The Thyroid Gland: Anatomy & Physiology

Thyroid system

Hypothalamus

Anterior pituitary gland

Thyrotropin-releasing hormone (TRH)

Negative feedback

Thyroid-stimulating hormone (TSH)

Thyroid gland

Thyroid hormones (T3 and T4)

Increased metabolism

Growth and development

Increased catecholamine effect

JABIRAMI KRITHIGA
• FROM GREEK *thyreoeides* = SHIELD SHAPE

• ENDOCRINE GLAND

• REGULATES BMR

• STIMULATES SOMATIC AND MENTAL GROWTH

• PLAYS IMPORTANT ROLE IN CALCIUM METABOLISM
Thyroid Gland, Anterior and Posterior Views

Thyroid Gland: anterior view (left); and posterior view (right)
• TWO LOBES JOINED BY ISTHMUS
• PYRAMIDAL LOBE (80%) PROJECT UPWARDS FROM ISTHMUS OR EITHER OF THE LOBES
• A FIBROMUSCULAR BAND levator glandulæ thyroideæ DESCEND FROM THE BODY OF THE HYOID BONE TO ISTHMUS OR TO PYRAMIDAL LOBE
• Gland lies against C5, 6, 7 & T1 vertebrae

• Clasps the upper part of trachea

• Each lobe extends from middle of thyroid cartilage to 4th or 5th tracheal ring

• Isthmus extends from 2nd to the 3rd tracheal
• EACH LOBE MEASURES 5cmX2.5cmX2.5cm
• ISTHMUS MEASURES 1.2cmX1.2cm
• GLAND MEASURES 25gms
• LARGER IN FEMALES THAN MALES
• INCREASES IN SIZE DURING PREGNANCY AND MENSTRUATION
• **TRUE**- PERIPHERAL CONDENSATION OF CONNECTIVE TISSUE OF GLAND

• **FALSE/SURGICAL**- FROM PRETRACHEAL LAYER OF DEEP CERVICAL FASCIA
APPLIED ANATOMY

• A DENSE CAPILLARY PLEXUS IS PRESENT DEEP TO THE TRUE CAPSULE. TO AVOID HGE DURING SURGERY, THYROID IS REMOVED ALONG WITH TRUE CAPSULE

• BUT, IN PROSTATE THE VENOUS PLEXUS LIES B/W 2 CAPSULES AND THEREFORE DURING PROSTATECTOMY, BOTH THE CAPSULES ARE LEFT BEHIND
Thyroid:
- **False capsule**
- **Plane of cleavage**
- **True capsule**
- **Venous plexus**
- **Gland substance**

Prostate:
- **False capsule**
- **Venous plexus**
- **True capsule**
- **Plane of cleavage**
- **Gland substance**

During surgery, the thyroid is removed along with the true capsule. However, during prostatectomy, both the capsules are left behind to avoid HGE.
• DEEP CERVICAL FASCIA

• HAS 4 COMPONENTS

1. INVESTING LAYER
2. PREVERTEBRAL LAYER
3. PRE TRACHEAL LAYER
4. CAROTID SHEATH
SUSPENSORY LIGAMENT OF BERRY

- THE PRETRACHEAL LAYER IS THIN ALONG THE POSTERIOR BORDER OF THE LOBES, BUT THICK ON THE INNER SURFACE OF THE GLAND WHERE IT FORMS A SUSPENSORY LIGAMENT OF BERRY WHICH CONNECTS THE GLAND TO THE CRICOID CARTILAGE
WHY THYROID MOVES WITH DEGLUTITION

• DURING 1st STAGE OF DEGLUTITION
• HYOID MOVES UP
• PULLS PRETRACHEAL FASCIA UP
• THIS PULLS LIGAMENT OF BERRY UP
• THIS PULLS THYROID UP
ALL STRUCTURES ENCLOSED IN THE PRETRACHEAL FASCIA MOVES UP WITH DEGLUTITION

