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RECOVER evidence and knowledge gap analysis on veterinary CPR. Part 5: Monitoring

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Abstract

Objective – To systematically examine the evidence on patient monitoring before, during, and following veterinary CPR and to identify scientific knowledge gaps.
Design – Standardized, systematic evaluation of the literature, categorization of relevant articles according to level of evidence and quality, and development of consensus on conclusions for application of the concepts to clinical practice. Relevant questions were answered on a worksheet template and reviewed by the Reassessment Campaign on Veterinary Resuscitation (RECOVER) monitoring domain members, by the RECOVER committee and opened for comments by veterinary professionals for 3 months.
Setting – Academia, referral practice, and general practice.
Results – Eighteen worksheets evaluated monitoring practices relevant for diagnosing cardiopulmonary arrest (CPA), monitoring CPR efforts, identifying return of spontaneous circulation (ROSC), and post-ROSC monitoring.
Conclusions – Although veterinary clinical trials are lacking, experimental literature using canine models and human clinical trials provided relevant data. The major conclusions from this analysis of the literature highlight the utility of end-tidal carbon dioxide ($EtCO_2$) monitoring to identify ROSC and possibly to evaluate quality of CPR. In addition, recommendations for ECG analysis during CPR were addressed. Unless the patient is instrumented at the time of CPA, other monitoring devices (eg, Doppler flow probe) are likely not useful for diagnosis of CPA, and the possibility of pulseless electrical activity makes ECG inappropriate as a sole diagnostic tool. Optimal monitoring of the intra- and postcardiac arrest patient remains to be determined in clinical veterinary medicine, and further evaluation of the prognostic and prescriptive utility of $EtCO_2$ monitoring will provide material for future studies in veterinary CPR.

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Abbreviations

AED	automatic external defibrillator
AMSA	amplitude spectrum area transformation
CPA	cardiopulmonary arrest
CPP	coronary perfusion pressure
EGDT	early goal directed therapy
$EtCO_2$	end-tidal carbon dioxide
ETT	endotracheal tube
LOE	level of evidence
MAP	mean arterial blood pressure
PCO_2	partial pressure of CO_2
PEA	pulseless electrical activity
PEEP	positive end-expiratory pressure
PICO	population, intervention, control group, outcome
RECOVER	Reassessment Campaign on Veterinary Resuscitation
ROSC	return of spontaneous circulation
VF	ventricular fibrillation
VT	ventricular tachycardia

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V_d	alveolar dead space volume
Vt	tidal volume

Introduction

The goals of a separate domain devoted exclusively to monitoring are 3-fold. First, because of the striking differences in cardiovascular physiology during CPR compared to states of spontaneous circulation, special considerations apply to the use of familiar hemodynamic monitoring technology under these circumstances. Second, by recommending monitoring equipment and techniques necessary for the performance of high-quality CPR, guidance for practitioners aiming to update clinical CPR practices and preparedness is provided. Lastly, by highlighting deficiencies in the current literature specifically concerning evidence for monitoring protocols and techniques during CPR, this document may provide a source of future research hypotheses aimed toward improving monitoring during veterinary CPR.

The monitoring domain is divided into 3 important aspects of veterinary CPR. The first is focused on methods to confirm cardiopulmonary arrest (CPA) and endotracheal intubation. The second section, and the bulk of this domain, evaluates monitoring options during CPR, covering both commonly used monitoring protocols as well as newer options for assessing adequacy of CPR and return of spontaneous circulation (ROSC). The final section of this domain is concerned with suggested monitoring protocols for small animal patients following ROSC. The key monitoring recommendations made in this consensus statement for canine and feline CPR are as follows:

- Time spent verifying an absent pulse may delay onset of CPR; chest compressions should be initiated immediately for apneic, unresponsive patients.
- ECG analysis of an unresponsive patient may help to rule out CPA, or be used to evaluate for rhythms requiring specific therapeutic approaches (eg, ventricular fibrillation [VF]).
- End-tidal CO_2 ($EtCO_2$) should not be used as the sole confirmation of endotracheal intubation in cardiac arrest patients.
- Pauses in chest compressions to evaluate the ECG rhythm should be minimized.
- $EtCO_2$ monitoring is useful to identify ROSC, and may be prognostic for the likelihood of ROSC.
- Patient monitoring following ROSC should be directed at identifying abnormalities that may portend another CPA, and should be individually tailored to each patient.

Although not all of the initial relevant questions in this domain could be addressed and remain material for future initiatives, the prioritized worksheet topics provide excellent guidance for veterinary practitioners involved in treating patients with CPA and represent a major step for direction of further veterinary CPR research.

Summary of the Evidence

Diagnosis of CPA and confirmation of endotracheal intubation

The first part of the monitoring domain investigates the diagnosis of CPA, and specifically addresses the criteria to diagnose CPA in otherwise unresponsive, apneic patients prior to starting CPR. The modalities investigated range from relatively simple procedures such as palpation of a femoral pulse to the utility of more advanced monitoring that may be more relevant for those animals that experience CPA during an anesthetic event, where monitoring equipment is already in place. These questions touch on the conundrum that it is easier to rapidly diagnose life than death, and have been evaluated with the thought that the faster an accurate diagnosis of CPA can be made, the sooner CPR may be initiated. Surprisingly, there is little information available in the literature addressing the consequences of performing CPR on patients who have not actually experienced CPA. Worksheets evaluated femoral pulse palpation (MON03), electrocardiography (MON05), absence of pulse identified by a Doppler ultrasonic flow probe (MON04), and $EtCO_2$ monitoring (MON02) for diagnosis of nonperfusing cardiac rhythms or CPA.

The value of pulse palpation for diagnosing CPA (MON03)

PICO Question

In dogs and cats with suspected cardiac arrest (P), is the palpation of femoral pulses (I) versus assessment for other signs of life (eg, pupil size, agonal breathing, thoracic auscultation) (C) a reliable tool for diagnosis of cardiac arrest (O)?

Conclusion

Evidence from many human studies (level of evidence [LOE] 6) shows that lay rescuers and healthcare professionals are often unable to accurately and swiftly determine the presence or absence of a pulse. Palpation for a lack of a femoral pulse in dogs and cats is not a reliable diagnostic tool for cardiac arrest when used in the absence of corroborating evidence such as apnea, agonal breathing, or lack of an auscultable heartbeat. Moreover, the time needed to confirm the lack of a pulse may delay the initiation of CPR.

Summary of the evidence

No clinical veterinary studies or experimental animal studies have been conducted to investigate the reliability of pulse palpation for diagnosis of cardiac arrest in dogs or cats. Multiple, well-designed, prospective, randomized human studies (LOE 6) have shown both diagnostic inaccuracy and delayed diagnosis of the presence or absence of a pulse in adult and pediatric simulated cardiac arrest.[1–9] Studies used mannequins, healthy volunteers, or patients on extracorporeal circulation with pulsatile or nonpulsatile flow. The sensitivity and specificity of pulse palpation varied between studies, but was generally in the region of 0.9 (sensitivity) and 0.65 (specificity). One study (LOE 6) separately analyzed the data from pulseless patients, and found that only 2% of participants correctly recognized a pulseless patient within 10 seconds, suggesting that sensitivity may be much lower in the absence of a pulse.[3] Another study found that experienced doctors and nurses working in acute care areas were able to detect a pulse more quickly and accurately than those working in nonacute areas, but confirmation of the absence of a pulse still required longer than 10 seconds.[8] Patient positioning may have an effect on the ability of a rescuer to palpate a carotid pulse.[10] Human guidelines no longer recommend a pulse check (lay rescuer) or limit the permissible time for pulse check to less than 10 seconds (healthcare provider) prior to starting resuscitation efforts in unresponsive human patients.[11]

Human anatomy and the usual site of pulse palpation are, however, different from those in cats and dogs. Most studies investigated palpation of the carotid pulse, although the site was variable. In hypotensive infants, however, palpation of the femoral pulse was more rapid and accurate than palpation of the carotid or brachial pulses for confirmation of a heartbeat.[12] There are no studies investigating palpation of the femoral pulse during cardiac arrest in any species.

