1. Preoperative Preparation. Evaluation of Cardiac Patient

Once the surgical indication is established, the anesthesiologist’s role is to assess the cardiac patient for appropriate anesthetic management.

In pre-anesthetic assessment of the cardiac patient for cardiac surgery intervention, we must consider issues related to the surgical technique and the physiological impact of extracorporeal circulation and elective heart stop:

- Previous surgery to chest, heart, great blood vessels or lungs can influence (by adherence and / or changes in anatomy) intraoperative surgical management.
- History of peripheral vascular disease (atherosclerotic vascular disease) should be considered. Significant carotid artery stenosis is an indication of carotid thrombendarterectomy which may be performed before or simultaneously with cardiac surgery.
- Coagulopathy- patients with a history of heparin-induced thrombocytopenia develop thrombotic disorders important for the administration of heparin.
- Chronic renal failure requires special kidney protection measures during ECC and postoperatively.
- Post-ECC respiratory failure can be important; therefore, patients with lung disorders will benefit before surgery from therapy, antibiotics, bronchodilators, corticosteroids and kinesiology.

The cardiac assessment of the patient determines the characteristics of cardiovascular anatomy and physiology and the functional resources of the heart.

1. Radionuclide imaging investigations show areas at risk of myocardial ischemia.
2. Radionuclide ventriculografia determines telesistolic volumes (VTSVS) and telediastolic volumes (VTDVS) of the left ventricle, ejection fraction (EF).
3. Echocardiography provides data on ventricle contractility and valve kinetics. The hypo / dis/akinezia of areas in the ventricular wall indicates ischemia or infarcted areas.
4. Cardiac catheterization provides anatomical and functional data that can not be obtained through non-invasive investigations.
   - anatomical data - The coronary angiography shows the location and extension of coronary stenosis, distal loading, collateral flow and the dominant coronary. Significant stenosis means the reduction in arterial diameter of over 70%. The posterior descending coronary artery starts from the dominant coronary.
   - functional data - Ventriculography may show contractility deficiency, mitral regurgitation or intracardiac shunts. Left ventricular ejection fraction is normally higher than 0.6.
   - hemodynamic data are obtained both from right heart catheterization and the left heart. Pressure values reflect the volemic status, the functionality of heart valves and presence of pulmonary hypertension. Increased left ventricular telediastolic pressure (LVTDP) may be due to expansion and ventricular failure, volume overload (mitral or aortic regurgitation), decreased ventricular
compliance (due to ischemia or hypertrophy) or a constrictive phenomenon (cardiac tamponade). LVTDP may significantly increase in coronary patients following dye injection for ventriculography or coronaryography.

- normal values of intracardiac pressure and O2 blood saturation

<table>
<thead>
<tr>
<th>Structure</th>
<th>Pressure (mmHg)</th>
<th>Saturation in O2 (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Superior vena cava</td>
<td>-</td>
<td>71</td>
</tr>
<tr>
<td>Inferior vena cava</td>
<td>-</td>
<td>77</td>
</tr>
<tr>
<td>Right atrium</td>
<td>1-8</td>
<td>75</td>
</tr>
<tr>
<td>Right ventricle (systolic / diastolic)</td>
<td>15-30/0-8</td>
<td>75</td>
</tr>
<tr>
<td>Pulmonary artery (systolic / diastolic)</td>
<td>15-30/4-12</td>
<td>75</td>
</tr>
<tr>
<td>PAOP (systolic / diastolic)</td>
<td>2-12</td>
<td>-</td>
</tr>
<tr>
<td>Left atrium</td>
<td>2-12</td>
<td>98</td>
</tr>
<tr>
<td>Left ventricle (systolic / diastolic / telediastolic)</td>
<td>100-140/0-8/2-12</td>
<td>98</td>
</tr>
<tr>
<td>Aorta (systolic / diastolic)</td>
<td>100-140/60-90</td>
<td>98</td>
</tr>
</tbody>
</table>

- Left-right intracardiac shunts are proven by the increase of O2 saturation in arterial blood (SaO2) in the right heart cavities.
- The cardiac output is determined by thermodilution and hemodynamic indices are determined by the following formulas.

