Anatomy, Principles, and Techniques for Transcranial Doppler

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Principles of Doppler ultrasound

- \( F_d = 2 \cdot f_0 \cdot v \cdot \cos \theta / c \), where \( f_0 \) = source frequency, \( v \) = scatter speed, \( c \) = propagation speed
- \( V (cm) = \frac{77 \cdot f_d (kHz)}{f_0 (MHz)} \cdot \cos \theta_D \)
Vascular Doppler

Spectral Analysis Parameters

- Flow direction
- Peak systolic velocity
- End-diastolic velocity
- Spectral pattern

Acoustic properties of the skull

- Three layers
  - Middle layer (dipole): important on the attenuation and scattering of the sound
  - Outer and inner tables: refraction
- Temporal region: absence of bony spicules
- Power loss depends on the thickness of the skull
Circle of Willis

Insonation Depth

Insonation Depth
Criteria for vessel identification I

1. Insonation depth
2. Direction of the flow
3. Traceability of vessels
4. Flow velocities
5. The site of the probe's position
   - Temporal
   - Orbital
   - Suboccipital
   - Submandibular

Criteria for vessel identification II

6. Spatial relationships
7. Transducer angle
8. Direction of the ultrasonic beam
   - posterior, anterior, caudad, or cephalad
9. Response to carotid oscillation or compression

Criteria for Normal TCD

<table>
<thead>
<tr>
<th>Artery</th>
<th>Depth(mm)</th>
<th>Direction</th>
<th>MFV(Cm/s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>M2-M1</td>
<td>40 - 65</td>
<td>➞</td>
<td>&lt; 80</td>
</tr>
<tr>
<td>MCA</td>
<td>62 - 75</td>
<td>➞</td>
<td>&lt; 80</td>
</tr>
<tr>
<td>A1-ACA</td>
<td>60 - 64</td>
<td>➞</td>
<td>&lt; 70</td>
</tr>
<tr>
<td>ICA Siphon</td>
<td>50 - 62</td>
<td>➞</td>
<td>Variable</td>
</tr>
<tr>
<td>OA</td>
<td>60 - 68</td>
<td>➞</td>
<td>&lt; 50</td>
</tr>
<tr>
<td>BA</td>
<td>80 - 100</td>
<td>➞</td>
<td>&lt; 60</td>
</tr>
<tr>
<td>VA</td>
<td>45 - 80</td>
<td>➞</td>
<td>&lt; 50</td>
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</table>

Parameter

- Peak systolic flow velocity(PV)
  - The maximum value of flow velocity in systole, at the apex of the waveform
- End-diastolic flow velocity(EDV)
  - The velocity measured at end diastole, usually at the lowest point before a new waveform begins
- Mean flow velocity(MV)
  - Estimated as the average of the edge frequency over a cardiac cycle
  - The edge frequency is the envelope of instantaneous peak velocities throughout the course of a cardiac cycle
  - MV=(PV+2EDV)/3 = EDV+(PV-EDV)/3

TCD waveform
**Time-averaged Mean of Maximum Velocity**
- The time mean of the peak velocity envelope, the envelope being a trace of the peak velocity as a function of time
- Automatic electronic measurements: TAMMX, TAMX (Time average of the maximum; Siemens, Acuson), TAP (Time average peak; Philips, ATL)
- Manual tracing
- Manual Measurement: a horizontal line or cursor can be placed so that the area above the line and under the peak of the waveform is the same as the area below the line and above the waveform outline

**Indices**
- Pulsatility Index (PI)
- Resistance Index (RI)
- Flow Acceleration (FA)

**Pulsatility Index I**
- Gosling and King
- PI = PV - EDV/MV
- The shape of the waveforms as displayed by TCD equipment
  - Rounded waveform: a lower PI
  - Peaked waveform: a higher PI
- An estimate of downstream vascular resistance
  - Low-resistance vascular beds: low PI (PI = PV - EDV/MV)
  - High-resistance vascular beds: high PI → Peripheral vessels
Pulsatility Index II

- The low PI of the cerebral vasculature
- The brain's unique metabolic needs
- The brain requires continuous blood flow throughout the cardiac cycle
- High diastolic flow with low downstream resistance
- Ophthalmic artery: low EDV, high downstream resistance, high PI
- ICA siphon: high EDV, low downstream resistance, low PI

Resistance Index

- Pourcelot
- Measure downstream vascular resistance
- \( RI = \frac{PV - EDV}{PV} \)
- Increased RI
  - Reflect increased downstream vascular resistance
  - \( 0.56 (0.07) \)
  - Elevated RI ( > 0.6 ) : Increased resistance to flow

