Ultrasound examination of the extracranial carotid and vertebral arteries has been used for many years to diagnose and manage patients with cerebrovascular disease. However, evaluation of the intracranial circulation has until now required angiography. Extracranial ultrasound examinations utilize probe frequencies ranging from 3 to 10 MHz. These frequencies are unable to penetrate bone sufficiently to reflect signals from the intracranial arteries. Aaslid demonstrated that signals could be obtained from the middle cerebral artery (MCA) and anterior cerebral artery (ACA) with a 2-MHz probe directed intracranially from the temporal bone just above the zygomatic arch. The intracranial internal carotid artery (ICA), posterior cerebral artery (PCA), intracranial vertebral artery (VA), and basilar artery (BA) can also be insonated by the transcranial approach.

Transcranial Doppler (TCD) examinations are based on the same basic principles as extracranial Doppler. A signal emitted from the probe reflects off moving objects (red blood cells), and the frequency of the reflected signal is shifted in direct proportion to the velocity of the moving object (Doppler principle). When a vessel narrows, regardless of the cause, velocity of blood increases to allow the same volume of blood to pass a narrowed lumen. This increase in velocity is detected by TCD. Velocity also increases when there is augmentation of flow due to collateral contributions to other vascular territories or supply to a large arteriovenous malformation (AVM). TCD techniques have recently been applied to evaluate vasospasm following subarachnoid hemorrhage, intracranial stenosis, and AVMs. In addition, the patterns of collateral circulation in patients with extracranial carotid artery disease may be determined with this technique. Some of the clinical applications remain speculative, and additional work will be necessary to establish the clinical utility of TCD. Initial studies indicate that this technique will become a valuable addition to other noninvasive evaluations in a variety of clinical situations.

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often bidirectional, and as the Doppler signal depth is increased, ACA flow is detected. ACA flow is usually away from the transducer, though in pathological conditions the flow may be reversed as a collateral channel. The PCA is detected by using the branching ICA point as a reference, increasing the depth by 5 mm, and directing the probe slightly posteriorly. The signal from the proximal PCA is towards the transducer. Values for normal peak and mean velocities in the above mentioned arteries have been published.¹³

The OA and CS are insonated through the orbital window. With the ultrasound probe placed over the closed eyelid, at a depth of 40–50 mm, flow can be detected from the OA. At this depth and through the orbit, no intracranial vessels would be insonated. In addition, OA flow has a waveform characteristic of extracranial vessels, i.e., it is more pulsatile than the waveforms of intracranial vessels. Flow from the OA is characteristically directed towards the probe, though in pathological conditions such as ICA occlusion with collateral flow through the eye via the external carotid artery, the flow may be reversed and thus be away from the probe. As the depth is progressively increased, the CS is insonated. At depths of 55–70 mm the supraclinoid, the genu, and the parasellar components of the ICA can be studied. Signals towards the probe are from segments below the genu, whereas those showing flow away are from the supraclinoid region.

When the ultrasound probe is placed in the region of the foramen magnum, the VA and BA are insonated. The patient flexes his head forward with his chin on his chest; this opens up the gap between the atlas and the cranium and aids in investigation.

The BA flow is detected at a depth of 85–100 mm with the probe in the midline. By moving the direction of the probe, and thus the ultrasound beam, slightly laterally from side to side while decreasing the depth of the ultrasound beam down towards 60 mm, the VAs are insonated. The direction of Doppler shifts when insonating the VA is typically away from the probe, as in the BA. Signals from the posterior inferior cerebellar artery can sometimes be found at depths between 50 and 70 mm; these flows are more likely to be towards the probe.

A more detailed discussion of TCD technique can be found elsewhere.⁴

**Intracranial Vascular Disease**

Stenosis of intracranial arteries causes characteristic TCD abnormalities. Mild stenosis increases peak velocity, often without other changes in the Doppler pattern. Moderate or severe stenosis leads to greater increases in peak velocity with spectral broadening, increased diastolic velocities, and evidence of turbulent flow (Figure 1, Table 1). A drop in peak velocity often occurs distal to the stenotic segment when the stenosis exceeds 60–80%.⁵⁶ Several studies indicate a good correlation between TCD abnormalities and angiographically demonstrated stenosis (Figure 2).⁵⁸ MCA, ACA, intracranial ICA, and BA stenoses have been identified by TCD. Lindegaard et al⁵ found that velocity correlated inversely with residual lumen diameter in a small group of patients with intracranial stenosis. Other authors failed to confirm this finding.⁶ The number of patients with intracranial stenosis studied to date remains small, and the sensitivity and

