CHAPTER ONE

Introduction

1.1. Introduction:

The interaction between kidney and thyroid function are known for years. Thyroid hormones (TH) are necessary for growth and development of the kidney and for the maintenance of water and electrolyte homeostasis. On the other hand kidney is involved in the metabolism and elimination of (TH) (Feinstein EI, etal, 1982). Both hypothyroidism and hyperthyroidism are accompanied by remarkable alterations in the metabolism of water and electrolyte as well as cardiovascular function. All these effects generate changes in water and electrolyte kidney mangment.it is known that hypothyroidism reduce and hyperthyroidism increase the kidney-to-body weight ratio by a not fully under stood mechanism. On the other hand, children with congenital hypothyroidism have an increase prevalence of congenital renal anomalies. These finding support an important role of TH during embryogenesis thyroid dis function cause significant changes in kidney function, effect renal blood flow, GFR tubular function, electrolytes homeostasis, electrolytes pump function, and kidney structure (Feinstein EI, etal, 1982).

Doctor mistaken to correlate between thyroid hormones and kidney.

1-2 Statement of problem

The kidney is not an organ for metabolism and elimination of thyroid hormones but also a target organ of some of iodiothyronine actions ,thyroid dysfunction causes remarkable change in gluomerular and tubular function and electrolytes and water homeostasis which may lead to acute and choric kidney injury.

1-2-1 general objective:

To evaluate the effect of thyroid hormone on the kidney using ultrasound.

1-2-2 Specific objective:

- To detect the main effect of thyroid hormones disorders on the kidney.
- To detect the kidney size and kidney echo-texture for thyroid hormone disorder patients.

1-3 Hypothesis:

There are interaction between thyroid hormone and kidney.

1.4 Overview of study:

This study consist of five chapters , Chapter One contents Introduction, hypothesis, objectives and overview of the study.   Chapter tow deals with Literature reviewwhich includes anatomy, physiology and pathology of the thyroid gland , normal and abnormal sonographic features of thyroid gland and previous studies.   Chapter Three contains   methodology of the study. Chapter four contains results of the study. Chapter five contains Discussion of the results, Conclusion and recommendation, finally there are list of refrences and .appendices which include ultrasound images

Chapter Two

Literature Review

2 Literature Review:

2-1 Thyroid Anatomy:

Medial portion of thyroid gland, Medial gland, Arises from the endodermal tissue tissue of the base of tongue posteriorly, , the foramen cecum-lack of migration results in a retrolingualmass mass ,Attached to tongue by the thyroglossal duct -lack of atrophy after thyroid descent results in midline cyst formation (thyroglossal duct cyst)duct ,Descent occurs about fifth week of fetal life --remnants may persist along track of descent along descent, Lateral lobes of thyroid gland Lateral gland, Derived from a portion of ultimobranchial body, part of the body, fifth branchial pouch from which C pouch cells are also derived (calcitonin secreting cells). (Agur *etal*, 2009)
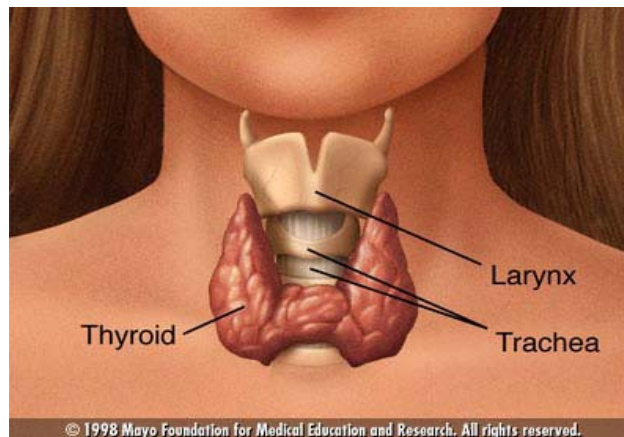

Figure (2-1) Thyroid gland anatomy (Clinical Anatomy A revision and applied anatomy for clinical students, 11th edition, 2006)

Lingual Thyroid (failure of descent) Verification that lingual mass is thyroid by its ability to trap

Significance: May be only thyroid tissue in body (~70% of time), removal resulting in hypothyroidism; treatment consists of TSH suppression to shrink size. (Agur *etal*, 2009)
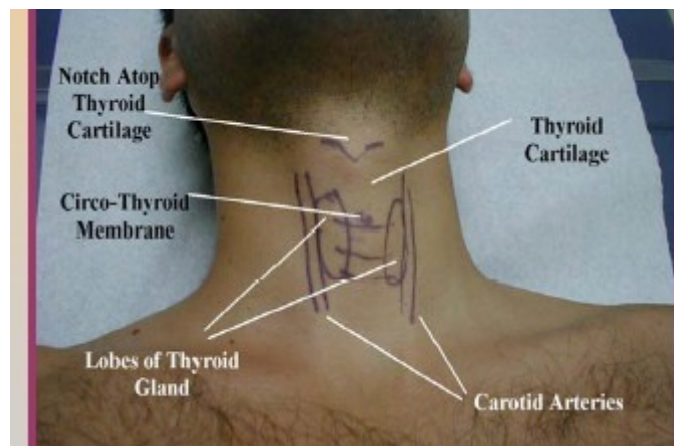
Figure (2-2) Thyroid gland position (gray's anatomy for the students, 2007)

Brownish-red and soft during life Usually weighs about 25-30g (larger in women) Surrounded by a thin, fibrous capsule of connective tissue External to this is a "false capsule "formed by pre tracheal fascia Right and left lobes United by a narrow isthmus, which extends across the trachea anterior to second and third tracheal cartilages In some people a third "pyramidal lobe "exists, ascending from the isthmus towards hyoid bone. (Agur *etal*, 2009)
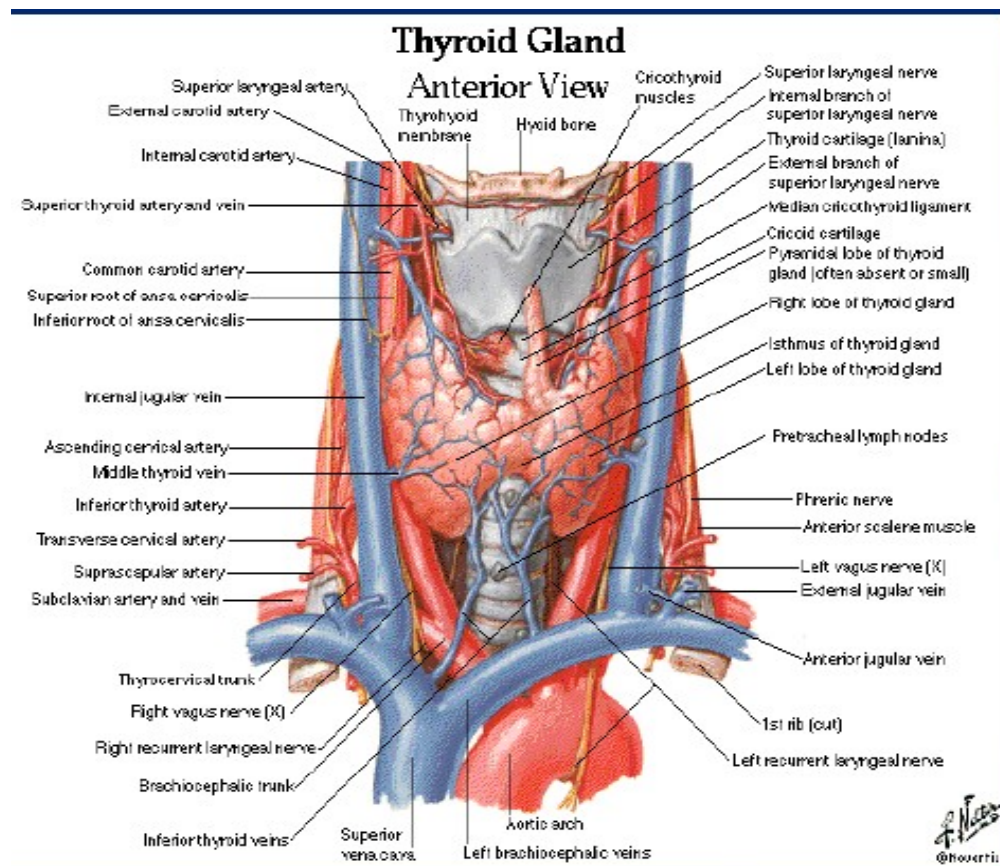

Figure (2-3) Position and relations Anterior View (Clinical Anatomy A revision and applied anatomy for clinical students, 11th edition, 2006)

- Clasps anterior and lateral surface of *pharynx, larynx, esophagus and trachea* "like a shield"

- Lies deep to steno thyroid and steno thyroid muscles

- Parathyroid glands usually lie between posterior border of thyroid gland and its sheath (usually 2 on each side of the thyroid), often just lateral to anastomosis between vessel joining superior and inferior thyroid arteries

- Internal jugular vein and common carotid artery lie postero-lateral to thyroid

- Recurrent laryngeal nerves an important structure lying between trachea and thyroid may be injured during thyroid surgery →ipsilateral VC paralysis, hoarse voice

- Each lobe Pear-shaped and ~5cm long extends inferiorly on each side of trachea (and esophagus), often to level of 6thtracheal cartilage

- Attached to arch of cricoid cartilage and to oblique line of thyroid cartilage moves up and down with swallowing and oscillates during speaking. (Agur *etal*, 2009)

Figure (2-4) Arterial supply (gray's anatomy for the students, 2007)

- highly vascular

- main supply from superior and inferior thyroid arteries lie between capsule and pretracheal fascia (false capsule).

- All thyroid arteries anastomose with one another on and in the substance of the thyroid, but little anastomosis across the median plane (except for branches of superior thyroid artery).

- Superior thyroid artery first branch of ECA descends to superior pole of gland, pierces pretracheal fascia then divides into 2-3 branches

- Inferior thyroid artery branch of thyroid-cervical trunk runs superomedially posterior to carotid sheath reaches posterior aspect of gland divides into several branches which pierce pretracheal fascia to supply inferior pole of thyroid gland intimate relationship with recurrent laryngeal nerve in ~10% of people the thyroid ima artery arises from aorta, brachiocephalic trunk or ICA, ascends anterior to trachea to supply the isthmus. (Agur *etal*, 2009)

2-1-1 Venous drainage:

- Usually 3 pairs of veins drain venous plexus on anterior surface of thyroid superior thyroid veins drain superior poles middle thyroid veins drain lateral parts.

- Superior and middle thyroid veins empty into internal jugular veins inferior thyroid veins drain inferior poles empty into brachia-cephalic veins often unite to form a single vein that drains into one or other brachia-cephalic vein. (Agur *etal*, 2009)

2-1-2 Lymphatic drainage:

Lymphatic's run in the interlobular connective tissue, often around arteries communicate with a capsular network of lymph vessels pass to prelaryngealLN's → pretracheal and paratracheal LN's lateral lymphatic vessels along superior thyroid veins pass to deep cervical LN's. Some drainage directly into brachio-cephalic LN' sor directly into thoracic duct. (Agur *etal*, 2009)
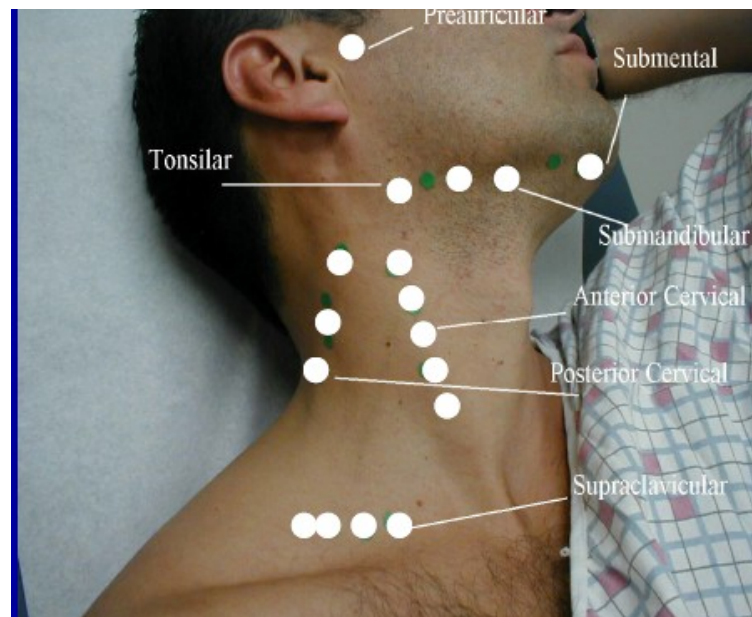
Figure (2-5) Lymph nodes of the neck (gray's anatomy for the students, 2007)

2-1-1-4Innervation:

