# STRIDOR IN CHILDHOOD

## THESIS

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By

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# INTRODUCTION

The children and infants are the men and women of the future, which will build up their natives and countries.

So we must take care of them, protect them and find out their developmental problems and try to treat as much as we can.

Stridor is a symptom present in many diseases of child hood; This review is to here the researcher wants to clarify that stridor may be an emergency case and there are dangers of asphyxia and death if the treatment was delayed.

Stridor is more common in children and infants because their central nervous system is not well developed and so there is delayed development of the Respiratory centres in the brainstem.

The Review ~~The researcher~~ gives an idea about the importance of nutrition for the pregnant mother because in the lower socio-economic group and poor nutrition status there is increased incidence of congenital malformations in the newborns e.g. laryngomalacia where there is flaccidity of the supraglottic portion due to defective deposition of calcium within the cartilagionus framework of the larynx leading to Stridor.

The larynx of the child is different anatomically from adult's larynx, so any slight inflammation leads to edema of the m.m. of the larynx which narrows the lumen ( 1 mm. of edema in infantile larynx will reduce the glottic space by 50%).

Also the infantile larynx is higher in position than in adults, this predisposes to aspiration of foreign bodies easily ( F.B. like pins, coins , buttons, tacks) ; and it is also softer in infants i.e., more susceptible to collapse leading to Stridor ( J.F. Birrell 1977).

Subepithelial tissues are less dense in infantile larynx leading to accumulation of tissue fluid in inflammatory conditions giving rise to stridor due to obstruction of the infraglottic and supraglottic areas.

Infantile epiglottis is an omega shaped, which may oblitrate the vestibule and provide less support for the aryepiglottic folds.

There is natural tendency among children to carry small objects in the mouth, and they constantly experiment with the feel of different objects with their teeth. It may be that the protective reflex action is less well

developed in children, so that such objects are more easily inhaled or swallowed, H.N. Bhatnagar (1974) recorded a case of acute fatal pulmonary edema secondary to impacted foreign body in larynx.

A large piece of meat impacted over the enterance of the larynx will cause sudden asphyxia ( R.M.Handfield 1967) or large foreign body behind the larynx may exert pressure on the soft cartilage infantile larynx leading to stridor ( J.F. Landon 1953) .

Children suffering of repeated attaks of chest infections must be examined well for fear of laryngeal tumors or tracheal tumors giving rise to repeated bouts of brassy cough, dyspnea, inspiratory and expiratory difficulties accompanied with suprasternal and infrasternal retractions. So repeated attacks of chest infections may lead to stridor or there is an underlying cause leading to stridor ( Joseph G. Gilbert 1953 and P.H. Halinger 1950) .

Care must be taken during delivery for fear of birth injury to the head leading to stridor of central origin without laryngeal deformity ( H.C. Ballenger 1954).

Or direct birth trauma by the direct action of instrumentation delivery or by action of traction ( J.F. Landon 1953).

One of the important aims of this work is to direct the attention to the very severe dangerous effects on the larynx due to drinking or inhalation of corrosive chemicals by children like : Lye ( NaOH, KOH), clorox and lysol as a result of negligance of their mothers. The Lye ingestion specially leads to severe burns in larynx, hypopharynx and esophagus ( J.J. Ballenger 1969 and Douglas R. 1971).

Repeated endoscopic procedures in a short time, or prolonged endotracheal intubation leads to traumatization of the m.m. of larynx --- fibrosis ---- laryngeal stenosis and air way narrowing --- obstruction of airway and stridor. ( Blair Fearon 1966, 1971).

Tracheal collapse was observed in newborns and older infants suffering of stridor due to airway obstruction ( M.H. Wittenborge et al. 1967).

Every case of stridor must be examined by laryngoscope for fear of faulty diagnosis. A case of a piece of bone

between the vocal cords with stridor was diagnosed : Acute laryngotracheobronchitis , after lary - ngoscopy and removal of the bone recovery was rapid. ( James Crooks 1954).

The researcher tries to give a reasonable description for the embryology, anatomy and physiology, of the larynx.

Also tries to find out and collect the important ideas from the previous litreatures that deals with stridor.

Also tries to find and collect the etiological factors that lead to stridor and tries to discuss in details for the most important causes and in brief for the least importance. Then deals with the different methods of management according to the cause of stridor . Putting and adding a humble effort to the medical library.

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## EMBRYOLOGY OF THE LARYNX

The primordium of the larynx, trachea, bronchi, and lungs arises as a kell-shaped outgrowth from the floor of the pharynx during the third embryonic week. The pharyngeal site of this outgrowth is a depression which elongates posteriorly, forming the laryngotracheal groove. The anterior end of the groove, which is immediately posterior to the hypobranchial eminence, becomes the primitive laryngeal aditus. The aditus lies between the bases of the sixth branchial arch. The margins of the laryngotracheal groove begin to fuse in a caudocranial direction, about the fourth embryonic week, to form the tracheoesophageal septum which separates the trachea from the esophagus and hypopharynx. Closure to the level of the cricoid cartilage is completed by the fifth week, leaving the small pharyngotracheal canal connecting the pharynx and trachea. ( Joseph H. Ogura 1977).

The sagittal slit of the primitive laryngeal aditus is altered by the growth of three tissue masses around it. Anteriorly, the posterior portion of the hypobranchial eminence separates to become the furcula, a mound of mesoderm which is the primitive epiglottis. Lateral to

the slit, the ventral ends of the sixth branchial arches grow and form two raised mounds, the arytenoid eminences, on the floor of the pharynx. These approximate each other medially, the furcula forming a T-shaped sulcus anteriorly. The pharyngotracheal canal lies between the bases of the arytenoid eminences. The vestibule of the larynx develops from the T-shaped sulcus, which is largely part of the floor of the pharynx and lies anterior to the pharyngotracheal canal. Later, the pharyngotracheal canal moves anteriorly into the posterior part of the vestibule. At this time ( eighth week), the analogue of the vocal cords is formed by an epithelial and mesodermal mass separating the floor of the vestibule from the upper trachea, with the pharyngotracheal canal posterior to it . By the tenth week, mass splits sagittally, giving rise to both pairs of vocal cords. ( Richard W. Mallem 1977).

During the seventh week, a fissure appears on the medial surface of each arytenoid eminence and extends anteriorly and laterally into the floor of the primitive vestibule. This is the laryngeal ventricle, the extremity of which extends laterally past the margins of the primitive

vestibule to form the sacculus or appendix of the ventricle. The sacculus is intimately related to the fourth branchial arch and cleft. As the ventricles develop, the true and false vocal cords are separated ( Joseph H. ogura 1977).

The hyaline cartilages of the larynx develop from branchial arch mesoderm, but the elastic cartilages are derivatives of the mesoderm of the floor of the pharynx. The hyaline cartilages first appear during the fourth week. The arytenoids develop in the arytenoid eminences, and at first they are fused to the two halves of the cricoid inferiorly. They are gradually separated from the cricoid by the development of the cricothyroid joint and by the twelveth embryonic week are well developed. The vocal processes are developed separately in association with the vocal cords; and as a result, consist of elastic cartilage. ( Richard W. Mallen 1977).

The cricoid cartilage is also a sixth arch derivative which initially develops as two mesodermal masses which fuse anterior to the pharyngotracheal canal by the sixth week. Fusion of the posterior lamina is delayed until

the eighth or ninth week. The cricoid fuses as the pharyngotracheal canal shifts forward to become a part of the laryngeal lumen. The last portion of the laryngotracheal groove to be obliterated is the interarytenoid sulcus, which is present until the eleventh week. ( Joseph H. ogura 1977).

The thyroid cartilage develops from the ventral portions of the fourth branchial arches, which fuse anterior to the pharyngotracheal canal. Development is fairly complete by the tenth week with the formation of the cricothyroid joint. The fifth branchial arch is vestigial in humans and does not contribute to laryngeal development.( Richard W. Mallen 1977).

The epiglottis and cuneiform cartilages of Wrisberg are derivatives of the furcula, which is part of the floor of the pharynx, although the fourth arch, which forms the lateral glossoepiglottic fold, may contribute to formation of the epiglottis.( Joseph H. Ogura 1977).

The hyoid bone is derived from the second and third branchial arch cartilages; the second arch forms the lesser cornu and part of the body, and the third arch is the origin of the greater cornu and remainder of the body.

The major portion of the supraglottic larynx derives its nerve supply from the fourth arch nerve( the superior laryngeal), which must enter the larynx inferior to the hyoid through the thryohyoid membrane. Remnants of the fourth branchial groove will be attached to the thyrohyoid membrane posterior to the point of nerve entry.(Joseph H. ogura 1977).

The intrinsic muscles, with the exception of the cricothyroid, are derived from the mesoderm of the sixth branchial arches which invades the epithelial and mesodermal primordium of the vocal cords. The cricothyroid muscle is derived separately from the anterior portion of the pharyngeal sphinter at the level of the fourth arch and is supplied by the fourth arch nerve, the superior laryngeal. The extrinsic muscles are derived from the epicardial ridge, as is the sternomastoid muscle, and are therefore supplied by the hypoglossal nerve. ( Joseph H. Ogura and Richard W. Mallen 1977).

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## ANATOMY OF THE LARYNX

The larynx lies in front of the hypopharynx in the anterior midline of the neck, opposite the third to the sixth cervical vertebrae. It is composed of a cartilaginous skeleton that has articulations with three cartilages, the cricoid and the two arytenoids. They are held togather by ligaments and muscles and move upon one another as a result of these muscle actions. ( Gerald M.English 1976).

The location of the larynx between the gastrointestinal and respiratory systems is strategic in protecting the airway during Swallowing and breathing. This is probably its most important function, with vocalization of secondary importance.(Gerald M. English, 1976).

In adult males the larynx is about 4.4 cm. long, 4.3 cm. across and 3.5 cm. in its anteroposterior extent. In adult females it is smaller it is 3.5 cm. long, and 4.1 cm. across and 2.5 cm. in its antero posterior diameter ( W.J. Hamilton 1971).

Thyroid Cartilage : The thyroid cartilage is a hyaline cartilage and is the largest in the larynx. It consists of two alae or wings which meet anteriorly at an acute

angle. The angle varies between sexes, being 90 degrees in the adult male and 120 degrees in the female. In the superior portion of the angle forms a subcutaneous prominence, the Adam's apple. The upper portions of the alae are separated by a deep notch, the superior thyroid notch. Each ala is quadrangular in shape, and at each posterior " corner " is a process or cornu. The superior cornua serve as attachements for the lateral thyrohyoid ligaments. The inferior cornua articulate with a facet on the posterolateral surface of the cricoid to form the cricothyroid joint ( W.J. Hamilton 1971).

Each surface of the cartilage is covered by thick perichondrium. The external surface of each ala is marked by an oblique ridge or line running in an anterior inferior direction from the base of the superior cornu to the inferior tubercle at the midpoint of the inferior margin. This ridge serves as the attachement for the sternothyroid, thyrohyoid, and inferior pharyngeal constrictor muscles. ( R.J. Harrison 1971).

The inner surface is fairly smooth, but about half the distance between the thyroid notch and the inferior margin is a small prominence which is deficient in perichondurium and serves as the attachment for the anterior

commissure tendon ( ligament of Broyles). The petiole of the epiglottis is attached by the thyroepiglottic ligament approximately 1 cm. above this point. (W.J. Hamilton 1971).

Cricoid Cartilage : The cricoid cartilage is an unpaired, ring-shaped, hyaline cartilage. It is formed by a narrow anterior arch and a wide quadrate lamina posteriorly. The arch is 5 to 7 mm. in height, while the lamina is 20 to 30 mm. in vertical length. On the posterolateral aspect of each side is a small, slightly raised facet which articulates with an inferior thyroid cornu. On the anterosuperior surface of the quandrate lamina are two facets with the long axes parallel to the line of the lamina. These are the sites of articulation with the arytenoid cartilages. The posterior surface of the quadrate lamina is marked by a vertical ridge to which are attached the longitudinal esophageal muscle fibers, also, the ridge separates the posterior cricoarytenoid muscles. The inner superior margin of the arch forms the inferior attachment of the cricothyroid membrane and conus elasticus. ( J.J. Ballenger 1977).

1. Arytenoid Cartilages:

Are the largest and pyramidal in shape .

a. Posterior surface: Triangular and concave extending laterally into a muscular process .

b. Anterolateral surface: Convex, extends forwards into a vocal process.

c. Inferior surface ( base) : Concave, articulates with the cricoid cartilage.

d. Medial surface : Narrow , Smooth and flat.

e. Apex : Curves backwards to articulate with corniculate cartilage. ( J. Groves 1978).

2. Corniculate cartilages of Santorini :
Small fibroelastic nodules attached to the apices of the arytenoid cartilages ( J.J. Ballenger 1977).

3. Cuneiform cartilages of wrisberg :
rod like structures of elastic cartilage imbeded in the margin of aryepiglottic folds.( J.J. Ballenger 1977).

Epiglottis : The epiglottis is a thin, flexible , leaf-shaped, fibroelastic cartilage. The narrow inferior portion, the petiole , is attached to the thyroid cartilage just superior to the anterior commissure. Near the inferior

end of the petiole is a prominence, the tuberculum epiglotticum, which often obscures the anterior commissure when the larynx is examined indirectly. The cartilage is perforated by several foramina below the attachment of the hyoepiglottic ligament . This portion of the epiglottis forms the posterior wall of the pre-epiglottic space, an important area in the spread of laryngeal carcinoma. Unlike the perichondrium of the hyaline cartilages, the epiglottic perichondrium is tighly adherent. As a result, infections tend to remain localized when involving the epiglottis, whereas they cause widespread destruction of the hyaline cartilages elsewhere because of the perichondrial elevation ( J.J. Ballenger 1977).

## LARYNGEAL LIGAMENTS AND MEMBRANES

I. Intrinsic : Uniting the cartilages of the larynx to one another.

   a. Elastic Membrane of Larynx : is the fibrous framework of the larynx. It lies beneath the laryngeal muscoa and is divided into upper and lower parts by the ventricle of the larynx. The upper part supports the aryepiglottic and ventricular folds. ( John groves 1978).

The ventricular ligament is a thickening of the free edge ( John Groves 1978).

b. Conus Elasticus, or cricovocal membrane, it is the lower part of the elastic membrane of the larynx. It is composed mainly of yellow elastic tissue. Below it is attached to the superior border of the cricoid cartilage. Above it is attached :

1. Infront to the deep surface of the angle of the thyroid cartilage.
   - The median cricothyroid ligament is formed by the thickened anterior part of the conus.
2. Behind to the vocal process of the arytenoid cartilage.
   - The vocal ligament is the free upper edge of the conus between these points of attachement ( charles H. Edwards 1978).

c. Thyro-epiglottic ligament attaches the epiglottis to the thyroid cartilage. ( Charles Harold Edwards 1978).

II. <u>Extrinsic</u> :

Uniting the cartilages of the larynx to the skeletal structures outside the larynx.

a. Thyrohyoid Membrane :

Is a broad sheet of fibro-elastic tissue.

- Below it is attached to the thyroid cartilage at :
  1. Upper border of alae.
  2. Front of Superior Cornua.
- Above it is attached to the hyoid bone at :
  1. Upper margine of posterior Surface of body.
  2. Upper margine of greater horns ( John Groves 1978).

The Subhyoid bursa Separates the membrane from the posterior surface of the body.

- Upward movement of the larynx during deglutition is facilitated by the bursa.

The membrane is pierced on each side by :

1. Superior laryngeal vessels.
2. Internal branch of superior laryngeal nerve.
3. Supraglottic lymphatic pedicle which pierces the membrane at a point approximately 1 cm. above and anterior to the junction of the superior cornu and the ala of the through cartilage ( John Ballantyne 1978).

b. Median Thyrohyoid Ligament :

Is the thickned median portion of the thyrohyoid membrane.

c. Lateral thyrohyoid ligaments :

From the thickened posterior border of the thyro-hyoid membrane. They are attached on each side :

1. Below : To the tips of the superior cornu of the thyroid cartilage.
2. Above : to the posterior ends of the greater horns of the hyoid bone.

The cartilago triticea is a small cartilage often found in each ligament. ( John C. Ballantyne 1978).

d. Cricotracheal Membrane is attached :

Below to the first ring of the trachea.

Above to the lower border of the cricoid cartilage ( David Downton 1978).

e. Hyo-epiglottic Ligament :

Joins the anterior surface of the epiglottis and the posterior surface of the hyoid to form the roof of the pre-epiglottic space and the floor of the valleculae. ( J.C. Ballantyne et al. 1978).

Laryngeal Joints :

1. Crico thyroid Joint : Between the inferior cornu of the thyroid cartilage and the facet on the cricoid cartilage at the junction of the arch with lamina. It is a synovial joint with a capsular ligament.

   Movements : Rotation and gliding ( J.C. Ballantyne 1978).

2. Crico-Arytenoid Joint : Between the base of the arytenoid cartilage and the facet on the upper border of the lamina of the cricoid cartilage. It is a synovial joint with a capsular ligament.

   Movements : Rotation and gliding ( J.J. Ballantyne 1978).

MUSCLES :

a. Extrinsic : These muscles are concerned with the movement and fixation of the larynx as a whole. The depressor group consists of the thyrohyoid, sternohyoid, and omohyoid muscles and is supplied by the ansa hypoglossi from C2 and C3, the elevtor group includes anterior and posterior digastric , stylohyoid, geniohyoid, and mylohyoid muscles supplied by cranial nerves V and VII . These muscle groups

are important in the laryngeal functions of deglutition and phonation. ( J.J. Ballenger 1977).

The middle and inferior constrictor muscles of the pharynx are also important extrinsic laryngeal muscles. The middle constrictor is attached to the greater cornua of the hyoid bone. The inferior constrictor is attached to the oblique lines of the thyroid cartilage, to a fibrous band bridging the cricothyroid space laterally, to the cricothyroid muscle, and to the cricoid cartilage. These muscles influence the position of the larynx during deglutition ( J.J. Ballenger 1977).

b. Intrinsic : Between one laryngeal cartilage and another. . Are all paired except for the interarytenoid muscle. These muscles are responsible for the function of the vocal cords.

1. Abductors of the Vocal Cords :

   a. Posterior Crico-Arytenoid muscle :

   Origin : from the depression on the posterior surface of the cricoid lamina.

   Insertion: into the back of the muscular process of the arytenoid cartilage.

2. Adductors of the vocal cord.

   a. Lateral Crico-arytenoid muscle :

      Origin : from the upper border of the arch of the cricoid cartilage.

      Insertion : Into the front of the muscular process of the arytenoid cartilage.

   b. Transverse portion of interarytenoid muscle :

      Origin : From the back of the muscular process and lateral border of one arytenoid cartilage.

      Insertion: Into the corresponding portion of the other

   c. External Portion of thyro-arytenoid muscle :

      Origin : anteriorly, from the lower half of the angle of the thyroid cartilage and from the cricothyroid ligament.

      Insertion : Into the anterolateral surface of the arytenoid cartilage, its upper part forms the ventricular band.

3. Tensors of the Vocal Cords :

   a. Cricothyroid muscle :

      Origin : From the front and lateral part of the outer surface of the cricoid cartilage.

Insertion : into the lower border of the ala and the anterior border of the inferior cornu of the thyroid cartilage " External Tensor ".

b. Internal Portion of Thyro-arytenoid( Vocalis)muscle :

It is the lower and deeper fibres of the thyro-arytenoid muscle.

Insertion : Into the lateral surface of the vocal process and anterolateral surface of the arytenoid cartilage. Some fibres gain attachment to the vocal ligament .( Internal tensor).

4. Opener of the Laryngeal Inlet :

a. Thyro-epiglottic muscle :

Part of the thyro-arytenoid muscle whose fibres are prolonged into the ary-epiglottic fold. Some of these reach the margin of the epiglottis.

5. Closers of the laryngeal Inlet :

a. Oblique Portion of Inter arytenoid muscle :

Origin : From the back of the muscular process of one arytenoid cartilage.

Insertion : Into the apex of the other.

b. Ary-epiglottic muscle :

Is a continuation of the oblique portion of the

interarytenoid muscle into the false vocal cord and

Inserted : Into the quadrangular membrane and the margin of the epiglottis. ( John C. Ballantyne et al. 1978).

INTERIOR OF THE LARYNX :

Extends from the inlet of the larynx to the lower border of the cricoid cartilage.

It is divided into 3 parts by :

- False vocal cords : Formed of mucous membrane covering the ventricular ligament and the upper part of thyro-arytenoid muscle.
- True vocal cards : Project in a lower position, its covering epithelium is closely bound down to the underlying vocal ligament.

They appear white in colour due to poor blood supply.

1. Vestibule : Between the inlet and the edges of the false cords.

   Boundary :- Infront : The posterior surface of epiglottis.

   - Behind : The interval between the arytenoid cartilages.

- Eachside : The ary-epiglottic folds and upper surfaces of the false cords.

2. Ventricle : A recess between the false and true vocal cords, it is 2cm. long in adults. Its mucous membrane is covered externaly by the thyro-arytenoid muscle.

- Saccule : Canical pouch , between the inner surface of the thyroid cartilage and the false cords.

- Glottis : The interval between :

  i. Tue vocal cords in its anterior $\frac{3}{5}$

  ii. Vocal processes of the arytenoid cartilage in its posterior $\frac{2}{5}$ .

its length in males : 2.5 cm., females 1.6 cm.

3. Subglottic Space : Between true vocal cords and the lower border of the cricoid cartilage . ( J.C. Ballantyne et al. 1978).

HISTOLOGY OF THE LARYNX :

The mucous membrane lining the larynx is continuous above with that of the pharynx and below with that of the trachea.

It is loosely attached to the walls, except over the posterior surface of the epiglottis, over the corniculate

and cuneiform cartilages, and over the vocal ligaments, where it is firmly adherent to the underlying structures ( W.J. Hamilton 1971).

The epithelium is mainly stratified squamous in the upper part of the larynx and vocal folds.

The lower part of the vestibule and middle and lower portions are ciliated columnar epithelium. (W.J. Hamilton 1971)

The submucosal tissues consist of loose, fibrous stroma except over the epiglottis and true vocal cords, thus there is a propensity for accumulation of inflammatory exudates or edema fluid. The epithelium of the epiglottis is fairly lightly adherent; therefore, edema tends to occur more slowly. ( J.J. Ballenger 1977).

Mucous glands are distributed in the mucous membrane specially on the posterior surface of the epiglottis forming identations into the cartilage and in the margins of the aryepiglottic folds and saccules. Vocal folds have no glands but libricated by those in the saccule. ( W.J. Hamilton 1971).

BLOOD SUPPLY :

1. Laryngeal branches of the superior thyroid artery, pierces the posterior inferior part of the thyrohyoid membrane on each side deep to thyrohyoid muscle.
2. Laryngeal branches of the Inferior thyroid artery, which accompanies the recurrent laryngeal nerve.
3. Cricothyroid branches of the Superior thyroid artery it crosses the midline at the upper part of the cricothyroid membrane. ( J.C. Ballantyne 1978).

THE VENOUS DRAINAGE :

The veins leaving the larynx accompany the arteries,

(1) The superior vessels enter the internal jugular vein via the superior thyroid or facial vein.

(2) The inferior vessels pass via the inferior thyroid veins into the branchiocephalic veins.

(3) Some veins from the larynx also pass by the middle thyroid vein which drains into the internal jugular vein. ( W.J. Hamilton&R. J. Harrison 1971).

NERVE SUPPLY OF THE LARYNX :

The larynx is supplied by branches of the vagus nerve.

I. SUPERIOR LARYNGEAL NERVE :

Arises from the inferior ganglion of the vagus and it receives a branch from the superior cervical sympathetic ganglion. The nerve descends lateral to the pharynx, behind the internal carotid, and at the level of the greater horn of the hyoid, divides into:

1. Internal Laryngeal branch :

Entirely( sensory) . Descend to the thyrohyoid membrane pierce it above the enterance of the superior laryngeal artery and vein and divides into two main branches.

a. The Upper Branch : supplies the mucous membrane of the lower part of the pharynx, epiglottis , vallecula and vestibule of the larynx.

b. The Lower Branch :Passes medial to the pyriform fossa beneath the mucous membrane and supplies the aryepiglottic fold and the mucous membrane of the posterior part of the rima glottidis. Terminal branches of the nerve end in the inferior constrictor muscle of the pharynx.

2. External Laryngeal Branch :

Travels down on the inferior constrictor muscle of the pharynx. It supplies the cricothyroid muscle.

II. RECURRENT (INFERIOR)LARYNGEAL NERVE :

Has a much longer course on the left side than on the right, on the left side it turns round the arch of the aorta. On the right side it runs round the subclavian artery. In the neck it lies between the trachea and oesophagus. Its terminal part passes upwards, under cover of the ala of the thyroid cartilage, immediately behind the inferior cricothyroid joint. It then divides into :-

a. Anterolateral ( Motor) Branch : which supplies all the intrinsic muscles of the larynx except the cricothyroid muscle. No fibres cross the midline and there is no spatial differentiation between those supplying abductors and those supplying adductors.

b. Postero medial( Sensory ) Branch which supplies the cavity of the larynx below the level of the vocal cords. The loop of Galen is formed by nerve fibres which pass between postero medial branch of the recurrent laryngeal nerve and the internal branch of the superior laryngeal nerve.

LYMPHATICS OF THE LARYNX :

A knowledge of the lymphatic drainage of the larynx is of the utmost importance in determining proper and adequate treatment of carcinoma of the larynx.

The lymphatics in the region of the free borders of the vocal cord are small and sparse due to a paucity of subepithelial tissue in this area. The laryngeal lymphatic system is divided into two parts by this area, one draining superiorly and the other inferiorly. The lymphatic system is much better developed in infants and children, in whom the channels are longer and more numerous, but regression takes place with increasing age. ( J.J. Ballenger 1977).

(1) The lymphatic network of the supraglotic structures is extensive, especially in areas with abundant submucosa. The channels collect in a pedicle at the anterior end of the aryepiglottic fold, passes laterally, anterior to the anterior wall of the pyriform fossa, and leavesthe larynx with the neurovascular bundle through the thyrohyoid membrane. Almost all (98%) of the channels end in the upper deep cervical nodes between the digastric tendon and

the omohyoid muscle. The remainder pass to the lower cervical drain or the spinal accessory chain. ( J.J. Ballenger 1977).

(2) The lymphatics of the infraglottic area have a more variable drainage pattern than those of the supraglottic network. The channels leave the area in three pedicles. The anterior pedicle passes through the cricothyroid membrane, and many vessels end in the prelaryngeal ( Delphian) nodes in the region of the thyroid isthmus. Channels then leave these nodes with the remaining anterior channels to travel to the deep interior cervical nodes. The two posterolateral pedicles leave the larynx through the cricotracheal membrane, with some channels going to the paratracheal chain of nodes while others pass to the inferior jugular chain. The superficial lymph node systems of the neck are of secondary importance with regard to laryngeal drainage. Generally, lymphatic drainage from each half of the larynx is quite separate, and little crossover or mixing occurs. (J.J. Ballenger 1977).

There is evidence that lymphatic channels do cross the midline in the supra-and infraglottic areas.

Lymph flow through these areas is normally negligible, but this is altered when drainage is obstructed on either side of the neck. Contralateraldrainage is more likely to occur spontaneously from the infraglottic areas; thus lesions of this area may be associated with less consistent patterns of metastases. The presence of direct lymphaticovenous communications has been demonstrated in dogs and may also account for abnormalities in drainage patterns in humans. ( J.J .Ballenger 1977).

