Sudan University of Science and Technology (Sudan)

College of Graduate Studies

with collaboration of

Afro-Asian Institute of Medical Sciences
Lahore – Pakistan

CAROTID ASSESSMENT IN PAKISTANI POPULATION

A thesis submitted as partial fulfillment of
M.Sc in Medical Diagnostic Ultrasound

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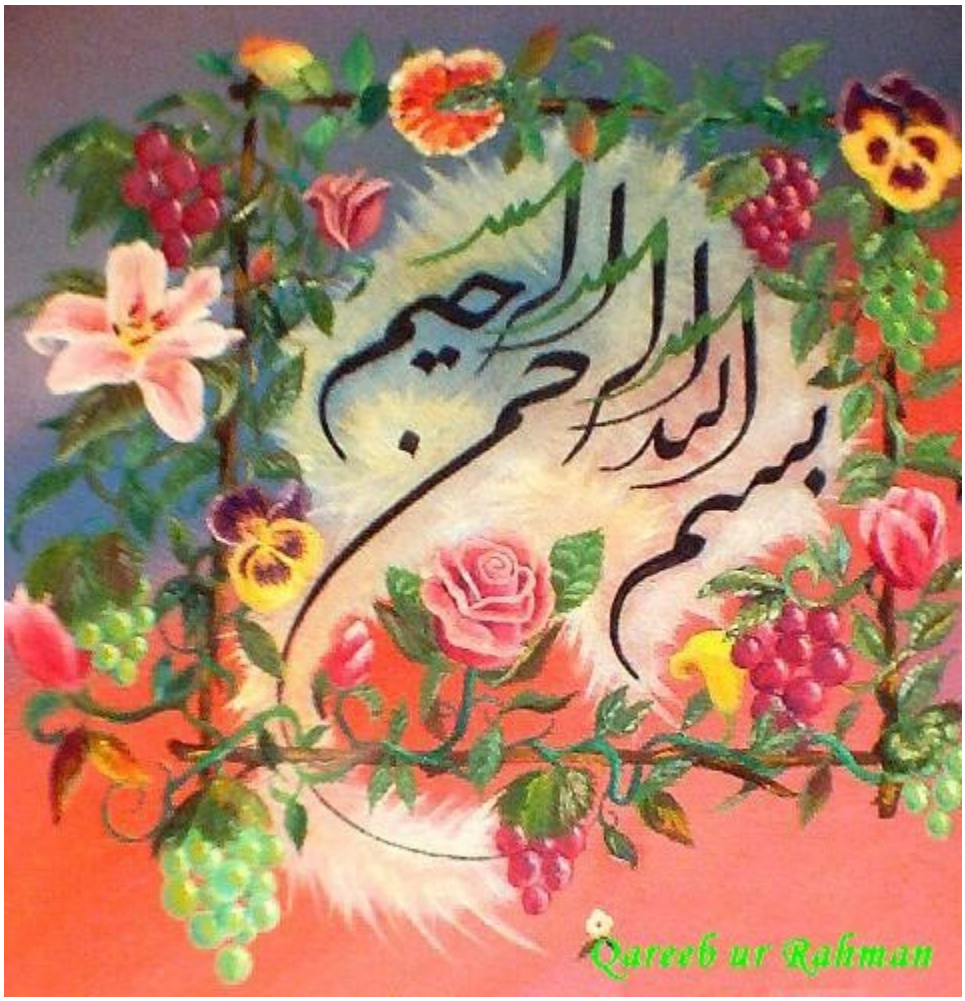
بسم الله الرحمن الرحيم
Qareeb ur Rahman

DEDICATION

TO MY PARENTS

WHO HAVE ALWAYS ENCOURAGED ME AND PRAYED FOR ME TO BE SUCCESSFUL IN MY LIFE.

AND TO MY TEACHERS

PROF. DR.SYED AMIR GILANI AND
DR. TAUQEER ABBASI

ACKNOWLEDGEMENT

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Dr. Muhammad Afzal

Dated:…….

SUPERVISOR'S COMMENT

I have gone through thesis on "CAROTID ASSESSMENT IN PAKISTANI POPULATION" by Dr. MUHAMMAD AFZAL and found it satisfactory for submission.

I wish him ever success

Prof. Dr. Syed Amir Gilani **Date …….**

M.B;B.S (Pb), PhD(Swiss)

ABSTRACT
(ARABIC)

ABSTRACT
(English)

Objectives

To study the definition role of Real time B- made and Doppler Ultrasound for Evolution of IMT of CCA peak systolic velocities of CCA, I.C.A & ICC/CCA ratio of both sides and their comparison.

To study the definition rule for follow up

Method

One rounded Asymptomatic individuals of different ages of both sex were renamed for CCA and ICA of both sides:

Took in time meet a illness of CCA of both sides

Peak systolic valuation of CCA and ICA of both sides were taken

ICC/CCA

Ratios were taken of both sides –Valuation taken with angle of 55

-Machine used were Toshiba anemia x G and Toshiba xeric equipped with 9-11 MHZ linear problem.

Results

The over all rounded of my study is 21% of asymptomatic individuals have I.M.T about than 0.8 mm having 09-1.3 –PSV of CCA and ICA are with normal ranges.

ICA/CCA valuation ratio with normal lioness ie less than 1.80.

Conclusion

Therefore I concluded through my study that ultrasound has high sanctified speedily and caesurae rate for measurements of IMT/ PSV of CCA, ICA for and ICA / CCC ratio and any theories if bound.

TABLE OF CONTENTS

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CHAPTER ONE

INTRODUCTION

Carotid arteries are vital vessels supplying blood to neck, face and brain: Carotids run along anterolateral side of neck and divided into internal and external carotid arteries.

Embryologically carotids arteries arise from third aortic arch cranial portion of dorsal aortic arch.

Right CCA arises from in nominate artery and left CCA arises directly from aorta. Common carotid artery enclosed in carotid sheath along with internal jugular vein and vague nerve.

Common carotid artery bifurcates at level of upper border of laryngeal cartilage in to internal and external carotid artery. ECA supplies face and I.CA supplies to brain after entering in skull through carotid foramen. ICA is lateral, larger, limbless, & low resistance vessel than ECA. Carotid arteries are medium sized arteries. Histology composed of three layers.

Carotid sinus

Present at junction of CCA and ICA supplied by glossopharngeal and sympathetic nerve. It acts as baroreceptor which regulates blood pressure.

Carotid body

Radish-brown body present at bifurcation having rich nerves supply from vague and sympathetic nerves. Carotid body also has chemoreceptor. Carotid body responds to change in O_2, CO_2, and PH.

Carotid arteries may have congenital, arteriosclerosis, Aneurysms, inflammatory, neoplastic and other diseases.

Above stated diseases especially atherosclerosis, aneurysm and other diseases cause TIA, RIND, HTN, CVA and subclavian steel and etc.

Real time B-Scan and Doppler has definitive role in assessing anatomy, extent of disease and management of carotids after medically and surgically treated patients.

SYNOPSIS

Carotid arteries are vital vessels supplying blood to neck, face, and brain. Doppler Assessment has sensitivity and specificity in detection of vascular pathology.

INDICATIONS FOR CAROTID ULTRASOUND

Atherosclerotic lesion appears in early child hood and clinically appears in 40 - 60 years.

- Transient ischemic attacks.
- RIND
- Hypertensive patients.
- High risk patients for brain strokes.
- Smokers.
- Hyperlipidemia.
- Resolving strokes in young patients.
- Obesity.
- Screening for vascular pathology

HYPOTHESIS

Real time B-mode. color and spectral Doppler have definitive role in evaluation of anatomy, pathology and management of carotids.

RESEARCH METHAD

The study will be taken at Afro Asian Institute of Medical Sciences Lahore Pakistan. Intima media thickness, pathological lesions and color and sF5cmd"analysis, will be taken in Pakistan population.

Each patient will be scanned under scanning guide lines by candidate and diagnosis will be confirmed by highly qualified sonologist.

All daily data sheets will be signed by candidate's local supervisor who will see the details of causes and will check sonograms with pathological findings.

FACILITIES AVAILABLE

1 Nemio - XG 3 - D

Convex probe, linear probe. Color and power Doppler Directional angio. Tissue harmonics,

2 Xario 4-D

With linear probe and convex

CAROTID ASSESSMENT IN PAKISTANI POPULATION

COMMON CAROTID AND INTERNAL CAROTID ARTERIES

For research topic, the target population will be patients either asymptomatic or symptomatic for carotids.

DATA STORAGE

All data collected will he stored on computer protected by password. All paper format data will be stored in locked cabinet.

DATA ANALYSES

The analysis data is done to evaluate intima-media thickness, spectral color and Doppler assessment of pathologies.

Application of advantages and characteristics of Doppler ultrasound investigation to carotid arteries.

CONCLUSIONS

The results will be conducted from processed data and will be discussed in detail to see the carotid in Pakistan population and its comparison with international standard data-Discussion will be done and result of study will be summarized to draw conclusion.

REFERENCES FOR SYNOPSIS

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CHAPTER TWO

Literature Review

EMBRYOLOGY

The third aortic arch forms the common carotid artery and the First part of the internal carotid artery. The remainder of the internal carotid is formed by the cranial portion of the dorsal aortic artery. The external carotid artery is a sprout of the third aortic arch. The bud that grows cranially to form external carotid artery. Some portion of disappearing first and second arch arteries may contribute.

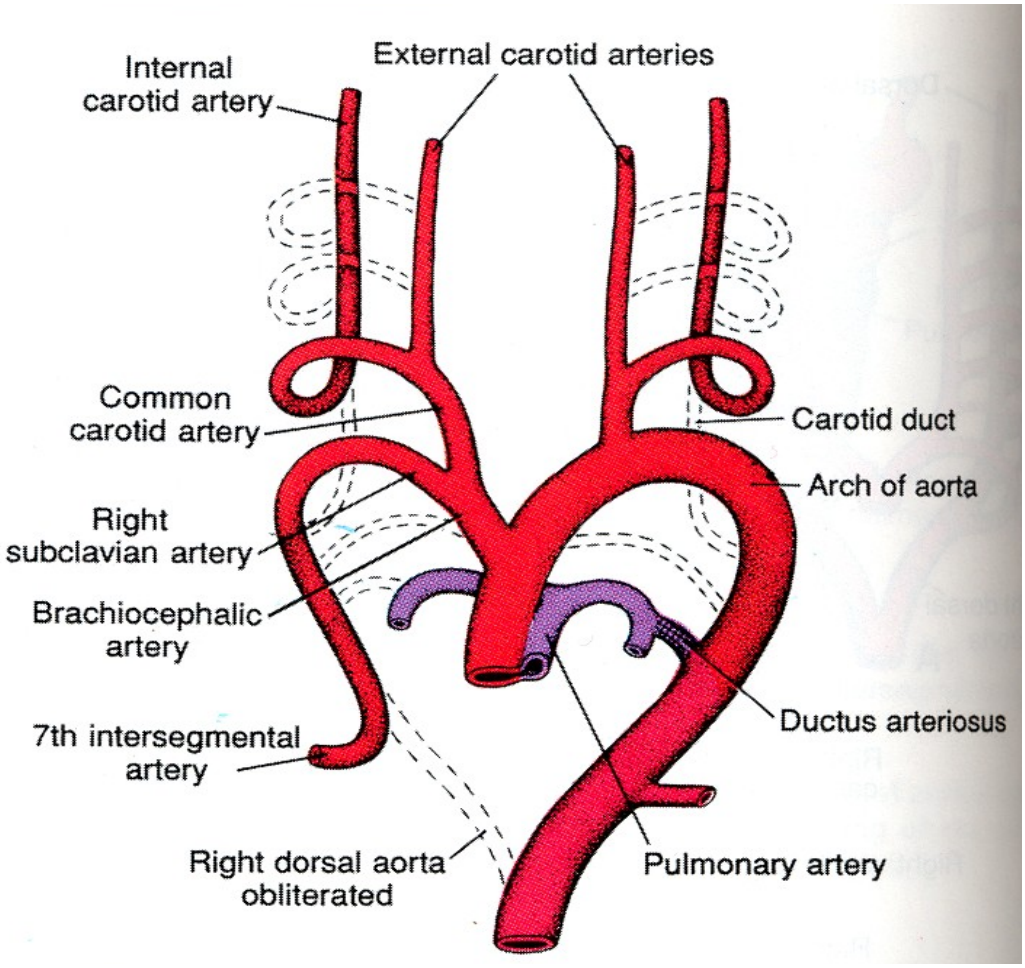

Fig. 1

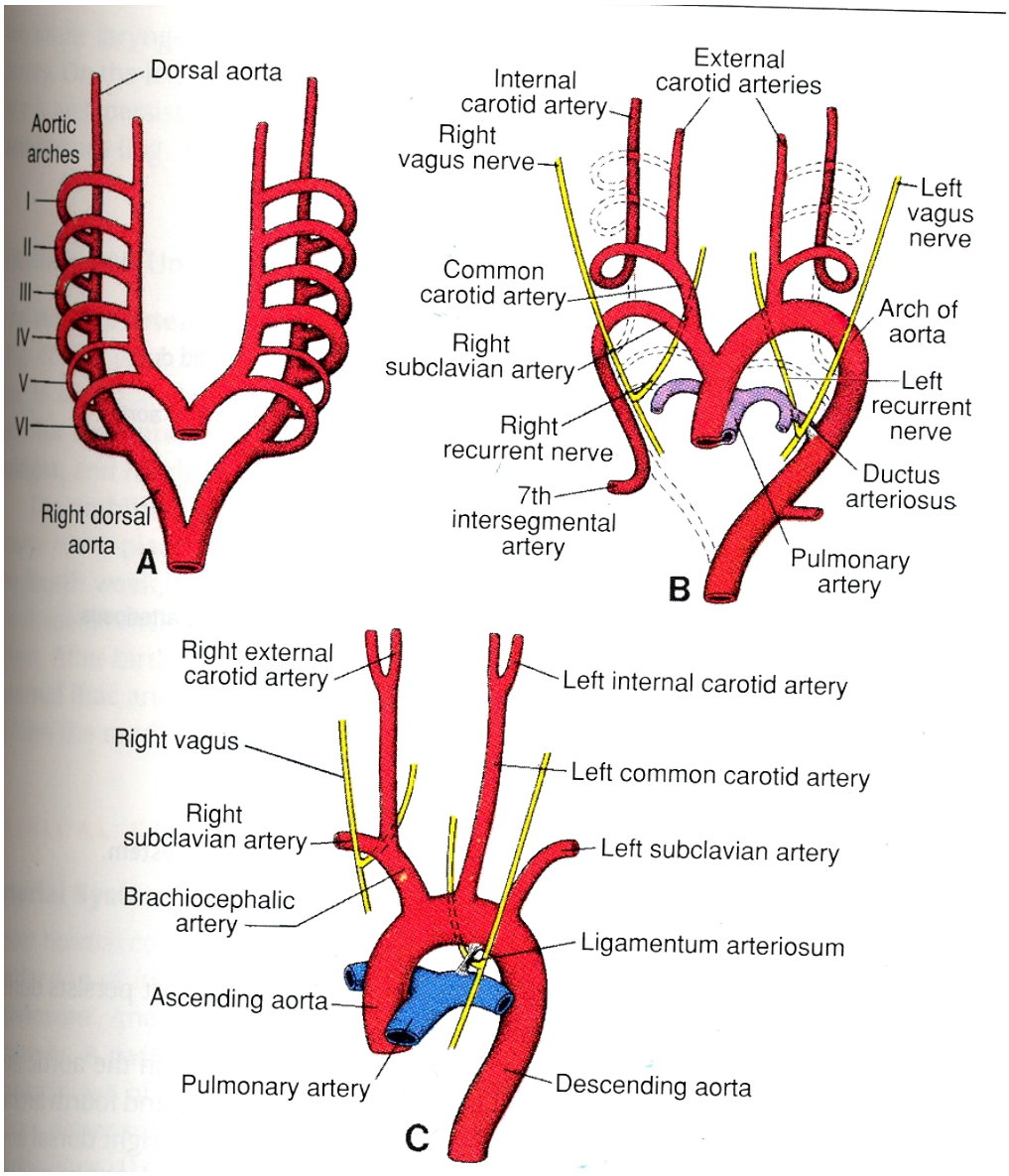

Fig. 2 On the either side, the part of third arch artery proximal to external carotid bud, forms common carotid artery. The portion of third arch artery distal to bud, along with original dorsal aorta cranial to attachment of third arch artery forms internal carotid artery. As the right third and fourth arch arteries from the right horn of aortic sac. The common carotid artery and subclavian arteries become branches of brachio-cephalic artery.

ANATOMY

Common Carotid Artery

The right common carotid artery is a branch of the brachiocephalic artery. It begins in the neck behind the right sternoclavlcular joint. The left common carotid artery is branch of the arch of the aorta. It begins in the thorax in front of the trachea opposite a point a little to the left of the centre of the manubrium. It ascends to the back of the left sternoclavicular joint and enters the neck.

In the neck both arteries have a similar course. Each artery runs upwards within carotid sheath, under cover of anterior border of sternocleidomastoid. It lies in front of lower four cervical transverse processes. At the level of the upper border of thyroid cartilage the artery ends by dividing into external and internal carotid arteries.

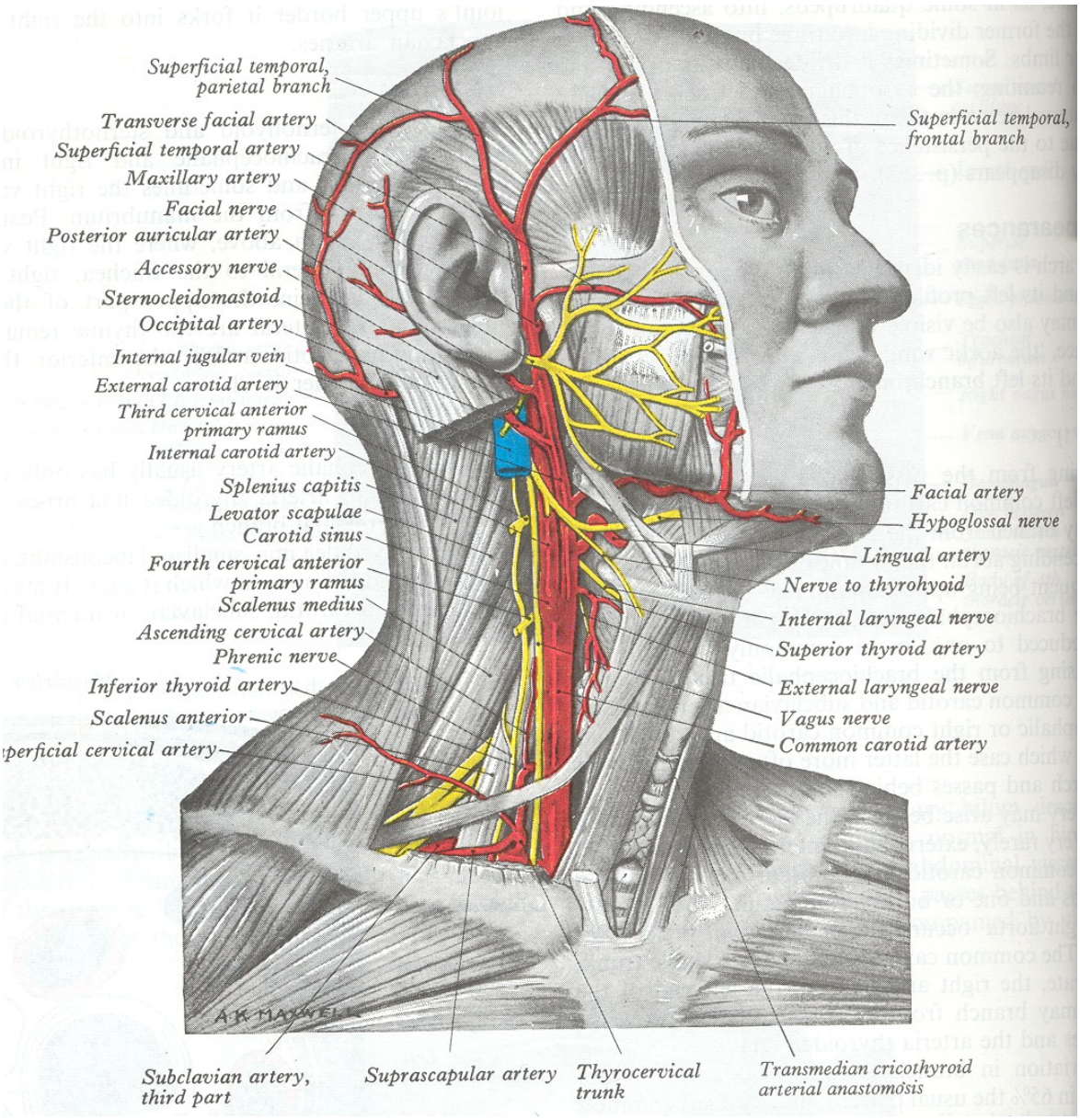

Fig. 3

COMMON CAROTID ARTERY

Surface Marking

Common Carotid Artery

It is marked by a broad line along the anterior border of the stemocleidomastoid muscle by joining the following two points.

(a) A point on the stemoclavicular joint.

(b) A second point on the anterior border of the stemocleidomastoid muscle at the level of the upper border of the thyroid cartilage.

The thoracic part of the left common carotid artery is marked by a broad line extending from a point a little to the left of the centre of the manubrium to the left stemoclavicular joint.

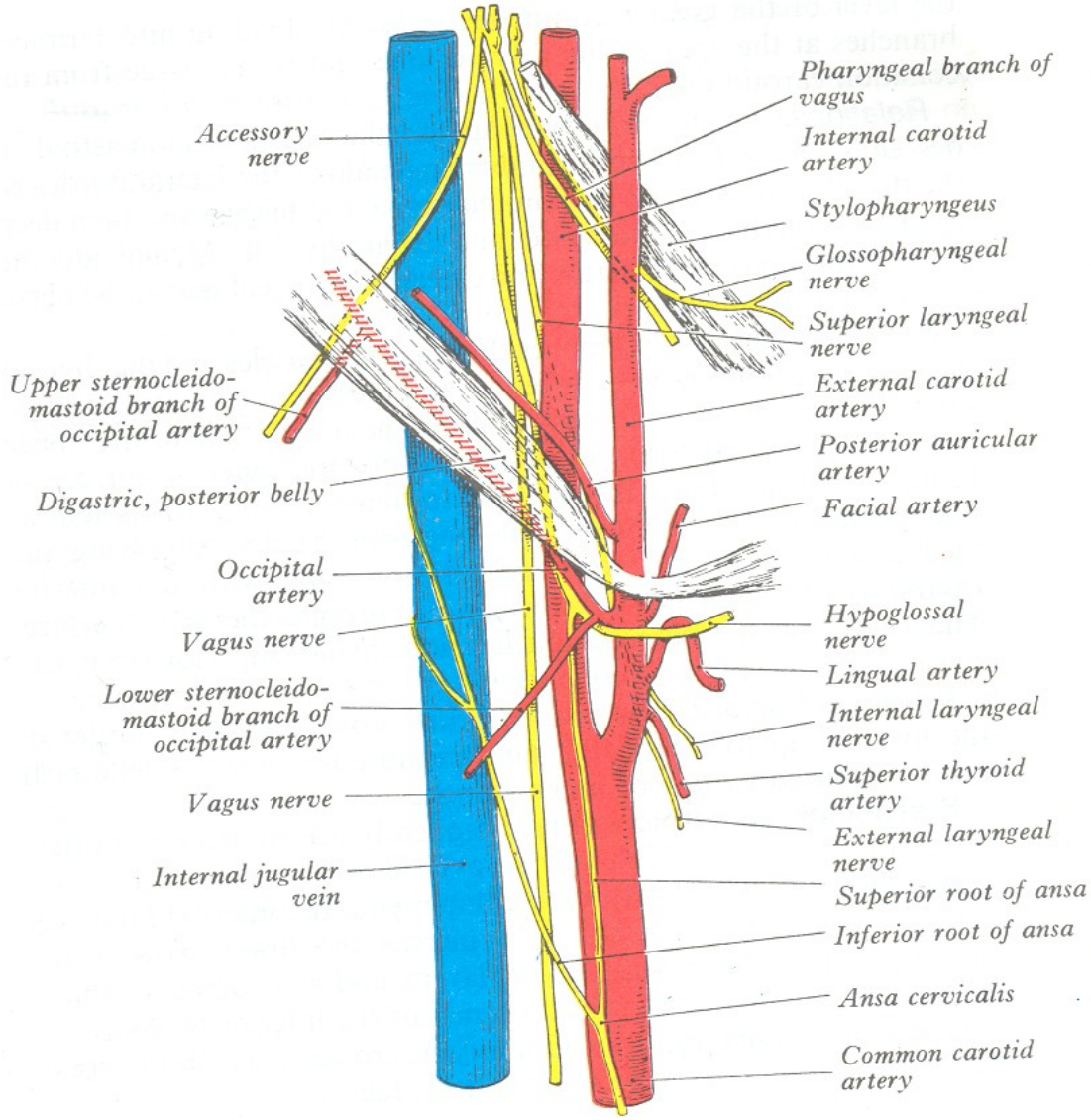

Fig. 4 The structures crossing the internal jugular vein and carotid arteries and those intervening between the external and internal carotid arteries.

It is marked by a broad line joining the following 1 points.

(a) A point on the anterior border of the sternorcleidomastoid muscle at the level of the upper border of the thyroid cartilage.

(b) A second point on the posterior border of condyle of the mandible.

The origin and course of the common carotid arteries. The relation of the artery in the neck are given:

The common carotid artery is enclosed in carotid sheath. The three contents of the sheath;

(1) The common carotid artery medially;

(2) internal jugular vein, laterally; and

(3) the vagus nerve between the artery and the vein, posteriorly

Anterior Relations

(a) The common carotid artery is crossed by superior belly of the omohyoid at the level of cricoid cartilage

(b) Above the omohyoid (in the carotid triangle the artery is comparatively superficial, and is related to: (1) The medial (anterior) margin of the sternorcleidomastoid, deep to which there are; (2) descending hypoglosal nerve; (3) the superior thyroid vein; (4) the stemothyroid; and (5) the ansa cervical

(c) At the level of the omohyoid there are : (1)' stemocleidomastoid; (2) the omohyoid; (3) stemohyoid; (4) the stemothyroid; and (5) the anterior cervicalis.

(d) Below the omohyoid, the artery is deeply situated, and is covered by; (1) The stemocleimastoid; (2) the anterior Jugular vein; (3) stemohyoid; (4) The stemothyroid and the middle thyroid vein.

Carotid Sinus

The termination of the common carotid artery, or the beginning of the internal carotid artery shows a slight dilatation, known as the carotid sinus. In this region, the tunica media is thin, but the adventitia is relatively thick and receives a rich innervation from the glossopharyngeal and sympathetic nerves. The carotid sinus acts as a baroreceptor or pressure receptor and regulates blood pressure.