Whereas there is good human evidence opposing pulse palpation for diagnosis of cardiac arrest, extrapolation to veterinary medicine should be cautious, given the differences in anatomy, site of pulse palpation, arrest setting, and the training and specialization of veterinarians.

Knowledge gaps

There is no information in the veterinary literature that evaluates the accuracy of pulse detection using any specific pulse location, whether performed by lay people or those involved in veterinary care. Ideally, these studies should be performed in animals with normo-, hyper-, and hypotension, as well as in patients who have experienced a cardiac arrest. In addition to assessment of femoral pulses, palpation of metatarsal pulses and the apex beat should be evaluated.

The utility of peripheral Doppler pulse evaluation for identification of CPA (MON04)

PICO Question

In dogs and cats with suspected cardiac arrest (P), is the evaluation of a Doppler pulse (I) versus assessment for other signs of life (eg, pupil size, agonal breathing, femoral pulse) (C) a reliable tool for diagnosis of cardiac arrest?(O)

Conclusion

Loss of femoral pulse and radial artery Doppler signal may occur before complete cardiac arrest. In addition, CPA events are often not predictable such that a Doppler can be placed in advance, and valuable time may be spent attempting to locate an absent pulse with the Doppler probe. Doppler pulse sounds are not a reliable tool for the diagnosis of cardiac arrest, although their disappearance may indicate impending arrest in animals where the Doppler probe was placed prior to CPA.

Summary of the evidence

In an experimental canine model (LOE 3) designed to detect pseudo-PEA (pulselsess electrical activity), femoral pulses and a radial artery Doppler signal were no longer detectable even though an esophageal Doppler signal and thoracic aorta blood movement were still present, and were lost an average of 100 seconds before aortic root fluctuations and esophageal Doppler sounds were no longer detectable.[13] For this reason, loss of radial artery Doppler signal may be a clinically relevant means of detecting critical reduction of peripheral blood flow and impending CPA. In clinical veterinary practice, CPA may not always be predicted in time to place a Doppler probe. In anesthetized patients, the loss of Doppler pulse sounds may be a reasonable indicator of profound hemodynamic deterioration or CPA.

Some human case reports (LOE 6) have described the use of the radial Doppler pulse to monitor for events such as PEA and verification of CPR-related blood flow, although these studies do not address the actual diagnosis of CPA.[14–16]

Knowledge gaps

Based on lack of clinical evidence, controlled animal studies in target species and clinical trials evaluating at-risk patients monitored by Doppler flow probe are

recommended before Doppler pulse sounds can be considered a reliable tool for the diagnosis of CPA.

The value of ECG assessment for confirmation of cardiac arrest (MON05)

PICO Question

In dogs and cats with suspected cardiac arrest (P), is the evaluation of an ECG (I) versus assessment for other signs of life (eg, pupil size, agonal breathing, femoral pulse) (C) a reliable tool for diagnosis of cardiac arrest (O)?

Conclusion

Some ECG rhythms (eg, PEA) can easily be mistaken for perfusing rhythms, emphasizing the importance of accurate physical examination or additional monitoring modalities for the diagnosis of CPA. In addition to assessment for signs of life alone, ECG analysis enables identification of rhythms that can be treated with defibrillation (eg, VF). In cases where the ECG rhythm is consistent with CPA (eg, asystole or VF), the use of ECG may help to clarify the status of a patient with clinical signs of CPA. In patients with syncope or collapse but not CPA, ECG may assist in ruling out CPA.

Summary of the evidence

There is little evidence in the veterinary literature that directly addresses the value of evaluating the ECG versus other signs of life for diagnosing cardiac arrest. Certain cardiac arrhythmias (eg, PEA, pulseless ventricular tachycardia [VT]) that may appear to be consistent with a perfusing rhythm but, in fact, are not, may delay the onset of CPR while additional verification of CPA is obtained. Since CPA is a clinical diagnosis, it is essential that the ECG is not regarded as the sole indicator of life or of a perfusing cardiac rhythm. Given the difficulty in diagnosing CPA using pulse palpation alone (see MON03), ECG analysis may help to more rapidly alert the caregiver to CPA. In the anesthetized patient (LOE 2 and 4), where physical signs of CPA such as unconsciousness and apnea are not available, and where the animal is likely monitored with an ECG prior to CPA, ECG alterations may prove helpful as a supporting diagnostic tool for confirmation of CPA.[17,18]

In cases of animals with syncope or collapse not associated with CPA, ECG findings may aid in diagnosis. One LOE 4 study described the use of an implantable ECG loop recorder in dogs with syncope, collapse, or intermittent weakness and identified a variety of cardiac rhythms such as slow ventricular escape and supra-VT.[19] Cardiac activity that is premonitory of sudden cardiac arrest (tachycardia, bradycardia, ventricular ectopy) has been documented in the human literature via ambulatory electrocardiography, thus monitoring the ECG may allow the veterinary team to anticipate CPA in susceptible patients (LOE 6).[20]

Knowledge gaps

Continued investigation into veterinary CPA ECG rhythms is indicated, as well as studies to evaluate if rapid ECG analysis (eg, as generated by "quicklook" defibrillator paddles) can augment CPA diagnosis.

The value of $EtCO_2$ for diagnosing CPA (MON02)

PICO Question

In dogs and cats with suspected cardiac arrest (P), is the evaluation of $EtCO_2$ (I) versus assessment for other signs of life (eg, pupil size, agonal breathing, femoral pulse) (C) a reliable tool for diagnosis of cardiac arrest (O)?

Conclusion

The correlation of cardiac output and $EtCO_2$ during CPA has been demonstrated in animal models and human clinical studies. At constant ventilation, a rapid decline in $EtCO_2$ value is expected during the evolution of CPA such that a sudden decrease of $EtCO_2$ to near zero in intubated, consistently ventilated animals is a strong indicator of CPA. In nonintubated patients, different causes of CPA (asphyxial versus primary cardiac), may alter the $EtCO_2$ level detected immediately following intubation, with the $EtCO_2$ frequently normal or elevated in asphyxial CPA. Consequently, the initial $EtCO_2$ value cannot be used alone to diagnose CPA. Subsequent $EtCO_2$ values, however, will rapidly decrease after the first few delivered breaths unmasking the presence of CPA.

Summary of the evidence

The correlation of $EtCO_2$ with cardiac output has been shown in many animal and human studies (LOE 3 and 6),[21–28] and may be a better reflection of cardiac output than arterial blood pressure (LOE 3).[29] In a canine model of cardiac arrest (LOE 3), $EtCO_2$ was documented to quantitatively reflect cardiac output when minute ventilation was held constant.[21] At the onset of cardiac arrest, $EtCO_2$ fell immediately to near zero. During cardiopulmonary resuscitation, $EtCO_2$ increased with onset of chest compressions. A sudden, large rise of $EtCO_2$ occurs at ROSC (LOE 3 and 6).[21,30,31] Although $EtCO_2$ is also affected by ventilation parameters, the linear relationship of $EtCO_2$ and cardiac output was consistently observed in porcine CPA models (LOE 6).[23,32–37] This

has also been demonstrated in a number of human case studies (LOE 6), in which $EtCO_2$ also inversely correlated to systemic oxygen extraction ratio, which served as an indicator of tissue oxygen delivery.[22,38–40] Additionally, $EtCO_2$ correlates with coronary perfusion pressure (CPP) during CPR (LOE 3).[41,42] Therefore, $EtCO_2$ may be used as an index of cardiac output and aid in detecting circulatory arrest.

