Received: 22 March 2024 | Accepted: 25 April 2024

DOI: 10.1111/vec.13390

SPECIAL ARTICLE

Journal of Veterinary Emergency and Critical Care | WILEY

2024 RECOVER Guidelines: Monitoring. Evidence and knowledge gap analysis with treatment recommendations for small animal CPR

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Funding information
Boehringer Ingelheim Animal Health; Zoetis Animal Health

Abstract

Objective: To systematically review evidence on and devise treatment recommendations for patient monitoring before, during, and following CPR in dogs and cats, and to identify critical knowledge gaps.

Design: Standardized, systematic evaluation of literature pertinent to peri-CPR monitoring following Grading of Recommendations, Assessment, Development, and Evaluation (GRADE) methodology. Prioritized questions were each reviewed by Evidence Evaluators, and findings were reconciled by Monitoring Domain Chairs and Reassessment Campaign on Veterinary Resuscitation (RECOVER) Co-Chairs to arrive at treatment recommendations commensurate to quality of evidence, risk:benefit relationship, and clinical feasibility. This process was implemented using an Evidence Profile Worksheet for each question that included an introduction, consensus on

Abbreviations: ABP, arterial blood pressure; AKI, acute kidney injury; ALS, advanced life support; aOR, adjusted odds ratio; BG, blood glucose; BLS, basic life support; CI, confidence interval; CPA, cardiopulmonary arrest; CPC, Cerebral Performance Category; DBP, diastolic arterial blood pressure; DopBP, Doppler blood pressure; EDD, esophageal detection device; $ETCO_2$, end-tidal CO_2; ETT, endotracheal tube; GRADE, Grading of Recommendations, Assessment, Development, and Evaluation; HR, heart rate; ICP, intracranial pressure; IHCPA, in-hospital cardiopulmonary arrest; IQR, interquartile range; MAP, mean arterial blood pressure; NEWS, National Early Warning Score; OHCPA, out-of-hospital cardiopulmonary arrest; OR, odds ratio; PCA, postcardiac arrest; PEA, pulseless electrical activity; PICO, Population, Intervention, Comparator, and Outcome; RECOVER, Reassessment Campaign on Veterinary Resuscitation; ROSC, return of spontaneous circulation; RR, respiratory rate; SAP, systolic arterial blood pressure; SpO_2, oxygen saturation percentage as measured by pulse oximetry; SPP, systemic perfusion pressure.

science, treatment recommendations, justification for these recommendations, and important knowledge gaps. A draft of these worksheets was distributed to veterinary professionals for comment for 4 weeks prior to finalization.

Setting: Transdisciplinary, international collaboration in university, specialty, and emergency practice.

Results: Thirteen questions pertaining to hemodynamic, respiratory, and metabolic monitoring practices for identification of cardiopulmonary arrest, quality of CPR, and postcardiac arrest care were examined, and 24 treatment recommendations were formulated. Of these, 5 recommendations pertained to aspects of end-tidal CO_2 ($ETCO_2$) measurement. The recommendations were founded predominantly on very low quality of evidence, with some based on expert opinion.

Conclusions: The Monitoring Domain authors continue to support initiation of chest compressions without pulse palpation. We recommend multimodal monitoring of patients at risk of cardiopulmonary arrest, at risk of re-arrest, or under general anesthesia. This report highlights the utility of $ETCO_2$ monitoring to verify correct intubation, identify return of spontaneous circulation, evaluate quality of CPR, and guide basic life support measures. Treatment recommendations further suggest intra-arrest evaluation of electrolytes (ie, potassium and calcium), as these may inform outcome-relevant interventions.

KEYWORDS

capnography, cardiopulmonary arrest, ECG, electrolytes, pulse oximetry

1 | INTRODUCTION

Monitoring of patients during and following CPR allows identification of factors that can impact return of spontaneous circulation (ROSC), favorable neurologic outcome, and survival to hospital discharge. These factors may provide prognostic information, but more importantly, they may inform the actions of rescuers to potentially improve outcome through alteration of CPR technique or administration of supportive treatments. The Monitoring Domain focuses on elements that can be assessed during CPR, in the postcardiac arrest (PCA) period, and in patients who are at risk of cardiopulmonary arrest (CPA). Identifying problems in patients at risk of CPA is particularly important to address, as prevention of CPA before it happens likely provides the best opportunity for a positive outcome.

The most significant monitoring guidance from the 2012 Reassessment Campaign on Veterinary Resuscitation (RECOVER) publication was a strong recommendation to integrate end-tidal CO_2 ($ETCO_2$) monitoring into every CPR event.[1] The current document reviews new data about $ETCO_2$ targets and uses. Rescuers performing CPR should focus on optimizing the conditions for ROSC for individual patients given the available resources. This document also assesses the utility of point-of-care electrolyte and biochemistry analysis during CPR, focusing on prognostication and identification of opportunities to treat abnormalities and improve chances of ROSC, neurologic outcome, or PCA clinical course.

A portion of the current document is devoted to monitoring patients who are at risk of CPA. Many veterinary practices have the ability to monitor ECG, systemic arterial blood pressure (ABP), and pulse oximetry in dogs and cats. This manuscript assesses the application of these physiologic monitors to assess patients who may be at risk of CPA, with the aim of characterizing changes that may indicate impending arrest. Earlier alerts to patient decompensation can provide the opportunity for intervention, and some CPA events may be avoided entirely.

2 | METHODS

Full explanation of the methods used to generate the Monitoring Domain's treatment recommendations is available in a companion paper.[2] What follows here is an overview. This Monitoring Domain Paper and the associated 2024 RECOVER CPR Guidelines[3] were generated using a modified version of the GRADE (Grading of Recommendations, Assessment, Development, and Evaluation) system for guidelines generation in health care.[4]

The RECOVER Co-Chairs assigned content experts to serve as chairs for the Monitoring Domain (BB, SL). These Domain Chairs generated research questions in the Population, Intervention, Comparator, and Outcome (PICO) format including multiple relevant outcomes for each PICO question. PICO questions were rated as high priority, moderate priority, or lower priority. Nineteen PICO questions

were developed for evidence evaluation in the Monitoring Domain; 6 were rated as moderate or lower priority. Because of the number of PICO questions generated and the number of volunteers available to review and summarize evidence and generate treatment recommendations, only high-priority PICO questions were evaluated. Of the 13 high-priority Monitoring PICO questions evaluated, 5 questions were re-evaluated from the 2012 RECOVER CPR Guidelines and 8 new questions were added.

Domain Chairs prioritized the outcomes for each PICO question by clinical importance so that treatment recommendations could be generated based on the evidence pertaining to the highest priority outcomes for which clinically relevant evidence was available. Outcomes used for most PICO questions included favorable neurologic outcome, survival to hospital discharge, ROSC, and surrogate markers of perfusion, in this order of priority. Additional or different outcomes were investigated for various PICO questions where Domain Chairs deemed this appropriate.

Specialist librarians (Information Specialists) worked with Domain Chairs to create search strings for entry into medical databases. Search strings were developed using an iterative process among Information Specialists and Domain Chairs to optimize the number and type of articles returned in the searches.[a] Peer review of search strategies occurred using modified Peer Review of Electronic Search Strategy Guidelines and informal meetings.[5] Once potentially relevant articles were identified, 2 Evidence Evaluators (specialist veterinarians, general veterinarians in emergency or specialty practice, or veterinary technician specialists in relevant fields such as emergency and critical care, anesthesia, and cardiology) reviewed abstracts independently to eliminate irrelevant material and leave only pertinent primary literature for review. Domain Chairs resolved any conflicts. Relevant publications were then reviewed for each PICO by the same Evidence Evaluators.

A purpose-developed, web-based evaluation system was used to guide Evidence Evaluators through a systematic review using a predetermined, standardized set of questions designed to identify key aspects of evidence quality (eg, risk of bias, consistency with population of interest, consistency of outcomes). This evaluation system used these data to generate Evidence Evaluation Summary Tables for each outcome for every PICO question. Evidence Evaluators also wrote overview summaries of the evidence for their PICO question. Finally, the Domain Chairs generated Evidence Profile Worksheets consisting of a structured summary (introduction, consensus on science, treatment recommendations, justifications for the treatment recommendations, and knowledge gaps for future study) and additional notes made during evaluation of individual studies for each PICO question. These Evidence Profile Worksheets were reviewed and edited by the Co-Chairs. The Co-Chairs and Domain Chairs met to reach consensus on these documents. The treatment recommendations and links to the Evidence Profile Worksheets were then posted at the RECOVER Initiative website[b] for a 4-week open comment period beginning in August 2023; Evidence Evaluators and listservs for relevant specialty and other professional organizations were notified directly of this comment period. Following this period, comments were considered by the Co-Chairs and Domain Chairs, and relevant treatment recommendations honed to create a finalized set of treatment recommendations for CPR in dogs and cats, which appear in this paper. The structured summary for each Monitoring PICO question appears below, and the additional study evaluation notes appear in the full Evidence Profile Worksheets.[b]

BOX 1: Major updates

- The minimum target end-tidal CO_2 ($ETCO_2$) during CPR is 18 mm Hg.
- Proper endotracheal tube placement can be confirmed with $ETCO_2 \geq 12$ mm Hg.
- Plasma potassium concentration should be measured in all animals undergoing CPR.
- Plasma lactate concentration should be measured serially in the postcardiac arrest (PCA) period, and therapy should be adjusted with the goal to normalize plasma lactate concentration as quickly as possible.
- Serum creatinine concentration should be measured serially in the PCA period to monitor, identify, and optimally treat acute kidney injury.
- We suggest routine measurement of blood glucose concentration in the PCA period.
- Patients at risk of CPA or re-arrest should be monitored with continuous ECG and frequent blood pressure measurement.