![Figure 1. Flow velocity waveforms recorded by transcranial Doppler ultrasound in a patient with a tight middle cerebral artery stenosis.](http://stroke.ahajournals.org/)

```latex
\begin{figure}
\centering
\includegraphics[width=\textwidth]{figure1.png}
\caption{Flow velocity waveforms recorded by transcranial Doppler ultrasound in a patient with a tight middle cerebral artery stenosis.}
\end{figure}
```
TABLE 1. Transcranial Doppler Ultrasonography in Patient With Tight Right Middle Cerebral Artery Stenosis

<table>
<thead>
<tr>
<th>Artery</th>
<th>Normal values</th>
<th>Patient values</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Depth (mm)</td>
<td>Velocity (cm/sec)</td>
</tr>
<tr>
<td>MCA</td>
<td>50–55</td>
<td>62±12</td>
</tr>
<tr>
<td>ACA</td>
<td>65</td>
<td>52±12</td>
</tr>
<tr>
<td>PCA</td>
<td>70</td>
<td>42±10</td>
</tr>
<tr>
<td>OA</td>
<td>45–55</td>
<td>24±8</td>
</tr>
<tr>
<td>CS</td>
<td>70</td>
<td>54±13</td>
</tr>
<tr>
<td>VA</td>
<td>60–80</td>
<td>36±9</td>
</tr>
<tr>
<td>BA</td>
<td>85–100</td>
<td>42±10</td>
</tr>
</tbody>
</table>

Waveforms shown in Figure 1. Note focal area of high velocity in right MCA. MCA, middle cerebral artery; ACA, anterior cerebral artery; PCA, posterior cerebral artery; OA, ophthalmic artery; CS, carotid siphon; VA, vertebral artery; BA, basilar artery.

specificity of this technique in a larger population requires further study.

Occlusion of the MCA can also be identified by TCD. The absence of signals from a depth at which the MCA is usually recorded (50–60 mm), while good signals are obtained from the ACA and PCA, indicates an intact temporal window but no flow in the MCA. Hennerici et al correctly diagnosed six of seven MCA occlusions by TCD and one BA occlusion. The absence of a signal from the ACA or PCA is not a reliable indicator of occlusion even when an adequate temporal window is demonstrated. A BA signal can be found in nearly all individuals, but when no signal is recorded, differentiation between occlusion and technical difficulties remains problematic.

Figure 1. Angiogram in patient in Figure 1 showing right middle cerebral artery stenosis.

Noninvasive demonstration of intracranial arterial occlusion may be an important clinical tool. Halsey demonstrated patency of the MCA by TCD in a patient with a progressive stroke and infarction by computed tomography (CT) in the corona radiata. In patients with acute infarcts, the MCA can be studied serially to identify proximal occlusions and possibly demonstrate recanalization. The recent use of thrombolytic agents for treatment of acute stroke raises the possibility that TCD may be useful to monitor the efficacy of this therapy following identification of an occluded vessel.

Extracranial Carotid Artery Disease

Extracranial carotid noninvasive techniques are highly reliable in identifying hemodynamically significant stenosis. Changes also occur in the intracranial circulation as a result of extracranial stenosis. MCA velocity decreases ipsilateral to severe stenosis or occlusion, and pulsatility of the waveform also often decreases. These changes are likely due to the diminished flow from the ipsilateral carotid artery as well as the effects of collateral circulation. Vasodilation in the distal arterial circulation ipsilateral to stenosis probably contributes to the diminished pulsatility. Not all patients with severe stenosis or occlusion demonstrate these abnormalities, and the reasons for individual variability remain unknown. One study found that MCA velocity characteristics did not differ in patients with extracranial carotid artery disease, with and without collateral circulation via the circle of Willis. Several factors contribute to the velocity profile, and the relative contribution of each under normal and pathological conditions remains to be determined. Lindegaard et al reported that pulsatility transmission index (PTI; ratio of the pulsatility index of the ipsilateral MCA to a reference artery) correlated better with severity of extracranial carotid stenosis than did the absolute velocity. In this study and others, MCA velocity decreased ipsilateral to severe stenosis and occlusion; however, the variability between individuals with similar degrees of
stenosis was large. Extracranial ICA stenosis cannot be easily diagnosed based on absolute MCA velocity; however, MCA velocity ratio (ratio of ipsilateral MCA velocity to contralateral MCA velocity) or PTI may be more reliable, at least in patients with unilateral disease.