Nerves derived from superior, middle and inferior cervical sympathetic ganglia reach thyroid through cardiac and laryngeal branches of vague nerve which accompany arterial supply postganglionic fibers and vasomotor –indirect action on thyroid by regulating blood vessels. (Agur *etal*, 2009)
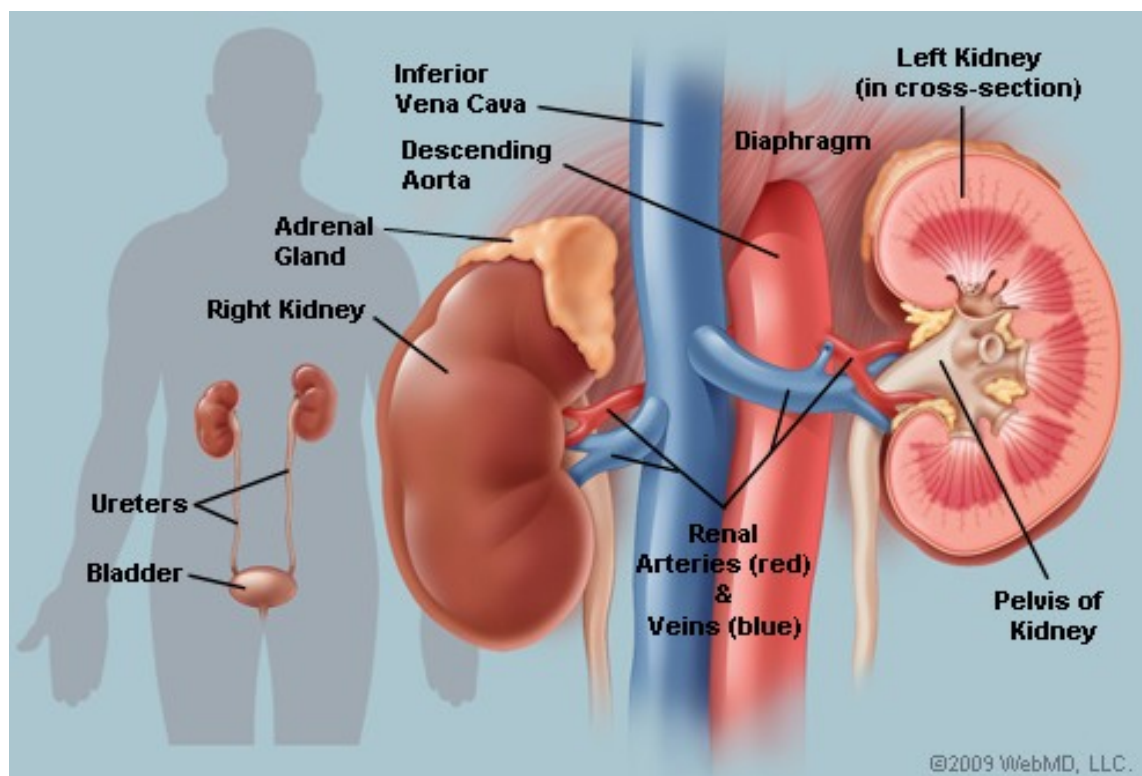
2-2 Anatomy of the Kidneys:

2-2-1 Location:

Figure (2-6) location of the kidney (Clinical Anatomy A revision and applied anatomy for clinical students, 11th edition, 2006)

The kidneys are a pair of organs found along the posterior muscular wall of the abdominal cavity. The left kidney is located slightly more superior than the right kidney due to the larger size of the liver on the right side of the body. Unlike the other abdominal organs, the kidneys lie behind the peritoneum that lines the abdominal cavity and are thus considered to be retroperitoneal organs. The ribs and muscles of the back protect the kidneys from external damage. Adipose tissue known as per renal fat surrounds the kidneys and acts as protective padding.

Structure the kidneys are bean-shaped with the convex side of each organ located laterally and the concave side medial. The indentation on the concave side of the kidney, known as the renal hilus, provides a space for the renal artery, renal vein, and ureter to enter the kidney. (John E. Hall, 2006)
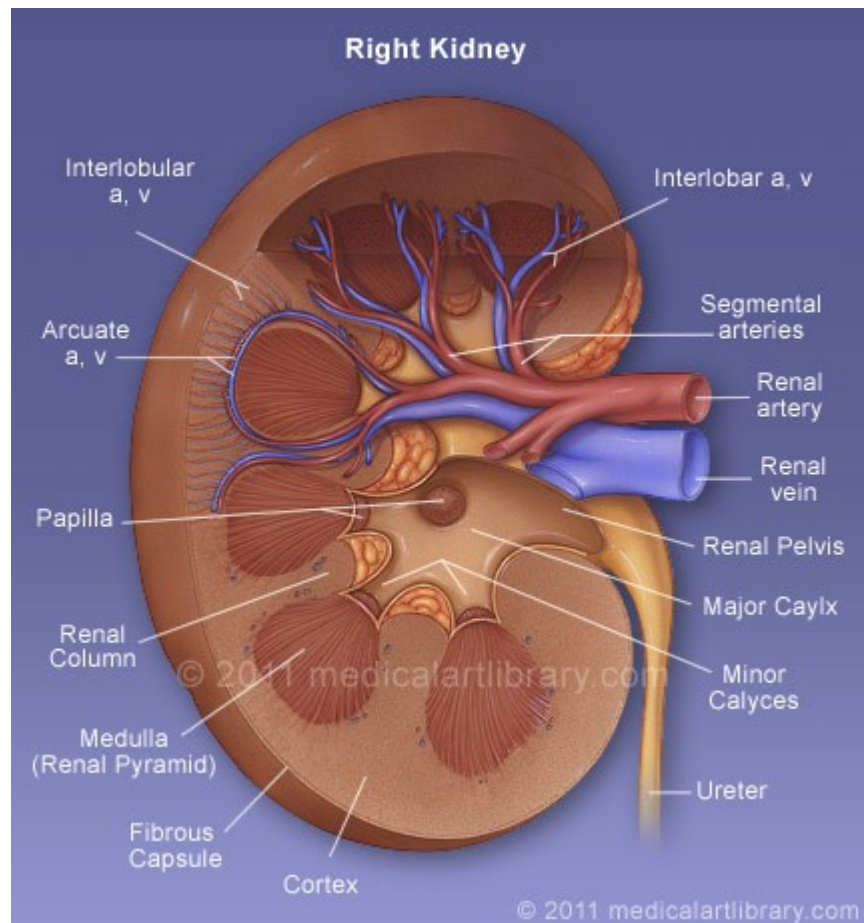
Figure (2-7) Right kidney in cross section (gray's anatomy for the students, 2007)

A thin layer of fibrous connective tissue forms the renal capsule surrounding each kidney. The renal capsule provides a stiff outer shell to maintain the shape of the soft inner tissues.

Deep to the renal capsule is the soft, dense, vascular renal cortex. Seven cone-shaped renal pyramids form the renal medulla deep to the renal cortex. The renal pyramids are aligned with their bases facing outward toward the renal cortex and their apexes point inward toward the center of the kidney.

Each apex connects to a minor calyx, a small hollow tube that collects urine. The minor calyces merge to form 3 larger major calyces, which further merge to form the hollow renal pelvis at the center of the kidney. The renal pelvis exits the kidney at the renal hilus, where urine drains into the ureter Blood Supply. (John E. Hall, 2006)
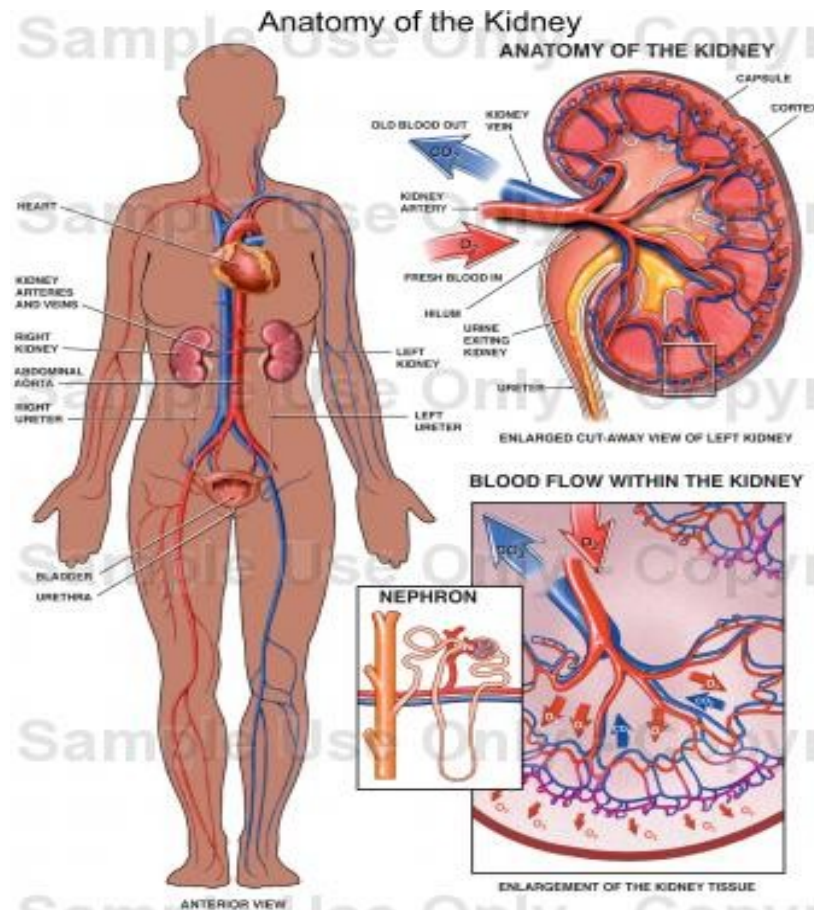
Figure (2-8) renal blood supply (Clinical Anatomy A revision and applied anatomy for clinical students, 11th edition, 2006)

2-2-2 Renal blood supply:

- The renal arteries branch directly from the abdominal aorta and enter the kidneys through the renal hilus.
- Inside our kidneys, the renal arteries diverge into the smaller afferent arterioles of the kidneys.
- Each afferent arteriole carries blood into the renal cortex, where it separates into a bundle of capillaries known as a glomerulus.
- From the glomerulus, the blood recollects into smaller efferent arterioles that descend into the renal medulla.
- The efferent arterioles separate into the peril tubular capillaries that surround the renal tubules.

- Next, the peritubular capillaries merge to form veins that merge again to form the large <u>renal vein</u>.

- Finally, the renal vein exits the kidney and joins with the <u>inferior vena cava</u>, which carries blood back to the heart. (John E. Hall, 2006)

2-2-3 The nephron:

Each kidney contains around 1 million individual nephrons, the kidneys' microscopic functional units that filter blood to produce urine. The nephron is made of 2 main parts: the renal corpuscle and the renal tubule.

Responsible for <u>filtering the blood</u>, our renal corpuscle is formed by the capillaries of the glomerulus and the glomerular capsule (also known as Bowman's capsule). The glomerulus is a bundled network of capillaries that increases the surface area of blood in contact the blood vessel walls. Surrounding the glomerulus is the glomerular capsule, a cup-shaped double layer of simple squamous epithelium with a hollow space between the layers. Special epithelial cells known as podocytes form the layer of the glomerular capsule surrounding the capillaries of the glomerulus. Podocytes work with the endothelium of the capillaries to form a thin filter to separate urine from blood passing through the glomerulus. The outer layer of the glomerular capsule holds the urine separated from the blood within the capsule. At the far end of the glomerular capsule, opposite the glomerulus, is the mouth of the renal tubule.

A series of tubes called the renal tubule concentrate urine and recover non-waste solutes from the urine. The renal tubule carries urine from the glomerular capsule to the renal pelvis.

- The curvy first section of the renal tubule is known as the proximal convoluted tubule. The tubule cells that line the proximal convoluted tubule reabsorb much of the water and nutrients initially filtered into the urine.

- Urine next passes through the loop of Henle, a long straight tubule that carries urine into the renal medulla before making a hairpin turn and returning to the renal cortex.

- Following the loop of Henle is the distal convoluted tubule.

- Finally, urine from the distal convoluted tubules of several nephrons enters the collecting duct, which carries the concentrated urine through the renal medulla and into the renal pelvis.

From the renal pelvis urine from many collecting ducts combines and flows out of the kidneys and into the ureters. (John E. Hall, 2006)

2-3 Thyroid Physiology:

The Thyroid Produces and Secretes 2 Metabolic Hormones

- Two principal hormones

  –Thyroxin (T4) and tri iodo thyronine (T3)

- Required for homeostasis of all cells

- Influence cell differentiation, growth, and metabolism

- Considered the major metabolic hormones because they target virtually every tissue.

TRH

 Produced by Hypothalamus

 Release is pulsatile, circadian

 Down regulated by T 3

 Travels through portal venous system to adeno hypothesis

 Stimulates TSH formation

Thyroid-Stimulating Hormone (TSH)

- Up regulated by TRH

- Down regulated by T4, T3

- Travels through portal venous system to cavernous sinus, body.

- Stimulates several processes

  – Iodine uptake

  – Colloid endocytosis

  – Growth of thyroid gland

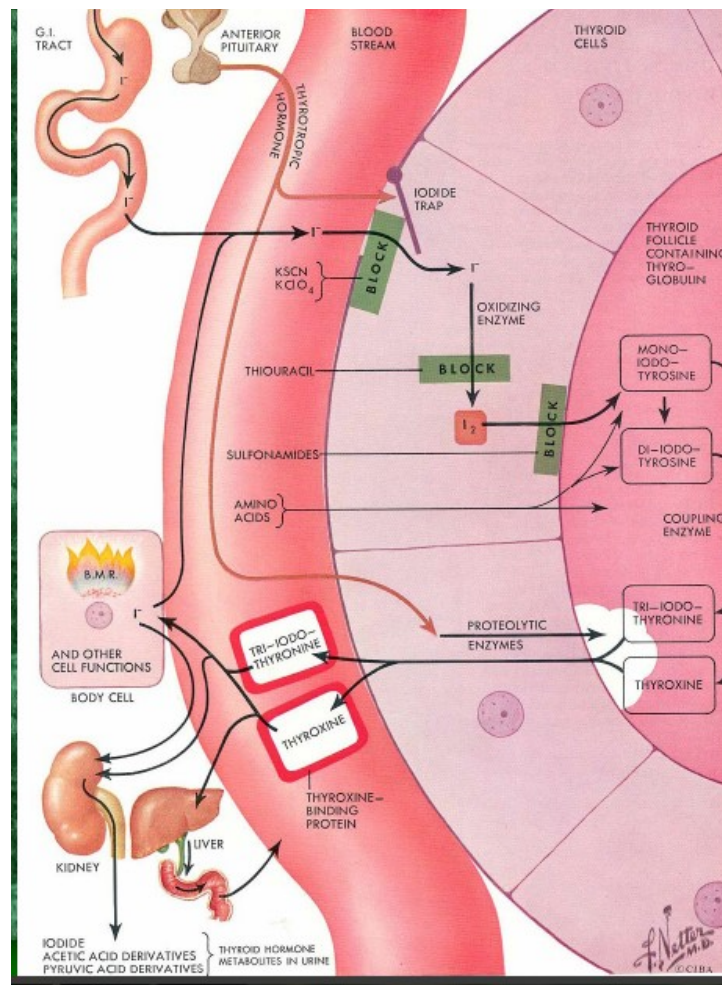- Produced by Adeno hypophysis Thyrotrophs. (Harold Elis, 2006)

