Some Possible Reasons on the Failure of Cardiopulmonary-cerebral Resuscitation——The Enlightenments of a Successful Resuscitation of a Sudden Ventricular Fibrillation Patient during Resection of Lower Lob of Left Lung

Chen Zhiyang¹,²

¹Department of Anesthesia, Cancer Hospital, Shanghai, 200032, P.R. China, ²Department of Oncology, Shanghai Medical College, Fudan University, Shanghai, 200032, P.R. China

ABSTRACT

A patient had received resection of lower lobe of left lung under general anesthesia combined with the epidural block. By the end of the operation, ventricular fibrillation was shown on the monitor after thoracic surgeons had washed the cavitas thoracis with NS. cardiopulmonary-cerebral resuscitation (CPCR) was performed and 2 min later, all of the patient’s monitoring indexes were not improved, and even more that the patient’s heart had enlarged markedly (1.5 times as large as normal size or so) and become more and more stiff and purple, the massaging surgeon complained that the heart was too hard to massage. Two-way in-synchronism electric defibrillation was used out-of-body and succeeded. From this case, author mention some reasons on the failure of CPCR and some suggestions about CPCR.

Key words: Cardiac intervention resuscitation, CPR, LVEDP, CPP

CASE HISTORY AND THE COURSE OF ANESTHESIA MANAGEMENT AND RESUSCITATION

A 28-year-old man, 72 kg, with cancer of lower lob of left lung, American Society of Anaesthesiologists I. He had no hobbies of smoking and alcohol, no any other systemic diseases. He had received resection of the lower lobe of left lung under general anesthesia combined with the epidural block. The epidural acupuncture site was T5-6, and 15ml 0.375% ropivacaine was injected into his epidural space in all, continuous monitoring invasive blood pressure (BP) through the right radial arterial puncture catheter. Central venous catheter was placed through the right internal jugular venepuncture, natrium lacticium D-glucitol Ringer’s solution 500 ml and voluvem 500 ml were infused into. Induction of general anesthesia: Midazolam 3 mg, Vecuronium Bromide 8 mg, fentanyl citrate 0.2 mg, and Diprivan 120 mg were given intravenously. A 37⁰ right double-lumen tube was intubated, the patient’s life signs were stable during the operation, BP was among 110-125/70-85 mmHg. However, by the end of the operation, ventricular fibrillation was shown on the electrocardio monitor after thoracic surgeons had washed the cavitas thoracis with NS (30°C), heart rate (HR) amounted to 500–550 bpm and BP decreased down to 40/20 mmHg, ETCO₂ down to 5 mmHg, and SpO₂ down to 30%. The operation was temporarily interrupted, and cardiopulmonary-cerebral resuscitation (CPCR) had been performed. Adrenaline (AD) 1 mg and lidocaine 100 mg were injected through central vein and intrathoracic heart massage were performed by a surgeon after slitting arcula cordis. 2 min later, all of the patient’s monitoring indexes...
were not improved except that HR decreased to 350–400 bpm, and even more that the patient’s heart had enlarged markedly (1.5 times as large as normal size or so) and become more and more stiff and purple, the massaging surgeon complained that the heart was too hard to massage. Two-way in-synchronism electric defibrillation was used out-of-body, 100j was used in the 1st time, but the electrical conversion had not succeeded, 150j was used again then EKG showed sinus rhythm, and the size of the patient’s heart had gradually recovered. 5 min later, BP had gone up among 145–170/85–100 mmHg, HR among 100–110 bpm. The results of the radial artery blood gas analysis showed pH6.01, PaCO265 mmHg. 150 ml 5% NaHCO3 was infused intravenously drip, 200 mg hydrocortisone and 250 ml 20% Manicol were injected intravenously. After having been sewed the incision and stopped providing anesthetics, the patient had been analepsia, all of the indexes were normal, and he was extubated and sent to ICU. 2 days later, he was delivered to his ward and was discharged in 5 days.

