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Anesthesia and Intensive Care

# **Unexpected Cardiac Arrest during Spinal Anesthesia**

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#### Abstract

Case Report

Intraoperative cardiac arrest in the operating room (CAIOR) is an unexpected and potentially serious event. Two types of CAIOR must be distinguished: those directly attributable to anesthesia and CAIOR of other etiologies, notably surgical. The incidence of CAIOR directly attributable to anesthesia is between 0.5 and 1.86 per 10,000 anesthesias, Through the clinical case of a 62-year-old woman who presented a per operative caradiac arrest during a spinal anesthesia for operative hysteroscopy, and through a review of the literature, we will expose the risk factors of CAIOR, as well as the main etiologies. Symptomatic and etiological treatments must be implemented as soon as possible to avoid the mainly neurological consequences of circulatory failure. The management is based on symptomatic treatment with effective cardiopulmonary resuscitation, especially associated with the etiological treatment of cardiac arrest. But sometimes, the etiology of the cardiac arrest remains unidentified, and the treatment is therefore essentially symptomatic. The mortality rate of CAIOR remains high, the crude mortality rate observed 90 days after CAIOR is estimated at 48% (14) The survival of patients after CAIOR is intimately linked to the precocity and quality of their initial initial management.

Keywords: Cardiac arrest, intraoperative, Spinal anesthesia, hysteroscopy.

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# INTRODUCTION

Cardiac arrest (CA) may occur, although very rarely, during each anaesthetic process. Cardiac arrests in the operating room (CAIOR) represent about 2% of intra-hospital CA [1] and occur between 1.1 and 34.6/10,000 surgical procedures [2].

Two types of CAIOR must be distinguished: those directly attributable to anesthesia and CAIOR of other etiologies, notably surgical.

The incidence of CAIOR directly attributable to anesthesia is between 0.5 and 1.86 per 10,000 anesthesias, i.e., approximately 11% of CAIORs (3). Mortality directly attributable to anesthesia is estimated to be between 0.55 and 1.4 cases per 10,000 anesthesias [4, 5].

## CASE REPORT

We report the case of a 62-year-old female patient. Without any notable pathological history, never operated. She consulted her gynecologist for postmenopausal metrorrhagia. The ultrasound showed an intra uterine mass of 3\*2cm. The patient did not tolerate hysteroscopy without anesthesia, so she was scheduled for an operative hysteroscopy under anesthesia.

The patient was seen in pre anesthetic consultation. She was in good general condition, her weight was 82 Kg, her height was 167cm, her BMI was 29.4. She had no dyspnea at rest or during exercise, no chest pain, her functional capacity was > 4 METs.

The cardiorespiratory examination was normal, there were no heart murmurs. His vitals were normal with BP 132/68, HR 67Bpm, respiratory rate 16 and SpO2 97%. His preoperative workup was strictly normal, with a hemoglobin level of 12.7 g/dl, and normal renal function.

His ECG was also normal, with a regular sinus rhythm, HR at 68 bpm, he had no conduction or repolarization disorder. His frontal lung x-ray was normal, with a cardiothoracic index < 0.5.

The patient was not taking any long-term treatment, so she was classified as ASA I, scheduled for

a low-risk cardiovascular surgery. A benzodiazepinebased premedication was prescribed for the day before the operation.

After a preoperative fasting of 8 hours, the patient was admitted to the operating room, installed on an operating table, took an 18G peripheral venous line and filled with 250 of saline. Then the patient was installed in a sitting position, a spinal anesthesia was performed by an anesthetist at the level of the L3-L4 space, by injection of 7.5mg of hyperbaric bupivacain, with 25micrograms of fentanyl as an adjuvant, with a cofillment of 500 ml of isotonic saline.

The patient was then placed in the supine position and the operation began after 15 minutes of spinal anesthesia. Verbal contact was maintained with the patient, who responded perfectly to the questions of the anesthesiologist. The patient remained hemodynamically stable and did not present a postspinal hypotension.

20 minutes after the beginning of the procedure, the patient suddenly presented a cardiac arrest, objectified by a flat tracing on the cardio scope, and absence of carotid pulse.

Cardiac massage was started immediately, with direct IV injection of 1mg of adrenaline, orotracheal intubation was performed, with connection to the respiratory system, tidal volume at 400ml, FR 12, FiO2 at 100% and a PEEP at 0.

After 1min of cardiac massage the patient recovered a sinus rhythm, adrenaline was introduced at a dose of 2mg/h with a self-pulsing syringe. BP was 68/35 and HR was 90.

3min later the patient presented 3 successive episodes of ventricular fibrillation, requiring 3 external electric shocks, successively of 150, 200 and 200J. With introduction of amiodarone 300mg in loading dose, then 600mg/24h with auto pulsed syringe. The patient resumed a sinus rhythm thereafter, but required even more adrenaline with a dose of 4mg/h. The patient was then transferred to the intensive care unit for further management.