In accordance with the GRADE system, each treatment recommendation is written either as a *recommendation* where the RECOVER group found stronger evidence (or perceived risk/benefit relationship, where evidence was poor or not available) or as a *suggestion* where the RECOVER group found weaker evidence (or perception of risk/benefit relationship, where evidence was not available), for or against the intervention.

3 | $ETCO_2$ MEASUREMENT

Exhaled CO_2 detection has the potential to help confirm endotracheal tube (ETT) placement at initial intubation and throughout the CPR effort. Additionally, continuous $ETCO_2$ measurement during CPR may be helpful to confirm adequate blood flow. The PICO questions that follow aim to determine how $ETCO_2$ can be used to optimize CPR outcomes in dogs and cats.

3.1 | CO_2 detection to confirm tracheal intubation (MON-01)

In cats and dogs in respiratory or cardiac arrest (P) following attempted endotracheal intubation, does CO_2 detection (capnometer or

colorimetric CO_2 detector) (I) compared to standard clinical assessment (laryngeal visualization, cervical palpation) (C) affect survival to discharge, ROSC, correct intubation, time to start CPR, or complications (O)?

3.1.1 | Introduction

Timely airway management is essential to maximize success of recovery efforts for patients experiencing CPA. Confirmation of endotracheal intubation can allow the rescuer to quickly support ventilation and oxygenation during CPR. There are several studies in which high $ETCO_2$ concentrations have a high sensitivity and specificity to confirm endotracheal intubation; however, many investigators warn that low-perfusion states like those encountered in CPA may result in lower sensitivity of the technique, and that the clinician may need other methods to confirm correct intubation.[6–12]

3.1.2 | Consensus on science

Considering the primary outcome of *survival to discharge*, only 1 observational study in people was identified (very low quality of evidence, downgraded for very serious risk of bias, very serious indirectness, and serious imprecision) that found that (in a registry of 176,054 people experiencing in-hospital CPA) there was an association between documentation of $ETCO_2$ measurement or esophageal detection device (EDD) usage to confirm ETT placement and survival to discharge (adjusted odds ratio [aOR]: 1.093, 95% confidence interval [CI]: 1.033–1.157).[13] Survival to discharge in this cohort was 18.2% (7800/42,955) for patients with correct intubation confirmed by capnometer or EDD compared to 17.2% (3339/19,464) when confirmation was done only by auscultation, and compared to 17.3% (5674/32,712) for patients for whom there was no documentation of correct intubation or when confirmation was done by auscultation alone. No statistical comparison was made between capnometry and confirmation of intubation via other means.[13] This study also identified an association between documentation of $ETCO_2$ or EDD to confirm ETT placement and *ROSC* (aOR: 1.229, 95% CI: 1.179–1.282) when compared to no documented method to determine correct ETT placement. ROSC was achieved in 53.6% (23,061/43,034) of patients with correct intubation confirmed by capnometer or EDD compared to 48.7% (9489/19,480) when confirmation was done only by auscultation, and compared to 48.9% (16,002/32,743) for patients who did not have documentation of correct intubation or when confirmation was done by auscultation alone.[13]

The use of capnography to identify *correct endotracheal intubation* was addressed in 10 observational studies in people (very low quality of evidence, downgraded for serious indirectness and serious imprecision)[6–9,12,14–18] and 1 experimental study in dogs (very low quality of evidence, downgraded for serious indirectness and serious imprecision).[19] All included studies were performed in pulseless people and animals. The largest study[17] included 246 adults with out-of-hospital cardiopulmonary arrest (OHCPA) who were intubated in the field; capnometry in this report was 100% specific and 88% sensitive for tracheal intubation. The positive predictive value of auscultation (99%) and capnometry (100%) to confirm proper ETT placement was similar; however, the negative predictive value of auscultation (ie, the absence of breath sounds confirming improper ETT placement) was significantly better (100%) than that of capnometry (13%; ie, the absence of detected CO_2 reflected improper tube placement).[17] Takeda et al showed that in people in CPA, lung auscultation and $ETCO_2$ measurement had sensitivities of 92.6% and 67.9% for confirmation of endotracheal intubation, respectively.[18] The negative predictive value of auscultation (ie, the absence of breath sounds accurately identified improper tube placement) in this report was 60%, and statistically equivalent to the negative predictive value of $ETCO_2$ measurement (25.7%).[18] The remaining 8 observational studies in people in CPA had similar conclusions: CO_2 presence is uniformly consistent with tracheal ETT placement, while the absence of detection of CO_2 may indicate either tracheal or extratracheal intubation; these reports were either small or did not consistently compare $ETCO_2$ to other methods of confirmation.[6–9,12,14–16] In the single experimental study in dogs in CPA, tubes were preplaced in either the esophagus or the trachea prior to fibrillation, and their placement was confirmed by fiberoptic scope. While the $ETCO_2$ was significantly higher in the correctly intubated dogs during CPR, the $ETCO_2$ measured from esophageally placed tubes was not 0 mm Hg (median: 3 mm Hg, range: 2–11 mm Hg).[19]

No studies were identified that addressed the influence of $ETCO_2$ detection on *the time to start CPR*, although there is some evidence in the human literature to support that the use of colorimetric CO_2 detection devices may allow for earlier confirmation of endotracheal intubation over capnography or lung auscultation.[10,20,21] Likewise, no studies were identified that addressed the impact of $ETCO_2$ measurement at the time of intubation on *complications*.

3.1.3 | Treatment recommendations

In dogs and cats undergoing CPR, detection of $ETCO_2$ using a waveform capnograph attached to the breathing circuit is adequate to confirm proper ETT placement if a waveform is present and CO_2 is consistently detected (strong recommendation, very low quality of evidence).

In dogs and cats undergoing CPR with a CO_2 detection device in place, an $ETCO_2$ of $\geq$12 mm Hg likely indicates proper ETT placement; if the $ETCO_2$ <12 mm Hg, we recommend the rescuer confirm tracheal intubation by other means (strong recommendation, very low quality of evidence).

In intubated dogs and cats undergoing CPR that are instrumented with any CO_2 detection device, when $ETCO_2$ is 0 or very low (eg, <5 mm Hg) despite high-quality chest compressions, we recommend confirmation of tracheal intubation by other means (eg, direct visualization of the tube passing through the arytenoid cartilages, lung auscultation during the pause between chest compression cycles) and reintubation if indicated (strong recommendation, very low quality of evidence).

3.1.4 | Justification for recommendations

All available evidence shows that higher $ETCO_2$ concentration at intubation likely predicts correct tracheal intubation; however, in poor perfusion states such as CPA, low $ETCO_2$ concentrations do not rule out tracheal intubation and may instead indicate the need for improved circulation (eg, improved chest compression technique). Other methods were more accurate in confirming esophageal intubation in human studies, such as lung auscultation (which likely requires a pause in chest compressions), confirming chest wall excursions during ventilation (challenging to assess during CPR), and laryngoscopy (which is not always available). Considering the ease with which the larynx can be visualized in most dogs and cats, the committee recommends confirmation of ETT placement in the case of low $ETCO_2$ by direct visualization when possible—this can be done during chest compressions with adequate staff. When visualization is not possible, the evidence suggests that auscultation of breath sounds during a scheduled 2-minute cycle pause may be more reliable than absence of CO_2 at detecting incorrect tube location.

3.1.5 | Knowledge gaps

There is no clinical evidence in dogs or cats that evaluates the impact of $ETCO_2$ monitoring or other clinical verification methods of successful endotracheal intubation.

3.2 | Use of $ETCO_2$ monitoring during CPR in dogs and cats (MON-07)

For cats and dogs in CPA undergoing CPR (P), does no $ETCO_2$ monitoring (I), compared to continuous $ETCO_2$ monitoring (C), improve favorable neurologic outcome, survival to discharge, ROSC, surrogate markers of perfusion, or chest compression quality (O)?

3.2.1 | Introduction

It is generally accepted that $ETCO_2$ during CPR reflects circulation and, by extension, the quality of chest compressions, with higher $ETCO_2$ values associated with (presumably) better blood circulation and (definitively) improved chances of ROSC. Because of the relationship between $ETCO_2$ and quality of chest compressions, $ETCO_2$ may be used to adjust chest compression technique to improve outcome. This question aimed to determine whether $ETCO_2$-guided chest compressions led to improved outcome compared to CPR performed without this guidance.