<table>
<thead>
<tr>
<th>Formula</th>
<th>Units</th>
<th>Normal Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Volume-beat SV=(CO/HR) *1000</td>
<td>ml / beat</td>
<td>60-90</td>
</tr>
<tr>
<td>Index Volume-beat SI=SV/BSA</td>
<td>ml/bataie/m²</td>
<td>40-60</td>
</tr>
<tr>
<td>LVSWI = [1.36(MAP-PAOP)/100]*SI</td>
<td>g*m/m²/beat</td>
<td>45-60</td>
</tr>
<tr>
<td>RVSWI = [1.36(PAP-CVP)/100]*SI</td>
<td>g*m/m²/beat</td>
<td>5-10</td>
</tr>
<tr>
<td>SVR= (MAP-CVP)/CO*80</td>
<td>dyne*sec/cm5</td>
<td>900-1500</td>
</tr>
<tr>
<td>PVR= (PAP-PAOP)/CO*80</td>
<td>dyne*sec/cm5</td>
<td>50-150</td>
</tr>
</tbody>
</table>

CO = cardiac output; HR = heart rate, BSA = body surface area, MAP = mean arterial pressure, PAOP = pulmonary artery occlusion pressure, PAP = pulmonary artery mean pressure, CVP = central venous pressure, SVR = systemic vascular resistance; PVR = pulmonary vascular resistance; LVSWI, RVSWI

- Routine laboratory tests for patients scheduled for cardiac surgery include: hemoleucogram, prothrombin time (PT), APTT, ionogram, urea, creatinine, blood glucose, AST, ALT, LDH, CK, brief examination of urine, chest X-ray (for heart size and pulmonary vasculature) and 12 Lead ECG (for analysis of pace, frequency, axis, QRS complex, ST segment elevation, signs of malignant arrhythmia, ischemia, infarction and ventricular hypertrophy).
2. Intraoperative Monitoring

Anesthesia in cardiac surgery requires knowledge of Physiology and Pathophysiology of cardiovascular system, pharmacology notions of anesthetic, vasoactive and inotropic drugs, and associated notions of specific surgical procedures and extracorporeal circulation.

Monitoring has an important role in checking and control of vital functions during anesthesia, surgery and immediate postoperative period.

In cardiac surgery and great blood vessels, the two derivations ECG are standardly monitored, blood pressure using Korotkoff method (sleeve) and bleeding, central venous pressure (CVP), the central and peripheral temperature, oxygen saturation in peripheral blood / peripheral pletismography, carbon dioxide in expired air (EtCO2) and optionally artery pressure / pulmonary capillary (with pulmonary artery catheter), the pressure in the left atrium. (?)

- **ECG.** ECG is used to monitor the pace, heart rate, detection and diagnosis of arrhythmias, pacemaker functionality and myocardial ischemia detection. This route does not guarantee the existence of ECG cardiac mechanical activity. We use the simultaneous and continuous monitoring of derivatives D II and V5, and ST segment analysis.

- **Temperature monitoring.** The "central" temperature measurement is used (the sensor is placed in the nasopharynx and reflects the brain temperature and other central infused internal organs); direct measurement of blood temperature (by means of the pulmonary artery catheter) and "peripheral" temperature (the sensor is positioned intrarectally or into the bladder and measures the temperature of less infused, peripheral tissues).

- **BP monitoring.** Normally, by self-regulation most organs have the capacity to maintain a relatively constant blood flow to relatively large variations of infusion pressure. BP measuring monitors infusion of blood infusion. We use non-invasive measurement (tonometer with sleeve) and invasive measurement (bleeding blood pressure), a more rigorous approach that provides for frequent harvesting of arterial blood samples. Bleeding BP is measured directly by placing a catheter in the radial, brachial or femoral artery, a catheter connected to an external pressure transducer. Pressure is converted into electrical signal and then displayed on a screen.

- **CVP monitoring.** To monitor central venous pressure (CVP), catheterization of a central vein is required (internal jugular vein or subclavian vein). Central venous pressure measurement is obtained by coupling the catheter lumen whose tip is in the superior vena cava to an external pressure transducer. The obtained CVP curve has 3 positive deflections (a, c and v) corresponding to atrial contraction, alteration in systolic heart and filling the right atrium. The CVP normal value is 2.6 mmHg.