Identification of sources of collateral flow in patients with extracranial carotid artery disease may be possible by TCD. Common carotid artery compression maneuvers test patency of the circle of Willis, but compression should probably not be performed in patients with cerebrovascular disease because of the possibility of dislodging clot or debris from atherosclerotic plaques. Increased velocity in the ACA contralateral to ICA stenosis with reversal of the direction of flow in the ipsilateral ACA suggests collateral circulation from the contralateral ICA via the anterior communicating artery. Similarly, increased velocity in the PCA suggests collateral flow from the posterior circulation via the posterior communicating artery. Increased velocity also occurs in the BA in patients with bilateral carotid artery occlusion and collateral flow to both hemispheres from the posterior circulation. The reported correspondence between TCD findings and angiographically demonstrated patterns of collateral flow has been variable. Large increases in contralateral ACA or ipsilateral PCA velocities usually correspond to collaterals from these vessels by angiography. However, the direction of flow in the ACA ipsilateral to severe stenosis or occlusion is not always reliable evidence of flow reversal demonstrated by angiography. Unfortunately, angiography is not necessarily accurate since reversal of ACA flow may be dependent on the pressure of contrast injection. It is possible that TCD provides a more accurate indication of patterns of collateral flow under existing physiological conditions. Extracranial Doppler and duplex Doppler often combined with oculoplethysmography and supraorbital Doppler remain the mainstay of noninvasive diagnosis of extracranial vascular disease. TCD may increase the reliability of diagnosis when used in concert with these other techniques and add information regarding the status of intracranial collateral pathways.

Increased velocity with turbulence in intracranial arteries occurs both in stenotic arteries and in arteries providing collateral flow. These conditions cannot reliably be distinguished on the basis of the TCD patterns alone. Doppler information from other intracranial arteries and from extracranial noninvasives in most cases contributes to proper diagnosis. Increased velocity also occurs in intracranial arteries when they supply medium-sized or large AVMs. Velocity increases are found throughout the course of such arteries, distinguishing them from localized stenosis in which velocity decreases distal to the stenotic segment. Pulsatility index typically decreases in arteries feeding AVMs. These abnormalities can be found in most patients with AVMs; however, small lesions and venous angiomas may not be identified. Abnormal TCD patterns occur in those arteries that supply the AVM, while Doppler patterns in other intracranial arteries are normal. In addition to identification of AVMs, TCD may permit noninvasive monitoring of regression after treatment with gamma knife or proton beam radiation.

**Intraoperative Monitoring**

When performing carotid endarterectomy (CEA), temporary cross-clamping of the ICA is felt in certain circumstances to put the patient at risk for intraoperative stroke. Current methods of monitoring include electroencephalography, regional cerebral blood flow (rCBF) monitoring, and measurement of stump pressure; however, these methods may be difficult to monitor throughout the operation and may not be accurate.

There has been increasing interest in the possibility of using TCD to monitor MCA flow velocity (V_MCA) while cross-clamping the ICA during CEA. Monitoring can be done using a flat probe, fixed preoperatively in the temporal region of the skull, with a means of easily adjusting the probe within the frame until the optimal waveform is obtained. The patient can be studied preoperatively to assure that monitoring of the MCA is possible through the patient's temporal window.

In a study of 19 patients during CEA, Padayachee et al monitored V_MCA, using TCD on the side ipsilateral to the CEA, throughout the entire operation including anesthesia, diathermy, carotid cross-clamping, shunting, and in the recovery room. During cross-clamping there was a significant decrease in V_MCA in all patients studied, with three patients showing a drop in V_MCA to 0. Backbleeding in the ICA before shunt insertion was associated with diminished V_MCA in 10 of 19, oscillatory forward/reverse flow in three of 19 (thought to be secondary to an "ICA-steal" phenomenon, i.e., stealing blood from the MCA to flow down the ICA), and cessation of flow (V_MCA dropped to 0) in the remaining six patients. Bishop et al measured "stump" pressures during CEA while monitoring V_MCA by TCD and found stump pressures <50 mm Hg were associated with low V_MCA during clamping.