IMPORTANT SIGNS OF THIS PATIENT AND SUGGESTIONS

Ventricular fibrillation is a kind of very serious arrhythmia because of losing blood-pumping function of the heart. A patient with ventricular fibrillation may die in several minutes if he will not receive effective treatment in time. This patient’s important signs were that:

• This patient had received resection of lower lobe of the left lung, we had observed the changes of the size of his heart during the CPCR, from normal size to markedly enlarged dimension, and returned to normal again.
• The patient’s heart enlarged markedly and become more and more stiff 2 min later after ventricular fibrillation, and the massaging surgeon complained that the heart was too hard to massage. This might show that the effects of intrathoracic cardiac massage had being decreased gradually if the patient’s life signs were not improved. Actually, neither extrathoracic cardiac massage nor intrathoracic cardiac massage could pump blood effectively now.
• What his heart become stiff and enlarged might suggest that the end-diastolic pressure of left ventricle (LVEDP) had been going up increasingly, considering the decreasing of DP, the coronary perfusion pressure (CPP) = DP-LVEDP, the CPP may be lowered down gradually and may be negative even, blood volume of coronary circulation might be reduced and stopped. In fact, the injected medicines could not be got into myocardial tissue to contribute their pharmacological effects because of myocardial ischemia now.
• Why his heart enlarged markedly might be mainly for the reasons of left ventricular enlargement, and myocardial cells were prolonged. Once the most appropriate initial length of myocardial cells was exceeded, the starting of the constriction of myocardial cells might be very difficult.
• Two-way in-synchronism electric defibrillation was used out-of-body, 100j was given in the 1st time, but the electrical conversion had not succeeded, 150j was given again then EKG showed sinus rhythm. This may demonstrate that electric defibrillation is effective to sudden ventricular fibrillation, and should be performed as soon as possible if the equipment is available.
• The actual blood volume of circulation might be very small; this may lead to the difficulty of playing the role of rescue medications which need to be transported to their target organs by blood flow.

SOME POSSIBLE REASONS ON THE FAILURE OF CPCR

When a patient has cardiac arrest unexpectedly during anesthesia or perioperative period, doctors will take the actions of CPCR. While we cheer for the successful rescues, we also have to learn from the failure cases, which directly lead to the loss of patients’ life. Based on this case of CPCR and my clinic experiences, I try to list some reasons of the failure of CPCR and bring out some suggestions here.

The condition of cardiovascular system of a patient in the state of cardiac arrest
While a patient being performed CPCR in the state of cardiac arrest, his condition of cardiovascular system may be quite different from that of in normal.

Blood pumped from the left ventricle is decreased substantially or stopped
Due to cardiac arrest, blood pumped from the left ventricle is stopped also. Extrathoracic cardiac massage cannot pump the same amount of blood like that of the normal heartbeat. An unskilled doctor or operating in wrong methods cannot make the heart pump enough blood. Moreover, it is even more important that the effects of cardiac massage may have been decreasing gradually if the pressure of the left ventricle is going up.

Artery system ischemia and vein system congestion
Due to the stop of heartbeat, left ventricle stops pumping and leads to artery ischemia and low BP. Since pulmonary circulation does not depend on the power from heartbeat, blood in pulmonary veins keeps flowing into the left atrium and left ventricle, which will make the latter enlarging. Blood in systemic circulation keeps flowing back to vena cava, right atrium, right ventricle, pulmonary artery, and pulmonary vein, left ventricle, and left atrium because of the gravitational effect. Together with the injected fluid and drugs, the vein system is congested with a lot of blood, injected fluid and drugs.
The changes of heart preload and cardiac contractility
Quantity of injected fluid and rescue drugs being congested in superior, inferior vena cava, right atrium, right ventricle, pulmonary vein, left atrium, left ventricle, and less of blood pumped from heart, the heart may be enlarged enormously, and the preload will be extremely heavy. Myocardial ischemia and the reducing of cardiac contractility, together with heavy heart preload resulting in myocardial initial length much longer than optimum length, will make it very difficult to restart the heartbeat.

Does cardiac massage work or not?
Does extrathoracic or intrathoracic cardiac massage, proved being an effective method in the early stage of CPRC, work for all patients and in whole period of CPRC? Apparently, due to doctors’ different proficiency, patients’ different cardiac, vascular condition, the massaging effects may be different very much. The massaging effects can be detected by either the wave shape of SpO2 or invasive artery BP monitoring. Increasing of the BP and wide and high wave of pulse prove an effective massage while acute and flat wave indicates failed massaging. Cardiac massage may be effective when a cardiac arrest has just happened while the pressure of the left ventricle is not so high. But after a while, the blood pumped out will less than the blood flowing into, and the pressure in left ventricle will increase gradually, cardiac massage will not be so effective now.