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## DEVELOPMENTAL DIFFERENCES FROM THE INFANT TO THE ADULT LARYNX

Size :

The infantile larynx is of equal size in both sexes. However, it is smaller in relation to body size than the adult larynx and therefore forms a relatively a smaller isthmus to the respiratory tract. The infraglottic area is the narrowest area of the larynx in the infant, whereas the glottic area is the narrowest in the adult. The relative laryngeal dimensions are found in the following table. Thus the area of the glottis in the newborn is some 24 $mm^2$. From this it is apparent that 1 mm. of oedema in the neonate will reduce the area of the glottic space by 50 perdent to 12 $mm^2$. The diameter of the immediate subglottis at birth is 5-7 mm. and that of the trachea is 6-8 mm. The area of this region is thus about 9 $\pi$ and this is reduced by 1 mm. of oedema to 4 $\pi$ or to 44 percent of normal. By the same token 1 mm. of oedema in a 3-mm. bronchus reduces its area to 11 percent of normal. The trachea is 4 cm. long at birth, 5.5 cm. about the age of 7 years and 9-15 cm. in length in the adult. ( J.F. Birrell 1977). See table No. I

The larynx grows fairly rapidly until the child is 6 years of age; then growth proceeds slowly until adolescence. At this time there is rapid growth of the male larynx& by time growth is complete , the male larynx is considerably larger, as we as having much greater internal dimensions than the female larynx. The female larynx enlarges slightly after puberty.

Dimensions of The Larynx

Table I

| | Infancy | Puberty | Adult | |
|---|---|---|---|---|
| | | | Male | Female |
| Vocal Cord-Length | 6-8 mm. | 12-15mm. | 17-23mm. | 12.5-17 mm. |
| Membranous portion. | 3-4 mm. | 7 - 8mm. | 11.5-16mm. | 8 - 11.5 mm. |
| Cartilaginous. | 3-4 mm. | 5 - 7mm. | 5.5-7mm. | 4.5-5.5 mm. |
| Glottis-Width at rest | 3mm. | 5 mm. | 8mm. | 6 mm. |
| Maximum. | 6 mm. | 12 mm. | 19 mm. | 13 mm. |
| Infraglottis-Sagittal | 5-7mm. | 15 mm. | 25 mm. | 18 mm. |
| Transverse. | 5-7mm. | 15 mm. | 24 mm. | 17 mm. |

( J.J. Ballenger 1977).

Position : At birth, the inferior margin of the cricoid cartilage is at the level of the upper border of the fourth cervical vertebra, and the tip of the epiglottis is opposite the first cervical vertebra. Thus, the epiglottis may be seen easily over the dorsum of the tongue in most infants. The larynx descends slightly in the neck between birth and age 2 years, but then the position is relatively stationary until age 11 to 12 years. During this period, the cricoid is opposite the intervertebral disc between the fourth and fifth vertebrae, and the epiglottis is at the level of the second intervertebral disc. After puberty, the larynx lengthens rapidly and the cricoid descends to the level of the seventh cervical vertebra in males and to the level of the sixth vertebra in females. The tip of the epiglottis remains almost stationary opposite the third cervical vertebra. ( Joseph H. Ogura 1977).

Consistency : In infants, the cartilaginous framework of the larynx is softer and the supporting ligaments more lax, making the larynx more susceptible to collapse when an internal negative pressure is exerted on it.( Richard W. Mallen 1977).

The subepithelial tissues are less dense as well as more abundant and vascular in infants, which predisposes to accumulation of tissue fluids. This is an important factor contributing to the frequency of obstruction of the infraglottic and supraglottic areas by inflammatory edema in small children. ( Richard W. Mallen 1977).

Shape : Several laryngeal structures differ in shape in infants. The epiglottis tends toward an omega shape; therefore, there is a greater tendency for it to obliterate the vestibule should it become edematous. Also, the margins of an omega-shaped epiglottis provide less support for the aryepiglottic folds than do the margins of the flat adult epiglottis which help to hold the folds in á lateral position ( Joseph H. Ogura 1977).

In neonates, the vocal cords are half membranous and half cartilaginous, but the membranous portion relatively longer with growth, forming two thirds of the vocal cord in adult ( Joseph H. Ogura 1977).

The angle of the thyroid cartilage is relatively constant at 110 to 120 degrees until puberty, when it becomes much narrower in the male (90 degrees) and widens

slightly in the female (120 degree). ( Joseph H. Ogura 1977).

The peculiarities of the infantile larynx are important factors in the predisposition of infants to particular laryngeal diseases and to aspiration of foreign bodies. ( Joseph H. Ogura & Richard W. Wallen 1977).

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## PHYSIOLOGY OF THE LARYNX

The principal function of the larynx is the protection of the lower ariway. When this aspect of function is altered, serious lifethreatening problems airse. There are a number of other important functions, and these are listed : ( Gerald M. English 1976).

1. PROTECTION OF THE AIRWAY :

The protection of the airway is phylogenically the earliest function of the larynx to develop. When food or fluids are swallowed, the inlet or laryngeal aditus is closed. The mechanisms include the aryepiglottic folds, arytenoid cartilages, false vocal cords, true vocal cords and the epiglottis. Respiration ceases automatically when food or water contact the posterior pharyngeal wall, base of the tongue or tonsillar pillars. These areas are supplied by the glossopharyngeal nerve (IX) , and this nerve is the afferent pathway of this reflex.( Gerald M. English 1976).

Coughing is an efficient method for removing any particles that pass into the tracheobronchial tree from above or that form within this structure.

Hiccup represents a violent and sudden contraction of the diaphragm and expansion of the thoracic cage with vocal cords wide open. This first phase is quite similar to that of cough or deep inspiration . In the midst of this inspiratory effort, the vocal cords close sharply, and the inspiration is abruptly terminated producing the characteristic sound. ( Gerald M. English 1976).

Yawning is ordinarily an involuntary act that is characterized by the rapid inhalation of large quantities of air with the mouth wide open and the vocal cords markedly abducted toward the lateral walls . This produces a wide open, unobstructed airway from the mouth to the trachea. Gerald M. English 1976).

2. PHONATION :

Phonation develops later in the phylogenic evolution of the larynx. Sound is produced by the vibration of the vocal folds, and this sound is amplified by the resonating chambers of the mouth, pharynx, nose and chest. The larynx produces the sound or raw material that is ultimately converted into speech by the action of the pharynx, tongue, lips and related structues. To produce a wide range of sounds, the

vibrating vocal cords must be relatively long, and man has long vocal folds. Only a short segment does not vibrate because the arytenoid cartilages insert into these folds. The edges of the vocal folds have free margins that are moderately rounded and freely mobile. In the production of sound these folds act as a valve. They are momentarily separated by the inspiratory current, and they spring back into the adducted position by virtue of their own elasticity. This rapidly recurring cycle cuts off the air column intermittently from the lungs and produces small puffs of air that are the sound waves. During this process the arytenoids are in firm opposition and motionless. The vibratory cycles occur when the cords are tensed and in the position of phonation. Movements of the arytenoids precede the vibratory cycle and are not a part of that cycle .( Gerald M. English 1976).

STAGES OF SOUND PRODUCTION :

Production of sound occurs in several stages for any given tone :

1. The vocal cords are adducted to the midline and tensed by the action of the adductor muscles. ( Vanden Berg 1958).

2. The entire length or segments of the vocal cord, depending upon the tone, are forceably pulled apart by the internal fibers of the thyroarytenoid muscles that insert into the vocal folds. This occurs without any movement of the arytenoid cartilages that remain closely approximated. ( Pressman 1942; Sonninen 1954).

3. The hiatus between the vocal cords allows air to escape under pressure, and this air everts the separated free margins of the vocal cords. ( Smith 1954).

4. By virtue of their elasticity the everted cord edges spring back into position without affecting the degree of the opening established by the thyroarytenoid muscle. ( Hollien and Curtis 1960).

5. This cycle is rapidly repeated, and this represents the vibrations of the vocal cords. ( Fink 1962).

The false vocal cords play a negative role in phonation. They flatten out against the lateral wall of the larynx and open the larynx above the true vocal cords more widely to allow an uninterrupted, upward passage of the sound waves. When they approximate during phonation an unpleasant, muffled sound is produced, dysphonia

plicae ventricularis. ( Gerald M. English 1976).

3. ACTION OF THE LARYNX DURING RESPIRATION :

The glottis opens a fraction of a second before air is drawn in by descent of the diaphragm ( Green and Neil, 1955). This opening is brough about by contraction of the posterior cricoarytenoid muscles. The recurrent nerve supply to this muscle provides a rhythmic burst of motor activity which begins just before that in the phrenic nerve ( Bianconi and Raschi 1964) . It is driven by the respiratory center and, like the activity in the phrenic nerve, is accentuated by hypercaphia and ventilatory obstruction and is depressed by increases in arterial oxygenation and by hyperventilation. These rhythmic inspiratory bursts in the recurrent laryngeal nerve persist after respiratory movements have been arrested by succinylcholine paralysis ( Suzuki and Kirchner 1969) . They persist after tracheostomy, indicating that the mechanism does not depend on stimulation of laryngeal receptors by passing air (Nakamura et al.,1958).

As a result of variations in the size of the glottic aperture during respiration, the larynx may be an important contributor to adjustments in the intrinsic airway resistance

during respiration. Abduction of the vocal cords produces glottic dilation and reduction of resistance during inspiration. Adduction with constriction of the glottis produces increased expiratory pressures which may assist air mixing in the lungs. Rattenborg , (1961) concluded from his studies that adjustments in the glottic aperture compensate for changes in total airway resistance arising in the nose and bronchi . ( John A. Kirchner 1973).

O'Neil (1959), in this regard, reported a lowered maximum breathing capacity and an impaired intrapulmonary mixing in laryngectomized subjects ( John A. Kirchner 1973).

4. ACTION OF THE LARYNX DURING SWALLOWING :

The usual mechanisms which protect the laryngeal inlet during swallowing include :

(1) reflex inhibition of respiration.

(2) Closure of the glottic sphincter.

(3) Elevation and anterior displacement of the larynx, bringing its inlet under the protection of the base of tongue.

(4) clearing of ingested material from the pharynx before inspiration is resumed. ( John A. Kirchner 1973).

In man, unlike the deer, respiration ceases during deglutition. This is a reflex act resulting from stimuli arising in the pharynx as food enters, the stimuli being conducted centralward through the ninth and tenth crainal nerves. The reflex is involuntary, occurs in decerebrated animals, and is triggered by the receptor end organs which exist in great abundance in the mucous membrane of the pharynx and larynx ( Pressman Kelemen 1955 , Doty and Bosma, 1956,) The most densely innervated regions of the laryngeal mucosa are those on the laryngeal aspect of the epiglottis, in the aryepiglottic folds, the ventricular bands, and the inter-arytenoid area ,(Konig and Von Leden 1961).

Closure of the glottic sphincter is a reflex act, initiated by stimuli carried centrally in the internal branch of the superior laryngeal nerve. Electrical stimulation of the central cut end of the superior laryhgeal nerve produces swallowing movements closure of the glottic sphincter and inhibition of respiration.( Murtaugh, 1945 ; Ogura and Lam, 1953 , Doty and Bosma 1956). Closure begins by approximation of the true vocal cords. Next the false cords close against

one another and against the base of the epiglottis. The posterior commissure is sealed off by an inward rotation and approximation of the arytenoid cartilage . When the false cords have been brought into by apposition, a squeezing effect results, occurring as a result of intrinsic muscular activity within the mass of the false cords themselves. Passive forces may also contribute to the sphincteric closure of the supraglottic structure. Elevation of the larynx and increased intrapharyngeal pressure during swallowing by compressing the pre-epiglottic body between the thyroid cartilage and hyoid bone, pushes the base of epiglottis posteriorly against the elevated ventricular bands and helps to complete closure of the laryngeal inlet. ( Fink1956).

5. CARDIOVASCULAR REFLEXES FROM THE LARYNX :

Arrythmia, bradycardia and occasionally, cardiac arrest may result from stimulating the larynx, particularly in infants, a fact well known to the bronchoscopist. The mechanism appears to be related to stimulation of nerve fibers which arise in aortic baroreceptors and in some individuals, travel to the central nervous system by way of the recurrent laryngeal nerve, ramus communicans, and superior laryngeal nerve. These nerve fibers, when stimulated

within the larynx, slow the heart rate. They pass through the larynx in the deep tissues near the thyroid ala , thus are not influenced by topical anesthetics. They are most effectively stimulated when the larynx is dilated, as with a bronchoscope, or a tight endotracheal tube. The reflex cardiac effects can be controlled by atropine, and are enhanced by morphine ( Reid and Brace 1940 & Suzuki 1967).

Burstein et al. (1950) reported electrocardiographic distrubances in 68 percent of patients anesthetized with the common agents, with intravenous procaine, the incidence fell to 24 percent . Factors which enhanced these disturbances include light anesthesia, prolonged laryngoscopy, repeated attempts at intubation, respiratory obstruction, or tracheal irritation. Hypoxia and hypercapnea are also though to contribute to reflex cardiac disturbances( Converse et al., 1952 ; Denson and Joseph, 1954) . These distrubances, during intubation are generally transient and are unaccompanied by decreased cardiac output as reflected by the blood pressure.( J.A. Kirchner 1973).

6. FIXATION OF THE CHEST : Serves an essential purpose in such acts as :
   - Valsalva's maneuver.
   - Lifting , climbing, digging etc.
   - Straining efforts: As at stool or during childbirth ( Gerald M. English 1976).

## REVIEW

Verylittle was known about laryngeal diseases in children until the begining of the twentieth century. The term " Croup" covered a generality of the disease.

The noisy respiration heared soon after birth and mostly on inspiration is called congenital laryngeal stridor.

Von Luschka, (1871) described the infantile larynx which contains special features that are the main causes of stridor :

Folding of the epiglottis, longitudinally, to form a narrow tube; pulling together of the aryepiglottic folds with the arytenoids and their group of sesamoid cartilages by the inward-rolled edges of the tube-like epiglottis and increase in the angle of enterance to the larynx.

Sutherland and Lack, (1897) , said that the anatomical abnormality is the cause of stridor and was recognant regnant for several decades. By laryngoscope they described the epiglottis to be sharply folded in itself, the two lateral folds lying in close opposition, and in some cases in contact.( J.A. Martin 1963).

**Thomson and Logan Turner (1900)** , came to the conclusion that there was no proof of any congenital malformation and that the deformity was purely aquired. They thought the lesion to be due to a disorder of coordination of respiratory movements, possibly resulting from developmental backwardness in the centres, acting upon a larynx in which there was undue flaccidity of the upper parts.

Paterson and Brown kelly described the epiglottis in case of stridor as being long and tapering with its margins rolled backwards so as to meet forming a complete cylinder.(J.A. Martin 1963).

Hill ,(1930), said, since stridor is a symptom there must be many different causes either local,structural or congenital malformation of the larynx.

Kennedy and New,( 1931) and Bowman and Jackson (1939) reported their experiences with congenital laryngeal stridor " under the broad term of " chronic stridor inchildhood".( G. Kelemen 1953).

Schwartz , (1940) , who gave an excellent historical survey, advocated replacing " congenital laryngeal stridor"

by " inspiratory laryngeal collapse".

Stridor can be produced almost everywhere in the larynx where abnormal ridges are protruding or where compression narrows the normal lumen.

Schall and Johnson , (1940), recorded a case with rattling and squeezing, the persistence of both embryonic fourth arches, forming a left and right aortic arch, built together a circle enclosing the esophagus and the trachea, with a narrowing of the latter lcm. above the carina.

A similar etiology, resulting in a grunting sound was described by Faber, Hope and Robinson, (1945) in a hyperemic but otherwise normal larynx.( G. Kelemen 1953).

Chevalier Jackson (1945), used the term "chondromalacia " to denote flaccidity of the cartilaginous rim of the laryngeal orifice. In-rolling of the lateral margins of the epiglottis occuring on each inspiration, with indrawing of the flaccid folds over the orifice causes the stridor( J.A. Martin 1963).

Wilson , (1953), studied the infantile larynx and found that either healthy or stridorous larynx varies only

with the epiglottis which ranges from the " exaggerated infantile " to the " Omega-Shaped " type.

Thomdson and Negus (1955), used another term which is laryngomalacia to denote that anatomical abnormality is the cause of stridor.

Holinger (1961), considered laryngomalacia a more satisfactory term than congenital laryngeal stridor commenting on a high speed motion photography film of the condition he said that the arytenoid movement was obviously passive., Others said that the movements of the laryngeal inlet are the result of active muscular contraction. ( J.A. Martin 1963).

Chevalier Jackson was interesting in investigation and treatment of croup in infants and children and so asto see the larynx he designed a special infant's laryngoscope which he used without any form of anaesthesia . It was the only method to the laryngologists giving them facility in examination of very small infants and children. But it needs practice and it was fearfull for the patient.

Gray and Halton, (1946), introduced the muscle relaxants in anaesthesia, so this opened new fields in the

surgery of children of all ages.

Rees (1950), helped in development of the paediatric anaesthesia as a specialty in it own right.

Bush (1965), Modified Rees's technique in order to provide an even more satisfactory field for endoscopy. Oxygen saturation and halothane, followed by endotracheal catheterization with a small rubber or plastic catheter, allowed the surgeon to inspect the larynx.( G. Kelemen 1953).

Since the introduction of the Zeiss operating microscope the camera attachment has made possible the making and preservation of accurate records of the conditions found.

Infantile stridor has been a perquisite of laryngologists for so long that it is not surprising to find the many descriptions of it devoted largely to laryngeal factor. A great deal of attention has been paid to the larynx but very little to the infant so ( John Apley 1953) , recorded the changes in the larynx and also the changes in the general physical and mental development in infants, by following up of 80 cases of stridor for several years, and found that stridor cases in males are more than females, also males with stridor have higher

weight than females with stridor.

Few cases of congenital cardiac malformation were recorded, but it was not the cause of stridor.

Feeding difficulties and pulmonary complications were frequent, also many cases with chest deformities were observed. In the largest group of cases stridor was produced by an anatomical anomaly which impaired the patency of the upper respiratory tract. In the majority the anomaly was epiglottic. In other group, stridor was due to dysfunction of the nervous system.

So in the investigation of infantile stridor it is essential to make early and repeated observations, not only of the larynx and respiratory tract, but of the patient as a whole.(R. Pracy 1979).

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## STRIDOR IN CHILDHOOD

The Oxford English dictionary defines stridor as: " A harsh vibratory sound produced by a bronchial, tracheal or laryngeal obstruction"( John Apley1953).

(Wilson 1952 ) rightly affirms that stridor may be caused by almost any lesion of the respiratory system". (John Apley 1953).

Stridor is a harsh sound during respiration high-pitched and like the blowing of the wind due to obstruction of air passages ( Clarence Wilbur Taber 1964).

Stridor is a noise made by obstruction of the passage of air into or out of the lower respiratory tract where the obstruction lies at the enterance to the tracheobronchial tree the noise appears on inspiration and is called inspiratory stridor (R.Pracy 1971).

J.F, Birrell (1977), defines stridor as the noise produced by an obstruction to the passage of air in and out of the lower respiratory tract, and added that it may be inspiratory, expiratory and mixed inspiratory and

expiratory, and said that laryngeal stridor is inspiratory, bronchial stridor is usually expiratory and the rare inspiratory and expiratory variety must bring to mind the possibility of an abnormal vessel arrising from the aortic arch.

Stridor is a Noisy, Crowing, respiratory sounds usually associated with inspiration are relatively common during neonatal period and the first year of life. ( Robert C. Stern 1979).

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## THE INFANT WITH STRIDOR

Normally the baby may crow with delight and this is a common experience to hear a healthy infant makes an occasional stridorous inspiration with pleasure or excitement, or, still more commonly, at the onset of a bout of crying. Differentiation between the normal and abnormal is, however, generally obvious; in true stridor the sound is repeated or sustained, though it ranges from a mild purr, easily audible only with deep breathing, to a harsh croak which may be heard outside the room or, indeed, the house in which the child is lying( John Apley 1953).

1. Character and Intensity :

The most vivid descriptions of stridor were provided by mothers. They varied from crowing like a cock' to cooing like a dove . Between these extremes fell the wheezers and the graters, the creakers and the croakers, and many more, including the child who was thought to have swallowed a whistle . Grunting was a common descriptive term, asthma " not unusual, and 'hissing " or bubbling in the throat', or even ' a sieving noise ' have been applied

to describe what was heard,( John Apley 1953).

The character and intensity of the stridor were evidently related to the degree of obstruction of the airway, but bore no apparent relation to the type of underlying anomaly.

2. Changes in Character :

In one patient a " crow" changed to a " whistle" at 21 months, and to a squeak a few months later. Quite frequently the stridor was extremely soft; it could readily be missed by the observer in the first few days of life, but became louder after a variable period of days or weeks( Wilson 1952).

On reaching its peak of intensity the stridor in most cases remained fairly constant for a few months, and then receded gradually. Later it was heard only with crying or excitements, or eventually only in the presence of superimposed respiratory infections, before it was finally lost completely ( John Apley 1953).

3. Timing :

In the large majority of cases stridor was confined to the inspiratory phase of respiration; in cases which it is infective in origin, stridor was audible both in

inspiration and expiration. ( John Apley 1953).

4. Beginning of Stridor :

Usually whatever the cause, stridor was heard initially within a few days of birth, most commonly on the first day ( John Apley 1953).

Sometimes it is evident for the first time before the end of the second, and in a few the third, week. Stridor was heard for the first time three weeks after birth in one infant with a tracheal anomaly; at five weeks in one with micrognathia & in another with paralysis of a vocal cord; at seven weeks in a mongol, and as late as three months in a baby with congenital heart disease ( John Apley 1953).

Stridor with congenital laxness or deformity of the epiglottis and supraglottic structures begins in the first few days , nearly always on the first day. Stridor associated with laryngeal oedema or trauma of the larynx during birth, or with cerebral irritation, also began without delay. Where stridor was due to anatomical aberrations outside the larynx the onset was usually, but not always, immediately after birth. Stridor associated with upper respiratory infection the characteristic sound was usually

heard soon after birth, but sometimes the onset was delayed for two or three weeks. Stridor associated with collapse of a pulmonary lobe or segment, the onset of stridor is after the first month of life( John Apley 1953).

5. Termination of Stridor :

In small minority of cases stridor disappeared in the first few months, or occasionally weeks, usually with some temporary lesion like laryngeal oedema or paralysis of a vocal cord.

Stridor persisted bore no apparent relation to the under lying cause, though in cases with chronic upper respiratory infection it tends to last longer ( John Apley 1953).

Stridor by the age of 1 year, may disappear , in majority of cases 2 years. In two mongols it was still present at 5 years. In one child stridor, probably infective in origin, was still audible at 7 years in another, where the cause was undetermined, it was lost finally at 8 years of age ( John Apley , 1953 ).

SYMPTOMS AND SIGNS OF LARYNGEAL DISEASE IN THE NEWBORN

1. STRIDOR :

Stridor is a noise made by obstruction of the passage of air into or out of the lower respiratory tract. Where the obstruction lies at the enterance to the tracheobronchial tree the noise appears on inspiration and is called "insporatory stridor . The actual sound may vary with the obstructing lesion but the most common form of inspiratory stridor has a musical crowing quality and is traditionally called croup ( R.Pracy 1971).

2. CRYING AND COUGH

Obstruction of the airway gives rise to distress and the infant tends to be restless and to cry. In cases where the lesion prevents a complete apposition of the vocal cords the cry may be weak or wheezy" and on occasion the cry may be absent although the child is making all the other signs of crying such as grimacing and turning his head from side to side and rotation his clenched fists.

Cough frequently accompanies the cry where there is an irritative lesion of the larynx and this cough may have a harsh metallic sound ( R.Pracy 1971).

3. DYSPNOEA :

Many children with laryngeal stridor have no dysponea, but where the obstruction narrows the airways by more than 1.5 mm. in a 3.15 kg. baby some degree of difficulty in breathing is always present. Signs of difficult breathing may be sought :

(1) In the face, where the nostrils dilate and the lips may be cyanosed and surrounded by an area of pallor. ( R-Pracy 1971).

(2) In the neck, where violent inspiratory movements cause the whole larynx and trachea to move downwards towards the mediastinum. This is due to high negative pressure in the pleural cavity which in effect sucks the larynx and the trachea into the thorax, and if the tracheal cartilages are soft may lead to tracheal collapse. This movement of the trachea into the thorax is known as " tracheal plunging ". When tracheal plunging is very marked it may also be possible to see movements in the muscles of the neck which are used to increase the movements of the thoracic cage ( R.Pracy 1971).

(3) In the chest, where excessive muscular effort directed to drawing air through a narrow glottis will be accompnied by recession of the intercostal spaces. The

sternum may be sucked in towards the spinal column. ( R. Pracy 1971).

(4). HOARSENESS :

A child who is born with a hoarse voice must always be suspected of a laryngeal lesion. In the young child hoarseness may be associated with webs between the vocal cords, subglottic stenosis or papillomas or an impacted foreign body) ( A.G.D. Maran 1979).

(5) Feeding difficulties :

Nearly all children with airway obstruction from whatever cause have difficulty in feeding. Feeds are taken slowly and sometimes spill over into the tracheo-bronchial tree. The child fails to gain weight, airway obstruction therefore may be a cause for failure to thrive. In other cases the airway obstruction only becomes apparent when the child has fed too well in the early weeks of life and has gained weight too rapidly. It is often necessary and usually unpopular to insist that the baby should lose weight.( P.M. Stell 1979).

(6) RECURRENT CHEST INFECTIONS :

Many children with airway obstruction develop chest infections. A decrease in the frequency and severity of

the infection may indicate that the problem causing the obstructions is resolving, and that the surgeon should temporize and resist the temptation to seek radical surgical solutions to the problem. ( A.G.D. Maran 1979).

General symptoms and Signs :

The child who cannot breath easily and who has difficulty in feeding does not thrive. He will therefore tend to be weak and underweight. However there are other general effects which are less obvious. Repeated overspill into the tracheobronchial tree leads ultimately to chest infections and absorptive toxaemia. Inadequate oxygen intake and a high $PCO_2$ result in alterations in the acid-base equilibrium. There will also be some measure of cerebral anoxia and to compensate for this the cardiovascular centre will be stimulated leading to a rise in pulse rate, Prolonged severe cerebral hypoxia may lead to brain damage and physical and mental retardation. It is therefore extremely important that assessment of the cause of the obstruction to the respirations should be made at the earliest possible opportunity. Before a description is given of the various lesions which may give rise to obstructed airways, the routine assessment

of the patient presenting with symptoms of laryngeal obstruction will be described . This assessment may be difficult and time-consuming. It requires patience on the part of the laryngologist and the use of keen clinical observation) ( R.Pracy 1971).

ASSESSMENT

When symptoms and signs of laryngeal obstruction are manifest the baby is transferred to hospital and nursed in an incubator with humidity and oxygen supplied. If time permits, that is to say if the patient is not in need of urgent relief of the obstruction, the history may be obtained from the mother or person who first noticed the symptoms of distress. ( R. Pracy 1971).

The surgeon will want to known :

The following questions :

1. When was the stridor first noticed?
2. At what time of day or night is it loudest ?
3. Is the voice or cry abnormal or weak?
4. Is the child a slow feeder ?
5. Does feeding cause coughin cyanosis or apnoea?
6. Have there been chest infections?
7. What is the weight ? What was the birth weight? Has the child been slow to gain weight?

8. Was pregnancy, labour and delivery normal?
9. Did the baby cry immediately ?
10. Have any other abnormalities been detected.( A.G.D. Maran 1979).

An assessment must then be made of the child's general condition, and particular attention should be paid to the general nutrition. The child with a paralysed vocal cord will experience considerable feeding difficulty and will in consequence tend to be under-nourished. The overweight baby obviously does not have a feeding difficulty but may well have a feeding problem. Since oxygen requirements are related to surface area of the body the overweight baby is particularly at risk . His larynx will be of normal size for a child of his age yet his oxygen requirements will be greater. Therefore, if for any reson the laryngeal air way is further narrowed he will be plunged into respiratory distress more rapidly. This is the sort of child who at six months develops the laryngismus stridulus. Once the general impression of the state of nourishment has been obtained the baby should be examined for signs of dyspnoea. The general colour, whether there is pallor or cyanosis should be noted. Abnormal movements of the alae nasi, trachea, larynx and neck muscles or indrawing of the

intercostal spaces should be recorded . If it is at all possible it is useful to hear the noise which it is claimed the child is making, and the place in the respiratory cycle at which the noise appears is very important. The quality of the noise will help in making the diagnosis. It is often helpful to listen both at the nares and the larynx to hear if the noise is present. Much, too, can be learned by placing the examining ear on the baby's back and listening to perhaps 30 or 40 respirations. Help may also be obtained by placing a stethoscope over the child's larynx and listening to the respiration. It may be possible to determine by the use of this instrument whether the obstruction is supraglottic, glootic or subglottic . Once the position of the noise in the cycle has been fixed the baby should be placed in various positions. Sometimes a change in position may eliminate the noise altogether. Thus, in Pierre-Robin syndrome, in which the micro-gnathia associated with a relatively large tongue and a cleft palate causes pressure on the supraglottis, the distress can be relieved at once by placing the baby face-down on the surgeon's knee. In less clear-cut conditions it can be very profitable to spend perhaps half to three -

quarters of an hour turning the baby from side to side and adjusting the position of the head relative to the trunk in order to find out in which position the noise is least obtrusive. However, even if the probably diagnosis can be arrived at by such manoeuvres it is imperative that every child with stridor should undergo a direct examination of the larynx in order that the cause may be determined and treatment if possible instituted. ( R. Pracy 1979).

