"EVALUATION OF CAROTID ARTERIES IN ISCHAEMIC STROKE PATIENTS USING COLOUR DOPPLER IMAGING"

By

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Dissertation submitted to the

BLDEUNIVERSITY, BIJAPUR, KARNATAKA

In partial fulfilment of the requirements for the degree of

DOCTOR OF MEDICINE

IN

RADIO DIAGNOSIS

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KARNATAKA

2014

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ACKNOWLEDGEMENT

With regard to the dissertation titled "**EVALUATION OF CAROTID ARTERIES IN ISCHAEMIC STROKE PATIENTS USING COLOUR DOPPLER IMAGING"** I consider it a privilege to express a few words of gratitude and respect to all those who guided and inspired me in completing the dissertation.

Words are not enough to express my deep sense of gratitude and heartfelt thanks to my esteemed guide **Dr. R C PATTANSHETTI** Professor, Department of Radio Diagnosis, Shri B M Patil Medical College, Bijapur, who has been the source of propelling force, complete guidance and inspiration for this dissertation / research work. He has guided me by his constant encouragement and valuable suggestions to carry out and complete this dissertation.

My heartfelt gratitude to **Dr. M M PATIL**$_{DMRD,}$ Senior Resident, Shri B M Patil Medical College, Bijapur, for his constant support and encouragement.

My sincere thanks are due to **Dr. M S BIRADAR** M.D. Principal, Shri B M Patil Medical College, Bijapur, for his support and encouragement.

I want to thank all the department staff **Dr. Shivanand Patil, Dr. Praveen M. Mundaganur, Dr. Vishal N S, Dr. Ajey Bhagwat, Dr. Shriganesh Patil, Dr. Sanket Shah and Dr. Avinash Vernekar** for their help, support and advice.

I thank my Co-PGs, **Dr. Vinod and Dr. Chaitra,** my juniors **Dr. Shivendra, Dr. Sheetal, Dr. Uday** and **Dr. Puneeth,** PG's in Department of Radio Diagnosis who rendered immense help and support during my postgraduate course. I thank them from my heart.

My sincere thanks to **Sapna** and **Bhagyashree,** staff members of the Dept. of Radio diagnosis for their co-operation.

I am thankful to all the Technical and non-teaching Staff of the Department of Radio Diagnosis, Shri. B M Patil Medical College, Bijapur for their co-operation.

I thank **Mrs.Vijaya Soraganvi** of Community Medicine Department for her valuable help in statistical analysis of data.

I sincerely acknowledge the support and kindness shown towards me by all the staff of Central Library, Shri B.M. Patil Medical College, Bijapur, at all times.

I would like to thank **my parents Shri. Gullappa S** and **Smt. Keerti G,** for their constant encouragement, love, sacrifice and support in all the endeavours of my life and work.

I am grateful to **Mr. Kalyankumar, Preeti net zone** who have helped in the editing, printing and binding of this thesis work. I am also thankful to my uncle, **Mr. Basavaraj Kalyani** who helped me with the technical aspects.

Last but not the least, I would like to acknowledge all the participants for providing with necessary data and forming the backbone of this study, without whom this study would not have been possible.

I thank **GOD**, for all his blessings.

Date: **Dr. SACHIN G SHATAGAR**

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LIST OF ABBREVIATIONS USED

USG	-	ULTRASONOGRAPHY
CCA	-	COMMON CAROTID ARTERY
ICA	-	INTERNAL CAROTID ARTERY
ECA	-	EXTERNAL CAROTID ARTERY
PSV	-	PEAK SYSTOLIC VELOCITY
EDV	-	END DIASTOLIC VELOCITY
SVR	-	SYSTOLIC VELOCITY RATIO
NASCET	-	NORTH AMERICAN SYMPTOMATIC CAROTID STENOSIS TRIAL.

ABSTRACT

Aims and Objectives:

1. To assess carotid arteries with the help of colour Doppler and B mode imaging in ischemic stroke patients.
2. To correlate cerebrovascular accidents with extracranial carotid artery status.

Materials and Methods:

Prospective study in 63 patients during the time period from October 2012 to may 2014; on patients subjected to stroke protocol/CT for stroke/TIA.

Observation is done for the following:

- Plaque characteristics as seen on real time image.
- Percentage reduction of area/diameter of carotid lumen if significant.
- Peak systolic velocity of common carotid artery.
- Peak systolic velocity of internal carotid artery.
- Systolic and diastolic velocity ratios between internal carotid artery and common carotid artery.

Results:

Thirty four out of 63 patients had carotid stenosis caused by atheromatous plaques.

Thirty out of 63(47.6%) patients had evidence of ipsilateral carotid stenosis distributed as:18 out of 30 patients (60%) had < 50% stenosis, 8 patients (26.7%) had 50-69% stenosis, and 4 patients (13.3%) had > 70% stenosis.

Homogenous hypoechoic and ulcerated plaques were more frequent in patients with large size infarcts. 19 patients (57%) had non-calcified, homogenously hypoechoic soft plaques.

Conclusion:

- Our study concludes that colour Doppler examination is noninvasive,economic, safe, reproducible and less time consuming method of demonstrating the cause of cerebrovascular insufficiency in the extracranial carotid artery system and will guide in instituting treatment modalities.
- The study also showed atherosclerosis as the most common cause of carotid artery disease leading to stroke consistent with other studies done in the past to evaluate cause of stroke.

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INTRODUCTION

Atherosclerotic disease of the carotid arteries outside the cranial cavity has long been recognized as the most common source of emboli that travel to the brain causing stroke.[1]

Arteriography has been long regarded as the gold standard diagnostic tool for carotid stenosis. It is a costly and invasive technique with potentially serious complications. The results of arteriography have not been standardized which makes comparison of results from different laboratories difficult. Duplex ultrasound is inexpensive, non-invasive and can provide functional and anatomical information about vessel stenosis and plaque morphology.[2,3]

Colour duplex flow ultrasonography has thus become the most widely used noninvasive method of assessing extracranial cerebrovascular occlusive disease because it avoids the expense and risk of routine arteriography.[4]

The principal appealing points in favour of sonography are patient comfort, lack of risk and accuracy. In contrast, angiography is invasive and expensive. Moreover contrast related effects also contribute to significant morbitdity. In symptomatic patients like those with hemisphere symptoms of TIA, carotid ultrasound may be the only diagnostic modality performed before carotid endarterectomy.[5]

Besides estimating the degree of stenosis, the biggest advantage of sonography is its ability to identify and characterize plaque and identify plaques with higher risk of embolization With the use of high resolution ultrasound, plaque can be characterized into relatively high risk groups from containing intra-plaque haemorrhage which is thought by many to the precursor of plaque ulceration.[6,7]

Colour Doppler sonographic examination consists of real time B- mode imaging with colour Doppler flow imaging with built in Doppler capabilities.

Though the prime indication of colour Doppler sonographic examination of carotid vasculature is stroke prevention, it has now-a-days ramified to evaluate various miscellaneous subjects such as suspected patients of aneurysm, dissection, chemodectoma and takayasu's arteritis which is relevant in a south-asian set up.

CLINICAL INDICATIONS FOR CEREBROVASCULAR DUPLEX. SONOGRAPHY.[8]

- Transient ischemic attacks (TIA)
- Reversible ischemic neurological deficits (RIND)
- Stroke patients with focal neurological deficit
- Mild resolving strokes in younger patients
- Atypical , non-focal symptoms which may have a vascular etiology
- High – risk patient pre-op evaluation.
- Post – Endarterectomy
- Pulsatile neck masses
- Arterial Dissection.
- Screening.

AIMS AND OBJECTIVES

1. To assess carotid arteries with the help of colour Doppler and B mode imaging in ischemic stroke patients.
2. To correlate cerebrovascular accidents with extracranial carotid artery status.

REVIEW OF LITERATURE

Medical echography resulted from the creativity and hard-working investigation by physicists, engineers and medical men. The first practical and commercialized applications of 1-D (A-scans) and 2-D (B-scans) in medicine were introduced in the 1950's. The rapid evolution of electronic gear (the solid state revolution !) and transducers led the way to the so-called vascular "Doppler device" (i.e. a blood-flow detector using the Doppler effect).

In the 1960's rapid B-scan repetition was made possible for the same reasons : "real-time" was born .

Also in the 1960's Strandness delivered pioneering work both on the interpretation of Doppler curves and the development of new equipment. He was lucky to be among a group of motivated and creative engineers at the Washington State University in Seattle.

Pulsed wave Doppler technology was successfully applied to an artery for the first time in 1971.[8]

Thanks to perseverance and engineering skill, pulsed wave technology was applied to embody the 2-D imaging and the Doppler probe into one device : duplex, a self-evident name.

The history of duplex scanning itself can be summarized as follows : in the 1970's duplex was born, in the 1980's the electronic probe and the colour-code came in and colour duplex eventually gained recognition for accuracy, especially in the detection and investigation of carotid artery disease.[9]

MIYAZAKI and KATO et al – 1965: first reported the use of Doppler ultrasound for the assessment of extra cranial carotid vasculature.

BLAISDELL et al; HAGS et al and CAROLL et al – 1965: demonstrated that 30-60% of strokes are produced by atherosclerosis disease involving extra-cranial arteries within 2 cm of carotid bifurcation.[10]

BARBER et al 1974: was first to propose the use of ultrasound for the investigation of carotid bifurcation disease.[11]

WOOD et al, DREISBACH et al and GRANT et al 1983: opined that evaluation of CAD by duplex Sonography had 92-95% accuracy compared to conventional angiography. They pointed out that the positive predictive value of the test is 97% and that is 3% false positive result.[12]

PATEL M R; KUNTZ KM et al; 1995: opined preoperative assessment of the carotid bifurcation by MRA and duplex Sonography can replace angiography.

BLUTH EI; KAY D et al 1986: characterized sonographically atherosclerotic plaque and detected hemorrhage within plaque.[13]

TAYLOR D.C; E STRANDNESS 1987 demonstrated the Doppler sensitivity of 99 % and specificity of 84 % when compared with angiography.[14]

WILLIAM D MIDDLETON.J et al 1988: stated that flow reversal is a normal phenomenon and occupied 33% of carotid bulb lumen and duration is 22% of total cardiac cycle.[15]

BLUTH E.I STARROS A.T; MARICH K.W et al 1988: recommended after multicenter trial for standardized imaging and Doppler criteria to diagnose the critical stenosis.[16]

ERICKSON.S.J; MEWISSEN.M; W.D FOLEY et al 1989: assessed stenosis of internal carotid artery using colour Doppler flow imaging and compared with angiography and correlated with angiography in 75%.[17]

ZWIEBEL AND KNIGHTON 1990: suggested that a very fresh thrombus was

virtually anechoic and very old thrombus was moderate to markedly echogenic.[19]

BLUTH E.I; SUNSHINE L. H et al 2000: evaluated power Doppler imaging as a possible exam for screening of carotid artery stenosis and found results are comparable to efficacy of mammography as a general screening method.[20]

IVAN N STAIKOV, KRASSEN et al 2002; stated that the use of duplex Sonography has a high level of sensitivity and negative predictive value for diagnosing severe caroid stenosis. In addition most stenosis can be diagnosed with a high level of specificity and positive predictive value. They found that the optimal values for PSV and EDV for diagnosis of a severe i.e >70% stenosis were =220cm/sec and = 80 cm / sec.[21]

North American symptomatic carotid stenosis trial (NASCET) is the largest multi-centric study conducted in this field. In this trial a total of 2885 patients who had a TIA or non disabling stroke (Rankin score) were included and randomized to medical therapy or medical therapy with endarterectomy. Patients with a likely cardiac source of embolism were included. Carotid stenosis was evaluated by angiography and Sonography. The mean follow up period was five years.[22]

NASCET demonstrated an average cumulative ipsilateral stroke rate at two years of 26 % for patients with 70% stenosis who were treated medically. In the group treated with the same drugs along with endarterectomy, the risk was 9 %. Thus there was 17% reduction in the surgical group.

Noor ulHadi et al studied on 100 patients with ischemic infarction on CT brain, 56 (56%) had carotid stenosis on color Doppler ultrasonography of carotid arteries. 64.3% had mild stenosis, 26.8% moderate and 8.9% severe stenosis. The prevalence of hypertension and diabetes was 59% and 44% respectively.

Niaz Ahmed Shaikh et al studied on one hundred (100) patients, sixty one (61%) were males and thirty nine (39%) were females. Thirty nine patients (39%) were found to have carotid artery stenosis, all of them on the ipsilateral side corresponding to the ischaemic lesion. Eleven (11) of these patients had stenosis on the contra lateral side as well. The presence of stenosis was significantly correlated with older age and the presence of multiple risk factors. Majority (52%) of the lesions were severe to critical as determined by Doppler peak systolic velocity. Fifty nine (59%) were non calcified.

Dr.Moazzam Ali Atif studied on 100 patients over a period of 5 months 66% of these were having cerebral infarction. Hypertension (72%), diabetes (35%), smoking (29%) abdominal obesity (20%) were common risk factors in these patients. The significant carotid atherosclerosis in acute ischemic stroke patients was 21%.

Dr. Praveen Reddy et al and F. Todua et al: stated that majority of the patients with CAD are of 5^{th} and 6^{th} decade with male predominance.

VASCULAR ANATOMY:

The extra-cranial carotid system consists of two common carotid arteries which bifurcate into right and left internal and external carotid arteries on their respective sides. The two internal carotid arteries supply most of the anterior part of cerebral hemispheres.

The blood supply for the central nervous system derives from the three great vessels arising from the aortic arch in the superior mediastinum the innominate, the left common carotid, and the left subclavian arteries. The innominate artery travels upward, Slightly posteriorly from thearch to the right of the neck for its 4-5 cm length, dividing into the right common carotid artery and the right subclavain artery at the upper border of the right sterno clavicular junction.

