ASN 2021 VIRTUAL ANNUAL MEETING

Practice Updates Neurosonology: Transcranial Doppler

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Disclosures

- Honorarium and travel compensation for participation in continuing medical education courses offered by Society of Critical Care Medicine and Neurocritical Care Society

- No other relevant disclosures

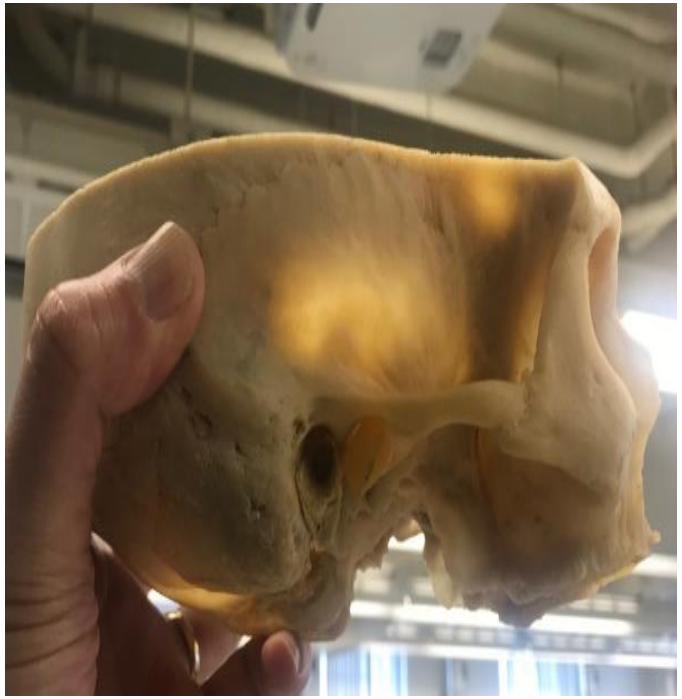
Course Objectives:

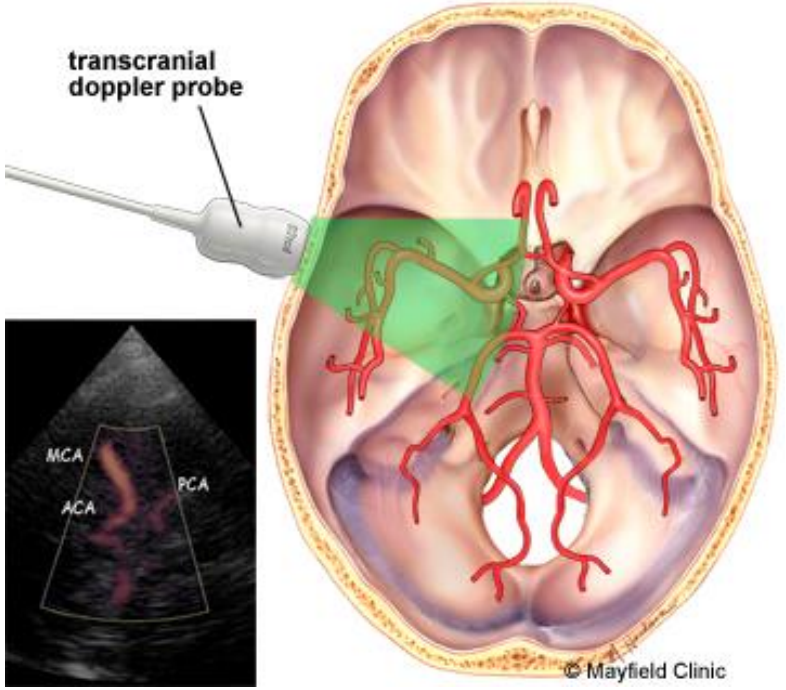
1.Explain physics of transcranial Doppler ultrasonography

2. Review the clinical indications and usefulness of TCD and apply contemporary protocols and practices in common neurovascular disorders

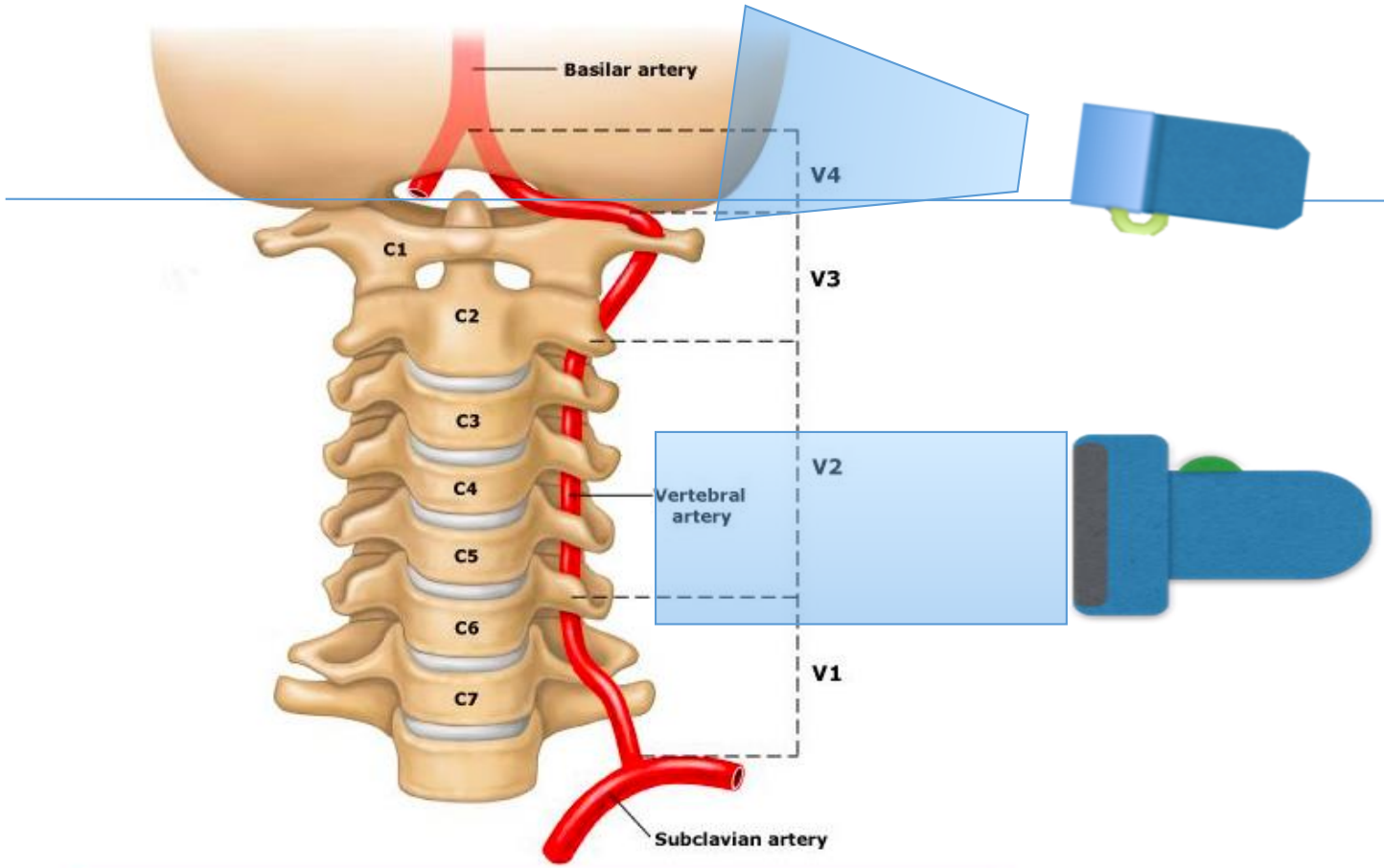
3. Discuss the coding and billing for neurosonology such as the appropriate CPT and Medicare and local carriers' coverages

Physics of Transcranial Doppler Ultrasonography





Segments of the vertebral artery



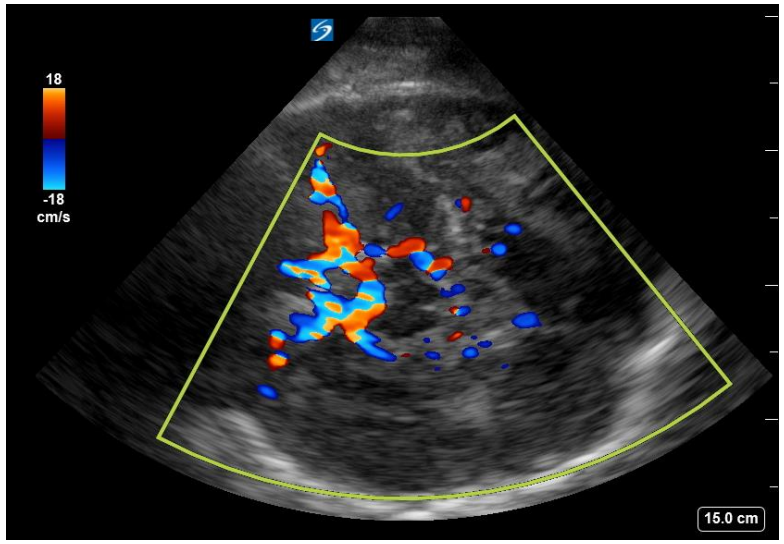
The vertebral artery is divided into four anatomic segments:

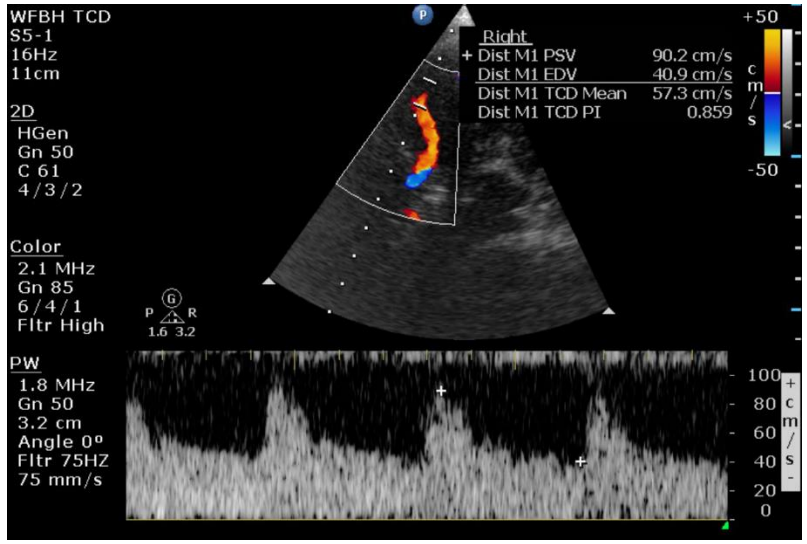
- V1 – Origin of the vessel to the foramina of the sixth cervical (C6) transverse process
- V2 – Intraforaminal segment from the sixth to the second cervical vertebral body (C6 to C2)
- V3 – From the second cervical (C2) foramina to the base of the skull
- V4 – Intracerebral segment of the vertebral artery. The vertebral arteries merge to form the basilar artery and are intradural.

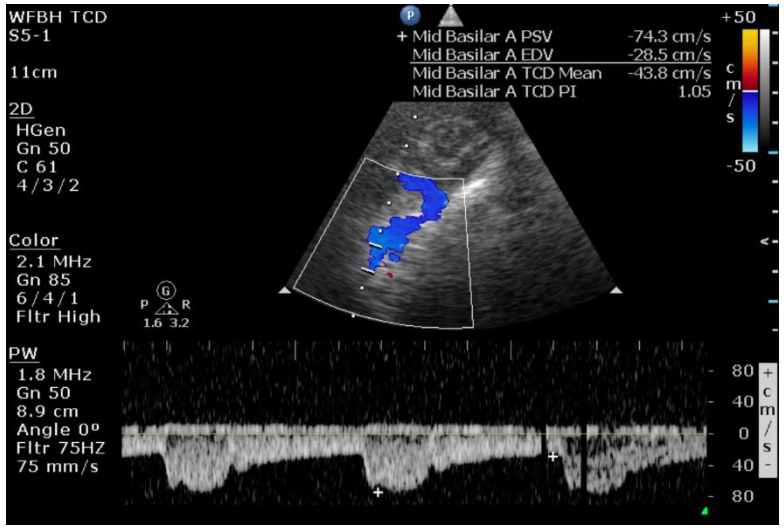
Phased Array
Low Frequency

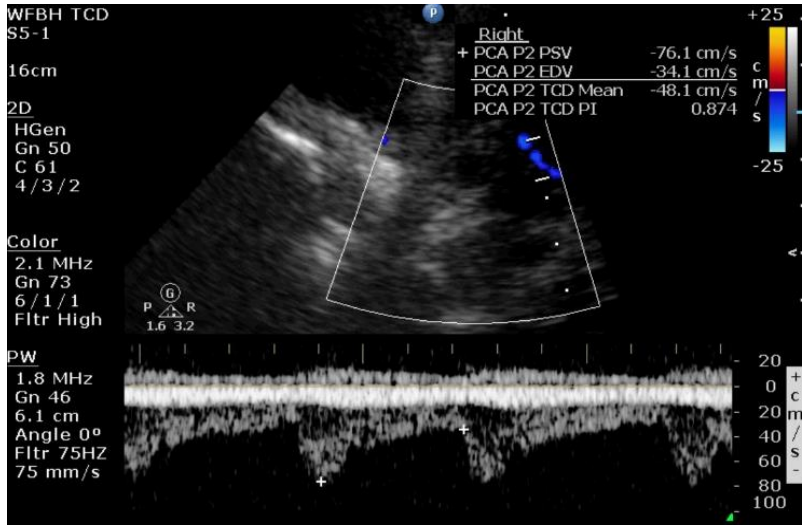
Linear
High Frequency

NeuroUltrasound



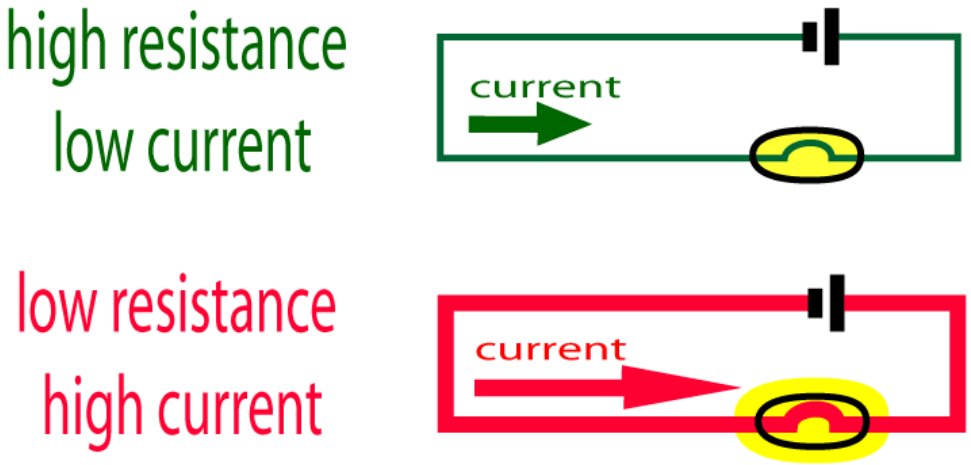






Spectral Doppler Key Parameters

- Vital organs like brain, heart, liver, placenta and kidney all maintain a **low resistance circulation** to allow preferential blood flow to them in times of shock
- Musculoskeletal system and gut where blood vessels feed a **high peripheral vascular resistance** circulation



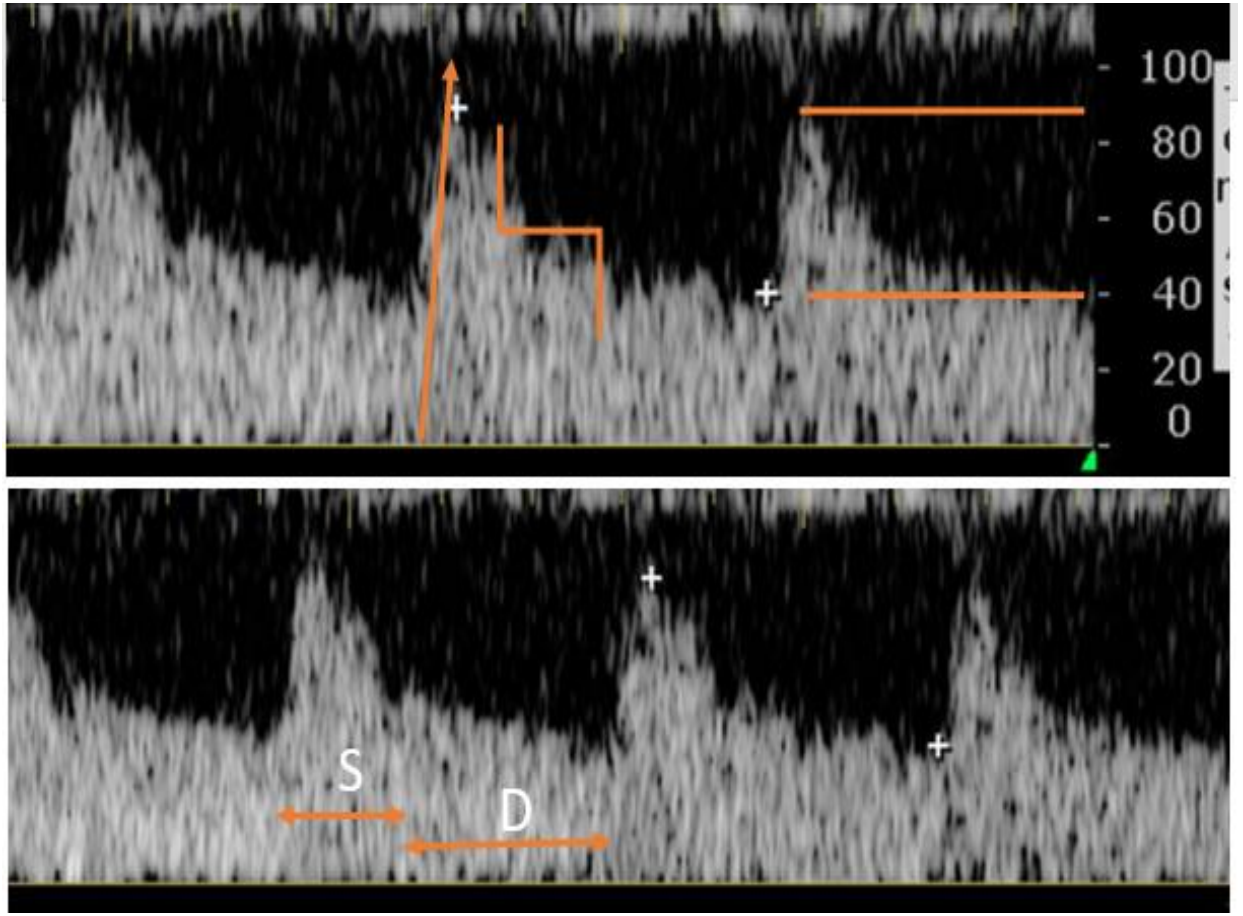
Spectral Doppler Key Parameters

Low resistance circulation

- Systole vs diastole
- Shape of the Waveform
- Rapid upstroke
- Stepwise deceleration
- Diastole <1/2 systole

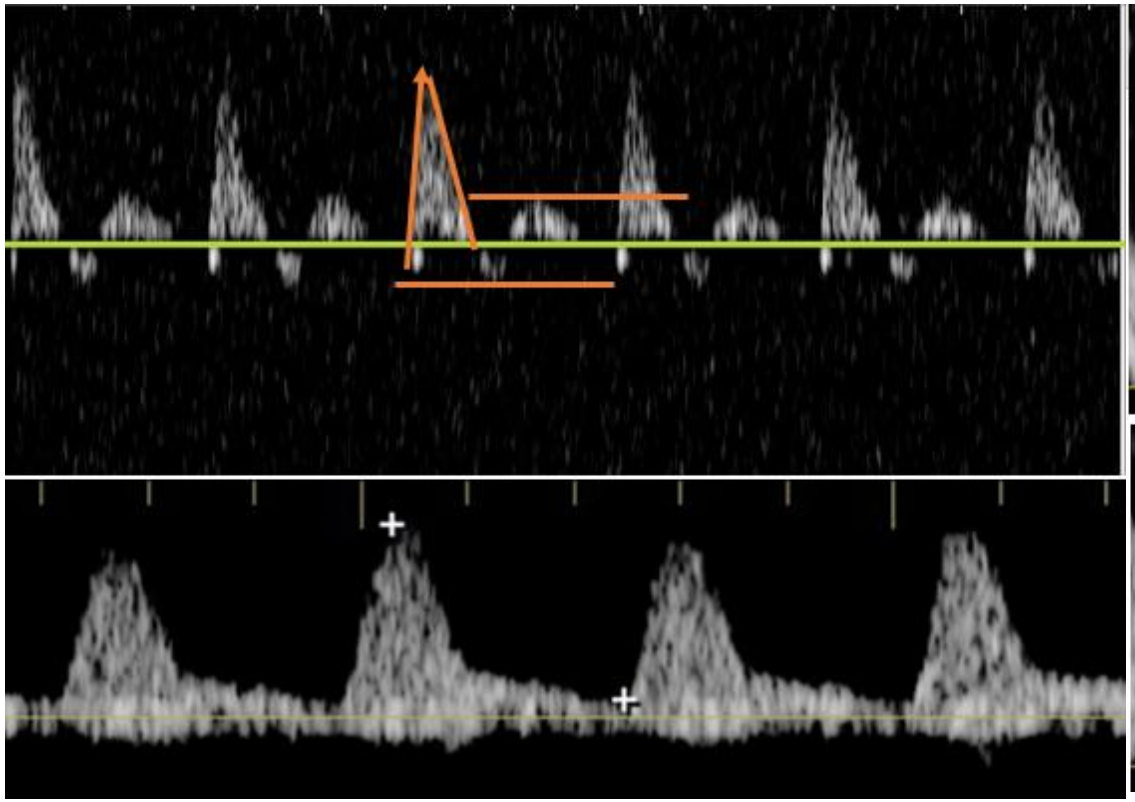
“Blood pressure”