3.2.2 | Consensus on science

For the most critical outcomes of *favorable neurologic outcome* and *survival to discharge*, 1 prospective observational study in people was identified (very low quality of evidence, downgraded for serious risk of bias and serious indirectness)[22] that evaluated the impact of monitoring $ETCO_2$ and/or diastolic arterial blood pressure (DBP) (grouped) in 9096 adults with in-hospital cardiopulmonary arrest (IHCPA) and found no association between this monitoring and survival with a favorable neurologic outcome (odds ratio [OR]: 0.97, 95% CI: 0.75–1.26) or survival to discharge (OR: 1.04, 95% CI: 0.92–1.18).[22]

For the next most critical outcome of *ROSC*, the same study as above[22] and 1 retrospective study[23] that included 87 children undergoing CPR were identified (very low quality of evidence, downgraded for serious risk of bias and serious indirectness) that addressed the PICO question. Sutton et al found that monitoring using either $ETCO_2$ or DBP was associated with higher odds of ROSC (OR: 1.22, 95% CI: 1.04–1.43) compared to no monitoring.[22] The Bullock study found that in pediatric resuscitation, ROSC was achieved more often among subjects where capnography use was documented (64% vs 14%).[23] Also, the mean duration of CPR was significantly longer among cases where capnography was used (29 vs 17 min, 95% CI: 2.1–20.2), which may indicate the use of $ETCO_2$ to inform decisions to continue CPR or to change CPR technique, although this was not specifically stated.[23]

Three experimental studies (very low quality of evidence, downgraded for very serious indirectness, serious imprecision, and serious inconsistency) were identified that addressed the PICO question.[24–26] All 3 of these studies compared the effects of $ETCO_2$-guided chest compression delivery to $ETCO_2$-blinded, standard chest compressions on ROSC in fibrillatory[24] and asphyxial–fibrillatory[25,26] models of CPA in piglets. In the fibrillatory CPA model, ROSC was not different when $ETCO_2$ was used to guide compressions (14/20: 70%) compared to real-time audio (pulse oximeter, arterial pressure monitor), verbal, and video ("control") feedback (13/20: 65%).[24] In 1 asphyxial–fibrillatory piglet model, the same authors[25] found that the $ETCO_2$-directed group had a higher incidence of ROSC (7/14; 50%) compared to the control group (2/14; 14%). A final model of prolonged asphyxial–fibrillatory CPA in piglets[26] found that after a long duration of arrest (23 min), survival was equally poor regardless of method used to guide chest compressions (control or $ETCO_2$-directed). After only 17 minutes of arrest, ROSC was 9/14 (64%) in the $ETCO_2$-directed group and 3/14 (21%) in the control/$ETCO_2$-blinded group, though this finding failed to reach significance.[26]

The impact of $ETCO_2$ monitoring on *surrogate markers of perfusion* was investigated in 4 experimental piglet studies (very low quality of evidence, downgraded for very serious indirectness, serious imprecision, and serious inconsistency).[24–27] In a porcine fibrillatory CPA model, mean arterial blood pressure (MAP) was better when $ETCO_2$ was used to guide compressions (27.7 ± 5.4 mm Hg) compared to "control" feedback consisting of real-time audio (pulse oximeter, arterial pressure monitor), verbal, and video cues (21.5 ± 6.4 mm Hg).[24] Mean central venous pressure was higher with $ETCO_2$ guidance in this study, although systemic perfusion pressure (SPP) was not different between groups.[24] In an asphyxial–fibrillatory piglet model, the same authors [25] found that the $ETCO_2$-directed group had higher DBP during advanced life support (ALS), which resulted in a greater increase in DBP after epinephrine administration, compared to the control group. In a model of prolonged asphyxial–fibrillatory CPA in piglets,[26] after

17 minutes of asphyxial CPA, there was no difference between the groups in DBP, MAP, central venous pressure, SPP, coronary perfusion pressure (CoPP), or intracranial pressure (ICP) when comparing $ETCO_2$-guided basic life support (BLS) to standard without $ETCO_2$.[26] In the same study, after 17 minutes of arrest, only MAP, SPP, and COPP were higher during ALS in the $ETCO_2$-directed group than in the control group. After 23 minutes of asphyxial arrest, none of the aforementioned hemodynamic metrics differed between groups during BLS, and only ICP was higher in the $ETCO_2$-directed group during ALS.[26] The final study included a cohort of piglets with asphyxial cardiac arrest and compared a DBP-guided resuscitation strategy to an $ETCO_2$-guided strategy (alternating between the 2 every 2 min). The $ETCO_2$-guided chest compressions resulted in higher ICP (21.7 ± 2.3 vs 16.0 ± 1.1 mm Hg), while DBP-guided chest compressions were associated with a higher myocardial perfusion pressure (6.0 ± 2.8 vs 2.4 ± 3.2) and COPP (9.0 ± 3.0 vs 5.5 ± 4.3).[27]

3.2.3 | Treatment recommendation

We recommend continuous measurement of $ETCO_2$ to guide chest compression quality during CPR in dogs and cats (strong recommendation, very low quality of evidence).

3.2.4 | Justification of treatment recommendation

Few studies were identified that evaluated the presence of continuous $ETCO_2$ monitoring to its absence for chest compression guidance. However, of the studies identified, most showed an improvement in ROSC or various surrogate markers of perfusion when patient-derived feedback (continuous $ETCO_2$ or DBP measurement) was used to guide chest compressions as opposed to nonpatient, guideline-driven feedback (eg, visual or audio cues based on general, prespecified metrics). No studies showed a clear risk of guiding chest compressions with continuous $ETCO_2$; additionally, $ETCO_2$ monitoring is relatively noninvasive, inexpensive, and simple to use.

3.2.5 | Knowledge gaps

No studies are available in dogs and cats that evaluate the guidance of chest compressions based on continuous $ETCO_2$, on audio/visual feedback based on general guideline principles, or on any other method.

3.3 | Optimal $ETCO_2$ target during CPR (MON-10)

In cats and dogs with CPA (P), does achieving any other specific $ETCO_2$ during CPR (I), compared to achieving $ETCO_2$ of ≥15 mm Hg (C), improve favorable neurologic outcome, survival to discharge, or ROSC (O)?

3.3.1 | Introduction

While it has been established that higher $ETCO_2$ values are associated with better outcomes in CPR in people, the minimum $ETCO_2$ target in dogs and cats is unknown.[28] Hofmeister et al reported that in their population of dogs, 17 of 18 (94%) with an $ETCO_2$ of <15 mm Hg during CPR were not successfully resuscitated, whereas 25 of 29 (86%) with an $ETCO_2$ of ≥15 mm Hg achieved ROSC.[29] In the same study, 5 of 9 cats with an $ETCO_2$ of <20 mm Hg were not successfully resuscitated, whereas 9 of 10 cats with an $ETCO_2$ of ≥20 mm Hg achieved ROSC. The best way to use $ETCO_2$ to improve chest compression technique in dogs and cats is unknown.

3.3.2 | Consensus on science

Considering *favorable neurologic outcome* after ROSC, 3 observational studies in people (very low quality of evidence, downgraded for very serious indirectness and inconsistency) were identified that addressed the PICO question. In 803 adults with IHCPA, Sutton found that the achievement of an $ETCO_2$ of >10 mm Hg during CPR was significantly associated with favorable neurologic outcome (OR: 2.31, 95% CI: 1.31–4.09).[22] Calbay, however, found no association between $ETCO_2$ during CPR and favorable neurologic outcome in 155 adults with OHCPA.[30] Finally, in 43 pediatric patients with IHCPA, Berg et al found that all children who survived to discharge did so with favorable neurologic outcome, and that both survivors and nonsurvivors achieved median $ETCO_2$ values of >20 mm Hg during CPR.[31]

Considering the impact of the PICO question on *survival to discharge*, 3 observational studies in people (very low quality of evidence, downgraded for very serious indirectness) were identified. In 803 adults with IHCPA, Sutton found that $ETCO_2$ >10 mm Hg during CPR was associated with survival to hospital discharge (OR: 2.41, 95% CI: 1.35–4.30).[22] A prospective, observational study in 102 adults undergoing CPR in the emergency department found that median $ETCO_2$ during CPR was higher (35 mm Hg) in patients who survived to hospital admission than in nonsurvivors (13.7 mm Hg, $P < 0.01$). In this cohort, $ETCO_2$ >16 mm Hg predicted survival to hospital admission.[32] In 43 pediatric patients with IHCPA, Berg et al found no difference in survival to discharge when comparing pediatric patients with mean $ETCO_2$ >20 mm Hg during CPR to those with lower mean $ETCO_2$.[31]