LARYNGOSCOPY

Once again a routine procedure is all-important. When a baby is to be examined to determine the cause of respiratory obstruction the surgeon must look for.

(1) Abnormalities of structure .

(2) Abnormalities of function.

(3) Pathological processes superimposed upon an otherwise normal laryngotracheobronchial tree. ( G.Pestalozza 1974).

Both hands may be required in order to carry out some endoscopic surgical procedure and it is therefore essential that the laryngoscope should be fixed in position. No endoscopic examination of a case of stridor is complete without

tracheobronchoscopy.

The following instruments are required :

1. Kleinsasser's infant laryngoscopes-blackened.
2. Negus infant tracheoscope.
3. Infant and suckling bronchoscopes if possible, with Hopkin's telescopes.
4. Loewy Laryngostat and fixation ( C. Cioce 1974).

The instrument is introduced through the right-hand side of the patient's mounth, while the first finger of the operator's left hand is placed between the upper and lower alveolus. When the laryngoscope tip reaches the level of the uvula it is possible to see the catheter and this acts as a reliable guide to the larynx. The first stage in the examination is to pass the beak of the laryngoscope anterior to the epiglottis into the vallecula. In the infant this causes the laryngeal introitus to come into line with the optical axis of the laryngoscope. It allows a good overall view of the larynx and it ensures that in looking for paralysis or paresis of a cord a false diagnosis of paralysis is avoided because there is no pressure on the aryepiglottic fold. A diagnosis of vocal cord paralysis is only made

when failure of the cords to move is clearly seen with the laryngoscope in this position. The beak should now be used to lift up the epiglottis and in this way the vocal cords, false cords and subglottic regions are inspected. If for some reason subglottic stenosis is suspected the rubber catheter can be removed ( C.Pestalozza and C. Cioce 1974).

The operating microscope has made it possible to obtain a much better view of the larynx and a standard 400 mm. objective used for adult laryngoscopy is also used for examination of the infant's larynx. This enables the surgeon to gain a satisfactory view and at the same time to carry out any required operative endoscopic treatment.

When laryngoscopy has been completed the laryngoscope should be removed and the tracheobronchial tree examined for abnormal narrowing evidence of a tracheooesophageal fistula and for possible abnormal pulsation due to aberrant artery formation ( G. pestalozza and C. Cioce 1974).

## CLASSIFICATION OF STRIDOR CAUSES IN CHILDHOOD

I. CONGENITAL

1. Laryngo Malacia :( Congenital Laryngeal Stridor)

Is a spesific entity refering to excessive flaccidity of the supraglottic portions of the larynx and is only one of the causes of congenital laryngeal stridor ( Ballenger 1969) .

Laryngo malacia is the most common laryngeal anomaly- it comprises three-quarters of all congenital anomalies of the larynx ( Holinger 1976).

( Birrell 1960), added that it is a rare condition and is more common in boys than in girls.

(P.H. Ward , 1973) , called it chondromalacia which means a flaccidity of the cartilaginous super structure of the larynx usually secondary to defective or latent deposition of calcium within the cartilaginous frame work of the larynx.

The infant epiglottis is typically" omegashaped " with the lateral margins being parallel with the aryepiglottic folds. The result is that the aryepiglottic folds are poorly supported. Exaggeration of

the omega shape of the epiglottis, bulky aryepiglottic folds and excessive tissue flaccidity are probably present to some degree in each case. The syndrome, appears to be more common in lower socio-economic groups, and poor nutritional status may be an etiologic factor.
( J.J. Ballenger 1969).

The term laryngomalacia refers to a larynx in which the tissues are generally flaccid, so that the arytenoids, epiglottis, and the aryepiglottic folds all tend to fall inwards on inspiration, thus producing the fluttery inspiratory stridor which is so typical of the condition.

( P.F. Mc Swiney and N.P.C. Cavanagh . and P. Languth 1977).

Etiology : The stridor in congenital laryngeal stridor is an active contraction of the muscles comprising the inlet sphicter of the larynx. At this point it is admitted that passive movements of the soft tissues surrounding the inlet must occur. The turbulence of the air stream created by passage through the narrowed inlet must induce vibration in the containing chamber or there would be no noise. It is maintained that this phenomenon is secondary to the active tilting forwards of the arytenoid cartilages and in no way causative.

If it is conceded that muscular contraction causes the stridor by narrowing of the airway during inspiration, two questions remain to be answered . Where is the site of the primary lesion ? and what is its nature ?( J.A.M. Martin 1963) .

1. Rhythmically recurring motor activity in the inlet sphincter suggests that one must look away from the larynx itself for an explanation.
2. The motor neurones supplying the laryngeal musculature lies in the medulla in the vagus and accessory nerve elements of the nucleus ambiguus.
3. Inlet constriction is occurring at the most inconvenient moment in the whole cycle of respiration. Respiratory embarrassment must inevitably follow. If the patterns were transferred to the expiratory phase , the symptoms might well pass unnoticed.
4. The initiation of motor activity by the respiratory centre is being closely interlinked with laryngeal motor activity of a special pattern.
5. It was shown during the discussion on the mechanism of inlet closure, that this special pattern of activity conforms in all respects with the behaviour of the

laryngeal inlet during the second stage of swallowing.

6. The respiratory, cardiac and swallowing centres lie in close proximity to one another in the reticular system of the medulla, being placed in the floor of the fourth ventricle at its lower end.

7. Sinus arrhythmia is said to be due to some sort to excitation of the cardiac centre by the respiratory centre during inspiration.

8. By analogy with sinus arrhythmia it is suggested that an abnormal neural pathway exists in these children, through which the respiratory centre facilitates discharge from the swallowing centre at the inception of inspiration.

9. In consequence there is a motor discharge from the nucleus ambiguus of each side which, passing along the recurrent laryngeal nerves, reaches the inlet sphincter of the larynx at the very start of the respiratory cycle. Contraction is maintained in the sphincter until inspiratory activity is inhibited in the brain stem. ( J.A.M. Martin 1963).

**Microscopic Anatomy :** In cases of congenital laryngeal stridor even autopsy records are rare, histological observations are conspicuous by their absence, and the number of biopsy reports is minimal. ( Iglauer) found the amputated part of the epiglottis to be normal. ( Hasslinger) on excising of flabby ridges from the aryepiglottic folds saw beneath the squamous epithelium edematous tissue with few cells. ( Beneke , quoted by Bagg) found structural displacement of the cartilaginous cells through pressure of the thymus in fetal life.( Gunn), in the case of a 4-month-old infant with inspiratory stridor, which disappeared when the child was turned on its back, found microscopically a slight increase in the lymphatic and polymorphonuclear cellular elements of the submucosa, but this was insufficient to constitute an inflammatory infiltrate. The thickness of the cartilaginous layer of the epiglottis was about one-half the thickness of a control specimen taken from a child of approximately the same age. ( Gunn) thought that the histological examination gave no definite information as to the cause of the anomaly. ( George Kelemen 1953).

Clinical Manifestations :

The symptoms of inspiratory stridor, suprasternal supraclavicular and epigastric retractions may be present at birth or may arise several weeks later. The noisy, severe inspiratory stridor; often inconstant being absent one moment and extremely marked a short while later , it is described as a crowing sound and is the most common presenting symptom ; in extreme cases inspiration may have a fluttering , staccato quality. It tends to be worse when the child in the supine position or feeding and is usually better with the infant in the prone position, also, it is often absent when the infant is asleep. Usually, cyanosis and severe dyspnea arenot present( Raul H. Ward 1973 and Lauren D. Holinger 1976).

If the condition persists the infant will have funnel breast( Pectus excavatum). The child may be slow to gain. weight due to feeding difficulties and so fail to thrive, also he suffers many chest infections on the way through childhood ( R.Pracy 1979).

(Wilson, 1952) , considered that the mechanism of the production of stridor , and the type of noise produced may vary with the shape of the epiglottis whether long and tapering, omegashaped, or intermediate in form being tightly curled.

Wilson also stated that it's abening condition and disappears by the age of 2½ years. Very few may die of their abnormality but experiences show that noise persist up to five years.

Diagnosis :

Confirmed by direct laryngoscopy ,it is important to rule out other causes of stridor in the infant . In many cases the epiglottis is the structure from which the fluttering sound originates, although the arytanoids may participate with the epiglottis which is curled or tubular in extreme instances ( Holinger 1967).

The laryngoscope blade must be inserted in the vallecula to observe the motions of the epiglottis. On inspiration, the structures around the vestibule, particularly the aryepiglottic folds, will be seen to invaginate with the synchronous production of the stridorous sound.

The stridor can be relieved by inserting the laryngoscope into the vestibule of the larynx ( Ballenger 1969).

Most dyspneic episodes can be terminated merely by Placing the child in a prone position ( Holinger 1976).

2. CONGENITAL SUBGLOTTIC STENOSIS :

Is a condition which has attrcted attention in the past 20 years. This has been associated with improvements in paediatric anaesthesia and the introduction of routine direct laryngoscopy under general anaesthesia.(R.Pracy 1979).

The condition is one of the most common laryngeal anomaly; if the degree of stenosis is severe it will give rise to a severe harsh barking kind of stridor and dyspnoea ( R. Pracy 1979).

Aetiology :

Congenital subglottic stenosis refers to a laryngeal stenosis occuring between the glottis ( true vocal cords) and the first tracheal ring. The most common cause is a thickening of the subglottic soft tissue( Conus elasticus) and sometimes the true vocal cords themselves. The narrowest point is usually 2-3 mm. below the superior surface of the true vocal cords. The stenosis may also be caused

by considerable thickening of the cricoid cartilage and the abnormal cartilage extends round the circumference of the airway. Subglottic stenosis, sometimes is accompanied with other congenital abnormalities, such as tracheo-oesophageal fistula. This increases the difficulties of treatment .

Tucker (1932) determined the normal diameter of the subglottic lumen in a fullterm newborn to be 6mm. He considered 5mm. small but compatible with life, and 4mm. a definite stenosis. Wilson (1953) , noted that 4.5-5 mm. was the normal range.( L.D. Holinger 1976).

CLINICAL MANIFESTATIONS :

Children with subglottic stenosis are not usually seen at birth unless the abnormality is accompanied by other conditions such as tracheo-oesophageal fistula.

The diagnosis is made by the anesthetist trying to intubate the child for the repair of the fistula. (R.Pracy 1979). Less severe stenoses may manifest themselves by recurrent episodes of " Croup" with every upper respiratory infection ( U.R.I. ) since 1 mm. of edema of the

subglottic mucosa narrows the lumen to 32% of its original size. And the respiratory obstruction is manifested by the characteristic brassy-two-way stridor aggravated by upper respiratory tract infection which often cause further narrowing due to mucosal swelling. So those children are slow feeders and found to be underweight for their age. Abnormalities of phonation are generally not present. (Lauren D. Holinger 1976), ( Paul H. Holinger, Joyce A. Schild, Steven L. Kutnick).

DIAGNOSIS :

The correct diagnosis suggested by the symptoms and X-rays, is established by direct laryngoscopy.And is made by the anaesthetist and the laryngologist together. The soft tissue type of stenosis appears as a concentrid narrowing or a bilateral subglottic swelling which leaves a narrow oblong lumen. The cartilaginous type is observed anteriorly; it leaves a small posterior lumen. If the tip of a 3 mm. bronchoscope cannot pass, a subglottic stnosis is present ( Normally 4.5 mm.)(Lauren D. Holinger 1976). Whenever a stridor is being investigated one must do a thorough endoscopic examination, not only of the larynx, but do the tracheobronchial tree as well, to distinguish

the cause of stridor ( Blair Fearon, David Ellis 1971).

PROGNOSIS :

The child who manages to survive the first two or three months can be expected to survive to adult life unless he is attacked by severe infections as acute laryngotracheobronchitis.

In case of upper respiratory tract infections, extra care must be taken and it is advicable to nurse the baby in hospital where oxygen and added humidity are at hand.

3. CONGENITAL LARYNGEAL WEB :

( J.J. Ballenger 1969) . Congenital webs are the result of failure of normal splitting of the vocal cord primordium. Therefore, the patency of the laryngeal lumen is deficient because of persistent attachment between the two halves of the larynx. The morphology of the webs varies widely, some are paper thin while others are quite thick. Also, they may occur at various sites in the larynx. Congenital webs are most commonly found at the level of the glottis between the anterior portions of the vocal cords. Less commonly, they occur in the infraglottic area, in the supraglottic area between the ventricular

bands or in the posterior commissure. Infraglottic webs may be associated with cricoid cartilage abnormalities. ( J.J. Ballenger 1969).

Clinical Manifestations :

Symptoms will vary with the site and degree of webbing, GLOTTIC WEBS : Produce dysphonia of varying degree which is marked by a weak, hoarse cry in infants. Frequently this may be the sole symptom and when minimal, may not be noted until speech is developing. The presence of a web tends to delay the development of speech. Inspiratory stridor and other signs of laryngeal obstruction, which are made worse by respiratory infections, are common symptoms. More extensive webs cause obstruction which may be so severe that emergency tracheostomy is necessary immediately after birth ( J.J. Ballenger 1969).

WEBS IN THE INFRAGLOTTIC : area tend to produce little voice change, but they produce stridor which may have an expiratory component. The smaller webs may produce symptoms only when there is superimposed respiratory tract infection. When obstruction is fairly severe there is usually some feeding difficulty ( J.J. Ballenger 1969).

DIAGNOSIS : Diagnosis of a laryngeal web is made by direct laryngoscopy which may be performed without anesthesia in newborn infants. The site and thickn ss of the webs should be noted in newborn infants. The site and thickness of the webs should be noted in order to plan treatment. Soft tissue roentgenograms and laryngograms are helpful in determining the configuration of the web.

4. ABNORMALITIES OF THE EPIGLOTTIS : Per se are very rare.

More commonly they are associated with other structural abnormalities and on occasion with genetic abnormality such as the cri du chat syndrome. For this reason attempts to correct air way obstruction on the basis of a diagnosis of isolated epiglottic pathology should be resisted( A.G.D. Maran, P.M. Stell 1979).

a. Brif Epiglottic : Extremely rare condition, it appears that one half of the epiglottis becomes sucked into the glottis and obstructs the airway. So the symptoms are more acute in onset and more severe in effect than congenital laryngeal stridor( R. Pracy 1979).

b. Quadrate Epiglottis : The epiglottis is square in shape and much larger than usuall . It is one of the characteristic features of the cri du chat syndrome, associated with an abnormal gene on the short arm of chromosome 5. The glottis here is rhombodal and the vocal cords are difficult to see. There is also facial changes and poor muscle tone and mental retardation. This must be born in mind when examination of a case of stridor.

## THE LARYNX IN THE CRI DU CHAT SYNDROME

As its name suggests, the syndrome is characterized by a cat like cry in new-born infants. In all cases a chromosomal abnormality has been found, a loss of a segment of the short arm of chromosome number 5 in the B group ( K.P. MANNING 1978).

Females are more commonly affected than males. The gestation period is normal . The infants tend to be of low birth weight. They have feeding difficulties and fail to thrive after birth. They are mentally retard ed. Despite this many survive childhood.( W. Roy, Berg. 1969).

Clinically, cases are characterized by some or all of the following features, a cat-like cry, microcephaly, mental retardation, micrognathia, a round facies, epicanthic folds, antimongoloid-slant of the palpebral fissures, hypertelorism, strabismus, and low set ears( Warkany, 1971) Other features that may be seen are congenital heart lesions muscular Hypotonia , hypertonicity J. Lavillaurey 1972) . scoliosis, dislocation of the hip, hyperextensibility of the fingers and toes, incurved little fingers, cleft

palate, high arched palate and bifid uvula.

Abnormal dermatoglyphics are found and simian creases or transverse but interrupted palmar creases are the rule. ( K.P. Manning 1977).

THE LARYNX : Direct laryngoscopy under general anaesthesia showed that the epiglottis was larger than normal and quadrate in form and somewhat thicker than normal (Junien Lavillaurey et al. 1972) . The glottis was rhomboidal in shape. The vocal cords joined anteriorly but merged into the false cord and aryepiglottic folds about half way along their expected length. The false cords were deeply shelving and the arytenoids were larger than normal. There is the appearance of a cleft between the arythenoids which continued down to the level of the posterior commissure.

As the child gets older it tends to lose its cat-like quality. Ward et al. (1968), . Gordon (1965) , and Berg et al. (1970); ( K.P. Manning 1977).

Cause of the Cry :

- Lé Jeune et al. (1963) , thought the flaccidity of the larynx accounted for the cry.

- Micheau and Schlumberger (1968), said that a malformation of the nerve centres or pathway controlling phonation must be responsible for the cry.
- Schroeder et al. (1967) , thought that an organic or functional cerebral lesion is the cause of the cry.
- Gordon (1965), thought that some of the abnormality of the cry must be due to hypotenia. ( K.P. Manning 1978).

5. CONGENITAL LARYNGEAL CLEFT

This very rare condition has in many cases been diagnosed in the post-mortem room. This is because

(a) the extreme rarity means that the surgeon does not consider the possibility and ,

(b) the area is difficult to visualize adequately. The cricoid lamina and a variable portion of the posterior wall of the trachea are divided by a cleft. The protective action of the supraglottic sphincter is lost and food and saliva spill through the cleft into the tracheobronchial tree. Frequent chest infection occurs, there is considerable difficulty in feeding and the child fails to gain weight . Cyanosis and

apnoeic attacks are not uncommon. ( R. Pracy 1979).

SYMPTOMS AND SIGNS :

Feeding difficulties, and stridor which is usually "two way " are present from birth. Coughing, spluttering and cyanosis may all be the cause for the initial consultation . The baby is restless and fails to gain weight. On examination there may be obvious discolouration of the face and perhaps excess saliva around the lips. The baby may be febrile and coarse sounds may be heard in the chest ( R.Pracy 1979).

DIAGNOSIS :

The laryngotracheobronchial tree should be examined under general anesthesia, The cleft may , not be easy to visualize even with modern endoscopies. However, it may be possible to pass a probe or bougie from the trachea to the oesophagus without withdrawing it from the larynx. If this appears to be the case then the area should be examined further by contrast radiography. (R. Pracy 1979).

6. CONGENITAL CYSTS :

These may be of three types.

(1) They may be isolated , in which case they are cysts of mucous gland in the false cords and may extend laterally.

(2) They may be part of a generalized lymphangiomatous condition, in which case the treatment of the lymphangioma as a whole will have to be taken into account.

(3) They may be associated with a laryngocoele. These are seen later in childhood and may be diagnosed accidentally. ( A.G.D. Maran 1979).

Cysts usually give rise to acute airway obstruction and this obstruction is seen when the cyst suddenly increases in size. When this is part of a lymphangiomatous process it is probably wise to carry out a tracheostomy and to wait because many of these tumours do not grow as fast as the baby . In this case no definitive treatment for the lymphangioma will be required and in due course it should be possible to remove the tracheostomy cannula and to leave a normal voice and airway. (P.M. Stell 1979) .

7. CONGENITAL HAEMANGIOMA OF THE LARYNX :

Hemangiomata, usually of the cavernous type, tend to occur anteriorly in the infraglottic region of the

larynx. They occur most commonly in females in a ratio of more than 5 to one and, one-half of the patients will have cutaneous hemangiomas ( William T. Brown 1967).

CLINICAL MANIFESTATIONS : The symptoms are those of laryngeal obstruction including : Stridor ( which may be biphasic) dyspnea, cyanosis and indrawing. Symptoms may be absent at birth and may first appear in association with an upper respiratory infection, then they persist after the infection resolves. They are made worse by placing the infant in a trendelenburg position and are often improved when the infant is held erect. ( J.J. Ballenger 1969).

The presence of cutaneous hemangiomas and the absence of symptoms in the early postnatal period may be a clue to the diagnosis. Direct laryngoscopy discloses a red or purple mass covered by normal epithelium in the area beneath the vocal cords. A helpful diagnostic maneuver is the subcutaneous injection of 1 or 2 minums of 1: 1000 epinephrine which will cause blanching and shrinking of the hemangioma and transient relief of symptoms. This procedure should be done at the time of direct laryngoscopy when the changes may be visualized.

Biopsy of the lesion is dangerous if a tracheostomy has not been done. After tracheostomy and evaluation of the lesion, a small biopsy may be taken with reasonable safety, if it is felt to be necessary although it is perhaps best avoided. Soft tissue roentgenograms of the larynx are of help in delineating the size and extent of the lesion ( Max K. Pierce 1962).

8. CONGENITAL VOCAL CORD PARALYSIS :

Some degree of weakness of one or both vocal cords is one of the more common congenital lesions of the larynx. About 25% of all cases of stridor in infancy are found to have vocal cord weakness ( R.Pracy 1979).

The cause is obscure in many cases, in the case of unilateral paralysis, the right cord is most frequently involved. When the left cord is paralyzed, there may be associated cardiac abnormalities, such as patent ductus arteriosus. Bilateral cord paralysis is usually associated with other central nervous system abnormalities such as Arnold Chiari syndrome. In these cases, the onset of paralysis is probably related to increasing hydrocephalus, since the cord paralysis is not manifested until four to

six weeks after birth when hydrocephalus becomes marked. When cord paralysis is present, it will often disappear after ventricular tap and insertion of a Holter valve. (Ballenger 1969).

CLINICAL MANIFESTATIONS : The infant has a weak or hoarse cry inspiratory stridor and difficulty with feeding . Stridor is most marked during activity and crying and often disappears at rest if paralysis is unilateral. Bilateral paralysis causes more severe obstructive symptoms. ( Ballenger 1969) .

DIAGNOSIS : Is made by direct laryngoscopy with the blade of the laryngoscope inserted in the valleula. If the laryngoscope is inserted in the larynx, it may tense the vocal cord and give a false impression of paralysis, (R. Pracy 1979).

9. ATRESIA OF THE LARYNX :

Complete occlusion of the larynx is rare as far as the clinical otolaryngologist is concerned since most of the afflicted infants are classified as stillborn or die before tracheostomy can be performed. The condition is due to a failure of the epithelial primordium of the vocal

cords to split in a sagittal plane. Therefore, atresia may be limited to the supraglottic, glottic or infraglottic level or may involve all levels of the endolarynx. The pharyngotracheal canal is the residuum of the laryngotracheal groove and it is only secondarily incorporated into the laryngeal lumen. Therefore, when atresia is present, the pinhole-sized pharyngotracheal canal can often be found in the posterior part of the larynx between the arytenoids( J.J. Ballenger 1969) .

10. TRACHEO-ESOPHAGEAL FISTULAS :

The esophagus and tracheobronchial tree arise from a common tube and initially they are connected by a long laryngotracheal groove which progressively closes in a caudocranial direction to leave the pharyngotracheal canal. Deficiencies in the closure of this groove give rise to a variety of defects in about I out of every 2500 births;( J.J. Ballenger 1969) .

SYMPTOMS : These anomalies usually give rise to symptoms immediately after birth. The afflicted infants will have persistent barking cough with episodes of choking and cyanosis which are markedly increased by feeding . Aspiration eventually leads to severe bronchopneumonia and

death unless the abnormality is corrected.( Ballenger 1969).

DIAGNOSIS : Tracheo-esophageal fistula may be associated with other abnormalities of the trachea and esophagus and four types of this defect occur. Three varieties are associated with esophageal atresia. The diagnosis may be easily established by passing a small nasogastric tube and demonstrating its failure to enter the stomach . A lateral roentgenogram of the chest may demonstrate a blind upper esophageal pouch with air in the stomach indicating an inferior tracheoesophageal connection or there may be a complete absence of air in the abdomen which would suggest the opposite defect. The use of a radiopaque dye is not necessary and invites aspiration pneumonia; also the use of barium sulfate is contraindicated in such patients. (Ballenger 1969).

The difficulty in diagnosis is with the H-type fistula not associated with atresia. The fistula may occur at any level in the posterior wall of the larynx or trachea. Esophagoscopy is of little value because the loose esophageal mucosal flap hides the tract; however, tracheoscopy may disclose the fistula. Frequently, a dye contrast examination using cinefluoroscopy is diagnostic. In a small number

of cases , it may be necessary to inject methylene blue into the trachea through an endotracheal tube and watch for the appearance of the dye in the esophagus with an esophagoscope in order to determine the site of the fistula. ( J.J. Ballenger 1969).

11. MICROGNATHIA , GLOSSOPTOSIS AND RESPIRATORY OBSTRUCTION ( PIERRE ROBIN SYNDROME ) :

Micrognathia with glossoptosis is responsible for respiratory obstruction in some newborn infants. When these deformities are associated with a cleft palate, the entity is termed the "Pierre Robin syndrome ". Although cleft palate is frequently associated with these abnormalities, the association of cleft lip is rare. The cause of the deformity is unknown, although it is familial in some instances.

Since the hyoid bone, base of the tongue and epiglottis are dependent upon their attachments to the mandible to hold them forward, a deficiency in the size of the mandible results in loss of support. The base of the tongue, therefore, falls posteriorly and approximates the posterior pharyngeal wall occluding the airway. The tongue itself is probably of normal size and macroglossia is not an important factor.

When cleft palate is present, the tip of the tongue may be trapped in the cleft which will hold the tongue in a posterior position. Tongue-tie( ankyloglossia) is often present and prevents normal forward movement of the tongue. ( Ballenger 1969) .

DIAGNOSIS : The infant presents a typical appearance with a small recessed chin with the lower alveolus one cm. or more posterior to the upper alveolus. The term "Andy Gump" has been applied to this picture.

Respiratory obstruction results from a ball valve obstruction of the pharynx secondary to glossoptosis. The obstruction may not be marked at birth but becomes much worse during the next few days as the infant becomes weaker because of feeding difficulties. Frequently the obstruction is episodic with attacks of cyanosis, stridor and indrawing occurring when the infant is supine, being fed or asleep. The airway can be improved by placing the infant in a prone position.

Feeding problems are marked. Because of glossoptosis, deglutition is altered and these infants eat only small amounts, while aspirating frequently. Failure to thrive and repeated respiratory infections result in a worsening

of the obstructive symptoms producing a syndrome of respiratory cachexia ( Ballenger 1969).

12. DYSPHAGIA LUSORIA :

Is caused by compression of the oesophagus by an unusually located right subclavian artery. The term is used to include dysphagia caused by any aberrant great vessel. The condition is confined to the upper third of the oesophogus. ( J.C. Ballantyne, J. Groves, C.H. Edwards, D. Downton 1978) .

Pressure on the larynx or trachea from without can cause signs of respiratory obstruction. In dysphagia lusoria the aortic arch may pass between the trachea or the oesophagus. In this case a pulsating narrowing of the trachea may be seen on bronchoscopy and indentation of the oesophagus and separation of the oesophagus from the trachea may be noted in the contrast radiogram of the swallow . Once the diagnosis is made the patient should be referred to the cardiothoracic surgeon for correction of the vascular anomaly).

13. TRACHEOBRONCHIAL COMPRESSION BY CONGENITAL CARDIOVASCULAR ANOMALIES IN CHILDREN " SYNDROME OF APNEA "

(Blair Fearon, Robert Shortreed, 1963) wrote a report that deals with a series of 104 patients with

tracheobronchial compression by cardiovascular anomalies in which per-oral endoscopy has been carried out.