There are important limitations to the utility of $EtCO_2$ to diagnose CPA, however. $EtCO_2$ immediately following intubation varies depending on whether CPA is of asphyxial or primary cardiac origin. In a canine asphyxial arrest model (LOE 3), the initial mean $EtCO_2$ (35.0 ± 15 mm Hg) was higher than the prearrest mean $EtCO_2$ level (31.9 ± 4.3 mm Hg).[43,44] Subsequent $EtCO_2$ values in this cohort were lower during cardiopulmonary resuscitation (mean $EtCO_2$ of 12.4 ± 3.5 mm Hg during CPR) but rapidly increased to a mean $EtCO_2$ of 27.0 ± 7.2 mm Hg immediately prior to or at ROSC.[44] These findings were mirrored in a study of human patients (LOE 6) comparing subjects with asphyxial cardiac arrest (mean initial $EtCO_2$ of 66.4 ± 17.3 mm Hg) with those with cardiogenic cardiac arrest (mean initial $EtCO_2$ of 16.5 ± 9.2 mm Hg).[45] Studies of porcine CPA models (LOE 6) further confirmed these observations.[46] Other conditions may lead to a sudden decline in $EtCO_2$ values in the absence of CPA. For example, the presence and severity of pulmonary embolism has been correlated to a decrease in $EtCO_2$ in dogs undergoing total hip replacement surgery (LOE 2)[47] and in experimental rodent cardiac arrest models (LOE 6).[48]

$EtCO_2$ is a useful index of pulmonary blood flow and cardiac output in endotracheally intubated animals receiving constant ventilation. In conjunction with physical examination findings, it may aid in early detection of CPA in these animals. In animals endotracheally intubated and ventilated after CPA (or as part of CPR), the initial $EtCO_2$ value may not reliably correlate with cardiac output, particularly in the setting of asphyxial arrest. Therefore, best evidence suggests that $EtCO_2$ cannot be used as the sole modality to diagnose CPA.

Knowledge gaps

Although at least 1 veterinary CPR study has noted peak $EtCO_2$ during resuscitation, values obtained at the initiation of CPR have not been reported, nor have they been classified by the cause of CPA.[18] Prospective studies should aim to verify experimental findings (eg, the difference between asphyxial and primary cardiac arrest) in clinical veterinary patients, and endeavor to clarify the utility of $EtCO_2$ in early accurate recognition of CPA.

Confirmation of endotracheal intubation (MON06)

A patent airway is essential for supporting oxygenation and ventilation during CPR, and guidelines are necessary to verify endotracheal intubation (as opposed to esophageal intubation) in the context of CPR. The utility of capnometry to confirm endotracheal intubation in humans, dogs, and cats with intact circulation is well established; however, the extent to which the same is true in dogs and cats with CPA is less clear.[49]

PICO Question

In dogs and cats with cardiac or respiratory arrest (P), is the use of $EtCO_2$ monitoring (I) versus observance of chest wall motion (c) a more accurate tool for verification of endotracheal intubation?

Conclusion

Verification of endotracheal tube (ETT) placement in dogs and cats with cardiac or respiratory arrest is best accomplished using a combination of clinical assessment (ie, direct visualization of the ETT between the arytenoid cartilages, auscultation of air movement in both hemithoraces, observation of chest wall motion, or ETT condensation) and secondary confirmatory tools such as $EtCO_2$ monitoring. In patients where a high $EtCO_2$ value is obtained following intubation, endotracheal intubation is likely, due to the low amount of CO_2 in the stomach and esophagus.

Summary of the evidence

Eleven studies in nontarget species (all LOE 6) document concern for the use of $EtCO_2$ monitoring alone (via capnography or colorimetric indicator devices) when verifying proper ETT position during cardiac arrest.[50–60] One human study (LOE 6) documented that auscultation (not chest wall motion) for verification of ETT position was superior to $EtCO_2$ monitoring in cardiac arrest patients.[52] However, another human study (LOE 6) demonstrated no significant difference between the use of capnography and auscultation to confirm ETT placement in patients during CPR.[61] One experimental study (LOE 3) confirmed the ability of $EtCO_2$ to identify esophageal intubation in dogs undergoing CPR.[62]

Although the preponderance of studies verified correct ETT placement using primary clinical assessments (including direct visualization of the ETT between the vocal cords, observation of chest rise, presence of breath sounds on bilateral auscultation of lung fields, absence of breath sounds over the epigastrium, and presence of condensation in the ETT), there are no studies that

directly compared $EtCO_2$ measurement to these clinical assessments.

In patients with respiratory but not cardiac arrest, esophageal intubation is not expected to produce a sustained high $EtCO_2$ value, and in this context, $EtCO_2$ monitoring may be used to verify ETT position. Two human studies (LOE 6) support this conclusion.[51,52] In an additional study (LOE 6), the use of $EtCO_2$ for ETT verification was reliable for confirming ETT placement in patients with respiratory distress or arrest.[58]

In patients with cardiac arrest, a low $EtCO_2$ value may be encountered despite appropriate ETT placement, and may not distinguish endotracheal from inadvertent esophageal intubation. This is supported by the majority of studies assessed (LOE 6).[50,52–54,56–59,63–65] In contrast, 2 small human studies (LOE 6)[66,67] and 1 experimental canine study (LOE 3)[62] obtained a high test sensitivity, supporting the use of $EtCO_2$ monitoring for verifying endotracheal intubation in subjects with CPA. None of the dogs with esophageal intubation achieved an $EtCO_2$ value above 11 mm Hg at any time during CPR, while all endotracheally intubated animals achieved an $EtCO_2$ value of 13 mm Hg or higher.[61]

In summary, there is not sufficient evidence to support the superiority of a single observation or measurement, such as $EtCO_2$, for verification of ETT placement in dogs or cats with CPA. Generally, the sensitivity of $EtCO_2$ monitoring for identification of endotracheal intubation in patients with CPA is low. In small animal patients, visualization of the ETT between the arytenoid cartilages may be less difficult than in humans and would be considered positive confirmation of endotracheal intubation.

Knowledge gaps

Despite physiologic similarities, evidence regarding the evaluation of successful endotracheal intubation using $EtCO_2$ with or without confirmatory physical parameters is absent in both healthy small animal patients and those with cardiovascular collapse or CPA.

Monitoring during CPR

A large part of the monitoring domain focused on recommendations for assessments that should be performed during CPR, as well as for the appropriate application of these techniques. Because these modalities are inextricably linked to other worksheets across the Reassessment Campaign on Veterinary Resuscitation (RECOVER) domain (eg, in order to treat VF, it must first be identified by electrocardiography), some cross-referencing is necessary across the RECOVER report. While MON14 addresses the question of the utility of ECG monitoring during CPR, MON11 and MON12 discuss the application of these techniques, especially in the context of interruptions in chest compressions. The remaining questions regarding monitoring during CPR are focused on improvement of CPR outcome, including the monitoring of physiologic feedback and $EtCO_2$ (MON15, MON23), ventilatory parameters (MON19), general feedback on the quality of CPR mechanics (MON16), blood gases (MON20), and blood electrolytes (MON21). In addition, a final question addressed the utility of ECG waveform analysis as a predictor of successful defibrillation (MON24).

The relevance of ECG monitoring during CPR (MON14)

PICO Question

In dogs and cats in cardiac arrest (P), does the use of ECG monitoring during CPR (I), compared with no ECG monitoring (C), improve outcome (eg, ROSC, survival to discharge) (O)?

Conclusion

Given the available evidence, the ECG should be utilized to identify and treat arrhythmias amenable to defibrillation (pulseless VT and VF) during CPR. As the majority of the veterinary patient population has an initial arrest rhythm of PEA or asystole that may convert to VF during CPR, ECG monitoring beyond documentation of the first identified rhythm may be of benefit.[18] However, ECG monitoring should be weighed against the risk of interrupting chest compressions for ECG analysis (see MON11).

Summary of the evidence

There are no studies that directly investigate the outcome effect of ECG usage during CPR in animals or people. The 2000 American Heart Association CPR guidelines for pediatric advanced life support recommend the use of ECG monitoring for detection of arrhythmias, and subsequent modifications of the guidelines (2005, 2010) make recommendations for treatment of cardiac arrhythmias.[68] Three studies (all LOE 6) demonstrate a better outcome when CPR is performed in adult humans presenting with an initial arrhythmia of VF or pulseless VT both in- and out-of-hospital.[69–71]

Continuous monitoring of an ECG during CPR may be useful to detect conversion to a rhythm that can be treated with defibrillation. The majority of studies in humans have shown higher survival rates for patients with initial nonshockable rhythm (ie, rhythm not amenable to defibrillation) and subsequent conversion to VF/VT

followed by successful defibrillation (LOE 6),[72–74] although 1 study opposed this finding (LOE 6).[75]

Evidence is mixed regarding outcome associated with ECG monitoring during CPR beyond determination of the presenting rhythm and the associated benefit of early defibrillation. The delay or interruption in chest compressions that occur to allow setup and review of the ECG trace during CPR must also be considered (see MON11).

