

# Intraoperative Hypotension Attributable To Septicemia And Diagnosed By Measurement Of Central Venous Oxygen Saturation

V Krishnamoorthy, K Beckmann, A Gustin, C Laurito

## Citation

V Krishnamoorthy, K Beckmann, A Gustin, C Laurito. *Intraoperative Hypotension Attributable To Septicemia And Diagnosed By Measurement Of Central Venous Oxygen Saturation*. The Internet Journal of Anesthesiology. 2010 Volume 29 Number 1.

## Abstract

Intraoperative hypotension may contribute to significant post-operative morbidity and mortality. We present a case of sudden, profound intraoperative hypotension during Harrington rod revision for scoliosis. During resuscitation of the patient, measurement of the central venous oxygen saturation (CVO<sub>2</sub>) allowed us to narrow our differential diagnosis. We noted a significantly elevated CVO<sub>2</sub> and, in the setting of hypotension, diagnosed intraoperative sepsis. After rod removal and stabilization of the patient's hemodynamics, we cancelled the remainder of the case due to the concern of bacteremia and infection of any newly placed hardware. Cultures from the wound later grew methicillin-resistant staphylococcus aureus. This case highlights the value of central venous oxygen saturation in a clinical picture to diagnose the cause of intraoperative hypotension

## INTRODUCTION

Intraoperative hypotension may contribute to significant post-operative morbidity and mortality. We present a case of sudden, profound intraoperative hypotension during Harrington rod revision for scoliosis diagnosed by measurement of the central venous oxygen saturation.

## CASE REPORT

The patient is a 12 year-old female with idiopathic kyphoscoliosis. One year prior to presentation, she underwent a posterior spinal fusion and Harrington rod placement. Her post-operative course was complicated by hardware infection treated conservatively with parenteral antibiotics. She presents one year later for revision of this fusion due to rod migration, explant of the Harrington Rods, and hardware replacement. The patient has no complaints, stable vital signs, and no changes in her medical condition.

In the operating room, standard monitors were placed, and an uneventful intravenous induction of anesthesia with propofol, fentanyl, and vecuronium was performed. A 7.0 endotracheal tube was placed without difficulty, and anesthesia was maintained with 2.0% (expiratory concentration) Sevoflurane and fentanyl; no additional muscle relaxant was given to allow for sensory and motor-

evoked potential monitoring. A right internal jugular vein catheter was placed using ultrasound guidance; the central venous pressure was monitored. Two large-bore peripheral lines were then placed. Multiple attempts to cannulate the radial, dorsalis pedis, and axillary arteries were unsuccessful. The decision was made to proceed with the operation using an oscillometric blood pressure cuff (cycled every 3 minutes); our plan included reattempting radial catheter placement with a small catheter intraoperatively, and (if unsuccessful) potentially abandoning arterial line placement for the procedure.

The patient was positioned prone and 750mg of Vancomycin was slowly infused over one hour for surgical prophylaxis. Surgery proceeded uneventfully and the wound was dissected to the level of the lamina. A serous pocket was encountered, drained, and fluid sent for gram stain and cultures. At this time, the patient became acutely tachycardic to 140 beats per minute; the cuff blood pressure, which had been 126/64, measured 50/32. The end-tidal CO<sub>2</sub> decreased to 22 from 36. One liter of normal saline was rapidly infused, and the patient was given a total of 400 mcg of phenylephrine and 20 mg of ephedrine with minimal increase in blood pressure to 64/36. 12 mcg of epinephrine was injected over 2 minutes, and the cuff measured 96/54.