Compression of the trachea or tracheobronchial tree by congenital cardiovascular anomalies is a rather frequent cause of stridor and/or respiratory difficulty in the infant. Many of these anomalies can be diagnosed by roentgenograms and by clinical examination. However, peroral endoscopy is not only a valuable adjunct in the diagnosis, but certain compressions, and in particular that caused by an anomalous innominate artery, may be accurately diagnosed by bronchoscopy. In certain patients with such anomalies there appears to be a syndrome of reflex apnea which makes surgical intervention almost mandatory.

Etiology : The main anomalies are:

- Anomalies in the innominate artery.
- Anomalies in the pulmonary artery.
- Anomalies in the subclavian artery which is mainly double Aortic Arch.
- Cardiac or Aortic hypertrophy and dilatation of the superior vena cava.

Symptoms and Signs :

Appear from birth, the more severe the compression, the earlier the symptoms present.

Mild stridor, wheezing, dyspnea with cyanosis.Minor respiratory infection accentuates the expiratory wheezing and brassy cough; so those children are described as having a short staccato cough ( W.H. Hermann 1928). Difficulty with feeding is common as well as recurrent chest infections because of the difficulty in coughing up secretions through the narrowed area in the trachea .

Those children lie with the head extended to stretch the trachea.

Chest examination : Biphasic stridor, laryngomalacia, intercostal indrawing and sternal retraction. Chest is hyper resonant with expiratory rhonchi.

Chest X-Ray : Shows areas of atelectasis or emphysema ( Bair Fearon and R. Shortreed 1963).

14. Laryngocele Ventricularis :

Laryngocele ventricularis is an air-filled dilatation of the sacculus or appendix of the laryngeal ventricle.

Etiology : The ventricles of the larynx develop from an evagination into the primordium of the endolaryngeal structures beginning during the second month. The primitive ventricles extend quite far laterally under the floor of the pharynx and the distal extension, the appendix, may be related, in origin, to the 4th. branchial cleft. At birth, the appendix is relatively large but about the age of six years it begins to regress in size, relative to the remainder of the larynx and ventricle. It occurs in persons with congenitally large ventricular appendices, the deformity is rare in children. The most common predisposing factor is activity which produces a recurrent, persistent increase in intralaryngeal pressure, such as straining, coughing , playing wind instruments and excessive straining with voice use.( J.J. Ballenger 1969 ).

Clinical Manifestations : Three types of laryngocele are recognized including internal, external and mixed .

The internal form consists of an air-containing sac confined to the area of the false cord and aryepiglottic fold and within the thyrohyoid membrane. The external form presents as a cystic mass on the lateral surface of the thyrohyoid membrane which is connected to the ventricle by a narrow patent tube. The tube passed through the thyrohyoid membrane in the region of the neurovascular bundle although it is usually situated inferior to the bundle. The mixed type consists of connected air-containing cavities on both sides of the thyrohyoid membrane . The symptoms tend to fluctuate, increasing with straining and disappearing at rest. The internal and mixed forms will cause hoarseness, stridor and dyspnea which worsens with continued voice use. The external type may present only as a compressible, spherical mass over the thyrohyoid membrane which moves with the larynx on swallowing . Bryce's sign may be present and is characterized by a gurgling and hissing in the throat when the neck mass is compressed. The mass may vary widely in size from time to time and may be absent at times. The patient may have a persistent productive cough, especially if infection is present ( J.J. Ballenger 1969) .

Occasionally, the opening to the laryngocele may become obstructed and fills with musus, becoming a secondary mucocele, which may become infected producing a laryngopyocele .

Diagnosis : A soft tissue roentgenogram of the neck during the Valsalva maneuver will disclose the presence of the air-containing cystic structures. Direct laryngoscopy and inspection of the ventricle is indicated to rule out an underlying neoplasm.

Treatment : Small asymptomatic laryngoceles require no therapy and will seldom cause any difficulty if causative factors are removed . Symptomatic laryngocles and laryngopyoceles should be excised . External and mixed laryngoceles are best excised by a lateral approach through the neck .

The internal laryngocele should be excised via a lateral pharyngotomy. ( J.J. Ballenger. 1969).

15. Spasmodic ( Laryngismus Stridulus ) or ( Laryngeal Tetany ) : Is a condition which is met with in children under two years of age, the majority of whom suffer also from rickets, but teething, digestive disturbances, the presence of enlarged bronchial glands, and intestinal parasites, may all be aetiological factors. Accordings to(Semon and Horsley), the condition is due to irritability of the cortical adductor centres. ( John P. Stewart 1961).

Laryngospasm may be due to hypocalcaemia. When poor nutrition was commonplace, particularly with low vitamin D intake, children were frequently seen with laryngeal tetany. Similar episodes have been reported in adults but are usually associated with hypocalcaemia due to hypoparathyroidism (Williams and Brown, 1974). The spasm follows increased, reflex excitability at the neuromuscular junctions of the recurrent laryngeal nerves. The condition is often worse during the night because of the collection of pharyngeal secretions (Jackson and Jackson 1937) . Laryngospasm due to hypocalcaemia is usually accompanied by other features of tetany including the following :( H.A. Young & I.T. Ferguson 1977).

Symptoms : the child has suddenly a few crowing inspirations, followed by apnoea for some seconds, the movements of respiration coming to a stop. The face assumes an expression of great terror , it is at first flushed but soon turns pale ; the eyes are staring, the pupils contracted; the head is thrown back, and the skin is covered with sweat . The attack ends as it began, with some stridulous inspirations. In rare cases the child dies during the seizure. The attacks may be repeated several times a day, or may occur only at long intervals. In severe cases there may be spastic

contractions of the feet and hands, the thumbs being flexed into the palms and the fingers either folded over them or rigidly extended. ( H.A. Young & I.T. Ferguson, 1977).

Prognosis : The prognosis depends on the cause. It is commonly good; some patients have died from asphyxia during an attack( John P. Stewart 1961).

Diagnosis : The diagnosis has to be made from false croup and depends on the sudden onset, the complete apnoea during the attack, and the absence of laryngeal symptoms in the free intervals ( R.B. Lumsden 1961).

II. INFLAMMATORY CAUSES :

(1) Acute Nonspeciefic Laryngitis :

Acute infectious layrngitis is usually a mild, self-limited inflammatory condition which is often a manifes - tation of a more diffuse upper respiratory infection . It is relatively common in children and especially in boys ( J.F. Birrell 1960).

Etiology : The cause of this infection is almost always a virus. Bacterial invasion may be secondary. The laryngitis is almost always associated with acute rhinitis( coryza) or nasopharyngitis. The onset of infection may be associated with

exposure to sudden temperature changes, dietary deficiencies, malnutrition and lack of immunity. The disease is more common in winter and is readily transmitted( J.J. Ballenger 1969).

Pathology : There is capillary dilatation and hyperemia associated with generalized extracellular edema, submucosal leuko cytic infiltrate, mostly of mononuclear cells, is present initially; later polymorphonuclear cells appear if a secondary bacterial infection develops. The superficial mucosal layers usually slough and shallow ulcerations covered by pseudomembranes may develop( Ballenger 1969).
Initial dryness is followed by the production of a thick mucopurulent secretion ( R.E. Ryan 1970).

Clinical Manifestations : The usuall symptoms are those of a common cold with associated hoarseness. The first symptoms involving the throat are dryness, rawness and a change in voice pitch. Cough is common. If cordal edema is marked, aphnoia ensues and mild inspiratory stridor may be evident. Fever is usually of low grade (102°F) . Early in the course of the disease, laryngeal secretions are scanty, later they may become abundant

but viscid and may be blood tinged. ( J.J. Ballenger 1969 & Lauren D. Holinger 1976).

The laryngeal mucosa is variably inflammed and edematous. The mucosa may be granular with patchy superficial ulceration and areas of grey pseudomembrane formation. Vocal cord motion is normal although the margins of the cords may be red and edematous and actually appear polypoid because of edema of Rienke's space( Ballenger 1969) .

Diagnosis : Diagnosis is made by the history and laryngeal appearance. Throat cultures are expensive and indicated only in prolonged or resistant illness. Isolation of the causative viral agent from throat washing is only necessary in the face of a possible epidemic( Maxwell Ellis 1977).

(2) ACUTE SPASMODIC CROUP (ACUTE LARYNGITIS OF CHILDHOOD)

This is a mild inflammatory disease of children which is significant because of associated laryngeal spasm and attacks of severe dyspnea. It is differentiated from laryngisms stridulus by the fact that there is no abnormality of calcium metabolism.

Etiology : The condition typically occurs in children, mainly boys, between one and four years but may occur up to age six.( J.F. Birrell 1960). There is no specific organism or disease which causes the condition but it does tend to occur in association with mild upper respiratory infections. There is undoubtedly psychogenic factor associated with the recurrent form of the disease which occurs in some children ( J.J. Ballenger 1969).

Pathology : The disease is not a true infection and there are only minor tissue changes in the larynx. Some patients manifest a redness and swelling on both sides of the infraglottic area. These changes are present only after an attack and are transient. Edema of the supraglottic areas may be present ( J.J. Ballenger 1969).

Clinical Manifestations : Typically , the child goes to bed feeling well and soon is asleep. A few hours later, he awakes with a croupy cough, stridor and dyspnea. Because of fear and panic, the respiratory effort is increased which, in turn, causes worsening of the stridor and dyspnea. Thus laryngeal, spasm is precipitated. Supraclavicular and subcostal indrawing occur and eventually a dusky to cyanotic tinge of the skin develops.

At this point, the child may become unconscious or may vomit and terminate the attack. The child then falls asleep and awakens in the morning with nothing but slight hoarseness and cough.

The attacks may be slolitary or recur for two or three nights and then disappear completely. In other children, because it is an attention-getting mechanism and because of psychogenic factors, it may recur for some time. Occasionally an attack may not subside spontaneously and obstructive symptoms persist necessitating more active therapy ( J.J. Ballenger 1969).

(3) ACUTE EPIGLOTTITIS

Inflammation of the supraglottic structures is a condition that tends to affect small children from ages of three to six years, but it occurs quite frequently in adults and is as potentially lethal as in children. It affects children especially common between september and March ( Camps 1953).

Etiology : Haemophilus influenzae type B has been the bacteria cultured most consistently from afflicted individuals. However Staphylococcus, Streptococcus and Pneumococcus have also been implicated, It is probable that

some viruses may also cause the disease. The disease is of most significance in children because of the configuration of the supraglottic larynx which predisposes to obstruction by a small amount of swelling of structures around the laryngeal vestibule ( J.J. Ballenger 1969).

Pathology : There is a severe cellulitis of the tissues of the epiglottis and aryepiglottic folds. The mucous membrane is bright red and edematous.( Bruce Benjamin 1976) . Excretions on or in the larynx tend to be thick and inspissated. Obstruction of the laryngeal vestibule prevents effective cough and removal of secretions creating a second obstructive problem ( J.J. Ballenger 1969).

Clinical Manifestations : The course of this diseáse is, in most instances, rapid, progressing from the symptoms of mild upper respiratory infection to almost complete respiratory obstruction over a period of six to twelve hours. The disease begins with fever which is often high (104°F) Brian O'Reilly 1976) Both adults and children are extremely ill, appearing pale and lethargic. They tend to sit or lie quietly concentrating only on breathing. It is common to see the children sitting or standing in bed

leaning slightly forward and with their mandible projected to facilitate respiration. The mouth may be held open with the tongue protruding. The voice is not usually hoarse but has a muffled quality and the patient has the appearance of having a " hot potato" in his mouth. (J.J. Ballenger 1969).

The respirations are variable but may change markedly in a few moments. Initially, the patients are amazingly quiet, compared to those with laryngotracheitis, because maximum ventilation is obtained by slow, steady, inspiration whereas vigorous inspiration will cause the swollen epiglottis to obstruct the vestibule. Stridor is not an outstanding feature and it is variably present as an inspiratory and/or expiratory rattle. From this point, any increase in respiratory effort causes the swollen epiglottis to act as a ball valve completely obstructing the larynx at each inspiration. The signs of marked indrawing, increasing restlessness and cyanosis will appear rapidly. As distress continues, the children develop a peculiar ashen-gray color which is a significant sign of impending respiratory arrest. As respiratory distress increases, they become restless and disoriented( J.J. Ballenger 1969).

Dysphagia is an important symptom. Early in the course of the disease there may be complaints of sore throat and pain on swallowing . However, with progression, the patients will have difficulty swallowing liquids and secretions and will refuse oral feedings. ( J.F. Birrell 1960).

General reactions to increasing obstruction and dysphagia include dehydration, hyperpyrexia, tachcardia, anxious facies and restlessness. Finally, exhaustion with respiratory and circulatory collapse develops ( J.J. Ballenger 1969).

Oral examination will show the cropharynx to be somewhat injected with pooling of secretions in this area. Using a tongue depressor, the cherry-red edematous epiglottis may be seen in most children projecting above the posterior tongue ( Imrich Friedmann 1973). Caution must be used during examination as acute obstruction may be precipitated. If direct laryngoscopy is done to insert an airway , the true vocal cords may be seen to be slightly injected but otherwise normal. Auscultation of the chest will often disclose a remarkable paucity of breath sounds denoting decreased air exchange( J.J. Ballenger 1969).

Diagnosis : made in the clinical findings . Direct laryngoscopy is rarely necessary . X-ray of soft tissues of neck shows the supra glottic dwelling . X-ray chest is indicating since atelectasis and pneumonia may accompany the disease ( J.J. Ballenger 1969).

A greatly increased neutrophil polymorph count in the peripheral blood may assist in the differential diagnosis from a viral disease. Blood culture is an important investigation because it may provide unequivocal retrospective evidence about the nature of the infection. Isolation of an untyped H. influenzae strain from the throat or epiglottis is of no significance, because nonencapsulated strains of this species can be recovered from such sites in 50 percent or more of healthy children. The isolation of a type B strain is carried by only a few members of most healthy communities but has been found responsible for nearly all cases of epiglottitis. ( Imrich Friedmann 1973).

Differential Diagnosis : In uncomplicated cases however the condition must be differentiated from:

1. Acute laryngotracheitis : This is the most common clinical problem and Table II shows the distinguishing features :

TABLE II

FEATURES DISTINGUISHING ACUTE LARYNGOTRACHEITIS FROM ACUTE EPLGLOTTITIS

| Acute Laryngotracheitis | Acute Epiglottitis |
|---|---|
| 1. More in males. | 1. Males and females equal. |
| 2. Occurs below the age of five and often recurs. | 2. Occurs at any age and never recurs. |
| 3. Severe airway obstruction takes from one to several days to occur, with a variable clinical course. | 3. Severe airway obstruction occurs within 6 to 24 hours with a progressive clinical course. |
| 4. There is a firm indurated pink subglottic swelling. | 4. An acute inflammatory fulminating swelling in the supraglottic laryngeal tissues. |
| 5. Little toxemia or generalized effect as the etiology is almost certainly a localized viral infection. | 5. Severe toxemia & prostration from septicemia due to H influenzae type B. |
| 6. Cool moist air is the main therapy and steroids are probably beneficial, antibiotics being used only for secondary bacterial infection. | 6. Parenteral antibiotics always necessary and steroids probably of little benefit. |
| 7. Very seldom requires tracheotomy. | 7. Usually requires tracheotomy. |

( Imrich Friedmann 1973).

2. Foregin body in the pharynx , larynx, trachea or upper esophagus. Although a sudden choking attack may have been observed, there is frequently no history. Foreign body should be consisdered where laryngospasm, husky voice, stridor or inspiratory wheeze dominate the clinical picture. An upper esophageal foreign body may develop periesophgeal swelling and cause tracheal compression ( Bruce Benjamin& Brian O'Reilly 1976).

3. Laryngopharyngeal diphtheria : The child is seldom proprerly immunized. There may be evidence during a minor epidemic, of contact with other known cases of diphtheria, the child is toxic and a diphtheritic membrane may or may not be seen in the oropharynx. ( B. Benjamine and Brian O'Reilly 1976).

4. Gross hypertrophy of the tonsils and adenoids, the diagnosis is confirmed by inspection of the oropharynx.

5. Acute bacterial abscess of the pharyngeal area. This includes peritonsillar abscess, retropharyngeal abscess, pharyngomaxillary space abscess and para-pharyngeal abscess. There are usually features of a preceding infection of the tonsils or pharynx,

then persistent high fever, irritability, enlarged cervical laymph nodes and airway obstruction. Respiratory obstruction may be especially serious in infants with a retropharyneal abscess. ( Bruce Benjamin & Brian O'Reilly 1976).

(4) ACUTE LARYNGOTRACHEOBRONCHITIS :

Is an acute infection of the lower respiratory passages, extending from the larynx down into the smaller subdivisions of the bronchial tree. Since the control of diphtheria and the virtual extinction of diphtheritic laryngitis, acute laryngotracheobronchitis prime importance as the leading cause of respiratory obstruction in small children. It is endemic throughout the year, but may reach epidemic proportions in any locality during the winter months( R.E. Ryan 1970).

Etiology : Atmospheric conditions ( i.e. low humidity and cold ) may play a part in production of the disease.

The basic etiologic agent is probably a virus. A myxovirus, parainfluenza type I, is the agent most frequently isolated but other myxoviruses, parainfluenza 3 and influenza A and B , may also cause the disease . The

adenoviruses have also been implicated. After the viral infection is well established secondary bacterial infection by organisms indigenous to the nose and throat may develop. Most commonly hemolytic streptococcus and Streptococcus viridans staphylococcus aureus and Pneumococcus are cultured ( J.J. Ballnger 1969) .

Pathology : The pathologic picture is that of a descending inflammation of the mucous membrane lining the lower respiratory tract, followed by congestion, edema , and exudation of a thick, tenacious secretion. Ulceration and crusting occur in the more severe cases. If the respiratory obstruction is permitted to continue over a period of time ,secondary vascular changes take place as the result of increased intrathoradic pressure ( R.E. Ryan 1970).

Symptoms : At the onset the disease is not unlike an ordinary cold except for the early presence of a croupy cough which is frequent at night. The term croup has been lossely applied to several laryngeal conditions which are characterized by the triad of inspiratory stridor ( or more severe respiratory obstruction), hoarseness and barking cough. Several distinct pathologic entities of various etiologies

may present with these symptoms. The term is used primarily with children and specifically refers to acute laryngotracheobronchitis ( viral croup).( Lauren D. Holinger 1976).

Hoarseness is noted shortly thereafter, either in the cry or in the speaking voice of the child. As the swelling increases, laryngeal stridor develops, this noise,which is caused by the passage of air through the narrowed glottis , is more marked on inspiration. Retractions occur as the patient finds it increasingly difficult to draw air into his lungs. This indrawing of the soft tissues around the rigid thoracic skeleton is most marked in the supraclavicular and infraclavicular regions ; in a small child the anterior abdominal wall in the epigastric area nearly touches the vertebtal column during the violent excursions( R.E. Ryan and J.H. Ogura 1970).

Lesser retractions may be observed in the intercostal spaces and above the sternum. Circumoral pallor and cyanosis usually precede a decrease in breath sounds that, in turn, is an indication that death is imminent. In addition to these symptoms of respiratory embarrassment, anorexia and fever are common in the early stages, while restlessness,

dehydration, and exhaustion may be noted later. The most striking phenomenon about this disease is the rapidity of its course. While it may take one or two days to develop the characteristic symptoms, death from respiratory, obstructions may intervene within hours in a fulminating case.( R.E. Ryan and J.H. Ogura 1970 ).

As the condition progresses, the picture may change rapidly. The child becomes stuporous, lies quietly and develops a glazed facial expression. There is decreased air entry to the chest and little effort to cough. True cyanosis is not present and the color becomes ashen-gray. Respiratory efforts are diminished and this may lead one to believe that improvement is occurring, much to the detriment of the patient. The signs of tachycardia, ashen-gray color, reduced air entry to the lungs, and lassitude are evidence of exhaustion and impending respiratory failure. This situation should be anticipated and tracheostomy done before it develops ( J.J. Ballenger 1969).

Diagnosis : The diagnosis may be suspected by the history, but supraglottic laryngitis must be ruled out. A lateral roentgenogram of the neck will reveal the infraglottic

narrowing, but direct laryngoscopy is indicated to rule out other obstructive conditions ( J.F. Birrell 1960) Direct inspection of the larynx may be performed without anesthesia in children. A small diffuse redness is noted in the infraglottic area and this may be associated with an apparent increase in tissue mass. The supraglottic structures may be red but are otherwise normal. The lack of a significant membrane helps differentiate this disease from diphtheria. A bronchoscope or endotracheal tube and tracheostomy set must be on hand before laryngoscopy is performed in the event obstruction is precipitated. A smear and culture of the larynx should be taken for significant pathogens. Serum antibody levels may be helpful in the diagnosis of a viral infection particularly if there is a rising titer.

Blood counts will vary as to the stage of the disease. Initially there may be leukopenia but in the more severe later cases, a leukocytosis of moderate degree is present. ( J.J. Ballenger 1969).

Prognosis : The prognosis depends entirely on early recognition of the disease and upon timely hospitalization and treatment ( R.E. Ryan 1970).

(5) MEMBRANOUS LARYNGITIS :

Membranous laryngitis is common but it is usually associated with other disease processes such as infectious mononucleosis , viral diseases and Vincent's infection.

Membranous laryngitis of nonspecific etiology may occur in adults but is most frequent in children, it may occur in infants. In small children, it may represent a form of acute laryngotracheobronchitis. It tends to occur during epidemics of respiratory infections both in children and in adults . Recently, we have observed the disease in two neonates born after premature rupture of placental membranes ( J.J. Ballenger 1969).

Etiology : Various organisms have been cultured from these cases including Pneumococcus, Streptococcus and Staphylococcus. However, a significant number of patients have had gram-negative organisms such as Pseudomonas aeruginosa. Proteus vulgaris and Proteus mirabilis isolated from the membrane. Other predisposing factors appear to be upper respiratory infections, depressed immune responses , debilitation and diabetes mellitus ( J.J. Ballenger 1969). It occures also as a complication of zymotic disease and exposure to cold and damp. ( Maxwell Ellis 1977) .

Clinical Manifestations : The symptoms are hoarseness, croupy cough and dyspnea with inspiratory stridor. There is loos of appetite and the older patients complain of thirst and odynophagia. As cough becomes worse, post-tussive laryngeal spasm occurs which increases the respiratory difficulty. Sudden episodes of obstruction may occur, especially in small children secondary to glottic obstruction by a loose piece of membrane. During such episodes, the patient is aphonic and crowing inspiratory stridor is evident ( J.J. Ballenger , 1969).

Examination discloses the surfaces of the aryepiglottic folds, arytenoids and false cords to be covered with discrete patches of loosely attached yellow-white membrane. The membrane is friable and easily detached while the underlying mucosa is red and granular but does not bleed. In contrast to specific forms of membranous laryngitis, the membrane formation is often confined to the larynx ( Maxwell Ellis 1977).

In addition to the laryngeal symptoms episodes of gram negative septicemia may occur marked by high fever, prostration and shock. A blood culture should be obtained in such instances.

(6) SPECIFIC ACUTE INFECTIONS OF THE LARYNX :

a. Diphtheria Diphtheria is an acute infective disease which may involve a part of or all of the upper respiratory tract. The disease occursin children over six years of age but adults may be affected. This disease is uncommon in most areas in the United States because of active immunization in infancy ( J.J. Ballenger 1969).

Etiology : The disease is caused by Corynebacterium diphtheriae, a gram-positive organism of which there are three types:
Gravis, intermedius and mitis. Disease caused by the gravis type is rare in North America. The types are separated by colony morphology on the tellurite medium and their ability to ferment sugars( Maxwell Ellis 1977).

The disease is usually spread by droplet and fomities. It has been noted that this organism may change from a nonvirulent to a toxin-producing virulent form which may account for the occurrence of isolated cases. Since the advent of widespread immunization, the disease is uncommon but it may occur in a mild form in immunized persons ,( J.F. Birrell 1960).

Pathology : The infection is superficial, involving the mucous membranes of the nose, pharynx and larynx, producing epithelial necrosis. There is an outpouring of serum and this agglutinates into a firmly attached membrane of necrotic epithelium, bacteria, fibrin and phagocytes. The membrane may be present anywhere in the nose oropharynx, larynx and trachea.

The bacteria elaborates on exotoxin which specifically attacks heart muscle and peripheral nerves. Death results from airway obstruction or heart failure (Maxwell Ellis 1977).

Clinical Manifestations : The incubation period is one to seven days. The onset is insidious with slight sore throat, malaise and low-grade fever. There may be a mild tachycardia. Inspection of the pharynx will usually reveal the presence of a grayish-white membrane on a tonsil, the pharyngeal wall or the larynx. Laryngeal involvement is indicated by hoarseness first, followed by cough , stridor and progressive signs of respiratory obstruction. The membrane is firmly attached and leaves a bleeding bed when removed. Cervical lymphadenitis is

usually present and may be marked, giving the characteristic bull-necked " appearance ( J.J. Ballenger 1969).

Diagnosis : The diagnosis must be suspected in any form of membranous pharyngitis or laryngitis. The diagnosis can usually be made by microscopic examination of the membrane which will disclose the organisms . This is confirmed by culture of the organism on Loeffler's and tellurite media( L.D. Holinger 1976).

b. Herpes of the Larynx :

Etiology : This virus is peculiar in that it has developed a successful host-parasite relationship with man. Most people harbor the virus from infancy. Since most adults develop circulating antibodies to the virus, manifestations of infection are most common in small children between six months and three years ( J.J. Ballenger 1969).

Pathology : The virus probably resides in the cells of the mucous membranes. Usually there is an initiating factor in the onset of an acute infection such as an upper respiratory infection or trauma to the mouth. The lesions begin as punctate vesicles surrounded by a red areola. The vesicles rupture leaving a small

ulcer covered by a whitish exudate. The infection usually involveds the mouth and pharynx as well as the larynx. (Maxwell Ellis 1977) .

Clinical Manifestations : Acute herpetic laryngitis usually occurs in association with herpetic gingivostomatitis. This is a severe febrile disease of infants and small children. The lesions in the oral cavity are usually obvious. Symptoms of laryngeal involvement include hoarseness, croupy cough and stridor. Enlargement of the cervical lymph nodes is usually present . In rare instances, isolated involvement of the larynx may occur.

Diagnosis : The diagnosis may be confirmed by identification of the virus by culture on amniotic membrane or rabbit cornea. A biopsy of a typical lesion will disclose typical intranuclear inclusion bodies . Antibodies appear in the blood within four to five days after the onset of infection and a rising antibody titer is a significant indication of the causative organism. ( J.J. Ballenger 1969).

(7) LARYNGEAL EDEMA :

Etiology : Edema of the larynx may be the result of trauma, inflammation, or an allergic reaction .

neoplasms, perichondritis, intrathoracic diseases or generalized diseases as in cardiac failure and renal disease. In any case the condition usually develops swiftly and leaves little time for prolonged deliberation. The inflammatory type is commonly seen in acute laryngotracheobronchitis , diphtheria, and other infectious disease. It is particularly common in children with their loose, spongy subareolar tissues. An allergic reaction with laryngeal edema occasionally follows the injection of an antigen in which the individual has been sensitized previously ( R.E. Ryan 1970).

Pathology : The pathologic picture varies with the underlying cause. If the swelling is the result of trauma, the tissues bear the signs of injury, lacerations, or hemorrhages, if the cause is inflammatory, the picture is one of generalized hyperemia with obliteration of the normal landmarks. In allergic or angioneurotic edema the appearance is similar , but the colour of the mucous membrane is pale and the tissues appear water logged . ( R.E. Ryan and J.H. Ogura & H.F. Biller and L.L. Pratt 1970) .

The rapid spread of oedema in the laryngeal vestibule is due to the looseness of the laryngeal submucosa. Logan

Turner's investigations have shown that if fluid was injected between the epiglottis and the base of the tongue it would fill the vallecula and the anterior surface of the epiglottis, and under greater pressure, the pyriform fossa and the aryepiglottic folds. Fluid injected into the ary-epiglottic folds caused them to swell until they were pear-shaped, obstructing the laryngeal inlet. Injection of fluid into the false cords was limited to them until greater force was used , when it spread to the aryepiglottic folds. ( J.F. Birrell 1960).

