LARYNGOLOGY

CLINICAL REFERENCE GUIDE

Justin Ross, DO
Haig Panossian, MD
Mary J. Hawkshaw, RN, BSN, CORLN
Robert T. Sataloff, MD, DMA, FACS

PLURAL PUBLISHING INC.

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5521 Ruffin Road
San Diego, CA 92123

e-mail: information@pluralpublishing.com
Website: https://www.pluralpublishing.com

Typeset in 9/11 Adobe Garamond by Flanagan's Publishing Services, Inc.
Printed in the United States of America by Integrated Books International

Library of Congress Cataloging-in-Publication Data:
Names: Ross, Justin, DO author. | Panossian, Haig, author. | Hawkshaw, Mary, author. | Sataloff, Robert Thayer, author.
Title: Laryngology : clinical reference guide / Justin Ross, Haig Panossian, Mary J. Hawkshaw, Robert T. Sataloff.
Description: San Diego, CA : Plural Publishing, [2020] | Includes bibliographical references and index.]
Identifiers: LCCN 2019017790| ISBN 9781635501407 (alk. paper) | ISBN] 1635501407 (alk. paper)
Subjects: | MESH: Otorhinolaryngologic Diseases | Laryngeal Diseases | Otolaryngology | Handbook
Classification: LCC RF46 | NLM WV 39 | DDC 617.5/1--dc23
LC record available at https://lccn.loc.gov/2019017790

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CHAPTER

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Embryology and Development of the Larynx

Haig Panossian, Mary J. Hawkshaw, and Robert T. Sataloff

STAGES AND STRUCTURES OF DEVELOPMENT

Histology-Based Theories of Development

- early descriptions of development theorized by His in 1885
- **Respiratory primordium (RP)**: an outpouching of the cephalic portion of the pharynx which gives rise to lungs and bronchi
- theorized that tracheoesophageal (TE) separation was an ascending process initiated by groove posterior to RP separating foregut into ventral trachea and dorsal esophagus
- led to decreased distance between floor of pharyngeal pouch (IV) and RP over time
- Zaw-Tun (1982) developed alternative theory based on study of Carnegie and Shatner collections of human embryos
- **Primitive laryngopharynx (PLPh)**: segment of foregut that separates pharyngeal floor (at level of pharyngeal pouch IV) from RP
- PLPh lengthens (descending process) as embryo matures and distance between pharyngeal pouch IV and RP remains constant
- PLPh becomes supraglottis
- primitive pharyngeal floor develops into glottis

Carnegie Stages 11 Through 14 (According to Zaw-Tun's Theory)

- hepatic primordium (HP) and RP are both within the septum transversum (ST)
- rapid proliferation of HP causes foregut to lengthen and RP to separate from HP
- separation of RP allows it to dilate and bifurcate into lung buds
- as RP descends from pharyngeal floor, gives rise to funnel-shaped structure
    1. cephalic end becomes infraglottis; caudal end becomes trachea, bronchi and lungs
- concurrently, foregut segment develops into esophagus, stomach, and cephalic half of duodenum
- **Laryngeal inlet**: median slit in pharyngeal floor in Stage 13 (described by Arey in 1965)
- proliferation of pharyngeal mesoderm and arteries of fourth branchial arch lead to arytenoid swellings lateral to entrance of slit
- growth of median epiglottic swelling derived from hypobranchial eminence causes slit to become a T-shaped laryngeal outlet
- transverse pouch of the T descends along epithelial lamina to form laryngeal cecum (see later section, Stages 15–18)

- recanalization of epithelial lamina forms supraglottis (see later section, Stages 19–23)
- controversy regarding interpretation of median slit:
  1. Kallius (1897) and Frazer (1910) reported it represented the cephalic portion of the trachea/glottis
  2. Zaw-Tun demonstrated that it was entrance to PLPh and hence the entrance to supraglottis

Computer Model-Based Theories of Development

- Henick et al (1993) used computer-generated 3-dimensional studies of mouse embryos
- led to discovery of several additional structures but largely supported Zaw-Tun's theories, with the following additional details