Flow Acceleration

- The inclination or slope of the systolic upstroke of the waveform
- Flow acceleration (FA) \( = \frac{PV - EDV}{\Delta t} \)
- Low FA: increased upstream resistance, such as severe proximal ICA stenosis, aortic stenosis, or decreased cardiac performance

Lindegaard Index or Ratio

- The MCA-ICA flow velocity ratio; VMCA/VICA
- Distinguish vasospasm from states of systemic hyperemia
- ICA blood flow: measure from the neck, insonation depth of 40 to 50 mm
- Normal: 1.76 (0.10)
- Increased index of at least 3: Consistent with angiographic vasospasm in the MCA in patients with subarachnoid hemorrhage

Stenosis of Large Cerebral Artery

- Typical Features of stenosis of large basal cerebral artery
  - Acceleration of flow: increased flow velocity
  - Disturbed flow
  - Co-variation phenomena
- Mild stenosis
  - Increased in peak velocity with minimal change in the rest of the Doppler pattern
- Moderate to severe stenosis
  - Greater increase in peak velocity with spectral broadening, increase diastolic velocity, turbulent flow, poststenotic drop in peak velocity

Abnormal Waveforms

- Dampened signal: Pulsatile flow with normal flow acceleration and decreased MFV (30% difference between hemispheres); any PI values
- Blunted signal: Delayed flow acceleration with stepwise maximum velocity arrival during mid to late systole compared with contralateral side and focal decreased MFV and positive end-diastolic flow (low PI <= 1.1).
- Minimal signal: Presence of a flow signal with no end diastolic flow; PI >= 1.2.
- Absent signal: No detectable flow
Reversed flow in ipsilateral ACA

Increased velocity of contralateral ACA; ACA > MCA by at least 25%

Reversed ophthalmic artery

Decreased pulsatility in ophthalmic artery: internalization of ophthalmic artery

• The presence of any one of these parameters ⇒ the battery “positive”
• a 95% sensitivity for identifying greater than 70% ICA stenosis (NASCET)

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
An average rate of rise in Flow Velocities of > 20 cm/s/day between days 3 and 7 after SAH

A rapid early rise in FVs ( > 25%/day)

A mean absolute rise in MCA-FVs or ACA-FVs of 65±5 cm/s over 24-hour period and a higher VMCA/VICA ratio (6±0.2)

Specific clinical application of TCD

<table>
<thead>
<tr>
<th>Applications</th>
<th>Rating</th>
<th>Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sickle cell disease</td>
<td>Effective</td>
<td>Class I</td>
</tr>
<tr>
<td>Ischemic cerebrovascular disease</td>
<td>Established</td>
<td>Class II</td>
</tr>
<tr>
<td>Subarachnoid hemorrhage</td>
<td>Established</td>
<td>Class II</td>
</tr>
<tr>
<td>Aneurysms/malformations</td>
<td>Established</td>
<td>Class III</td>
</tr>
<tr>
<td>Cerebral circulatory arrest</td>
<td>Established</td>
<td>Class III</td>
</tr>
<tr>
<td>Perioperative monitoring</td>
<td>Possibly useful</td>
<td>Class III</td>
</tr>
<tr>
<td>Meningeal infection</td>
<td>Possibly useful</td>
<td>Class III</td>
</tr>
<tr>
<td>Periprocedural monitoring</td>
<td>Investigational</td>
<td>Class III</td>
</tr>
<tr>
<td>Migraine</td>
<td>Doubtful</td>
<td>Class II</td>
</tr>
<tr>
<td>Cerebral venous thrombosis</td>
<td>Doubtful</td>
<td>Class III</td>
</tr>
</tbody>
</table>

Applications Rating Evidence

Clinical application of TCD

Application

Screening of children aged 2-16 years with sickle cell disease for assessing stroke risk

Detection and monitoring of angiographic vasospasm after spontaneous subarachnoid hemorrhage

Cerebral Thrombolysis: monitoring thrombolysis of acute MCA occlusions

Cerebral Microembolism Detection: the detection of cerebral microembolic signals in a variety of cardiovascular/ cerebrovascular disorders/procedures

Application of TCD

Functional tests
- blood flow velocity during activation of circumscribed cortical areas: light and mental stimulation of the visual cortex, etc
- Noninvasive ancillary tests and monitoring procedures in animal experiments
- Monitoring during experiments in space.