- Peak systolic velocity
- End Diastolic velocity

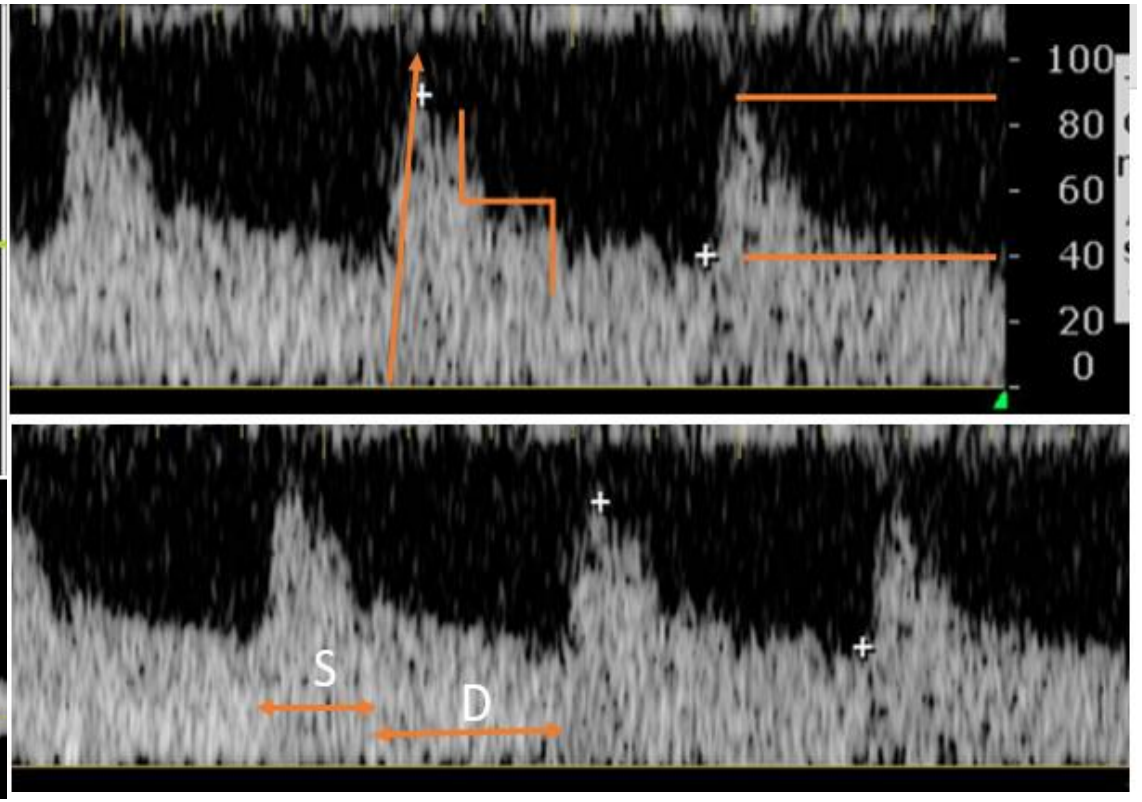


Blood Resistance

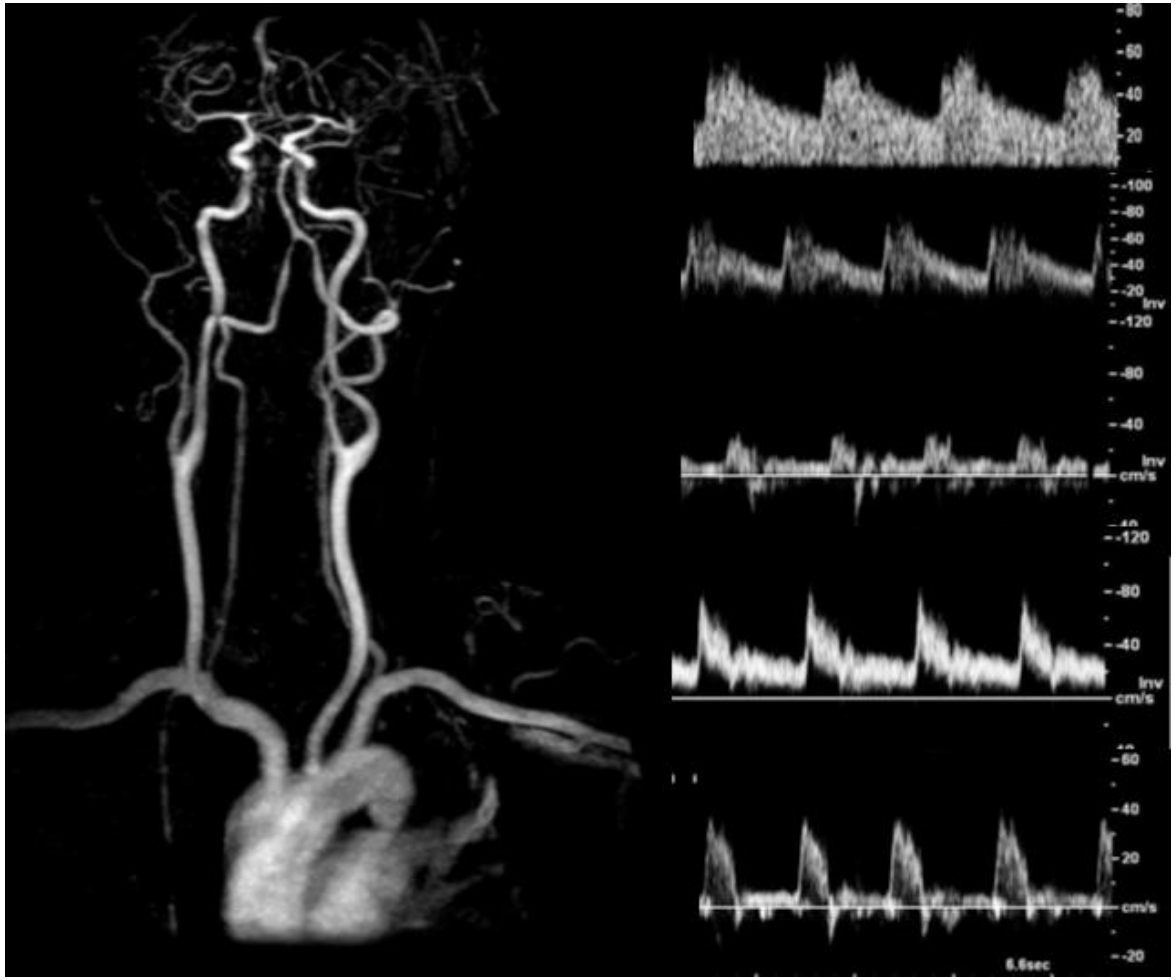
High resistance circulation



Low resistance circulation



Blood Resistance



Andrei V. Alexandrov MD, RV Cerebrovascular Ultrasound in Stroke Prevention and Treatment.

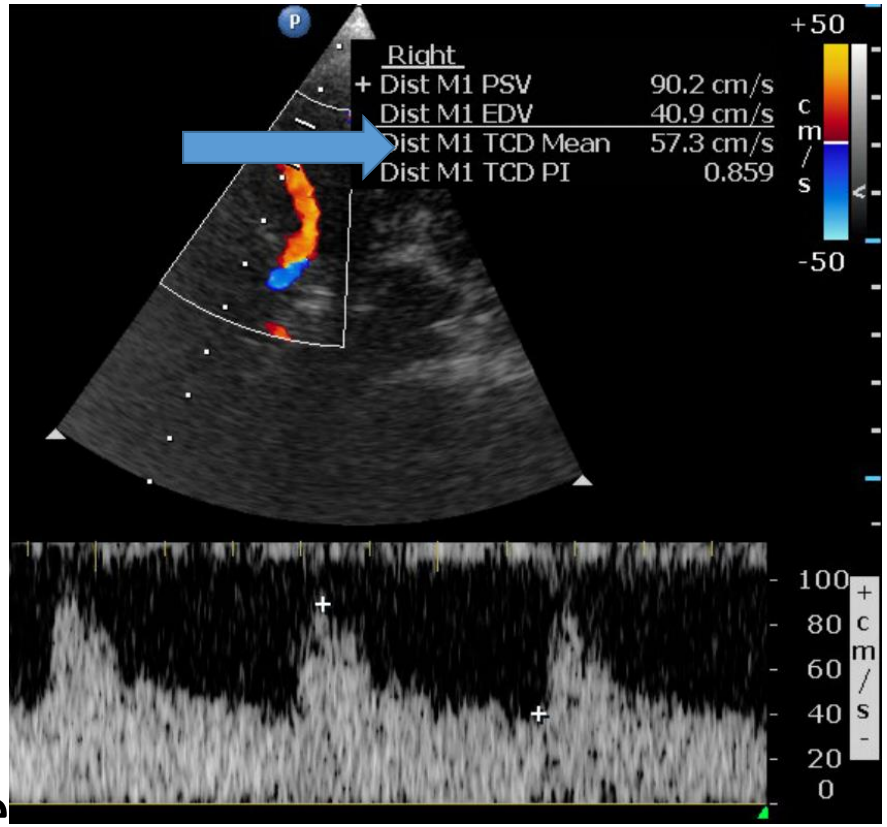
Transcranial Doppler

Key Parameters

- Flow direction
- Peak Systolic Velocity
- End Diastolic velocity
- Time Average Mean Flow Velocity
- Pulsatility Index (PI) index of distal resistance

$$\frac{PSV - EDV}{MV}$$

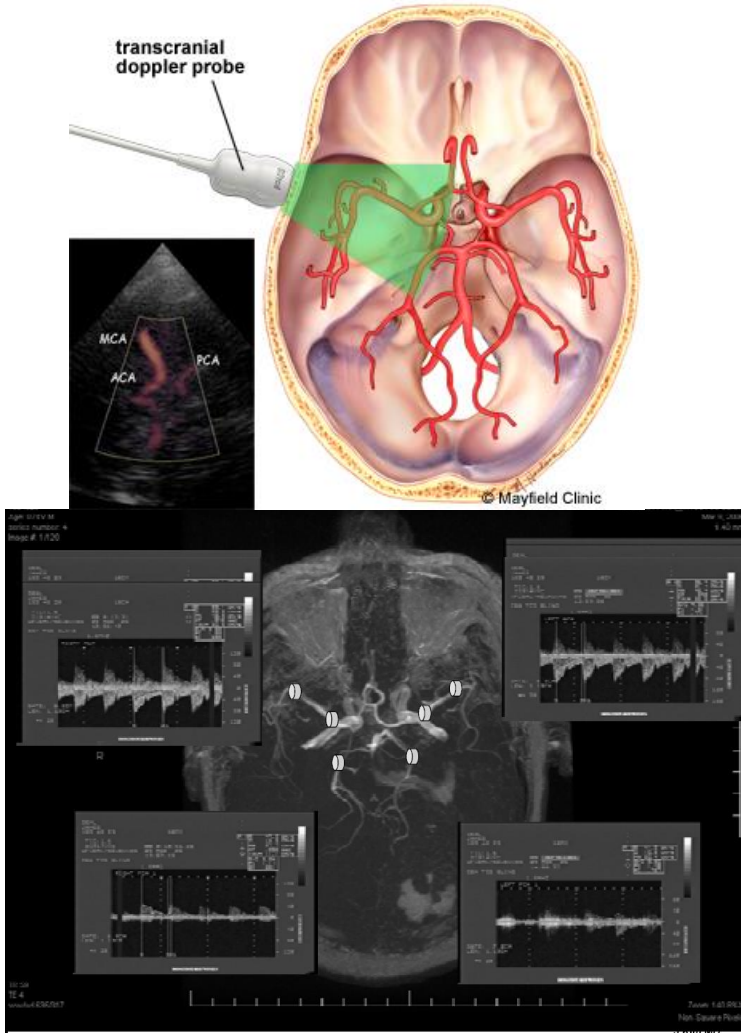
- Normal PI 0.6-1.2



Criteria for Normal TCD

Artery	Depth(mm)	Direction	MFV(Cm/s)
M2-M1 MCA	40 - 65	⇨ ▸	< 80
A1-ACA	62 - 75	⇦ ▸	< 80
ICA Siphon	60 - 64	⇔ ▸	< 70
OA	50 - 62	⇨ ▸	Variable
PCA	60 - 68	⇔ ▸	< 50
BA	80 - 100	⇦ ▸	< 60
VA	45 - 80	⇦ ▸	< 50

Transcranial Doppler



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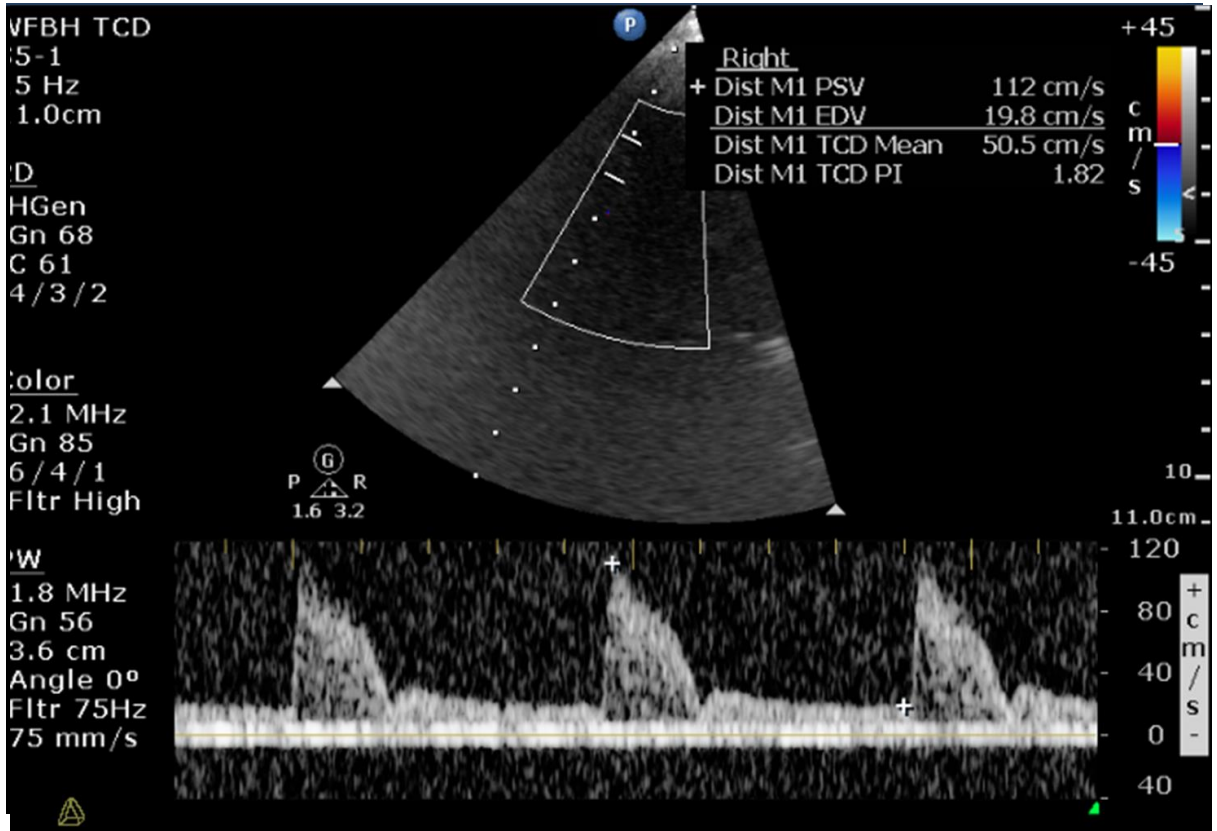
PCA	60 - 68	⇔ ▸	< 50
BA	80 - 100	⇦ ▸	< 60
VA	45 - 80	⇦ ▸	< 50

Aaslid R. Transcranial Doppler Sonography. New York: Springer-Verlag; 1986

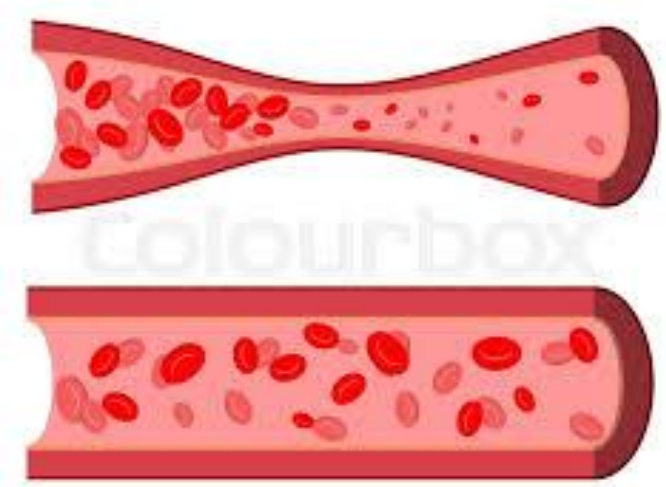
Gosling's Pulsatility Index

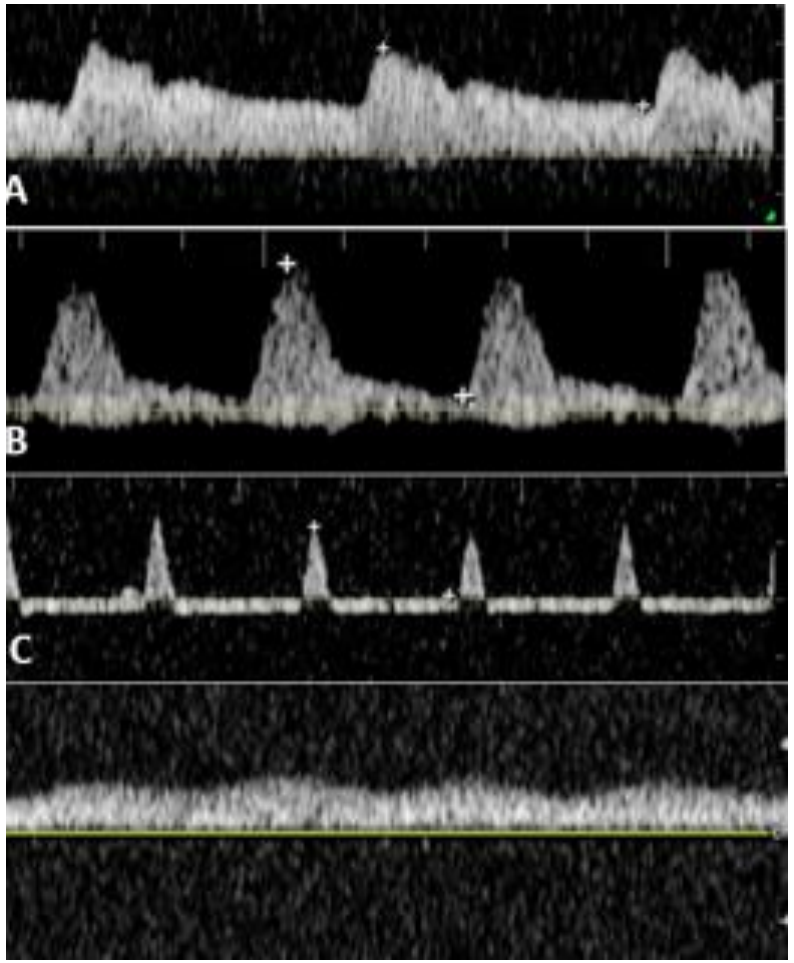
- (PSV-EDV)/MFV
- 0.5 to 1.19



- Quantitative & qualitative changes in the morphology of TCD waveform resulting from CPP changes
- ICP & PI are positively correlated during increases of ICP
- Increase in PI
  - drop in CPP
  - decrease in PaCO2
  - increase in pulsatility of ABP waveform
  - distal resistance
- Decrease in PI
  - proximal stenosis or occlusion due to downstream arteriolar vasodilation
  - AVM

Resistance





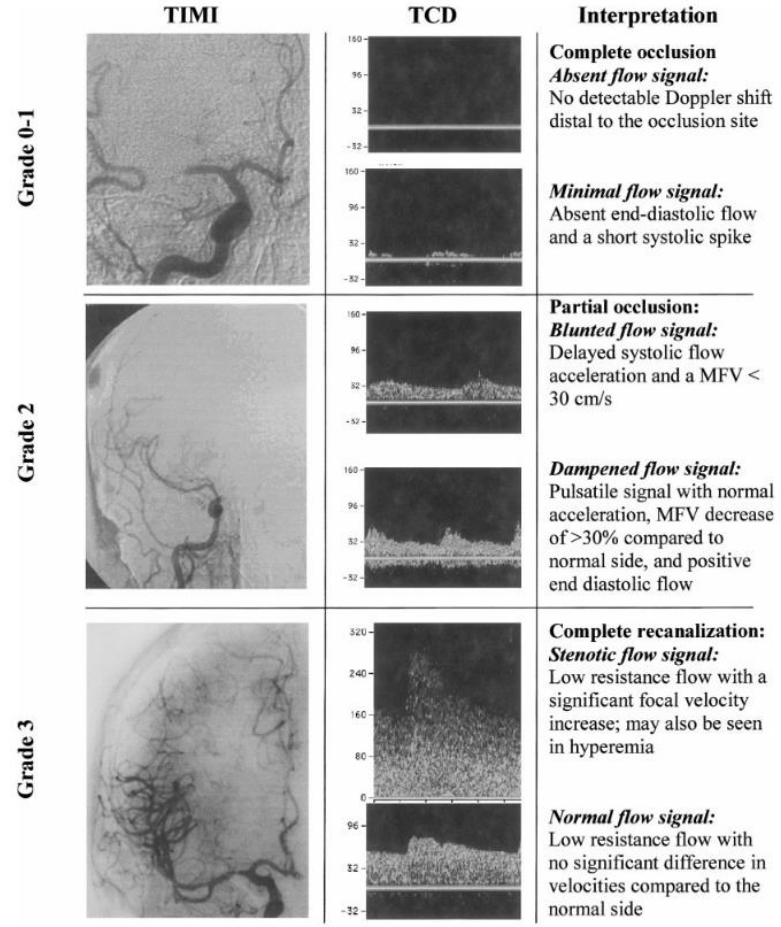


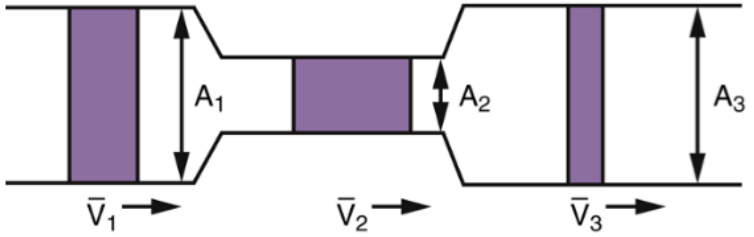
Figure 1. TIMI flow grades and TCD waveforms. MFV indicates maximum flow velocity.

Burgin Stroke 2000

Arterial stenosis

- Mild stenosis
  - Increased PSV
  - No change in doppler pattern
- Moderate to Severe Stenosis
  - Highly increased PSV
  - Decreased EDV
  - Turbulent flow
  - Post stenotic drop in PSV

Hagen-Poiseuille law

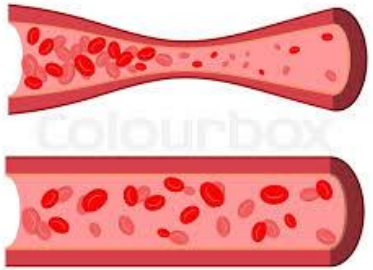


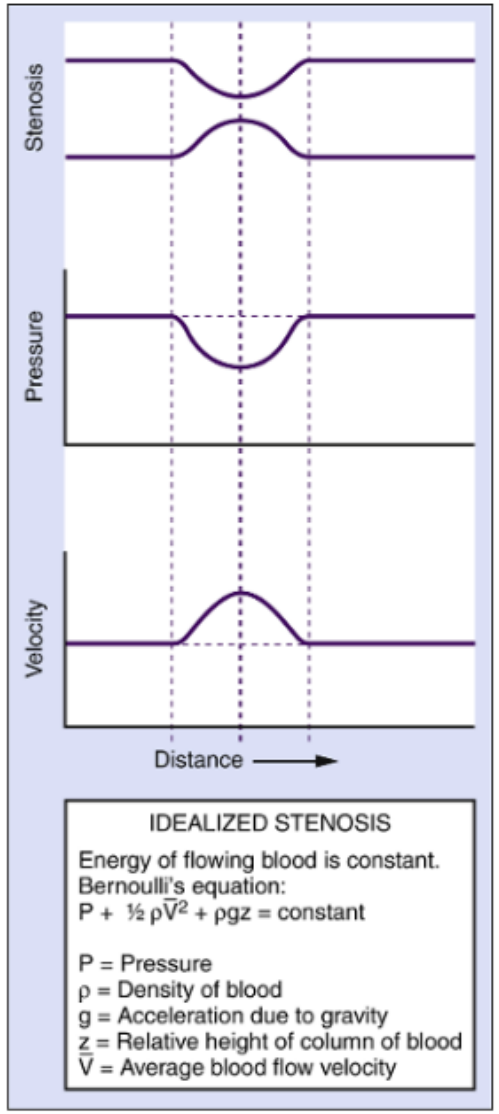
CONSERVATION OF MASS

Volume of blood flowing through a vessel without branches is constant.

