Critical Incidents During Cardiopulmonary Bypass

Filip De Somer

Since the introduction of cardiopulmonary bypass (CPB) over 60 years ago there has been a major evolution in both CPB components as perfusion techniques. In opposition to many other extracorporeal therapies such as dialysis where these innovations resulted in standard circuit configurations this did not happen in CPB. The heterogeneity and complexity of circuits and perfusion techniques makes CPB potentially prone to incidents.

Risk for incidents depends not only on the equipment and supplies used but also on training and education of the perfusionist as well as on the use of correct communication with other specialties during the procedure.

Several retrospective surveys were conducted over the last 20 years to investigate the occurrence of critical incidents and accidents during CPB procedures. Reported incident rates varied between 1:16 and 1:198 whereas the number of incidents resulting in serious injury or death were reported as 1:1236 to 1:3220.

A problem with all published surveys is that although all of them were retrospective in nature, the questionnaires and definitions of incidents were not similar what in part explains the reported differences.

In this chapter we will discuss some of the more common incidents during CPB.

Aortic Dissection During Cannulation

Intraoperative aortic dissection is a rare but potentially fatal complication. Although the incidence rate is low, this complication frequently lead to catastrophic results with high operative mortality. The most common sites of aortic injury are the ascending aorta cannulation site, cross-clamp site, partial occlusion clamp, proximal

F. De Somer (✉)
Chief Perfusionist, Ghent University Hospital, Ghent, Belgium
e-mail: Filip.DeSomer@ugent.be

M. Vives and A. Hernandez (eds.), *Cardiac Anesthesia and Postoperative Care in the 21st Century*, https://doi.org/10.1007/978-3-030-79721-8_9

anastomosis site and the cardioplegia cannula site. Femoral artery cannulation has a higher risk for dissection, compared to ascending aorta cannulation.

Prevention

Optimal blood pressure during cannulation with systolic blood pressure (SBP) around 100 mmHg. Too high SBP increases the risk of Aortic dissection. If SBP is too low (< 80 mmHg), aorta tend to collapse making more difficult to make the incision and having greater risk of tear too.

Diagnosis

Intraoperative recognition of aortic dissection is not straightforward. Suspicious clinical signs are sudden disappearance of radial arterial pressure, a sudden drop in cerebral oxygenation as measured by NIRS or a major change in EEG pattern. However, none of these signs is uniquely related to the occurrence of aortic dissection and differential diagnosis remains vital. Differential diagnosis can be made by Transesophageal Echocardiography (TEE) which has a high specificity and sensitivity or by epiaortic scanning.

Management

Once aortic dissection is confirmed it is key to know whether it is a type A or type B dissection. For type A, the aortic cannula should be replaced towards another insertion site most often the femoral artery or axillary artery. The patient is then cooled to 22–24 °C in order to stop the circulation and head cooling is applied. The use of selective cerebral perfusion should be considered. Perfusion techniques used during cooling down such as pH-strategy, flow rate and perfusate temperature will all influence outcome. A type B dissection can be often treated medically or with an endograft.

Aortic Cannula Malposition

Proper position of the aortic cannula tip is important. 1 to 2 cm into the aorta, directed toward the middle of transverse arch. The cannula might be inserted too much or misdirected, entering the cannula to the left carotid artery or misdirecting the flow to the innominate artery. The diameter of the aortic cannula can be too

small causing a jet effect redirecting the flow to the brain. Intramural placement of the cannula is another potential complication.

Diagnosis

- High systemic line pressure plus facial edema and a sudden increase of cerebral oxygenation by NIRS.
- Intramural placement of the cannula may be diagnosed by the absence of noting pulse flow in the systemic line pressure.

Treatment

Repositioning the cannula or changing to longer or shorter one.

Aortic Cannula Outlet Bleeding

Diagnosis

In some cases, blood loss is observed at the arterial cannula insertion site.

Management

- Maintain mean arterial pressure low.
- Place an additional suture string around the cannula.