Application

Diagnosis of intracranial occlusive disease

Ancillary test for confirmation or exclusion of extracranial occlusive disease
- Confirmation of well-collateralized chronic ICA occlusions
- Diagnosis and follow-up of internal carotid artery
- Evaluation of hemodynamic effects of extracranial occlusive disease on intracranial blood flow velocities
  - ICA stenosis or ICA occlusion
  - Subclavian steal mechanism
  - Extracranial lesions

Application

TCD monitoring: probably useful to detect hemodynamic and embolic events that may result in perioperative stroke during and after carotid endarterectomy in settings where monitoring is felt to be necessary

Monitoring during surgery for hemodynamic status

Vasomotor Reactivity Testing

Detection of right-to-left shunts
Cerebrovascular Disease

- **Indication**
  - Ischemic stroke
  - Transient ischemic attack
  - Asymptomatic patients with high risk

- **Usage**
  - Proximal intracranial arterial stenosis
  - Arterial occlusion
  - Collaterals
  - Evidence of microembolization
  - Proving recanalization
  - Enhancement of thrombolysis

Transcranial Color Duplex

- **Transcranial Doppler with Imaging**

Transcranial Color Doppler

- **Anatomical information**

References

- CH Tegeler Neurosonology 1996
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- Cerebrovascular ultrasound in stroke prevention and treatment
Noninvasive transcranial Doppler ultrasound recording of flow velocity in basal cerebral arteries

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In this report the authors describe a noninvasive transcranial method of determining the flow velocities in the basal cerebral arteries. Placement of the probe of a range-gated ultrasound Doppler instrument in the temporal area just above the zygomatic arch allowed the velocities in the middle cerebral artery (MCA) to be determined from the Doppler signals. The flow velocities in the proximal anterior (ACA) and posterior (PCA) cerebral arteries were also recorded at steady state and during test compression of the common carotid arteries. An investigation of 50 healthy subjects by this transcranial Doppler method revealed that the velocity in the MCA, ACA, and PCA was 62 ± 12, 51 ± 12, and 44 ± 11 cm/sec, respectively. This method is of particular value for the detection of vasospasm following subarachnoid hemorrhage and for evaluating the cerebral circulation in occlusive disease of the carotid and vertebral arteries.

KEY WORDS • arterial flow velocity • collateral flow • ultrasonics • transcranial Doppler ultrasound • cerebral arteries • internal carotid artery • middle cerebral artery

Doppler ultrasound recording of the blood flow velocity in the extracranial arteries supplying the brain was reported by Miyazaki and Kato in 1965 and is now used routinely in neurological and neurosurgical practice. The velocity in the intracranial vessels has been observed by Doppler technique during surgery, and in children with open fontanelles. In adults, however, the skull is a severe obstacle to the penetration of ultrasound. Bone strongly attenuates the ultrasonic wave, making it impossible to record noninvasively the blood flow velocity from intracranial arteries by conventional Doppler instruments operating in the range from 5 to 10 MHz. At lower frequencies, 1 to 2 MHz, the attenuation in bone and soft tissues is considerably less. The skull bones are of varying thickness, and because the bone of the temporal region is thin, this would appear to be the most promising area for penetration of ultrasound. In fact, determination of midline deviation, using echo techniques, has demonstrated that some penetration of ultrasound is possible.

The present study investigates the blood flow velocities in the middle, anterior, and posterior cerebral arteries (MCA, ACA, and PCA) using a noninvasive transcranial Doppler ultrasound technique.

Clinical Material and Methods

Fifty healthy subjects with no history of cerebral vascular disease were investigated. Their ages ranged from 20 to 65 years, with a mean of 36 years.

For the present study we used a laboratory prototype range-gated Doppler instrument with the following characteristics. Emitted ultrasonic frequency 2 MHz; burst repetition rate 6.8 to 18 kHz; burst length 10 μsec; high pass filter 100 Hz; low pass filter 3.4 to 9 kHz; and emitted ultrasonic power 350 mW. The effective range for this apparatus is from 3.0 to 10 cm. Sampling can be done at preselected distances from the probe within this range by means of a gating system.

The emitting area of the ultrasonic transducer was 1.5 sq cm, which is about 10 times larger than the cross-sectional area of the MCA in adults. Without focusing, only a small portion of the ultrasonic energy can be directed at the location of interest. In addition, the transducer is not effective in receiving the weak Doppler shifted signals from the blood flow. For this
FIG. 1. Upper: Spectral display of the Doppler signal from the middle cerebral artery (MCA). The horizontal line through the spectra represents a cursor that can be controlled up or down on the display. Lower: The outline of the spectra shown above. The cursor was placed so that the areas $A_1$ and $A_2$ were judged equal. The velocity $v$ corresponding to this cursor position was calculated using the Doppler equation.