- THYROGLOSSAL CYST
- SUBHYOID BURSITIS
- PRE TRACHEAL LYMPH NODES
- PRE LARYNGEAL LYMPH NODES
RELATIONS

LATERAL(SUPERFICIAL)
Relation w/ Strap muscles

- Lateral - sternothyroid
- Anterior
  - omohyoid muscle
  - sternohyoid
- Inferior - SCM (lower portion)

** motor nerve supply from the ansa cervicalis enters these muscles inferiorly.**
STRAP MUSCLES

- STYLOHYOID
- STERNOHYOID
- OMOHYOID
- STERNOTHYROID
- THYROHYOID

SUPRAHYOID MUSCLES

HYOID

BONE

INFRAHYOID OR STRAP MUSCLES
Thyroid gland
Larynx
Carotid artery
Trachea
Pharynx
Parathyroid glands
Esophagus
Trachea
VASCULAR ANATOMY

• HIGHLY VASCULAR

• SUPERIOR THYROID ARTERY

• INFERIOR THYROID ARTERIES
• 3 VEINS
• SUP THYROID VEIN DRAINS INTO IJV OR COMMON FACIAL V.
• MIDDLE THYROID VEIN DRAINS TO IJV
• INFERIOR THYROID VEIN INTO LEFT BRACHICEPHALIC V.
• A 4\textsuperscript{TH} VEIN OF KOCHER’S EMERGE B/W MIDDLE AND INFERIOR VEIN AND DRAIN INTO IJV
Innervation

Principally from **ANS**

- *Parasympathetic* fibers – from vagus
- *Sympathetic* fibers – from superior, middle, and inferior ganglia of the sympathetic trunk

Enter the gland along with the blood vessels.
LYMPHATICS

- **PRIMARILY TO INTERNAL JUGULAR NODES**
- **SUPERIOR POLE & MEDIAL ISTHMUS TO SUPERIOR GROUP**
- **LOWER POLE OF THYROID TO INFERIOR GROUP**
- **EMPTY INTO PRETRACHEAL & PARATRACHEAL NODES**
APPLIED ANATOMY

• RECURRENT LARYNGEAL NERVE SUPPLIES THE INTRINSIC MUSCLE OF LARYNX EXCEPT CRICOTHYROID WHICH IS SUPPLIED BY EXTERNAL LARYNGEAL NERVE

• ACCIDENTAL DAMAGE TO THIS NERVE DURING SURGERY CAUSES IPSILATERAL VOCAL CORD PARALYSIS

• & DIFFICULTY IN PHONATION
• RT SIDE IT ORIGINATES FROM VAGUS CROSSES FIRST PART OF SUBCLAVIAN.A LOOPS UNDER IT RUNS OBLIQUE TO ENTER LARYNX AT LEVEL OF CRICOID
• LEFT SIDE AFTER ORIGIN FROM VAGUS CROSSES AORTIC ARCH LOOPS POSTERIORLY AROUND LIGAMENTUM ARTERIOSUS ASCENDS MEDIALLY IN TRACHEO ESOPHAGEAL GROOVE

• SUPERIOR LARYNGEAL NERVE HAS INTERNAL BRANCH(SENSORY) & EXTERNAL BRANCH(MOTOR) HELPS IN VOCAL CORD TENSION AND PITCH OF VOICE
• SUPERIOR LARYNGEAL NERVE RUNS IN CLOSE PROXIMITY TO SUPERIOR POLE VESSELS, TO AVOID INJURY SUPERIOR POLE VESSELS SHOULD BE INDIVIDUALLY LIGATED & DIVIDED LOW ON THYROID GLAND AND DISSECTED LATERALLY TO CRICOTHYROID MUSCLE
SURFACE ANATOMY

- Notch Atop Thyroid Cartilage
- Circo-Thyroid Membrane
- Lobes of Thyroid Gland
- Thyroid Cartilage
- Carotid Arteries
HISTOLOGY

- COMPOSED OF CLOSELY PACKED SACS CALLED FOLLICLES
- FOLLICLES CONSIST OF:
  - FOLLICULAR CELLS: SECRETE THYROXINE (T4) AND TRIIODOTHYRONINE (T3)
- **Colloid**, a jellylike substance inside a follicle that contains thyroglobulin.

