Title:
Approach to Cardiopulmonary-Cerebral Resuscitation

Session Description:
A firm grasp on the goals and techniques for CPCR is essential in maximizing the chance of a successful outcome. In this hour, we will explore an evidence-based approach to CPCR using guidelines set forth by the RECOVER initiative.

Lecture Notes:
Publication of the RECOVER initiative in 2012 marked the first attempt to characterize our approach to veterinary CPCR within the context of available experimental and clinical research. Although many knowledge gaps were uncovered, it was encouraging to see a scientific background to our approach as well as to give future investigators spaces to explore. The information available to the committee and its many investigative arms allowed for modification and refinement of previous CPCR recommendations. There were 5 domains explored by the committee, including preparedness, basic life support (BLS), advanced life support (ALS), monitoring and post-arrest care. In the interest of time, we will explore preparedness, ALS, BLS and monitoring. It should be mentioned that although the discussion occurs in a linear fashion, the latter three points occur simultaneously during CPCR.

As in the case of any acute emergency, an efficient team approach is necessary to maximize the chance of a successful outcome during a CPCR event. Delays in starting CPCR can be detrimental to the patient. An organized crash cart with a weekly census can help ensure quick access to everything needed for an arrest event. Near this station, cognitive aids such as dosing charts, CPCR algorithms, and checklists can be posted to improve adherence to guidelines. Additionally, regular training of doctors and technical staff can help improve psychomotor skills and comfort during situations that may cause anxiety for CPCR participants.

Basic life support (BLS) includes detection of cardio pulmonary arrest (CPA), chest compressions, intubation and provision of ventilation. Identification of CPA can be challenging in the emergency situation. Pulses can be difficult to identify in the obese or hypovolemic patient and agonal breaths may be misinterpreted for spontaneous breathing. For this reason, it is recommended to start CPCR immediately upon suspicion of CPA. The detriment associated with a delay in CPCR outweighs the risk of injury to a living patient. If CPCR is unwarranted, the patient will let you know immediately. In the unresponsive recumbent patient, an airway, breathing, circulation (ABC) assessment is recommended within 10 seconds. Our preferred method to identify CPA is brief focused cardiac ultrasound between rounds of compressions. This allows us to assess contractility as well as to identify potential causes for CPA, including pericardial effusion. The goal for chest compressions is to achieve good quality compressions without delay. With the patient in lateral recumbency and your hands placed either over the heart (cardiac technique for patients <10kg) or over the widest portion of the thorax (thoracic pump technique for patients >10kg), the chest should be compressed 1/3 – 1/2 its width at a rate of 120-150 compressions per minute. It is important not to lean on the patient and to allow for full recoil of the chest and for the heart to fill completely with blood, thereby increasing cardiac output. It is important to engage your core while performing chest compressions. Locking the elbows and driving your shoulders down through the compression point allows decreases the risk of fatigue. Ideally, each member of the CPCR team performs compressions for 2 minutes, allowing for ECG assessment between cycles. While ventilation may not be as important for CPA of cardiac origin, our patients are more likely to experience CPA of non-cardiac origin. Therefore, intubation and provision of ventilation are important steps during CPCR. We prefer to intubate while in lateral recumbency to allow for ongoing chest compressions. The tube cuff should be inflated and tube tied in place. The goal is a rate of ~10 breaths per minute while titrating to an $EtCO_2$ of 35mmHg-45mmHg. It is important not to overinflate the lungs during positive pressure ventilation. Pressures over $20cmH_2O$ may decrease venous return and cardiac output. We typically aim for a peak inspiratory pressure of about 10-$15cmH_2O$. While hypoxia is known to be detrimental, hyperoxia has been found to be associated with a poor outcome in people who experience CPA. Excessive oxygen supplementation causes an increase in reactive oxygen species and free radical formation, which may further neurologic injury. For this reason, flow-by oxygen through an AMBU® bag may be a better option than a system that delivers 100% oxygen (e.g. standard anesthesia circuit).

Advanced life support involves therapy with vasopressors and anticholinergics; and correction of acid-base disturbances and volume deficits. Dilute medications can be given through the endotracheal tube, however evidence for the effectiveness of this technique is lacking. Ideally, intravenous access should be obtained as soon as possible through standard peripheral catheter placement, a jugular cut-down technique or an intraosseous catheter. At the onset of CPCR, doses of epinephrine (low dose) and atropine can be given, followed by repeat doses of epinephrine every other round of compressions as needed until return of spontaneous circulation (ROSC) is achieved. Blood pressure is titrated using vasopressors (e.g. norepinephrine, dopamine, etc.) and volume resuscitation. It is important to reserve fluid therapy for hypovolemic patients, as excessive fluid therapy can lead to decreased coronary perfusion pressure. Administration of sodium bicarbonate to correct acid-base disturbances has fallen out of favor due to its use being associated with a poor outcome. Rather, the typical disturbances, metabolic (lactic) respiratory acidosis, are corrected with good-quality chest compressions and ventilation, respectively.

Monitoring patients during CPCR revolves around ECG and $EtCO_2$. ECG is an invaluable monitoring tool, however it is important to not interrupt compressions for the sake of ECG monitoring. Rather, the ECG tracing should be assessed every 2 minutes between compression cycles. Additionally, ECG is prone to over interpretation. Pulseless electrical activity is a common arrhythmia associated with CPA, and can look like a normal sinus rhythm. For this reason, ECG should not be used as a sole indicator of ROSC. A more useful indicator of ROSC, as well as compression quality is $EtCO_2$. This can be considered a surrogate for cardiac output (CO). As the heart beats, CO_2 travels to the lungs through the pulmonary circulation. Cardiac output diminishes during CPA, so a downward trend of $EtCO_2$ may be seen in an intubated patient (e.g. a patient under general anesthesia) experiencing CPA. During CPCR, however, good-quality chest compressions will increase CO enough to move CO_2 to the lungs. A reasonable goal for $EtCO_2$ during compressions is about 10-15mmHg, meaning reasonable CO has been achieved. An EtCO2 >20mmHg is likely associated with ROSC.

While CPCR can be daunting, basic knowledge and routine training can help improve outcomes for patients who experience CPA. A firm grasp on BLS alone is truly enough to give our patients a chance to survive an arrest event. Once ROSC is achieved, however, the real challenge begins. Close monitoring and goal-directed therapy are essential to avoid and/or overcome post-arrest syndrome. At the point of ROSC, a 24-hour facility with critical care may be your patient's best chance of survival.

REFERENCES

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