

Case Report

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Cardiac arrest in the operating room: what to think? About 4 cases and review of the literature



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Introduction

Cardiac arrest in the operating theatre is a serious and dramatic incident, with an incidence of 5.6 per 10,000 procedures. The context of the operating theatre generates specificities in relation to the pre-hospital approach, both in terms of diagnosis and management. Under general anesthesia, the diagnosis and conduct of resuscitation is based on the monitoring that is already in place. Etiologies are primarily respiratory with the result of hypoxemia but also allergic and toxic accidents. Resuscitation is carried out in parallel with the control of the ventilation system. The prognosis is better due to the speed of diagnosis and resuscitation manoeuvres, which justifies prolonged resuscitation [1].

We report 4 observations of patients who had intraoperative cardiac arrest during a period from December 2015 to September 2019, which occurred in the operating theatre of the Moulay Ismail military hospital in Meknes.

Observations

Observation 1

64-year-old patient, with no significant pathological anteriority, with a normal basic preoperative check-up, admitted to the operating theatre for a surgical treatment of an acute intestinal obstruction on volvulus of the sigmoid. A standard anesthetic monitoring was performed, Hemodynamic and respiratory parameters were correct. The induction was made by fentanyl, propofol and rocuronium adapted to weight, the entretiens provided by the isoflurane. The hemodynamic parameters remained stable. Prior to the incision, the patient received an infusion of two grams amoxicilline-clavulanic acid. 10 minutes after the incision, blood pressure decreased from 125/85mmHg to 80/40 without responding to ephedrine injection and filling with elevated ventilation pressures. The evolution was

rapidly adverse by worsening the low blood pressure reaching up to 40/23mmHg and then settled a progressive bradycardia with wide QRS complexes. Then an adrenaline syringe shoot was set up. Gradually the hemodynamic parameters stabilized with lower ventilation pressures and improved saturation. A biological assessment was carried out after the introduction of the VVC and having objectified a correct ionogram with retained kidney function and a normal troponin at 0.0052 g/l (VN-INF 0.0350) and a negative CRP.

Biological monitoring of troponin is summarized below (Table 1):

Table 1

Time	H0	H6	H12	J24	J36
Troponin	0.0052	0.7079	1.7406	0.8983	0.0873

A labial edema settled 2 hours after the state of shock. And the interrogation with the family revealed a history of labial edema following the taking of NSAIDs. Electrocardiographic (ECG) recordings of the patient after 10min, at H6 and H24 did not reveal signs of ischemia or myocardial lesions, Transthoracic echocardiography did not objectify toxic disorders or valvulopathy and found a preserved ejection fraction. The patient was operated on 12 hours after this incident with invasive monitoring and without intraoperative complications

Observation 2

A 16-year-old patient with no particular history, who consults for lower limb edema set to a heart-compressing intrathoracic mass (Figure 1). She was admitted for exeresis of this mass. The patient presented a stop at anesthetic induction following heart failure, recovered by internal cardiac massage and adrenaline and an extreme bradycardia during pericardiorrhaphy also recovered.



Figure 1: Intrathoracic mass compressing the heart.

Observation 3

58-year-old patient, with no particular history, admitted for exeresis of a large mediastinal tumor (Figure 2) who had

intraoperative cardiac arrest following a rhythm disorder such as supraventricular tachycardia and haemorrhage following difficulty with surgical dissection.



Figure 2: large mediastinal mass resulting in intraoperative cardiac arrest

Observation 4

75-year-old patient, diabetic on insulin and hypertensive on ARA II, admitted for revascularization of lower limb ischemia

under rickets following intraoperative myocardial ischemia revealed by an ST segment underlay and positive troponin dosage (Table 2)

Table 2: Types of intervention, causes and evolution of cardiac arrests in the operating theatre of the Moulay Ismail Military Hospital in Meknes.

