ESOPHAGEAL DYSFUNCTIONS

M. S. THESIS

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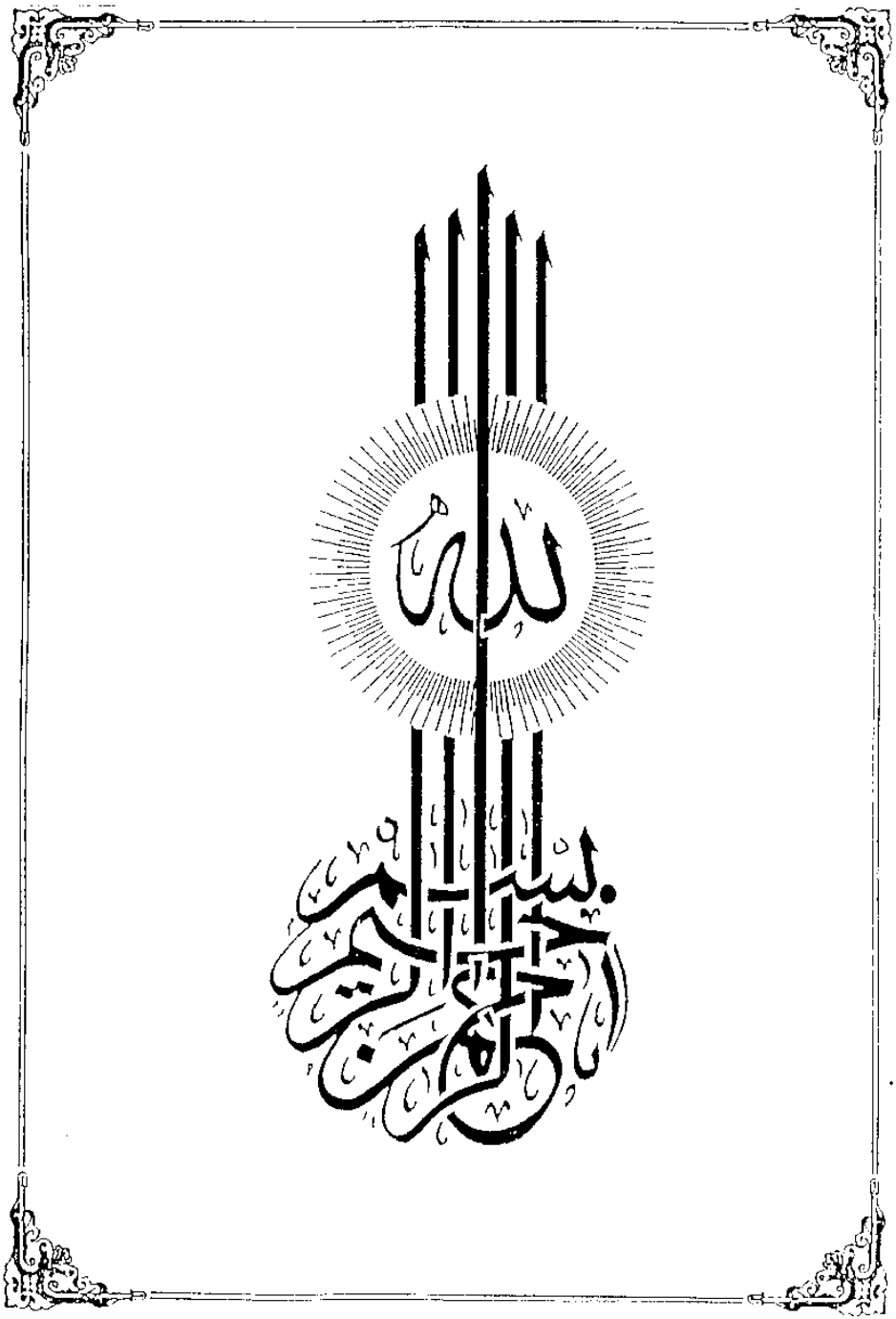

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C O N T E N T S

INTRODUCTION

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The purpose of this work is to present an up-to-date review of the normal physiology and pathophysiology, and of the diagnosis and treatment of clinical disorders of esophageal and pharyngeal motility. The esophagus is essentially a hollow tube, which is about 25 cm long in adult. The act of swallowing initiates a chain of events that results in transport of ingested material from the mouth to the stomach. The events constitute orderly contractions of muscles of pharynx and esophagus, and relaxations at both ends of the esophagus. In clinical practice an esophageal motility study should be considered in any patient whose esophageal complaints are not readily explained by a structural abnormality.