Carotid Body

Carotid body is a small, oval reddish-brown structure situated behind the bifurcation of the common carotid artery. It receives a rich nerve supply mainly from the glossopharyngeal nerve, but also from the vagus and sympathetic nerves. It acts as a chemoreceptor and responds to changes in the oxygen and carbon dioxide and pH content of the blood.

Other allied chemoreceptors are found near the arch of the aorta, the ductus arteriosus, and the right subclavlan artery. These are supplied by the vagus nerve.

Internal Carotid Artery

The internal carotid artery is one of the two terminal branches of the common carotid artery. It begins at The common carotid artery can be compressed against the carotid tubercle, i.e. the anterior tubercle of the transverse process of vertebra C6. which lies at the level of the cricoid cartilage.

The level of the upper border of the thyroid cartilage opposite the disc between the third and fourth cervical vertebrae, and ends inside the cranial cavity by supplying the brain. This is the principal artery of the brain and the

eye. It also supplies the related bones and meninges.

For convenience of description the course of the artery is divided into four parts: (a) Cervical part. in the neck;

(b) petrous part, within the petrous temporal bone; (c) cavernous part. within the cavernous sinus: and (d) cerebral part in relation to the base of the brain.

Cervical Part

1. It ascends vertically in the neck from its origin to the base of the skull to reach the lower end of the carotid canal. This part is enclosed in the carotid sheath (with the internal jugular vein and the vagus.

2. No branches arise from the internal carotid artery in the neck.

3- Its initial part usually shows a dilatation, the carotid sinus which acts as a baroreceptor

4. The lower part of the artery (in the carotid triangle) is comparatively superficial. The upper part, above the posterior belly of the digastric, is deep to the parotid gland, the styloid apparatus, and many other structures.

Relations

Anterior or Superficial

I. In the carotid triangle: (1) Anterior border of sternocleidomastoid: (2j hypoglossal nerve;

(3) occipital artery; (4) descendens hypoglossi;

(5) common facial and lingual veins; and (6) the external carotid artery is anteromedial to it.

II. Above the carotid triangle: (1) Posterior belly of the digastric: (2) stylohyoid; (3) posterior auricular artery; (4) stylopharyngeus; (5) styloid process;

(6) parotid gland with structures within it;

(7) glossopharyngeal nerve; (8) pharyngeal branch of vagus nerve: and (9) the cartilaginous part of the auditory tube and the tensor veli palatini (at the base of the skull)

Posterior

(1) Longus capitis: (2) prevertebral fascia: (3) superior cervical ganglion: (4) superior laryngeal nerve; (5) carotid sheath; (6) pharyngeal veins; (7) vagus posterolateral throughout: and (8) the glossl-opharyngeal, vagus. accessory and hypoglossal nerves at the base of the skull,

Medial

(1) Pharynx; (2) ascending pharyngeal artery

(3) internal and external laryngeal nerves; (4)The external carotid is anteromedial to it below the| parotid; and (5) levator veli palatini (at me base of the skull).

Lateral

(1) Internal jugular vein; and (2) the bony part of the auditory tube, tympanic plate and the temporo-mandibular joint (at the base of the skull).

Petrous Peat

1. In the carotid canal, the artery first runs upwards, and then turns forwards and medially at right angles. It emerges at the apex of the petrous .temporal bone, in the posterior wall of the foramen lacerum where it turns upwards and medially.

2. Relations: The artery is surrounded by venous -aid sympathetic plexuses. It is related to the middle ear and the cochlea (posterosuperiorly): the auditory tube and tensor tympani (anterolaterally); and the trigeminal ganglion (superiorly).

3. Branches: (a) Caroticotympanic branches enter [lie middle ear, and anastomose with the anterior and posterior tympanic arteries; and (b) the pterygoid branch (small and inconstant} enters the pterygoid canal with the nerve of that canal and anastomoses with the greater palatine artery.

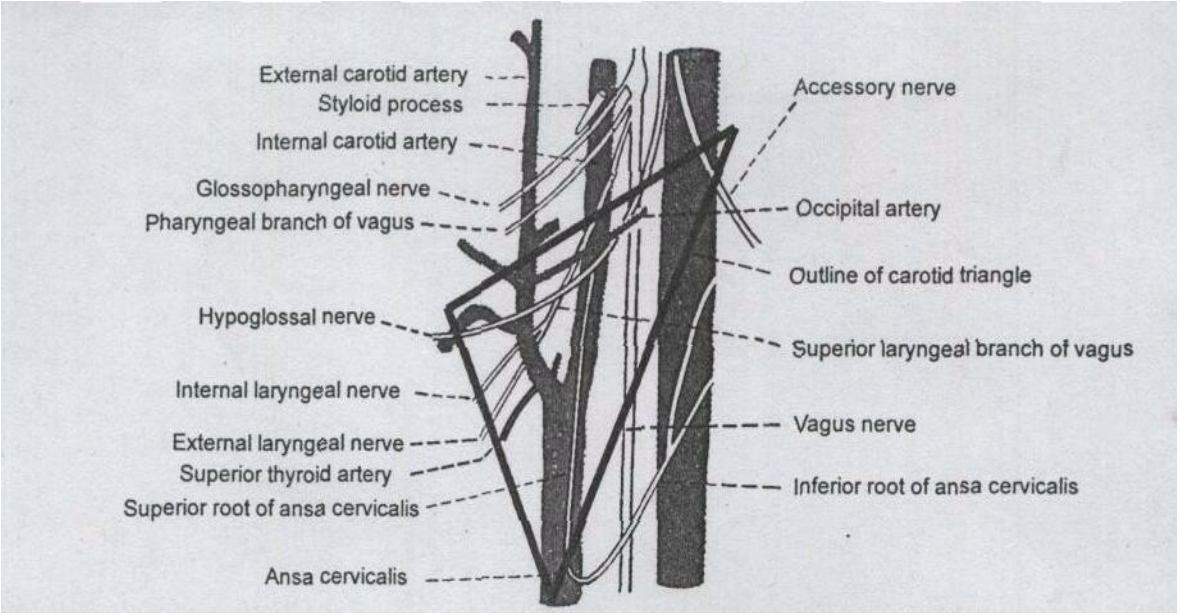

Fig. 5 The ninth, tenth, eleventh and twelfth cranial nerves and their branches related to the carotid arteries and to the interne ! jugular vein, in and around the carotid triangle.

HISTOLOGY

This plate illustrates various types of blood and lymphatic vessels, surrounded by loose connective and adipose tissue (13, 28). Most vessels have been cut in a transverse or oblique plane of section.

A small artery (4), with its basic wail structure, is shown at the top center of the plate. In contrast to a vein (22), an artery has a relatively thick wall and a small lumen. In cross section, the wall of an artery exhibits the following layers:

a. tunica intima, composed of an inner layer of endothelium (16), a subendothelial (17) layer of connective tissue and an internal elastic lamina (membrane) (19), which marks the boundary between the tunica intima and tunica media.

b. tunica media (4), composed predominantly of circular smooth muscle fibers. A loose network of fine elastic fibers is interspersed among the smooth muscle cells.

c. tunica adventitia (6), composed of connective tissue containing small nerves (14) and blood vessels (15). The blood vessels in the adventitia are collectively called vasa vasorum (15), or "blood vessels of blood vessel."

When arteries acquire about 25 or more layers of smooth muscle in tunica media, they are called muscular or distributing arteries- Elastic fibers become more numerous, but are still present as thin fibers and networks.

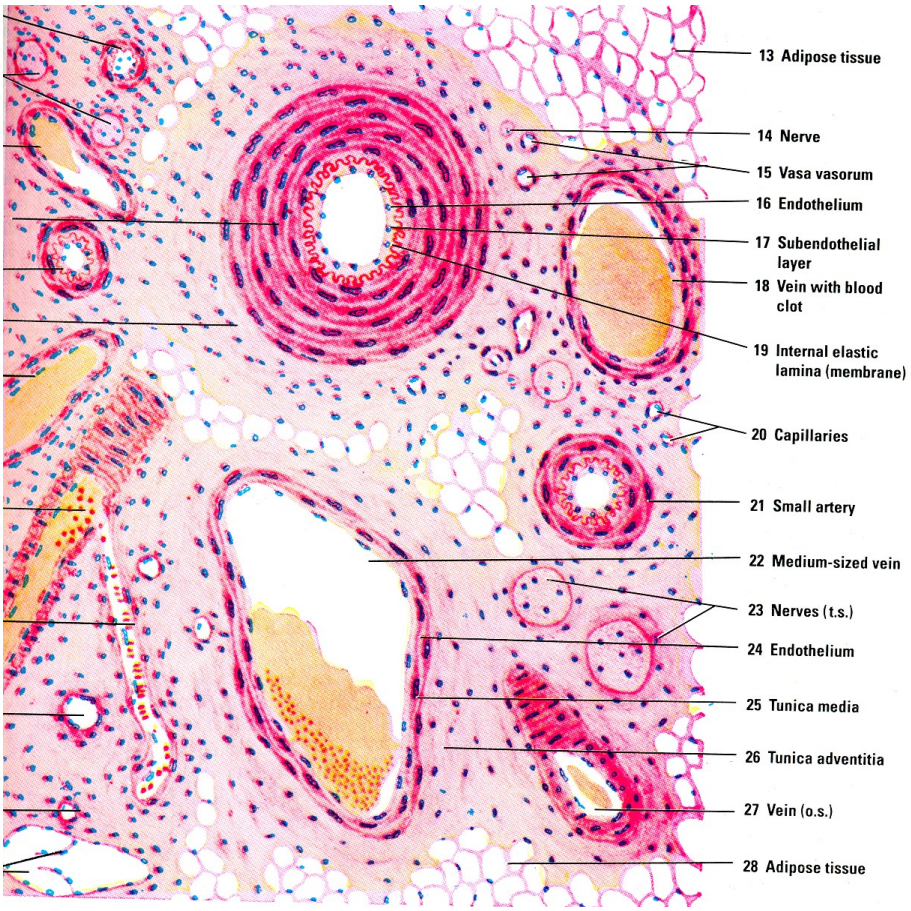

Fig. 6 Blood vessel

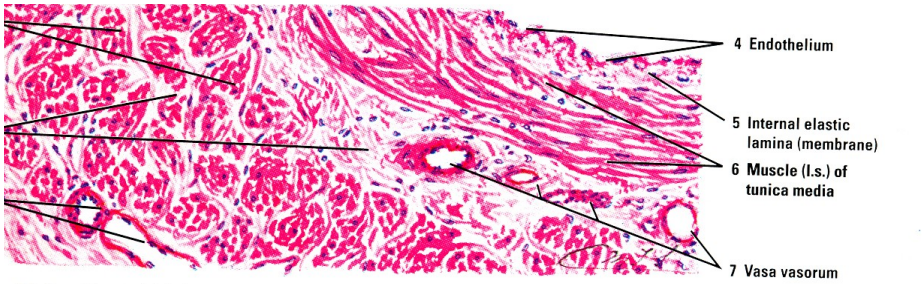

Fig. 7 large vein portal vein (Transverse section). Stain hematoxylin-eosin Medium magnification.

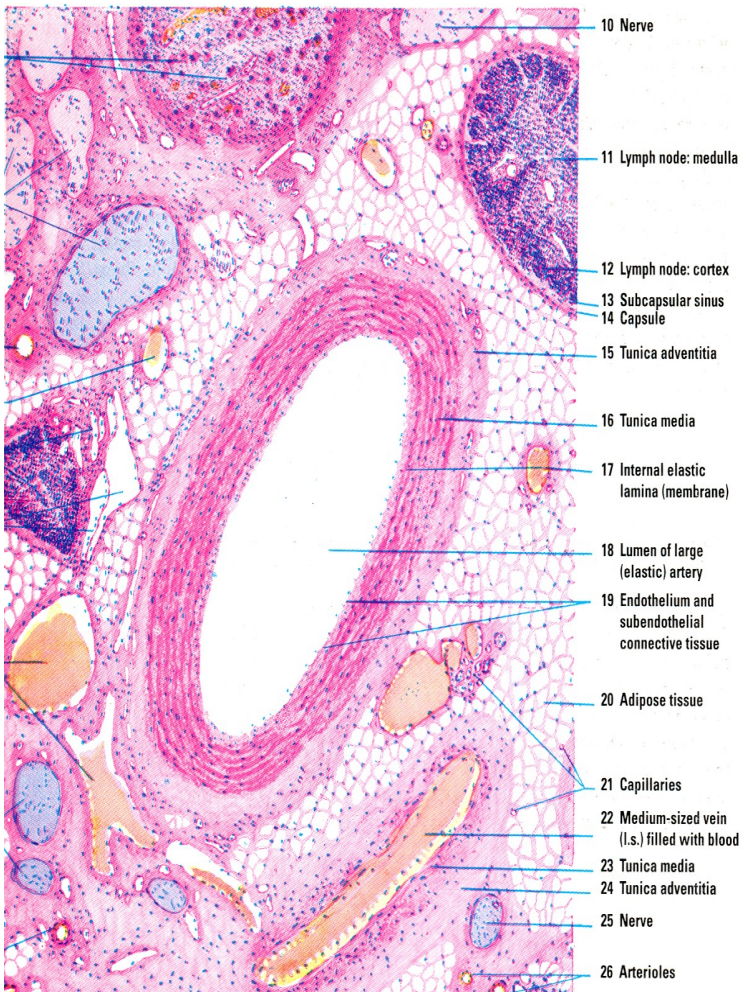

Fig. 8 Histology

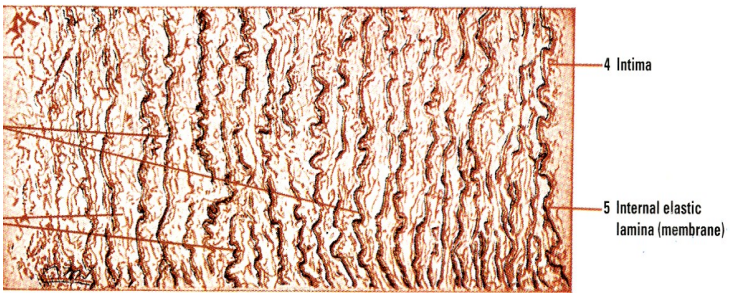

Fig. 9 Large artery: aorta (vein (Transverse section). orcein stain aorta elastic fibers selectively stained between high magnification.

PATHOLOGY

CLASSIFICATION OF ARTERIAL LESIONS:

(A) Congenital:

1. Congenital (or Berry) Aneurysm
2. Coarctation of aorta
3. Arteriovenous fistulas

(B) Arterioscleosis:

1. Atheroscleosis
2. Arteriolosclerosis

(i) Hyaline

(ii) Hyperplastic

3. Monckeberg's Sclerosis

(C) Aneurysms:

1. Atherosclerotic
2. Syphilitic
3. Dissecting

(D) Inflammatory:

1. Non-Specific arteritis
2. Giant cell arteritis
3. Rheumatic arteritis
4. Rheumatoid aortitis and arteritis
5. Syphilitic aortitis

6. Polyarteritis nodosa
7. Thromboangitis obliterans (Buerger's disease)
8. Wegener's synd

(E) Neoplastic:

1. Hemangioma
2. Hemangioendothelioma
3. Angiosarcoma

(F) Others:

1. Raynaud's disease
2. Acrocyanosis
3. Idiopathic cystic medionecrosis

ATHEROSCLEROSIS (AS)

DEFINITION:

"It is characterized by atheromas (or fibrofatty plaques) which are raised focal plaques within the intima having a core of lipids (mainly chlesterol and cholesterol esters) and a covering fibrous cap. It affects large and medium-sized muscular arteries as well as elastic arteries."

ETIOLOGY AND RISK FACTORS

(A) CONSTITUTIONAL OR IMMUTABLE FACTORS:

1. Age: Early lesions of AS appear in childhood but clinically significant disease appears in 40-60 yrs.
2. Sex:

i. Male are more affected

ii. Females are protected due to estrogen production

iii. After menopause, incidence increases and becomes equal to men by 70-80 years.

3. Familial Predisposition: It may be due to.

i. Hereditary derangements in lipoprotein metabolism e.g., familial hypercholesteremia and dyslipoproteinemias.

ii. Familial clusteing of other risk factors e.g., hypertension and diabetes.

(B) ACQUIRED OR CONTROLLABLE FACTORS:

1. Hyperlipidemia:

(i) LDL: Inc. LOL level (or hypercholesterolemia) has a linear correlation with AS, as:

(a) Diets rich in saturated animal fat like butter and eggs -> inc. LDL level -> in risk of AS.

(b) Diets rich in monounsaturated (olive oil) or polyunsaturated fat (fish or corn oil) -> dec. LDL level - dec. risk of AS

(c) Genetic or acquired disorders like diabetes mellitus or hypothroidism -> cause hypercholesterolemia -> lead to AS.

(d) When serum cholesterol levels are lowered -> atherosclerotic plaques regress or fail to progress.

(ii) HDL: Inc. HDL2 and HDL3 levels have inverse relationship with as.

(a) HDL participate in reverse transport of cholesterol.

(b) Exercise and moderate alcohal consumption -> inc. HDL level dec. risk of AS.

(c) Obesity and smoking ->. dec. HDL level -> inc. risk of AS.

(iii) VLDL: Inc. VLDL (or hypertriglyceridemia) have linear correlation with AS, but it is weaker than for LDL

2. Hypertension:

i. Hypertension (esp. diastolic level) inc. the risk fivefold

ii. It is more important than hypercholesterolemia after 45 years of age.

3. Smoking:

i. One pack/day inc. the risk 200%

ii. Cessation of smoking dec. the risk.

4. Diabetes Mellitus:

It causes hypercholestremi -> inc. risk of AS -> inc. risk of Ml, brain stroke and gangrene of lower limbs.

(C) SOFT RISK FACTORS:

Their effect on AS is either less clearly defined or is controversial. There is inc. risk of AS with:

1. Lack of physical activity
2. Obesity (more than 30% overweight)
3. Type A personality behaviour
4. Hyperuricemia
5. Use of oral contraceptives.
6. Coffee consumption
7. High carbohydrate intake.

Multiple factors have a multiplied effect rather than an additive one.

PATHOGENESIS:

There are five hypotheses to explain the origin of atheromas. These are:

1. Response to injury hypothesis
2. Monoclonal hypothesis
3. Thrombogenic or encrustation hypothesis
4. Insudatian hypothesis
5. Intimal cell mass hypothesis

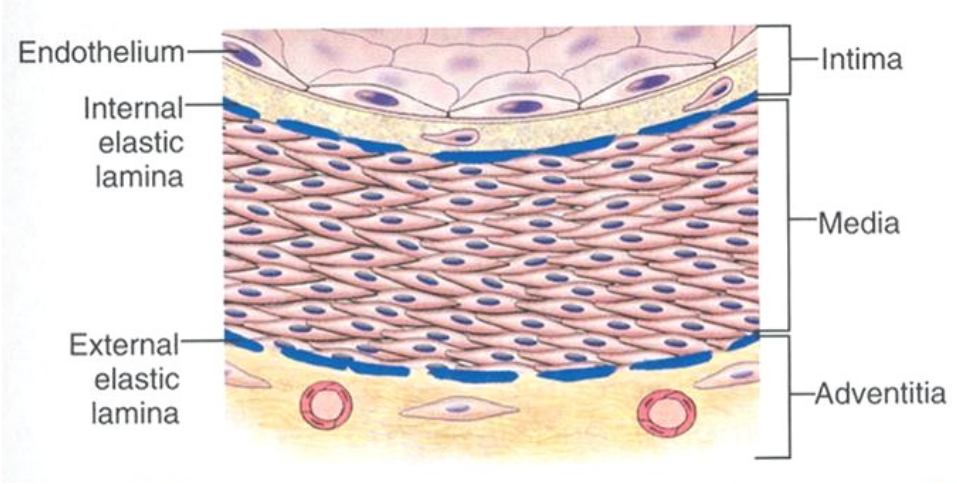

Fig. 10 Histology

RESPONSE TO INJURY HYPOTHESIS:

It states that, 'The lesions of AS are initiated as a response to some form of endothelial injury."

1. Chronic or Repeated Endothelial injury:

It may be denunding or nondenuding injury, and can be caused by

i. Anoxia
ii. 'Circulating endotoxins
iii. -CO or other products derived from cigarette smoke.
iv. Specific endotheliotoxins, e.g., homocysteine in homocystinuria Cardio-

Vascular System

v. Viruses

vi. Hemodynamic disturbances e.g., shear stress and turbulent flow. These occur due to twists and turns as well as branchings of arteries. These causes inc.-

a. Endothelial permeability

b. Cell turnover

c. Receptor-mediated LDL-endocytosis

vii. Hyperlipidemia (esp. hypercholesterolemia). Role of

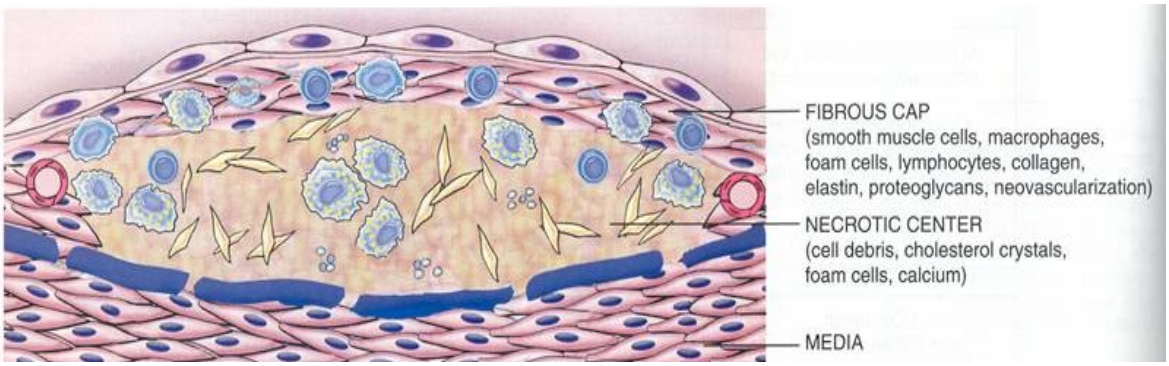

Fig. 11 Schematic depiction of the major components of well developed atheromatous plaque: fibrous cap composed of proliferating smooth muscle cells, macrophages lymphocytes, from cells and extracellular matrix. The necrotic core consist of cellular debris extracellular lipid with cholesterol crystals, and foamy macrophages.

Hypercholesterolemia:

1. Inc. cholesterol-phospholipid ratio of endothelial cell membranes which become rigid -> dec. intercellular associations -> inc. permeability.
2. Stimulate synthesis of adhesion molecules by endothelial cells -> adherence of monocytes and lymphocytes to the focus of injury.
3. Causes changes in platelet membrane composition - -> activation and inc. adhesiveness of platelets.
4. Causes lipoprotein accumulation within the intima at sites of endothelial injury.
5. Provides the opportunity for oxidation of lipoproteins

2. Insulation of Lipoproteins:

I. LDL

ii. Oxidized LDL

iii. VLDL

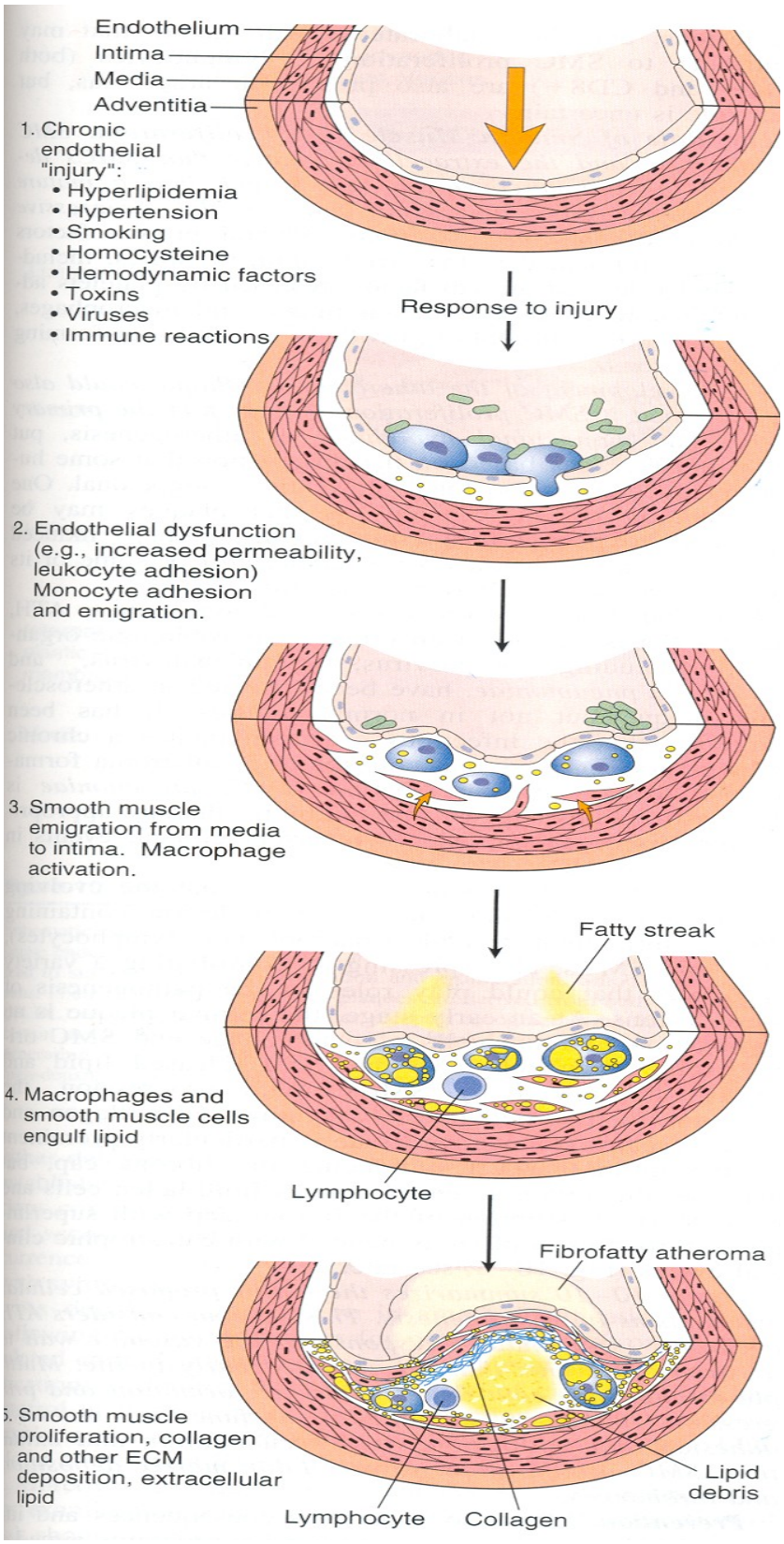

Fig. 12 steps in the formation of atheroma

Oxidized LDL:

Formation:

It is derived from oxidation of LDL due to the action of free radicals released by monocytes and endothelial cells.

