The anesthetic considerations for the patient undergoing total laryngectomy

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Patients with laryngeal tumors may present with a deviated trachea or with a reduced tracheal lumen size due to preoperative radiation therapy. A short-acting induction agent and muscle relaxant is indicated in the event of difficult intubation and a smaller than expected endotracheal tube may be necessary should the tracheal lumen be reduced. A reversible muscle relaxant is suggested in order to provide spontaneous breathing intraoperatively at the surgeon’s request.

A total laryngectomy may be performed for multiple laryngeal papillomatosis or for laryngeal carcinoma. Laryngeal papillomas are frequently found in children, and reoccurrence following surgical removal is common. The reoccurrence may be severe enough to require laryngectomy in an effort to alleviate respiratory obstruction.

Carcinoma of the larynx occurs ten times more frequently in males than in females. It generally appears in the fifth, sixth, and seventh decade of life. The most common cell type noted is squamous and the degree ranges from in situ to undifferentiated carcinoma. Laryngeal cancer is highly curable if detected early. The overall cure rate of laryngopharyngeal carcinoma is 57%. When the carcinoma is localized to the larynx, the five year survival rate is 76%, and when the tumor is limited to the mid-third of the true vocal cord, cure rates as high as 96% have been reported. However, if regional nodes are involved the five year survival rate can be as low as 29%.

Guidelines for appropriate treatment have been established by the American Joint Committee for Cancer Staging and the International Union Against Cancer. Frequently, the extent of the disease requires a partial to total removal of the larynx. A thorough understanding of the anatomy of the larynx and pathophysiology of the disease, as well as of the surgical technique intended, is essential to develop a plan of care which will provide a physiologic anesthetic for these patients.

Anatomical review

The larynx opens posteriorly into the pharynx and inferiorly into the trachea. It extends vertically to the level of the fourth through sixth cervical vertebrae. This position may be somewhat higher in the female and in the child.

Until puberty, the larynx of the male differs little in size from that of the female. During puberty, the male cartilages enlarge considerably—the thyroid cartilage becomes prominent in the midline while the rima glottidis doubles.

The entire larynx is covered with mucous membrane; the anterior surface and the upper half of the epiglottis, the aryepiglottic folds and the vocal folds are covered with stratified squamous cell epithelium while the rest of the laryngeal membrane is covered by columnar ciliated cells.
The larynx is supported by nine cartilages which are held together by corresponding ligaments and moved by corresponding muscles. The muscle groups are responsible for adduction of tissue for vocalization or breath holding, or abduction for strenuous respiration.

**Cartilages.** The nine cartilages, (three paired and three unpaired), consist of one thyroid, one cricoid, two arytenoids, two corniculate, two cuneiform, and the epiglottis. The largest of the cartilages is the thyroid which forms the laryngeal prominence in the neck. The thyroid cartilage, as its name implies (shield shaped), does not extend around the larynx. The right and left lamina meet anteriorly in the midline forming the laryngeal prominence (Adam’s apple).

The cricoid cartilage (signet ring), although smaller, is stronger than the thyroid. It forms the caudal and dorsal parts of the wall of the larynx. The two major parts of the cricoid are its lamina and arch. The cricoid is the smallest area of the child’s airway.

The two arytenoids are located at the upper border of the cricoid cartilage and are pyramidal in shape. The base presents as a synovial articular surface on which the arytenoid cartilage can slide laterally and medially, forward and back, or rotate upon the cricoid. A muscular base projects laterally from the base, and the vocal process to which the vocal cords and folds are attached projects anteriorly.

The corniculate cartilages are situated in the aryepiglottic folds of mucous membrane and are fused with the arytenoids.

The cuneiform cartilages are placed on either side of the aryepiglottic folds where they give rise to the cuneiform tubercle.

The epiglottis is leaf shaped and projects behind the base of the tongue, ventral to the entrance of the larynx.

**Ligaments.** The two major groups of ligaments, which arise from the cartilages of the larynx, are the extrinsic and the intrinsic ligaments. The extrinsic ligaments function to connect adjacent organs. They include: (1) the thyroid membrane which attaches the upper border of the thyroid cartilage and the thyroid bone, (2) the cricotracheal membrane which links the cricoid to the first ring of the trachea, (3) the cricothyroid which links the thyroid and cricoid (emergency laryngotomy site), and (4) the hyoepiglottic ligament which connects the epiglottis to the hyoid.