Average velocity × Area = Constant

$$\bar{V}_1 \times A_1^2 = \bar{V}_2 \times A_2^2 = \bar{V}_3 \times A_3^2$$





Arterial Stenosis

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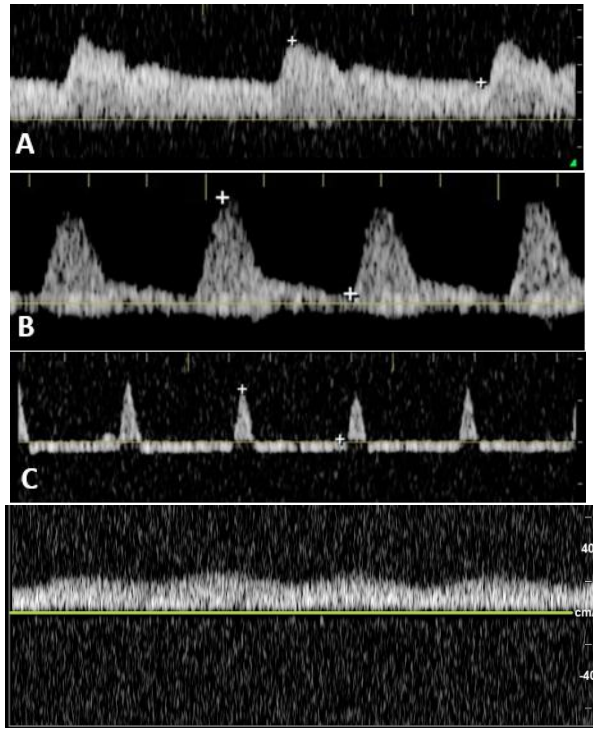
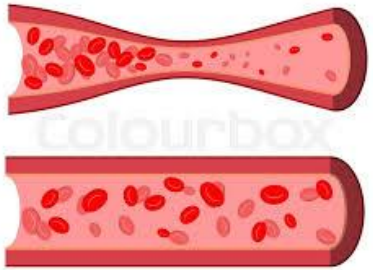
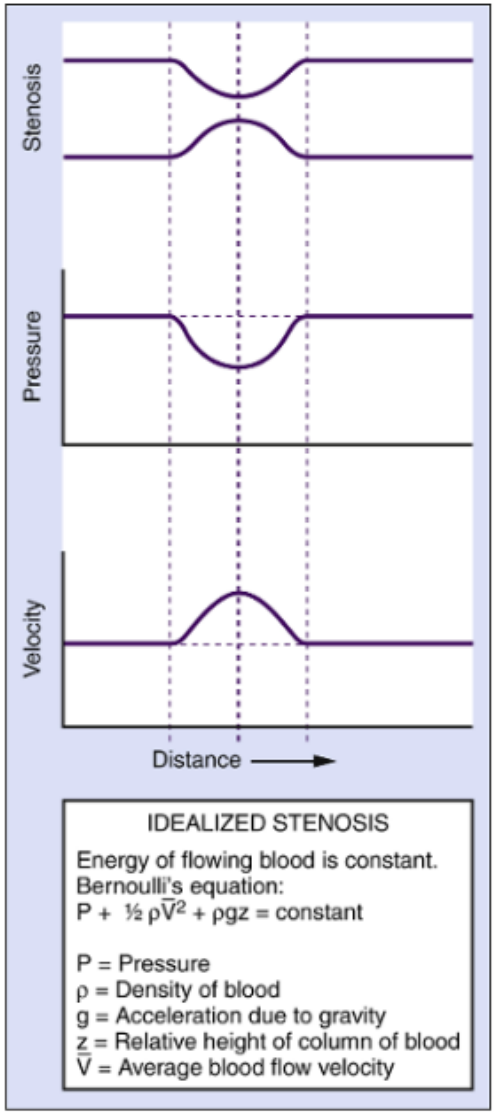
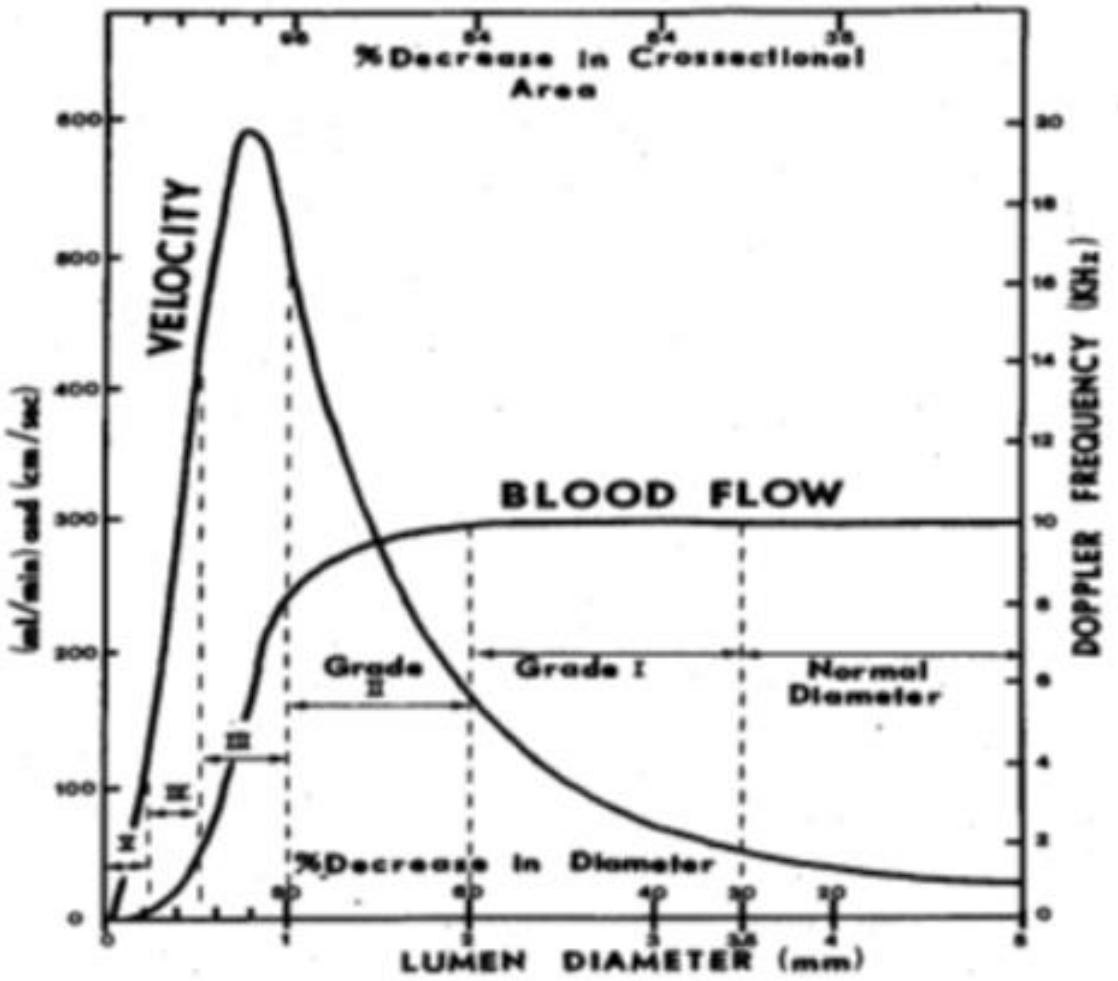
Hagen-Poiseuille law







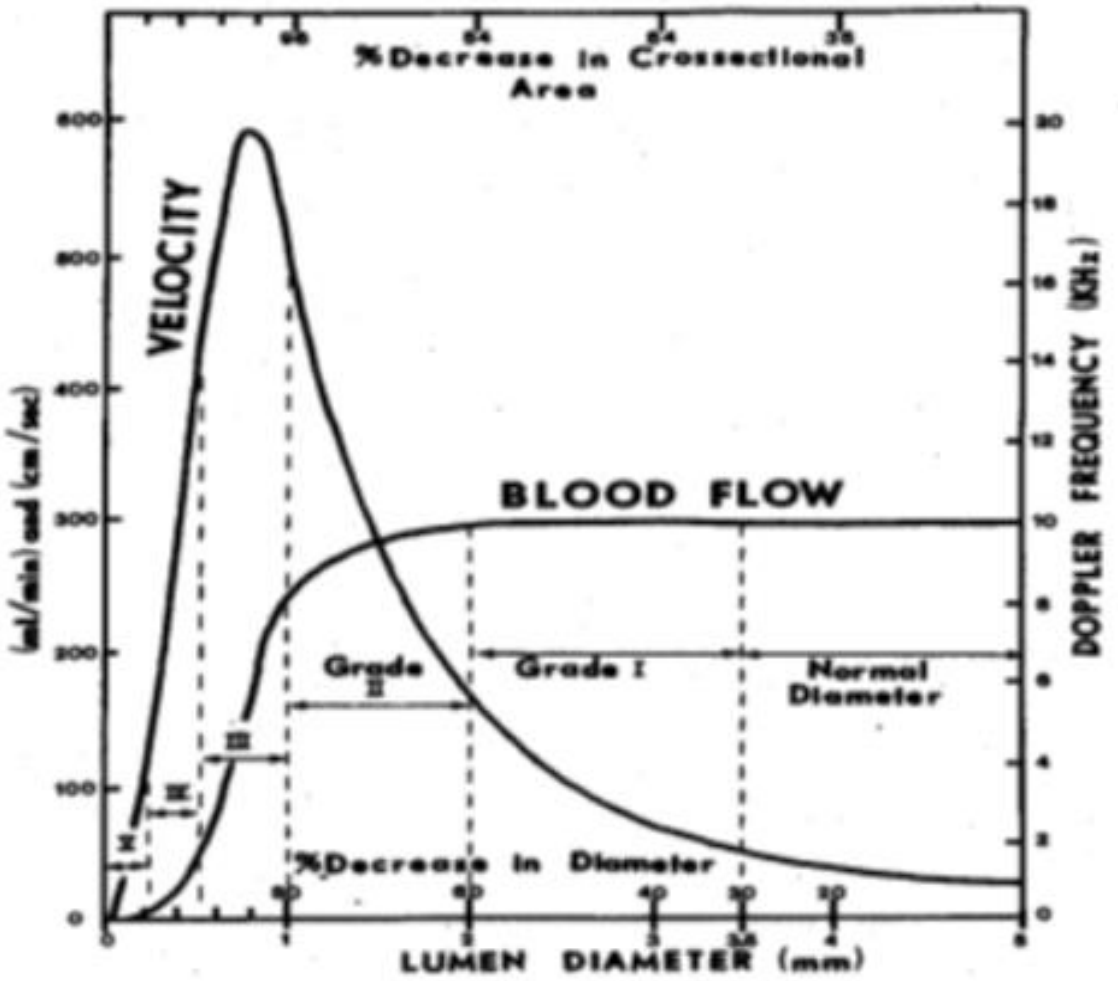
Spencer and Reid Curve

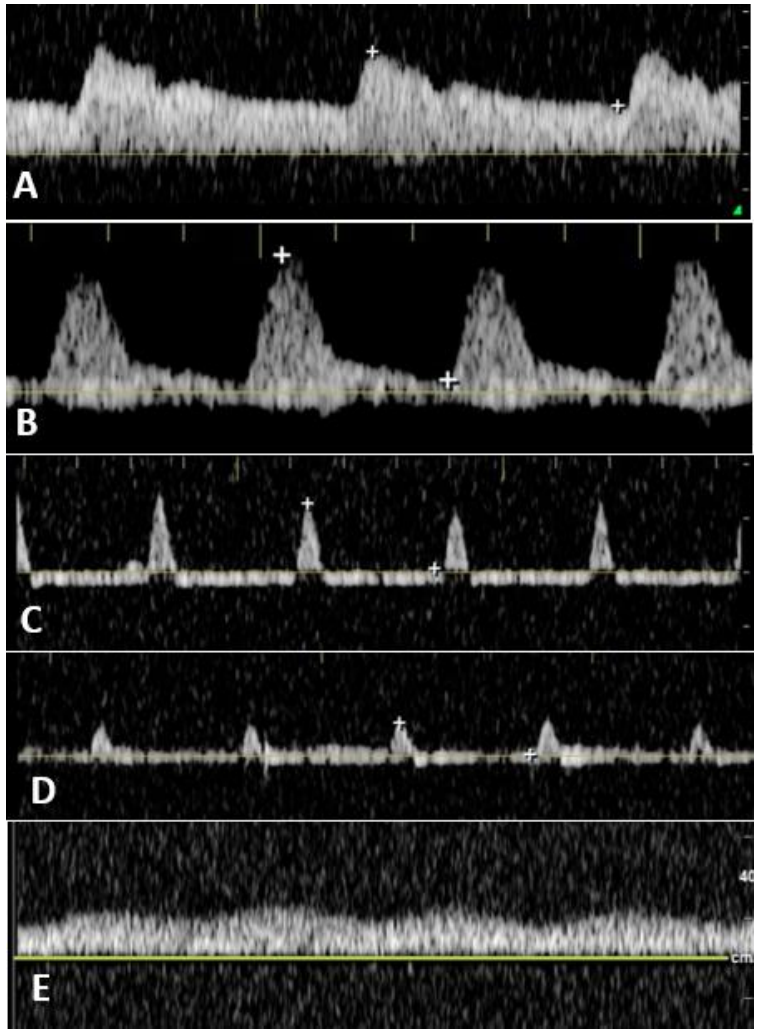


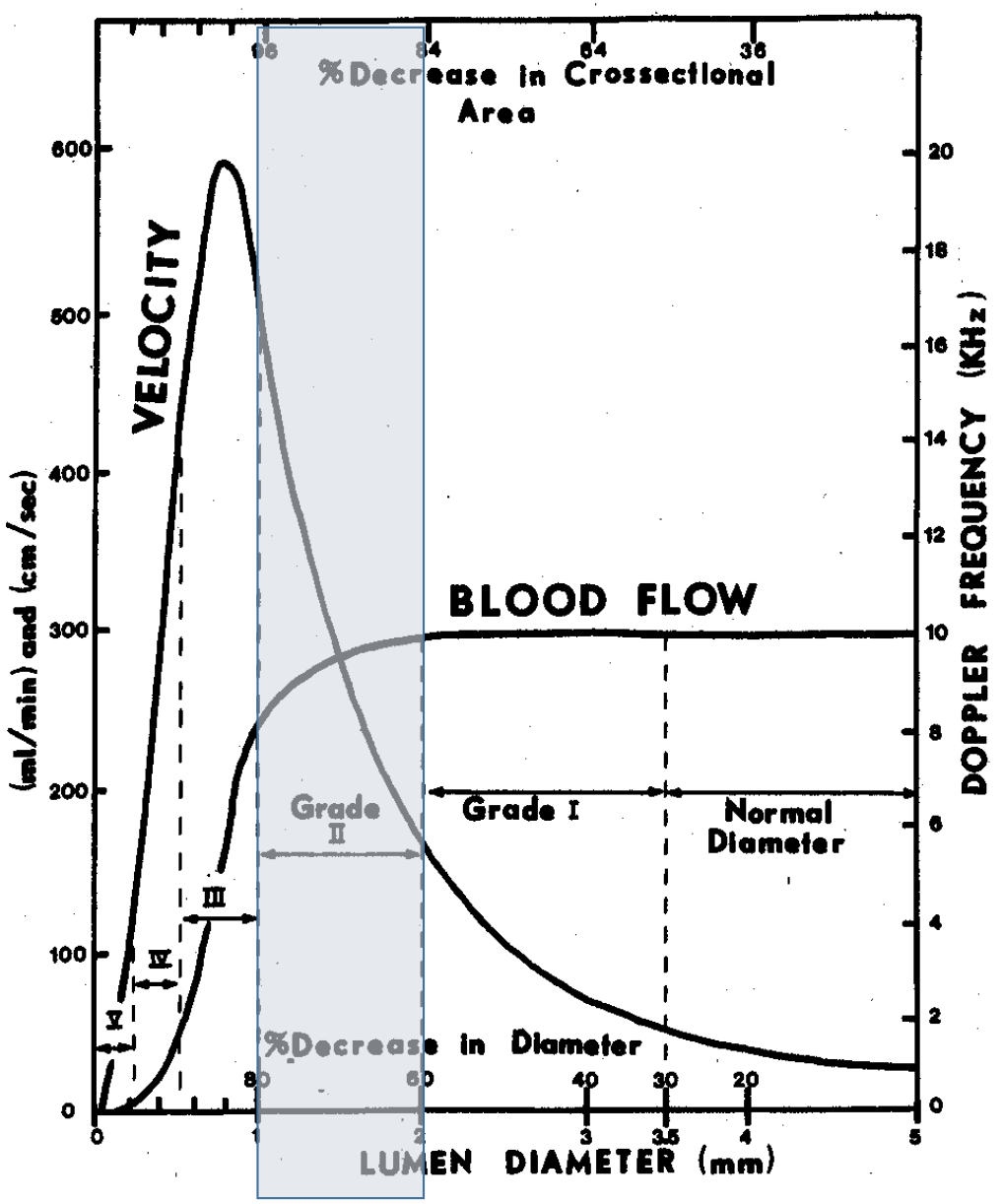
A theoretical model of the relationship between the flow velocity, flow volume, and decreasing size of the residual vessel lumen

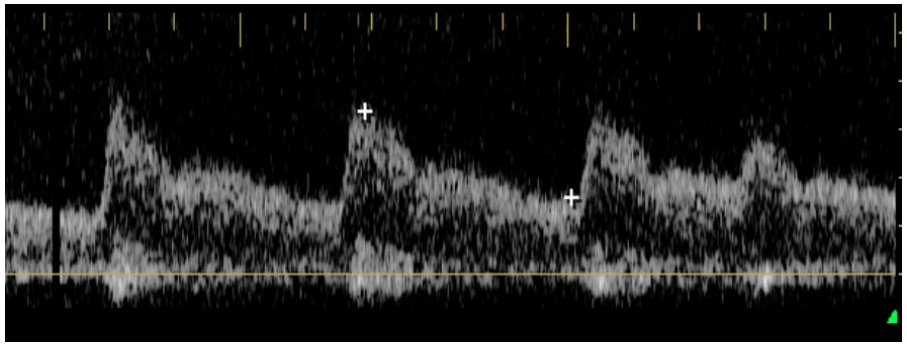
Based on the **Hagen-Poiseuille law**, **continuity principle**, and **cerebrovascular resistance**