- **Pulse oximetry.** Continuous measurement of peripheral oxygen saturation of hemoglobin is the standard method of monitoring oxygenation during anesthesia.
and immediate postoperative period. Pulse oximetry uses pletismography and spectrophotometric analysis (light-emitting diodes emit light waves on two wavelengths, light waves passing through the arterial bed represented by finger or ear lobe). There are several factors limiting the accuracy or pulse oximeter ability to measure oxygen saturation in hemoglobin: the absence of capillary pulse (hypothermia, hypotension, use of vasoactive drugs), movement (awake patient, shivering), falsely-elevated results (increased carboxyhemoglobin or methemoglobin) or falsely-low (nail polish, administration of methylene blue or intravenous indocyanina green).

- **Capnometry and capnography.** By this method we measure the concentration of carbon dioxide in expired air. Capnography is continuous monitoring of the capnogram. It is used to confirm orotracheal intubation for ventilation assessment and detection of pathological conditions (malignant hyperthermia, pulmonary thromboembolism, fat embolism, absorbing CO2 from the peritoneal cavity during laparoscopic interventions), the calculation of cardiac output.

- **Pulmonary artery catheter (Swann-Ganz).** It is mounted in the pulmonary artery, passing consecutively through the subcalvian vein / internal jugular-superior vena cava-right atrium-right ventricle, and provides data on central venous pressure (CVP), pulmonary artery pressure (PAP), pulmonary artery occlusion pressure (PAOP), oxygen saturation in central venous blood and cardiac output. Like any invasive pressure, CVP, PAP and PAOP are measured by coupling lumen Swann-Ganz catheter to an external pressure transducer. PAP normal value is 15-25 mmHg in systole and 5.12 mmHg in diastole, and PAOP normal value is 5.12 mmHg. Mounting pulmonary artery catheter is indicated in patients with compromised ventricular function (EF <40%), in patients with pulmonary hypertension and in complex surgery (lung thrombendarterectomy, heart transplant).

- **Transesophagian echocardiography (TEE).** It is used in preoperative evaluation of valvulopathies for surgical correction (plasty or replacement); for preoperative evaluation of intracardiac thrombus, intracardiac shunts, or aortic atheromatous plaques; for preoperative evaluation of dissection of the aorta, to assess perioperative myocardial ischemia, the segmentary motility of ventricles, to evaluate intracardiac air bubbles before separation from ECC; for postoperative evaluation of plasty/valvular replacement or intracardiac shunt closing.

3. Anesthesia in Cardiac Surgery

Anesthesia represents all pharmacological and technical resources that allow the patient to endure the surgical act without feeling its negative effects (pain, fear, anxiety). It also lays the
foundation for conducting surgery in good conditions: hypnosis, analgesia, muscle relaxation and homeostasis maintenance.

General anesthesia includes the following steps: preanesthesia, induction, maintenance, awakening from anesthesia.

- **Preanesthesia.** Preanestezia includes preanesthetic examination, risk assessment anesthetic premedication.
- **Premedication.** Patient education (explaining the procedures pre-, intra-and postoperative) is a good anxiolytic.
- **Cardiac specific medication.** Beta-blockers, calcium channel blockers and nitrates (including nitroglycerin iv) are administered under the scheme of treatment to patient arrival in the operating room. Digital preparations usually stop 24 hours before surgery because of potential toxicity (especially in the presence of hypokalaemia) and long half-life. Administration of antihypertensives, including converting enzyme inhibitors (CEI) and diuretics, stop in the morning of the surgery. Antiarrhythmics continue until the time of surgery. Amiodarone has a half-life of 30 days, so its interruption a few days before surgery does not significantly affect serum concentration and is not indicated.
- **Platelet antiaggregants-** (aspirin, ticlopidine, clopidogrel) is interrupted 7-10 days before surgery. Oral anticoagulants (dicumarinic preparations) are interrupted 2-3 days before surgery. On the day of surgery the prothrombin time (PT) and INR are checked. Continuous intravenous infusion of unfractionated heparin in patients with unstable angina or obstruction of left coronary artery trunk continues with preoperative routine.
- **Sedation and analgesia.** It uses a combination of benzodiazepines and morphine to ensure amnesia, anxiolysis and a good analgesia for insertion of catheters, and the degree of cardiorespiratory depression is acceptable. 5mg midazolam i.m. and morphine 0,10-0,15 mg / kg i.m are recommended.