While maintaining a broad differential diagnosis, we entertained intraoperative sepsis and anaphylaxis at the top of our list. We continued supportive treatment of the patient, and a central venous blood gas was sent for analysis. The results revealed: pH: 7.29, PCO<sub>2</sub>: 43.2, PO<sub>2</sub>: 80.8, HCO<sub>3</sub><sup>-</sup>: 20.1, O<sub>2</sub> Saturation: 94.9%, HgB: 11.5, Glu: 119, K: 3.6, Na: 141. We noted the abnormally high central venous oxygen saturation; and we deduced that a decrement in oxygen extraction at the tissue level was occurring. Thus, the diagnosis of severe transient septic shock was entertained. The patient had stable hemodynamics after this episode and required no additional pressor support. Once the hardware was removed, the remainder of the case was cancelled due to presumed bacteremia and concern for infectious seeding of any newly placed hardware. With intraoperative treatment consisting of IV fluid, supportive therapy with vasopressors, and central venous pressure and oxygen monitoring, as well as infectious source control by the surgical service, the patient's hemodynamics were restored to normal. Central venous oxygen saturation was subsequently measured at 81.4% one hour after the previous sample. Cultures from the fluid pocket revealed methicillin-resistant staphylococcus aureus (MRSA), and long-term antibiotic therapy was initiated. The patient was discharged home in one-week in stable condition with significantly decreased back pain.

## **DISCUSSION**

This case highlights the value of central venous oxygen saturation in a clinical picture to diagnose the cause of intraoperative hypotension. The central venous blood gas data informed our decision to cancel the remainder of the case due to the risk of infection of the new hardware. The pathophysiology of septic shock includes hypoperfusion to vital tissue beds eventually resulting in cellular dysfunction. Uncoupling of oxygen delivery from oxygen extraction at

the tissue level is a hallmark of septic shock. Left untreated, anaerobic metabolism ensues, lactic acidosis occurs, and cell death occurs. Early goal-directed therapy in this setting decreases 28-day mortality; a marker of clinical improvement is restoration of SVO<sub>2</sub> to normal levels. Several case reports in the literature have demonstrated the use of early goal-directed therapy in patients who develop septic shock intraoperatively by following central venous oxygen saturation. Our case further illustrates the usefulness of this technology in a patient who developed sudden intraoperative hypotension. Recognition of an elevated central venous oxygen saturation solidified our diagnosis and preempted placing new hardware in a patient with MRSA bacteremia.

## **References**

1. Anesthetic management and one-year mortality after non-cardiac surgery  
Monk, et al; *Anesth Analg*, 2005 Jan; 100(1): 4-10
2. Intraoperative hypotension and 1-year mortality after non-cardiac surgery  
Bijker, et al; *Anesthesiology*, 2009 Dec; 111(6): 1183-1184
3. Early goal-directed therapy in the treatment of severe sepsis and septic shock  
Rivers, et al; *N Engl J Med*, 2001 Nov; 345: 1368-1377
4. Medical progress: The pathophysiology and treatment of sepsis  
Hotchkiss, et al; *N Engl J Med*, 2003 Jan; 348: 138-150
5. Early goal-directed therapy (EGDT) using continuous central venous oxygen saturation monitoring in a patient with septic shock  
Oyama, et al; *Masui (Japanese)*, 2008 Apr; 57(4): 443-446
6. Venous oximetry: physiology and therapeutic implications  
Blasco, et al; *Ann Fr Anesth Reanim (French)*, 2008 Jan; 27(1): 74-82
7. Should we use central venous saturation to guide management in high-risk surgical patients?  
Pearce, et al; *Crit Care*, 2006; 10(6): 181
8. The role of mixed venous oxygen saturation in perioperative monitoring and therapy. A critical stock taking  
Wiesemans, et al; *Anesthesiol Intensivmed Notfallmed Schmerzther (German)*, 1993 Aug; 28(5): 269-278

**Author Information**

**Vijay Krishnamoorthy, M.D.**

Department of Anesthesiology, University of Illinois College of Medicine

**Katharina Beckmann, M.D.**

Department of Anesthesiology, University of Illinois College of Medicine

**Allen Gustin, M.D.**

Department of Anesthesiology, University of Illinois College of Medicine

**Charles E. Laurito, M.D.**

Department of Anesthesiology, University of Illinois College of Medicine