Managing patients in cardiopulmonary arrest due to reversible conditions (Proceedings)

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Review the common pathophysiology – Cause – Affect of the reasons cardiopulmonary arrest occurs in clinical practice and then review the current views on basic and advanced cardiac life support and provide my personal views on the subject based on over 35 years of clinical and some years of experimental research experience.

Objectives of this presentation

Review the common pathophysiology – Cause – Affect of the reasons cardiopulmonary arrest occurs in clinical practice and then review the current views on basic and advanced cardiac life support and provide my personal views on the subject based on over 35 years of clinical and some years of experimental research experience. Some of my experience comes from my emergency medical technician experience. In this regard I am able to carefully compare, in my opinion, the drastic differences as well as similarities between CPR in animals and CPR in humans. The title reminds us of which patients should be resuscitated.

Pathophysiology

Cardiac Arrest leads to dysoxia in the brain mitochondria... energy failure. Calcium influx into mitochondria AND THE cytosol occurs leading to a literally melt down of cellular enzyme pathways making even getting and using oxygen into the metabolic energy manufacturing pathways. Also it should be remembered that ROS production, following the restarting of perfusion occurs and these oxygen radicals combine with inducible NO and produces peroxylnitrite. This compound literally blows the cell up! It is like intracellular TNT. The cells most vulnerable are in the brain & heart (the heart by a factor of half that of the most sensitive areas of the brain (cortical-subcortical occulocephalic reagions involved with visual recognition). Within a few minutes of severe dysoxia as seen with cardiac arrest, this area is rendered into various stages of cell injury and death – from Stage I where no morphological changes are seen on scanning EM to Stage V where cells are literally autolyzing. It is estimated by some that the percentage of the number of cells reaching the stage IV level of cell injury, where the 'point of no return' is for recovery must reach approximately 35-45% before irreversible neuro-injury occurs, so long as this primary 'hit' does not propagate into a secondary injury caused by reperfusion injury mechanisms. These can be suppressed by such things as hypothermia, the most important, and use of neuro-excitation blockers such as lidocaine, calcium channel blockers,

superoxide radical blockers and scavengers, etc., but the most important is the IMMEDIATE USE OF LOCAL AND REGIONAL – GLOBAL HYPOTHERMIA starting at the time of the arrest and lasting at least several hours post resuscitation.

Principle goal of CPR or the new term cardiopulmonary and cerebral (CPCR) circulation

To resume normal or near normal blood flow and oxygenation to the brain and heart within a maximum of only several minutes. From my experience that means at the maximum 5 minutes and at the minimum 3 minutes. Any time period beyond this critical 5 minutes leads to impairments that could be life-long. EVERY SECOND IS IMPORTANT.

In review of 10 cases that were successfully resuscitated it appears that the use of Doppler flow detection using the cornea of the eye in dogs and cats as the landing zone for the pediatric flat probe of the Doppler Blood Flow Detector to assess cranial blood flow during CPCR Provide adequate blood flow was the most accurate means, outside of visual and palpable assessment of cardiac performance, was the best means of determining the meeting of the principle objective – the normalization of enough blood flow to prevent cardio-cerebral injury past the point of no return where pet function after the arrest would be poor, with such things as permanent blindness or cerebral palsy. It appeared in the analysis of the ten survivors with good neuro function that other organ systems were also resuscitated adequately to prevent irreversible injury, including spinal cord, lung, liver, GI tract, kidney and endocrine organs if the brain and heart were resuscitated adequately.

