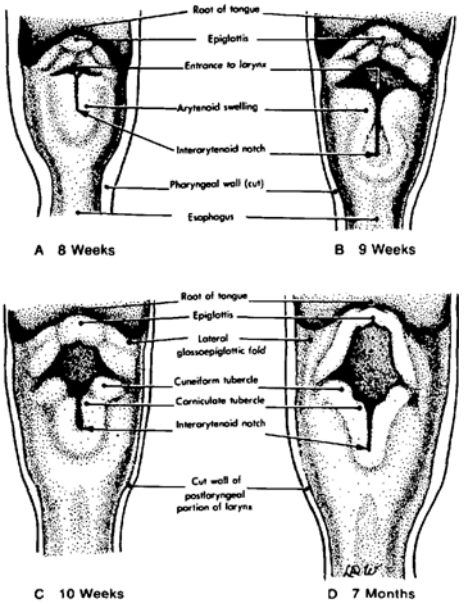

FIGURE 1 Embryology of the larynx. Four stages in the development of the larynx, which is formed from branchial arches III–VI. The laryngotracheal groove appears in the floor of the pharynx in the 4 mm embryo at four weeks of gestation, and continues to develop over the next six months, until the fetus is 28 wk (seven months). (Reproduced, with permission, from Corliss CE, 1976[2])

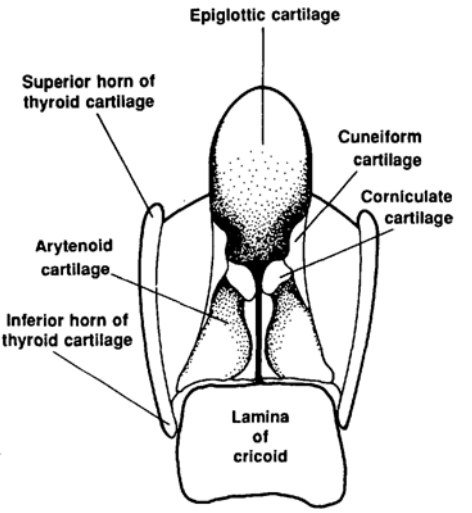

FIGURE 2 Posterior view of the laryngeal cartilages.

tract and forms the foundation of the larynx. The cricoid is also the narrowest part of the infant larynx. The rima glottidis is higher in the newborn being opposite the C_{3-4} interspace, descending to the adult level of the C_{4-5} interspace at 12 years. In addition, the glottis is angled antero-inferiorly, rather than horizontally as in adults.[4]

The framework consists of cartilages and membranes, which are partly ossified. There are nine cartilages, three single and three paired. The single cartilages are the epiglottic, thyroid, and cricoid cartilages. The paired cartilages are the arytenoids, corniculates and cuneiforms. The thyroid cartilage forms the shield of the larynx. The inferior horns of the thyroid, and the arytenoids articulate with the cricoid (Figure 2).

The superior opening of the larynx is bound anteriorly by the epiglottis, laterally by the aryepiglottic folds, and posteriorly by the arytenoids. In this way, the sphincter of the aditus is formed.

The sphincter operates by contraction of the aryepiglottic muscles, drawing the arytenoids anteriorly to the back of the epiglottis. The epiglottis is also capable of rolling posteriorly by the action of the paired thyroepiglottis muscles.

Pathophysiology of CLM

Anatomically, in addition to the presence of redundant supraglottic mucosa, six mechanisms of CLM have been reported. The first three are the most commonly seen (Figure 3, Table I).

Neuromuscular and/or cartilaginous defects or immaturity are thought to contribute to this disorder. However, no specific association has been confirmed with CLM in the recent literature.[5,7]

As the aetiology of these mechanisms is undetermined, the term laryngomalacia is inaccurate (Greek: malakia = softening).

History

The condition has been recognized for many years (Table II).

Severe congenital laryngomalacia – clinical features

Stridor (Table III)

In one study,[1] stridor presenting secondary to congenital laryngeal malformation was reported to be due to CLM in 59.8% of cases. Its onset is usually in the neonatal period.[14] The stridor is high pitched in nature and in-

