

Aneurysm Rupture during Endovascular Coiling: Effects on Cerebral Transit Time and Neurophysiologic Monitoring and the Benefits of Early Ventriculostomy: Case Report

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Abstract

Objective and Importance: We report a case of intra-operative aneurysm rupture during endovascular therapy and document the effects of rupture on cerebral transit times and neurophysiologic monitoring. The effects of early ventriculostomy are clearly documented.

Clinical Presentation: A 42-year-old man with Hunt and Hess grade 1, Fisher grade 3 subarachnoid hemorrhage secondary to a 5 mm anterior communicating artery aneurysm underwent coil embolization.

Intervention: Endovascular therapy was complicated by intra-procedural aneurysm rupture. Changes in cerebral transit time and electroencephalography along with somatosensory evoked potentials were documented as were improvement in these parameters following aneurysm obliteration and ventriculostomy placement. The patient awoke without deficit and was discharged 2 weeks later with a grossly normal examination.

Conclusion: Early recognition of aneurysm rupture during coil embolization and prompt aneurysm obliteration and reduction in intracranial hypertension can lead to acceptable patient outcomes. Use of neurophysiologic monitoring in the intubated patient can help the neurosurgeon determine the need for cerebrospinal fluid drainage in such situations.

Key words

Cerebral aneurysm · rupture · endovascular therapy · ventriculostomy · neurophysiology

Introduction

The incidence of intraoperative aneurysm rupture during coil embolization is approximately 2–4% [4–6,9]. We recently published a series of 274 intracranial aneurysms treated with coil embolization with a 2% incidence of intraprocedural rupture (n=6) (Neurosurgery, October 2001). Glasgow Outcome Score (GOS) at a mean follow-up of 8 months was one or two in four patients and five in two patients (mean 2.5) (1 = normal pre-operative function; 2 = moderate disability but independent; 5 = dead). Early rupture recognition, control of patient airway and blood pressure, immediate heparin reversal, rapid aneurysm obliteration, and ventriculostomy placement are key factors in achieving meaningful patient survival. This report documents the effects of rupture on angiographic transit times and neurophysiologic parameters and vividly illustrates the effects of corrective measures on cerebral perfusion and function.

Case Report

A 42-year-old right-handed man presented with a Hunt and Hess grade 1, Fisher grade 3 subarachnoid hemorrhage (SAH) secondary to a 5 mm anterior communicating artery aneurysm. The usual medications were administered including anti-hypertensives to maintain systolic blood pressure 90–100 mm Hg, anticonvulsants (Dilantin), and nimodipine. Once in the angiography suite the patient was intubated, pharmacologically paralyzed, and sedated with narcotic and propofol. A 6F sheath was placed in the right femoral artery and a 6F guiding catheter was advanced into the left internal carotid artery (ICA). Baseline images revealed a 0.34 second delay between initial ICA opacification and

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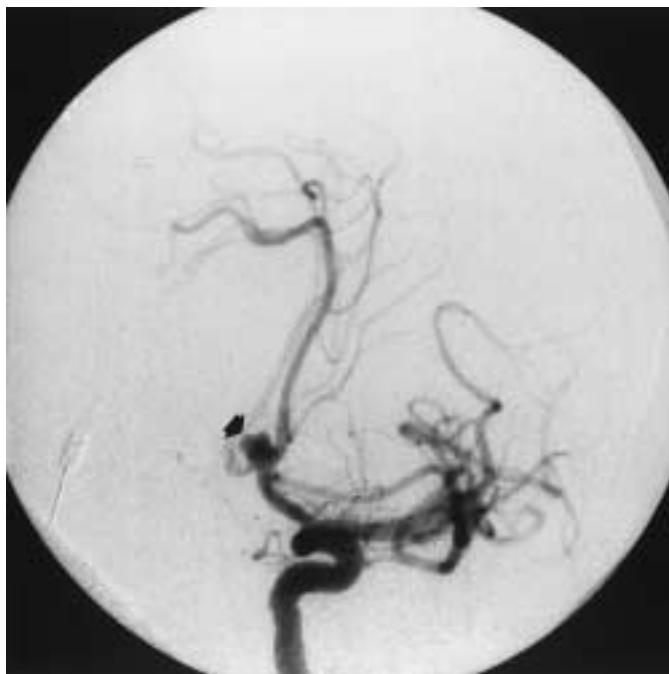


Fig. 1 Left oblique ICA arteriogram demonstrating the aneurysm arising from the left A1 – A2 junction (arrow). The transit time for contrast to opacify the ICA and the A2 was 0.34 seconds.