Knowledge gaps

The effect of ECG monitoring on CPR outcome in small animal patients has not been clearly identified. Many veterinary practices have ECG equipment.[76] Definitive study of the frequencies of heart rhythms during CPR and their association with outcome merits further study. Specifically, the role of intraarrest occurrence of VF in patients with nonshockable initial rhythms demands further investigation.

Interruption of chest compressions for rhythm check (MON11)

PICO Question

In dogs and cats undergoing resuscitation for cardiac arrest (P), does the interruption of CPR to check circulation or ECG rhythm (I), as opposed to no interruption of CPR (C), improve outcome (O) (eg, ROSC, survival)?

Conclusion

Although primary VF remains a rare cause of CPA in veterinary patients, evaluation of the ECG is valuable for identification of a shockable rhythm that develops as a consequence of CPR efforts. Despite this, it seems prudent to avoid or minimize interruptions in chest compressions in dogs and cats during CPR efforts.

Summary of the evidence

Interruption of CPR efforts for any reason does not improve outcome (majority LOE 6, 1 LOE 3).[77–85] One study in swine (LOE 6) revealed that continuous chest compression CPR produces greater neurologically normal 24-hour survival than standard CPR.[79] Recommendations (LOE 6) have been made that providers performing chest compressions switch out every 2 minutes to minimize rescuer fatigue, but that they minimize no-flow time during the switch.[82,85]

Significant pauses in chest compressions have been documented as a result of endotracheal intubation, ECG analysis, or other activities associated with airway management (eg, suctioning).[83,84] Another study (LOE 6) of 61 human out-of-hospital cardiac arrests found that chest compressions were provided for an average of only 40% ± 21% of the first 5 minutes during resuscitation by trained rescuers.[80]

Knowledge gaps

Further research is indicated to better describe the occurrence of interruptions in chest compressions in veterinary CPR, and delineate the causes and the effects of interruptions in chest compressions, including specific information on the effect of pauses on ROSC and neurologic outcome.

The effects of hands-off time for rhythm check after defibrillation (MON12)

PICO Question

In dogs and cats with cardiac arrest (P), does the minimization of hands-off time after defibrillation for rhythm check (I), as opposed to standard care (C), improve outcome (O) (eg, ROSC, survival)?

Conclusion

Extrapolation of human research to veterinary patients remains difficult as many human studies investigate the use of equipment such as automatic external defibrillators (AEDs) that have not been used in veterinary CPR. There are not enough data in the human literature to definitively support the hypothesis that minimization of hands-off time after defibrillation for rhythm check improves CPR outcome (defined either as ROSC or survival to discharge). It seems prudent to resume chest compressions as soon as possible after defibrillation and rhythm identification.

Summary of the evidence

Several experimental animal and clinical human studies (1 LOE 3 and 9 LOE 6 studies) have found that minimization of hands-off time after defibrillation improves outcome (ROSC, survival), leading to recommendations that the interval between discontinuation of chest compressions and delivery of a shock should be kept as short as possible.[77,80,85–92] Interruptions following defibrillation may arise from rescuer change out, mandatory delay with AED use, or attempted adherence to CPR guidelines.

It may take up to 2 minutes of mechanical CPR to reestablish adequate CPP following interruptions of CPR (LOE 6).[86] Adequate heart massage before and during defibrillation greatly improved the likelihood of ROSC in 1 study of porcine VF (LOE 6).[86] Four

experimental studies in swine (LOE 6) were neutral regarding the effect on resuscitation success of duration of hands-off time following defibrillation.[79,93–95] One of these studies found that resuscitation success lies more in the quality of chest compressions than hands-off time (LOE 6).[94] One human clinical study identified an association between shallow chest compressions with longer preshock pauses for rhythm analysis and defibrillation failure (LOE 6).[89] It has been documented, however, that short interruption of chest compressions (8 seconds) required for rhythm analysis and defibrillation resulted in adequate postresuscitation myocardial and neurologic function in swine (LOE 6).[93] Of practical interest, in a retrospective study of adult human patients with recurrent VF (LOE 6), VF recurred within 6 seconds of successful defibrillation in only 20% of patients, while recurrent VF had developed in 72% of patients by 60 seconds.[96] This implies that a rhythm check immediately following defibrillation may not result in an accurate determination of defibrillation success, and it may be better practice to continue CPR immediately following the shock, with a short pause for rhythm analysis after completion of a 2-minute cycle of CPR.

Most recently a relationship between early postdefibrillation chest compression and refibrillation rate was established, which could emerge as a potential argument against immediate resumption of chest compressions after defibrillation. In humans with out-of-hospital cardiac arrest, an 8-second (range: 7–9 sec) versus a 30-second (range: 21–39 sec) pause after successful defibrillation before the resumption of chest compressions was compared.[97] While the overall recurrence of VF was the same in both cohorts, VF reoccurred earlier in the patients who received early resumption of chest compressions, potentially resulting in a longer duration of untreated VF until subsequent rhythm check and defibrillation. However, patient outcome was not reported in this study, and the overall relevance to veterinary CPR is unclear.

Knowledge gaps

The impact of pauses in CPR following defibrillation has not been critically evaluated in dogs or cats, and studies evaluating immediate versus delayed rhythm diagnosis may be indicated prior to studies that evaluate different delay times prior to the resumption of CPR. Importantly, having a CPR reporting system and template in place that includes recording of chest compression pauses will be essential to answer some of these questions in veterinary medicine.

The effect of physiological feedback monitoring on quality of CPR (MON15)

PICO Question

For dogs and cats requiring resuscitation (P), does the use of physiological feedback regarding CPR quality (eg, $EtCO_2$ monitoring, blood gas analysis) (I), compared with no feedback (C), improve any outcomes (eg, ROSC, survival) (O)?

Conclusion

No research was identified that directly examined the effect of physiological feedback devices on the outcome of resuscitation. However, there is evidence that documents the value of $EtCO_2$ on prognostication and early identification of ROSC, as well support for its use as surrogate measure of CPP during CPR.

Summary of the evidence

Only 1 veterinary study is available that provides information on prognostic indicators for CPA in dogs and cats undergoing nonexperimental arrests (LOE 2).[18] The mean highest recorded $EtCO_2$ was significantly higher in dogs that achieved ROSC (36.6 $\pm$ 19.7 mm Hg) than those that did not (10.3 $\pm$ 10.2 mm Hg); however, the difference was not significant in cats. Ninety-four percent of the dogs that did not achieve ROSC had maximal $EtCO_2$ values of <15 mm Hg, whereas 86% with values $\geq$15 mm Hg achieved ROSC. In cats, ROSC was achieved in 90% with peak $EtCO_2$ of $\geq$20 mm Hg, but in only 55% with values <20 mm Hg. The value of $EtCO_2$ as predictor of ROSC is a finding echoed by clinical research in humans (LOE 6).[98,99] Two studies have shown that $EtCO_2$ values correlate with CPP during CPR and thus provide real-time physiologic feedback during CPR. Kern et al used an experimental canine VF model (LOE 3) to evaluate $EtCO_2$ as a prognostic guide for resuscitation.[42] A decline in $EtCO_2$ was seen during CPR in the dogs in which ROSC was not obtained, while $EtCO_2$ remained constant in the dogs that were successfully resuscitated. $EtCO_2$ was also significantly correlated to CPP, which was found to be a good predictor of outcome. A porcine VF model of CPA (LOE 6) showed significantly higher $EtCO_2$ and $PaCO_2$ values in surviving animals after 6 minutes of CPR (18 $\pm$ 7 versus 5 $\pm$ 4 mm Hg).[34] In this study, changes in $EtCO_2$ and $PaCO_2$ occurred in parallel and correlated with CPP. When ventilation is held constant, $EtCO_2$ may provide information regarding ROSC. In a pediatric dog model of CPA (LOE 3), the mean $EtCO_2$ during CPR was 12.37 $\pm$ 3.5 mm Hg and suddenly increased to 27 $\pm$ 7.2 mm Hg at or just prior to ROSC.[43]

Knowledge gaps

Objective data on the effects of physiologic feedback on CPR quality and success are lacking in the human and veterinary literature. The effect of real-time feedback (eg, $EtCO_2$) on parameters such as depth of chest compression or CPR success will be important to evaluate in future studies.

