



# Real-Time Detection of Cerebral Artery Rebleeding by Transcranial Doppler Ultrasound: Hemodynamic Changes and Response to Treatment

Eric J. Marrotte<sup>a</sup>  
Panayiotis Mitsias<sup>a</sup>  
Leonard Melvin<sup>a</sup>  
Asim Mahmood<sup>b</sup>  
Georgios Tsivgoulis<sup>c</sup>  
Panayiotis Varelas<sup>a,b</sup>

<sup>a</sup>Departments of Neurology and

<sup>b</sup>Neurosurgery, Henry Ford Hospital,  
Detroit, MI, USA

<sup>c</sup>Second Department of Neurology, School  
of Medicine, University of Athens, Athens,  
Greece

Dear Editor,

Aneurysmal rupture and subsequent subarachnoid hemorrhage (SAH) lead to multiple complications including vasospasm, delayed cerebral ischemia, cerebral edema, and cerebral herniation.<sup>1-3</sup> Transcranial Doppler ultrasound (TCD) is a noninvasive method for evaluating the blood flow velocities in intracranial vessels and the presence of vasospasm.<sup>3</sup> An elevated intracranial pressure (ICP) leads to decreased mean and diastolic blood flow velocities and increased pulsatility index (PI), which can also be monitored in patients with SAH.<sup>3</sup> We present a case report of a patient with SAH and a clipped aneurysm who rebled while undergoing a TCD study, and provide unique images, vitals graphs, and a video of the TCD study before and during the bleeding as well as after applying emergency treatment.

A 68-year-old Caucasian man presented for the evaluation of sudden-onset headache and neck pain. His blood pressure (BP) was initially elevated (240/117 mm Hg) and he had experienced a tonic-clonic seizure for which he received fosphenytoin and was subsequently intubated and started on a propofol drip. Brain computed tomography (CT) revealed SAH. Brain CT angiography showed an irregular anterior communicating artery aneurysm (2.5×2×1.5 mm) and increased hemorrhage within the ventricles, septum pellucidum, and corpus callosum (Supplementary Fig. 1 in the online-only Data Supplement).

An attempt to coil the aneurysm was unsuccessful, and so the patient underwent right frontotemporal craniotomy, clipping of the aneurysm, and placement of a ventriculostomy catheter (Supplementary Fig. 2 in the online-only Data Supplement). After surgery he was admitted to the neurointensive care unit (NICU), where an interval decrease in the subarachnoid blood was noticed in follow-up brain CT. Postoperatively he remained intubated, but was awake and followed commands briskly, and the results of neurological examination were normal except for a slight drift in the right upper extremity.

The patient exhibited rebleeding at 8 days after the index event, secondary to clip dislocation (Supplementary Fig. 3 in the online-only Data Supplement). TCD waveforms were captured before and during the bleeding as well as after emergency treatment (Fig. 1) (Supplementary video 1 in the online-only Data Supplement) in parallel with NICU multimodal monitoring that captured minute-by-minute changes in the vital signs (Fig. 1). PI increased markedly during aneurysmal rebleeding (PI=1.7) compared to before the rebleeding (PI=1.0) and following rebleeding treatment (PI=1.4) with propofol (two doses of 30 mg) and mannitol (75 g). The patient remained comatose (score on the Glasgow Coma Scale of 4 out of 15) throughout the following few weeks, and the family decided to withdraw life-support treatment at 40 days after admission.

To the best of our knowledge, this is the first case in which the rebleeding of a cerebral aneurysm was detected in real time using TCD monitoring. This report highlights the poten-

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## Correspondence

Georgios Tsivgoulis, MD  
Second Department of Neurology,  
School of Medicine, University of Athens,  
Iras 39, Gerakas Attikis, Athens 15344,  
Greece