Fig. 3 Left oblique ICA arteriogram demonstrating petrous ICA opacification (arrow) with delay in supraclinoid opacification (1.67 – 2.36 second transit times).

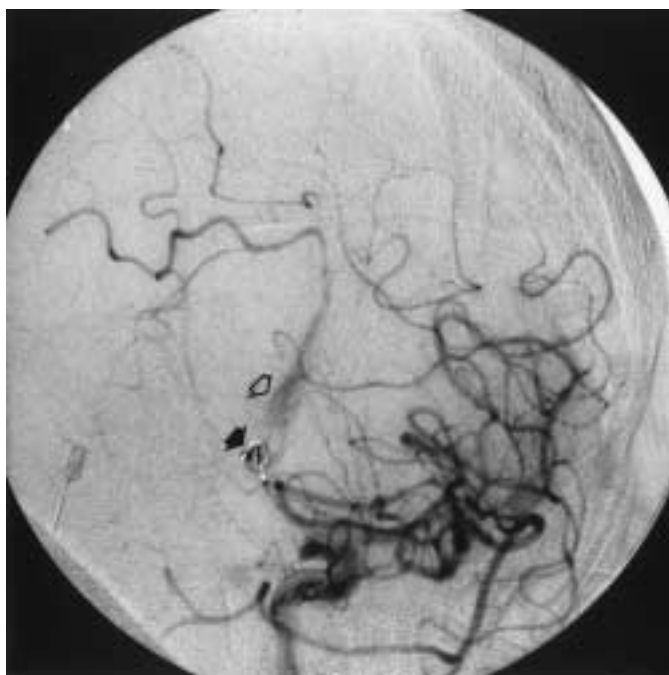


Fig. 2 Left oblique ICA arteriogram demonstrating the first coil in the aneurysm fundus (closed arrow) and contrast extravasation from the aneurysm fundus (open arrow).

subsequent A2 opacification (Fig. 1). Using road mapping guidance a 0.018 inch microcatheter was advanced over a 0.016 inch wire into the aneurysm fundus. During deposition of the first coil (T10 5 mm × 10 cm) the catheter tip suddenly advanced and both coil and catheter appeared to extend beyond the confines of the road mapped aneurysm. Initial heparinization with 3000 units was immediately reversed with 100 mg protamine. The catheter tip was withdrawn back into the fundus and the coil was de-

ployed. An angiographic run performed during coil deposition demonstrated contrast extravasation (Fig. 2). The delay between ICA and A2 opacification immediately increased to 1.67 seconds (Fig. 3). Arterial blood pressure rose 33 mm Hg and the patient's electroencephalogram (EEG) flattened within 5 seconds of rupture (Fig. 4a,b). Median and posterior tibial somatosensory evoked potential responses (SSEP) began to flatten within 30 seconds of rupture and completely flattened bilaterally within 2.5 minutes following rupture (Figs. 5a,b, 6a). The lesion was quickly obliterated with three additional coils. Sequential control arteriographic runs showed absence of extravasation after the second coil was deposited with delay between ICA and A2 opacification increasing to 2.36 seconds. There was no discernible SSEP or EEG response (Figs. 4c, 5c, 6b). At this point a right coronal ventriculostomy was placed (CSF access obtained 23 minutes after documented aneurysm rupture). Opening pressure was greater than 700 mm H₂O. Repeat ICA angiography two minutes following ventriculostomy insertion documented aneurysm obliteration and ICA to A2 contrast transit time < 0.05 seconds (Fig. 7). Within 5 minutes of ventriculostomy insertion the patient's EEG and SSEP began to improve (Figs. 4d, 5d, 6). A post-coiling CT scan revealed no intraparenchymal hematomas. He awoke 30 minutes later with a normal, baseline neurologic examination and was discharged 14 days later without deficits. Angiography performed the day after the initial coiling continued to show aneurysm obliteration.