Do emergency drugs work?
There are two ways in which emergency drugs are absorbed. One is the drugs being perfused into the myocardium tissue through a coronary artery; the other is the drugs being absorbed through microvessels under endocardium. Because the amount of the drugs being absorbed through microvessels under endocardium is usually small after all, plus the increasing pressure in the cardiac chamber and the loss of periodical changing of pressure in left ventricle when cardiac arrest, the way through a coronary artery is the first choice. Pharmacological effectiveness of some cardiovascular drugs such as AD and lidocaine rely on the blood flow of the coronary artery. In some clinical cases, doctors try to inject the drugs into blood as much as he can, the results are that the drugs and the liquid injected congest in superior, inferior vena cava, right ventricle, right atrium, pulmonary vein and artery, left atrium and left ventricle, which lead to the substantial expansion of the heart, heavy preload, and high LVEDP, if the heart does not pump out sufficient blood or stop pumping blood actually. Meanwhile, because less blood is pumped out from the heart, DP decreases dramatically. Hence, CPP will decrease furthermore and may be negative even, the blood flowing into the coronary artery becomes even less, then the drugs being injected cannot show their effectiveness.

Injection through central vein or peripheral veins?
Based on the analysis above, the effects of emergency drugs will have a substantial difference between through central vein and peripheral vein. Compared to central vein injection, drugs injected through peripheral veins will be further diluted by blood, and the way to myocardial tissue is much longer; the effects will come slowly and be uncertain. Hence, central vein injection is preferred if circumstance permits.

As an example of the usage of AD, which can be injected into peripheral veins, central vein, air tube, and injected directly into the left ventricle. The way of the AD may process peripheral vein, central vein, right ventricle, pulmonary circulation, left atrium, left ventricle, aorta, coronary artery, and myocardium. For the reasons of the special state of the cardiovascular system in cardiac arrest, the most appropriate way is that AD should be injected directly into the left ventricle, and the next selection may be through air tube or central vein. The administration of AD through peripheral veins may not be chosen as far as possible.

Bolus intravenous injection or dripping injection?
Emergency drugs are dripped intravenously by some doctors in CPRC usually in some hospitals. Based on the analysis above, diluted and flowing with blood through so long way before they reach myocardial tissue, the drugs in the myocardial tissue will be very small actually. Plus the decrease of the blood supply of coronary artery, the effects of these drugs will come slower. Some experienced doctors inject AD at the dosage of 5–10 mg at a time through the peripheral veins sometime, rash it is, but for the reason that their infusing way is intravenous dripping. Bolus intravenous injection through a central vein can make these drugs not be diluted too much and contribute their effects fastly.

1 Mg ad, not enough or too many?
Some doctors inject AD at the dosage of 1mg while doing CPRC as recommended.[1] This method has been proved effective for many years. In clinical, sometimes 1 mg is far more not enough. Some doctors may inject AD 5–10 mg[2–6] at a time in CPRC. When heartbeat has stopped, AD can only be absorbed through endocardium. As you can see from above, AD 1 mg is far more not enough if through peripheral veins dripping. If extrathoracic cardiac massage works well or a temporary pacemaker is installed, the heart works again, BP increases to some extent, and the heart has not stopped beating for a long time, 1 mg AD may be too many because of its side effects. Our suggestion is that it will be safe and effective that AD was injected in batches after diluted 100 times through the central vein while monitoring invasive artery pressure. Because in clinical, the BP of some patients may increase unduly after injected AD 1 mg, result in cerebrovascular accidents, heavy heart afterload, which
are also very dangerous and can lead to the failure of CPCR. Hence, it is with isoproterenol (IP) and noradrenaline (NE), for the reason that overdosage of these drugs cannot be drawn back.

**Analysis of Artery Blood Gas and Vein Blood Gas**
When heartbeat has just stopped suddenly, artery blood gas is normal and maybe normal also several minutes later because blood in the aorta or some slender arteries like radial artery is just staying where it is for a while. If the heart does not pump out enough blood or extrathoracic cardiac massage does not work, the artery blood refreshes slowly. Hence, the results of artery blood gas do not reflect the real organ metabolism condition. Meanwhile, metabolism in tissues keeps going on, the extent of anoxia, oxidosis becomes worse. Analysis of vein blood gas will be quite different from that of several minutes ago considering the flowing of vein blood caused by gravitational effect. Hence, it is inappropriate to judge the extent of anoxia or oxidosis based on the analysis of artery blood gas. It may be more reasonable to decide the extent of oxidosis and correct it based on analysis of blood gas in venule, especially the cerebral blood. In clinical, jugular venous oxygen saturation (SO2) is often be used to reflect cerebral metabolism. Because the brain consumes the most oxygen in all organs, SO2 in jugular veins <50% indicates insufficiency for cerebral metabolism. Hence, we should monitor cerebral metabolism while doing CPCR, blood gas in jugular vein may reflect well the extent of anoxia and oxidosis of cerebral tissue as compared with radial artery blood gas.