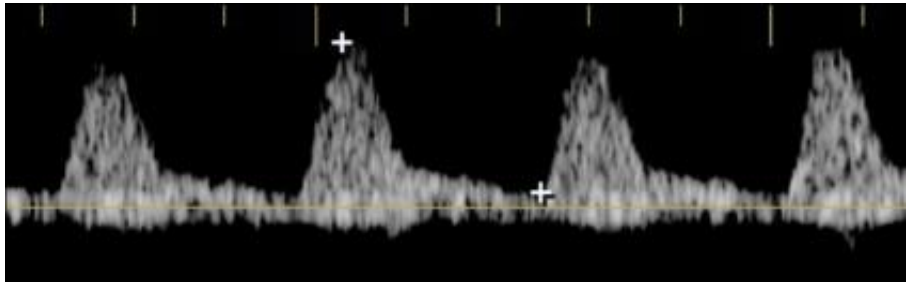
Spencer and Reid Curve

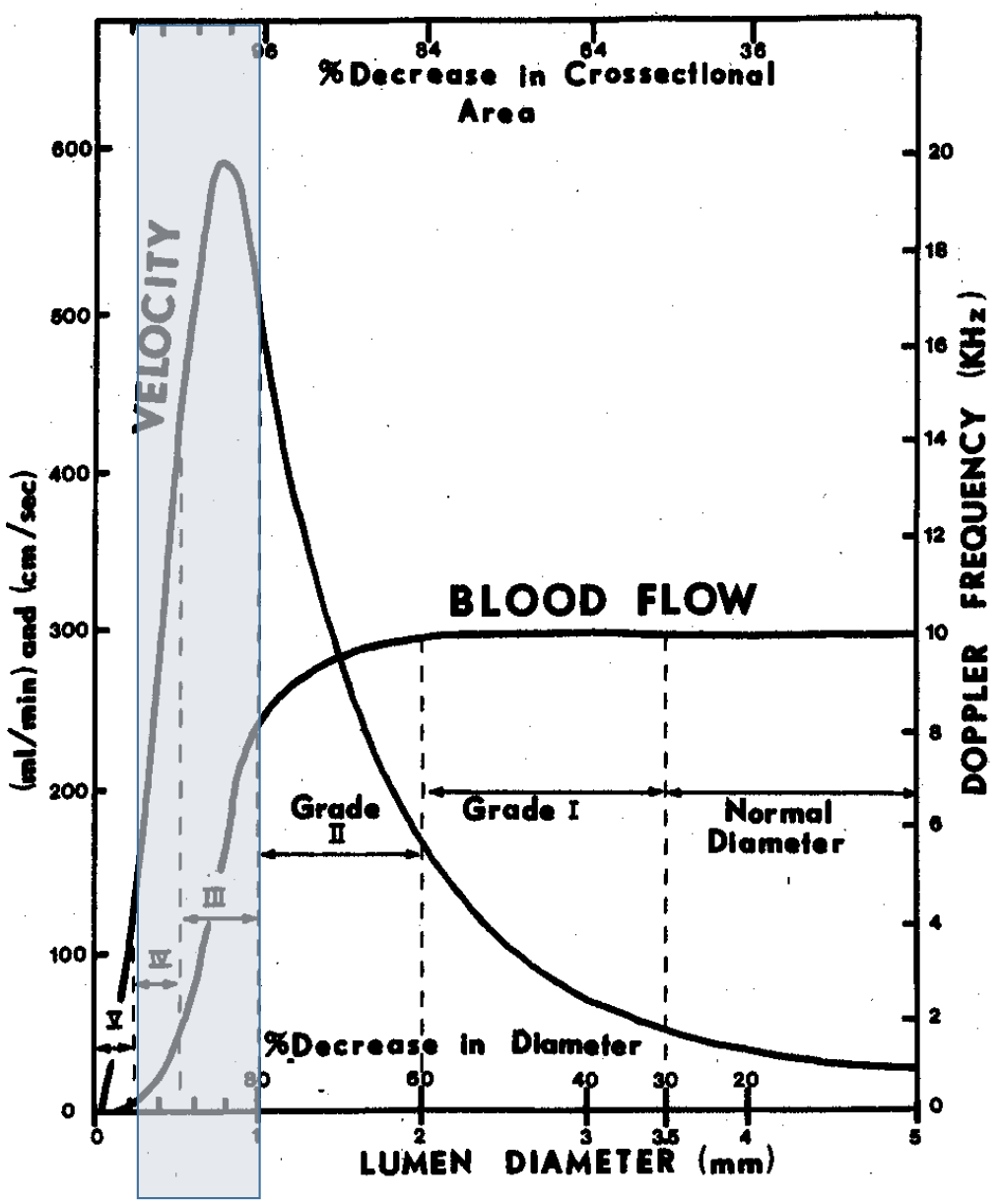


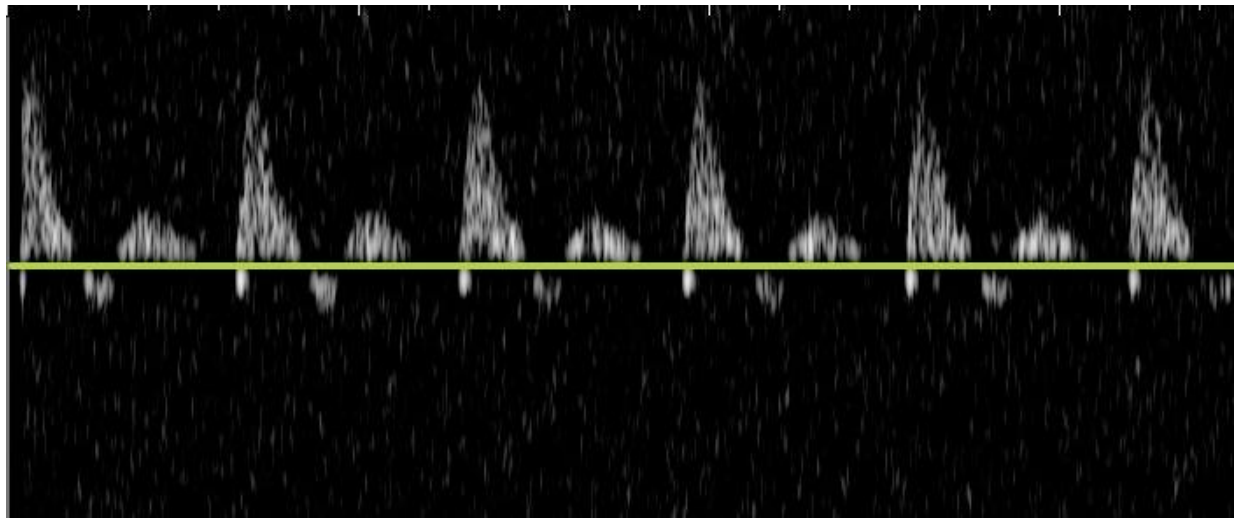


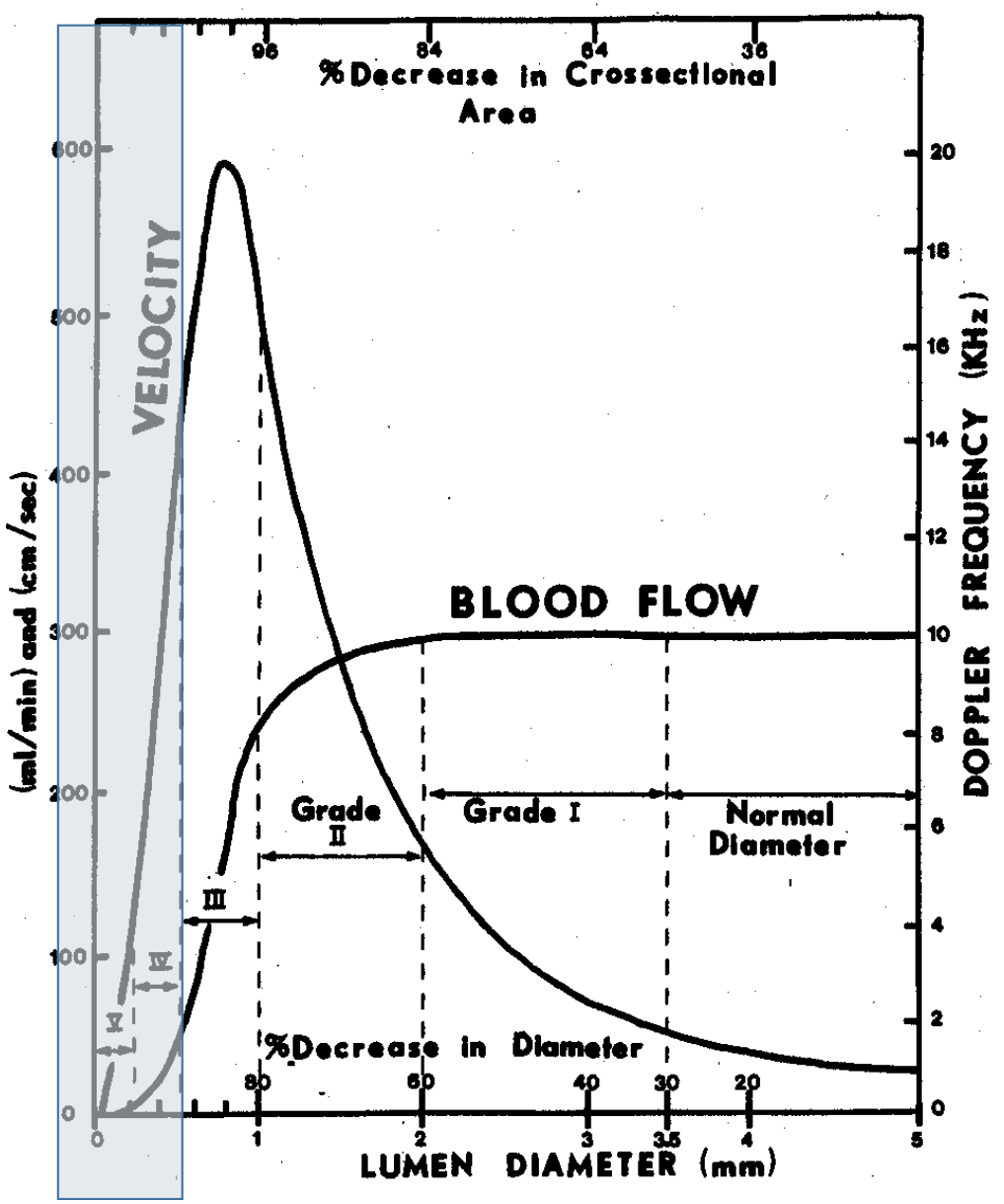

%Decrease in Crossectional Area
VELOCITY
BLOOD FLOW
(ml/min) and (cm/sec)
DOPPLER FREQUENCY (KHz)
Grade II
Grade I
Normal Diameter
%Decrease in Diameter
LUMEN DIAMETER (mm)

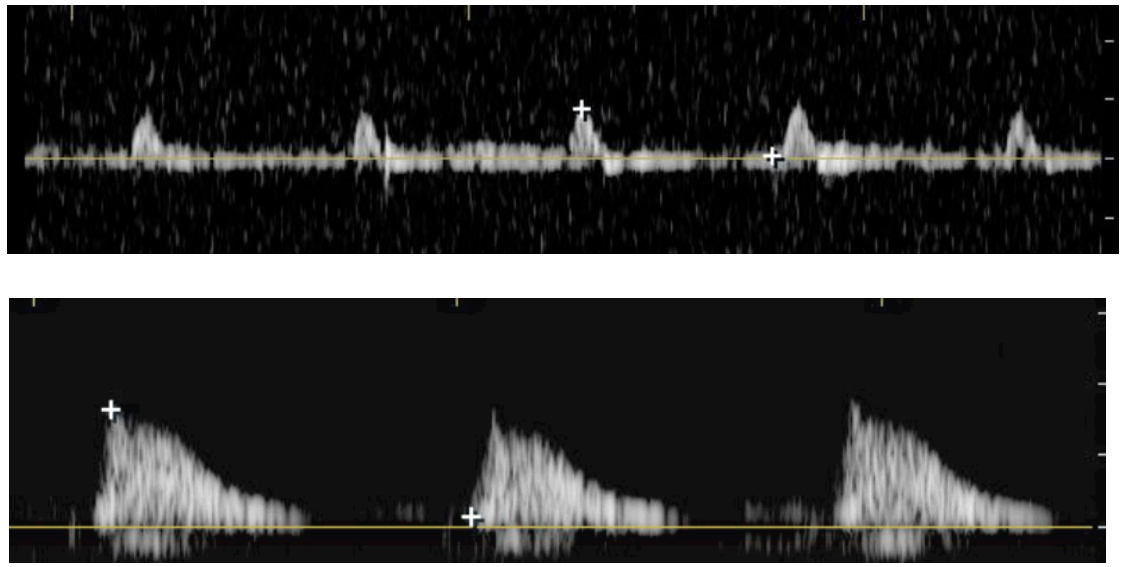





%Decrease in Crossectional Area
96
84
64
36
VELOCITY
BLOOD FLOW
(ml/min) and (cm/sec)
DOPPLER FREQUENCY (KHz)
600
500
400
300
200
100
0
20
18
16
14
12
10
8
6
4
2
0
Grade II
Grade I
Normal Diameter
III
IV
V
%Decrease in Diameter
80
60
40
30
20
0
2
3
3.5
4
5
LUMEN DIAMETER (mm)

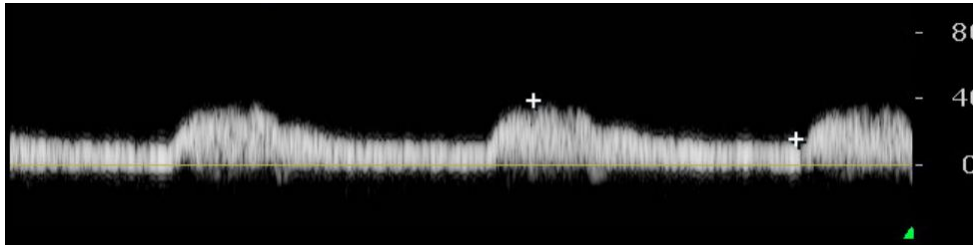


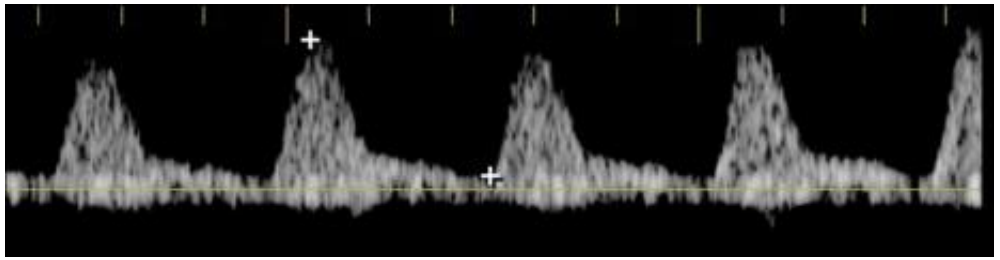

%Decrease in Crossectional Area
96
84
64
36
VELOCITY
BLOOD FLOW
(ml/min) and (cm/sec)
DOPPLER FREQUENCY (KHz)
Grade II
Grade I
Normal Diameter
III
%Decrease in Diameter
80
60
40
30
20
LUMEN DIAMETER (mm)
0
1
2
3
3.5
4
5
100
200
300
400
500
600
2
4
6
8
10
12
14
16
18
20



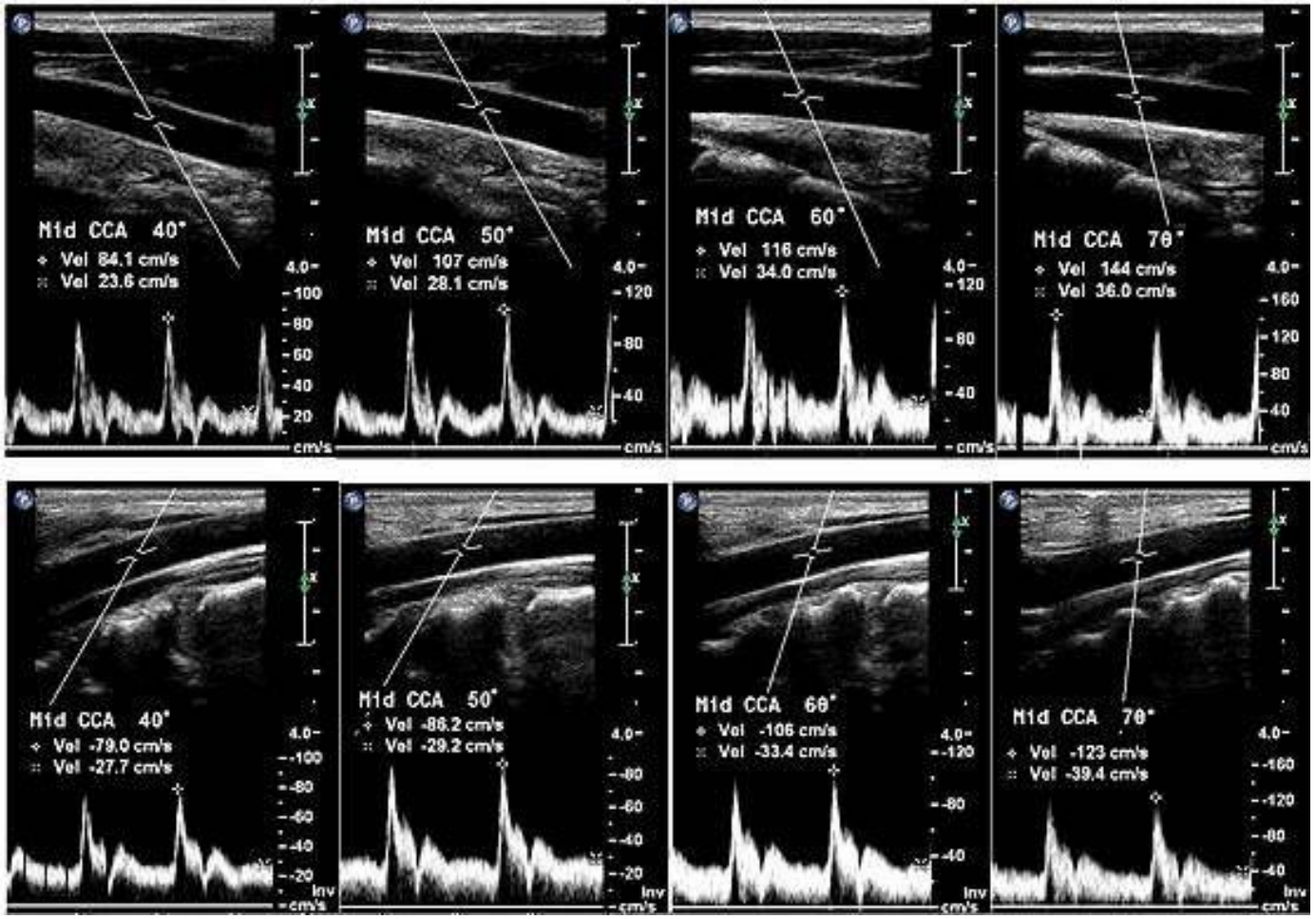
Lesion localization

- **Post stenotic Pattern- Lesion Proximal to insonation if**
  - Low velocity, slow upstroke – low resistance dampened waveforms Parvus (diminished) Tardus (delayed)
  - Delayed flow acceleration, +flow deceleration
  - Spectral narrowing
  - Global parvus et tardus – aortic stenosis
- **Pre-stenotic Pattern- Lesion Distal to insonation if**
  - High velocity – high resistance(low EDV) upstroke
  - High resistance sharp stump

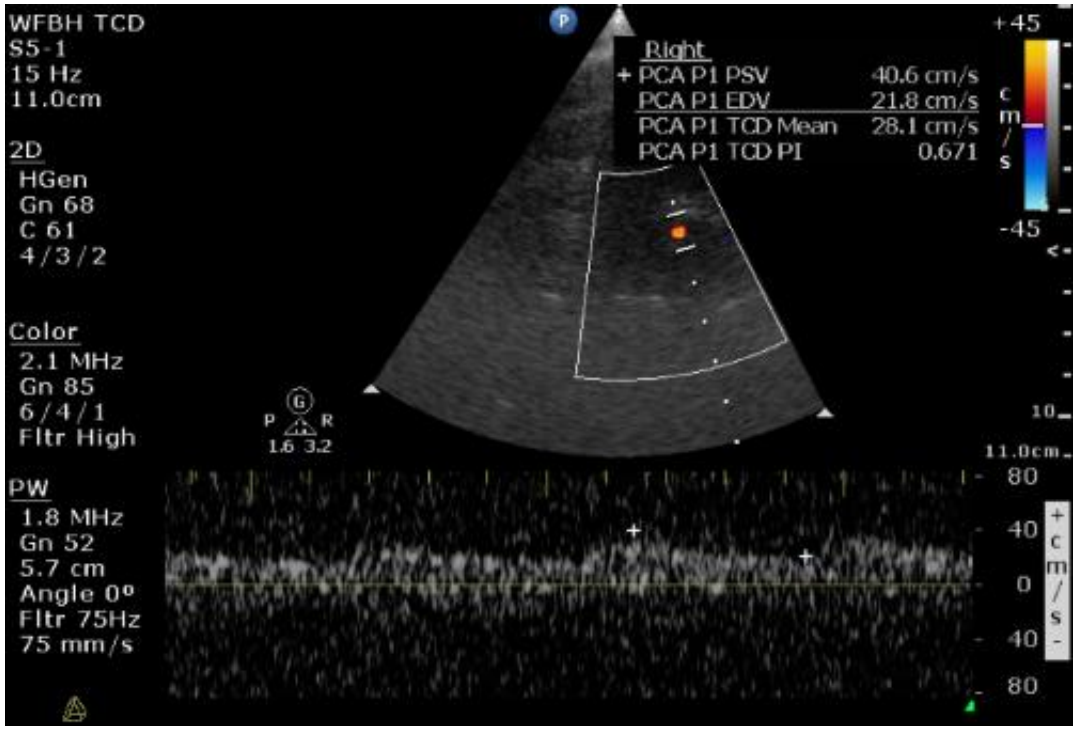




Angle of insonation

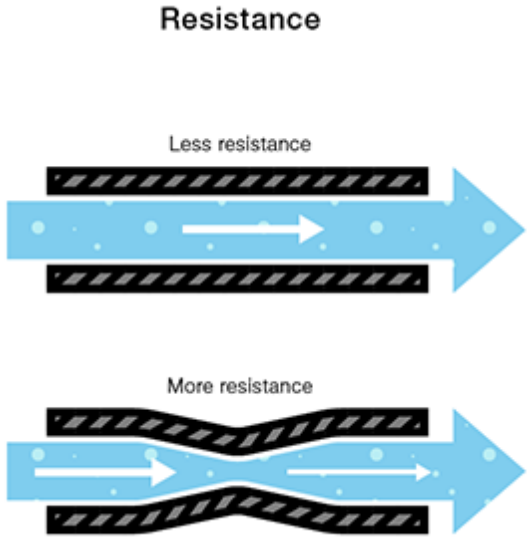


As angle of insonation increases, waveforms become blunted and ultimately disappear

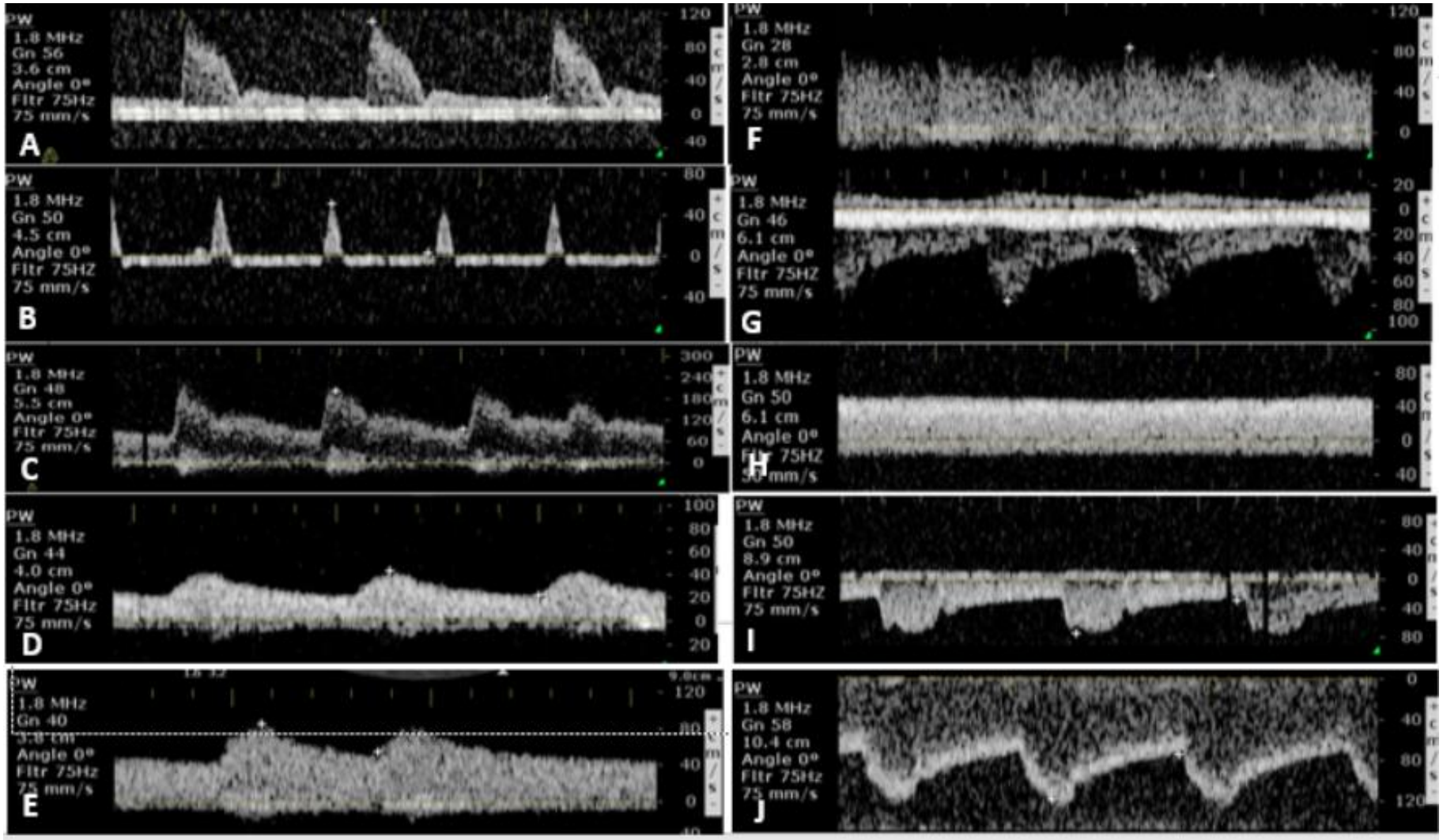


Resistance

	Resistance of the cerebral vessels	Resistance of the distal vascular bed
Increased	Intracranial stenosis, cerebral vasospasm Vasoconstriction	Increased intracranial pressure Distal atherosclerotic disease.
Decreased	Arteriovenous shunting	Peripheral vasodilatation Hypercarbia, acidosis Reperfusion of ischemic brain



