

Title: EFFECTS OF NITROPRUSSIDE ON AORTIC AND INTRASPINAL PRESSURES DURING THORACIC AORTIC CROSSCLAMPING

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Introduction. Paraplegia is an uncommon but devastating complication of thoracic aortic crossclamping during aortic surgery. Blaisdell and Cooley in 1962 reported increased intracranial pressure (ICP) during aortic crossclamping in the dog, and found that draining off cerebrospinal fluid (CSF) or pretreatment with urea decreased the incidence of paraplegia, while increasing ICP with saline increased the incidence of paraplegia.¹ Increases in ICP during aortic crossclamping are probably secondary to increased cerebral blood flow occurring when arterial pressure exceeds the upper limit of cerebral autoregulation. A recent report of a patient who developed postoperative paraparesis described an elevation of intraspinal pressure (ISP) after thoracic aortic crossclamping that was further increased by administration of sodium nitroprusside (ISP = 41 mmHg, MAP distal to crossclamp = 38 mmHg). Using a model reported to yield 40-60% paraplegia after thoracic aortic crossclamp in the dog,³ we evaluated the effects of nitroprusside administration upon hemodynamics, ISP and postoperative neurologic status.

Methods. Twenty-one pentobarbital-anesthetized dogs were studied during 60 minutes of thoracic aortic crossclamping just below the left subclavian artery.³ The right carotid and femoral arteries were cannulated to measure pressures above and below the crossclamp. A laminectomy at L3-L4 was performed and an 18 gauge Jelco Teflon catheter placed subdurally under direct vision to measure ISP. Respiration was adjusted to maintain PaCO₂ at 41 ± 1 mmHg (mean ± standard error) and PaO₂ at 199 ± 3 mmHg. In seven dogs nitroprusside was administered intravenously to reduce the carotid mean arterial pressure to the pre-crossclamp value. Dogs were evaluated for neurologic deficit 48 hours after aortic crossclamping.

Results. An increase in ISP was seen after thoracic aortic crossclamping (4 ± 1 mmHg pre-crossclamp to 7 ± 1 mmHg during cross-clamp, range of increase 0-5 mmHg). Concomitantly, MAP above the crossclamp increased from 125 ± 7 to 156 ± 6 mmHg. Addition of sodium nitroprusside resulted in further increases in ISP in 4 of 7 animals (not significant) and a 28% decrease in aortic pressure beyond the crossclamp (25 ± 4 mmHg pre-nitroprusside to 18 ± 3 mmHg during nitroprusside). In two nitroprusside animals, increases in ISP equaled or exceeded femoral pressures distal to the aortic crossclamp (Figure 1). The large number of deaths 48 hours post-crossclamp (7/14 non-nitroprusside and 6/7 nitro-prusside dogs) made comparisons between the two groups regarding spinal cord damage impossible. At autopsy histology from three nitroprusside dogs dying less than 48 hours after crossclamping showed evidence of bowel necrosis, massive hepatic necrosis, and hemorrhagic congested lungs.

Discussion. The present study supports a recent

case report of increases in ISP after cross-clamping of the thoracic aorta and the further increase caused by nitroprusside.² A 28% decrease in aortic pressure distal to the aortic crossclamp seen after the addition of nitroprusside is similar to the recent findings of Gelman et al.⁴ The fact that nitroprusside decreased aortic pressure distal to the crossclamp and further increased ISP in some animals should theoretically have a deleterious effect on spinal cord perfusion. In spite of this, nitroprusside has been used in a large surgical series in which a low incidence of spinal cord damage is reported. Further studies, possibly in a model with double aortic crossclamping and shorter crossclamp times, are needed to evaluate the effects of sodium nitroprusside on neurologic outcome during thoracic aortic crossclamp.

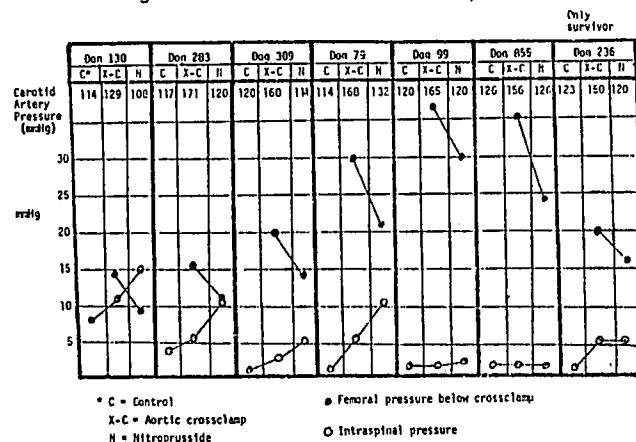


Figure 1: Intraspinal and femoral pressures for animals treated with nitroprusside. Pressures are recorded for each dog during a control period, 5 minutes after aortic crossclamp, and 10 minutes after nitroprusside infusion was begun.

References.

1. Blaisdell FW, Cooley DA: The mechanism of paraplegia after temporary thoracic aortic occlusion and its relationship to spinal fluid pressure. *Surgery* 51:351-355, 1962
2. Berendes JN, Bredee JJ, Schipperheyn JJ, Mashhour YAS: Mechanisms of spinal cord injury after crossclamping of the descending thoracic aorta. *Circulation* 66:1112-1116, 1982
3. Symbas PN, Pfaender LM, Drucker MH, Lester JL, Gravanis MB, Zacharopoulos L: Crossclamping of the descending aorta, hemodynamic and neurohumoral effects. *J Thorac Cardiovasc Surg* 85:300-305, 1983
4. Gelman S, Reyes JG, Fowler K, Samuelson PN, Leil WA, Smith LR: Regional blood flow during cross-clamping of the thoracic aorta and infusion of sodium nitroprusside. *J Thorac Cardiovasc Surg* 85:287-291, 1983