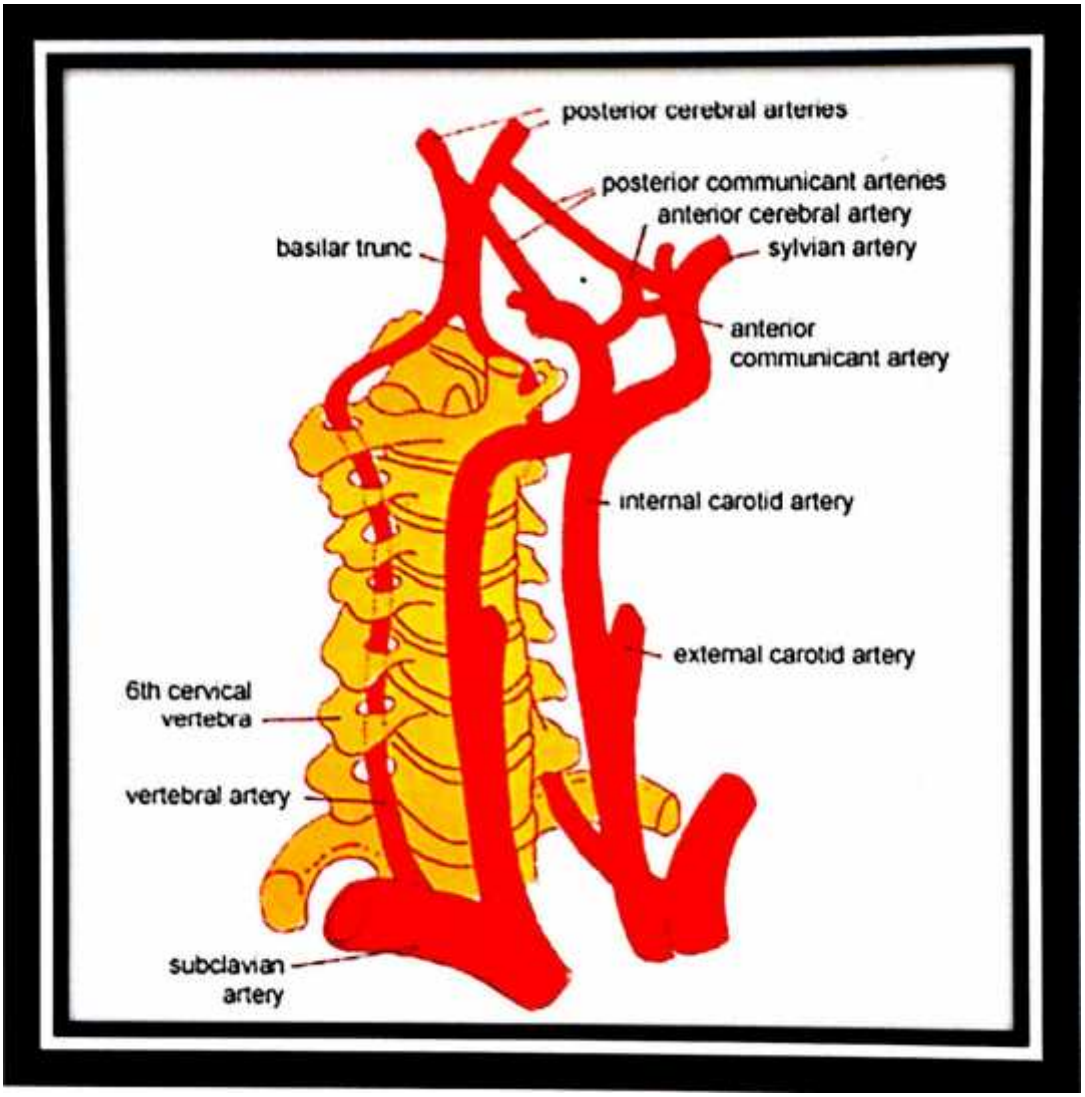


Fig.1 THE EXTRA-CRANIAL CAROTID SYSTEM

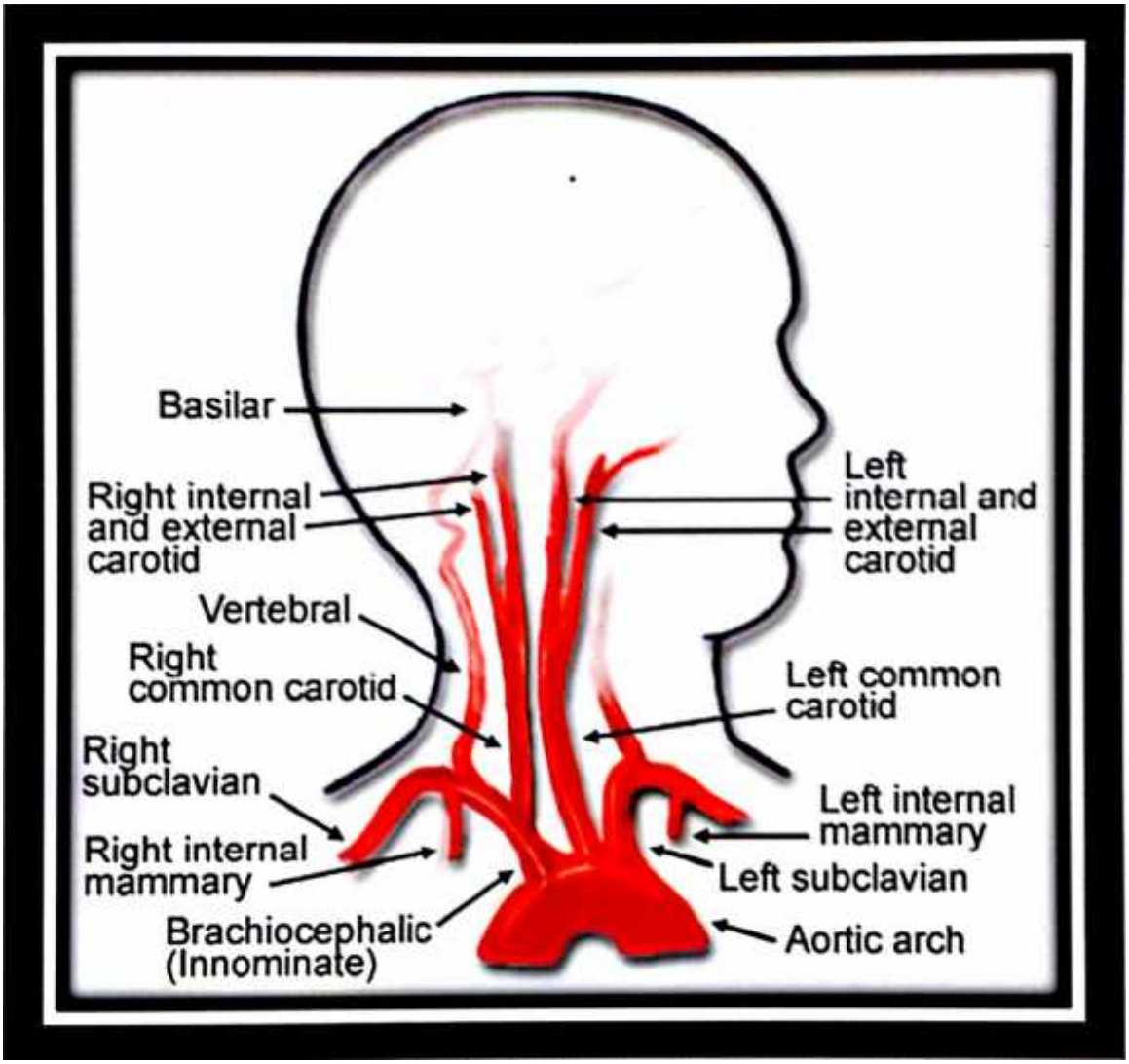


Fig.2 ORIGIN AND DIVISION OF CAROTID SYSTEM

Common Carotid artery

The left common carotid artery ascends from the arch and passes beneath the left sternoclavicular joint. Each common carotid artery divides into the internal and external carotid arteries at the level of the upper border of the thyroid cartilage.

Internal Carotid artery

The internal carotids supply most of the anterior circulation to the cerebrum. In their cervical portion, the internal carotid arteries may be relatively straight or may curve tortuously as they travel to the base of the skull. There are no branches of the internal carotid arteries in the neck.

As they proceed intracranially, the internal carotid arteries give rise to the caroticotympanic branches in the petrous bone, the meningohypophyseal branches in the cavernous sinus region, and the ophthalamic arteries immediately distal to the cavernous sinus. 8 mm beyond the cliniod process, within the duramater, the internal ceratoid arteries give rise to the posterior communicating arteries, which join with the posterior cerebral arteries. Further cephalad the internal carotid arteries divides into the middle and anterior cerebral arteries and give rise posteriorly to the anterior choroidal arteries.

External Carotid arteries

The external carotid arteries normally supply no blood to the brain. However, several of their branches can become important collateral pathways if occlusion occurs in the internal carotid or vertebral arteries.

The branches of the external carotid artery are the ascending pharyngeal, the superior thyroid, the lingual, the external maxillary, the occipital, the facial, the posterior auricular, the internal maxillary, the transverse facial and superficial temporal arteries.

The external carotid branches most vital to collateral circulation are those in communication with the ophthalmic artery and those that interconnect between the muscular branches of the occipital and vertebral arteries.[23]

STROKE:

The stroke syndrome consists of rapid development of a focal neurologic deficit that is usually localized to an area of brain supplied by a specific artery.

Common causes of ischemic stroke:

a) Atherosclerosis with superimposed thrombosis, with or without distal embolization.
b) Embolus from the heart.
c) Hypertensive arteriolar sclerosis.
d) Dissection of carotid artery, vertebral artery.
e) Prothrombotic states.
f) Vasospasm: subarachnoid haemorrhage, idiopathic.

BASIC PRINCIPLES AND DOPPLER PHYSICS

Doppler Effect

The Doppler effect is a change in the frequency of a detected wave when the source or the detector is moving. Doppler shift occurs when reflectors move relative to the transducer. The frequency of echo signals from moving reflectors is higher or lower than the frequency transmitted by the transducer, depending on whether the motion is toward or away from the transducer. The Doppler shift frequency or simply the Doppler frequency is the differences between the received and transmitted frequencies.

Doppler Equation

An Ultrasonic transducer is placed in contact with the skin surface; it transmits a beam whose frequency is f_o The received frequency f_r will differ from f_o When echoes are picked up from moving scatterers, such as red blood cells.

The Doppler frequency f_D is defined as the difference between the received and

transmitted frequencies and is given by

$f_D = f_r - f_O = \frac{2 f_o V \cos\theta}{C}$

Where C is the speed of sound

V is the flow velocity

θ is angle between in the insonating beam and the direction of flow of blood is the angle between the direction of flow and the axis of the ultrasound beam, looking towards the transducer is called the Doppler angle and strongly influences the detected Doppler frequency for a given reflector velocity.

When flow is directly towards the transducer, the angle is zero degrees and cos θ is 1. In practice the transducer beam is usually oriented to make 30-60 degree angle with the arterial lumen.

Continuous wave Doppler

The typical transducer used in continuous wave Doppler instruments contains two separate piezoelectric elements, one for transmitting and the other for receiving ultrasonic waves. A Doppler shift is generated when the moving red cells receive the ultrasound wave, and a second Doppler shift is generated when the moving cells reflect the ultrasound back to the stationary transducer.

If the flow of blood is towards the transducer, the higher frequency is received, if blood flow is away from the transducer the Doppler shift will be negative.

Thus the sign of the frequency change carries information about the direction of blood flow.

Pulsed Doppler

A pulsed Doppler instrument provides for discrimination of Doppler signals from different depths, allowing for the detection of moving interfaces and scatterers only from within a well-defined sample volume. Frequency information is usually

displayed on a pulsed Doppler image as a spectral display that detects the Doppler shift frequencies in a blood vessel as they vary with time.

Doppler colour flow imaging

Doppler colour flow imaging has been developed throughout an entire real time image, allowing visualization of blood vessels and their flow characteristics plus images of tissues surrounding the vessels. Colour Doppler presents flow information by superimposing a colour image on the grey scale real time image. This presents a real time image of both anatomy and blood flow. Detected motion is assigned a colour, usually red to flow towards the transducer and blue away from the transducer.

The Duplex Scanner

Duplex ultrasound instruments are real time B-mode scanner with built in Doppler capabilities.

In typical applications, the pulse echo B-mode image obtained with a duplex scanner is used to localize areas where flow is to be examined using Doppler.

The region of interest for Doppler studies may be selected on the B-mode image by placement of a sample volume indicator, or cursor.[24]

STROKE

The stroke syndrome consists of rapid development of a focal neurologic deficit that is usually localized to an area of brain supplied by a specific artery.

Transient ischemic attack – ischemic neurological deficit that lasts less than 24 hours.

Common causes of ischemic stroke:

1. Atherosclerosis with superimposed thrombosis, with or without distal embolization.
2. Embolus from the heart.
3. Hypertensive arteriolar sclerosis
4. Dissection of carotid artery, vertebral artery.

Diagnostic modalities in cerebrovascular insufficiency

The correct detection and quantification of carotid artery disease are of decisive impact on patient prognosis and adequate treatment.[25]

The availability of aggressive and potentially harmful treatments for acute ischaemic stroke demands fast and noninvasive examination of the intracranial vasculature.[26]

Ultrasound is the most widespread diagnostic procedure in obstructive disease of the arteries supplying the brain which is noninvasive and low cost.[27] In acute stroke ultrasound techniques offer bedside options to localize the underlying pathogenic process.[28]

Ultrasonic examination allows to detect carotid stenoses with high accuracy.[29] B mode evaluates the fine echotexture of plaque and also measures intima media thickness, an increase in which is generally considered as marker of atherosclerosis and helps to identify different subtypes of ischaemic stroke patients.[30]

Noninvasive assessment of carotid lesions can be useful in predicting the existence of silent cerebral infarction even in patients free from neurological deficits.[31]

Colour Doppler sonography suffers from disadvantages as high inter observer variability and problems with data presentation and storage.3D –color Doppler sonography showed good sensitivity and diagnostic accuracy for detection of significant stenosis of extracranial carotid arteries providing three dimensional data sets that can be acquired and reconstructed within 10 minutes giving the opportunity for complete datastorage.[32]

Magnetic resonance angiography has progressively gained clinical relevance in the evaluation of cerebrovascular disease[33] and has become powerful tool for accurate and early diagnosis of cerebral ischaemia.[34]

In computed tomography angiography with spiral CT, large parts of the vascular tree can be examined with rather small quantities of contrast agent.[34]

CT angiography has been found to be an excellent examination for the detection of carotid occlusion and categorization of stenosis in either 0-29% or >50% ranges. However CT angiography is unable to reliably distinguish between moderate (50-69%) & severe (70-90%) stenosis, which is important limitation in investigation and treatment of carotid stenosis.[35]

The European carotid surgery trial (ECST) and the North American Symptomatic carotid Endarterectomy Trial (NASCET) demonstrated that endarterectomy is highly helpful in Symptomatic high grade Stenosis (>70%).[36]

An accurate and reliable preoperative diagnostic evaluation is a indispensable prerequisite.[37]

Although digital substraction angiography is the gold standard in evaluating internal carotid arteries, it is an invasive procedure in which adverse reactions could occur due to potential complications at a rate of 0.45% to 2.6%.[38,39]

The duplex method has been recommended as the most effective means, because it is able to detect both morphological and hemodynamical changes.

Duplex ultrasound has replaced arteriography as the first choice technique for preoperative assessment of carotid arteries.[40]

It is considered the first step in the diagnostic work up of carotid atherosclerosis and evaluation of plaque conspicuity to select which plaques need to be treated with endarterectomy or percutaneous transluminal angioplasty.[41]

Many studies indicate that duplex sonography is sufficient to establish the need for surgery in symptomatic and asymptomatic patients.[42]

Carotid endarterectomy can be safely performed without preoperative angiography in cases with conclusive duplex scan findings.[43]

There was a high correlation between the Doppler ultrasound results and angiography.[44,45]

The Duplex ultrasound velocity criteria to grade the severity of carotid disease in 10% intervals is reliable and accurate.[46]

The transverse images obtained with colour B mode imaging were compared with endarterectomy specimens and was concluded that the colour B mode imaging is appropriate in determining stenosis.[47]

Also correlation with planimetric measurements of the corresponding post mortem specimens has proved that colour Doppler permits reliable detection and quantification of carotid stenosis and occlusions.[34]

Though a clear Doppler sonographic finding normal or pathologic is still a large diagnostic value, ambiguous or uncertain findings require further diagnostic work up.[48]

In cases where duplex assessment does not allow full visualization of carotid bifurcation angiography is required.[49]

Also to identify significant intracranial disease many stroke clinicians recommend angiography after screening with ultrasound before a decision regarding intervention is made.[50,51]

Magnetic resonance angiography have been developed as alternative techniques to digital substraction angiography. Magnetic resonance angiography probably gives a more precise estimate of stenosis because it provides a direct measurement of stenotic lumen[52].