	Intervention	ASA	Cause of Cardiac Arrest	Heart Rhythm	Evolution
1	Volvulus sigmoïde	I	Unknown	QRS larges	Favorable
2	mass intrathoracique	I	tamponnade	Asystolia and extreme bradycardia	Death
3	Mediastinal tumour	I	Rhythm disorder And bleeding	Supraventricular tachycardia	Death
4	Lower limb ischemia	III	Myocardial infarction	Under ST lag	Death

Discussion

The occurrence of an ACC during the intraoperative phase is an exceptional but dramatic event. Its management is standardized by recommendations established by the learned anesthesia societies. The etiological diagnosis is often directed by the pre-existing terrain of the patient. However, the etiological investigation

must be methodical and systematically include the search for anaphylaxis by the dosage of specific histaminemia, tryptademia and IgE, including in a patient with a major cardiovascular risk [2].

Epidemiology and prognosis

The incidence of cardiac arrests in the operating theatre has decreased since the publication of the anesthesia safety decree

that made monitoring, including that of exhaled CO₂ and oxygen arterial saturation, mandatory for patients under anesthesia [3]. This incidence is estimated at 5.6 per 10,000 interventions. An intraoperative cardiac arrest can occur at any time of anesthesia and regardless of the technique used. In fact, 23% of circulatory arrests occur at the time of induction and 29% during the maintenance of anesthesia. In the case of locoregional anesthesia, the risk of cardiac arrest mainly concerns the installation phase of the anesthesia, with an incidence of 3/10,000 [1]. In patients with coronary artery disease, the incidence of perioperative myocardial infarction (IDM) is 4% for all surgeries combined and varies according to the type of surgery [4].

The risk factors reported are emergency anesthesia, extreme ages of life and a high ASA score. Obstetrics, radiological and diagnostic procedures are associated with an even lower risk. In our study the risk factors found are extreme age in one patient, surgical urgency in one patient, and high-risk surgery in two patients (neoplastic chest surgery).

Poor prognosis factors have been described in terms of survival, such as the starting heart rate (asystolia) and the terrain (congestive heart failure, kidney failure, septicaemia, respiratory failure, high age, major trauma, metabolic abnormality, my line hemorrhage or metastatic neoplasia) [1].

The prognosis for cardiac arrest in the perioperative period is less bleak than in extrahospital. The survival rate is 31.7% in in-hospital, while it is 3% in out-of-hospital. In our study survival was 25% (1/4).

Etiological orientation to perioperative cardiac arrest

No cause is identified precisely in 10-30% of cardiac arrests in the operating theatre. In a review of the literature published in 2005, the heart rhythms found on the electrocardiogram are bradycardia in 23% of cases, asystolia in 22% of cases, ventricular arrhythmia (FV or TV) in 14% of cases. ECG is normal in 7% of cases (33% undocumented) [1]. The most rediscovered etiologies are mentioned in Table 3.

Table 3: Etiologies Most Commonly Associated with Intraoperative Cardiac Arrest [1].

Respiratory Causes	Hypoxia (inhalation, failure of intubation, accidental extubation, pneumothorax, selective intubation, equipment breakdown, respiratory depression during locoregional anesthesia) Bronchospasm
Cardiovascular Causes	Hypovolemic and/or hemorrhagic shock Surgical manoeuvres associated with decreased organ infusion Cruoric, gaseous, greasy, cement pulmonary embolism Hydro-electrolytic imbalance (kaliemia) Increased intra-abdominal pressure Transfusion accident Anaphylactic shock Pneumothorax compressive Acute coronary syndrome (infarction, Takotsubo...) Severe pulmonary arterial hypertension Pacemaker malfunction Long Q-T syndrome Conduction Disorder (Full BAV) Hypothermia
Anesthetic Causes	Overdose of anesthetic agents Medication errors Neuraxial anesthesia with brain stem block Intoxication with local anesthetics Hyperthermia malignant Anaphylactic shock
Neurological Causes	Stroke Intracranial hypertension

Diagnosis, management and resuscitation of circulatory arrest

The diagnosis of cardiac arrest during anesthesia is quickly made on the basis of the monitoring installed for anesthesia. Notably by the cardioscope, the pulse oxymeter and the monitoring of the exhaled CO₂ [3]:

Support

a) Once the diagnosis of circulatory arrest has been confirmed, especially after checking the reliability of the monitoring, **resuscitation** should immediately begin with an external or internal cardiac massage (depending on the type of surgery) at a rate of 100/minute [5], trying to minimize the duration and frequency of interruptions.

b) The administration of all anesthetic agents must be interrupted during the massage, a purge of the respirator circuits is performed, FiO₂ is set to 100%.

c) The patient is of course intubated and ventilated if the procedure was performed under locoregional anesthesia. A reinforcement must be called simultaneously using the emergency procedures of the operating theatre.

d) It should be noted that in the case of ventral decubitus surgery, a cardiac massage in the ventral position should be performed immediately, although its effectiveness is inconsistent and the patient should be placed back in the back position as soon as possible. Further care is codified by SFAR's recommendations: Intravenous adrenaline remains

the vasopressor of choice, regardless of etiology. The intra-tracheal pathway is no longer recommended.

In cases of ventricular fibrillation or ventricular tachycardia without a pulse, an external electric shock of 200 joules (8-10 joules internally) must be performed (biphasic defibrillator) followed by a resumption of cardiac massage for 2 minutes before looking for the presence of a rhythm. Amiodarone, lidocaine and magnesium sulphate may be useful for defibrillation-resistant ventricular fibrillations. Infusion solutes must be isotonic.

Etiological diagnosis and specific resuscitation

The research of the etiology of circulatory cessation is done simultaneously because it can allow to offer a specific treatment essential.

Two approaches can be proposed:

a) The first, described by Moitra et al. [1], is based on the analysis of the starting heart rate. However, this approach is often difficult due to the low specificity of the initial rhythm and a poor analysis of the ECG (parasite) route.

b) A second approach is to conduct a series of audits based on the context and frequency of events: The first step is to look for an orientation according to the context: the surgical gesture (hypovolemia, blood loss, gas embolism...), the type of anesthesia (local anesthetic poisoning...), the terrain (e.g. coronary), the clinic (signs of anaphylaxis...) and finally the data of the monitoring (desaturation, hyperthermia...). In the absence of obvious guidance, the search for a ventilatory mechanism is the second step, especially in the intubated patient. Indeed, the most common causes are obstruction or displacement of the intubation tube, bronchospasm and pneumothorax. The slightest doubt about the proper functioning of the ventilation system leads to manual ventilation in pure oxygen.

c) The third step requires a quick check of the anesthesia tray to detect an administrative error;

d) The fourth stage is based on a cardiovascular assessment with analysis of the ECG for a rhythm disorder, a repolarization disorder or conduction. Some studies have analyzed the interest of the initial cardiac rhythm [1]. (Table 4)

Table 4: Heart Rhythm and Etiology of Cardiac Arrest [1].

Bradycardia or asystolia		Tachycardia or Ventricular Fibrillation
“H”	« T »	
Hypoxia Hypovolemia Hypokalemia Hyperkalemia Hydrogen (acidosis) Hypothermia Hypoglycemia Malignant Hyperthermia “Hypervagal” reaction	Toxicity (anaphylaxis, anesthesia) Pneumothorax compressive Thromboembolism (pulmonary, coronary) Tamponnade Trauma (haemorrhage) QT elongated	Coronary ischemia Dyskaliémie

Main etiologies of intraoperative cardiac arrests

1. Acute coronary syndrome and Ischemic heart disease

Ways to detect ischemic heart events are:

- a) ECG changes (T-waves, ST segment, arrhythmias)
- b) hemodynamic instability (hypotension, elevated occlusion pulmonary hair pressure);
- c) Segmental kinetic disorders visible on transesophageal ultrasound [5].

Most of the time, perioperative infarctions are manifested by sub-shifting of the ST segment, while an extrashift, as reported, is less common. The appearance of intraoperative ST sub-shift is frequently associated with the occurrence of a Q-uniclinked infarction, which evokes a phenomenon of silent ischemia that therefore requires continuous peropoop monitoring of the ST segment. In perioperative, DO1/VO2 imbalance is as common as unstable plaque thrombosis. Intracoronarian embolisms and prolonged coronary spasms are rarer causes. However, it has been shown in patients with prior coronary artery disease that perioperative infarction often occurs in a different territory than preoperatively suspected during coronary or stress echocardiography assessment [5,6].