- **Parafollicular cells** - located outside follicles secrete calcitonin. They are located in the upper poles of the thyroid lobes, reflecting their origin as neuroectodermal cells derived from the ultimobranchial bodies, and are part of the amine containing precursor uptake decarboxylase (APUD).

- **Medullary CA** arises from C cells.
Figure 5.3.3
Effect of TSH on the thyroid gland. 

A. Hypothyroid follicles. 
B. Euthyroid (normal) follicles. 
C. Stimulated hyperplastic follicles.
EMBRYOLOGY

- 1\textsuperscript{st} ENDOCRINE ORGAN
- \approx 24\textsuperscript{th} DAY OF GESTATION
- PROLIFERATION OF THE ENDODERMAL CELLS ON THE MIDLINE PHARYNGEAL FLOOR
• BY THE 4\textsuperscript{TH} WK OF GEST. A ENDODERMAL THICKENING DEVELOPS IN THE FLOOR OF THE PHARYNX B/W TUBERCULUM IMPAR (2) AND THE HYPOBRANCHIAL EMINENCE (OR COPULA 4)

• "THYROID PRIMORDIUM"
• The thickened region soon depresses below the surface to form a diverticula called thyroglossal duct.

• The depression is then called foramen caecum.
• THE DUCT DESCENDS IN FRONT OF THE PHARYNGEAL GUT, HYOID BONE AND LARYNGEAL CARTILAGES AND REACHES ITS FINAL POSITION IN FRONT OF THE TRACHEA IN THE 7TH WEEK
• The proliferation of the cells of the bifid ends of the duct gives rise to the two lobes of the thyroid.
– THE INF. PARATHYROID DEVELOPS FROM THE ENDODERM OF THE THIRD PHARYNGEAL POUCH

– SUP. PARATHYROID DEVELOPS FROM THE ENDODERM OF THE FOURTH PHARYNGEAL POUCH

N.B. THE SUP. PARATHYROID DEVELOPS FROM THE CAUDAL PHARYNGEAL COMPLEX ALONG WITH THE PARAFOLLICULAR C CELLS
SURGICAL SIGNIFICANCE OF THYROID EMBRYOLOGY

- ANOMALIES OF SHAPE
- ANOMALIES OF POSITION
- ECTOPIC THYROID
- THYROGLOSSAL CYST
- THYROGLOSSAL FISTULA
ANOMALIES OF SHAPE

1. **PYRAMIDAL LOBE**
   - IN UP TO 80% PEOPLE
   - PERSISTENCE OF THE INFERIOR END OF THE THYROGLOSSAL DUCT
   - MAY BE ATTACHED TO THE HYOID BONE OR MAY BE INCORPORATED INTO A THYROGLOSSAL DUCT CYST

   • **ABSENT ISTHUMUS**
   • **ABSENT LOBE**
ANOMALIES OF POSITION

• LINGUAL THYROID
• MASS OF ECTOPIC THYROID TISSUE-BASE OF TONGUE
• F:M=4:1
• INCIDENCE 1/1,00,000
• ONSET OF SYMPTOMS COINCIDE WITH PREG, PUBERTY AND MENOPAUSE
OTHER POSITION ANOMALIES

- INFRAHYOID THYROID
- SUPRAHYOID THYROID
- INTRA THORACIC THYROID
- INTRA LINGUAL THYROID
ECTOPIC THYROID TISSUE

- SMALL MASSES OF THYROID TISSUE PRESENT IN ABNORMAL SITES
- ECTOPICS ARE SEEN IN LARYNX, TRACHEA, ESOPHAGUS, PONS, PLEURA, PERICARDIUM AND OVARIRES
- ECTOPICS IN RELATION TO THE DEEP CERVICAL LYMPH NODES WERE ONCE THOUGHT TO BE LATERAL ABERRANT THYROID, IS NOW BELIEVED TO BE NODAL METS FROM CA THYROID (WILLIS)
THYROGLOSSAL CYST

