Paro Intraoperatorio

Navarro-Vargas José Ricardo*

*Professor of Medicine, Universidad Nacional de Colombia, Member of the Committee of Resuscitation S.C.A.R.E, Colombia

Corresponding Author: Navarro-Vargas José Ricardo, Professor of Medicine, Universidad Nacional de Colombia, Member of the Committee of Resuscitation S.C.A.R.E, Colombia.

Received: October 01, 2019; Published: November 26, 2019

Intraoperative arrest differs from out-of-hospital unemployment because it is usually witnessed and frequently anticipated, and the main cause is not acute myocardial infarction, other causes such as drug overdose (21%), hypovolemia (15%), hypoxemia (14%), anaphylaxis (7%), vagal reflexes (18%) [1,2].

Human error is involved in up to 91% of all cases, mainly due to inadequate preoperative estimation of anesthetic and surgical risk, intraoperative errors (lack of attention, rush, lack of checklist, inexperience, distraction, fatigue), communication problems, difficulties in monitoring, wrong clinical judgment, etc [2,3].

At present, it is considered that general anesthesia can cause intraoperative arrest in up to 5.5/10,000 anesthesias; regional anesthesia 1.5/10,000 anesthesia; When monitoring is used appropriately, these causes can be reduced to 0.7/10,000 anesthesias [4]. In children younger than 1 month, the intraoperative unemployment rate can be as high as 54.2/10,000 anesthesia with a mortality of 43/10,000 anesthesias [4].

Rhythms of unemployment

The most frequent rates of unemployment are bradycardia 23%, asystole 22%, ventricular tachycardia/VF 14%, sinus/AESP 7%, not determined 33% [1]. The following relationship describes mortality in different studies [4-7].

<table>
<thead>
<tr>
<th>Author</th>
<th>Years of the study</th>
<th>Rate for anesthesia (10,000)</th>
<th>Mortality (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stephen</td>
<td>1950-1959</td>
<td>6.0</td>
<td>68%</td>
</tr>
<tr>
<td>Pottecher</td>
<td>1978-1982</td>
<td>6.0</td>
<td>56%</td>
</tr>
<tr>
<td>Keenan</td>
<td>1969-1984</td>
<td>1.7</td>
<td>52%</td>
</tr>
<tr>
<td>Keenan</td>
<td>1969-1988</td>
<td>1.5</td>
<td>48%</td>
</tr>
<tr>
<td>Biboulet</td>
<td>1989-1995</td>
<td>1.08</td>
<td>54%</td>
</tr>
<tr>
<td>NACOR</td>
<td>2010-2013</td>
<td>5.6</td>
<td>58.4%</td>
</tr>
<tr>
<td>Ellis</td>
<td>1999-2009</td>
<td>0.6</td>
<td>29%</td>
</tr>
</tbody>
</table>

Table 1

Studies carried out before 1970 record an incidence of cardiac arrest of anesthetic cause of 4 - 6 per 10,000 anesthetic-surgical procedures, while in more recent studies there is a markedly reduced rate of 0.5 - 1.5 unemployment per 10,000 anesthesia [7,8]. It is associated with hypoxemia and adverse events due to medication administration. The most important predictive factors are intraoperative blood transfusion volume, high ASA rating (grades 4.5), emergency surgery and high-risk surgical procedures (such as cardiovascular surgery) [9].

Surveillance of the ventilatory status of the patient and the checklist have been fundamental in reducing the incidence of anesthesia-related strikes in recent years, however, mortality after an intraoperative arrest remains high, up to 46% - 48% [4].

The POCA (Pediatric Perioperative Cardiac Arrest) study, which began in 1994, reported an incidence of perioperative cardiac arrest of 1.4/10,000 anesthesias and a mortality of 26%. Among the causes were those related to administration (overdose) of medications (37%), cardiovascular depression caused by the inhalation agent Halotano and toxicity by local anesthetics after a caudal block.

Cardiovascular causes (arrhythmias, massive transfusions, hydroelectrolytic alterations) were more frequent than those related to respiratory depression (32% vs. 20%).

Children under 1 year were the most affected, representing 55% of all cases, and of this group, children under 5 months presented 43% of transoperative stoppages [10].

Management of intraoperative arrest
The handling of these episodes should be done as follows:

1. Early measures to manage the crisis and prevent unemployment:
   a) Recognize hemodynamic instability conditions quickly
   b) Ask for help Request the cardio-defibrillator
   c) Stop surgery and anesthesia if necessary
   d) Administer 100% oxygen
   e) Perform the DONE (check if the tracheal tube is displaced, obstructed, if there is pneumothorax or if there is a failure in the respiratory equipment or circuit [11].
   f) Evaluate capnography and its tendency with hemodynamic status.