The exact positioning of the ultrasound probe was rather critical in most subjects. A satisfactory signal could only be obtained in a restricted region above the zygomatic arch, from 1 to 5 cm in front of the ear (Fig. 2). An “ultrasonic window” had to be located in each individual by searching this region to obtain a maximum amplitude of the Doppler signals.

In order to record the velocity in the MCA, we first set the depth of the range-gate to 5.0 cm. Usually the signal was found after a short search (Fig. 3). In difficult cases, probing for several minutes was necessary before obtaining the Doppler signal. Then the depth setting was increased stepwise until the MCA signal became weak. This occurred at a depth of about 6 cm, depending on the skull diameter. By aiming the probe slightly caudally, we obtained signals from the terminal portion of the internal carotid artery (ICA). This artery runs at a blunt angle with the ultrasonic beam. The Doppler signals from the intracranial ICA have lower frequency shifts than those from the MCA.

The probe was then reaimed at the MCA, and the depth of the range-gate was reduced in steps of 0.5 cm. The probe was adjusted for maximum signal at each depth. From 4.5 to 3.5 cm, we could obtain signals from two or more branches. This tracking or scanning procedure could be performed with only slight adjustments in the direction of the probe, indicating that the ultrasonic beam was intercepting the artery at a sharp angle.

The signal from the proximal ACA was obtained by scanning the MCA signal progressively deeper until a velocity in the opposite direction was found. The depth of the range-gate and the tilt of the probe were then adjusted for the best signal from the ACA. The proximal ACA is rather short, and we were not able to track it over a distance of more than 0.5 to 1 cm. The instrument has a finite resolution, and it was sometimes difficult to obtain a proximal ACA signal without interference from the MCA. However, this never caused serious difficulty in interpreting the data.

Doppler recording of cerebral arterial flow

as the spectrum analyzer has direction discrimination, allowing velocities in both arteries to be recorded simultaneously.

The PCA signal was obtained by the following procedure. The MCA was located first, then the depth of the range-gate was increased stepwise until the signal became weak and disappeared. Then the probe was tilted and aimed at a location posterior and slightly caudal to that of the MCA signal. This area was searched until we found the Doppler signal. The depth was increased further until the low-frequency Doppler shift from the basilar artery was detected. This was the distal portion of the basilar artery which runs at a blunt angle with the ultrasonic beam. Advancing the depth control still further disclosed flow in the opposite direction. This came from the ipsilateral PCA on the contralateral side. We then tracked the ipsilateral PCA from its origin at the basilar artery and laterally until the Doppler shift was maximal. This depth was used to determine the PCA velocity.

The velocity in the ICA's in the neck was measured using the same Doppler instrument and probe that was used for the transcranial recordings. The probe was placed slightly below the mandibular angle and aimed cranially. The depth of the range-gate was set in the range from 3.5 to 4.0 cm to achieve insonation at a sharp angle (less than 30°). The external carotid artery and the common carotid artery (CCA) were identified so as to ensure that we were recording well above the bifurcation.

Results

Doppler recordings of bilateral MCA blood flow velocities were obtained in all 50 subjects. However, the Doppler signal was not of sufficient intensity to allow ACA velocity determination in 20% of the arteries investigated. For the PCA this failure rate was 40%.

An MCA velocity recording from a healthy 51-year-old man is shown in Fig. 4 upper. The ipsilateral CCA was compressed for approximately 4 seconds, causing an instant drop in the MCA velocity to 60% of control. In this case, the MCA velocity waveform became damped. When the compression was released, the velocity rose to 130% of the control value for a period of 4 to 5 seconds, then returned to the pre-occlusion level (not shown). The probe was then directed slightly caudally until a signal from the terminal ICA was obtained. A new compression test was performed (Fig. 4 lower). The velocity fell to zero, and some backflow caused by an external carotid artery "steal" was observed during the systole.

In the same subject, the proximal ACA exhibited a velocity pattern as shown in Fig. 5. The upper panel illustrates a reversal of flow in this artery when the ipsilateral CCA was compressed. The proximal ACA was supplying collateral flow to the MCA on the same side in this situation. During compression, irregular flow or turbulence could be heard in the Doppler signal, particularly in systole. This showed up in the spectra as a brief period of low-frequency noise. We interpret this as the effects of a high-velocity jet from the anterior communicating artery. A recording of the proximal ACA velocity during compression of the contralateral CCA is shown in Fig. 5 lower. The velocity increased to 280% of the control value, thus demonstrating an excellent collateral capacity of the anterior circle of Willis.
FIG. 4. Spectral display of the Doppler signal from the middle cerebral artery (MCA, upper) and the terminal internal carotid artery (ICA, lower) during test compressions of the common carotid artery (CCA) on the ipsilateral side in a 51-year-old man. The MCA velocity fell by 40% during compression. Note the reversed systolic flow in the terminal ICA. This indicates external carotid artery "steal."