Cerebral Edema Caused by Venous Cannula Obstruction

Vascular access remains a challenge during CPB procedures. Optimal venous drainage is essential for the prevention of tissue edema in organs. So, correct positioning of the venous cannula is important in preventing cerebral edema. The tip of the venous cannula might be malpositioned into the hepatic vein or the coronary sinus or the azygos or into the left atrium (LA) across the atrial septal defect.

Diagnosis

- Sudden rise in central venous pressure (CVP) and excessive flow resistance e.g. when the purse string is placed around the cannula. A sudden rise in CVP may cause RV dilatation and contractility impairment.
- Sudden or continuous drop in cerebral oxygenation.
- TEE may play an important role to detect malpositioning of the venous cannula.

Management

Reposition the cannula as soon as possible.

Misplacement of Venous Cannula

Over the years the use of minimally invasive cardiac surgery (MICS) has expanded. There is no questions that MICS is technically more demanding for the cardiac team. In most cases vascular access is done through the femoral blood vessels. Especially the insertion of the venous cannula towards the right atrium can pose difficulties. In rare cases the cannula tip might be positioned in the coronary sinus or hepatic vein (20). Prevention of malpositioning is done by TEE monitoring of the guide wire until it is at the level of the right atrium.

Diagnosis

- Difficulty in obtaining full flow with scattering of the venous line.
- TEE observation of the cannula positioned in the coronary sinus or hepatic vein.

Management

- Control with TEE volume status and cannula position.
- Replace the cannula.

Coronary Sinus Rupture During Retrograde Cardioplegia Administration

Retrograde cardioplegia offers several advantages such as avoidance of coronary ostium injury, more effective cooling of the myocardium in the case of distal coronary lesions, no interruption of surgery, diminished atrial rewarming and effective cardioplegia delivery in case of aortic regurgitation. Disadvantages are inadequate preservation of the right ventricle, delayed cardiac arrest and injury to the coronary sinus (CS). Typical CS injuries are hematoma, perforation and laceration of the CS or right ventricle. They can be caused by traumatic insertion of the catheter with stylet, overinflation of the CS catheter balloon, elevated CS infusion pressure, excessive flow during cardioplegia delivery, or excessive retraction of the heart when the catheter is in place or with the balloon inflated.

Prevention

When administering retrograde cardioplegia maintain:

- Delivery pressure between 0–40 mmHg
- Start delivery slowly, 125 mL/min/m^2, or approximately 200–250 mL/min
- Observe the pressure wave during inflation of the balloon and maintain pressure below 40 mmHg

Diagnosis

Any sudden increase or decrease in delivery pressure is suspicious and the surgeon should be immediately informed.

Management

In case of hematoma stop retrograde cardioplegia delivery and choose an alternative delivery site (aortic root or coronary ostium).

- In case of a perforation perform a direct suture repair.
- In case of laceration a pericardial patch is mostly used.
- Use TEE for follow up during the operation and the immediate postoperative period.

New Left Persistent SVC (LSVC) Diagnosis During Retrograde Cardioplegia Administration

- LSVC is present 0.3–0.5% of the population.
- LSVC usually drains into coronary sinus and then into the Right Atrium.
- In some cases, LSVC drains into the LA.
- LSVC should be rule out when large coronary sinus is observed on TEE.
- Suspect LSVC when Right SVC is small and left innominate vein is small or absent.

Diagnosis

Observing saline in the coronary sinus and then entering the RA on TEE when saline administered through a vein in the left arm.

Management

Use cardiotomy suction in the coronary sinus and Cannulate LSVC.

Circuit Thrombosis

According to all surveys coagulation disorders occur regularly during CPB. Circuit thrombosis can be caused by low heparin sensitivity, antithrombin III deficiency, platelet activation, diffuse intravascular coagulation (DIC), heparin induced thrombocytopenia (HIT) and in rare situations by erroneous administration of protamine.

Diagnosis

The detection of ongoing thrombosis is done by:

- Controlling ACT, a normal or marginally prolonged ACT value is a strong indicator of inadequate anticoagulation.
- Measuring the pressure drop over the oxygenator: an increase in pressure drop over the oxygenator is an indicator of beginning thrombosis in the hollow fiber bundle. The need for continuous increase of the revolutions per minute (RPM) of a centrifugal pump to maintain the desired blood flow without any changes in patient hemodynamics can also be a first sign of beginning thrombosis in the fiber bundle.