Role in AS:-It causes:

i. Endothelial injury

ii. Monocyte chemotaxis

iii. Immobilizes macrophages at the site of injury

iv. Smooth muscle injury

v. Foam cell formation because it is taken up by macrophages and smooth muscle cells much more avidly than LDL.

3. A Complex Series of cellular Events:

i. Monocyte adhere to endothelium -> migrate between endothelial cells —> reach intima-»take up lipoproteins-> become Foam Cells.

ii. Smooth muscle cells (SMCs) enter intima -> take up liproproteins -> become Foam Cells.

iii. With the persistence of hypercholesterolemia, more and more foam cells are formed. These aggregate to form Fatty Streaks.

4. Proliferation of SMCs about the Foci of Foam Cells:

SMCs proliferate and synthesize collagen, elastin and proteoglycans which are deposited, within the atheroma esp. on its intimal aspect creating fibrous cap. With this fatty streaks are converted into fully mature Fibro-Patty Atheromas:

Cardio-Vascular System

Growth Factors responsible for SMCs Proliferation are:

i. PDGF: It is released from platelets, macrophages, SMCs and endothelial cells in response to cytokines (like TNF, IL-land-IFN-a) released by macrophage- lymphocyte interaction.

ii. FGF

iii. EGF

iv. TGF-a

v. Loss of growth inhibitors, e.g. heparin-like molecules and TGF-

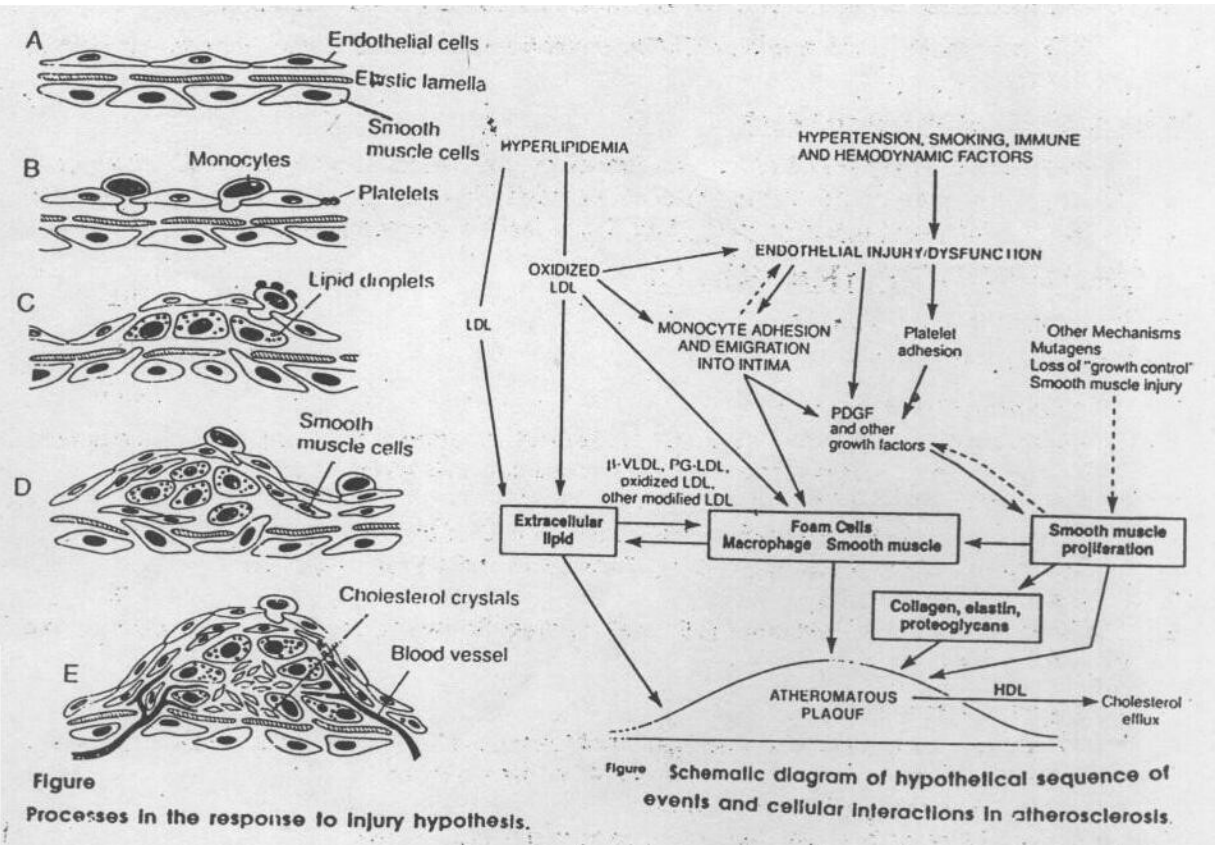

Fig. 13 Schematic steps in the pathogenesis atheroma

Monoclonal Hypothesis

It states that the atheromas are derived from the proliferation of single (mono-clonal) or few SMCs (Oligo-clonal). SMCs are stimulated to proliferate by mutagens like viruses, products derived from cholesterol or tobacco smoke. With the passage of time, these cellular lesions accumulate lipoproteins in hypercholesterofemic persons.

This hypothesis has been disproven.

Thrombogenic or Encrustation Hypothesis

It states that the atheromas arise from mural thrombi formed over the foci of endothelial injury. Organization and re-endotheliazation of the thrombi incooporate them into the intimal layer, which then accumulate lipoproteins in hypercholesterolemic persons.

This hypothesis is also doubtful.

Insudation Hypothesis

it states that the focal accumulation of fat within the vessel wall is derived from plasma lipoproteins. Insudation of lipoproteins may be receptor-mediated or via macrophages.

This-hypothesis cannot explain SMCs proliferation and thrombosis that are important features of AS.

Intimal cell mass hypothesis:

It explains the origin of AS at vascular branch points. Due to hemodynamic disturbances at branch points/there are normally present focal accumulation of SMCs and extracellular matrix in the intima. Called Intimal Custion or Intimal Pad. These lesions eventually develop into atheromas.

MORPHOLOGY:

FATTY STREAKS:

Gross:

1. Origin: in first years of life.
2. Location: aortic valve ring, post. wall of descending thoracic aorta, adjacent to the orifices of intercostal arteries in thoracic aorta and coronary arteries (at 10 yrs.)
3. Colour: yellowish intimal discolorations
4. Size: Initially 1 mm in size. Eventually become thicker and slightly elevated, and elongate in long axis of the vessel to produce 1-3 mm wide and 1.5 cm long streaks.
5. Shape: Linear streaks
6. Fate: These are present universally in all children. Some regress while others develop into atheromas .

Microscopic:

Fatty streaks are the intimal aggregation of foam cells having finely vacuolated cytoplasm. Foam cells are derived from macrophages and SMCs.

There may be:

i. Few marginal T lymphocytes
ii. Little extracellular debris derived from dead foam cells.
iii. Little cellular proliferation
iv. Little enclosing connective tissue
v. Delicate fibrous cap between the lesion and overlying endothelium.

Atheromatous Plaque

Gross:

1. Origin: 30 - 40 yrs. of age

2. Location: In descending order of involvement: lower abdominal aorta, coronary arteries, popliteal arteries, descending thoacic aorta, internal carotid arteries and circle of Willis. But the vessels of upper limb, mesenteric arteries, renal arteries and aortic arch are spared.

3. Colour: Bright yellow to gray Cardio-Vascular System

4. Size: Range upto several cms. in largest dimension, and raised several mms. above the surface of the intima

5. Shape: Irregular in shape and may coalesce to form map-like configurations.

6. Cut Section: Shows:

i) Central core of yellow grumous debris enclosed within a firmer, poorly defined wall.

ii) Covered by a tough gray-white fibrous cap.

7. Fate: May regress or enlarge to become complicated plaques (calcification, ulceration, superimposed thrombosis, intra-plaque hemorrhage and atrophy of the underlying media with formation of aneurysm).

Microscopic:

1. Components: There are 3 basic components:

i. Cells: SMCs, monocytes/macrophages and lymphocytes

ii. Connective tissue: Fibers and matrix

iii. Lipids:

2. Parts: There are 2 parts of an atheroma:

i. Deeper part called Narcotic Center: Foam cells, cellular debris, cholesterol crystals, plasma proteins and calcium. .

B. Superficial part called Fibrous Cap: SMCs, macrophages,lymphocytes, foam cells, collagen, elastin and proteoglycans.

3. Variations:

i. Some plaques may have less lipids but more SMCs and connective tissue called

"Fibrous Plaques"

ii. May develop vascularization about their margins

iii. May cause atrophy and fibrosis of the unerlying media to form aneurysms

iv. May develop superimposed thrombosis, which may be incorporated into the plaque.

Complications

1. Calcifications -> form pipestem arteries
2. Fissuring or Ulceration of the luminal surface -> there is discharge of debris into the blood giving rise to cholesterol Emboli.
3. Superimposed thrombosis -> gives rise to Emboli as well as narrowing of arterial lumen. This may lead to ischemia or infarction of the organs
4. Intraplague hemorrhage -> may balloon the plaque and cause rupture. May narrow the arterial lumen with ischemia or infarction of the organs
5. Aneurysmal dilatation due to atrophy of the underlying media -> it may rupture.

Organs Affected:

1. Heart-> ischemic heart disease (IHD)
2. Brain -> brain infarct (strokes)
3. Kidneys -> ischemia
4. Small intestine-> ischemia
5. Lower limbs -> ischemia

LAB DIAGNOSIS

1. Blood Cholesterol:

Blood sample is taken before breakfast. Adults above 50 yrs. of age with cholesterol level above 250 mg/100ml are considered at a higher risk to AS.

2. Measuring of Velocity with Doppler Probes:

Lesions of AS well be suspected in the vessels in which the velocity of the blood flow is altered.

3. Radiography:

to show calcified atheromas

4. Angiography:

to show lesions of AS by the filling defect produced by atheromas

5. Other tests:

For example. Exercise tolerance test, Echocardiography or Cardiac scintigraphy may be done to see the impact of AS on heart function

ARTERIOSCLEROSIS

It refers to the thickening and inelasticity of arteries. It includes:

1. Atherosclerosis

Arteriolosclerosis: It is characterized by hyaline or hyperplastic thickening of

the small .arteries and arterioles. It leads to luminal narrowing and ischemic injury to the tissues, e.g.. kidneys (nephropathy). It is associated with hypertension and diabetes mellitus. 3. . Monckeberg's Medial Calcific Sclerosis: It is characterized by ring or plate-like calcification of the media of medium to smal-size muscular arteries. It creates nodularity on . palpation and may undergo ossification. It does not produce luminal narrowing, so clinically

ANEURYSMS

DEFINITION

"Aneurysm is a localized abnormal dilatation of an artery or vein. It develops wherever there is marked weakening of vessel wall."

CLASSIFICATION

(A) On the basis of causative factor;

Congenital:

1. Berry aneurysm Crisoid aneurysm
2. Traumatic:
3. Acquired:

i. Atherosclerotic

ii. Syphilitic

iii. Dissecting following cystic medionecrosis

iv. Mycotic

In polyarteritis nodosa

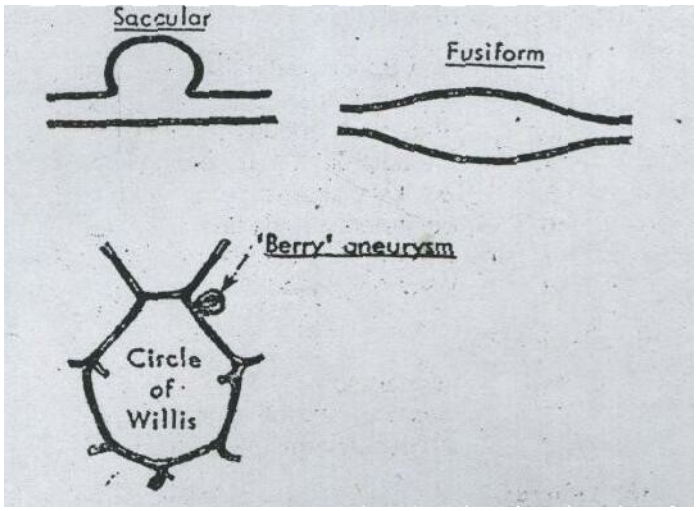

Fig. 14 Berry-shaped aneurysm

(B) On the basis of Gross appearance:

1. Berry-shaped
2. Saccular
3. Fusiform
4. Cylindrical!
5. Dissecting

LAB DIAGNOSIS:

T. Chest/Abdominal X-ray:

Show calcified aneurysms

2. Ultrasound,
3. Aortoqram.
4. CTandMRI:

May be done in dissecting aneurysm to show abnormal thickening of aortic wall in the area of dissection.

Cardio-Vascular System

ATHEROSCLEROTIC ANEURYSM

ETIOLOGY:

Atherosclerosis. Risk; factors for AS are the risk factors for these aneurysms.

PATHOGENESIS:

Atheroma formation -> atrophy and fibrosis of the underlying media - marked Weakening of the vessel wall -> aneurysmal Dilatation

MORPHOLOGY:

Gross:

1. Location: Abdominal aorta between renal and common iliac arteries.
2. Appearance: Saccular, cyindrical or fusiform
3. Size: upto 15 cm India and 25 cm in length

Microscopic:

1. Atheromas: are seen within the intima
2. Atrophy and fibrosis of the underlying media.
3. Mural Thrombi may be seen. In Saccular form, they completely fill the dilatation while in cylindrical or fusiform forms, they partially fill the dilatation.

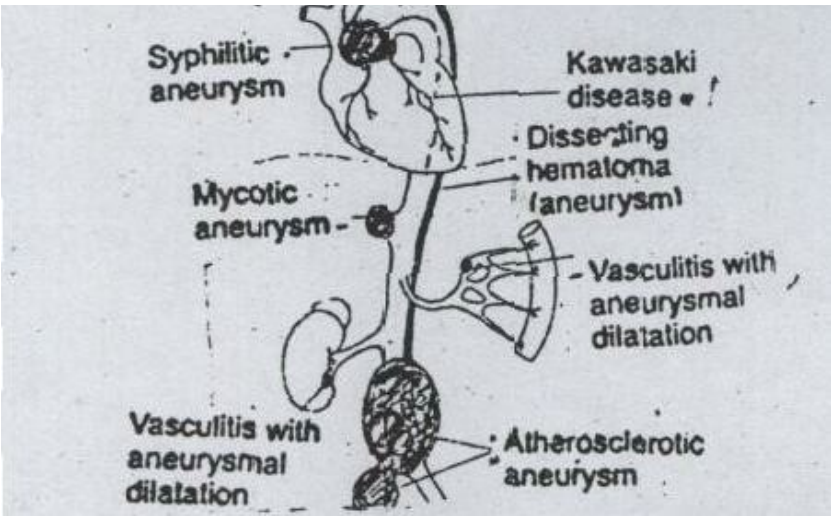

Fig. 15 Atherosclerotic aneurysm

CLINICAL FEATURES AND COMPLICATIONS:

1. Occlusion of iliac, renal or mesenteric arteries either by direct pressure or by thrombus.
2. Embolism from the thrombus
3. May Erode adjacent structure e.g.. ureter, vertebrae, etc.
4. May rupture to cause fatal hemorrhage
5. May form: an abdominal mass

Incidence:

ETIOLOGY: (Rs)

Syphilis caused by Treponema pallidum. Syphilitic aortitis occurs in the tertiary stage of syphilis. .

PATHOGENESIS:

1. In Syphilis: Following basic tissue reaction occur:

Obliterative endarteritis (there is concentric endothelial and fibroblastic

proliferative thickening of the small vessels in the involved area. ii. Perivascular cuffing (there is mononuclear esp. plasma cell infiltrate around theaffected vessel). Gumma formation (a type of chronic granuloma).

2. In Syphilitis Aortitis: there is obliterative endarteritis and perivascular cuffing of the vasa-vasorum of the aorta -> narrowing of the lumen of the vasa-vasorum -> ischemia of the aortic media -> pathchy loss of elastic fiber and smooth muscles (called Windowing) -> development of fibrous scars in the media which contract to produce longitudinal wrinkling of the intimal surface called 'Tree Barking". Weakness of the wall -> aneurysmal dilatation. Secondary atherosclerotic involvement may occur and further weaken the aortic wall.

MORPHOLOGY:

Gross:

1. Location: Ascending thoracic aorta and aortic arch. Aortic valve ring may be. involved leading to valve incompetence and hypertrophy and dilatation of the left ventricle upto 1000 gms (cor Bovinum). There may be narrowing of the ostia of vessels arising from aorta including coronary arteries.
2. Appearance: Saccular or fusiform
3. Size: upto 15-20 cm in dia.

Cardio-Vascular System

Microscopic:

1. Vosa-Vasorum of the aorta shows: obliterative endarteritis (concentric endothelial and fibroblastic proliferation) and perivascular cuffing (surrounding mononuclear esp. plasma cell infiltrate).

2. Aortic media show patchy loss of elastic fibers and smooth muscles (called Windowing) with development of fibrous scars. -

3. Aortic Intima shows fibrous thickening and longitudinal wrinkling due to contracture of fibrous scars, called tree barking.

4. There may be: Secondary atherosclerotic involvement, mural thrombi or calcification of the aorta

CLINICAL FEATURES AND COMPLICATIONS

1. Difficulty in respiration. due to pressure on the lungs and airways.
2. Difficulty in swallowing due to pressure on the esophagus
3. Persistent brassy cough due to pressure on recurrent laryngeal nerve. '.
4. Pain due to erosion of ribs and vertebrae
5. Cardiac disease (Cor Bovinum)
6. Death due to:

i. Rupture -> fatal hemorrhage
ii. Heart failure
iii. Myocardial infarction
iv. Erosion of vital organs (bronchi and esophagus)

Incidence:

1. Male to female ratio is 3:1
2. Commonly seen in 40-55 yrs of age.

DISSECTING ANEVRYSM (Dissecting Hematoma; Aortic Dissection)

DEFINITION:

Dissecting aneurysm arises when blood enters the wall of the artery, dissects between its layers and creates a cavity within the vessel wall

CLASSIFICATION:

(M De Bakev's Classification:

Type arises in ascending aorta but extends beyond it Type II: arise in ascending aorta but is confined to it. Type III: arise in descending thoracic aorta

B) Recent Classification:

Type A: includes all dissections involving the ascending aorta

PREDISPOSING FACTORS

1. Connective tissue disorders, e.g., Marian's synd. and Ehler's - Danlos synd.
2. Hypertension
3. Pregnancy
4. Aortic coarctation
5. Bicuspid aortic valves

PATHOGENESIS:

Dissecting aneurysm begins with Cystic or Laminar Medionecrosis (v/hich is focal loss of elastic and smooth muscle fibers and the defect is filled with amorphous basophilic ground substance) -» weakening of the aortic wall -> formation of intimat tear when pressure waves pass along the aortic wall -> blood enters the aortic wall through the intimal tear due,to its own pressure (that is raised in hypertension) -> blood dissects between the medial layers -> produces cavity " within the aortic wall -> Dissecting Aneurysm

Causes of Cystic Medionecrosis:

1. Hypertension
2. Biochemical defects in the synthesis or maintenance of the proteoglycans, collagen and elastic fibers, For Example:

i. In Marian's Synd defective cross-linking of collagen

ii. Copper deficient diet, administration of penicillamine or ft - aminopropionitrite - defective cross-linking of collagen - dissecting aneurysm.

MORPHOLOGY:

1. Intimal Tear:

* It is located within 10 cm of the aortic valve in 90% cases
* It is longitudinal or oblique

Cardio-Vascular System

* 4-5 cm long
* Edges are sharp, clear and irregular
* It is absent in 5-10% cases.

2. Blood enters the media through the intimal tear and dissects between outer and middle third of the media. It may involve either the entire circumference of the aorta or a portion of »t.

3. Blood may:

i. Dissect proximally into the heart or coronary arteries

ii. Dissect distally into the iliac or femoral arteries.

iii. Rupture into the pericardial Sac or pleural cavity.

iv. Re-rupture back into the aortic lumen through a second distal intimal tear to produce a false lumen or double barrelled aorta which may become endotheliazed.

Microscopic:

1. Cystic or Laminar Medionecrosis: There are focal areas of elastic 'and smooth muscle loss in the media that are filled by amorphous basophlic ground substance
2. Elastic fragmentation is present even outside the focal areas of cystic medionecrosis
3. Focal fibrosis of the media may be present:
4. Accompanying atherosclerosis may be present

CLINICAL FEATURES AND COMPLICATIONS

1. The dissection itself -> excruciating chest pain.
2. Extension of the dissection into the aortic branches causing obstruction of the vessels:

I. Neck arteries -> ischemia of the arm or brain
ii. Coronary arties -> myocardial ischemia
Hi. Mesenteric arteries -> abdominal pain
Iv. Iliac arteries -> sensory and motor changes, in lower half of the body.

3. Extension of the dissection into aortic root —> aortic regurgitation
4. Aneurysm may rupture --> fatal hemorrhage.

POLYARTERITIS NODOSA (Periarteritis Nodosa; PAN)
(NUQ)

DEFINITION:

It is characterized by transmural acute necrotizing inflammation of medium to small sized arteries.

ETIOLOGY AND PATHOGENESIS:

The vascular lesions are mediated by type 111 hypersensitivity reaction.

(1) Formation of Antibodies:

i. PAN is often associated with hepatitis b- antigenemia and antibodies to HBAg play

a role in PAN.

ii. Some believe that the lesions of PAN resemble those in the Arthus reaction.

iii. Other believe that ANCA (anti-neutrophic cytoplasmic antibodies) are formed in some way. These are of 2 types:

* c-ANCA - stain cytoplasm diffusely and react with proteinase-3

* P-ANCA -» produce nuclear and perinuclear staining and react with Myeloperoxidase.

(2) Type III Hypersensitivity Reaction:

Antigen antibody complexes are formed -> activate the complement -> attract neutrophils -> release of enzymes and oxygen radicals -> endothelial damage -> vascular necrosis -> PAN.

MORPHOLOGY:

1. Location: It affects medium to small-sized arteries of any organ esp. Kidneys, heart. liver, GIT, pancreas, testis, skeletal muscle, neurons system, and skin with the exception of lungs and aorta with its primary branches.

2. Individual Lesions: are sharply segmental and tend to be located at branch points and bifurcations.

3. May Produce:

i. Aneurysmal dilatation

ii. Nodularity

iii. Intravascular thrombosis

iv. Ulceration, infarction or hemorrhages in the affected organs.

4. Microscopic PAN: shows no gross changes.

Microscopic:

1. Acute Stage:

Transmural inflammation of the arterial wall with neutrophils, eosinophils and mononuclear cells. Inflammation may extend into the adventitia.

ii. Fibrinoid necrosis of the inner half of the vessel wall.

iii. The lumen may become thrombosed.

2. Healing Stage:

i. Acute inflammatory infiltrate begins to disappear and is replaced by fibrous thickening and mononuclear infiltration of the vessel wall. The fibrosis may extend into the adventitia.

ii. Fibrosis produces firm nodularity of the vessels.

3. Healed Stage:

i. Marked fibrotic thickening of the affected vessel

ii. Inflammatory infiltrate absent

* In PAN all the three stages may coexist in different vessels or even within the same vessel.

CLINICAL FEATURES:-

1. Non-Specific Symptoms: Like malaise, fever, weakness and weight loss

2. Organ-Damage: Any organ except lungs:

* Kidneys ->Venous impairment, hypertensions

* Heart-> heart failure, Ml

* GIT -> abdominal pain, diarrhea and melena

* Nervous system -> peripheral neuropathy.

Incidence: Affects middle-aged persons. Male: female ratio is 2 : 1 or 3 :1.

COMPLICATIONS:

1. Aneurysmal dilatation.
2. Nodularity of the vessels.
3. Intravascular thrombosis
4. Vascular obstruction.
5. Infarction of the organs. .

LAB. DIAGNOSIS:

1. Blood Exam:

ESR T

2. Serological Tests:

ELISA can be done to detect serum liters of ANCA

3. Biopsy:

Histological exam of biopsy material taken from kidney and skeletal muscles, show characteristic lesions of PAN

INTRODUCTION OF DOPPLER STUDY

CAROTID EXAMINATIONS

PRACTICAL CONSIDERATIONS & TECHNIQUE - HEADINGS PATIENT COMFORT

- Explanation of technique
- Low pillow
- Knee support

SONOGRAPHER COMFORT

- Height adjustable bed and chair
- Leg room under bed
- Adjustable lighting

ULTRASOUND EQUIPMENT

- High resolution linear transducer
- Colour Doppler/Amplitude imaging
- Spectral Doppler with audible signal
- Adjustable sample volume size and depth
- Variable PW Doppler angle
- Adequate hard copy

B-MODE IMAGE OPTIMISATION

- Depth of field of view

Also modified by patient and transducer positioning

- Positioning of focal zone to region of interest
- Intima / media interface best visualised when 90° to ultrasound beam
- TGC and gain appropriately adjusted

B-MODE IMAGING OF CAROTID VESSELS

- Vessel pathway
- Level of bifurcation
- Vessel orientation
- Presence and location of disease
- Extent of disease
- Echogenic characteristics of plaque
- Transverse and longitudinal

CAROTID EXAMINATION

CAROTID ORIENTATION

- Longitudinal
- Head to left
- Feet to right
- Transverse
- Right side - medial right, lateral (left
- Left side - medial left. lateral right

VESSEL IDENTIFICATION

ECA

- Smaller diameter
- Medial with respect to ICA
- Extracranial branches
- High resistance flow
- Superficial temporal artery tap

ICA

- Larger diameter
- Lateral with respect to ECA
- Bulb
- No extra cranial branches (usually)
- Low resistance flow

COLOUR DOPPLER IMAGING

Colour presets for carotids

Colour assignment: although colour is based on the direction of blood flow (toward or away) in relation to the transducer, red is usually assigned to arteries and blue to venous blood flow

Appropriate colour map

Appropriate colour "velocity settings"

The colour scale (PRF) should be set for each individual patient by checking homodynamic appearance of colour map at the proximal CCA with the systolic upstroke being assigned a lighter hue. The colour can then be used

throughout the examination to evaluate the changing velocity patterns by observing any aliasing

Colour box size

The colour "box" width affects frame rates (number of image frames per second) so the colour display should be kept as small as possible

Colour wall filter is set medium (approx. 100Hz)

Colour gain, persistence Colour gain should be adjusted throughout the examination as the signal strength changes.

