Role of Color Coded Duplex Imaging in the Assessment of Carotid Arteriosclerosis

By

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List of Abbreviations

CCA ..... Common Carotid artery

ICA.....Internal Carotid artery

ECA.....External Carotid artery

PSV.....peak systolic velocity

END.....End diastolic velocity

TIA.....transient ischaemic attack

RBCs.....Red blood cells

RNID.....reversible neurological deficit

HZ.....Hertz

PWD.....pulsed wave Doppler

CWD.....Continuous wave Doppler

IJV.....Internal Jugular vein

Exam.....Examination

Acknowledgement

Thanks to God for helping me to complete this work.

I would like to express my deep thanks and supreme gratitude to ***Prof. Dr. Sayed Ahmed Sayed,*** Prof. Of Diagnostic Radiology , Faculty of medicine, Al-Azhar University, for his kind guidance, valuable support, sincere directions and encouragement throughout this work. I find no words that could express my gratitude to him.

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INTRODUCTION

Until the last decade, the only reliable diagnostic test for carotid atherosclerosis was angiography. This test gives good information about the lesion severity and some reliable information about lesion complication (ulcerative thrombus). ***(Pollak, and O.Leary et al.,1990).***

Doppler ultrasound is promising as a screen modality for extra-cranial atherosclerotic disease. It is non invasive and inexpensive relative to magnetic resonant angiography (MRA).and very high sensitivity and specificity has been reported for detection of clinical significant disease ***(Derdeyn et al., 1995).***

Atherosclerosis is a degenerative disease causing narrowing of the lumen and obstruction of blood flow or ulceration and thrombosis ***(Zarins and Glagov., 2000).***

Doppler screening of carotid artery is helpful in detecting the subgroup of patient at increasing risk of stroke patients with critical carotid artery stenosis should be subjected to angiography ***(Rath et al., 2001).***

Thus, the practice of using Doppler screening to exclusion patients from arteriography is superior to performing arteriography in all symptomatic patients ***(Derdeyn et al., 1995).***

Alternatively some authors have advocated the use of Doppler ultrasound alone or in conjunction with MRA preoperatively to define the degree of stenosis and anatomy of extra cranial carotid artery. These approaches sacrifice diagnostic accuracy in return to return to reduce arteriography complication ***(Derdeyn et al., 1995).***

Aim of the work

The aim of this work is to evaluate the role of color duplex sonography in diagnosis of carotid atherosclerotic disease and detection of early atherosclerotic change in carotid artery and detection of complication.

PHYSICS OF COLOR DOPPLER ULTRASOUND

Doppler Effect:

This effect was discovered by Christian Doppler in 1842, and was named after him. He realized that the wave length recorded by an observer depends on movement of the source and the observer to one another ***(Klews., 1995).***

The Doppler Effect is the change or shift in frequency or wavelength of a wave due to relative movement between the sound source and the receiver ***(Tegeler and Ratanakorn., 1999).***

Doppler ultrasonography in medical diagnostic imaging concerns behavior of high frequency sound waves as they are reflected off moving fluid usually blood. When high frequency sound beam meets a moving structure, such as blood flow in vessels, the reflected sound returns from the moving red blood cells at different frequency. The returning frequency will be increased if flow is toward the sound source (transducer) and decreased if flow is away from sound source ***(prince et al., 1998).***

The difference between transmitted and reflected frequencies is called "The Doppler frequency" ***(Tegeler and Ratanakorn, 1999).***

In daily life has probably already noticed the Doppler effect. This effect is responsible for the variation in pitch of the sound wave

from an ambulance siren as it moves towered and away from observer. The siren pitch becomes higher as the ambulance approaches and lowers as vehicle departs. In medicine, the Doppler effect is helpful in localizing blood vessels and determining optimal site for velocity measurements. The Doppler frequency shift can be converted to audible signal, which help in differentiation between veins and arteries. Typically veins have a low-pitched hum, whereas arteries have an alternating pattern with a high-pitched systolic component and low-pitched diastolic component ***(prince et al.,1998)***

The Doppler Effect can be used to determine the speed and direction of flow in blood vessels and sometimes the flow disturbance or turbulence (spectral broadening). ***(Tegeler and Ratanakorn., 1999).***

Doppler Equation:

The Doppler frequency shift is directly related to the speed of the reflector and transmitted frequency; it is inversely related to the angle of insulation ***(Tegeler and Ratanakorn., 1999)***. The Doppler equation is:

$$\Delta F = 2 \frac{FO\ V\ COS\ \theta}{C}$$

WHERE:

ΔF = measure Doppler frequency shift
V= Velocity of moving blood
F0=frequency emitted from the transducer
C= Speed of sound in tissue
Θ= Doppler angle (angle between direction of blood and direction of propagation of sound wave) ***(Landwehr., 1995).***

Then the direction of sound beam is parallel to the direction of flow (angle of insulation θ = 0 and cos θ =1) then the highest Doppler frequency shift is obtained, whereas it become impossible to accurately determine at insulation angle 90, cos 90 =0 ***(Tegeler and Ratanakorn., 1999).***

Doppler instrument:

There are two type of Doppler instrument in clinical medicine, the continuous wave (CW) and pulsed wave (PW) Doppler machine.

1. **Continuous wave Doppler system (CWD):**

Uses two transducer crystals mounted side by side, one continuously emitting and the other continuously receiving sound waves. Very high velocities can be recorded and peak velocities obtained but significant depth resolution which easy with pulsed Doppler is not possible ***(Sutton et al., 1998).***

Continuous wave Doppler transducer cannot localize the depth at which signal originated. ***(Tegeler and Ratanakorn., 1999)***.

Since this is a simple system it is cheap it can be used when superficial vessels are examined. CWD is used to check for the presence of flow in superficial arteries and to monitor umbilical artery flow since the cord lies in amniotic fluid no other confusing vessels are within ultrasound beam ***(Prince et al., 1998).***

2. **Pulsed wave Doppler:**

Utilizes a single transducer to emit short bursts of energy, which are received and recorded in the intervals between emission .this method permits precise focusing of the ultrasound beam on sample volumes as small as 2 or 3 mm diameter. How ever the necessity for repeated pulses render the method less accurate than continuous wave Doppler for measuring high velocity flows and recording high peak velocity. ***(Sutton et al.,1998)***

PWD is used to detect the presence of blood flow in a selected vessel at a given depth when there are several vessels within the ultrasound beam. Only those signals from a vessel at a known depth are displayed and analyzed. Angle gate, is not necessarily a better since it diminishes the sensitivity of the signal detection to obtain the highest velocity in a stenotic vessel, move a similar gate though the vessel and listen for the highest audible sound ***(Prince et al., 1998)***

Doppler imaging combination:

Duplex system:

A duplex system is one that enables two dimensional ultrasound pulse echo imaging to guide the placement of an ultrasonic Doppler beam and allow anatomical location of the origin of Doppler signals to be identified. Imaging systems may be static or real time. ***(Peter., 1995)***

Color-coded Duplex imaging:

Method allowing the simultaneous visualization of soft tissue structure and blood flow. The color-coded flow information is superimposed on gray scale imaging .when motion is detected, the gray scale value displaced that location is replaced by appropriate color value. ***(Klews., 1995)***

The red color indicate positive Doppler shift (flow towered the transducer). And the blue color indicate negative Doppler shift (flow away from transducer) and the green color for turbulent flow with increasing depth proportionate to the turbulence ***(Kremakau., 1992).***

Power Doppler:

Power Doppler also superimposes Doppler information but on gray scale image, but with out any directional information .it displays only the amount of energy and more sensitive. The advantage of power Doppler the signal is stronger allowing identification of smaller vessels with lower velocity flow than for color Doppler and not angle dependent than color Doppler. This useful for vessels, which run perpendicular to beam such as inferior vena cava .because power Doppler, is not angle dependent the flow is demonstrated throughout the vessels even at 90 to beam. ***(Bates., 1999)***

Doppler Artifact:

Artifact that distort the size shape or brightness of the image, (Aliasing). Aliasing is an artifact occurring in the pulsed Doppler instrument.Aliasing cross from one map end to another one with

bright boundary between .Flow reversal cross the base line through the wall filter and has dark boundary in between .Aliasing is more liable to occur with high velocity jets associated ***(Middelton et al., 1988).***

Aliasing can be diminished by the following methods:

1- Increasing the beam flow angle, which has effect of decreasing the measuring velocity.

2- Increasing pulse Repetition frequency or velocity rang either directly or by decreasing transducer –target distance.

3- Base line shift to set the zero velocity at either the tope or bottom of the recording paper. This allows all velocity range to either positive or negative direction .this simplest method to over come aliasing.

(Middelton et al., 1988).

Vascular Anatomy of Carotid System

Common Carotid Artery Fig (1):

The right common carotid artery arises from the brachocephalic artery behind the right sternoclavicular joint. The left artery arises from the arch of the aorta in the superior mediastinum. The common carotid artery runs upward through the neck, from the sternoclavicular joint to the upper border of the thyroid cartilage, where it divides into the external and internal carotid arteries. At its point of division, the terminal part of the common carotid artery or the beginning of the internal carotid artery shows a localized dilatation, called the carotid sinus. The tunica media of the sinus is thinner than elsewhere, but the adventitia is relatively thick and contains numerous nerve endings derived from the glosso-pharyngeal nerve. The carotid sinus serves as a reflex pressoreceptor mechanism: A rise in blood pressure causes a slowing of the heart rate and vasodilatation of the arterioles. The carotid body is a small structure that lies posterior to the point of bifurcation of the common carotid artery. It is innervated by the glosso-pharyngeal nerve and it is chemoreceptor, being sensitive to excess carbon dioxide and reduced oxygen tension in the blood. Such a stimulus reflexly produces a rise in blood pressure and heart rate and an increase in respiratory movements.

The common carotid artery is embedded in the carotid sheath throughout its course and is closely related to the internal jugular

vein and vagus nerve. A part from the two terminal branches, the common carotid artery gives off no branches.

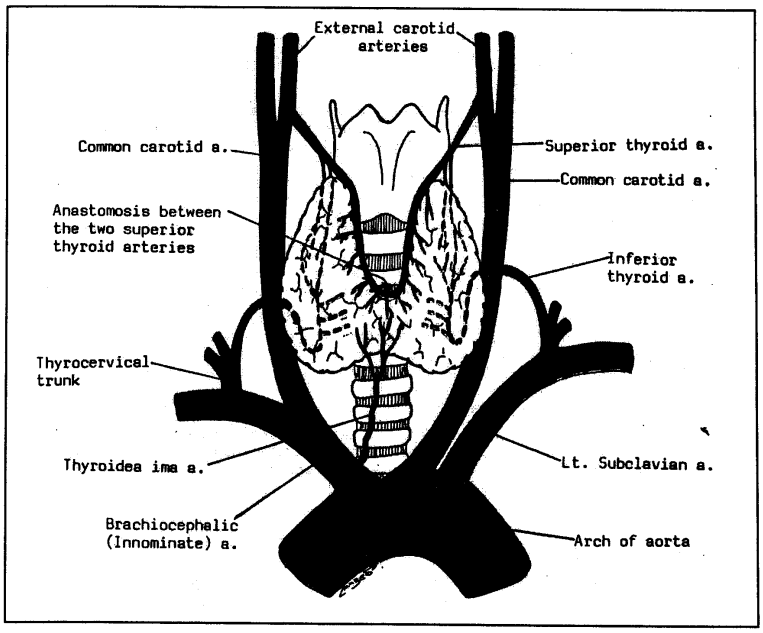


Fig (1): Diagram show the origin of common carotid artery internal and external carotid artery. ***(Quoted from clinical Anatomy., 1996)***

Relations:

Anterolaterally: The skin, the fascia, the sterno-cleidomastiod, the sterno-hyiod, the sterno-thyroid, and the superior belly of the omoyhoid).

Posteriorly: The transverse processes of the lower four cervical vertebrae the prevertebral muscles, and the sympathetic trunk. In the lower part of the neck are the vertebral vessels.

Medially: The larynx and pharynx and below these, the trachea and esophagus. The lobe of the thyroid gland also lies medially.

Laterally: The internal jugular vein and, posterolaterally, the vagus nerve.

External Carotid Artery:

The external carotid artery is one of the terminal branches of the common carotid artery. It supplies structure of the neck, face, and scalp, it is also supplies the tongue and the maxilla. The artery begins at the level of the upper border of the thyroid cartilage and terminates in the substance of the parotid gland behind the neck of the mandible by dividing into the superficial temporal and maxillary arteries.

At its origin, where its pulsations can be felt, the artery lies within the carotid triangle. At first medial to the internal carotid artery, but as it ascends in the neck, it passes backward and laterally. It is crossed by the posterior belly of the digastric and the stylohyoid.

Relations:

Anterolaterally: The artery is overlapped at its beginning by the anterior border of the sternocleidomastoid. Above this level the artery is comparatively superficial being covered by skin and fascia. It is crossed by the hypoglossal nerve, the posterior belly of the digastric muscle, and the stylohyoid muscles. Within the parotid gland it is crossed by the facial nerve .The internal jugular vein first lies lateral to the artery and then posterior to it.

Medially: The wall of the pharynx and the internal carotid artery. The stylopharyngeus muscle, the glossopharyngeal nerve, and the pharyngeal branch of the vagus pass between the external and internal carotid arteries.

Branches Fig (2):

The branches of the external carotid artery are as follows:

1. Superior thyroid artery.
2. Ascending pharyngeal artery.
3. Lingual artery.
4. Facial artery.
5. Occipital artery.
6. Posterior auricular artery.
7. Superficial temporal artery.
8. Maxillary artery.

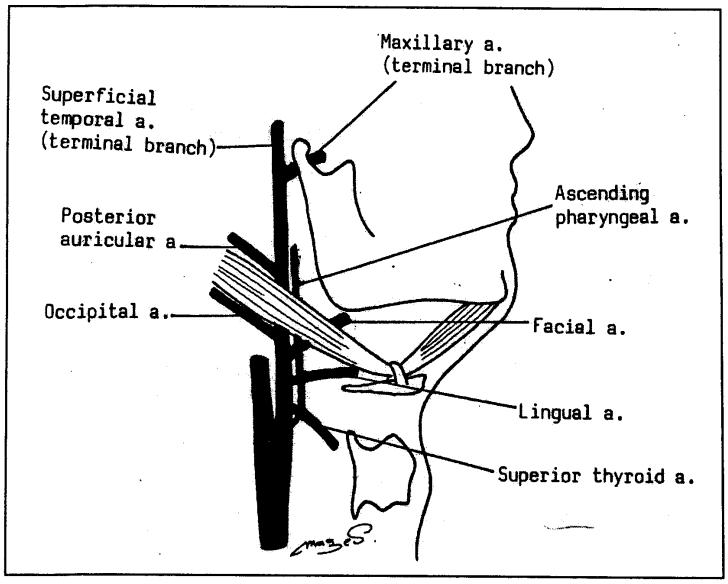


Fig (2): Branches of external carotid artery ***(Quoted from Clinical Anatomy., 1996)***

1) The superior thyroid artery arises from the external carotid artery near its origin). It passes almost vertically downward to reach the upper pole of the thyroid gland. It gives off (a) a branch to the sternocleidomastoid muscle and (b) the superior laryngeal artery, which pierces the thyrohyoid membrane with the internal laryngeal nerve.

2) The ascending pharyngeal artery is a long, slender vessel that ascends on the wall of the pharynx, which it supplies.

3) The lingual artery arises from the external carotid artery, opposite the tip of the greater cornu of the hyoid bone. It loops upward to enter the submandibular region. The loop of the

artery is crossed superficially by the hypoglossal nerve. It supplies the tongue.

4) The facial artery arises from the external carotid artery, just above the level of the tip of the greater cornu of the hyoid bone. It arches upward deep to reach the posterior part of the submandibular salivary gland supplies the face.

5) The occipital artery arises from the external carotid artery, opposite the facial artery passes upward and reaches the back of the scalp. It terminal part accompanies branches of the greater occipital nerve to supply the back of the scalp.

6) The posterior auricular artery arises from the external carotid artery, at the level of the upper border of the posterior belly of the digastric. It passes backward to reach the auricle.

7) Superficial temporal artery.

8) Maxillary artery.

Internal Carotid Artery:

The internal carotid artery is one of the terminal branches of the common carotid artery. It supplies the brain, the eye, the forehead, and part of the nose. The artery begins at the level of the upper border of the thyroid cartilage and ascends in the neck to the base of the skull. It enters the cranial cavity through the carotid canal in the petrous part of the temporal bone. .It lies embedded in the carotid sheath with the internal jugular vein and vagus verve. At its beginning, it lies superficially in the carotid triangle and then ascends deep to the parotid gland. The internal carotid artery gives off no branches in the neck.

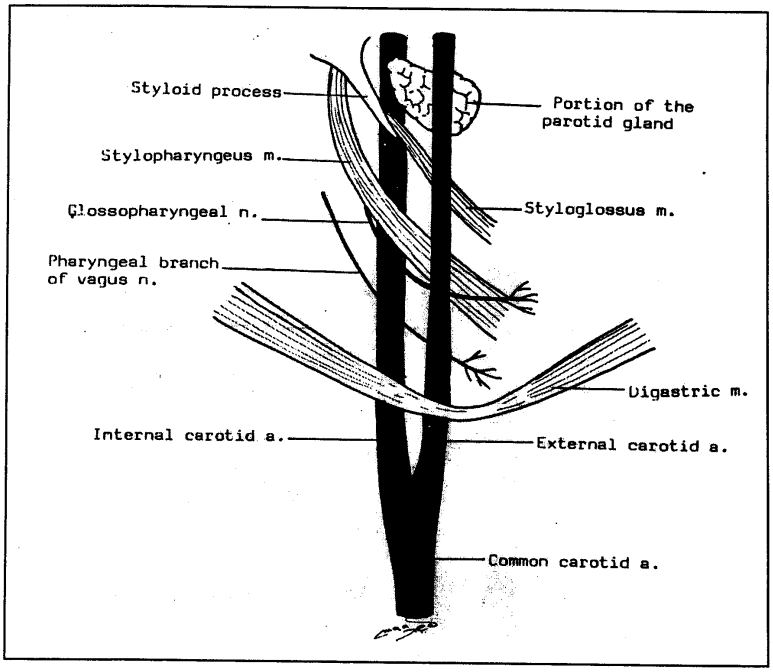


Fig (3): The structures pass between internal carotid and external carotid artery ***(Quoted from clinical Anatomy., 1996)***

Relations Fig (3):

Anterolaterally: Below the digastric lies the skin, superficial, fascia, the anterior border of the sternocleidomastoid, and the hypoglossal nerve. Above the digastric lie the stylohyoid muscle, the stylopharyngeus muscle, the glosso-pharyngeal nerve, the pharyngeal branch of the vagus, the parotid gland, and the external carotid artery.

Posteriorly: The sympathetic trunk, the longus capitis muscle, and the transverse processes of the upper three cervical vertebrae.

Medially: The pharyngeal wall and the superior laryngeal nerve.

Laterally: The internal jugular vein and the vagus nerve.

Course of cerebral portion of Internal Carotid Artery:

The internal carotid artery emerges from the cavernous sinus on the medial side of the anterior clinoid process. It then turns backward to the region of the lateral cerebral sulcus. Here, it divides into the anterior and middle cerebral arteries.

Branches of the Cerebral Portion of the Internal Carotid Artery:

1) The ophthalmic artery arises as the internal carotid artery emerges from the cavernous sinus. It enters the orbit through the optic canal, below and lateral to the optic nerve. It supplies the eye and other orbital structures, and its terminal branches supply the frontal area of the scalp, the ethmoid and frontal sinuses, and the dorsum of the nose.
2) The posterior communicating artery is a small vessel that runs backward to join the posterior cerebral artery.
3) The choroidal artery, a small branch, passes backward, enters the inferior horn of the lateral ventricle, and ends in the choroid plexus.
4) The anterior cerebral artery runs forward and medially and enters the longitudinal fissure of the cerebrum. It is joined to the artery of the opposite side by the anterior communicating

artery. It curves backward over the corpus callosum, and its carotid branches supply all the medial surface of the cerebral cortex as far back as the parieto-occipital sulcus. They also supply a strip or cortex about 1 inch (2.5 cm) wide on the adjoining lateral surface. The anterior cerebral artery thus supplies the "leg area" of the precentral gyrus. Several central branches pierce the brain substance and supply the deep masses of gray matter within the cerebral hemisphere.

5) The middle cerebral artery, the largest branch of the internal carotid, runs laterally in the lateral cerebral sulcus. Carotid branches supply the entire lateral surface of the hemisphere, except for the narrow strip supplied by the anterior cerebral artery, the occipital pole, and the inferolateral surface of the hemisphere, which are supplied by the posterior cerebral artery. This artery thus supplies all the motor area except the "leg area". Central branches enter the anterior perforated substance and supply the deep masses of gray matter within the cerebral hemisphere.(**Staskin et al., 1996**)

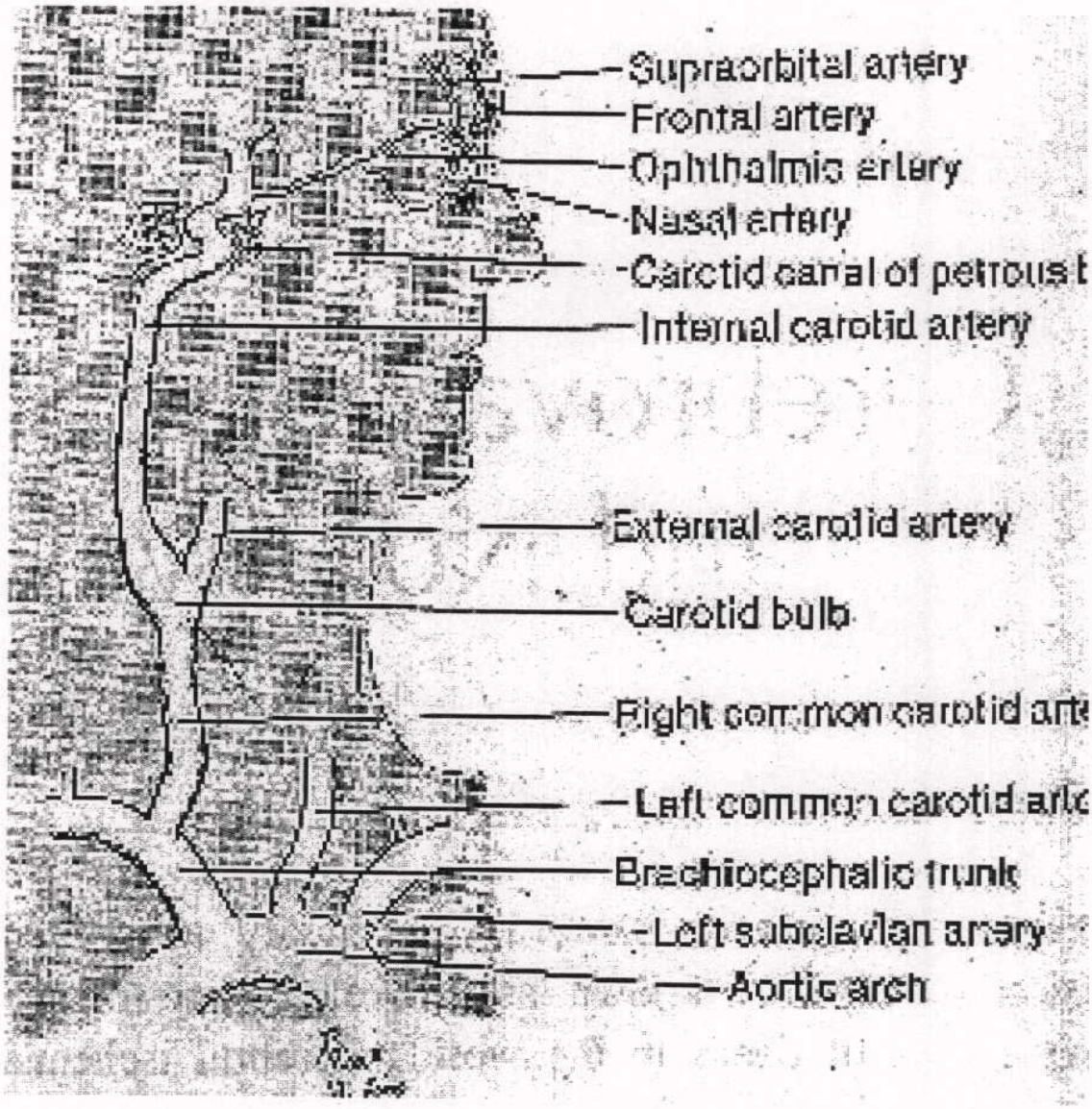


Fig (4): Aortic branches and internal carotid arterial system ***(Quoted from Diagnostic ultrasound, 1998)***

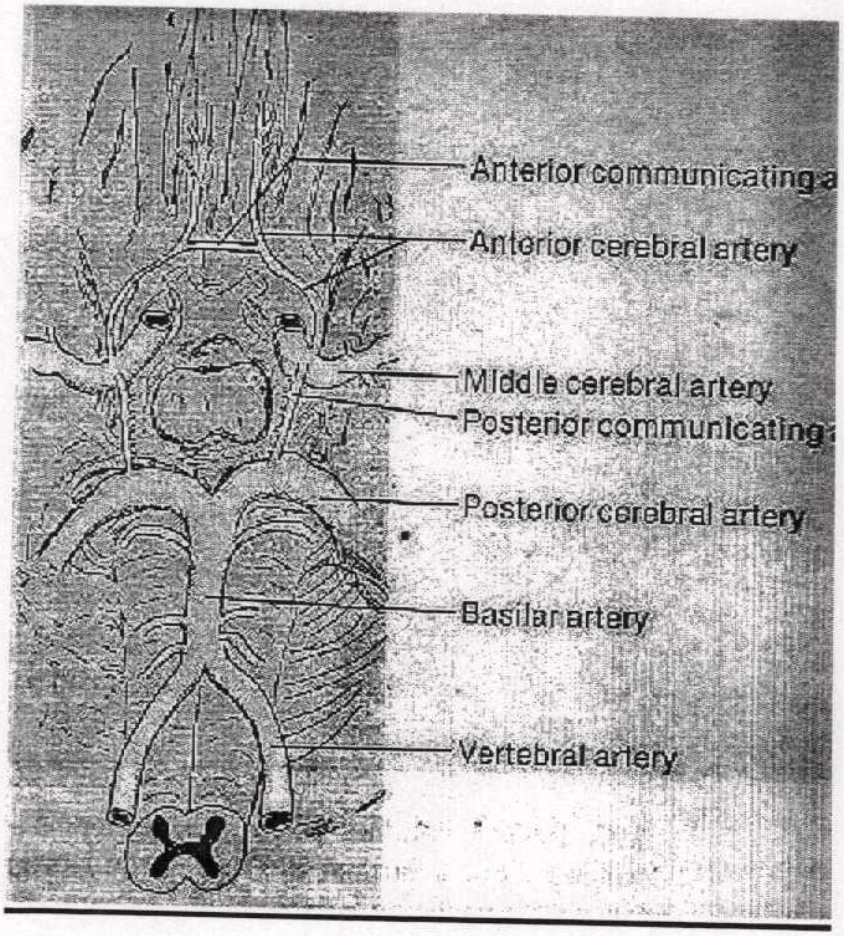


Fig (5): Diagram show intracerebral branches of ICA and Circle of wills ***(Quoted from Diagnostic Ultrasound, 1998)***

Vertebral Artery:

The vertebral artery arises from the first part of the subclavian artery and ascends in the neck between the longus coli and the scalenus anterior muscles. It passes in front to transverse process of the seventh cervical vertebra and enters the foramen in the transverse process of the sixth cervical vertebra. It then ascends through the foramina in the transverse processes of the upper six cervical vertebrae. Having emerged from the transverse process of the atlas, it curves backward behind the lateral mass of the atlas. It then passes medially, pierces the dura mater, and enters the vertebral canal. The vertebral artery then ascends into the skull through the foramen magnum to supply the brain.