The intrinsic ligaments are actual synovial joints which lie between the arytenoids and cricoid and the thyroid and cricoid cartilage. The cricothyroid membrane, the upper free border of the cord, is white in appearance due to the lack of mucous membrane. The second intrinsic ligament is the quadrangular membrane.

**Muscles.** There are also extrinsic and intrinsic groups of muscles. The extrinsics include the sternothyroid muscle which depresses the larynx, and the thyrohyoid which elevates the larynx. There are three groups of intrinsic muscles. The adductors, which serve to close the cords, are three in number —two lateral cricoarytenoids and one interarytenoid. The abductors, which open the cords, include two posterior cricoarytenoids. The regulators of cord tension include two tensors, the cricothyroids, two relaxors, the thyroarytenoids, and the vocalis.

<table>
<thead>
<tr>
<th>Table I</th>
<th>Summary of muscle action</th>
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<tbody>
<tr>
<td><strong>Cricoarytenoids</strong></td>
<td>-separate the vocal folds.</td>
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<td>-open the glottis by rotating the arytenoid cartilages outward so that their vocal processes and the vocal folds attached to them are widely separated.</td>
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<tr>
<td></td>
<td>-close the glottis by rotating the arytenoids so the vocal processes are approximated.</td>
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<tr>
<td><strong>Arytenoids</strong></td>
<td>-approximate the arytenoid cartilages and thus close the opening of glottis.</td>
</tr>
<tr>
<td><strong>Cricothyroids</strong></td>
<td>-produce tension and elongation of vocal folds by drawing up the arch of the cricoid cartilage and tilting back the lamina. The result is an increase in the angle of the thyroid and distance between the vocal process, and thus, elongation of the folds.</td>
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<tr>
<td><strong>Thyroarytenoids</strong></td>
<td>-draw the arytenoids upward and thus shorten and relax the vocal folds.</td>
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<td></td>
<td>-narrow rima glottidis.</td>
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for fine adjustment. Table I summarizes the physiologic activity of the laryngeal muscles. (Laryngospasm is defined as prolonged contraction of the adductor muscles.)

Interior of the larynx. The laryngeal cavity extends from the cricoid cartilage to the trachea. It is divided into two parts by the projection of the vocal cords.1 The vestibule is that portion of the cavity above the vocal cords. The vocal cords are ligaments of yellow elastic tissue which are attached to both the thyroid and arytenoid cartilages. The rima glottidis is a narrow triangular opening or fissure between the vocal cords. The width and shape of the rima glottidis varies with the movements of the vocal cords and the arytenoids during respiration and phonation.

Blood supply. The arterial supply to the larynx is provided by the superior laryngeal artery which arises from the superior thyroid artery, a branch of the external carotid, and the inferior or recurrent laryngeal artery which arises from the inferior thyroid, a branch of the subclavian artery. Venous drainage is provided via the inferior and superior thyroid veins.

Innervation. The nerve supply to the larynx is derived from the vagus nerve via its superior and inferior branches. The superior branch divides at the hyoid into the internal branch, which is sensory to the area above the cords, and the external branch, which supplies the motor function of the cricothyroid. The inferior (recurrent) branch supplies the remaining muscles of the larynx. The right branch wraps around the right subclavian artery and then back to the larynx. The left branch travels to the aortic arch.

Pathophysiology
The larynx can be viewed as a three-dimensional structure, with compartments, connective tissue barriers and lymph circulators that channel the progression and dissemination of cancer.3 Embryologically speaking, the supraglottic or vestibular portion of the larynx and the glottic and subglottic portions arise from different structures.9 This provides a basis for the fact that the larynx consists of two hemilarynxes, each with its own derivation and with independent blood supply.9 Cancer originating in the upper larynx quite exceptionally spreads to the lower larynx and vice versa.9

Anatomically, the larynx can be divided into four regions: supraglottic, glottic, subglottic, and transglottic (Figure 1).