Etiologies of an intraoperative Acute coronary syndrome [5].

Intraoperative ACS is due to an increase in O2 requirements, the causes of which are

- a) Severe aortic stenosis;
- b) Severe mitral insufficiency;
- c) High blood pressure;
- d) tachycardia
- e) Or a decrease in O2 intakes due to
- f) arteriosclerosis;
- g) low blood pressure;
- h) Anemia;
- i) hypoxemia.

Attitude in case of perioperative ACS

The goal of the treatment is to reduce Oxygen consumption and increase intakes with FiO2 to 100%; ensure noecocodemia; deepen anesthesia and ensure adequate analgesia and monitor hemoglobin levels and transfer if necessary, with a tolerance threshold of 100g/l;

- a) administer 500mg of acetylsalicylic acid (Aspirin®) if the patient is not already treated, and possibly 180mg of ticagrelor (to be evaluated according to the type of surgery and anesthetic strategy);
- b) beta-blockers in case of tachycardia - 60b/min, but only if ventricular function is normal or little impaired
- c) infusion nitroglycerin: 0.5-10g/kg/min (between 1-10mg/h), until pain in the case of ALR disappears;
- d) maintenance of coronary infusion pressure unless VG dysfunction: norepinephrine: 0.03-0.5g/kg/min.

In case of cardiogenic shock: catecholamines (dobutamine [Dobutrex®], norepinephrine).

Intra-aortic counterpulsation balloon if amines are ineffective.

The assessment should be completed as soon as possible with an ECG 12 bypass, a dosage of cardiac enzymes and an echocardiography; again, it is necessary to be able to recognize an SCA with sus-shifting ST that requires revascularization as soon as possible [5]. The 2004 ACC/AHA recommendations state that in the face of an IDM with a sus-shift of the ST segment (STEMI) complicated by cardiogenic shock, an emergency revascularization must be carried out as soon as possible, either by percutaneous angioplasty or by fibrinolysis [3]. However, in a perioperative context, fibrinolysis is naturally contraindicated due to hemorrhagic risk and only coronary angioplasty can be proposed, with hemorrhagic problems related to anti-stick anti-aggregators and anticoagulation [6].

2. Perioperative rhythm disorders

Rhythm disorders associated with anesthetic agents are among the well-known side effects in general anesthesia. Long QT (QTL) (according to Bazett's formula) is defined by: QTc 440ms (severe if 500ms), this parameter is a known risk factor for spike twisting, which can lead to ventricular fibrillation and circulatory arrest. Moreover, this phenomenon is all the more formidable because significant variations in QT have been demonstrated in the postoperative period [7] (Figure 2).

3. Intraoperative anaphylactic shock

The incidence of peraphylactic allergic or anaphylactic (AA) accidents varies from one in 3,500 to one in 13,000 studies. In children, the incidence is estimated at 1 in 7741 for all types of anesthesia combined. The main substances responsible are latex, curares, antibiotics and hypnotics [8]. In Morocco, no prospective studies have looked at this impact. The results obtained are most often derived from an estimate, the result of a simple evaluation based on retrospective studies. This incidence is in the order of 1 in 4970. Moreover, there are few systematic studies linking the dual epidemiological aspect (frequency, risk factors) and osotic etiol (responsible drug).

General anaphylaxis (GA) are severe accidents that can be fatal. Mortality is estimated at between 2% and 6%. All anesthetic

agents may be involved. In France, curares are the most blamed, with rates between 49.6 and 81.0% according to studies [9].

Curares are the leading cause of intraoperative anaphylaxis in countries such as France, Norway, Spain and Australasia [10]. Responsibility for other causes such as antibiotics, latex is called into question [11].

The symptomatology varies depending on the severity. In intraoperative, cardiovascular signs (hypotension, tachycardia, arrhythmia.) appear with or without skin signs. These can be masked by surgical draping. The presence of bronchospasm is less frequent and unspecific, especially during general anesthesia [11]. The clinical signs observed are the same for both types of reactions (IgE dependent or not). These signs are not always complete and the absence of skin-omu signs does not exclude diagnosis [9].