Relations of the Vertebral Artery: (Fig. 6)

Anteriorly: The common carotid artery; on the left side, it is crossed by the thoracic duct.

Posteriorly: The transverse process of the seventh cervical vertebra, the cervicothoracic sympathetic ganglion (stellate ganglion), and the anterior rami of the seventh and eighth cervical nerves. As the artery ascends through the foramina in the transverse processes, it lies in front of the anterior rami of the cervical nerves.

Branches: Spinal and muscular branches arise from the vertebral artery. The spinal branches enter the vertebral canal through the intervertebral foramina. ***(Staskin et al., 1999)***

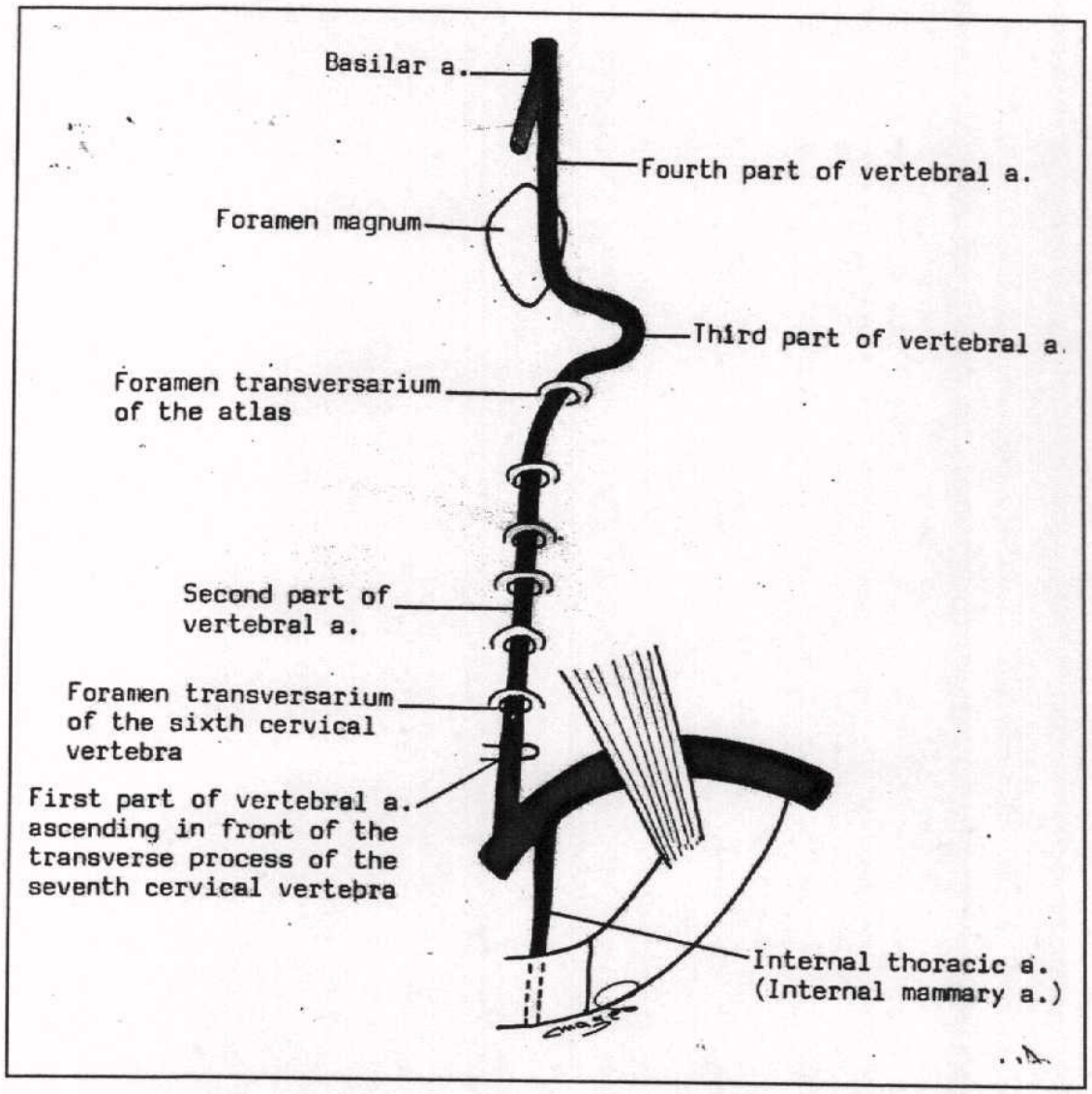


Fig (6): Diagram show course of vertebral artery ***(Quoted from Atlas of color Anatomy, 2000)***

HISTOLOGY OF THE ARTERIAL WALL

Structures of the arterial wall composed:

1- **Intima**

The intima, the innermost layer of the arterial wall, extend from the luminal surface to the internal elastic lamina. The luminal surface is lined by the endothelium, Between the endothelium and internal elastic lamina, the intima is normally very narrow, with endothelium lying directly on the internal elastic lamina and containing only few scatter leucocytes, smooth muscle cells and connective tissue fibers, It the region that atherosclerotic change develop. ***(Zarins and Glagov, 2000)***

Under normal circumstances, the vascular endothelium functions as an anti-thrombotic surface and contributes to the regulation of vascular tone and artery lumen diameter through the secretion of vasoconstrictors. (e.g. angiotensin II) and vasodilator and inhibitors of platelet aggregation (.e.g. Prostacyclin and endothelium derived relaxing factor) such as factors maintain the smooth muscle cells of the media in contractile, non proliferate phenotype with low cholesterol ester content. In response to endothelial cell activation or injuries, endothelial cell become increasingly permeable to low density lipoprotein, have higher replicative rates, develop prothrombotic properties, and express surface glycoprotein that promote the adhesion and ingress of neutrophils, monocytes and platelets. Endothelial cells and

monocytes reales cytokines grows factors and leucotrienes inducing prostacyclin production, which further promotes monocytes adhesions. The net effect of cytokine and growth factor production is the stimulation of smooth muscle cells proliferation and migration. As a result of these changes, extra cellular lipid as well as foam cells containing cholesterol esters accumulate in the intima. Also physical and mechanical endothelial disruption and denudation may not be reactions that initiate or precipitate events in atherosclerotic plaque formation, biological reactions of the endothelium and arterial wall during injury and repair may play important roles in the proliferative and lipid deposition stage of plaque formation. ***(Zarins and Glagov, 2000)***

2- Media.

The media extends from the internal elastic lamina to the adventitia although an external elastic lamina demarcates the boundary between the media and adventitia in many vessels, distinct external elastic lamina may not be present particularly in vessels with a thick and fibrous adventitial layer. The media consist of closely packed layers of smooth muscle cells in close association with elastin and collagen fibers. The smooth muscle cell layers are composed of groups of similarly oriented cells, each surrounded by a common basal lamina and a closely associated interlacing basketwork of type 3 collagen fibrils arranged so as to tighten about the cell groups as the media is brought under tension; this

configuration tends to hold the groups of cells together and prevent excessive stretching or slippage. ***(Zarins and Glagov,, 2000)***

3- Adventitia

The adventitia is composed of fibrocellular connective tissue and contains a network of vasa vasorum composed of similar arteries, arterioles, capillaries and venous channels as well as nerves that mediate smooth muscle tone and contraction. The adventitia varies in thickness and organization. In atherosclerotic arteries, however, increasing intimal plaque thickness may be associated with atrophy of the underlying media. Under these circumstances, a thickened adventitia may contribute to tensile support. ***(Zarins and Glagov., 2000).***

SONOANATOMY OF THE CAROTID ARTERIES

In transverse scan, the common carotid artery (CCA) appears as a rounded lumen posterior or lateral to the irregular shaped jugular vein. The normal carotid retains its rounded appearance to the level of the bulb, where the diameter of the vessels becomes larger and begins to elongate as the bifurcation approaches ***(Rae, 1995).*** The carotid bulb appear as club–like dilatation in the area of carotid bifurcation ***(Landwehr, 1995).***

The majority of the carotid diameters by ultrasound range from 6.0 to 8.0 mm for the common carotid artery in the women. The carotid diameters are approximately 0.5 to 1.0 mm larger in men ***(Cranley et al., 1989).*** The internal carotid artery (ICA) and external carotid artery (ECA) are seen as two separate round lumina forming from the bulb as the transducer moves superiorly ***(Lee et al., 1999).***

Just above the bifurcation, the typical appearance of the ICA, which is slightly larger and located above, lateral and superficial to ECA. Normal ultrasound diameter for distal ICA range from 3.5 to 4.5 mm. And 3.0 to 4.0 for the ECA in the women. The carotid diameter are approximately 0.5 to 1.0 mm larger in men ***(Cranley et al., 1989).***

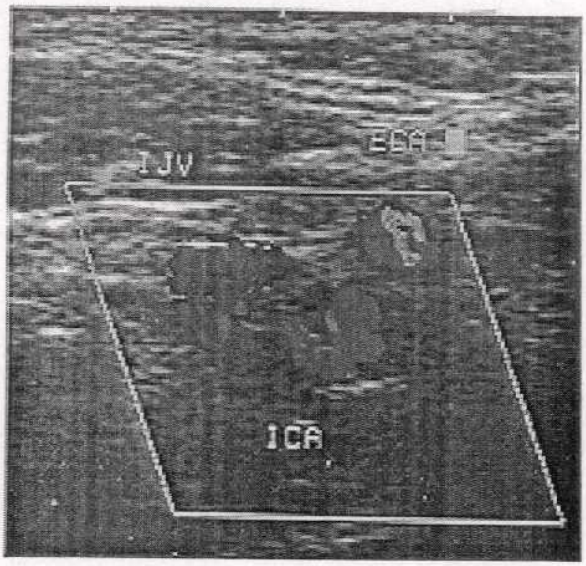


Fig. (7): Transverse image show internal, external carotid and internal jugular vein ***(Quoted from Taylor, 1995)***.

Sometimes the ICA and ECA are seen to be rotated even farther relative to each other in what is term "reversal bifurcation" with the ICA below and medial to the ECA and sometimes, the two vessels are of more equal size. There are sex basic criteria by which one can identify the two arteries:

- The most frequent criterion is a relative orientation in transverse view. This holds true in perhaps 90 % of cases.
- The second criterion is relative size just above the bifurcation .the ECA generally does not have a bulb as the ICA.
- The third criterion is distal direction .Although the arteries appear to be reversed at the level of the

bifurcation. By following them distally they sometimes seen cross back over and one can appreciate the ICA lying more medially than the ECA at a higher level.

- The ECA is directed anteriorly toward the mandible and face, whereas the ICA is directed posteriorly and superiorly, roughly toward the mastoid process of the skull. ***(Zwiebel., 1982).***
- The fifth criterion is Doppler signal characteristic biphasic signal is obtained in the ICA because of out flow resistance to the brain.
- The final criterion is the presence of branches of the ECA. The ICA has no braches in the neck ***(Cranley et al., 1989).*** The ECA taper markedly as it ascends, due to its numerous side branches. ***(Land wehr., 1995).***
- In longitudinal scan (Fig. 6), the CCA, ICA, and ECA appear to have echo-free fluid–filled Lumina bordered by two bright reflications, the arterial wall ***(Rae., 1995).*** On scan occasionally detect reflection from blood cells in areas of sluggish flow (lumina jugular vein), but this phenomenon is seen in arteries only when significant obstruction is present. ***(Zwiebel., 1982).***

The CCA can be followed superiorly from the supra-clavicular area. the irregular without thicker walled the internal jugular vein lies anterior or on one side of the CCA, to differentiate between them the vein has lack of pulsatility, physic, dilatation and collapse with respiration and collapse from light pressure from transducer while the artery thicker

wall, regular pulsatility and much more stable appearance ***(Rae, 1995)***.

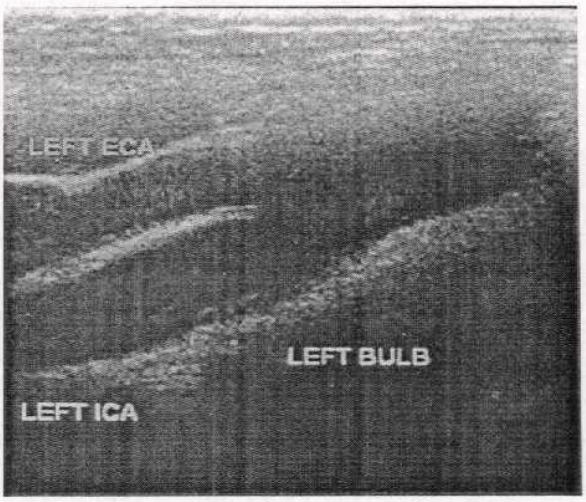


Fig (8): Longitudinal scan show common carotid external carotid & internal carotid artery ***(Quoted from Talyor, 1995).***

As the transducer moves up the neck, the carotid is seen to widen up to the area of the carotid bulb. The Y-shape of the carotid bifurcation is infrequently seen. The bifurcations tend to be rotated differently in certain individuals, and the appearance depends on ability to obtain the both vessels in the same view. Then the ICA and ECA appear. As one proceeds outward from the echo-free central lumen, one first encounters the dark gray zone. This zone is often referred to as the "intimal stripe", but in fact, the true intima is only a few cell layers thick, measured in microns. The surface echo is bright not because one is seeing the intimal tissue but because this solid/liquid interface with the flowing blood

makes a reflective boundary and one is seeing the abrupt transition between tissues of different densities. The rest of this intimal strips in fact is composed of the inner two third of the media or smooth muscle layer of the arterial wall. This is the portion that does contain vasa vasorum. It's similar to echo density to the sternomastiod muscle .Normally the intima measure up to 1mm in thickness. As one proceeds outward from the intimal stripe, one see a layer of very bright echoes representing outer medial and adventitial layers of the arterial wall. Superficial to the CCA is the IJV. Preceding outward the third layer that is not bright as adventitial layer but more brightly echogenic than the surrounding tissue. This corresponding to peradventitial tissue, carotid sheath ***(Cranley et al., 1989).***The following structures are then identified as we proceeded more superficial the strap muscles (sternothyriod, sternohyiod and sternocleidomastoid) then the deep cervical fascia platysma muscle the superficial fascia and finally the skin. ***(Williams, 1995).***

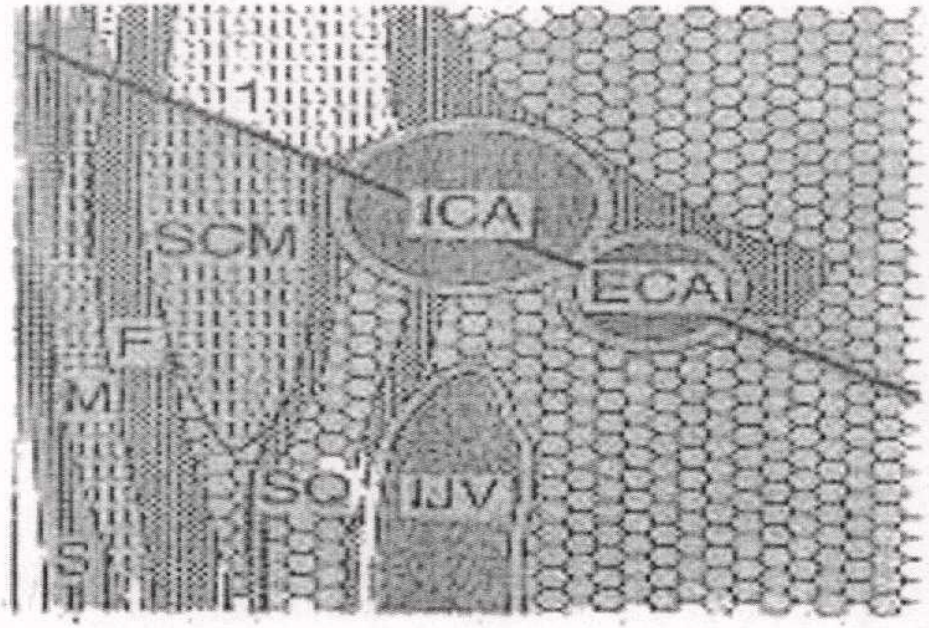


Fig (9): Diagram of the CCA just above the bifurcation. Note that ICA is located above and superficial to the ECA. S = Skin, M = Platysma muscle, F = Fascia, SCM = Sternocleidomastoid muscle, SQ = Subcutaneous tissue, IJV = Internal jugular vein, ICA = internal carotid artery, ECA = External carotid artery ***(Quoted from Cranley et al., 1989).***

CAROTID DUPLEX TECHNIQUE

General consideration:

A technical protocol should be established within each vascular laboratory for every examination performed in that laboratory. The use of technical protocols ensures that each exam. is comprehensive and includes the diagnostic features that are deemed important by the laboratory directors ***(Zwiebel et al., 1992).***

PROTOCOL FOR CAROTID DUPLEX EXAM:

Longitudinal survey:

lateral transducer position:

- Begin at the clavicle and move cephalad.
- Identify carotid bifurcation, ECA & ICA.
- Localize plaque and areas of obstruction, to the CCA, ICA or ECA.

Postero-lateral transducer position:

Trace the ICA as far cephalad as possible searching for evidence of pathology.

Longitudinal evaluation of pathology:

Lateral or postero-lateral transducer position:

- Document the extent of plaque deposition.
- Document the surface & internal characteristics of plaque.
- Measure the peak systolic and end-diastolic velocities and, compare with the proximal CCA velocities.
- Note degree of poststenotic flow disturbance.

Anterior transducer position:

Re-examine the entire carotid bifurcation to double check the findings made previously from a different perspective.

Transverse examination:

Transverse transducer position:

- Begin at the clavicle & move cephalad.
- Identify the carotid bifurcation, ECA & ICA.
- Localize plaques and areas of obstruction to the CCA, ECA or ICA.
- Document surface & Internal characteristics of plaque.
- Measure residual lumen size and percent diameter reduction ***(Wolverson et al., 1983).***

These aspects of the carotid examination (exam.) are included for the sake of completeness but their value is questionable.

Ascertain whether or not the B mode measurements of stenosis correspond with the Doppler spectral measurements.

The carotid arteries are examined with the patient in a supine position with the examiner seated at the patient's head. In some laboratories, the sonographer may choose to sit at the side of the patient. Another method is to examine the patient in a reclining chair equipped with a headrest. Such as a dental chair. Exposure of the neck is maximized by having the patient drop the ipsilateral shoulder as far as possible. Neck exposure also is facilitated by rotating the patient's head away from the side being examined. Sonographer

should feel free to vary the position of the head and neck in ways that optimize the examination. Methods for recording the duplex carotid exam, includes video tape, hard copy images, and written notes. In most laboratories, a report form is used, upon which important data are noted, as well as some method for recording images. Regardless of the method employed. It is imperative to document clearly all findings referable to the severity of plaque and the degree of stenosis. This documentation is particularly important for the follow up assessment of plaque stenosis ***(Comerate et al., 1981)***.

Step by step carotid exam technique:

In general terms, the carotid duplex examination follows these steps:

Step 1: The examination begins with a longitudinal survey of the cervical carotid arteries, with the transducer in a lateral position. The common carotid is identified at the clavicle, and the transducer is moved cephalad along the artery until the carotid bifurcation is seen. The carotid bifurcation may be profiled in some patients.

In other individuals it may not be possible to obtain this view. The location of the bifurcation is then ascertained by shifting back and forth between the internal and external carotid branches. The identity of the external and internal carotid arteries must be confirmed without question using the observations as follow (Table 1).

After confirming the internal carotid artery and external carotid artery identity, the internal carotid artery is followed as far cephalad as possible, using a posterolateral transducer position.

Table (1): Shows the main differences between internal and external carotid arteries.

	ECA	ICA
Size	Usually smaller	Usually larger
Branches	Present	Non
Orientation	Oriented anteriorly towards the face	Oriented posteriorly towards the mastoid process
Doppler characters	High pressure flow pattern	Low pressure flow pattern

The location of plaque and major points of obstruction are noted during the survey examination, but the severity of individual lesions is not addressed in details until the survey is completed.

Step 2: After the longitudinal survey is completed, and the bifurcating branches are correctly identified, areas where plaque had been identified are returned to and each abnormal area is scrutinized.

The extent of plaque formation is documented as well as internal plaque characteristics, and the degree of luminal narrowing (from color Doppler and Spectral Doppler information).

This detailed examination of atheromatous lesions is initially conducted from a lateral and posterolateral transducer positions.

Step 3: After the carotid vessels have been examined from the lateral and posterolateral transducer positions, the carotid arteries are reexamined from an anterior transducer position which is roughly at right angles to the other longitudinal positions. The plaque and obstruction are reevaluated from this position to enhance the three dimensional perspective of atheromatous lesions.

Step 4: When the Longitudinal exam, is completed, the carotid vessels should be studied from a transverse position. The transverse exam, is begun at the clavicle. The transducer is then moved cephalad where the carotid arteries are observed on the color Doppler image, at the carotid bifurcation the identification of the internal and external carotid branches should once again be confirmed. The location of plaque stenosis (common, external or internal carotid segments) should be reaffirmed. Visual (Color Doppler) assessment of stenosis severity also is recommended during transverse exam. The visual findings should correlate with spectral Doppler data. If a discrepancy is evident between the visual and spectral data, then further assessment of a lesion is indicated ***(Zwiebel et al., 2000).***

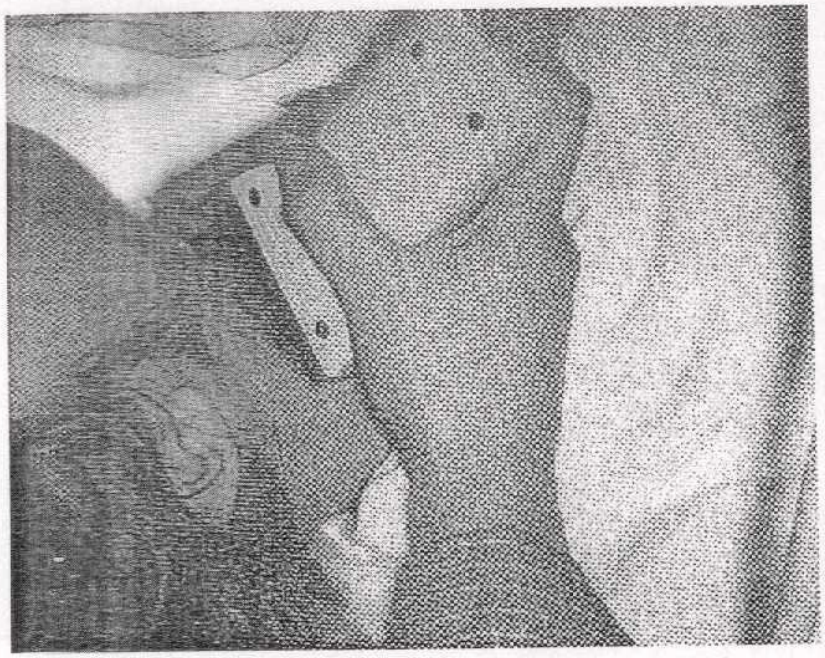


Fig (10): Transducer positioning for transverse imaging plan ***(Quoted from Diagnostic ultrasound, 1998).***

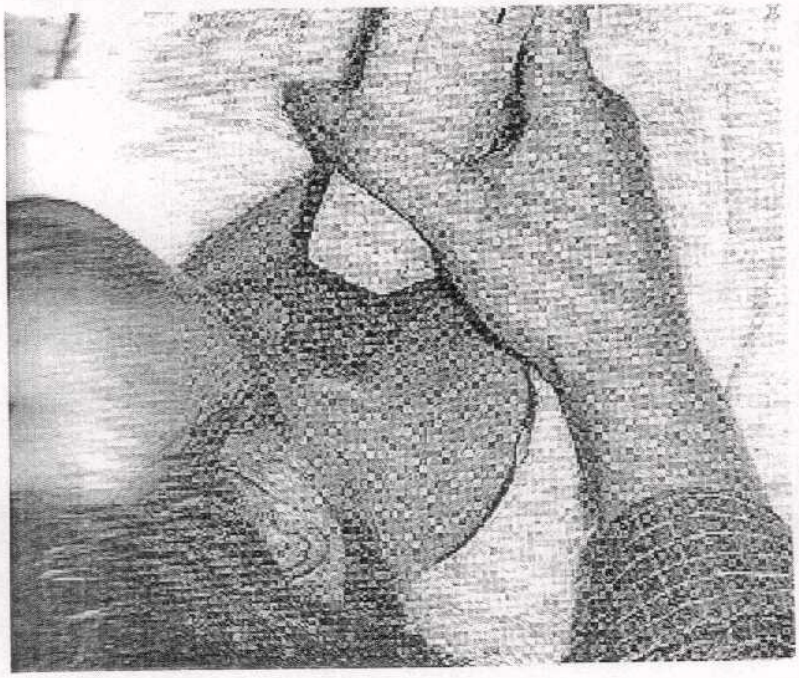


Fig (11): Transducer positioning for longitudinal duplex scanning of carotid arteries in the anterior imaging ***(Quoted from Diagnostic ultrasound, 1998).***

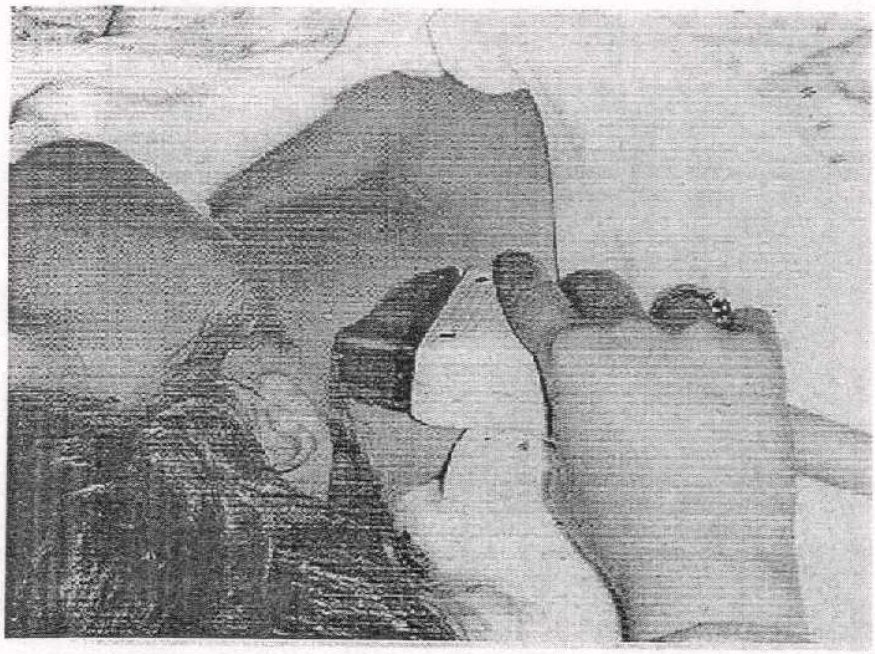


Fig (12): Transducer positioning for longitudinal duplex scanning of carotid arteries in the posterior imaging ***(Quoted from Diagnostic Ultrasound, 1998).***

