Cerebral aneurysm clipping requires periods of hypotension to facilitate dissection and clip application. We describe the use of rapid ventricular pacing to facilitate controlled hypotension for an 18 month old child presenting for giant basilar artery aneurysm clipping. This technique is an alternative to pharmacologic methods of inducing hypotension for neurosurgical procedures.

**Case Report**

An 18 month old male with history of severe traumatic brain injury due to motor vehicle crash several months prior presented with giant basilar artery pseudoaneurysm. The neurosurgical team required periods of hypotension during the procedure to facilitate dissection of the aneurysm from surrounding tissue and dissection of pontine perforating branches from the aneurysm. Following induction of anesthesia, femoral venous access was obtained and a pacing wire placed under radiographic guidance in the right ventricle. The feasibility of inducing hypotension with rapid ventricular pacing was confirmed; a ventricular rate of 300 beats per minute lowered the mean arterial pressure (MAP) to 23 mm Hg. A Medtronic Model 5328 Programmable Stimulator (Medtronic, Minneapolis, Minnesota) was utilized for pacing.

Ventricular pacing was employed to facilitate surgical dissection and clip placement at seven time points during the procedure. Specifically, the dome of the aneurysm was adherent to surrounding tissue due to scar formation following the brain injury. Prior to each episode of pacing, a propofol bolus was administered to attempt to induce EEG burst suppression by Bispectral Index (BIS) Monitoring; the patient was then paced for up to 75 seconds. A ventricular pacing rate of 300 bpm produced an average MAP of 26 mm Hg (Table). We noted rebound hypertension, with a peak MAP occurring two minutes after return to sinus rhythm, of decreasing magnitude throughout the procedure. The patient experienced ventricular fibrillation after the final pacing episode that was immediately terminated following asynchronous defibrillation (2 J/kg), returning the sinus rhythm.

**Discussion**

Cerebral aneurysm clipping requires balance between cerebral perfusion pressure (CPP) and the aneurysmal transmural pressure gradient (TMPG) (Priebe 2007). Normal to elevated CPP reduces the likelihood of cerebral ischemic injury; minimal TMPG reduces the propensity for aneurysm rupture. These parameters are each defined by the difference between mean arterial pressure (MAP) and intracranial pressure (ICP).

The requirement for discreet periods of hypotension during the procedure complicates the competing goals of maintaining CPP while minimizing TMPG. Hypotensive periods must be minimized to maintain CPP, but such periods are required to facilitate aneurysm preparation. The anesthesiologist is challenged to provide episodes of hypotension of reliable and reproducible magnitude and of a short and controlled duration. Such an ideal technique must be available, easy to administer and pose minimal risk to the patient. Current options are often limited to pharmacologic agents, both inhalational and intravenous.

Inhalational agents (isoflurane, sevoflurane) decrease systemic vascular resistance (SVR) and MAP but cause concurrent cerebral vasodilation, thus increasing cerebral blood volume (Tobias 2002). Furthermore, patients exhibit resistance to the hypotensive effects of these agents (Tobias 2002).

Similarly, while intravenous agents including sodium nitroprusside and nitroglycerin are direct acting peripheral vasodilators (via nitric oxide activity), their cerebral effects include vasodilation resulting in increased blood flow and intracranial pressure. Yaster et al found that nitroglycerin exhibits a ceiling effect regarding magnitude of hypotension in children (Yaster et al 1986). Nicardipine is a cerebral vasodilator which reliably decreases MAP, but return to baseline is prolonged (Tobias et al 1996). Adenosine is an alternative to these agents; it causes decreased cardiac output and decreased SVR (Tobias 2002) but displays a wide dose range with variable time to response and duration of asystole (Hashimoto 2000).

Rapid ventricular pacing is an alternative to pharmacologic options. The uncoupling of atrial and ventricular synchrony decreases ventricular filling and cardiac output, thus avoiding the problems of increased cerebral blood volume. This technique has been successfully employed in adult patients for placement of transcutaneous aortic valves and thoracic endografts (Billings et al 2009). Benefits of pacing to induce hypotension include reliable, reproducible hypotension, quick onset and offset, and ability to titrate mean pressure to desirable level. A limitation of this technique is the possibility of ventricular fibrillation post-pacing, as occurred in our patient, likely due to hypotension-induced myocardial ischemia.

**Conclusion**

This case report demonstrates that rapid ventricular pacing is a novel, reliable method for providing discreet periods of controlled hypotension in children. Further study is anticipated.

**References**