The effect of $EtCO_2$ monitoring on ROSC (MON23)

PICO Question

In dogs and cats in cardiac arrest (P), does the use of $EtCO_2$ monitoring during CPR (I), compared with no $EtCO_2$ monitoring (C), improve chances for ROSC (O)?

Conclusion

$EtCO_2$ measurement serves as a noninvasive surrogate measurement of cardiac output and CPP during CPR. $EtCO_2$ is frequently used as a reflection of quality of CPR when comparing approaches or devices. Rescuers can and likely should alter circulatory support methods to optimize $EtCO_2$ values. No study has been performed comparing the use of a capnometer during CPR to CPR without a capnometer. Specific target values for $EtCO_2$ are addressed in MON 22.

Summary of the evidence

The monitoring of $EtCO_2$ during CPR gives information regarding cardiac output and cardiac index (LOE 3 and 6),[32,35,100] CPP (LOE 3 and 6),[41,42,101,102] and mean aortic pressure (LOE 6)[101] during CPR. Studies focusing on the utility of $EtCO_2$ monitoring for predicting CPR outcome (primarily evaluating adult humans with nontraumatic, primary cardiac arrest [LOE 6]), uniformly found that patients with higher $EtCO_2$ over the course of CPR were more likely to achieve ROSC, survival to hospital discharge, or both.[22,32,33,39,45,56,58,98,99,101,103–114] Only a single indexed study (LOE 6) was neutral on the subject of $EtCO_2$ monitoring pertaining to CPR outcome, finding no difference between $EtCO_2$ in CPA patients that could not be resuscitated (13.7 ± 7 mm Hg) and $EtCO_2$ in CPA patients that achieved ROSC (12.9 ± 5 mm Hg).[31]

There is sufficient evidence to support the routine use of capnometry during CPR. Higher $EtCO_2$ values are associated with higher cardiac output and CPPs, and are strongly associated with ROSC in people. Thus, targeting a specific $EtCO_2$ value by optimizing CPR technique is likely to benefit patients and improve the likelihood of ROSC.

Knowledge gaps

Optimal cutoff values for $EtCO_2$ that may improve the likelihood of ROSC when used as a feedback goal have not been determined in veterinary medicine.

The importance of audio-visual feedback on CPR delivery characteristics (MON16)

PICO Question

For dogs and cats in cardiac arrest (P), does the use of feedback regarding the mechanics of CPR quality (eg, rate and depth of compressions and/or ventilations) (I), compared with no feedback (C), improve any outcomes (eg, ROSC, survival) (O)?

Conclusion

There is no direct evidence that feedback about the mechanics of CPR quality affects outcome in dogs and cats, or that current methodology is applicable to veterinary species. Studies in humans do not show a clear benefit of the use of real-time feedback monitoring in terms of outcome improvement, although such technology was shown to improve adherence to CPR guidelines.

Summary of the evidence

Newer defibrillators can record and report on data about the depth, rate, and decompression (recoil) time of chest compressions using accelerometers, force transducers, or induction of ECG artifacts via sternal movements. Ventilation data may be derived from changes in thoracic impedance or from integrated capnography. Real-time feedback from these defibrillators usually consists of verbal messages and/or visual prompts displayed on the monitor.

Most human studies that evaluated the effect of real-time feedback on ROSC were neutral in outcome. One large study (LOE 6) investigated a defibrillator that provided audio-visual prompts regarding adequacy of chest compressions and ventilation in human adults with out-of-hospital cardiac arrest.[115] While the authors found that compliance with CPR guidelines and quality of CPR (eg, number and depth of chest compressions) was improved, the study failed to identify a positive effect on either ROSC or hospital discharge. A smaller study (LOE 6) evaluating in-hospital CPA also failed to demonstrate a difference in ROSC or hospital discharge using a similar defibrillator feedback protocol.[116]

A prospective human study (LOE 6) that evaluated a defibrillator that provided real-time prompts when chest compression parameters (rate, depth, or duty cycle) and ventilation parameters (rate, depth, or inspiration time)

were not in accordance with guidelines failed to show a statistically significant difference in survival, but showed a trend toward a benefit for the use of real-time feedback (hospital discharge 2.9% for control versus 4.3% with feedback).[117] In this study, some CPR variables such as compression depth, percentage of compressions with adequate depth, and respiratory rate were significantly improved with feedback.

A recent study compared the use of limited CPR feedback (metronome and visual graphics of the depth of chest compressions) with more extensive feedback that integrated verbal prompts during CPR.[118] There was no change in CPR quality or rate of ROSC with the addition of the voice prompts.

Knowledge gaps

Randomized prospective studies using metronomic guidance of chest compression rates are feasible in veterinary medicine and merit investigation. Additional studies evaluating the use of accelerometers or other methods to quantify depth and rate of chest compressions may result in data that can be integrated into a real-time feedback system suitable for veterinary CPR. Moreover, such systems would provide an opportunity to record and analyze rescuer performance, and could be the basis for studying CPR quality in clinical settings and for training purposes.

The role of $EtCO_2$ in predicting ROSC (MON22)

PICO Question

In dogs and cats in cardiac arrest (P), does an $EtCO_2$ value above 15 mm Hg during CPR (I), compared with an $EtCO_2$ value below 15 mm Hg (C), predict ROSC (O)?

Conclusion

$EtCO_2$ values are positively correlated with CPP and rise steeply when ROSC occurs. Higher levels appear to predict ROSC while continued low $EtCO_2$ predicts nonsurvival. In humans, an $EtCO_2$ less than 10 mm Hg over a period of several minutes during CPR is a poor prognostic indicator for ROSC. However, studies have shown variability in exact changes in $EtCO_2$ measurements based on the underlying cause of cardiac arrest and medications used during resuscitation. Caution may be warranted when interpreting a rise in $EtCO_2$ after using either sodium bicarbonate or epinephrine during CPR.

Summary of Evidence

Higher $EtCO_2$ values are associated with an increased likelihood of ROSC (LOE 3 and 6).[32,33,38,39,41,99,103,106,119,120] Persistently low $EtCO_2$ during CPR is associated with a poor prognosis (LOE 3 and 6).[42,46,111] In a single study of humans with CPA (LOE 6), survivors to hospital discharge had higher $EtCO_2$ during resuscitation than nonsurvivors.[103]

In a cardiac arrest model in swine (LOE 6), piglets that did not achieve ROSC had $EtCO_2$ readings that remained below 15 mm Hg for the duration of CPR.[46] In an experimental canine study (LOE 3), the $EtCO_2$ increased from 13.9 $\pm$ 4 mm Hg during CPR to 27 $\pm$ 7 mm Hg at or just prior to ROSC.[44] In another experimental study in dogs (LOE 3), 6 of the 12 dogs were successfully resuscitated, and the dogs with ROSC achieved a mean $EtCO_2$ of 9.6 $\pm$ 3.2 mm Hg during CPR versus 3.2 $\pm$ 1.1 mm Hg for dogs that did not achieve ROSC.[41]

Absolute values for $EtCO_2$ that might be predictive of ROSC have been most closely examined in human studies (all LOE 6). Cantineau and colleagues concluded that an $EtCO_2$ value of greater than 10 mm Hg obtained during the first 20 minutes of CPR predicted ROSC with a sensitivity of 100% and specificity of 66%.[106] In 2 other human studies, $EtCO_2$ values consistently less than 10 mm Hg during CPR predicted failure to achieve ROSC.[99,111] Compared to these studies, Asplin and White reported much higher maximum $EtCO_2$ levels in human patients that did not achieve ROSC (22.7 $\pm$ 8.8 mm Hg), although these values were significantly lower than those in survivors (30.8 $\pm$ 9.5 mm Hg).[120] Another small observational study showed that all subjects that achieved ROSC had measured $EtCO_2$ values above 10 mm Hg during CPR, although several of those who did not survive also had $EtCO_2$ values greater than 10 mm Hg.[103]

The administration of sodium bicarbonate during CPR can increase the $EtCO_2$ independent of cardiac output and coronary perfusion.[121] An experimental canine study of VF (LOE 3) described CPPs that were highly correlated to $EtCO_2$ during CPR prior to the administration of epinephrine but not afterward.[122] This was also found in another study (LOE 3), where $EtCO_2$ was not different between nonresuscitated and resuscitated animals.[123] Despite these findings, epinephrine was used during CPR in several human observational clinical trials[111] and in one of the canine research studies[102] without reducing the prognostic value of $EtCO_2$ on ROSC.