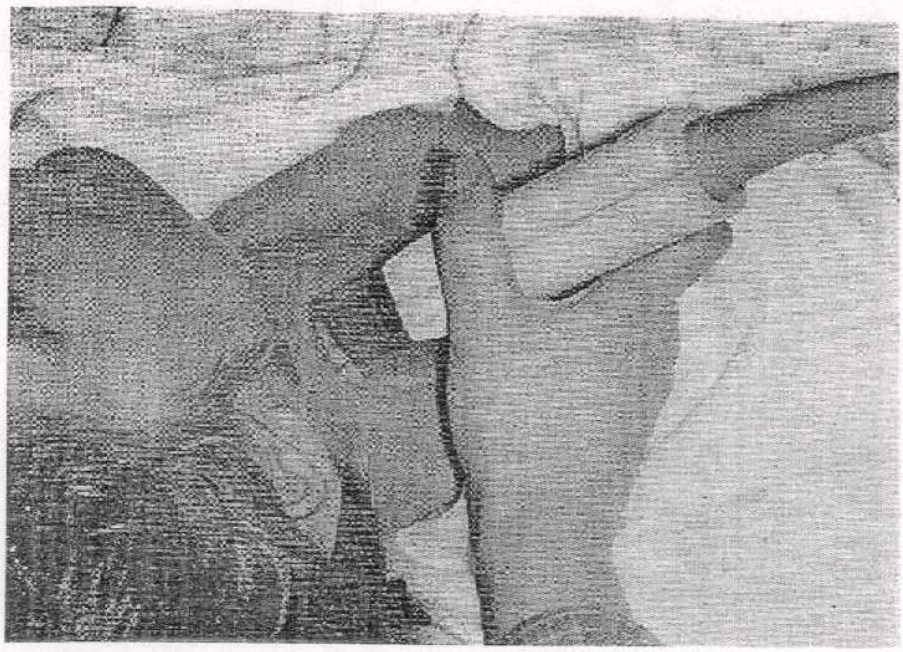


Fig (13): Transducer positioning for longitudinal duplex scanning of carotid arteries in the posterolateral imaging ***(Quoted from Diagnostic Ultrasound, 1998).***

DUPLEX FEATURES OF NORMAL CAROTID ARTERIES

NORMAL B-MODE APPEARANCE OF THE CAROTID ARTERIES:

The normal CCA has a uniform diameter to just below its bifurcation where a variable degree of widening occurs. The widened portion of the CCA is oval in configuration as seen in transverse sections, while the remainder of the CCA is rounded. A variable degree of localized widening also occurs in the proximal 1-2 cm of the internal carotid artery. ***(Cooperberg et al., 1979).***

Carotid wall structure:

The wall of the carotid artery produces parallel echoes corresponding to the adventitial and intimal layers of the arterial wall. The intervening hypoechoic region represents the media. Following endarterectomy, the intimal reflection is missing in the area of intimal removal. The media is often visible beneath plaque deposits. In such cases, the junction of the media with the plaque makes the original luminal boundary. The identification of this boundary assists with the measurement of plaque thickness and stenosis. ***(Zwibel et al., 1992).***

The lumen of the carotid vessels should be echo free; however, artifactual intra-luminal reflections are common, even with the development of the art of instruments. The sonographer, therefore, is faced with the challenge of differentiating between artifactual echoes and real ones e.g. from thrombus or plaque. One

should be highly suspicious cannot be documented from other perspectives (e.g. Longitudinal and transverse planes) ***(Zwiebel et al., 1992).***

Echoes seen only in one plane are probably artifactual. It also helpful to scrutinize the pulsation of the intraluminal echoes. Plaque will usually be seen to pulsate synchronously with the arterial walls whereas artifactual echoes are stationary, except for those generated by the walls of the jugular vein. One may occasionally detect reflections from blood cells in areas of sluggish flow, but this finding is only seen in the presence of significant obstruction ***(Wolverson, et al., 1983).***

Flow as revealed by the color Doppler images:

Laminar flow is readily apparent in the normal common or internal carotid artery, as manifested by gradation of the shades of color from the periphery to the center of the vessels. This manifestation of normal differences in flow velocities between the periphery and the center of the vessel may be seen both in transverse and in longitudinal images. In some cases, the highest velocities may be eccentric within the lumen due to the effects of bifurcations and curves proximal to the point of examination. ***(Merritt., 1986).***

Tortuousity of bifurcation of the vessels may produce flow disturbances that vary in severity in proportion to the curvature or angulation of the vessel. Such disturbance may be manifested by

mixtures of color (red and blue) representing forward and reversed flow. Flow disturbances such as those may occur in the absence of pathology. The most normal flow disturbance occurs at the carotid bulb where a vortex is established in the capacious portions of the common and internal carotid artery. The size of the vortex appears to be related to anatomical factors, including size of the bulb and the degree of angulations between the internal and external carotid arteries.

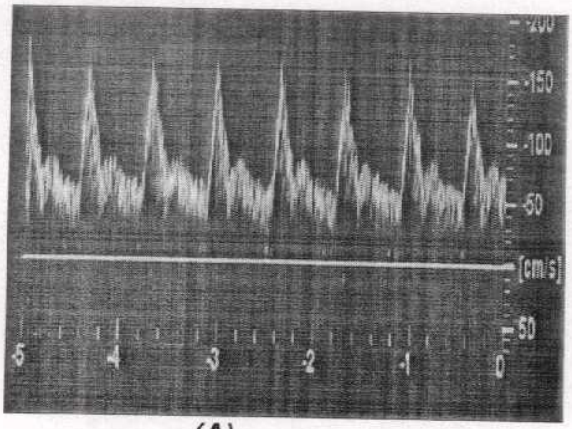
Aspects of carotid pulsatility that assist with identification of the external and internal carotid arteries are also manifested by the color Doppler image:

- Continues flow throughout diastole in the common and internal carotid arteries is indicated by persistence of color through the entire cardiac cycle.
- In contrast, cessation or marked diminution of diastolic flow in the external carotid artery is manifested by the disappearance of color during the diastolic portion of the cardiac cycle.

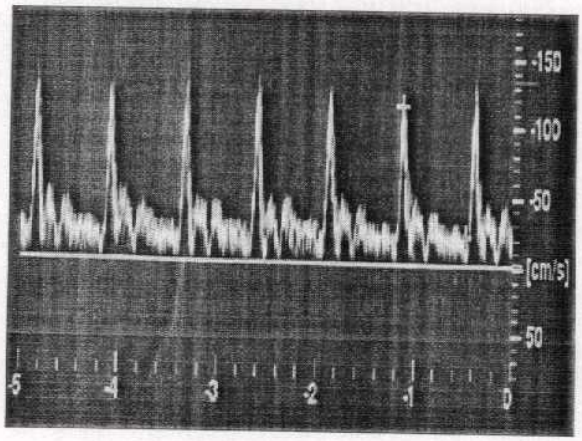
As seen in real time, the color of the common and internal carotid arteries undulates with each cardiac cycle, whereas it flickers on and off in the external carotid **(Zwiebel et al., 2000).**

Normal extra cranial carotid artery blood flow patterns Fig (6):

The sound spectral analysis patterns of the ECA, ICA and CCA should all be examined together as a unit. Approximately 80% of the blood flowing from the CCA goes through the ICA into the brain, whereas 20% goes through the ECA into the facial musculature as shown ***(Ricotta et al., 1984 & Spencer., 1981)***.



(A)



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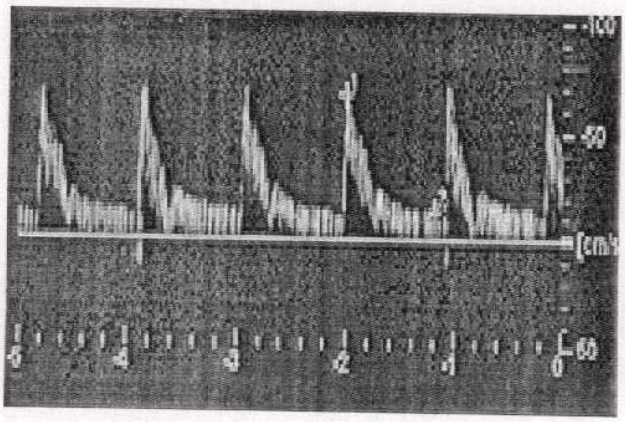

(C)

Fig (14): Normal spectral Doppler features of internal carotid artery (A), External carotid artery (B), Common Carotid artery (C). ***(Quoted from Taylor, 1995).***

The relative decrease in blood flow through the ECA will cause it to have a lower amplitude gray scale wave form throughout all three components of the cardiac cycle than that found in both ICA and CCA. This is noted as low amplitude for the ECA and high amplitude for the ICA and CCA.

The ECA carries blood into the high-resistance circulation of facial musculature; the ICA carries blood into the low-resistance brain circulation. As a result, the ECA has a steep systolic upward slope, a spiked systolic peak and a steep early diastolic downward slope.

Both CCA & ICA which are carrying blood to a lower resistance circulation have a gradual systolic upward slope, a rounded systolic peak, and a gradual early diastolic downward slope, because the ICA is a high flow-low resistance system. It has a higher systolic peak Doppler shift frequency (2.5 KHz) than does the ECA (1.2 KHz), which is a low flow high resistance system.

A comparison of the late diastolic component of the ECA, the CCA and ICA shows some distinct differences:

The ECA late diastolic blood flow is low because of the decreased run off blood into the high resistance vascular bed of the facial musculature. This results in the low late diastolic Doppler shift frequencies, which approach, the zero base line. The CCA & ICA late diastolic blood flow is increased as compared with the ECA late diastolic blood flow into the low resistance vascular bed of the brain.

This result in the elevated late diastolic Doppler shift frequencies above the zero baselines, A low, late diastolic blood flow velocity is recorded in the ECA chart, whereas it is recorded as high in the CCA & ICA. Chart.

Movement of RBCs in a normal direction is present because none of these vessels is occluded. Turbulence is usually found in the flow patterns of abnormal vessels therefore, it is not expected except at specific sites in the normal extracranial carotid arteries ***(Jacobs et al., 1997 & Courbier et al., 1998).***

COLOR CODED DUPPLER IMAGING FINDINGS IN CAROTID ARTERY DISEASES

B-MODE FEATURES OF CAROTID PLAQUES:

The principle arterial pathology detected with duplex sonography is atherosclerosis ***(Davenport et al., 1988).*** Atherosclerotic plaque is represented by echogenic material that encroaches on the arterial lumen and produces a flow-void in the color Doppler image ***(Leahy et al., 1998).*** The following are the important diagnostic features of carotid plaques:

(a) Its extent and thickness.
(b) The resultant degree of luminal narrowing.
(c) Its composition.

Plaque extent and thickness:

The cephalocaudal extent of plaque can be visualized reliably with longitudinal images of carotid arteries. The extent of plaque is communicated descriptively in duplex ultrasound repots; for example, plaque might be described as "focal within the internal carotid", "diffuse" or "extending into the internal carotid for 2 cm" ***(Steinke et al., 2000).***

The thickness of plaque is more difficult to be described sonographically for two reasons:

- Firstly, the plaque may be irregular, in which cases it varies in thickness from one location to another.

- Secondly, the plaque frequently has a crescentic configuration on cross section. Considering that the vessels and the plaque represent a complex three dimentional structure and that a two dimentional image is used to depict this structures it is not surprising that plaque severity may be over or under estimated ***(Fisher et al., 1999).*** The best means for assessing plaque severity is from transverse images. The transverse view depicts most accurately the maximum thickness of the plaque and the resultant degree of luminal narrowing. Longitudinal images are not reliable for evaluating the severity or thickness of plaque or for assessing the degree of luminal narrowing ***(Hallam et al., 1989 & and Langsfeld et al., 1989).***

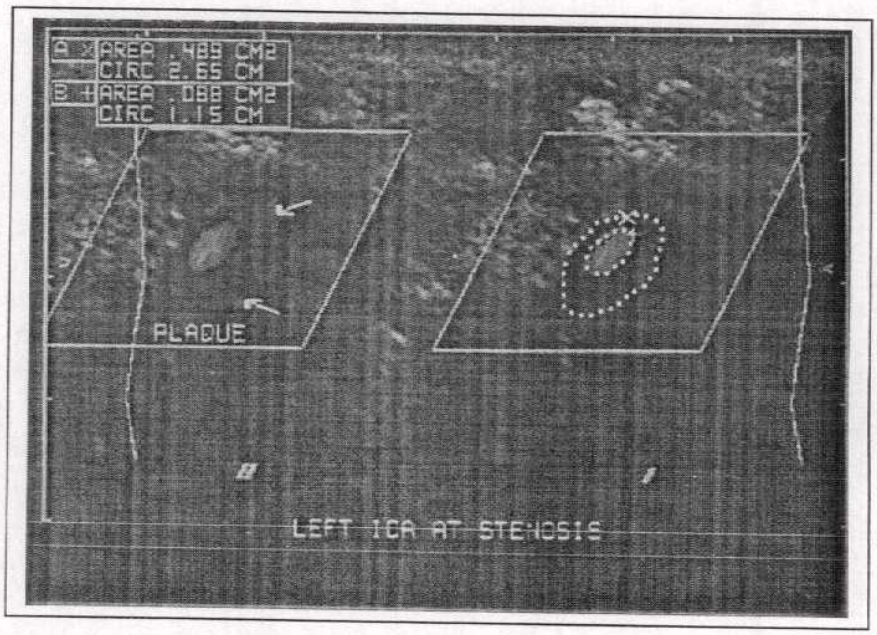

Fig (15): Color Doppler measurement of stenosis transverse images of a internal carotid artery. 74% ***(Quoted from Zwiebel., 2000).***

Plaque composition:

Sonography is unique among vascular imaging procedures because it can be used to assess the internal characteristics of plaques.

Fibrofatty plaque: which contains a large amount of lipid material, is the least echogenic type of plaque and this plaque may be so faintly echogenic as to be difficult to identify sonographically. In general, fibro fatty plaque is less echogenic than the sternomastoid muscle. The echogenicity of plaque increases in proportion to its collagen contents. ***(Fisher et al., 1999).***

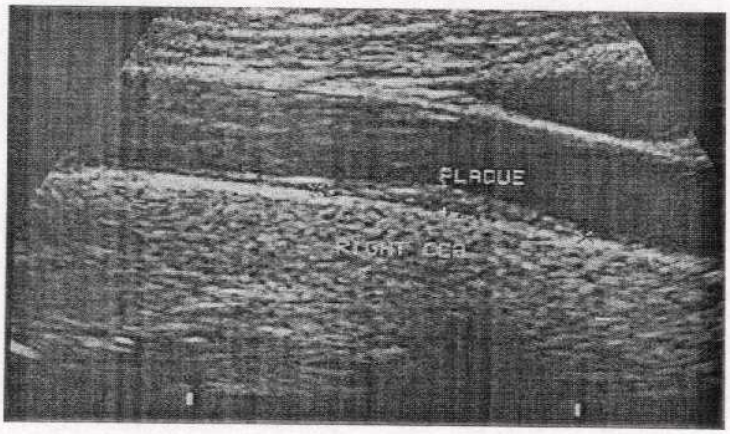

Fig (16): Poorly echogenic consistent with fibro-fatty composition ***(Quoted from Zwiebel., 2000).***

Fibrous plaque: in which collagen is a prominent component, is moderately echogenic. In general terms, this plaque is more echogenic than the sternomastoid muscle but less echogenic than the arterial adventitia. ***(Anderson., 1983).***

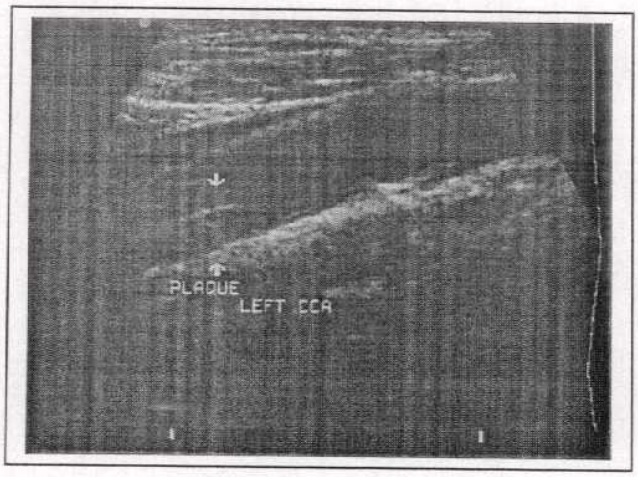

Fig (17): Fibrous plaque ***(Quoted from Zwiebel., 2000).***

Dystrophic calcification: which occurs in plaque at sites of hemorrhage and necrosis, generates strong reflections and distal acoustic shadows. Calcified areas are as bright as or brighter than any object in the ultrasonic image. No correlation has been found between the presence of calcification and neurological symptoms ***(Seeger et al., 1991 and Widder et al., 1994).***

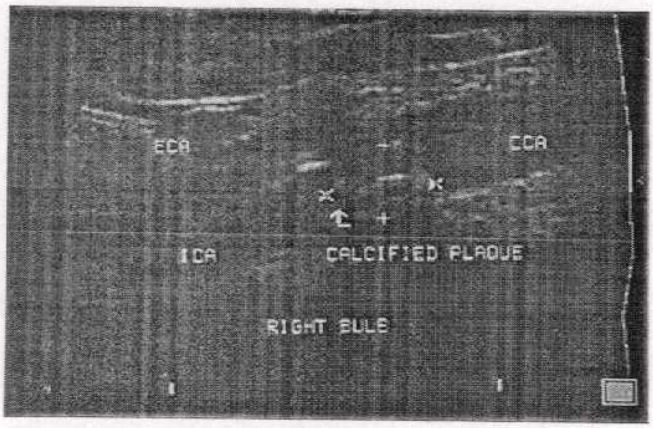

Fig (18): Calcified plaque causes bright reflections accompanied by an acoustic shadow ***(Quoted from Zwiebel., 2000).***

Plaque in homogeneity: is a sonographic feature that has received a great deal of attention, because in homogeneity has been associated with intraplaque hemorrhage ***(Bluth et al., 1988).*** The sonographic detection of intraplaque hemorrhage is sought to be important because such hemorrhage have been related to intimal inflammation, intimal disruption, frank ulceration, and rapid enlargement of plaque ***(Ammar et al., 2001).***

Plaque surface characteristics:

It is well established that embolic occlusion of the intracranial carotid arteries is the primary cause of stroke or TIA the surface characteristics of plaque, therefore, are of considerable interest, because denuded surfaces and ulcers are thought to be common sources of emboli. The ability of duplex sonography to detect surface features is questionable. Only on histologically based series of meaningful size. ***(O'Donell et al., 2001)*** has shown that duplex US was effective for detection of ulcers (100% sensitivity and specificity). Other histologically verified studies ***(O' Leary et al., 1987)*** have shown: either no correlation or poor results for ulcer detection (39-67% sensitivity & 31-84% specificity). On the basis of these studies it appears unlikely that duplex sonography is useful for the assessment of gross surface characteristics of plaque. Color Doppler sonography can demonstrate flow in large plaque craters, but this capability does not eliminate another major limitation of carotid plaque imaging namely that, sonography in any form cannot

determine whether the intimal lining of the vessel is intact, because the intimal layer is not visible per see ***(Zweible., 2000).***

Complications of plaques:

Atherosclerotic plaques are considered clinically significant because they may spontaneously undergo a series of changes, the complications of which lead to TIAs & strokes. It is of considerable diagnostic importance that all of these complications may be observed on the B-mode image of the plaque ***(Reilly et al., 1983).***

The complications include:

1. Intraplaque haemorrhage.
2. Surface ulcerations.
3. Intraluminal thrombus formation.

Significant intraplaque hemorrhage: is most frequently found in association with symptomatic plaque causing focal neurological deficits and is less frequently found in asymptomatic plaque. The hemorrhage results from rupture of the thin walled capillaries that vascularize the endothelium. Smooth muscle, and fibrous connective tissue adjacent to the lipid core. Hemorrhage can dissect and extend into the media muscularis, rupture into the lipid core of the plaque and elevate the fibrous cap of the plaque, causing it:

a) To protrude into the arterial lumen. One cause for the development of a critical stenotic arterial lesion. As the

hemorrhage elevates the fibrous cap, it tears the overlying thin endothelial surface, which soon becomes ulcerated. These ulcers erode down into the fibrous plaque, exposing its surface to the blood stream.

b) The ulcers may also extend down into the hemorrhagic lipid core, which now acts as a nidus for the microemolization of lipid debris, fibrin, and platelets into the blood stream ***(Persson et al., 1985 & Reilly et al., 1983).***

c) Both the non-endothelia lied surface of the fibrous plaque and the exposed hemorrhagic lipid core act to stimulate thrombus formation. This thrombus may be either gray, pink or red. Gray or pink thrombus is initiated by the adherence of fibrin and platelets to the plaque surface. It is microscopically characterized by the lines of Zhan, representing alternating bands of platelets and fibrin with relatively few trapped red blood cells. This type of thrombus cover plaque surfaces in arteries when there is rapid blood flow. Red clot forms in arteries with either sluggish or absent blood flow for example, it may be seen distal or proximal to a tight stenosis or in a totally occluded artery. It is microscopically characterized by absence of lines of Zhan and contain all of the blood elements with numerous trapped red blood cells. The complicated ulcerated plaque can be covered with both gray or pink thrombus and red clot. The proliferation of gray or pink thrombus can cause further stenosis of the artery until it

becomes totally occluded. It then becomes completely filled with fresh red clot. Within the period of weeks, the fresh clot becomes a densely organized thrombus within the vessel lumen ***(Imparato et al., 1983 & Gooding et al., 1982).*** The transition period between a fresh red clot and an organized thrombus explains why some totally occluded arteries appear anechoic, where as others show dense areas of echoes on the B-mode image.

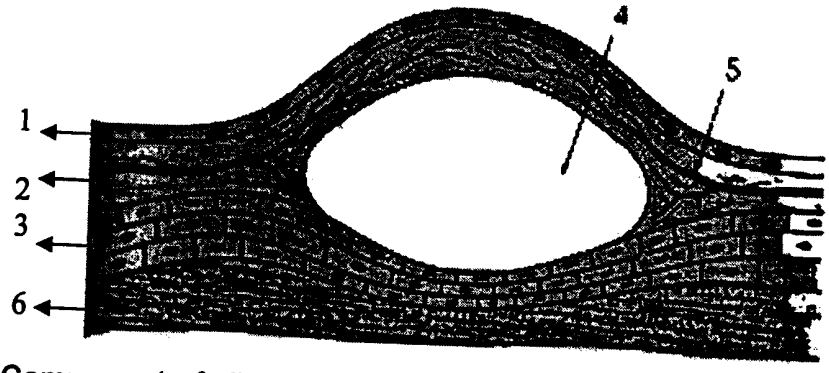

Fig (19): Component of clinical significant atherosclerotic plaque.1-endothelium 2- fibrous cap .3- Media muscularis. 4-Lipid core and cholesterol clefts. 5- Thin walled capllaries. 6- Adventitia. ***(Quoted Harrison., 2002).***

Recently clotted blood has little inherent echogenecity, and even a totally occluded artery can appear surprisingly normal on the B-mode image. This may be explained by the fact that a fresh clot is composed of blood elements, mainly red blood cells, and lacks an organized matrix of fibrous connective tissue (collagen). A careful adjustment of the gain settings to enhance low-amplitude signals, may be helpful in demonstrating these clots. If this fails, other

parameters that can be used to diagnose an occluded artery with fresh blood clot include (1) Pulsed Doppler sonography to detect absent blood flow, and (2) real time imaging to detect absent arterial pulsations and decrease in the diameter of the occluded artery as compared with the contralateral patent artery of the same size. Internal echoes begin to appear as the clot becomes an organized thrombus. Areas of liquefaction often appear as anechoic, cystic spaces, scattered throughout the echogenic thrombus. Old thrombosed arteries incite an inflammatory like reaction in the surrounding soft tissues. This causes intralumenal echoes from the organized thrombus of the occluded internal carotid artery (ICA) to blend in with the surrounding adjacent soft tissue, inflammatory like reaction ***(Wing et al., 1995).*** The result is a loss in the normal acoustical interface between the occluded artery and the surrounding soft tissues. A B-mode image of the chronically occluded artery cannot be obtained under these circumstances. The inability to visualize the ICA with B-mode is a clue for its occlusion. (D) Calcification is another complication that may occur in the intimal plaque. Almost always, atheromas in advanced disease undergo patchy or massive calcification. Hemorrhage beneath a calcified plaque may cause it to protrude into the lumen, resulting in an arterial stenosis. This is similar to the stenosis produced when hemorrhage occurs beneath a dense fibrous cap. ***(Eastcott et al., 1984).***

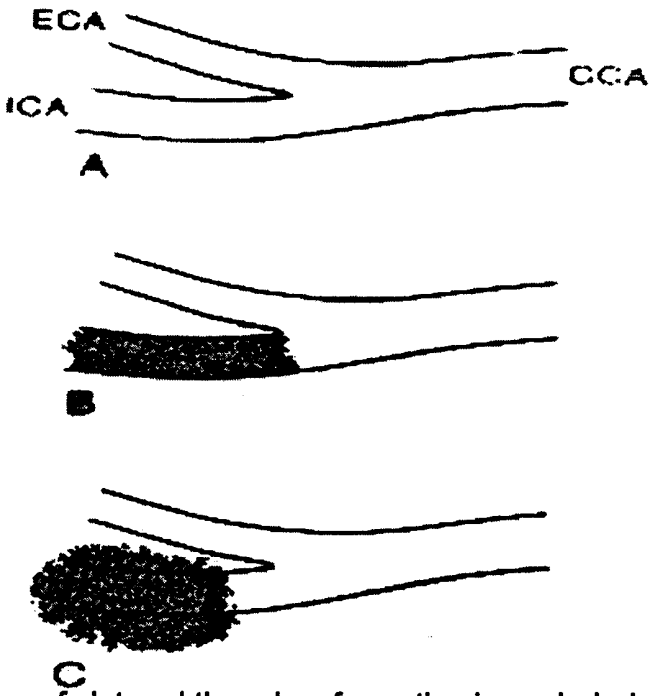

Fig (20): Pattern of clot and thrombus formation in occluded arteries.
A- Non echogenic fresh clot
B- Echogenic thrombus with cystic spaces
C- Echogenic thrombus in occluded artery surrounded by inflammatory reaction. ***(Quoted from Atlas of color Doppler., 1996).***

These characteristics are important because the morphology of the plaque seems to play an important role in the development of cerebral insufficiency symptoms and in the prediction of stroke risk ***(Imparato et al.,1983).*** U.S.scanning can assess plaque morphology and distinguish between heterogeneous & homogenous plaques. With an accuracy of 82% this is important because heterogeneous plaques are associated with a statistically greater incidence of pathological plaque hemorrhage and ulceration that correlate with a greater risk of TIA or stroke. Identification of plaque histology is valid in Identify clinically significant lesions and may ultimately aid in the choice of therapy ***(Persson et al., 1985).***

DOPPLER AND COLOR DOPPLER CRITERIA OF CAROTID ARTERY PLAQUE

Typical flow abnormalities that occur at various locations in the vicinity of an arterial stenosis:

Proximal to the stenosis, laminar flow pattern will be detected assuming that this part of the vessel is normal. Within the stenotic zone, velocities are increased, but flow generally remains laminar, some spectral broadening may occur in the stenotic zone when: a wide range of velocities exists across the lumen, the lumen is irregular or the Doppler sample volume is too large or positioned too near a vessel wall ***(Carroll., 1991).***

Spectral broadening in the stenotic zone, when the Doppler sample volume includes a portion of the post stenotic disturbed flow. At post stenotic lumen, the flow stream spreads out rapidly producing disturbed flow pattern proportionate in severity to the degree of stenosis (flow disturbance = spectral broadening). ***(Zwiebel, et al., 1987)*** Maximum flow disturbance occurs within 1 cm beyond the stenosis ***(Garth et al., 1999).***

In case of severe stenosis, the disturbed flow pattern, may cause the arterial wall to vibrate. Producing: high amplitude (strong), low frequency signals in Doppler spectrum, loss of definition of the upper border of the spectrum and simultaneous bidirectional flow. ***(Zwiebel et al., 1987).***

About one to 2 cm beyond the stenosis, the severity of flow disturbance subsides, laminar flow usually is re-established within 3 cm beyond the stenosis ***(Kassam et al., 1985).***

Post stenotic flow disturbance is represented by spectral broadening which has two values it may be the only indication that a major stenosis is present e.g. when the Doppler signal in the stenotic zone are blocked by calcification and the severity of post-stenotic disturbed flow is a rough indicator of the severity of carotid stenosis. ***(Black shear., 1980).***

The spectral broadening due to disturbed flow is first noticed along the down slope of systole and throughout the diastole. As the stenosis becomes severe (more than 60%) filling in of entire spectral window takes place. ***(Carroll., 1991).***

GRADING of CAROTID STENOSIS

Several of the classification or grading schemes employed for carotid stenosis

Table 2. Bluth's Carotid Stenosis Classification Scheme

Percentage of Stenosis	PSV (cm/s)	EDV (cm/s)	PSV ICA/CCA Ratio	EDV ICA/CCA Ratio
Normal	<110	<40	<1.8	<2.4
1-39	<110	<40	<1.8	<2.4
40-59	<130	<40	<1.8	<2.4
60-79	>130	>40	>1.8	>2.4
80-99	>250	>100	>3.7	>5.5

(Bluth's., 1988).