Colour filling of the vessel should be optimised to reach edges but not overwrite any plaque formation

PULSED WAVE DOPPLER IMAGING

- Sample volume placement
- Sample volume size
- Angle correction
- Reproducibility and error
- Artifacts
- Gain / spectral broadening

PW SPECTRAL MEASUREMENTS

- P.S,V. (peak systolic velocity)
- E.D.V. (end diastolic velocity)
- Velocity ratios
- Cardiac irregularity

- Aortic stenosis

CAROTID EXAMINATION OBJECTIVES

❖ Image the carotid vessels from origin to as far distally as possible

❖ Accurately identify the CCA, ICA, ECA, Vertebral & Subclavian arteries

❖ Detect the presence of pathology

➢ Stenosis

➢ Occlusion

➢ Carotid body tumour

❖ **Document B-mode characteristics of disease**

➢ Location

➢ Extent

➢ Severity

➢ Echogenicity

➢ Surface characteristics

❖ **Determine haemodynamic significance of disease**

➢ Category of stenosis

➢ Occlusion

➢ Retrograde flow

VERTEBRAL AND SUBCLAVIAN ARTERIES

❖ Vertebral

➢ Low resistance

➢ Antegrade flow

- Segments seen between transverse processes
- Where necessary, image vertebral origin

Vertebral Origin (Pipe Bowl & Stem)

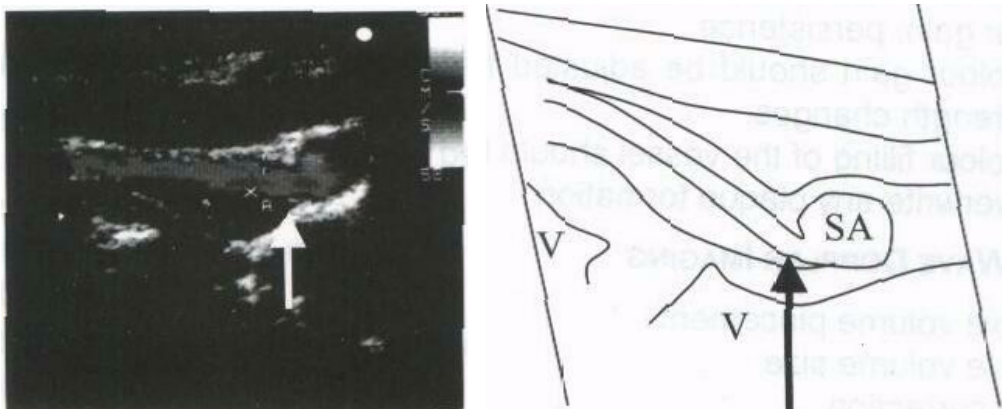

Fig. 16 subclavian artery

- **Subclavian**
- High resistance
- Right, from brachiocephalic artery
- Left. from aortic arch
- ✓ Left commonly associated with subclavian steal

SOME CRITERIA FOR ABNORMAL FINDINGS

EXTRA CRANIAL DISEASE

- Increased systolic velocities
- increased Diastolic velocities
- Increased ICA/CCA ratio
- Post stenotic turbulence
- Trickle flow
- No flow

INTRACRANIAL DISEASE 75% OR HIGHER

- Four "D's" - Distal Disease Decreases Diastole
- CCA, dampened waveform
- ICA, decreased diastolic flow
- ECA, normal flow pattern
- Contralateral, normal or increased flow

SUBCLAVIAN STENOSIS (STEAL)

- Varies with degree of stenosis
- Critical or occluded
- Reversed flow In ipsilateral vertebral
- Partial
  - Turbulence, decreased pulsatility,
  - bi-directional flow
  - Contralateral flow may be increased

ICA OCCLUSION

- Debris filled vessel
- No detectable flow
  - Increase sample volume size
  - Lower PRF, Lower Filters
- Absent wall pulsatility
- Ipsilateral CCA
- Dampened waveform

CCA OCCLUSION

- ECA - Retrograde flow
- ICA - Antegrade flow

Stroke Risk Factors, Warning Signs, And Symptoms

Several risk factors have been identified for stroke. Risk factors may be categorized into those that are not modifiable and those that are changeable or controllable.

NON-MODIFIABLE RISK FACTORS

- Age (risk increases with increasing age)
- Sex (incidence is higher in males, although females generally have a more severe deficit)
- Race (African-Americans have a higher stroke risk)

MODIFIABLE OR CONTROLLABLE RISK FACTORS

- Hypertension
- Elevated Cholesterol
- Smoking
- Atrial fibrillation & other cardiac diseases
- Obesity (patients have more hypertension, lipid abnormalities, etc.)
- Sedentary lifestyle

WARNING SIGNS

The five warning signs of stroke are:

- Sudden numbness or weakness of face, arm or leg, especially on one side of the body
- Sudden confusion, trouble speaking or understanding
- Sudden trouble seeing in one or both eyes
- Sudden trouble walking or experiencing dizziness, loss of balance or coordination
- Sudden headache with no known cause

CLASSIFICATION OF SYMPTOMS

- Stroke or cerebrovascular accident (CVA):
  - permanent ischaemic neurological deficit
- transient ischaemic attack (TIA):
  - ischaemic neurological deficit that lasts less than 24 hours
- amuarosis fugax:
  - transient partial or complete loss of vision in one eye
- he mi paresis:
  - unilateral partial or complete paralysis
- dysarthria:
  - difficulty with speech
- aphasia:
  - inability to communicate (speech, writing)

EXAMINATION PROTOCOL

The examination is explained and a history (risk factors, symptoms) is obtained from the patient.

Carotid imaging is performed with the patient lying in the supine position on

an examination table, with the sonographer seated at the patient's head. The patient's head is placed on a small round pad and turned slightly away from the side being scanned.

After applying ultrasound get to the neck, the transducer is placed midway between the clavicle and angle of the jaw in a transverse plane. The thyroid is located and the probe orientation checked. The CCA is located and is followed proximally as far as the clavicle will permit.

❖ Although the origin of the right CCA is often located as it arises from the innominate artery, the left CCA originates from the arch and is usually not accessible to ultrasound imaging.

❖ The transducer is moved cephalad following the CCA to the level of the carotid bifurcation (thyroid cartilage). B-mode images of the CCA S of the bifurcation are taken. Along with images to represent any disease location, extent & characteristics.

The transducer is rotated through 90° and the carotid system is examined in longitudinal section. The internal and external carotid arteries are individually followed distally to the angle of the mandible. Multiple longitudinal views (anterior, lateral, and posterior to the sternocleidomastoid muscle) are required to completely assess the cervical carotid arteries. Although the lateral approach provides the best visualization of the carotid system, the distal ICA is usually best visualised from a posterior approach.

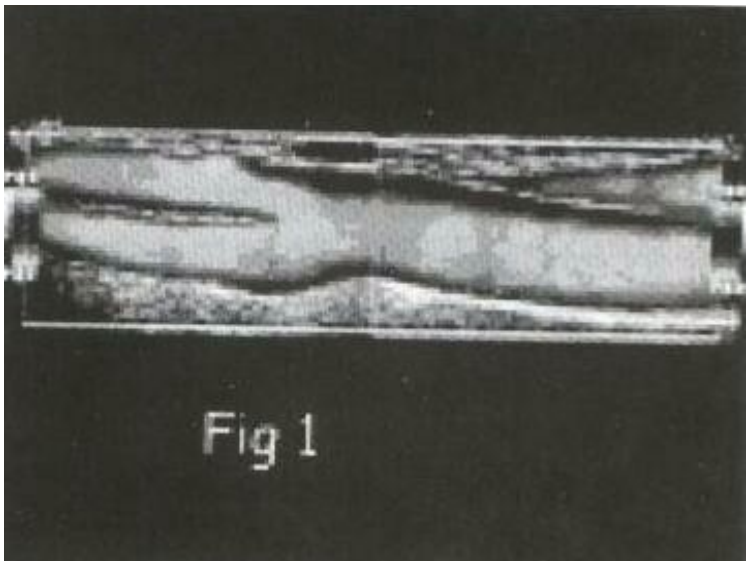

Fig. 17 Longitudinal image of the normal carotid artery and bifurcation into the internal and external carotid arteries.

Proper identification of the internal and external carotid arteries is not a problem in most patients. The most reliable method to distinguish the ICA from the ECA Is by the Doppler signal. The ICA has diastolic flow due to the low peripheral resistance of the brain, and the ECA demonstrates a more pulsatile Doppler signal (minimal diastolic flow) because it supplies blood to the muscular bed of the Jaw and face.

❖ Colour can be useful in this by observation of the pulsatility of the flow Additionally, the ECA is usually smaller in diameter, has cervical branches. and usually originates anterior and medial at the carotid bifurcation.

The vertebral arteries are located by angling the transducer slightly laterally from a longitudinal view of the mid/proximal CCA, The vertebral artery lies deeper than the CCA, to reliably Identify the vertebral artery, it should be followed distally, and periodic shadowing should be visualized from the

transverse processes of the vertebrae. The vertebral artery Is accompanied by the vertebral vein and proper identification of the artery is made by the Doppler signal. Once the vertebral artery has been correctly identified, it should be followed as far proximally as possible. The use of colour Doppler will greatly assist in locating the vertebral artery, its origin, and evaluating the direction of blood flow.

Doppler interrogation of the carotid system is performed in the longitudinal plane using a 60 degree or less angle between the ultrasound beam and the vessel walls (or parallel to the colour jet in advanced disease),

❖ Using a constant Doppler angle permits comparison of repeated studies in the same individual. However, some vessel orientations require angles of less than 60 in order to be accurate. In this case, documentation of the sample site and angle used should be made for follow up studies.

The sample volume size should be small and placed in the centre of the artery (or centre stream). The Doppler volume is moved slowly through the artery searching for the highest velocity- The colour Doppler display will help to guide the proper placement of the sample volume and is useful in locating sites of disease (aliasing).

Doppler signals are recorded from the mid and distal common carotid artery, the origin of the external carotid artery, the proximal, mid, and distal internal carotid artery, the origin of the vertebral artery, and the subclavian artery bilaterally. In addition, Doppler signals are obtained from any area of -stenosis. It is important to evaluate all Doppler signals bilaterally to correctly perform a carotid duplex imaging examination in an individual patient.

The location of any plaque visualised during the examination should be described, as well as its surface characteristics (smooth vs irregular) .and echogenicity (calcification).

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(or centre stream). The Doppler volume is moved slowly through the artery searching for the highest velocity- The colour Doppler display will help to guide the proper placement of the sample volume and is useful in locating sites of disease (aliasing).

Doppler signals are recorded from the mid and distal common carotid artery, the origin of the external carotid artery, the proximal, mid, and distal internal carotid artery, the origin of the vertebral artery, and the subclavian artery bilaterally. In addition, Doppler signals are obtained from any area of -stenosis. It is important to evaluate all Doppler signals bilaterally to correctly perform a carotid duplex imaging examination in an individual patient.

The location of any plaque visualised during the examination should be described, as well as its surface characteristics (smooth vs irregular) .and echogenicity (calcification).

Proper identification of the internal and external carotid arteries is not a problem in most patients. The most reliable method to distinguish the ICA from the ECA Is by the Doppler signal. The ICA has diastolic flow due to the low peripheral resistance of the brain, and the ECA demonstrates a more pulsatile Doppler signal (minimal diastolic flow) because it supplies blood to the muscular bed of the Jaw and face.

❖ Colour can be useful in this by observation of the pulsatility of the flow Additionally, the ECA is usually smaller in diameter, has cervical branches. and usually originates anterior and medial at the carotid bifurcation.

The vertebral arteries are located by angling the transducer slightly laterally from a longitudinal view of the mid/proximal CCA, The vertebral artery lies deeper than the CCA, To reliably Identify the vertebral artery, it should be followed distally, and periodic shadowing should be visualized from the transverse processes of the vertebrae. The vertebral artery Is accompanied by

the vertebral vein and proper identification of the artery is made by the Doppler signal. Once the vertebral artery has been correctly identified, it should be followed as far proximally as possible. The use of colour Doppler will greatly assist in locating the vertebral artery, its origin, and evaluating the direction of blood flow.

Doppler interrogation of the carotid system is performed in the longitudinal plane using a 60 degree or less angle between the ultrasound beam and the vessel walls (or parallel to the colour jet in advanced disease),

❖ Using a constant Doppler angle permits comparison of repeated studies in the same individual. However, some vessel orientations require angles of less than 60 in order to be accurate. In this case, documentation of the sample site and angle used should be made for follow up studies.

The sample volume size should be small and placed in the centre of the artery (or centre stream). The Doppler volume is moved slowly through the artery searching for the highest velocity- The colour Doppler display will help to guide the proper placement of the sample volume and is useful in locating sites of disease (aliasing).

Doppler signals are recorded from the mid and distal common carotid artery, the origin of the external carotid artery, the proximal, mid, and distal internal carotid artery, the origin of the vertebral artery, and the subclavian artery bilaterally. In addition, Doppler signals are obtained from any area of -stenosis. It is important to evaluate all Doppler signals bilaterally to correctly perform a carotid duplex imaging examination in an individual patient.

The location of any plaque visualised during the examination should be described, as well as its surface characteristics (smooth vs irregular) .and echogenicity (calcification).

It is very important for patient management to differentiate between a high

grade stenosis versus an occlusion of the ICA. An ICA occlusion is not amenable to surgical intervention. To characterise an ICA as occluded, the artery should be evaluated with Doppler, colour Doppler, and power Doppler to rule out the presence of trickle flow When determining if the ICA is occluded, the colour PRF should be decreased to document the presence of any slow moving blood flow and the colour gain should be increased to enhance any blood flow. Secondary ultrasound characteristics of an ICA occlusion are: echogenic material filling the lumen, lack of arterial pulsations, reversed colour blood flow near the origin of the occlusion, and the loss of diastolic blood flow (damping) in the ipsilateral CCA,

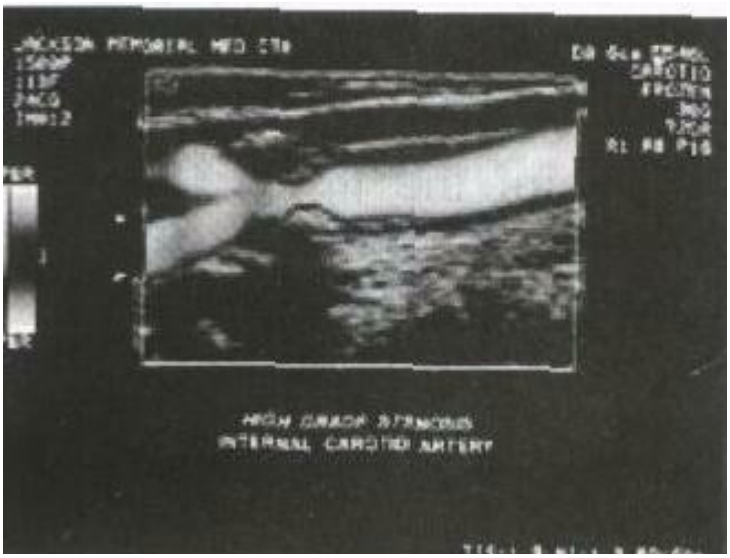


Fig. 18 High grade stenosis is shown Just distal to the bifurcation of the common carotid artery, (see area of narrowing)

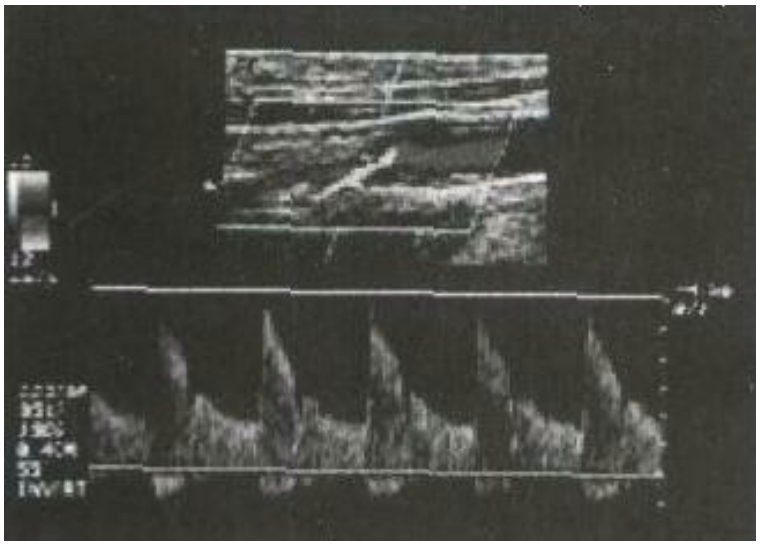


Fig. 19 Colour Doppler image shows aliasing (blue/yellow colour), the Doppler sample volume is placed within the area of highest velocity (turbulence) and the spectral waveform shows an abnormal waveform of 4.5 m/sec in this patient with internal carotid artery stenosis.

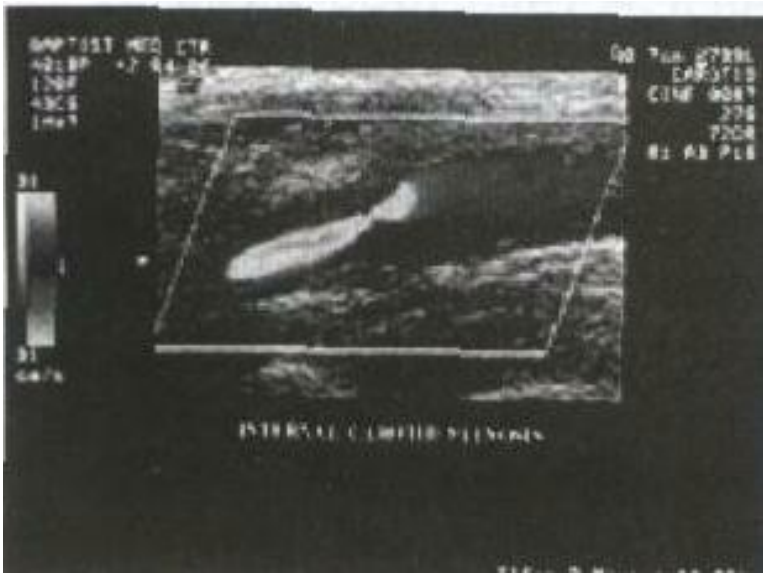


Fig. 20 Colour Doppler shows a longitudinal image of a patient with internal carotid artery stenosis. The internal carotid artery shows the area of narrowing with increased (aliased) flow denoted by the blue/yellow pattern.

In addition to documenting the presence of atherosclerotic disease during a

carotid duplex imaging examination, the sonographer should also be aware that carotid aneurysms, dissections, and carotid body tumours may be encountered. Fig.

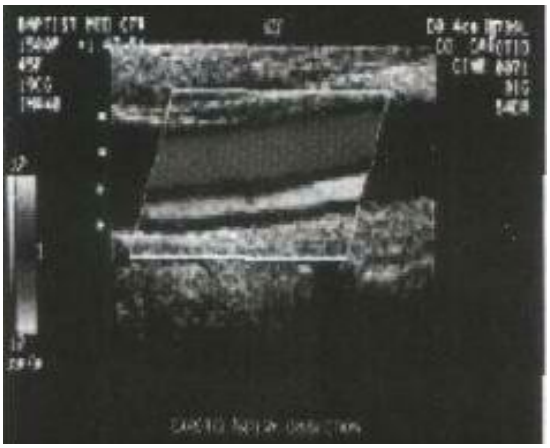


Fig. 21 A patient with a carotid artery dissection shows the "false" channel (yellow-orange) as separate from the normal lumen (red),

INTERPRETATION

The accurate interpretation of a carotid duplex imaging examination depends upon the quality and the completeness of the evaluation. Often the patient's body habitus will affect the quality of the image and the sonographer's ability to search the entire carotid system with Doppler. The sonographer must be prepared to switch transducers if necessary to complete the carotid examination and have a complete understanding of the equipment controls to optimise the duplex imaging system.

In addition to the peak systolic velocity, end diastolic velocity, and direction of blood flow, the shape of the Doppler spectral waveforms should be evaluated bilaterally. Abnormal waveform shape (increased or decreased pulsatility) may be an indicator of more proximal (innominate, subclavian) or distal (intracranial) disease.

To determine the degree of stenosis present, a complete Doppler evaluation of the artery is necessary. There should be an elevated velocity through the

narrowed segment and post-stenotic disturbances distal to the stenosis. The highest velocity obtained from a stenosis is used to classify the degree of narrowing, along with other secondary criteria such as end diastolic velocity, diagnostic criteria chart), Doppler signals obtained distal to the area of post-stenotic flow disturbance may be normal or diminished, and the upstroke of the distal Doppler spectral waveform may be slowed.

Because the recent carotid enarterectomy trials [North American Symptomatic Carotid Endarterectomy Trial (NASCET), Asymptomatic Carotid Atherosclerosis Study (ACAS), European Carotid Surgery Trial (ECST)] used specific thresholds for surgical treatment, ultrasound criteria for ICA stenosis >70% and >60% were needed to classify patients. Investigators have found that an ICA/CCA peak systolic velocity (PSV) ratio useful in grading ICA stenosis >70% and >60%. The following ratios are calculated using the highest PSV from the origin of the ICA divided by the highest PSV from the CCA (distal).

DIAMETER REDUCTION	ICA/CCA PSV RATIO
70-99%	>45
60-99%	>3.26

In the presence of a contralateral ICA occlusion the velocity from the ipsilateral ICA may be elevated. This may lead to over reading the extent of the ipsilateral ICA disease.

The evaluation of normal vertebral arteries produces a wide variation in velocities. Absolute velocities are not useful to diagnose a stenosis post-stenotic disturbances in the vertebral artery and changes in the waveform shape may suggest a proximal obstruction. It is important to note the direction of blood flow in the vertebra! artery. A subclavian steal is present if there is

reversal of vertebral artery blood flow direction secondary to a significant obstruction in the ipsilateral subclavian or innominate artery. The arterial obstruction must be located proximal to the origin of the vertebral artery. A vertebral artery Doppler signal can also display an alternating (toward and away) pattern. If an alternating pattern occurs in the vertebral artery it may change to reversal of blood flow direction with arm exercise or after reactive hyperaemia of the Ipsilateral arm induced by maintaining a blood pressure cuff inflated above systole for approximately 3 minutes.

The evaluation of normal subclavian arteries produces multiphasic high-resistance Doppler signals. If there Is a significant stenosis or occlusion of the proximal segment of this vessel then the Doppler signal distal to the stenosis is monophasic, A difference of blood pressure in the arms greater than 20mmHg is usually associated with significant disease of the subclavian artery on the side with the lower blood pressure.

Other important information to include In the interpretation of a carotid duplex imaging examination is: (1) the location of the stenosis. (2) the extent of the plaque and patency of the distal ICA> (3) the presence of tortuosity or kinking of the vessels, and (4) plaque characteristics (smooth vs irregular surface, calcification).

SUMMARY

The best carotid duplex imaging examinations will be achieved by the following suggestions:

(1) Optimise the gray scale image and know how each colour control affects the image and how the different instrument controls affect each other

(2) Compare the right and left side.

(3) Be aware of the spectral Doppler waveform configuration which may

suggest proximal or distal pathology-

(4) Use a consistent Doppler angle where possible and document angles and measurement sites on the worksheet,

(5) Use the colour Doppler and power Doppler display as a guide, to the Doppler examination, and

(6) Each institution should establish its own diagnostic criteria.

Diameter stenosis	**Peak systolic (m/s)**	**End diastolic (m/s)**	**Ica / cca**	**B-scan/colour**	**Clinical features**
0	<1.25	<0.40		Normal	Normal
1-15	<1,25	<0.40		Intima! thickening Minor Plaque	Minimal disease
16-49	<1.25	<0.40		Moderate Plaque	Moderate disease
50-69	>1.25	>0.40	>2	Colour flash Aliasing Prominent Plaque	Prominent Haemodynamicall y significant
70-79	>2.70	>1.10	>4	Colour Flash # Advanced Plaque	Prominent Haemodynamicall y significant stenosis, suggest surgical opinion

80-99	>2.70	>1.40	>4	Very narrow colour flash ## in thick plaque	Prominent Haemodynamically significant stenosis, suggest urgent surgical opinion
Occluded				No Flow Total luminal plaque or thrombus	Occluded

COLOUR DUPLEX DOPPLER ULTRASOUND

EXTRACRANIAL CAROTID ARTERY DISEASE

These guidelines for the reporting of extracranial carotid artery disease have been adopted by ASUM to assist those performing and reporting these examinations. The threshold values are recommendations only and older values may have similar validity. Laboratories should attempt to audit the results they obtain with the criteria they choose to use,

Stanosis Grade	Ultrasound Criteria - 1CA
0	Normal waveform and image
< 15% diameter reduction	Deceleration spectral broadening PSV < 125 cm/sec
16-49% diameter reduction	Pansystolic spectral broadening PSV < 125 cm/sec
50-69% diameter reduction	• Pansystolic spectral broadening • PSV > 125 cm/sec and EDV < 110 cm/sec or • ICA/CCA > 2
70 - 79% diameter reduction	• Pansystolic spectral broadening • PSV > 270 cm/sec or • EDV ~> 110 cm/sec or • ICA/CCA > 4
80-99% diameter reduction	As above plus • EDV > 140 cm/sec
Occluded	No flow Terminal thump

1CA	Internal carotid artery
CCA	Common carotid artery
PSV	Peak systolic velocity
(FDV	End diastolic velocity
ICA/CCA	Ratio of 1CA PSV lo CCA PSV

Plaque Classification	Surface	Bifurcation	Tortuosity	Technical
echogenic	smooth	normal	minimal	poor
hypoechoic	irregular	high	moderate	good
mixed	indeterminate	low	maximum	excellent
calcification				
indeterminate				

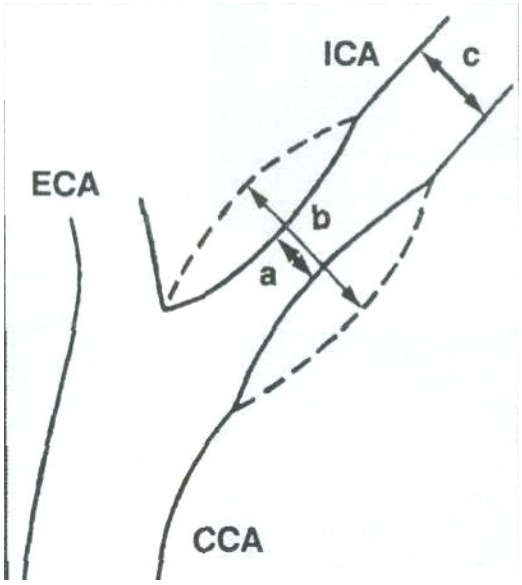


Fig. 22 NASCET method of ICA stenosis grading (1- a/c x100%)

For grades 15 - 50%. attempt to estimate per cent diameter reduction by visual assessment and preferably caliper measurements of lumen. Diameter reduction is measured according lo NASCEET method (see diagram above), Evidence suggests greatest benefit from surgery in > 70% and possibly > 50%

stenosis grades but me need to intervention is influenced by patient's symptoms and other factors.