Figure (2-09) thyroid hormones regulation (Medical Physiology 11th edition, 2006)
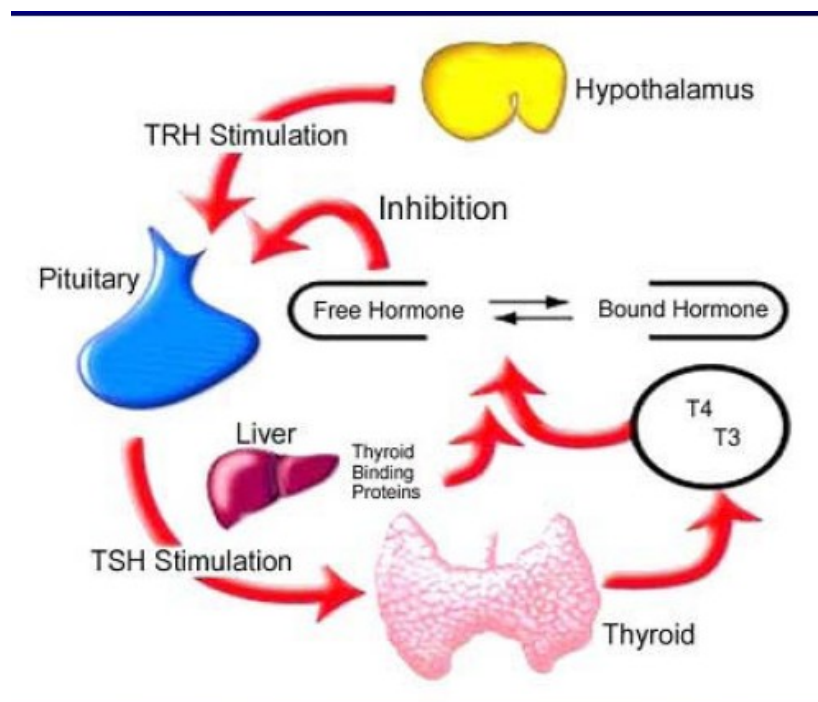
Figure (2-10) Hypothalamic-Pituitary-Thyroid Axis Negative Feedback Mechanism (Medical Physiology 11th edition, 2006)

2-3-1 Biosynthesis of T4 and T3:

The process includes:

- Dietary iodine (I) ingestion.

- Active transport and uptake of iodide (I-) by thyroid gland.

- Oxidation of I-andiodination of thyroglobulin (Tg) tyrosine residues.

- Coupling of iodotyrosine residues (MIT and DIT) to form T4 and T3.

- Proteolysis of Tg with release of T4 and T3 into the circulation.

- Iodine Sources.

- Available through certain foods (e.g., seafood, bread, dairy products), iodized salt, or dietary supplements, as a trace mineral.

- The recommended minimum intake is 150µg/day. (Harold Elis, 2006)

Active Transport and I-Uptake by the Thyroid:

- Dietary iodine reaches the circulation as iodide anion (I-)

- The thyroid gland transports I-to the sites of hormone synthesis

- I-accumulation in the thyroid is an active transport process that is stimulated by TSH.

Oxidation of I-and Iodination of Thyroglobulin (Tg) TyrosylResidues:

- I-must be oxidized to be able to iodinate tyrosyl residues of Tg

- Iodination of the tyrosyl residues then forms mono iodotyrosine (MIT) and di iodotyrosine (DIT), which are then coupled to form either T3 or T4

Thyroperoxidase (TPO):

- TPO catalyzes the oxidation steps involved in I-activation, iodination of Tg tyrosyl residues, and coupling of iodotyrosyl residues

- TPO has binding sites for I-and tyrosine

- TPO uses H_2O_2 as the oxidant to activate I-to hypoiodate (OI-), the iodinating species Both reactions are catalyzed by TPO.

- Proteolysis of Tg With Release of T4 and T3

- T4and T3 are synthesized and stored within the Tg molecule

- Proteolysis is an essential step for releasing the hormones

- To liberate T4 and T3, Tg is resorbed into the follicular cells in the form of colloid droplets, which fuse with lysosomes to form phagolysosomes

- Tg is then hydrolyzed to T4 and T3, which are then secreted into the circulation. (Harold Elis, 2006)

Production of T4 and T3

- T4 is the primary secretory product of the thyroid gland, which is the only source of T4

- The thyroid secretes approximately 70-90 μg of T4 per day

- T3 is derived from 2 processes

  – The total daily production rate of T3 is about 15-30 μg

  – About 80% of circulating T3 comes from de iodination of T4 in peripheral tissues

  – About 20% comes from direct thyroid secretion. (Harold Elis, 2006)

T4: A Prohormone for T3:

- T4 is biologically inactive in target tissues until converted to T3

  – Activation occurs with 5'iodination of the outer ring of T4

- T3 then becomes the biologically active hormone responsible for the majority of thyroid hormone effects.

Sites of T4 Conversion:

- The liver is the major extra thyroidal T4conversion site for production of T3

- Some T4 to T3conversion also occurs in the kidney and other tissues. (Harold Elis, 2006)

T4 Disposition:

- Normal disposition of T4

  – About 41% is converted to T3

  – 38% is converted to reverse T3(rT3), which is metabolically inactive

- 21% is metabolized via other pathways, such as conjugation in the liver and excretion in the bile

- Normal circulating concentrations

  - T4 4.5-11 µg/dL

  - T3 60-180 ng/dL (~100-fold less than T4). (Harold Elis, 2006)

Carriers for Circulating Thyroid Hormones:

- More than 99% of circulating T4and T3is bound to plasma carrier proteins

  - Thyroxin-binding globulin (TBG), binds about 75%

  - Trans thyretin (TTR), also called thyroxin-binding pre albumin (TBPA), binds about 10%-15%

  - Albumin binds about 7%

  - High-density lipoproteins (HDL), binds about 3%

- Carrier proteins can be affected by physiologic changes, drugs, and disease.

- Free Hormone Concept

- Only unbound (free) hormone has metabolic activity and physiologic effects

  - Free hormone is a tiny percentage of total hormone in plasma (about 0.03% T4; 0.3% T3)

- Total hormone concentration

  - Normally is kept proportional to the concentration of carrier proteins

  - Is kept appropriate to maintain a constant free hormone level.

Changes in TBG Concentration Determine Binding and Influence T4and T3 Levels.

- Increased TBG:

  - Total serum T4and T3levels increase

  - Free T4(FT4), and free T3(FT3) concentrations remain unchanged

- Decreased TBG:

  - Total serum T4and T3levels decrease

  - FT4and FT3levels remain unchanged.

Drugs and Conditions That Increase Serum T4 and T3 Levels by Increasing TBG:

- Drugs that increase TBG–Oral contraceptives and other sources of estrogen–Methadone–Clofibrate–5-Fluorouracil–Heroin–Tamoxifen.

- Conditions that increase TBG–Pregnancy–Infectious/chronic active hepatitis–HIV infection–Biliary cirrhosis–Acute intermittent porphyria–Genetic factors.

Drugs and Conditions That Decrease Serum T4 and T3by Decreasing TBG Levels or Binding of Hormone to TBG:

- Drugs that decrease serum T4 and T3

  – Glucocorticoids / Androgens / L-Asparaginase / Salicylates / Mefenamic acid/ Anti-seizure medications, e.g., phenytoin, carbama-zepine/ Furosemide

- Conditions that decrease serum T4 and T3:

  – Genetic factors/Acute and chronic illness. (Harold Elis, 2006)

2-3-2 Physiology of the Kidneys:

2-3-2-1 Excretion of waste:

The primary function of the kidneys is the excretion of waste products resulting from protein metabolism and muscle contraction. The liver metabolizes dietary proteins to produce energy and produces toxic ammonia as a waste product. The liver is able to convert most of this ammonia into uric acid and urea, which are less toxic to the body. Meanwhile, the muscles of our body use creatine as an energy source and, in the process, produce the waste product creatinine. Ammonia, uric acid, urea, and creatinine all accumulate in the body over time and need to be removed from circulation to maintain homeostasis.

The glomerulus in the kidneys filter all four of these waste products out of the bloodstream, allowing us to excrete them out of our bodies in urine. Around 50% of the urea found in the blood is reabsorbed by the tubule cells of the nephron and returned to the blood supply. Urea in the blood helps to concentrate

other more toxic waste products in urine by maintaining the osmotic balance between urine and blood in the renal medulla. (John, 2006).

Filtration, Reabsorption, and Secretion:

- The kidneys filter blood as it passes through the capillaries that form the glomerulus. Blood pressure forces most of the blood plasma through the lining of the capillaries and into the glomerular capsule. Blood cells are too large to pass through the capillary lining and so remain within the capillaries along with some residual plasma. The filtered plasma, now known as tubular fluid, begins to flow out of the glomerular capsule and into the proximal convoluted tubule. (John, 2006).

- At the same time, the concentrated blood that remains inside the capillaries of the glomerulus moves into the efferent arterioles and on to the peritubular capillaries surrounding the proximal convoluted tubule. Epithelial cells lining the tubule actively reabsorb valuable molecules of glucose, amino acids, and ions from the filtrate and deposit them back into the blood. These cells also absorb any waste products remaining in the blood (such as ammonia and creatinine) and secrete these chemicals into the filtrate. While these solutes are being exchanged, osmotic pressure pushes water from the dilute, hypotonic filtrate back into the concentrated, hypertonic blood. (John, 2006).

- From the proximal convoluted tubule, the tubular fluid next enters the loop of Henle, where water and ions are reabsorbed. The descending limb of the loop of Henle is permeable to water and carries the filtrate deep into the medulla of the kidney. Tissues in the medulla surrounding the tubule contain a high concentration of ions and very little water compared to the filtrate. Osmotic pressure between the hypotonic filtrate and hypertonic medullary cells pushes water out of the filtrate and into the cells. The cells of the medulla return this water to the blood flowing through nearby capillaries. (John, 2006).

- Filtrate next passes through the ascending limb of the loop of Henle as it exits the medulla. The tissues surrounding the ascending limb are not permeable to water but are permeable to ions. The filtrate is very concentrated after passing through the descending limb, so ions easily diffuse out of the filtrate and into the cells lining the ascending limb. These ions are returned to the blood flowing through nearby capillaries. (John, 2006).

- Tubular fluid exiting the loop of Henle next passes through the distal convoluted tubule and the collecting duct of the nephron. These tubules continue to reabsorb small amounts of water and ions that are still left in the filtrate. The tissues surrounding the collecting duct actively absorb excess potassium and hydrogen ions from the nearby capillaries and secrete these excess ions as waste into the filtrate. (John, 2006).

- When filtrate reaches the end of the collecting duct, almost all of the valuable nutrients, ions, and water have been returned to the blood supply while waste products and a small amount of water are left to form urine. The urine exits the collecting duct and joins with urine from other collecting ducts in the renal pelvis. (John, 2006).
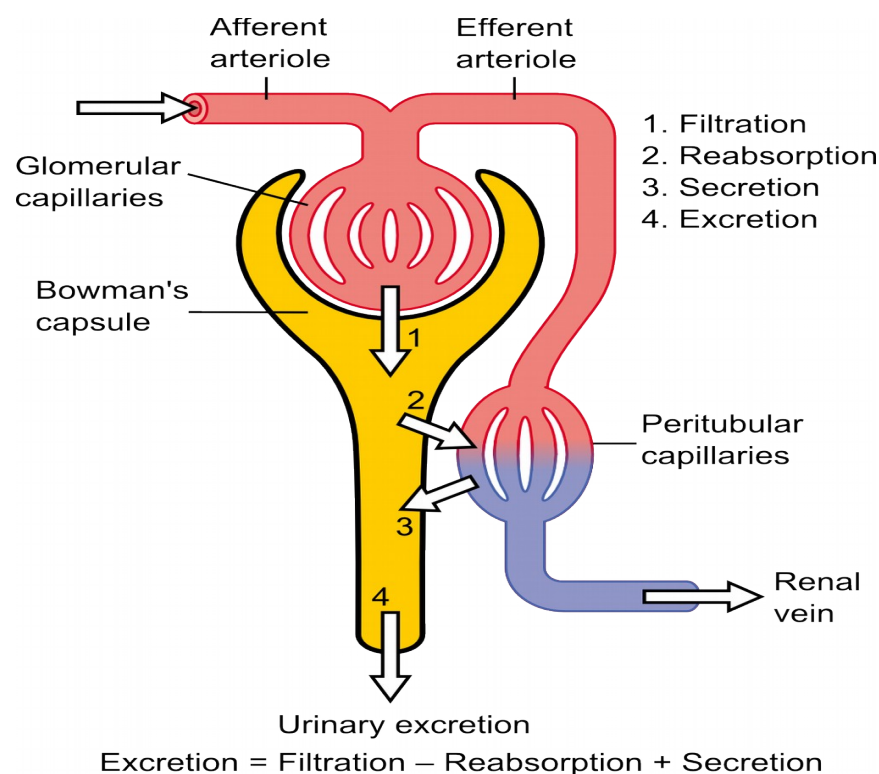

Excretion = Filtration − Reabsorption + Secretion

Figure (2-11) urinary excretion (Medical Physiology 11th edition, 2006)

2-3-2-2 Water homeostasis:

The kidneys are able to control the volume of water in the body by changing the reabsorption of water by the tubules of the nephron. Under normal conditions, the tubule cells of the nephron tubules reabsorb (via osmosis) nearly all of the water that is filtered into urine by the glomerulus. (John, 2006).

Water reabsorption leads to very concentrated urine and the conservation of water in the body. The hormones antidiuretic hormone (ADH) and aldosterone both increase the reabsorption of water until almost 100% of the water filtered by the nephron is returned to the blood. ADH stimulates the formation of water channel proteins in the collecting ducts of the nephrons that permit water to pass from urine into the tubule cells and on to the blood. Aldosterone functions by increasing the reabsorption of Na+ and Cl- ions, causing more water to move into the blood via osmosis. (John, 2006).