– Check if there is breakthrough of blood above the blood level in the venous reservoir as this represents partial filter obstruction in the defoamer/filter sock.

Management

– Administer immediately a bolus of 300 IU/kg unfractionated heparin (UFH) in case of a normal ACT or a bolus of 100 IU/kg UFH in case of a marginal prolonged ACT in the CPB circuit.
– Inform the other members of the cardiac surgical team and check whether clots are present in the surgical field.
– Check the circuit for visible clots and or fibrin deposits.
– Check the mass transfer of the oxygenator by taking a blood sample before and after the oxygenator.
– Order a spare CPB circuit.
– In case of severe thrombosis, check if it is possible to wean the patient from CPB. If not, cool down the patient and prepare for circuit/component exchange.

Oxygenator Failure

The term oxygenator failure can have different meanings. It can be defined as the inability of an oxygenator to transfer oxygen and to remove carbon dioxide, but also as a mechanical failure such as a blood or water leak. Exchange of a failing oxygenator is a technical demanding task and not without risk for the patient.

Diagnosis

A mechanical failure is relatively easy to diagnose as in most cases blood or water will be dripping out of the unit. Minor blood leaks should not be cured by exchanging the unit as the risk does not outweighs the benefit. Failure to oxygenate is mostly witnessed by dark colored blood leaving the oxygenator.

Management

Mechanical failure

Analyze where the leak is localized. A leak in a low pressure area such as the venous reservoir can often be cured by putting some bone wax over the fracture. The loss of small amounts of blood out of the gas exhaust can be caused by damage

to one or two hollow fibers and can be tolerated as it almost never will jeopardize gas transport. Larger leaks will necessitate component exchange.

Mass transfer failure

- This is witnessed by dark blood leaving the oxygenator eventually combined with a drop in cerebral oxygenation and low arterial oxygen saturation.
- Put the patient on 100% oxygen and check the complete gas circuit for lose connections.
- Warn the team that you experience an oxygenator failure.
- Take a blood gas before and after the oxygenator and calculate the oxygen transfer. If the oxygen transfer equals maximum oxygen transfer for the unit discuss with the anesthesiologist to deepen anesthesia level and check muscular relaxation.
- If venous saturation is low increase blood flow and check the calibration of the arterial pump.
- Check if foam is coming out of the gas exhaust. This indicates that plasma is leaking through the microporous oxygenation membrane. In patients on ECMO before undergoing cardiac surgery it is advisable to use a dense membrane instead of a microporous membrane.
- Take new blood gases before and after the oxygenator, if low oxygen transfer persist exchange the oxygenator.

Oxygenator exchange

- Mention loud and clear to the cardiac team that an oxygenator replacement is required.
- When available ask for a second perfusionist
- Bring a spare unit into the operating theatre
- If the aorta is not yet clamped, wean the patient from CPB and replace the unit afterwards.
- If the aorta is clamped, cool the patient down to at least 25 °C.
- Prime the spare oxygenator with an auxiliary pump.
- Once cooled exchange the oxygenator according to the hospital protocol. Each hospital should have an internal protocol for oxygenator exchange.
- After exchanging the oxygenator carefully check the circuit for remaining air.
- Restart CPB and check the functioning of the oxygenator by taking an arterial blood gas.
- Slowly rewarm the patient to the desired temperature.

Massive Air Embolism

Massive air embolism is a potential lethal complication that requires an immediate response. Causes of massive air embolism are (1) inattention to reservoir level, (2) reversal of pump head tubing or direction of pump head rotation, (3) unexpected

resumption of heartbeat, (4) inadequate steps to remove air after cardiotomy, (5) high-flow suction deep in a pulmonary artery, (6) defective oxygenator, (7) use of a pressurized cardiotomy reservoir, defective low level alarm (8) and (9) inadvertent detachment of oxygenator during bypass.

