The Alphabet of Cardiopulmonary Resuscitation.

For medical students by a medical student.

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What if you are in a supermarket and witness a man suddenly collapsing. He looks grey and stops breathing. Would you know what to do? And maybe more importantly why you are doing what you're doing?

In England in 2013 alone there were approximately 28.000 out of hospital cardiac arrests (OHCA) that Emergency Medical Services (EMS) tried to resuscitate. 8.6% survived until hospital discharge. This number unfortunately, has barely changed over the years. There are several parts to what helps increase survival rates and together they are called the chain of survival and it consists of four links:

1. Early recognition and call for help
2. Early CPR
3. Early defibrillation
4. Post resuscitation care

These links seem straightforward yet there still is room for improvement. In order to improve you must know what you are doing and why! The purpose of this article is to provide medical students with a more in-depth look to the practice of CPR by highlighting a few concepts.

A for Agonal breathing

Approximately half of Cardiac Arrest (CA) victims show a phenomenon called agonal breathing also known as gasping. It is thought that agonal breathing is a brainstem reflex. It is a surprising reflex, capable of reaching a minute volume of almost 4L. This often confuses bystanders into thinking that victims are suffering from labored breathing. This translates into that bystanders fail to recognize OHCA in 84% of victims. This is a major cause of serious delays in the chain of survival.

As previously mentioned gasping is capable of a reaching relatively large minute volume considering that the cardiac output during CPR is about a third of normal. For these circumstances this is an adequate ventilation-perfusion ratio and sufficiently ventilates the alveoli. Spontaneous gasping not only repositions the tongue, jaw and neck to allow unobstructed air passage, it also produces forward blood flow, most likely because of changes in intrathoracic pressure. Finally, gasping is associated with increased odds of survival and one could say that gasping is an effective life-sustaining reflex!

B for Breathing

Rescue breaths or maybe more aptly named Positive Pressure Ventilation (PPV) has been a central part of CPR since its inception. However, physicians are starting to reconsider its current role in CPR because of lacking evidence in favor of performing PPV. (1) There has been no conclusive evidence that PPV increases the odds of survival, excluding cases of hypoxic arrests. (2) High quality chest compressions can be capable of maintaining normal arterial levels of carbon dioxide and oxygen during the first few minutes post-arrest. (3) PPV increases intrathoracic pressure and consequently causes decreases in CO and Coronary Perfusion Pressure (CPP) during CPR. (4) Because chest-compressions can maintain normocarbia, adding PPV can thus result in hypocarbia. This causes vasoconstriction of cerebral blood vessels and therefor decreased cerebral blood flow and might have a detrimental effect on neurologic outcome. (5) In victims of CA there is always a risk of regurgitation and aspiration; PPV puts victims at greater risk of regurgitation and aspiration due to air entering the esophagus and stomach. (6) Performing PPV is demanding because the provider must perform complex airway maneuvers and preform mouth-to-mouth ventilation. This makes PPV prone to poor performance and unwillingness because of fear for disease-transmission.

In short we should be critical about PPV and it is something we should continue to investigate.

C for Compressions

Chest compressions have become a central part of CPR and high quality compressions the focus of guidelines. In order to understand what constitutes as high quality, you have to know a few physiological principles of chest-compressions.

There are two theories of how circulation is achieved during CPR: (1) the cardiac pump, where the heart is directly compressed between the sternum and spine and the valves open and close as if the heart were pumping by it self. (2) The thoracic pump, in which the intrathoracic pressure is equally applied to the vasculature and heart in the chest and forces blood to circulate. The consensus however is that CPR produces increases in intrathoracic pressure and forward flow by means of an indirect cardiac pump mechanism. This is supported by observations of patients who were experiencing ventricular fibrillation during catheterization and were able to maintain circulation by coughing. When applying physics you might think that greater negative intrathoracic pressures create larger venous return and therefor during the compression higher forward blood flow. Hence the inventions of tools that aim to create bigger pressure differentials, for example active decompression by ways of a vacuum device attached to the chest. In order to create the maximum pressure difference during CPR the chest must be compressed sufficiently, PUSH HARD!, and the chest must be allowed to fully recoil in order to generate the lowest pressure possible.

Capnography is an excellent tool for measuring CPR efficacy because studies have shown marked increases in venous PCO_2 and larger venous-arterial PCO_2 gradients during CPR thus $ETCO_2$ is a good quantitative measure for blood flow achieved during CPR. Using this method researcher demonstrated that

increasing the compression frequency increases the ETCO2, which we now know, corresponds with an increased CO. SO PUSH FAST!
Studies have shown that there are critical levels of CPP that dramatically increase the odds of successful defibrillation. Diastolic aortic pressure and right-atrial pressure are the primary determinants of CPP. CO and therefor CPP increase with rate and depth of compressions and also increase when compression are prolonged to about half of the compression-recoil cycle. If we look back at chapter B it mentions that PPV decreases CPP. This happens because PPV increases intrathoracic pressure, decreasing CO and causing an increase in right-atrial pressure resulting in a decrease in CPP.

The odds of detecting a shockable rhythm decrease with every minute from onset of CA. CPR greatly increases these odds, most likely because of blood circulating through the coronary arteries supplying the muscle with oxygen.
Interruptions of compressions during CPR are common. For example: rescue breaths, placing of electrodes, miscommunication etc. However, even short interruption during compression can have disastrous consequences for CA outcomes. Interruptions during CPR result in a run-off of blood out of the aorta causing a decrease in aortic pressure and as we now understand a decreased CPP. Even when quickly resuming compressions it takes several compressions to reach maximum levels of circulation. That is why there is such an emphasis on minimizing interruptions.

If you apply this knowledge to CPR you get high quality compressions as defined by the AHA 2010 guidelines:

- A compression rate of at least 100/min
- A compression depth of at least 2 inches (5 cm) in adults and a compression depth of at least one third of the anteriorposterior diameter of the chest in infants and children
- Allowing for complete chest recoil after each compression
- Minimizing interruptions in chest compressions
- Avoiding excessive ventilation

Unfortunately a large fraction of chest-compressions are not of high quality.

D for Defibrillators

Defibrillition is the definite treatment of pulseless arrhythmias. Early defibrillation significantly increases the odds of ROSC and survival. The increase of available Automated External Defibrillators (AED) has led to an increase of OHCA survivability. The purpose of giving a shock to the heart is to extinguish VF by depolarizing the myocardium in order give a perfusing rhythm the chance to establish.

AED unfortunately cause necessary interruptions during CPR. The analysis of the ECG requires bystanders to not touch the victim and during the actual delivery of the shock it not recommended.
However, good integration of the AED into CPR will most likely greatly improve outcomes.

In short, cardiac arrest and resuscitation physiology is what drives CPR and the continuous effort to better understand it is what will advance our skill in resuscitating patients suffering from cardiac arrest.

Knowledge alone is not enough to save the man at the supermarket. It's your action that will count! Get certified for BLS and drag your fellow students with you. Isn't this exactly why we want to become doctors?

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