• CAN BE FORMED IN ANY PART OF THE THYROGLOSSAL TRACT
• SWELLING MOVES WITH PROTRUSION OF TONGUE AS WELL AS SWALLOWING BECAUSE OF THE ATTACHMENT TO THE FORAMEN CAECUM
• CYST >1CM WHICH PERSISTS SHOULD BE EXCISED (SISTRUNK)
Body of Tongue
Foramen Caecum
Epiglottis
Hyoid Bone
Position of Thyroglossal Cysts
Thyroid Cartilage
Cricoid Cartilage
Thyroid Gland
THYROGLOSSAL FISTULA

• NEVER CONGENITAL
• FORMED DUE TO INFECTION OR INADEQUATE EXCISION OF THYROGLOSSAL DUCT
• LINED BY COLUMNAR EPITHELIUM AND DISCHARGES MUCUS AND IS A SEAT OF RECURRENT ATTACKS OF INFLAMMATION
Feedback control of thyroid hormone

TRH Thyrotrophin-releasing hormone
TSH Thyrotrophin
T₃ Tri-iodothyronine
T₄ Thyroxine

T₄ → T₃
in brain

T₄ → T₃
in pituitary

TSH

T₃
T₄

Thyroid gland

T₃
T₄

Target tissue
PHYSIOLOGY

• IODINE – RAW MATERIAL FOR THYROID HORMONE SYNTHESIS
• INGESTED IODINE CONVERTED TO IODIDE BEFORE ABSORPTION
• 150 µg OF IODINE MINIMUM REQD FOR NORMAL THYROID FUNCTION OF WHICH 120µg ENTER THYROID AT NORMAL RATES OF HORMONE SYNTHESIS AND SECRETION
• REDUCED INTAKE OF IODINE CAUSES ENDEMIC GOITRE
• THYROGLOBULIN IS A GLYCOPROTEIN SYNTHESIZED IN THYROID CELLS AND SECRETED INTO THE COLLOID BY EXOCYTOSIS

• THYROID PEROXIDASES (TPO) CATALYSES THE OXIDATION OF IODINE AND ITS ABSORPTION

• THYROGLOBULIN IS BOUND TO THYROID HORMONES TILL IT IS SECRETED INTO BLOOD, AFTER WHICH IT IS INGESTED BACK INTO THE COLLOID

• IMMUNOGLOBULIN AGAINST TSH RECEPTOR OR TPO ANTIBODY IS FOUND IN HASHIMOTOS
STEPS OF THYROID HORMONE SYNTHESIS

1. IODINE TRAPPING
2. OXIDATION
3. IODINATION
4. COUPLING
5. STORAGE
6. RELEASE
IODINE TRAPPING

• AVAILABLE THROUGH CERTAIN FOODS (EG, SEAFOOD, BREAD, DAIRY PRODUCTS), IODIZED SALT, OR DIETARY SUPPLEMENTS ETC

• THYROID CELL MEMBRANES FACING THE CAPILLARIES CONTAIN A SYMPORTER OR IODINE PUMP THAT TRANSPORTS Na⁺/I⁻ AGAINST ELECTROCHEMICAL GRADIENT

• IT IS CALLED A NIS (Na-I SYMPORTER)
APPLIED PHYSIOLOGY

- THIOCYANATES AND PERCHLORATES BLOCK THIS STEP
- IN FAMILIAL GOITRE OR DYSHORMONOGENSES THERE IS IMPARIED IODINE TRAPPING OR COUPLING
OXIDATION

• THE IODIDE TRANSPORTED INSIDE THE CELL HAS TO BE CONVERTED BACK TO IODINE
• THIS IS DONE BY THE THYROID PEROXIDASES (TPO)
IODINATION

- THE IODINE MOLECULE COMBINES WITH TYROSINE AT 3\textsuperscript{RD} POSITION TO FORM 3-MONIOIODOTYROSINE (MIT)

- $I + \text{TYROSINE} \rightarrow 3\text{-MIT}$
• IODINATION OF MIT PRODUCES 3,5-DIIODOTYROSINE (DIT)

• $I + MIT \rightarrow 3,5, \text{ DIT}$
COUPLING REACTIONS

• MIT+DIT —— 3,5,3’TRIIODOTYROSINE (T₃)