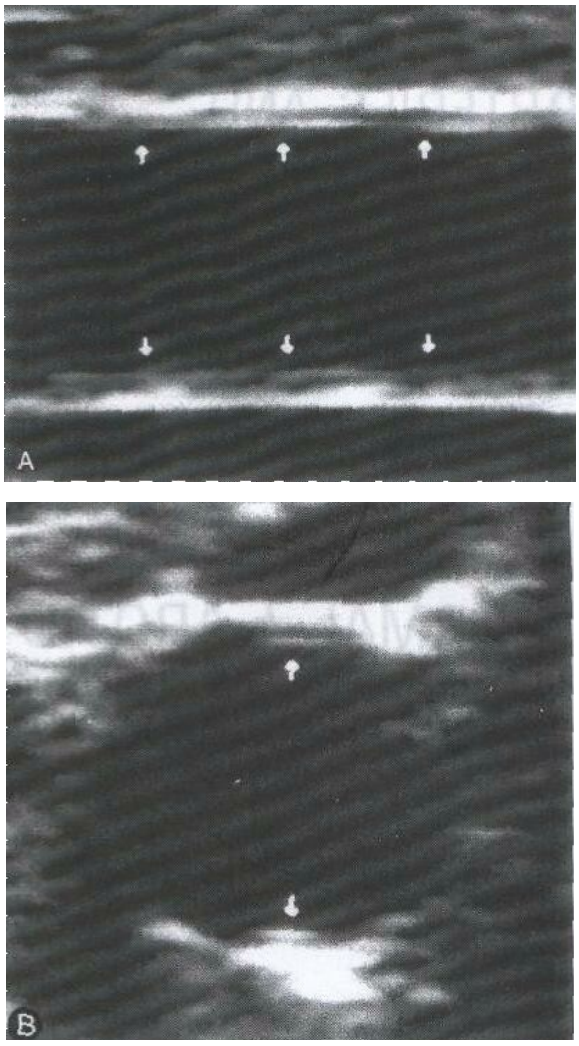


Fig. 23 Normal arterial anatomy. Longitudinal (4) and transverse (B) images of the common carotid artery demonstrate a sharp line (specular reflection) that emanates from the intimal surface (arrows). The black line peripheral lo this reflection represents the media of the artery. The outermost white line is the adventitia of the artery.

Distinguish between the ICA and ECA, section. Furthermore, altered carotid pulsatilitv is an important clue about the presence of carotid occlusive disease. In some cases, pulsalility changes are the only indication of abnormality.

The normal range of velocities in the CCA, ICA, and EGA has not been studied extensively and velocities may vary with physiologic differences among individuals. With regard lo peak systolic velocity in the ICA, reported mean values for normal adults range from 54 to B8 cm/sec (in the postbulbar region). Peak systolic velocities as high as 120 cm/sec have been reported in some normal individuals, but these values are exceptional and an ICA velocity exceeding 100 cm/sec should he viewed as potentially abnormal. Peak systolic velocity in the ECA is reported as 77 cm/sec (mean) in normal individuals, and the maximum velocity does not normally exceed 115 cm/sec- Consider able patient-to-patient variability occurs in EGA flow velocity, however, particularly if

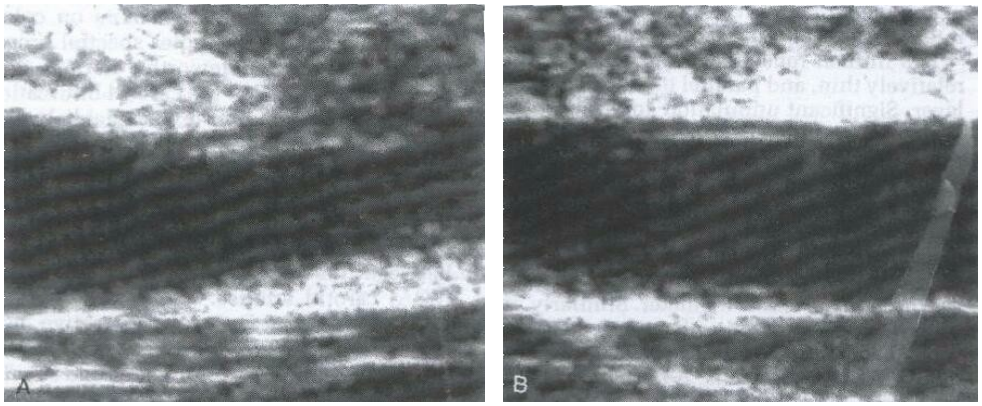


Fig. 24 False-positive plaque diagnosis due to off-diameter image plane. A, It appears that plaque is present, resulting in stenos is, in this off-diameter section of the common carotid artery. B, The same artery is demonstrated lo be normal by moving the transducer very slightly, such that the plane of t tie section passes through the diameter of the vessel. Note the clearly seen intimal reflections.

Laminar flow pattern. Darker red shades are seen at the periphery of this common carotid artery, because flow is slower near the wall. Lighter colors are present throughout the rest of the vessel, in which flow is faster.

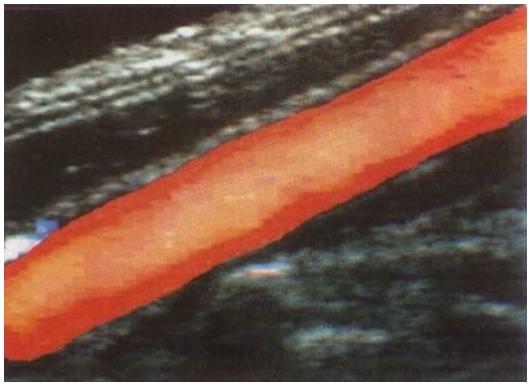
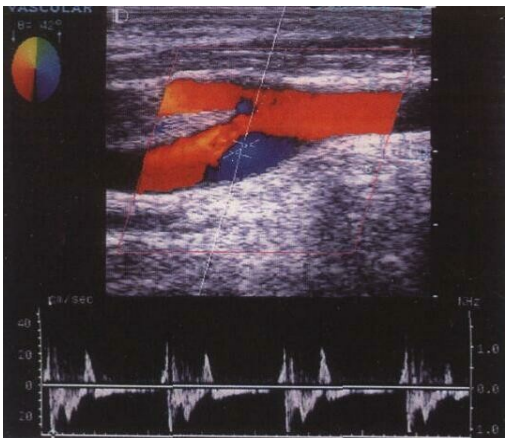
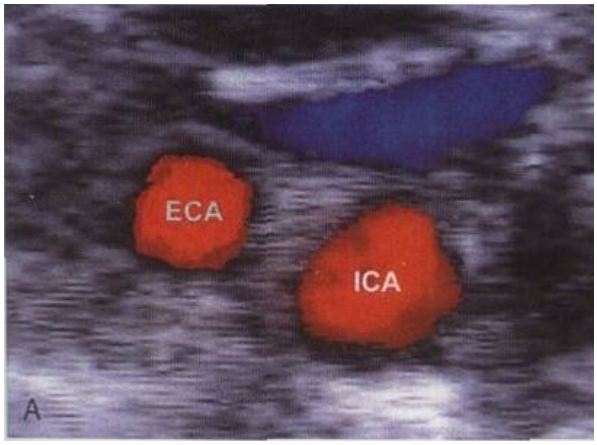


Fig. 25 Long-axis view of the carotid bifurcation. The blue area in the bulbous portion of the internal carotid artery represents the normal flow reversal zone. The Doppler spectrum seen at the base of the image shows a disturbed to-and-fro pattern caused by the combined forward- and reverse-flow components present in the bulbous portion.





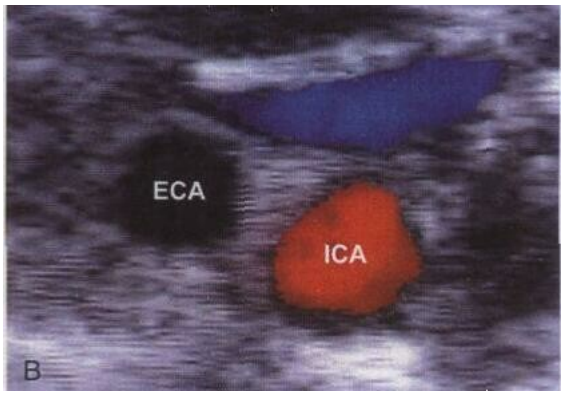


Fig. 26 B-a Color- Doppler visualization of pulsatilitv patterns A, In systole, flow (rod color) is evident in both the internal (ICA) and external (EGA) carotid arteries. S, In diastole, flow is absent in the external carotid artery (ECA) but persists In the internal carotid artery <ICA). When seen in real time, the EGA "blinks" on and off, whereas the ICA undulates in brightness,

Normal Carotid Arteries and Carotid Examination Technique

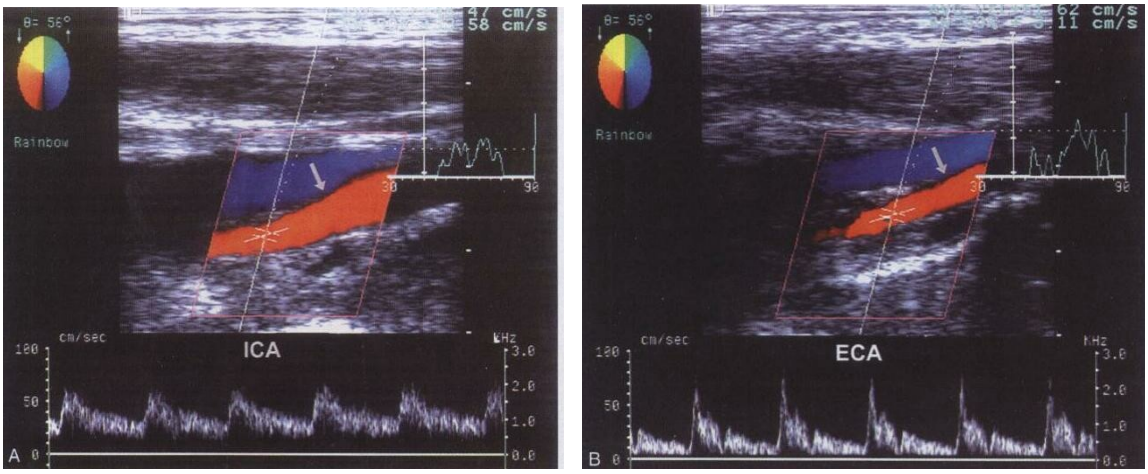


Fig. 27 Identifying the internal and external carotid arteries. By shifting back and forth between the internal (A) and the external (8) carotid arteries (ICA and ECA, respectively), the sonographer has determined that the junction of the vessels is at the approximate location of the arrows. The pulsatilitv of the ICA is clearly different from that of the EGA.

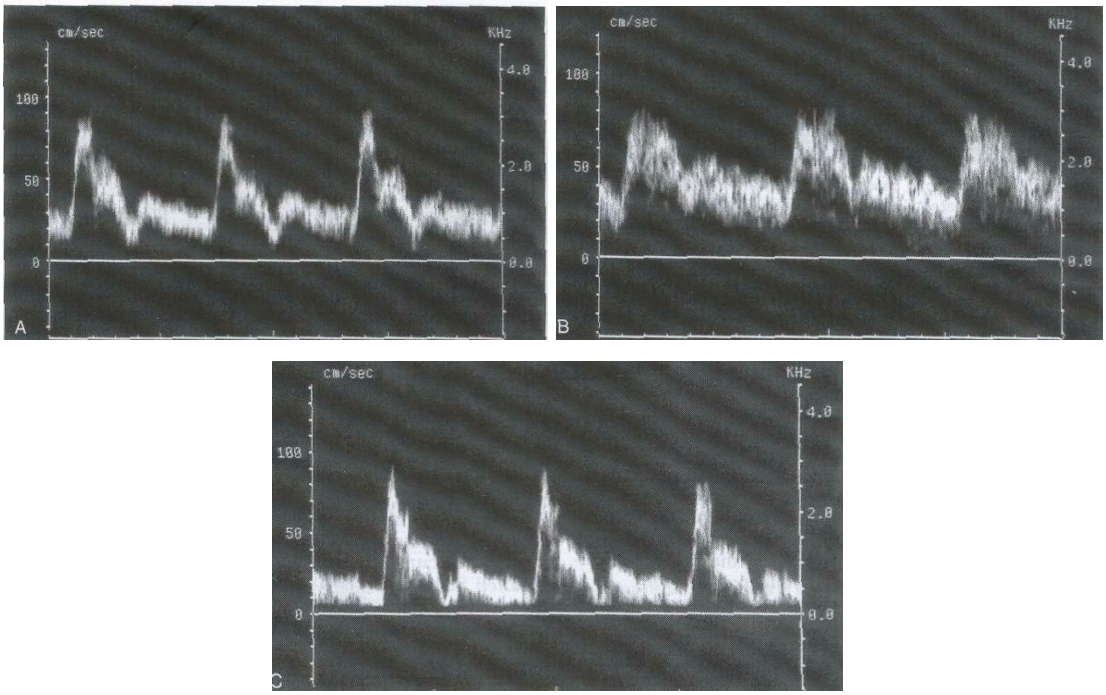


Fig. 28 Normal carotid artery Doppler waveform. A, Waveforms in the common carotid artery have moderately broad systolic peaks and a moderate amount of flow throughout diastole. B. The internal carotid artery waveforms have broad systolic peaks and a large amount of flow throughout diastole. Audible Doppler signals in the internal carotid artery have a smoothly undulating sound. C. External carotid artery waveforms have sharp systolic peaks and relatively little flow In diastole. The audible signals in the external carotid artery have a whip-like sound that is usually quite different from the sound of the internal carotid artery Doppler signals,

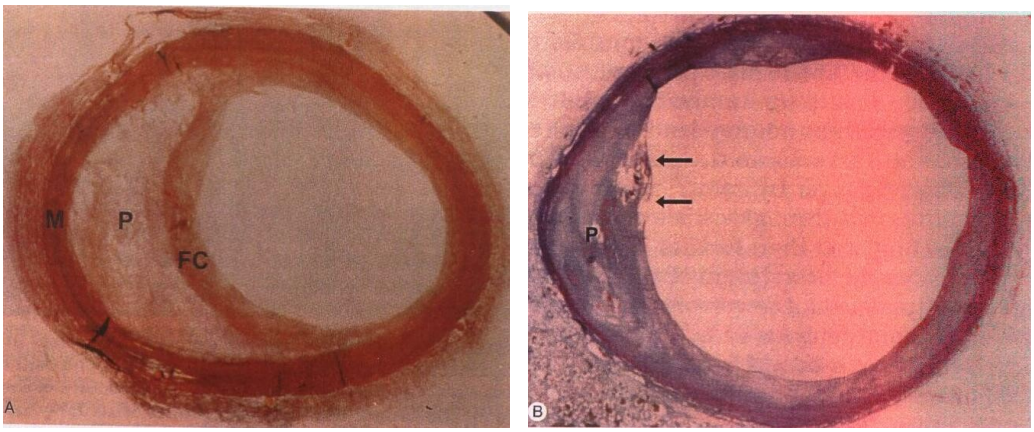


Fig. 29 Plaque histology. A. Microscopic section of an uncomplicated plaque (P), The fibrous cap FC) is intact and the plaque contents (P) are homogeneous. M = muscularis. B. Microscopic section of a complicated plaque. The fibrous cap is ruptured and an area to cavilation is present {arrows} The plaque contents (P) are heterogeneous. (From O'Leary D, olagov S, Zarlns C, Giddens D Carotid artery disease. In RIfkin W, Charboneau JW, Laing FC [eds]: Ultrasound 1991. Special Course Syllabus, 77tn Scientific Assembly and Annual Meeting. Oak Parti, IL, RSNA Publications, 1991, pp 189-200. Reproduced with the kind pennisaon of Daniel O'Leary MD.)

INTERNATIONAL STUDY

1. ARTICLES

Cerebrovascular evaluation: assessment of Doppler scanning of carotid arteries, ophthalmic Doppler flow and cervical bruits

RD Shoumaker and S Bloch

In 38 patients who underwent cerebrovascular evaluation followed by angiography the Doppler ultrasound scanning technique was found to be an excellent screening procedure for detecting marked stenosis (greater than 50%) or occlusion of the internal carotid artery (93% correlation). It is noninvasive, easily reproducible and can be performed by a qualified technician. The major problems are: the inability to detect ulcerated plaques without marked stenosis, the requirement for patient cooperation (lying still for periods of 15 minutes), and the fact that it assesses only the extracranial circulation. Screening with just the directional ophthalmic Doppler flow signal yielded a high percentage of false negatives (13%). The presence of a cervical bruit may indicate an underlying stenosis of the internal carotid artery, but may also be due to stenosis of the external carotid artery or other factors such as increased blood flow, vessel tortuosity, etc. (12% false positives). Absence of a cervical bruit does not exclude internal carotid artery disease (ulcerated nonstenotic plaque or occlusion).

2. INTEROBSERVER VARIABILITY IN THE ASSESSMENT OF CAROTID DISEASE BY DOPPLER ULTRASOUND: IMPLICATIONS FOR STROKE PREVENTION.

Mead GE, Lewis SC, Wardlaw JM.

Annu Meet Int Soc Technol Assess Health Care Int Soc Technol Assess Health Care Meet. 1999; 15: 81.

Department of Clinical Neurosciences, Western General Hospital, Edinburgh EH4 2DN, UK.

OBJECTIVE

Duplex Doppler ultrasound (DUS) alone in the assessment of internal carotid artery (ICA) disease prior to endarterectomy avoids the risks of angiography, is quick to perform, uses accessible equipment and is inexpensive. Two DUS examinations by different observers might be adequate to confirm the severity of ICA stenosis but it is not known whether there is clinically important interobserver variability in the assessment of ICA stenosis. Our aim was to measure the interobserver variability of DUS. METHODS: Consecutive patients attending for carotid DUS underwent two examinations on the same day by two of three observers (an experienced neuroradiologist, observer 1; an experienced radiographer, observer 2, and a stroke physician with three months experience of DUS, observer 3). All observers were blind to the reasons for referral and each others' results. Severity of stenosis was assessed by velocity measurements and the lesion appearance. RESULTS: A total of 189 patients were scanned (378 ICAs). Of the 134 ICAs scanned by observers 1 and 2, observer 1 classified 11 as >80% stenosis, compared with only 9 by observer 2. Thus, among those classified as >80% stenosis by observer 1, 18% were classified differently by observer 2. Of the 206 ICAs scanned by

observers 1 and 3, observer 1 classified 11 as >80% stenosis compared with only 5 by observer 3 (55% discrepancy). Of the 38 ICAs scanned by observer 2 and 3, observer 2 classified 2 as >80% stenosis compared with none by observer 3 (100% discrepancy). CONCLUSIONS: There was clinically important interobserver variability in the assessment of ICA disease by DUS. It would be unwise to perform carotid endarterectomy on the basis of a single Doppler examination as there may be considerable disagreement on the number of operable stenoses. If two observers disagree on the appropriate treatment, then a third opinion should be sought.

Publication Types:

- Meeting Abstracts

Keywords:

- Angiography
- Carotid Artery, Internal
- Endarterectomy, Carotid
- Humans
- Observer Variation
- methods
- radiography
- surgery
- therapy
- hsrmtgs

Other ID:

References

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3. INCREASED CAROTID ARTERY INTIMA-MEDIA THICKNESS IN SUBJECTS WITH PRIMARY HYPOALPHALIPOPROTEINEMIA

Damiano Baldassarre; Mauro Amato; Linda Pustina; Elena Tremoli; Cesare R. Sirtori; Laura Calabresi; Guido Franceschini

Ultrasonography

Ultrasound scanning and reading of carotid arteries were performed by a single expert sonographer, using an 8-MHz transducer with an axial and lateral resolution of ≈0.385 and ≈0.500 mm, respectively.[6,21] The sonographer was blinded to the subject's identity. A standard protocol, scanning the near and far walls of the right and left common, internal and external carotid arteries, and bifurcations in three different projections (anterior, lateral, and posterior), was performed.[21] Eight segments of the right and left carotid arteries in each projection were examined, and the 48 IMT measurements were averaged to calculate the average IMT (Avg-IMT) for each subject. Less than 4% of all IMT measurements was missed because of anatomical reasons; the missing value was replaced in the calculation of Avg-IMT with the average of the remaining measurements for that segment. The highest IMT value among the 48 segments was defined as the maximum IMT (Max-IMT). As age has been shown to be the strongest predictor of IMT,[7,22] age-adjusted IMT values were used in some analyses.

Statistical Analysis

The number of subjects needed to detect a difference in Avg-IMT between the three groups of 0.20 mm with an SD of 0.30 mm, a power of 80%, and α=0.05

is 45 per group. Therefore, the study is adequately powered to disprove multiple null hypotheses.

Results are reported as mean± SEM, if not otherwise stated. Logarithmic transformation was performed on individual values of skewed variables. Group differences in continuous and categorical variables were determined by using ANCOVA and a χ^2 test of significance, respectively. Pearson correlation coefficients were computed to assess the association between parameters. Multiple stepwise regression analysis was performed with Avg-IMT or Max-IMT as the dependent variable, and by entering the independent variable with the highest partial correlation coefficient at each step, until no variable remained with an F value of 4 or more. Group differences or correlations with $P<0.05$ were considered statistically significant.

Results

The characteristics of the recruited subjects are shown in Table 1. A trend toward lower BMI and fasting glucose concentrations with increasing average HDL-C levels was found, possibly indicative of the presence of some insulin-resistant subjects in the Hypo ALP group; none of them, however, was diabetic. Plasma triglycerides were significantly higher in Hypo ALP than in Hyper ALP and control subjects. There was no difference in plasma Lp(a), blood pressure, or prevalence of smokers in the three examined groups.

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COMPARISON OF MY STUDY WITH THREE INTERNATIONAL STUDIES

When I am going to compare my study with international studies I found that ultrasonography is excellent screening procedure measuring normality about carotids

With respect of IMT, PSV and ICA/CCA Ratio. It is good modality to detect mild to marked stenosis with comparison to international study for symptomatic patients to find pathology in carotids like atherosclerosis, aneurysm and also for thrombotic lesions but my study was on Asymptomatic normal patients.

Since it is non invasive, quick inexpensive procedure. We can also avoid the risks of angiography. Before referring to surgeon about three inter observer studies should be taken.

We can match our results about degree of stenosis with results of angiography.

We can extend our study with respect to all risk factors about stenosis of carotid like smoking, Hyperlipidemia, hypertension, obesity and etc.

CHAPTER THREE

MATERIAL AND METHODOLOGY

Research Method

The study will be taken at Afro Asian Institute of Medical Sciences Lahore Pakistan. Intima media thickness, pathological lesions and color and sF5cmd"analysis, will be taken in Pakistan population.

Each patient will be scanned under scanning guide lines by candidate and diagnosis will be confirmed by highly qualified sonologist.

All daily data sheets will be signed by candidate's local supervisor who will see the details of causes and will check sonograms with pathological findings.

Facilities Available

1 Toshiba: Nemio - XG 3 - D

Convex probe, linear probe. Color and power Doppler Directional angio. Tissue harmonics,

2 Toshiba: Xario 4-D

With linear probe and convex

Carotid assessment in Pakistani population

Common carotid and internal carotid Arteries

For research topic, the target population will be patients either asymptomatic or symptomatic for carotids.

Data Storage

All data collected will he stored on computer protected by password. All paper format data will be stored in locked cabinet.

Data Analyses

The analysis data is done to evaluate intima-media thickness, spectral color and Doppler assessment of pathologies.

Application of advantages and characteristics of Doppler ultrasound investigation to carotid arteries.

Conclusions

The results will be conducted from processed data and will be discussed in detail to see the carotid in Pakistan population and its comparison with international standard data-Discussion will be done and result of study will be summarized to draw conclusion.

CHAPTER FOUR

RESULT

The study of character Arteries U, S, C. was carried out in a Afro Asian Institute of Medical sciences at Shadman Lahore Pakistan.

One hundred individuals of both sides of different ages for randomly for ICA/CCA ratios of both sides of arteries.

Method of Data Collection

The target population of my study was all patients coming for U.S.O for agencies of their diseences but on my request Institute allowed me to Sean their carotids.

Techniq ues

Machine used are:

1. Toshiba Nemio x G with Colour Spectral Doppler and tissues harmonics e 9- 11 MHz linear problem.
2. X erio lives Colour +Power, Colour and spectral Doppler, Tissues harmonics 4 D with linear Problem. Frequently used 9- 11 MHz Angle used for meaning valuation is 55.

Individual Scanned

Name and identify Specification are not requested in data Sheet.

I asked about Age Sex, family history of CVA, Obesity smoking occupation and lipid profile (but no one presented it) Because all about are rile feature for carotid Pathology. All patient visited to me were asymtontable.

Scanning Technique

The Know scanning protocols were followed for carotid arteries scanning in study.

Age

In my study out of hundred individuals 10% are of Age ranges 20-29 years. 20% are of Age ranges 30-39years. 19% are of age ranges 40-49years 13% are of age ranges 50-59years 21% are of age ranges 60%69year. 14% are of age ranges of 70-79Years. 2% of age ranges of 80-89%years

Occupation

Wide range of occupation individuals came with my study 30% are house wives. 26% of individuals were Employee in different apartments of life. 27% were students of different subjects. 10% were teacher of different schools, colleges and Universities 7% were doctors of different Hospitals.

Obesity:-

Out of 100 Individuals, 21% were Obese and 79% of them were no obese.

Smoking

As Smoking is also risk feature. 29% of individuals were smoking where are 71% of individuals were Non Smokers.

Intima – Media Thickness

Individuals Scanned out of hundred. 21% of the person with 0.2 -0.3mm on both sides. 21% with IMT of 0.4-0.5mm on Right side. , 22% of 0.4-0.5mm

on left side. 35% on Right and 33% on left sides with IMT ranges 0.6-0.7mm. 21% of right and 23% of left sides with ranges up to 0.8mm. 21% of right side and 20% of left sides were above or equal to 0.9mm.

Peak Systolic velocities of ICA

1% Individual of both sides with Velocities ranges 50-59 cm per second. 50% on right side and 47% on left side with Velocities ranges 60-69 cm per second. 44% on right and 43% on left side individuals with Velocities ranges 70-79 cm per second. 5% on right side 8% on left sides of individuals having Velocities ranges 80-89 cm per second. Only one 1% of left side with Velocities ranges 90-99 cm per second.

ICA/CCA Ratios

In study, 27% of right side, 25% of left side of individuals having ratios ranges 1.0-1.09.