DUS, CTA, and MRA all show similar accuracy in the diagnosis of symptomatic carotid stenosis. But no technique on its own is accurate enough to replace DSA. Two non invasive techniques in combination and adding a third if the first two disagree appears more accurate.[53]

It appears that the combined use of DUS, MRA and CTA can improve the diagnostic accuracy for evaluation of carotid stenosis so that application of DSA can be reduced.

Duplex features of normal carotid arteries

Features that identify the external and internal carotid arteries

Table.1 FEATURES

CRITERIA	ECA	ICA
1.Size	Usually smaller	Usually larger
2.Branches	Yes	No
3.Orientation	Proceeds anteriorly towards face	Proceeds posteriorly towards mastoids
4. Doppler	High resistance flow pattern	Low resistance flow pattern
5. Temporal tap	Waveform deflection on tap	No waveform defection.

Normal carotid wall structure

The walls consist of three distinct layers, intima, media and adventitia. The intima and adventitia produce parallel echogenic lines, with an intervening echo void that represents the media.

Normal flow characteristics

In normal arteries that are relatively straight, blood flow is laminar, meaning that the blood cells move in parallel lines. The laminar pattern may be disturbed by vessel tortuosity, kinks or branching. The most noteworthy normal flow disturbance occurs at the carotid bifurcation where a vortex is established in the bulbous portion of the common carotid artery. Peak systolic velocity in the internal carotid artery, reported mean values for normal adults range from 54 to 88 cm/ second. ICA velocity exceeding 100 cm/ second should be viewed as potentially abnormal. Peak systolic velocity in ECA is reported as 77cm/second (mean) in normal individuals, and the maximum velocity does not normally exceed 115 cm /second.

Normal carotid artery Doppler wave form

Wave forms in the common carotid artery have moderately broad systolic peaks and a moderate amount of flow throughout diastole. The internal carotid artery wave forms have broad systolic peaks and a large amount of flow throughout diastole.

The external carotid artery wave forms have a sharp systolic peaks and relatively little flow in diastole.[53]

Duplex features of abnormal carotid arteries

Atherosclerosis should be considered a systemic disease, however it tends to be segmental. The commonest sites are the coronary artery, the superficial femoral artery, the subrenal aorta and the carotid arteries at the level of bifurcation.

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. A measurement greater than 1.2 mm appears to accurately indicate the presence of plaque.

Plaque pathogenesis

Three endothelium mediated processes occur in the course of plaque formation, the migration of smooth muscle cells into the subendothelial layer, the accumulation of intracellular and extracellular lipid and the development of a collagenous matrix within the evolving plaque. Plaque has been shown to occur more frequently at regions of flow seperation and of low and oscillating shear stress. A similar hemodynamic situation with flow seperation and low oscillating shear stress is found in the internal carotid artery and in the carotid bulb. Uncomplicated or stable plaque consists largely uniform, lipid and cellular deposit covered by a subendothelial fibrous tissue cap made of smooth muscles and connective tissue. The architecture of complicated plaque is disturbed by degenerative processes including necrosis, haemorrhage, calcification, thinning or disruption of the fibrous cap, disruption of the endothelial layer and ulceration.

Plaque characterization

Several studies have reported on the correlation of ultrasonic carotid plaque morphology, cerebrovascular symptoms and intra plaque haemorrhage. These studies have suggested that ultrasonic carotid plaque morphology can be critical in producing cerebrovascular is chaemic events and may be helpful in patient selection for carotid endarterectomy.[54]

Mechanisms by which the atheromatous plaque represents a cause of ischaemia involves two factors: the embolic factor and the hemodynamic factor.[55]

Plaque rupture may play an important role in acute cerebral events by embolization.[56]

Some investigators have concluded that hypoechoic plaques and echolucent plaques are associated with an incidence of cerebrovascular symptoms that is higher than that for hyperechoic plaques.

The major predictors of risk for cerebrovascular events besides the degree of stenosis, are the progression of the degree of stenosis, irregular surface and non homogenous echographic appearance.

Plaques can be characterized has homogeneous or heterogeneous with the latter being described as a combination of hyperechoic, isoechoic and hypoechoic plaques. The homogeneous plaques consisted of uniformmly high to medium level echoes, whereas heterogeneous plaques consisted of mixed high level, medium level and low level echoes. They also concluded that homogeneous plaques were correlated with the fibrous lesion on pathological examination and heterogeneous plaques were correlated with the presence of intra plaque haemorrhage, ulceration and loose stroma containing lipids, cholesterol and proteinaceous deposits. Some investigators have classified plaques into four major sub types: [56]

Type 1- Echolucent

Type 2- Echolucent with small Echogenic areas

Type 3- Echogenic with small Echolucent areas

Type 4- Echogenic

Cerebrovascular events were mainly present in type 1 and type 2 lesions whereas type 3 and type 4 lesions were mainly asymptomatic. They also reported that type 1 and type 2 lesions were associated more frequently with intra plaque haemorrhage or ulceration.

The higher the degree of stenosis the more likely it was associated with heterogeneous plaques. Heterogeneous plaques were also associated with an incidence of cerebrovascular symptoms (TIA/stroke) that was higher than in homogeneous plaques for all grades of stenosis . [56,21]

One of the limitations of ultrasonic characterization of plaque morphology is reproducibility. Ultrasonography is usually a technologist dependent study with variation also occurring from machine to machine. [56]

Doppler evaluation of carotid arteries

There are three important areas to consider in Doppler evaluation of an arterial stenosis: the pre-stenotic region, the stenosis itself and the post stenotic region. The most important Doppler measurements are made in the stenotic zone.

The flow velocity in the stenotic lumen is elevated in proportion to the degree of luminal narrowing and this principle is the basis for Doppler stenosis assessment. The increase in stenotic zone velocity is small until the lumen diameter is reduced to about 50% of its original size. Thereafter the velocity goes up rapidly as severity of stenosis increases.

Three stenotic zone velocity measurements are key to carotid stenosis diagnosis : the peak systolic velocity, the end diastolic velocity and the systolic velocity ratio. These

measurements are called cardinal Doppler parameters. Doppler ultrasound grading of internal carotid stenosis using the two parameters of spectral analysis and internal carotid to common carotid artery peak systolic velocity ratio is well established.[57]

Peak systolic velocity

The best documented Doppler parameter for carotid stenosis is the stenotic zone peak systolic velocity. At any level of arterial narrowing , the peak systolic velocity is affected by the length of the stenosis (i.e, the longer the stenosis the lower the velocity) and by a host of physiologic factors that differ from one patient to another.

End Diastolic Velocity

With arterial stenosis of less than 50% diameter reduction, the diastolic velocity remains normal because no appreciable pressure gradient is present across the stenosis during diastole. As stenosis progress beyond 50%, a pressure gradient across the stenosis is present in diastole and diastolic velocities increase in proportion to the severity of the gradient . This parameter is quite valuable for detecting high grade carotid stenosis.

Systolic velocity ratio

The systolic velocity ratio is recommended to avoid errors caused by physiologic factors or collateralization. This ratio is the peak systolic velocity in the stenotic ICA divided by the peak systolic velocity in the ipsilateral CCA.

North American Symptomatic Carotid Endarterctomy Trial (NASCET) suggest that ratio measurements are more accurate than peak systolic velocity measurements for ICA stenosis.

Table 2

Doppler Spectrum Analysis[58]

Diameter	Peak systolic velocity	Peak End diastolic velocity	ICA/CCA peak systolic velocity ratio	ICA/CCA Peak End diastolic velocity ratio
1-39%	<110cm/sec	< 40 cm / sec	< 1.8	< 2.4
40-59 %	<130cm/sec	<40 cm/sec	< 1.8	<2.4
60-79%	> 130cm/sec	>40 cm/sec	> 1.8	>2.4
80-99 %	>250cm/sec	>100cm/sec	>3.7	>5.5

North American Symptomatic Carotid Endarterectomy Trial (NASCET) and European Carotid Endarterectomy Trial (ECET) clearly demonstrated that the long term benefits of endarterectomy were significantly greater than the medical treatment in patients with 60% or70% Internal carotid artery stenosis, whether symptomatic or asymptomatic. Second, the endarterectomy trials established that 60-70% diameter reduction as clinically significant levels of Internal carotid artery stenosis. The trials showed that endarterectomy was effective at or above these levels.[58]

CLINICAL APPLICATIONS

The clinical use of duplex scanning is divided into four areas mainly:

1. Symptomatic
2. Asymptomatic
3. Follow up of surgical patients
4. Research

1. Symptomatic patients

Patients with hemispheric symptoms including those with amaurosis fugax, transient ischemic attack (TIA) and ischemic strokes are advised cerebral angiography

as soon as possible, when otherwise healthy patients have TIA traceable to the carotid artery.

These patients have a high risk of stroke particularly in the first three months after the onset of symptoms. Only about one half will be found to have carotid stenosis of more than 50% on the symptomatic side are good candidates for carotid Endarterectomy. Non invasive carotid evaluation may increase the sense of urgency in such cases if it demonstrates critical narrowing of the artery.

In patients considered as borderline candidates for surgery because of either advanced age or a systemic medical illness such as heart failure , angiography and surgery might be reconsidered if a critical cervical carotid disease by Doppler evaluation may also be a deciding factor in taking up further cerebrovascular investigations when the diagnosis of TIA is uncertain. Ultrasound evaluation of the carotid system is rarely of the view when the clinical examination and CT scanning indicate cerebral infarction, since the discovery of even significant carotid disease is unlikely to lead to further investigations.

In patients showing good recovery from their stroke the demonstration of critical carotid stenosis may suggest the need for further investigation and for carotid Endarterectomy to prevent further strokes.

Another valuable use of duplex scanning in symptomatic patient is to determine what type of angiogram to perform.

If the duplex scan shows a high grade stenosis at the carotid bifurcation, this can be easily assessed by intra-arterial digital subtraction angiography (DSA). If the duplex scan shows minimal to no disease, the appropriate investigation is a standard angiogram. This is because in the second case the offending lesion is likely to be either very small or at some site not accessible to duplex examination.

2. Asymptomatic patients

The management of asymptomatic carotid disease remains controversial. The finding of carotid bruit has been used as a marker of carotid disease in asymptomatic patients and studies have suggested these patients are at an increased risk of TIA and strokes when compared with age matched controls.

Some investigator however feel the risks of stroke is not increased in this patients group but they are at a increased risk of death from cardiac diseases. It is therefore argued that non-operative management of asymptomatic carotid lesion is the preferred approach.

One reason why this problem is so unclear is that a cervical bruit is a poor marker of carotid stenosis. But clearly occurs in patients without carotid stenosis.

Moreover, not all patients with carotid stenosis have cervical bruits.

Taylor and Strandness found that a stenosis reducing vessel diameter by greater than 80% was associated with a 35% risk of developing symptoms of arterial occlusion within 16 months and a 46% risk at 12 months. Conversely, only 12.15% of the lesions that remained in the less than 80% category produced acomplication.

Thus, on this basis they now recommend carotid Endarterectomy to those patients with an asymptomatic carotid stenosis of greater than 80% diameter reduction if they are good operative candidates. On the other hand, it is recommended that it is safe to observe patients with stenosis less than 80%. Thus, they have identified a group of patients having asymptomatic carotid stenosis by duplex scanning who are at risk of cerebro-vascular complications.

These results in patients with asymptomatic carotid stenosis have been further substantiated by Baird who found that asymptomatic patients with greater than 75% of stenosis detected by duplex scanning went on to suffer unheralded stroke. He also

found that patients with asymptomatic carotid stenosis less than 75% had a low risk for stroke. Bogousslava et al followed 38 patients with asymptomatic carotid stenosis greater than 90% by duplex scanning for a mean period of 4 years. He found that 50% suffered an adverse neurological deficit in follow up (TIA, stroke or occlusion).

These data show that duplex scanning in patient with asymptomatic carotid disease discovers a group at high risk for adverse cerebro-vascular events. With the widespread use of this technology more and more patents with asymptomatic carotid disease will be identified. The Doppler scanner has a major role to play in the management of these patients.

3. Follow up of surgical patients:

Duplex scanning can define the natural history of the endarterectomised carotid artery. On clinical symptoms the rate of recurrence of carotid stenosis is less than 5%. However, when duplex scanning became widely used on patients following Endarterectomy, it became evident that the incidence of restenosis was much high.

Zeigler et al showed an initial restenosis rate of 36% with subsequent regression in many and a persistent restenosis rate of 19%. Majority of these lesions were asymptomatic.

These unexpected findings have been confirmed by others. Particularly interesting is the generally benign course followed by the lesions in that they tend not to progress to symptoms or occlusion and infarct often regresses in early post-operative period.

These lesions are not atherosclerotic but are related to smooth muscle proliferation in the endarterectomized vessel. Follow- up of these lesions with duplex scanning will provide information regarding the process of arterial healing and interventional strategies designed to reduce its incidence.

4. Research Duplex ultrasound is an ideal research tool.

It is non invasive, accurate and reasonably inexpensive. It will continue to have a major role in better defining the natural history of atherosclerotic lesions of the extra-cranial arteries in both symptomatic and asymptomatic patients.

High resolution imaging can better define the association between morphology and out come. It has been suggested that complex plaques noted on ultrasound have worse prognosis than simpler ones and that ulceration can be accurately defined by ultrasound. These ideas can be explored further with the use of duplex technology.

LIMITATIONS OF DOPPLER AND DOPPLER ARTIFACTS IN CAROTID DOPPLER

Doppler sonography provides the information about the direction of flow. The instrument asses the flow relative to the transducer by calculating doppler shift. Doppler Shift frequency it is displayed in two methods,

1. On a velocity spectrum flow in one direction is shown above the baseline and in opposite direction below the base line.
2. On colour flow imaging flow in one direction (generally flow towards probe) in red and the opposite flow in blue.

Frequency of the transducer and depth of location of vessel pose a significant limitation to the Doppler evaluation, in the form of aliasing.

DOPPLER ARTIFACTS

Artifacts abound in Doppler. The contributions from the machine and scanning process are frequently regarded as “artifacts” which distract from, or sometimes conceal the diagnostic information.

1. Aliasing in pulsed Doppler:

Aliasing is a production of artifactual, lower frequency signals when the sampling rate (PRF) is less than twice the Doppler signal frequency: when the PRF equals 2 x F. This is known as Nyquist Sampling rate.