FIG. 5. Spectral display of the Doppler signal from the proximal anterior cerebral artery (ACA) during test compression of the common carotid artery (CCA) on the ipsilateral side (upper), and on the contralateral side (lower). Arrows indicate irregular flow during systole.
Doppler recording of cerebral arterial flow

Figure 6 displays a recording of the velocity in the proximal PCA during ipsilateral CCA compression. The velocity instantly rose to 160% of the control value, indicating its potential as a collateral flow source. When the depth of the range-gate was set to a slightly more distal portion of the PCA, we did not record any appreciable change in the velocity during ipsilateral CCA compression. Compression of the contralateral CCA did not influence the ipsilateral PCA velocity (not shown).

The MCA velocities at different depths in 10 subjects are shown in Fig. 7. The lower panel illustrates the mean values of these velocities at the standard depths for MCA recording. Our data show that the MCA velocity was relatively constant over a depth range from 6.0 to 4.0 cm, with slightly more occurring at 5.5 and 5.0 cm.

The MCA velocity in the whole series was 62 ± 12 cm/sec (mean ± standard deviation), with a range of 33 to 90 cm/sec. The ratio between the MCA velocity on the left side and that on the right side was 1.01 ± 0.14:1. Thus, in the normal adult the MCA velocities are nearly equal on the two sides. The MCA velocity did not correlate with age (r = 0.23) in this series. The velocity in the ACA was 51 ± 12 cm/sec and the PCA was 44 ± 11 cm/sec. The velocities in the extracranial ICA were 37 ± 6.5 cm/sec. The ratio between the velocity in the MCA and that in the extracranial ICA was 1.7 ± 0.4:1. The end-tidal pCO2 was 5.1 ± 0.5 kPa during these studies.

Discussion

A range-gated Doppler instrument with a frequency of 2 MHz has provided satisfactory intracranial recordings of the velocities in the MCA. This artery runs almost directly toward the probe and is thus ideally located for Doppler ultrasonic recording with the technique described.

The ACA and PCA describe comparatively sharp angles with the ultrasonic beam in their proximal parts. The velocities calculated from the spectral display probably reflect values close to true velocities in most individuals. In some subjects, however, the velocities in these arteries may be slightly underestimated and this must be kept in mind when evaluating readings from these two arteries. Our results indicate that the ACA and the PCA will also be within reach in practically all individuals with improvements in the instrumentation, particularly the probe design.

The velocities observed in the present study were in the same range as those found by Doppler techniques during surgery. Also, the responses we obtained to test occlusions of the carotid arteries followed the same pattern as those found in these previous studies. We have never observed higher velocities in the branches of the MCA than in the parent artery, and this concurs with our Doppler findings at operations. The velocities in the ACA and the PCA were generally somewhat lower than in the MCA, but higher than in the extracranial ICA. Thus, our results indicate that in the normal subject the highest velocities in the cerebral circulation are found in the basal cerebral arteries.

The systolic peak velocity in the ACA and the MCA was about 1 m/sec in more than 50% of the individuals in these series. This velocity is of the same magnitude as the systolic velocity in the aorta. It is unusual in the human circulation that arteries only 2 to 4 mm in diameter can exhibit the same peak velocity as in the aorta. Furthermore, it is of particular interest to note that, in humans, aneurysms are most apt to occur in these two arterial areas.

Transcranial Doppler recording gives useful information on the intracranial flow directions and distributions. A special application of the method is the determination of the collateral capacity of the circle.
of Willis. One can assume that the arterial lumen stays relatively constant during CCA test compression. Therefore, the flow velocity in the MCA provides direct information on the relative change in the volume flow when the CCA is occluded. Furthermore, the velocities in the ACA and PCA can be studied in the same way in order to determine their relative contribution to collateral flow.

The transcranial approach can be used for the detection of vasospasms following subarachnoid hemorrhage. When the artery contracts, or the lumen is otherwise reduced, the velocity is practically inversely proportional to the vessel lumen area. Velocities exceeding 200 cm/sec have been observed in spastic arteries (material to be published). Because the method is noninvasive, it can be repeated as often as necessary, and thus be a guide in the timing of operations and in the general handling of these patients.

References


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