39% of right side, 40% of left side of individuals having ratios ranges 1.10-1.19

30% of right side, & 28% of left side of individuals having ratios ranges 1.20-1.29.

3% of right side, 6% of left side of individuals having ratios ranges 1.30-1.39.

1% of right side, 1% on left side of individuals having ratios ranges 1.40-1.80.

TABLES AND GRAPHS

Table: 1. The Age group of 100 individuals for carotid scan at Afro-Asian Center Shadman Lahore Pakistan from Feb 2007 to Feb 2008. Age ranges with there percentage.

Age Group (Yrs)	No of Individual	Percentage
20-29	10	10%
30-39	21	21%
40-49	19	19%
50-59	13	13%
60-69	21	21%
70-79	14	14%
80-89	2	2%

Summarize

Case Processing Summary(a)

	Cases					
	Included		Excluded		Total	
	N	Percent	N	Percent	N	Percent
NoOfPatients	7	100.0%	0	.0%	7	100.0%
percentage	7	100.0%	0	.0%	7	100.0%

a Limited to first 100 cases.

Case Summaries(a)

		NoOfPatients	percentage
1		10.00	.10
2		21.00	.21
3		19.00	.19
4		13.00	.13
5		21.00	.21
6		14.00	.14
7		2.00	.02
Total	N	7	7
	Mean	14.2857	.1429
	Std. Deviation	6.87300	.06873
	Range	19.00	.19
	Std. Error of Skewness	.794	.794

a Limited to first 100 cases.

Descriptives

Descriptive Statistics

	N	Minimum	Maximum	Mean	Std. Deviation
NoOfPatients	7	2.00	21.00	14.2857	6.87300
percentage	7	.02	.21	.1429	.06873
Valid N (listwise)	7				

T-Test

One-Sample Statistics

	N	Mean	Std. Deviation	Std. Error Mean
NoOfPatients	7	14.2857	6.87300	2.59775
percentage	7	.1429	.06873	.02598

One-Sample Test

	Test Value = 0					
					95% Confidence Interval of the Difference	
	t	df	Sig. (2-tailed)	Mean Difference	Lower	Upper
NoOfPatients	5.499	6	.002	14.28571	7.9293	20.6422
percentage	5.499	6	.002	.14286	.0793	.2064

Two-stage Least Squares Analysis

Model Description

		Type of Variable
Equation 1	percentage	dependent
	NoOfPatients	predictor & instrumental

MOD_6

Model Summary

Equation 1	Multiple R	1.000
	R Square	1.000
	Adjusted R Square	1.000
	Std. Error of the Estimate	.000

ANOVA

		Sum of Squares	df	Mean Square	F	Sig.
Equation 1	Regression	.028	1	.028	.000	.000
	Residual	.000	5	.000		

Total	.028	6			

Coefficients

		Unstandardized Coefficients		Beta	t	Sig.
		B	Std. Error			
Equation 1	(Constant)	-1.85E-017	.000		.	.
	NoOfPatients	.010	.000	1.000	.	.

Graph: 1

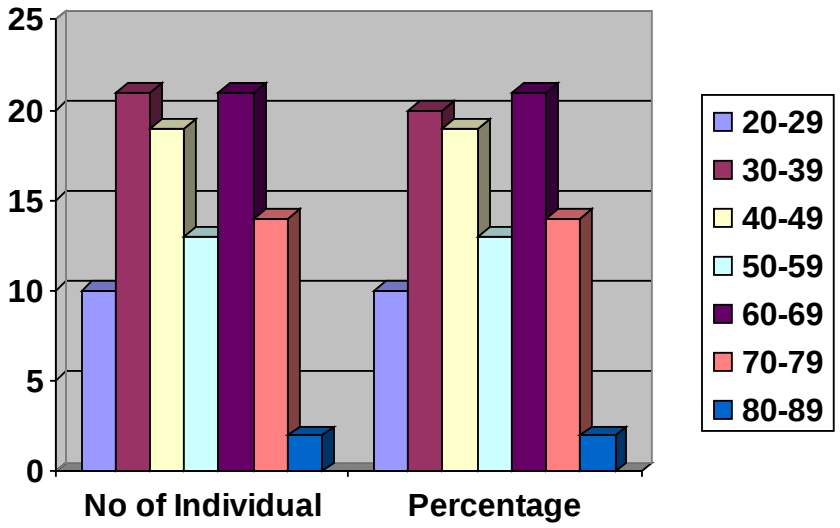


Table: 2. Occupation of 100 Individual with their percentage

Occupation	No of Patients	Percentage
House Wife	30	30%
Employee	26	26%

Student	27	27%
Teacher	10	10%
Doctor	7	7%

Summarize

Case Processing Summary(a)

	Cases					
	Included		Excluded		Total	
	N	Percent	N	Percent	N	Percent
NoOfPatients	5	100.0%	0	.0%	5	100.0%
Percenrage	5	100.0%	0	.0%	5	100.0%

a Limited to first 100 cases.

Case Summaries(a)

		NoOfPatients	Percenrage
1		30.00	.30
2		26.00	.26
3		27.00	.27
4		10.00	.10
5		7.00	.07
Total	N	5	5
	Mean	20.0000	.2000
	Std. Deviation	10.65364	.10654
	Range	23.00	.23
	Std. Error of Skewness	.913	.913

a Limited to first 100 cases.

Descriptives

Descriptive Statistics

	N	Minimum	Maximum	Mean	Std. Deviation
NoOfPatients	5	7.00	30.00	20.0000	10.65364
Percenrage	5	.07	.30	.2000	.10654
Valid N (listwise)	5				

T-Test

One-Sample Statistics

	N	Mean	Std. Deviation	Std. Error Mean
NoOfPatients	5	20.0000	10.65364	4.76445
Percenrage	5	.2000	.10654	.04764

One-Sample Test

	Test Value = 0					
					95% Confidence Interval of the Difference	
	t	df	Sig. (2-tailed)	Mean Difference	Lower	Upper
NoOfPatients	4.198	4	.014	20.00000	6.7718	33.2282
Percenrage	4.198	4	.014	.20000	.0677	.3323

Two-stage Least Squares Analysis

Model Description

		Type of Variable
Equation 1	Percenrage	dependent
	NoOfPatients	predictor & instrumental

MOD_7

Model Summary

Equation 1	Multiple R	1.000
	R Square	1.000
	Adjusted R Square	1.000
	Std. Error of the Estimate	.000

ANOVA

		Sum of Squares	df	Mean Square	F	Sig.
Equation 1	Regression	.045	1	.045	.000	.000
	Residual	.000	3	.000		
	Total	.045	4			

Coefficients

		Unstandardized Coefficients		Beta	t	Sig.
		B	Std. Error			
Equation 1	(Constant)	6.94E-018	.000		.	.
	NoOfPatients	.010	.000	1.000	.	.

Graph: 2

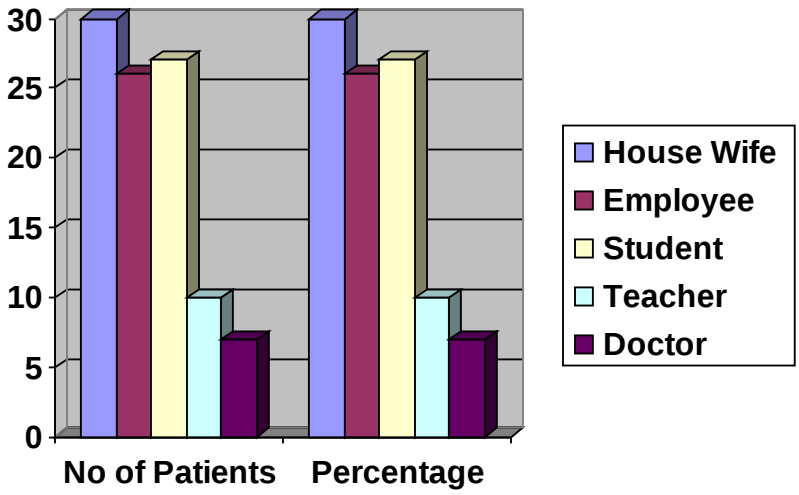


Table: 3. No of obese patients and with their percentage non obese

Patients	No of Patients	Percentage
Obese	21	21%
Non Obese	79	79%

Summarize

Case Processing Summary(a)

	Cases					
	Included		Excluded		Total	
	N	Percent	N	Percent	N	Percent
NoOfPatients	2	100.0%	0	.0%	2	100.0%
Percentage	2	100.0%	0	.0%	2	100.0%

a Limited to first 100 cases.

Case Summaries(a)

		NoOfPatients	Percentage
1		21.00	.21
2		19.00	.19
Total	N	2	2
	Mean	20.0000	.2000
	Std. Deviation	1.41421	.01414
	Range	2.00	.02
	Std. Error of Skewness	.	.

a Limited to first 100 cases.

Descriptives

Descriptive Statistics

	N	Minimum	Maximum	Mean	Std. Deviation
NoOfPatients	2	19.00	21.00	20.0000	1.41421
Percentage	2	.19	.21	.2000	.01414
Valid N (listwise)	2				

T-Test

One-Sample Statistics

	N	Mean	Std. Deviation	Std. Error Mean
NoOfPatients	2	20.0000	1.41421	1.00000
Percentage	2	.2000	.01414	.01000

One-Sample Test

	Test Value = 0					
					95% Confidence Interval of the Difference	
	t	df	Sig. (2-tailed)	Mean Difference	Lower	Upper
NoOfPatients	20.000	1	.032	20.00000	7.2938	32.7062
Percentage	20.000	1	.032	.20000	.0729	.3271

Two-stage Least Squares Analysis

Model Description

		Type of Variable
Equation 1	Percentage	dependent
	NoOfPatients	predictor & instrumental

MOD_8

Model Summary

Equation 1	Multiple R	1.000
	R Square	1.000
	Adjusted R Square	1.000
	Std. Error of the Estimate	.000

ANOVA

		Sum of Squares	df	Mean Square	F	Sig.
Equation 1	Regression	.000	1	.000	.000	.000
	Residual	.000	0	.		
	Total	.000	1			

Coefficients

		Unstandardized Coefficients		Beta	t	Sig.
		B	Std. Error			
Equation 1	(Constant)	1.11E-016	.000		.	.
	NoOfPatients	.010	.000	1.000	.	.

Graph: 3

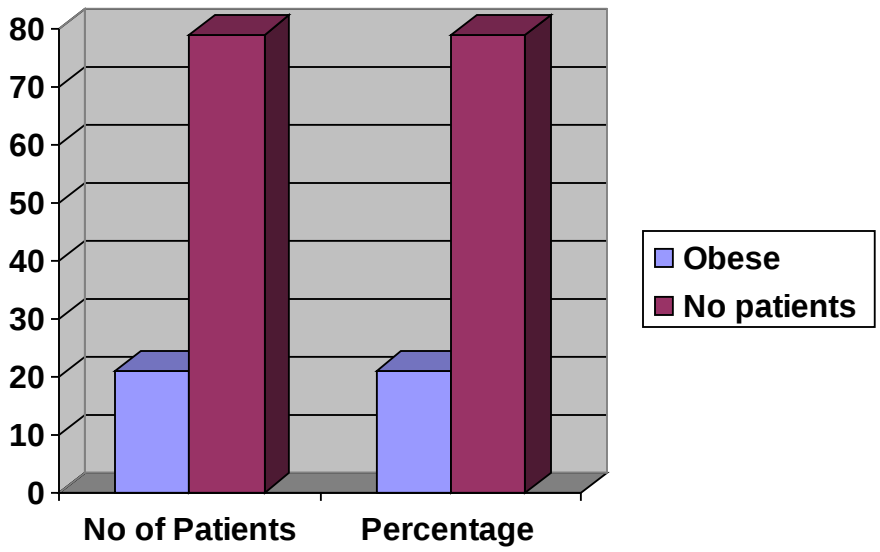


Table: 4. Smoker Patient and Non Smoker

Patients	No of Patients	Percentage
Smoker	29	29%
No Smoker	71	71%

Summarize

Case Processing Summary(a)

	Cases					
	Included		Excluded		Total	
	N	Percent	N	Percent	N	Percent
NoOfPatients	2	100.0%	0	.0%	2	100.0%
Percentage	2	100.0%	0	.0%	2	100.0%

a Limited to first 100 cases.

Case Summaries(a)

	NoOfPatients	Percentage

1		29.00	.29
2		71.00	.71
Total	N	2	2
	Mean	50.0000	.5000
	Std. Deviation	29.69848	.29698
	Range	42.00	.42
	Std. Error of Skewness	.	.

a Limited to first 100 cases.

Descriptives

Descriptive Statistics

	N	Minimum	Maximum	Mean	Std. Deviation
NoOfPatients	2	29.00	71.00	50.0000	29.69848
Percentage	2	.29	.71	.5000	.29698
Valid N (listwise)	2				

T-Test

One-Sample Statistics

	N	Mean	Std. Deviation	Std. Error Mean
NoOfPatients	2	50.0000	29.69848	21.00000
Percentage	2	.5000	.29698	.21000

One-Sample Test

	Test Value = 0					
					95% Confidence Interval of the Difference	
	t	df	Sig. (2-tailed)	Mean Difference	Lower	Upper
NoOfPatients	2.381	1	.253	50.00000	-216.8303	316.8303
Percentage	2.381	1	.253	.50000	-2.1683	3.1683

Two-stage Least Squares Analysis

Model Description

		Type of Variable
Equation 1	Percentage	dependent
	NoOfPatients	predictor & instrumental

MOD_9

Model Summary

Equation 1	Multiple R	1.000
	R Square	1.000
	Adjusted R Square	1.000
	Std. Error of the Estimate	.000

ANOVA

		Sum of Squares	df	Mean Square	F	Sig.
Equation 1	Regression	.088	1	.088	.000	.000
	Residual	.000	0	.		
	Total	.088	1			

Coefficients

		Unstandardized Coefficients		Beta	t	Sig.
		B	Std. Error			
Equation 1	(Constant)	-1.04E-017	.000		.	.
	NoOfPatients	.010	.000	1.000	.	.

Graph: 4

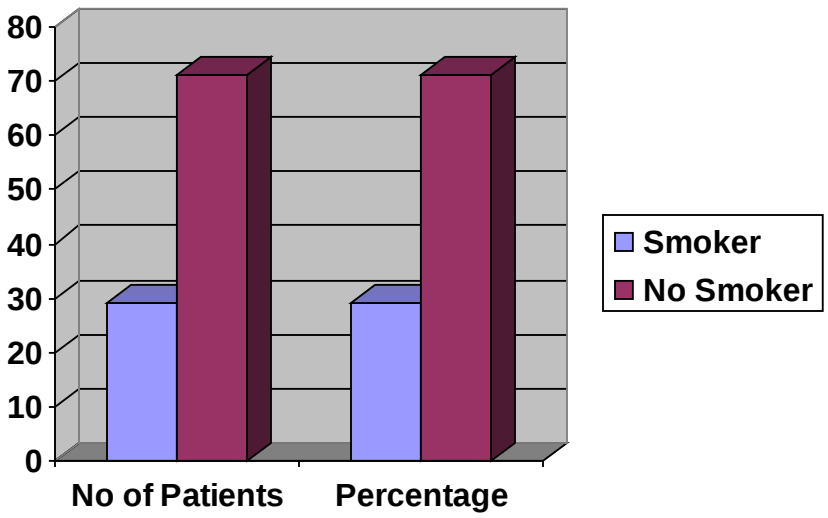

80
70
60
50
40
30
20
10
0
No of Patients
Percentage
Smoker
No Smoker

Table: 5. IMT Ranges with their percentages.

IMT (mm) Ranges	Right CCA		Left CCA	
	No of Patients	Percentage	No of Patients	Percentages
0.2- 0.3	2	2%	2	2%
0.4-0.5	21	21%	22	22%
0.6-0.7	35	35%	33	33%
Up to 0.8	29	29%	23	23%
≥ 0.9	21	21%	20	20%

Summarize

Case Processing Summary(a)

	Cases					
	Included		Excluded		Total	
	N	Percent	N	Percent	N	Percent
NoOfPatients	5	100.0%	0	.0%	5	100.0%
Percentage	5	100.0%	0	.0%	5	100.0%

a Limited to first 100 cases.

Case Summaries(a)

		NoOfPatients	Percentage
1		2.00	.02
2		21.00	.21
3		35.00	.35
4		29.00	.29
5		21.00	.21
Total	N	5	5
	Mean	21.6000	.2160
	Std. Deviation	12.44186	.12442
	Range	33.00	.33
	Std. Error of Skewness	.913	.913

a Limited to first 100 cases.

Descriptives

Descriptive Statistics

	N	Minimum	Maximum	Mean	Std. Deviation
NoOfPatients	5	2.00	35.00	21.6000	12.44186
Percentage	5	.02	.35	.2160	.12442

Valid N (listwise)	5				

T-Test

One-Sample Statistics

	N	Mean	Std. Deviation	Std. Error Mean
NoOfPatients	5	21.6000	12.44186	5.56417
Percentage	5	.2160	.12442	.05564

One-Sample Test

	Test Value = 0					
					95% Confidence Interval of the Difference	
	t	df	Sig. (2-tailed)	Mean Difference	Lower	Upper
NoOfPatients	3.882	4	.018	21.60000	6.1514	37.0486
Percentage	3.882	4	.018	.21600	.0615	.3705

Two-stage Least Squares Analysis

Model Description

		Type of Variable
Equation 1	Percentage	dependent
	NoOfPatients	predictor & instrumental

MOD_10

Model Summary

Equation 1	Multiple R	1.000
	R Square	1.000
	Adjusted R Square	1.000
	Std. Error of the Estimate	.000

ANOVA

		Sum of Squares	df	Mean Square	F	Sig.
Equation 1	Regression	.062	1	.062	.000	.000
	Residual	.000	3	.000		
	Total	.062	4			

Coefficients

		Unstandardized Coefficients		Beta	t	Sig.
		B	Std. Error			
Equation 1	(Constant)	-1.14E-017	.000		.	.
	NoOfPatients	.010	.000	1.000	.	.

Left Side

Summarize

Case Processing Summary(a)

	Cases					
	Included		Excluded		Total	
	N	Percent	N	Percent	N	Percent

NoOfPatients	5	100.0%	0	.0%	5	100.0%
Percentage	5	100.0%	0	.0%	5	100.0%

a Limited to first 100 cases.

Case Summaries(a)

		NoOfPatients	Percentage
1		2.00	.02
2		22.00	.22
3		33.00	.33
4		23.00	.23
5		20.00	.20
Total	N	5	5
	Mean	20.0000	.2000
	Std. Deviation	11.24722	.11247
	Range	31.00	.31
	Std. Error of Skewness	.913	.913

a Limited to first 100 cases.

Descriptives

Descriptive Statistics

	N	Minimum	Maximum	Mean	Std. Deviation
NoOfPatients	5	2.00	33.00	20.0000	11.24722
Percentage	5	.02	.33	.2000	.11247
Valid N (listwise)	5				

T-Test

One-Sample Statistics

	N	Mean	Std. Deviation	Std. Error Mean
NoOfPatients	5	20.0000	11.24722	5.02991
Percentage	5	.2000	.11247	.05030

One-Sample Test

	Test Value = 0					
					95% Confidence Interval of the Difference	
	t	df	Sig. (2-tailed)	Mean Difference	Lower	Upper

NoOfPatients	3.976	4	.016	20.00000	6.0347	33.9653
Percentage	3.976	4	.016	.20000	.0603	.3397

Two-stage Least Squares Analysis

Model Description

		Type of Variable
Equation 1	Percentage	dependent
	NoOfPatients	predictor & instrumental

MOD_11

Model Summary

Equation 1	Multiple R	1.000
	R Square	1.000
	Adjusted R Square	1.000
	Std. Error of the Estimate	.000

ANOVA

		Sum of Squares	df	Mean Square	F	Sig.
Equation 1	Regression	.051	1	.051	.000	.000
	Residual	.000	3	.000		
	Total	.051	4			

Coefficients

		Unstandardized Coefficients		Beta	t	Sig.
		B	Std. Error			
Equation 1	(Constant)	6.94E-018	.000		.	.
	NoOfPatients	.010	.000	1.000	.	.

Graph: 5

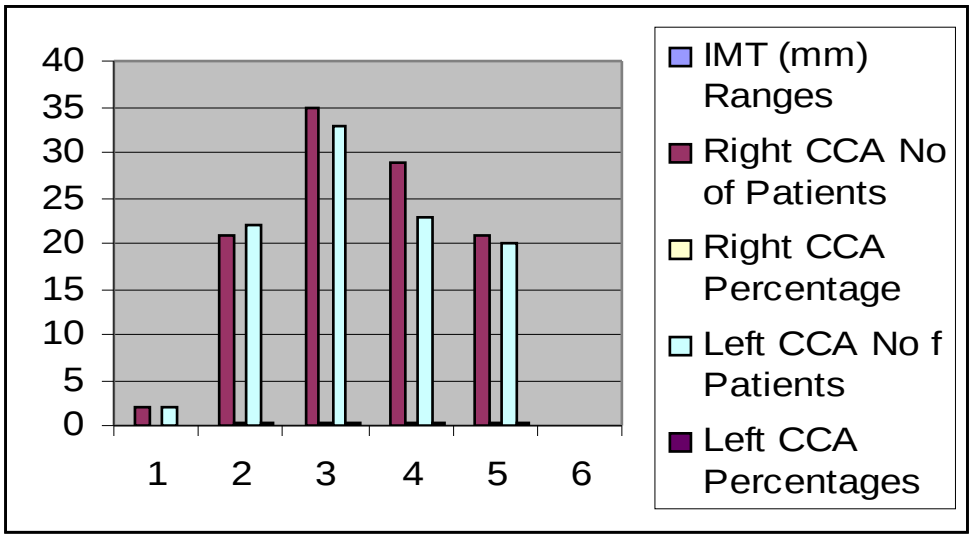

40
35
30
25
20
15
10
5
0
1
2
3
4
5
6
IMT (mm) Ranges
Right CCA No of Patients
Right CCA Percentage
Left CCA No f Patients
Left CCA Percentages

Table: 6.Percentage of ICA/CCA Ratio with following ranges

Range	No of Patient	Percentage	No of Patients	Percentage
1.0-1.09 cm/s	27	27%	25	25%
1.10-1.19	39	39%	40	40%
1.20-1.29	30	30%	28	28%
1.30-1.39	3	3%	6	6%
1.40-1.80	1	1%	1	1%

Right side

Summarize

Case Processing Summary(a)

	Cases					
	Included		Excluded		Total	
	N	Percent	N	Percent	N	Percent
NoOfPatients	5	100.0%	0	.0%	5	100.0%
Percentage	5	100.0%	0	.0%	5	100.0%

a Limited to first 100 cases.

Case Summaries(a)

		NoOfPatients	Percentage
1		27.00	.27
2		39.00	.39
3		30.00	.30
4		3.00	.03
5		1.00	.01
Total	N	5	5
	Mean	20.0000	.2000
	Std. Deviation	17.02939	.17029
	Range	38.00	.38
	Std. Error of Skewness	.913	.913

a Limited to first 100 cases.

Descriptives

Descriptive Statistics

	N	Minimum	Maximum	Mean	Std. Deviation

NoOfPatients	5	1.00	39.00	20.0000	17.02939
Percentage	5	.01	.39	.2000	.17029
Valid N (listwise)	5				

T-Test

One-Sample Statistics

	N	Mean	Std. Deviation	Std. Error Mean
NoOfPatients	5	20.0000	17.02939	7.61577
Percentage	5	.2000	.17029	.07616

One-Sample Test

	Test Value = 0					
					95% Confidence Interval of the Difference	
	t	df	Sig. (2-tailed)	Mean Difference	Lower	Upper
NoOfPatients	2.626	4	.058	20.00000	-1.1448	41.1448
Percentage	2.626	4	.058	.20000	-.0114	.4114

Two-stage Least Squares Analysis

Model Description

		Type of Variable
Equation 1	Percentage	dependent
	NoOfPatients	predictor & instrumental

MOD_12

Model Summary

Equation 1	Multiple R	1.000
	R Square	1.000
	Adjusted R Square	1.000
	Std. Error of the Estimate	.000

ANOVA

		Sum of Squares	df	Mean Square	F	Sig.
Equation 1	Regression	.116	1	.116	.000	.000
	Residual	.000	3	.000		
	Total	.116	4			

Coefficients

		Unstandardized Coefficients		Beta	t	Sig.
		B	Std. Error			
Equation 1	(Constant)	4.16E-017	.000		.	.
	NoOfPatients	.010	.000	1.000	.	.

Left Side

Summarize

Case Processing Summary(a)

	Cases					
	Included		Excluded		Total	
	N	Percent	N	Percent	N	Percent

NoOfPatients	5	100.0%	0	.0%	5	100.0%
Percentage	5	100.0%	0	.0%	5	100.0%

a Limited to first 100 cases.

Case Summaries(a)

		NoOfPatients	Percentage
1		25.00	.25
2		40.00	.40
3		28.00	.28
4		6.00	.06
5		1.00	.01
Total	N	5	5
	Mean	20.0000	.2000
	Std. Deviation	16.17096	.16171
	Range	39.00	.39
	Std. Error of Skewness	.913	.913

a Limited to first 100 cases.

Descriptives

Descriptive Statistics

	N	Minimum	Maximum	Mean	Std. Deviation
NoOfPatients	5	1.00	40.00	20.0000	16.17096
Percentage	5	.01	.40	.2000	.16171
Valid N (listwise)	5				

T-Test

One-Sample Statistics

	N	Mean	Std. Deviation	Std. Error Mean
NoOfPatients	5	20.0000	16.17096	7.23187
Percentage	5	.2000	.16171	.07232

One-Sample Test

	Test Value = 0					
					95% Confidence Interval of the Difference	
	t	df	Sig. (2-tailed)	Mean Difference	Lower	Upper
NoOfPatients	2.766	4	.051	20.00000	-.0789	40.0789
Percentage	2.766	4	.051	.20000	-.0008	.4008

Two-stage Least Squares Analysis

Model Description

		Type of Variable
Equation 1	Percentage	dependent
	NoOfPatients	predictor & instrumental

MOD_13

Model Summary

Equation 1	Multiple R	1.000
	R Square	1.000
	Adjusted R Square	1.000
	Std. Error of the Estimate	.000

ANOVA

		Sum of Squares	df	Mean Square	F	Sig.
Equation 1	Regression	.105	1	.105	.000	.000
	Residual	.000	3	.000		
	Total	.105	4			

Coefficients

		Unstandardized Coefficients		Beta	t	Sig.
		B	Std. Error			
Equation 1	(Constant)	6.94E-018	.000		.	.
	NoOfPatients	.010	.000	1.000	.	.