Table 3. Zwiebel's Carotid Stenosis Classification Scheme

Percentage of Stenosis	Peak Systolic Velocity (cm/s)	End Diastolic Velocity (cm/s)
0-39	<110	<40
40-59	<130	<40
60-79	>130	>40
80-99	>250	>100

ICA, CCA ratio of 3.2 to 3.5. These authors also reported that prior studies list criteria for 70% as a PSV of 325 cm / second and a PSV ICA / CCA ratio of 4. *(Zwiebel's., 1992).*

Table 4. Strandness's Carotid Stenosis Classification Scheme.

Percentage of Stenosis	Criteria
1-15	No flow separation in bulb
16-49	Spectral broadening
50-79	PSV> 125 cm/s, EDV <140 cm/s
80-99	EDV >140 cm/s

(Strandness's., 1990).

Table 5. Carroll's Carotid Stenosis Classification Scheme

Percentage of Stenosis	PSV ICA/Cca Ratio	EDV ICA/Cca Ratio	PSV (cm/s)	EDV (cm/s)
0-40	<1.5	<2.6	>25,<110	<40
41-59	<1.8	<2.6	>120	<40
60-79	>1.8	>2.6	>130	>40
80-99	>3.7	>5.5	>250	>80-135

(Carroll's., 1989).

Table 6. Proposed New Categories and Criteria for Classification of Carotid Stenosis

Percentage of Stenosis	PSV (cm/s)	EDV (cm/s)	PSV ICA/CCA Ratio	EDV ICA/CCA Ratio
<15	<110 and	*		
16-39	<110 and	<40	–	–
40-59	<130 and	<70 #	–	–
Near 60	130-170 and	<70 #		
60-79	>170 or	>40,70-110$	>2.0	>2.4
70-99	>250 or	>110	–	–
80-99	–	>140 or	>3.7	>5.5
90-99	<110 #	–	–	–

PSV, peak systolic velocity; EDV, end diastolic velocity.
*No spectral broadening and visual estimate < 15% stenosis.
+ PSV 110-130 and / or EDV 40-70.
# Indeterminate as to whether actually 40%-59%or 60%-79%.

(Withers., 1990).

Role of Color Doppler Imaging:

Although duplex scanning of the carotid artery by combining B-mode ultrasound and Doppler ultrasound in one instrument overcomes many of the individual limitations of each, it has some limitation due to technique as inability to image vessels other beyond than those in the neck, calcified plaque may obscure an area of stenosis from proper interrogation, the high velocity jet may be missed in severe stenosis and the external carotid artery may be mistaken for internal carotid artery in cases of ICA occlusion of collateralization develop. ***(Hallam. et al., 1989).***

A quick transverse and longitudinal color Doppler U/S survey of the carotid artery pin points areas of abnormal flow which appear

as heterogeneous color patterns ***(Mosaic colors),*** lumenal narrowing or both. Identifications of abnormal areas of color allows the operator to place the pulsed Doppler sample volume in an area of color Doppler abnormality, thus streamlining the time consuming process of pulsed Doppler spectral analysis along the entire course of a vessel. Although qualitative estimations of flow abnormality can be made by observing a heterogeneous, narrowed, color pattern, spectral analysis remains essential; the color patterns reflect only mean velocity values, whereas peak velocity value (be Doppler analysis) are necessary for accurate determination of degrees of stenosis ***(Carroll., 1991).***

Early diastolic color flow reversal proximal to the orifice of an occluded internal carotid artery, also color Doppler U/S with low velocity settings revealed no evidence of color Doppler flow in the occluded vessel. These findings, combined with color Doppler U/S features of damping in the CCA (the appearance of color flow in systole followed by a conspicuous decrease or absence of color in diastole) are highly suggestive of occlusion. These color Doppler findings correspond to the Doppler spectral wave form abnormalities of damping associated with decreased or absent diastolic flow. ***(Polak et al., 1990).***

Color Doppler has some limitations as: It may be limited by the framing rate of the imaging device (15-25 frames/sec), it may aliase at lower frequency shifts than a pulse Doppler map and the mean velocity rather than the peak velocity is displayed. ***(Erikson et al., 1989).***

PITFALL AND ARTIFACTS

Pitfalls and artifacts are separated in three categories:

1- Anatomic pitfalls.
2- Physiologic pitfalls.
3- Technical error and artifacts

1- Anatomic pitfalls

a) Distinguishing the internal from the external carotid artery distinguishing feature are lasted in table one about 95% ICA is posterior and lateral two ECA. ***(Polak., 1993).***

b) Tortuous carotid elongated vessels are commonly found in chronically hypertensive and in elderly patient. At time the arteries may be difficult to interrogate with Doppler because of their orientation and tortuous. In such cases, artifactually high velocity may be suggested by misapplying the angel correction cursor and by approaching the vessel from a non standard angel of insinuation. In such difficult cases, qualifying the certainty of result is advisable, especially when velocity value is in border line ranges. ***(Polak., 1993).***

c) High bifurcation in some patients, particularly those with relatively short necks. The carotid bifurcation may not be satisfactory seen because of their high position. Depending in the degree of difficulty of the examination, this situation may be one of the uncommon causes of a non diagnostic study. ***(O'leary et al., 1987).***

d) Congenital absence of the common carotid artery is rare in such cases the ICA and ECA arises directly from the innominate artery or the aortic arch

e) Postoperative changes finding in patient with prior carotid surgery or other intervention can vary. Spectral broadening often persist after carotid endarterecotomy in the absence of significant residual or recurrent disease ***(Russell., 1998).*** Postoperative spectral broadening is often striking in vein patch graft carotid surgery. Spectral analysis may demonstrate discordenatly high or low velocity in the area of the vein patch graft suggesting markedly disturbed flow in this area of disturbed flow can result in erroneous velocity determination. Relatively normal flow is demonstrated in the CCA proximal to and in the ICA distal to the graft. ***(Carroll., 1991)***.

2- **Physiologic pitfalls.**

a) Low diastolic flow in the internal or common carotid artery:

- Distal occlusion or high grade stenosis often because a high resistance flow pattern with little or no diastolic flow (high resistivity index).This may be associated with low velocities throughout the ICA. This pattern is similar to the normal flow pattern of the ECA, this appearance may be termed **externalization of flow**. This pattern is most seen in the CCA, proximal to a high grade stenosis or complete occlusion. When seen in proximal ICA, externalization may signify one of the following conditions.

- Low cardiac output.
- Aortic valvular insufficiency.
- Distal ICA stenosis.
- Distal small vessels disease. ***(Lincoln et al., 1998).***

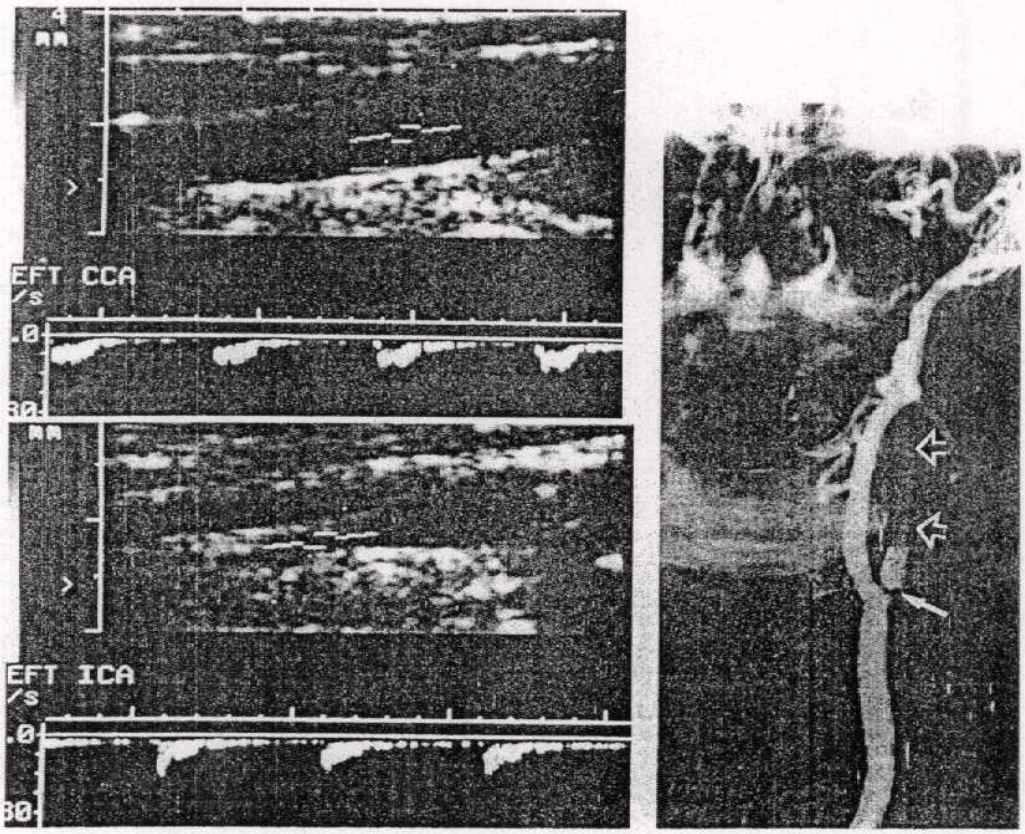

Fig. (21): High-grade stenosis producing decreased peak velocity with elevated resistance. (a) CCA demonstrates decreased diastolic flow (increased resistivity index), also known as externalization of flow. (B) ICA shows similar externalization pattern in narrowed vessel. (C) Angiogram demonstrates critical stenosis at the origin of the ICA (arrow). ***(Quoted from, Diagnostic ultrasound, 1998)***.

b) High diastolic flow in the internal or common carotid artery:

High–grade stenosis proximal to the area of insonation may cause damped flow in the CCA or ICA recognized by low–amplitude waves with round peak and little difference between the peak systolic and end diastolic flow velocities. ***(Robinson., 1992).***

c) High diastolic flow in the external carotid artery:

Just as the diastolic flow velocity may be suppressed in the CCA and ICA may be unusually elevated in the ECA. Complex intracarinal–extracrinal anastomoses are second only to circle of Willis in importance in supplying collateral circulation. occlusion of the ICA produces collateral circulation to the carotid siphon through the ECA and ophthalmic arteries .if this decreased systolic–diastolic difference is not caused by more proximal stenosis, this may be represent the situation termed **internalization** of the flow .in the presence of the ICA occlusion, particularly chronic and ECA collateral flow to the internal circulation ,the ICA may note be seen and the primary collateralized trunk of the ECA is dilated with an ICA –like waveform. In such case, the ECA may mistaken for the ICA, and first ECA branch may be mistaken for main ECA. This may lead to missing complete or near complete occlusion of ICA. ***(Lincoln et al., 1998).***

High velocity flow without apparent vascular cause

The flow velocity appear elevated without vascular disease in the following condition.

- hypertension
- systemic arterio-venous fistula
- Intra cardiac shunt
- Hyperpyrexia
- Other causes of increased cardiac output. ***(Lincoln et al, 1998).***

CONTRALATERAL STENOSIS

High–grade stenosis or occlusion in one carotid artery can significantly affected velocities in the contralateral vessels. ***(Beckett., 1990)***. Severe ICA stenosis or occlusion can produce collateral shunting of increased blood flow through the contralateral carotid artery. This increased flow may artifactually increase velocity measurements in the contralateral vessels, particularly in areas of stenosis. Similarly, a tandem high- grade intracrinal narrowing may reduce anticipated velocity abnormalities. Therefore, it is important to continue Doppler interrogation as far cephalad as possible to avoid missing distal tandem lesion. ***(Zwiebel., 1992).***

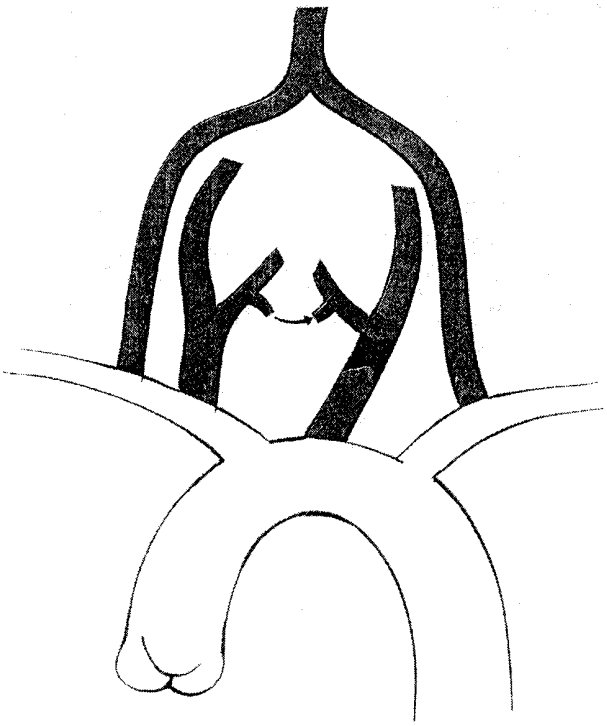

Fig. (22): Diagram of potential pattern of collateral flow in a patient with an occluded CCA, but patent ipsilateral ICA and ECAs. ***(Quoted from Diagnostic ultrasound, 1998).***

Internal carotid artery occlusion versus minimal residual flow.

In case of an apparent internal carotid occlusion, additional efforts are required to distinguish between complete or near complete occlusion. This important because patient with high –grade stenosis are candidates for surgical endarterectomy or carotid stint placement, while patient with complete occlusion are note depending on quality of the Doppler unit and technique uses, duplex

ultrasound may note be able to detect all cases of minimal residual flow. ***(Bridges., 2001).*** The introduction of color Doppler and more sensitive flow detection capabilities in Doppler systems has facilitate more accurate diagnosis of occlusion by allowing easier identification of residual string of blood flow in preocclusive stenosis. ***(Berman et al.,1995).***

3- Technical Errors and artifact

a) *External carotid artery*

Generally, only the origin of the ECA is evaluated ultrasonographically because plaque there is rarely clinically significant. Stenosis of the external carotid artery should be noted however, because it may account for a worrisome cervical bruit when the ICA is normal. ***(Holman., 1990)***

Assisning Angle Theta

Selecting an inaccurate Doppler angle correction is probably the most common error encountered in carotid ultrasonography. Angles greater than 60 degrees or less than 30 degrees are associated with artifactual elevation of calculated flow velocity. Another possible technical error results from using the inappropriate positioning of the angle cursor parallel to the vessel walls, rather than to the direction of the flow jet in stenosis, in examples of high-grade stenosis, the jet is commonly directed obliquely by the

irregularity of the plaque, if the cursor is not placed parallel to the direction of the flow jet, error may also be introduced. ***(Jacobs et al., 1997).***

VERTEBRAL DUPLEX TECHNIQUE

Duplex examination of the vertebral arteries is performed in conjunction with the carotid examination. The patient is positioned in the same manner as carotid study and the following steps are used to examine the vertebral arteries.

Step 1: To find the vertebral artery, begin with an image of the common carotid artery and then shift the image plane laterally until shadows are identified that represent the transverse process of the cervical vertebra. Next adjust the plane until one or more segment of the vertebral artery is seen between the transverse processes.

Step 2: Note the direction of flow in the vertebral artery and confirm that flow is cephalad by comparing the vertebral and carotid flow direction.

Step 3: survey the entire visible length of the vertebral artery. Begin this survey at the point where the artery was first identified and move inferiorly to visualize the vertebral artery origin below the C6 level. Next follow the vertebral artery cephalad as far as possible tracing its course from one transverse foramina to the next.

Step 4: Measure the diameter of a normal appearing segment of the vertebral artery and note the waveform shape and peak systolic velocity in that segment.

Step 5: Scrutinize any area with spectral Doppler in which color Doppler has revealed abnormal flow (high – velocity jet or flow disturbance). ***(Zwiebel., 2000).***

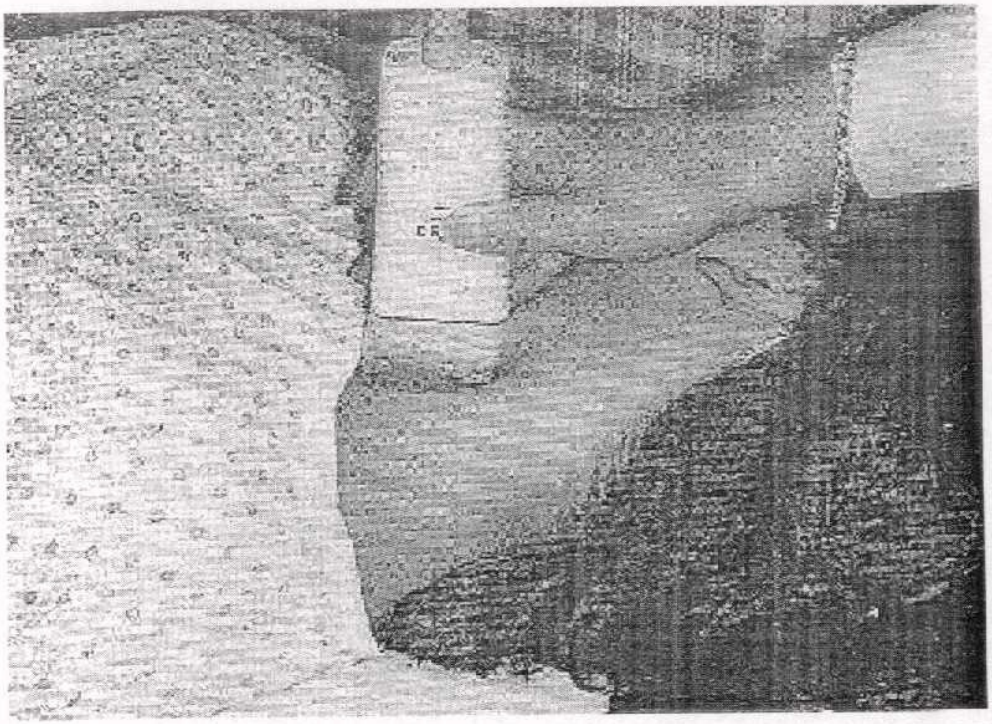

Fig (23): Transducer placement for Duplex scan-Doppler spectral evaluation of the vertebral artery ***(Quoted from Diagnostic Ultrasound, 1998).***

Duplex Feature of Normal Vertebral Arteries

Most normal vertebral arteries are easily seen between the transverse processes. The color Doppler image demonstrates cephalad flow throughout the cardiac cycle and uniform flow pattern. The mean vertebral artery diameter is 4mm ***(Touboul, 2001)***, but vertebral artery size is variable and the vertebral arteries are asymmetric in 73% of normal individuals ***(Meyer, 1995),*** because the size of vertebral arteries is variable. No diagnostic information is conveyed directly by vertebral caliber, except when the arteries are very small or unusually larger. When the vertebral arteries are asymmetric the left is larger in about 80% of cases ***(Ackerstaff, 1998).*** The origin of normal vertebral artery may be difficult to demonstrate clearly because the proximal portion of the vessel often tortuous. Further more the origin may lie quite low, beneath the clavicle. The success rate for standard duplex (non color) visualization of vertebral artery origin is 82-90% on the right side and 50-63% on the left side. Color Doppler may prove to be more effective than standard duplex for identifying the vertebral artery origin. ***(Visona, 1986).***

Vertebral Doppler signal exhibit low resistance characteristics identical to those seen in the internal carotid artery. Large volume of cephalad flow is present throughout diastole and the systolic wave form is relatively broad. The

waveform pattern generally is symmetric in vertebral arteries, but systolic and diastolic velocities may differ in normal individuals if the arteries are symmetric in size. Normal peak systolic velocities range from 20-40 cm/sec. and systolic velocities lower than 10 cm /sec are considered pathologic ***(Bendick., 1990).*** Miner flow disturbances may be present in the late systole and throughout diastole in normal vertebral arteries regardless of the site of examination. ***(Jak., 1998).***

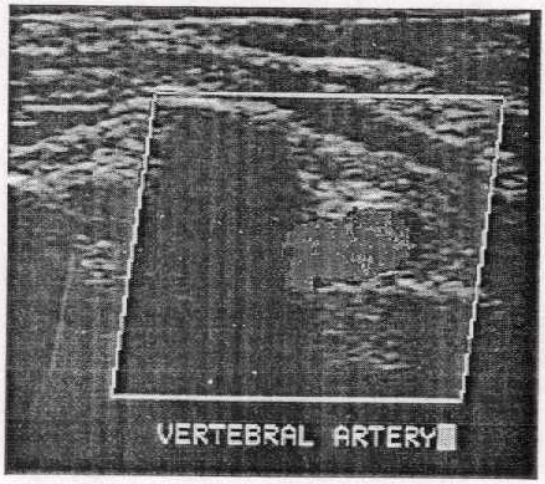

Fig (24): Normal vertebral artery an arrow marks a transverse process ***(Quoted from Zwiebel., 2000).***

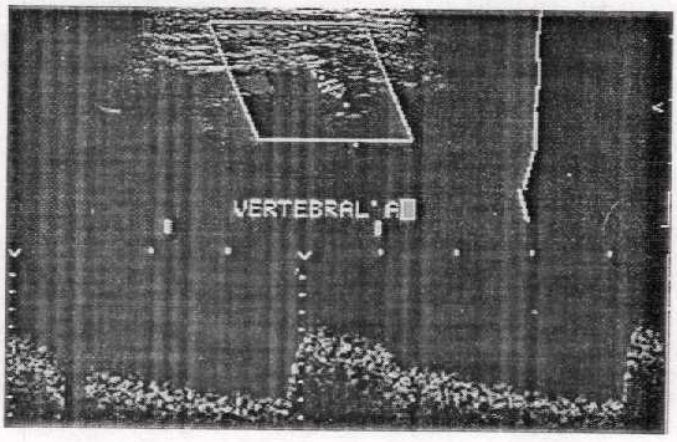

Fig (25): Doppler spectral wave form a normal vertebral artery ***(Quoted from Taylor., 1995).***

DUPLEX FEATURE OF ABNORMAL VERTEBRAL ARTERIES

Duplex finding in abnormal vertebral arteries may be categorized as follow.

- Non visualization.
- Small size.
- Absence flow.
- Increase flow velocity.
- Decreased velocities.
- Abnormal flow direction.

1- Non visualization

Failure to identify a vertebral artery indicates that vessels are occluded. Care must be taken. However to avoid a false-positive diagnosis of occlusion related to poor visualization. If image quality is poor because of large patient size or other factors, failure to visualize a vertebral artery should be reported in caution term. ***(Bornstein., 1998).***

2- Small Size

As noted above, vertebral artery size is variable, and large side–to-side differences may occur in normal individuals. However a vertebral smaller than 2mm in diameter suggests abnormalities. The origin of the small size vessels should be examined for flow disturbances indicative of stenosis, and vertebral waveform should be carefully compared with waveform of opposite vertebral artery. ***(Zwiebel., 2000).***

3- Absent flow

Absence of flow in successfully imaged vertebral artery indicates occlusion of the vessel false–positive has been however, in highly stenosed vessels with only trickle of flow ***(Bendick., 1990).***

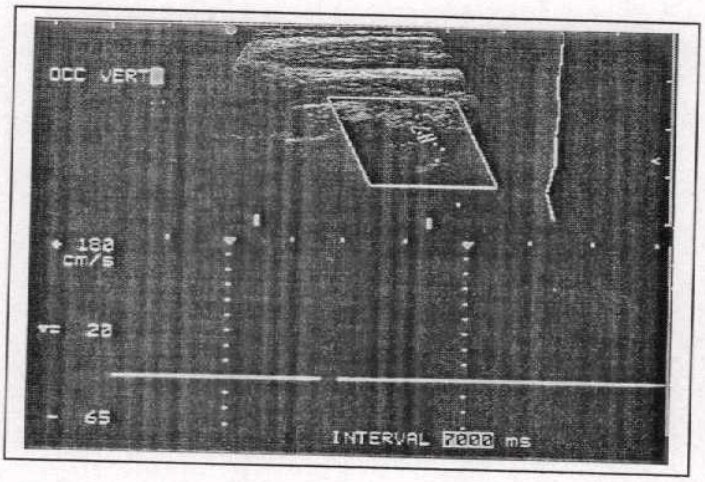

Fig (26): Absent vertebral artery flow the vertebral artery is seen between the transverse processes but no flow is evident on this color Doppler image ***(Quoted from Zwiebel., 1992).***

4- Increased velocity

A focal velocity increase (exceeding 40 cm/sec, peak systole) accompanied by disturbed flow in the adjacent (downstream) portion of the vessel suggests vertebral stenosis. With diameter reduction of at least 50%. The relationship between the severity of the stenosis and the magnitude of the velocity increase has been defined. It is important to note that the velocity increase must be focal and should be associated with poststenotic disturbed flow. ***(Ackerstaff., 1998).***

When stenosis is located in one of the transverse process canals. The high velocity may be inaccessible to duplex detection. Disturbed flow should be apparent. However immediately distal to the transverse process. ***(Kotval et al., 1990).***

5- Decreased velocity

With sever stenosis at the vertebral artery origin. Velocity may be reduced through the remainder of the vertebral artery and the velocity waveform may have a damped appearance. The finding suggests reduced perfusion pressure and volume flow distal to the stenosis. Reduced vertebral artery velocity may also be seen with distal obstruction of the vertebral and basilar artery or of their tributaries in the brain in such cases. The systolic peak remains sharp as in the normal vertebral waveform. ***(Strandness., 1990).***

6- Decreased volume flow

That total blood flow in both vertebral arteries normally exceeds 200ml /min. Total volume less than this suggests that vertebro-basilar insufficiency may because of non localizing neurological symptoms. Total volume although may be reduced by cardiac dysfunction. ***(Benedict., 1990)***.

7- Abnormal flow direction

Normal vertebral artery flow is cephalad in direction through out the cardiac cycle. And any deviation from this

pattern is abnormal. The vertebral to subclavian steal may cause vertebral flow reversal or TO and fro flow pattern. The vertebral to subclavian steal syndrome is an example of the use of collateral pathway to compensate the effect of proximal subclavian artery stenosis or occlusion ***(Erickson., 1989).*** The ischaemic limb steals blood flow from basilar circulation through retrograde vertebral artery flow to supply the arm. Subclavian stenosis and subclavian steel have left side predilection about 85% involved the left subclavian artery and only 15% of the right artery ***(Bornstein., 1988).*** It has been found that the subclavian steal is almost harmless homodynamic phenomenon. Three duplex finding may be seen in subclavian steel syndrome.

a) Vertebral flow reversed throughout cardiac cycle.

b) Flow is bidirectional (forward in systole and reversed in diastole).

c) Flow normal with patient at rest. The steal may be intensified by inducing hyperemia with blood pressure cuff placed at the brachial level. If subclavian steal present, the vertebral waveform quickly inverts or become biphasic after pressure is released.