In situations where there is too much water present in the blood, our heart secretes the hormone atrial natriuretic peptide (ANP) in order to increase the excretion of Na+ and Cl- ions. Increased concentration of Na+ and Cl- in urine draws water into the urine via osmosis, increasing the volume of urine produced. (Harold Elis, 2006)

2-3-2-3 Acid/Base homeostasis:

The kidneys regulate the pH level of the blood by controlling the excretion of hydrogen ions (H+) and bicarbonate ions (HCO3-). Hydrogen ions accumulate when proteins are metabolized in the liver and when carbon dioxide in the blood reacts with water to form carbonic acid (H2CO3). Carbonic acid is a weak acid that partially dissociates in water to form hydrogen ions and bicarbonate ions. Both ions are filtered out of the blood in the glomerulus of the kidney, but the tubule cells lining the nephron selectively reabsorb bicarbonate ions while leaving hydrogen ions as a waste product in urine. The tubule cells may also actively secrete additional hydrogen ions into the urine when the blood becomes extremely acidic. (Harold Elis, 2006)

The reabsorbed bicarbonate ions enter the bloodstream where they can neutralize hydrogen ions by forming new molecules of carbonic acid. Carbonic acid passing through the capillaries of the lungs dissociates into carbon dioxide and water, allowing us to exhale the carbon dioxide. (Harold Elis, 2006)
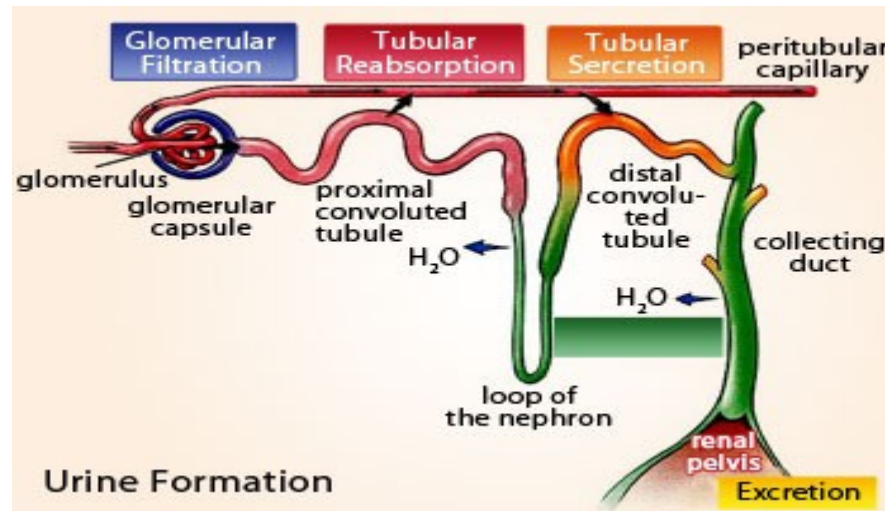

Figure (2-12) Urine formation (Medical Physiology 11th edition, 2006)

2-3-2-4 Electrolyte homeostasis:

The kidneys maintain the homeostasis of important electrolytes by controlling their excretion into urine.

Sodium (Na+): Sodium is a vital electrolyte for muscle function, neuron function, blood pressure regulation, and blood volume regulation. Over 99% of the sodium ions passing through the kidneys are reabsorbed into the blood from tubular filtrate. Most of the reabsorption of sodium takes place in the proximal convoluted tubule and ascending loop of Henle. (John, 2006)

Potassium (K+): Just like sodium, potassium is a vital electrolyte for muscle function, neuron function, and blood volume regulation. Unlike sodium, however, only about 60 to 80% of the potassium ions passing through the kidneys are reabsorbed. Most of the reabsorption of potassium occurs in the proximal convoluted tubule and ascending loop of Henle. (John, 2006)

Chloride (Cl-): Chloride is the most important anion (negatively charged ion) in the body. Chloride is vital to the regulation of factors such as pH and cellular fluid balance and helps to establish the electrical potential of neurons and

muscle cells. The proximal convoluted tubule and ascending loop of Henle reabsorb about 90% of the chloride ions filtered by the kidneys. Calcium (Ca2+): Calcium is not only one of the most important minerals in the body that composes the bones and teeth, but is also a vital electrolyte. Functioning as an electrolyte, calcium is essential for the contraction of muscle tissue, the release of neurotransmitters by neurons, and the stimulation of cardiac muscle tissue in the heart. The proximal convoluted tubule and the ascending loop of Henle reabsorb most of the calcium in tubular filtrate into the blood. Parathyroid hormone increases the reabsorption of calcium in the kidneys when blood calcium levels become too low. (John, 2006)

Magnesium (Mg2+): Magnesium ion is an essential electrolyte for the proper function of enzymes that work with phosphate compounds like ATP, DNA, and RNA. The proximal convoluted tubule and loop of Henle reabsorb most of the magnesium that passes through the kidney. (Harold Elis, 2006)
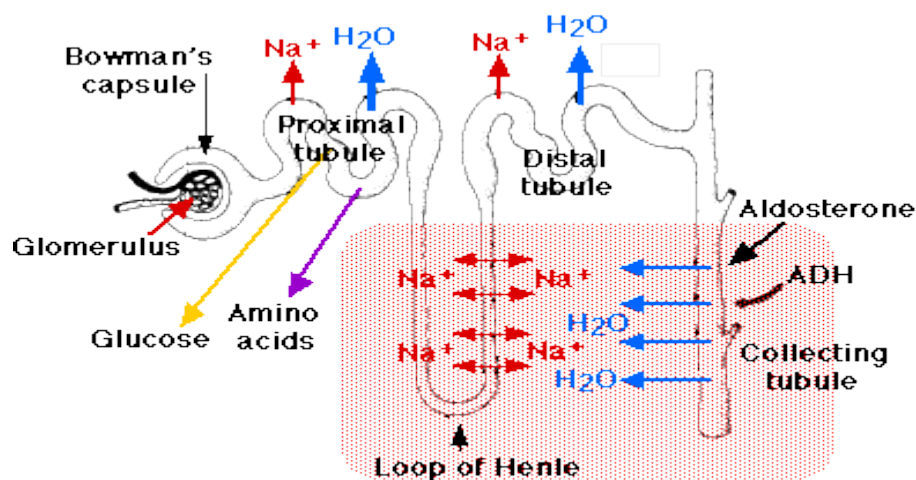

Figure (2-13) Electrolyte homeostasis (Medical Physiology 11th edition, 2006)

2-3-2-5 Blood pressure homeostasis:

The kidneys help to control blood pressure in the body by regulating the excretion of sodium ions and water and by producing the enzyme renin. Because blood is mostly made of water, an increased volume of water in the body results in an increase in the volume of blood in the blood vessels. Increased blood

volume means that the heart has to pump harder than usual to push blood into vessels that are crowded with excess blood. Thus, increased blood volume leads to increased blood pressure. On the other hand, when the body is dehydrated, the volume of blood and blood pressure decrease. (Harold Elis, 2006)

The kidneys are able to control blood pressure by either reabsorbing water to maintain blood pressure or by allowing more water than usual to be excreted into urine and thus reduce blood volume and pressure. Sodium ions in the body help to manage the body's osmotic pressure by drawing water towards areas of high sodium concentration. To lower blood pressure, the kidneys can excrete extra sodium ions that draw water out of the body with them. Conversely, the kidneys may reabsorb additional sodium ions to help retain water in the body. (Harold Elis, 2006)

Finally, the kidneys produce the enzyme renin to prevent the body's blood pressure from becoming too low. The kidneys rely on a certain amount of blood pressure to force blood plasma through the capillaries in the glomerulus. If blood pressure becomes too low, cells of the kidneys release renin into the blood. Renin starts a complex process that results in the release of the hormone aldosterone by the adrenal glands. Aldosterone stimulates the cells of the kidney to increase their reabsorption of sodium and water to maintain blood volume and pressure. (Harold Elis, 2006)

2-3-2-6 Hormones:

The kidneys maintain a small but important endocrine function by producing the hormones calcitriol and erythropoietin.

Calcitriol is the active form of vitamin D in the body. Tubule cells of the proximal convoluted tubule produce calcitriol from inactive vitamin D molecules. At that point, calcitriol travels from the kidneys through the bloodstream to the intestines, where it increases the absorption of calcium from food in the intestinal lumen. (Harold Elis, 2006)

Erythropoietin (EPO) is a hormone produced by cells of the peritubular capillaries in response to hypoxia (a low level of oxygen in the blood). EPO

stimulates the cells of red [bone marrow](#) to increase their output of red blood cells. Oxygen levels in the blood increase as more red blood cells mature and enter the bloodstream. Once oxygen levels return to normal, the cells of the peritubular capillaries stop producing EPO. Several hormones produced elsewhere in the body help to control the function of the kidneys. (John, 2006)

Antidiuretic hormone (ADH), also known as vasopressin, is a hormone produced by neurosecretory cells in the brain's hypothalamus. These cells extend into the posterior pituitary, which stores and releases ADH. ADH production is stimulated by a decrease in blood volume and increased blood osmolarity. ADH helps the body retain water by increasing the number of water channels in the cells of the collecting ducts of the kidneys. These water channels allow water remaining in urine to be reabsorbed into the blood, resulting in extremely concentrated urine. (John, 2006)

Angiotensin II is a hormone made in the liver and activated by the enzymes renin and angiotensin-converting enzyme. Once activated, angiotensin II increases the reabsorption of sodium and chloride ions in the proximal convoluted tubule, leading to an increased reabsorption of water as well.

Aldosterone is a hormone produced in the adrenal cortex in response to Angiotensin II. Aldosterone binds to target cells in the walls of the nephron's collecting ducts. These cells reabsorb additional sodium and chloride ions that would have been excreted as urine. The target cells also remove potassium ions from the blood and excrete it into urine. (John, 2006)

Atrial natriuretic peptide (ANP) is a hormone produced by cardiac muscle cells in the atria of the heart. These cells produce ANP in response to high levels of sodium in the blood or increased blood pressure. In the kidneys, ANP increases the glomerular filtration rate so that more blood plasma is forced into the glomerular capsule and into the renal tubules. ANP also removes some solutes from the cells of the renal medulla, making the loop of Henle less efficient in reabsorbing water and ions from the filtrate. The net result of ANP is that more

sodium and water end up being excreted into urine, blood volume decreases, and blood pressure decreases as well. (Harold Elis, 2006)

2-4 Thyroid Pathology:

2-4-1 Thyroid cancer:

Most people with thyroid cancer have a thyroid nodule that is not causing any symptoms. If you have a thyroid nodule, there is a small chance it may be thyroid cancer. To tell if the nodule is cancerous, your doctor will have to do certain tests. A few people with thyroid cancer may have symptoms. If the cancer is big enough, it may cause swelling you can see in the neck. It may also cause pain or problems swallowing. Some people get a hoarse voice. (GB Health watch. (Kaptein EM, 1986)

Thyroid cancer is rare compared with other types of cancer. It is more common in people who: Have a history of exposure of the thyroid to radiation (but not routine X-ray exposure, as in dental X-rays or mammograms) Have a family history of thyroid cancer. (Kaptein EM, 1986)

2-4-2 Goiter:

A goiter is an abnormally enlarged thyroid gland. Causes of goiter include:
Iodine deficiency. Iodine is a mineral that your thyroid uses for making thyroid hormones. Not getting enough iodine in your food and water can cause your thyroid to get bigger. This cause of goiter is uncommon in the United States because iodine is added to table salt. Hashimoto's disease, Graves' disease, Thyroid nodules, Thyroiditis and Thyroid cancer. Usually, the only symptom of a goiter is a swelling in your neck. But a very large or advanced goiter can cause a tight feeling in your throat, coughing, or problems swallowing or breathing. (Kaptein EM, 1986)

Having a goiter does not always mean that your thyroid is not making the right amount of hormones. Depending on the cause of your goiter, your thyroid could be making too much, not enough, or the right amount of hormones. (GB Health watch website). (Kaptein EM, 1986)

2-4-3 Effects of thyroid dysfunction on the kidney

Thyroid dysfunction causes significant changes in kidney function. Both hypothyroidism and hyperthyroidism affect renal blood flow, GFR, tubular function, electrolytes homeostasis, electrolyte pump functions, and kidney structure. (P Iglesias *etal*, 2006)

2-4-3-1 Hypothyroidism:

The most common kidney derangements associated to hypothyroidism are: elevation of serum creatinine levels, reduction in GFR and renal plasma flow (RPF), and disruption of the capacity to excrete free water and hypo natremia. These alterations may be absent in patients with central hypothyroidism due to the fact that this kind of thyroid hypo function is often accompanied by other pituitary hormone deficiencies that might affect directly or indirectly the kidney function. (P Iglesias *etal*, 2006)

Primary hypothyroidism is associated with a reversible elevation of serum creatinine in both adults and children. This increase is observed in more than half (∼55%) of adults with hypothyroidism. Moreover, some authors have reported an elevation of serum creatinine associated with subclinical hypothyroidism. (P Iglesias *etal*, 2006)

Primary hypothyroidism is associated with a reduction of GFR and RPF that are normalized following levothyroxine administration. Similarly, normalization of circulating TH concentrations with replacement therapy in hypothyroid patients with chronic kidney disease (CKD) can significantly improve GFR. However, it has recently been reported that kidney function recovers slowly in hypothyroid children, and sometimes partially, after the introduction of replacement with levothyroxin. The long-term clinical implications of these findings are unknown. Hypothyroidism-associated kidney dysfunction seems to be more related with the decline in thyroid hormone levels rather than with thyroid autoimmunity. Among the mechanisms involved in hypothyroidism-associated kidney derangements are direct effects of TH on the cardiovascular system (increased peripheral resistance and reduction of myocardial contractility and stroke

volume) and metabolism (hyperlipidemia), and indirect effects through paracrine or endocrine mediators, such as insulin-like growth factor type 1 (IGF-1) and vascular endothelial growth factor . (P Iglesias *etal*, 2006)

Hypo natremia is the commonest electrolyte derangement in hypothyroid patients. Hypo natremia appears in 45% of hypothyroid patients who have elevated serum creatinine, but in less than a quarter (21%) of those with normal creatinine levels. It is mainly due to a reduction in GFR causing diminished water delivery to the distal tubular segments. This becomes evident after water load, although ADH may be appropriately suppressed. Other possible mechanism of hypothyroidism induced hyponatremia is an inappropriate ADH secretion syndrome (SIADH)-like disorder. *(*Kaptein EM, 1986*)*

2-4-3-2 Thyrotoxicosis:

Thyrotoxicosis is characterized by an increase in RPF and GFR resulting in a reduction of serum creatinine levels. These changes are normalized after the control of thyroid function with appropriate treatment. Hyperthyroidism may be linked to a decrease in total body water and exchangeable K. By contrast, the amount of exchangeable Na tends to increase. However, serum concentrations of Na, K, and Cl are normal. These alterations are typical of endogenous hyperthyroidism and exogenous thyrotoxicosis. However, central hyperthyroidism may not be accompanied by these changes when it is associated with other pituitary disorders. The reduction of serum creatinine has also been reported in subclinical hyperthyroidism. However, changes in water and electrolyte metabolism have not been reported by other authors.