The glottic region is the area most frequently invaded by cancer. Glottic tumors originate from the vocal cord and/or anterior commissure4 and are usually squamous cell. The most common symptom is hoarseness followed by aphonia, dyspnea and pain in the latter stages. When the tumor is limited to the vocal cord or anterior commissure, and free movement of the cord exists, the incidence of lymphatic metastasis is significantly low.
If the tumor invades adjacent laryngeal regions, the incidence of lymph metastasis increases to 25-30%. Supraglottic cancer includes lesions around the epiglottis, aryepiglottic fold, arytenoid, false cord and the laryngeal ventricle. It is the second most commonly occurring laryngeal carcinoma. Symptoms include a change in voice quality or the feeling of a lump in the throat. Hemoptysis, dyspnea and ear pain occur in advanced stages. Lymph node metastasis is frequent (25-50%) due to the abundance of closely located lymph nodes. Subglottic carcinoma is the least common form of laryngeal carcinoma. These lesions occur in the area below the vocal cords to the cricoid cartilage. Dyspnea is the most common symptom. Because the tumor presents as a large mass, procedures to improve the airway are necessary.

Transglottic tumors occupy the ventricular cavities. They are infiltrative and can produce vocal cord fixation.

**Treatment.** The appropriate treatment for laryngeal carcinoma is determined by: (1) the degree of mucosal or submucosal involvement, (2) the mobility of the tumor, (3) the presence of metastasis, and (4) the probability of metastasis.

The clinical setting for laryngeal cancer was developed by the American Joint Committee for Cancer Staging and End Results Reporting and the International Union Against Cancer (Tables II, III, and IV). The tumor, node, metastasis (TNM) classification defines the degree of tumor involvement in each region of the larynx and any mode of treatment should be based on the TNM categories.

The goal of treatment is to provide maximum cure rates while preserving uninvolved laryngeal tissue. Conservative therapy is maintained whenever possible, because it has been shown that only 50% of totally laryngectomized patients over 60 years of age develop a serviceable voice. If conservative therapy is not appropriate for a maximum cure, surgical intervention is necessary. The more

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**Table II**

<table>
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<th>TNM categories for cancer of the larynx</th>
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<td><strong>T:</strong> Primary tumor</td>
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<tr>
<td><strong>T1S:</strong> Carcinoma in situ</td>
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**Supraglottis**

Both anterior and posterior surfaces of epiglottis (including the tip), aryepiglottic fold, arytenoid, ventricular bands (false cords), ventricular cavities (right and left).

<table>
<thead>
<tr>
<th><strong>T1S:</strong> Carcinoma in situ.</th>
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<tr>
<td><strong>T1:</strong> Tumor confined in site of origin with normal mobility.</td>
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<tr>
<td><strong>T2:</strong> Tumor involves adjacent supraglottic site(s) or glottis without fixation.</td>
</tr>
<tr>
<td><strong>T3:</strong> Tumor limited to larynx with fixation and/or extension to involve postcricoid area, medial wall of pyriform sinus, or pre-epiglottic space.</td>
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<tr>
<td><strong>T4:</strong> Massive tumor extending beyond larynx to involve oropharynx, or soft tissues of neck, or destruction of thyroid cartilage.</td>
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**Glottis**

True vocal cords (right and left), anterior glottic commissure.

<table>
<thead>
<tr>
<th><strong>T1S:</strong> Carcinoma in situ.</th>
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<tbody>
<tr>
<td><strong>T1:</strong> Tumor confined to vocal cord(s) with normal mobility (includes involvement of anterior or posterior commissures).</td>
</tr>
<tr>
<td><strong>T2:</strong> Supraglottic and/or subglottic extension of tumor with normal or impaired cord mobility.</td>
</tr>
<tr>
<td><strong>T3:</strong> Tumor confined to larynx with cord fixation.</td>
</tr>
<tr>
<td><strong>T4:</strong> Massive tumor with thyroid cartilage destruction and/or extension beyond confines of larynx.</td>
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**Subglottic**

Subglottic region, exclusive of the under surface of true cords.

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<thead>
<tr>
<th><strong>T1S:</strong> Carcinoma in situ.</th>
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<tr>
<td><strong>T1:</strong> Tumor confined to subglottic region.</td>
</tr>
<tr>
<td><strong>T2:</strong> Tumor extension to vocal cords with normal or impaired cord mobility.</td>
</tr>
<tr>
<td><strong>T3:</strong> Tumor confined to larynx with cord fixation.</td>
</tr>
<tr>
<td><strong>T4:</strong> Massive tumor with cartilage destruction or extension beyond confines of larynx.</td>
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advanced forms require partial or total laryngectomy. (The reader should refer to Tables II, III and IV for differentiation and preferred method of treatment.) The following discussion reviews the case history, surgical treatment and anesthetic management of a patient whose laryngeal carcinoma was treated by total laryngectomy.