**Preinduction.** On patient arrival in the operating room, electrodes for ECG, the pulse oximeter and the tonometer cuff are mounted. A peripheral venous line, 14-16G thick, is placed. When it is necessary, premedication is supplemented with IV midazolam 1-2mg i.v. Address pressure is the practice of first intent radial artery. The following options are femoral, brachial or axillary artery. In the operating room the following are ready: defibrillator, external pacemaker and 2-4 units of whole blood or isogroup concentrated red blood cells, izoRh. The following drugs are also prepared: heparin, calcium chloride / calcium gluconate, xilina 10mg/ml, inotropic (adrenaline in different dilutions 0.1 mg / ml and 0.01mg/ml, atropine 0.1 mg / ml), vasopressors (ephedrine 10mg/ml, phenylephrine 50µg/ml), nitroglycerin.

**Induction.** It consists of anesthesia setting by the administration of (inhaled and / or intravenous) anesthetic drugs.

The cardiac patient induction is one of the critical moments in anesthesia. At induction time, a surgeon must be available and the ECC pump prepred if the patient's hemodynamic status deteriorates. Choice of anesthetic drugs dependents on specific cardiac lesions, the general condition of the patient and type of surgery. Drugs used in induction are:
- Opioids i.v. Fentanyl is used in doses of up to 50-100µg/kgc, as a single agent both in induction and as maintenance agent. We are currently using lower doses (10-25µg/kgc) in combination with other anesthetic drugs, this type of anesthesia being known as "balanced technique".
- Hypnotic and amnesic sedatives. Thiopental, propofol (1.5-2mg / kgc), and etomidate (0.2-0.3 mg / kgc) are used as coinduction agents in particular cases.
- Volatile inhaled anesthetics are especially useful in hypertensive patients.
- Muscle relaxers. We use nondepolarizing relaxers: pancuron (0,08-0,1 mg / kgc) or vecuron (0,08-0,15 mg / kgc), which have minimal cardiovascular effects.

**Preoxygenation**

- After administration of these drugs, trachea is intubated with orotracheal intubation probe with internal diameter ballon of 7-7.5 mm for women and 8-8.5 mm for men. Parameters of the fan of anesthesia apparatus are established: 10ml/kgc VT = tidal volume, respiratory FR = 10-14 resp / min, maximum inspiratory pressure Pi max = 25mmHg.

- Central venous catheter is placed on subclavian vein or internal jugular vein. If the patient is unstable / haemodynamically fragile, the central venous catheter is placed before induction / trachea intubation in order to administer vasoconstrictor medication if necessary.
- An arterial blood sample is analyzed to measure partial pressure of blood gases (O2, CO2), pH, hematocrit, the degree of inhibition of rennet formation is checked (ACT = activated clotting time, normal levels :80-150S )

3.1. Specific considerations of anesthetic induction according to valvulopathy.

From a pathophysiological point of view, valvulopathies are characterized by volume loading or atrium or ventricle pressure.

- **Aortic stenosis.** Normal aortic valve consists of 3 semilunar cups located at the base of the aorta. The normal diameter of the valve ring is 1.9 - 2.3 cm with aortic orifice area of 2 - 4cm². Aortic stenosis has usually rheumatic etiology. Aortic stenosis stages are: stage I, mild aortic stenosis, asymptomatic, with valvular orifice area between 1 - 2cm², stage II, moderate aortic stenosis, symptomatic, the area between 0.7-0.9 cm² and stage III, critical aortic stenosis with aortic orifice area less than 0.7 cm². Symptoms appear late in the course of the disease. Without surgery, life expectancy is 5 years from angina appearance and 2 years of onset of heart failure. Chronic obstruction of left ventricular ejection results in concentric hypertrophy of ventricular wall. Atrium contraction is critical for ventricular filling and beat volume. Heart is susceptible to ischemia (even in the absence of coronary disease) due to increased intraventricular pressure and hypertrophied myocardium by coronary infusion of low pressure. During anesthesia induction, it is necessary to maintain an adequate intravascular volume, the sinus rhythm, the inotropismului and systemic vascular tone. Avoid using anesthetic drugs that lower inotropism or produce systemic vasodilation (eg thiopental). Hypotension, tachycardia (decreases ventricular filling and increases oxygen
consumption), severe bradycardia (decreases cardiac output) and arrhythmias are poorly tolerated and should be treated aggressively to maintain pressure of coronary infusion.