Independent risk factors for fatal anaphylaxis were male, cardiovascular disease, b-blockers at the time of reaction, and injection of the muscle relaxant in an emergency. The type of curare administered was not one of them, a controversial observation because other series show discordant results. In the case of death, the average time to experience shock after curare injection was 2.27 min (1-5) and the average dose of adrenaline administered was 15.34 mg (1-60) [10].

The diagnostic approach

The diagnostic assessment seeks to prove a dependent IgE reaction, the role of the suspect product and possible cross-awareness. The immediate investigation is based mainly on the dosage of serum tryptase, with three samples needed in the acute phase. A remote sample of the reaction is required to interpret the small increases. The dosage of plasma histamine levels is also part of the immediate balance sheet. Concordance with tryptase concentrations is generally good, but some patients have only an increase in histamine concentration and others only tryptase. Histamine appears more sensitive than tryptase in moderate reactions. The association of histamine and tryptase dosages increases diagnostic sensitivity [9].

The subsequent investigation is carried out as part of an allergeo-anaesthesia consultation. The balance includes the dosage of specific IgE with skin tests (prick-tests then IDR) and will concern curares, hypnotics and morphinics. If skin tests quantify the IgE attached to the skin mast cells, the IgE dosage evaluates serum free IgE. Specific IgEs concern quaternary antiammoniums in the case of curares [9]. **The management of anaphylactic shock** is well codified. It must be started early. Treatment consists of stopping the administration of any medication, the momentary cessation of the intervention, the administration of pure oxygen, vascular filling with crystalloids and the administration of vasopresand and corticosteroids [11] For vasopressors, adrenaline is the first-line drug. It is recommended to adapt adrenaline doses to the hemodynamic response by rereading a titration until the correct blood pressure is restored. Doses of 1 to 10 ug/kg can be relayed by a continuous infusion started at 0.1 mg/kg per minute.

Glucocorticoids are often given in the acute phase of anaphylactic shock, although their effects are delayed by several hours. After hemodynamic stabilization, a biological check-up should be done quickly involving a dosage of histamine and tryptase [12,13].

Prevention

Primary prevention consists of patients not being exposed to drugs that may induce immediate hypersensitivity in intraoperatively. This concept is difficult to achieve with general anesthetics [9].

Secondary prevention consists of not reexposing the patient to a product against which he is sensitized, hence the interest of the systematic allergological investigation after an immediate hypersensitivity reaction during an AG. It is noted here that premedication in this case has been ineffective. Better yet, locoregional anesthesia should be preferred and myorelaxation should be avoided when it is not justified (digestive endoscopy, ENT surgery, etc.) [9]. Paraesthetic allergic reactions are a reality in the Moroccan context, hence the interest in expanding this study to other services, and in raising awareness among anaesthetists of the practice of systematic blood samples during a reaction. hypersensitivity, and then refer patients to the allergo-anaesthesia consultation.

The latest SFAR/SFA recommendations “preventing allergic risk in anesthesia” will help to raise awareness among different specialists (anaesthetists, surgeons, allergists) and to harmonize procedures [9].

Epidemiology of curare allergies

Epidemiological data on allergic perioperative complications in France show that curares are the drugs that induce the greatest number of anaphylactic reactions. The curares most frequently involved were suxamethonium (68.2%) and atracurium (13.9%), followed by rocuronium (10.6%) [10-14].

Pathophysiological mechanisms of reactions to curares

Curares can induce genuinely allergic reactions (type I, IgE-mediated) or by nonspecific histaminolibration. The latter is referred to as a differential diagnosis before a negative allergological assessment [10].

Severity of allergic reactions to curares

Clinical symptomatology alone does not direct suspicion towards one molecule over another. Nevertheless, allergic reactions to curares are considered severe (anaphylactic), and rapid, typically occurring at the induction of anesthesia, within seconds or minutes after intravenous administration of muscle relaxants [10].