Knowledge gaps

While there is evidence that $EtCO_2$ is a reliable indicator of ROSC when it occurs, and that higher peak $EtCO_2$

values during CPR are associated with ROSC in dogs, there are no prospective studies that evaluate the change in $EtCO_2$ values during CPR in clinical veterinary patients, or definitively identify a cut-off point or goal for measured $EtCO_2$ during a CPR episode. These data also need to be reevaluated in cats.

The importance of monitoring ventilation during CPR (MON19)

PICO Question

For dogs and cats requiring resuscitation (P), does the monitoring and control of ventilatory parameters (eg, ventilation rate, minute ventilation, and/or peak pressures) (I), as opposed to standard care (without ventilatory monitoring) (C), improve outcome (O) (eg, ROSC, survival)?

Conclusion

Alterations in ventilation parameters (ventilation rate, compression:ventilation ratio, end expiratory pressure) may have significant effects on ROSC, survival, and hemodynamics; however, strong evidence on the effect of monitoring ventilatory parameters on outcome is lacking and research in small animal patients is absent. High ventilation rates (greater than 10–12 breaths/min) during CPR should be avoided, as time spent without cardiac compressions in single-rescuer scenarios and increased time with positive intrathoracic pressure will have a negative effect on hemodynamics. Optimal ventilation rates are unknown.

Excessive ventilation during CPR commonly occurs, even with trained personnel, and retraining results in minimal improvement. Because of this fact, and because traditional tidal volume (V_t; eg, 10 mL/kg) may not be necessary during CPR, the monitoring and control of ventilatory parameters may be beneficial in small animal patients during CPR.

Summary of the evidence

Only 1 LOE 3 study was identified,[124] and this, in addition to several LOE 6 studies,[78,125,126] was focused on evaluation of compression:ventilation ratios, providing limited information on other ventilation parameters.

One study in swine (LOE 6) compared animals receiving a V_t of 12.5 ± 0.6 mL/kg to those receiving 50% of that V_t during CPR, and did not demonstrate a difference in ROSC between groups.[127] Lower tidal volumes will reduce peak airway pressure during CPR (LOE 6), and measurement of V_t may lead to more focused recommendations for small animal CPR.[128]

Administration of high ventilation rates is a common occurrence during CPR (LOE 6) and can result in persistent positive intrathoracic pressure and a significant decrease in CPP.[128,129] Hyperventilation can result in decreased ROSC (LOE 6), with 1 study showing a 69% decrease in the rate of ROSC between pigs ventilated at 30 breaths/min compared to 12 breaths/min.[129]

The measurement and provision of positive end-expiratory pressure (PEEP) may be indicated, and 1 study in rats suggested that 5 cm H_2O PEEP during CPR may result in greater survival rates compared with zero end expiratory pressure (LOE 6), although this technique also increases intrathoracic pressure.[130]

Knowledge gaps

Data regarding respiratory rates and compression:ventilation ratios should be confirmed in small animal patients, in addition to further studies evaluating the effects of different V_t and PEEP. Moreover, pulmonary gas exchange and respiratory mechanics during CPR are not well understood and deserve further exploration.

Monitoring arterial and venous blood gases during CPR (MON20)

PICO Question

In dogs and cats with cardiac arrest (P), does the measurement of arterial blood gases during resuscitation (I) versus measuring venous blood gases (C) improve the chances of ROSC (O)?

Conclusion

Venous blood gas values have better predictive value for ROSC than arterial blood gas values and are directly related to physiological properties such as cardiac output and tissue perfusion.

Summary of the evidence

Blood gas analysis (arterial or venous) during CPR is controversial due to the clinical paradox that results in relatively normal blood gas values early in CPA despite significant hypoperfusion of tissues (LOE 6).[131] An evaluation of the association between arteriovenous partial pressure of CO_2 (PCO_2) difference and cardiac output during CPR in dogs (LOE 3) found that mixed venous PCO_2 and pH was better associated with low cardiac output states than arterial values.[132] A laboratory evaluation of arterial and venous blood gas, pH, and lactate changes in dogs during CPR (LOE 3) found that mixed venous blood gas values changed to a greater degree during the resuscitation period than did the arterial samples.[133] A

significant arteriovenous difference in pH, PCO_2, and PO_2 was found during CPR in this group of dogs, and supported the premise that venous values more accurately reflect tissue acid-base balance than arterial values. A swine CPA model (LOE 6) showed similar results.[131] Venous pH also was found to have a greater predictive value for defibrillation outcome in dogs compared to arterial pH (LOE 3),[134] and in humans with CPA secondary to severe hypothermia (LOE 6).[135]

The value of arterial blood gases is less clear. A study evaluating arterial blood gases in dogs undergoing various resuscitation techniques (LOE 3) found that surviving dogs had lower arterial pH and higher $PaCO_2$ during CPR compared to dogs that did not survive.[123] However, a clinical study in humans (LOE 6) showed the opposite, that patients with CPA who did not experience ROSC had significantly lower arterial pH and higher $PaCO_2$ than patients that achieved ROSC.[136] Due to the delay between initiation of CPR and blood sampling, this study primarily confirms that prolonged and worsening elevations of $PaCO_2$ are associated with lower rates of ROSC in human CPR. In addition, another canine model of CPR (LOE 3) did not find significant associations between arterial blood gas values and ROSC.[137] The same was true in cats (LOE 3).[138] Thus, the value of arterial blood gas abnormalities for quality assessment or prognostication of CPR success remains unclear. Even after ROSC, arterial base excess was not an independent predictor of mortality in resuscitated patients, but merely correlated with duration of CPR (LOE 6).[139]

Knowledge gaps

Changes in blood gas parameters in clinical small animal patients undergoing CPR have not been investigated and the value of blood gas analysis during CPR has not been examined. Such studies are necessary. In addition, acquisition of more experimental data, for example, in asphyxial cardiac arrest models, is warranted prior to recommending the measurement of venous blood gas data in clinical patients.

Monitoring electrolytes during CPR (MON21)

PICO Question

In dogs and cats with cardiac arrest (P), does the measurement of arterial or venous electrolytes (Na^+, K^+, Ca^{2+}) during resuscitation (I) versus not measuring electrolytes (C) improve the chances of ROSC (O)?

Conclusion

In cases where the cause of cardiac arrest may be due to electrolyte abnormalities (eg, hyperkalemia), the measurement of serum electrolytes may allow directed therapy in addition to standard CPR. Ionized hypocalcemia may be prognostic for ROSC. The imperative for therapy of other electrolyte abnormalities that occur during CPR is less clear. The measurement of electrolytes in certain cases may identify abnormalities that, if corrected, may promote ROSC.