Case report

Mr. C., age 61, was examined in his physician’s office to determine the cause of his chief complaint of hoarseness, dysphagia, and pain in his left ear. Physical examination revealed a large intralaryngeal lesion which involved the left epiglottis and false cords. There were no palpable lymph nodes.

He was admitted for elective surgery, scheduled as a laryngoscopy, biopsy with frozen section, and possible total laryngectomy. Mr. C. had a 35 pack/year history of smoking. Ethanol intake was limited to social events. He also had a history of seizures, although his last occurrence had been four to five years ago. Drug therapy included phenytoin Na 100mg and phenobarbital 15 mg t.i.d. In 1977, a benign tumor was removed from Mr. C.’s right lung. No anesthetic complications were noted. A left corneal implant was performed in 1980 under local anesthesia.

On admission, Mr. C. weighed 84 kg and was 69 inches tall. He gave no history of recent weight loss and claimed no allergies. Laboratory data was essentially normal with the exception of macrocytic red blood cells. Electrocardiogram revealed a septal infarction of undetermined age. There was no history of familial anesthetic complications.

Preoperative preparation and operative procedure. An interview by the social worker, speech

Table IV
Summary of stage groupings

<table>
<thead>
<tr>
<th>Stage</th>
<th>T1, N0, M0</th>
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<tbody>
<tr>
<td>II</td>
<td>T2, N0, M0</td>
</tr>
<tr>
<td>III</td>
<td>T3, N0, M0</td>
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<tr>
<td></td>
<td>T1 or T2 or T3, N1, M0</td>
</tr>
<tr>
<td>IV</td>
<td>T4, N0 or N1, M0</td>
</tr>
<tr>
<td></td>
<td>Any T, N2 or N3, M0</td>
</tr>
<tr>
<td></td>
<td>Any T, any N, M1</td>
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Table III
TNM categories for regional and distant metastasis

The following regional node classification is applicable to all malignant head and neck tumors. In clinical evaluation the actual size of the nodal mass should be measured and allowance made for intervening soft tissues. It is recognized that most masses over 3 cm in diameter are not single nodes but confluent nodes or tumors in soft tissues of the neck. There are three stages of clinically positive nodes: N1, N2 and N3. The use of subgroups a, b, and c is not required but is recommended. Midline nodes are considered homolateral nodes.

N: Regional nodes

N0: No clinically positive node.
N1: Single clinically positive homolateral node less than 3 cm in diameter.
N2: Single clinically positive homolateral node 3 cm in diameter or multiple clinically positive homolateral nodes, none over 6 cm in diameter.
  - N2a: Single clinically positive homolateral node 3 cm to 6 cm in diameter.
  - N2b: Multiple clinically positive homolateral nodes, none over 6 cm in diameter.
N3: Massive homolateral node(s), bilateral nodes, or contralateral node(s).
  - N3a: Clinically positive homolateral node(s), one over 6 cm in diameter.
  - N3b: Bilateral clinically positive nodes (in this situation each side of the neck should be staged separately; that is, N3b—right N2a, left N1).
  - N3c: Contralateral clinically positive node(s) only.

M: Distant metastasis

M0: No distant metastasis.
M1: Clinical or radiographic evidence of distant metastasis.

A pathologist and anesthesia personnel was conducted prior to surgery. Fear over the outcome of his surgery and/or whether or not he would be able to communicate appeared to be foremost in Mr. C's thoughts.

Meperidine HCl 50 mg, hydroxyzine 50 mg, cefazolin Na 1 gm and phenytoin Na 100 mg were given one hour preoperatively to minimize seizure activity.

Upon arrival to the operating room suite, Mr. C. was identified, the chart reviewed, and NPO status was established. A five lead electrocardiogram was applied, and blood pressure was determined by electronic measurement. A 16- and 18-gauge catheter were placed in his right and left hand respectively. Intravenous solutions consisting of 5% dextrose and lactated Ringer's solution and lactated Ringer's injection. Patency of the intravenous lines was determined, pressure points were protected and the patient's arms were positioned at his side.