In oedema of the larynx the arytenoids are first involved, and the swelling spreads to the ary-epiglottic folds. There may also be some swelling of the epiglottis. ( J.F. Birrell 1960).

Symptoms : The symptoms of laryngeal edema are so obvious and its course is so stormy that no one should remain in doubt as to the diagnosis. Hoarseness is usually noted first, sometimes followed by a cough. The gradual closing of the glottis is indicated by increasing inspiratory stridor, which assumes a higher pitch with the decrease in the airway. Retractions are most obvious in children

and presage serious difficulties , anxiety and restlessness are other warning signals ( R.E. Ryan 1960) Circumoral pallor often precedes the development of a frank cyanosis, which is in turn indicative of severe anoxemia, Unless something drastic is done to interrupt the progress at this stage, cessation of breathing may be expected shortly. ( J.H. Ogura 1960).

In asthmatic cases, the differentiation of acute laryngeal obstruction may pose a real problem. While the typical asthmatic wheeze is bronchial in origin and expiratory in phase, visualization of the larynx may be necessary for a correct diagnosis ( R.E. Ryan et al.1960).

Pfognosis : The prognosis of acute respiratory obstruction is excellent if the obstruction is relieved in time . One cannot expect good results after obstruction or cardiac exhaustion has passed beyond the stage of possible recovery (Maxwell Ellis 1977).

B. CHRONIC INFLAMMATIONS OF THE LARYNX :

a. Chronic nonspesific

1. Chronic laryngitis of childhood :

Aetiology : A raucous voice occasionally develops in children usually between the ages of five and ten (Maxwell Ellis 1971).

Infection and vocal abuse are the chief factors, and of the former the acute specific fevers are the most important. Severe laryngitis with mucosal erosion and sometimes ulceration very occasionally complicates measles, and similar condition may occur in scarlet fever, the enteric fevers, influenza, penumonia and diphtheria. This laryngitis usually resolves, but persistence may be induced by other abnormalities such as mouth breathing, sinusitis and infected tonsils (Maxwell Ellis 1971).

Many children seem incapable of speech or conversation or enjoyment in general except at the tops of their voices, particularly in city life where the level of background noise is high. Other children sign in choirs in an unnatural range. Benign growths, particularly papillomas, sometimes occur in children and provoke undue effort in an endeavour to produce a clear voice. All these types of vocal abuse may induce chronic changes in the larynx.

Finally, a condition of hereditary hoarseness is known in which the vocal cords may seem normal on inspection, although atrophy and scarring have both been recorded. ( Maxwell Ellis 1971).

Pathology : The pathology is exactly as in chronic laryngitis in adults. Depending upon the severity of the condition and its duration, pathologic changes may involve only the superficial layers of the mucous membrane, or they may extend into the deeper structures.

Patchy or diffuse hypermia of the mucous membrane covering the vocal cords is generally marked.

Various degrees of oedema and fibrosis.

Myositis occurs in the intrinsic muscles.

The production of excessive thick, tenacious mucus and adherent crusts.

Diffuse polypoid thickening may be found in more advanced cases.

The hyperaemic and oedematous stage often passes to a granular hypertrophic one rarely to an atrophic one.

( Robert E. Ryan et al. 1970).

Symptoms and Physical Signs :

Hoarseness is the only symptom and the voice has an unpleasant harsh and raucous timbre which is constant. Examination of the larynx with a mirror is often possible, with patience and persuasion, but when the child is

unco-operative direct laryngoscopy must be performed with or without an anaesthetic, depending on the technique used in the particular clinic, Garel (1921) has described the following three distinct laryngeal changes :

First , nodules are symmetrically placed on the cords identical with the condition of vocal nodules in adults.

Secondly , the whole anterior two-thirds of each vocal cord is slightly hyperaemic and uniformly swollen and rounded into a semi-elliptical shape with the convexity along the free margin so that on apposition a small space remains between the vocal processes.

Thirdly , the portion of the cord adjacent to the free margin atrophies and a line or groove of demarcation running the whole, or almost the whole, length of one or other cord, or both, appears .

( Garel ) gave the names laryngite a type nodulaire, laryngite a grains d'orge and laryngite a sillons atrophiques to these respective appearances, and in addition, maintained that the lastnamed occurs only in children, and if seen in an adult is pathonomonic of such affection in childhood. It is due to atrophy of the thryoarytenoid

muscle and is usually a late change seen in older children. This atrophy may extend to the interarytenoid muscles (Maxwell Ellis 1971).

Diagnosis : Tuberculous laryngitis is rare in children, as is nowadays hereditary syphilis . For practical purposes it remains only to differentiate the condition from a benign growth , readily achieved by an examination of the larynx which will also disclose the type of laryngitis present .

2. CHRONIC SPESIFIC DISEASES :

SYPHILITIC LARYNGITIS :

B. Chronic Spesific Diseases :

Congenital syphilis, rarely affects the larynx :

1. Early form : occurs in the first few months of life. Perichondritis is the main lesion. Acute laryngeal obstruction may be caused by the resulting oedema other luetic lesions are usually present.
2. Late Form : occurs between the ages of 2-10 y . Mucosal hyperplasia with granulations is the commonest lesion. Ulceration and necrosis follows. The epiglottis is the commonest site and may be completely, eroded. The cords may be extensively ulcerated.

Stenosis may follow . Hoarseness in a child must lead to suspicion of a leutic infection. Stridor may occur. Other stigmata may or may not be present and the W.R. may be negative or only weakly positive in a few cases ( J.C. Ballantyne et al. 1978).

3. CHRONIC TUBERCULOSIS OF THE LARYNX (LARYNGEAL PHTHISIS)

Aetiology : Secondary to pulmonary lesion. Most infections are sputogenic, a few are haematogenous. This is rare in children.

Pathology : The tubercle bacillus infect the posterior third of the laryngeal mucosa -- Myositis with weakness, then submucosal small round-cell infiltration. Surface nodules appear which caseate and ulcerate forming granulation tissue + psuedo-oedema in the epiglottis, aryepiglottic folds, arytenoids and ventricular bands. Lesions are asymmetrical. Later on perichondritis and cartilage necrosis with true oedema occures.

Clincial Picture : 1. Weakness of voice 2. Hearseness 3. Cough 4. Pain on Swallowing 5. Referred otalgia 6. Dyspnea 7. Localized tenderness.

Laryngoscopic appearances:

1. Slight impairment of adduction early sign due to myositis.

2. Marked injection of one vocal cord.
3. Ulceration of the edge of the cord ( mouse-nibbled appearance).
4. Granulations of the interarytenoid region.
5. Oedema of the mucosa of the ventricle.
6. Pseudo-oedema of the epiglottis and arytenoids.
7. Subglottic infiltration.
8. Tuberculoma.
9. Perichondritis and cartilage necrosis.
10. Vocal cord paralysis.

Diagnosis : Chest X-ray, Sputum examination and Biopsy .
( J.C. Ballantyne 1978).

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III. TUMOURS OF THE LARYNX :

A. Benign Tumours :

1. Laryngeal papillomate are the most common tumours of the larynx in childhood. Histologically, the tumour is similar to the single papilloma found in adults, but the characteristic in childhood is the multiplicity of the growths and their rapid recurrence after removal . They are not uncommon in infancy, and are presumed to be congenital . More commonly the growths cause symptoms about the age of 2 years, and occasionally no features are found until the child is aged 6 or 7 years( J.F. Birrell 1960).

Aetiology : The aetiology of these tumours is unknown but many theories have been proposed :

a. Congenital : ( Gerhardt) states that 20 percent of the juvenile type of tumours are congenital in origin.

b. Trauma : ( D'Aunoy and Zoeller) suggested that tracheal papillomata may be produced by the chronic irritation of a tracheostomy tube. There was no evidence that this was operative in any of our cases.

c. Chronic Irritation and Inflammation : In 28 such cases treated by ( Jackson and Jackson) there was a history of inhalation of poisonous gases. The histology of laryngeal papillomata is similar to that of plantar warts, and these lesions are thought to follow chronic irritation.

Tumours due to chronic irritation and inflammation are common in the larynx, but there was no clinical or histological evidence of these factors in any of our cases .

d. Hormonal Factors : These appear to play some part in the aetiology of the juvenile type of laryngeal papilloma because :

(a) There is an active phase of growth during childhood which has been called the " Juvenile phase" by ( Broyles). At puberty these tumours usually either regress or enter an inactive phase .

(b) The success of hormonal therapy as claimed by (Broyles).

(c) A case, recorded by ( Holinger), of a woman who

had suffered from multiple papillomata which regressed with each pregnancy and recurred with the onset of menstruation.

e. The Virus Theory : Many laryngologists believe that multiple juvenile papillomata are caused by a virus infection. The supporting evidence includes :

(a) Innoculation experiments of ( Ullmann 1923) . Ullmann claimed to have produced cutaneous papillomata in human volunteers, and genital papillomata in dogs, by innoculating them with " cell free filtrate " from the multiple laryngeal papillomata of children. Innoculation with filtrates from an adult papilloma failed to produce any lesion.

(b) ( Hajek) reports that the mother of small body with multiple laryngeal papillomata suffered herself from genital papillomata. It was suggested that the boy became infected with the virus during parturition.

(c) The resemblance to the common wart. The viral cause of these lesions has been conclusively proved by ( Lyell and Miles).

(d) Viral inclusion bodies . ( Ullmann) calims to have identified viral inclusion bodies in the nuclei of papilloma cells. ( Willis) stated that these " viral inclusion bodies " in tumour cells are only abnormal cell products.

The abscence of viral inclusion bodies, in papillomata does not preclude a viral cause, because( Lyell and Miles) were only able to identify inclusion bodies in 40 percent, of wards, and these lesions are undoubtedly of viral origin. ( D.J. Brain 1956).

Pathology :

Grossly the tumours are glistening, elevated, mulberry-like nodular masses which vary in color from a whitish pink to red. They may occur anywhere in the larynx, but chiefly on the true and false cords and in the anterior commissure. Frequently they extend subglottically and occasionally into the trachea and bronchi or upwards on the epiglottis, pharyngealwall , tonsil and soft palate. They vary in size from small nodules to sessile placques or large nodular masses the size of a cherry. The tumours are usually friable and bleed easily with slight trauma, a quality which makes difficult complete removal by a single operation. ( P.H. Holinger et al. 1950).

Microscopically the papilloma tissues are sessile or papillary structures composed of a vascular connective tissue core covered by stratified squamous epithelium in many layers. There are usually secondary and even tertiary stalks of vascular fibrous tissue covered by the epithelium. Cells in mitosis are frequent, indicating growth activity, but the cells are well differentiated, mature epithelial cells. The growths have no tendency to invade the stroma or submucosa. No histologic difference is recognized between the papilloma of adults and those in children. But a distinct clinical difference seems to be present. The papillomata in adult seem to be more friable, leaving a cleaner surface on forceps removal, whereas in children the tumours appear to be more deep seated and have a tougher consistency. (Paul. H. Holinger, Kenneth C. Johnston and George C. Anison 1950.).

Spread :

The spread of laryngeal papillomata has been attributed to :

(1) Contact This could occur on the true and false cords.

(2) Aspiration ;

a. Viable papilloma cells. This is supported by

(Hitz and Oesterlin) who demonstrated histologically an intrabronchial plug of papiloma cells.( Willis) has shown, however, that the lung provides poor soil for the establishment of tumour emboli

b. Viral particles, some writers consider that this is most likely to occur during biopsy or removal of the laryngeal lesions.

(3) Multicentric Origin ( D.J. Brain 1956)
Clinical Features : In the infant the first symptom is stridor which may be allied to a hoarse cry. The older child usually presents with changes of respiration and phonation. Changes of phonation usually occur first, with a change in the character of the voice followed by progressive hoarseness or huskiness, and finally aphonia. Respiratory changes are those of croupy cough, stridor, dyspnea, cyanosis and ultimately asphyxia . Compensatory phenomena to aid the patient with a slowly obstructing airway may occasionally be observed. A change in personality in children is not infrequent as slow-moving, read rather than play, and remain alone as much as possible. They begin to actively use the accessory muscles of respiration, and indrawing of the supra-and infra-sternal notches becomes pronounced ( Paul H. Holinger et al. 1950).

Clinical Course : These tumours pass through an active "juvenile phase " . Early recurrence followed the removal of these lesions when performed before puberty was reached. Spontaneous regression did not occur in any of our cases after puberty, but they appeared to enter an inactive phase, which enabled treatment to be effected with much less risk of recurrence.

The cause of this regression with puberty is unknown, but theories include :

(1) An increasing immunity to a viral infection. This theory is in direct conflict with the experimental results of Ullmann, who found that the successive incubation periods diminished, when human volunteers were repeatedly innoculated with viral filtrates.

(2) Hormonal factors.

(3) The location of the tumour and its mobility, resulting in the repeated injury to the delicate blood vessels which supply it( Schrotter).

(4) ( Hopp) has shown that the infantile cords are covered by cubical epithelium, which later differentiates into the stratified squamous type . This form of epithelium is more resistant to infection, and this is probably

a contributing factor to the frequent regression of the disease at puberty. ( D.J. Brain 1956).

Malignant Change :

All types of laryngeal papillomata are more liable to malignant change than is normal epithelial tissue. This change is much less frequent in the multiple juvenile type of lesion ( Le Jeune, McCart, Stout,). No sharp distinction can be made between benign and malignant papillary growths of the larynx. This accounts for the widely differing incidence of malignant change in laryngeal, papillomata, recorded by different authors( Holinger, Johnston and Anison o percent, Jackson and Jackson 3 percent, Putney 5 percent. Cunning 13 percent). It is impossible to be sure from a single biopsy specimen whether or not actual invasive anaplastic changes have not occurred in an adjacent part of the lesion ( D.J. Brain 1956).

Diagnosis : By Laryngoscopy that shows the pale , wart-like masses arising from any part of the laryngeal vestibule, the glottis, or the subglottis. Occasionally the growth may be confined to the anterior commissure or to the subglottic region immediately below this. Large masses of the

tumour are easily identified, but in infants the earliest evidence of the growth may appear like a drop of clear water on the surface of a vocal cord, and may be extremely difficult to recognize. ( J.F. Birrell 1960).

Papillomata may be mistaken for the somewhat similar formations of phthisis laryngea, but their anterior insertion, along with the absence of any clinical evidence of tubercle elsewhere, will serve to distinguish them from the latter variety. The absence of any marked alteration of the adjoining mucosa should also be noted and, where passible, a portion of the growth should be removed for histopathological examination ( John P. Stewart 1961).

Prognosis : Papillomata are chiefly dangerous in regard to the dyspnoea which they cause. They tend to recur after removal , but sometimes disappear spontaneously about puberty. ( J.P. Stewart 1961). Death may occur from asphyxia due to obstruction of the larynx or blockage of the tracheostomy tube by tracheal papillomatosis, or following a tracheostomy in an infant. ( J.F. Birrell 1960).

II. HEMANGIOMAS :

Hemangiomas in children occur subglottically are sessile, & may beassociated with cutaneous hemangiomas. The lesion is usually unilateral, compressible, and of red or dark blue color. These lesions usually manifest symptoms of fluctuating airway obstruction by six months of age. Biopsy is contraindicated because of hemorrhage. Diagnosis is made on the history and endoscopic findings.

III. Adenoma :

These are rare lesions. They arise from the mucous glands, and therefore the most common of origin is usually the false cords or the ventricle . Because the tumour formation lacks duets, adenomas become cystic and pedunculated. If oncocytes are present histologically, the tumour is termed oxyphilic adenoma or ( oncocytic cyst adenoma) ( J.H. Ogura, 1973).

IV. FIBROMA :

Fibromas are small pedunculated lesions which usually arise from the true vocal cords. Hoarseness is the only symptom.

Fibroma is the next to (Papillomata ) most common benign tumour in childhood. It may occur singly , but more frequently a small nodule is found at the junction of the anterior and middle thirds of each cord. These are known as( Screamer's nodes) because they are thought to be due to vocal abuse. The only symptom is hoarseness. If the nodes are small, they may be left alone, and will disappear in time, if the child can be persuaded to rest the voice. Should they enlarge they may be removed at direct laryngoscopy with sharp cut-ended forceps( J.F. Birrell 1960).

Laryngoscopic removal is adequate and recurrence is rare ( J.H. Ogura 1973).

V. CHONDROMAS

A chondroma is a slow-growing lesion . It is composed mainly of hyaline cartilage and affects males more often than females in a ratio of 10:1 . Chondromas may arise from the internal or external aspect of the laryngeal cartilage, the most frequent site of origin is the internal aspect of the posterior plate of the cricoid, followed by thyroid, arytenoid, and epiglottis. Though it may present supraglottically, subglottic involvement is more frequent ( Joseph H. Ogura 1973).

The symptoms are insidious because of the slow growth. Hoarseness, dyspnea, and dysphagia, Hoarseness is related to the restriction of cord mobility by the expanding mass.

Indirect laryngeal examination indicates a smooth firm, round or nodular, fixed tumour covered by normal mucosa. Pallor of the overlying mucosa may be present. Palpation of the tumour at direct laryngoscopy confirms the firmness of the mass. Chondromas of the thyroid, cricoid, or tracheal cartilages may present as a hard

neck mass attached to the thyroid, cricoid or tracheal cartilages. Biopsy may be difficult to obtain because of the firmness of the lesion. The mucosa, should be removed prior to biopsy of the underlying lesion ( Hugh F. Biller 1973) .

Soft tissue films, laminograms, and laryngograms will delineate the extent and site of origin of the lesion. The majority of patients exhibit evidence of calacification on X-ray. The diagnosis can be suspected in every case ( J.H. Ogura 1973).

VI. NEUROFIBROMA :

Neurofibromas of the larynx are solitary tumours, but may be associated with generalized neurofibromatosis( von Recklinghausen's disease) (Mary chang-LO 1977) . The incidence favours females 2:1 and the tumour may occur in any age group. The site of origin is from the neurolemma( Sheath of Schwann) and not from the axis cylinder. They arise most commonly from the aryepiglottic (AE) fold, but can arise from the true and false cord. Symptoms are hoarseness and dyspnea, When these tumors reach great size and involve the posterior arytenoid area, dysphagia may be

the only presenting complaint ( J.H. Ogura 1973).

Indirect laryngoscopy indicates a rounded, solid encapsulated tumour with intact overlying mucosa, Biopsy can be performed without difficulty. ( Joseph H. Ogura, and Hugh F. Biller, (1973).

B. MALIGNANT TUMOURS OF THE LARYNX :

1. CARCINOMA :

95% of all malignant neoplasms of the larynx are squamous cell carcinoma histologically; sarcoma, adenocarcinoma and metastatic neoplasms comprise the remaining 5%.

Laryngeal carcinoma remains localized for a long period of time due to paucity of lymphatics at the true cord level and the resistance of tumour invasion by the perichondrium.

Squamous cell carcinoma affects males more than females (9:1) with the greatest frequenty in the age group of 50 to 70 years, but it is very rare in children.

Cancer larynx is either glottic, supraglottic, infra glottic or transglottic ( J.H. Ogura 1973).

Symptoms and Signs :

1. Hoarseness of the voice . 2. Airway obstruction due to occlusion of the airway by the mass or by vocal cord fixation. 3. Sore throat 4. Dysphagia. 5. Reffered pain to the ear of short duration and sharp in quality ( H.F. Biller 1973)

Diagnosis :

- Indirect or Direct laryngoscopy.
- Palpation of vallecula and base of tongue
- Laryngo grams and Biopsy.

2. SARCOMA :

Neoplasms of the larynx are certainly not uncommon, but ( Fibrosarcoma) is only infrequently seen in any large tumour clinic . Serious neoplasms of the larynx in children are rare ( Daniel Miller 1950).

Cancer has become the second ranking cause of death from diseases in children between one and 14 years of age.

(Jackson and Jackson 1941) reported one death from cancer of the larynx in a body under four years.

( Rigby and Holinger 1943) , described a case of fibrosarcoma of the larynx in an infant aged 17 days.

The infant died soon after a tracheotomy. The diagnosis of fibrosarcoma was not made until autopsy.

Fibrosarcoma is mainly of neurogenic origin, composed of fiberforming C.T. cells and is considered highly malignant with high mortality rate. Lymph node metastasis is highly rare.

IV. FOREIN BODIES AND TRAUMA OF LARYNX :

A. FOREIGN BODIES IN THE ESOPHAGUS OR RESPIRATORY TRACT :

Introduction : Geographically and structurally the larynx is the most logical place for an obstruction of the airway. Located at the end of the combined respiratory and alimentary channel, the larynx is exposed to all foreign bodies entering the nose, mouth, or throat. It construction as the narrowest portion of the respiratory tract predisposes it to any blockage, and the presence of the loose areolar tissue promotes rapid obstruction of the soft tissues by edema ( Robert E. Ryan 1970).

The natural tendency in young children is to carry any portable object to the mouth, while older children are continually experiementing on the feel of different objects with their teeth. In children, probably, the protective reflex action is not so efficient as in the adult, and objects held in the mouth slip into the oesophagus much more easily, or are inhaled into the air-passages( John P. Stewart , R.B. Lumsden 1961).

Etiology : The propensity for small children to put whatever comes into their grasp into their mouths is well known. To this may be added their tendency to imitate adults. The mother who holds an open safety pin in her mouth while she diapers her child should not be at all surpirised when the infant puts an open safety pin in his own mouth at the first available opportunity. Foreign body ingestion may be encouraged by failure of the patient's protective mechanisms in several ways. Diminution of perception and reflex action in an epileptic seizure, deep sleep , or unconsciousness is also a contributing cause.

Carelessness may contribute to foreign body ingestion in many ways: improper preparation of food, hasty eating

and drinking, permitting children to play while eating, talking with food in the mouth, giving food such as peanuts, to children who do not have the proper molar teeth to chew them, improper supervision of small children playing in the vicinity of infants. On a number of occasions small children have been seen to deliberately feed an object they knew to be dangerous( such as a safety pin ). to an infant sibling. Such primitive solutions to the problem of sibling rivalry can be eliminated only by careful supervision. ( Gabriel F. Tucker, 1973).

Symptoms : Initial contact of the foreign object with the respiratory mucosa, classically produces, choking gagging , coughing and wheezing, depending on the exact location ( Jackson and Jackson 1959). As the stimulated mucosa accommodates to the presence of the foreign object, a symptomless interval often follows such manifestations. If the initial reaction caused either the expulsion of the offending foreign object or its passage into the lower gastrointestinal tract, the episode may be quickly forgotten by the patient without further sequelae. The initial symptomatology may have been unrecognized or

forgotten if the foreign objectis retained. The occurrence of late symptoms depends on patient reaction to the foreign object. This may have occurred in a variety of ways depending on whether the object ingested cause chemical or mechanical irritation or interfered with physiological functions such as the passage of air or food. Persistent pain is not common in the esophagus or tracheobronchial tree. Intermittent pain in association with attempts to swallow in the presence of an esophageal foreign body is , of course, not unusual. ( Gabriel F. Tucker, 1973).

Laryngeal Foreign Body :

Foreign bodies lodging in the larynx which are completely obstructive usually cause sudden death. Objects which are only partially obstructive and thus compatible with life may cause hoarseness, croupy cough, aphonia, odynophagia, hemoptysis,wheezing and varying degrees of dyspnea. These symptoms may be due to the foreign body itself still lodged in the larynx or to a foreign body which has migrated to the trachea and left a residual laryngeal reaction. Such symptoms may also be caused by laryngeal reaction to digitál or instrumental attempts at

removal. If the foreign body is in fact lodged in the esophagus, there may still be sufficient periesophageal reaction and obstruction to cause secretions to overflow through the larynx and cause the outlined symptoms as secondary manifestations of the presence of a foreign body. When large or odd shaped foreign bodies find lodgement behind the larynx, they may exert sufficient pressure on the soft and yeilding cartilaginous structures of the infant's larynx as to cause severe stridor ( John Fitch Landon, 1953) foreign body in the larynx and the trachea had partially shut off the exchange for a long time, resulting in hypoxia, yet still maintaining life, and finally causing acute pulmonary edema, which had almost cleared after removal of the foreign body and positive pressure breathing through a face mask. However, the long duration of the foreign body and obstruction had apparently caused cerebral or neurological changes which resulted in a secondary reflex episode of acute massive pulmonary edema. ( H.N. Bhatnagar 1974).

F.B. of the Pharynx :

Less common in the pharynx than in the oesophagus. Smooth rounded objects are usually held up at, or just

below , the cricopharyngeal sphincter. These are considered with oesophageal foreign bodies.

Sharp and Irregular Foregin bodies of the pharynx :

may be arrested in the tonsils, fauces, base of the tongue, valleculae or pyriform fossae. Small fish bones and toothbrush bristles are the commonest and are usually impacted in the tonsil. They can usually be removed perorally by forceps, but direct endoscopy under general anesthtesia is preferable for foreign bodies in the pyriform fossae or valleculae ( John C. Ballantye et al. 1978) . It is important to remove F.B. of Pharynx as an acute inflammation of the pharynx may be caused. If the wall of the pharynx is perforated surgical emphysema may result, still worse, cellulitis of the neck and mediastinitis or a parapharyngeal abscess which may cause haemorrhage from the great vessels. Endoscopyremoval for F.B. in laryngopharynx; but F.B. above the Postcricoid portion may sometimes be removed by Mackenzie's angular laryngeal forceps guided by a mirror (R.M. Handfield - Jones and Sir Arthur E. Porritt 1957).

Tracheal Foreign Body : Pathognomonic are :

(1) audible slap, (2) palpatory thud, and (3) asthmatoid wheeze. Diagnosis is by radiographic examination,

auscultation, palpation, and bronchoscopy. The a dible slap is best heard at the open mouth during cough. The asthmatoid wheeze is best heard with the ear at the patient's open mouth. This is especially significant if there is a history of initial choking, gagging, and wheezing( G.F. Tucker 1973).

Esophageal Foreign Body : There is often a history of initial choking and gagging. However, even without these there may be a subjective feeling of " something " foreign in the throat. This is occasionally constant, but is more often present on the act of swallowing. Associated muscle spasm or incipient perforation may give rise to substernal or epigastric pain that sometimes extends to the back. Flat objects such as coins, when lodged just behind the cricoid or in the narrow space between the trachea and the cervical spine at the thoracic inlet, most often lie in the coronal plane. Disc-shaped objects are most often found just below the cricopharyngeus in the cervical esophagus. This is thought to be the increased retention by the circular fibers of the true esophagus once the coin has dropped below the slot-shaped lumen

of the cricopharyngeus at its point of attachement to the cricoid cartilage .( G.F. Tucker, 1973).

Radiologicaids : current radiologic techniques assist in the establishment of the diagnosis and removal of foreign bodies so incomplete studies may lead to errors in diagnosis. Also inspiration-expiration films and fluoroscopy for radiolucent foreign bodies help in diagnosis . Newer Fluoroscopic-techniquies with television-type monitoring may add a third dimension of perspective to the endoscopic image. ( G.F. Tucker, 1973).

Neck : The lateral soft tissue film of the neck is one of the most useful single films available to the endoscopist. Even without supplementary contrast material, the caliber of the airway is often readily appreciated. Holinger (1962) , has suggested an endoscopic lateral film.

Chest : In addition to the posteroanterior, anteroposterior, and lateral films of the chest normally taken during inspiration similar films taken at the end of expiration are often most helpful. Such studies are especially useful to delineate obstructive emphysema since the trapping of air behind the foreign object and the failure

of the trapped air to empty on expiration will be most apparent on this type of film. By fluoroscopy the entire respiratory cycle can be continuously observed during chest motion ( G.F. Tucker 1973).

Contrast Study : Contrast materials such as barium should be used with great caution and only after plain films have been found to be inadequate to meet the specific problem. An object such as a chicken or pork bone may be apparent in the lateral soft tissue film of the neck. Administration of contrast material in such a situation normally tends to delay the performance of the endoscopic procedure until such material has passed beyond the stomach. Contrast material retained above an obstructive foreign body not only would complicate the attempt at the removal of the foreign object but might also be aspirated into the respiratory tract. Bronchography has occasionally been useful to demonstrate the relation of a given foreign object to the bronchial tree or even to localize an otherwise radiolucent plastic foreign object( Gabriel F. Tucker, 1973) .