(Bornstien.,1988).

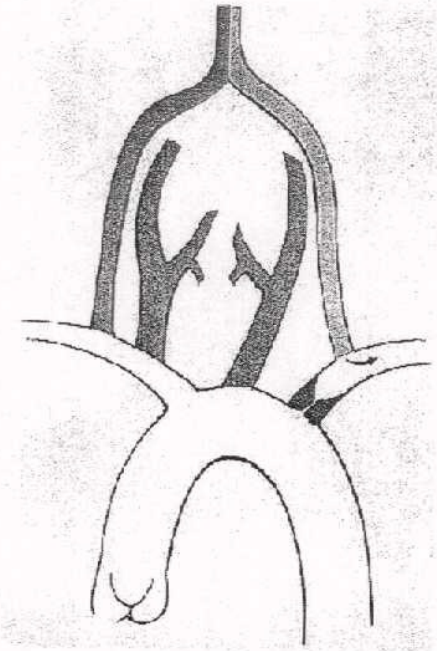

Fig (27): Diagram of location of atherosclerotic lesion in a patient with subclavian steal syndrome ***(Quoted from Diagnostic Ultrasound, 1998).***

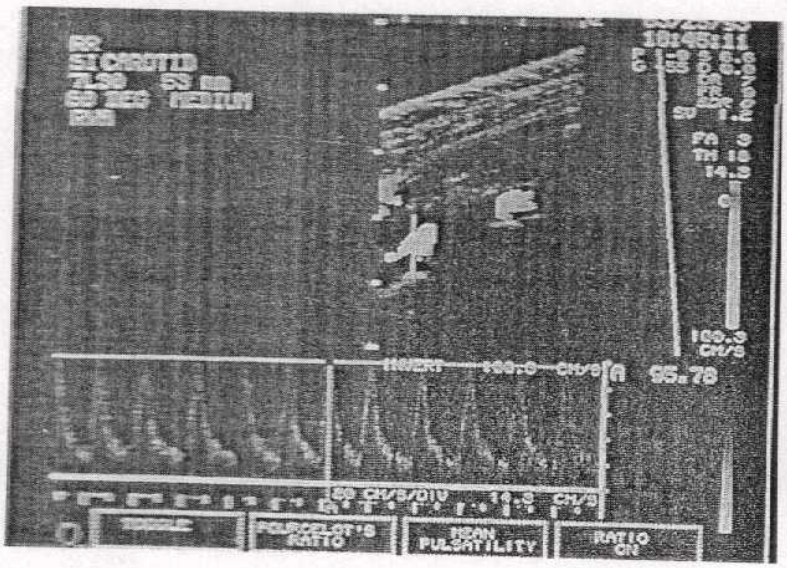

Fig (28): Vertebral arteries in left subclavian steal. A) Antegrade Doppler flow signal in Rt vertebral artery. ***(Quoted from Diagnostic Ultrasound, 1998).***

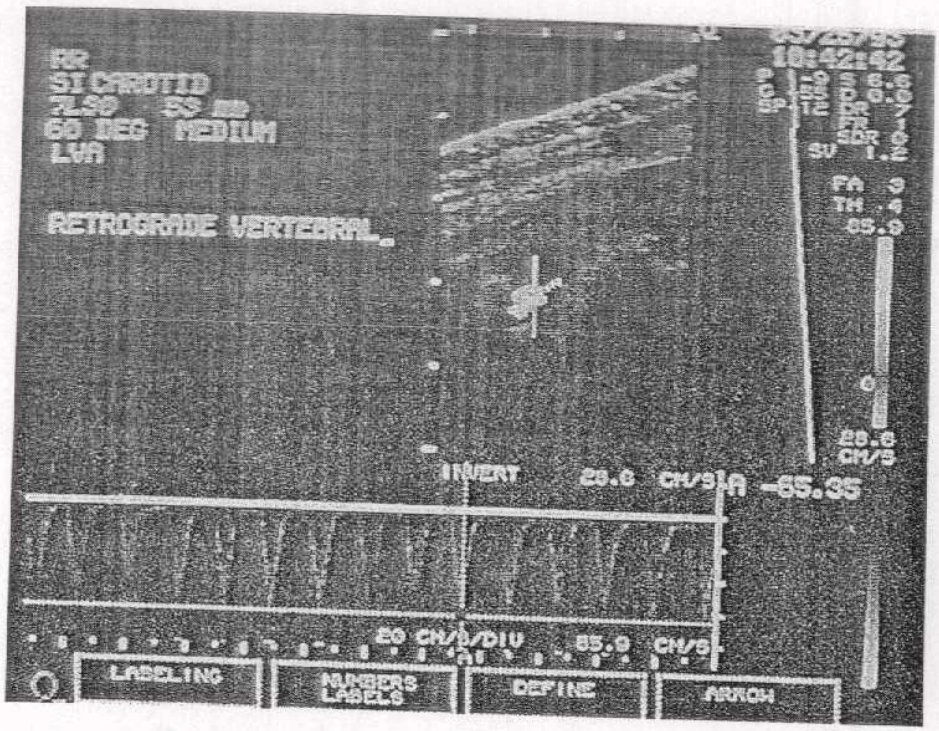

Fig (29): Vertebral arteries in left subclavian steal. B) Fully retrograde Doppler flow signal in the left vertebral artery. Note that the flow is below baseline with negative velocity reading. ***(Quoted from Diagnostic Ultrasound, 1998).***

OTHER DIAGNOSTIC MODALITIES FOR IMAGING EXTRA CRANIAL CAROTID ARTERIES.

1- Direct Angiography:

A- *Carotid angiography:*

It was originally performed by pecutaneous puncture of the common carotid artery in the neck in most centers needles of 18 S.W.G. were used. Carotid angiography is now generally practiced by introduction of catheter into lumen of the common carotid artery from pecutaneous transfemoral approach. The latter technique permits selective catheterization of the internal and external carotid arteries. About 10 ml of contrast medium are usually injected within 1-2 sec into CCA. A slight smaller quantity about 8 ml is adequate for selective injection of the IC and ECA. In case of ECA injection can be made more slowly taking 2-4 sec. As with other form of arteriography, it is important to obtain rapid serial film as contrast medium pass through circulation. Many types of apparatus were available for achieving this but today most imaging is performed by DSA. A standard series could consist of seven images taken at 1per sec. For 7 sec and timed as that arterial, capillary, and venous phase of the angiogram are all covered. ***(Rothwel PM et al., 1995).***

2- Digital Subtraction Angiography

Digital technique is new widely used for vascular studies. It is enable rapid manipulation of multiple images with subtraction of

bones and other soft tissues from opacified blood vessels. Adequate arterial studies may be obtained with low intra arterial concentration of contrast medium and thus can be obtained following simple intravenous injection. Advantage of intravenous technique. Performed without anesthesia, less discomfort to patient, not requires hospitalization and very safe. Disadvantages Resolution is not as good as direct arteriography. Intra-arterial digital subtraction angiography has several advantages. Selective study is possible with smaller dose of contrast medium. Good arterial study using only one third of amount required for conventional studies. The only apparent disadvantage compared with conventional studies is inability to resolve tiny blood vessels. ***(Vanniner et al., 1994).***

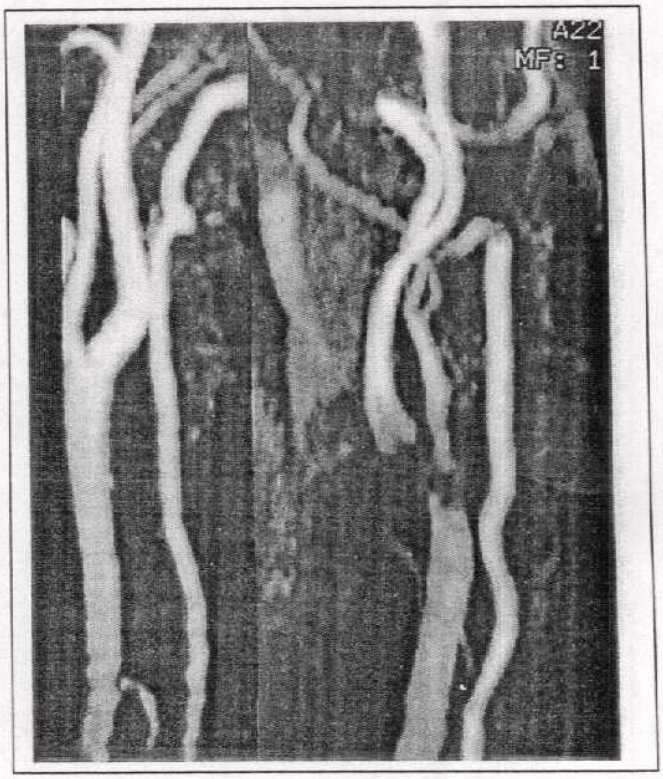

Fig (30): Digital Subtraction Angiography of carotid artery ***(Quoted from Suttonl., 2002).***

3- Spiral CT Angiography

CT angiography is a noninvasive modality compared to DSA that can be used for studying the extra cranial portion of the carotid arteries. ***(Buelmke DA et al., 1995).***

CT Angiography is a highly accurate and precise technique for determining the percentage of stenosis. ***(Cinat M et al., 1998).***

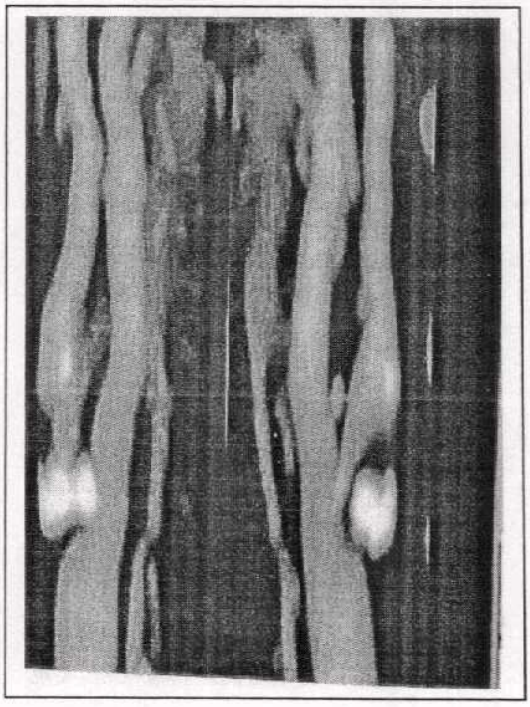

Fig (31): Spiral CT Angiography shows plaque at origin internal carotid artery ***(Quoted from Suttonl., 2002).***

4- Magnetic Resonance Angiography of the Carotid artery (MRA)

It is a non–invasive imaging modality that makes use of the unique properties of moving protons in an applied magnetic field to provide a representation of blood flow ***(White JE et al., 1994).*** This technique has expanded greatly in recent years as it is considered an ideal method for demonstrating lesions of major vessels. Tow

basic strategies are used for imaging, namely time of flight effect (TOF) and phase contrast (PC).

Pitfall of MRA is lack of differentiation between severely stenosed and occluded category and lack of differentiation between arteries and vein of the neck. To eliminate signal from jugular veins, prostrations can be applied in an axial plane at or above the skull base so jugular vein will saturated and give no signal on imaging the carotid arteries. ***(Mattle HP et al., 1991).***

Fig (32): Magnetic Resonance Angiography of the Carotid artery (MRA) **(Quoted from Suttonl., 2002).**

Patients AND METHODS

The study was performed on 40 patients with strokes related to atherosclerotic or ischemic changes in their extra cranial carotid arteries. These patients were selected from medical Neurology, intensive care units and surgical departments of Sayed Galal Hospital, Al-Azhar University from August, 2003 to March 2004.

They were 21 males (52.5%) and 19 females (47.5%). Their age range from 41 years to 75 years with average age of 59 years.

All these patients were subjected to

1- Complete history taking

2- Clinical examination.

3- The presences of any risk factor for carotid atherosclerosis were recorded including: smoking, D.M, hypertension, obesity, coronary heart disease, and lower limb ischemia.

4- They were all subjected to color coded Duplex of the extra cranial carotid and vertebral arteries, Plaques are assessed using the B-scan regarding the common sites (mainly bifurcation), degree of stenosis that was calculated in the transverse and longitudinal axis, also plaque features were assessed including its homogeneity, echo characters and wall regularity.

Assessment of the carotid wall thickness searching for any irregularities, thickness or disappearance of the subintimal lucency was also done.

Many correlations were made between the degree of stenosis and associated age, sex, risk, factors, and plaque features. The aim of this correlation is to study the effect of each factor separately on the occurrence of plaques using statistical analysis.

Different degrees of stenosis were studied and assessed by taking different velocity parameters using P.S.V., E.D.V., and systolic & diastolic velocity ratios.

The Ultrasound equipment used was Philips and GE Logiq. 9, using 7.5 MHZ linear probe. No special preparation for the examination.

A- Carotid artery examination:

1- B-scan:

The patient lies supine with his neck exposed. The neck is extended and in cases of short neck or obesity, the shoulder can be placed on pillow to elongate the neck. The head is slightly rotated away from the side being examined so as to make the vessel more perpendicular to the transducer. The examiner sits beside the patient on his right side and the machine on his left side.

Examination starts by locating the CCA in the lower neck in the transverse plane. The CCA is followed proximally until the transducer is blocked by the clavicle and caudal angulations is tried to evaluate the common carotid origin if possible. The sternocleidomastoid muscle and jugular vein are used as acoustic shadow. The jugular vein is easily identified as it collapses by minimal probe pressure and engorged by suspension of respiration. The CCA is followed upwards till it widens to form the carotid bulb, then it bifurcates into internal and external branches. The transducer is then rotated 90 degree to be parallel to the CCA to have longitudinal scanning on the CCA, the bifurcation, the ICA and the ECA. The ICA was followed distally as far as possible and optimally until it is lost behind the mandible.

Scanning was done through anterior, lateral, and posterior approaches till we have good images for all vessels. We tried to demonstrate the bulb, ICA, ECA in one view; however it was difficult in many patients.

Differentiation of both internal and external carotids was then done. The vessels were evaluated meticulously for the presence of subintimal lucency, atherosclerotic plaques that bulge into the lumen, or generalized wall thickening as well as the lumen echogenecity. The wall is considered thick if it was more than 1mm. Any plaque was studied regarding its location, size, extent, surface contour and its intrinsic echo-pattern. The plaque echogenecity is

compared with that of the surrounding fascia. The presence of calcification with acoustic shadow is recorded. Plaque ulceration was diagnosed if there is discrete deep defect in the plaque surface.

The percentage of diameter stenosis is calculated from longitudinal scan using the formula.

$$\frac{\textit{Residual lumen diameter (RLD)}}{\textit{Vessel lumen diameter (VLD)}} \times 100$$

- VLD is measured from intima to intima.
- RLD is the diameter of the lumen at the point of maximum stenosis.

2- *Pulsed Doppler:*

B-scan imaging is used to put the Doppler sample volume in the area of interest. The Doppler gate must be in the vessel center and the sample volume is kept smaller than the vessel diameter, except when occlusion is suspected. The transmitted power and received gain were adjusted to have clear spectrum with minimal background noise.

Sampling is done for CCA, ICA, ECA on both sides. When there is difficulty in differentiating internal from external carotids, we do tapping test. Simply, tapping sharply over the superficial temporal artery as it passes over the tempero-mandibular joint will produce flow alterations in the proximal ECA, while if the sample volume is in

the ICA, no changes will be observed. When any plaque is identified, sampling was done proximal, at and distal to it, special care was taken to ensure sampling at the point of maximum stenosis.

We used the following pulsed Doppler parameters:

1- Peak systolic velocity (PSV) at the point of maximum stenosis, pre and post stenosis.

2- End diastolic velocity (EDV) at the point of maximum stenosis, pre and post stenosis.

3- Ratio between PSV at maximum stenosis and CCA (PSVR).

4- Ratio between EDV at maximum stenosis and CCA (EDVR).

These Doppler parameters were used to measure the percentage of stenosis according to the principles of ***(Bluth EL, et al., 1988).***

B- Examination of vertebral arteries by pulsed Doppler:

The position of the patient as the same as in the carotid artery examination. The transducer is placed on the anterior neck to identify the CCA in long plane, then slight lateral rotations to bring the vertebral artery into view as tubular sonolucent structure with periodic shadowing from the cervical transverse processes that

interrupt the vessel during its passage through the transverse foramina.

Caudally, the vertebral artery could be traced down to its origin from the subclavian artery. Pulsed Doppler sampling from the vertebral artery reveals low resistant spectrum with spectral broadening as the vessel is small in caliber.

The vertebral artery is examined for the following:

1- Non visualization.

2- Size.

3- Presence or absence of flow.

4- Velocity.

5- Volume flow.

6- Flow direction.

RESULTS

Color Doppler examination on the extra cranial carotid arteries was done for 40 patients complaining of ischaemic strokes on different sides of the brain.

The 40 patients with carotid atherosclerosis comprised 40 carotid systems affected as follows:

22 patients had right carotid plaques.

18 patients had left carotid plaques.

Among these 40 diseased carotid, 25 carotids showed mild stenosis, 9 carotids showed moderate stenosis, 4 carotids showed severe stenosis, and 2 carotid showed complete occlusion of the ICA.

Table (7) : Number of affected carotid arteries with defferies with different degree of stenosis.

Degree of stenosis	Frequency	%
Mild (1 – 39)	25	62.5
Moderate (40 – 59)	9	22.5
Severe >60	4	10.0
Occlusion 100%	2	5.0
Total	**40**	**100**

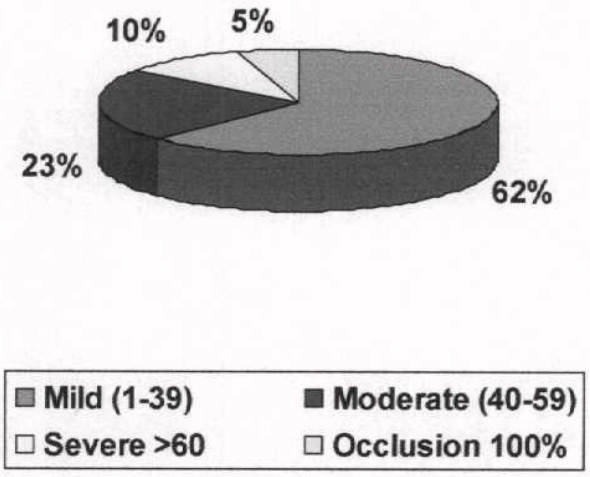

Fig. (33): Pie chart showing number of affected carotid arteries with different degree of stenosis

Table (8): Relation between age of patient and severity of stenosis.

Lesion severity	>60 years		<60 years	
	Frequency	%	Frequency	%
Mild (n = 25)	11	44	14	56
Moderate (n = 9)	6	66.7	3	33.3
Severe (n = 4)	3	75	1	25
Occlusion	2	100	0	0

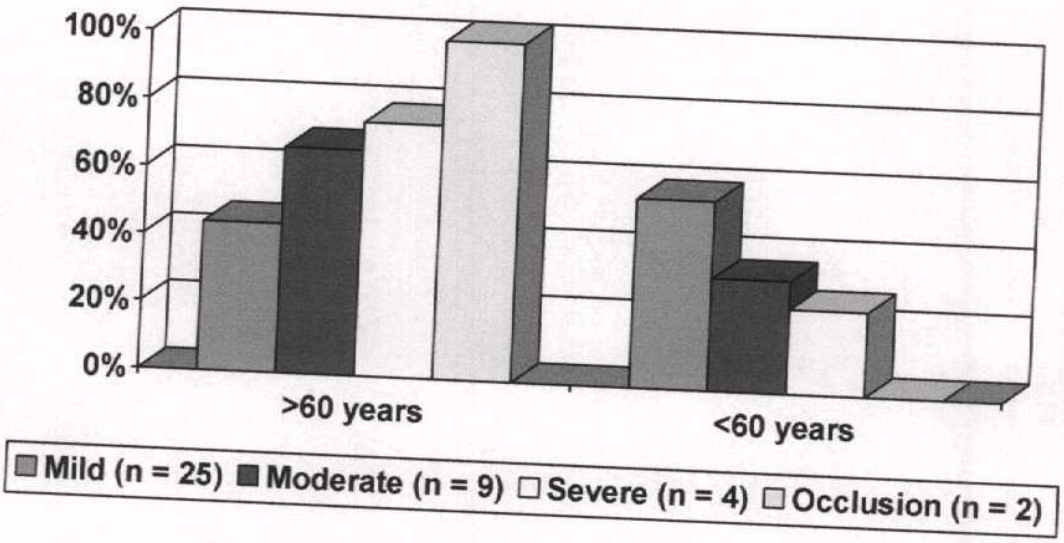

Fig. (34): Bar graph showing relation between age of patients and severity of stenosis.

Most of severely stenotic lesions occur in old age above 60 years.

Table (9): Distribution of risk factors in studied group (n =40)

Risk factor	+ve		-ve	
	Frequency	%	Frequency	%
Smoking	19	47.5	21	52.5
Hypertension	26	65	41	35
D . M	15	37.5	25	62.5
Coronary attacks	13	32.5	27	67.5
T . I . A	12	30	28	70
Hyper - cholesterolaemia	15	37.5	25	62.5

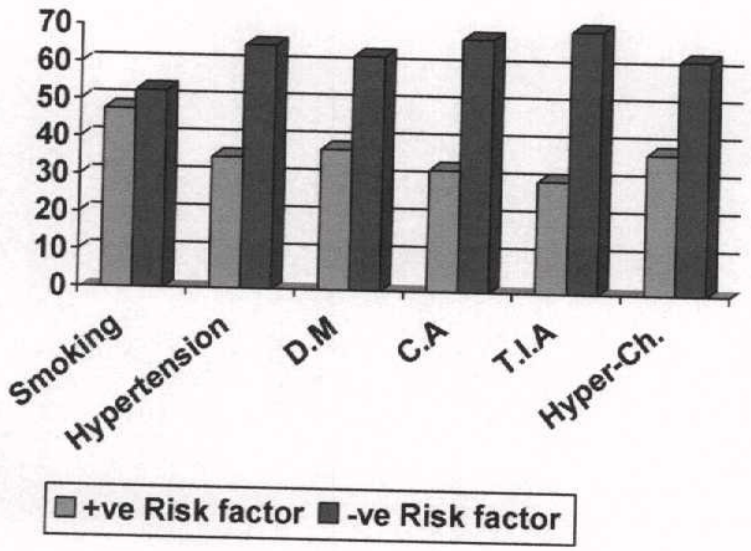

Fig. (35): Bar graph showing distribution of risk factors in studied group.

Table (10): Relation between risk factors and severity of stenosis

	Smoking		Hypertension		D.M	
Mild (n=25)	9	36	18	72	9	36
Moderate (n=9)	6	66.7	5	55.5	4	44.4
Severe (n=4)	3	75	2	50	2	50
+ Occlusion (n=2)	1	50	2	100	1	50

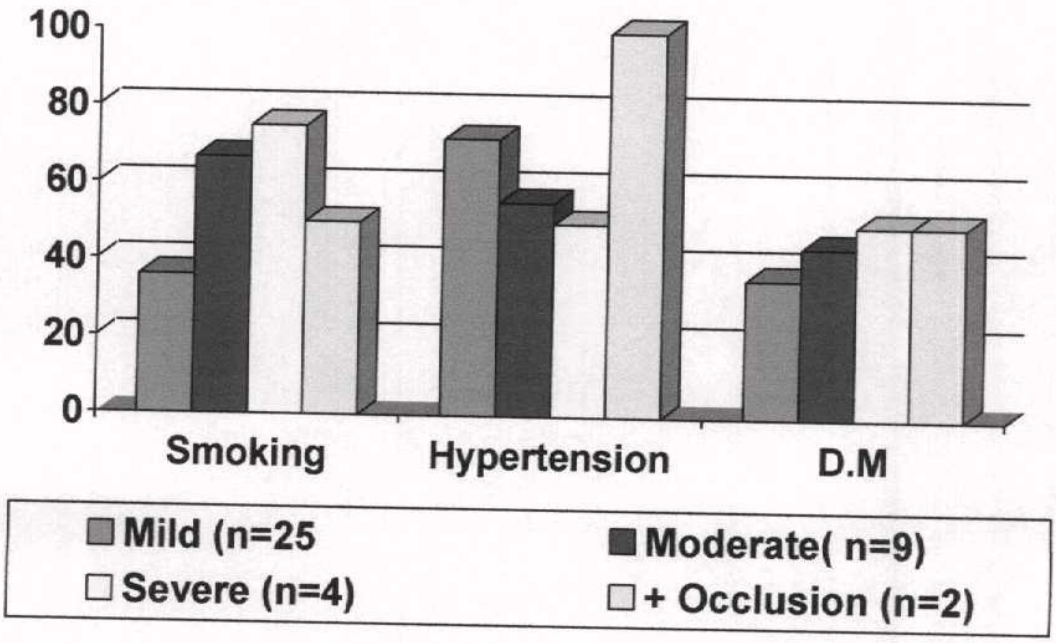

Fig. (36): Bar graph showing relation between severity of stenosis and different risk factors

Table (11): Relation between carotid wall thickness and severity of stenosis

Degree of stenosis	No of affected carotids	No of patient with carotid wall thickness	%
Mild	25	5	20
Moderate	9	6	66.6
Severe	4	4	100

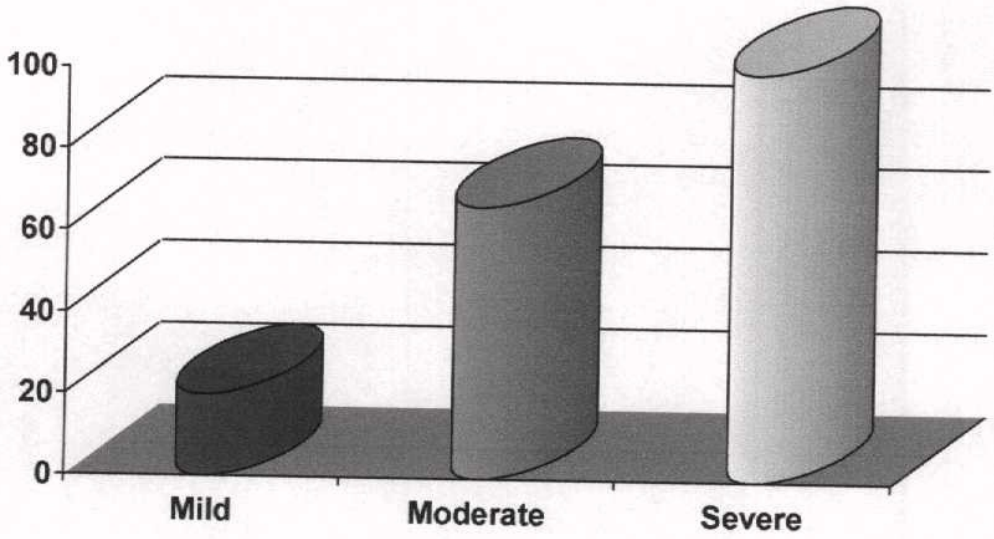

Fig. (37): Bar graph showing relation between carotid wall thickness and severity of stenosis

100 % of severely stenotic lesions have increased wall thickness

Table (12): Number of affected carotids with different sites of stenosis

Site of plaque	Frequency	%
Bifurcation	15	37.5
Bifurcation & I . C . A	10	25
I . C . A alone	8	20
C . C . A	7	17.5