Hemodynamic changes, i.e., increase in systolic volume, heart rate, and cardiac output coupled with a reduction of peripheral vascular resistance, also participate in alteration in renal function reported in patients with hyperthyroidism. These changes are due to the increased circulating demands as a result of hyper metabolism and the need to dissipate excess heat associated with hyperthyroidism. *(*Kaptein EM, 1986*)*

2-4-4 Kidney disease associated to thyroid dysfunction:

The different types of kidney diseases can be associated with various disorders of thyroid function. (P Iglesias *etal*, 2006)

2-4-4-1 Glomerular disease:

Thyroid disease may be linked to different forms of glomerulonephritis. Both hypothyroidism and hyperthyroidism can coincide with different forms of glomerular disease. The more frequent form is membranous glomerulopathy associated with nephrotic syndrome (NS). Thyroid dysfunction has been reported to be associated with IgA glomerulonephritis, mesangiocapillary or membrane oproliferative glomerulonephritis, and minimal change glomerulonephritis. (P Iglesias *etal*, 2006)

Several mechanisms have been involved in these associations. Proteinuria may promote the development of primary hypothyroidism, and the immune activation of the thyroid or kidney disorders could induce the formation of immune complexes. The presence of immune complexes is common in patients with thyroid disease. In a study performed in 171 patients with thyroid disease, the presence of immune complexes was detected in 26% of patients in comparison with 8% of the control subjects. This percentage increased to 33–55% in patients with an autoimmune process and was correlated with the presence of thyroid peroxidase antibodies, but not with the titer of these antibodies. Also, immune complexes deposits in the basement membrane of thyroid follicular epithelium and the glomeruli have been reported in patients with Hashimoto's thyroiditis and membranous glomerulopathy. Therefore, several data support the autoimmune pathogenesis for this association: i) the association of kidney and thyroid diseases of autoimmune origin, ii) its association with other autoimmune diseases such as type 1 diabetes, and iii) the presence of deposits of immune globulins and thyroglobulin in the glomeruli of some patients. Although autoimmune thyroid disease has occasionally been reported in patients with glomerulonephritis, no causal relationship between the two disorders has been proved so far. Glomerular disease in general is associated

and occasionally caused by autoimmune disease (e.g. lupus nephritis, anti-neutrophil cytoplasmic antibodies (ANCA) associated vacuities) that can be associated to autoimmune thyroid disease. (P Iglesias *etal*, 2006)

2-4-4-2 Tubular disease:

Although less frequently than glomerular disease, tubular or tubule interstitial damage has also been reported to be associated with thyroid dysfunction. Isolated cases of hyperthyroidism have been reported in association with tubule interstitial nephritis and uveitis, a self-limited syndrome of unknown etiology that responds to glucocorticoids. In these cases, the etiology of hyperthyroidism was not Graves' disease, but rather a destructive thyroiditis with the absence of thyroid autoimmunity, low uptake in thyroid scintigraphy, and adequate response to steroid therapy. Tubulo interstitial nephritis and hyperthyroidism has been reported to be associated in patients under treatment with rifampicin. (P Iglesias *etal*, 2006)

2-4-4-3 Nephrotic syndrome:

NS is associated with changes in serum TH levels. Urinary losses of binding proteins, such as thyroxin binding globulin (TBG), trans thyretin or pre-albumin, albumin, and TH binded to them, result in a reduction in serum total thyroxin (T_4) and, sometimes, in total T_3 levels. These hormonal changes are related both to the degree of proteinuria and to serum albumin levels. However, patients often remain euthyroid, because free T_4 and T_3 levels are usually normal. This suggests that thyroid is able to compensate for hormonal urinary losses keeping the patient euthyroid. However, in patients with low thyroid reserve overt hypothyroidism can develop. Similarly, NS may increase the exogenous levothyroxine needs in patients with hypothyroidism. (P Iglesias *etal*, 2006)

Primary hypothyroidism linked to congenital NS (CNS) has been reported. TH urinary loss associated with the intrauterine massive proteinuria stimulates the hypothalamus–pituitary–thyroid axis increasing serum thyrotropin (TSH) concentrations. Other involved factors are malnutrition and iodine depletion. However, the main cause is TH urinary losses, since it was observed that

bilateral nephrectomy followed by extra renal purification treatment reverses completely the CNS associated hypothyroidism and permits the withdrawal of hormonal treatment with levothyroxine. Some authors recommend treatment with levothyroxine supplementation in children with CNS as it facilitates their normal development. (P Iglesias *etal*, 2006)

2-4-4-4 Acute kidney injury:

Acute kidney injury (AKI) is associated with abnormalities in thyroid function tests similar to those found in euthyroid sick syndrome (ESS). Contrary to the usual form of the ESS, patients with AKI may not exhibit an elevation or reverse (r) T3 levels. The hypothyroidism-associated rise in serum creatinine may be of relevance in patients with thyroid carcinoma in which the withdrawal of levothyroxine treatment for total body scan preparation can lead to accumulation of drugs whose metabolism and elimination is primarily renal. Furthermore, the development of AKI has been associated with rhabdomyolysis in patients with primary or secondary hypothyroidism treated or not with statins. (P Iglesias *etal*, 2006)

2-4-4-5 Chronic kidney disease:

CKD affects both hypothalamus–pituitary–thyroid axis and TH peripheral metabolism. Uremia influences the function and size of the thyroid. Uraemic patients have an increased thyroid volume compared with subjects with normal renal function and a higher prevalence of goiter, mainly in women. Also, thyroid nodules and thyroid carcinoma are more common in uraemic patients than in the general population. (P Iglesias *etal*, 2006)
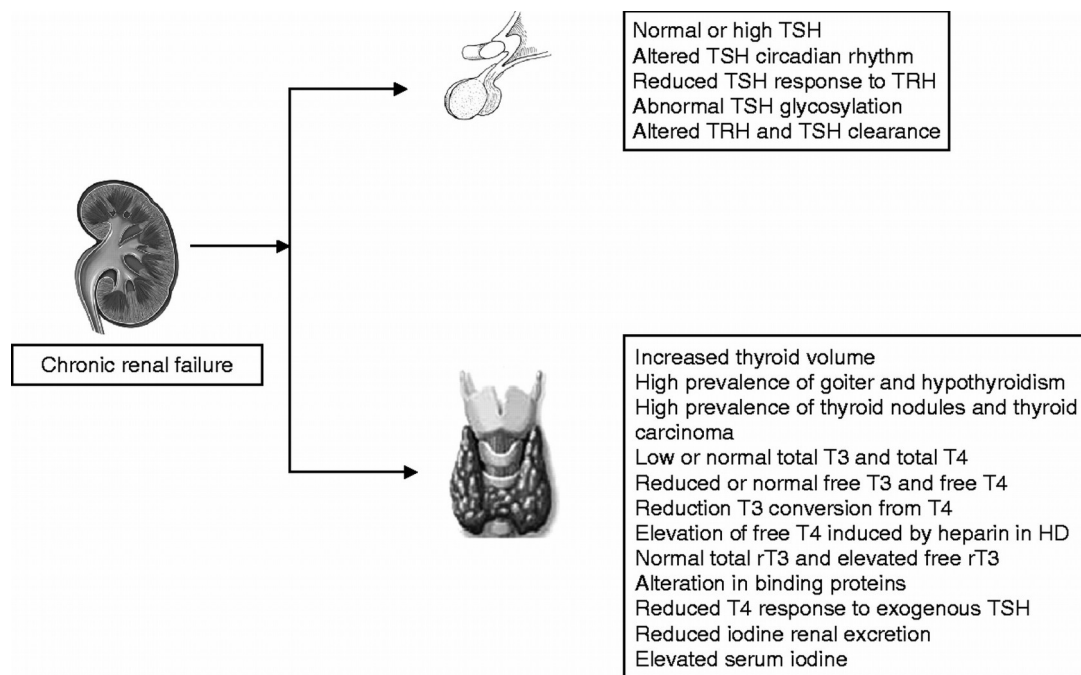
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┌─────────────────────────────────────────┐
│ Normal or high TSH                      │
│ Altered TSH circadian rhythm            │
│ Reduced TSH response to TRH             │
│ Abnormal TSH glycosylation              │
│ Altered TRH and TSH clearance           │
└─────────────────────────────────────────┘
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Chronic renal failure

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┌─────────────────────────────────────────────────┐
│ Increased thyroid volume                         │
│ High prevalence of goiter and hypothyroidism     │
│ High prevalence of thyroid nodules and thyroid   │
│ carcinoma                                        │
│ Low or normal total T3 and total T4              │
│ Reduced or normal free T3 and free T4            │
│ Reduction T3 conversion from T4                  │
│ Elevation of free T4 induced by heparin in HD    │
│ Normal total rT3 and elevated free rT3           │
│ Alteration in binding proteins                   │
│ Reduced T4 response to exogenous TSH             │
│ Reduced iodine renal excretion                   │
│ Elevated serum iodine                            │
└─────────────────────────────────────────────────┘
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Figure (2-14) effects both hypothalamus-pituitary-thyroid axis and TH peripheral metabolism (Medical Physiology 11th edition, 2006)

Serum TSH concentrations are usually normal or elevated in CKD, but its response to its releasing hormone (TRH) is generally low. These findings suggest the presence of intra thyroidal and pituitary disturbances associated with uremia. Also, both TSH circadian rhythm and TSH glycosylation are altered in CKD. The latter may compromise TSH bioactivity. Free and total T_3 and T_4 concentrations are usually normal or low in patients with CKD. The reduction in T_3 levels (low T_3syndrome) is the most frequently observed thyroid alteration in these patients. This reduction in T_3 concentrations has been linked to a decrease in the peripheral synthesis of T_3 from T_4. Chronic metabolic acidosis associated with the CKD may contribute in this effect. Although free and total T_4 concentrations may be normal or slightly reduced, sometimes free T_4 may be high due to the effect of heparin used in anticoagulation during hemodialysis (HD), which inhibits T_4 binding, to its binding proteins. In CKD patients, the ESS is characterized by the absence of total rT_3 rising, a typical feature in other patients with non-thyroidal disease. Despite the fact that the total rT_3 clearance in CKD patients is diminished, there is a redistribution of rT_3 from the vascular

to the extravascular space and an increase in rT_3 cellular uptake. However, free rT_3 concentrations are high due to a reduction in its renal clearance. (John, 2006) CKD is associated with a higher prevalence of primary hypothyroidism, both overt and subclinical, but not with hyperthyroidism. In fact, the prevalence of primary hypothyroidism, mainly in the subclinical form, increases as GFR decreases. A recent study has shown a prevalence of subclinical hypothyroidism of 7% in patients with estimated GFR $\geq$90 ml/min per 1.73 m^2 that increased to 17.9% in subjects with GFR <60 ml/min per 1.73 m^2. The prevalence of hypothyroidism is higher in women and is associated with an increased frequency of high titers of anti-thyroid antibodies. (P Iglesias *etal*, 2006)

A greater prevalence of non-autoimmune primary hypothyroidism has been reported in patients with advanced diabetic nephropathy under conservative treatment in comparison with non-diabetic patients with nephropathy. It is possible that these patients had impaired renal handling of iodine resulting in an elevation of serum iodine levels with a prolongation of the Wolff–Chaikoff effect. The prevalence of hyperthyroidism in CKD is similar to that found in general population (∼1%), in areas with inadequate intake of iodine. On the other hand, uraemic patients undergoing dialysis with hyperthyroidism due to either Graves' disease or toxic multi nodular goiter can be adequately treated with therapeutic doses of [131]I. Moreover, hyperthyroidism has been considered as one of the many causes of anemia resistant to recombinant human erythropoietin (rh-EPO) in CKD patients on HD with an adequate response to anti thyroid treatment. (P Iglesias *etal*, 2006)

The kidney contributes to the iodine clearance primarily through glomerular filtration. Serum iodine concentrations are high in CKD but are not correlated with the degree of kidney failure. This iodine excess has been linked to increased prevalence of goiter and hypothyroidism reported in CKD. A high exposure to iodine facilitates the development of hypothyroidism in CKD patients. Some authors have reported that a restriction of dietary iodine in

uraemic patients on HD can correct the hypothyroidism avoiding the need for hormone replacement with levothyroxine. (P Iglesias *etal*, 2006)

2-5 Ultrasound:

Is an oscillating sound pressure wave with a frequency greater than the upper limit of the human hearing range. Ultrasound is thus not separated from 'normal' (audible) sound by differences in physical properties, only by the fact that humans cannot hear it. Although this limit varies from person to person, it is approximately 20 kilohertz (20,000 hertz) in healthy, young adults. Ultrasound devices operate with frequencies from 20 kHz up to several gigahertz.