Prognosis : The prognosis varies with the size of the foreign object and the degree of the obstruction.

Endoscopy for Foreign Body : Ingested foreign bodies should be regarded as true emergencies only when the airway is embarrassed or there is imminent of perforation of if it is felt that the foreign body may readily migrate to a more dangerous position . A minimal morbidity and mortality will depend on an adequate preparation of the patient, the instrumentarium and the endoscopic team before starting the procedure, If two hours are spent in such preparation, the safe endoscopic removal of the foreign body may take only two minute. (Holinger 1962).

An unsuccessful attempt-for whatever reason -may well imply that repeated instrumentation should not be undertaken for another several days. During which steroid-antibiotic coverage are given to lessen not only laryngeal reaction but also bronchial reaction at the site of lodgement of the foreign object.

Only in the most desperate emergency should removal of a foreign object be attempted at the scene of ingestion.

As pointed out previously, ill advised attempts at first aid may precipitate a fatal respiratory obstruction. Most community hospital emergency rooms are equipped to provide basic X-rays and such lifesaving procedures as tracheotomy, however, few are staffed around the clock with personnel sufficiently experienced to handle more than the most urgent problems.

(Holinger, 1962 ; Tucker, 1968 ; Adriani, 1969 ; Fearon 1970). Said that the endoscopic team should be capable of working safely with awake patient whenever general anesthesia would increase the risk of the procedure. This is especially true in those situations in which the anesthesiologist and the endoscopist must share a respiratory tract which is not functioning optimally, the induction of anesthesia should be begun in the endoscopic operating room after the endoscopic team is fully prepared to proceed. This is required since the induction of anesthesia may precipitate coughing, retching and so forth, which may in turn cause the foreign object to shift into an obstructive position and thus require the immediate availability of the endoscopic team to preserve the patient's airway.

A foreign body in the esophagus, the following points should be considered : The orotracheal tube must not enter the esophagus during intubation if the foreign body is in the cervical esophagus. A nasogastric tube is contraindicated in the presence of an esophageal foreign body. With endotracheal intubation the anaesthesiologist squeez the inflatable bag creating an endothoracic pressure which is greater than atmospheric pressure to collapse the esophagus around the foreign object which may displace the posterior tracheal wall anteriorly and thus make intubation difficult. ( Gabriel. F. Tucker 1973).

B. TRAUMA OF THE LARYNX :

Causes : I. Mechanical :

1. External :

a. Auto accidents.

b. Other blunt neck injuries, neck.

c. Complication of tracheostomy.

d. Cricothyrotomy.

2. Internal :

a. Endoscopic procedures.

b. Endotracheal intubation.

c. Indwelling nasogastric tubes.

II. Burns of Larynx :

1. Thermal burns :

a. Ingestion of hot food or liquid.

b. Inhalation of hot air or gas.

2. Chemical :

a. Lye ( NaOH, KOH ) b. Ammonia.

c. Sodium hypochlorite ( Clorox).

d. Orthophenylphenol ( Lysol) .

III. Irradiation Injury :

IV. Autogenous Trauma ( Voice abuse ) ( J.J.Ballenger 1969).

TYPES OF TRAUMA OF THE LARYNX : 1. Mechanical .

Aetiology : The cause is often related to the age of the patient. In small children, internal injury is most frequent. Iatrogenic injuries caused by traumatic or prolonged tracheal intubation are the most frequent causes. Aspiration of sharp foreign bodies or corrosives are other common causes of internal injury. Any game involving contact, swinging of bats, sticks etc. may cause injury to the larynx. In older children and adolescents, collision with a steretched wire or cable is frequent. The patient may have been riding a minibike, motorcycle or bicycle, or water skiing and run into the cable or other object. The force in this instance, may completely separate the larynx and trachea, but it usually throws the patient backward before dislocating the cervical spine. ( Jerry W. Tempeler 1976).

Acute Laryngeal Injury : The structure and location of the larynx are such that injury to the organ is followed by the development of lesions producing distinctive signs and symptoms. The mucosal lining of the larynx and pharynx is easily torn by traumatic forces and this may be followed by the rapid appearance of subcutaneous emphysema. Also the dehiscence of the mucosal barrier permits contamination

of the deep tissues of the neck followed by the development of cellulitis, abscess and, perhaps, fistulous tracts. ( J.J. Ballenger 1969)

Fractures and dislocations of the laryngeal cartilages and joints of a wide variety occur and they tend to be more severe with the less resilient, calcified cartilages of older individuals. Perichondrial injury is often associated with andlead to subperichondrial hematoma and devascularization and necrosis of hyaline cartilage. If the area contaminated by connection with the laryngeal lumen perichondritis and chondritis may follow . ( J.J. Ballenger 1969).

Healing of laryngeal injuries is by granulation and eventual fibrosis. Since the wounds are usually secondarily infected epithelization is often delayed with the result that excessive granulation and fibrous tissue are laid down. Cicatrization is followed by deformity and considerable permanent alteration in laryngeal function and patency may develop secondaryto relatively minor injuries. ( J.J. Ballenger 1969).

Laryngeal Trauma due to prolonged endotracheal intubation :

Due to the following factors :

1. Period of intubation : The longer the tube is in position the more likely the tissue damage results. ( Donnelly 1969) Said that after only 3 hours there may be loss of epithelium. The increase in the number of cases is mainly due to traffic injuries and to laryngeal and tracheal trauma caused by prolonged resuscitation and intubation. ( U. Siirala (1974).

2. Trauma at intubation when it is done quickly as during cardiac arrest.

3. Size and shape of tube : Too large a tube more likely to presson the laryngeal wall resulting in pressure necrosis of the epithelium overlying the vocal process of the arytenoid and posterior plate of the cricoid. The ulcerated areas heals normally but infection or continuing truma leads to granulation tissue and finally establishes intubation granulomas which is treated by surgicalremoval done endoscopically or by the use of oral zinc sulfate. ( George L. Adams 1978).

4. Movement of the tube within the larynx causing epithelial damage.
5. Sustained tissue hypoxia ( P.D.M. Ellis and J. Bennett 1977).
6. Composition of endotracheal tube or chemical irritants on the tube ( Donald B. Hawkins 1977).

In 1965 ( McDonald and Stocks) advocated the use of prolonged endotracheal intubation for the relief of various airway problems but resulting in an increase in the incidence of acquired subglottic stenosis ( U. Siirala 1974) ,(Blair Fearon and David Ellis 1971).

So the advice for the avoidance of laryngeal injuries is :

1. Prolonged in tubation is indicated in early infancy.
2. A continuing laryngological control is necessary from the fifth day onwards :
3. The following findings oblige the surgeon to remove the tube :
   - edema or ulcers of the arytenoid or interarytenoid region.
   - Circumscribed blue spots at the laryngeal inlet.
   - the appearance of fibrous tissue in the lumen of the cricoid cartilage ( W. Kup 1974).

The patient must be examined every day. In most cases a Mcintosh laryngoscope is sufficient. If there is reason to suspect deeper damage. It is necessary to introduce a tracheoscope. ( W. Kup 1974) .

Clinical Manifestations of Acute Laryngeal Injury :

In any patient who has sustained a possible laryngeal injury, the following symptoms are indicative of some derangement of laryngeal structure : (1) Increasing airway obstruction with dyspnea and stridor. (2) Dysphonia or aphonia. (3) Cough. (4) Hemoptysis and hematemesis . (5) Neck pain . (6) Dysphagia and odynophagia ( J.J. Ballenger 1969).

The preceding symptoms may be accomplished by distinctive clinical signs including : (1) Deformities of the neck including alterations in contour and swelling . (2) Subcutaneous emphysema (3) Laryngeal tenderness (4) Bony creptitus . The presence of air-way obstruction and subcutaneous emphysema are diagnostic of a severe disruption of laryngeal or tracheal structure.( J.J. Ballanger 1969).

Diagnosis : The presence of any of the preceding clinical manifestations is a basis for assuming a severe injury and is an indication for indiredt and direct laryngoscopy and bronchoscopy to determine the presence of edema, hematoma, mucosal tears, cartilage displacement and vocal cord paralysis. Roentgenograms of the neck and chest must be obtained to detect laryngeal fractures, tracheal injuries and pneumothorax, ( J.J. Ballenger 1969).

Edema and Hematoma post laryngeal Injury : Edema and hematoma formation in the loose supraglottic and subglottic tissues is common to almost all laryngeal injuries. They may constitute the sole pathologic findings in some injuries, but care must be taken to rule out the presence of any underlying cartilaginous injury ( J.J. Ballenger 1969)

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II. BURNS OF THE LARYNX :

A. THERMAL BURNS OF THE LARYNX:

Etiology : Burns of the laryngeal mucosa occur after the inhalation of hot gasses or smoke and are a common injury of firemen. They may also be secondary to the ingestion of excessively hot food or liquid.( Douglas Ranger 1971).

Pathology : As with external burns, there may be first, second and third degree burns. Third degree burns are uncommon since the period of contact is usually short. First and second degree burns cause supraglottic edema with respiratory obstruction ( J.J. Ballenger 1969).

Clinical Manifestations: Hoarseness, pain, stridor and respiratory obstruction may be present. Indirect laryngoscopy reveals the false cords to be red, swollen and edematous. There may be patches of grayish exudate covering the areas of second degree burns. The true cords, if involved are usually burned over the anterior two-thirds with redness and swelling occurring in this area ( J.J. Ballenger 1969).

B. Chemical Burns of the Larynx :

Etiology : There are many corrosive chemicals which may cause burns by ingestion or inhalation but sodium hydroxide is the most common offender. Potassium hydroxide, Lysol and Clorox are other commonly ingested corrosive agents; however, burns secondary to the latter two are usually not severe. Burns of the larynx are most often secondary to lye ingestion and are associated with hypopharyngeal and esophageal burns ( J.J. Ballenger 1969).

Pathology : Burns may be first, second and third degree. The burned areas of the larynx include the epiglottis, the aryepiglottic folds, the arytenoids and the postcricoid area. Third degree burns are not uncommon and are associated with full thickness loss of mucosa. Ulceration and infection develop secondary to the burns and are followed by granulation and fibrosis. Cicatricial stenosis of the supraglottic portion of the larynx may be the end result due to adhesions between the false vocal cords or adhrence of the epiglottis to the posterior pharyngeal wall, Postcricoid burns may be associated with perichondritis and destruction of the cricoid cartilage. ( J.J. Ballenger 1969).

Clinical Manifestations : There is usually a history of chemical ingestion and burns will be seen in the mouth

and oropharynx. It is uncommon to have a burn of the larynx or hypopharynx without burns in the mouth. With severe burns, respiratory obstruction may develop secondary to edema and will appear within an hour after the burn. ( Douglas Ranger 1971).

Indirect laryngoscopy may disclose only redness and edema of the supraglottic structures. However, with severe burns there may be actual charring with gray-black membranous exudate covering the involved areas. The epiglottis may be denuded of epithelium and appear quite white in contrast to the surrounding black areas. Cervical esophagoscopy is necessary to detect postcricoid burns. ( J.J. Ballenger 1969).

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## V. PEDIATRIC VOCAL CORD PARALYSIS

Unilateral or bilateral vocal cord paralysis by itself in the pediatric age group is unusual. A neonate, infant, or child usually presents with unilateral or bilateral laryngeal paralysis as only one manifestation of a multiple system anomaly. Frequently, the vocal cord paralysis is overlooked due to the life threatening situation of the other congenital disorders, Early detection of pediatric laryngeal paralysis requires a high index of suspicion and is important to prevent catastrophes during periods of acute respiratory embarrassment. (D.D. Dedo 1979).

Etiology : A. Congenital :

1. Hydrocephalous, meningomyelocele ( cervical, lumbar, sacral), and meningocele have been associated with the Arnold-Chiari malformation, caudal displacement of the brain stem by increasing intracranial pressure stretches the vagi and causes recurrent nerve and vocal cord paralysis ( Graham 1962) .
2. Other development anomalies of the central nervous system ( C NS ) such as encephalocele cerebral agenesis

and nucleus ambiguus dysgenesis have been complicated by vocal cord paralysis . ( Holinger 1961 , Priest 1966, Hart 1970 ) .

3. Congenital anomalies of the cardiovascular system, left recurrent nerve is more affected than the right because of its longer course. ( Holinger 1976).

4. Embryological development of the laryngopharynx, esophagus, and tracheobronchial tree ( Cohen 1975).

B. Acquired :

1. Newborns with V.C. paralysis and no apparent cause is due to stretching of the laryngeal nerve during delivery this paralysis may be temporary or permanent ( Douglas D. Dedo 1979).

2. During surgery in ,

- Thyroid : incidence of paralysis 0.3% - 9.4% ( Doyle 1967 ) .
- Cardiovascular surgery ( Marlin Weaver 1976).
- Anterior surgical approach to cervical vertebrae ( Heeneman, 1973 ) .

3. Laryngeal Trauma and Tracheal intubation.
4. Inflammatory disease ( Fex, 1973 , Wirth 1972 ).
5. Neoplasms and its therapy complications as antimetabolities ( Brook 1971) .
6. Antibiotics and immunizations ( Vaccinations) reduced the infectious types of Vocal Cord paralysis as whooping cough, encephalitis , polio, Diphtheria, rabies and tetanus ( Cavanaugh 1955).
7. Idiopathic, following a viral infection is thought to be due to a peripheral neurities simillar to Bell's Palsy ( Graham 1962 & Marlin Weaver 1976).

CLINICAL PICTURE :

Voval cord paralysis may affect any of the normal laryngeal functions of voice production, respiration, or deglutition. For unilateral vocal cord paralysis, the cry tends to be breathy and weak while it is often normal in bilateral vocal cord paralysis . This normal cry misleads the physician into believing that the vocal cords move normally. When these children are stressed, they develop significant airway obstruction as they inspire

deeply and draw the vocal cords together from the paramedian or midline position ( Douglas D. Dedo, 1979).

Stridor is the harsh respiratory sound produced when air passes a partially vibrating obstruction - in this case the vocal cords. Cavanaugh (1955), reported that stridor was the most frequent symptom of children with vocal cord paralysis for which parents sought advice. (Douglas D. Dedo, 1979).

Abnormal deglutition is frequently seen in those children with vocal cord paralysis. Drooling , recurrent choking and aspiration suggest either a development anomaly or a neuromuscular disorder of the laryngopharynx. ( Douglas D. Dedo, 1979 ) .

Diagnosis : A neonate, infant, or child with an airway problem, feeding difficulty, weak, or absent cry, deglutition abnormality or known anomaly of the esophagus, heart or central nervous system must be suspected of having a vocal cord problem. The method of choice to evaluate cord mobility is direct laryngoscopy without anasthesia. Oxygen and pediatric endotracheal tubes. Laryngeal movement must be evaluated during inspiration and phonation or crying.

To examine the configuration of the vocal cords, anterior commissure, and subglottic surface. In children with severe dyspnea. If there is no apparent pathology to explain the stridor or partial upper airway obstruction, direct laryngoscopy under general anaesthsia should be done( D.D. Dedo 1979).

In unilateral paralysis of Recurrent laryngeal Nerve the cords are in a paramedian position. While in Bilateral paralysis it is intermediate position . ( Dedo 1970) . Radiological studies for chest and neck and X-rays and dye studies of larynx, trachea oesophagus and cardiovascular system are important for presence or abscence of other congenital anomalies. ( Kahn 1977).

VI. MISCELLANEOU CAUSES :

I. THYROID GLAND AS A CAUSE OF STRIDOR :

1. Cretinism : Congenital deficiency in thyroid secretion dating from birth. Errors of embryologic development may be responsible for either complete abscence of thyroid gland( Thyroid aplasia) or anomalous differentiation of the gland( Thyroid dysplasia) . Athyrotic children have no detectable uptake of $^{131}I$ in the neck

and develop severe clinical manifestations of hypothyroidism in early infancy. ( Judson J. Van Wyk 1968).

Diagnosis :

Diagnosis is considered in newborns who maintain a :

1. Noisy Breathing due to the obstructive effects of the large myxoedematous tongue.
2. Feeding difficulties due to the large myxoedematous tongue.
3. Subnormal temperature 4. exhibit excessive circulatory mottling .
5. Inactive cry infrequently.
6. Constipation. 7. Persistent unexplained Jaundice.
8. Umblical hernia classical face due to myxoedema in subcutaneous tissues and tongue. The cry is hoarse due to myxoedema of the vocal cords ( Judson J. Van Wyk 1968 ).

Diagnosis of Hypothyroidism in Childhood :

By comparison the weight age which exceeds the hight age. The retardation of bone age equall or exceeds the retardation in linear growth. Hypothyroidism is a life long disease.

diagnosis should be supported by tests of thyroid function before giving the treatment, which substitution therapy ( Judson J. Van Wyk. 1968 ) .

2. Goiter in Childhood :

A. Congenital Goilter : infants with cretinism due to an inborn error of metabolism e.g., with severe peroxidase deficiencies having large neonatal goiters than infants with other hereditary defects to threaten respiration .
In endemic Goiter congenital Goiter leads neonatal death by asphyxiation. Neonatal G. is due to ingistion by mothers of large doses of Iodids during pregnancy . Many drugs given to patients with asthma contain substance that influence thyroid function ( Harvey D. Klevit 1969) .

B. Intratracheal Goiter : One of the many ectopic locations of thyroid tissue is within the frachea. The intra luminal thyroid is beneath the trechial mucosa and is frequently continuous with the normally situated extratracheal thyroid. The thyroid tissue is susceptible to goitrous enlargement, which involves the normally situated as well as the ectopic thyroid.

when there is obstruction of the air way associated with a goiter , it must be ascertained whether the obstruction is extratracheal or endotracheal. ( Angelo M. DiGeorg 1979).

3. THYROTOXICOSIS : ( Graves diseases )

Due to diffuse thyroid hyperplasia more than hyperfunction , built on genetic basis and Psychosomatic illness and Iodine metabolism abnormality. It occurs in infants born to mother with Graves disease. Exophthalmous diseppear in 3 months.

LATS activity in plasma of these infants are threated by asphyxiation by tracheal compression as well as by thryotoxic state it self ( Judson J. Van Wyk 1968).

Treatment : Propylthiouracil, Iodides, reserpin. ( Judson J. Van Wyk 1968) .

4. RETROSTERNAL EXTENSION :

The trachea may be constricted by the thyroid gland which may be enlarged congenitally, and which may involve the tracheal lumen either in the neck or in the thorax from a retrosternal extension of the gland. The thymus gland is more frequently blamed for tracheal compression resulting in stridor and dyspnea ( J.F. Birrell 1960).

5. HASHIMOTOS STRUMA : It is increased inchildren, females more than males. It is insidious in onset there is no painful enlargement or fever, There is tachycardia, nervousness, no exophalmous. The gland is irregularly enlarged, firm but it may get atrophy with emergency myxedema. The satellite lymphnodes enlarged (Delphian Node above the Isthmus . It is due to Autoimmune reaction of delayed hypersensitivity type ( Judson J. Van Wyk 1968).

6. THYROID CARCINOMA :

Cervical metastases and Pulmonary metastases are frequent. Although the majority of children with thyroid carcinoma are euthyroid, incidences of associated hyperthyroidism have been reported( George W. Clayton 1969).

Therapy :

Carcinoma of the thyroid is compatible with long life and that the disease, has a very slow progression. Long survival after the diagnosis has been is not uncommon, and recurrenee, after as a long as twenty years, is not unusual, This is particularly true in childhood. Despite its benign

nature in children, a fatal outcome occurs in a number of the patients afflicted( George W. Clayton 1969) .

## 2. DISEASES OF THE MEDIASTINUM

### INFECTIONS OF THE MEDIASTINUM ( ACUTE MEDIASTINITIS :

Acute infection of the mediastinum is most frequently the result of trauma. Infection may be introduced from without as in gunshot or stab wounds, or from within as in perforation of the trachea or esophagus by foreign bodies or instrumentation, or secondary to infections in neighboring structures, but may develop in the course of a general infection with septicemia . Infection may extend into the mediastinum from the neck as in Ludwig's angina or retropharyngeal abscess; it may extend from the esophagus, ( as in perforation of a carcinoma of the esophagus); it may arise from pneumonia or empyema, or osteomyelitis of various parts of the thoracic skeleton may be the source( Carl Muschenheim 1951).

1. Acute nosuppurative mediastinitis : May accompany pericarditis, pleurisy, pneumonia or an infection in the neck. The symptoms are usually mild and consist

in an abrupt increase of fever accompanied by pain beneath the sternum or between the shoulder blades, painful cough, and discomfort on swallowing . Physical examination may reveal slight fullness in the suprasternal notch, tenderness over the sternum and crepitations along the sternal borders, or there may be no positive signs. The symptoms may subside spontaneously in a few days, and there are no sequelae. ( Carl Muschenheim 1951).

2. In Acute Suppurative Mediastinitis : there is rapid spread throughout the mediastinal tissues from the point of origin of the infection, and the symptoms are severe, consisting in high fever chills, severe prostration, painful shallow respiration and pain on swallowing. The pain is referred beneath the sternum , or in posterior mediastinitis, between the shoulder blades. On physical examination there may be , in addition to the signs noted in the nonsuppurative form, evidence of compression of the mediastinal organs. The course in acute suppurative mediastinitis is usually fulminating and the case mortality is high( Carl Muschenheim 1951).

3. Acute Mediastinal Abscess : formation results from localization of an infection either in the anterior or the

posterior mediastinum. The anteriorabscesses are caused most commonly by extension of infections of the neck, posterior ones by perforation of the esophagus. Either may arise from suppuration of lymph nodes in pulmonary infections. The symptoms and signs of mediastinal compression ( superior vena cava syndrome) commonly associated with abscesses vary according to the location , Pressure on the trachea causes stridor, dyspnea and cough. Pressure on the esophagus causes dysphagia and pain on swallowing. On the recurrent laryngeal nerve paralysis of the vocal cord. In the larger abscesses there may be dullness to percussion anteriorly or posteriorly. The roentgenogram may be helpful in revealing the presence and location of an abscess. ( Carl Muschen Heim 1951).

Chronic Mediastinitis :

Chronic diffuse inflammation of the mediastinum is rare. It usually arises either as a sequel of acute suppurative mediastinitis or in association with chronic infections of neighboring structures( Carl Muschenheim 1951).

The symptoms and signs of chronic mediastinitis, abscess and mediastinal lymphadenitis are variable, depending

on the nature of the infection and the part of the mediastinum which is invaded. The cases of tuberculous mediastinal lymphadenitis may be symptomatically entirely latent, until the lymph nodes attain large size, or there may be mild constitutional symptoms. Tuberculosis is the most frequent cause of chronic mediastinitis and the origin is usually a tuberculous mediastrinal and bronchopulmonary lymphadenitis. When the nodes become large, they may compress the bronchi or trachea sufficiently to cause cough and stridor. Signs of secondary pulmonary involvement from the bronchial obstruction may then appear ( Carl Muschenheim 1951).

The roentgenogram reveals the presence size and location of any discrete lesions, or it may show diffuse widening of the mediastinal shadow. In the tuberculus cases, evidence of tuberculosis elsewhere may clarify the diagnosis ( Carl Muschenheim 1951).

Cysts and Tumours of The Mediastinum:

Mediastinal cysts : Simple cysts derived from the thymus, the esophagus or the bronchi occur in the mediastinum. They usually cause no symptoms unless infection supervenes. Occasionally they may cause pressure symptoms and signs

simulating those of solid tumours or aortic aneurysm. Dermoid cysts and cystic teratomas are the most important of the mediastinal cysts. ( Carl Muschenheim 1951).

Benign connective tissue tumours of the mediastinum fibromas and neurofibromas are the most common are discovered most frequently in the course of routine physical or x-ray examinations and do not cause thoracic symptoms until they have attained large size ( Carl Muschenheim 1951).

Intrathoracic ( retrosternal) goiter and tumours of the thymus, thought not usually classed as tumours of the mediastinum, are actually situated in the anterior mediastinum and may produce pressure effects similar to those from other tumours in this location. Malignant thymomas of various types are the most common thymic tumours in adults. Nonmalignant tumours of the thyrmus, and a simple enlarged or persistent thymus, are of particular interest because of their occasional association with myasthenia gravis ( Carl Muschenheim 1951).

Symptoms and Signs : The symptoms and signs of mediastinal cysts and tumours depend mainly on their size and on the pressure which they may exert on vital structures situated in the mediastinum.

The earliest symtpms may indeed be cough and dyspnea Thymic tumours and substernal extensions of thyroid enlargements are particularly prone to cause tracheal compression in which severe dyspnea is associated with stridor.( Carl Muschenheim 1951) .

Diagnosis : By the X-ray and fluoroscopic examinations. Lateral or oblique views are usually necessary, and biopsy; in doubtful cases angiocardiography will reveal whether or not the mass is vascular ( Carl Muschenheim 1951) .

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## MANAGEMENT OF THE NEWBORN CHILD WITH LARYNGEAL OBSTRUCTION

Two main factors have to be considered in planning the management of the patient . These are :

(1) An adequate supply of correctly humidified air

(2) Adequate nourishment.

Many babies with strider do not need assistance with their airway unless they develop some form of respiratory inflammation . However, about one-third of those babies will experience some feeding difficulty in the early months of life. They will be slow feeders and show a marked tendency to cough and splutter as the milk goes the wrong way. It has been found that such babies often progress more rapidly if they are introduced to a semi-solid weaning diet at an earlier age (4-5 weeks ) than is usual. If some artificial assistance with the airway is necessary two possibilities are open to the surgeon; which one he chooses will depend upon how long it will have to be maintained. In the short term the most satisfactory of airway . If it can be introduced, is the in-dwelling endotracheal tube; for all other cases the method of choice is tracheostomy,( R.Pracy 1979 ) .

A formal tracheostomy is carried out through the second, third and fourth tracheal rings and a No. 14 or 16 tracheostomy tube is introduced. The tubes are of

two sorts, plastic and the more traditional silver ( R. Pracy 1979).

Where it is felt desirable that the expiratory thrust should be maintained, a valved tracheostomy tube should be fitted once the situation has stabilized. Whatever form of artificial airway is decided upon it is imperative that the air supply to the baby is properly humidified. Adequate cold humidification greatly reduces the problems such as crusting and the cleaning of the tubes. This can be supplied by one of several forms of ultrasonic atomizer which deliver a fine mist of particles of less than 5 um in diameter. If this is done and aspiration of excessive secretions using a no-touch technique with disposable sterile gloves is followed there are few difficulties in maintaining a small baby's tracheostomy in the short term ( R. Pracy 1979).

In the long term , however, humidification in an incubator cannot be maintained because of the water-logging effect it has upon the baby's skin. Once the cause of the obstruction has been treated and de-cannulation is possible, the cannula may be removed. If it has been present for some months it may be helpful either to pass an endotracheal tube for 12 hours or to carry out such measures. ( R. Pracy 1979).

1. LARYNGOMALACIA :

Treatment : In most cases, active treatment is not necessary and the condition usually subsides spontaneously by the age of two years ( Ballenger 1969).

Pulmonary complications are treated as they arise. Vitamin D has been recommended with a view to strengthening the flabby cartilage.

Partial amputation of the epiglottis only rarely necessary, ( Birrell 1960) .

Surgical procedures, such as partial epiglottectomy or denuding the surface of the base of tongue and epiglottis to cause adhesions and increase the rigidity of the supra glottis, have been described but are rarely necessary ( Ballenger 1969) .

Most patients are from lower socioeconomic groups and attention should be given to assure an adequate diet. ( J.J. Ballenger 1969 and Paul H. Ward 1973 ).

A temporary tracheotomy is rarely necessary because of extreme dyspnea or exacerbation of symptoms due to an upper respiratory tract infection, particularly if the infant is unable to feed properly and does not gain weigh. ( Holinger, 1976).

Tracheostomy : is necessary when feedings were either accompanied by severe cyanosis or had to be interrupted so frequently to allow the infant to breathe that the infant failed to gain weight( Holinger, W.T. Brown 1967).