For the next critical outcome of *ROSC*, 12 observational studies (very low quality of evidence, downgraded for very serious indirectness)[31,33–43] and 1 experimental study in dogs (very low quality of evidence, downgraded for serious indirectness and serious imprecision)[44] were identified that addressed the PICO question. In a registry study of 109 dogs and cats undergoing CPR,[33] median $ETCO_2$ was significantly higher (23 mm Hg) in those that achieved ROSC than in those that did not (15 mm Hg); this report recommended an $ETCO_2$ of 16.5 mm Hg as a target to maximize likelihood of ROSC (sensitivity 75%, 95% CI: 60%–86%; specificity 64%, 95% CI: 52%–75%).[33] One clinical observational study in 35 dogs and cats undergoing CPR found that patients that achieved ROSC had higher initial, peak, mean,

and change in $ETCO_2$ than patients that did not achieve ROSC; optimal $ETCO_2$ cutoff to predict ROSC in this population was 18 mm Hg.[34] An experimental study in dogs supported the relationship of higher $ETCO_2$ with ROSC; however, it demonstrated a much lower mean $ETCO_2$ in the survivor group than reported in other reviewed canine and human studies (9.6 ± 3.2 mm Hg).[44]

In people, a prospective, observational study in 737 adults with OHCPA found that $ETCO_2$ at 20 minutes into ALS was higher in patients who achieved ROSC (32.8 ± 9.1 mm Hg) than in those who did not (6.9 ± 2.2 mm Hg).[37] One observational study in 575 adults with OHCPA found that mean $ETCO_2$ was higher in patients with ROSC than in those who did not achieve ROSC, regardless of cause of CPA or arrest rhythm.[43] One prospective observational study of 114 adults with OHCPA found significantly higher $ETCO_2$ from 5 minutes into CPR to the final value obtained in people achieving ROSC than in those who did not, regardless of cause of CPA.[42] Finally, Singer described an optimal $ETCO_2$ target of ≥19 mm Hg based on the prospective observational study of 100 adult human patients with OHCPA.[39] An additional observational study in 97 adults with IHCPA or OHCPA found that the final $ETCO_2$ (36.4 ± 4.46 vs 11.74 ± 7.01 mm Hg) was greater in patients who achieved ROSC than in those who did not.[38] An observational study of OHCPA in 90 adults found that people who achieved ROSC had significantly higher $ETCO_2$ just prior to ROSC (31 ± 5.3 mm Hg) than those who did not achieve ROSC by 20 minutes of ALS (3.9 ± 2.8 mm Hg).[40] Savastano evaluated defibrillation success of 207 defibrillation events in 62 people with OHCPA and shockable rhythms and determined that a higher $ETCO_2$ was associated with defibrillation success; no shocks administered to patients with $ETCO_2$ >45 mm Hg were unsuccessful.[36] Finally, a study in 32 young to middle-aged adults suffering nontraumatic OHCPA or IHCPA found that mean $ETCO_2$ during CPR was lower in those who failed to achieve ROSC (19.1 ± 7.8 mm Hg) than in those who achieved ROSC (26.3 ± 6.5 mm Hg).[35] Only 2 identified studies found a lack of association between $ETCO_2$ and ROSC, including 1 in a small population of adults[41] and 1 in pediatric patients.[31]

3.3.3 | Treatment recommendation

We recommend optimizing CPR to maximize $ETCO_2$ to no less than 18 mm Hg in dogs and cats undergoing CPR (strong recommendation, very low quality of evidence).

3.3.4 | Justification of treatment recommendation

Observational veterinary and human studies, in addition to some experimental data, suggest that higher $ETCO_2$ targets are positively associated with ROSC. There were no identified studies that directly compared an $ETCO_2$ of 15 mm Hg versus higher $ETCO_2$ values. Therefore, this recommendation is to target the high value found in clinical observational studies of dogs and cats ($ETCO_2$ > 18 mm Hg), with the understanding that as a concept, higher $ETCO_2$ values (even into the low 30s in mm Hg) are associated with ROSC.

3.3.5 | Knowledge gaps

Optimal $ETCO_2$ targets in dogs and cats undergoing CPR are unknown, and larger studies are warranted. The effects of precise targets and the harm (ie, more damage to thoracic structures) that may occur with more aggressive CPR to achieve higher $ETCO_2$ raise questions about how high an $ETCO_2$ is too high, if such a value exists, or if targets should differ among species.

4 | OTHER CARDIOVASCULAR MONITORING DURING CPA

Other forms of cardiovascular monitoring, such as pulse palpation or direct ABP monitoring, can provide beat-to-beat (compression-to-compression) information about the movement of blood in the body. The following 2 PICO questions aimed to determine whether cardiac apex or femoral pulse palpation is valuable for diagnosing CPA, and whether there is a role for direct ABP monitoring during CPR efforts.

4.1 | Femoral pulse or cardiac apex palpation to recognize CPA (MON-11)

In cats and dogs suspected to be in CPA (P), does the addition of femoral pulse or cardiac apex palpation (I) compared to assessment of only mental responsiveness and attempts to breathe (C) improve favorable neurologic outcome, survival to discharge, ROSC, accurate diagnosis of CPA, or time to start CPR (O)?

4.1.1 | Introduction

The 2012 RECOVER CPR Guidelines recommend against pulse palpation as part of the initial assessment of the unresponsive patient.[45] This PICO question investigates whether there is evidence that pulse palpation or apex beat palpation should be incorporated into the initial assessment of the unresponsive patient when attempting to rule out CPA.

4.1.2 | Consensus on science

No studies were found to inform the answer to the PICO question for *favorable neurologic outcome*, *survival to discharge*, or *ROSC*. One observational study (very low quality of evidence, downgraded for very serious indirectness) investigated the influence of pulse palpation on the *time to start of CPR*, specifically assessing the time required by rescuers of varied experience to accurately diagnose the presence or absence of a pulse in children and infants receiving nonpulsatile extracorporeal circulation for cardiac arrest or with cardiac failure.[46] Across all rescuers, the accuracy of diagnosing pulselessness was 76% and required 30 ± 19 seconds, while the presence of a pulse was correctly

diagnosed in 79% of cases and required 13 ± 13 seconds. Experienced rescuers achieved faster diagnoses of both pulselessness (25 ± 14 s) and the presence of pulses (9 ± 5 s) than novices (37 ± 24 and 21 ± 19 s, respectively; $P = 0.042$).

In addition to the observational study described for the outcome above, 1 observational study was identified that evaluated the accuracy of pulse palpation for the *diagnosis of CPA* in children and infants (very low quality of evidence, downgraded for serious indirectness).[47] When a pulse was present, rescuer pulse palpation accuracy was 78% (95% CI: 70–82), sensitivity was 0.86 (95% CI: 0.77–0.90), and specificity was 0.64 (95% CI: 0.53–0.74). When the pulse was absent, rescuer accuracy was 89% and sensitivity was 0.89. Two additional observational studies of the diagnostic accuracy of carotid pulse palpation in adult humans with CPA by first responders were identified from the RECOVER 2012 guidelines process that were not included in the GRADE analysis for this PICO question. The results of these 2 studies were similar to the 2 studies described above. The first showed that only 16.5% of participants were able to reach any pulse diagnosis within 10 seconds, and that only 15% of assessments that were made within 10 seconds were correct and were almost all in patients with pulses.[48] In a second study of first responders assessing carotid pulses in adult patients, sensitivity for pulselessness was 90%, but specificity was only 55%, and median time for diagnosis of pulseless patients was 30 seconds, with a minimum diagnostic time of 13 seconds.[49]

4.1.3 | Treatment recommendation

In apneic, unresponsive dogs and cats, we recommend that BLS be started without attempting to palpate femoral or apex pulses (strong recommendation, very low quality of evidence).

4.1.4 | Justification of treatment recommendation

The evidence suggests that pulse palpation in people is inaccurate even when performed by experienced rescuers, and that the diagnosis of pulselessness commonly takes ≥30 seconds. This procedure will likely lead to delays in the start of chest compressions in dogs and cats with CPA, which could have a negative impact on prognosis. In addition, in human patients not in CPA in whom CPR was initiated, only 2% sustained injuries likely caused by the CPR, and none experienced visceral organ injury.[50] Taken together, the available evidence suggests that the risk:benefit ratio of starting CPR in a patient without delays to palpate the pulse or apex beat palpation is likely favorable.

Pauses in chest compressions of greater than 10 seconds are detrimental to patients during CPR (see BLS-16),[51] which further suggests an unacceptable risk:benefit ratio of the delay caused by pulse palpation to diagnose CPA. In addition, a retrospective observational veterinary study showed that a shorter time to initiation of CPR is associated with more favorable frequency of ROSC in small animal patients.[52] In that study, median time (range) from CPA to start of CPR in all patients that achieved ROSC was 0.5 minutes (0.08–10 min) versus 1 minute (0.08–30 min) in animals that did not experience ROSC ($P = 0.04$). In another retrospective veterinary study, animals with witnessed CPA, and hence likely shorter delays to the start of CPR, were more likely to regain ROSC than patients with unwitnessed arrests.[53] Pulse palpation may be a reasonable adjunctive monitoring option during CPR, as both of these retrospective studies found that patients with palpable femoral pulses during CPR were more likely to achieve ROSC.

4.1.5 | Knowledge gaps

Given current guidelines and existing evidence suggesting that pulse palpation as a diagnostic test for CPA results in longer time to start CPR (which is associated with a poorer outcome), accompanied by minimal evidence of adverse effects of initiation of CPR in patients not in CPA, it is unclear that this question requires further investigation. However, studies investigating the utility of pulse palpation during CPR would be warranted.

