

# Differential diagnosis of intraoperative cardiac arrest after spine surgery in prone position

### ABSTRACT

Intraoperative cardiac arrest is one of the most feared events by anesthesiologists and surgeons. Although there are many possible causes, three differential diagnoses stand out in the presented scenario: pulmonary embolism, gas embolism, and acute myocardial infarction. A 61-year-old female patient was admitted in the hospital to C2-C5 arthrodesis. Despite no major bleeding during surgery, immediately after supination the patient developed refractory hypotension, a decrease in end tidal CO<sub>2</sub>, progressive bradycardia that ultimately led to pulseless electrical activity. Resuscitation maneuvers were promptly performed, sustained return of spontaneous circulation was attained after 50 minutes, and the patient was transferred to the ICU. This paper discusses the main causes for an episode of cardiac arrest in the context of cervical arthrodesis, with a markedly prolonged resuscitation time, in which the patient survived.

**Key words:** Air embolism, cardiac arrest, gas embolism, intraoperative cardiac arrest, intraoperative myocardial infarction, pulmonary embolism, spine surgery

### Introduction

Intraoperative cardiac arrest (IOCA) is among the most feared events by anesthesiologists. It is an ominous occurrence with a 50% lethality rate.<sup>[1]</sup> Three differential diagnoses stand out in the presented scenario: pulmonary embolism (PE), gas embolism (GE), and acute myocardial infarction (MI). Reaching a specific diagnosis is challenging, since patients cannot refer to symptoms and full physical examination is often not possible. The clinician must react quickly and decisively to emerging clues.


### Case History

A 61-year-old female suffering from depression, fibromyalgia, and hypertension was being treated for an L2 extramedullary plasmacytoma that had disseminated to C5-T2. She underwent arthrodesis between C5-T4 and T12-L4, during which she developed hemodynamic instability and transitory ST segment elevation, later diagnosed as a type 2 MI. Diagnostic coronary catheterization was executed, but no major issues were found. A transthoracic echocardiography reported normal right and left ventricular function.

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A year later, the patient was readmitted due to upper limb weakness and a burning neck pain irradiating to both hands. An uneventful decompressive C4-T4 arthrodesis was performed but ultimately failed after a week, due to loosening of osteosynthesis material attributed to poor bone quality, ending up in cervical instability.

A new C2-C5 arthrodesis in the prone position lasting 5 hours was performed. Despite no major bleeding nor hemodynamic instability until wound closure, the patient developed sudden and refractory hypotension immediately after supination, accompanied by decreased end tidal CO<sub>2</sub> (ETCO<sub>2</sub>) and progressive bradycardia that ultimately led to pulseless electrical activity (PEA). Resuscitation maneuvers were promptly initiated, and sustained return of spontaneous circulation was attained after 50 minutes, but the patient remained hemodynamically unstable and a deterioration in oxygenation was perceived, such that PaO<sub>2</sub>/FiO<sub>2</sub> ratio was 60.4 and PaCO<sub>2</sub> 101.4 mmHg, despite normal tidal volumes. Point-of-care echocardiography showed marked hypocontractile left ventricle (LV) and an even worse right ventricle (RV). A poor echocardiographic window prevented more elaborated evaluation at the time. The patient was transported to intensive care receiving norepinephrine, vasopressin, epinephrine, and dobutamine.

Echocardiography performed 2 days later showed hypocontractile and dilated RV, TAPSE 13 mm, and PSAP of 37 mm. Meanwhile, LV had a mild systolic dysfunction. Five days later, LV had normal systolic function, but RV remained hypocontractile. Postoperative troponin I was 11.600 ng/L, and D-dimer was not measured due to institution limitations.

After several weeks, the patient woke up and was transferred to infirmary care, still wearing a neck brace. Upon examination, Glasgow 15/15, the patient preserved strength and sensitivity in all four limbs, except for difficulty closing her left hand. Mild respiratory effort persisted, and an oxygen flow of one liter per minute was necessary to maintain normal saturation.

## Discussion

Among the many possible causes for IOCA, three stand out here: pulmonary embolism, gas embolism, and myocardial infarction.

Prone position creates a state of blood stasis and decreased venous return in lower extremities. The cushions used keep the legs below the heart, and the flexed position of the hips

and knees also interferes with blood circulation. Abdominal compression further exacerbates this process. Thus, it is no surprise that PTE after spinal surgery has an incidence of 19–25%.<sup>[2]</sup> Its intraoperative diagnosis, however, is challenging, given that classic signs such as tachypnea and dyspnea are absent. When there is sufficient magnitude, clues include sudden ETCO<sub>2</sub> reduction, increased PaCO<sub>2</sub>, and appearance of hemodynamic instability.<sup>[3]</sup> Lethality rates of cardiac arrest due to pulmonary embolism are very high, 65–90%. PEA is observed in up to 50% of patients, and shockable rhythms are rarely seen.<sup>[4]</sup>

GE can occur in any procedure where the surgical site is located above the heart. Besides, many veins in the cervical vertebrae are devoid of valves, so their intraluminal pressure is directly dependent on their relation to the heart position. Although GE is common in cervical surgery in the prone position, it is usually asymptomatic. Nevertheless, significant air volumes can precipitate right ventricular outflow tract obstruction, causing dramatic drops in cardiac output, with consequent hemodynamic instability, abrupt reduction in ETCO<sub>2</sub>, and oxygen saturation.<sup>[5]</sup> Lethality rates are about 20%.<sup>[6]</sup> The lethal volume is around 200–300 ml or 3–5 ml/kg, and the closer the vein of entrainment is to the right heart, the smaller the expected lethal volume.<sup>[7]</sup> The main therapeutic interventions are: interrupting air entrainment by flooding the surgical site, placing the patient in the Trendelenburg position to increase venous pressure, and forcing the exit of air emboli from the heart through inotropic stimulation. There are few reports in the literature of GE cases during spinal surgery with major hemodynamic repercussions.<sup>[5]</sup>

As for MI, there are two main mechanisms: Type 1 occurs due to unstable or vulnerable plaque, whereas type 2 implies oxygen supply–demand imbalance.<sup>[8]</sup>

Given that our patient had ordinary preoperative echocardiography and coronary angiography, type 1 MI was ruled out. Furthermore, since there was minimal blood loss and hemodynamic stability was maintained up until supination, type 2 MI was considered less likely as a primary cause for the sudden hemodynamic instability. Therefore, the high troponin levels later obtained were attributed to ischemic insult secondary to the cardiac arrest combined with the trauma from chest compressions. Thus, although type 2 MI could be a cause, it was more likely a consequence.

PE and GE share several clinical and laboratorial manifestations. Both are compatible with the cardiovascular instability that

developed upon supination, both can explain hypoxemia, elevated  $\text{ETCO}_2$ - $\text{PaCO}_2$  gap, and right heart failure, and either could justify PEA.

Therefore, in this scenario, we cannot assertively rule out either of them. Perhaps, the return to spontaneous circulation without thrombolysis and the extremely high lethality of PE-related cardiac arrests might suggest gas embolism as a primary hypothesis.

The author's aim is to raise awareness toward the main causes of IOCA, so that prompt interventions can be undertaken, and lives can be saved.

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#### **Conflicts of interest**

There are no conflicts of interest.

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