Graph: 6

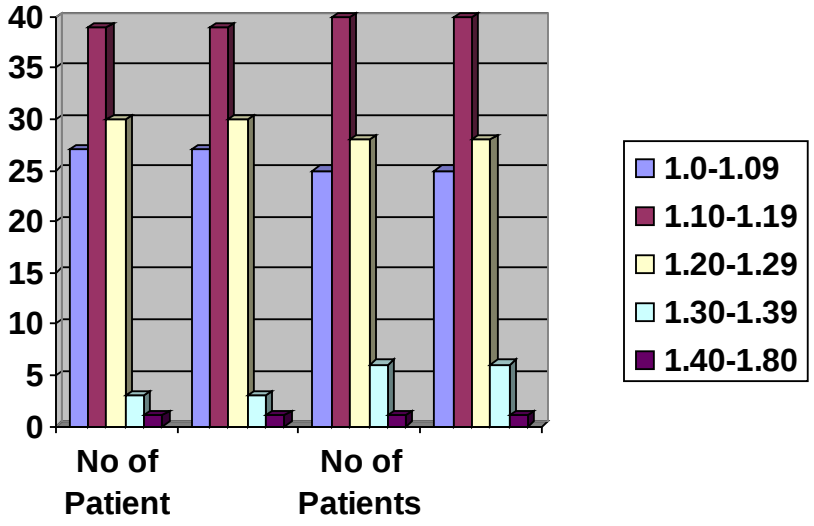


Table: 7. Percentage of PSV of ICA with following ranges.

Ranges	Rt. Side PSV R/ICA	Percentage	Lt. Side PSV L/ ICA	Percentage
50-59	1	1%	1	1%
60-69	50	50%	47	47%
70-79	44	44%	43	43%
80-89	5	5%	8	8%
90-99	0	0%	1	1%

Right Side

Summarize

Case Processing Summary(a)

	Cases					
	Included		Excluded		Total	
	N	Percent	N	Percent	N	Percent
RtSidePSV	5	100.0%	0	.0%	5	100.0%
Percentage	5	100.0%	0	.0%	5	100.0%

a Limited to first 100 cases.

Case Summaries(a)

		RtSidePSV	Percentage
1		1.00	.01
2		50.00	.50
3		44.00	.44
4		5.00	.05
5		.00	.00
Total	N	5	5
	Mean	20.0000	.2000
	Std. Deviation	24.80927	.24809
	Range	50.00	.50
	Std. Error of Skewness	.913	.913

a Limited to first 100 cases.

Descriptives

Descriptive Statistics

	N	Minimum	Maximum	Mean	Std. Deviation
RtSidePSV	5	.00	50.00	20.0000	24.80927
Percentage	5	.00	.50	.2000	.24809
Valid N (listwise)	5				

T-Test

One-Sample Statistics

	N	Mean	Std. Deviation	Std. Error Mean
RtSidePSV	5	20.0000	24.80927	11.09504
Percentage	5	.2000	.24809	.11095

One-Sample Test

	Test Value = 0					
					95% Confidence Interval of the Difference	
	t	df	Sig. (2-tailed)	Mean Difference	Lower	Upper
RtSidePSV Percentage	1.803	4	.146	20.00000	-10.8048	50.8048
	1.803	4	.146	.20000	-.1080	.5080

Two-stage Least Squares Analysis

Model Description

		Type of Variable
Equation 1	Percentage	dependent
	RtSidePSV	predictor & instrumental

MOD_14

Model Summary

Equation 1	Multiple R	1.000
	R Square	1.000
	Adjusted R Square	1.000
	Std. Error of the Estimate	.000

ANOVA

		Sum of Squares	df	Mean Square	F	Sig.
Equation 1	Regression	.246	1	.246	.000	.000
	Residual	.000	3	.000		
	Total	.246	4			

Coefficients

		Unstandardized Coefficients		Beta	t	Sig.
		B	Std. Error			
Equation 1	(Constant)	4.16E-017	.000		.	.
	RtSidePSV	.010	.000	1.000	.	.

Left Side

Summarize

Case Processing Summary(a)

	Cases					
	Included		Excluded		Total	
	N	Percent	N	Percent	N	Percent
ltSidePSV Percentage	5	100.0%	0	.0%	5	100.0%
	5	100.0%	0	.0%	5	100.0%

a Limited to first 100 cases.

Case Summaries(a)

		ltSidePSV Percentage
1		1.00 .01
2		47.00 .47
3		43.00 .43
4		8.00 .08
5		1.00 .01
Total	N	5 5
	Mean	20.0000 .2000
	Std. Deviation	23.04344 .23043
	Range	46.00 .46

	Std. Error of Skewness	.913	.913

a Limited to first 100 cases.

Descriptives

Descriptive Statistics

	N	Minimum	Maximum	Mean	Std. Deviation
ltSidePSV	5	1.00	47.00	20.0000	23.04344
Percentage	5	.01	.47	.2000	.23043
Valid N (listwise)	5				

T-Test

One-Sample Statistics

	N	Mean	Std. Deviation	Std. Error Mean
ltSidePSV	5	20.0000	23.04344	10.30534
Percentage	5	.2000	.23043	.10305

One-Sample Test

	Test Value = 0					
					95% Confidence Interval of the Difference	
	t	df	Sig. (2-tailed)	Mean Difference	Lower	Upper
ltSidePSV	1.941	4	.124	20.00000	-8.6122	48.6122
Percentage	1.941	4	.124	.20000	-.0861	.4861

Two-stage Least Squares Analysis

Model Description

		Type of Variable
Equation 1	Percentage	dependent

	ltSidePSV	predictor & instrumental

MOD_15

Model Summary

Equation 1	Multiple R	1.000
	R Square	1.000
	Adjusted R Square	1.000
	Std. Error of the Estimate	.000

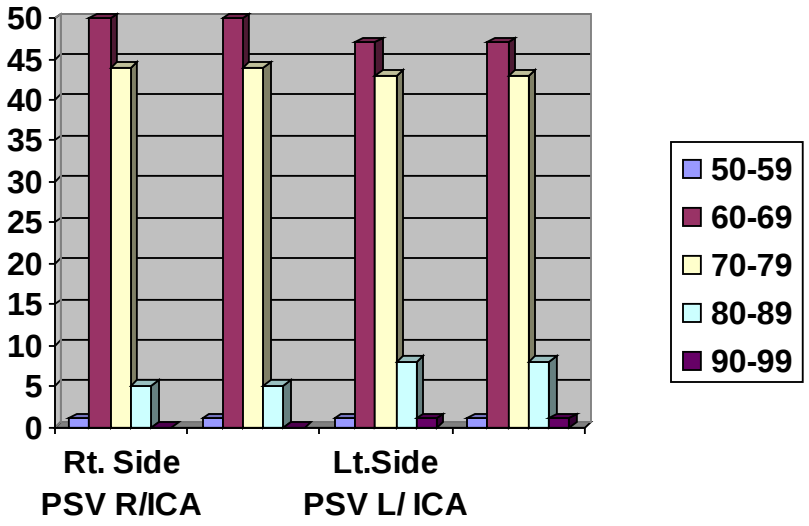
ANOVA

		Sum of Squares	df	Mean Square	F	Sig.
Equation 1	Regression	.212	1	.212	.000	.000
	Residual	.000	3	.000		
	Total	.212	4			

Coefficients

		Unstandardized Coefficients		Beta	t	Sig.
		B	Std. Error			
Equation 1	(Constant)	4.86E-017	.000		.	.
	ltSidePSV	.010	.000	1.000	.	.

Graph: 7


50
45
40
35
30
25
20
15
10
5
0
Rt. Side
PSV R/ICA
Lt.Side
PSV L/ ICA
50-59
60-69
70-79
80-89
90-99

CHAPTER FIVE

DISCUSTION

Real time B-Scan and Doppler Ultrasound is easy and cheap but highly skilled modality for measurements of IMT, Velocities of carotids and there ratios we can determined the anatomy, Pathology & management after medically and Surgically Treated Carotids.

I conducted my study at Afro Asian Institute of Medical Sciences in Lahore, Pakistan for hundred Individuals during period of April 2007-Dec 2007. During my study all Individuals came to me were Asymptomatic.

In my study IMT of 79% of right CCA & 80% of left CCA were with in normal ranges i.e; 0.8 mm.

Only 21% of patients having IMT 0.9 to 1.3 mm of right CCA. 20% of Individuals having IMT ranges 0.9-1.3 mm of left CCA.

The average IMT of right CCA is 0.68 mm and IMT of left CCA is 0.71 mm in hundred Individuals.

The average PSV of right CCA is 60.16 cm/second. And PSV of left CCA is 60.54 cm/second in one individuals.

The average PSV of right ICA is 70.40 cm/second. And the average of PSV of left ICA is 71.30 cm/second in hundred individuals.

The average ICA/CCA Ratio of right side is 1.16 in hundred individuals.

The average ICA/CCA Ratio of left side is 1.16 in hundred individuals.

If we will extend our study, with symptomatic patient like T1A, CVA. cardiac patients like atherosclerotic aneurysmal thrombotic lessons.

We can detect pathologies in carotid arteries, likes grade and stage of stenosis which affects the PSV of ICA, CCA. With sever stenosis velocities may be reduced.

We can detect location, extant, severity, echogenicity and surface characteristics. We can determent haemodynamic significances of disease like category of stenosis, occlusion and retrograde flow. We can also diagnose lesion in subclavian, vertebral arteries and subclavian steal.

Contrast agents can also be used for vascular study to enhance the diagnoses in real time B-scan and Doppler study helpful for further managements of carotid after medically and surgically treated patients. It is useful and helpful modality for surgeon for there.

CONCLUTION

One hundred Individuals of different ages, both sexes, having different occupation, obese, non obese, smoker, & non smoker, were scanned in Afro Asian Institute of Medical Sciences Shadman Lahore, Pakistan.
All of the hundred Individuals were Cooperative: - All Measurements regarding of IMT, PSV & ICA/CCA Ratios were recorded.
But the images taken on paper for reference were 30 only. All individuals were Asymptomatic.

The over all average of measurements of IMT, PSV & ICCA/CCA ratios regardless of age, age sex occupation obesity are given in table no# 08

Overall average measurements of 100 Individuals.

I.M.T of CCA		PSV of CCA		PSV of ICA		ICA/CCA Ratios	
Rt	**Lt**	**Rt**	**Lt**	**Rt**	**Lt**	**Rt**	**Lt**
0.68mm	0.71mm	60.16cm/sec	60.54cm/sec	70.40cm/sec	71.30cm/sec	1.16	1.16

RECOMMENDATION

1. I found that Ultrasonography has high Accuracy in evaluating Anatomy, pathology and post operative management
2. The study should be extended for Symptomatic patients like TIA, CVA & for other neurological and CVS.
3. The size of population for study can be increased.
4. The study can also be connected in Cardiology and Nero- Surgery departments in different Hospitals in different Cities.
5. New and Separate
6. Ultrasound Machine should be allocated for research purpose only.

7. Awareness should be given to general population, the hazards of obesity, smoking, fat consumptions and Diabetes Mellitus through handbills, banners and through Media
8. As it growing Modality it should be sported by government.

REFERENCES

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2 Human embryology By Inderbir Singh 4th edition