Discussion

Our group and others have previously reported a 2–4% incidence of coiling related aneurysm rupture. We have suggested in a previous publication that outcomes can depend on time of rupture and pre-rupture control of the patient's airway and

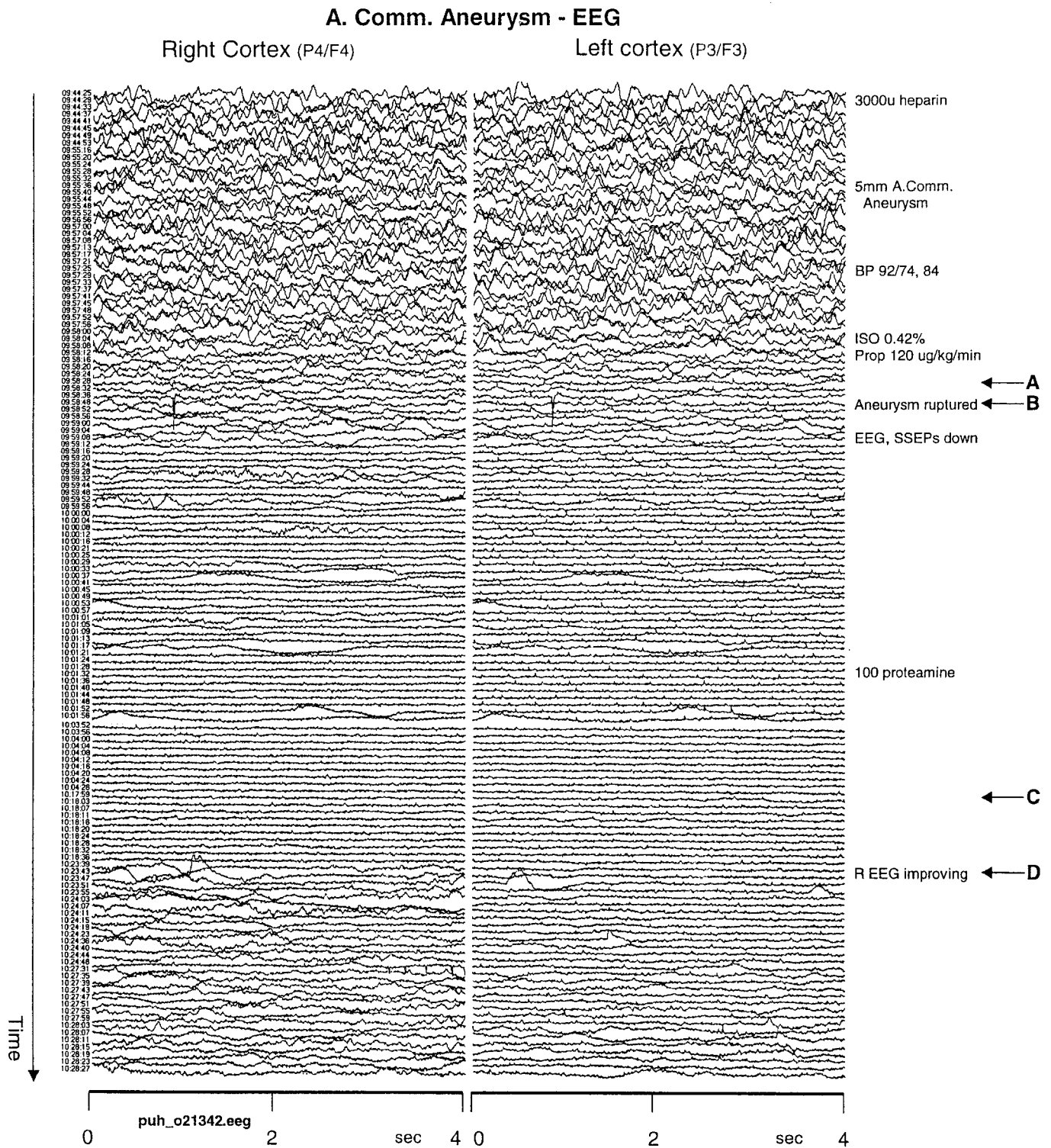


Fig. 4 EEG was continuously acquired from a two-channel scalp montage with one pair of electrodes recording between the right (P4/F4 electrode sites) and a second pair between the left (P3/F3) parietal and frontal lobes. The two channels of EEG data were recorded simultaneously (amplified at 5K, filtered at 1–100 Hz) and displayed in 4-second intervals. The real time at which each pair of EEG tracings was acquired is shown to the left of the figure. Selected EEG trials are plotted with the earliest trial at the top and the latest at the bottom as illustrated by the arrow to the left. Comments entered into the data record are shown to the right of corresponding EEG trials. Arrows indicate the times of significant events. **a** The EEG begins to decrease in amplitude and spectral content. **b** Data comment when aneurysm rupture was reported. **c** Time at which EVD placement began. **d** Onset of recovery of the EEG recorded at the right parietal/frontal cortex.