Keys to successful resuscitation

Requires several steps that all must be done well

1. Being Prepared with Oxygen, bag-valve mask, Crash Cart, defibrillator, Doppler blood flow detector that is able to assess cranial blood flow ;

2. Team work that is efficient, which includes the use of training drills, keeping in mind that the minimum number in the resuscitator team is 2 and best, based on watching over 30 actual CPCR events in progress, with 4-5 team members IF COORDINATED well (I shoot for three as this is the most common scenario of team members involved in most practices that I have visited:

a. A team member - Being the person that provides cardiac or thoracic compressions at a rate of from 80-100 per minute, or does other advanced maneuvers such as a resuscitative thoracotomy, opening of the pericardium, direct cardiac massage, delivery of intra-cardiac epinephrine, aortic cross clamping, other intrathoracic maneuvers as necessary such as clamping off a leaking lung lobe and closure of the chest following irrigation and chest tube-placement

b. B team member - Being the person who ventilates the patient if not on a volume cycled ventilator which is often the case, at a rate that satisfies CO2 unloading, commonly only 6-10 breaths per minute.(NOTE: it is my experience that NOT having anesthetized patients on a ventilator predisposes them to arrest – one of the most common causes of anesthesia associated arrests is poor ventilation leading to respiratory acidosis and sensitivity to vaso-vagal or sympathetic discharge influences that may occur with movement, hypothermia, pain response, parasympathetic discharging events such as ovarian ligament 'strumming', and rolling patients over.) And this is the person that has to do all other functions then if no other team members are available (team member C)

c. C team member - Being the person that is really moving with many duties from getting monitors on board, gets drugs drawn up and injected after gaining an iv site if this is not yet present, records time and events, sets and charges the defibrillator, monitors Doppler flow during compressions and ECG. As can be appreciated this members responsibilities is easily not possible for one member to do all these duties. Assigning ONE to be doing the Doppler Monitoring is more realistic as this is a full time job, and having two others working together to get the other duties completed.

3. Immediate recognition using LOC and noting an unconscious 'PNB' (pulseless non-breathing), and if anesthetized or unconscious, then monitors such as Doppler blood flow on a beat-by-beat basis, ECG, SpO2, ETCO2, Arterial BP;

4. Immediate treatment that is effective in producing good (adequate) blood flow to brain and heart (enough that you hear pulsatile blood flow of sufficient strength that it is easy to detect with ocular Doppler produced either by closed chest or open chest techniques – understanding that the open chest technique, because it is much more effective in generating forward blood flow, by a 3-1 ratio; and is used immediately if closed chest techniques do not produce adequate flows within the first 1-2 minutes, 3 minutes maximum if drugs are given a try to increase effectiveness; The reason for more effectiveness my the open chest technique has to do with two reasons: a. Differences in generated perfusion pressures (arterial-venous pressure) and b. a more direct ability to produce flows with less effort and mechanical forces . With the closed chest technique venous pressure is also increased (approx 20-30 mmHg)and therefore the AV difference is less than the open chest technique which does not increase the venous pressures (staying at 5-10 mmHg), therefore with closed chest techniques perfusion pressure is commonly 80-30 = 50 mmHg whereas the open technique provides perfusion pressures that are 90-10 = 80 mmHg as can be seen this also means more coronary circulation for open chest techniques because coronary perfusion pressures are also better.

5. Defibrillation if it is found indicated on ECG or by direct visualization of the epicardium that is effective and uses the minimal amount of joules necessary to cardiovert a ventricular fibrillation arrhythmia to a normal sinus rhythm with attendant pulsatile flow with each beat, appearing adequate based on visualization, palpation, blood flow detection using a Doppler Blood Flow Detector. Good CPCR – FLOW!!! Frequently in anesthesia associated asystole and ventricular fibrillation.

6. Monitoring cardiac function and flow with continuous Doppler blood flow – with the flat pediatric probe on the palmar arterial arch area of the front paw – area just proximal to the metacarpal pad on the midline, taped in place firmly, and ECG monitoring as well as SpO2 probe on the tongue or lip and watching the graphic waveform of the oximetry sensor ; which allows