Trans thoracic echography showed no valvulopathy, nor signs of myocardial ischemia. Blood volume was normal. The patient's hemodynamic status continued to worsen, with a progressive increase in adrenaline doses. The patient died 12 hours later from refractory shock.

### **DISCUSSION**

Romain Jouffroy *et al.*, [6] has identified different predictive factors for the occurrence of CAIOR:

- The patient's ASA status
- The patient's age
- Late time of entry to the operating room
- The urgency and type of surgery [7, 8].
- The period of anesthesia: 23% of CAIOR occur during induction and 29% during maintenance of anesthesia [9]
- The type of anesthesia: general anesthesia (GA) is more frequently involved than locoregional anesthesia with frequencies of 5.5 and 1.5 per 10,000 anaesthesias respectively, and a complication concerning airway management is in 64% of cases. In regional anesthesia, the frequency of CAIOR is higher in axial anesthesia, especially spinal anesthesia, and is estimated to be about 1.8 per 10,000.

Cardiac arrests during spinal anesthesia are described as "very rare," "unusual," and "unexpected," but are actually relatively common [10]. The two largest prospective studies designed to evaluate the incidence of complications during spinal anesthesia reported two arrests in 1881 patients [11] and 26 arrests in 40,640 patients [12] for an overall incidence of seven arrests for every 10,000 (0.07%) spinal anesthetics.

Lena-Quintard D [9] has grouped the etiologies of CAIOR by nosological framework:

respiratory causes	Hypoxia: failed intubation, inhalation, pneumothorax, accidental extubation extubation,
	equipment failure, respiratory depression during regional regional anesthesia
	Bronchospasm
	Compressive pneumothorax
cardiovascular	True hypovolemic shock (hemorrhage) or relative hypovolemic shock (anaphylaxis)
causes	Transfusion accident
	Gas tamponade (compressive pneumothorax) or liquid tamponade (pericardial effusion)
	Pulmonary embolism
	Acute coronary syndrome
	Severe PAH
	Electrolyte abnormalities (dyskalemia)
	Intra-abdominal pressure
	Rhythm and/or conduction disorder
	Pacemaker malfunction
neurological	Stroke

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causes	Intracranial hypertension
Anesthetic causes	Malignant hyperthermia
	Intoxication by local anesthetics
	Overdose of local or general anesthetic agents
	Errors in administration

The occurrence of an CAIOR is a rare event that requires a well thought-out and established team organization to be efficient [6].

Symptomatic and etiological treatments must be implemented as soon as possible to avoid the mainly neurological consequences of circulatory failure.

The management is based on symptomatic treatment with effective cardiopulmonary resuscitation, especially associated with the etiological treatment of cardiac arrest.

External cardiac massage should be started as soon as possible according to the recommendations. Mechanical ventilation is usually performed in controlled assisted ventilation mode with protective ventilation.

Invasive blood pressure measurement is used to guide resuscitation and to take biological samples, but should not delay symptomatic and etiological management, with a target MAP of 60-65 mmHg.

IV and volatile anesthetic agents should be discontinued [11] due to the vasodilatory and/or negative inotropic properties of these agents.

Defibrillation and external electric shock (EES) should be performed as soon as possible if indicated, as they improve survival (between 150 and 200 Joules [1]. It is recommended to immediately resume chest compressions after external electric shock for 2 minutes before performing a new rhythm analysis.

Monitoring of EtCO2 is recommended, it also has a negative prognostic value if it is below 10mmHg after 20 min of CPR.

Cardio-thoracic ultrasound may identify hypovolemia, pulmonary thrombosis, choking pneumothorax or tamponade as reversible causes of CA.

Epinephrine remains the first-line vasopressor drug for the management of CA. The recommended dose is 1 mg every 2 cycles of CPR.

A 300-mg bolus of amiodarone is recommended for VF and VT refractory to 3 external electric shocks, followed by a maintenance dose of 900 mg/24 h if recurrent. Reversible causes of cardiac arrest must be identified and treated as soon as possible [6]. 8 reversible etiologies are to be sought and treated immediately in CAIOR: Hypoxia, Hypovolemia, Hypo/hyperkalemia, Hypothermia, Thrombosis (coronary or pulmonary), Suffocating pneumothorax, Tamponade and Intoxications [13].

This last cause regroups 3 etiologies, 2 of which are specific to anesthesia, anaphylaxis, malignant hyperthermia and intoxication to local anesthesics.

Sometimes, the etiology of the cardiac arrest remains unidentified, as in the case of our patient, and the treatment is therefore essentially symptomatic.

# CONCLUSION

Unfortunately, the mortality rate of CAIOR remains high, the crude mortality rate observed 90 days after CAIOR is estimated at 48% [14]. The survival of patients after CAIOR is intimately linked to the precocity and quality of their initial initial management.

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