Ultrasound is used in many different fields. Ultrasonic devices are used to detect objects and measure distances. Ultrasonic imaging (sonography) is used in both veterinary medicine and human medicine. In the nondestructive testing of products and structures, ultrasound is used to detect invisible flaws. Industrially, ultrasound is used for cleaning and for mixing, and to accelerate chemical processes. Animals such as bats and porpoises use ultrasound for locating prey and obstacles.

2-5-1 The parts of an ultrasound machine

- **Central Processing Unit (CPU)**
  The CPU is the hub of an ultrasound machine. The CPU is like a computer that contains the microprocessor, memory, amplifiers and power supplies for the microprocessor and transducer probe. The transducer receives electrical currents from the CPU and sends electrical pulses that are created by returning echoes. The CPU does all of the calculations to produce an image on the monitor, and also stores the processed information on a disk.
- **Transducer Pulse Controls**
  The operator, called the ultrasonographer, uses the transducer pulse controls to set and change the frequency and duration of the ultrasound pulses. The transducer pulse controls also allow for scanning the mode of the machine.

Commands from the operator are changed into fluctuating electrical currents that are applied to the piezoelectric (PZ) crystals in the transducer probe.

- **Display**

  The display turns processed data from the CPU into an image. This image can be either in black-and-white or color, depending upon the model of the ultrasound machine.

- **Keyboard/Cursor**

  Ultrasound machines have a keyboard and a cursor. The keyboard allows the operator to add notes and to take measurements of the image.

- **Disk Storage**

  The processed data and/or images can be stored on disks. These disks can be hard disks, floppy disks, compact disks (CDs), or digital video disks (DVDs). Most of the time, ultrasound scans are filled on floppy disks and stored with the patient's medical records.

- **Printers**

  Most ultrasound machines have printers which are thermal. These can be used to capture a printed picture of the image from the monitor.

2-5-2 Renal Scanning Protocol:

- **Preparation of the patient:**

  The patient should take nothing by mouth for 8 hours preceding the examination. If fluid is essential to prevent dehydration, only water should be given. Infants should be given nothing by mouth for 3hours preceding the examination.

- **Position of the patient:**

  Lie comfortable on his/her back (supine). Head may be rest in a small pillow. To visualize Rt. and Lt. Kidneys apply gel to the Rt. & Lt. upper abdomen consequently. Scanning is best performed with the patient holding the breath in.

- **Choice of transducer:**

  - Use 3.5 MHz for adults. Curvilinear probe. Use 5 MHz for children and thin adults. Linear probe.
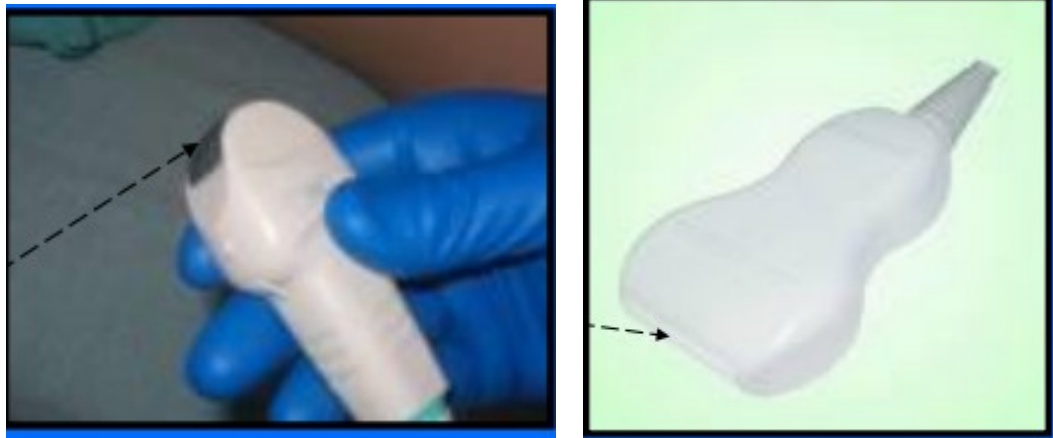

Figure (2-15) curvilinear probe and linear probe

- **Setting the correct gain**:

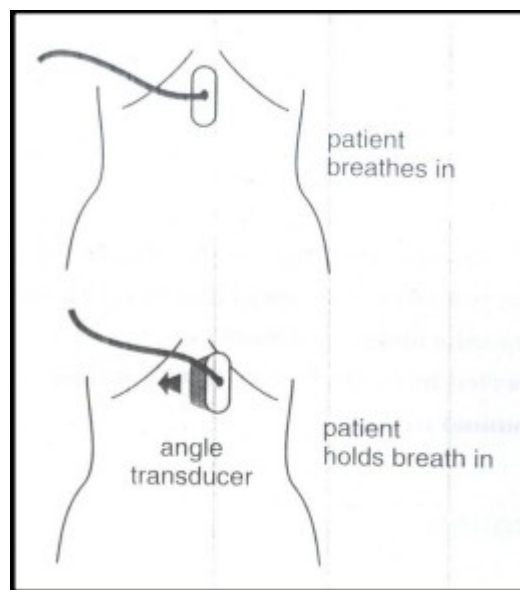 - Start by placing the transducer longitudinal central

Figure (2-16) Locations of the probe

And at the top of the abdomen (the xiphoid angle). Ask the patient to take a deep breath and hold it in. Angle the transducer beam towards the right side of the patient to image the liver.

Adjust the gain settings so that the image has normal homogeneity and texture. It should be possible to recognize the strongly reflecting lines of the diaphragm next to the posterior part of the liver.

Longitudinal scan: the normal liver and diaphragm.
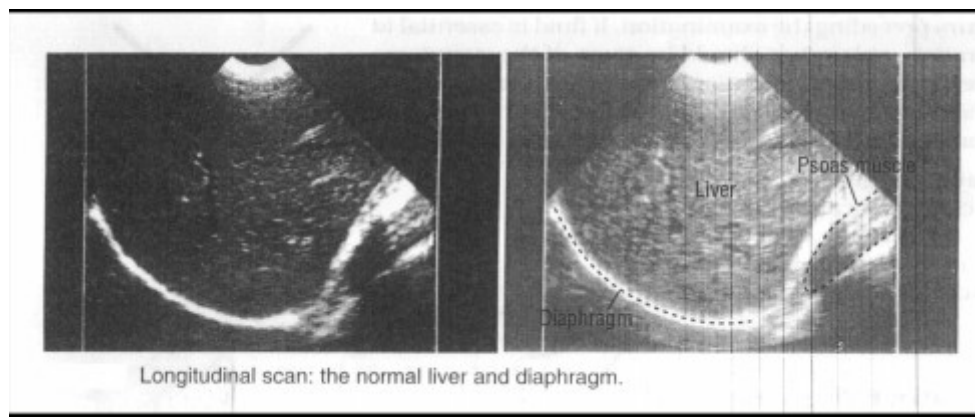
Figure (2-17) longitudinal scan: the normal liver and diaphragm for setting the gain (Manual ultrasound)

For the right kidney: Supine (as basic). Left posterior oblique; Left lateral decubitus and; Prone   as needed. For the left kidney. Right lateral decubitus (as basic) and; Prone as needed.

2-5-3 Sonographic Appearance:

Because of fat, the renal sinus is echogenic with variable contour. Parenchyma surrounds the sinus. The infundibula and renal

pelvis are not seen if collapsed; otherwise, they

appear anechoic. The cortex is homogenous. The medullary pyramids

appear as triangular, round hypoechoic areas. The ureters are not normally

seen.

Longitudinal Survey: Sagittal plane, anterior approach (Rt. Kidney). Begin with transducer perpendicular, just inferior to the most lateral edge of the right costal margin.
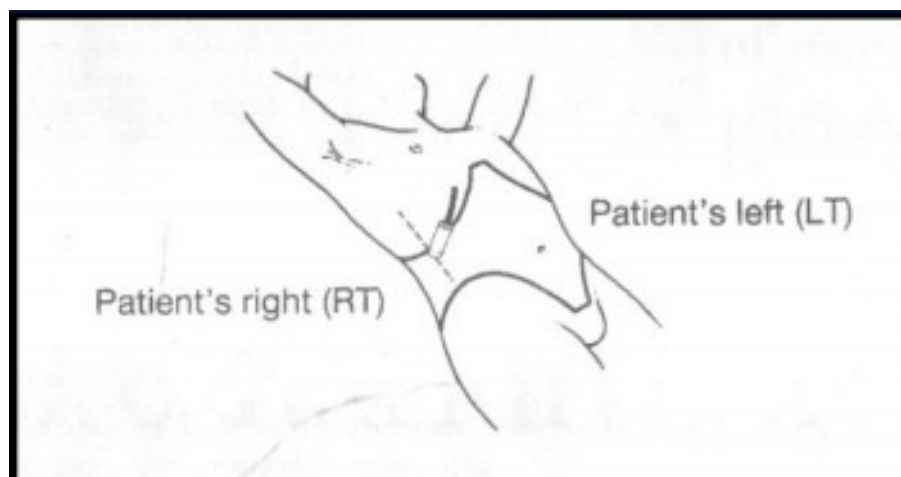

38

Figure (2-19) Patient Position

Transverse Survey: Transverse plane, anterior approach (Rt. Kidney). Still in sagittal scanning plane, locate the long axis of the right kidney, rotate the transducer 90 degrees into the transverse scanning plane and traverse the kidney.

2-6 Previous study:

Zahoor Ahmed (etal) said the study was basically designed to see the normal and healthy individuals to investigate the effect of T_3.T_4 on the normal hormonal levels are difference for difference gender. Males were found to be 24.5% and females 75.5%. The age distribution of study subject group divided into some different age. (2010)

P Iglesias (etal) said: thyroid dyes function caused significant change in kidney function structure and size both hypothyroidism and hyperthyroidism affect renal morphology the most renal kidney derangements associated to hypothyroidism. (2009)

Anjali (etal) said: difference drugs used in thyroid disease may have adverse effect on the kidney. Hypothyroidisms induce by thionamides. Can cause kidney fairly. Thionamides can affect kidney function by differ immunological mechanisms leading to development of different types of glomerulonephritis. (2009)

Laura H.Mariani (etal) said: thyroid hormones affect kidney size weight and structure histological studies the effects of thyroid on cortical and outer medullary tubular segment and medullary thick ascending limb. These change invariable reuser with thyroid hormones replacement in ultrasound examination for patient reveal alteration in size (small) and increase in texture. (2011)

Thyroid hormones play a very important role regulating metabolism, development, protein synthesis, and influencing other hormone functions. The two main hormones produced by the thyroid are triiodothyronine (T3) and thyroxine (T4). These hormones can also have significant impact on kidney

disease so it is important to consider the physiological association of thyroid dysfunction in relation to chronic kidney disease (CKD). CKD has been known to affect the pituitary-thyroid axis and the peripheral metabolism of thyroid hormones. Low T3 levels are the most common laboratory finding followed by subclinical hypothyroidism in CKD patients. Hyperthyroidism is usually not associated with CKD but has been known to accelerate it. One of the most important links between thyroid disorders and CKD is uremia. Patients who are appropriately treated for thyroid disease have a less chance of developing renal dysfunction. Clinicians need to be very careful in treating patients with low T3 levels who also have an elevation in TSH, as this can lead to a negative nitrogen balance. Thus, clinicians should be well educated on the role of thyroid hormones in relation to CKD so that proper treatment can be delivered to the patient.

CHAPTER THREE

Materials and Methods

3- Methodology:

3-1 Types of the study:

Descriptive analysis observation study.

3-2 population of the study:

Male and female from different part of the Sudan with different age, Selected randomly from the out patients whom they are with thyroid hormone Defect.

3-3 Sample size:

50 patients with thyroid hormones disorders.

3-4 Data source:

Patient's files.

Internet.

Previous research.

3-5 Data collection:

Old data will be put in sheet. Pt whom sent for ultrasound are computed by medical. SPSS Version 20 used for analysis.

3-6 Sample:

Sudan nationality, both sex, different age, different part of the Sudan and apparently suffering from thyroid hormone defect.

3-7 Equipment used:

Thyroid hormone lab result and ultrasound machine.

3-8 Area of the study:

Department of ultrasound in military hospital N.M department and private clinic.

3-9 Study period:

Approximately 3-6 month.

3-10 Study design:

Cross sectional-descriptive- observation study.

3-11 Material:

Random sample of 50 patients who were clinically has thyroid hormone disorder, undergo ultrasound examination to the kidney to defect the effects of thyroid hormones on the kidney, their average age range between (4-66) years, 43 patients are females and 7 are males.

3-12 Machine used:

The sinologist who did ultrasound examination used different types of ultrasound machine in different hospitals and clinic, e.g. Toshiba 3.5-7 MHZ probe in Military Hospital and Aloka in private clinic.

3-13 Data collections:

The data collected by using data collecting sheet, a random sample of 50 patients with thyroid hormones disorders was examined. (Thyroid hormones normal value T3 normal range is1.0 – 3.3 µg/dL. T4 normal range is 55 - 177 µg/dL.) The data collecting sheet was designed to cover the gender of patients, age of pt., Thyroid hormones, the duration of the thyroid hormones defect, the weight of the patient, and the monograph finding of the kidney

3-14 Technique:

3-14-1 patient preparation:

Pt. should be hydrated.

3-14-2 patient's position:

Patient lie supine.

3-14-3 Technique:

The kidneys are scanned using high resolution electrical focused sector or higher array real times. Using 3-3.5 MHZ transducer. We examined the Rt a Lt kidney using coronal, sagittal, transverse plane with patients supine after applying the gel some patient with gaseous abdomen we use the prone position to see the outline of the kidney. Sometimes we use clock protocol.

Sagittal plane: approximately 1 cm interval relative cortical and medullary echogenicity should be noted and compare to the echogenicity of the linear and the previous studies of the kidney.

3-14-4 Data analysis:

The data analyzed using computer SPSS program Version 20 to represent frequencies tables, graphs, cross-tabulations and correlations.