Diagnosis

Visual air passing through the arterial line. Once noticed immediately warn the whole team and look for the cause of the air embolism.

Management

- Stop CPB, clamp arterial and venous line.
- Place the patient in Trendelenburg position (Head down).
- Remove the aortic cannula and de-air the aorta by using a pump sucker.
- De-air the CPB circuit including arterial line and aorta cannula.
- Start retrograde venous cerebral flow over the superior vena cava at 2 L/min until no air is observed in the aortic root.
- Intermittent compression of the carotid arteries during retrograde cerebral perfusion will improve de-airing of the vertebral arteries.
- Induce hypertension by means of vasoconstrictor drugs.
- Ventilate the patient and the oxygenator with 100% oxygen.
- Administer steroids.
- Barbiturate anesthesia to decrease the cerebral metabolic rate of oxygen (CMRO2).
- After completing the de-airing of the patient re-establish full antegrade flow and maintain hypothermia (20 °C) for 45 min in order to optimize gas absorption.
- Slowly rewarm the patient to 35 °C and wean the patient from bypass
- When available the use of a hyperbaric chamber is advisable.

Summary

Although the number of incidents resulting in serious adverse effects or death remain low during CPB, vigilance of the cardiac team is important. Well trained perfusionists, use of check lists and simulation training sessions are all necessary in the prevention of incidents during CPB (Table 1).

Table 1 Summary of most common critical incidents its diagnosis and treatment

Incident	Diagnosis	Management
Aortic dissection during cannulation	– Sudden disappearance of radial arterial pressure – Sudden drop in cerebral oxygenation as measured by NIRS or a major change in EEG pattern – Differential diagnosis can be made by TEE which has a high specificity and sensitivity or by epiaortic scanning	– For a type B dissection can be often treated medically or with an endograft – For type A, the aortic cannula should be replaced towards the femoral artery or axillary artery and Asc Aorta and/or aortic arch replaced – The patient should be cooled to 22–24 °C in order to stop the circulation and head cooling is applied – Selective cerebral perfusion should be considered
Aortic cannula malposition	– High systemic line pressure plus facial edema and a sudden increase of cerebral oxygenation by NIRS – Intramural placement of the the cannula may be diagnosed by the absence of noting pulse flow in the systemic line pressure	– Repositioning the cannula or changing to a longer or shorter one
Aortic cannula outlet bleeding	– Blood loss is observed at the arterial cannula insertion site	– Maintain mean arterial pressure low – Place an additional suture string around the cannula
Cerebral edema caused by venous cannula obstruction	– Sudden rise in CVP and excessive flow resistance – Sudden rise in CVP may cause RV dilatation and contractility impairment – Sudden or continuous drop in cerebral oxygenation	– Reposition the cannula as soon as possible
Misplacement of venous cannula	– Difficulty in obtaining full flow with scattering of the venous line – Observing the the cannula positioned in the coronary sinus or hepatic vein by TEE	– Control volume status and cannula position by TEE – Replace the cannula
Coronary sinus rupture during retrograde cardioplegia administration	*Prevention* – Delivery pressure between 0–40 mmHg. Start delivery slowly, 125 mL/min/m^2, or approximately 200–250 mL/min. Observe the pressure wave during inflation of the	– In case of hematoma stop retrograde cardioplegia delivery and choose an alternative delivery site (aortic root or coronary ostium) – In case of a perforation perform a direct suture repair

(continued)

Table 1 (continued)