blood pressure. Improved outcomes appeared to be related to rupture during late coil deposition as well as pre-rupture airway control. We do not routinely place ventriculostomies in Hunt and Hess grade 1–3 patients prior to endovascular therapy because

of the belief that such placement can induce aneurysm rupture as ICP is lowered. In addition, the vast majority of our better grade patients never require temporary CSF diversion during hospitalization. By not routinely placing ventriculostomies we

A. Comm. Aneurysm – LE SSEPs

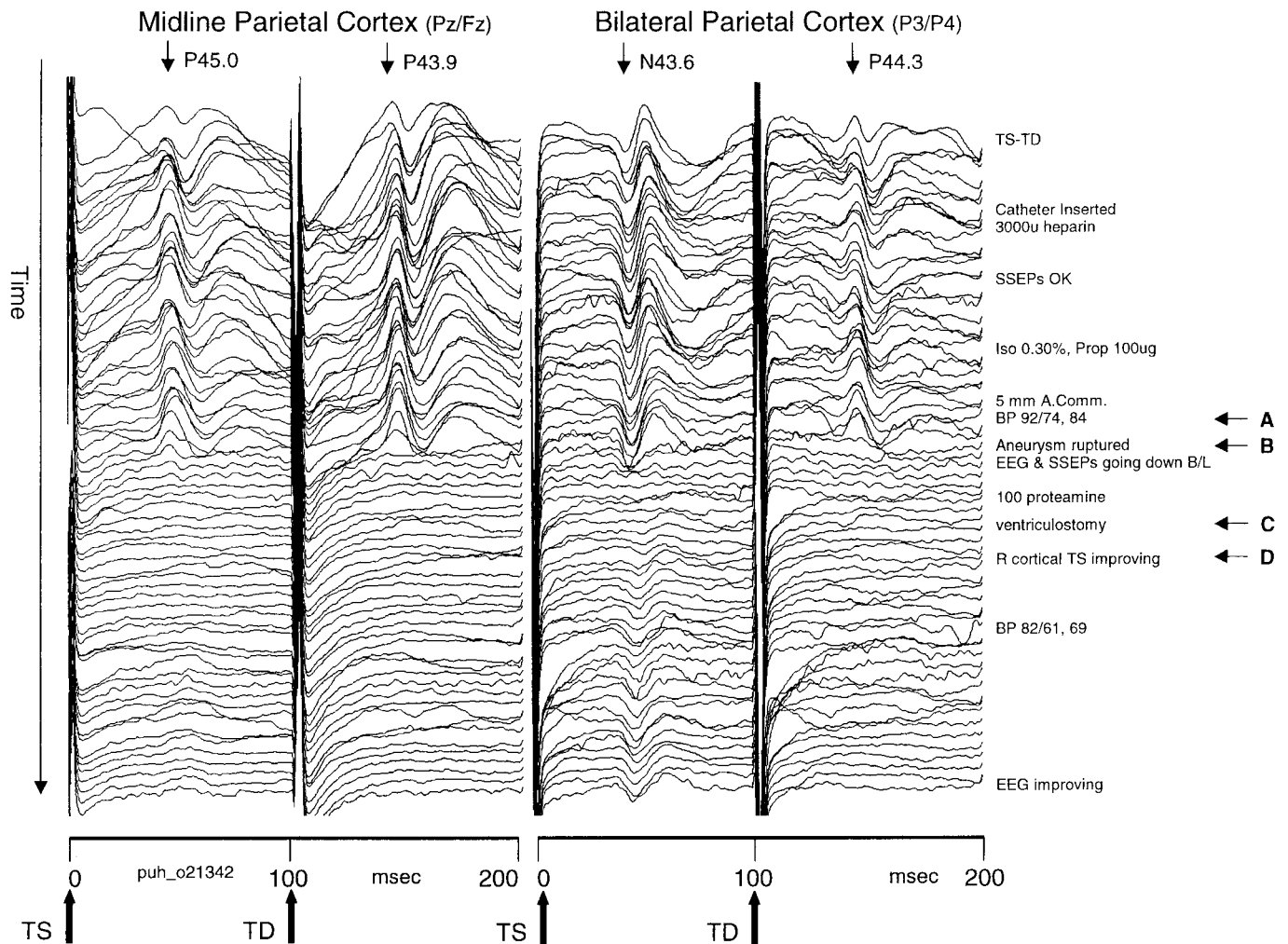


Fig. 5 Lower extremity (LE) somatosensory evoked potentials (SSEPs) were obtained from stimulating the left (TS) and right (TD) posterior tibial nerves at the ankles (110 V pulses of 0.2 ms duration applied at a rate of 3.43 Hz) and recording evoked potentials from the scalp (100 ms duration trials of EEG were accumulated, each time locked to TS and TD stimulus presentation and 128 such trials were averaged to form a single SSEP waveform) using subcutaneous needle electrodes at all sites. A two-channel electrode montage on the scalp recorded the cortical SSEP components from both midline, sagittal electrodes (amplified $\times 20$ K, filtered 3–300 Hz) between parietal and frontal lobes (Pz/Fz from the 10–20 system for electrode placement) and bilateral parietal lobe electrodes (P3/P4). The LE SSEP averages were acquired throughout the procedure with the earliest, baseline responses shown at the top and the last recorded waveforms at the bottom. The SSEPs obtained from the midline montage are on the left, with those from the bilateral montage on the right, each in response to both TS and TD stimulation. Comments entered into the data record are shown to the right of corresponding waveforms. Arrows indicate the times of significant events.