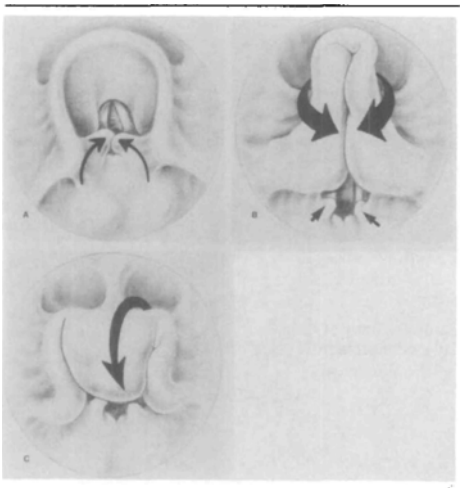

FIGURE 3 The three most common anatomical abnormalities of congenital laryngomalacia. All three variations may coexist during the relatively negative pressure created during inspiration. Although not described below, the aryepiglottic folds also prolapse inferio-medially, following the cartilages. Figures **A, B** and **C**, represent **Types 1, 2,** and **3** respectively of upper airway obstruction that have been described in association with CLM. **Type 1**. Antero-inferior collapse of the cuneiforms (small arrows). **Type 2**. The above mechanism (small arrows), accompanied by the arytenoids inferiorly. This often occurs in association with obstruction by an omega-shaped epiglottis, curling about its vertical axis (large arrows). **Type 3**. The epiglottis may roll posteriorly to form a seal against the posterior pharyngeal wall (large arrow), and may even prolapse between the vocal cords. (Reproduced, with permission from Holinger & Konior, 1989.[5])

TABLE I Anatomical mechanisms of upper airway obstruction in CLM[5,6]

Type 1. The cuneiforms are drawn inwards during inspiration.
Type 2. A long tubular epiglottis (exaggerated omega shaped Ω), curls on itself.
Type 3. The arytenoids collapse inwards.
Type 4. The epiglottis is displaced against the posterior pharyngeal wall or vocal folds.
Type 5. Short aryepiglottic folds.
Type 6. Overly acute angle of the epiglottis at the laryngeal inlet.

TABLE II Historical view of CLM[5,7]

1853. First description of congenital stridor.[8]
1897. First review and description of supraglottic collapse.[9]
1900. Respiratory incoordination postulated as cause.[10]
1942. Laryngomalacia, term first used by Jackson and Jackson (malakia the Greek for the morbid softening of an organ).[11]

TABLE III Chronological steps in the description of Stridor in CLM[7]

1897. Usually present at birth, intermittent, positional, worse on activity.[9]
1953. Precipitated by an URTI.[6]
1977. May develop only months after birth.[12]
1983. May develop weeks after birth.[13]
1984. It is the commonest manifestation of CLM and is characteristically *high pitched*, inspiratory with sternal recession and presents at birth. The latter may lead to a pectus excavatum and eventually cor pulmonale. In the mild form the severity increases until nine months then reaches a plateau, declines and has usually gone by two years.[14]
1987. May lead to cor pulmonale.[15]
1987. The stridor may develop months after birth, is intermittent, positional, being worse in the supine position and on activity. It may even persist into early adolescence.[16]

spiratory in timing.[1,17] It is loudest when the child is upset. The stridor is also more evident in the supine, relative to the prone position. The positional difference is probably due to the dual effect of the tongue and neck extension on the supraglottic structures.[17,18]

Severe CLM may cause upper airway obstruction, cyanotic spells, feeding difficulties, failure to thrive, cor pulmonale, and developmental delay. Death can occur from either upper airway obstruction or heart failure.[5,15,19,21]

Cardiorespiratory failure

The spectrum of signs ranges from nasal flaring to chest retraction, tachypnoea, tachycardia, cyanosis, and finally a diminished level of consciousness.[7]

Feeding difficulties

Feeding difficulties are a result of gastro-oesophageal reflux (GER), which is secondary to upper airway obstruction. These infants and children are slow to feed, vomit easily and therefore fail to thrive. The incidence of GER has been reported at 33% in a series of 297 patients,[17] and in another series as high as 75%.[5]

Relative incidences of complications

The relative incidences of these complications have been published in a study (Table IV).

Definitive Diagnosis of CLM

It is essential to obtain a definitive diagnosis of the cause of stridor. Congenital laryngomalacia is responsible in the majority, but not in all. Diagnosis is by endoscopy, particularly laryngoscopy, but tracheobronchoscopy should also be considered. Congenital laryngomalacia represents

TABLE IV Preoperative signs and symptoms of CLM in a series of 13 patients.[5]

Symptom/sign	*No. of patients*	%
Stridor	13	100
Feeding difficulty	10	77
Failure to thrive	8	62
Obstructive apnoea	8	62
Cyanosis	5	38
Gastro-oesophageal reflux	3	23
Cor pulmonale	1	8

75% of paediatric laryngeal abnormalities that present for endoscopy,[22] but even when the diagnosis is made, 27% of patients with CLM have additional airway problems.[23]