Waveforms

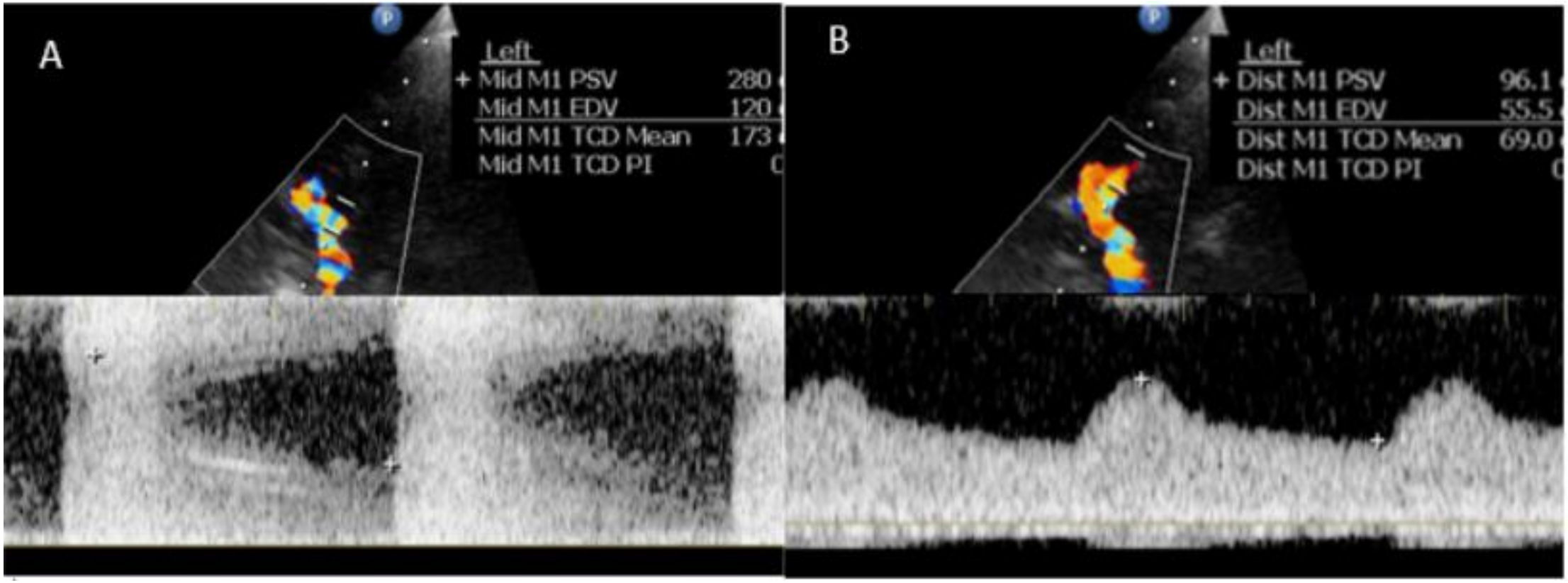

PW
1.8 MHz
Gn 56
3.6 cm
Angle 0°
Fltr 75Hz
75 mm/s
A
PW
1.8 MHz
Gn 50
4.5 cm
Angle 0°
Fltr 75HZ
75 mm/s
B
PW
1.8 MHz
Gn 48
5.5 cm
Angle 0°
Fltr 75Hz
75 mm/s
C
PW
1.8 MHz
Gn 44
4.0 cm
Angle 0°
Fltr 75Hz
75 mm/s
D
PW
1.8 MHz
Gn 40
Angle 0°
Fltr 75Hz
75 mm/s
E
PW
1.8 MHz
Gn 28
2.8 cm
Angle 0°
Fltr 75HZ
75 mm/s
F
PW
1.8 MHz
Gn 46
6.1 cm
Angle 0°
Fltr 75HZ
75 mm/s
G
PW
1.8 MHz
Gn 50
6.1 cm
Angle 0°
Fltr 75HZ
H
PW
1.8 MHz
Gn 50
8.9 cm
Angle 0°
Fltr 75HZ
75 mm/s
I
PW
1.8 MHz
Gn 58
10.4 cm
Angle 0°
Fltr 75Hz
75 mm/s
J

Clinical Indications

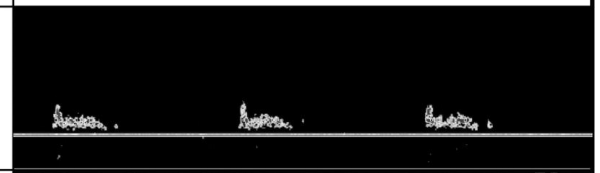
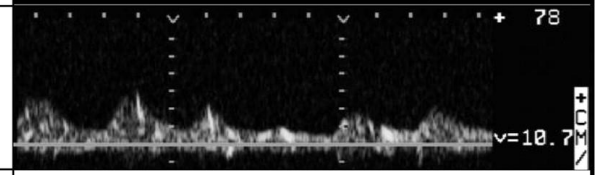
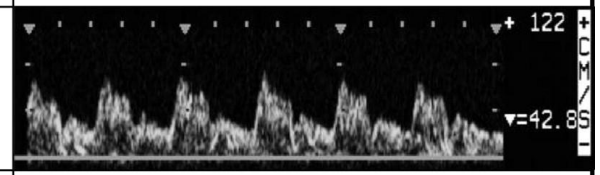
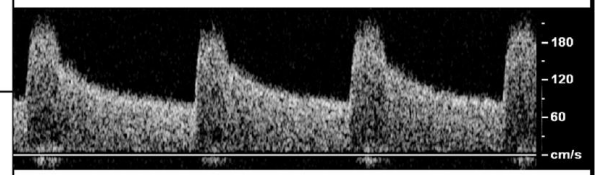
- Ischemic Stroke Evaluation
- Subarachnoid Hemorrhage
- Vertebral Steal
- Cerebral Circulatory arrest assessment
- Noninvasive Intracranial Pressure Measurement

Ischemic Stroke Evaluation

Intracranial stenosis



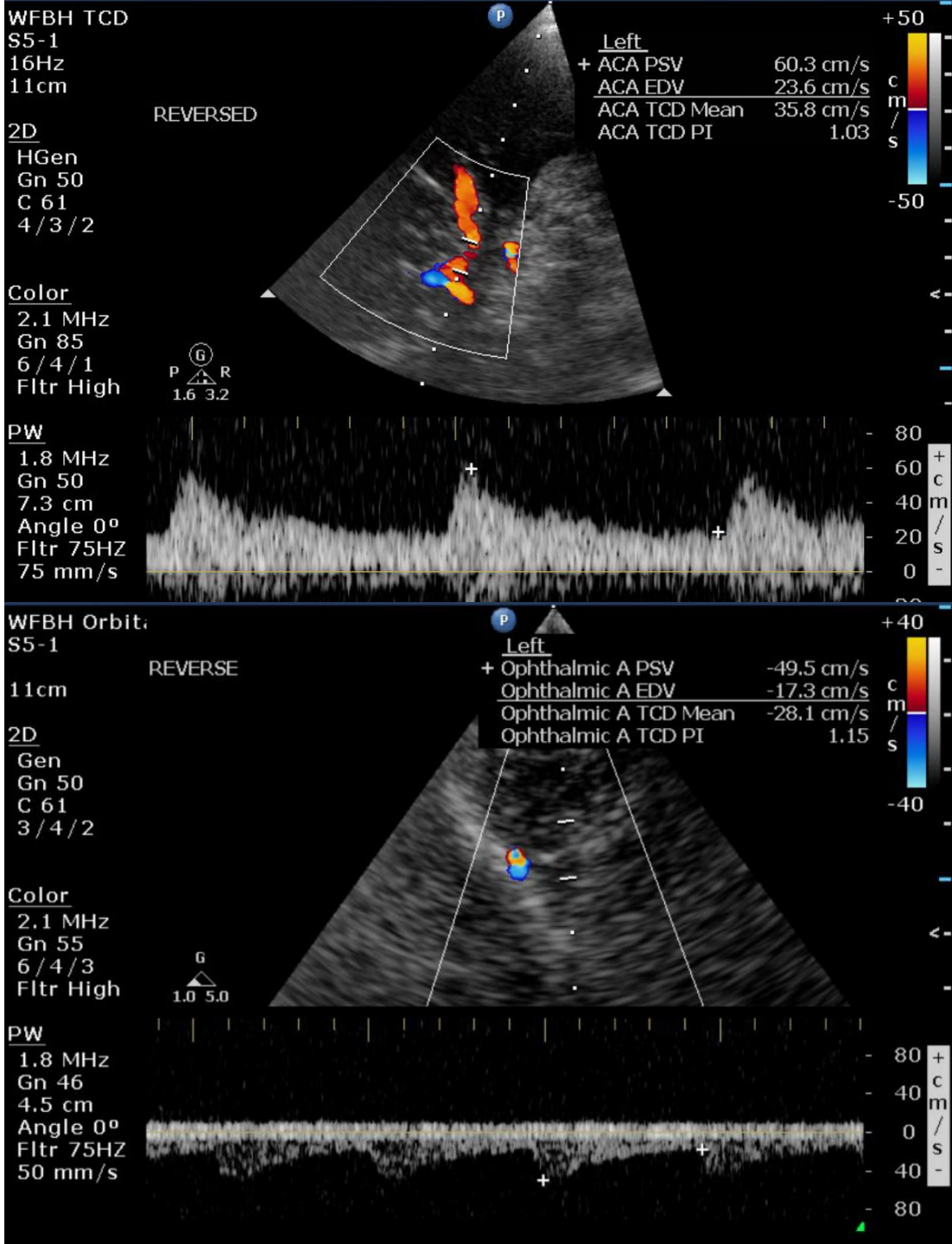
Waveforms during recanalization

Hemodynamic pattern	Example
No flow	
Low flow velocities without diastolic flow	
Low flow velocities with diastolic flow	
Established perfusion:	
a) flow velocities equal to contra-lateral side	
b) high focal flow velocities (i.e. stenosis)	
c) high segmental flow velocities (hyperperfusion)	

Max Nedelmann et al. Stroke. 2009;40:3238-3244

Assessment of collaterals

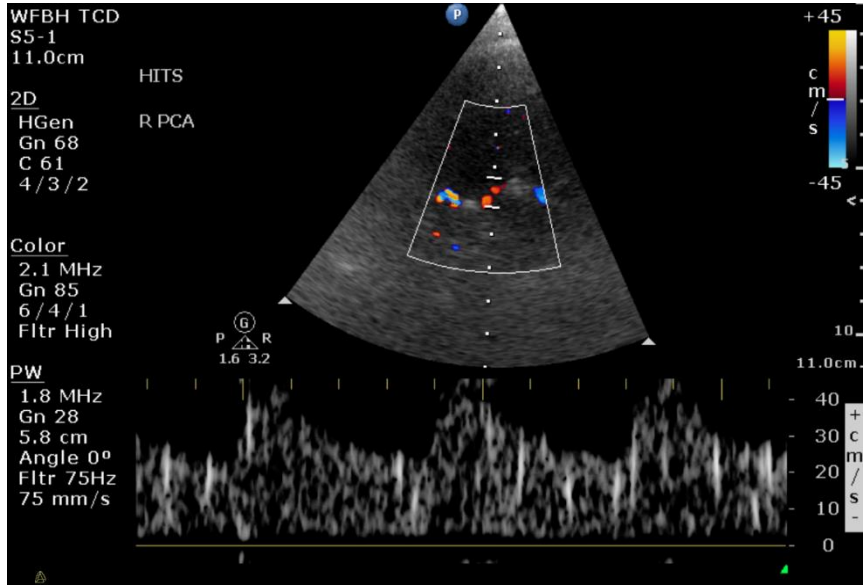
- A Comm collateral
  - C/L ACA >MCA, I/L ACA reversed
- P Comm collateral
  - PCA >MCA
- Reversed opthalmics
  - ECA to ICA siphon
  - Relative inadequacy of Acom , Pcom collaterals

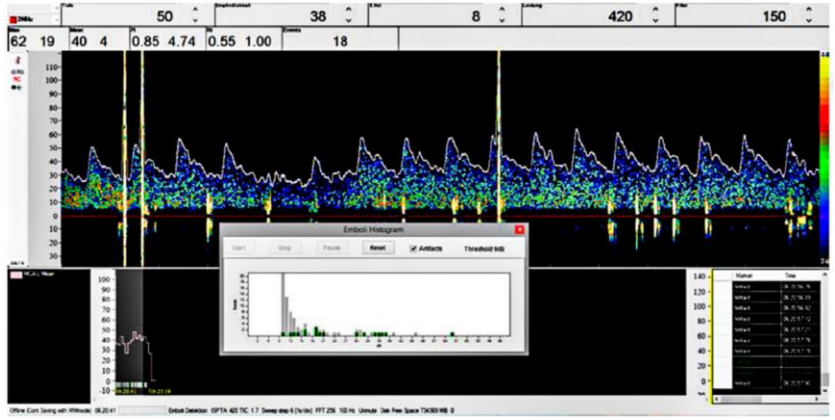


Lancet Neurol 2011; 10: 909–21

Emboli monitoring

- Coronary artery bypass
- Carotid endarterectomy
- Cerebral angiograms
- ECMO
- Infective endocarditis





Embolic Signals during Routine Transcranial Doppler Ultrasonography in Aneurysmal Subarachnoid Hemorrhage

Fernando Mendes Paschoal Jr., Karla de Almeida Lins Ronconi, Marcelo de Lima Oliveira, Ricardo de Carvalho Nogueira, Eric Homero Albuquerque Paschoal, Manoel Jacobsen Teixeira, Eberval Gadelha Figueiredo, and Edson Bor-Seng-Shu

Cerebral Microemboli During Coronary Artery Bypass Using Different Cardioplegia Techniques

Andrew J. Baker, MD, Basem Naser, MBBS, Mark Benaroia, and C. David Mazer, MD

Department of Anaesthesia, St. Michael's Hospital, University of Toronto, Toronto, Ontario, Canada

Transcranial Doppler Emboli Monitoring for Infective Endocarditis

Glen Huang, Leilani L. Johnson, James E. Peacock Jr., Charles Tegeler, Kyle Davis, Aarti Sarwal

From the Department of Internal Medicine, University of California Los Angeles, Los Angeles, CA (GH); Department of Neurology, Wake Forest Baptist Medical Center, Winston-Salem, NC (LLJ, CT, AS); Department of Internal Medicine, Wake Forest Baptist Medical Center, Winston-Salem, NC (JJP); and Department of Pharmacy, Wake Forest Baptist Medical Center, Winston-Salem, NC (KD).

Vasomotor reactivity

- Co2, Diamox , Breath Holding
- Intracranial hemodynamic status in patients with carotid occlusive disease with the intent of predicting the occurrence of ischemic brain events
- Compare intracranial hemodynamics before and after carotid endarterectomy/ carotid stent or EC MCA bypass procedures
- Compare autoregulation and collateral circulation in the different parts of the circle of Willis specially with bilateral lesions

Ringelstein EB, et al. Stroke 1988;19:962-9, Sorteberg W, et al. Acta Neurochir 1989;97:139-45, Markus HS, et al. Stroke 1992;23:668-73, Silvestrini M, et al. JAMA: March 2000. Neurol Neurochir Pol. 2001

Value despite CTA, MRI in stroke

- 25 of 198 acute stroke patients admitted in 2017 underwent TCD and/or CUS after having CTA head and neck during their hospital admission

The Clinical Contribution of Neurovascular Ultrasonography in Acute Ischemic Stroke

Jonathan R. Gomez, Kyle S. Hobbs, Leilani L. Johnson, Quang D. Vu, John Bennett, Charles Tegeler, Stacey Q. Wolfe, and Aarti Sarwal

Table 3. Results of Review by Two Vascular-Trained Neurologists to Determine Whether Ultrasound added Clinical Information to Management Decisions Despite Having CTA Results

	tPA patients DSA	tPA patients No DSA	No tPA DSA	No tPA No DSA	Totals
Ultrasound changed management after CTA	4	4	2	0	10
No change in management after ultrasound	1	10	3	1	15
Totals	5	14	5	1	

The numbers reflect cases where consensus was reached. A third expert (AS) was used to reach consensus in cases of disagreement using a nominal group process. tPA = tissue plasminogen activator; DSA = digital subtraction angiogram; CTA = computed tomography angiogram.

Table 4. Notable Abnormalities Found on Neurovascular Ultrasound

Most notable abnormalities that were determined to have impacted management

1. Detection of mobile thrombus requiring anticoagulation;
2. Distinguishing carotid near occlusion from occlusion;
3. Confirming hemodynamic significance of intra/extracranial stenosis helping emergent stenting/endarterectomy;
4. Detecting hyperperfusion/hyperperfusion on TCDs inciting workup for noncerebrovascular etiology;
5. Establishing chronicity of carotid stenosis based on collateral flow patterns, especially in patients with no known history or prior medical evaluation.

Most notable abnormalities that did not affect immediate clinical management

1. Anterior circulation velocity asymmetry;
2. Waveform patterns consistent with known CTA findings of intracranial stenosis;
3. Incidental vertebral steal phenomena.

Value despite CTA, MRI in stroke

- 86 patients 2012-2015- CTA, TCD, MRI
- Patients already studied with CTA, TCD during the acute period provide
    - additional useful information in 1 out of 2 patients
    - changes in management are indicated in 1 out of 6 cases.
- The most frequent additional information was
    - **collateral pathways**
    - information related to patency of vessels
    - active microembolization

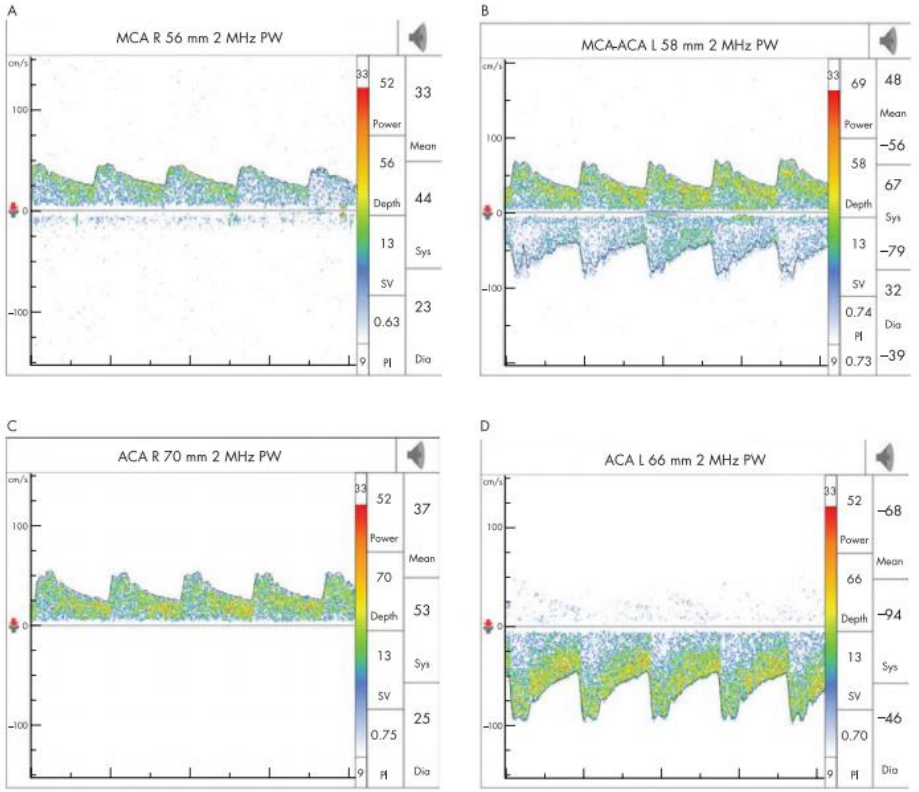
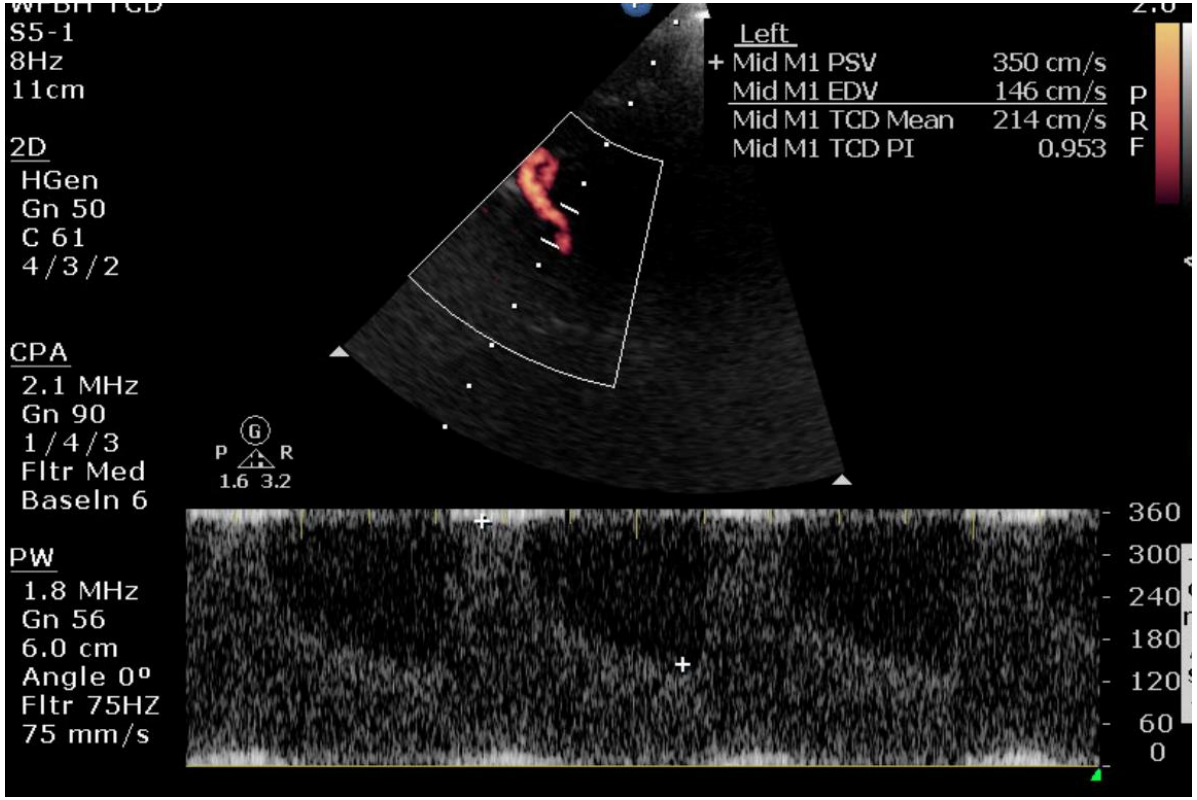


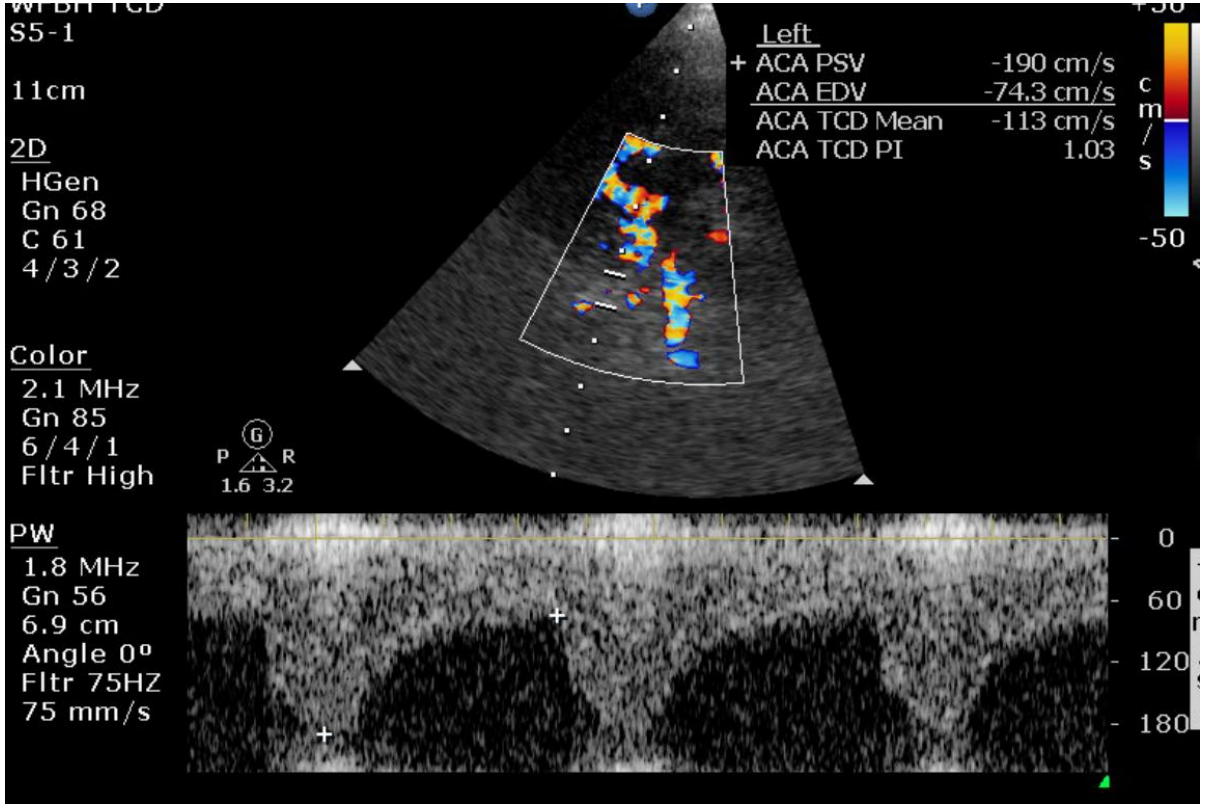
Figure 5 Effect of extracranial internal carotid artery (ICA) stenosis on cerebral haemodynamics (patient had high grade stenosis of proximal right ICA). (A) Decrease in mean flow velocity of right (ipsilateral) middle cerebral artery (MCA). (B) Normal flow in left (contralateral) MCA with increased flow in left (contralateral) anterior cerebral artery (ACA) (due to collateral flow). (C) Reversal of flow in right (ipsilateral) ACA. (D) Increased flow in left (contralateral) ACA.