4.2 | Measurement of direct ABP during CPR (MON-12)

In cats and dogs in CPA undergoing CPR (P), does direct ABP monitoring to titrate BLS and ALS interventions (I) compared to no direct ABP monitoring (C) improve favorable neurologic outcome, survival to discharge, ROSC, or surrogate markers of perfusion(O)?

4.2.1 | Introduction

There is some evidence to suggest that direct ABP monitoring, specifically that of the DBP, may be beneficial during CPR.[54] Direct ABP monitoring may not be available or feasible in many dogs and cats with CPA, but if an arterial catheter is in place at the time of arrest or can be placed during CPR, such monitoring may be possible and desirable. This PICO question was designed to evaluate the usefulness of direct ABP monitoring during CPR in dogs and cats.

4.2.2 | Consensus on science

For the most critical outcome of *favorable neurologic outcome*, 2 observational studies in children were identified (very low quality of evidence, downgraded for serious indirectness and inconsistency)[55,56] as well as 4 experimental studies in swine (very low quality of evidence, downgraded for very serious indirectness and serious imprecision)[57–60] that addressed this PICO question.

In a prospective, multicenter cohort study of 164 children with IHCPA, mean DBP ≥25 mm Hg during CPR in infants and ≥30 mm Hg in children ≥1 year of age was associated with greater likelihood of survival with a favorable neurologic outcome; however, systolic

arterial blood pressure (SAP) targets of ≥60 and ≥80 mm Hg, respectively, were not associated with favorable neurologic outcome.[56] A prospective, multicenter, cohort study of neurologic morbidity following 244 CPR events in children who survived following IHCPA did not find an association between the DBP or SAP achieved during CPR and the development of new neurologic morbidity.[55]

In an experimental porcine model of fibrillatory CPA, piglets administered hemodynamic-directed chest compressions targeting SAP of 100 mm Hg and COPP of >20 mm Hg had better favorable neurologic outcome than piglets treated with a depth-guided chest compression strategy.[59] In an experimental asphyxial–fibrillatory CPA porcine model, 24-hour favorable neurologic outcome was more likely in pigs resuscitated with a CPR strategy that targeted SAP of 90 mm Hg and COPP of >20 mm Hg compared to standard, depth-guided CPR.[58] In another asphyxial–fibrillatory porcine study, this hemodynamic-guided strategy resulted in superior neurologic scores and cerebral mitochondrial bioenergetics compared to standard CPR.[60] Similarly, improved favorable neurologic outcome was demonstrated following asphyxial–fibrillatory CPA in swine when targeting SAP of 100 mm Hg and a COPP of >20 mm Hg during CPR, compared to standard, depth-guided CPR.[57]

Concerning the outcome measure of *survival to discharge*, 2 observational studies in people[56,61] (very low quality of evidence, downgraded for serious indirectness and serious imprecision) and 5 experimental studies in swine[57–59,62,63] (very low quality of evidence, downgraded for very serious indirectness and serious imprecision) were identified.

A prospective, multicenter cohort study of 164 children with IHCPA found that achieving a mean DBP ≥25 mm Hg during CPR in infants and ≥30 mm Hg in children older than 1 year of age was associated with greater likelihood of survival to discharge; however, the attainment of SAP targets of ≥60 and ≥80 mm Hg, respectively, was not associated with survival.[56] A small retrospective study in 35 adults in CPA undergoing percutaneous coronary intervention found that no ABP parameter measured during CPA was associated with survival, though survival was not defined.[61]

In an experimental porcine model of fibrillatory CPA, piglets administered hemodynamic-directed chest compressions targeting an SAP of 100 mm Hg and COPP of >20 mm Hg had better 24-hour survival than piglets treated with a depth-guided chest compression strategy[59]; this same group had similar findings in an asphyxial–fibrillatory swine model.[57] However, in another experimental asphyxial–fibrillatory CPA porcine model, ROSC and 24-hour survival were not different between pigs resuscitated with a CPR strategy that targeted SAP of 90 mm Hg and COPP of >20 mm Hg compared to standard, depth-guided CPR.[58] Finally, 2 experimental studies of short-term (45-min) survival in swine found that 45-minute survival was improved in both asphyxial–fibrillatory and fibrillatory CPA models when targeting COPP of >20 mm Hg as opposed to depth-guided chest compression strategies during CPR.[62,63]

Regarding the PICO question with an outcome measure of *ROSC*, 3 observational studies in people (very low quality of evidence, downgraded for very serious indirectness and serious inconsistency)[56,61,64] and 4 experimental studies[57,62,63,65] (very low quality of evidence, downgraded for serious indirectness and serious imprecision) were identified.

In 1 clinical study of 100 adults, the majority with OHCPA, maximal COPP was predictive of ROSC, and only patients with maximum COPP of ≥15 mm Hg achieved ROSC. This study also found that maximal aortic relaxation pressure (a surrogate for DBP) was significantly higher in people who achieved ROSC than in those who did not (35.2 ± 11.5 vs 24.1 ± 15.2 mm Hg).[64] Similarly, a small retrospective study in 35 adults in CPA undergoing percutaneous coronary intervention found that higher early (but not late) DBP measured during CPA was associated with ROSC.[61] However, a prospective, multicenter cohort study of 164 children with IHCPA found no association between achieving specific SAP or DBP targets and ROSC.[56]

One experimental canine model of fibrillatory CPA showed that during CPR, aortic diastolic pressure was significantly higher (always >30 mm Hg) in dogs that maintained ROSC for 10 minutes than in nonsurvivors.[65] Two experimental studies of short-term (45-min) survival in swine found that ROSC was improved in both asphyxial–fibrillatory and fibrillatory CPA models when targeting COPP of >20 mm Hg, as opposed to depth-guided chest compression strategies during CPR.[62,63] Similarly, improved ROSC was demonstrated following asphyxial–fibrillatory CPA in swine when targeting SAP of 100 mm Hg and a COPP of >20 mm Hg during CPR, compared to standard, depth-guided CPR.[57]

Evidence for the outcome of *surrogate markers of perfusion* was not summarized since a recommendation could be made based on data from the higher priority outcomes.

4.2.3 | Treatment recommendation

In dogs and cats in CPA with an arterial catheter in place, we recommend optimizing BLS and ALS interventions to maximize DBP to no less than 30 mm Hg (strong recommendation, very low quality of evidence).

4.2.4 | Justification of treatment recommendation

There are adequate experimental and observational human data to suggest that maintaining COPP during CPR is associated with improved outcomes. In the absence of direct measurement of COPP, DBP may act as a surrogate measure, and DBP >30 mm Hg should be targeted, based on human observational studies. A DBP of 30 mm Hg should ensure a COPP of ≥20 mm Hg in most cases. This recommendation is most relevant for those animals that experience CPA with an arterial catheter already in place, as placement of an arterial catheter and assembly of a pressure monitoring system are difficult to perform during the CPR effort. In large-enough resuscitation teams with the expertise and equipment to rapidly place arterial monitoring devices, this may be a helpful adjunct to other techniques (eg, $ETCO_2$) to assess the quality of CPR. In 1 human case series of OHCPA, arterial access was achieved in 1.5 (range: 1–2) attempts, on average 7.9 ± 1.2 minutes after patient presentation. Blood pressure measurement and recording

began 10.5 ± 2.4 minutes after presentation.[66] This finding suggests that well-trained teams may be able to achieve arterial access and direct ABP monitoring within a relevant time frame in animals of adequate size.

4.2.5 | Knowledge gaps

There are very limited experimental and no clinical data regarding the utility of direct blood pressure monitoring (for any target measure including DBP) in dogs or cats.

Retrospective reports of clinical data regarding the usefulness of direct ABP monitoring in dogs and cats undergoing CPR would be helpful to determine whether experimental studies in asphyxial models in dogs and cats could be justified.

5 | MEASUREMENT OF ELECTROLYTES AND OTHER BLOOD CHEMISTRY COMPONENTS

With the broader availability of cage-side analyzers for blood gas, electrolyte, and blood biochemistry components, a broader question may be posed about whether the results of measurement of specific analytes may impact the provision of CPR or care following ROSC. Additionally, some analytes (eg, lactate) can indicate resolving or persistent states of poor oxygen delivery, and others (eg, blood glucose [BG] concentration) may have indirect impacts on prognosis. The following PICO questions aim to determine whether measurements of specific analytes may impact critical outcomes in the periarrest period.

5.1 | Serial plasma lactate concentration measurement (MON-02)

In cats and dogs that have experienced ROSC after CPA (P), does serial plasma lactate measurement (lactate clearance) (I), as opposed to single time-point plasma lactate measurement (C), improve favorable neurologic outcome, survival to discharge, prediction of recurrent CPA, or duration of postarrest hospitalization (O)?

5.1.1 | Introduction

Inadequate tissue perfusion likely results in hyperlactatemia commonly in the PCA period. Persistently increased lactate concentrations in the PCA period may indicate ongoing tissue hypoperfusion that may be associated with worse outcomes. The aim of this question was to evaluate whether monitoring lactate clearance in the PCA period is more helpful for guiding therapy than a single-point lactate measurement.