• DIT+DIT —— 3,5,3’,5’,TETRAIODO TYROSINE (THYROXINE T₄)
• THYROID PEROXIDASE CATALYSES
  1. OXIDATION
  2. IODINATION
  3. COUPLING

• TPO INHIBITORS CARBIMAZOLE, METHIMAZOLE & PROPYLTHIOOURACIL CAN ACT AT ALL THESE LEVELS
STORAGE & RELEASE

- AFTER FORMATION, T₃ AND T₄ ARE TRANSPORTED TO THE FOLLICLES WHERE THEY REMAIN STORES ASA COLLOID
- ON STIMULATION VIA TSH, THESE HORMONES ARE RELEASED INTO CIRCULATION
Synthesis of Thyroid Hormone

1. Thyroglobulin is synthesized and discharged into the lumen

2a. Trapping (active uptake) of iodide (I⁻)
2b. Oxidation

3. Iodine enters follicle lumen where it is attached to tyrosine in colloid forming DIT and MIT

4. Iodinated tyrosines are linked together to form T₃ and T₄

5. Thyroglobulin colloid is endocytosed and combined with a lysosome

6. Lysosomal enzymes cleave T₄ and T₃ from thyroglobulin colloid and hormones diffuse from follicle cell into bloodstream

To peripheral tissues

MIT (T₂) DIT (T₁)
Figure 118-2. Algorithm for using the TSH level in the evaluation of thyroid function.
• REFETOFF SYNDROME—T4 IS INCREASED WITH NORMAL TSH
• T3 THYROTOXICOSIS – ISOLATED INCREASE OF T3 WITH NORMAL T4 & RADIOIODINE UPTAKE
• T₄ is biologically inactive in target tissues until converted to T₃

• The liver is the major extrathyroidal T₄ conversion site for production of T₃

• Some T₄ to T₃ conversion also occurs in the kidney and other tissues
THYROID HORMONE BINDING PLASMA PROTEINS

- ALBUMIN
- THYROXINE BINDING PREALBUMIN (TBPA) = TRANSTHYRETIN
- THYROXINE BINDING GLOBULIN (TBG)
- HIGH DENSITY LIPOPROTEIN
• MORE THAN 99% OF CIRCULATING T3 AND T4 ARE IN BOUND FORM
• BOUND FORM IS NOT AVAILABLE FOR METABOLIC ACTION
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<th>T4</th>
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<td><strong>POTENCY</strong></td>
<td>8 TIMES MORE</td>
<td>LESS</td>
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<td><strong>ACTIVITY</strong></td>
<td>MORE ACTIVE</td>
<td>INACTIVE IN TARGET TISS</td>
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<td><strong>CIRCULATION</strong></td>
<td>LESS IN CIRCULATION</td>
<td>MAIN CIRC. HORMONE</td>
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<td><strong>DURATION OF ACTION</strong></td>
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<td>TSH</td>
<td>0.5-4.7 mU/L</td>
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<td>T4</td>
<td>4.5-10.9 µg/dL</td>
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<td>T3</td>
<td>60-181 ng/dL</td>
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<td>FREE T4</td>
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<tr>
<td>FREE T3</td>
<td>1.4-4.4 pg/mL</td>
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<td>Tissue</td>
<td>Physiological Action</td>
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<td>Heart</td>
<td>Cardiac output: Heart rate</td>
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<td>Adipose tissue</td>
<td>Stimulate lipolysis</td>
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<td>Muscle</td>
<td>Increases protein breakdown</td>
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<tr>
<td>Bone</td>
<td>Promotes normal growth and skeletal development</td>
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<td>NERVOUS SYSTEM</td>
<td>PROMOTE NORMAL BRAIN DEVELOPMENT</td>
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<tr>
<td>GUT</td>
<td>INCREASES RATE OF CHO ABSORPTION</td>
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<td>STIMULATE LDL RECEPTOR FORMN.</td>
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<td>CALORIGENIC ACTION STIMULATE O2 CONSUMPTION</td>
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THANK YOU