J Neuroimaging 2016;26:420-425.

Subarachnoid Hemorrhage

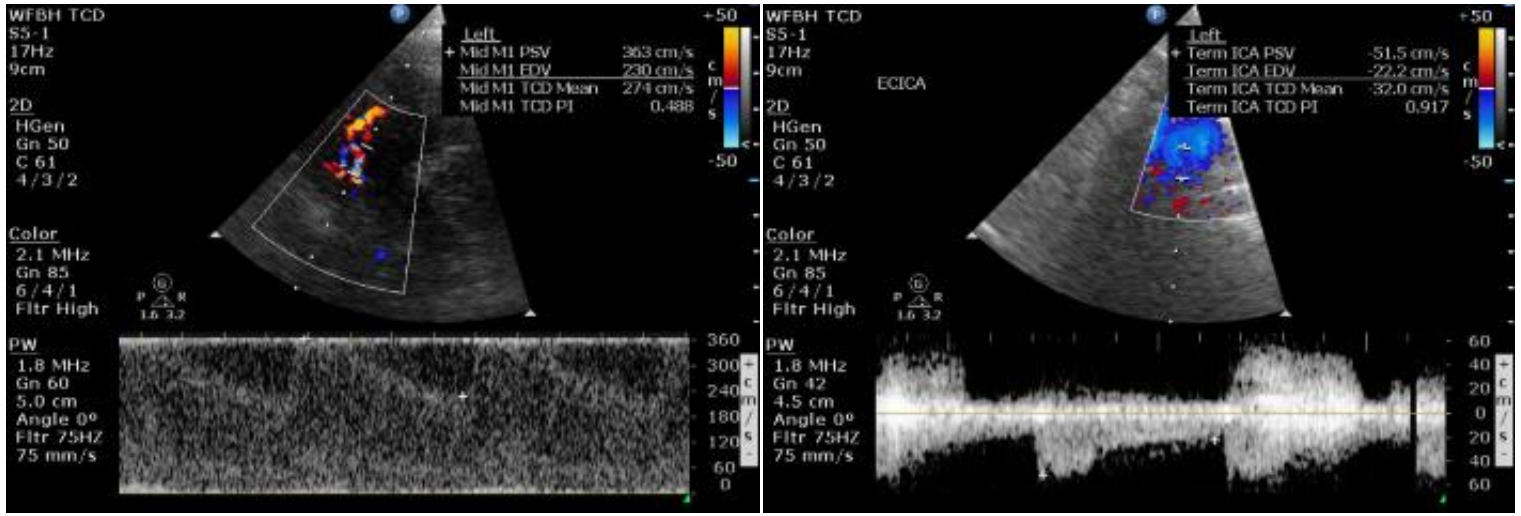
Mean flow velocity and PI





Lindegaard ratio

- Mean velocity MCA/ Mean velocity ipsilateral extracranial ICA
- Submandibular insonation ICA with TCD probe
- High velocity in MCA >120- hyperemia or vasospasm
  - <3 hyperemia
  - >3 mild spasm
  - 3-6 moderate spasm
  - >6 severe



- Post circulation LR (Sviri ratio or Soustiel index) cutoff 2

=Basilar MFV/Mean of L & R extracranial VA MFV

- Sloan's hemispheric ratio =ACA MFV/ECICA MFV

Kumar J of Ultrasound in Med 2015, LindegaardActaNeurochir1989,SviriNeurosurgery2006,SoustielStroke 2002

Grading vasospasm

Mean MCA Velocity	MCA/ICA Velocity ratio	Interpretation
< 120 cm/s	< 3	Normal, nonspecific elevation or distal MCA spasm
> 120 cm/s	3 – 6	Vasospasm of proximal MCA
> 200 cm/s	> 6	Severe spasm of proximal MCA

Table 3: Grading of vasospasm severity

Degree of MCA or ICA vasospasm	MFV (cm/s)		LR
Mild (<25%)	120–149	A	3–6
Moderate (25–50%)	150–199	N	3–6
Severe (>50%)	>200	D	>6
Degree of BA vasospasm	**MFV (cm/s)**		**Modified LR**
May represent vasospasm	70–85	A	2–2.49
Moderate (25–50%)	>85	N	2.5–2.99
Severe (>50%)	>85	D	>3

AaslidJ Neurosurg.1984

Vasospasm vs Hyperemia

MFV, cm/s	MCA/EC ICA MFV (Lindegaard) Ratio	Interpretation
<120	≤3	Hyperemia
>80	3–4	Hyperemia + possible mild spasm
≥120	3–4	Mild spasm + hyperemia
≥120	4–5	Moderate spasm + hyperemia
>120	5–6	Moderate spasm
≥180	6	Moderate-to-severe spasm
≥200	≥6	Severe spasm
>200	4–6	Moderate spasm + hyperemia
>200	3–4	Hyperemia + mild/residual spasm
>200	<3	Hyperemia

Artery	MFV, cm/s		
	Possible Vasospasm	Probable Vasospasm	Definite Vasospasm
ICA	>80	>110	>130
ACA[a]	>90	>110	>120
PCA	>60	>80	>90
VA	>60	>80	>90

MFV, cm/s	BA/EC VA MFV (Sviri) Ratio	Interpretation
>70	>2	Vasospasm
>85	>2.5	Moderate or severe vasospasm
>85	>3	Severe vasospasm

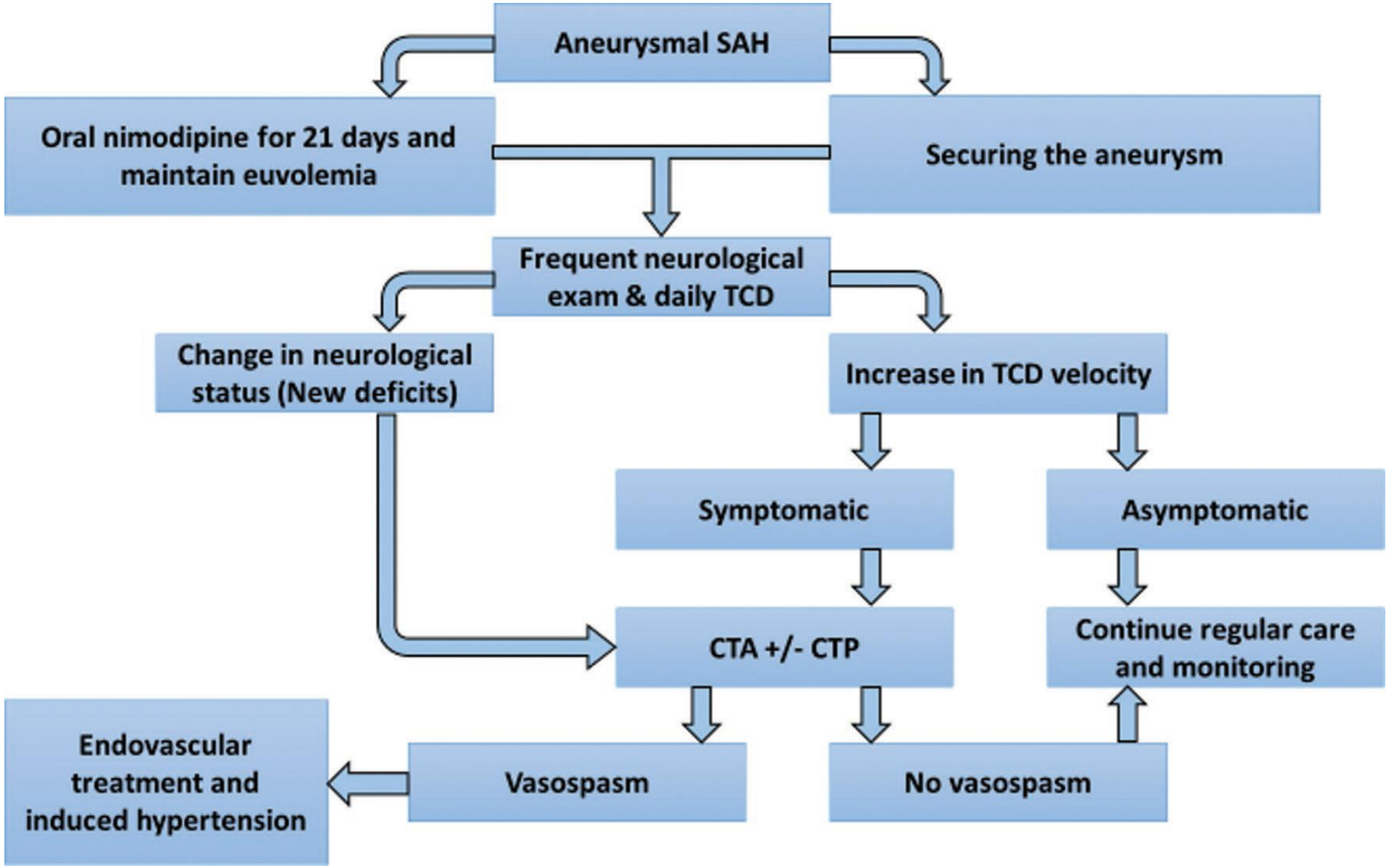
Kumar J of Ultrasound in Med 2015

Table 4: The prediction of cerebral vasospasm based on the various range of middle cerebral artery velocities

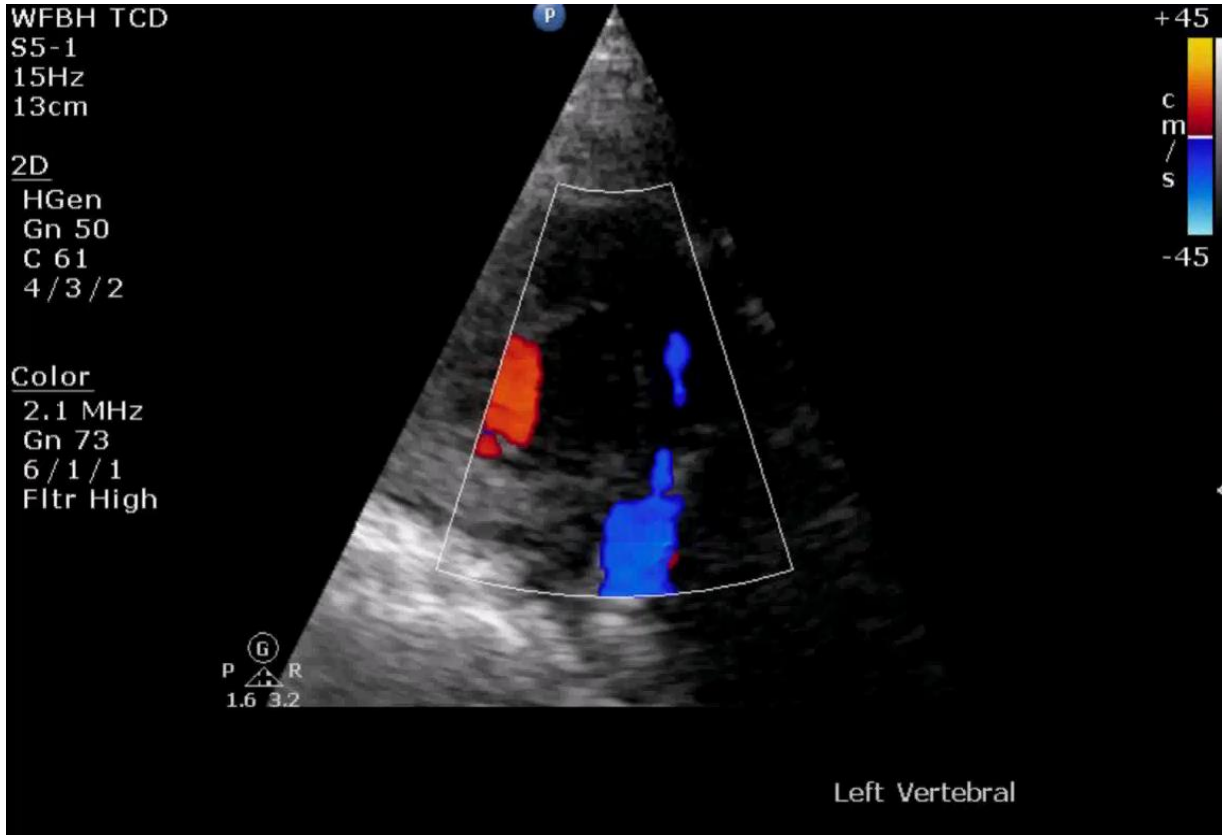
TCD velocity	Sensitivity	Specificity	LR+	LR−	PPV	NPV
<120	0.88	0.72	3.14	0.17*	0.55	0.94*
120-159	0.40	0.80	2.00	0.75	0.44	0.77
160-199	0.31	0.93	3.98	0.85	0.56	0.75
≥200	0.27	0.98	16.39*	0.74	0.87*	0.77

**Significant values. LR+=Positive likelihood ratio; LR−=Negative likelihood ratio; PPV=Positive predictive value; NPV=Negative predictive value*

SamaghJ AnaesthesiolClinPharmacol. 2019

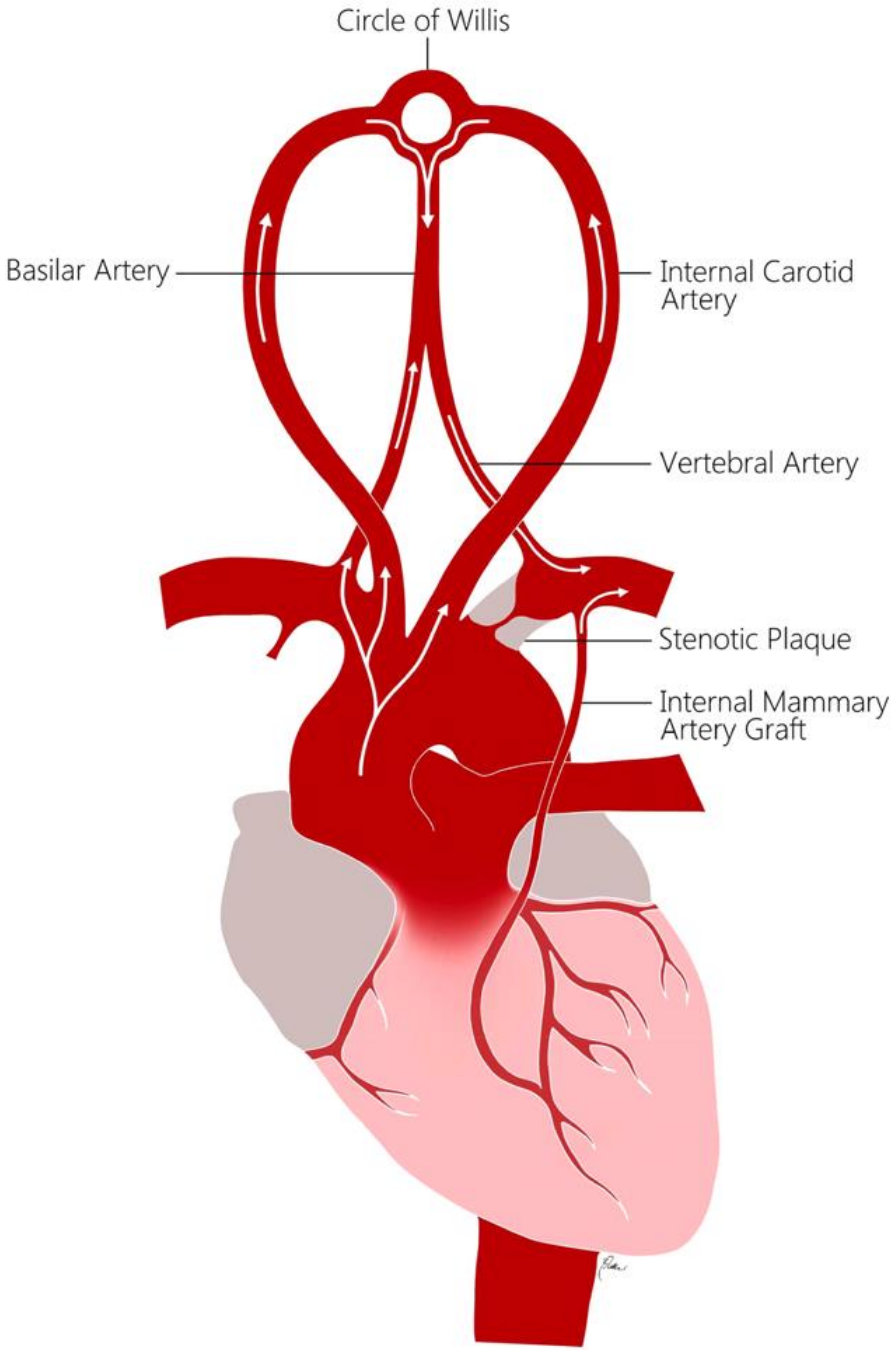

Aneurysmal SAH
Oral nimodipine for 21 days and maintain euvolemia
Securing the aneurysm
Frequent neurological exam & daily TCD
Change in neurological status (New deficits)
Increase in TCD velocity
Symptomatic
Asymptomatic
CTA +/- CTP
Continue regular care and monitoring
Endovascular treatment and induced hypertension
Vasospasm
No vasospasm

Vertebral Steal



Steal phenomena

- Vertebral steal- posterior circulation signs
- Coronary-Subclavian steal phenomenon if CABG and stenosis in the subclavian artery proximal to the takeoff of an IMA graft utilized to perfuse the heart



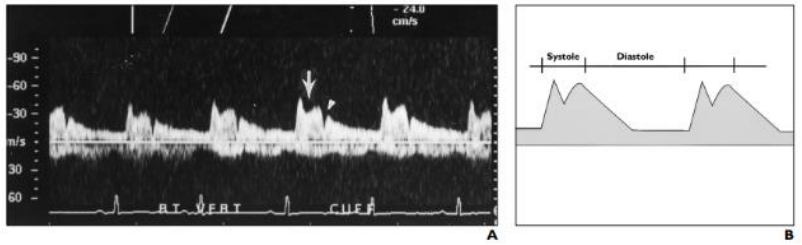


Fig. 1.—Type 1 waveform in 63-year-old woman with asymptomatic carotid bruit.
A, Doppler waveform shows antegrade flow throughout cardiac cycle. Note transient sharp decline in velocity at mid systole producing notch in Doppler trace (*arrow*). Notch creates two systolic peaks. First rises to acute angle and second has rounded contour. Velocity at nadir of notch is greater than that of end diastole. Also note diastolic notch (*arrowhead*).
B, Drawing shows timing of waveform fluctuations in cardiac cycle.

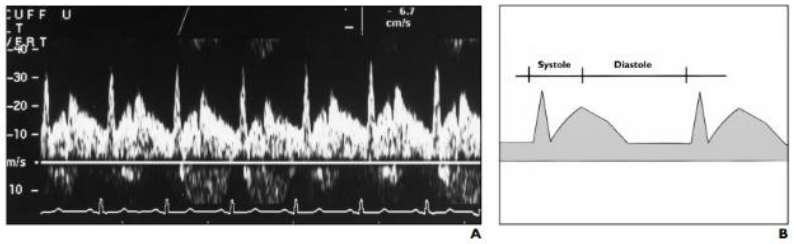


Fig. 2.—Type 2 waveform in 53-year-old woman with coronary artery disease.
A, In this waveform, more pronounced and deeper cleft is evident between two systolic peaks. Nadir of this cleft reached velocity at or just below that of end diastole. Second systolic peak tends to be smaller and broader than corresponding peak of type 1 waveform.
B, Drawing delineates waveform changes in cardiac cycle. Outline resembles body profile of rabbit and is sometimes referred to as "bunny waveform."

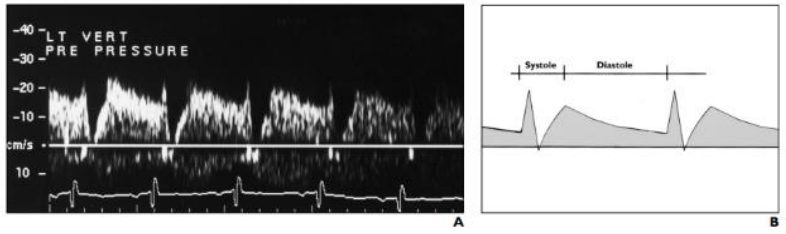


Fig. 3.—Type 3 waveform in 54-year-old man with angina.
A, Nadir of mid systolic cleft is at or below baseline, but rapid recovery of forward flow before diastole is shown.
B, Drawing shows waveform outline.