**Tel** +30 6937178635

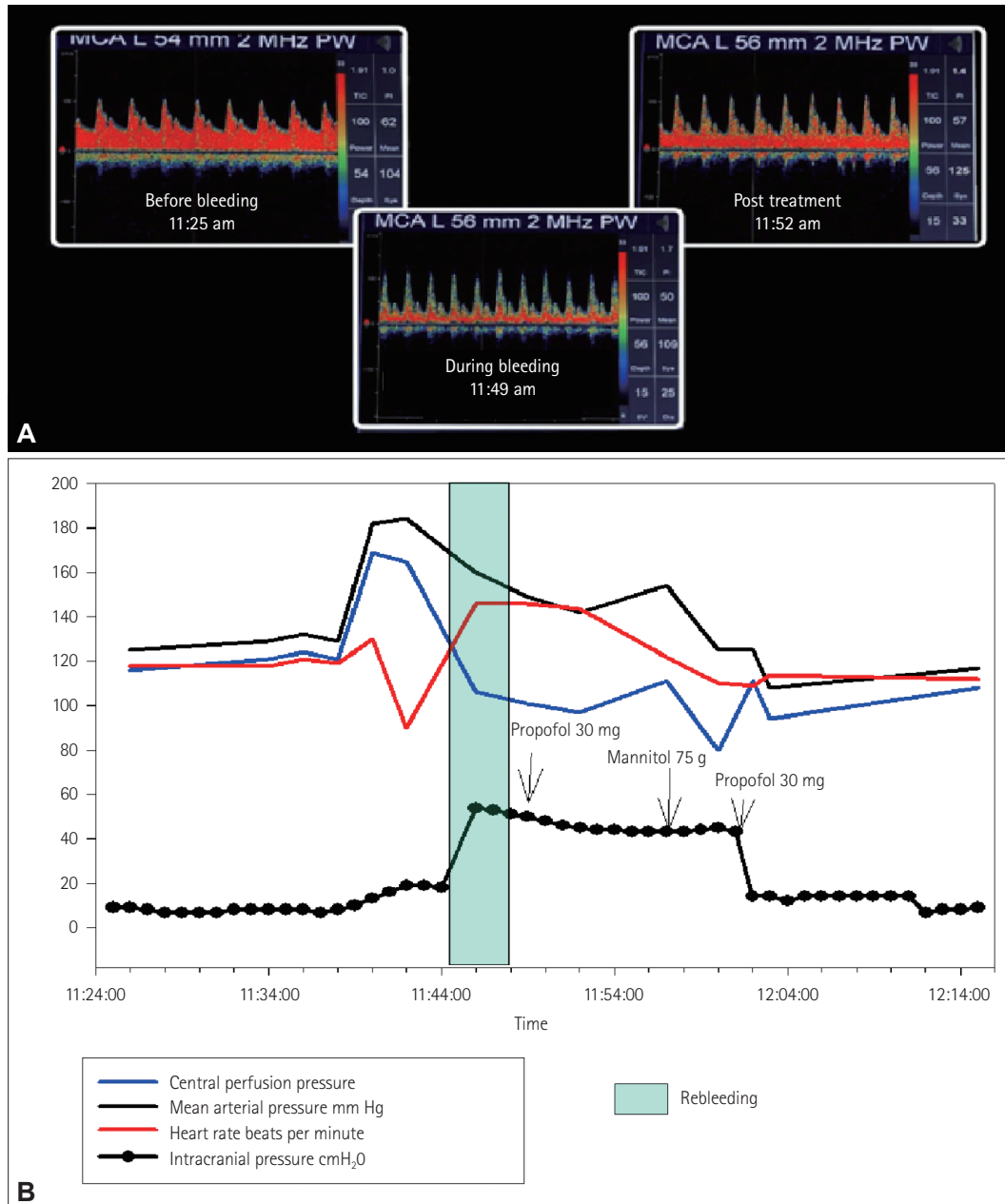
**Fax** +30 2105832471

**E-mail** [tsivgoulisgiorg@yahoo.gr](mailto:tsivgoulisgiorg@yahoo.gr)

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tial of TCD [coupled with multimodal monitoring of the cerebral perfusion pressure (CPP), ICP, and vital signs] in documenting hemodynamic changes occurring during clip failure and rebleeding in realtime. It is noteworthy that in addition to the elevation of the mean arterial pressure and CPP, a PI increase preceded the ICP surge, suggesting that hypertension caused the dislodgment of the clip and rebleeding.

These observations are consistent with the results of animal studies suggesting that BP elevation triggers rather than follows aneurysm rebleeding.<sup>4</sup> The association between high BP and aneurysm rupture is unclear. Although there are reports of patients who rebleed having higher mean BPs,<sup>5</sup> a decrease in the rebleeding rate by tight BP control has not been demonstrated definitively. Moreover, several authors have suggested



**Fig. 1.** A: TCD waveforms before, during the bleed and post treatment (A and B left middle cerebral artery). Prior to bleed: ICP was 8 mm Hg, left MCA flow velocity was 61 cm/sec and PI was 1.0. During bleed: ICP increased to 54 mm Hg and PI to 1.7, with the waveform showing a narrow peak and decreased diastolic and mean velocity. 75 g of mannitol was given and the ventriculostomy was opened to drain. Within 5 minutes the ICP decreased to 14, PI improved to 1.4, the waveform widened and the velocities returned to previous levels. Please also see supplemental video. B: Telemetry data, mean arterial pressure increased and cerebral perfusion pressure increased before the rebleed. During rebleed there was a trend for ICP elevation (up to 54 mm Hg, but with poor waveform). After therapy with propofol and mannitol all normalized. ICP: intracranial pressure, MCA: middle cerebral artery, PI: pulsatility index, TCD: Transcranial Doppler ultrasound.

that rebleeding could be induced by variations or changes in BP rather than the absolute BP level.<sup>6</sup> A very recent case—control study found that systolic BP variability was associated with increased rates of acute aneurysmal rebleeding, emerging thus as a novel and independent risk factor for rebleeding after SAH.<sup>7</sup>

The present unique case underscores the diagnostic utility of TCD in offering real-time assessment of the hemodynamics and ICP changes during SAH. TCD combined with other imaging modalities such as multimodal magnetic resonance imaging may further enhance our understanding of the pathophysiology of SAH.<sup>8</sup> We have also provided evidence that BP elevation leading to an increased PI is the cause of aneurysmal rebleeding, and argue against the hypothesis that a BP increase documented in patients with recurrent SAH is secondary to aneurysmal rerupture. The association between BP control and the risk of aneurysm rebleeding should be evaluated in future randomized clinical trials.

### Supplemental Video Legend

Transcranial Doppler ultrasound recordings in left M1 middle cerebral artery before, during aneurysmal rerupture and post elevated intracranial pressure (ICP) treatment. Note the high resistance flow during aneurysmal rerupture in comparison to normal, low-resistance before the rebleeding of the aneurysm. Also, note the decrease in PI and the increase in diastolic flow following the control of elevated ICP levels complicating the rebleeding of the aneurysm.

### Supplementary Materials

The online-only Data Supplement is available with this arti-

cle at <https://doi.org/10.3988/jcn.2017.13.1.109>.

### Conflicts of Interest

The authors have no financial conflicts of interest.

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