Bradycardic Asystolic Arrest in an Obese Parturient During Caesarian Section Under Spinal Anesthesia

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Bradycardic Asystolic Arrest in an Obese Parturient During Caesarian Section Under Spinal Anesthesia
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INTRODUCTION
Unexpected cases of delayed bradycardia and asystole during neuraxial anesthesia in young healthy patients has been reported. True incidence and predisposing factors and the exact pathophysiology has yet to be completely defined.
We are reporting a case of bradycardic asystolic arrest in an obese parturient during caesarean section under spinal anesthesia.

POSSIBLE ETIOLOGY
The exact mechanism of bradycardia/asystole during spinal anesthesia is not completely well defined. Cardiac arrest has been reported within 12 to 72 minutes of spinal anesthesia.

Common physiological explanation may include:
• High or total Spinal with blockade of cardio accelerator sympathetic fibers.
• Decrease venous return/preload may activate low pressure baroreceptors in right atrium and mechanoreceptors in Left ventricle stimulating Bezold-Jarisch reflex and cause severe bradycardia.
• Unopposed parasympathetic activity may produce significant degree of bradycardia and hypotension resulting in cardiac arrest.
• Hemorrhage or Aortocaval compression
• Other differentials include local anesthesia toxicity/premedication with sedative/vagolytics medications pulmonary or amniotic emboli.

CASE DESCRIPTION
A 26 year old obese parturient with BMI of 41 presented for emergency caesarean section for fetal distress under spinal anesthesia with hyperbaric Bupivacaine 0.7% 1.6 ml, Astramorph 3mg, Fentanyl 10 ug.

Twelve minutes post-delivery, patient presented with acute desaturation, loss of consciousness and progressive sinus bradycardia that progressed to asystole. CPR was initiated as per ACLS protocol along with administration of atropine/ephedrine/epinephrine and brief cardiac compression of less than 1 minute.

Patient was successfully intubated and responded to resuscitative measures with return of spontaneous circulation.
Surgery was completed uneventfully. Series of investigation including: EKG-Echo-cardiogram Head CT and lab work was conducted in attempt to identify etiology of cardiac arrest concluded with normal results. Patient was extubated 12 hours later without any neurological sequelae.

MANAGEMENT
• Anesthesiologist shall be vigilant; constant monitoring during spinal anesthesia
• Uterine displacement post spinal.
• Early recognition of bradycardia and impending asystole. Prompt intervention and CPR is essential to decrease frequency of and improve survival associated with cardiac arrest during neuraxial block.
• Epinephrine should be considered early in treatment of sudden bradycardia especially if conventional doses of atropine/ephedrine are not effective.
• Prompt intravascular fluids and correction of blood volume deficits
• Alpha and Beta agonist and vagolytics therapy has been proposed as measures of intervention.
• Our patient responded promptly to Atropine/Ephedrine/Epinephrine and cardiac compression with maintenance of secure airway with adequate ventilation and oxygenation.

REFERENCES
3. Liu SS, McDonald SB. Current issues in spinal anesthesia. Anesthesiology 2001; 94: 885-906