Indirect laryngoscopy using a mirror is not possible in infants and young children. The diagnosis of CLM has therefore to be confirmed by flexible endoscopy using a nasopharyngoscope, or failing this by rigid endoscopy. The latter will require general anaesthesia.[24]

Suction placed at the level of the cords will augment inward collapse of the supraglottic structures,[25] and assist in the diagnosis of CLM. This is referred to as a positive "Narcy sign."[7] This "suction test" may also be used during the operation, to detect redundant supraglottic mucosa, which is removed by microsurgery, employing microinstruments,[7,14,25] or the CO_2 laser.[5,26]

The aim of laryngoscopy, is to identify three anatomical abnormalities that occur either separately or in combination: (1) flaccid epiglottis; (2) poorly supported arytenoids; (3) short aryepiglottic folds. However, several problems may hinder the diagnosis of CLM.[23]

It has been recommended that, in the neonate, the diagnosis should be based on awake flexible endoscopy.[5,18,24,27] However, this may cause cord abduction, as a result of crying, and mask the presence of CLM.[20] During suspension laryngoscopy, the laryngoscope blade may splint the vallecula, preventing prolapse of supraglottic redundant mucosa, especially that on the epiglottis. The shallow breaths of spontaneous ventilation, during deep inhalational anaesthesia, will also lessen the likelihood of the inward prolapse of offending mucosa.

General anaesthesia for a definitive diagnosis

Preoperative preparation

In mild cases all that may be required is a history, clinical examination, complete blood count and electrolytes, with lateral neck and chest *x*-rays. An ECG may be ordered if there are signs of cor pulmonale. Barium studies, chest CT-scan and angiography are reserved for those cases where a vascular anomaly may be present.

As GER may be present, failure to ensure an empty stomach can result in pulmonary aspiration leading to a lower respiratory tract infection and atelectasis.[17] Prolonged fasting, apart from having no effect on gastric volume and pH, may be detrimental to a small patient. We use an NPO period of six hours for solids, and three hours for clear fluids. A further measure to achieve the desired gastric volume and pH, may be by the use of an H_2 antagonist. Cimetidine may be administered, over at least 20 min as an *iv* infusion in 0.9% saline or 5% dextrose (neonates 10 $mg \cdot kg^{-1} \cdot day^{-1}$ in divided doses every four to six hours; and for infants and children 20 $mg \cdot kg^{-1} \cdot day^{-1}$).

Premedication should be non-sedative. The major indication is to block cardiovagal reflexes. An antisialagogue is also an advantage.

Atropine appears to be the drug of choice and it should be administered *iv* prior to induction of anaesthesia.

Induction

After placement of monitors, *iv* access must be established, if not already secured. Halothane in 100% oxygen is used for induction. The time taken to reach an adequate depth of anaesthesia will take longer than normal, depending upon the degree of upper airway obstruction that is present. However, as anaesthesia is deepened and a small amount of CPAP (5 cm H_2O), is applied to the reservoir bag, the stridor usually disappears. This makes the airway easier to manage, and therefore increases the rate of induction. When an adequate depth of anaesthesia has been reached, a laryngeal spray of 4 $mg \cdot kg^{-1}$ lidocaine, diluted, may be used to obtund airway reflexes (each spray = 10 mg). The local anaesthetic is better absorbed by a dry mucosa. Atropine and lidocaine may also have the additional benefit of delaying the onset of bradydysrhythmias and tachydysrhythmias, respectively.

Spontaneous ventilation is maintained to assess supraglottic movement, and to avoid the potential problems that may be associated with other causes of stridor. Ketamine is used by some but may be relatively contraindicated, as it has been reported to stimulate laryngospasm and also to increase oxygen demand by hyperventilation.[28]

Maintenance of anaesthesia

WITH SUSPENSION LARYNGOSCOPY

Anaesthesia may be maintained with topical analgesia and spontaneous ventilation, with insufflation into the pharynx. Spontaneous ventilation without an ETT allows an unobstructed view of the larynx. It also allows the flaccid supraglottic tissue to prolapse inwards during inspiration. After anaesthesia has been established the mask

is removed and a suspension laryngoscope is introduced. Halothane in 100% oxygen is then insufflated by one of the following methods: (i) directly into the mouth; (ii) by the use of a #8 nasopharyngeal catheter;[26] (iii) a nasal ETT pulled back to the nasopharynx;[27,29] (iv) via the side-arm of the laryngoscope;[29] or (v) with a modified anterior commissure retractor.[30]

Of the above choices, the use of a nasopharyngeal catheter appears to be the most suitable, and is our practice. It must be borne in mind though, that the anaesthetist does not have complete control of the airway, so that excellent communication between the anaesthetist and surgeon is imperative.