Results

Table 4-1:

Study group of age distributions

Age	Frequency	Percent
4-10 Yrs	2	40
11-20 Yrs	1	2.0
21-30 Yrs	19	38.0
31-40 Yrs	10.	20.0
41-50 Yrs	12	24.0
51-60 Yrs	4	8.0
61-70 Yrs	2	4.0
Total	50	100.0
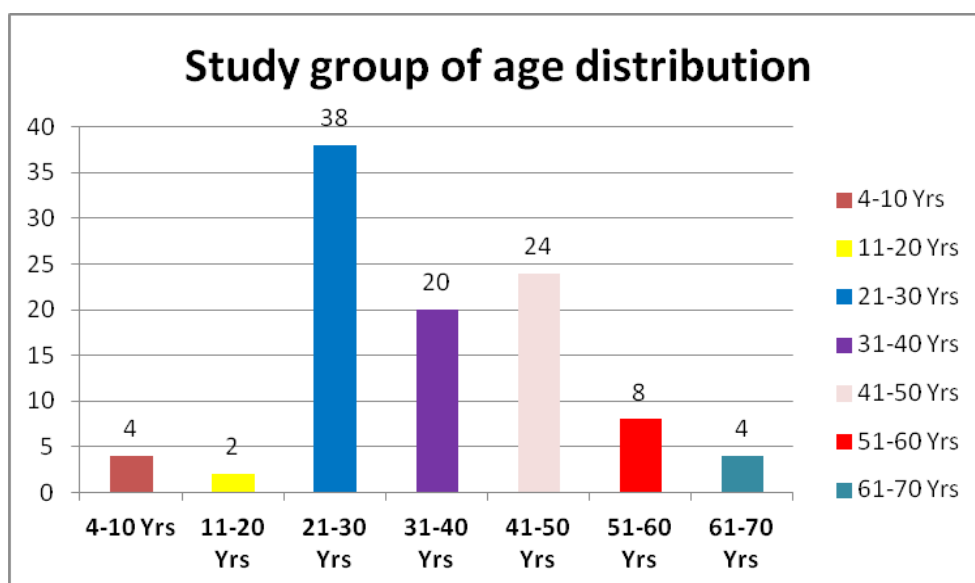
Figure 4-1: showing the study group of age

Table 4-2:

Study group of Sex distribution

Sex	No of patients	Percentage
Male	7	14
Female	43	86
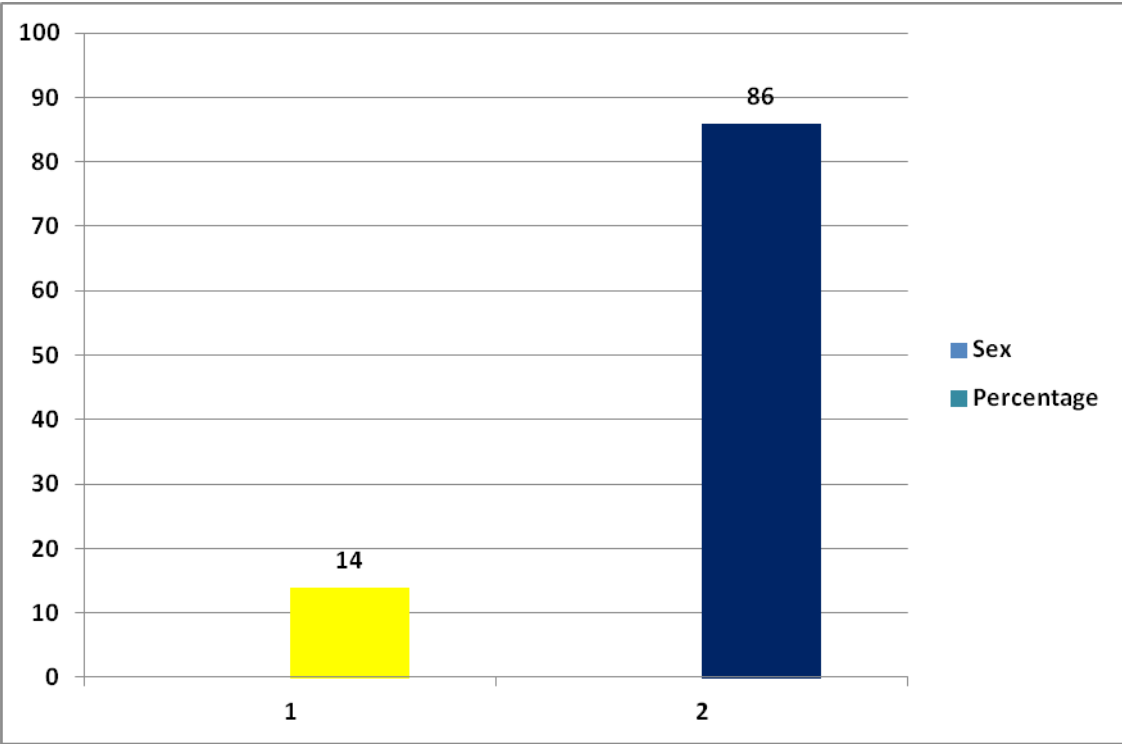
Figure 4-2: showing the Study group of Sex distribution

Table 4-3:

Study group of duration of thyroid hormone disorder distribution:

Duration	Frequency	Percent
One month	3	6.0
3 months	16	32.0
6 Months	5	10.0
1 Yr	8	16.0
2 Yrs	5	10.0
3 Yrs	2	4.0
4 Yrs	1	2.0
5 Yrs	2	4.0
7 Yrs	3	6.0
10 Yrs	3	6.0
12 Yrs	1	2.0
13 Yrs	1	2.0
Total	50	100.0
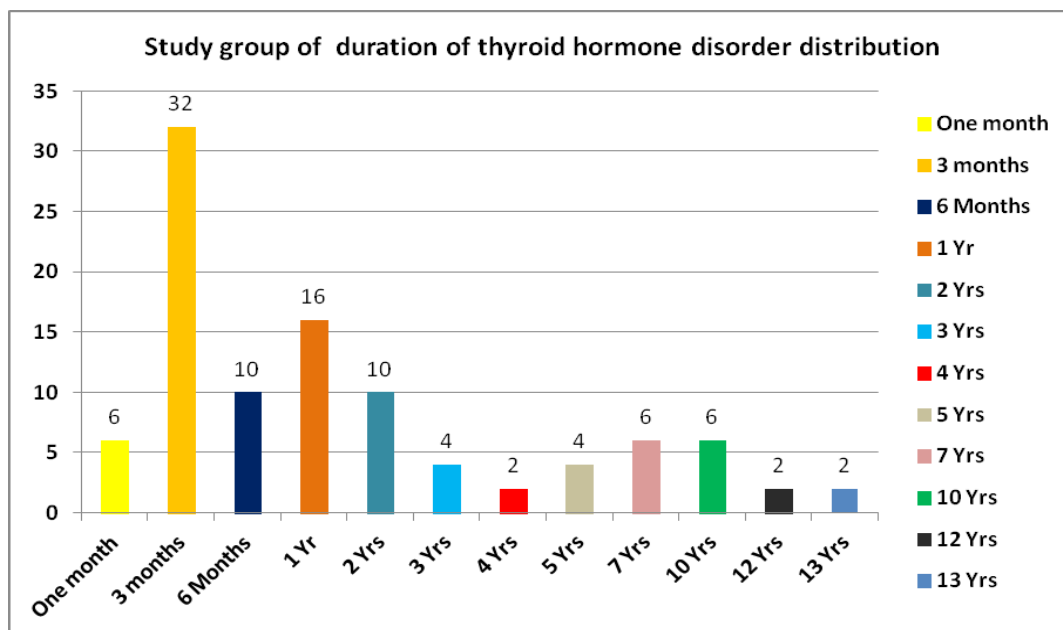
Figure 4-3: showing the Study group of thyroid disorder distribution

Table 4-4:

Showing the thyroid hormones disorder

		Thyroid hormone	No of patients	Percentage	
⇩	⇩	T3 T4	29	58%	hypothyroidism
⇧	⇧	T4 T3	21	42%	hyperthyroidism
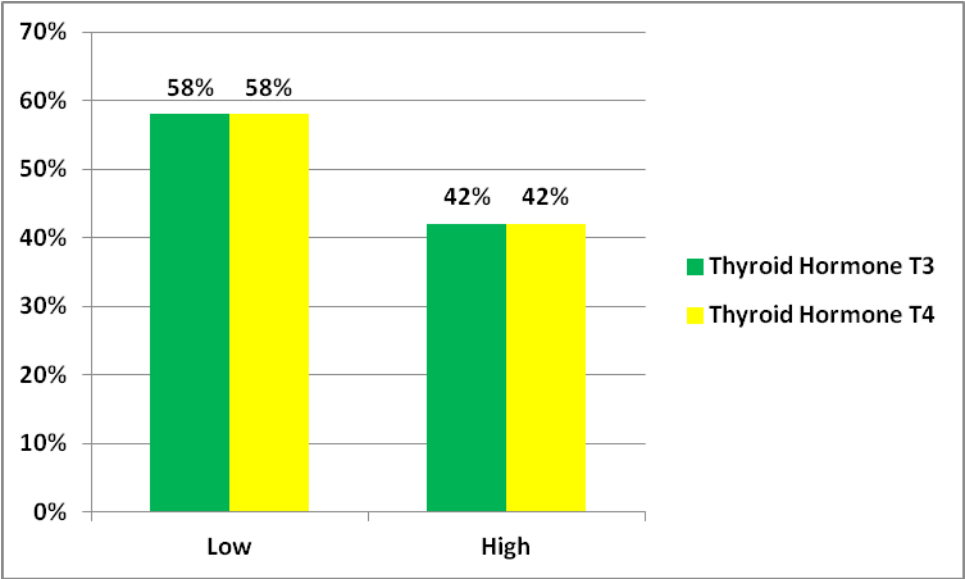
Figure 4-4: Showing the thyroid hormones disorder

Table 4-5:

Showing the Effect of treatment of thyroid on the kidney

Treatment	No of patients	Percentage
Treated	45	90 %
Untreated	5	10 %
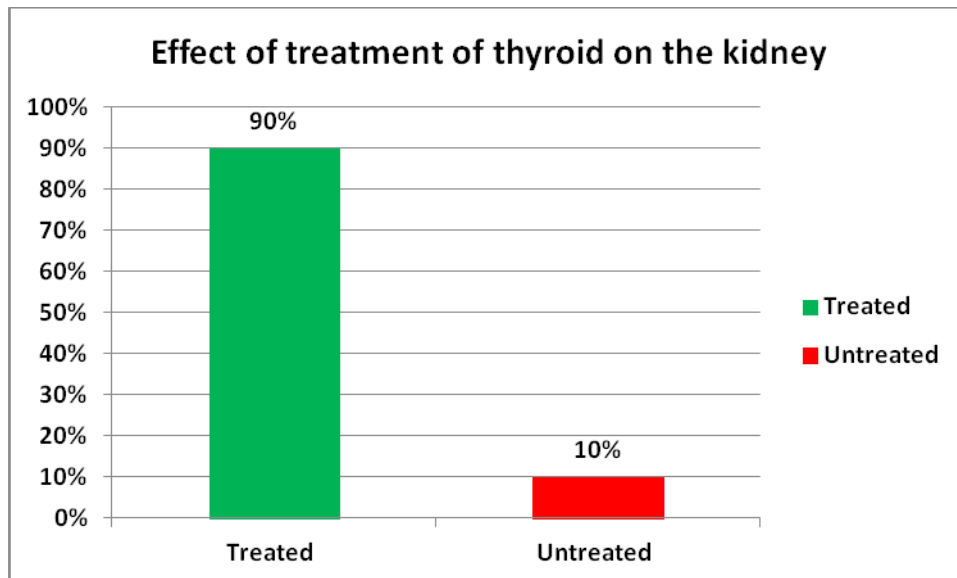
Figure 4-5: Showing the Effect of treatment of thyroid on the kidney

Table 4-6:

Showing ultrasound finding on the kidney with thyroid disorder

U/S Finding	Frequency	Percent
Small kidney	8	16.0
Mild effect	2	4.0
Normal	40	80.0
Total	50	100.0
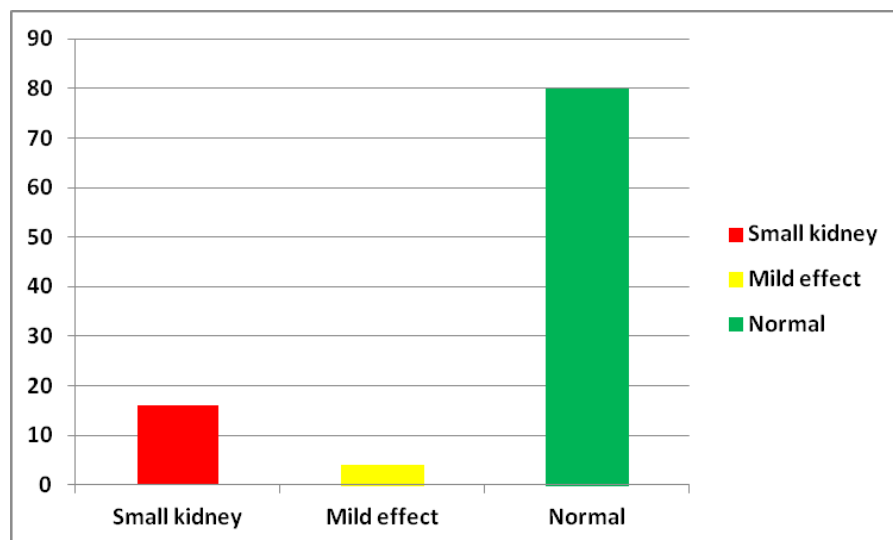
Figure 4-6: Showing ultrasound finding on the kidney with thyroid disorder