Summary of the evidence

Ionized hypocalcemia can occur during prolonged cardiac arrest and resuscitation in dogs (LOE 3).[140] Another laboratory investigation of canine CPR (LOE 3) found that arterial ionized calcium decreased within 5 minutes of starting CPR and continued to decrease in dogs that could not be resuscitated.[137] Other studies of ionized calcium levels during canine CPR (LOE 3) did not identify ionized hypocalcemia (LOE 3).[141]

Potassium levels can increase during CPR, and marked hyperkalemia (>8.0 mmol/L) was associated with lack of ROSC in both dogs (LOE 3)[137] and pigs (LOE 6).[142] A small human study evaluating the efficacy of hemodialysis during CPR for treatment of severe hyperkalemia found that ROSC was achieved in all 3 patients once the mixed venous potassium was decreased below 8.0 mmol/L (LOE 6), which suggests that treatment of hyperkalemia during CPR may improve resuscitative efforts.[143] In the special circumstance of hypothermia-associated CPA, severe hyperkalemia may indicate that cardiac arrest occurred prior to cooling (LOE 6), hypothermic cardiac arrest is prolonged (LOE 3), and advanced and prolonged resuscitative efforts are less likely to be fruitful.[135,144]

Knowledge gaps

The occurrence of electrolyte abnormalities over the course of CPA and CPR has not been described in clinical veterinary medicine, nor have the effects of electrolyte-directed therapy been tested. Efforts should be undertaken to identify and investigate subsets of patients with these abnormalities to further define clinical approaches.

Prediction of defibrillation success from VF waveform (MON24)

PICO Question

In dogs and cats with cardiac arrest (P), does the use of a technique for prediction of the likelihood of success of defibrillation (analysis of VF, etc) (I), compared with standard resuscitation (without such prediction) (C), improve outcomes (eg, successful defibrillation, ROSC, survival) (O)?

Conclusion

Based on laboratory investigations in dogs and swine, coarse VF is more likely to respond with ROSC following defibrillation than fine VF. Waveform analysis that can predict defibrillation success, however, remains a topic of investigation. Due to the small number of randomized clinical trials in human medicine, and the complete lack of clinical trials in veterinary medicine, it is uncertain whether these techniques will improve clinical outcomes in dogs and cats with CPA.

Summary of the evidence

The gold standard in determining the likelihood of response to defibrillation is measurement of the CPP.[145,146] Even in human medicine, the difficulties associated with arterial and central venous cannulation during CPR has led to investigations of other means for prediction of defibrillation success.

Newer techniques focus on ECG waveform analysis. These techniques investigate the VF wavelets for components that indicate a high chance of successful defibrillation if a counter shock is administered. The VF waveform changes from a higher amplitude wavelet (coarse VF) during the initial phase of untreated VF (less than 3-4 minutes of duration) to a lower amplitude wavelet (fine VF) with the progression in time (LOE 6).[147] As the amplitude and structure of the VF waveform decreases, there is a decreased likelihood of ROSC and survival (LOE 3 and 6).[147,148] Waveform analysis can also estimate the duration of VF, which guide timing of electrical defibrillation.

CPR generates significant ECG artifacts that complicate VF waveform analysis. Techniques (all LOE 6) including amplitude spectrum area transformation (AMSA)[149–152] and scaling exponents[153] have been applied to summarize the ECG waveform. Due to the complexity of these methods, their clinical utility has been restricted to more sophisticated AED's. AMSA analysis has been used to identify suboptimal chest compressions, and may be factored into AED-based prompts for bystander CPR (LOE 6).[152]

Human studies to date have focused on the use of waveform analysis to determine the duration of VF, and to investigate the subsequent question of whether a short period of CPR prior to counter shock is beneficial in patients with certain durations of VF. Randomized clinical trials in humans (LOE 6) to determine if waveform analysis and CPR prior to counter shock improved outcomes have not shown any survival benefit to a short period of CPR prior to counter shock.[154,155] However, one of the studies found a potential survival benefit for pre-shock CPR in patients with prolonged (>5 minutes) VF.[154]

Knowledge gaps

Waveform analysis has not yet been evaluated in veterinary medicine. However, more sophisticated defibrillators with data acquisition and review capability will allow for ECG waveform analysis to fill that gap.

The role of $EtCO_2$ in identification of ROSC (MON10)

PICO Question

In dogs and cats in cardiac arrest (P), does the use of $EtCO_2$ (I), compared with clinical assessment (C), improve accuracy of diagnosis of a perfusing rhythm (O)?

Conclusion

Only 1 study described an association between $EtCO_2$ and clinical assessment (auscultable heartbeat) during CPR and suggested in a neonatal pig model that an increase in $EtCO_2$ over 14 mm Hg should prompt evaluation for return of a heartbeat. Many clinical and experimental studies describe a marked increase in $EtCO_2$ upon ROSC. Given the clinical difficulty that may be associated with auscultation during CPR, the presumed negative effects of CPR interruptions for auscultation or ECG review, and the difficulties differentiating PEA from a perfusing rhythm, $EtCO_2$ monitoring may improve diagnosis of ROSC.

Summary of the evidence

Chalak et al examined the association between $EtCO_2$ readings and physical examination parameters in a piglet CPR study (LOE 6).[156] The objective was to determine the $EtCO_2$ level that best predicted the return of an audible heart beat, for purposes of minimizing interruption of chest compressions during CPR. The authors studied 46 anesthetized piglets in an asphyxiation model of CPA. The piglets underwent CPR with continuous $EtCO_2$ monitoring, while auscultation was performed every 30 seconds. An $EtCO_2$ value of 14 mm Hg had a sensitivity of 93% and a specificity of 81% for prediction of the presence of an audible heartbeat. Bhende et al reported a sharp increase in $EtCO_2$ values at the onset of ROSC in an experimental canine study (LOE 3), but did not compare this to clinical assessment.[44]

Knowledge gaps

There is a paucity of studies evaluating the use of $EtCO_2$ monitoring for prediction of ROSC in veterinary medicine, with much of the available data consisting of experimental studies directed toward human CPA and CPR (ie, the use of asphyxiation or VF models), rather

than naturally occurring CPA and clinical CPR in animals. More research evaluating $EtCO_2$ monitoring in conjunction with the clinical assessment of patients and the use of other monitors may help to define and improve the prompt diagnosis of perfusing rhythms in veterinary CPR.

Monitoring following ROSC

After spontaneous circulation is reestablished, physiologic monitoring devices to which clinicians are accustomed, such as pulse oximeters and noninvasive blood pressure monitors, regain their functionality. The postresuscitation population is extremely heterogeneous due to variability in duration and conditions of arrest, precipitating diseases, and other factors. Moreover, each time phase after ROSC demands different monitoring characteristics as the goals of therapy shift from prevention of rearrest immediately after ROSC to treatment of precipitating disease and the consequences of anoxic brain injury, cardiac dysfunction, and global reperfusion injury once stable ROSC is established.[157] These monitoring devices may also provide valuable prognostic information. Much of the postcardiac arrest monitoring follows principles customary to veterinary critical care. Additional specific postcardiac arrest monitoring considerations are the focus of MON 25.

The relevance of intensive continuous postcardiac arrest monitoring (MON25)

PICO Question

For dogs and cats with ROSC after cardiac arrest (P), does the use of intensive continuous monitoring (eg, continuous ECG, blood pressure, temperature, pulse oximeter, $\pm EtCO_2$) (I) versus standard intermittent monitoring (C) improve outcome (eg, survival)?

Conclusion

Veterinary studies evaluating monitoring as it relates to outcome following ROSC are lacking, and therefore any recommendations for intermittent or continuous monitoring following ROSC must be drawn from the human literature. Recommendations for monitoring after ROSC in the human literature tend to be extrapolated from studies in other shock states or based on expert opinion.

Although not supported by prospective research, there is a current trend toward early goal directed therapy (EGDT) in postcardiac arrest patients following ROSC. EGDT aims to establish early hemodynamic stability and adequate oxygen delivery parameters, a logical goal in patients following ROSC. Endpoints for resuscitation have yet to be determined in dogs and cats. Evidence from the human literature suggests that episodes of hypotension following ROSC are associated with a worse outcome, so it is reasonable to measure blood pressure continuously whenever possible to recognize and prevent hypotension. Given the occurrence of myocardial dysfunction in humans and animals (even in the absence of coronary artery occlusion) following ROSC, it is also reasonable to serially monitor veterinary CPA patients with continuous ECG and echocardiography.