Aliasing manifests on a Doppler spectral display as the Doppler spectrum wraps around the display, with high velocities being converted to reversed flow immediately at the point of aliasing and still higher velocities in the flow signal appearing as progressively lower velocities.

Methods to eliminate aliasing by;

I. Increasing the PRF, keeping it at the Nyquist limit for the maximum Doppler frequency shown on spectral scale.

II. Adjusting the spectral baseline, the line representing 'O' velocity, assigning the entire, spectral display to flow moving in just one direction.

III. Using a lower frequency transducer, which results in a lower frequency Doppler signal for a given velocity.

2. High pulse repetition frequency artifact (PRF)

High PRF causes a pulse to be emitted from transducer before the echoes caused by the structure of interest from the previous pulse have returned. When these do return, they are misinterpreted as coming from the second pulse. The result is a 'Phantom Image' of the vascular structure seen midway between the transducer and the real structure.

3. Spectral Mirror Image Artifact:

When the angle between the axis of the beam and the blood vessel is 90°, a signal is detected because of the geometric diversion of the beam, but it is displayed with a mirror image in the upper and lower spectral channels. In the colour image,

fluctuating, often random allocations of reds and blues can give the impression of flow reversal.

4. Temporal Resolution Artifact:

In this aliasing is seen at one of the colour flow, implying that Doppler shifts and hence velocities are higher in this location. In fact, this is the location at which the acquisition of Doppler data for the colour image coincided with systole. This Artifact occurs when the scan acquisition time is long compared to the duration of systole in pulsatile flow.

5. Transit Time Broadening Artifact:

When the aperture of a steered linear array is large in relation to the distance between the array and a blood vessel, there is an appreciable difference between the angle shown by the instrument between the axis of the Doppler beam and the blood vessel and the real Doppler angle which is responsible for the maximum detected Doppler shift frequency.

This effect is dependent on the aperture of the transducer and the distance between the target and the transducer. So transducers that subtend large angles will produce more severe broadening. This can be rectified by decreasing the aperture of array of Doppler.

6. Anechoic Space Artifact:

This occurs when in searching for flow, the gain has been increased to the point at which noise appears on the image. This manifests as colour flow in areas, which does not have blood vessel i.e., anechoic space. This artifact manifest itself as colour in haematomas and cysts.

7. Periodic Tissue Motion (Bruit) Artifact

Relatively slow motions of solid tissue create the flash artifact, rapid periodic movements of solid tissue create a characteristic Doppler shift that contains a predictable structure.

8. Pulse Echo Mirror Image Artifact:

This occurs in a situation where Doppler sample volume doesn't help in identifying the true location of flow is when there is a strong refractor orthogonal to the beam just distal to blood vessel. The reflection of echoes from the target back to the transducer via the reflector cause a mirror image artifact.

With Doppler, signals are detected in both colour and spectral modes from the reflected, or 'phantom"

MATERIALS AND METHODS

This study was carried out in patients with ischemic stroke diagnosed on CT/ stroke protocol from the period of Oct 2012 to May 2014.

The study was carried out on 63 patients who had undergone CT brain / stroke protocol for clinical signs of stroke or transient ischemic attack.

The data gathered from the color Doppler examination consisted of

- Peak Systolic velocity of common carotid artery
- Peak systolic velocity of internal carotid artery
- Velocity ratios between internal carotid artery and common carotid artery
- Plaque characteristics as seen on the real time image
- The presence of Spectral broadening / aliasing.

All the examination was performed with a Doppler angle of 60 degrees.

Equipment

Color Doppler examination of the carotid arteries was done using Philips HD-11 color Doppler with 7MHz linear array electronic transducer.

Examination Technique:

Patient position:

Carotid arteries were examined with patient in the supine position. Neck exposure was enhanced by tilting and rotating the head away from the side being examined, and ipsilateral shoulder was dropped as far as possible. The examiner was seated on the right side of the patient.

Transducer Position

The postero-lateral and far postero-lateral transducer positions are used to examine the carotid arteries in long axis (longitudinal). Short axis (transverse) views of the carotid arteries are obtained from an anterior, lateral or posterolateral approach.

The Examination Sequence

Step 1.0

The first step begins with a longitudinal survey of the cervical carotid arteries, with the transducer in a lateral position. The common carotid artery is identified at the clavicle, and the transducer is moved cephalad along the artery until the carotid bifurcation is seen. The location of the bifurcation is then ascertained by shifting back and forth between the internal and external carotid branches. The identity of the internal and external carotid arteries must be confirmed.

The internal carotid artery is followed as cephalad as possible using aposterolateral transducer position. The location of plaque and major points of obstruction are noted during survey examinations.

Step 2

Each abnormal area is scrutinized for the extent of plaque formation, internal plaque characterization and degree of luminal narrowing is noted (from colour Doppler and spectral Doppler information). The detailed examination of atheromatous lesions is initially conducted from a lateral and postero-lateral transducer position.

Step 3

The carotid arteries are re-examined from an anterior transducer position, which is roughly at right angles to the other longitudinal positions. Re evaluate plaque and obstruction from this position to enhance the 3-D perspective of atheromatous lesion.

Step 4

When the longitudinal examination is completed, the carotid vessels should be studied from transverse position, begining from the clavicle moving cephalad while the carotid arteries are observed on the Colour Doppler image.

At the carotid bifurcation, the identity of the internal and external carotid branches should be confirmed. The location of plaque and stenosis (common, external or internal

carotid segments) should be reaffirmed. Visual or Doppler assessment of stenosis severity are done.

PROTOCOL FOR CAROTID DUPLEX EXAMINATION

1. Longitudinal Survery

Lateral transducer position

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

Posterolateral transducer position

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

1. Longitudinal evaluation of pathology

Lateral or posterolateral transducer position.

Document extent of plaque deposition.

Document surface and internal characteristics of plaque.

Measure peak-systolic and end-diastolic velocities and compare with proximal CCA velocities. Note degree of post stenotic flow disturbance.

Anterior transducer position

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

TRANSVERSE EXAMINATION

- Begin at the clavicle and move cephalad.
- Identify the carotid bifurcation, ECA, ICA.
- Localize plaque and areas of obstruction to the CCA, ECA, or ICA.
- Document surface and internal characteristics of plaque.
- Measure residual lumen size and percent diameter reduction.

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

RESULTS

The study was carried out in patients with ischemic stroke on CT/ stroke protocol referred to department of radiodiagnosis.

A total of 63 patients were examined by colour Doppler sonography and of these 34 showed carotid artery disease. A detailed work up of these 34 patients was done, their clinical history and laboratory data was recorded.

Among 34 patients studied 25 patients were males and 9 were females. The highest number of patients with carotid artery disease in our study was found to be of age group 41 to 60 years which were 52 %. 30 patients had unilateral involvement while 4 patients had bilateral involvement of bilateral carotid vasculature.

The results are tabulated and percentages calculated with depiction in the form of pie diagrams and graphs for easy understanding.

TABLE 3: AGE AND SEX DISTRIBUTION

AGE in years	MALE	FEMALE	TOTAL
21 TO 30	2	0	2
31 TO 40	4	1	5
41 TO 50	7	4	11
51 TO 60	6	1	7
61 TO 70	3	2	5
71 TO 80	4	0	4
TOTAL	25	9	34

Graph 1: SEX WISE DISTRIBUTION

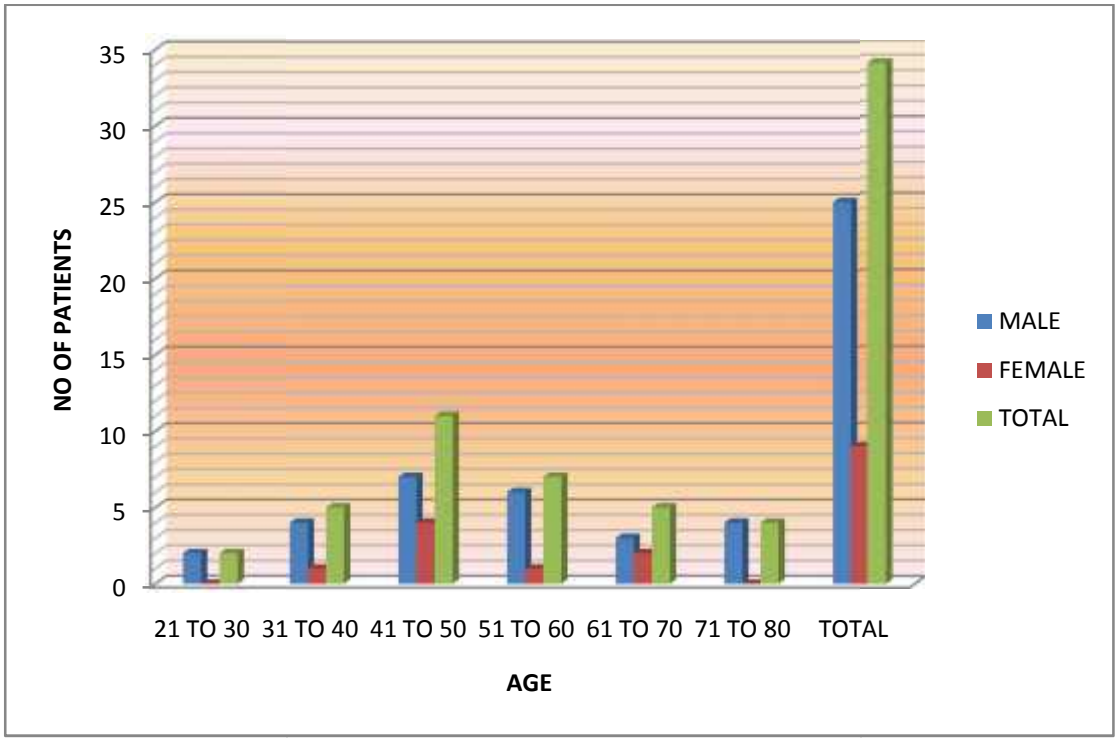


TABLE 4: SIDE OF INVOLVMENT.

SIDE OF INVOLVMENT	NO OF PATIENTS
RIGHT SIDE ONLY	19
LEFT SIDE ONLY	11
BILATERAL	4
TOTAL	34

GRAPH 2. SIDE OF INVOLVEMENT

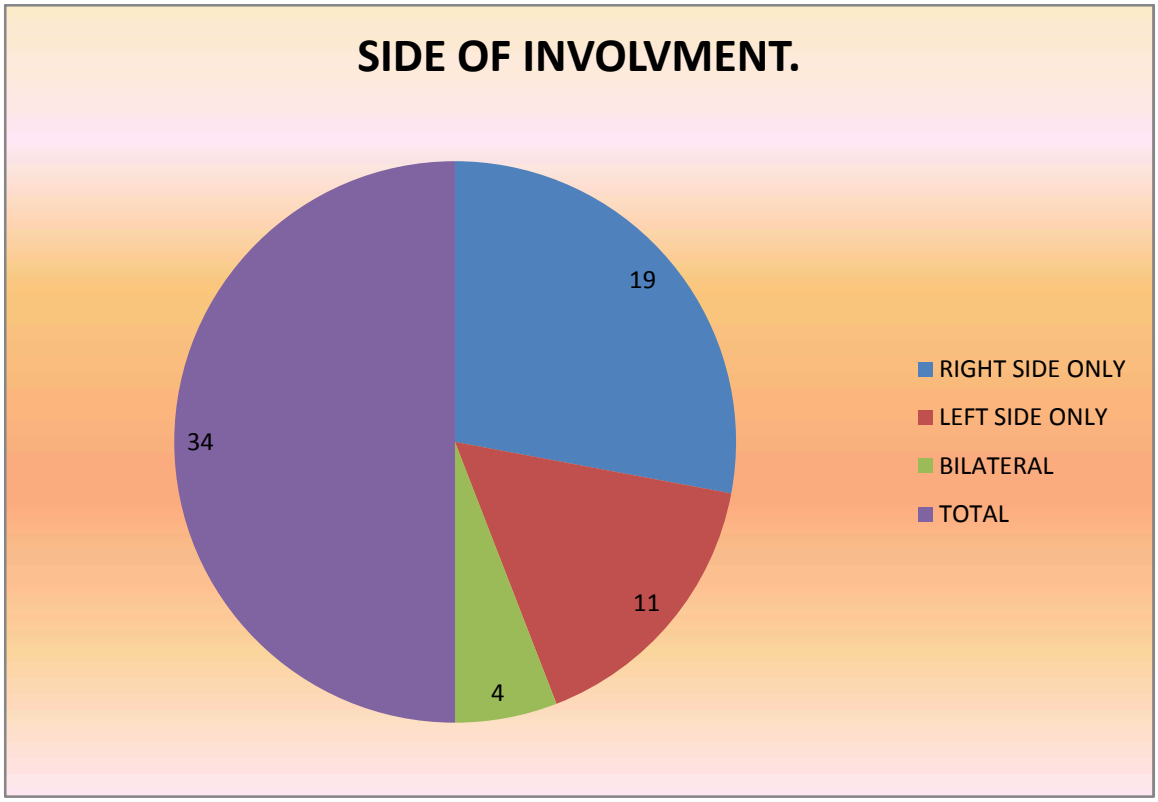
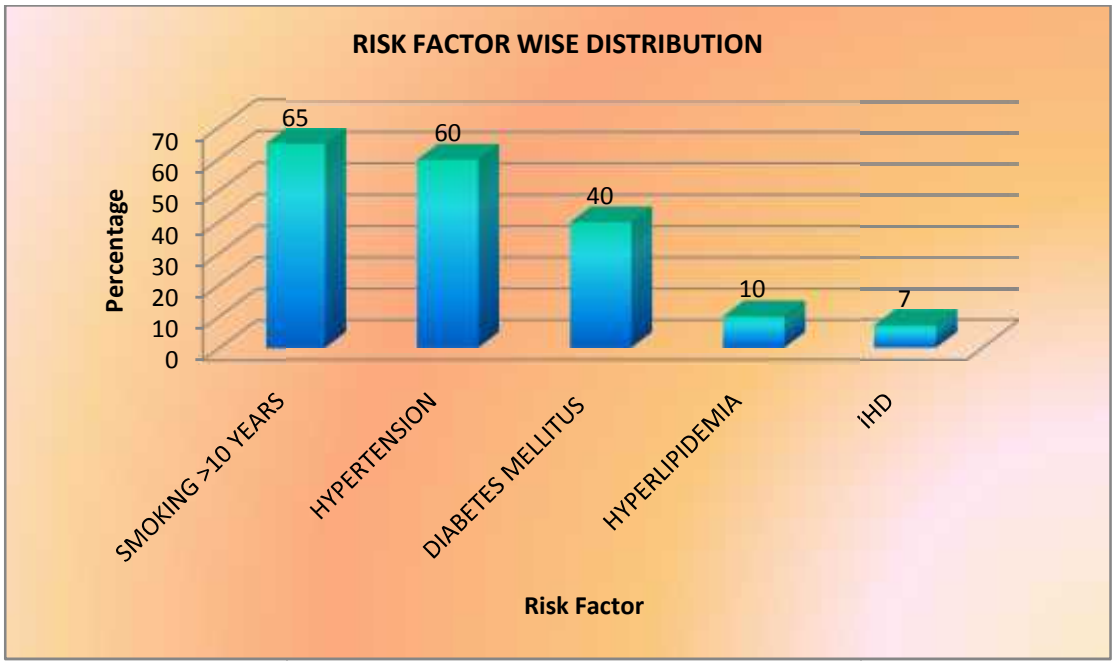


TABLE5: RISK FACTOR WISE DISTRIBUTION

RISK FACTOR	SYMPTOMATIC PATIENTS (N=30)	PERCENTAGE
SMOKING >10 YEARS	19	65
HYPERTENSION	18	60
DIABETES MELLITUS	12	40
HYPERLIPIDEMIA	3	10
IHD	2	7

GRAPH 3: RISK FACTOR WISE DISTRIBUTION



Among the symptomatic patients smoking was the most common risk factor (65%) followed by hypertension (60%), diabetes mellitus (40 %) , hyperlipidemiia (10%) and ihd (7 %).