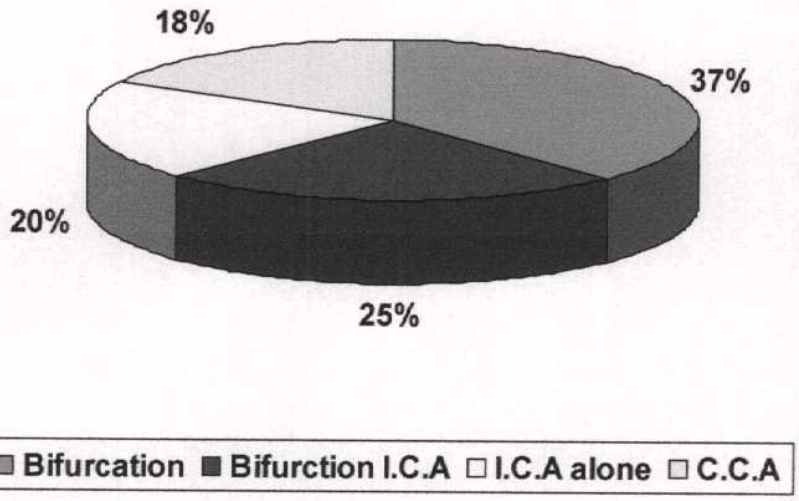

Fig. (38): Pie chart showing number of affected carotids with different sites of stenosis

Bifurcation is the commonest site for plaque formation

Table (13) : Echo characteristics of plaque

Plaque homogenicity	Frequency	%
Homogenous	12	30
Heterogeneous	28	70
Total	**40**	**100**

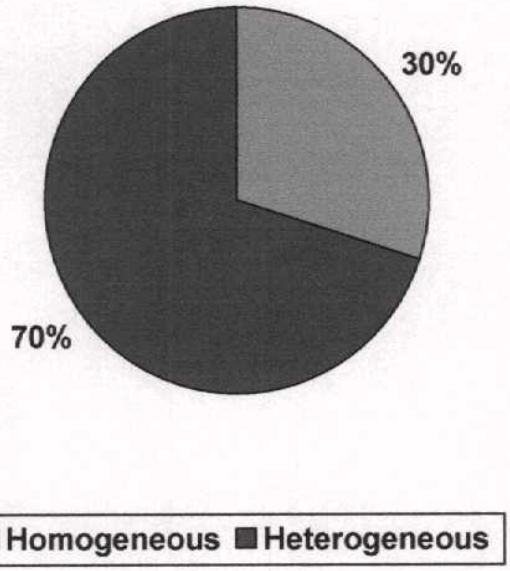

Fig. (39): Pie chart showing echo characteristics of plaque

Plaques are mostly heterogeneous

Table(14): Relation between plaque homogenicity and severity of stenosis

Degree of stenosis	Homogenous		heterogeneous	
Mild (n=25)	10	40	15	60
Moderate (n=9)	2	22.2	7	77.7
Severe (n=4)	0	0	4	100

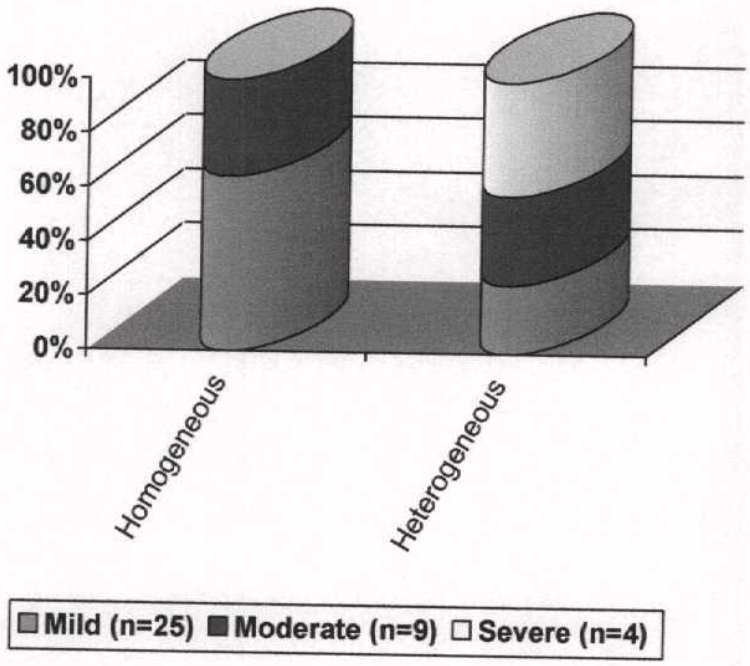

Fig. (40): Bar graph showing relation between plaque homogenicity and severity of stenosis

All cases of severe stenosis were caused by heterogeneous plaques

Table (15) : Plaque echogenicity

Plaque echogenicity	Frequency	%
Hyperechoic	28	70
Isoechoic	5	12.5
Hypoechoic	7	17.5
Total	**40**	**100**

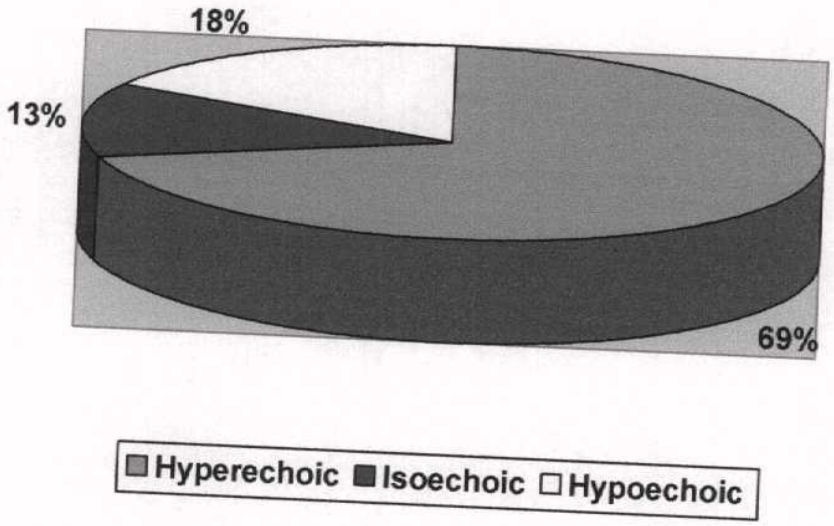

Fig. (41): Pie chart showing plaque echogenicity

Most of plaques were hyperechoic

Table (16) : Plaque surface.

Plaque homogenicity	Frequency	%
Smooth	14	35
Irregular	26	65
Total	**40**	**100**

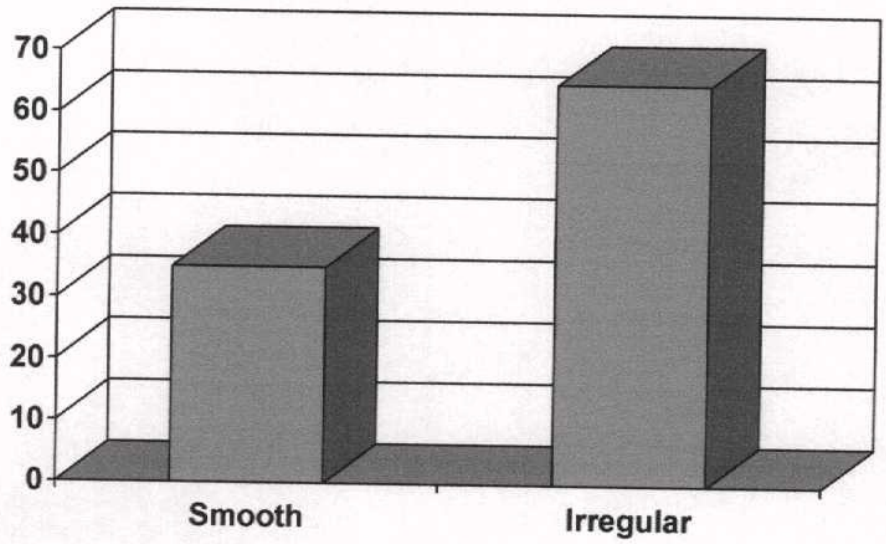

Fig. (42): Pie chart showing plaque surface

Most of plaques have irregular surface.

Case (1)

Male patient 40 years present with Rt sided hemiparasis. He is smoker. Non diabetic or hypertensive. (Fig. 43 a,b,c,d,e).

Gray Scale:

Normal arterial wall with normal intima-media thickness 0.6 mm. No atheromatous plaque

Color flow:

Normal color flow of both carotid and vertebral arteries.

Spectral wave form:

- Normal spectral wave of Lt CCA, ICA, ECA & vertebral arteries.

	PSV	EDV
CCA	110.05 cm/sec	40 cm/sec
ICA	102.28 cm/sec	27.64 cm/sec
PSV ratio	0.93	
EDV ratio	0.69	
Vertebral artery	45.22 cm/sec	8.4 cm/sec

Impression:

Normal duplex finding of left carotid system.

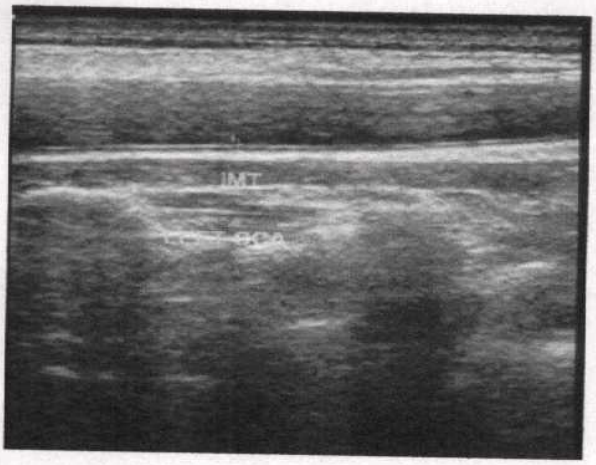

Fig. (43 A): B-Mode showing normal intima-media thickness (0.6 mm).

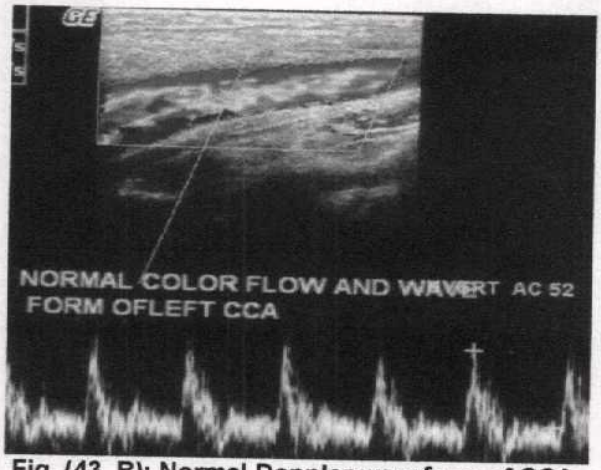

Fig. (43 B): Normal Doppler waveform of CCA.

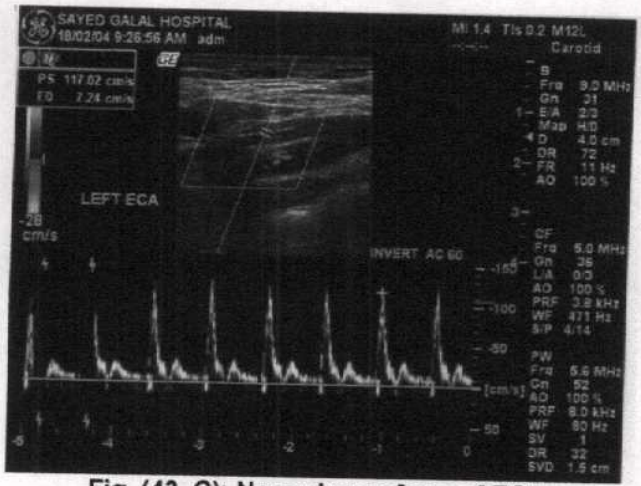

Fig. (43 C): Normal waveform of ECA.

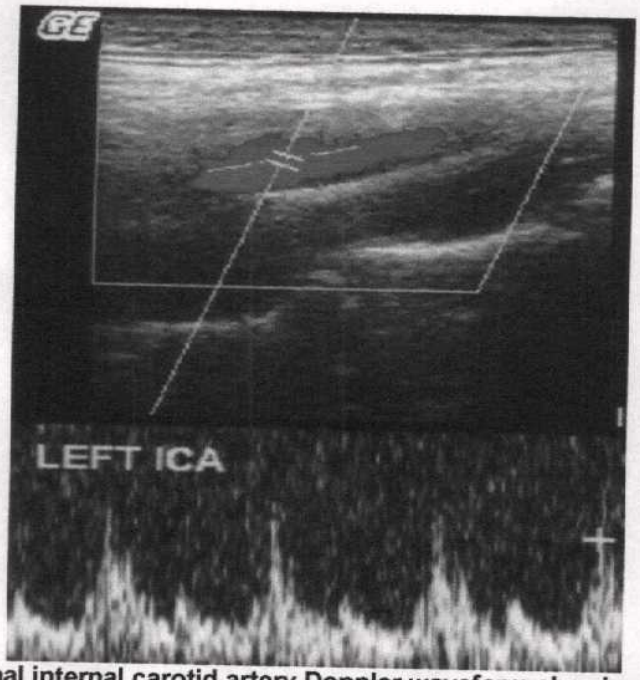

Fig. (43 D): Normal internal carotid artery Doppler waveform showing relatively broad peaks and large amount of flow throughout diastole.

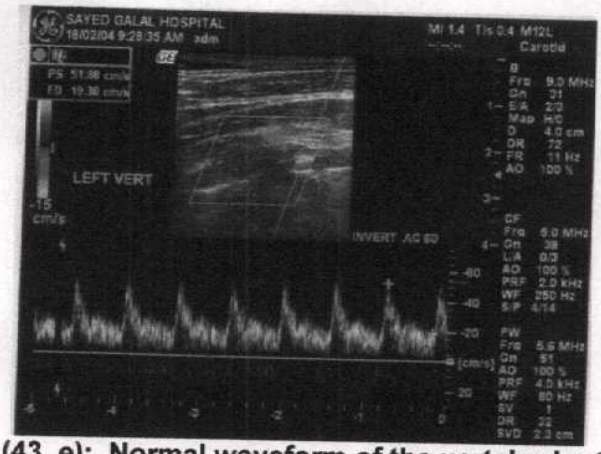

Fig. (43 e): Normal waveform of the vertebral artery.

Case (2)

Male patient 63 years old, present with Lt sided hemiplegia. Rt eye blindness. He is hypertensive. Diabetic and obese. (Fig. 44 a,b,c).

Gray Scale:

Diffuse atherosclerotic changes of arterial wall. Evident by increase intima – media thickness 1.3 mm with acute hypoechoic thrombus at the origin of Rt ICA.

Color flow:

No color flow at Rt ICA.

Spectral analysis:

- No signal wave form of Rt ICA.

Conclusion:

Totally occlusion of right internal carotid artery with diffuse a atherosclerosis of Rt carotid system.

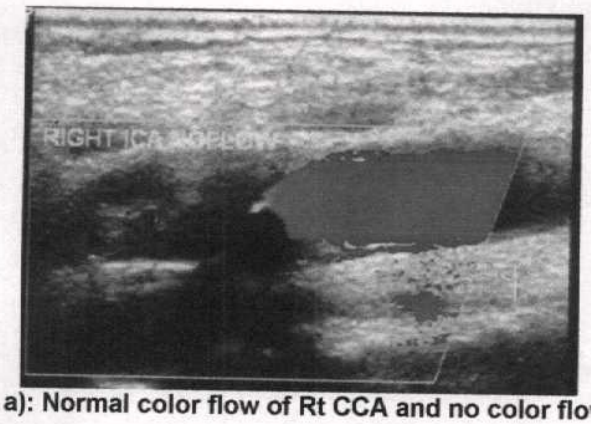

Fig. (44 a): Normal color flow of Rt CCA and no color flow of ICA.

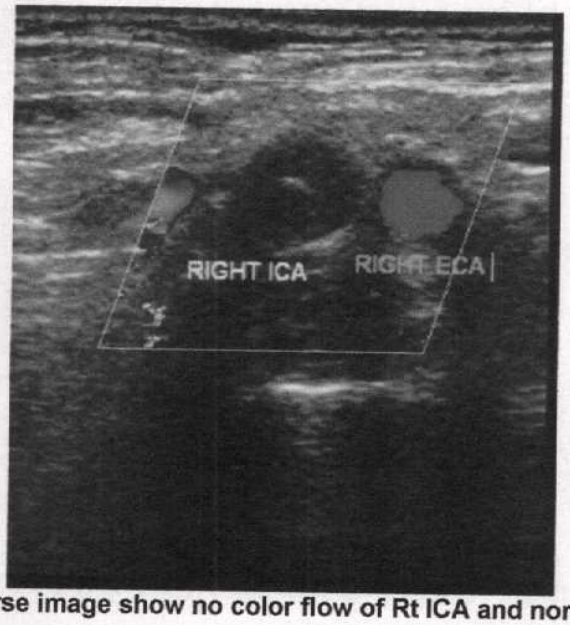

Fig. (44 b): Transverse image show no color flow of Rt ICA and normal color flow ECA.

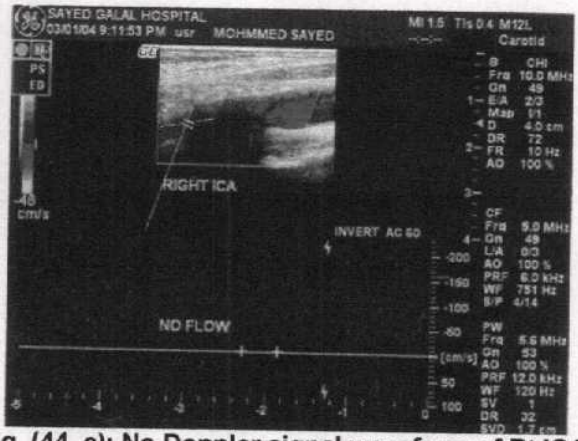

Fig. (44 c): No Doppler signal waveform of Rt ICA.

Case (3)

Male patient 64 years present with Lt side hemiplegia. He is diabetic. (Fig. 45 a,b,c,d).

Gray Scale:

Atherosclerotic change of arterial wall evident by increase intima-media thickness 1.2 mm. with calcified heterogenous plaque measuring 1.7 cm x 0.42 cm is in Rt ICA.

Color flow:

Post stenotic disturbed (turbulence) flow.

Spectral analysis.

Elevated PSV at stenotic segment 121.9 cm/sec & elevated EDV 40.6 cm/sec.

PSV ratio 2.17

EDV ratio 1-7.

Conclusion:

55% stenosis at proximal right ICA with atherosclerotic change at the right extra-carotid wall.

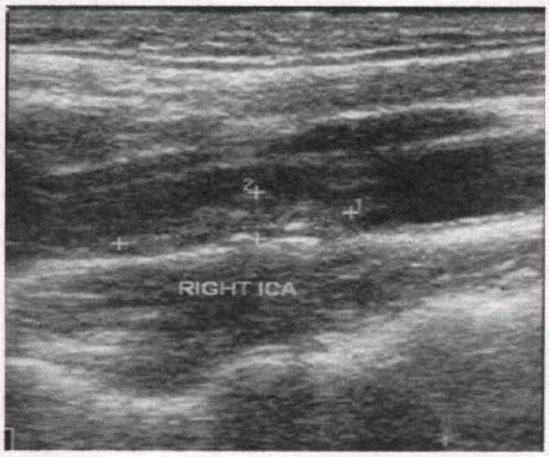

Fig. (45 a): Calcified heterogenous plaque at origins of Rt ICA & extending to it's proximal part.

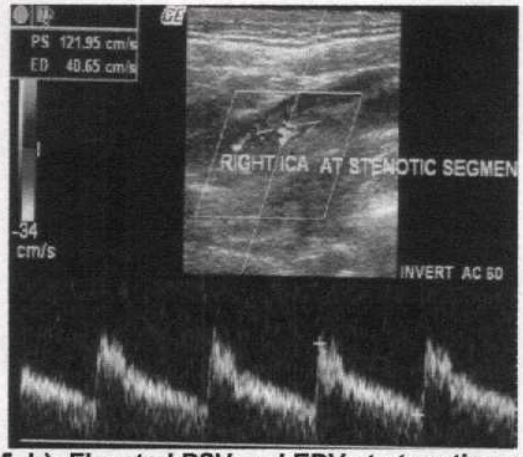

Fig. (45 b): Elevated PSV and EDV at stenotic segment.

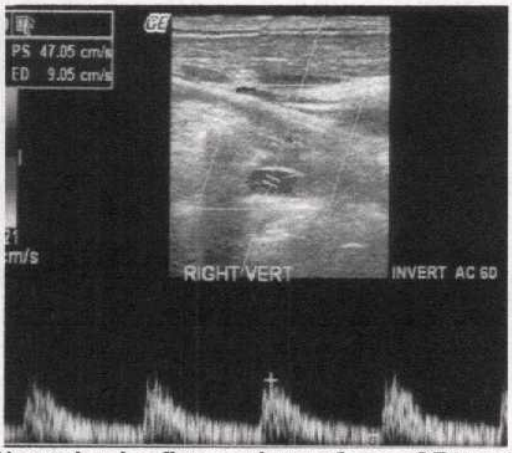

Fig. (45 c): Normal color flow and waveform of Rt vertebral artery.

Case (4)

Male patient 58 years present with Rt sided hemiplegia. He is hypertensive, diabetic smoker, and hypercholesterlemia. (Fig. 46 a, b, c, d, e).

Gray Scale:

Atherosclerotic change of arterial wall evident by increase intima-media thickness 1.1 mm and with iso-echoic plaque at Lt carotid bulb measuring 1.56 x 0.37 cm with stenosis 34.21%.

Color flow:

Normal color flow of Lt carotid and vertebral arteries.

Spectral wave form:

	PSV	EDV
CCA	47.65 cm/sec	12.50 cm/sec
ICA	59.72 cm/sec	17.19 cm/sec

Impression:

34.24% stenosis at the Lt carotid bulb with diffuse atherosclerotic change Lt carotid system.

Fig. (46 a): Increase IMT of Lt carotid system 1.1 mm.

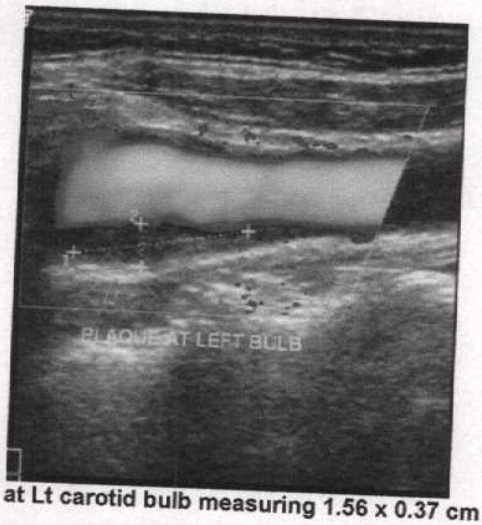

Fig. (46 b): Fibrous plaque at Lt carotid bulb measuring 1.56 x 0.37 cm causing stenosis 34.2%.

Fig. (46 c): Longitudinal image determine percent of stenosis.

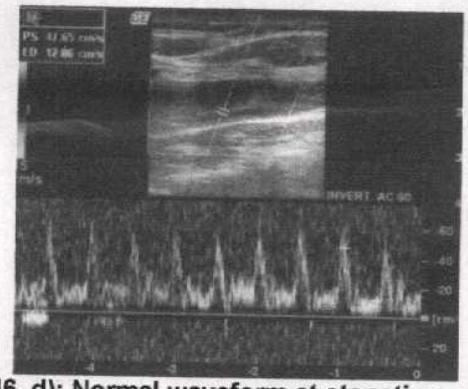

Fig. (46 d): Normal waveform at stenotic segment.

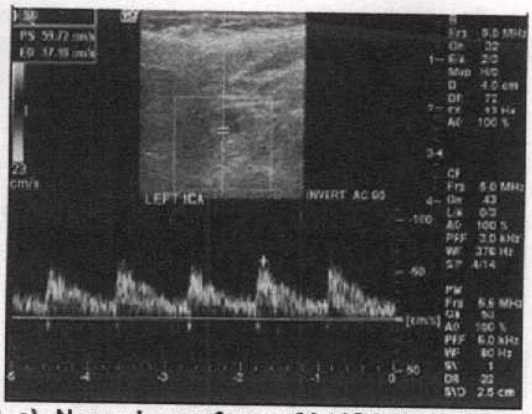

Fig. (46 e): Normal waveform of Lt ICA distal to stenosis.

Case (5)

Male patient 48 years present with Lt sided hemiplegia. He is smoker. Diabetic and hypertensive. (Fig. 47 a,b,c).

Gray Scale:

Increase intima-media thickness 1.0 mm with calcified heterogeneous plaque measuring 1.64 x 0.39 cm with stenosis 72.8%.

Color flow:

Post stenotic disturbed flow.

Spectral wave form:

At stenotic segment:

Elevated PSV	149.6 cm/sec
Elevated EDV	53.08 cm/sec
PSV ratio	2.3
EDV ratio	2.9

Impression:

Stenosis 72.82% of Rt carotid bulb with atherosclerotic changes of right extra-cranial carotid system.

Fig. (47 a): Calcified heterogeneous plaque at Rt carotid bulb with posterior a caustic shadow.

Fig. (47 b): Longitudinal image determine present of stenosis.

Fig. (47 c): Elevated PSV 149.5 cm/sec. and EDV 53.08 cm/sec. at stenotic segment.

Case (6)

Male patient 68 years present with Lt sided hemiplegia. He is not smoker. Diabetic and hypertensive. (Fig. 48 a,b,c).

Gray Scale:

Diffuse atherosclerotic change of arterial wall with increase initima-media thickness 1.2 mm with hyperechoic plaque at Rt bulb and extending to proximal part of ICA measuring 1.6 x 0.4 cm.

Color flow:

Post stenotic disturbed flow.

Spectral wave form:

Proximal to stenosis:

PSV	57.9 cm/sec
EDV	12.06 cm/sec

At stenotic segment:

Elevated PSV	271.9 cm/sec
Elevated EDV	78.40 cm/sec
PSV ratio	4.6
EDV ratio	6.5

Impression:

Critical stenosis more than 80 % of Rt internal carotid artery with atherosclerotic changes of right extra-cranial carotid system.

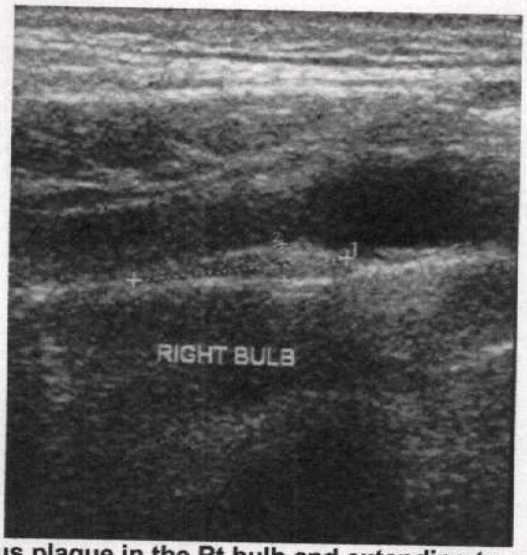

Fig. (48 a): fibrous plaque in the Rt bulb and extending to proximal Rt ICVA.

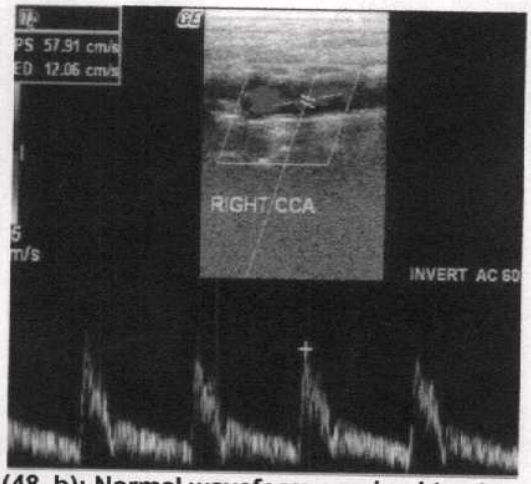

Fig. (48 b): Normal waveform proximal to stenosis.