- **aortic regurgitation** is the etiology of acute articular rheumatism, endocarditis, collagenosis, diseases which dilate the aortic ring (e.g. Marfan syndrome, aneurysm of ascending aorta, syphilis). Chronic aortic regurgitation has 3 stages: stage I, asymptomatic minimal regurgitation, stage II, moderate aortic insufficiency with regurgitation fraction of 60% of the beating, stage III, severe aortic insufficiency with significant ventricular dysfunction. Chronic regurgitation results in eccentric ventricular hypertrophy with minimal change in filling pressure. Symptoms may be minimal until late in the course of the disease, when left heart failure occurs. Acute aortic regurgitation may cause sudden volume filling of left ventricle with increasing telediastolic pressure (LVTDP) and occlusion pressure of pulmonary capillary (PAOP). Acute clinical regurgitation is manifested by low cardiac output syndrome, heart failure, tachycardia and vasoconstriction. In anesthetic induction, volemia must be maintained haemodynamically, an increased cardiac rate 80-100b/min (to shorten diastolic regurgitation time), inotropism and a degree of peripheral vasodilation (to decrease postload).

- **mitral stenosis** is in most cases of rheumatic etiology. Normal mitral valve area is 4-6cm². There are 3 stages in the evolution of mitral stenosis: stage I, mild mitral stenosis, valve area is 1.5-2.5 cm², symptoms occur at moderate effort, stage II, moderate mitral stenosis with valve area of 1-1.5 cm², symptoms occur in small efforts, stage III, tight mitral stenosis with valve area under 1cm² and symptoms of rest. Symptoms appear late in the course of the disease (20 years after the rheumatic episode) and consists in fatigue, palpitations, effort pulmonary edema, nocturnal paroxysmal dyspnea, chest pain. Increasing pressure and volume loading of the left atrium leads to increased atrium volume and atrial fibrillation setting (which increases the risk of thrombus formation in left atrium and the risk of embolic stroke). Increased pressure in the left atrium leads to increased pressure in the pulmonary veins and increased pulmonary vascular resistance. Thus, pulmonary hypertension can lead to tricuspidian regurgitation and right ventricle failure. During anesthesia induction we must maintain: an adequate intravascular volume, low heart rate (and a sinus rhythm if possible), inotropism and systemic vascular resistance. Worsening pulmonary hypertension by hypoventilation or ventilation with PEEP (positive pressure at the end of expire) should be avoided.

- **mitral regurgitation** has in most cases rheumatic etiology and is associated with mitral stenosis. Volume overloading of left atrium is the main pathophysiological mechanism in mitral regurgitation. Chronic mitral insufficiency has 3 stages: stage I, asymptomatic mild mitral insufficiency, stage II, mitral insufficiency with the appearance of symptoms, and stage III, severe mitral insufficiency, with higher regurgitation fraction, which leads to pulmonary hypertension. Acute mitral insufficiency has nonrheumatic etiology (by ischemia and rupture of papillary muscles, bacterial endocarditis) and causes rapid deterioration of cardiac function with sudden volume overload of left atrium and acute pulmonary edema. The echographic determination of ejection fraction is not relevant in the case of mitral regurgitation due to systolic blood ejection both antegrade (to aorta) and retrograde (by valve
failure in the left atrium). In anesthesia induction, we must maintain volemia, inotropism, normal or elevated heart rate (bradycardia increases left ventricular diastolic filling and implicitly increases regurgitation fraction) and low peripheral vascular resistance (which leads to decreased regurgitation fraction and increased cardiac output).

• In patients with mixed valvular lesions, the most haemodynamically significant valvulopathy will determine anesthetic conduct.

3.2. Specific considerations in anesthesia induction in surgical cardiovascular emergencies

• **Cardiac tamponade.** The patient with cardiac tamponade is a hemodynamically unstable patient, tachycardia, increased CVP, hypotensive, with signs of low cardiac output, oliguria. Induction of anesthesia and positive pressure ventilation may precipitate cardiovascular collapse, so the patient must be isolated before induction (for the thorax to be opened as soon as possible and the fluid/blood from the pericardium discharged); ketamine is used as inducing agent in doses of 1 -1.5 mg/kg (it maintains blood pressure through vasoconstriction). It is also necessary volemic support and administration of an inotrope and/or a vasoconstrictor.