**Failure of support therapy**
We have found that some patients get a successful cardiac, pulmonary resuscitation, and their conditions were not too serious, cerebral resuscitation fails occasionally, however, the possible reasons may be:

**Inappropriate treatment of cerebral edema**
Brain is one of the organs what are impacted extremely early by the lack of blood and oxygen after heart arrest, and encephal edema must emerge at first and will past 1–2 weeks. The level of conjunctival edema can reflect the extent of edema indirectly. Sufficient mannitol, corticoid must be applied in this period.

**Oxygen consumption**
After ischemic brain damage, some patients will have a muscular spasm, which will increase substantially the oxygen consumption of striated muscle all over the body. If not being treated in time, cerebral anoxia, which will exaggerate the injury of the brain and hinder a successful cerebral resuscitation, will happen inevitably. The treatment to muscular spasm includes using calmatives muscle relaxant, and dormancy treatment.

**Pulmonary infection**
After being ventilated mechanically, patients cannot evacuate sputum autonomously and need sputum suction while the glands of the respiratory tract can secrete regularly. Patients are susceptible to pulmonary infection after using a ventilator for a long time. Applying a large amount of antibiotics can cause flora imbalance and fungal infection occasionally. Clinical doctors must be cautious about that.

**SOME SUGGESTIONS FROM THE AUTHOR**

**Installing temporary pacemaker and using electrical defibrillation as soon as possible**
The rescue conditions and equipment are the best usually if patients have a cardiac arrest during the perioperative phase. Electric defibrillation must be employed as soon as possible while ventricular fibrillation occurring and a temporary pacemaker should be installed at once without X-ray in case of no heartbeat. Benefiting from helping patient’s heartbeat autonomously, the effects of pumping blood of employing a temporary pacemaker are superior to any kind of cardiac massage actually.

**No transfusing too much and too fast**
If a patient’s heartbeat has stopped already and the effects of cardiac massage are uncertain, it is wise to slow down infusion speed or just stop infusion until cardiac massage works.

**To correct oxidosis based on the results of jugular vein blood gas monitoring**
Jugular vein blood gas should be closely monitored while doing CPCR. Jugular vein blood gas can reflect relatively the extent of oxidosis of the brain. While slender arteries blood gas can only reflect the effect of treatment, it cannot be used as the reference to correct oxidosis especially in the earlier period of CPCR.

**Bolus injecting emergency drugs through central vein instead of through peripheral veins**
For a patient whose heartbeat stops, the blood does not flow also. Drugs delivered through peripheral veins can hardly reach vena cava; much less can reach to the coronary artery and myocardial tissue. Some doctors increase the pressure of the infusion tube, which can just inflate the vena cava more and more actually. To let emergency drugs reach myocardial tissue as fast as it can, we should use bolus injection through the central vein as far as possible instead of using dripping injection through peripheral veins.

Strong cardiovascular-activities drugs such as AD, IP, and NE should be diluted before injecting through a central vein in batches. Increasing little by little based on the BP measured by continuous invasive artery monitoring will prevent correcting
low BP too much. Increasing the dosage of AD through central vein or respiratory tract if no effects come out.

Expanding the capacitance vessels, such as pulmonary veins and systemic circulatory capacitance vessels will relieve the preload of the heart temporarily, which may help the restart of heartbeat.

Many cardiac emergency drugs, such as AD, can be injected through the left ventricle, and even injected through aorta or coronary artery, which the drugs will reach myocardial tissue easily while no blood-pumping or less of blood flow of coronary artery. This is the topic we are studying. In the sight of evidence-based medicine, this needs to be proved by sufficient clinical research evidence. Cardiac intervention techniques will provide strong support to this way of injection.

Some blood in left ventricle should be drawn out through a cardiac catheter in order to reduce the pressure of the left ventricle. By means of this method, the pre-load of left ventricle can be decreased, most appropriate initial myocardial length maybe obtained, and the restarting of hart beating maybe easily. Following this, the LVEDP maybe reduced and the CPP can be recovered, AD and LIDOCAINE can be circulated to myocardial cells. A new field which I name it as cardiac intervention resuscitation medicine is showing up.

REFERENCES


How to cite this article: Zhiyang C. Some Possible Reasons on the Failure of Cardiopulmonary-Cerebral Resuscitation——The Enlightenments of a Successful Resuscitation of a Sudden Ventricular Fibrillation Patient during Resection of Lower Lob of Left Lung. J Clin Res Anesthesiol 2018;1(1):1-5.