7. Neuroprotective Stratigies – This is so very important and can be done and should be done within moments of obtaining good flows returning. In fact, there is some research in children that suggests that these need to be initiated while the CPCR is in progress. The simplest is to induce mild to moderate hypothermia and especially to use ice around the head and neck. The length of time the hypothermia is required has not been determined, however I am going to give guidelines based on the length of time that the CPCR was required , the effectiveness of the CPCR based on the amount of flow effected by the compressions or massage, and the length of time taken before good continuous spontaneous arterial pulsitile flows were reestablished: minimum time of 2 -4 hours at 94-96 degrees or 1-2 hr at 86-90 degrees; moderate time of 4-6 hours of 94-96 degrees or 2-4 hours at 86-90 degrees; longest time of 8-12 hours of 94-96 degrees and 4-6 hours at 86-90 degrees. It is very important to use acepromazine iv to prevent rewarming ventricular fibrillation in any patient below 88 degrees and it is always important to depress shivering by use of this drug as well. Gradual rewarming in the extreme cases may take as long as 24 hours however in most cases it can be done with 12 hours. During this time it is best to have the patient under general anesthesia with small amounts of a narcotic, use of pento or phenobarbital and remaining on a ventilator throughout t the hypothermia period and at least the first half of the rewarming period.

Sources of information and references

The Ultrasonic Doppler Flow Detector with a 9.4 MHz 15 degree angle flate flow probe by either Parks Medical Electronics, Model 811-B w/ accessories, including a flat flow probe, and with an estimated to cost approximately $850.00 or the use of the Vet-Dop by DVM Solutions (who supply better service) is also effective as a good anesthetic guide to systemic arterial perfusion or flow which a the principle monitoring tool.

Evaluation of a Doppler flow detector and probe on the eye for determining effectiveness of blood flow generation with cardiac massage in dogs. Crowe DT Proceedings 3rd IVECCS, San Antonio TX, 1992 p 387 We used 3 anesthetized dogs with a carotid artery catheter a Doppler 9 mm flat probe on eye and graded the strength of the sound as Absent, Weak, Moderate, Strong. Ventricular fibrillation was induced with a 9 v battery attached to two epicardial needle. Open chest OC-CPCR was commenced. Doppler flows were recorded and the CPCR was performed. The aorta was cross clamped and again sounds were judged to be either zero, weak, moderate, strong. Euthanasia was then done and the carotid catheter position was verified at necropsy.

Evaluation of Doppler flow detection for clinical assessment of the effectiveness of CPCR in dogs and cats. Crowe, Kovacic, Kirby Proceedings 3rd IVECCS, San Antonio TX, 1992; there were 25 Patients: 15 dogs & 10 cats. All had had spontaneous cardiac arrest. Closed Chest CPCR was begun in all cases. A Doppler flow probe was placed on the cornea and swishing sound judged: strong, moderate, weak, not able to hear. Open chest CPCR was accomplished on those that did not have good flows, with 4 of the open chest cases surviving with good neuro function. All these had strong Doppler sounds and the strongest came from a patient that had his aortic cross-clamped.

Usefulness of ocular and metacarpal Doppler blood flow sounds in experimental canine CPR Spreng, Crowe, DeBehnke, Swart, Proceedings 4th IVECCS San Antonio TX, 1994 there were 40 anesthetized dogs that were Instrumented (Aortic and Rt atrial p), with Doppler probe on cornea, Doppler over metacarpal palmar arch. EMD was induced by asphyxia with the ET tube clamped in all these anesthetized dogs. Closed chest CPCR was started. Monitoring the Doppler sounds generated were graded 0 – 3, with 0 = none, 1– 2 = present, 3 = strong; Metacarpal Doppler could not be heard consistently and indicated poor peripheral flows. Ocular Doppler could be heard consistently and the sound strength correlated w/ systolic pressure as determined Doppler .0 =58 mm +24, 1 = 71 mm + 13, 2 = 114 mm +36, 4 = 144mm +23 - From this study it appeared that we could predict which animals had the best pressures generated by listening to the strength of the Doppler signal. H highly recommend the use of Doppler flow as a tool used to determine effectiveness of the blood flows both before, during and after cardiac arrest; and it can also be used to determine flow which is more important than P as Q = P/R.