Hypersensitivity reactions of propofol

Diprivan is composed of propofol, refined soybean oil and purified egg phosphatide. The incidence is estimated at one in 60,000 cases and represents 1.2% of perioperative

hypersensitivity reactions in France. Several teams report cases of hypersensitivity to propofol, including one in a child allergic to peanuts. Peanuts and soybeans belonging to the same botanical family, legumes, are similar from phylogenetic and antigenic point of view [8]. Cases of propofol-induced bronchoconstriction in patients with a history of rhinitis printanière are also reported. When injecting Diprivan, a hypersensitivity reaction may be related to cross-reactivity [8]

Prevention of propofol allergy

Given the data currently available, it seems appropriate to avoid propofol in patients with an isolated peanut allergy or an asthma allergic to birch pollen, given the increased risk of cross-allergy with It's Diprivan. It seems desirable to propose either locoregional anesthesia when it is feasible or general anesthesia by favouring other hypnotics such as halide or etomidate. These patients may also be discussed with an allergo-anesthesia consultation to test for soy [8].

Hypersensitivity reactions (HS) to betalactamines (BL) were described shortly after the commercialization of penicillins and then with the other BL groups immediately put on the market. It is classically accepted that nearly 10% of subjects treated by BL report suspicious allergic reactions. These reactions account for 25-45% of all suspected allergic drug side effects, significantly outpacing all other drug classes. However, it is currently shown that more than 80% of suspected penicillin-allergic subjects are not actually allergic [15]. The frequency of patients with a clinical history of penicillin allergy varies from 1 to 10% depending on the studies. Penicillin G has long been considered the most fusing BL for allergic reactions. However, recent data suggest that amoxicillin (AX) is increasingly involved due to the reversal of the prescribing trend in its favor [15].

AA symptomatology in BL

Anaphylactic shock occurs in 0.2% of cases with a mortality of 0.02%. It is estimated, however, that the number of deaths secondary to an anaphylactic reaction to BL accounts for 75% of fatal drug shocks [15]

Several hypotheses may explain the presence of specific IgE several years after the last therapeutic exposure to penicillins:

- Natural persistence of penicillin-specific IgEs.
- Some patients have very strong immunological memory and the secretion of antibodies by plasma cells can last for years after antigenic stimulation;
- Occult exposure to food penicillins;
- Skin or mucous contact with penicillins;

Cross-reactivity with environmental allergens/molecules. Occult exposures to penicillin during ingestion of milk or meat have been reported to cause allergic skin manifestations and respiratory problems. It is also possible that the patient may

have come into contact with low doses of penicillin, such as those that can be exposed to when preparing an amoxicillin drinkable suspension for the treatment of infections in his children. Finally, cross-reactivity against infectious agents or environmental molecules is possible, as suggested in the case reported by Guillet et al. of a subject who developed hives and anaphylactic shock from food allergy to *Penicillium italicum* [16].

4. Differential diagnosis and assessment of the severity of anaphylactic shock

Cardiovascular manifestations of anaphylaxis and anaphylactic shock may be confused with several other pathologies with a similar clinical picture (Table 2) when skin or bronchopulmonary signs suggestive of anaphylaxis are absent [17].

Differential diagnosis of anaphylactic shock. [17].

- a) Heart events
- b) Anesthetic overdose
- c) Activation vagale (syndrome vasovagal)
- d) Cardiac arrest of other etiologies
- e) Syndrome carcinoïde
- f) Massive pulmonary embolism
- g) Respiratory protests
- h) Obstruction of upper airways
- i) Intrabronchic foreign body
- j) Bronchospasm
- k) Asthma malcondition
- l) Spontaneous Pneumothorax

Should the intervention be continued?

In the event of an effective cardiac activity, the decision to postpone or continue the surgical procedure should be discussed on a case-by-case basis. Several factors fuel the discussion: the indication of the intervention and its vital nature, whether it is started or not, the degree of urgency, the etiology and duration of cardiac arrest, and the hemodynamic stability after cardiac arrest. If the procedure is not started and it is not vital, it must be postponed. If it is in progress, it is completed or shortened if possible [1].

In our study the procedure was delayed for a patient who had a delayed and shortened emergency in a patient who had a chest mass compressing the heart.

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