Under resting conditions, the esophagus remains as a collapsed tube with a sphincter at either end - a lower and an upper esophageal sphincter. The esophageal sphincters act as antireflux mechanisms to prevent entry of gastrointestinal contents into the esophagus and then into the pharynx. Incompetence of the lower sphincter allows reflux of gastrointestinal contents that may damage the esophageal mucosa. Dysfunctions of the upper sphincter may result in cervical dysphagia or esophago-pharyngeal regurgitation with subsequent fatal pulmonary aspiration. Motor disorders of the esophagus cause dysphagia and may also be the source of chest pain. The following list of the potential causes of dysphagia is quite

extensive and the subject of this study, is the esophageal motility disorders.

Causes of dysphagia:

A. Oropharyngeal dysphagia:

1. Neuromuscular diseases: cerebrovascular accidents, pseudobulbar palsy, brain stem tumours, hereditary or degenerative diseases, bulbar poliomyelitis, Parkinson's disease, peripheral neuropathy, polymyositis, dermatomyositis, myotonia dystrophica, occulopharyngeal muscular dystrophy, and myasthenia gravis.

2. Carcinoma of the piriform sinus; carcinoma of the base of tongue.

3. Cricopharyngeal incoordination.

4. Zenker's diverticulum.

5. Globus hystericus.

B. Esophageal transport dysphagia:

1. Plummer - Vinson syndrome.

2. Esophagitis: peptic, corrosive, candida invasion, bacterial, fungal, and viral infections (herpetic), granulomatous disease, syphilis.

3. Diverticulitis.

4. Esophageal webs.

3. Presbyesophagus.

4. Intrinsic tumours: benign and malignant.

5. Primary esophageal motility disorders: achalasia, and diffuse spasm.

6. Secondary esophageal motility disorders: scleroderma, diabetes mellitus, thyroid disease, chronic alcoholism, amyloidosis, neuromuscular diseases.

9. Extrinsic masses:

(a) Cervical: Thyroiditis, thyromegally, thyroid neoplasm, hypertrophic spurs of cervical spine.

(b) Mediastinal: tumours, pericarditis, pulmonary abscess, pleuritis, mediastinitis.

(c) Vascular: dysphagia lusoria, aneurysm.

C. Esophagogastric junction:

1. Spasm: hypertensive, hypercontracting, hypertonic sphincter.

2. Achalasia: cardiospasm, Chaga's disease.

3. Stricture postesophagitis: reflux esophagitis, post-traumatic (corrosive, nasogastric intubation, surgery, instrumentation, forign body injury).

4. Neoplasm - esophageal, pharynx cardia.

5. Zenker's diverticulum.

6. Hiatal hernia (paraesophageal).

7. Lower esophageal (Schatzki) ring.

CHAPTER I

ANATOMY & HISTOLOGY OF THE ESOPHAGUS

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DEVELOPMENT.

(Gray & Skandalakis, 1972)

The cranial end of both the esophagus and trachea becomes demarcated with the appearance of a median ventral diverticulum of the foregut about 22 or 23 days after fertilization. The esophagus will develop from the small area between the tracheal diverticulum and the stomach dilatation.

The tracheal diverticulum rapidly becomes a groove in the floor of the esophagus, and elongation of both structures begins. Two lateral speta develop in the tube during the third or fourth weeks. They grow, to meet and fuse in the midline. Hence, a partition is formed between the primitive trachea and esophagus (Fig. 1). Although separation proceeds toward the head, simultaneous elongation of both trachea and esophagus prevents the completion of the process before 34 to 36 days of age. By this stage, the beginning of the submucosal and muscular layers of both esophagus and trachea are visible. The elongation involves first the lower portion of the esophagus, and later the upper portion. At the level of tracheal bifurcation the diameter of the esophagus is reduced; it is here that most esophageal atresias will occur.

The esophageal lumen, round at first, becomes flattened dorsoventrally above and laterally below, during the 5th week. Four longitudinal folds are present by the tenth week. The dorsal and ventral folds develop caudad from the upper end and the lateral folds develop cephalad from the lower end. In the lower third, the ridges rotate 90° clockwise. This is a continuation of the rotation of the stomach, which begins before the esophageal folds are formed. During the 5th month, secondary folds appear by similar processes (Fig. 2).

During the seventh and eighth weeks, the esophageal epithelium proliferates until the lumen is nearly filled with cells, but complete occlusion of the lumen does not normally occur. However, the rare occurrance of a mucosal diaphragm across the lumen of the esophagus suggests that occlusion may occasionally take place. By the 10th week, recanalisation takes place and a single lumen is restored.

The superficial layer of epithelial cells has become ciliated by the 10th week. During the fourth month, the ciliated epithelium begins to be replaced, starting in the midesophagus, by islands of stratified squamous epithelium. Patches of ciliated epithelium may be present at birth.

Small areas of columnar cells toward the two ends of the esophagus are not replaced by stratified squamous