a The SSEP waveforms begin to change. **b** Data comment when aneurysm rupture was reported. **c** Time at which a right ventriculostomy and EVD placement began. **d** Onset of recovery of the N43.6 component of the SSEP waveform from left tibial nerve stimulation (TS) recorded at the bilateral parietal electrodes.

are able to avoid unnecessary procedures. Hunt and Hess grade 4 and 5 patients routinely have ventriculostomies inserted prior to therapy to best assess their neurologic examination without underlying intracranial hypertension. In those patients that do not have pretreatment CSF diversion, however, we have always felt that early ventriculostomy placement was critical to improved patient outcomes following intraprocedural rupture. If the aneurysm has been completely obliterated there should be no risk for subsequent aneurysm re-rupture once the ICP is lowered. In those cases where the aneurysm cannot be excluded from the circulation ventriculostomy is, in our opinion, also warranted in an effort to reduce life altering intracranial hypertension. The use of neurophysiologic monitoring in the intubated patient is the key in such cases of unsecured aneurysms because it can give

the surgeon an indication that cerebral blood flow has fallen below 15 cc/100 mg/min. It has been shown in experimental models that blood flow of about 2 ml/100 g of brain tissue/minute can be tolerated for several minutes with complete return of cortical electrical activity [3]. However, if cerebral ischemia is prolonged, irreversible damage can occur [7]. For example, Astrup et al. suggested that cerebral blood flow below 10 ml/100 g/min might lead to irreversible ischemia [1].

A cerebral blood flow below 15 ml/100 g/min has been correlated with significant EEG changes [3,8]. Experimental models of middle cerebral artery occlusion demonstrated significant changes in UE SSEP amplitude at cerebral flows less than 16 ml/100 g/min with no discernible responses at flows less than 12 ml/