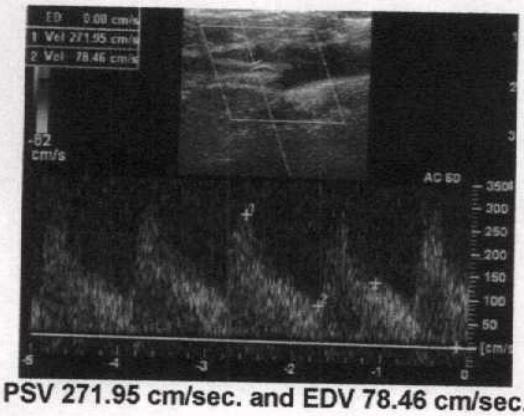

Fig. (48 c): Elevated PSV 271.95 cm/sec. and EDV 78.46 cm/sec. at stenotic segment.

Case (7)

Female patient 71 years present with coma, Lt sided hemiplegia. She is hypertensive, diabetic C.T. was done and revealed Rt deep parietal infarction. (Fig. 49 a,b,c,d,e).

Gray Scale:

Diffuse atherosclerotic change of arterial wall of Rt carotid arteries with increase initima-media thickness 1.1 mm and calcified atheromatous irregular surface, ulcerative plaque at Rt bulb and extending to Rt ICA measuring 1.57 x 0.4 cm. Causes stenosis 35.54%.

Color flow:

Normal color flow with flow in plaque.

Spectral wave form:

	PSV	EDV
Rt CCA	72.39 cm/sec	15.08 cm/sec
Rt ICA at stenotic segment	47.12 cm/sec	16.80 cm/sec
PSV ratio	0.65	
EDV ratio	1.1	

Impression:

Ulcerative plaque causing mild stenosis (35.54%) at Rt ICA with atherosclerotic changes of right extra-cranial carotid system.

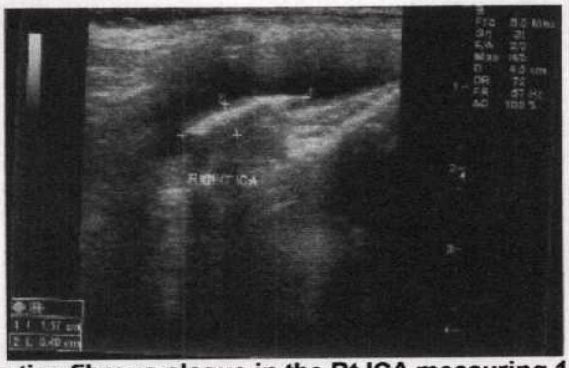

Fig. (49 a): Ulcerative fibrous plaque in the Rt ICA measuring 1.57 x 1.7 cm/sec.

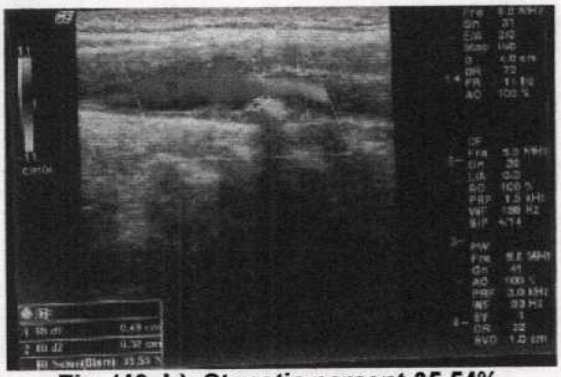

Fig. (49 b): Stenotic percent 35.54%.

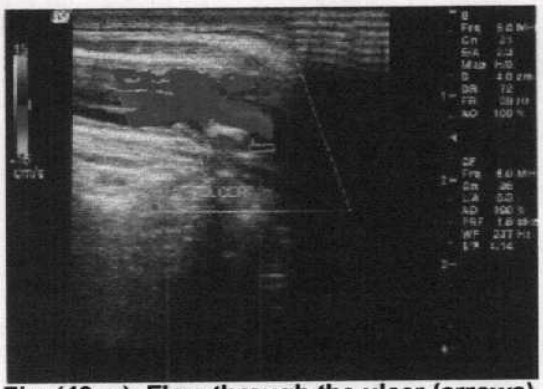

Fig. (49 c): Flow through the ulcer (arrows).

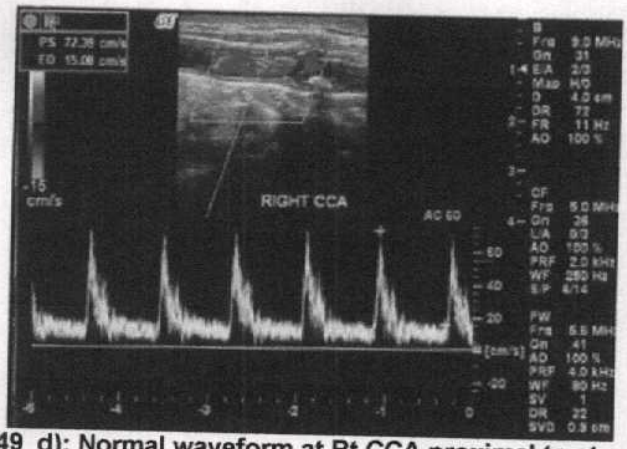

Fig. (49 d): Normal waveform at Rt CCA proximal to stenosis.

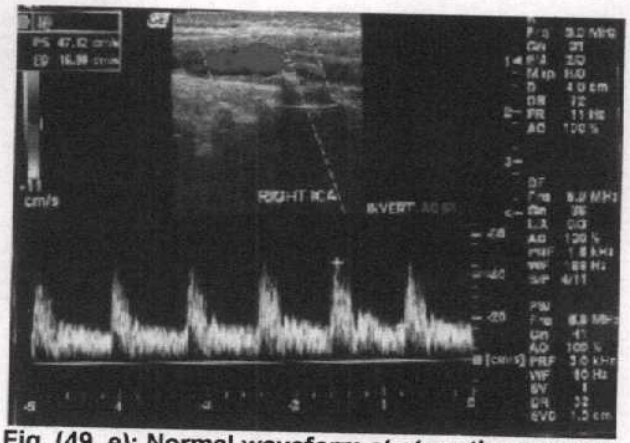

Fig. (49 e): Normal waveform at stenotic segment.

Case (8)

Female patient 68 years present with acute onset of Rt sided hemiplegia. She is diabetic and has A.F. and mitral stenosis, C.T. was done and revealed Lt temporoparietal infarct. (Fig. 50 a,b,c,d).

Gray Scale:

The lumen of Lt internal carotid artery is occupied by hypoechoic thrombus.

Color flow:

Show no color flow of Lt internal carotid artery.

Duplex findings:

Absence of pulsations in the left internal carotid artery, the left common carotid artery show high pulsatility consisting of sharp peaks with low amplitude, diastolic flow reversal in early diastole and relative decrease flow in late diastole, suggestive of distal obstruction of the left side.

Impression:

Totally occluded left internal carotid artery.

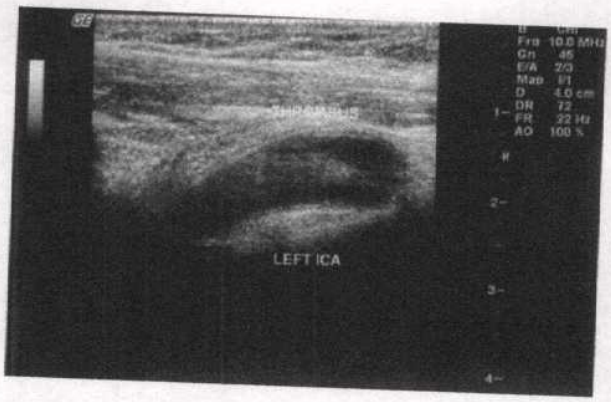

Fig. (50 a): Hypochoic thrombus in Lt ICA.

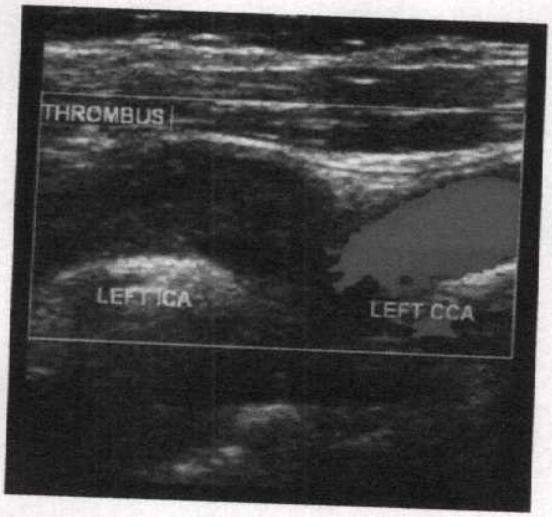

Fig. (50 b): Normal color flow of Lt CCA and no flow at Lt ICA.

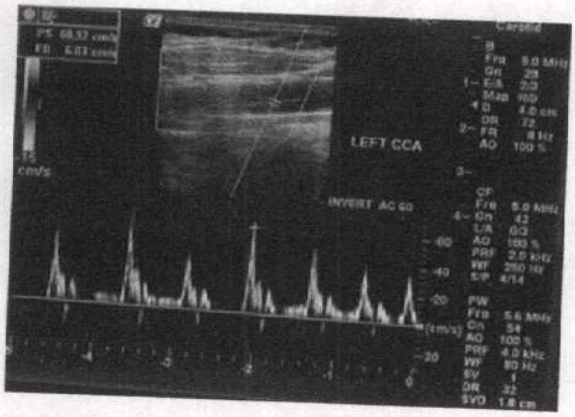

Fig. (50 c): High resistant flow in Lt CCA due to distal obstruction.

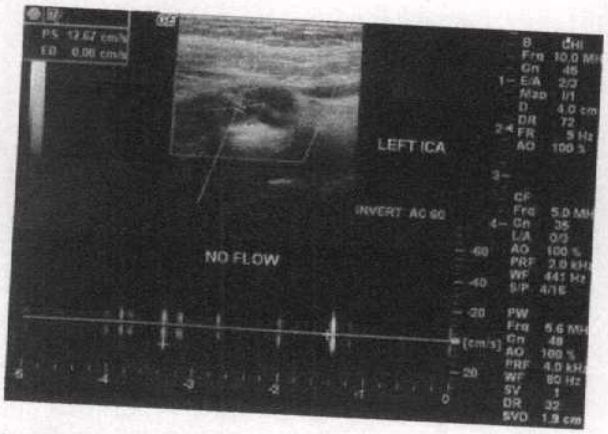

Fig. (50 d): No Doppler signal waveform of Lt ICA.

Case (9)

Male patient 59 years presented with left sided hemiparasis. He is diabetic hypertensive, smoker has recurrent attack of stroke. (Fig. 51 a,b,c,d).

Gray Scale:

Atheromatous calcified heterogenous plaque at right carotid bulb, measuring 0.8 x 0.22 cm.

Color flow:

Normal color flow.

Spectral wave form:

	PSV	EDV
Rt CCA	66.14 cm/sec	13.37 cm/sec
Rt ICA	44.34 cm/sec	13.57 cm/sec
Ratio	0.67	1.01

Impression:

Atheromatous calcified plaque at Rt carotid bulb with no hemodynamic significant stenosis.

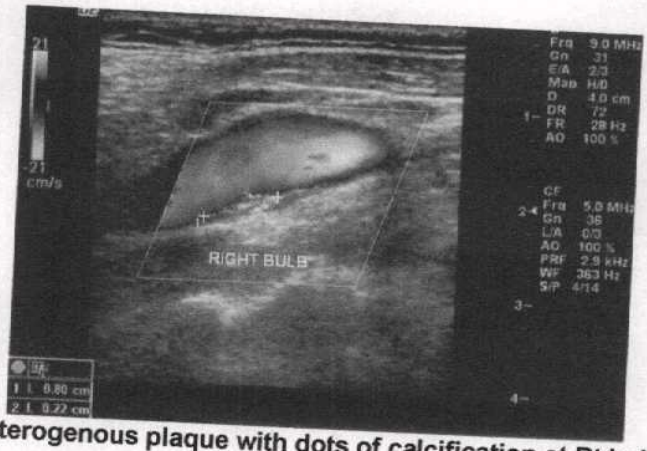

Fig. (51 a): Heterogenous plaque with dots of calcification at Rt bulb causing no significant stenosis.

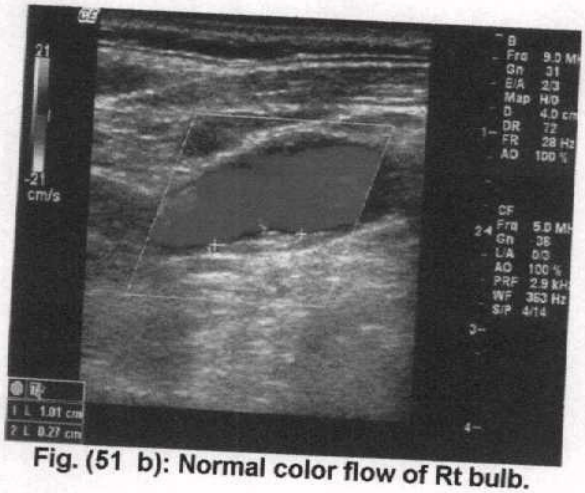

Fig. (51 b): Normal color flow of Rt bulb.

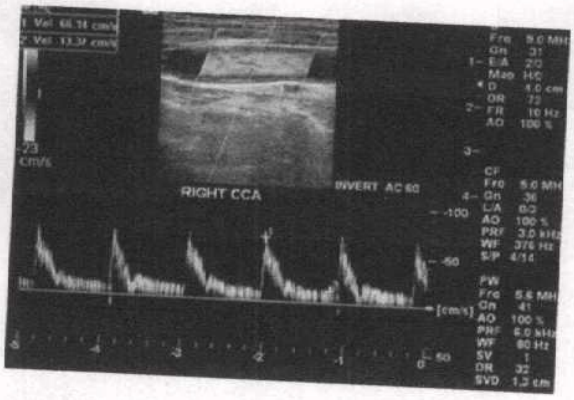

Fig. (51 c): Normal waveform of Rt CCA.

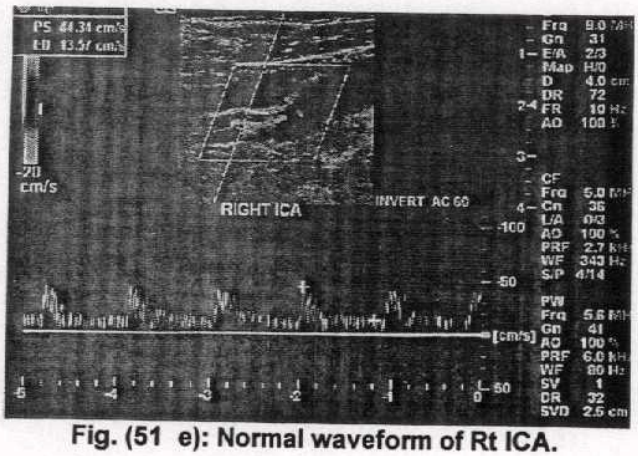

Fig. (51 e): Normal waveform of Rt ICA.

Case (10)

Female patient 69 years present with Lt sided hemiplegia. She is diabetic and hypertensive. (Fig. 52 a,b,c,d,e).

Gray Scale:

Diffuse atherosclerostic change of arterial wall evident by increase intima-media thickness 1.2 mm with regular smooth fibrous plaque is seen in Rt carotid bulb and extending to RT, ICA measuring 2.43 x 0.37 cm causing 33.7 stenosis.

Color flow:

Normal color flow.

Spectral wave form:

	PSV	EDV
Prestenotic Rt CCA	63.34 cm/sec	17.19 cm/sec
At stenotic segment	39.81 cm/sec	22.92 cm/sec

Impression:

Mild stenosis of Rt common carotid (33.7%) with diffuse atherosclerosis of Rt carotid system.

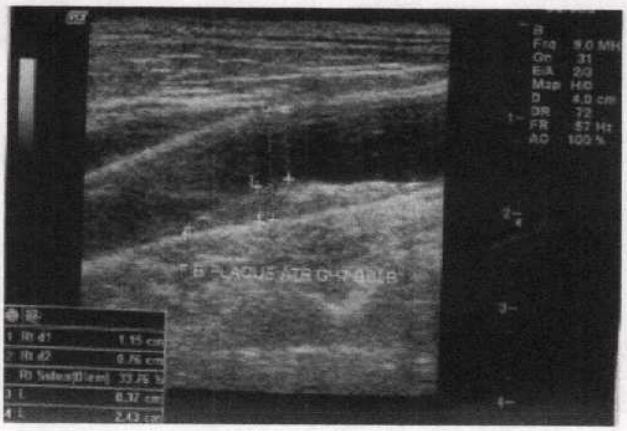

Fig. (52 a): Fibrous plaque at RT, bulb causing stenosis 33.76%.

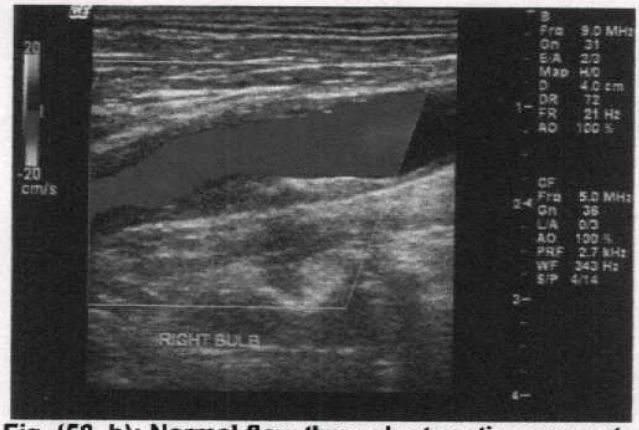

Fig. (52 b): Normal flow through stenotic segments.

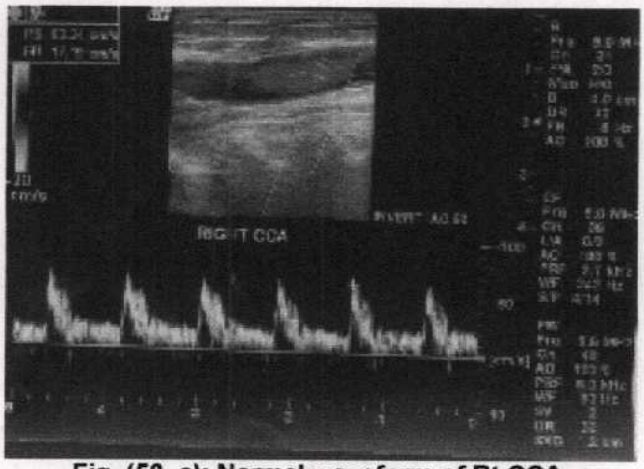

Fig. (52 c): Normal waveform of Rt CCA.

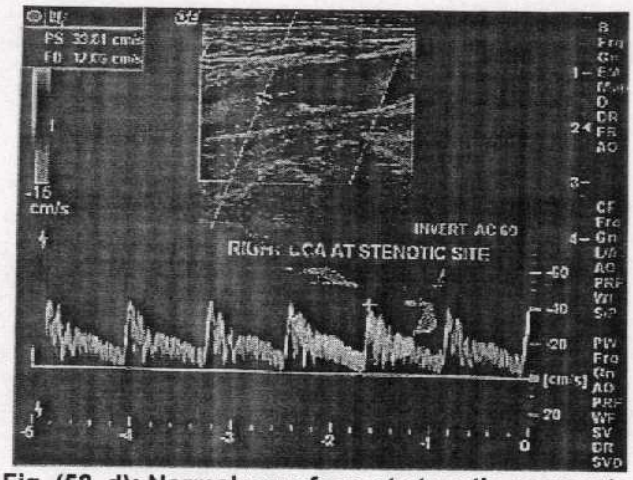

Fig. (52 d): Normal waveform at stenotic segments.

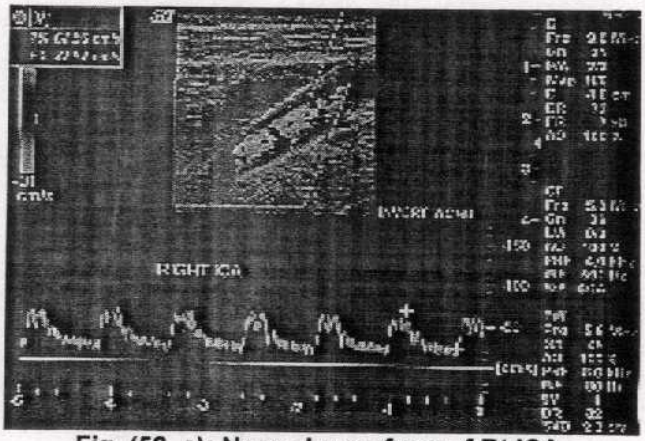

Fig. (52 e): Normal waveform of Rt ICA.

Discussion

Duplex scanning of the carotid arteries combines, B-mode ultrasound and Doppler ultrasound within a single instrument, thus providing both anatomical imaging of the vessel and flow velocity information. Combining these two unique aspects of ultrasound overcomes many of the individual limitations of each, and provides improved non-invasive diagnosis of cerebrovascular disease ***(Taylor and Strandness, 1987)***.

Atherosclerosis is a degenerative disease causing narrowing of the lumen and obstruction of blood flow or ulceration and thrombosis ***(Zarins and Glagov., 2000).***

The relation between the occurrence of severe stenosis and different risk for atherosclerosis had been demonstrated (table 10).

We noticed that the age of the patients significantly affects the degree of stenosis as 75% of the patients with severe stenosis were above 60 years old. The severity of stenosis decreases with younger age as only 25% of our patients developed severe stenosis was below 60 Years old. This is proved in statistical analysis. These results coincides with ***(Reneman RS., 1986)*** who said that people above 60 years old should be routinely screened for the presence of severe carotid atherosclerotic changes.

Smoking was associated with severe stenosis as also 75% of the group with severe stenosis was smokers. This is in agreement with ***(Muller HR et al., 1996)*** who determined that cigarette smoking is a determinant risk factor for carotid atherosclerosis.

Diabetes mellitus was found in 37.5% in our study, 50% of them developed severe carotid atherosclerosis, so it is also an important risk factor, and this agrees with ***(Arosio E et al., 1989)***.

Transient Ischemic Attack (T.I.A 's) was found in 30% of affected carotids in the study, and so it is considered as a warning sign for developing carotid stenosis, and so patients that had a history of T.I.A, should be screened for carotid atherosclerosis, and this agrees with ***(Dennis M et al., 1996)***, as they mentioned that T.I.A increases risk factor for developing stroke 5-10 times.

The association of coronary artery diseases and patients with hypercholesterolemia was significant in the study, and this agrees with ***(Homer D et al., 1991)***, when they mentioned that coronary artery diseases and hypercholesterolemia are risk factors that predisposes for carotid atherosclerosis.

Diagnostic ultrasound has the unique ability to evaluate the composition of atherosclerotic plaque.

Atherosclerotic plaque is represented sonographically by echogenic material that thickens the intimal reflection and encroaches on the arterial lumen. Minimal plaque formation may be

detected by measuring the total intimal-medial thickness. Widening of the carotid wall more than 1 mm. may represent an abnormal findings.

Having detected carotid plaque, it is important to describe its severity and to assess the plaque accurately; this is achieved by describing two features, extent and severity.

By extent, it means the length of the plaque (cephalocauded) affected by plaque, which is determined with longitudinal images of the carotid arteries (e.g, plaque extends from the distal common carotid artery into the proximal internal carotid artery)

By severity, it means thickness of plaque this is more difficult to define sonographically, because plaques vary in thickness from one location to another. The best means for assessing plaque thickness from transverse images, which most accurately show the maximum thickness of the plaque and the resultant degree of luminal narrowing. Plaque severity may be estimated in longitudinal images. From transverse images, plaque thickness is reported in millimeters, and Whether the Plaque is localized or circumferential.

Regarding Plaque location among the total number of the significant plaques in this study which was 40 plaques, 15 plaques were found at the bifurcation (37.5%), 10 plaques (25%) were found at the bifurcation and extends to the I.C.A, and 8 (20%) plaques were found in the I.C.A alone, and only 7 (17.5%) plaques were found in the C.C.A. No plaques were found in the E.C.A (Table 12).

This means that the large percent of plaques lies at the bifurcation (62.5%), and this is due to the turbulence of blood flow found at the carotid bulb ***(Middleton WD et al., 1988)***. This emphasizes much care during examining the bifurcation area, using the posterior approach that make the posterior wall of the bifurcation and the I.C.A more superficial to the probe.

E.C.A plaques is not found, this is important because some times examination of the E.C.A may be difficult because of its small caliber and different anatomical variations in its locations in relation to the I.C.A. Therefore, in these cases, terminating the examination without studying the E.C.A will reduce the examination time with minimal loss of diagnostic informations.

Regarding plaque characters, we found that 40% of cases with mild stenosis had homogenous plaques, while all cases of severe stenosis were caused by heterogenous plaques (Table 14).

In our study, most of plaques were hyperechoic, and these hyperechoic plaques were responsible for 84% & 77% of mild and moderate stenosis respectively, however in severe stenosis, 80% of affected groups were found to have hypoechoic plaque texture.

The surface of the plaques was also studied and are noticed that the percentage of severe lesions with irregular surface was 80% compared to severe lesions with smooth surfaces, which was found only in 20%, and this is significant according to our statistical analysis.

Our findings showed that plaques tend to be more heterogeneous, hypoechoic texture and irregular surface as the severity of the stenosis increases, and the greater the stenosis, the greater the likelihood of strokes. Thus, plaque characters are related to severity of stenosis, prevalent T.I.A and recurrent strokes and this was stated by ***(Carr S, et al., 1996)***.

The capability of ultrasound to detect heterogeneous plaques is of very important prognostic value, as all heterogeneous plaques are more liable to intraplaque hemorrhage and ulceration that correlate with increased incidence of strokes. The importance of intraplaque hemorrhage lies in the fact that it may lead to sudden elevation of the fibrous cap of plaques and sudden increase in the degree of stenosis or even occlusion ***(Widder B, et al., 1994)***.

B-Scan assessment of stenosis

All patients with different degrees of luminal stenosis had been examined (table 7). The carotid plaque thickness and the resultant degree of luminal narrowing were calculated from the transverse and longitudinal image. In fact a good quality transverse diameter was not early to get in every case. In approximately 25% of patients, an adequate transverse measurement could not be obtained and the percent of stenosis was based upon the sagittal view only. We used some terms in describing stenosis severity as mild, moderate, severe and critical stenosis. According to ***(Bluth EI et al., 1988)***, degrees of stenosis were categorized as mildly stenosed (1-39%),

moderately stenosed (40-59%), severely stenosed (60-79%), critical stenosis (80-99%) or occlusion.

This study showed that 62.5% of patients developed mild stenosis, 22.5% developed moderate stenosis, 10% developed severe stenosis, and finally 5% developed occlusion.

The walls of the carotid arteries were examined regarding their thickness and texture. In this study, the percentage of the patients with carotid wall thickness increased from 20% in mild stenosis, 66.6% in moderate stenosis, up to 75% in severe stenosis, thus the carotid arteries wall thickness increased as a function of severity of carotid artery stenosis, and this is in agreement with ***(Sutton-Tyrrell K et al, 1992)***.

The diagnostic role of Doppler ultrasound of the carotid arteries for detection of stenotic lesions is of high value; Doppler U\S also plays an important role in detection of high-grade stenotic lesions with sensitivity and specificity exceeding 90%. ***(Howard G et al, 1996)***.