A nitroglycerine patch was applied to the patient's forearm because of his previous myocardial infarction history. He was preoxygenated during preparation and was induced with thiopental 375 mg. Prior to thiopental administration, 5 mg of atracurium was given as pretreatment to avoid fasciculation from succinylcholine administration. Lidocaine 100 mg was given to reduce stimulation from endotracheal intubation and surgical manipulation of the trachea, and also to provide some myocardial protection given the patient's history of previous myocardial infarction.

After adequate anesthetic levels were established, the vocal cords were sprayed with a topical local anesthetic to assist in the reduction of stimulation during intubation and the period of hypotension. A bolus of succinylcholine 100 mg was given and the trachea was successfully intubated on the first attempt. The presence and equality of bilateral breath sounds were established and the endotracheal tube secured. An esophageal stethoscope was inserted to monitor temperature and pulse rate. The patient was maintained with isoflurane, nitrous oxide and oxygen. Muscle relaxation was assured by administration of atracurium 10 mg. Anti-epileptic drug therapy was continued perioperatively.

All fluids were warmed to 38°C and ventilation was controlled until the surgeon requested spontaneous respiration. Immediately prior to the development of the tracheal stoma, the tidal volume was measured to be 100-150 ml and reversal of the muscle relaxant with pyridostigmine (Mestinon®) 10 mg and glycopyrrolate 0.4 mg was necessary because spontaneous ventilation was desired. Upon request of the surgeon, the endotracheal tube and esophageal temperature probe were removed. A size eight endotracheal tube was placed directly into the stoma and temperature was then monitored by skin sensor. The larynx was removed and the tracheostomy site sutured. As the patient emerged from the anesthetic, the endotracheal tube at the stoma site was removed. When it was apparent that Mr. C. was fully awake and had regained reflex activity, he was transported to the recovery room with no apparent complications.

Operative procedure. Following general anesthesia, the patient underwent a laryngoscopy with biopsy. Frozen section revealed invasive squamous cell carcinoma. A roll was placed under the shoulder and skin preparation for total laryngectomy was performed. A U-shaped flap of skin, subcutaneous tissue and platysma were taken extending from the corner of the hyoid bone to one finger breadth above the suprasternal notch. The resection was limited from the hyoid to the first ring of the trachea. The strapesius muscles were excised along with the sternal hyoid and sternal thyroid muscles. The thyroid isthmus was split and the thyroid pushed off the trachea. Splitting of the cricothyroid muscle allowed access to the cricoid and tracheal cartilages.

Dissection proceeded cranially in order that the larynx be separated from the hyoid muscles. The muscles of the hyoid were cut, freeing the larynx. Dissection of the muscle was continued until the epiglottis was reached, leaving only the trachea and mucosa of the larynx attached. The endotracheal tube cuff was deflated and the tube slowly pulled out. The trachea was opened and sutured to suprasternal skin. The endotracheal tube was removed and a clean endotracheal tube was placed directly into the tracheal opening. The trachea was incised completely at the level of the first tracheal space. The esophageal stethoscope was removed and the esophagus was then entered. The pharyngeal opening was enlarged to the level of the hyoid. The mucosa was then cut transversely above the epiglottis so that the entire malignancy was removed.

The pharynx was closed over a size six endotracheal tube to maintain lumen size. The constrictor muscles at the base of the tongue were reapproximated and the tracheostomy was sutured to the skin. The flap was brought down over the drain and membranous portion of trachea sutured to the flap. A drain was inserted and the skin was then closed with staples.
Physiology of the head and neck pre- and post-laryngectomy can be seen in Figures 2 and 3.

Potential drug interactions. This patient had a history of seizure activity and was treated prophylactically with phenytoin Na and phenobarbital. Although there were no drug interactions noted, knowledge of potential problems was necessary to develop a complete plan of care for this patient. Phenytoin Na is structurally related to the barbiturates and exerts its anticonvulsive activity without causing CNS depression. It limits the development of maximal seizure activity by reducing the spread of seizure progress.