2. Establish a differential diagnosis:
   a) Evaluate the surgical procedure and establish effective communication with the surgical team.
   b) Review recently administered medications
   c) Perform chest Rx (with portable equipment) to rule out tension pneumothorax, if there is a sudden increase in airway resistance and hemodynamic instability.
   d) Perform echocardiogram (transesophageal echocardiography if the patient is intubated and is being operated on the chest) in order to determine ventricular filling, ventricular function, and valvular function and rule out cardiac tamponade with FAST.
   e) Consider empirical steroid therapy (dexamethasone 50 - 100 mg/E.V.).

3. If the patient is in cardiac arrest: BLS (basic life support) must be performed. If there is no pulse and the help has already been requested, the CAB starts: compressions/ventilations at a rate of 30 compressions for 2 ventilations for 5 cycles; if intubated, 100 compressions/minute and 10 ventilations/minute.

   If the strike is due to a defibrillable rhythm, defibrillation must be performed early. Subsequently, the ACLS (advanced vital cardiac support) continues: airway assurance, ventilation at a rate of 10 ventilations/minute - with a tidal volume of 7 ml/kg - and an I: E ratio (inspiration: expiration) of 1: 1.

   Epinephrine 1 mg E.V. bolus every 3 - 5 minutes and compressions/ventilations are continued. The differential diagnosis is decisive in the prognosis, which in transoperative cardiac arrest has 8 H and 8T [1].

Paro intraoperatorio

<table>
<thead>
<tr>
<th>Toxic</th>
<th>Hypoxia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypovolemia</td>
<td>Pneumothorax Tension</td>
</tr>
<tr>
<td>Hyper/hypokalemia</td>
<td>Thrombosis (venous embolism, PET)</td>
</tr>
<tr>
<td>Ion hydrogenation (acidosis)</td>
<td>Coronary thrombosis (AMI)</td>
</tr>
<tr>
<td>Hypothermia</td>
<td>Cardiac tamponade</td>
</tr>
<tr>
<td>Trauma</td>
<td>Hypoglycemia (hemorrhagic shock, Vascular injury)</td>
</tr>
<tr>
<td>Prolonged qT</td>
<td>Hyperthermia</td>
</tr>
<tr>
<td>Vagal hyperstimulation</td>
<td>Pulmonary hypertension</td>
</tr>
</tbody>
</table>

**Table 2: The 8 H and 8 T of the Intraoperative STOP.**

In the event of particular events, in addition to the ACLS [1,11], the indicated handling must be performed.

**In Anaphylaxis:** Epinephrine 0.5 - 3 ug/kg in bolus and start infusion at a rate of 5 - 15 ug/minute to maintain a systolic blood pressure above 90 mmHg; it is recommended to be aware of the cardiovisoscope to rule out myocardial ischemia.

**Toxicity by local anesthetics:** Administer 20% intralipid, I.V. 1.5 mL/Kg in bolus and continue with an infusion at a rate of 0.25 mL/Kg/hour. Cardiac arrest secondary to toxicity by local anesthetics requires prolonged resuscitation, usually 1 hour.

**Malignant hyperthermia:** Dantrolene sodium (you have to know how to prepare because this procedure requires a management protocol that takes time). A bolus of 2.5 mg/kg should be administered and if the signs of hyperthermia with hypercapnia persist, the doses should be repeated up to a dose of 10 mg/kg. The administration of calcium channel blocking agents should be avoided and if any type of arrhythmia persists, hyperkalemia and acidosis should be corrected. The administration of Dantrolene should be continued at a rate of 1 mg/kg every 6 hours or 0.25 mg/kg/hour. There is no demonstrated risk when administering Dantrolene to the sole suspicion of malignant hyperthermia, since this pharmacological agent does not produce major cardiovascular or pulmonary alterations.

**Left ventricular failure:** Left ventricular failure should be managed by decreasing afterload and sustaining cardiac output with inotropic agents. Mechanical assistance devices can be useful however, they are not always available.

**Right ventricular failure:** Right ventricular failure is managed with vasodilators of pulmonary circulation and positive inotropic agents. Unlike the management of the left failure, which requires a decrease in afterload, here vasoconstrictor agents are recommended to improve systemic perfusion and cardiac output.

According to the Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care, 2015, developed by the American Heart Association (AHA) make the consideration that extracorporeal membrane oxygenation (ECMO) is a measure contemplated in patients in whom there is suspicion of a cause reversible, with the possibility of recovery using cardiopulmonary mechanical support. It is recommended to start the ECMO within the first 30 minutes of the start of resuscitation [12].

In conclusion, cardiac arrest in the transoperative period is not frequent, it is largely due to human error; it is witnessed, with a better prognosis, because if a rapid evaluation and expedited management is done, adapting the ACLS maneuvers to the specific conditions that originated it, the patient’s recovery and survival can be complete with minimal neurological sequelae.

**Bibliography**


**Citation:** Navarro-Vargas José Ricardo. “Paro Intraoperatorio”. *EC Anaesthesia* 5.12 (2019): 19-22.


**Volume 5 Issue 12 December 2019**
©All rights reserved by Navarro-Vargas José Ricardo.