In a study by Halsey et al rCBF was compared with mean V_MCA during CEA. Little relationship was found between V_MCA and rCBF for all cases. On the other hand, if the ratio of systolic peak to diastolic nadir (S/D ratio) was used instead of mean V_MCA, a much closer relationship was found (the S/D ratio is related to the PTI described by Lindegaard et al). It was found that the S/D ratio decreased at the time of carotid occlusion (cross-clamping) when the V_MCA did not change and when rCBF decreased by 50%. The S/D ratio decreased in all seven patients in which it was recorded, whereas the rCBF decreased in five and did not change significantly in three, showing that the S/D ratio was
more sensitive to the occlusion than the mean $V_{\text{MCA}}$ or the rCBF. This study makes the point that rCBF and TCD are measuring different things in that rCBF reflects cortical flow at the cerebral convexity whereas TCD is measuring flow velocity in the proximal extraparenchymal vessels. It does illustrate that the S/D ratio measured by TCD has a greater sensitivity to proximal resistance than $V_{\text{MCA}}$ and rCBF. This has been noted by others as well.\textsuperscript{11}

It is still too early to know how valuable TCD monitoring will be during CEA, though the preliminary studies are quite promising. Additional support for its use is the fact that the monitoring equipment is small and mobile and in most instances does not interfere with the operative procedure. Also, monitoring with TCD can be used to supply ongoing information about true circulatory conditions within the insonated arteri-eties during cardiopulmonary bypass for open heart surgery\textsuperscript{4} as well as during certain neurosurgical or neuroradiological interventions.\textsuperscript{21}

Subarachnoid Hemorrhage

Vasospasm following subarachnoid hemorrhage is a significant cause of morbidity and mortality. Diagnosis often depends on recognition of typical clinical syndromes and exclusion of other causes of neurological deterioration such as rebleeding or hydrocephalus. Angiography is usually performed only when localization of an aneurysm becomes necessary prior to surgery and is potentially hazardous in patients with vasospasm. TCD allows noninvasive detection of vasospasm in the proximal MCA and ACA (Table 2). Increased peak systolic and mean velocities correlate well with angiographic\textsuperscript{21,22} and clinical\textsuperscript{23} evidence of vasospasm. Mean $V_{\text{MCA}}$ usually exceeds 200 cm/sec in patients with vasospasm,\textsuperscript{24} and most are $>250$ cm/sec. Not all patients with velocities in this range become symptomatic. Extremely high velocities occur in asymptomatic patients, probably due to adequate collateral flow. Increased velocities in both the MCA and ACA may be a more reliable predictor of symptoms than $V_{\text{MCA}}$ alone. $V_{\text{MCA}}$ correlates inversely with residual lumen diameter in patients with angiographic evidence of vasospasm, but ACA velocity correlates poorly.\textsuperscript{21,22}

In addition to diagnosis of vasospasm, TCD allows repeated studies at frequent intervals to monitor the time course of onset and resolution of vasospasm (Table 2). While symptoms of vasospasm usually appear between 4 and 14 days following subarachnoid hemorrhage, increases in velocity by TCD often occur earlier.\textsuperscript{7} Increased velocities typically precede the onset of symptoms by hours to days.\textsuperscript{22} The ability to detect vasospasm before symptoms develop permits prophylactic treatment in high-risk individuals. Treatment can also be tailored to the course of vasospasm since decreasing velocities signal resolution.\textsuperscript{7} In one study, a distinctive pattern of $V_{\text{MCA}}$ emerged in the first few days after subarachnoid hemorrhage in patients later develop-

### Table 2. Transcranial Doppler Ultrasonography in Patient With Vasospasm After Subarachnoid Hemorrhage

<table>
<thead>
<tr>
<th>Artery</th>
<th>Depth (mm)</th>
<th>Velocity (cm/sec)</th>
<th>Patient values</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Depths (mm)</td>
</tr>
<tr>
<td></td>
<td>Right</td>
<td>Left</td>
<td>Right</td>
</tr>
</tbody>
</table>