2. SUBGLOTTIC STENOSIS TREATMENT :

The subglottis enlarges as the child grows, particularly in the first 2 years, and the condition will right itself provided that adequate air is inspired.

When there is a marked subglottic narrowing there is insufficient inspired air to produce an adequate expiratory thrust, which provides the stimulus for the laryngeal growth. Thus in marked narrowing, repeated chest infections and failure to gain weight a tracheostomy may prove to be the short-term, management of choice. Since the expiratory thrust is trans laryngeal only when an expiratory valve is fitted , a valve and fenestrated tube must be used. ( A.G.D. Maran, P.M. Stell 1979).

( R. Pracy 1979 ) , Said that because the narrowing is due to cartilage rather than fibrous tissue nothing is gained from dilatation with bougies, But they damage the delicate mucous membrane in the area

of the cricoid narrowing and may lead to ulceration and further stenosis. If obstruction is severe the right treatment is the establishment of a tracheostomy below the stenosis with an expiratory valve kept by routine direct laryngoscopy and measurement of the lumen at the site of the obstruction.

(Blair Fearon, David Ellis 1971) and ( Lauren D. Holinger 1976), believed that gentle, endoscopic dilatation at regular intervals is considered helpful and improves the airway as well as the development of the larynx and speech.

This should not be attempted unless a tracheostomy is established.

SURGICAL TREATMENT :

The most satisfactory rasults are obtained by the use of Evans and Todd operation which is carried out under the operating microscope. A median thyrotomy is performed, the voval cords are visualized and the excessive narrowing of the ring is thinned by sculpting under direct vision. The cricoid is devided in steps as are the upper rings of the trachea, after removal of the redundant cartilage, the mucosa is spread to cover

as much as possible of the increased diameter. The cricoid and upper rings of tachea are distracted and sewn in a new position in order to increase the lumen up to 2-3 mm. ( R. Pracy 1979).

( Fearon and Cotton 1972) , advocate splitting the cricoid cartilage and interposing a segment of thyroid cartilage between the cut ends of the cricoid ring to enlarge the circumference.

( Blair Fearon, David Ellis 1971) , adviced splitting the cricoid cartilage vertically in the midline and the edges held apart by a piece of rib cartilage . The inner surface was lined with a graft of mucosa taken from inside the mouth, and a relatively good result was obtained.

3. WEB TREATMENT : Extensive webs of the larynx will require emergency treatment at birth because of obstruction. Tracheostomy is indicated in such instances. Onee tracheostomy has been necessary , definitive repair should be carried cut as soon as it is feasible so that normal development of the larynx, laryngeal reflexes and speech is permitted. Surgical correction can usually be carried out by one year of age :

Small laryngeal webs causing dysphonia only may

be left untreated until the larynx is of more substantial size. Repair, in such instances, is done at age five to six years when voice and speech become more important.

METHODS OF SURGICAL TREATMENT : Bougienage : Thin friable webs, particularly at the glottic level, may be treated by bougienage and dilatation of the larynx which will often cure the problem. Laryngeal dilators of appropriate size are passed by direct laryngoscopy. However, if there is any question of traumatizing the larynx by having to use unnecessary force for dilatation, the procedure should be abandoned. Repeated attempts to dilate a thick, resistant web will only worsen the stenosis ( J.J. Ballenger 1969).

McNaught Keel : After surgical division of a laryngeal web, the opposite sides of the larynx must be separated to prevent re-attachment. The use of a tantalum or silastic keel for this purpose , inserted through a thyrotomy will usually produce a good result.

4. TREATMENT OF ABNORMALITIES OF EPIGLOTTIS (BIFIDEPIGLOTTIS):

Amputation of the epiglottis after a preliminary tracheostomy is said to cure the condition. But with modern surgical techniques it is more logic to pare the

edges and try to suture the two halves togather ( R. Prácy 1979).

5. TREATMENT OF CLEFT OF THE LARYNX :

Once a cleft is diagnosed the only possible line of treatment if the child's life is to be saved is surgical closure of the cleft. However, before any attempt is made to carry out the closure, an attempt should be made to obtain the most favourable chest condition. Postural drainage, tube feeding, physiotherapy and aspiration all have their part to play Antibotics should be given pre-operatively. The cleft is closed by paring the edges and sewing up the deficit in layers. The child must be tube-fed for 3-4 days after the operation. ( J.E. Delahunty and Jerrie Cherry and Baltimore 1969).

6. TREATMENT OF CYSTS :

Simple cysts occurring in mucous glands or in cystic laryngocoeles should be removed. Where these are small they may be removed endoscopically using microdissection instruments. The dissection must be very gentle, incisions, must be made as far as possible from the laryngeal lumen and bleeding must be controlled

meticulously. By removing the top of cyst with forceps and evacuating the fluid but the cyst rapidly fills up and so it is necessary to repeat the procedure. The infant's larynx is so small and sensitive that repeated instrumentation should be avoided.

So a modified diathermy needle has been developed to destroy the redundant mucosa of the cyst after aspiration of its contents( A.G.D. Maran&P.M.Stell 1979). Larger cysts are better treated by the lateral pharyngotomy approach( Baker 1956).

7. TREATMENT OF HAEMANGIOMA : I

If symptoms are intermittent and there is no severe respiratory embarrass ment, the child may be watched. Often the hemangioma will regress spontaneously, as many cutaneous lesions do, within the first year or two of life. Spontaneous regression will usually begin within eight to ten months, but if involution is not evident within a reasonable length of time, then definitive treatment is indicated ( Ballenger 1969).

1. Irradiation : These lesions usually respond fairly well to small doses of irradiation from 500 to 750 r. There is , however, the question of irradiation in children being a factor in the etiology of carcinomata of the head and neck as well as the possibility of damage to growth centers. ( Ballenger 1969 & R. Pracy 1979 .)

2. Surgery : These lesions may be approached through vertical tracheal incision. Lesions of the anterior wall may be dissected out submucosally without entering the tracheal lumen. If the hemangioma is on the posterior wall, a vertical mucosal incision is made and dissection is done taking care to preserve the mucosa. The cricoid cartilage should not be injured. A stent of polyethylene tubing or Ivalon sponge must be left in the infraglottic area for three to four weeks, postoperatively, to prevent stenosis. Careful approximation of the tracheal rings is imperative to prevent formation of granulation tissue in the tracheal lumen. ( J.J. Ballenger 1969).

3. Sclerosing Agents : Sclerosing agents such as sodium morrhuate, have been used in the treatment of hemangiomas in other areas of the body. The difficulty of controlling

the injection of this material in a tiny larynx, the resulting fibrosis, and the possibility of inducing laryngeal stenosis precludes their use( R. Pracy 1979).

4. Destruction By Other Agents : Laser, diathermy and cryoprobles applied locally. Thus offer the best hope of treatment of the supra glottic lesions. The subglottic lesions ,are treated through a thyrotomy at the age of 3 years ( J.J. Ballenger 1969 and R.Pracy 1979).

8. TREATMENT OF CONGENITAL VOCAL CORD PARALYSIS :

In most cases of unilateral paralysis, spontaneous regression occurs within six months. However, a search should be made for any etiologic factor. Persistent unilateral paralysis usually does not require definitive therapy since it is well compensated for by infants.

Bilateral cord paralysis frequently requires tracheostomy which must be left in place until regression of cord paralysis or surgical widening of the glottis occurs. If movement fails to return, surgical widening of the airway may be required. Arytenoidectomy or arytenoid - opexy are best delayed untill age 5 or 6 ( J.J. Ballenger 1969 ).

9. TREATMENT OF ATRESIA OF THE LARYNX :

When it occurs it is diagnosed at birth. The breathing is immediately embarrassed and direct laryngoscopy in order to carry out intubation will reveal the diagnosis. Half of the larynx will be present and intubation may be extremely difficult. Usually only a very small tube may be passed( 2mm.) and this is not satisfactory for respiratory exchange. Since no difinitive solution will be proposed for some years it is necessary to make a semi-permanent tracheostomy. A valved tracheostomy tube should be used to promote normal laryngotracheal physiology. In time the child will learn to speak. However, the voice will in general be high pitched and poor in volume. Any operation which is proposed for the relief and reconstruction of the glottis should be delayed until the child's neck is long enough and the larynx large enough to make a worth while solution feasible. ( A.G.B. Maran and P.M. Stell 1979).

10. TREATMENT OF TRACHEO-OESOPHAGEAL FISTULA :

As soon as the presence of an abnormal laryngotracheal connection is suspected, all feeding by mouth must be stopped. Dehydration is treated by intravenous fluids. Antibiotics are given for pneumonia, which must be controlled

before surgical repair of the defect is attempted.

When atresia is present, the upper esophageal pouch should be kept empty by a soft catheter inserted into the pouch transnasally and attached to gentle suction. Elevation of the head will help prevent regurgitation into the trachea and all these infants should be nursed in this position. Gastrostomy is of value for feeding and prevention of regurgitation and is necessary for most patients( Ballenger 1969).

Definitive surgery should not be attempted until dehydration, nutrition and pneumonia have been controlled.

Surgical repair of most tracheo-esophageal fistulas with atresia must be done through a thoracotomy . However, high H-type fistulas may be repaired by an approach through the neck followed by isolation and division of the fistula tract and layered closure. A small local muscle flap should be interposed between the trachea and esophagus at the closure site ( Ballenger 1969).

11. TREATMENT OF MICROGNATHIA AND GLOSSOPTOSIS ( Pierre- Robin Syndrome ) :

The treatment will vary with the degree of respiratory obstruction and keeping an infant in prone position

will overcome minor degrees of obstruction. However, if an infant is having difficulty with feeding, the obstructive symptoms will usually get worse before improvement can be expected and surgical treatment should be instituted early. In the meantime, feeding should be done by gavage. In severely debilitated infants a feeding gastrostomy may be advantageous.

Tracheostomy will relieve the respiratory obstruction but it does not alter the problems of deglutition and aspiration, thus imposing a difficult nursing problem. Also, the complications , especially with decannulation, are greatly increased in these small , feeble infants. Therefore, tracheostomy is best avoided( Ballenger 1969).

The tongue must be fixed , in an anterior position and when this is done, the obstruction and feeding difficulties are usually relieved. Some form of temporary glossopexy is imperative when a history of cyanotic attacks and aspiration is elicited because the symptoms of glossoptosis will increase in severity before spontaneous improvement occurs. In the majority of cases, the growth of the micrognathic mandible " catches up " with the remainder of the facial structures by the end of the first year. Therefore,glossopexy is a temporary requirement.

The mortality of infants with this syndrome treated conservatively, has been variously reported between 30 and 65 percent. Douglas has reported, on the other hand a series of 30 patients treated surgically with only one death. Several procedures have been described for glossopexy ( Ballenger 1969).

Douglas Operation : This entails denuding an area on the undersurface of the tongue and posterior surface of the lower lip and approximating these surfaces in order to obtain an adhesion to hold the tongue forward.

Kirschner Wire : The insertion of a Kirschner wire through the angles of the mandible and the base of the tongue with the tongue pulled forward has been advocated.

Duhamel Suture : We have found this method of treatment consistent in giving adequate relief of dyspnea and avoiding tracheostomy. The advantages are that it is simple , can be done at bedside and gives immediate results.

12. TREATMENT OF DYSPHAGIA LASORIA :

Once the diagnosis is made the patient should be refered to the cardio thoracic surgeon for correction of the vascular anomaly ( D.Downton 1978).

13. TREATMENT OF TRACHEAL COMPRESSION BY VASCULAR ANOMALIES :

The child with vascular compression of the trachea may lie with the head extended since this tends to put the trachea on the stretch, lifting the offending vessel forward.

An unsuspected tracheal compression may make surgery for other diseases hazardous. If endotracheal anesthesia is used, the endotracheal tube may be partially or completely obstructed by the compressed area impinging on the open end of the tube. Furthermore, postoperatively the patient may have additional respiratory difficulty because of accumulated secretions in the tracheobronchial tree.

14. TREATMENT OF LARYNGEAL TETANY :

If the patient is seen during an attack, the clothes round the neck should be loosened, and the window thrown open to permit of a free draught of air; cold water should be applied to the face and neck, and smelling salts

held to the nostrils. If there is real danger of asphyxia, tracheostomy must be performed at once. The general treatment depends on the cause which is mainly rickets, diet must be carefully regulated, and that cod-liver oil and tonic treatment should be administered. If the attacks recur frequently, bromide, of potassium may be given ( J.P. Stewart and R.B. Lumsden 1961) .

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A. TREATMENT OF ACUTE INFLAMMATIONS :

1. Acute Non-Spesific Laryngitis

Treatment should include bed rest and medication for symptomatic relief of fever, cough and sore throat. Voice rest must be complete ( H.C. Ballenger and J.J. Ballenger 1954 ). Humidification in a cool room and the use of expectorants help to relieve dryness and tickle and to liquefy the secretions (R.Pracy 1972). If laryngitis is severe and stridor is present, a cold steam tent is indicated. A special effort should be made to maintain systemic hydration to avoid further inspissation of secretions. ( Ballenger 1969) . Broad-spectrum antibiotics may be helpful in preventing secondary bacterial infection of the ulcerated mucosa in the more severe cases ,( R.E. Ryan 1970) . The majority of patients will recover with simple conservative treatment; however, the disease tends to be more severe in elderly patients and may be complicated by pneumonia. Therefore patients in the older age group should be observed closely for progression of the infection ( Ballanger 1969).

2. Treatment of Acute Spasmodic Croup

Therapy is directed at abortion of the attack. Humidification is important and it is frequently noted that there is a rapid disappearance of stridor when the child is placed in the bathroom with the hot shower going ( R. Pracy 1972 ) . An emetic dose of ipecac ( ½ to 1 tsp. ) will induce vomiting and often terminate the attack ( Maxwell Ellis 1977) Oxygen therapy and tracheostomy are seldom required. Mild sedation at bedtime will often prevent recurrent attacks. Antibiotic therapy is rarely indicated unless evidence of a bacterial infection is found.( Pracy 1972 ). The parents must be reassured in order to prevent the situation from becoming a means of household control by the child when the attacks are recurrent ( J.J. Ballenger 1969) .

3. Acute Epiglottitis Treatment :

These patients, whether adults or children, should be hospitalized immediately and placed in an intensive care unit with bronchoscope, laryngoscope , endotracheal

tubes and tradheostomy set at the bedside( Bruce Benjamine 1976). Many physicians advocate, with complete justification, tracheostomy as soon as the diagnosis of supraglottic laryngitis is made( Brain O'Reilly 1976). Tracheostomy should be performed in any case where there is any degree of obstruction and whenever experienced personnel are not available to observe the patient continuously. Increasing dysphagia and decreased air entry to the lungs are also important signs indicating a need for tracheostomy .However, tracheostomy is not entirely innocuous and in many early cases, with vigorous conservative therapy and constant observation tracheostomy may be avoided( J.J. Ballenger 1969), For inflammatory conditions ( acute laryngotracheobronchitis as well as acute epiglottitis) nasotracheal intubation is the procedure of choice; Schuller and Birk (1975) , reported that the mortality rate of tracheotomy exceeds the complication rate of nasotracheal intubation in children. ( Lauren D. Holinger 1976).

If a patient is treated conservatively, equipment necessary to handle any airway problem must be kept at the bedside, should the patient experience respiratory embarrassment, an endotracheal airway should be inserted immediately. A tracheostomy may then be done under more orderly

circumstances with greater safety ( J.J. Ballenger 1969).

Cultures of the thorat and blood ( including cultures for diphtheria) should be taken as soon as possible . Should the organism prove to be ampicillin resistant. Chloramphenicol or a sulfonomide should be substituted for ampicillin . ( Lauren D. Holinger 1976).

Adequate hydration is maintained with parenteral fluids. Ampicillin 200 mg/kg/day, is given intravenously in four divided doses and should be started immediately upon establishing the diagnosis ( L.D. Holinger 1976).. The patient is placed in a croupette or mist tent to insure proper humidification. The patient should be pladed in an environment of 70° to 75°F with a humidity of 90 to 95% ( J.J. Ballenger 1969)

Oxygen Therapy :

The oxygen is used to nebulize water so that the atmosphere is saturated. Mucolytic agents such as Mucomyst and Tergemist may also be nebulized to help liquefy secretions. The use of carbon dioxide to stimulate respiration has no place in this disease because most patients are already hypercapnic ( J.J. Ballenger 1969).

Resolution begins within 24-48 hours and is usually complete within 4 days, when early decannulation can often be accomplished ( L.D. Holinger 1976).

Steroids may significantly reduce the duration of intubation and hospitalization. Dexamethasone is given intravenously : 1 mg/kg. for the first 5 kg. then 1 mg/ 5 kg additional, the dose is repeated in 4 hours, then half the dose every 6 hours for 48 hours until extubation. One-fourth of the original dose is then given at 6-hour intervals for the next 24 hours ( L.D. Holinger 1976 ).

Acute supraglottitis continues to have a significant death rate, not only in children but in adults as well. In spite of numerous publications advocating endotracheal intubation several deaths have occurred were intubation could not be accomplished, even by experts ( Blair Fearon 1977).

When endotracheal intubation is attempted to relieve airway obstruction in supraglottitis, tracheotomy should be planned, in the event that intubation should not succeed. (Michael Cinnamond 1977).

From 1970 to 1975 inclusive 5,000 patients were admitted with acute obstructive laryngitis. Of these 100

were of the supraglottic form of the disease. Sixty-nine of these were tracheotomized, whereas three were managed by intubation. There were no deaths in the patients tracheotomized; nor in fact, any serious complications. The choice of management of the severe cases of acute supraglottitis is to relieve the airway obstruction by bronchoscopy, without anesthesia, and with the patient anesthetized via the bronchoscope, carry out an orderly tracheoctomy( Blair Fearon, and Michael Cinnamond, 1977) .

4. Treatment of Acute Laryngotracheo-Bronchitis :

All treatment should be directed toward improving the failing respiration. In most cases this can be accomplished by medical means. Rest is helpful to conserve strength and to keep oxygen requirements at a minimum. Small doses of barbiturates may be used to allay anxiety. The administration of strong sedatives or narcotics is contraindicated, since these lower the struggle for air and prevent expectoration of accumulated secretions, by inhibiting the cough reflex. Atropine must not be used, since it increases the stickiness of the exudate. Corticosteroids often prove very beneficial in the presence of troublesome edema.

Fluids should be given in sufficient amounts to relieve dehydration and to liquefy secretions. Oxygen preferably administered through a nebulizer or through a special croup tent, relieves distress. Antibiotics should be prescribed. It mustbe emphasized that they are of no value in relieving acute respiratory distress( J.H. Ogura 1970).

In the early stages the most important active form of treatment is an effective system of humidification. A relative humidity of 90% or more at room temperature of 68° to 70°F. is ideal; it reduces loss of body fluids, liquefies tracheobronchial secretions, and soothes the inflamed mucous membrane. The cool air assists in the reduction of the high fever, thus lowering the metabolic rate and the oxygen requirements of the patient. In contradistinction, the ordinary croup kettle or other forms of steam production heat the surrounding air and thereby often actually decrease the relative humidity in the room ( R.E. Ryan 1970).

If medical treatment fails, or is inadequate to relieve the existing obstruction, prompt surgical intervention is indicated. Tracheotomy is the treatment of choice

since it provides immediate restoration of the airway and normal intrathoracic pressure ( R.E. Ryan et al.1970).

Acute laryngotracheobronchitis is a serious disease of small children requiring close observation and vigorous therapy. Patients are often lost because of sudden rapid respiratory failure when judicious tracheostomy may have been lifesaving. The timing of tracheostomy is important so as to avoid the complications of an unnecessary tracheostomy as well as the complications of delayed tracheostomy. When available, serial arterial $O_2$ saturation levels, $O_2$ tensions and arterial blood pH are helpful in determining a need for tracheostomy. Arterial $O_2$ saturation of less than 70 to 75 percent and any decrease in serum pH are indications for the need of tracheostomy. Also a decreasing $O_2$ saturation and acidosis in the face of increased respiratory effort with acute obstructive disease indicates a need for tracheostomy ( J.J. Ballenger 1969).

5. Membranous Laryngitis Treatment :

Initial treatment is directed towards airway maintenance. Tracheostomy is often necessary. Loose pieces of membrane should be removed if possible. This is easily accomplished by direct laryngoscopy without an anesthesia

in infants and small children. The membrane should be submitted for culture and antibiotic sensitivities(Maxwell Ellis 1979) .

Large doses of antibiotics are indicated and in the the acute phase should be given intravenously. Chloramphenicol is the drug of choice since it is active against most gram-negative organisms. In certain instances, ampicillin and colistimethate ( Colymycin ) may be added to the regimen ( J.J. Ballenger 1969).

The patient should be placed in a wet cool atmosphere. Intravenous fluid maintance is usually necessary for small patients. It may be necessary to give large amounts of fluids to overcome the initial dehydration. Despite chemotherapy and tracheostomy, this disease is frequently lethal in infants because of pneumonia, septicemia, shock and respiratory failure .( J.J. Ballenger 1969).

6. Specific Acute Infections of the Larynx :

a. Diphtheria :

Prophylaxis is the most important and successful method of therapy. Every child should be actively immunized as an infant and then receive periodic boosters until age ten ( L.D. Helinger 1976).

Anttitoxin should be given empirically if there is a reasonable suspicion of disease, rather than await a positive culture report, since it is the only specific treatment. It should be given in one dose of 20,000 to 100,000 units, one-half intramuscularly and one-half intravenously ( J.J. Ballenger 1969).

Penicillin will kill the causative organism but it will not alter the course of the disease. It is used to eliminate the carrier state and 125 mg. q.i.d. is given for ten days.

If the symptoms of respiratory obstruction are marked and there is evidence of tachycardia, tracheostomy is indicated. Bronchoscopy should be done first so that any membrane in the trachea or bronchi may be remeaved as to provide an airway during the performance of tracheostomy. The indirect insertion of O'Dwyer tubes is seldom indicated and is of historical interest now ( J. F. Birrell 1960) .

B. TREATMENT OF HERPES OF THE LARYNX :

The disease is usually selfilmited within one to two weeks and treatment is supportive. In severe cases, antibiotics may be necessary to control secondary

infection of the mucosal ulcers. Laryngeal involvement is seldom severe enough to warrant tracheostomy. Some patients may be subject to recurrent infections and in such instances, repeated vaccinia vaccinations may possibly be of value. Lactobacillus organisms given orally may also have a beneficial effect. ( John. Jacob Ballenger 1969).

7. TREATMENT OF LARYNGEAL EDEMA :

Acute laryngeal obstruction is a serious condition that calls for intervention at the earliest possible moment. All patients with acute laryngeal obstruction should be hospitalized immediately, with a physician in constant attendance until the danger has passed( J.J. Ballenger 1969) .

Specific medical treatment directed at the cause may retard or reverse the process. Thus, an early angioneurotic edema may respond to the administration of epinephrine or other vasoconstrictors, and corticosteroids or antihistaminics. However , once the condition has progressed to the point of stridor, no time should be wasted with dilatory measures that may induce a false sense of security. A change in stridor, the presence of restlessness in children, and the

development of pronounced retractions call for immediate restoration of an adequate airway by tracheotomy. Peroral intubation may be performed as a preliminary , temporary measure, while the patient is readied for the surgical procedure ..

Supportive measures include adequate humidification of the surrounding air , the administration of oxygen, the restoration of body fluids, etc. ( Robert E. Ryan 1970).

8. Treatment of Chronic Inflammations of The Larynx.

1. Chronic non spesific Laryngitis of Childhood.

Absolute silence is desirable, but this is almost impossible in a child. However, he can perhaps be prevented from singing, shouting or speaking in anything more than a quiet voice. All foci of sepsis must, of course, be eliminated. The results are frequently disappointing and the child may carry his raucous voice with him throughout life, especially if muscular wasting has occurred. On the other hand the appearance ( à grains d'orge) is rarely seen in adults, which suggests that in a number of cases resolution does occur . It may not be possible to persuade the child of the importance of treatment until he grows older

and more self-conscious, when willing co-operation often quite suddenly occurs, and a condition of some years' standing may clear up in a comparatively short time ( Maxwell Ellis 1971).

2. Chronic Spesific Laryngitis Treatment :

i. Syphilitic Laryngitis :

a. Systemic Penicillin.

b. Potassium iodide.

c. Tracheostomy: needed for laryngeal obstruction.

d. Reconstructive plastic operations can be carried out safely in inactive late cases of severe stenosis ( John. C.Ballantyne et al., 1978).

ii. Tuberculous Laryngitis :

1. Antituberculous chemotherapy ( Streptomycine , I. N. H. and other drugs . ).

2. Vocal rest.

3. Galvano cautery.

4. Heliotherapy.

5. Injection of the superior laryngeal nerve.

6. Tracheostomy rarely necessary( John C.Ballantyne et al. 1978).

TREATMENT OF TUMOURS OF THE LARYNX :

A. Benign Tumours :

1. Laryngeal Papillomata .

Crowe and Breitstein describe the treatment of multiple laryngeal papillomata as universally unsatisfactory. This statement is confirmed by the wide variety of therapeutic agents, which have been advocated. These include :

1. Tracheostomy This may be required as a temporary measure in order to maintain an airway. This operation is most frequently required for the multiple juvenile lesions. Tracheostomy as an elective procedure has been advocated by ( Mackenzie).He recommended that the operation be performed on children when the diagnosis was first made. It is claimed that the operation provides rest for the larynx and after puberty the possible regression of the lesions makes any further treatment unnecessary. This is a most uncertain form of treatment, because regression of the lesions with puberty is not quite sure ( D.J. Brain 1956).

2. Removal : (a) Endolaryngeal : This was introduced by ( Rosenberg) it is the method of choice in most cases. ( Jackson and Jackson) described this operation as scalping the papillomata off flush with the surface, by direct laryngoscopy. Recurrence is to be expected when performed during the juvenile phase, and removal must, therefore, be repeated as required . This may entail numerous operations. Recurrence followed this operation in most juvenile patients. The risk of damage to normal tissues is minimal.

In the past some laryngologists have cauterized the base of the papilloma after removal but did not prevent a recurrence . It also incurs the additional risk of damage to adjacent tissues.( D.J. Brain 1956).

b. Thyrotomy : Introduce by ( Brauer) this operation rapidly fell into disrepute in the treatment of multiple juvenile papillomata because of the high recurrence rate( Bruno 42 percent- Rosenberg 38 percent) and bedeasue of the frequency of post-operative functional laryngeal defects( Bruno 63 percent-Rosenberg 15 percent). ( D.J. Brain 1956).

3. Fulguration diathermy :

Advocated by ( Hubbard, and Galbraith, and New and Erich). It was introduced because the method had proved so successful in the treatment of vesical papillomata. ( New and Erich ) touched each papilloma lightly with the positive electrode, using a small amount of current. They claim the rapid destruction of the tumour without damaging the underlying tissue, and state that implantation of cells is less likely to occur.

Katz states that damage to the normal laryngeal tissues not infrequently occurs with resulting fibrosis. ( D.J. Brain 1956).

4. Hormones : Various hormones have been used in the treatment of multiple juvenile papilloma :

a. Local applications : of ( Estrogens introduced by ( Broyles) in 1940 . He advocates weekly applications of oestrogenic hormone ( Amniotin in oil 10,000 I.V. per. c.c.) which is sprayed endoscopically. The rationale of this treatment is :

(1) The observation that these lesions commonly regress at puberty.

(2) The success of the therapy in the treatment of vaginitis occurring in young girls.

(3) The differentiation from cubical to stratified squamous epithelium which occurs in the larynx during child hood, and its probable initiation by sex hormones. ( Swyer) has suggested the use of methallenestril because this oestrogenic hormone has been found to have twice the potency of oestrone on the vaginal epithelial cells of sprayed mature rats, and because of its minimal effects on the endometrium ( D.J. Brain 1956).

(b) Systemic sex hormones have been used to induce puberty. ( Holt) has used progesterone in boys without success. ( Strang) treated a 6 year old girl with stilboestrol and found this had no effect on the papillomata, but merely produced undesirable side effects.

(c) A.C.T.H. has been used by ( McCart) without success.