In the spontaneously breathing patient, a less popular technique, due to the movement produced, is *Intermittent apnoea*. This is achieved by the intermittent removal of the facemask or ETT.[31,32] The surgeon then has a series of short periods in which to make a diagnosis.

When ventilation must be controlled, Insufflation is still the method of choice. The alternative, *jet ventilation*, is not recommended in infants and young children, mainly due to the risk of barotrauma. When using the injector, inspection has to be limited to the times when the vocal cords are not abducted and vibrating. This technique will have the same deleterious effect during surgery.[32-36]

Continuous suction of the oropharynx by a catheter, taped to the cheek of the patient, will decrease pollution.[26]

WITHOUT SUSPENSION LARYNGOSCOPY

The procedure becomes more straightforward when a flexible fibreoptic system is used. After an inhalational induction, fibreoptic endoscopy may be performed through a facemask, using a Portex fibreoptic swivel adaptor between the facemask and the anaesthesia system; in this way the anaesthetist may have more control. The scavenging of waste gases is more efficiently handled if this method is adopted but it may be suitable only for older infants and children.

Postoperative care

Patients should be kept NPO for four hours postoperatively and placed in humidified oxygen, in an intensive care environment for observation of signs of airway obstruction. The signs include: stridor, nasal flaring, dyspnoea, tachypnoea, suprasternal, intercostal or subcostal indrawing, a low or falling transcutaneous oxygen saturation, or a decrease in the level of consciousness.

Racemic epinephrine and steroids are rarely necessary. However, the doses are: racemic epinephrine by facemask (0.2 ml of 2.25% solution, diluted in 2.5 ml of saline, nebulised with 100% oxygen, i.e., 2.25 mg), and dexamethasone 1 $mg \cdot kg^{-1}$.

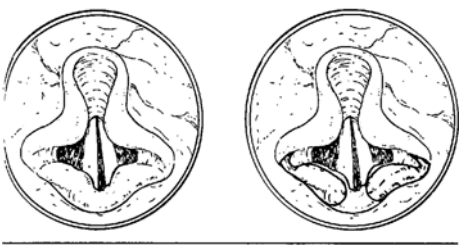

FIGURE 4 Aryepiglottoplasty. A form of supraglottoplasty, in which the redundant mucosa, over the arytenoids and aryepiglottic folds, is excised. The cuneiforms being located within these folds are also removed. (Reproduced, with permission, from Jani *et al.*, 1991.[7])

TABLE V Developments in surgery for severe CLM[5,7]

1922.	Amputation of the epiglottis.[40]
1944.	Wedge resection of the epiglottis.[41]
1971.	Epiglottis sutured to the base of the tongue.[42]
1971.	Hyomandibulopexy.[43]
1984.	The first microsurgical endoscopic surgery for CLM.[14]
1985.	Longitudinal aryepiglottic folds incision.[44]
1987.	Aryepiglottoplasty with glosso-epiglottopexy.[20]
1989.	Laser surgical supraglottoplasty.[5]
1991.	Microinstrumental aryepiglottoplasty.[7]

Mild laryngomalacia is benign and self-limiting, requiring reassurance and follow-up. Severe CLM, however, requires surgical intervention.[14]

Surgical options for severe CLM

Before the 1980s the mainstay of management was a tracheostomy. Today, the use of a tracheostomy has been abandoned due to the high morbidity, 46.5%, and mortality, 1.4% to 10%, with which it is associated.[37-39]

The current surgical aim is to perform a type of supraglottoplasty, i.e., removal of prolapsing redundant supraglottic mucosa: the amount and site of tissue removed varies. The terms epiglottoplasty, supraglottic trimming, anterior epiglottopexy, and aryepiglottoplasty have been used to describe these procedures, depending on what type of surgery has been performed.[5,7,14,15,19] However, the interarytenoid mucosa is never removed, but preserved to prevent postoperative stenotic scarring (Figure 4, Table V).

Laser surgery versus microsurgical instrumentation

Either of these options may be chosen with or without the use of an ETT. However, if the laser is not to be used, then an oral or nasal ETT may be placed. It offers complete control of the airway, and may even splint the larynx for the surgeon, making his task easier to perform.