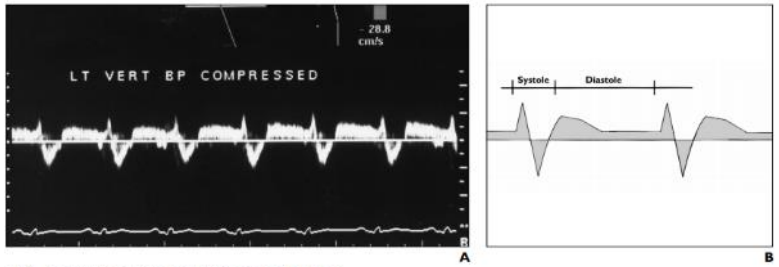
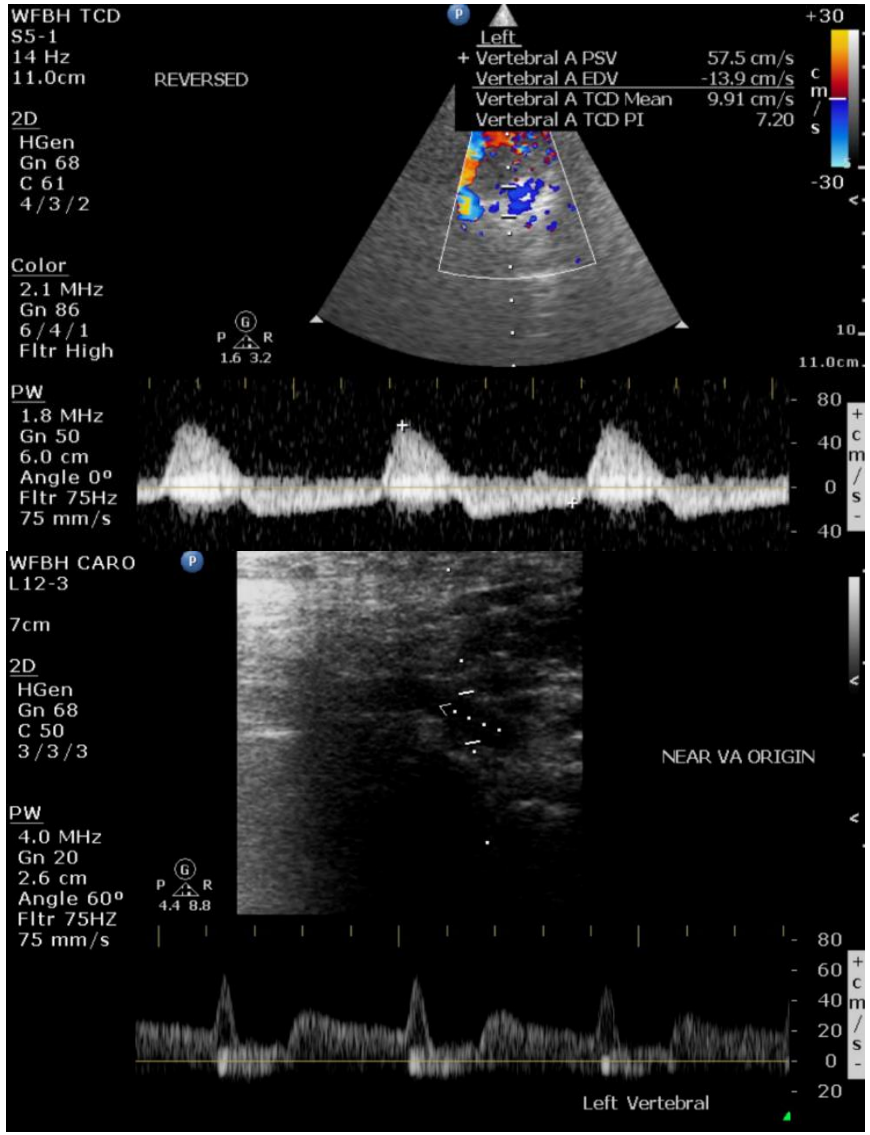


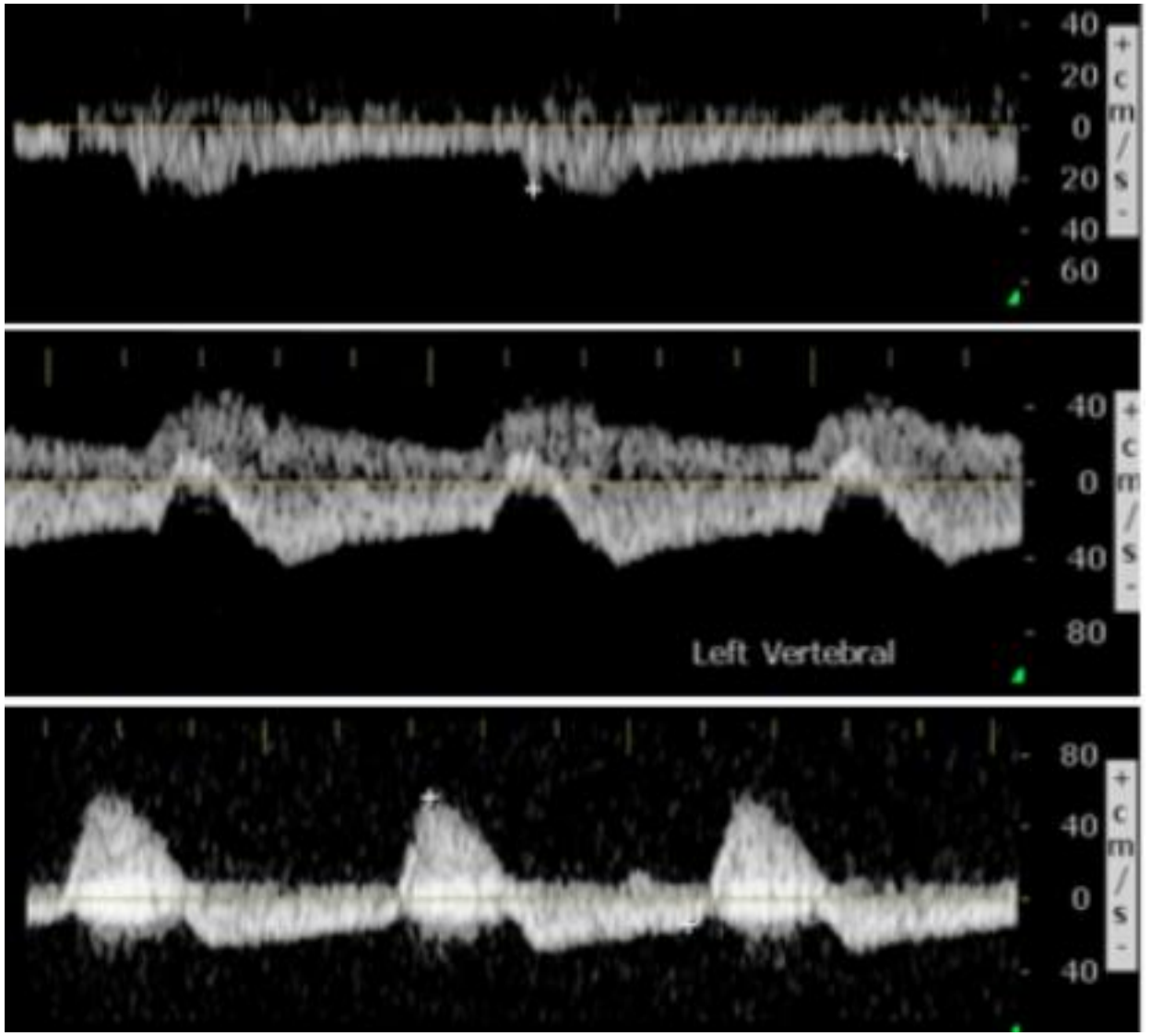
Fig. 4.—Type 4 waveform in 81-year-old man with coronary artery disease.
A, Nadir of mid systolic cleft falls well below baseline signifying greater reversal of flow during systole. Forward flow is restored in diastole.
B, Drawing indicates outline of pulse profile.

- 1914 vertebral arteries
- 40 transient sharp decline in velocities at mid or late systole. Reactive hyperemia induced in the ipsilateral arm with a blood pressure cuff.
- Same protocol performed in a control group of 52 patients with normal vertebral artery waveforms.
- Correlation between the waveforms and subclavian disease shown on angiography in 10 cases from prospective and 10 cases identified from a record search

Vertebral Artery Doppler Waveform Changes Indicating Subclavian Steal Physiology. AJR:174, March 2000

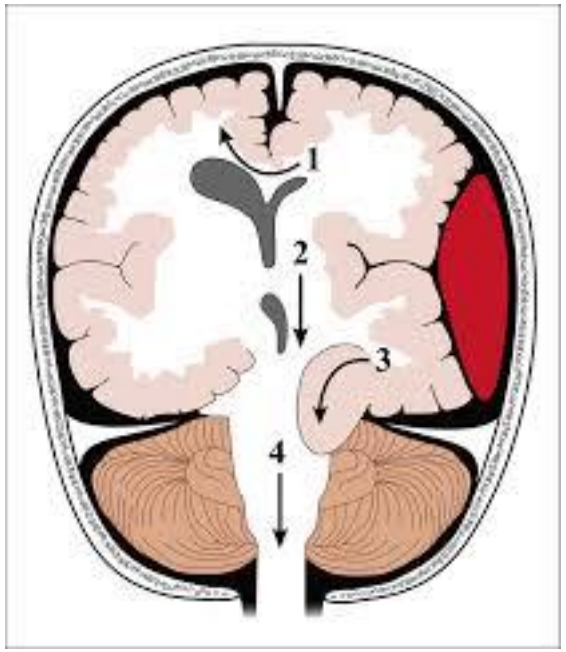
Steal

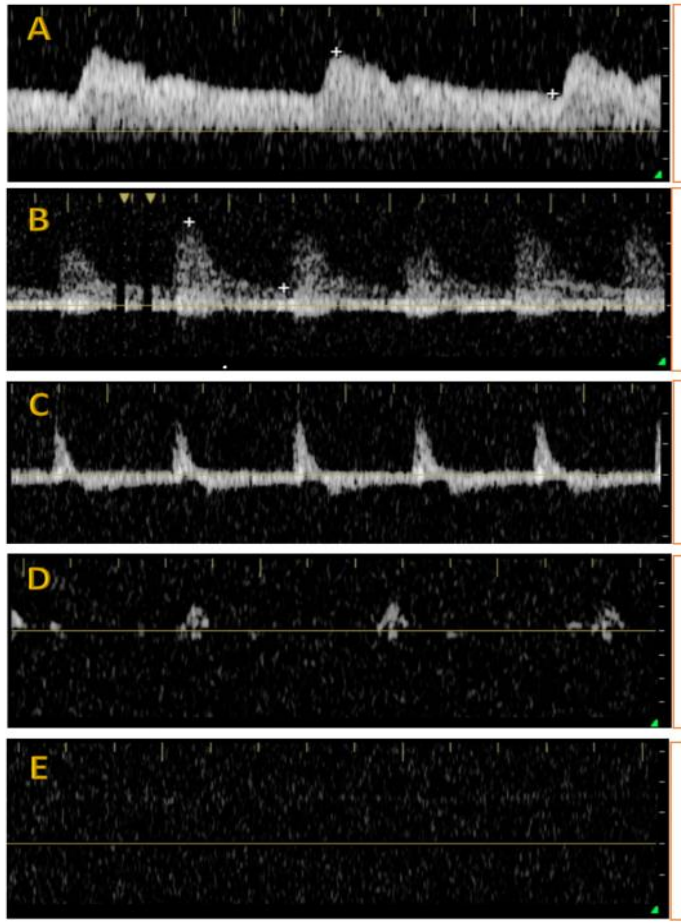

WFBH TCD
S5-1
14 Hz
11.0cm
REVERSED
2D
HGen
Gn 68
C 61
4/3/2
Color
2.1 MHz
Gn 86
6/4/1
Fltr High
PW
1.8 MHz
Gn 50
6.0 cm
Angle 0°
Fltr 75Hz
75 mm/s
Left
+ Vertebral A PSV 57.5 cm/s
Vertebral A EDV -13.9 cm/s
Vertebral A TCD Mean 9.91 cm/s
Vertebral A TCD PI 7.20
+30
-30
cm/s
1.6 3.2
11.0cm
80
40
0
40
WFBH CARO
L12-3
7cm
2D
HGen
Gn 68
C 50
3/3/3
PW
4.0 MHz
Gn 20
2.6 cm
Angle 60°
Fltr 75HZ
75 mm/s
4.4 8.8
NEAR VA ORIGIN
80
60
40
20
0
20
Left Vertebral


40
20
0
20
40
60
cm/s
40
0
40
80
Left Vertebral
80
40
0
40
cm/s

Cerebral Circulatory Arrest assessment

Resistance global increase





Normal arterial waveform
ICP significantly lower than diastolic BP

High resistance waveforms
ICP is elevated but lower than diastolic BP

Oscillating waveforms with reversed diastolic flow
ICP is elevated compared to diastolic BP but lower than systolic BP

Systolic spikes
ICP is significantly elevated compared to systolic BP

No flow visualized

Progressive increase in intracranial pressure

ELSEVIER

Journal of the Neurological Sciences 159 (1998) 145–150

JOURNAL OF THE NEUROLOGICAL SCIENCES

Consensus opinion on diagnosis of cerebral circulatory arrest using Doppler-sonography
Task Force Group on cerebral death of the Neurosonolgy Research Group of the World Federation of Neurology

Xavier Ducrocq[a], Werner Hassler[b], Kouzo Moritake[c], David W. Newell[d], Gerhard-Michael von Reutern[e,*], Toshiyuki Shiogai[f], Robert R. Smith[g]

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[g]*M.D. Methodist Neuroscience Associates, 1828 Hopstial Drive, Jackson, Mississippi 39204, USA*

Guidelines

- Prerequisites

1. The cause of coma has been established and is sufficient to account for a permanent loss of brain function.
2. Other conditions such as intoxication, hypothermia, arterial hypotension, metabolic disorders and others have been excluded.
3. Clinical evaluation by two experienced examiners shows no evidence of cerebral and brainstem function

- Criteria- Cerebral circulatory arrest can be confirmed if the following **extra- and intracranial Doppler sonographic findings** have been recorded and documented both intra and extracranially and **bilaterally on two examinations at an interval of at least 30 min.**

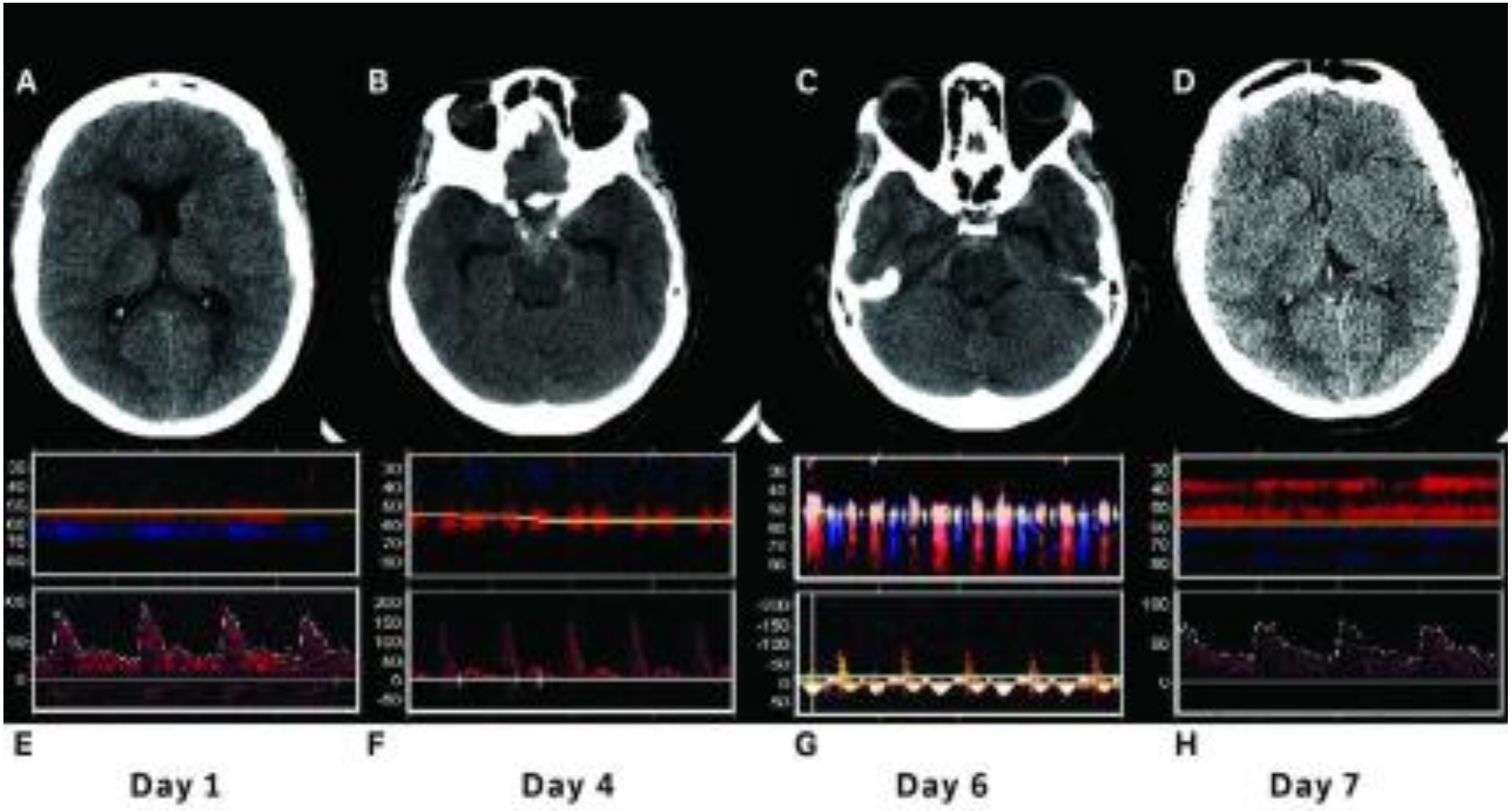
Guidelines

1. **Systolic spikes or oscillating flow** in any cerebral artery which can be recorded by **bilateral** transcranial insonation of the **ICA and MCA, respectively any branch or other artery which can be recorded (anterior and posterior circulation)**.
   a. Oscillating flow is defined by signals with forward and reverse flow components in one cardiac cycle exhibiting almost the same area under the envelope of the wave form (to and fro movement).
   b. Systolic spikes are sharp unidirectional velocity signals in early systole of **less than 200 ms duration, less than 50 cm /s peak systolic velocity,** and without a flow signal during the remaining cardiac cycle.
   c. Transitory patterns between oscillating flow and systolic spikes may be seen.
2. The diagnosis established by the intracranial examination must be **confirmed by the extracranial bilateral recording of the CCA, ICA and VA.**

Guidelines

3. The **lack of a signal** during transcranial insonation of the basal cerebral arteries is **not a reliable finding** because this can be due to transmission problems. But the **disappearance of intracranial flow signals in conjunction with typical extracranial signals can be accepted as proof of circulatory arrest**

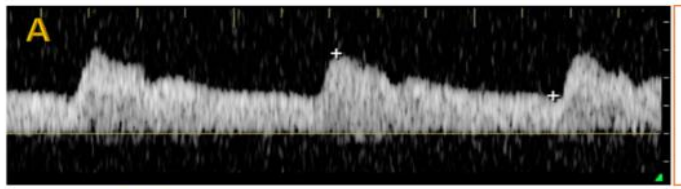
4. **Ventricular drains or large openings of the skull** like in decompressive craniectomy possibly interfering with the development of the ICP are **not present.**



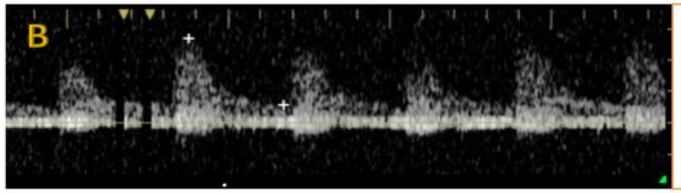
Journal of Ultrasound in Medicine
Volume 34, Issue 8, pages 1345-1350, 1 AUG 2015 DOI: 10.7863/ultra.34.8.1345
http://onlinelibrary.wiley.com/doi/10.7863/ultra.34.8.1345/full#jum20153481345-fig-0002

Noninvasive Intracranial Pressure Measurements

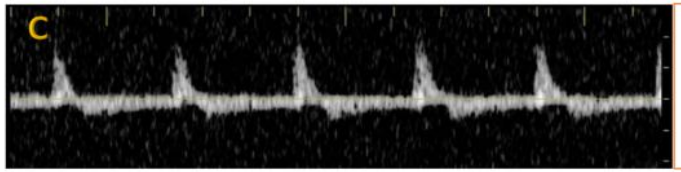
Waveforms during ICP crisis



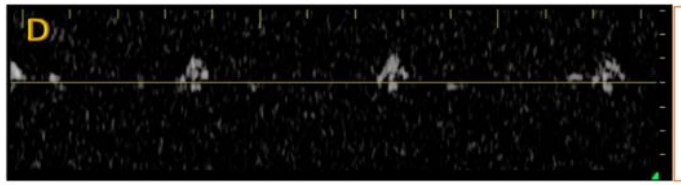
Normal arterial waveform
ICP significantly lower than diastolic BP



High resistance waveforms
ICP is elevated but lower than diastolic BP



Oscillating waveforms with reversed diastolic flow
ICP is elevated compared to diastolic BP but lower than systolic BP



Systolic spikes
ICP is significantly elevated compared to systolic BP

No flow visualized

Progressive increase in intracranial pressure

Brain Blood Interactions

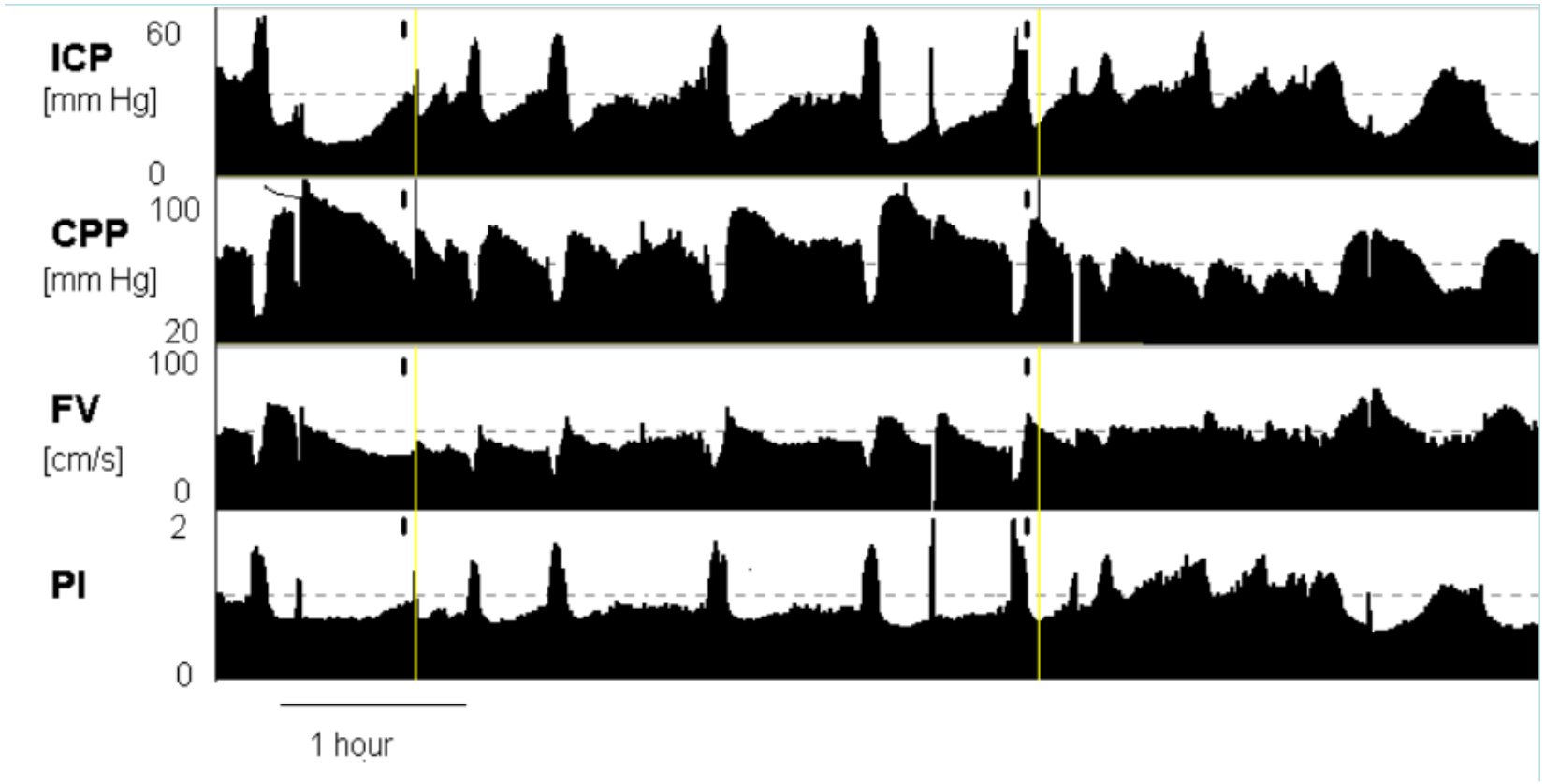
Serial assessment of resistance

Pulsatility index

PSV-EDV

Mean

Prognostic value explored in TBI and post arrest ROSC



CPP estimation from TCD

Bellner
nICP= 10.93*(PI) - 1.28

- nICP_Aaslid

$$\mathrm{nCPP} = \mathrm{FVm} * A/F.$$

- nICP_Schmidt

$$\mathrm{nCPP} = \mathrm{MAP} * \frac{\mathrm{FVd}}{\mathrm{FVm}} + 14\ \mathrm{mmHg}$$

- nICP_Edouard

$$\mathrm{nCPP} = \left(\frac{\mathrm{FVm}}{[\mathrm{FVm} - \mathrm{FVd}]}\right) \times (\mathrm{ABPm} - \mathrm{ABPd})$$

- nICP_ CrCP

$$\mathrm{nCPP} = \mathrm{ABP} \times \left[0.734 - \frac{0.266}{\sqrt{(\mathrm{CVR} \times \mathrm{Ca} \times \mathrm{HR} \times 2\pi)^2 + 1}}\right] - 7.026$$

neurocritical care society Neurocrit Care (2016) 25:473–491
DOI 10.1007/s12028-016-0258-6

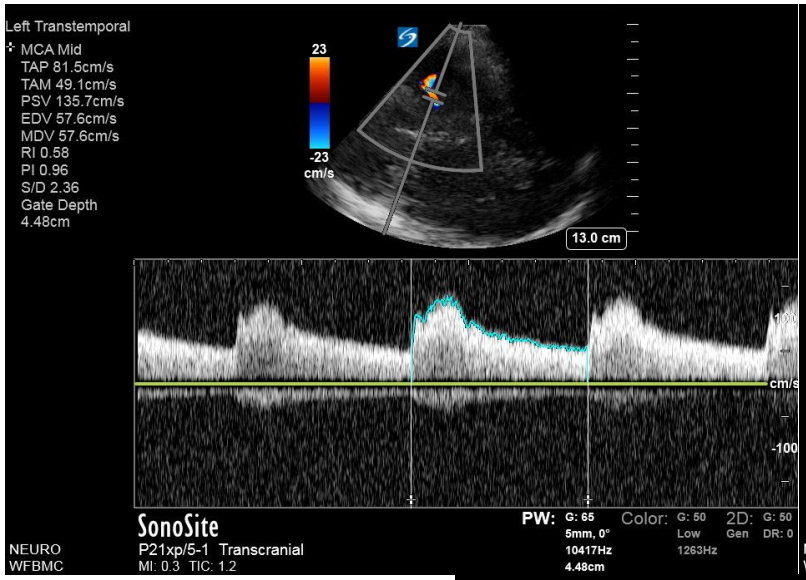
REVIEW ARTICLE

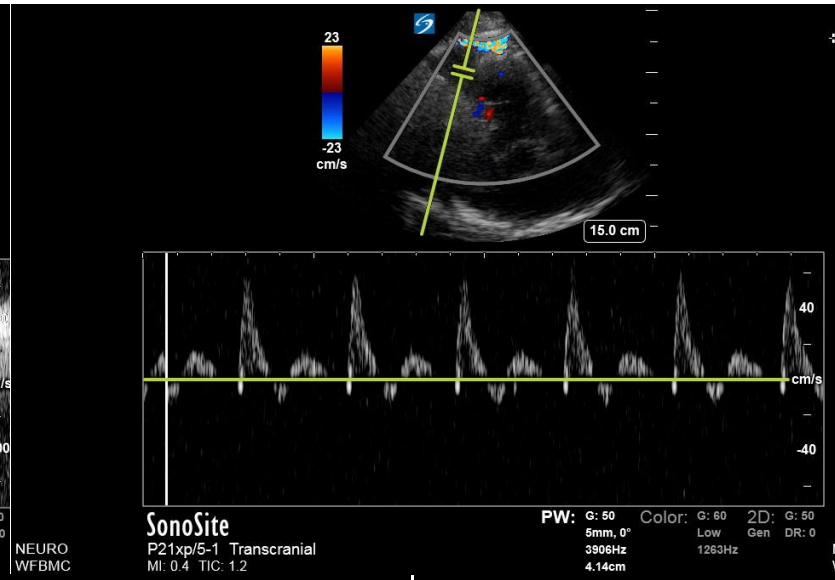
Non-invasive Monitoring of Intracranial Pressure Using Transcranial Doppler Ultrasonography: Is It Possible?