3 Kaplan Medical anatomy

4 Snell's anatomy 7th Edition

5 Robbin's pathology 7th Edition

6 Doppler ultrasound by Burwin Institute of diagnostic medical ultrasound.

7 Clinical Doppler ultrasound by Paul. L. Allan 6'"Edition

8 Lecture notes By Prof; Amir Gilani on Carotid color and spectral Doppler.

9 A regional Atlas of the human body by Carmine D. Clement 4th Edition

10 The Requisites in ultrasound by Kurtz and William D.

APPENDIX

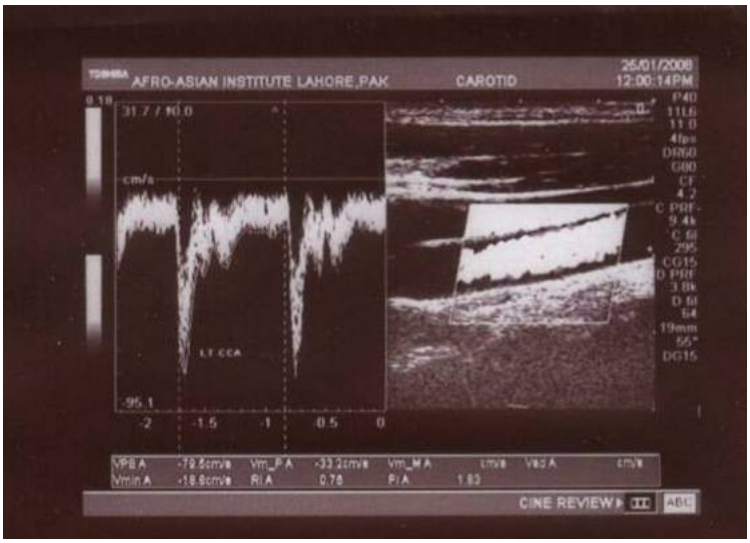

Fig: 1 PSV of Lt. CCA

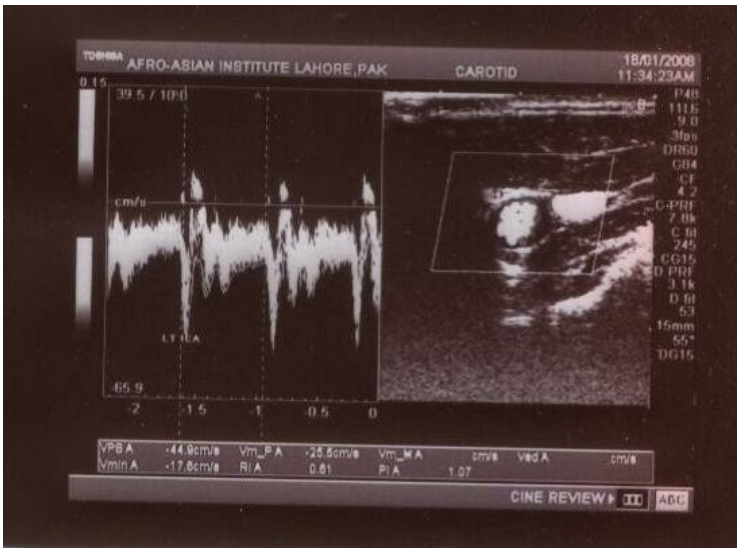

Fig: 2 PSV of Lt. ICA

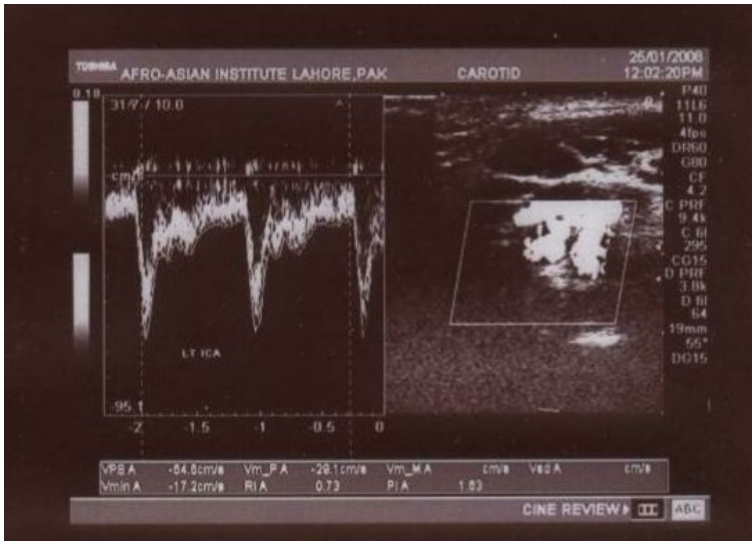

Fig: 3 PSV of Lt. ICA

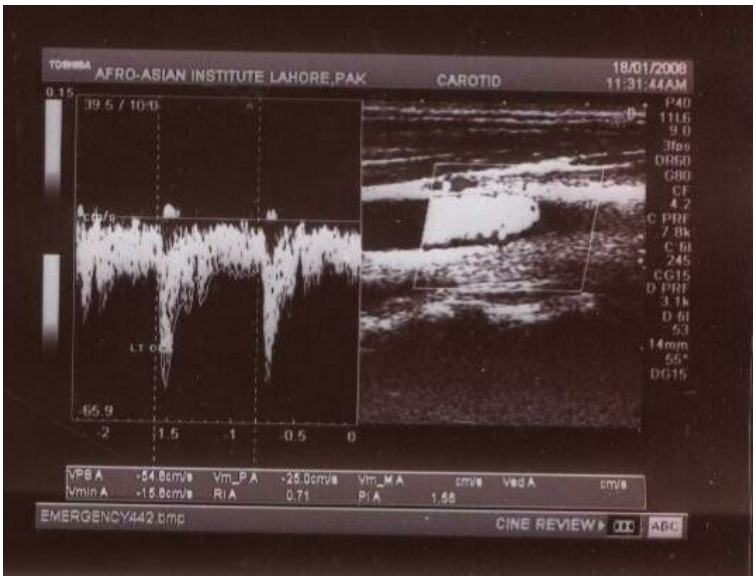

Fig: 4 PSV of Lt. CCA

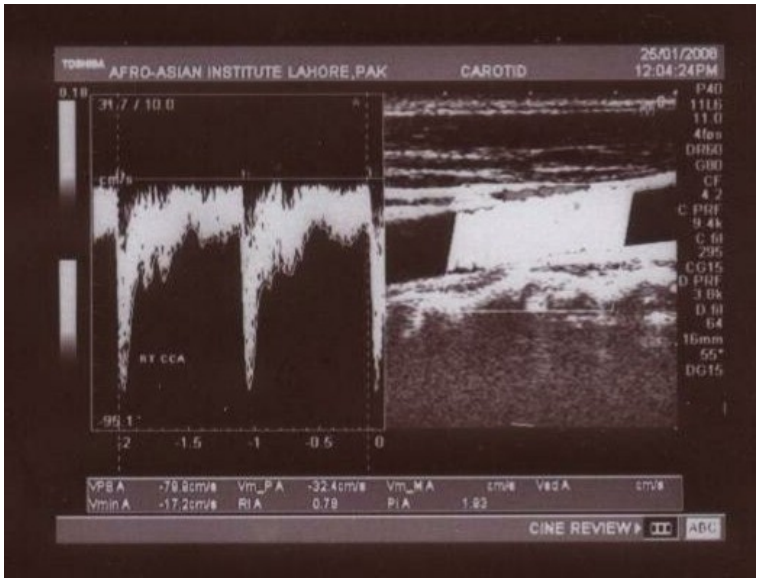

Fig: 5 PSV of Rt. CCA

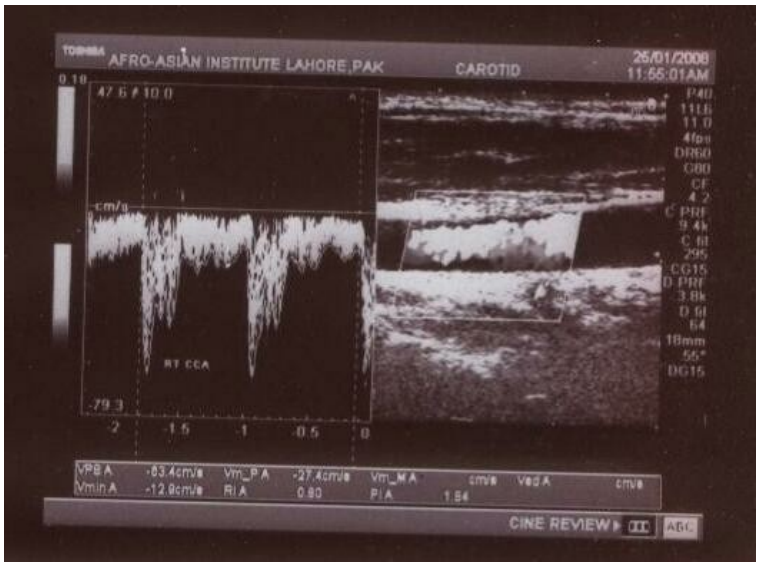

Fig: 6 PSV of Rt. CCA

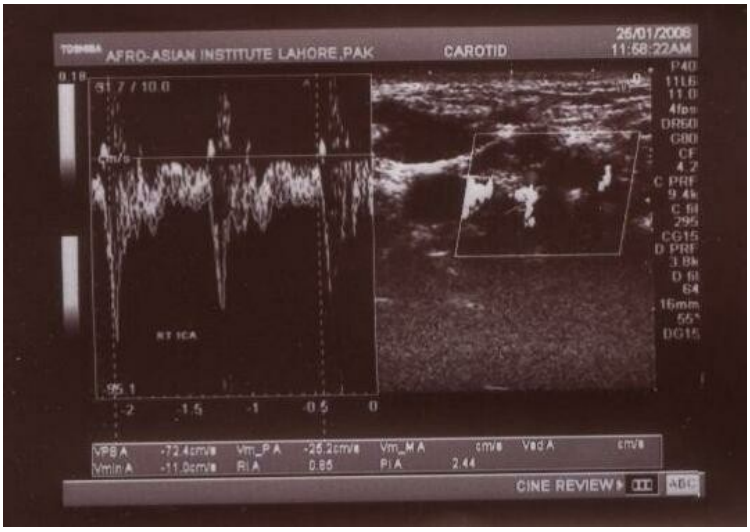

Fig: 7 PSV of Rt. ICA

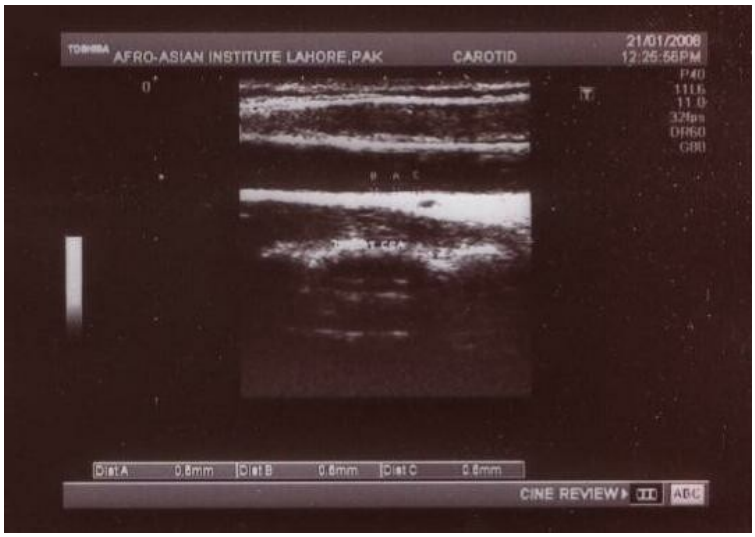

Fig: 8 IMT Rt. CCA

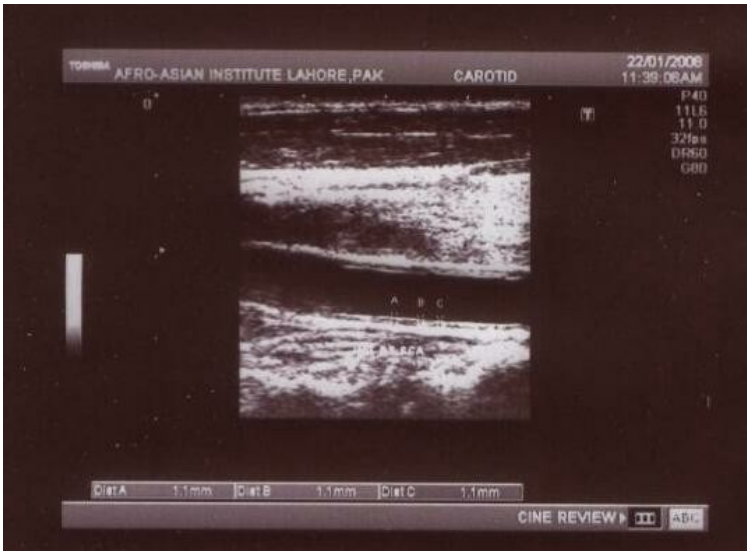

Fig: 9 IMT of Rt. CCA

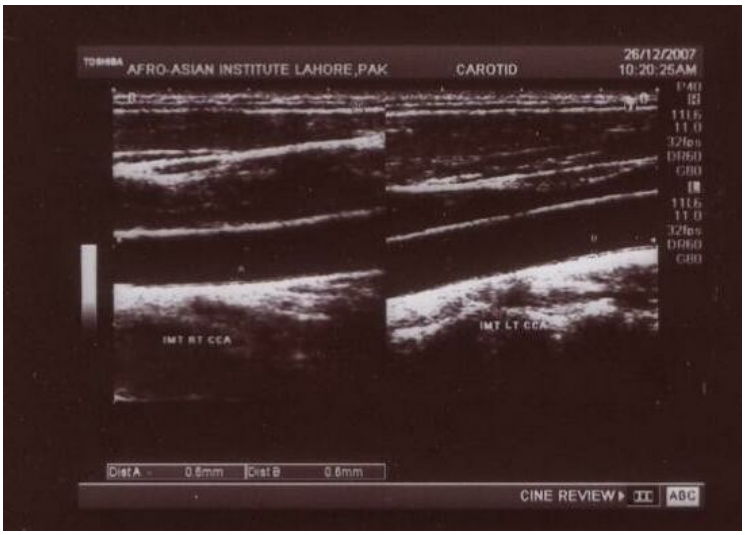

Fig: 10 IMT of Rt and Left. CCA

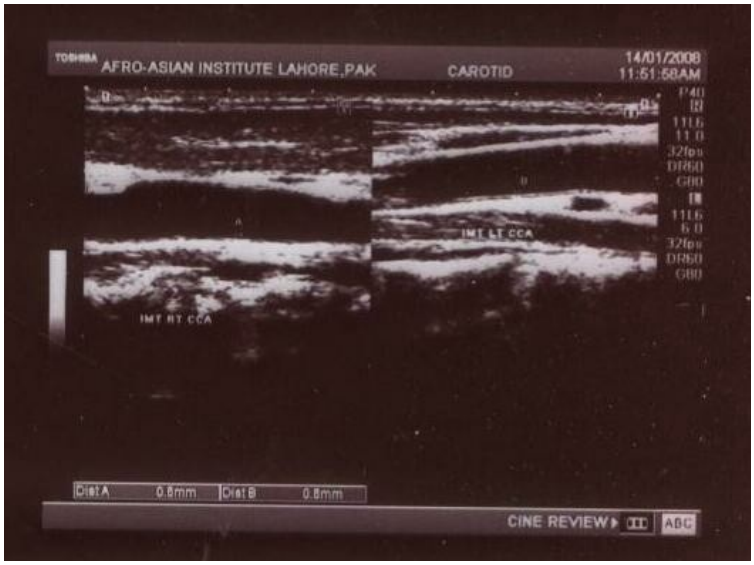

Fig: 11 IMT of Rt and Left. CCA

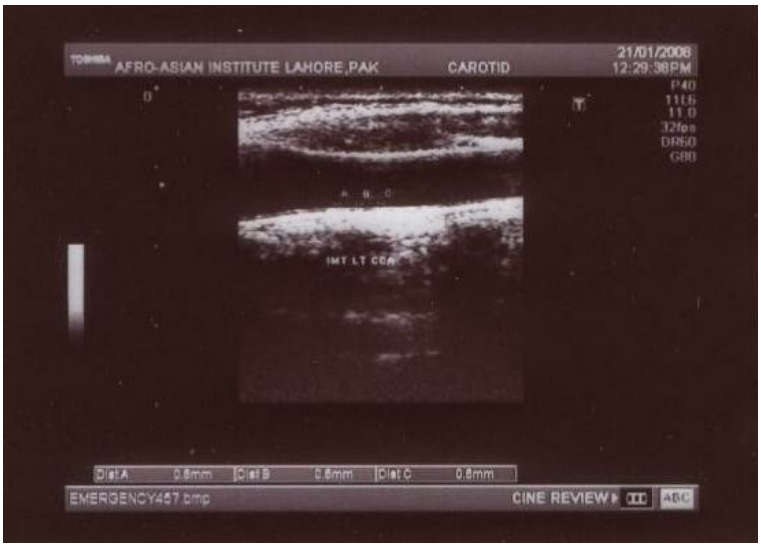

Fig: 12 IMT of Left. CCA

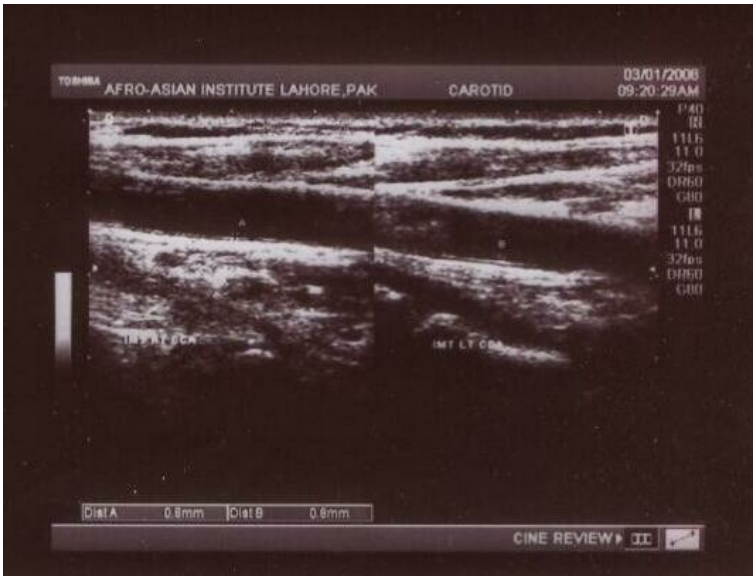

Fig: 13 IMT of Rt and Left. CCA

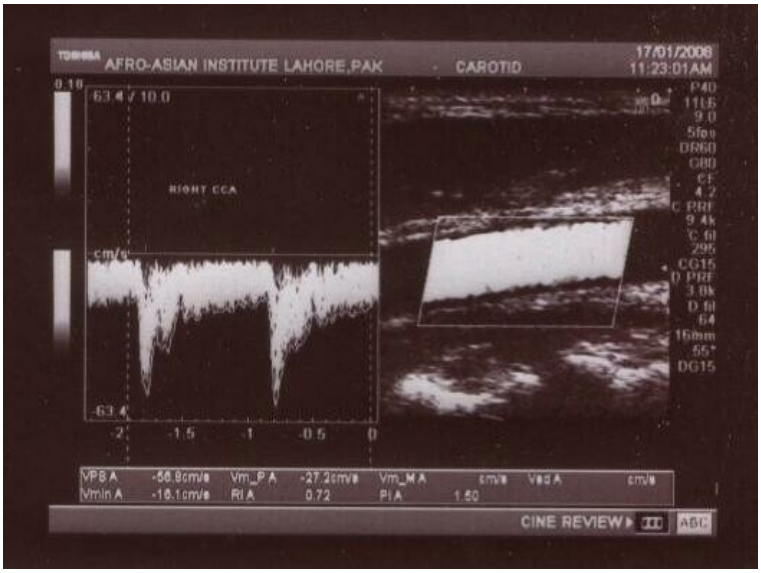

Fig: 14 PSV of Rt. CCA

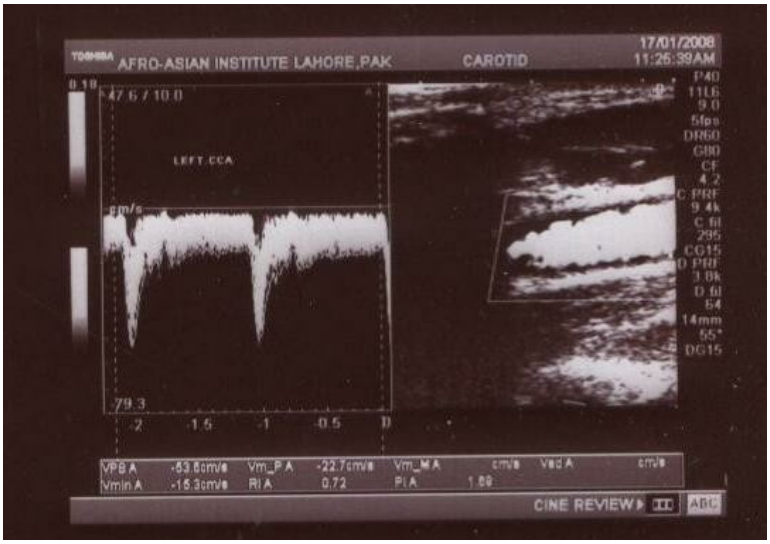

Fig: 15 PSV of Left. CCA

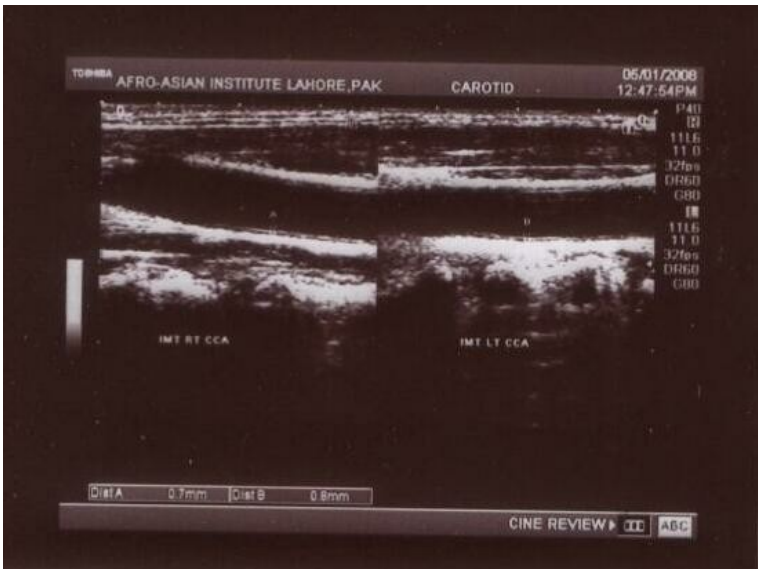

Fig: 16 IMT of Rt and Left. CCA

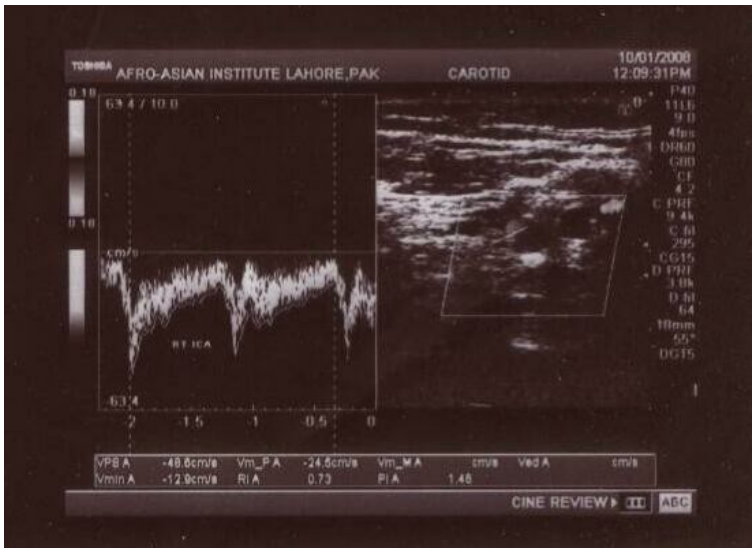

Fig: 17 PSV of Rt. CCA

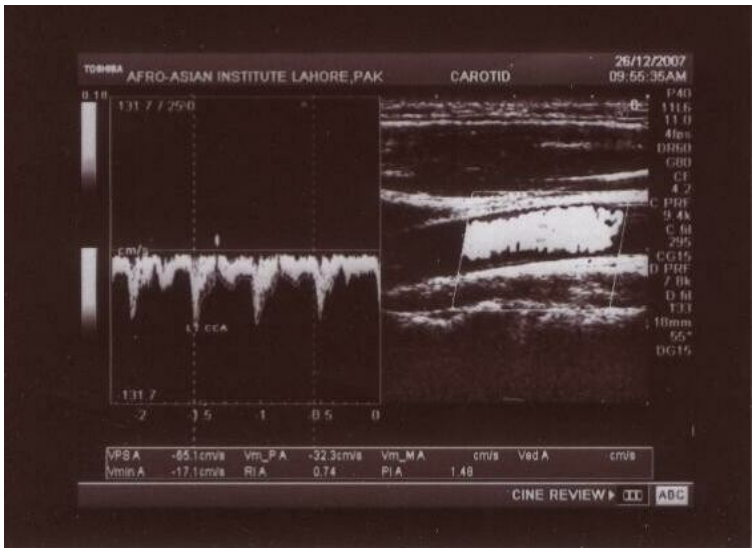

Fig: 18 PSV of Lt. CCA

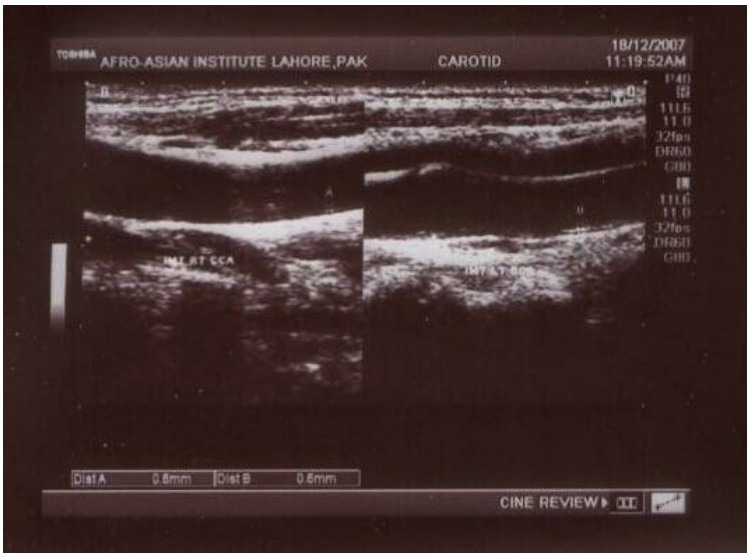

Fig: 19 IMT of Rt. and Left CCA

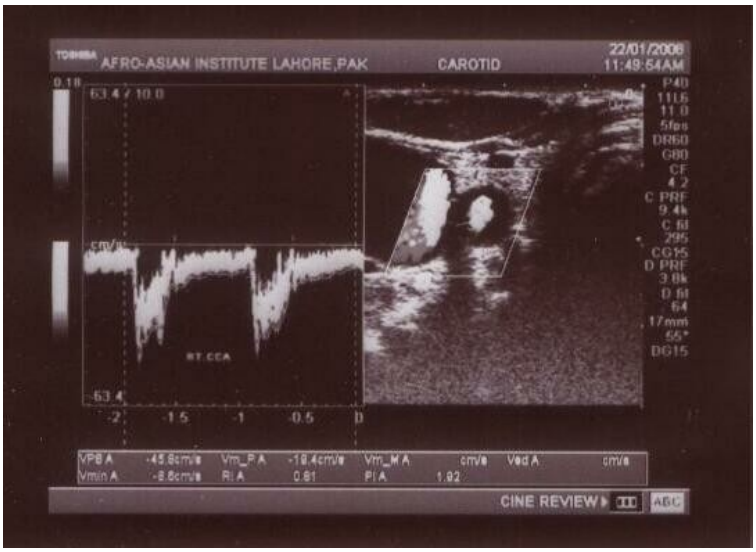

Fig: 20 PSV of Rt. CCA

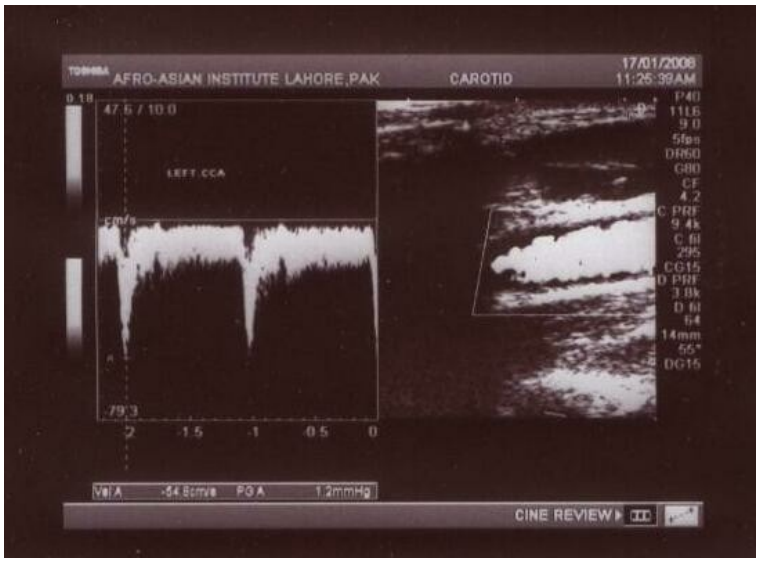

Fig: 21 PSV of Lt. CCA

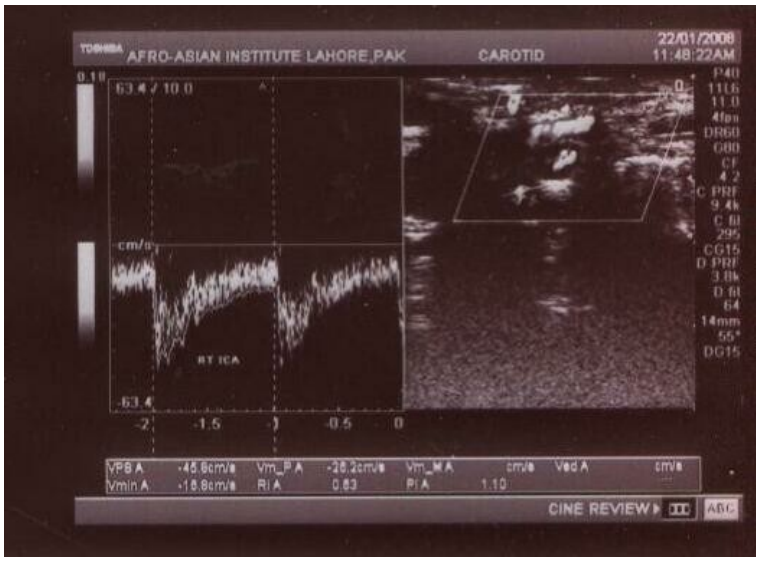

Fig: 22 PSV of Rt. ICA

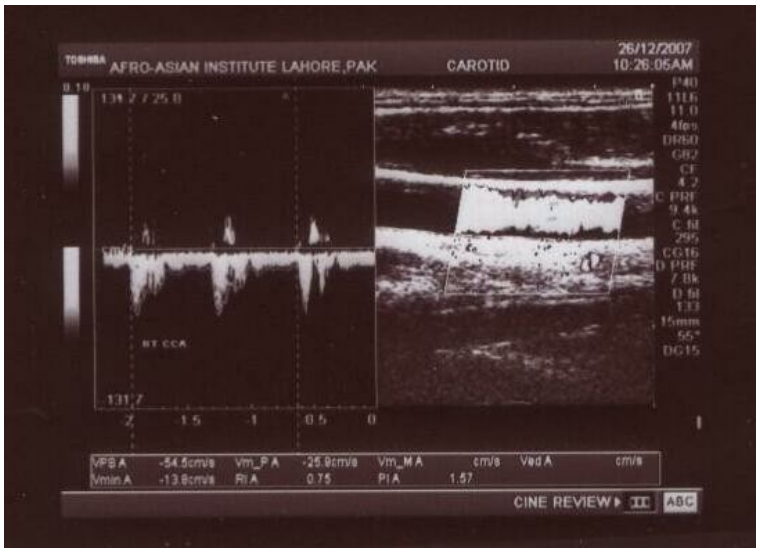

Fig: 23 PSV of Rt. CCA

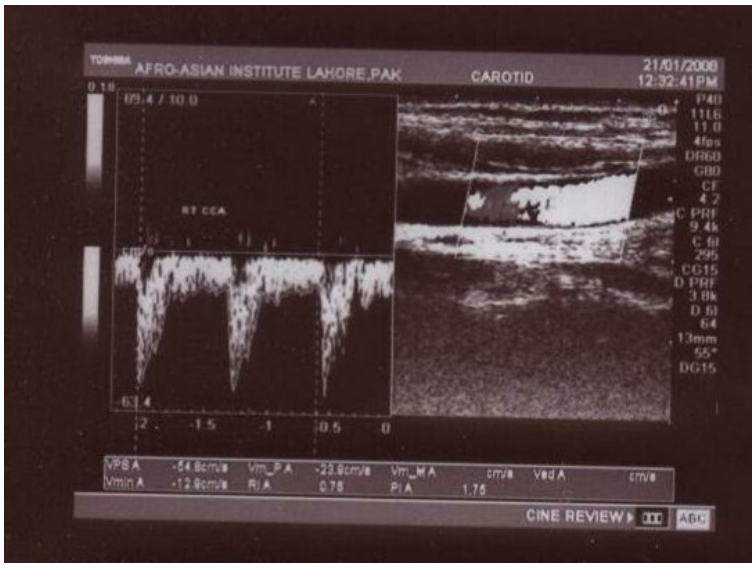

Fig: 24 PSV of Rt. CCA

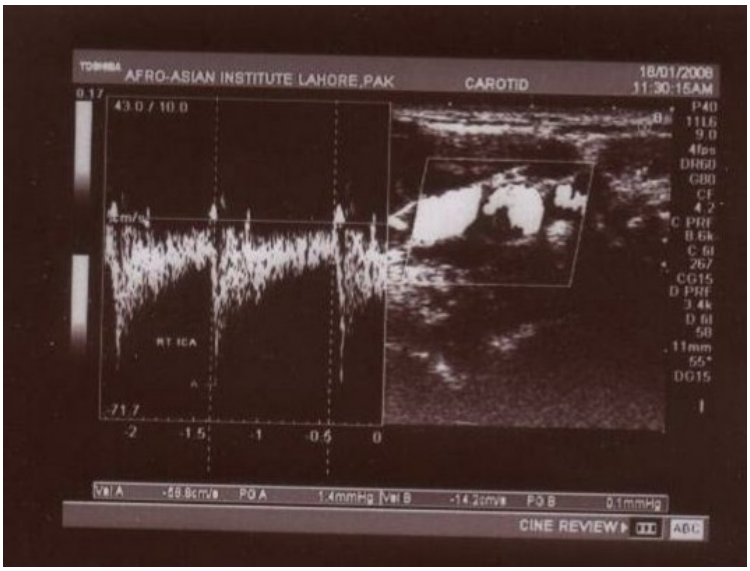

Fig: 25 PSV of Rt. ICA

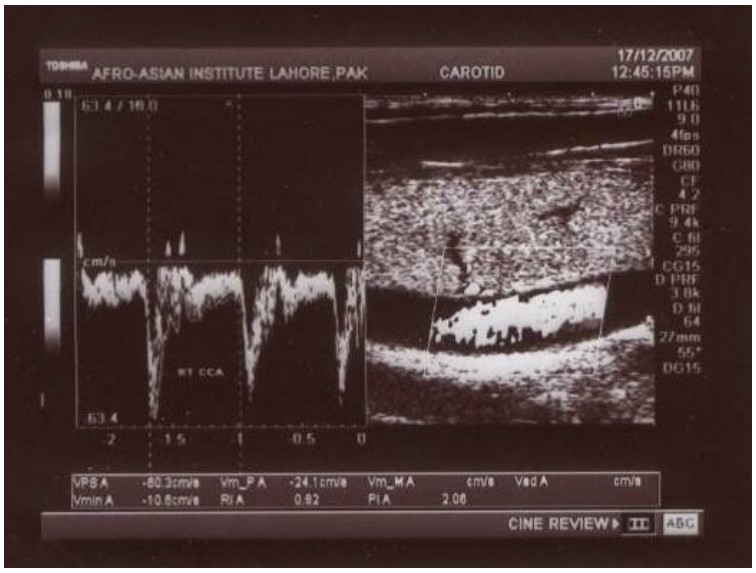

Fig: 26 PSV of Rt. CCA

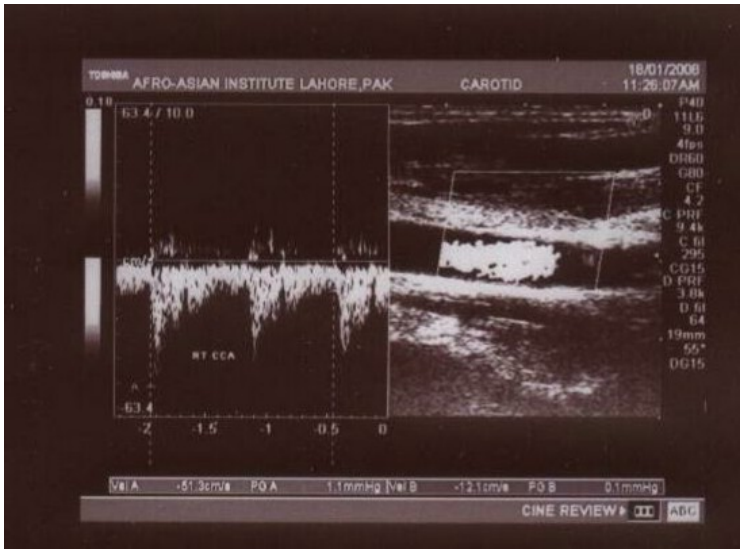

Fig: 27 PSV of Rt. CCA

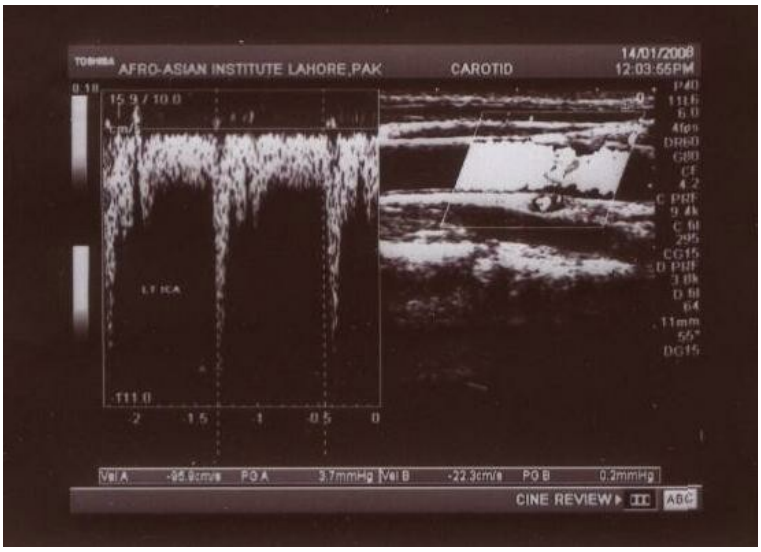

Fig: 28 PSV of Lt. ICA

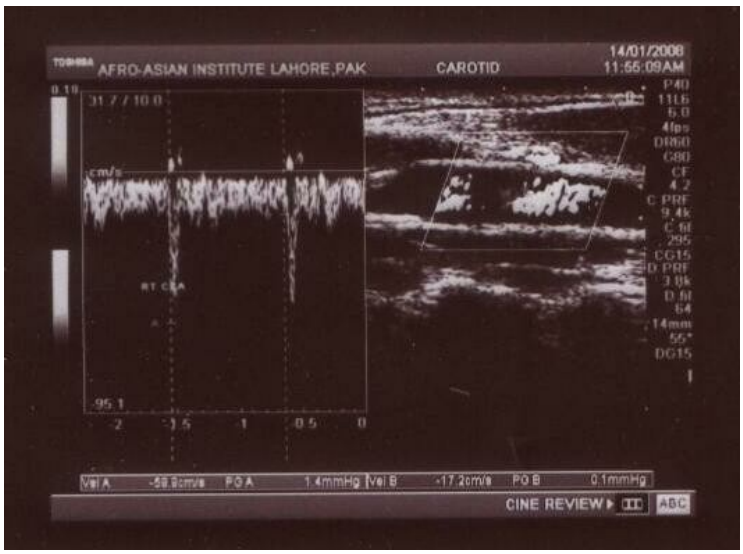

Fig: 29 PSV of Rt. CCA

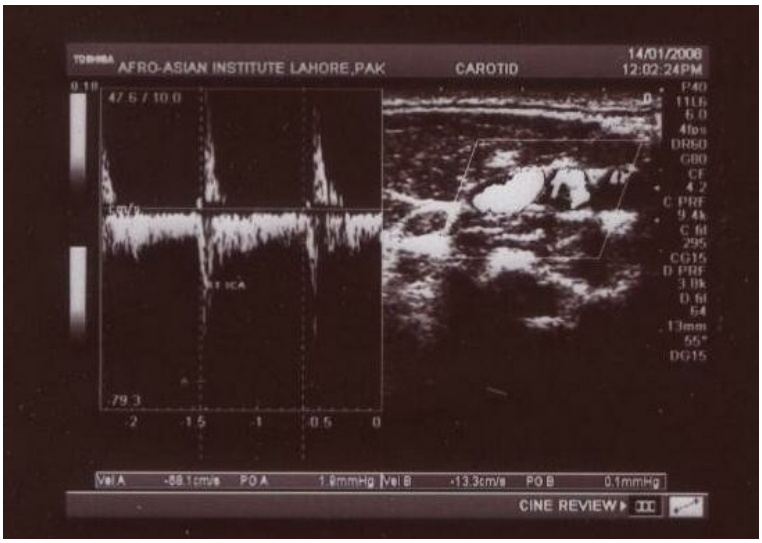

Fig: 30 PSV of Rt. ICA