There are then no limitations on the type of anaesthesia provided, and no scavenging problem. Carbon dioxide laser surgery has become popular recently, as it offers some advantages over the use of the conventional microsurgery. Tissue destruction is precise, immediate, and bloodless and tissue manipulation is unnecessary. The absence of instruments means that there is an unobstructed view of the operative field. Healing is faster as there is minimal oedema, pain and scar formation.[45]

However, there is controversy over the operative method used. Some surgeons prefer the feel of the tissue,[7] and obtain excellent results with the more traditional technique using microinstrumentation,[25] and feel that at times, it may be preferable to the use of a laser.[5]

General anaesthesia for severe CLM

Conventional microsurgery

A supraglottoplasty performed with conventional instruments, produces good results in certain centres,[5,7,25] and there is a reticence to change, particularly as new equipment is expensive and people to work with it must be trained. The anaesthetic is simplified in the absence of a technique that may result in an airway fire: specific gas mixtures and special ETTs become unnecessary. It is also easier to assist or ventilate the patient's lungs if an ETT is in place. If necessary the patient can also be made immobile with a muscle relaxant.

Carbon dioxide laser surgery

Some surgeons prefer the use of the CO_2 laser, despite its implications for the anaesthetist. We use the same anaesthetic technique, insufflation, as for diagnosis, as do others[26,27,29] with the additional precautions for laser surgery. Once the "best" airway is obtained with halothane in 100% oxygen, the inspired oxygen percentage, F_IO_2 should be lowered as far as possible by the addition of air. If the patient's oxygen saturation starts to decrease, the F_IO_2 increased incrementally until an acceptable level is reached. Should airway obstruction develop, the reflex to fill the reservoir bag with the oxygen flush must be suppressed until the surgeon is informed. Volatile anaesthetics in clinical concentrations are nonflammable and may be used with safety.[32] Nitrous oxide is also nonflammable but supports combustion as easily as oxygen at high temperatures, and should not be used.[29,46] Helium does not reduce the risk of fire compared with air as the carrier gas, but may offer an advantage, because of its reduced viscosity and density, in the severely compromised airway.[47]

The absence of an ETT obviates concerns over the type of ETT or protective tape required. However, if an ETT is indicated: for prolonged procedures or complete immobility, then the Laser-Flex (Mallinkrodt, St Louis, MO), is safe to use with the CO_2 laser, and does not require to be wrapped.[48] There is then the option of using a muscle relaxant.

The patient's exposed skin, and particularly eyelids, are protected with saline-soaked gauzes. The personnel also need protection, particulary by eye wear. Ideally a team should be formed which performs all the laser cases. There should be minimal personnel and movement in the room. A laser-linked visible and audible warning inside and outside the room is recommended.[28]

Postoperative care

Regardless of the treatment modality used, humidified oxygen, racemic epinephrine and dexamethasone may be required to treat signs of airway obstruction as detailed in the above section on diagnosis.

Why the carbon dioxide laser over other laser media?

The CO_2 laser is used in preference to other lasers in airway surgery because it is invisible, provides an unobstructed view of the surgical field, and is capable of being focussed down to <2 mm.[45] It is also able to coagulate vessels <0.5 mm diam. This precision helps to diminish postoperative pain, oedema, bleeding and scar formation,[29] all of which are critical in the narrow diameter of the paediatric airway.

Carbon dioxide laser hazards[32]

These include eye damage, bleeding into the airway, airway perforation, fire inside the airway or outside, and atmospheric contamination:

Eyes

The eye is the organ most susceptible to damage.[24] Corneal damage would be the result, but there is no risk to the retina. The patient's eyes must be protected by damp gauzes whilst the surgeon's are protected by the optics of the operating microscope. All other personnel in the room should wear goggles,[45] as optical spectacles do not offer wrap-around protection.

Bleeding and airway perforation

Vessels $>$ 0.5 mm are not coagulable, so bleeding into the airway may occur. Laryngeal and tracheal perforation have been reported, as has massive subcutaneous emphysema with bilateral pneumothoracies following laryngeal laser surgery.[49]

Atmospheric contamination

Unfortunately, the CO_2 laser produces the most smoke of all the laser media, tissue vaporization producing a plume of smoke and fine particles (0.31 μm). Ordinary

masks filtre only to 3.0 μm, therefore a high efficiency mask is required (The Protector II, Anago, Fort Worth, Texas).[32]

Fire

The greatest fear is that of an airway fire. Measures must be taken to minimize the risk of any fire, and to deal with the problem should it arise.

Conclusion

Congenital laryngomalacia can either be mild or severe. The former only requires investigation and follow-up. Severe CLM, however, needs a form of supraglottoplasty, using microinstrumentation or the CO_2 laser. When surgery is performed, we use a technique, without an ETT, in order to provide a clear view for the surgeon. However, this is controversial. If laser surgery is chosen by the surgeon, it is important to establish a protocol, and to develop a team that performs all the surgery in order to estabish proficiency.

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