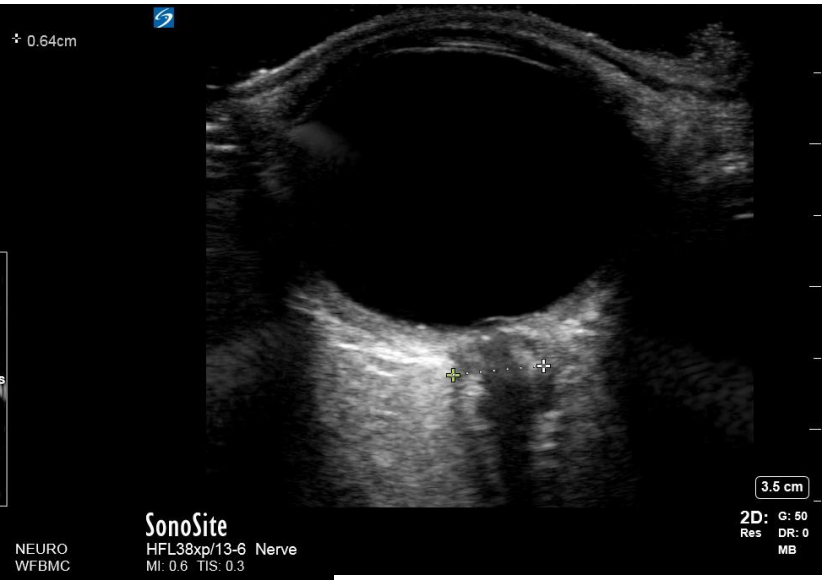
Danilo Cardim[1] · C. Robba[2] · M. Bohdanowicz[3] · J. Donnelly[1] · B. Cabella[1] · X. Liu[1] · M. Cabeleira[1] · P. Smielewski[1] · B. Schmidt[4] · M. Czosnyka[1]

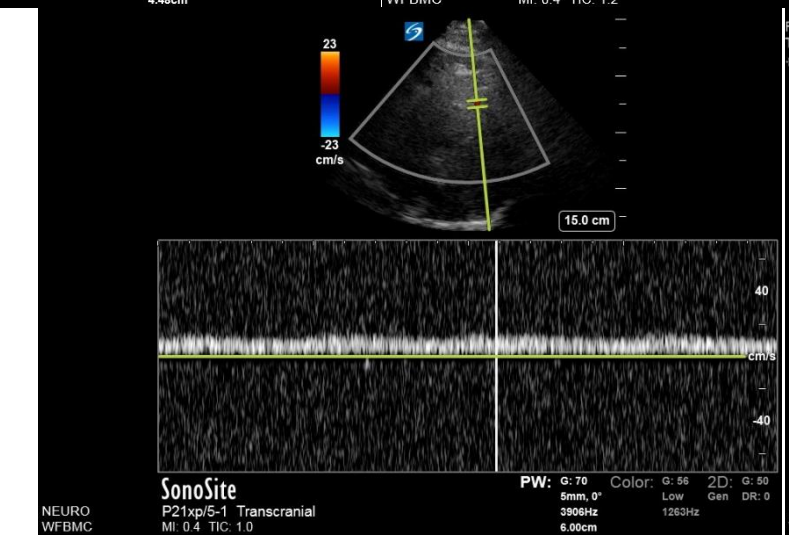
J Neurol Neurosurg Psychiatry 70(2):198–204 Br J Anaesth. 2005;94(2):216–21.

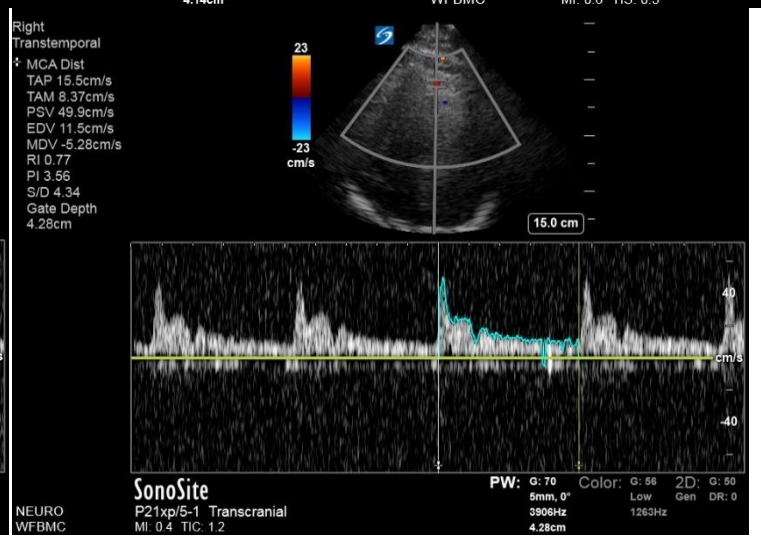
Case study

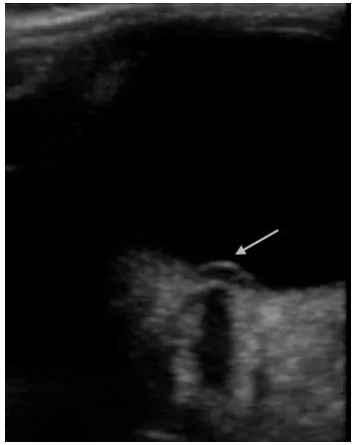

Left Transtemporal
MCA Mid
TAP 81.5cm/s
TAM 49.1cm/s
PSV 135.7cm/s
EDV 57.6cm/s
MDV 57.6cm/s
RI 0.58
PI 0.96
S/D 2.36
Gate Depth
4.48cm
23
-23
cm/s
13.0 cm
100
cm/s
-100
SonoSite
P21xp/5-1 Transcranial
MI: 0.3 TIC: 1.2
PW: G: 65 5mm, 0° 10417Hz 4.48cm
Color: G: 50 Low 1263Hz
2D: G: 50 Gen DR: 0
NEURO
WFBMC


23
-23
cm/s
15.0 cm
40
cm/s
-40
SonoSite
P21xp/5-1 Transcranial
MI: 0.4 TIC: 1.2
PW: G: 50 5mm, 0° 3906Hz 4.14cm
Color: G: 60 Low 1263Hz
2D: G: 50 Gen DR: 0
NEURO
WFBMC

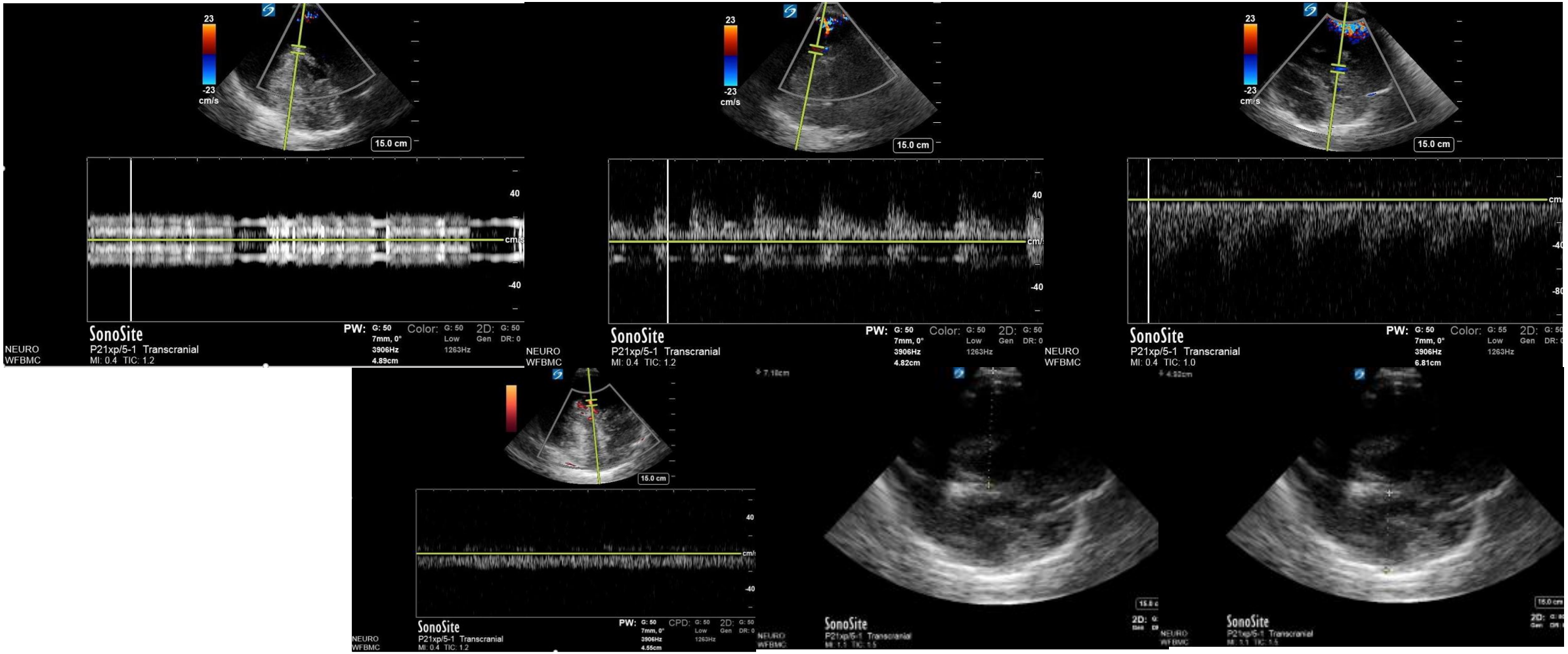

0.64cm
3.5 cm
SonoSite
HFL38xp/13-6 Nerve
MI: 0.6 TIS: 0.3
2D: G: 50 Res DR: 0 MB
NEURO
WFBMC


23
-23
cm/s
15.0 cm
40
cm/s
-40
SonoSite
P21xp/5-1 Transcranial
MI: 0.4 TIC: 1.0
PW: G: 70 5mm, 0° 3906Hz 6.00cm
Color: G: 56 Low 1263Hz
2D: G: 50 Gen DR: 0
NEURO
WFBMC

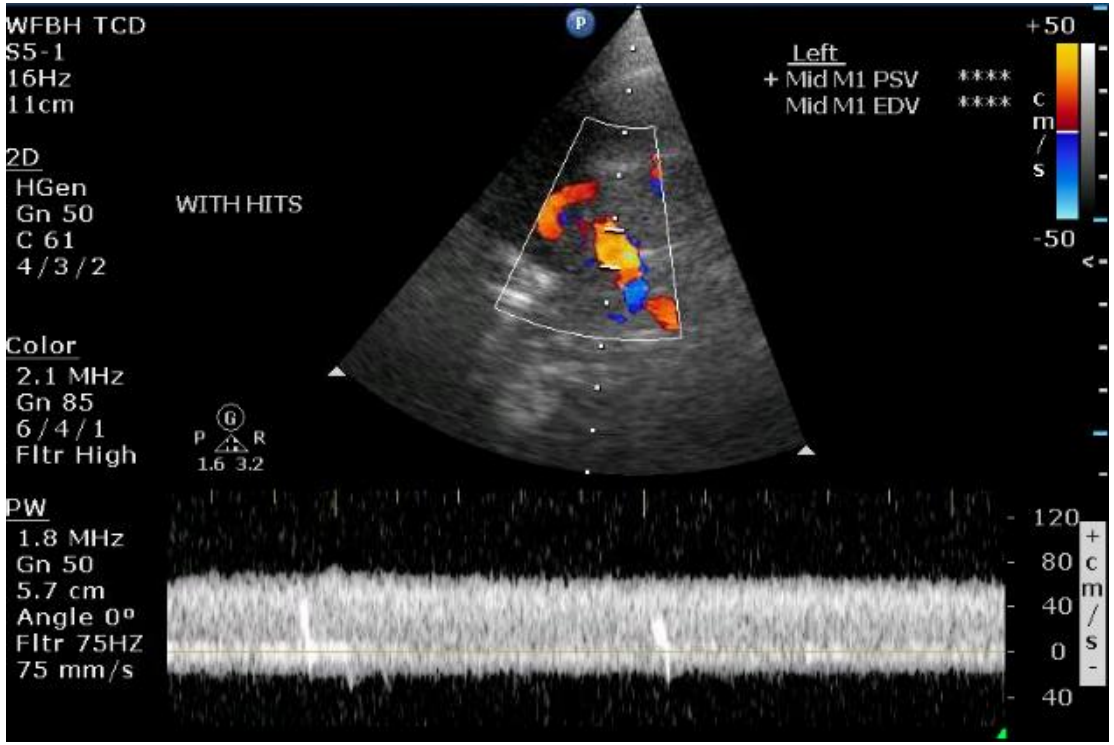

Right
Transtemporal
MCA Dist
TAP 15.5cm/s
TAM 8.37cm/s
PSV 49.9cm/s
EDV 11.5cm/s
MDV -5.28cm/s
RI 0.77
PI 3.56
S/D 4.34
Gate Depth
4.28cm
23
-23
cm/s
15.0 cm
40
cm/s
-40
SonoSite
P21xp/5-1 Transcranial
MI: 0.4 TIC: 1.2
PW: G: 70 5mm, 0° 3906Hz 4.28cm
Color: G: 56 Low 1263Hz
2D: G: 50 Gen DR: 0
NEURO
WFBMC

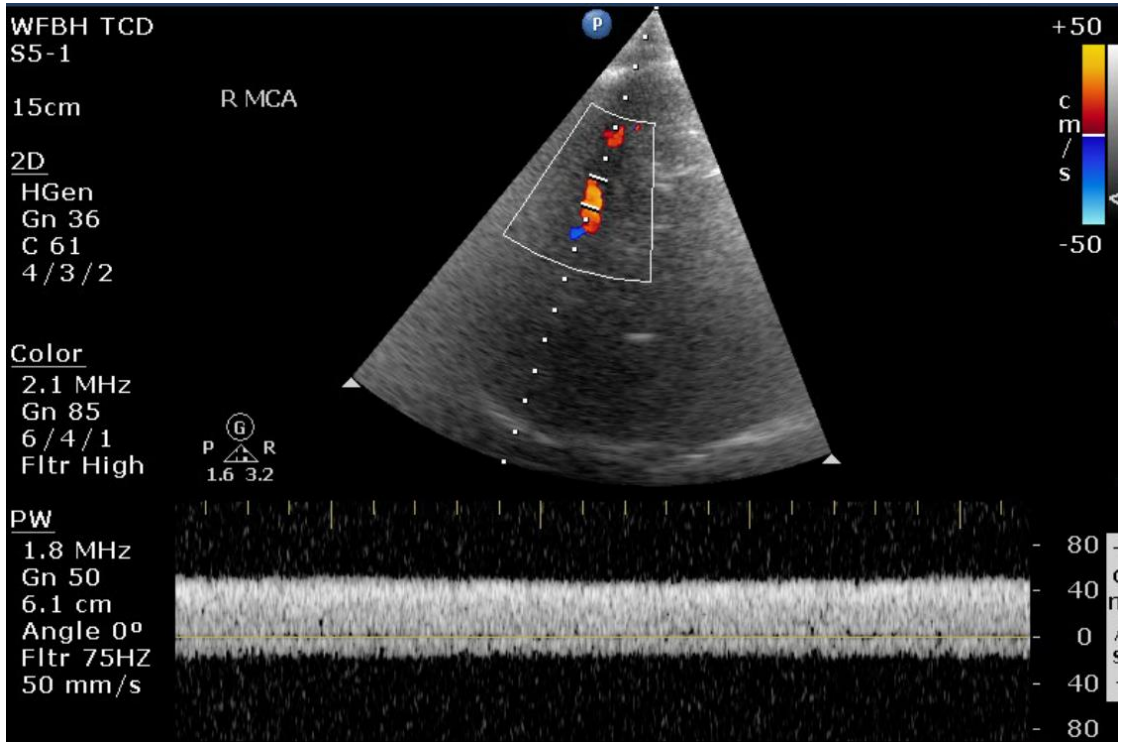


Case study


23
-23
cm/s
15.0 cm
40
-40
SonoSite
P21xp/5-1 Transcranial
MI: 0.4 TIC: 1.2
PW: G: 50
7mm, 0°
3906Hz
4.89cm
Color: G: 50 Low 1263Hz
2D: G: 50 Gen DR: 0
NEURO
WFBMC
SonoSite
P21xp/5-1 Transcranial
MI: 0.4 TIC: 1.2
PW: G: 50
7mm, 0°
3906Hz
4.82cm
Color: G: 50 Low 1263Hz
2D: G: 50 Gen DR: 0
NEURO
WFBMC
SonoSite
P21xp/5-1 Transcranial
MI: 0.4 TIC: 1.0
PW: G: 50
7mm, 0°
3906Hz
6.81cm
Color: G: 55 Low 1263Hz
2D: Gen
NEURO
WFBMC
SonoSite
P21xp/5-1 Transcranial
MI: 0.4 TIC: 1.2
PW: G: 50
7mm, 0°
3906Hz
4.55cm
CPD: G: 50 Low 1263Hz
2D: G: 50 Gen DR: 0
NEURO
WFBMC
SonoSite
P21xp/5-1 Transcranial
NEURO
WFBMC
SonoSite
P21xp/5-1 Transcranial
NEURO
WFBMC

Case study


WFBH TCD
S5-1
16Hz
11cm
2D
HGen
Gn 50
C 61
4/3/2
WITH HITS
Color
2.1 MHz
Gn 85
6/4/1
Fltr High
PW
1.8 MHz
Gn 50
5.7 cm
Angle 0°
Fltr 75HZ
75 mm/s
Left
+ Mid M1 PSV ****
Mid M1 EDV ****
+50
cm/s
-50
120
80
40
0
40
1.6 3.2


WFBH TCD
S5-1
15cm
R MCA
2D
HGen
Gn 36
C 61
4/3/2
Color
2.1 MHz
Gn 85
6/4/1
Fltr High
PW
1.8 MHz
Gn 50
6.1 cm
Angle 0°
Fltr 75HZ
50 mm/s
+50
cm/s
-50
80
40
0
40
80
1.6 3.2

Coding and Billing

Neurosonology CPT Codes

- **93875** Physiological testing extracranial arteries, complete bilateral study
- **93880** Duplex scan extracranial arteries, complete bilateral study
- **93882** Limited or unilateral duplex
- **93886** TCD intracranial, complete study
- **93888** TCD intracranial limited study
- **93890** Vasoreactivity study
- **93892** Embolus Detection without bubbles
- **93893** Embolus Detection with bubbles

Neurosonology CPT Codes

- Complete TCD: Includes attempted ultrasound evaluation of 3 circulations; the right and left anterior circulation by the temporal acoustic window and the posterior circulation including the vertebral and basilar arteries
- Anything less is limited TCD

- ICD coding is crucial to support medical necessity for CPT code
- Acceptable codes vary between carriers- Listed in LMRP/LCD for Medicare Carrier
- Must learn/know what is acceptable, and know denial and appeal policies
- Algorithms can be helpful

Acceptable ICD-9 Codes for TCD- NC Medicare

- 282.5
- 282.6-282.69
- 348.8
- 430
- 433.00- 433.01
- 433.10- 433.11
- 433.20- 433.21
- 434.00- 434.91
- 434.01, 434.11
- 435.9
- 436
- 447.1
- 747.81

Sickle cell trait
Sickle cell anemia
Other condition of brain… brain death
SAH
Occlusion/stenosis basilar artery (without/with stroke)
Occlusion/stenosis carotid artery (without/with stroke)
Occlusion/stenosis vert. Artery (without/with stroke)
Occlusion of cerebral arteries
Cerebral thrombosis, cardioembolic stroke
Unspecified transient cerebral ischemia (TIA)
Acute, ill-defined cerebrovascular disease
Stricture of intracranial artery
Anomalies of cerebrovascular system (AVM)

Keys to Reimbursement

- Correct coding of procedure done
- Correct, reimbursable diagnosis code
- Must list indication and ICD code in the report itself
- Be familiar with your local Medicare carrier medical review policy-Carrier Advisory Committee-reps from State Medical Societies and others
- Know local policies for denials and appeals policy

Key Websites

- American Society of Neuroimaging: www.asnweb.org
- Neurosonology Research Group of the World Federation of Neurology: www.nsrg.org.tw
- Intersocietal Commission of the Accreditation of Vascular Laboratories: www.icavl.org
- American Institute of Ultrasound in Medicine: www.aium.org
- American Registry of Diagnostic Medical Sonography: www.ardms.org

The art of medicine consists of amusing the patient while nature cures the disease….

-

Voltaire

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