A. Comm. Aneurysm – UE SSEPs

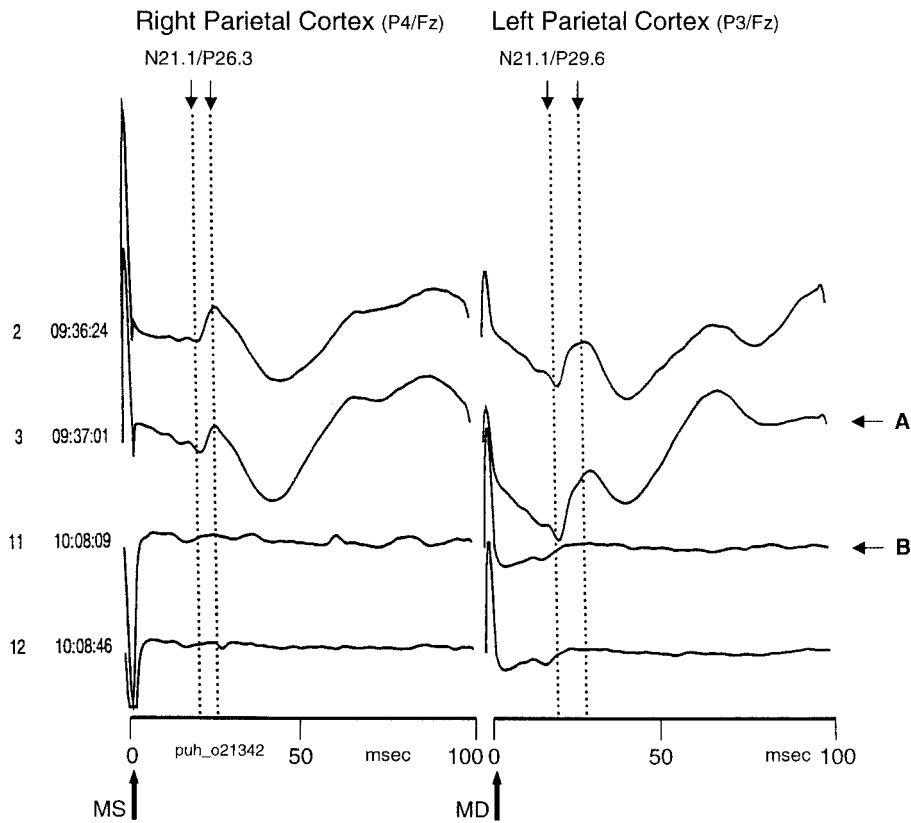


Fig. 6 Upper extremity (UE) somatosensory evoked potentials (SSEPs) were obtained from stimulating the left (MS) and right (MD) median nerves at the wrists (80 V pulses of 0.2 ms duration applied at 3.43 Hz) and recording evoked potentials from the scalp (100 ms duration trials of EEG were accumulated, each time locked to MS and MD stimulus presentation and 128 such trials were averaged to form the a single SSEP waveform) using subcutaneous needle electrodes at all sites. A two-channel electrode montage on the scalp recorded the cortical SSEP components from 2 pairs of electrodes (amplified $\times 20$ K, filtered 3–300 Hz) between the right (P4/Fz) and left (P3/Fz) parietal/frontal lobes. The UE SSEP averages were acquired periodically throughout the procedure with 4 simultaneously recorded SSEP waveforms shown. Waveforms 2 and 3 (indicated by arrow **A**) were baseline responses obtained at the outset of the procedure while waveforms 11 and 12 (arrow **B**) were obtained approximately 10 minutes after aneurysm rupture.



Fig. 7 Left oblique ICA arteriogram demonstrating embolized aneurysm. Transit time with the ventriculostomy inserted is now immediately < 0.05 seconds.

100 g/min [2]. If the ischemic interval was not longer than 15 minutes, restoration of SSEP activity resumed with the return of flow [2].

In this patient, the absence of discernible EEG and both UE/LE SSEPs following aneurysm rupture and excellent neurological outcome suggests that cerebral flow rates were lower than 15 and 12 ml/100 g/min for the approximately 25 minutes of cerebral ischemia and that during this time, although electrically silent, cerebral cortical neurons remained in a viable state. In patient with such a clinical condition ventriculostomy for raised ICP is warranted. If neurophysiologic changes are not seen then ventriculostomy placement can be avoided at a time when unnecessary placement might induce aneurysm re-hemorrhage from alterations in transmural pressure.

Conclusion

This case vividly documents the immediate effects of aneurysm rupture on cerebral circulation transit times, intracranial pressure, and neurophysiologic function. It also demonstrates the effect of early ventricular drainage on such parameters. Rapid reversal of intracranial hypertension and amelioration of its effects likely improves patient survival and clinical outcome. As neurosurgeons increasingly perform endovascular procedures or are called upon to assist their interventional colleagues with endovascular complications they must remain cognizant of the fact that intracranial hypertension kills and must be controlled to give patients the best chance for acceptable outcomes.

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