Stages 12 Through 14

- **Respiratory diverticulum (RD)**: ventral outpouching of foregut lumen that extends into RP
- originates at primitive pharyngeal floor—eventually becomes glottis
- cephalic portion of RD becomes infraglottis
- RD gives rise to bilateral projections known as bronchopulmonary buds
  1. tethered to superior aspect of septum transversum so are drawn caudally as foregut lengthens
  2. develop into lower respiratory tract
  3. carina develops from caudal aspect of RD
- foregut lengthens in cephalocaudal plane as heart and hepatic primordium proliferate on opposing surfaces of septum transversum
  1. gives rise to developing trachea and esophagus
  2. vascular compromise to developing esophagus or trachea can lead to developmental anomalies
- **Laryngeal mesodermal anlage**: triangular-shaped proliferation
  1. develops into laryngeal cartilages and muscles
- elevation of median pharyngeal floor leads to arytenoid swellings at level of fourth pharyngeal pouch

Stages 15 Through 18

- **Epithelial lamina**: formed as ventral aspect of PLPh compressed
- temporarily obliterates PLPh in ventral to dorsal direction
- obliteration incomplete, spares dorsal pharyngoglottic duct (PhGD) and ventral laryngeal cecum

- PhGD is last remnant of patent communication between hypopharynx and infraglottis
- **Laryngeal cecum**: triangular-shaped lumen that originates at ventral aspect of arytenoid swellings
  1. progresses caudally along ventral aspect of PLPh until reaches level of glottis in stage 18

Stages 19 Through 23

- epithelial lamina begins to recanalize from dorsocephalad to ventrocaudal direction
  1. forms the laryngeal vestibule and supraglottis
- Stage 21: laryngeal cecum becomes the two laryngeal ventricles
- Stage 23: glottis is last portion of PLPh to recanalize
  1. stenosis if process incomplete

Development of Laryngeal Cartilages and Muscle

- **Third pharyngeal arch**:
  1. stylopharyngeus muscle
  2. common and internal carotid arteries
  3. body and greater cornu of hyoid bone
  4. glossopharyngeal nerve
- **Fourth pharyngeal arch**:
  1. inferior pharyngeal constrictor, cricothyroid, and cricopharyngeus muscles
  2. subclavian artery (on right) and aorta (on left)
  3. thyroid cartilage, cuneiform cartilages
  4. superior laryngeal nerve; jugular and nodose ganglia
- **Sixth pharyngeal arch**:
  1. all intrinsic muscles of larynx (except cricothyroid)
  2. pulmonary artery (on right) and ductus arteriosus (on left)
  3. cricoid, arytenoid, and corniculate cartilages, trachea
  4. recurrent laryngeal nerve (left remains with ductus arteriosus, while right moves cranially and laterally in association with subclavian artery as the right homologue of the sixth arch disappears)
- **Extrinsic muscles develop from epicardial ridge**:
  1. superficial layer develops into sternohyoid and omohyoid muscles
  2. deep layer attaches to oblique line of thyroid cartilage as sternothyroid and thyrohyoid muscles

DEVELOPMENTAL ANOMALIES

Esophageal and Tracheal Anomalies

Esophageal Atresia

- present in ~8% of infants with TE anomalies
- Sx: increased salivation requiring frequent suctioning, pulmonary triad of coughing, choking, and cyanosis as saliva overflows from blind pouch of esophagus into airway
- aspiration greater in infants with associated TE fistula and direct airway connection

Tracheoesophageal Fistulae (Figure 2–1)

- esophageal atresia with distal TE fistula (~85% of affected infants)
- Sx: gastric distention as air ingested with each breath, respiratory symptoms result from direct aspiration of mix of air and stomach contents as well as decreased diaphragmatic excursion
- TE H-fistula without atresia (~8%)
- proximal TE fistula with atresia (~1%)
- proximal and distal TE fistulae with atresia (~1%)

Tracheal Anomalies

- not associated with laryngeal or pulmonary abnormalities because vascular insult limited to developing trachea
- tracheal agenesis/atresia
- tracheal stenosis with complete tracheal rings
  1. concentric circular tracheal cartilage with loss of posterior membranous trachea

Supraglottic and Glottic Atresia

- incomplete recanalization of PLPh can lead to supraglottic and glottic atresias
  1. Type I: complete supraglottic stenosis
  2. Type II: partial supraglottic stenosis, where communication between supraglottis and infraglottis maintained through patent pharyngoglottic duct
  3. Type III: glottic web
- associated subglottic stenosis in types I and II due to prevention of development of infraglottic region