SITES OF INVOLVEMENT:

30 out of the 63 patients studied had unilateral disease, 4 had bilateral disease. Therefore a total of 38 vessels were showing disease. Many of the patients showed plaques in CCA along with plaques in ICA. Bilateral involvement of ICA was seen in 2 patients and bilateral involvement of CCA/Bulb was seen in 2 patients.

TABLE 6: SITES OF INVOLVEMENT

SITE OF INVOLVEMENT	NO OF PATIENTS
Right common carotid artery	7
Right internal carotid artery	12
Left common carotid artery	4
Left internal carotid artery	7
Bilateral	4

GRAPH 4: SITES OF INVOLVEMENT

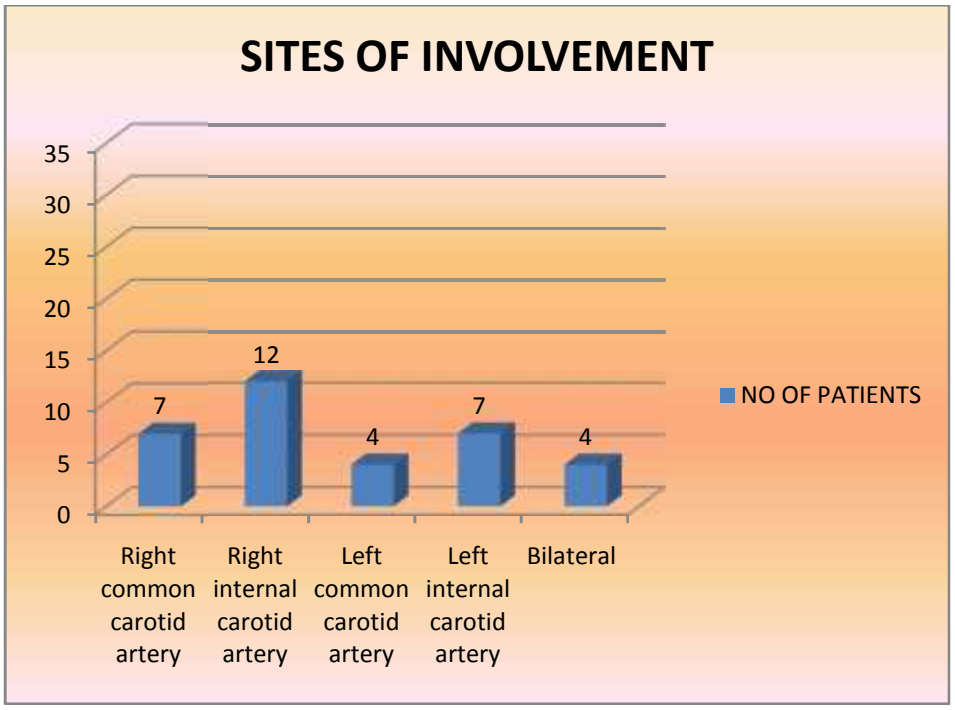
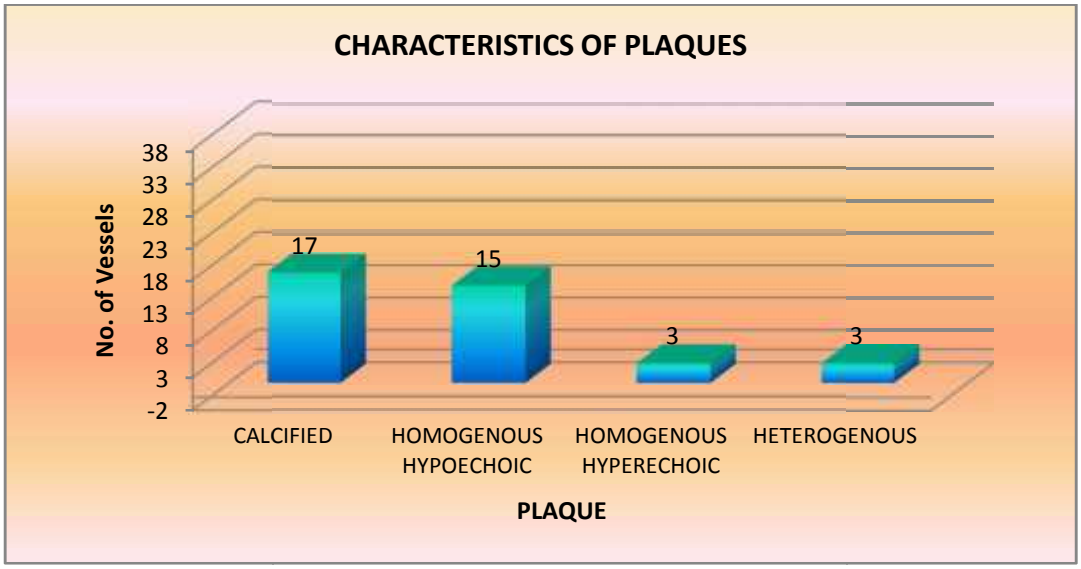


TABLE 7: CHARACTERISTICS OF PLAQUES

Plaque Echotexture	No. of Vessels
CALCIFIED	17
HOMOGENOUS HYPOECHOIC	15
HOMOGENOUS HYPERECHOIC	3
HETEROGENOUS	3
TOTAL	38

GRAPH 5 CHARACTERISTICS OF PLAQUES



On the right side, 11 were calcified 9 homogenously hypoechoic, on left side 6 were calcified and 6 were homogenously hypoechoic. homogenously hypoechoic plaques were associated with larger sized infarcts. no definite intraplaque haemorrhage was identified in this particular study.

Out of the 63 patients studied, 37 patients had right sided infarct 23 patients had left sided infarct and bilateral involvement was seen in 3 patients.

TABLE 8: PEAK SYSTOLIC VELOCITIES OF ICA:

PSV IN CM/S	% OF STENOSIS	NO OF VESSELS
<110	<40%	16
150-200	>60%	14
>210	>70%	6
TOTAL BLOCK	OCCLUSION	2
TOTAL		38

GRAPH 6 : PEAK SYSTOLIC VELOCITIES OF ICA

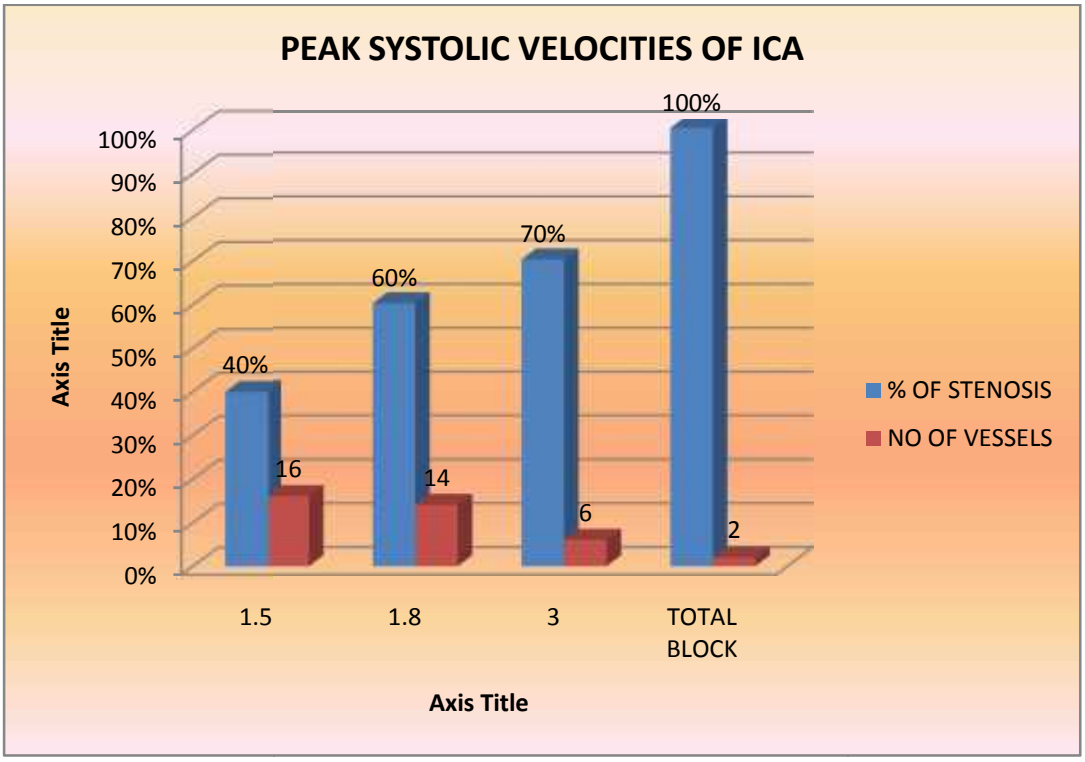


TABLE 9: PSV RATIO

PSV RATIO	% OF STENOSIS	NO OF VESSELS
<1.5	<40%	16
>1.8	>60%	14
>3	>70%	6
TOTAL BLOCK	OCCLUSION	2
TOTAL		38

GRAPH 7:PEAK SYSTOLIC VELOCITY RATIO

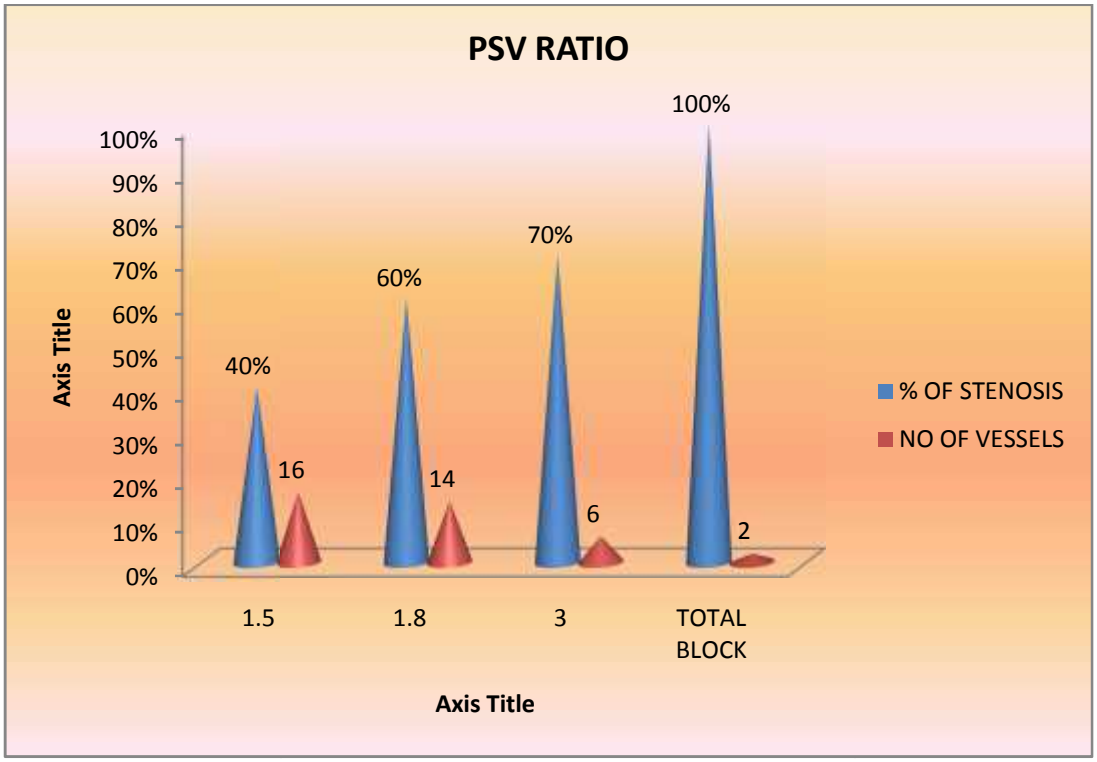


TABLE 10: END DIASTOLIC VELOCITIES OF ICA:

EDV IN CM/S	% OF STENOSIS	NO OF VESSELS
<40	<40%	16
40 TO 70	>60%	14
>70	>70%	6
TOTAL BLOCK	OCCLUSION	2
TOTAL		38

GRAPH 8: END DIASTOLIC VELOCITIES OF ICA

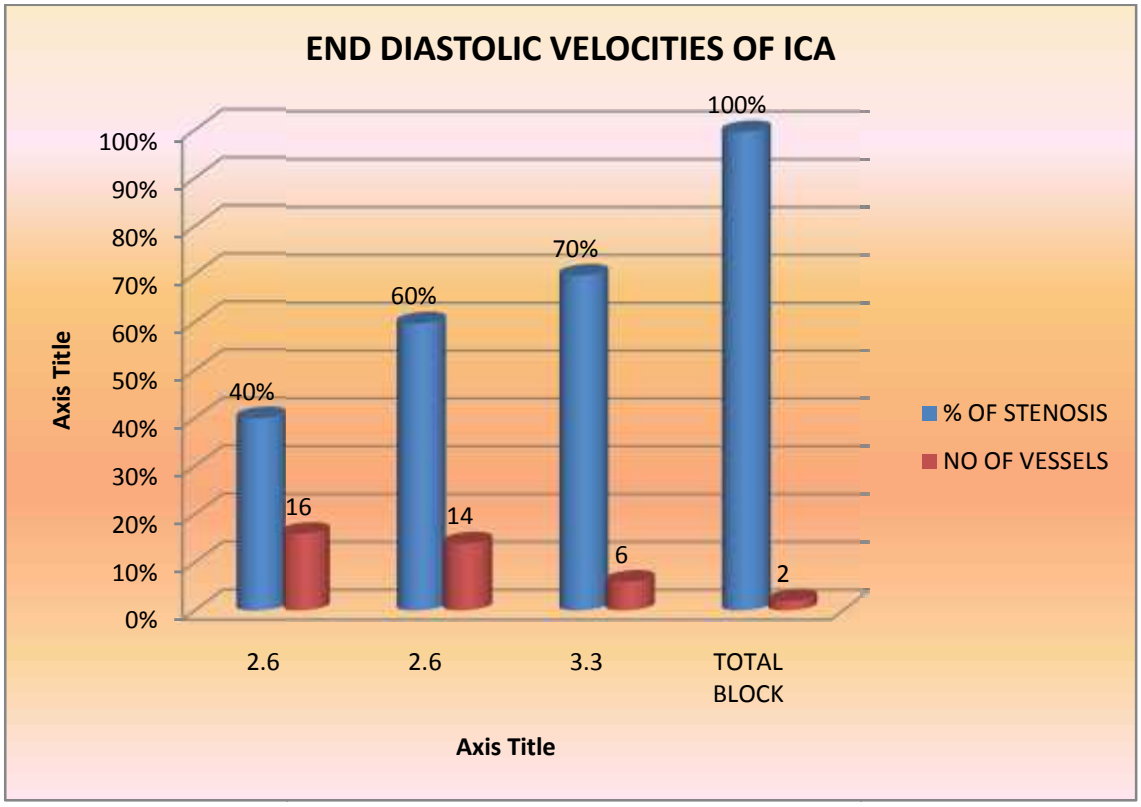


TABLE 11: RATIO OF END DIASTOLIC VELCITIES

RATIO OF EDV	% OF STENOSIS	NO OF VESSELS
<2.6	<40%	16
>2.6	>60%	14
>3.3	>70%	6
TOTAL BLOCK	OCCLUSION	2
TOTAL		38

GRAPH 9: RATIO OF END DIASTOLIC VELOCITIES

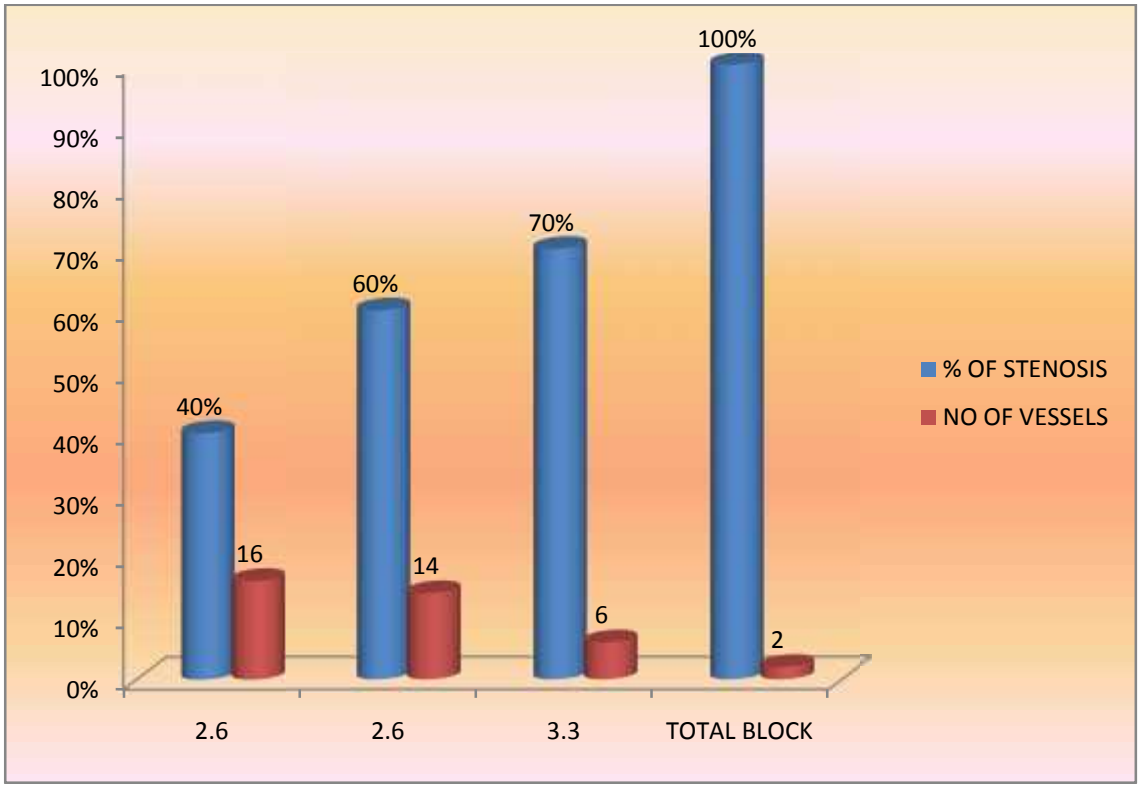


TABLE 12: ANALYSIS OF COLOUR FLOW PATTERN:

COLOUR FLOW PATTERN	NO OF VESSELS
ALIASING	19
TURBULENCE	11
REVERSAL OF FLOW	6
NO COLOUR SIGNAL	2
TOTAL	38

GRAPH 10: ANALYSIS OF COLOUR FLOW PATTERN

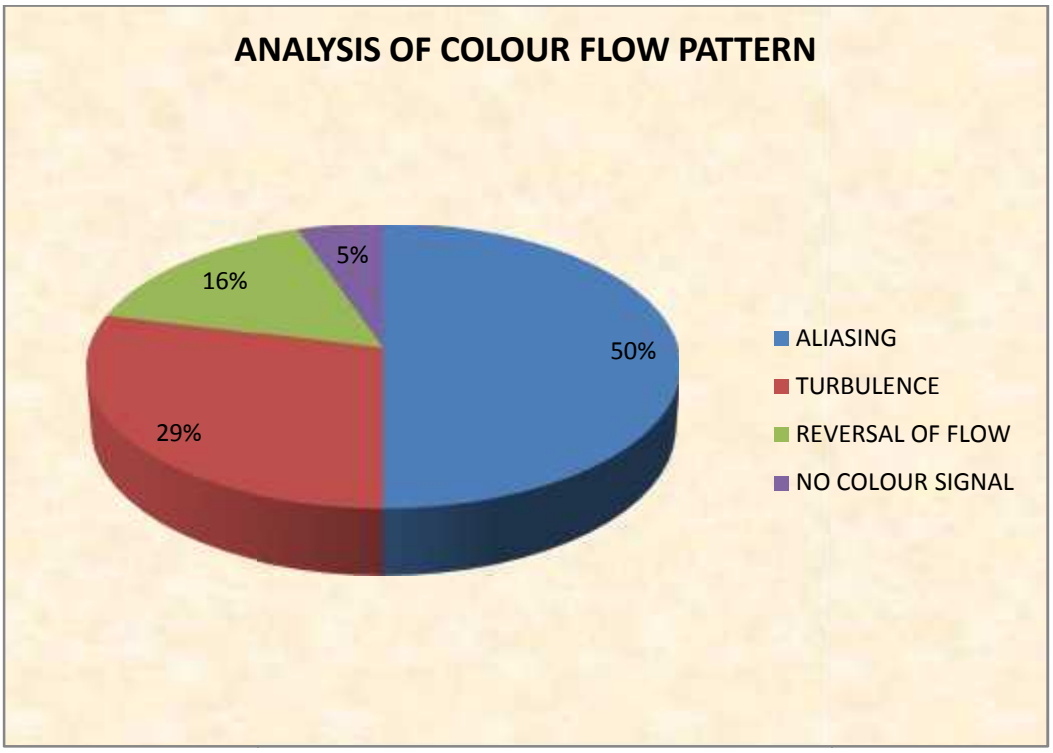
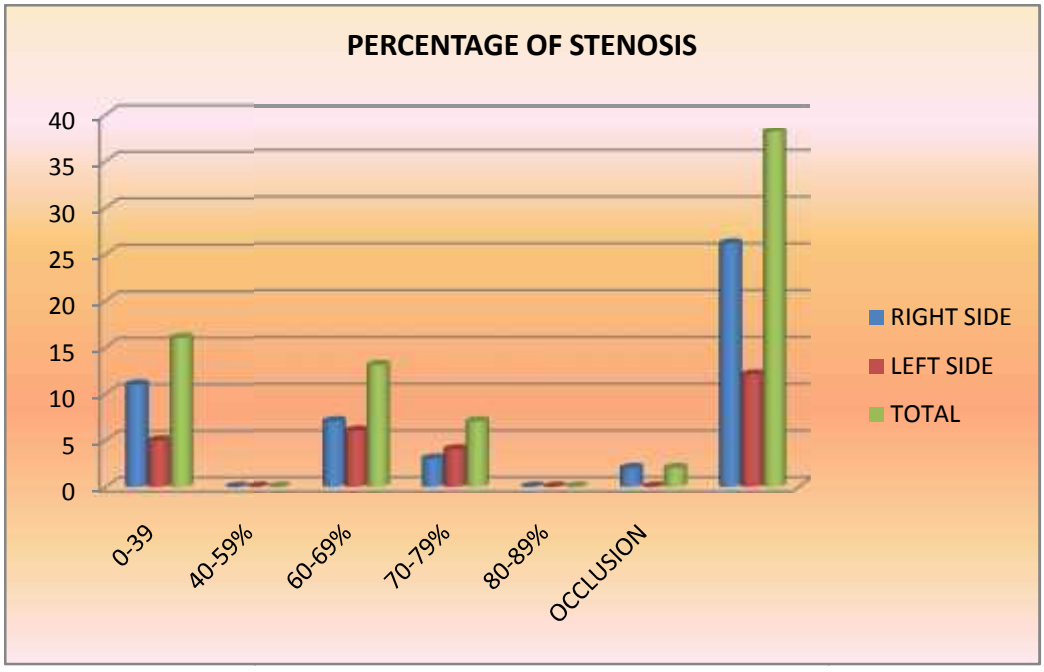


TABLE 13: PERCENTAGE OF STENOSIS:

% OF STENOSIS	RIGHT SIDE	LEFT SIDE	TOTAL
0-39	11	5	16
40-59%	-	-	-
60-69%	7	6	13
70-79%	3	4	7
80-89%	-	0	-
OCCLUSION	2	0	2
TOTAL	26	12	38

GRAPH 11 : PERCENTAGE OF STENOSIS



DISCUSSION

Cerebrovascular disease is the third leading cause of death after heart disease and cancer in the developed countries. About 30-60% of strokes are caused by atherosclerotic disease involving the extracranial carotid arteries usually within 2cms of carotid bifurcation.

Angiography is gold standard but invasive and expensive and involves significant risk to the patients. Sonography is unique among vascular imaging procedures in that it can assess plaque composition. Sonographically detected plaque characteristics may have prognostic value and may be useful for selection of medical and surgical therapy.

The present study was done to evaluate the extracranial carotid arterial system in the population who presented with ischemic stroke.

Out of the 34 patients who showed carotid artery disease, 21 patients had right sided stroke, 11 patients had left sided stroke. Bilateral involvement was seen in 2 patients.

Out of these, 16 patients had right MCA territory infarct, 9 had Left MCA territory infarct. 5 patients had right ACA territory infarct, 1 patient had left ACA infarct, one patient had left cerebellar infarct, 2 patients had bilateral infarcts

Palomaki H et. al studied the risk factors for cervical atherosclerosis in patients with ischemic stroke and transient ischemic attack and found that incidence of stroke increases after 60 years of age.[11] The highest number of stroke patients in our study were found in the age group of 41-60 years which was 29% (19/63).

Iemolo F et al. in his study showed that only 2.5% of stroke victims were females. In this study 82% of the patients (52/63) were males and only 18% were females (11/63). The risk factors that cause plaque formation and stroke were evaluated. Carlene Lawes et

al in their studies had studied 188000 patients with hypertension out of which 6800 had stroke events

In this study out of the 63 patients 18 patients had variable degree of hypertension out of which 7 had significant stenosis. Iadecola et al had proved that control of blood pressure leads to a substantially lower risk of stroke.[58]

Toshifumi Mannami et al confirmed a positive relationship between smoking and risk of stroke. They estimated that 22% of stroke was attributable to smoking. 19 of the patients in our study had history of smoking. Of them 8 had significant stenosis. .Diabetes mellitus is another risk factor for atherosclerosis. Lindberg Pertu and Roine Risto in their study had observed that two thirds of all ischemic stroke types on admission had diabetes mellitus. In this study 12 patients had diabetes mellitus of which 3 had significant stenosis. Schulz U. G. R, Flossmann E and Rothwell studied family history of stroke and found that 23% of stroke patients had positive family history.

The patients in our study were ruled out from cardiac problem, which will interfere in the velocity profiles of the carotid system. A diminished cardiac output will reduce both systolic and diastolic velocities.

In the ultrasound literature different authors say that one or another of the 3 major Doppler parameters that is peak systolic velocity, end diastolic velocity or systolic velocity ratio is the most accurate predictor of clinically significant ICA stenosis. Initially peak systolic velocities were defined for predicting the amount of stenosis, which are not very well defined due to physiological variability and obstructive lesions. Because a ratio compensates for patient to patient physiological variability and also compensate for instrument variability peak systolic velocity ratio has been considered best for assessing stenosis as proved by Zwiebel William J in his studies.[59]

North American symptomatic carotid endarterectomy trial (NASCET) and European Carotid endarterectomy trial clearly demonstrated that the long term benefits of

endarterectomy were significantly greater than medical treatment in patients with 60% or 70% internal carotid artery stenosis, whether symptomatic or asymptomatic. Second, the endarterectomy trials established 60-70% diameter reduction as clinically significant levels of ICA stenosis[58].

Arbeille Ph defined a peak systolic velocity ratio of greater than 1.8as an indicator of 60% or greater and a ratio of 3.7 as an indicator of more than 80% diameter stenosis.[59]

In this study the peak systolic velocity ratio of ICA / CCA is taken to define the percentage of significant stenosis. Out of 21 patients with significant stenosis (>60%), 11 (52%) patients had significant block on the right side, 9 (42%) had significant stenosis on the left side and bilateral significant block in 1 (0.04%) patients.

On the right side, 6 patients had 60-79% stenosis, 3 patients had 70-99% stenosis and 2 had total block. On the left side 7 patients had 60-79% stenosis, 3 had 70-99% stenosis.

The commonest cause for obstruction is due to the atheromatous plaque. Schulte Altedorneburg G et al found steno occlusive carotid lesion in 64% of the patients studied. He also confirmed his findings by postmortem studies.[60]

In our study 34 (54%) patients had plaque in the carotid system. 26 patients had plaque on the right side, 12 patients had plaque on the left side and 4 patients had bilateral involvement.

Zwiebel W J, found that the carotid bifurcation was commonly involved by the atherosclerotic plaque followed by the origin of carotid arteries.

In our study internal carotid artery was found to be the commonest site affected by the plaque. In the right internal carotid artery plaques were found in 14 patients and in 9 patients on the left. Plaques in the common carotid artery were also seen on the right side in 9 patients and 6 on the left side.

Aburahma Ali F, Wulu John T & Crotty Brad. have confirmed that's often plaques

and nonhomogeneous plaques are more positively correlated with symptoms than with any degree of stenosis and were the cause of adverse neurological events[56].

In this study 15 patients had homogenously hypoechoic plaques. On the right side., 11 patients had homogenously hypoechoic plaque. On the left side 4 patients had homogenously hypoechoic plaques.

No histopathological correlation was done in our study because surgery was not done in any of the above patients. These patients were managed conservatively.