5.1.2 | Consensus on science

For the most critical outcome of *favorable neurologic outcome*, 17 observational studies in people were identified that were relevant to the PICO question (very low quality of evidence, downgraded for very serious indirectness and serious inconsistency). Ten of the studies evaluated both single time-point blood lactate concentrations and either lactate clearance or blood lactate concentration at additional standardized time points.[67–76] Although there is some inconsistency among the studies, in 8 of the 10, multivariate analyses including both admission blood lactate concentration and either lactate clearance or blood lactate concentrations at later time points after ROSC showed that lactate clearance, blood lactate concentration at 48 hours post-ROSC, or the last measured blood lactate concentration after ROSC was independently associated with favorable neurologic outcome, while the admission blood lactate concentration or blood lactate concentrations at other time points was not.[67,69–74,76] Another report documented a negative association between favorable neurologic outcome and persistent hyperlactatemia 12 and 24 hours post-ROSC, but did not include a statistical analysis to compare serial lactate measurements to a single measurement.[68] In only 1 of the 10 studies, admission blood lactate concentration but not lactate concentrations at 6, 12, 24, or 48 hours after ROSC was an independent predictor of favorable neurologic outcome.[75] Taken together, 9 of these 10 observational studies provide evidence that serial blood lactate measurements documenting decreasing lactate concentrations in the initial 48 hours after ROSC likely provide better prognostic information regarding favorable neurologic outcome than the admission blood lactate concentration alone.

The remaining 7 observational studies only evaluated a single lactate concentration shortly after ROSC.[69,77–82] Four of the 7 studies used multivariate analyses including blood lactate concentration in addition to other potentially prognostic factors and showed an independent association between lactate concentration and favorable neurologic outcome.[69,78,81,82] One of the 7 studies was a retrospective evaluation of 55 elderly people who achieved prehospital ROSC and used receiver operating characteristic curve analysis to identify a cut-off admission lactate concentration of 7.8 mmol/L (70 mg/dL), which had a sensitivity of 90.5% and a specificity of 55.9% to predict favorable neurologic outcome.[79] The other 2 studies found no association between admission lactate and favorable neurologic outcome in adult people with OHCPA.[77,80]

Because of the large amount of evidence available to inform a recommendation for the most critical outcome, the outcomes of *survival to discharge*, *prediction of recurrent CPA*, and *duration of postarrest hospitalization* were not exhaustively evaluated. There were no studies identified that addressed *prediction of recurrent CPA* and *duration of postarrest hospitalization*, and most studies identified that addressed *survival to discharge* were the same as those used to evaluate the outcome of *favorable neurologic outcome*.

No studies were identified in people or veterinary species that specifically evaluated the impact on any outcome of using blood

lactate concentration in the PCA period to guide treatment. Most of the studies were retrospective in nature, and those that were prospective were observational and hence did not compare patients treated based on lactate concentrations to those in which lactate concentration was not used to guide therapy.

5.1.3 | Treatment recommendations

We recommend serial measurement of lactate in the PCA period (strong recommendation, very low quality of evidence).

We recommend that serial lactate measurements be used to guide and evaluate response to treatment in dogs and cats in the PCA period (strong recommendation, expert opinion).

5.1.4 | Justification of treatment recommendations

Although there is some variability in the primary findings of the observational studies evaluated this PICO question, the majority showed that measurement of blood lactate concentration in the PCA period offers prognostic information about favorable neurologic outcome and that serial measurements or evaluation of lactate clearance are likely superior to a single lactate concentration measurement for this purpose. Given that lactate is a well-established marker of decreased oxygen delivery to tissues, the committee members believe that a strong recommendation to use lactate and lactate clearance to guide resuscitation of patients in the PCA period is warranted.

5.1.5 | Knowledge gaps

There are no studies comparing the use of single blood lactate measurements to lactate clearance following ROSC in dogs and cats for prognostication. In addition, there are no studies in any species evaluating the impact of using lactate or lactate clearance data to guide therapy in the PCA period or evaluating the optimal interval between lactate measurements.

5.2 | Glucose measurement (MON-03a)

In cats and dogs that have experienced ROSC after CPA (P), does measurement of glucose (I), as opposed to nonmeasurement (C), improve favorable neurologic outcome, survival to discharge, prediction of recurrent CPA, or duration of postarrest hospitalization (O)?

5.2.1 | Introduction

BG monitoring is widely available in veterinary hospitals, inexpensive, and minimally invasive. Severe hypoglycemia and hyperglycemia, as well as targeted protocols to tightly regulate glucose concentrations in hospitalized patients, have been associated with both worse and better outcomes among the critically ill.[83–85] While glucose derangements can occur in CPA patients during the PCA period, the role of BG in the outcome of these patients is unknown.

5.2.2 | Consensus on science

We identified 5 observational studies in people that examined the association between PCA BG concentrations and *survival to discharge* (very low quality of evidence, downgraded for very serious indirectness).[86–90] Four of these studies are focused on patients with OHCPA. Skrifvars et al, in an observational study including 96 patients, found that the mortality rate at 6 months after ROSC increased with the average BG concentration over the first 72 hours after ROSC, noting a mortality rate of 9% for patients with an average BG between 5.5 and 6.8 mmol/L, 23% for those with average BG between 6.9 and 7.9 mmol/L (124–142 mg/dL), 50% for those with average BG between 7.9 and 8.9 mmol/L (142–160 mg/dL), and a mortality rate of 64% for patients with an average BG between 9.1 and 27.9 mmol/L (164–502 mg/dL).[90] In another observational study including 2028 patients with ROSC after OHCPA, Zhou reported increased odds for in-hospital mortality in patients with a mean BG concentration between 7.8 and 10 mmol/L (140.5–180 mg/dL; OR: 1.62, 95% CI: 1.26–2.08) and those with a mean BG concentration >10 mmol/L (OR: 1.80, 95% CI: 1.40–2.30) when compared to patients with normal BG concentration.[86] Two additional observational studies including a total of 1003 OHCPA patients suggest a similar association between hyperglycemia and nonsurvival.[87,88] Hyperglycemia also occurred after IHCPA in both diabetic and nondiabetic people, and in 1 study cohort, both hypo- and hyperglycemia were associated with decreased survival in nondiabetics, while only severe hyperglycemia (minimum BG concentration >13.3 mmol/L [239.6 mg/dL]) held this association in diabetic patients.[89]

With regard to the outcome measure of *favorable neurologic outcome*, 7 observational studies in people (very low quality of evidence, downgraded for very serious indirectness) and 1 experimental study in swine (very low quality of evidence, downgraded for very serious indirectness and serious imprecision) were identified.[87,88,91–96] Human studies assessing the association of BG concentrations with neurologic outcome in patients during the PCA period generally concluded that patients with hypoglycemia, hyperglycemia, and/or high variability of BG concentrations have worse neurologic outcomes compared to patients with normal BG concentrations and less variability of BG concentrations.[87,88,91–95]

In an observational study from registry data including 883 OHCPA patients treated with targeted therapeutic hypothermia, the mean BG concentration obtained 1 hour after ROSC was significantly lower in those with favorable neurologic outcome (Cerebral Performance Category [CPC] 1–2) (12.8 ± 5.0 mmol/L [230.6 ± 90 mg/dL]) compared to those with poor neurologic outcome (14.6 ± 7.6 mmol/L [263 ± 137 mg/dL]).[88] This association between PCA BG and favorable neurologic outcome was maintained when adjusting for confounders

in multivariate analysis (OR: 0.955, 95% CI: 0.918–0.994). In an additional post hoc analysis including 234 OHCPA patients undergoing targeted temperature management, subjects were categorized according to quartiles of median BG concentrations at 12 hours after ROSC: QI 5.6 mmol/L (3.7–6.4 mmol/L; 101 mg/dL [67–115 mg/dL]), QII 7.2 mmol/L (6.4–7.9 mmol/L; 130 mg/dL [115–142 mg/dL]), QIII 9.0 mmol/L (7.9–10.7 mmol/L; 162 mg/dL [142–193 mg/dL]), and QIV 14.7 mmol/L (10.7–25.8 mmol/L; 265 mg/dL [193–465 mg/dL]).[95] In a multivariate analysis adjusting findings for relevant confounders including duration of untreated CPA and duration of CPR, those in QI (OR: 4.55, 95% CI: 1.28–16.12) and QII (OR: 3.02, 95% CI: 3.29–49.9) were more likely to survive with good favorable neurologic outcome compared to those in QIV. Although not the objective of this study, the authors suggest that a more liberal BG target (eg, 3.7–7.9 mmol/L [67–142 mg/dL]) might be warranted, and that tight glycemic control in its original definition (ie, 4.4–5.6 mmol/L [79–101 mg/dL]) might not be necessary.[83] A further post hoc study including 939 patients hospitalized after OHCPA found that median postarrest BG concentrations were higher in patients with poor neurologic outcomes (12 mmol/L [interquartile range, IQR: 8.9–15.9 mmol/L]; 216 mg/dL [IQR: 160–286 mg/dL]) compared to those with favorable neurologic outcome (10 mmol/L [IQR: 7.71–13.5 mmol/L]; 180 mg/dL [IQR: 139–243 mg/dL]) and that 1 mmol/L (18 mg/dL) increase in BG concentration was associated with a 22% increase in the probability of a poor neurologic outcome.[93] An additional 4 observational studies, all small single-center retrospective studies including a combined total of 337 patients, showed a similar association between PCA hyperglycemia and neurologic outcome.[87,91,92,94]

We identified 1 experimental study, using an OHCPA fibrillatory pig model to determine whether early post-ROSC hyperglycemia affects neurologic function at 72 hours postarrest.[96] Animals were left untreated for 7 minutes after induction of ventricular fibrillation, and CPR was continued for up to 15 minutes, by which time 21 of 22 animals achieved ROSC. BG was measured within the first hour of ROSC. The investigators found no association between BG and neurologic function at 72 hours post-ROSC, as 20 of 21 resuscitated animals recovered with favorable neurologic outcome.