Continuous oximetry may be useful to guide titration of inspired oxygen concentrations to prevent both hypoxemia and hyperoxia. The actual targets for arterial oxygenation are unclear. Complications such as pulmonary edema, pneumonia, and pulmonary contusions are not uncommon following CPR and veterinary patients should be monitored for hypoxemia by pulse oximetry and/or arterial blood gases. There is evidence that the $PaCO_2$-$EtCO_2$ difference may be associated with outcome in people following ROSC and measuring arterial blood gases and $EtCO_2$ would be reasonable in ventilated veterinary patients following ROSC.

Glucose should be monitored serially to detect hypoglycemia and hyperglycemia. It seems prudent to avoid iatrogenic hyper- or hypoglycemia, and preliminary evidence in people (LOE 6) suggests that the severity of hyperglycemia in the postcardiac arrest phase is correlated with a worse outcome. Induced mild therapeutic hypothermia in humans that remain comatose after ROSC is rapidly becoming standard of care. Worse outcome has been described in people that have episodes of hyperthermia. Similarly, the severity of accidental hypothermia present at ROSC was associated with worse outcome. Serial monitoring of rectal or core temperature in veterinary patients after ROSC is reasonable. Seizures are not uncommon in people following ROSC and may be associated with a worse outcome. It is therefore reasonable to include monitoring for seizures and changes in neurologic status in veterinary patients with ROSC.

There is evidence in support of serial monitoring of ECG, arterial oxygenation, body temperature, blood glucose, and systemic blood pressure following ROSC, in addition to serial physical exams and neurologic monitoring. There is no clear evidence to delineate between recommendations for continuous monitoring versus intermittent monitoring of these variables, and this should be tailored to individual patients and circumstances, especially when determining the intervals for intermittent monitoring.

Summary of the evidence

After achieving ROSC, patients can enter a systemic inflammatory state not unlike SIRS or sepsis.[158,159] Early

optimization of systemic oxygen delivery has been subsequently applied to postcardiac arrest patients (LOE 6), with recommendations for monitoring arterial blood pressure, central venous pressure, serum lactate, arterial and central venous oxygen saturation, and cardiac output.[158,160,161] However, these studies were not designed to examine the value of individual monitoring elements or interventions, but rather the impact of the entire treatment package. There is very limited direct evidence in the human or veterinary literature to support or refute continuous or intermittent monitoring in the postcardiac arrest phase.

Several indications exist for the use of continuous or intermittent ECG following ROSC. Arrhythmias are commonly encountered in humans following ROSC (LOE 6) such that continuous ECG monitoring is included in most human postcardiac arrest monitoring guidelines.[162–164] In addition to identifying cardiac arrhythmias, continuous ECG monitoring has been used to assess postresuscitation heart rate variability, a metric suggestive of autonomic nervous system function (LOE 6).[159,163]

Manipulation of mean arterial blood pressure (MAP) following ROSC is an area that has received considerable attention in the human literature, although specific MAP goals have not been determined. Recent research (LOE 6) has demonstrated a worse outcome with the occurrence of episodes of hypotension in the first 6 hours after ROSC.[160,165,166] However, another study (LOE 6) evaluating blood pressure over a longer time period (24 hours) failed to show such an association.[167] MAP target values have varied among studies and ranged from 65 to 100 mm Hg. The optimal arterial blood pressure following ROSC has not been conclusively determined. Three human studies reported that early lactate clearance after resuscitation from cardiac arrest was associated with reduced mortality (LOE 6).[160,161,168] Reversible myocardial dysfunction, also known as myocardial stunning, has been documented in animals and people following ROSC and has been associated with lower survival rates in 1 clinical study (LOE 6).[169] A prospective study including 87 people that were successfully resuscitaed from in-hospital cardiac arrest found a 25% reduction in left ventricular ejection fraction in the postcardiac arrest phase when compared to prearrest values (LOE 6).[170] Left ventricular dysfunction was assessed with transthoracic echocardiography, which was shown to also be useful in guiding postcardiac arrest fluid therapy.[171]

A prospective observational study (LOE 6) evaluated $PaCO_2$-$EtCO_2$ difference and alveolar dead space ventilation ratio (V_d/V_t, where V_d is alveolar dead space volume) in mechanically ventilated patients 1 hour following ROSC.[160] A significant difference was found between survivors and nonsurvivors, with nonsurvivors having significantly elevated V_d/V_t and $PaCO_2$-$EtCO_2$ differences compared to survivors.

Oxygen therapy continues to be recommended in human CPR guidelines during resuscitation, as well as during the post-cardiac arrest care phase. However, hyperoxemia has been associated with brain injury, worse neurologic outcomes, and increased mortality in swine (LOE 6), dogs (LOE 3), and people (LOE 6).[165,172,173] Oximetry-guided reoxygenation following ROSC has been associated with improved neurological outcomes in dogs (LOE 3).[173] This prospective randomized experimental study in 17 dogs, using continuous pulse oximetry followed by blood gas analysis, demonstrated improved neurologic function and less histologic brain injury in the animals with oxygen administration titrated to an SpO_2 of 94% to 96% within 12 minutes of ROSC, when compared to dogs maintained on 100% O_2.

Seizures are common during the postcardiac arrest phase in humans, and human clinical studies suggest that patients experiencing seizures in the first 24 hours following ROSC have a worse long-term neurologic outcome (LOE 6).[162,174]

There is strong experimental and human clinical evidence that induced hypothermia following ROSC results in favorable outcomes and which suggests worse outcomes following hyperthermia in postcardiac arrest patients.[174–180] Interestingly, an episode of passive hypothermia following ROSC resulted in a decreased odds of survival to hospital discharge (LOE 6).[176]

Optimum blood glucose values in people have not been determined for patients with ROSC. Hyperglycemia occurring in the first 24–72 hours after ROSC was associated with a worse outcome (LOE 6).[162,174,181,182] A retrospective study in a human postcardiac arrest population (LOE 6) showed that patients with moderate-to-severe hyperglycemia did poorly; however, those with mild increases in glucose had a similar outcome as those with normal glucose values.[183]

Knowledge gaps

In 1 veterinary study, 54% of animals who achieved ROSC succumbed to another episode of CPA, highlighting the importance of postcardiac care and monitoring.[18] Studies are required to evaluate the need for strict control of body temperature, blood glucose, arterial oxygenation, and hemodynamic metrics, such as MAP, lactate, and central venous oxygen saturation in dogs and cats after cardiac arrest.

Discussion

Taken together, the evidence evaluated and presented here suggests that the monitoring of end-tidal

capnography may be useful for evaluation of the strength of, quality of, and physiologic response to chest compressions during CPR, for indicating ROSC, and possibly as a prognostic factor in animals with CPA. On the basis of this wide utility, it is recommended that an easily accessible $EtCO_2$ monitor be available in any situation where CPA is likely to be encountered (eg, in emergency rooms, as a component of a crash station, or in areas where patients are anesthetized). More studies are needed to evaluate the diagnostic, prognostic, and monitoring utility of $EtCO_2$ measurement in small animal CPR, but wider distribution of this technology will also benefit future studies.

One aspect of CPA diagnosis that was not addressed in the (primarily) human literature was the efficacy of apex beat palpation, as opposed to a peripheral pulse. Given the anatomical contours of small animal veterinary patients, palpation for an apex beat may even be possible as one is preparing to initiate CPR. As noted in MON03, it is easier to diagnose a heartbeat than a lack of a heartbeat, and this palpation will always require a delay prior to the institution of chest compressions. In the context of multiperson CPR, it may be possible to palpate the apex beat while stabilizing the patient for endotracheal intubation or ECG analysis (ie, interventions that are more difficult to perform while chest compressions are ongoing), but the bulk of the evidence still indicates that rapid institution of chest compressions is indicated and should be prioritized in any unresponsive animal.

As with all parts in the RECOVER initiative, the monitoring domain has highlighted significant knowledge gaps in the practice of veterinary CPR, thereby providing clear targets for researchers. At the same time, this domain summarizes available evidence as a basis for clinical guidelines for veterinary practitioners.

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Appendix

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