CONCLUSION

- Our study concludes that colour Doppler examination is noninvasive,economic, safe, reproducible and less time consuming method of demonstrating the cause of cerebrovascular insufficiency in the extracranial carotid artery system and will guide in instituting treatment modalities.
- The study also showed atherosclerosis as the most common cause of carotid artery disease leading to stroke consistent with other studies done in the past to evaluate cause of stroke.

SUMMARY

63 patients underwent colour Doppler examination of the carotid arteries in **BLDEA SHREE B M PATIL MEDICAL COLLEGE, BIJAPUR**

- The highest incidence of stroke was found in the age group of 41-60 years with male population commonly affected.
- The various risk factors included hypertension, diabetes mellitus, smoking and family history.
- Out of 63 patients 22 patients showed significant stenosis (>60%stenosis) as determined by the real time imaging, the peak systolic velocity ratio between the internal carotid artery and common carotid artery (ICA / CCA), spectral broadening and the colour flow pattern.
- Atherosclerotic plaques were seen in 34 (54%) patients.

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ANNEXURE-I

Institutional Ethical Committee
BIJAPUR-586 103.
OUTWARD
No. CH6/12
Date. 19/10/12
B.L.D.E. U's Shri B.M. Patil Medical College

B.L.D.E. UNIVERSITY'S
SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR-586 103
INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 18-10-2012 *at* 3-30pm *to scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis has been accorded Ethical Clearance.*

Title "Evaluation of carotid osteries in Stroke patients using color doppler imaging"

— x — x —

Name of P.G. student Dr. Sachin G. Shatagar
Radiodiagnosis

Name of Guide/Co-investigator Dr R.C. Pattanashetti
Prof & HOD. Radiodiagnosis

DR.TEJASWINI. VALLABHA
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
BLDEU'S, SHRI.B.M.PATIL
MEDICAL COLLEGE, BIJAPUR.

Following documents were placed before E.C. for Scrutinization
1) Copy of Synopsis/Research project.
2) Copy of informed consent form
3) Any other relevant documents.

CONSENT BY PARTICIPANT

The whole study and its procedure has been well explained in the language I can understand best.

I hereby consent voluntarily to participate as a study subject in **"EVALUATION OF CAROTID ARTERIES IN STROKE PATIENTS USING COLOR DOPPLER IMAGING."**

(Signature/Thumb Print of Patient)

Full name of the patient.....

Signature of Candidate.....

Date: .....

Place: .....

SAMPLE INFORMED CONSENT FORM

B.L.D.E.U's SHRI B.M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, BIJAPUR – 586103, KARNATAKA

TITLE OF THE PROJECT:	EVALUATION OF CAROTID ARTERIES IN STROKE PATIENTS USING COLOR DOPPLER IMAGING
PRINCIPAL INVESTEGATOR:	**DR. SACHIN G SHATAGAR.** DEPARTMENT OF RADIODIAGNOSIS EMAIL: sachinshatagar@gmail.com.
PG GUIDE:	**DR.R C PATTANSHETTI** M.D PROFESSOR AND HEAD DEPARTMENT OF RADIODIAGNOSIS B.L.D.E.U's SHRI B.M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, BIJAPUR – 586103

PURPOSE OF RESEARCH:

Role of computed tomography in evaluation of sonographically detected focal lesions in liver.

PROCEDURE:

I have been explained that I will be subjected to a Computed tomography scan and Doppler.

RISKS AND DISCOMFORTS:

I understand that my/my wards participation in this study, there will be risk of radiation exposure.

BENEFITS:

I understand that my/my wards participation in this study will help in finding out the role of carotid Doppler in ischemic stroke detected by computed tomography.

CONFIDENTIALITY:

I understand that medical information produced by this study will become a part of this Hospital records and will be subjected to the confidentiality and privacy regulation of this hospital. Information of a sensitive, personal nature will not be a part of the medical records, but will be stored in the investigator's research file and identified only by a code number. The code key connecting name to numbers will be kept in a separate secure location.

If the data are used for publication in the medical literature or for teaching purpose, no names will be used and other identifiers such as photographs and audio or video tapes will be used only with my special written permission. I understand that I may see the photograph and videotapes and hear audiotapes before giving this permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at any time. **Dr. SACHIN** is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of this study, which might influence my continued participation.

If during this study, or later, I wish to discuss my participation in or concerns regarding this study with a person not directly involved, I am aware that the social

worker of the hospital is available to talk with me. And that a copy of this consent form will be given to me for careful reading.

REFUSAL OR WITHDRAWL OF PARTICIPATION:

I understand that my participation is voluntary and I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital.

I also understand that **Dr. SACHIN** will terminate my participation in this study at any time after he has explained the reasons for doing so and has helped arrange for my continued care by my own physician or therapist, if this is appropriate.

INJURY STATEMENT:

I understand that in the unlikely event of injury to me/my ward, resulting directly to my participation in this study, if such injury were reported promptly, then medical treatment would be available to me, but no further compensation will be provided.

I understand that by my agreement to participate in this study, I am not waiving any of my legal rights.

I have explained to _____ the purpose of this research, the procedures required and the possible risks and benefits, to the best of my ability in patient's own language.

Date: Dr. R C PATTANSHETTI Dr. SACHIN

(Guide) (Investigator)

PROFORMA OF THE STUDY:

CASE SHEET PROFORMA

S. No.

Name Age/Sex

Address

Occupation

COLOUR DOPPLER FINDINGS

	RIGHT		LEFT	
	CCA	ICA	CCA	ICA
PSV (cms/sec)				
EDV (cms/sec)				
ICA/CCA ratio				
STENOSIS (percentage)				
PLAQUE MORPHOLOGY (core- calcified, non calcified),(surface – smooth, irregular)				

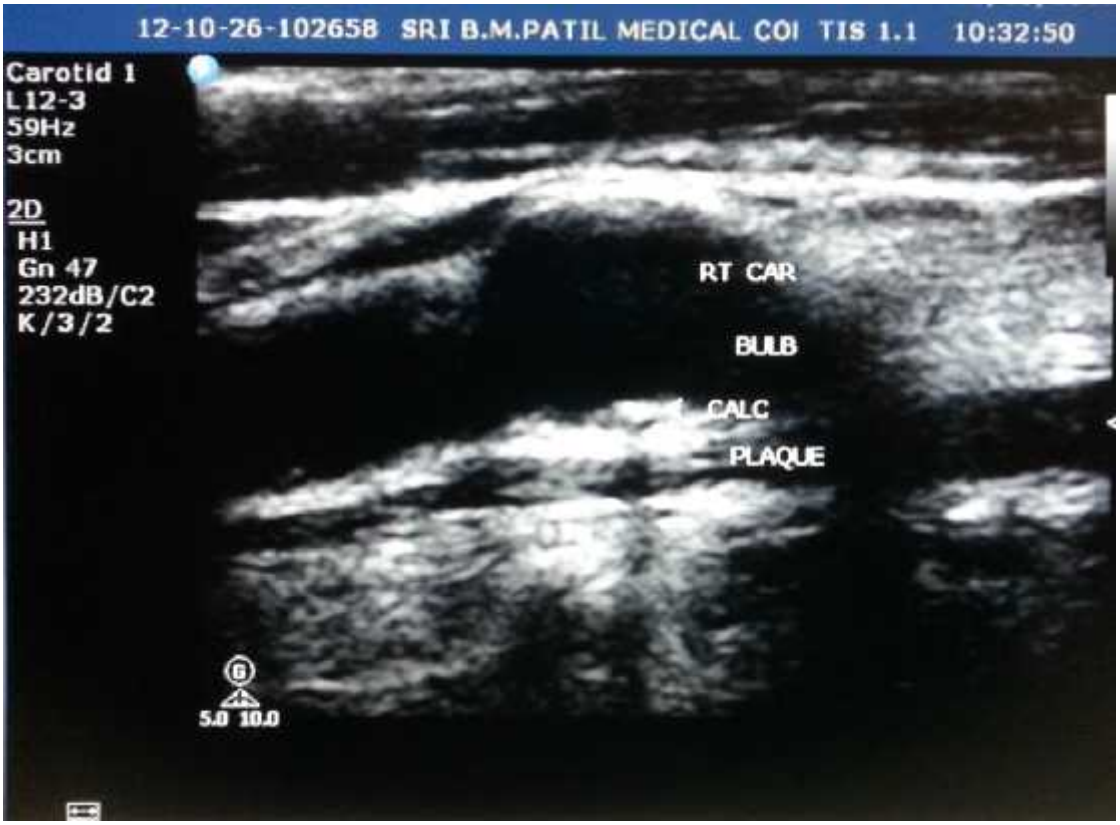
KEY TO MASTER CHART

Rt	-	Right
Lt	-	Left
CCA	-	Common carotid artery
ICA	-	Internal Carotid artery
PSV	-	Peak systolic velocity
EDV	-	End diastolic velocity
MCA	-	Middle cerebral artery
ACA	-	Anterior cerebral artery
B/L	–	Bilateral.
HS	–	Haemodynamically significant.

MASTER CHART

Sl No.	Name	Age	Sex	CT findings	RT. CCA	Rt. ICA	Lt. CCA	Lt. ICA	IMT	plaque echotexture	aliasing	FINAL DIAGNOSIS
1	Hampangouda	50	M	Lt. MCA infarct	soft plaque causing 36.4% narrowing	-	65 % plaque with irregular surface	-	0.4	Homogenously hypoechoic	+	HS Soft plaque in lt CCA
2	Dareppa	55	M	Rt. MCA infarct	-	62% plaque in proximal segment	-	-	1	calcified	+	HS plaque in rt ICA
3	Gurusidda	39	M	Rt. MCA infarct	-	38.4% plaque in proximal segment	-	-	0.5	Homogenously hypoechoic	-	soft plaque in Rt ICA - no HS stenosis
4	Bhimabai	48	F	Lt. MCA infarct	-	-	-	soft plaque causing 40% stenosis	0.5	Homogenously hypoechoic	-	Soft plaque in lt ICA
5	Jamil	42	M	Lt. MCA infarct	-	-	-	70 % plaque in proximal segment	0.8	calcified	-	Stable in lt. ICA - no HS stenosis
6	Anand	55	M	Lt. MCA infarct	-	-	<40 % plaque in mid segment	-	0.6	calcified	-	Small plaque in lt CCA
7	Murgeppa	64	M	Rt. MCA infarct	61% plaque at bulb	-	-	-	1.2	calcified	-	calcified plaque in lt. ICA
8	Chinnakka	69	F	Rt. ACA infarct	-	63% Plaque at proximal segment	-	-	1	Homogenously hypoechoic	+	Small plaque in rt ICA
9	Sharanu	34	M	Rt. MCA infarct	-	<40% plaque in proximal and mid segments	-	-	0.4	calcified plaque	-	Stable in Rt. ICA - no HS stenosis
10	Mahadevi Ingalgi	35	F	Rt. MCA infarct	soft plaque causing <40 % narrowing	-	-	-	0.8	Homogenously hypoechoic	-	Soft plaque in rt ccA - no HS stenosis
11	Digambar	68	M	Rt. MCA infarct	-	-	-	64.2% Plaque along posterior wall	2	Homogenously hypoechoic	+	HS Soft plaque in lt ICA
12	Ramesh ballolli	60	M	Rt. MCA infarct	-	18 x 10 mm sized soft plaque causing <40% stenosis	-	-	0.9	homogenously hypoechoic	-	Soft plaque in Rt. CCA
13	Mohammad	58	M	Rt ACA infarct	<40% Plaque in carotid bulb	-	-	-	0.7	homogenously hypoechoic	-	Soft plaque in rt CCA - no HS stenosis
14	Vittal	48	M	Rt. MCA infarct	-	echogenic thrombus occluding entire lumen	-	-	1.4	Echogenic	No flow	complete occlusion of right ICA
15	Kasbegouda	59	M	Rt. MCA infarct	-	long segment plaque causing <40% stenosis	-	-	0.6	homogenously hypoechoic	-	Soft plaque in Rt. CCA
16	Kamalabai	42	F	Rt. MCA infarct	68 % plaque in distal segment	-	-	-	1	calcified	+	Partly calcfied plaque in rt. CCA
17	ningangouda	75	M	Bilateral infarct	-	62% plaque in	-	75% Plaque in	1.3	bilateral calcified	+	Bilateral partly calcified