Relevant studies were not identified that reported the less critical outcomes of the association of BG concentration and *prediction of recurrent CPA* and *duration of PCA hospitalization*.

5.2.3 | Treatment recommendations

We suggest measuring BG in all dogs and cats as early as possible after ROSC (weak recommendation, very low quality of evidence).

We recommend measuring BG concentration in dogs and cats after ROSC in which hypoglycemia or hyperglycemia are known or suspected (strong recommendation, expert opinion).

5.2.4 | Justification of treatment recommendations

None of the studies we identified directly answer the PICO question on whether the measurement of BG concentration itself impacts critical outcomes in dogs, cats, or any other species recovering from CPA. However, there is ample evidence to suggest that BG abnormalities in the PCA period are common and that there is a consistent association between high BG concentrations and reduced survival rates and poor neurological outcomes. While we did not find any veterinary studies that report on the relationship between PCA BG and relevant outcomes, there are studies in dogs and cats that document hyperglycemia post-ROSC. One experimental study in a dog model of ventricular fibrillation ($n = 26$) evaluated BG and other metabolic parameters after resuscitation from 3 minutes of untreated arrest followed by 10 minutes of CPR. BG concentrations prior to CPA were 6.0 ± 1.9 mmol/L (108 ± 34 mg/dL) and increased to 12.2 ± 3.7 mmol/L (220 ± 67 mg/dL) 10 minutes after ROSC.[97] Hopper et al retrospectively reviewed acid–base, electrolyte, glucose, and lactate values during or immediately after CPR in dogs and cats in a clinical context.[98] Both hypoglycemia (9 of 42 patients; 21%) and hyperglycemia (26 of 42 patients; 62%) were identified during CPR and following ROSC in this patient population. The observed hypoglycemia was thought to be a result of preexisting disease or insulin administration (ie, as a treatment for hyperkalemia) and not a consequence of CPA. The median BG concentration 5 minutes after ROSC was 11.6 mmol/L (range: 0.7–22.8 mmol/L; 209 mg/dL [range: 13–411 mg/dL]). Taken together, these 2 studies suggest that postarrest hyperglycemia occurs in dogs (and possibly cats) to a similar extent as in people. Given the possible prognostic benefit and the relative ease of measuring BG concentration in dogs and cats, we suggest routine serial measurement of postresuscitation BG concentration.

The relationship between postresuscitation hyperglycemia and worse neurologic outcome is well-established in people. Hyperglycemia may be indicative of increased injury severity rather than the cause of further injury, and a benefit of therapy to alleviate hyperglycemia cannot be concluded from these studies. Experimental studies show that induction of hyperglycemia leads to worse neurologic outcome[99–101]; however, treatment of postarrest hyperglycemia in people has not been shown to improve outcome.[102,103]

5.2.5 | Knowledge gaps

While there is evidence in people to support the association between post-ROSC glucose derangements and decreased survival or poor neurologic outcomes, we did not identify any veterinary studies that addressed the relationship between glucose concentrations in the PCA period and relevant outcomes. Future research should focus on whether BG measurement after ROSC is of value to predict survival or time spent in hospital postarrest, if there are "cutoff" BG concentrations associated with favorable neurologic outcome, and if glycemic control in the periarrest period has any effect on patient outcomes.

5.3 | Measurement of sodium and potassium during CPR (MON-08)

In cats and dogs in CPA (P), does the identification and treatment of (arterial or venous) Na+ or K+ disorders during CPR (I) compared to not addressing Na+ and K+ disorders (C) improve favorable neurologic outcome, survival to discharge, or ROSC (O)?

5.3.1 | Introduction

Severe hyperkalemia (eg, >6.5 mmol/L) or hypokalemia (eg, <3 mmol/L) can cause life-threatening cardiotoxicity.[104–106] Severe potassium abnormalities can be the cause of CPA or can occur as a consequence of hypoxic–ischemic processes that evolve during CPR. Martin reported in dogs with experimental VF arrest and 5 minutes down time that serum potassium concentrations increased as CPR progressed (from 3.5 ± 0.4 to 4.6 ± 0.5 mmol/L after 5 min of CPR; $P < 0.001$), remained static until the end of CPR, and resolved rapidly after ROSC.[107] In addition, an experimental study in pigs documented a progressive increase in serum potassium concentration of nearly 10% per minute of CPR.[108] Changes in sodium concentrations are of less immediate concern as a much wider variation in extracellular sodium concentrations can be tolerated without fatal consequences, but fluid shifts can occur with rapid fluctuations in sodium concentrations, possibly leading to neurologic injury.[109] This question aims to explore whether routine assessment of potassium and sodium concentrations in dogs and cats undergoing CPR is warranted and if the potassium and sodium abnormalities should be addressed to improve outcome.

5.3.2 | Consensus on science

No studies were identified that directly compared the effect of measurement of sodium and potassium concentrations during CPR on *favorable neurologic outcome*, *survival to discharge*, or *ROSC*. However, multiple observational studies were identified that explored the association between blood potassium or sodium concentration and relevant outcomes and that thus provide indirect evidence to support or oppose the measurement of these electrolytes.

For the most critical outcome of survival with *favorable neurologic outcome*, 5 observational studies (very low quality of evidence, downgraded for serious risk of bias, and very serious indirectness) were evaluated. These studies examined the role of potassium concentration as a marker of severity of hypoxic-ischemic injury in populations of humans with OHCPA. Shin et al evaluated registry data from 2229 OHCPA patients analyzing the first electrolyte analysis examined at admission and found that none of the patients with blood potassium concentrations >8.5 mmol/L survived with good neurological function.[110] Torres and colleagues prospectively studied functional outcomes in 1552 nontraumatic OHCPA patients.[111] Venous blood samples collected with initial vascular access showed that blood potassium concentrations were significantly lower in those patients with favorable neurologic outcome (CPC 1–2; $[K^+] = 3.95 \pm 0.79$ mmol/L) compared to those with CPC 3–5 ($[K^+] = 4.49 \pm 1.21$ mmol/L; OR: 1.725, 95% CI: 1.506–1.977). Yanagawa et al conducted a medical chart review including nontraumatic OHCPA patients admitted to an emergency department ($n = 132$) and similarly showed significantly lower blood potassium concentration in patients with favorable neurologic outcome (CPC1–2: $[K^+] = 4.2 \pm 3.0$ mmol/L vs CPC 3–5: $[K^+] = 5.6 \pm 1.6$ mmol/L).[112] Choi analyzed data from a trial registry including 914 OHCPA patients with ongoing CPA or CPR in which electrolyte information was available.[113] After adjusting the covariates, hypokalemia was associated with favorable neurologic outcome (OR: 4.45, 95% CI: 1.67–11.91), while hyperkalemia had no statistically significant association with favorable neurologic outcome. Similar results were found by Shida in a prospective, multicenter observational study that included 1516 patients with OHCPA of cardiac origin and prehospital ROSC.[114] The population was grouped according to potassium concentrations at presentation to the emergency department into Q1 ($[K^+] \leq 3.8$ mmol/L), Q2 ($3.8 < [K^+] \leq 4.5$ mmol/L), Q3 ($4.5 < [K^+] \leq 5.6$ mmol/L), and Q4 ($[K^+] > 5.6$ mmol/L). One-month survival with favorable neurologic outcome (CPC 1–2) was best for Q1 (44.8%) and worst for Q4 (4.5%). The adjusted odds for survival with favorable neurologic outcome were 3 times worse for those patients in Q4 compared to those in Q1 (OR: 0.31, 95% CI: 0.15–0.66). Thus, taken together, higher blood potassium concentrations during CPR or immediately after ROSC were associated with worse outcomes.