• **Dissection of the aorta.** Dissection of the aorta in patients with hypertension control and ejection speed (by continuous infusion administration of nitroglycerin and beta-blocker) is required, because they are factors of propagation of dissection. Before induction, 8 units of isogroup blood, izoRh, must be prepared. Induction of anesthesia should be mild, with minimal hemodynamic disorders.

• Ventricular septal defect and papillary muscle rupture of acute postinfarct myocardial infarction. These patients show extreme hypotension (due to left-right shunt); large vessels cannulation and faster extracorporeal circulation are essential.

3.3. Maintaining anesthesia during surgery

• Pre-ECC stage/ECC incision is characterized by periods of surgical stimulation (skin incision, sternotomy, pericardial incision) that require deeper anesthesia.

• Preparing for the ECC includes heparinisation (iv300U heparin/kg is administered) for an ACT of more than 400S and large vessels cannulation (aorta, artery and upper and lower cave veins or right atrium). Heparinisation is required before the start of the ECC to prevent disseminated intravascular coagulation and clotting in the reservoir of ECC pump.

• When arterial and venous cannulae are positioned and secured, ACT has the desired value (> 400S) and cardiopulmonary bypass can start (extracorporeal circulation). ECC diverts the
vevous system blood flow from heart blood in the venous cannula, establishes gas exchanges (oxygenates blood and removes CO2), and redirects blood flow to a pressure vessel (usually the aorta artery). Thus, almost all blood flow through the heart and pulmonary circulation ceases. During ERC, mean systemic blood pressure is carefully monitored and maintained at values of TAm 50-60mmHg, blood flow is gradually increased to 2-2.5 l / m² sc (50-60ml/kgc/min). It is also monitored the amount of blood in the pump reservoir, the pressure in the arterial line (if the pressure is low, this may be due to inadequate venous return, dissection of the aorta or vasoplegia; if the pressure is high, it may be due to excessive flow of ECC plump or vasoconstriction), temperature, Astrup parameters (pH, pO2, pCO2), ACT, hematocrit (which is maintained between 20-25%), serum electrolytes (sodium, potassium), diuresis.

- myocardial protection during aorta clamping is achieved by reducing the consumption of O2 by hypothermia and / or by stopping the heart from hyperkalaemia (by administering cardioplegia solution). Moderate hypothermia (26-32 °C) and deep hypothermia (20-25 °C) is frequently used during ERC. Consumption (and hence the need) of O2 is reduced and blood viscosity is increased (reversed effect of haemodilution by priming solution). Metabolic acidosis and oliguria suggest inadequate systemic infusion and require the addition of f fluid (crystalloid or colloid). Oliguria (<1ml/kgc/h) is treated with volume, mannitol (0.25-0.5 mg / per kg of bodyweight), dopamine (1-5µg/kgc/h) or furosemide (10-20mg). Hemolysis during CEC is due to "trauma" suffered by the red blood cells through the circuits of ECC pump. Haemoglobinuria may cause acute renal failure and should be prevented by alkalinization of urine (sodium bicarbonate 0.5-1mEq / kgc should be administered).

- Separation from the ECC.

- Before ECC separation, laboratory values must be checked: hematocrit, arterial blood gases, potasemia (which may be increased due to cardioplegia solution); ventilation must be turned on, the patient must reach normothermia, ECG route and filling pressures must be evaluated, size, pace and heart contractility are estimated. If operations were performed involving open heart cavities (valve replacement, closure of interatrial or interventricular septal defects), de-aeration procedures are performed. Aorta declamping restores coronary perfusion. Ventricular fibrillation is treated with internal electric shock 20-30J. In order to restore pace, we need xiline administration of 1-1.5 mg / kgc, slow magnesium sulphate 1g i.v., amiodarone 150 mg bolus. If the heart starts in a slow pace (atrioventricular block gr.III, jonctional rate, etc.), atrial and / or ventricular internal pacing is required. Antiarrhythmic medication is used as beta-blockers iv (esmolol, metoprolol), calcium blockers (verapamil), amiodarone. The ECG route is examined in search of possible signs of ischemia. Visual examination of the heart in the operating field provides information about atrioventricular a/synchrony, contractility, the degree of filling of the ventricles. If myocardial dysfunction is anticipated or demonstrated (due to preoperative low performance or intraoperative ischemia), early administration of continuous inotropic infusion is indicated before separation from ECC.