						proximal ICA		proximal ICA				HS plaques
18	Girimalla	44	M	Rt. MCA infarct	-	66% plaque in proximal segment	-		1.2	calcified	+	Partly calcfied plaque in lt. ICA
19	Sharada	45	F	Lt. MCA infarct	-	-	-	65.8% plaque in proximal segment	1.2	calcified	+	Partly calcified plaque in lt ICA
20	Peerappa	45	F	Rt. MCA infarct	-	72% plaque in proximal segment	-	-	1.4	heterogenous	+	HS plaque in rt ICA
21	Shrikanth	26	M	Rt. MCA infarct	<40% plaque in distal segement extending into bulb	-	-	-	0.5	Homogenously hypoechoic		Soft plaque in rt CCA - no HS stenosis
22	Rafeeq	50	M	Lt. MCA infarct	-	-	67% plaque in distal segment extending into carotid bulb	-	1	Homogenously hypoechoic	+	Soft plaque in lt. CCA
23	Mutujabee	50	M	Rt. ACA infarct	-	25 x 10 mm soft plaque causing <40 % stenosis	-	-	1	homogenosly hypoechoic	+	Soft plaque in rt. ICA
24	Pandappa	37	M	Lt. MCA infarct	-	-	-	78% plaque in proximal segment	1.3	calcified	+	hemodynamically significant lt. ICA plaque
25	Parvati	66	F	Rt. MCA infarct	-	76% plaque with irregular surface	-	<40% plaque	1.5	calcified	+	Bilateral partly calcified plaques
26	Ishwarappa	70	M	Rt. MCA infarct	-	Echogenic thrombus occluding entire lumen	-	-	1.7	Echogenic	No flow	plaque in rt ICA causing complete occlusion
27	Mr. Udapudi	79	M	left cerebellar infarct	-		-	small calcified plaque in prox. ICA	0.8	calcified	-	Small calcified plaque in Lt. ICA
28	Safeena	54	F	Rt. ACA infarct	-	78% plaque in proximal segment	-	-	1.3	calcified	+	calcified plaque in Rt. CCA
29	Revanasidda	72	M	Rt. MCA infarct	64% plaque in distal segement extending into bulb	-	-	-	1.4	partly calcified, B/L heterogenous	+	Bilateral partly calcified plaques
30	Bhimangouda	77	M	Bilateral infarct	<40% plaque in distal segement extending into bulb	-	66% plaque in the mid segment	-	1.5	homogenous hypoechoic	+	Soft plaque in lt. CCA
31	shivayogappa	47	M	Lt. MCA infarct	-	-	76% plaque in distal segment extending into carotid bulb	-	1.2	homogenously hyperchoic	+	Echogenic plaque in lt cca
32	Naveen Arakeri	28	M	Lt. ACA infarct	-	-	<40 Plaque in distal segment	-	0.3	Homogenously hypoechoic	-	Soft plaque in lt CCA - no HS stenosis
33	Sadashiv	53	M	Rt. ACA infarct	40% plaque at bulb		-	-	1.2	calcified	-	Calcified plaque in rt. CCA
34	Thimappa	40	M	Lt. MCA infarct	-	-	-	67% plaque in proximal part and bulb	1.4	Homogenously hypoechoic	+	Soft plaque in lt ICA - no HS stenosis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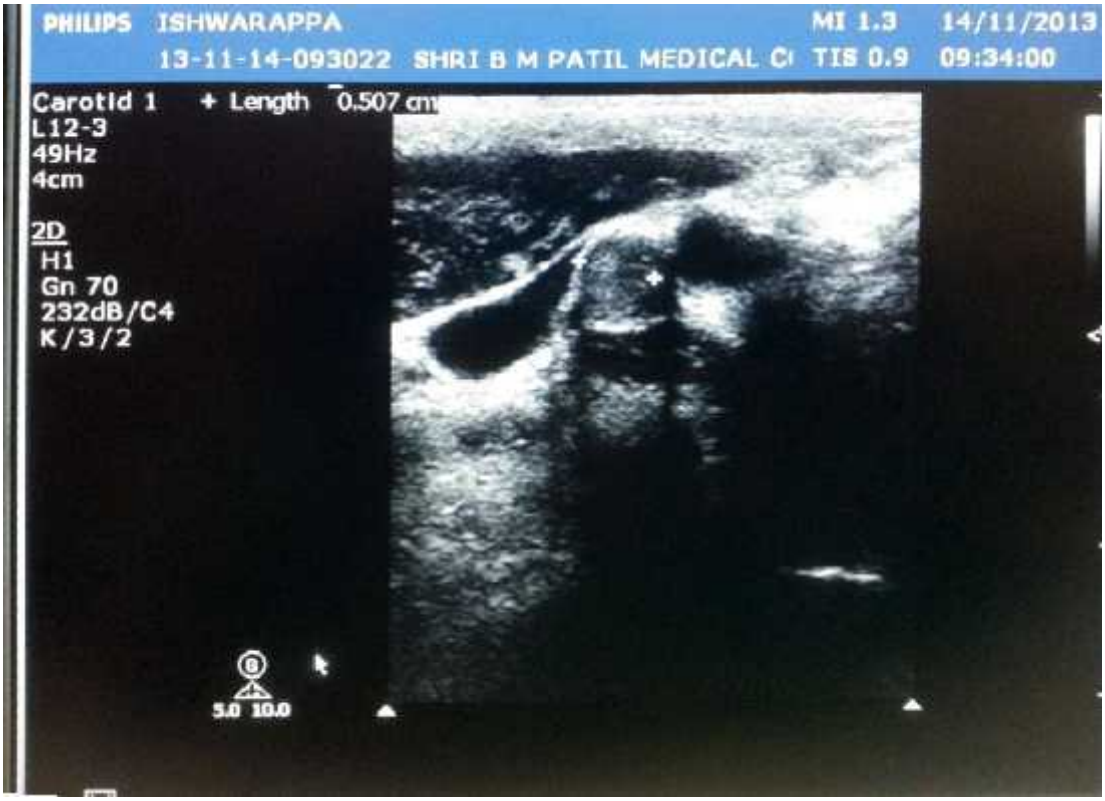
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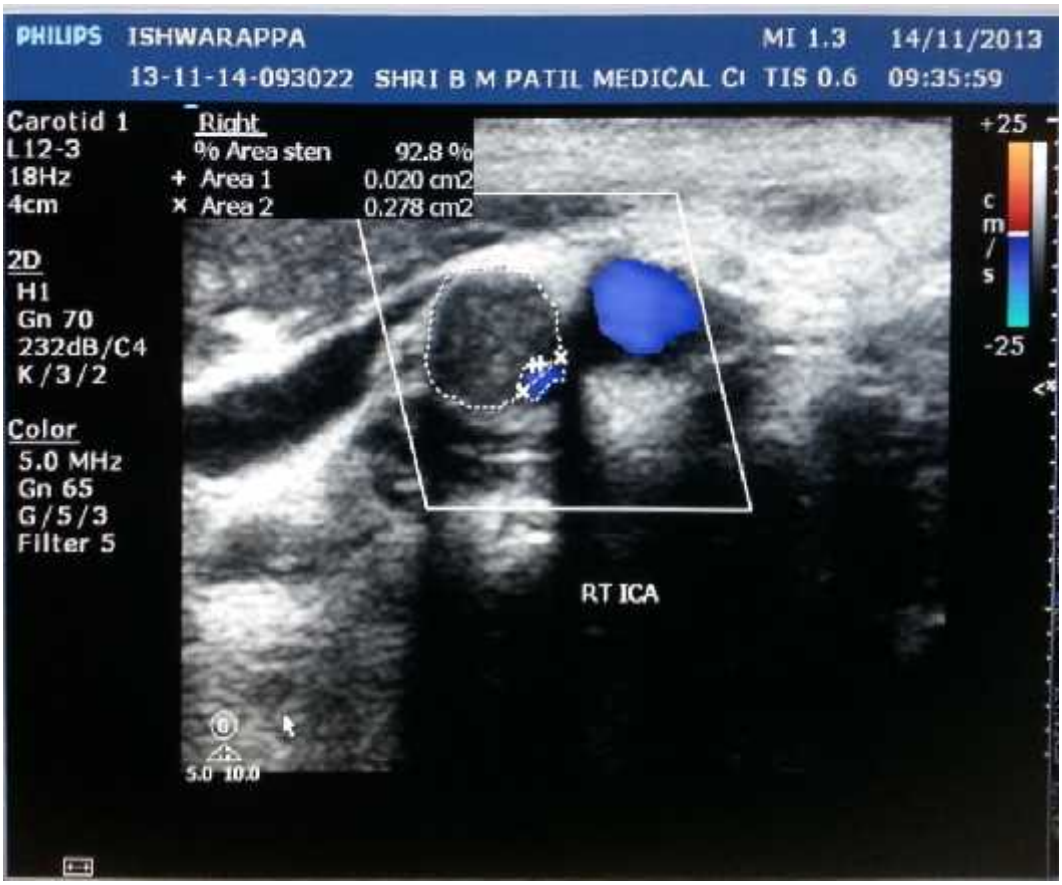
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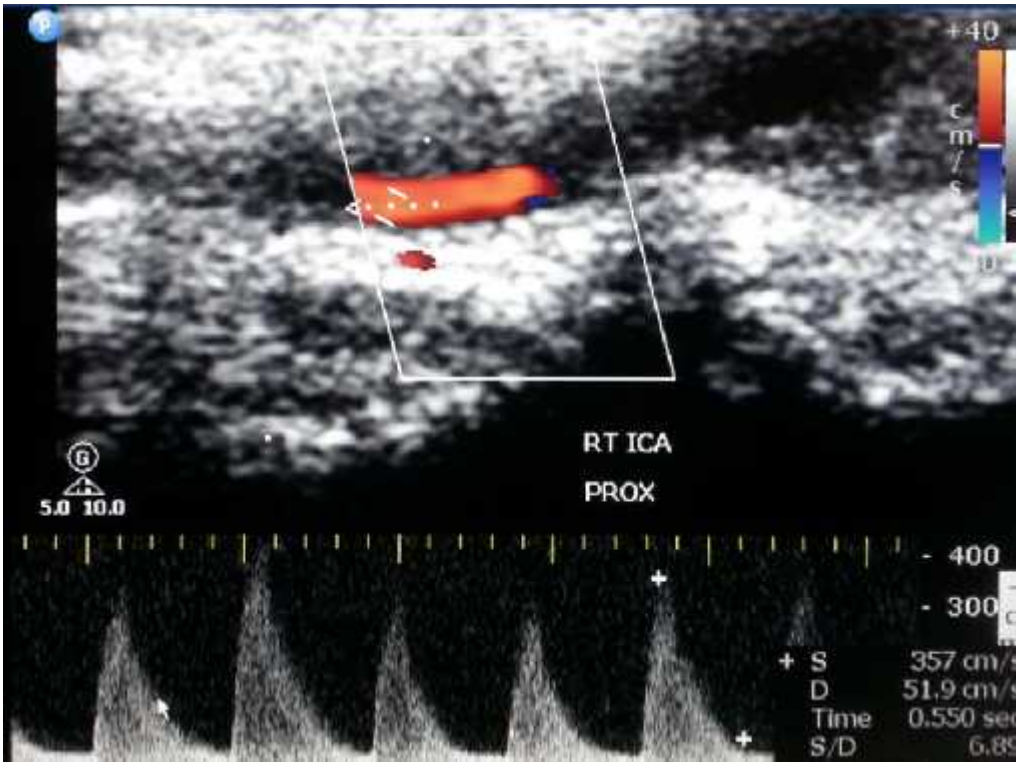
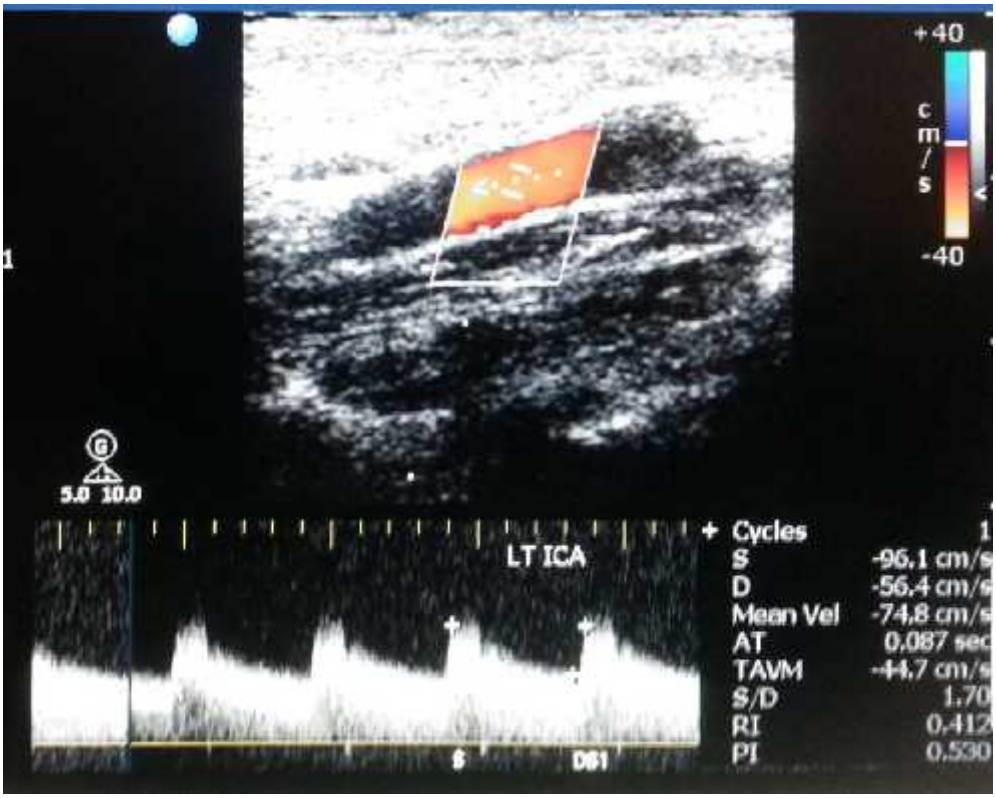
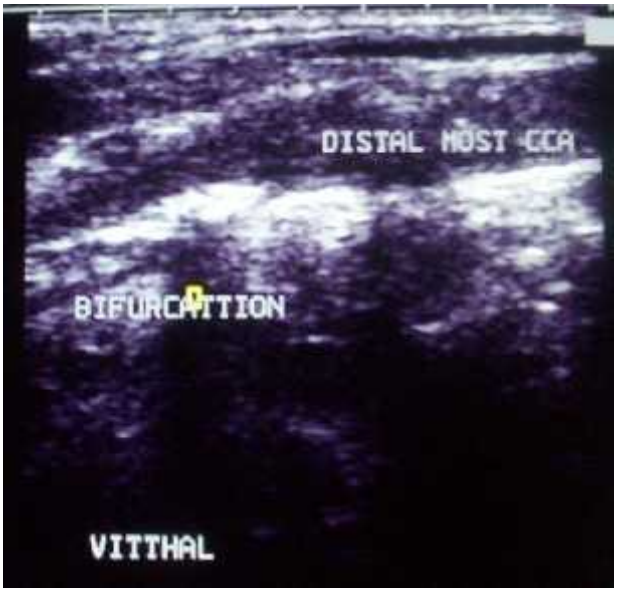
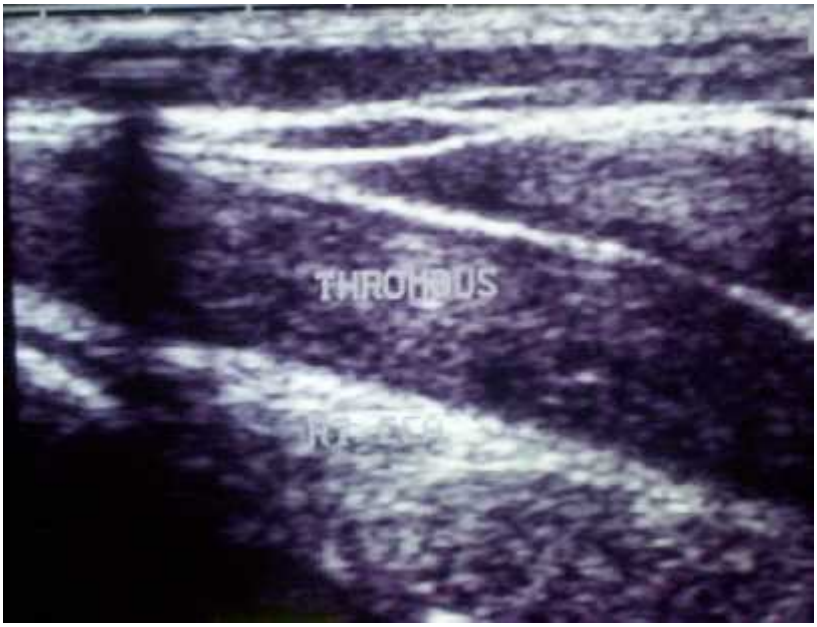
MI 1.3
14/11/2013
13-11-14-093022 SHRI B M PATIL MEDICAL C
TIS 0.6
09:35:59
Carotid 1
L12-3
18Hz
4cm
2D
H1
Gn 70
232dB/C4
K/3/2
Color
5.0 MHz
Gn 65
G/5/3
Filter 5
Right
% Area sten 92.8 %
+ Area 1 0.020 cm2
× Area 2 0.278 cm2
+25
cm/s
-25
RT ICA
5.0 10.0


+40
cm/s
RT ICA
PROX
5.0 10.0
- 400
- 300
+ S 357 cm/
D 51.9 cm/
Time 0.550 se
S/D 6.8


+40
cm/s
-40
5.0 10.0
LT ICA
Cycles
S
D
Mean Vel
AT
TAVM
S/D
RI
PI


DISTAL MOST CCA
BIFURCATION
VITTHAL


THROMBUS