**Four days after subarachnoid hemorrhage**

- **MCA**: 50–55
  - Normal values: $62 \pm 12$
  - Patient values: $50 \quad 50$
- **ACA**: 65
  - Normal values: $52 \pm 12$
  - Patient values: $65 \quad 65$
- **PCA**: 70
  - Normal values: $42 \pm 10$
  - Patient values: $65 \quad 70$
- **OA**: 45–55
  - Normal values: $24 \pm 8$
  - Patient values: $55 \quad 50$
- **CS**: 70
  - Normal values: $54 \pm 13$
  - Patient values: $65 \quad 65$
- **VA**: 60–80
  - Normal values: $36 \pm 9$
  - Patient values: $70 \quad 70$
- **BA**: 85–100
  - Normal values: $42 \pm 10$
  - Patient values: $85 \quad 75$

**Nine days after subarachnoid hemorrhage**

- **MCA**: 50–55
  - Normal values: $62 \pm 12$
  - Patient values: $50 \quad 55$
- **ACA**: 65
  - Normal values: $52 \pm 12$
  - Patient values: $65 \quad 70$
- **PCA**: 70
  - Normal values: $42 \pm 10$
  - Patient values: $70 \quad 70$
- **OA**: 45–55
  - Normal values: $24 \pm 8$
  - Patient values: $55 \quad 55$
- **CS**: 70
  - Normal values: $54 \pm 13$
  - Patient values: $70 \quad 65$
- **VA**: 60–80
  - Normal values: $36 \pm 9$
  - Patient values: $75 \quad 70$
- **BA**: 85–100
  - Normal values: $42 \pm 10$
  - Patient values: $85 \quad 80$

Subarachnoid hemorrhage from ruptured left MCA aneurysm. Note diffuse pattern of high velocities involving MCA, both ACAs, left PCA, and left CS at 4 days. Values at 9 days show resolving vasospasm. MCA, middle cerebral artery; ACA, anterior cerebral artery; PCA, posterior cerebral artery; OA, ophthalmic artery; CS, carotid siphon; VA, vertebral artery; BA, basilar artery.
ing symptomatic vasospasm. Early rapid rises were seen in those who developed infarction, and even more rapid rises occurred in those with fatal outcome. The maximum recorded velocity was greater in those with more extensive CT evidence of blood or focal clots. If larger studies confirm these initial results, it may be possible to identify patients destined to develop ischemic symptoms due to vasospasm based on TCD results in the first few days following hemorrhage.

There are several pitfalls in diagnosing a vasospasm by TCD. Narrowing of branches distal to the proximal segment of the ACA and PCA will usually be beyond the focal range of the Doppler probe and will be difficult to detect. Changes in the shape of the trace in the proximal arteries occur only if distal vasospasm is extensive. Severe vasospasm of the ICA diminishes distal flow and decreases velocity in the basal cerebral arteries. This may result in falsely low velocities when vasospasm coexists in the intracranial ICA and MCA or ACA. Finally, the reliability of TCD in detecting vasospasm of the BA remains unknown. Despite these reservations, TCD is likely to become an important tool to diagnose, follow, and treat vasospasm. Future studies of treatment for vasospasm will most likely include TCD because of its ability to noninvasively monitor the effects on vessel diameter.

**Subclavian Steal**

TCD can be used to evaluate patients with symptoms consistent with subclavian steal. If the steal is severe and present at rest, velocity in the stealing VA will be reversed (i.e., towards the ultrasound probe). If velocities in both VAs are away from the ultrasound probe), the possible presence of subclavian steal can be tested by inflating a blood pressure cuff while insonating the ipsilateral VA. If subclavian steal is present, the velocity in the insonated VA will reverse (i.e., will go from being away from the ultrasound probe to being towards it).

**Measurement of Cerebral Blood Flow**

TCD is primarily used for measuring flow velocities in the intracranial vessels. Interest has begun to develop in the possibility of using TCD to measure rCBF as well. Present methods for measuring rCBF include measurement of the clearance of intravenous or inhaled xenon-133 by means of extracranial recording, positron emission tomography after inhalation of oxygen-15, or CT scanning after inhalation of stable xenon. These methods are all quite invasive, require expensive equipment, and are difficult for screening or follow-up studies. TCD has the advantage of being noninvasive, and monitoring can be continuous over a period of time, recording changes in rCBF as they occur.