- effective ECC separation is performed by slow clamping of venous line, enabling the heart to fill gradually and eject in each contraction. Partial venous occlusion allows a "partial
bypass" during which cardiopulmonary function is divided by the heart-lungs and ECC pump, which allows assessment of haemodynamics. After complete venous clamping, once adequate filling pressures are obtained, blood flow through arterial cannula is stopped, the bypass heart – lungs is interrupted and the heart takes over all systemic infusion.

• After ECC separation, we must examine: ECG, systemic BP, filling pressures of right ventricle, left ventricle and cardiac output (CO). If any of these parameters is not considered appropriate, the administration of inotropic, vasodilators is adjusted by internal pacing, to evaluate graft patents, plasia or replacement of valves by transesophagian echocardiography.

Right or left ventricular dysfunction after ECC may have the following etiologies:

• Ischemia:
  - Inadequate myocardial protection
  - Intraoperative infarction
  - Reperfusion lesions
  - Coronary spasm
  - Coronary embolism (air, thrombi)
  - Difficulties of surgical technique (sutured grafts or thrombosis)

• Uncorrected structural defects:
  - Ungraftable vessels
  - Diffuse coronary ateromatosis
  - Residual or newly occured valvulopathy
  - Shunts
  - Previous cardiovascular dysfunction

• ECC-related factors:
  - Excessive cardioplegia
  - Unrecognized cardiac distension

The post-ECC:

• During the immediate post-ECC, hemodynamic stability must be maintained by keeping volemia, infusion pressure, pace and adequate rate. At the same time, the operating field must be continuously monitored and reevaluated.

• When the patient is stable and the surgeon controls hemodynamic hemostasis, protamine is administrated to antagonize residual circulating heparin. 1mg of protamine is administered for every 1mg (100U) of heparin at pump entrance. Initially 20-30mg protamine are administrated within 2-3min because of the risk of anaphylactic or anaphylactoid reactions or pulmonary hypertension crisis. After administering the entire dose of protamine, the activated clotting time (ACT) is measured and it must be at the control ATT (measured before entering the pump). Another important moment of the post-ECC is closing the sternum. At this time, due to the increasing intrachest pressure, filling pressures (which may increase) must be reevaluated and doses of inotropic or volemia adjusted.

4. Patient’s transportation in the intensive care unit

The patient must be hemodynamically stable before being transfered from the operating room to intensive care unit. During transportation, ECG route, blood pressure (BP) and peripheral oxygen saturation (SpO 2) must be monitored.
On arrival in the intensive care unit, the patient connects to the fan, pleural pericardic drainage tubes are connected to the source of aspiration and pressure transducers and ECG electrodes to the monitor. A chest X-ray and a 12-lead ECG are performed. As standard laboratory tests, we harvest: arterial blood gases, electrolytes, hematocrit, platelets, clotting time (prothrombin time PT and activated partial thromboplastin time APTT). The intensive care physician/doctor on duty and medical staff are informed on hemodynamic data, vasoactive doses and any possible events in the evolution of the case.

5. Immediate postoperative period

Depending on the patient, the type of intervention and local protocols,
• most patients still remain mechanically ventilated 2-12h postoperatively. Sedation continues with low doses of benzodiazepines and opioids.
• In the first postoperative hours hemodynamic parameters should be closely monitored (BP, pace, heart rate, CVP) and chest drainage (bleeding). In the first 2 postoperative hours, a chest drainage for more than 200-300ml/h in the absence of blood disorder (measured by PT, APTT?) requires surgical reintervention. Intrachest bleeding, which does not occur in drain tubes, produces cardiac tamponade and requires emergency surgery.
• hypertension unresponsive to sedatives and analgesics requires administration of hypotension (use of sodium nitroprusside and nitroglycerin. Beta-blockers are used in patients with good ventricular function.
• Volume administration is guided by filling pressures (CVP ± PAP). Hypokalemia and hypomagnesemia require electrolytic rebalancing.
• Extubation is considered when a patient is decurarized, conscious, cooperative, hemodynamically balanced. Special attention will be given to obese patients, elderly or with subjacent respiratory diseases.