Incident	Diagnosis	Management
	balloon and maintain pressure below 40 mmHg *Diagnosis* – Any sudden increase or decrease in delivery pressure is suspicious and the surgeon should be immediately informed	– In case of laceration a pericardial patch is mostly used – Use transesophageal echography for follow up during the operation and the immediate postoperative period
LSVC diagnosis during retrograde cardioplegia administration	– Observing saline in the coronary sinus and then entering the RA on TEE when saline administered through a vein in the left arm	– Use cardiotomy suction in the coronary sinus – Cannulate LSVC
Circuit thrombosis	– A normal or marginally prolonged ACT value is a strong indicator of inadequate anticoagulation – An increase in pressure over the oxygenator is an indicator of beginning thrombosis in the hollow fiber bundle. The need for continuous increase of the RPM of a centrifugal pump to maintain the desired blood flow without any changes in patient hemodynamics can also be a first sign of beginning thrombosis in the fiber bundle – Check if there is breakthrough of blood above the blood level in the venous reservoir as this represents partial filter obstruction in the defoamer/filter sock	– Administer immediately a bolus of 300 IU/kg unfractionated heparin (UFH) in case of a normal ACT or a bolus of 100 IU/kg UFH in case of a marginal prolonged ACT in the CPB circuit – Inform the other members of the cardiac surgical team and check whether clots are present in the surgical field – Check the circuit for visible clots and or fibrin deposits – Check the mass transfer of the oxygenator by taking a blood sample before and after the oxygenator – Order a spare CPB circuit – In case of severe thrombosis, check if it is possible to wean the patient from CPB. If not cool down the patient and prepare for circuit/component exchange
Oxygenator Failure	– A mechanical failure is relatively easy to diagnose as in most cases blood or water will be dripping out of the unit. Minor blood leaks should not be cured by exchanging the unit as the risk does not outweighs the benefit – Failure to oxygenate is mostly witnessed by dark colored blood leaving the oxygenator	– Analyze where the leak is localized – A leak in a low-pressure area such as the venous reservoir can often be cured by putting some bone wax over the fracture – The loss of small amounts of blood out of the gas exhaust can be caused by damage to one or two hollow fibers and

(continued)

Table 1 (continued)

Incident	Diagnosis	Management
		can be tolerated as it almost never will jeopardize gas transport. Larger leaks will necessitate component exchange Mass transfer failure: Dark blood leaving the oxygenator eventually combined with a drop in cerebral oxygenation and low arterial oxygen saturation – Put the patient on 100% oxygen and check the complete gas circuit for lose connections - Warn the team that you experience an oxygenator failure – Take a blood gas before and after the oxygenator and calculate the oxygen consumption. If the oxygen consumption equals maximum oxygen transfer for the unit discuss with the anesthesiologist to deepen anesthesia level and check muscular relaxation – If venous saturation is low increase blood flow and check the calibration of the arterial pump – Check if foam is coming out of the gas exhaust. This indicates that plasma is leaking through the microporous oxygenation membrane. In patients on ECMO before undergoing cardiac surgery it is advisable to use a dense membrane instead of a microporous membrane – Take new blood gases before and after the oxygenator, if low oxygen transfer persists exchange the oxygenator – Oxygenator exchange (see text)

Recommended Reading

1. Charriere JM, Pelissie J, Verd C, Leger P, Pouard P, de Riberolles C, et al. Analysis of incidents, monitoring and safety devices of cardiopulmonary bypass for cardiac surgery for the year 2005 in France. Ann Fr Anesth Reanim. 2007;26(11):907–15.
2. Williams ML, Sheng S, Gammie JS, Rankin JS, Smith PK, Hughes GC, Richard E, Clark Award. Aortic dissection as a complication of cardiac surgery: report from the Society of Thoracic Surgeons database. Ann Thor Surg. 2010;90(6):1812–6; discussion 6–7.
3. Winiszewski H, Perrotti A, Chocron S, Capellier G, Piton G. Malposition of the extracorporeal membrane oxygenation venous cannula in an accessory hepatic vein. J Extra Corpor Technol. 2018;50(3):167–9.
4. Kurusz M, Girouard MK, Brown PS Jr. Coronary sinus rupture with retrograde cardioplegia. Perfusion. 2002;17(1):77–80.
5. Mills NL, Ochsner JL. Massive air embolism during cardiopulmonary bypass. Causes, prevention, and management. J Thorac Cardiovas Surg. 1980;80(5):708–17.
6. Authors/Task Force M, Kunst G, Milojevic M, Boer C, De Somer F, Gudbjartsson T, et al. EACTS/EACTA/EBCP guidelines on cardiopulmonary bypass in adult cardiac surgery. British J Anaesth. 2019.