Phenytoin Na provides a stabilizing action against potentiation by repetitive stimuli by decreasing intracellular concentrations of sodium within brain cells as well as cardiac and skeletal muscle cells. This stabilization property permits phenytoin to be utilized as an antiarrhythmic agent. The stabilization includes the reduction of posttetanic potentiation (PTP) at synapses. Loss of PTP prevents cortical seizure foci from adjacent cortical areas which are firing impulses and reduces the maximal activity of brain stem centers responsible for the tonic phase of grand mal seizures.

Combination therapy of phenytoin and phenobarbital is advantageous because the differing side effects of the two drugs permit full dosage of each with a greater anticonvulsive therapy. Phenytoin and barbiturates are potent inducers of hepatic drug metabolizing enzymes. This means that a decrease in effectiveness of a drug occurs when it is given simultaneously with the inducer. An “increase” requirement for intravenous barbiturates may be necessary, and the addition of phenytoin to a patient receiving phenobarbital will result in enhanced metabolism. High doses of barbiturates can competitively inhibit the metabolism of phenytoin, and the potential for a fall in plasma concentration exists should barbiturate administration be abruptly stopped. Conversely, abrupt reduction in the use of the enzyme reducer may result in overdosage of drugs that are rapidly biotransformed in the presence of the inducer.

Patients who receive drugs that have enzyme-inducing properties may have an increased tolerance to the systemic effects of repeated lidocaine dosages, and patients receiving barbiturate therapy may have an increased number of premature ventricular contractions when sympathetic amines are used with inhalation agents.
Anesthetic considerations

The patients about to undergo laryngectomy should be appropriately sedated prior to arrival to the operating room suite. Although a thorough explanation of the procedure is provided, these patients fear suffocation, communication problems, aspiration, social rejection, and death. As a result, catecholamine response during induction is common.

To facilitate surgical access, the patient is positioned supine with both arms tucked at the side. Precautions should be taken against nerve injury and back strain from prolonged positioning. Elbow protectors minimize nerve damage resulting from pressure on the radial or ulnar nerves. Slight flexion (lawn chair position) of the operating room table releases strain on back muscles as well as enhances surgical exposure.

Two large bore peripheral lines are necessary because once surgery has commenced the arms are inaccessible, and drip failure on one line can be accommodated for by the other. In addition, the larynx is surrounded by many large neck vessels, and accidental incision can lead to sudden massive blood loss requiring immediate treatment.

Induction and maintenance agents should be selected based on the following desirable effects:

1. The induction agent chosen should allow a rapid smooth induction to provide a balance in myocardial oxygen supply/demand.
2. The induction agent and muscle relaxant chosen should provide a rapid recovery in the event of difficult intubation.
3. The maintenance agent chosen should provide a rapid return of reflexes once the procedure has been completed.
4. The non-depolarizing muscle relaxant should be able to be readily reversed at the request for spontaneous breathing.

Patients with laryngeal tumors may present with a deviated trachea, and/or preoperative radiation therapy may have reduced the lumen of the trachea, requiring a smaller than normal endotracheal tube.

Constant manipulation of the trachea can stimulate coughing on the tracheal tube; thus measures should be taken to minimize this response. Deeper anesthetic levels and/or adequate muscle relaxation with controlled topical spray to the vocal cords and surrounding area can provide some protection. As with any anesthetic, temperature should be closely monitored to alert the anesthetist for signs of malignant hyperpyrexia.

An additional endotracheal tube may be required as a guide to maintain pharyngeal lumen size once closure has begun.

Emergence should be early to facilitate spontaneous breathing, to aid in the elimination of secretions, and to allow for early evaluation of bleeding tendencies.

The superior laryngeal nerve is usually not damaged during supraglottic or total laryngectomy; however, if a radical neck dissection is also performed, the nerve is at risk. A hemithyroidectomy may be necessary in patients with transcartilaginous invasion of the thyroid. Postoperative thyroid studies should be obtained to determine the occurrence of hypothyroidism.

Summary

The complications encountered during anesthesia for removal of the larynx can be devastating. A thorough understanding of the anatomy, pathophysiology, surgical procedure, and anesthesia implications is essential to afford the patient maximum safety and quality care. Psychological support is extremely important in the immediate postoperative period in order that the fear of suffocation and anxiety caused from inability to communicate are reduced.

REFERENCES


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