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Cardiac Arrest from Peritoneal Vagal Stimulation Is Possible During Abdominal Insufflation: A Case Report

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Background

A clear association between episodes of potentially fatal cardiovascular collapse during abdominal insufflation due to venous gas embolization has been described in multiple previous reports since the 1970s. Other reports have described associations between unstable bradycardia and vagal stimulation from peritoneal stretch during insufflation. Bradycardia is often assumed to be a benign finding during abdominal insufflation and typically self-corrects or remains stable after decompression with reported frequency up to 28% of all abdominal laparoscopic cases. However, no cases of fulminant cardiac arrest from peritoneal stretch in the absence of venous gas embolization has been clearly described in this population. To our knowledge, this case report describes the first of its kind.

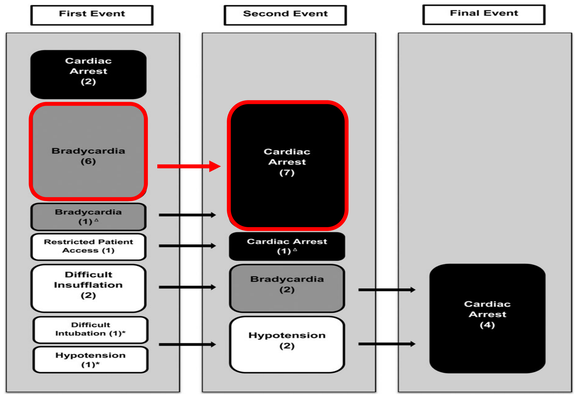

Figure 1: Reported events prior to cardiac arrest due to abdominal insufflation. (*) Anesthesia related averse events. (Δ) Associated hypocapnea.[4] Red box outlines sequence events specific to this case report.

Case Description

We present a 76-year-old male with no previous cardiac history who was 5 months status post emergent hemicolectomy and Hartmann's pouch creation for perforated sigmoid diverticulitis. After a thorough pre-anesthetic evaluation, he underwent general anesthesia for elective robotic-assisted colostomy reversal and parastomal hernia repair. Standard induction of general anesthesia was accomplished without complication. Shortly after pneumoperitoneum was established, the patient developed sinus bradycardia. He was administered 0.4 mg glycopyrrolate followed by immediate de-insufflation with recovery of heart rate. After re-attempt at insufflation, sinus bradycardia recurred and was accompanied by loss of plethysmography signal and unmeasurable BP by non-invasive BP cuff. No abrupt changes to end-tidal CO2 were identified by capnography and the patient was de-insufflated immediately without improvement of blood pressure or heart rate. He was examined to have no palpable carotid pulse and a code blue was initiated. After following 6 minutes of standard ACLS protocol for pulseless electrical activity, return of spontaneous circulation was achieved and the patient was transferred to the ICU in stable condition, the planned procedure aborted. Repeat ECG and chest x-ray demonstrated no significant changes from baseline. Initial cardiac troponins were elevated at 0.71 but repeat troponin down-trended to 0.52. Metabolic acidosis with elevated lactic acid initially at 3.2 improved to 1.7 after initiating hypothermic protocol in the ICU and IV fluid resuscitation. His postoperative course remained uncomplicated and the patient was deemed suitable for discharge home on postoperative day 6.

Discussion and Conclusion

Previous reports and larger clinical studies have established a common link between abdominal insufflation and unstable bradycardia for a number of underlying reasons with rare episodes of fulminant cardiovascular collapse due to CO2 gas embolization. These reports have not identified any cases of cardiovascular collapse due to peritoneal stretch in the absence of gas embolization. Bradycardia during insufflation has been associated with other comorbidities including those on chronic cardiac medications but a more recent report suggest unstable bradycardia can occur in otherwise healthy patients and may still result in cardiac arrest presumably due to air embolism.[4] This same study also showed that bradycardia associated with pneumoperitoneum most frequently preceded cardiac arrest (Fig. 1). Therefore, unstable bradycardia should be generally perceived as a possible harbinger to cardiac arrest, even in individuals without any previous risk factors for coronary artery disease. The prophylactic use of atropine has been advocated as an effective preventive measure[5] but, at the very least, a high level of vigilance by the anesthesiologist during each laparoscopic case may be warranted as our case report suggests there is more to understand about this underlying pathophysiology.

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