Changes of arterial carbon dioxide partial pressure (PaCO₂) considerably influence CBF and have become a basis for studying the above methods of measuring rCBF. Depression and elevation of PaCO₂ cause vasoconstriction and vasodilation of arteriolar channels, respectively, leading to alterations in cerebrovascular resistance, which is responsible for the changes in cerebrovascular circulation time, CBF, and flow velocity in cerebral arteries. The diameter of large basal cerebral arteries is felt to remain constant during hypocapnia and hypercapnia, and thus changes in CBF induced by alterations of PaCO₂ should also be proportional to changes in flow velocity in the basal arteries. Markwalder et al studied the CO₂ response for V_mCA in normal subjects and found that, provided that end-tidal Pco₂ was not influenced by changes in arterial blood pressure and represented a close approximation of PaCO₂, the response curves for V_mCA obtained in their study strongly resembled the CBF response curves obtained from experimental and clinical studies.

In a study that measured rCBF using xenon inhalation, rCBF and CO₂ responsiveness were measured in patients with carotid arterial disease. It was found that patients with carotid stenosis of ≥60% had normal resting flows but reduced responsiveness to CO₂ inhalation. Patients with ipsilateral carotid occlusion had both reduced resting flow and reduced CO₂ responsiveness, and the CO₂ responsiveness decreased more with increasingly severe bilateral carotid disease. These findings were much like those of Bishop et al who measured V_mCA using TCD above a carotid occlusion and found reduced responsiveness to hypercapnia. Lindegaard et al measured PTI (pulsatility index adjusted for individual variations as outlined in this reference) using TCD and found that carotid stenoses reducing the luminal area of the ICA by ≥75% reduced the PTI of the ipsilateral MCA; however, V_mCA correlated poorly with the degree of ipsilateral carotid stenosis. For stenoses in the 75–89% category, PTI reduction was significantly greater in patients with bilateral carotid stenosis, indicating impaired potential for collateral flow in these patients; however, there was no change in the 90–99% stenoses category. In spite of severe ICA stenosis or occlusion, the ipsilateral cerebral hemisphere often has blood flow within normal limits. In response to reduced inflow pressures, cerebral autoregulation maintains blood flow by lowering cerebrovascular resistance. In effect, MCA volume flow and flow velocity remain relatively constant when cerebral autoregulation is operative. Therefore, PTI is more sensitive than V_mCA to net hemodynamic effects of ICA stenosis. In conclusion, it was felt that PTI reduction reflects a combination of reduced MCA inflow pressure and lowered cerebrovascular resistance.

These studies offer only preliminary data, though the possibility exists that TCD may be able to be used to accurately measure and follow rCBF.

**Brain Death**

At the time of brain death, a characteristic and diagnostic pattern of flow has been noted by TCD in
large basal intracranial vessels. An oscillating re-verbatory or “to-and-fro” movement has been noted in the flow velocity waveforms. Depending on the cardiac output, these oscillations can be sharp and pulsatile or quite damped. The diagnosis is made based on the finding of a reflux phenomenon during late systole following antegrade injection of blood into the vascular tree.

Summary

TCD recording of flow velocities in intracranial vessels was first described by Aaslid in 1982. The utility of this instrument becomes more apparent as it is used in different clinical settings and compared with angiographic findings. Its importance in early detection of vaso-spasm in subarachnoid hemorrhage is now clearly known; increased flow velocity can be documented prior to neurologic deterioration and thus allow early institution of therapy. In patients with stroke or transient ischemic attack of unclear etiology, especially in blacks, Orientals, or females, who have a higher incidence of intracranial artery disease, TCD can be a very important noninvasive means for detecting stenosis of intracranial vessels. Its value for assessing collateral circulation, intraoperative monitoring, and measuring CBF is quite promising.

Hopefully, through further work with TCD, we will be able to clarify the spectrum of its usages as well as its limitations, though the preliminary data indicate that it should be an important addition to present noninvasive evaluations.

References


KEY WORDS • ultrasonics • carotid artery diseases • cerebral artery diseases
Transcranial Doppler.
L D DeWitt and L R Wechsler

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