Table 4-7

Showing cross-tabulation between duration of illness and U/S findings results

Ultra Sound Findings * Treatment Cross-tabulation				
Count				
		Treatment		Total
		Yes	No	
Ultra Sound Findings	Small kidney	6	2	8
	Mild	1	1	2
	Normal	38	2	40
Total		45	5	50
7 years	2	1	0	
10 years	3	0	0	
12 years	1	0	0	
13 years	1	0	0	
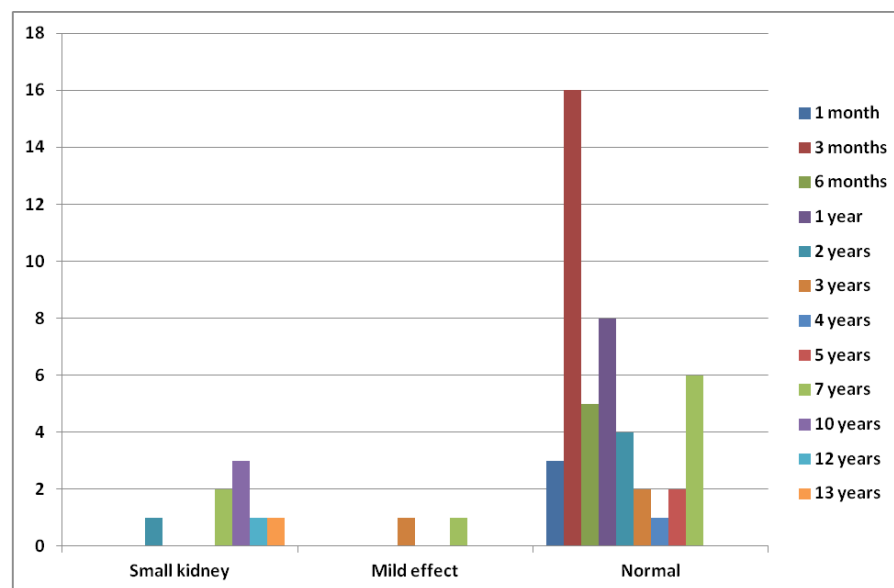
Figure 4-7: Showing cross-tabulation between duration of illness and U/S findings results

Table 4-8

Showing cross-tabulation between treatment of thyroid hormones disorder and U/S findings results
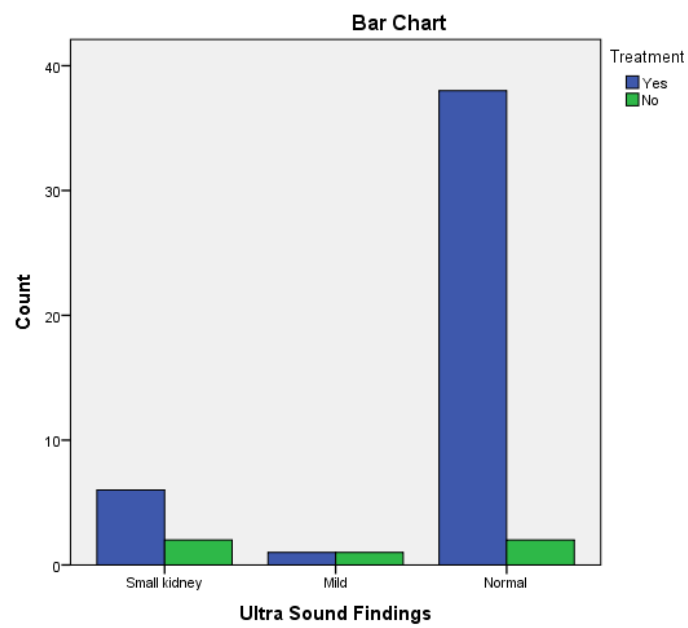
Figure 4-8: Showing cross-tabulation between treatment of thyroid hormones disorder and U/S findings results

Table 4-9

Showing cross-tabulation between Thyroid hormones disorders and U/S findings results

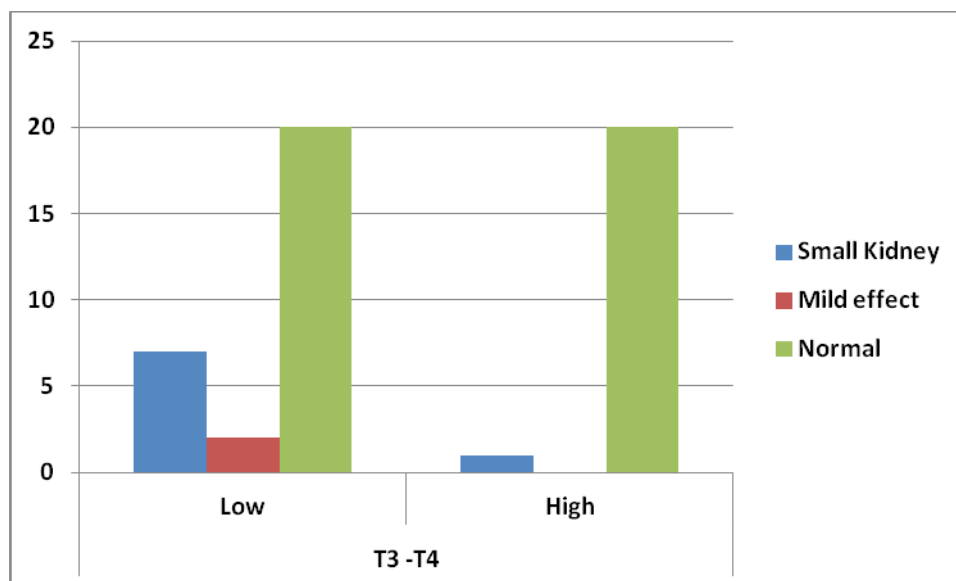| U/S Findings	T3 -T4	
	Low	**High**
Small Kidney	7	1
Mild effect	2	0
Normal	20	20

Figure 4-9: Showing cross-tabulation between Thyroid hormones disorders and U/S findings results

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Chapter Five

Discussion, Conclusion & recommendations

5.1 Discussion

In this study we found that the common group age was (21-30) 19 which is (38%), (41-50) 12 which is 24%, (31-40) 10 which 20%, (51-60) 4 which 8%, (4-10) 2 which 4%, (61-70) 2 which 4% and the last (11-20) 1 which 2%.

The most common effect were female patient (43) which is (86%), this was agree with (Zahoor Ahmed (etal) (2010)).Table (4-1) and (4-2).

16 patients which were (32%) had history of three months thyroid hormones disorders, 8 patients had history of 1 year (16%), 5 patients had history of 2 years (10%), same as 6 months, 3 patients had history of 3 years (6%) same as 7 years and 10 years, 2 patients had history of 3 years (4%) same as 5 years and the last 1 patient had history of 12 years (2%) same as 13 years. (Table 4-4).

We found that when the T_3T_4 level was lower the most effects on kidney size and texture, and the effect reduced when T_3T_4 was elevated, this agree with P Iglesias (etal) (2009)(table 4-5).

Our study showed that 45 patient had got medical treatment which is 90%, some of them showed changing of kidney size and texture while only 5 patient (10%) were not treated and there was no change in kidney size and texture this the same result as (Anjali *etal*, 2009) (table 4-6).

In our study through results finding from U/S sound we found effect on the kidney depend on the duration of illness as follow; 40 patients were normal (80%), 8 patients had small kidney (16) and the last 2 patients had mild effect (4%).this agree with Laura H. Mariani (*etal*)(2011)

(Table4-7):

Showing cross tabulation between duration of illness and the ultrasound findings which revealed that the morphological changes of the renal parenchyma is affected prolonged duration of Thyroid hormones disorders.

(Table4-8):

Showing a cross tabulation between treatment of the Thyroid hormones disorders and the ultrasound findings revealed that some drugs that used for thyroid disorder treatment have a reversed effect on the kidney.

(Table4-9):

Showing a cross tabulation between treatment of the Thyroid hormones disorders and renal parenchymal changes, in which the hypothyroidism have high potential harmful effect on the renal parenchyma than the hyperthyroidism.

5.2. Conclusion

Kidney, thyroid function and dysfunction are interrelated through several mechanisms, it is important to know the effect of hypothyroidism and hyperthyroidism on renal function.

Drugs used in treatment of thyroid may induced change in renal physiology in appropriated investigation leading to delay proper management.

This study aim to determine the effect of thyroid hormone disorder on the kidney by U/S as a rapidly evolving imaging modality in diagnosis the effect.

U/S imaging of the kidney with trans-abdominal techniques has contribute greatly to understand and diagnose.

The most effect on the kidney are decreasing in size (small) and mild effect (increase echogensity).

Lastly the research found that, long duration of Thyroid hormones disorder effect on size and texture of the kidney as appear during U/S findings.

5.3. Recommendations

- U/S is an important modality for detection and diagnosis the effect of thyroid hormone disorder on the kidney.
- Patients with thyroid hormones disorder must checked for renal problem through U/S to discover the effect early.
- Further research by Doppler must be done because color Doppler plays important roles in diagnosis effects on the kidney.
- Further research can be done on specified samples their illness duration above 5 years to gain more specific results.

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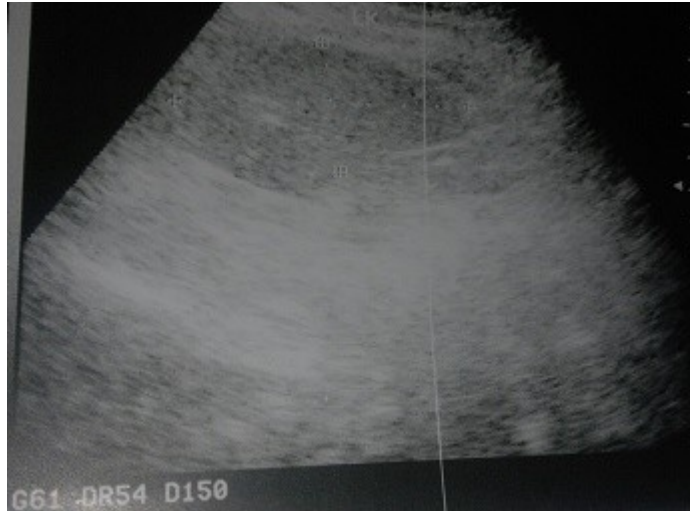**Appendix**

Appendix 1

Figure 5-1: female 54 years, U/S show small kidney
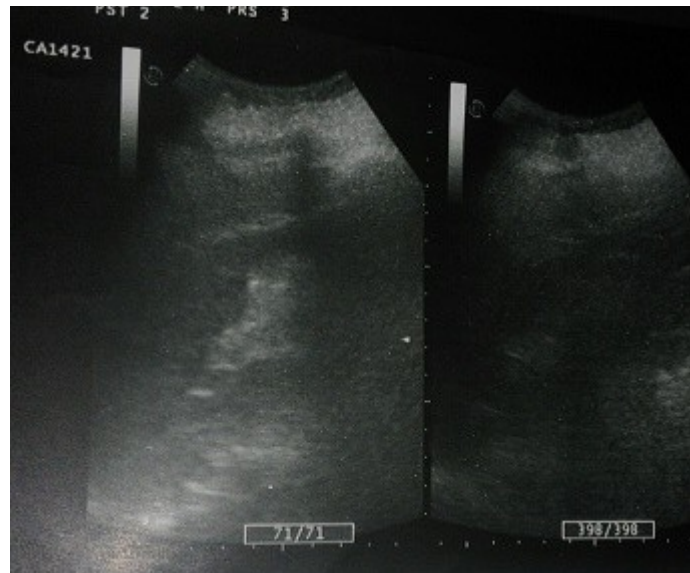

Figure 5-2: female 58 years, U/S show chronic renal disease

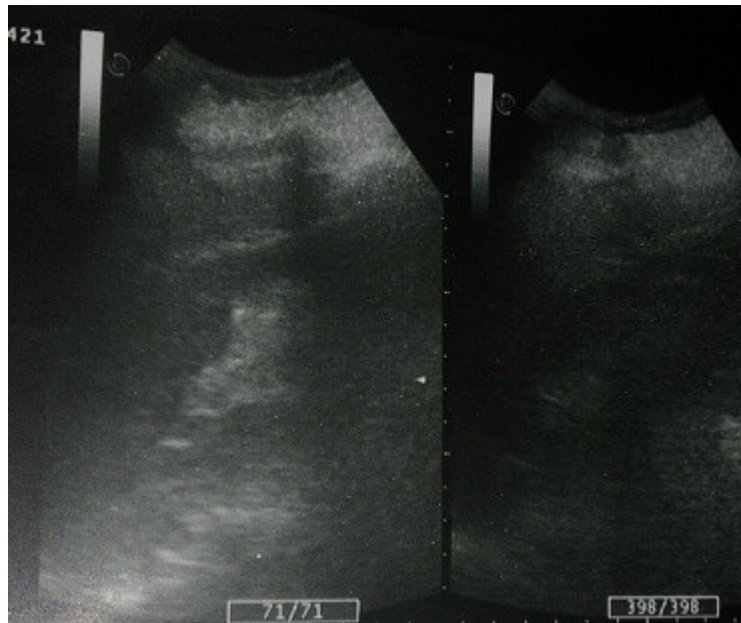**Figure 5-3: male 39 years, U/S show glomular nephritis**

Figure 5-4: female 48 years, U/S show small kidney

Figure 5-5: female 28 years, U/S show normal kidney

Figure 5-6: male 26 years, U/S show normal kidney

Figure 5-7: male 51 years, U/S show chronic renal disease

Appendix-2

Sudan University

Data Collection Sheet

Evaluation the effects of thyroid hormones on the kidney using ultra sound

Date: ………………………………………   No (   )

Patient Data: ……..………………….

   *Patient age:

   (4-24) ☐   (25-45) ☐   (46-66) ☐

   *sex distribution:

    Female ☐   Male ☐

   *Duration of thyroid disorder:

   (5month-5year) ☐   (5-10) ☐   (10-15) ☐

   *Thyroid hormones disorder:

↓ ↓ T_3 T_4 ☐↑ T_3 T_4 ↑ ☐

   *Treatment:
    Treated ☐   untreated ☐ s

*Ultrasound finding on kidney with thyroid disorder:

First effect ☐   Mild effect ☐   last effect ☐ s