The degree of stenosis of the internal carotid artery depending on Doppler criteria. Peak systolic velocity less than 110 cm/sec. is found in normal or mild degree of stenosis, less than 130 cm/sec. for moderate stenosis (40-59%), more than 130 cm/sec for severe stenosis and more than 250 cm/sec for critical stenosis ***(Bluth et al., 1988)***.

The peak systolic velocities greater than 100 cm/sec in the internal carotid artery are highly predictive of stenosis greater than 50% ***(Garth et al., 1999)***.

In our study, peak systolic velocity was measured in all cases and it was found to be less than 110 cm/s in mild degrees of stenosis (25 carotids), less than 130 cm/sec for moderate stenosis (9 carotids) and more than 130 cm/sec for severe stenosis (4 carotids).

The peak systolic velocity ratio(PSVR) was less than 1.3 in mild stenosis, 1.9 in moderate stenosis, reaching 2.8 in severely stenosis, thus it also shows linear increase with the severity of stenosis.This agrees with ***(Bluth EL et al, 1988)*** when he mentioned that significant stenosis should be suspected when the PSVR exceeds 1.8.

As arterial stenosis increases in severity, flow velocity rises initially only in systole, as luminal narrowing progresses beyond 50% diastolic velocities also increase, and the systolic/diastolic velocity ratio falls. In severe stenosis that exceeds 70% diameter reduction, very high velocities are present throughout the cardiac cycle and the ratio of systolic to diastolic velocities is statistically reduced ***(Dreisbach et al., 1983)***, the high grade lesions cause diastolic velocity to increase, which represents the final effort to transport as much blood as possible across a tight lesion, diastolic flow greater

than 4.5 KHz (> 80-135 cm/sec) indicates a stenosis greater than 80% ***(Roederer et al., 1983)***.

The end diastolic velocity less than 60% is less than 40 cm/sec., while it is more than 40 cm/sec. for severe stenosis (60-79%) and more than 100 cm/sec. for critical stenosis (80-99%) ***(Bluth et al., 1988)***.

In our study, we used end diastolic velocity, in different degrees of stenosis, we met 34 carotid with end diastolic velocity less than 40 cm/sec. these represented normal and stenotic carotid plaques up to 59%. And we also met 4 carotids in which end diastolic velocity was more than 40 cm/sec, and these three carotids were diagnosed as severe stenosis (60-79%) and critical stenosis (80-99%).

Role of Color:

Color Doppler gives a real time, global overview of areas of flow abnormalities within vessels. A quick transverse and Longitudinal color Doppler US survey of the carotid artery pinpoints areas of abnormal flow which appear as heterogeneous color pattern, luminal narrowing or both. Identification of abnormal areas of color allows the operator to place the pulsed Doppler sample volume correctly in an area of color Doppler abnormality, thus streamling the time consuming process of pulsed Doppler spectral analysis along the entire course of a vessel.

In addition, although qualitative estimations of flow abnormality can be made by observing a heterogeneous, narrowed color pattern, spectral analysis remains essential. The color patterns reflect only mean velocity values, whereas peak velocity values (by Doppler analysis) are necessary for accurate estimation of degrees of stenosis ***(Carroll, 1991)***.

In our study, we used color Doppler for global overview of areas of flow abnormalities within the vessel and correct placement of the sample volume of pulsed wave Doppler in the area of maximum, color Doppler abnormality (post stenotic jet), this was carried out in all cases, however color Doppler data were not able to provide more than mean velocity values and so spectral analysis was essential for ultimate determination of degree of stenosis.

Arterial occlusion is diagnosed with duplex sonography by: absence of arterial pulsations, lumen filled with echogenic material, subnormal vessel size and absence of Doppler signal ***(Zwiebel, 1987)***.

Tight carotid artery stenosis or occlusion usually creates increased resistance to flow in the epsilateral vessel proximal to the lesion. This results in a damped wave form with decreased, absent or even reversed and diastolic flow, except when collateralization to the intracranial circulation has resulted (usually through braches of the epsilateral external carotid artery) ***(Carroll, 1991)***.

In this study, we have two cases of complete carotid occlusion, which were accurately diagnosed by duplex before doing angiography. Duplex sonography is very important in cases of complete carotid occlusion as color coded duplex sonography can detect very low traces of blood flow thus differentiating between complete occlusion (inoperable), and incomplete occlusion (operable).

SUMMARY & CONCLUSION

A major cause of thromboembolic cerebral stroke is atherosclerotic narrowing and ulceration in the region of carotid bifurcation. The indication for carotid surgery may or may not include the presence of symptoms, but all indications include the presence of a significant plaque that may result in embolic or occlusive disease ***(Dogan & Dempsey, 2000)***.

Stroke is a non-transient, acute neurological injury, resulting from interruption of the blood flow to cerebral tissue. It is the third leading cause of death in elderly patients after heart disease and cancer. Stroke is considered a serious disease because it has 25 % mortality rate and 75 % morbidity rate with residual neurological defect. Many hemispheric events result from atherosclerotic disease in the extracranial carotid arteries, a location amenable surgical corrections as carotid endarterectomy. The risk factors of carotid arteriosclerosis include age, sex, smoking, hypertension, diabetes mellitus, and history of coronary attacks.

Patients who complaining of transient ischaemic attacks (TIAs) or stroke should be screened for extracranial carotid arteries diseases.

Duplex sonography with color Doppler techniques is widely used to screen patients with suspected carotid artery diseases.

Forty patients complaining of cerebrovascular strokes were examined using duplex and color-coded Doppler.

The most common location of atheromatous plaques is the carotid bifurcation.

High resolution B-scan is very useful in demonstrating characters of plaque including its extension and echogenic texture. Recurrent strokes are more prevalent when the lesion is heterogeneous, hypoechoic, and irregular. These are the characters found in severely stenotic lesions of the extracranial carotid arteries.

Many pulsed Doppler parameters are used to evaluate stenosis, like PSV, EDV & systolic and diastolic velocity ratios; the most accurate is the peak systolic velocity.

Color Doppler U/S can detect ulcerated plaques by filling of color within the lumen of the ulcer, and this is important because ulcerated plaques show high incidence of recurrent strokes.

Color coded duplex is very sensitive in detecting minimal flow in a severely occluded artery and differentiating subtotal from total occlusion, which is confirmed by detecting

absence of pulsations. Also color coded Duplex demonstrates the post stenotic aliasing or flow turbulence.

Finally, it is obvious that Duplex sonography is an ideal method for evaluating patients with carotid arteries diseases, because of its accuracy and in addition, it is economic if compared to magnetic resonance angiography, it is non invasive if compared to angiography and the hazards of contrast media, so the patient always accept duplex sonography than other imaging modalities especially that there is no any contraindication to the technique.

- ***Ackerstaff, R.G.A.; Grosved, W.J.H.M.; Eikelbook, B.C.; Ludwig, J.W.:*** Ultrasound scanning of the prevertebral segment of the vertebral artery in patient with cerebral atherosclerosis . Eur. J. Vasc.Surg. 2:387-393. 1998.
- ***Ammar A.D; Wilson, R.L;Travers, H; et al:*** Intraplaque hemorrhage: its significance in cerebrovascular disease. Am. J. Surg, 148: 840-843, 2001.
- ***Anderson DC, Loewenson R, Yock D, Farber R, Larson D, Bromer M:*** B-mode, real-time carotid ultrasonic imaging. Arch Neurol; 40: 484-488, 1983.
- ***Arosio E, Pancera P, Zannon M, Aracar G, Fincati E:*** Progression of carotid atheromatous involvement in cerebrovascular patients. Advances in vascular pathology. (Strano A and Novo S) eds. 77, 1989.
- ***Bates JA:*** Abdominal ultrasound: How, Why and When. Churchill Livingstone, Edinburgh ,Chap.1,p.1-3,1999.
- ***Beckett W.W.Jr, David, P.C., Hoffman, J.C.Jr .:*** Duplex Doppler sonography of the carotid artery: false positive results in an artery contralateral to an artery with marked stenosis ,AJNR .11:1049.1990.
- ***Bendick, P.J; Glover, J.L.:*** Homodynamic evaluation of evaluation of vertebral arteries by duplex ultrasound . Surg Clin. North Am .70 (1) 235-244. 1990.
- ***Berman S.S., Devine J.J., Erdoes, L.S., et al:*** Distinguishing carotid artery pseudo-occlusion with flow Doppler. Stroke. 26: 434.1995.
- ***Black shear, E.I; Merritt, C.R.B; et al:*** Sonographic characterization of carotid plaque: Detection of hemorrhage. AJR . 146:1061-1065, 1980.
- ***Bluth EI, Stavros AT, Marich KW, et al.*** Carotid duplex songraphy: a multicenter recommendation for standardized imaging and Doppler criteria. Radiographics; 8:487, 1988.

- ***Bluth,E.I ; Mcvay , L.V. et al :*** The identification of ulcerative plaque with high resolution duplex sonographic carotid scanning .J. Ultrasound Med .7:73-76,1988.
- ***Bornstein, N. M.; Krajewski, A. Norris, J.W.:*** Basilar artery blood flow in subclavian steal.Can J. Neurol. Sci 15:417-419. 1988.
- ***Bornstein, N.M.; Norris, J.W.***: Subclavian steal: A harmless haemodynamic phenomenon? Lancet Aug.9:303-309. 1998.
- ***Bridges S.L.***: Clinical correlates of Doppler/ultrasound errors in the detection of internal carotid artery occlusions. Stroke. 20-612. 2001.
- ***Buelmke, D.A, Chambers, T.P.***: Spiral CT angiography .an alternative to conventional angiography. Radiology; 195:317, 1995.
- ***Carr S, Farb A, Pearce WH***: Atherosclerosis plaque rupture in symptomatic carotid artery stenosis. J. Vasc Surg., 12: 755, 1996.
- ***Carroll BA***. Duplex sonography in patients with hemispheric symptoms. J ultrasound Med; 8:535, 1989.
- ***Carroll,B.A.***: Carotid sonography: state of the art. Radiology. 178-303,1991.
- ***Cinat, M. Lane CT, Pham, H. Lee, A.:*** Helical CT angiography in the preoperative evaluation of carotid artery stenosis . J. Vasc. Surg.:28:290, 1998.
- ***Comerata, A.J.; Cranley , JJ. and Cook, S.E:*** Real-time B-mode carotid imaging in diagnosis of cerebrovascular disease .Survey ,6:718-729 ,1981.
- ***Cooperberg, P.L; Robertson, W.D; Fry, P.; et al***: High resolution ultrasound of the carotid bifurcation. J. Clinical Ultrasound 7:13-17, 1979.

- ***Courbier, R,; Reggi, M. and Jausseran, J.M.:*** Non invasive cerebrovascular diagnosis :Current Concepts .1st edn . Baltimore University, park Pp.61-73, 1998.
- ***Cranley JJ, Karkow WS & Baldridge ED:*** Atlas of duplex scanning: carotid arteries .WB Saunders Company, Phialdelphia Chap 1,p.1-39 1989.
- ***Davenport,L.; Sterpetti,A.V ; Schultz ,R.D.; et al.:*** Carotid artery plaque imaging and the risk of subsequent stroke. J. Vasc. Technol., 12:245-249.1988.
- ***Denis M, Bamford J, Sandercock P, Warlow CP:*** Incidence of transient ischaemic attacks in oxfordshire, England Stroke; 20: 333, 1996.
- ***Derdeyn CP, Powers WJ, Moran CJ Cross III DT and Allen BT:*** Role of Doppler US in screening for carotid atherosclerotic disease. Radiology, 197; 635-643, 1995.
- ***Dogan A. and Dempsey R.J.:*** Diagnostic modalities for carotid artery disease. Neuro. Surg. Clin. N. Am. 11: 205-220, 2000.
- ***Dreisbach, J.N.; Serbert, C.E.; Smazal, S.F. et al.:*** Doppler sonography in the evaluation of carotid artery disease. Am. J. Neuro. Radio., 4: 678-680, 1983.
- ***Eastcott, H.H.G; Pickering, G.W. and Rob, G.C:*** Reconstructuion of internal carotid artery in a patient with intermittent attacks of hemiplegia. Lancet, 2:994-999.1984.
- ***Erickson S.J., Mewissen M.W., Foly W.D, et al .:*** Color Doppler Evaluation of arterial stenosis and occlusions involving the neck and thoracic inlet. Radiographics. 9-389: 1989.
- ***Fisher, M.;Blumenfeld, A.M.: Smith, T.W.:*** The importance of carotid artery plaque disruption and haemorrhage. Arch. Neurol., 44:1066-1088.1999.
- ***Garth, K.E; Carroll, B.A.; Sommar,F.G. et al.:*** Duplex ultrasound scanning of carotid arteries with velocity spectrum analysis. Radiology, 174: 823-827. 1999.

- ***Gooding, G.A.W and Effency D.G.:*** Static and Real-time B-mode sonography of arterial occlusions. AJR. 139: 949-954 1982.
- ***Hallam, M.J.; Reid , J.M. and Cooperberg, P.L.:*** Color flow Duppler and conventional duplex sonographic scanning of the carotid bifurcation : prospective double blind, correlative study. Am. J. Radiol., 152: 1101-1105. 1989.
- ***Holman BL:*** The research that radiologists do; perspective based on a survey of the literature. Radiology 176: 329, 1990.
- ***Homer D, Ingall TI, Baker HL***: Serum lipids and lipoproteins are less powerful predictors of extra cranial carotid artery atherosclerosis than cigarette smoking and hypertension. Mayo Clinic Proc., 66: 259, 1991.
- ***Howard G, Baker WH, Chambless LE***: An approach for the use of Doppler ultrasound as a screening tool for haemo-dynamically significant stenosis. Stroke, 27: 1951, 1996.
- ***Imparato, A.M.; Riles, T.S., Mintzer, K.; et al:*** The importance of hemorrhage in the relationship between growth morphologic characteristics and cerebral symptoms in 376 carotid artery plaques. Ann. Surg., 97: 159-198, 1983.
- ***Jacobs NM, Grant EG, Shellinger D, et al.:*** Duplex carotid sonography: criteria for stenosis, accuracy, and pitfalls. Radiology 154: 385, 1997.
- ***Jak, J.G., Hoeneveld, H., Vander. Windt, J.M, et al ,:*** Asix year evaluation of duplex scanning of the vertebral artery . Noninvasive technique compared with contrast angiography. J Vasc Technol .13:26-30.1998.
- ***Kassam, M.; Kohoston, K.W. and Cobbold, R.S.C.:*** Quantitative estimation of spectral broadening for the diagnosis of carotid arterial disease : method and in vitro results. Ultrasound Med. Biol., 11:425-433., 1985.

- ***Klews PM***: Introduction to color duplex sonography .In Color duplex sonography .Wolf KJ & Fobbe F. Thieme, New York, Chap.1, P1 1995.

- ***Kotval PS, Babu SC, Shah PM***: Doppler diagnosis of partial vertebral/subclavian steals convertible to full steals with physiologic maneuvers. J. Ultrasound Med 9: 207, 1990.

- ***Kremakau FW:*** Doppler principle seminars in Roentgenology. Radiology, 27: 6-16, 1992.

- ***Landwehr P:*** Carotid and vertebral arteries . In:Color duplex sonography . Wolf KJ & Fobbe F Thieme ,New York, Chap. 5, P. 45-48,1995

- ***Langsfeld, M.; Gray-Weale, A.C. and Lusby, R.J.:*** The role of plaque morphology and diameter reduction in the development of new symptoms in asymptomatic carotid arteries.J. Vasc. Surg., 9: 548-557,1989.

- ***Leahy, A.L,;McCollum, P.T.; Feeley, T.M; et al.***: Duplex ultrasonography and selection of patient for carotid endarterectomy :plaque morphology or luminal narrowing .J. Vasc. Surg., B:558-562, 1998.

- ***Lee VS, Hertzberg BS, Kliewer MA and Carroll BA***: Assessment of stenosis implications of variability of Doppler measurement in normal appearing carotid arteries. Radiology, 212; 493-498, 1999.

- ***Lincoln L.B.; Therese M.W.:*** Diagnostic ultrasound. In Textbook of Lippincott-Raven Publishers, Philadelphia. Vol. 1, Ch. 31, P. 1016, 1998.

- ***Mattle ,H.P., Kent, K.C., Edelman, R.R.:*** Evaluation of the extra cranial Carotid arteries. Correlation of magnetic resonance angiography, duplex ultrasonography, and conventional angiography . J Vasc Surg , 13 : 838, 1991.

- ***Merritt, C.R.B.***: Doppler Blood flow imaging: Integrating flow with tissue Data. Diag. Imaging,11:149 – 155, 1986.

- ***Meyer, J.S.; Lobe, C.:*** Stroke due to vertebro-basilar disease. Springfield, I.L.: Charles, C Thomas .:137. 1995.

- ***Middleton WD, Foley WD Lawson TL :*** Flow reversal in the normal carotid bifurcation : Color Doppler flow imaging analysis .Radiology .167:207-210,1988.
- ***Muller HR, Buser MW:*** Smoking and hypertension; risk factors for carotid stenosis. J Neurol, 238: 97, 1996.
- ***Ó Donnell, T.R.; Erodoes, L.; Mackey, W.C.; et al.:*** Correlation of B-mode ultrasound imaging and arteriography with pathologic finding at carotid endarterectomy. Arch. Surg., 120:443-449 ,2001.
- ***O'leary DH, Holen J, Ricotta JJ, et al.:*** Carotid bifurcation disease; Prediction of ulceration with B-mode US. Radiology 162: 523, 1987.
- ***Persson, A.J.; Robichaux, W.T. and Silverman, M.:*** The natural history of carotid plaque development . Arch .surg. , 118:1048-1052, 1985.
- ***Peter, N.T.:*** Instrumentation including color flow imaging In:Taylor, K.I.W., et al. ed. Clinical Application of Doppler ultrasound , 2nd Ed. New York, Raven , Pp . 10-35, 1995.
- ***Polak JF, O'Leary DH, Quist WC, Greeger MA and Logerfo FW:*** Pulse and color Doppler analysis of normal carotid bifurcation flow dynamics using an in vitgro model. Angiology, 41; 241-246, 1990.
- ***Polak JF:*** Noninvasive carotid evaluation; carp diem. Radiology 186: 329, 1993.
- ***Prince BD, Saylor MM & Sanders RC :*** Doppler and color flow principles. In Clinical sonography. Sanders RC &Miner AS (eds.).Lippincott, Philadelphia.3rd ed., Chap 5, P.22-29, 1998.
- ***Rae RE:*** Cerebrovascular sonography, Doppler, and non invasive examination. In: Textbook of diagnostic ultrasonography. Hagen–Ansert SL(ed.). Mosby, London, Vol.1, Chap 22.p 615-625,1995.
- ***Rath PC, Agarwala MK, Dhar PK, Lakshmic, Ahsan SA, Debt Kumar S. Narasimham RR, Rao PS and Dixity:*** Carotid artery involvement in patient of atherosclerotic

coronary artery disease undergoing coronary artery by pass grafting Indian. Heart J, 53 (6); 761-765, 2001.

- ***Ratliff, D.A.; Gallagher, P.J. and Hames, T.K.:*** Characterization of carotid artery disease: comparison of duplex scanning with histology. Ultrasound Med. Biol., 11-835-840, 1985.

- ***Reilly, L.M.; Lusby, R.J. ; Hughes, L.; et al.:*** Carotid plaque histology using real-time Ultrasonography. Am. J. Surg., 146:188-193, 1983.

- ***Reneman RS, Vanmerode T, Ttick P and Hocks AP***: Flow velocity patterns and dispensability of the carotid artery bulb in subjects of various ages. Circulation; 71: 500, 1986.

- ***Ricotta, J.J.; Holen, J.; Schenk, E.; et al:*** Is rountine angiography necessary prior to carotid endarterectomy? J. Vasc. Surg., 1:96:102, 1984.

- ***Robinson, M.L.*** Duplex sonography of the carotid arteries. Semin Roentgenol. 27-17, 1992.

- ***Roederer, G.O.; Langlois, Y.E.; Lussiani, L.G.:*** The natural history of the contralateral; non operated carotid artery. Presented at the annual meeting of the International Society for cardiovascular surgery, San Sransico, June 16-17, 1983.

- ***Roth Well, P.M., Salinas, R., Ferrando, L.A., Warlow, C.:*** Dose the angiographic appearance of a carotid stenosis predict the risk of stroke independently of the degree of the stenosis Clinical Radiology ; 50:830, 1995.

- ***Russel,D, Bakke,S.J., Wiberg ,J., et al.:*** Patency and flow velocity profiles in the internal carotid artery assessed by digital subtraction angiography and Doppler studies three months following endarterectomy. J. Neurol Neurosurg. Psychiatry. 49-183, 1998.

- ***Seeger, J.M and Klingman, N.:*** The relationship between carotid plaque composition and neurological symptoms . J. Surg. Res. 43: 78-85, 1991.

- ***Spencer, M.P.:*** Heamodynamic of carotid stenosis .In Spencer,M.P., Reid, J.M.,(eds.): cerebrovascular Evaluation with Doppler ultrasound. The Hague, Martinus Nyhoff,1st edn. Pp113:121, 1981.
- ***Steinke, W.; Klortzsch, C. and Hennerici, M.:*** Carotid artery disease assessed by color Doppler flow imaging : correlation with standard Doppler sonography and angiography . Am. J. Radiol. 154:1061-1068.2000.
- ***Strandness DE Jr.*** Extracranial arterial disease. In: Duplex scanning in vascular disorders. New York, Raven, 92, 1990.
- ***Sutton D & Stevens***: Angiography in Neuroradiology :In textbook of radiology and imaging .Churchill Livingston, Edinburgh vol 2 Chap 56 ,P 1527,1998.
- ***Sutton-Tyrrell k, Wolfson SK Jr, Thompson T, Kelsey SF:***measurment variability in duplex scan assessment in carotid atherosclerosis. Stroke. 23:215-220.1992.
- ***Staskin D.R., Hadley H.R.,Leach G.E.:***Reproduced from clanical anatomy of the Head and neck.Norton Medical publishers, Chapter 8; P.606-613,1996.
- ***Taylor, D.C. and Strandness, D.E.:*** Carotid artery duplex scanning. J. Clinical Ultrasound. 15: 635, 1987.
- ***Tegeler CH & Ratanakorn D***: Physics and principles In : Trans cranial Doppler ultrasonography. Babikian VL, Wechsler LR &Toole JF (eds.). Butterworth-Heinemann, Boston ,2nd ed ., Chap .1 P. 3-12, 1999.
- ***Touboul, P.J.; Bousser, M.G.; LaPlane, D.; Castaigne, P.:*** Duplex scanning of normal vertebral arteries Stroke. 17(5): 921 –923. 2001.
- ***Vanniner , R., Manninen, H., Koivisto,K.:*** Carotid stenosis by digital subtraction angiography. Reproducibility of the European carotid surgery trial and North American symptomatic carotid Endareterectomy trial measurement methods and visual interpretation. Amer. J. of Neuroradiology: 15 : 1635, 1994.

- ***Visona, A.; Lusiani, I.S; Castellani, V.:*** The echo Doppler (duplex) system for detection of vertebral artery occlusive disease. Camparason with angiography. J Ultrasound Med: 5:247, 1986.
- ***Wells, P.N.T.:*** Doppler Ultrasound in medical diagnosis. Br. J. Radiology, 62: 399-420, 1989.
- ***White, J.E., Russell,W.L., Greer, M.S., Whittle,M.T.:*** Efficacy of Screening MR angiography and Doppler Ultrasonography in the evaluation of carotid artery stenosis . Amr. Surg. :60: 340, 1994.
- ***Widder,B.; Paulat,K.;Hackspacher J. et al .:*** Morphological characterization of carotid artery stenosis by ultrasound duplex scanning. Ultrasound Med. Biol.16:349-354, 1994.
- ***Williams PL :*** Grays anatomy. The anatomical basis of medicine and surgery .Churchill Livingstone, New York ,38th ed., 1995.
- ***Wing, V. and Scheible, W.:*** sonography of jagular vein thrombosis. AJR, 140:333-339 ,1995
- ***Withers CE, Gosink BB, Keightley AM, et al.*** Duplex carotid sonography: Peak systolic velocity in quantifying internal carotid artery stenosis, J Ultrasound Med. 9: 345, 1990.
- ***Wolverson, M.K.; Bashiti, H.M. and Peterson, G.J.:*** Ultrasonic tissue characterization of athromatous plaque using high resolution real-time scanner Ultrasound Med. Biol., 6: 669-609, 1983.
- ***Zarins CK & Glagov S:*** Artery wall pathology in atherosclerosis . In : Vascular surgery . Rutherford RB (eds.). WB Saunders company , Philadelphia, Chap. 18, P 313-349, 2000.
- ***Zierler, R.E.; Phillips, D.J.; Beach, K.W.; et al.:*** Non invasive assessment of normal carotid bifuraction haemodynamics with color flow ultrasound imaging. Ultrasound Med. Biol., 13:471-476, 1987.

- ***Zwiebel WJ*** : Introduction to vascular Ultrasonography non imaging Doppler cerberovascular techniques. Grune and startton , New York ,Chap 5 , P.61-76 1982.
- ***Zwiebel WJ.*** Color-duplex features of normal and abnormal carotid arteries. In: Zwiebel WJ, ed. Introduction to vascular ultrasonography, ed 3. Philadelphia, WB Saunders, 123, 1992.
- ***Zwiebel, W.J. :*** Cerebrovascular Doppler application . In : Taylor K.J.W. et al., clinical application of Doppler . Ultrasound ., 2nd Edn., New York ., Pp.: 109-131. 2000.
- ***Zwiebel, W.J. and Cruamy, A.B.:*** Doppler diagnosis of carotid occlusive disease. Am. J. Neuro. Radio. 137: 1-12, 1987.
- ***Zwiebel, W.J. and Knighton, R.:*** Duplex examination of carotid arteries Semin ultrasound ,135:11-97,1990.

الملخص العربي

استهدف هذا العمل دراسة موجات دوبلر فوق الصوتية الملونة في تقييم تصلب الشريان السباتي خارج الجمجمة.

وقد تم في هذا البحث مناقشة النواحي الفيزيائية لموجات دوبلر فوق الصوتية الملونة، كما تضمن هذا البحث وصف تشريحي وطريقة فحص الشريان السباتي.

وفي الوقت الحالي يعتبر الدوبلكس والدوبلر الملون من الفحوصات التي يعتمد عليها بنسبة كبيرة في تشخيص توسعات تصلب الشرايين.

وقد أجريت هذه الدراسة علي ٤٠ مريضا يعانون من مرض السكتة الدماغية بالكشف علي الشرايين السباتية بواسطة جهاز فوق الصوتية والدوبلكس والدوبلر الملون.

ولوحظ من خلال الدراسة قدرة الموجات فوق الصوتية والدوبلكس والدوبلر الملون في تشخيص ترسبات تصلب الشرايين وتحديد نسبة الضيق وأشكال الترسبات في الشرايين السباتية.

وقد أظهرت الدراسة أيضا قدرة الدوبلكس والدوبلر الملون علي تشخيص الانسداد في الشرايين السباتية والتفرقة بين الانسداد الكامل والغير كامل.

وأخيرا يعتبر الدوبلكس والدوبلر الملون فحص مثالي للشرايين السباتية إذا قورن بالفحوصات الأخرى مثل الرنين المغناطيسي أو الأشعة المقطعية الحلزونية أو الأشعة بالصبغة حيث أنه رخيص وغير تداخلي ولا يحتاج المريض إلي الصبغة وما قد ينتج عنها من أضرار كما أنه لا يوجد أي موانع لإجراء هذا الفحص.

دور موجات دوبلر فوق الصوتية الملونة في تقييم تصلب الشريان السباتي

رسالة مقدمة من

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