(5) Radiotherapy : Reported in 1911 by ( L. Polyak ) at an international congress held in Berlin. Later experience has shown that :

(a) It is not certain of success.

(b) There is no insurance against recurrence.

(c) After irradiating the juvenile larynx it has been noted that further development may be arrested(Salinger and Clerf). This precludes use of radiotherapy in the treatment of multiple juvenile laryngeal papillomata.

(d) Further complications, such as perichondritis, laryngeal stenosis and tracheo-oesophageal fistula have been reported.

(e) ( Simpson, Hemplemann, and Fuller) have shown that the incidence of carcinoma of the thyroid is much higher in patients whose neck was irradiated during childhood.

(6) Antibiotics : The use of chlortetracycline in the treatment of multiple juvenile papillomata was suggested by ( Evans ) in view of their probably viral aetiology and because the spectrum of this antibiotic has been shown to include some viruses. This was preceded by simple endoscopic removal.

( Blackburn) has treated four cases with oxytetracycline and simple endoscopic removal . Recurrence occurred in three cases,

(7) . Other methods : In the past many other forms of treatment have been advocated, such as calcinated magnesia, various acids, bismuth, arsenicals, potassium iodide and oil of Thuja. ( D.J. Brain 1956).

Recently the carbon dioxide laser coupled with microlaryngoscopy provides an exceptionally safe and efficacious technique for the treatment of benign laryngeal tumours and lesions in pediatric patients of all ages including neonates.

The specific advantages of $CO_2$ laser surgery in children include :

1. Precise control 2. Protection of surrounding structures with preservation of anatomy and function .
3. Airway preservation without tracheotomy, 4. hemostasis,
5. Rapid excellent healing without stricture formation or functional disturbance . 6. Possibility of repeated surgery without trauma , cosmotic defect or danger of wound infection. 7. Lessened anesthesia risk; 8. possibility of cost effectiveness superior to other methods, and 9. avoidance of potential hazards of radiation and sterioids ( George T. Simpson Gerald B. Healy, Trevor McGill and, M. Stuart Strong, 1979) .

8. Immunological, the most frequently reported being autogenous vaccine which has shown the best results to date, and transfer factor( Quick, 1975 ). Which has been used in an attempt to influence the thymus dependent small lymphocytes to become immunologically effective in attempting to control the lesion. ( Charlene B. stephens, Godfrey E. Arnold, et al., 1979).

2. HAEMANGIOMAS :

Resolution by 1 year of age.

Tracheostomy may be necessary ( J.H. Ogura and H.F. Biller 1973).

3. ADENOMA : Removal perorally or by Thyrotomy ( J.H. Ogura 1973 ) .

4. FIBROMA : Removal by direct laryngoscopy with sharp cup-ended forceps.

5. NEUROFIBROMA : Small lesions are removed perorally. larger lesions require removal by lateral pharyngotomy. ( J.H. Ogura and H.F. Biller 1973 ).

6. CHONDROMA : The treatment is surgical. The site of origin determines the approach-either thyrotomy or lateral pharyngotomy. Chondromas of the anterior

aspect of the cricoid are approached by thyrotomy. Chondromas of the thyroid , posterior aspect of cricoid, or arytenoid are approached by a lateral external approach with or without pharyngotomy. The entire lesion must be removed or it will recur. Peroral removal, therefore, is not indicated in the usual case. Total laryngectomy may be required for the treatment of recurrences. The histologic differentiation between chondroma and chondrosarcoma may be difficult. Conservative procedures are the treatment in either instance because of the very slow growth of even recurrent tumour. ( Joseph H. Ogura, and Hugh F. Biller 1973 ).

B. MALIGNANT TUMOURS :

1. Carcinoma Treatment :

1. Irradiation : In small superficial cancers, and in lesions of the free border of the epiglottis and in patients of poor surgical risk.

2. Surgical :

a. Hemi laryngectomy : for lesions less than 1 cm. and mobile vocal cords.

b. Total laryngectomy : For lesions more than 1 cm. and fixed vocal cords, either

one or both and with subglottic extention ( J.H. Ogura and H.F. Biller 1973 ).

2. Sarcoma Treatment : ( Fibrosarcoma ).
   Local surgery; but if the tumour is anaplastic with many mitoses radical surgery is indicated, even to an amputation of an extremity or a total laryngectomy. Ratio therapy is of no help in these tumours ( Daniel Miller 1950).

F.B. Treatment : In the treatment of patient with a foreign body in the larynx, speed is the great essential. In the case of a small youngster it is advisable to hold the child upside down by his heels in the hope that this procedure will dislodge the foreign object. If this approach is not successful, the patient should be rushed to a properly equipped hospital without delay, since nothing can be accomplished at home. At such an institution the removal of a foeign body in this location can be performed through the direct laryngoscope. If the obstruction threatens life , it may be necessary to perform a temporary emergency tracheotomy until the foreign object can be removed( R.E. Ryan 1970). Bronchoscopy is performed under

general anaesthesia . The younger child, the greater is the danger and the more difficult is the successful removal. The first bronchoscopy is used to aspirate secretions and to locate the foreign body and note its position. A small object, such as a pin, may have moved peripherally so that it cannot be seen through the bronchoscope, and if it cannot be extracted by a magnetized probe, its removal may have to be undertaken by the thoracic surgeons at á thoracotomy . ( J.F. Birrell 1977)

Objects in the larynx may be removed by indirect or direct laryngoscopy. Indirec laryngoscopy, in which laryngeal mirrors and used, is satisfactory in many instances if the object large or easily grasped. Direct laryngoscopy, in which a distally or proximally lighted laryngoscope is used, is preferable in other cases as direct vision is obtained.

Foreign objects in the trachea, bronchi and esophagus are removed in 98 percent of the cases by means of peroral endoscopy. ( H. Charles Ballenger and John. J. Ballenger 1954 ).

General anasthesia in case of a large foreign body, such as a jack, in the esophagus of a child may be rapidly

fatal by asphyxia because of the compressibility of the tracheoesophageal party wall . The best forceps for a jack is the ball forceps applied to one of the four globular extremities. Any presentation other than a globular extremity is to be regarded as a malpresentation that should be converted into a favourable one.

Safety Pins in the Esophagus : These are dangerous. They may be dealt with by 16 different methods. The most frequently used are (1) endogastric version (2) endogastric straightening (3) the point-sheathed and (4) closing methods. After the for

After the foreign body has set up extensive pathologic changes from prolonged sojourn. This is in marked contrast to pulmonary suppuration of other cause. Foreign bodies in the esophagus can be removed by esophagoscopy through the mouth in almost all cases. The public press reports show that ingested foreign bodies are the cause of many deaths by asphyxia before the victim can reach a physician. A large piece of candy may asphyxiate a child. ( Galbriel F. Tucker 1973 ).

TREATMENT OF TRAUMA OF THE LARYNX :

I. Mechanical :

a. ( Acute laryngeal injury ).

Early treatment : 1. A patient with an acute laryngo-tracheal trauma must be looked upon as a very complicated emergency case. In the presence of other serious injures, there is a risk that moderate laryngeal symptoms might be overlooked and therapy delayed. Early examination by a laryngologist is therefore necessary.

2. The first step must be focused on the patient's ventilation, cardiovascular situation, bleeding ,aspiration and subcutaneous emphysema, and followed by a judgement of the type and extent of the injury ( N.G. Toremalm 1974).

3. A patient with an acute laryngotracheal contusion must be kept under observation because of the risk of delayed oedema or haematoma. ( N.G. Toremalm 1974).

4. The initial procedure should not be limited to routine nasotracheal intubation or tracheotomy ( N.G. Toremalm 1974).

5. The necessary examinations and the operative planning are most effectively done by an experienced team and it ought to be made during the emergency situations. ( N.G. Toremalm 1974 ).
Then the Treatment depends on the tissues involved as follows :

1. Mucosal oedema is treated by corticosteroids, antibiotics and if necessary nasotracheal intubation for few days.
2. Submucosal haematoma is treated by drainage via a fenestration of the thyroid cartilage, antibiotics and possibly temporary intubation.
3. Soft tissue lacerations with emphysema are controlled by multiple drainage, manual compression or pure oxygen ventilation. The inlet must be found ( N.G. Toremalm 1974).
4. Cartilage fractures with one dislocated fragment can sometimes be repositioned endoscopically ( J.J. conley 1953 ) Comminuted fractures combined with soft tissue lacerations must , however, be treated by open surgery and reconstruction. Tissues with good vitality should be spared for reconstructive

purposes. A free graft may be necessary. Acute tracheal injuries can usually be treated by an end-to end anastomosis without stent support. In the larynx, however, a stent is necessary in order to establish not only a sufficiently wide air passage but also to rebuild a normal laryngeal cavity ( N.G. Toremalm 1974).

The following demands should characterize and ideal laryngeal stent : (1) an anatomically correct design, (2), a non-irritant material (3), a thin yet stable wall, (4) a hollow design which allows a whisper-like voice without any risk for aspiration and (5) a stent easy to remove endoscopically. ( N.G. Toremalm 1974).

b. Treatment of edema or haematoma post laryngeal injury

Usually , edema and hematoma will resolve spontaneously and completely in the absence of any other injury. Treatment should include absolute voice rest and humidification of inspired air. The use of corticosteroid pre parations is of value in reducing inflammatory response and preventing progression of edema . Sometimes temporary tracheostomy is necessary to relieve obstruction.

## II. TREATMENT OF BURNS :

### a. Thermal Burns

Emergency tracheostomy may be necessary for edematous obstruction of the larynx. However, the edema usually subsides rapidly. Adequate humidification and complete voice rest are indicated. Healing is usually prompt and the complication of chronic stenosis is rare with this type of burn. ( J.J. Ballenger 1969).

### b. Chemical Burns :

These burns are treated in association with hypopharyngeal and esophageal burns. Attempts to neutralize the caustic substance are usually a waste of time by the time the child is seen. The introduction of substances such as vinegar, lemon juice and sodium bicarbonate invites aspiration, because of alteration in sphincter function , and the risk of aspiration pneumonia. Vomiting should not be induced as this may subject the esophagus to a second exposure to the corrosive agent. After the airway is controlled, by tracheostomy if necessary, every effort must be made to prevent laryngeal stenosis. Broad-

spectrum antibiotics and steroid preparation are given in adequate doses to retard infection and fibrosis. Insertion of a finger-cot stent in the supraglottic area may be of value in preventing stenosis, since most of the burning is usually around the superior margins of the laryngeal vestibule ( J.J. Ballenger 1969).
( Kilner 1954 ), adviced plastic surgery to repair the burns. Patients with postcricoid burns must be watched for the development of perichondritis. The use of naso-gastric tubes for feeding such patients is contraindicated and a gastrostomy should be inserted for this purpose. Chronic supraglottic laryngeal stenosis may be the end result despite preventative efforts. ( J.J. Ballenger 1969).

TREATMENT OF PEDIATRIC VOCAL CORD PARALYSIS :

Maintenance of an airway and adequate nutrition are the two main concerns in neonates and children with vocal cord paralysis. Tracheotomy is required in approximately one-half of the patients with bilateral cord paralysis while only 20% with unilateral paralysis require tracheotomy ( D.D. Dedo 1979).

Feeding tubes and gastrostomy may be required to provide adequate nutrition until swallowing is relearned.

The congenitals are dependent upon the survival rate of the underlying CNS or cardiovascular anomaly ( Rontal M. and Rontal E. 1979).

Surgecal Treatment

1. a. Arytenoidectomy : The most frequently used approach for arytenoidectomy is through an incision in the lateral neck exposing the thyroid cartilage and in turn the cricoid cartilage. ( Woodman 1946).

   b. An arytenoidectomy may be accomplished endoscopically by transoral intralaryngeal approach as described by ( WM. C. Thornell 1957).

   c. A third approach is through a thyrotomy insision. Each of these operations is designed to increase the glottic opening which is required in cases of bilateral Recurrent laryngeal Nerve paralysis ( Marlin Weaver 1976).

2. Teflon Implant : Teflon granules , suspended in glycerine may be injected in the soft tissue of a paralyzed vocal cord, which leaves the vocal cord immobile and 3-5 mm. lateral to the midline ( Arnold, 1964).

3. Nerve Repair : Approximation of the divided end is generally followed by nerve regeneration. Recovery of nerve function is limited to tonus in a position of adduction ( Malin weaver 1976).

4. Nerve-Muscle pedicle : Transplant of a nerve-muscle pedicle offers a prospect of return of laryngeal function ( Marlin Weaver 1976).

TREATMENT OF THYROID GLAND :

Preparations available for the treatment of cretinism are : desicrated thyroid ( USP ), purified thyroglobulin ( proloid), cytomel, and synthroid ( Judson J. Van Wyk. 1968).

TREATMENT OF CONGENITAL GOITER :

If Goiter is severe leading to dyspnea so full substitution therapy with thyroid hormone to achieve immediate euthyroidism and more rapid shrinkage of gland. The drug of choice is $T_3$ .

If respiratory obstruction is severe : Tracheal compression must be carried out by surgical resection of thyroid isthmus or partial thyroidectomy. Trachestomy alone hazardous because it ends fatally ( Handelsman and Sussman, 1957).

TREATMENT OF INTRATHORACIC GOITER :

Administration of sodium-L-thyroxin ( 100 to 300 Mg/ 24 hr). Will usually cause the goiter to decrease in size. When symptoms are severe, surgical removal of the endotracheal goiter is indicated ( Angelo M. DiGeorg 1979).

TREATMENT OF THYROID CANCER :

Total thyroidectomy is the treatment of choice. Affected lymph nodes should be removed at the time of thyroidectomy, and administering therapeutic doses of radio-iodine.

Thyroid hormone, in full doses, should always be given after surgical or radioiodine therapy has been carried out. ( George W. Clayton 1969).

TREATMENT OF ACUTE MEDIASTINITIS :

Acute nonsuppurative mediastinitis usually requires no treatment beyond that of the primary condition. Treatment of acute suppurativemediastinitis and of acute mediastinal abscess is surgical drainage , antibacterial therapy is always indicated ( Carl Muschen Heim 1951).

TREATMENT OF CHRONIC MEDIASTIUTIS :

The treatment of chronic mediastinitis depends on the nature of the infection ( Carl Muschenheim 1951).

TREATMENT OF TUMOURS AND CYSTS :

Benign tumours and cysts of the mediastinum should usually be removed, even if causing no symptoms. When they are discovered early in life ( Carl Muschenheim 1951).

Substernal or intrathoracic goiter also should be removed in most cases, especially if there are any symptoms or signs of tracheal or superior vena cava compression. Malignant mediastinal tumours are usually inoperable . Roentgen therapy is effective; especially in Hodgkin's disease and in lymphosarcoma ( Carl Muschenheim 1951).

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## SUMMARY AND CONCLUSION

Stridor is the difficult harsh, vibratory , noisy and high pitched sound heared during respiration. It is one of the problems confronting the otolaryngologists with increasing frequency.

Very little was known about stridor and laryngeal diseases before the twentieth century, but nowadays there is a wide knowledge of the different causes of stridor, and many modern facilities are present to help in investigation, diagnosis and treatment of nearly every case of stridor.

The condition may or may not be serious, but its cause should be determined. The history and clinical study of the type and phase of stridor will suggest a tentative diagnosis , but radiologic, laryngoscopic , bronchoscopic or esophagoscopic examination may have must be made to provide the final estimate of the true nature of the lesion.

In this humble ~~thesis the researcher~~ work it is tried to mention ad in chapter I the embryology, anatomy and physiology of the larynx, and the main important differences

between the adult larynx and the infantile larynx and why the infantile larynx is softer and very sensitive and easily, rapidly and severely affected by any inflammation which leads to oedema giving rise to laryngeal obstruction ( 1 mm. edema in the subepithelial tissues leads to obstruction of the glottic area by 50%).

The high position of the infantile larynx makes it more susceptible for foreign bodies inhalation.

The infantile epiglottis is long tapering and folded which may obliterate the vestibule of the omega-shaped larynx and provides less support for the aryepiglottic folds. The lumen of the infantile larynx and trachea is smaller in proportion to the body as a whole than in the case with the adult.

The not well developed central nervous system of infants ~~makes the larynx gets spasm~~ irritates spasm easily and rapidly leading to stridor.

The researcher mentioned in chapter II the historical review of stridor and tried to explain in brief what is meant by Stridor and how to investigate a case of stridor.

In chapter III the researcher discussed nearly most of the important causes of stridor.

In congenital lesions, the history generally indicates that symptoms have been present since birth. Stridor during the first months of life is most often due to congenital soft fladcid larynx, although congenital cysts, webs, paralysis of the larynx and congenital vascular anomalies compressing the trachea will give similar signs and symptoms.

Laryngomalacia is the commonest due to excessive flaccidity of the supraglottic portion of the larynx is more common in lower socio-economic groups and poor nutritional status . Congenital subglottic stenosis is one of the most common laryngeal anomalies. If it is severe it gives severe harsh barking stridor and dyspnea.

Children with congenital subglottic stenosis are not seen at birth unless it is accompanied by other condition like tracheo-oesophageal fistula, and is diagnosed during intubation for fistula repair.

In inflammatory processes and trauma the stridor is characterized by an acute onset. Acute epiglottitis

is a common and dangerous dissease leading to stridor with cherry-red edematous epiglottis. Direct laryngoscope is rarely necessary fordiagnosis, unless a bronchoscope or endotracheal tube is immediatly available for fear of respiratory obstruction.

Acute laryngo tracheobronchitis and diphtheritic laryngitis are also common and serious disease leading to respiratory obstruction.

Laryngeal papillomata are the most common infantile benign tumour , it is present multiple and it may be due to either congenital, traumatic, chronic irriation , hormonal or viral cause.

They are dangerous in regard to the dyspnea which they cause. They tend to recur after removal. But disappear at puberty.

Foreign bodies ingestion is one important cause of stridor, it is common in children and infants, because children put whatever comes into their grasp into their mouths adding the protective reflex action is not so efficient, carelessness may contribute to foreign body inhalation by improper preparation of food, hasty eating, permitting

children to play while eating or talking with food in the mouth.

Laryngeal foreign bodies which are completely obstructive usually cause sudden death. So inhalation of foreign bodies should be regarded as true emergency only when embarrassement of air way or there is danger of perforation or the foreign body may migrate to more dangerous position .

The researcher also mentioned the dangerous effects and burns due to ingestion or inhalation of lye and other chemicals e.g. clorox orlysol which is most common in children.

Prolonged endotracheal intubation is a common cause of stridor by the dangerous trauma to the larynx. So in case of endotracheal intubation take care for . Period of intubation, size and shape of the tube, trauma of intubation, movement of the tube, sustained tissue hypoxia and the chemical composition of the tube which may be irritant to the larynx.

Vocal cord paralysis was mentioned also as an important cause of stridor inchildhood.

Early detection of Pediatric laryngeal paralysis requires a high index of suspicion and is important to

prevent catástrophes during periods of acute respiratory embarrassment.

In chapter IV the researcher discussed nearly the different methods of treatment of stridor according to the underlying cause. Some cases of stridor like acute laryngitis with severe respiratory obstruction or edema of the larynx need cricothyrotomy laryngotomy which must be performed in cases of real urgency and without proper facilities and training.

Tracheostomy is indicated in emergency cases or essential in others, it is performed nearly in most cases of stridor like , congenital web, closed and opened injuries of the larynx, subglottic stenosis, burns with dyspnea, foreign bodies inhalation and in cases of intubation injury. Also in acute inflammations of the larynx acute epiglottitis where tracheostomy is urgently necessary with nasotracheal intubation. Also acute laryngotracheo-bronchitis and indiphtheritic laryngitis it is indicated early before signs of cardiac failure appears.

Valved tracheostomy tube is indicated in Atresia of the larynx to promote normal laryngo tracheal physiology and surgical treatment is delayed till the larynxis large enough.

The researcher discussed the treatment of multiple papillomate of the infantile larynx either by tracheostomy, removal by direct laryngoscopy or by thryotomy. Fulguration diathermy was used, also hormonal treatment locaiy applied or systemic sexhormones, Radiotherapy, antibiotics and recently carbon dioxide cryosurgery, laser coupled with microlaryngoscope proved to be a good and safe method. Immunological method by autogenous vaccine showed good results.

Burns are treated by tracheostomy, antibiotics, steroids, finger -cot stent in supraglottic to prevent stensosis . Gastrotomy is essential for feeding in certain cases In cases of vocal cord paralysis, tracheostomy is needed at first, than surgical treatment by doing arytenoidectomy either through lateral ~~neck~~ pharyngotomy, transoral intralaryngeal or thyrotomy insicion. Then teflon implant or nerve repair.

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بسم الله الرحمن الرحيم

## الملخص العربي للبحث

## " الاختناق عند الاطفال "

يعتبر هذا الموضوع من الاسباب الهامة التى قد تؤدى الى الوفاة اذا لم يتم التصرف فى علاجها بسرعة وحكمة .

حتى بداية القرن العشرين لم يكن يعرف الشئ الكثير عن امراض الحنجرة بشكل خاص ، ولكن مع تقدم العلم وتوفر الاجهزة العلمية والطبية الحديثة التى تساعد على دراسة الحالة جيدا تمكن من التوصل الى معرفة التشخيص بدقة ، وكذلك الى معرفة الاسباب التى تؤدى الى هذه الحالة .

ففى كل حالة لابد من معرفة التاريخ الكامل للمريض وفحص كل حالة فحصا دقيقا شاملا بالمنظار ، والاشعة لتحديد مكان الاصابة .

ففى هذا البحث المتواضع تناول الباحث وصف حنجرة الاطفال تشريحيا لمعرفة الاسباب التى تؤدى الى الاصابة بالاختناق بسهولة عند الاطفال اكثر من البالغين وهى :-

١) المكان المرتفع للحنجرة عند الاطفال .

٢) الحجم الصغير للحنجرة بالنسبة للجسم بعكس البالغين .

٣) التكوين الغضروفى والعضلى لحنجرة الاطفال اكثر ليونة من البالغين ، كذلك الغشاء المخاطى والطبقة التى تليه رخوة ومتسعة وغير ملتصقة كما فى البالغين .

حيث انها فى حالة الالتهاب تتورم محدثة ضيقا بفراغ الحنجرة يؤدى الى الاختناق ، حيث أن ١مم من محيط الحنجرة يؤدى الى ضيق ٥٠٪ من حيز ( فراغ ) الحنجرة .

٤) الشكل الطولى والمدبب والمنثنى كالاسطوانة للسان المزمار ( اللهاة ) .

٥) أما من الناحية الفسيولوجية فان الجهاز العصبى عند الاطفال غير كامل النمو كما أنه غير متزن ويمكن ان يتهيج بسهولة مؤديا الى انقباض فى الحنجرة واختناق الطفل .

ثم قدم الباحث نبذة تاريخية عن الاختناق مع شرح تفصيلى لمعنى الاختناق وكيف يتأثر الاطفال به ، والتغيرات التى تطرأ على أصوات التنفس والدورة التنفسية .

كما قدم الباحث بابا كاملا للاسباب المختلفة التى تؤدى الى الاختناق عند الاطفال وركز على التشوهات الخلقية لما لها من دور هام فى اصابة الطفل بالاختناق بعد الولادة مباشرة أو بعد عدة أشهر من الولادة ، حيث هذه هى الفترة المهمة فى حياة الطفل اذ أنه لو لم يتم علاجه فسيكون من الصعب

عليه أن يتمكن من الرضاعة التى ستبنى جسمه وتغذيه ، وبذا تقل مقاومته للأمراض ويضعف جسمه وقد تؤدى به الحالة الى التهابات متكررة فى الجهاز التنفسى والرئتين مما قد ينتج عنه الوفاة .

كذلك من الاسباب الهامة الالتهابات الحادة التى تصيب حنجرة الطفل وخاصة الدفتيريا - ولو أنها اصبحت قليلة فى السنوات الاخيرة نتيجة التطعيم ضد الدفتيريا - الا أن بعض الالتهابات الحادة التى تصيب الحنجرة والقصبة الهوائية والتفرعات الرئوية فى آن واحد اصبحت على درجة عالية من الخطورة على الاطفال لانها قد أخذت مكان الدفتيريا فى خطورتها .

ومن الاسباب المهمة ابتلاع الاجسام الغريبة عند الاطفال حيث أنه لديهم عادة فطرية بأن يضعوا فى افواههم اى شئ يقع فى قبضتهم فيسهل انزلاقه الى الحنجرة التى لها وضع مرتفع عند الاطفال مؤدية الى الاختناق الحاد الذى قد يؤدى الى الوفاة .

كذلك فان اهمال الأم بوضع (السوائل) (مثل الصودا الكاوية والبوتاس) فى متناول ايدى الاطفال حيث يشربونها مؤدية الى الاصابة بحروق شديدة ومؤلمة جدا مع نزيف من الفم والبلعوم والمرئ والحنجرة وقد تكون الحروق من الدرجة الاولى ، أو الثانية أو الثالثة حيث لها مضاعفات خطيرة جدا من تقرحات مؤلمة جدا تعقبها فترة تليف وضيق بالمرئ

والحنجرة ، والتهابات وتدمير لغضاريف الحنجرة .

كما تعرض الباحث الى ذكر اسباب الاصابات فى الحنجرة التى تؤدى الى تهتك الغشاء المخاطى ثم الى ضيق فى الحنجرة خاصة فى المنطقة الواقعة اسفل الاحبال الصوتية وذلك نتيجة لكثرة استعمال المنظار ، واطالة فترة بقاء انابيب التخدير فى القصبة الهوائية اكثر من اللازم حيث يجب مراعاة الفترة الزمنية وحجم الانبوبة وشكلها كذلك يجب تثبيتها جيدا مع التأنى فى وضعها .

كما بين الباحث أن من الاسباب المهمة للاختناق عند الاطفال هو اصابة الأحبال الصوتية بالشلل نتيجة العنف فى سحب المولود لحظة الولادة حيث ينتج عنه شلل مؤقت او مستديم للاحبال الصوتية .

كذلك فان وجود نوع شائع جدا من الاورام الحميدة فى حناجر الاطفال واسمه ( الورم الحلمى Multiple Papillomas ) يعتبر عاملا مهما يؤدى الى الاختناق عند الاطفال ، حيث انه لم يعرف سبب هذه الاورام بالتحديد لكن هناك افتراضات تقول انها ربما تكون اوراما خلقية من الاصل ، او انها نتيجة الالتهابات المزمنة فى الحنجرة أو نتيجة للاحتكاك المباشر بين انابيب التخدير والغشاء المخاطى للحنجرة أو انها تعود الى مؤثرات هرمونية أو فيروسية ، وهى تصيب الأطفال الحديثى الولادة بالاختناق ، اما فى الاطفال الاكبر سنا فتؤدى الى تغيرات

فى الصوت قد تصل الى حد اختناقه تماما ، وتغيرات فى التنفس مثل السعال وصعوبة فى التنفس واحتقان ثم اختناق ويميل هذا النوع فى الأطفال الى السكون وقلة النشاط .

كما أنفرد الباحث بابا خاصا لعلاج الحالات المختلفة من الاختناق تبعا للاسباب المؤدية الى ذلك ، الا أنه فى معظم الحالات نضطر الى عمل شق حنجرى أو فتحه بأعلى القصبة الهوائية وتركيب أنبوبة فى فتحة القصبة الهوائية متصلة بالخارج للتنفس تكون اما مؤقتة أو مستديمة تبعا للحالة ، وغالبا ما تكون مستديمة . ( Tracheostomy tube )

وأرجو أن اكون قد وفقت فيما وصلت اليه ، وما توفيقى الا بالله ﷻ

بسم الله الرحمن الرحيم

بناء على قرار فضيلة الاستاذ الدكتور / رئيس جامعة الازهر بتشكيل لجنة مناقشة والحكم على رسالة الطبيب زهير زكى عبد الحفيظ محمود بتاريخ

وعنوانها : " الاختناق عند الاطفال "

اجتمعت اللجنة بتاريخ

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# " الاختناق عند الاطفـــــال "

البحث المقدم من :

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لنيـل درجـة الماجستير فـــــى

أمراض وجراحة الانف والاذن والحنجـرة

اشـراف

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