Differential considerations of Tcd pulsatility (Gosling’s) and resistance (Pourcelot) indices after AVM surgery

JJ Sierra¹, R Hanel, MD², L Mooney³, and WD Freeman, MD⁴⁵*

¹ Mayo Clinic Observer, Jacksonville, FL, USA
² Department of Neurosurgery, Mayo Clinic, Jacksonville, FL, USA
³ Department of Nursing, Mayo Clinic, Jacksonville, FL, USA
⁴ Department of Neurology, Mayo Clinic, Jacksonville, FL, USA
⁵ Department of Critical Care, Mayo Clinic, Jacksonville, FL, USA

Neurolmage/Case report

A 29-year-old man previously healthy described the worst headache of his life prior to admission where a CT scan was performed at the local emergency department (Figure 1a). This showed an acute intraparenchymal hemorrhage of 5.7 cm × 3.4 cm × 5.7 cm dimensions, or ~54 cm³ volume [1]. The patient had acute right-sided hemiplegia and aphasia and a NIHSS stroke of 16. The patient underwent an acute digital subtraction angiogram (DSA) which revealed a Spetzler Martin grade 3 arteriovenous malformation (AVM) (Figure 1b) [2]. The patient was transferred to our facility where he underwent acute left-sided craniotomy, hematoma evacuation, AVM resection, and clipping. Because of the concerns about some blood being seen on the cortex and the theoretical concerns about arterial subarachnoid hemorrhage (Figure 1c) occurring from his AVM, transtemporal transcranial Doppler imaging was performed to screen for large vessel vasospasm [3,4] which are shown in Figure 1d. Although mean flow velocities (MFVs) of the left MCA-M1 were not indicative of vasospasm (i.e., >120–200 cm/s), the pulsatility (Gosling’s) index (PI) and resistance (Pourcelot) index (RI) were markedly abnormal on the left compared with the right, and raised the question about downstream resistance or poor intracranial compliance (Table 1, differential of raised PI and RI) based on TCD differential [5]. However, on careful review of the DSA, the patient’s AVM had a high-flow left MCA-M2 branch which after surgery (Figure 1e and f) was dramatically reduced in flow. This is demonstra-
Table 1. Differential diagnoses of elevated pulsatility (Gosling’s) and resistance (Pourcelot) Indices by Tcd5

<table>
<thead>
<tr>
<th>Etiology</th>
<th>PI and RI pattern</th>
<th>Physiologic interpretation and management</th>
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<tbody>
<tr>
<td>Poor intracranial compliance, elevated intracranial pressure (ICP)</td>
<td>PI increase steadily &gt;2.0 and RI increase &gt;0.8 suddenly or gradually</td>
<td>ICP monitoring, hypertonic saline or manitol to reduce ICP, surgery (e.g., decompressive hemicraniectomy to decompress cerebral edema, hematoma evacuation etc.</td>
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<tr>
<td>Brain death</td>
<td>PI and RI increase and may develop “short spikes” and even biphasic or “reverberating flow” before complete cessation on intracranial flow</td>
<td>Absence of brainstem reflexes, and findings consistent with brain death</td>
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<tr>
<td>Hyperventilation</td>
<td>Elevated PI without RI</td>
<td>Hyperventilation noted at the bedside, ventilator, etc., or on arterial blood gas (low pCO2)</td>
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<tr>
<td>Atherosclerotic (“stiff”) arteries</td>
<td>Elevated PI without major RI elevation, and stable on serial examination</td>
<td>Atherosclerotic stiffening of the vessels, without elevated ICP, etc.</td>
</tr>
<tr>
<td>High-flow proximal vessel which has its flow reduced</td>
<td>Lower mean flow velocity, elevated PI and RI</td>
<td>Clinically correlate with patient’s examination, findings for edema, elevated ICP, versus more extensive vascular imaging such as angiogram</td>
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**Figure 1c. Post CT.**

**Figure 1d. TCD left MCA-M1.**

**Figure 1e. Arrow pointing turbulent flow within MCA branch feeding AVM.**

**Figure 1f. Arrow pointing MCA branch occluded by aneurysm clip at site of AVM reaction.**

...ing the venous phase and filling of other major cerebral veins. The following case illustrates the importance of the TCD as a means to gauge for vasospasm but by considering the interpretation of the elevated RI and PI with the entire clinical picture such as the patient’s neurologi-
cal exam, the CT scan findings (lack of major cerebral edema), and postoperative DSA which shows the relative low flow of a prior high flow vessel. The patient’s neurological outcome on postoperative day 3 was excellent with return of his speech and right-sided extremity function (NIHSS = 9), and he was later discharged to an inpatient rehabilitation to work on speech and hemiparesis.

References