The results of the studies reporting the next most important outcomes of *survival to discharge* (4 observational studies; very low quality of evidence, downgraded for serious risk of bias and very serious indirectness) [110,114–116] and *ROSC* (5 observational studies; very low evidence, downgraded for serious risk of bias and very serious indirectness)[98,107,111,116,117] are similar to those reported for favorable neurologic outcome. In 1 report of 8 cats and 16 dogs in which blood samples were collected during ongoing CPR, blood potassium and sodium concentrations in animals with and without ROSC were not different.[98] This is similar to 1 human paper that also found no association between intra-arrest blood sodium concentrations and survival to discharge,[110] but different from another retrospective observational study of human trauma victims undergoing open chest CPR, which found that patients who did not experience ROSC had higher serum sodium and potassium concentrations than those who did regain ROSC.[117]

Only 1 study was identified that examined the association of treatment on ROSC in patients with hyperkalemia. Wang and colleagues conducted a retrospective study including 109 adults with IHCPA and serum potassium concentration >6.5 mmol/L.[113] In patients with serum potassium concentrations between 6.5 and 7.9 mmol/L, sodium bicarbonate administration was significantly associated with sustained ROSC (OR: 10.51, 95% CI: 1.50–112.89). Likewise, calcium administration was significantly associated with sustained ROSC in patients with serum potassium concentrations between 6.5 and 9.4 mmol/L (OR: 51.11, 95% CI: 3.12–1639.16).

Five case reports in people describe CPA associated with severe hypokalemia ranging from 0.9 to 2.9 mmol/L, the administration of potassium, and its contribution to recovery.[118–122] Allen et al report a dog that developed CPA due to severe hypokalemia (1.5 mmol/L) associated with leptospirosis; this dog received periarrest potassium at 0.9–2.0 mmol/kg/h, IV, and survived to discharge with good neurologic function.[123] Five further case reports or case series document successful CPR in individuals with severe hyperkalemia due to kidney disease, rhabdomyolysis, and other conditions.[124–128] Treatment in these cases consisted of varying combinations of calcium gluconate, sodium bicarbonate, insulin, and dextrose administration, as well as hemodialysis in addition to standard basic and ALS measures.

5.3.3 | Treatment recommendations

We suggest measuring sodium and potassium concentrations in all dogs and cats during CPR (weak recommendation, very low quality of evidence).

We recommend measuring potassium concentrations as early as possible in dogs and cats during CPR in which severe potassium abnormalities are suspected (strong recommendation, expert opinion).

5.3.4 | Justification of treatment recommendations

Evidence suggests that hyperkalemia can develop during prolonged CPR, but in most cases, it does not require treatment. Accordingly, a weak recommendation is provided for the routine determination of potassium concentrations during CPR.

Multiple case reports suggest circumstances in which severe hypo- or hyperkalemia was associated with onset of CPA and in which aggressive intra-arrest measures were undertaken to correct the electrolyte disorders. Survival to discharge with a favorable neurologic outcome was achieved in these cases. There are multiple disease processes in dogs and cats that can lead to hyperkalemia, including acute kidney injury (AKI), urethral obstruction, reperfusion injury in cats with aortic thromboembolism, or accidental intravenous overdose with potassium. We therefore recommend the timely determination of blood potassium concentrations in cases in which a precipitating cause for hyper- or hypokalemia is known or suspected.

Very little evidence was found indicating sodium concentration (or treatment aimed at altering sodium concentration) was related to CPA outcomes; monitoring for meaningful sodium concentration derangement and treating any if found seems reasonable.

5.3.5 | Knowledge gaps

While moderate hyperkalemia has been noted with prolonged CPR, no studies have been conducted regarding the effectiveness of and the need for treatment of this hyperkalemia. In addition, it remains unclear whether there is an association between hypo- or hyperkalemia and survival to discharge or ROSC in veterinary species.

5.4 | Measuring calcium and treating dyscalcemias during CPR (MON-09)

In cats and dogs in CPA (P), does the identification and treatment of (arterial or venous) calcium disorders during CPR (I) compared to not addressing calcium disorders (C) improve favorable neurologic outcome, survival to discharge, or ROSC (O)?

5.4.1 | Introduction

Calcium is important for myocardial function and vasomotor tone. Alterations in plasma calcium concentrations during CPA could result in a decreased chance of ROSC and survival if adequate myocardial contractility cannot be generated to maintain spontaneous circulation. This PICO question investigated the question of whether management of plasma calcium concentration abnormalities during CPR impacts outcome.

5.4.2 | Consensus on science

No studies were identified that addressed the PICO question for any of the outcomes of interest. Studies were therefore examined to determine if hypocalcemia occurs during CPR and if those changes are associated with outcome, and also if administration of calcium during CPR is associated with improved outcomes.

Multiple studies have documented decreased plasma calcium concentrations in patients in CPA. Although the mechanism is unclear, there is some evidence that ionized calcium complexes with lactate as progressive lactic acidosis develop during CPA.[129] Gando et al showed a significant correlation between transport time to the hospital and plasma ionized calcium concentrations in 30 human patients with OHCPA, suggesting that decreases in ionized calcium concentrations in patients in CPA are progressive over time ($r = -0.371$, $P < 0.05$).[130] In a study of 22 patients with traumatic CPA and open-chest CPR, 12 (55%) had ionized hypocalcemia.[117] Multiple experimental studies in dogs and swine have documented statistically significant decreases in plasma ionized calcium during CPR that persist in the PCA period.[97,131–134] In a clinical retrospective study, ionized hypocalcemia was found in 18% of dogs and cats undergoing CPR (mean: 1.21 mmol/L, range: 0.68–1.59 mmol/L, reference interval: 1.1–1.5 mmol/L).[98]

Two observational studies in people investigated the association between serum calcium concentrations and outcomes in human patients in CPA.[111,135] In a study of 1552 adults with OHCPA, ionized calcium concentration at catheter placement was not associated with neurologic outcome.[111] Univariate analysis of these data showed that plasma ionized calcium concentrations at catheter placement were

associated with ROSC (OR of no ROSC 0.580 per mmol/L, 95% CI: 0.347–0.971), but in a multivariate analysis, only pH and P_{CO_2} (but not ionized calcium) showed an independent association with ROSC. In a retrospective observational study of 33 adults who achieved ROSC, the lowest serum ionized calcium concentration in the first 48 hours after ROSC was lower in patients with poor neurologic outcome than in patients with good neurologic outcome (0.96 ± 0.06 vs 1.02 ± 0.06 mmol/L).[135] Neither of these studies investigated the effect of treating hypocalcemia on outcome.

The routine administration of calcium during CPR has not been shown to improve the frequency of ROSC,[117,130,136–141] favorable neurologic outcome,[111] or survival to hospital discharge.[137,142] In addition, some studies have documented increased mortality in patients in CPA treated with calcium.[143,144] However, 2 studies have demonstrated improved outcomes in hyperkalemic humans in CPA treated with calcium.[113,145]

Early studies suggested some benefit to the administration of calcium to adults and animals during CPR, but the evidence overall does not support routine administration of calcium to patients in CPA. A retrospective study including 480 people with OHCPA showed that calcium administration was associated with ROSC in patients with pulseless electrical activity (PEA; 11% developed a perfusing rhythm after administration) but not in patients with asystole or ventricular fibrillation.[146] In contrast, another clinical trial in patients with OHCPA and PEA found a similar frequency of ROSC in patients who received calcium (8/48) to those who did not receive calcium (2/42).[147] In addition, a retrospective study including 210 people with asystole or PEA reported a lower frequency of ROSC in those who received calcium (8% in those with asystole and 16% for those with PEA) in a prehospital setting compared to those who did not (33% for those in asystole, 44% for PEA).[138] Another prospective, randomized, masked clinical trial of 70 people with OHCPA found that calcium chloride administered during refractory asystole did not improve the rate of ROSC (5.1% in calcium group vs 14.7% in saline group, $P = 0.37$).[148] A multicenter, randomized, controlled clinical trial including 773 people experiencing IHCPA showed that calcium administration during CPR was significantly associated with nonsurvival at 1 hour after ROSC (17.3% nonsurvivors vs 7.1% survivors). Likewise, an experimental study in 16 dogs with PEA showed that calcium administration during CPR was not more effective than placebo in achieving ROSC (4/11 in calcium group, 4/9 placebo group).[149] A larger experimental study in 40 dogs with PEA confirmed that calcium administration during CPR was not more effective than placebo for the achievement of ROSC (6/10 vs 2/10).[141]

5.4.3 | Treatment recommendations

In dogs and cats in CPA, we suggest monitoring of plasma ionized calcium during CPR (weak recommendation, expert opinion).

In dogs and cats in CPA with documented hypocalcemia (ionized calcium <0.8 mmol/L), we suggest administration of 10% calcium gluconate (50 mg/kg, IV or IO, over 2–5 min) or 10% calcium chloride (15 mg/kg, IV or IO, over 2–5 min) (weak recommendation, expert opinion).

In dogs and cats in CPA, we recommend against routine administration of calcium in patients in the absence of documented hypocalcemia or other specific indications (eg, calcium channel blocker overdose) (strong recommendation, very low quality of evidence).

5.4.4 | Justification of treatment recommendations

There is limited evidence that ionized hypocalcemia impacts outcomes in people with CPA, and there are no studies in dogs or cats evaluating the impact of hypocalcemia during CPR on outcomes. However, plasma ionized calcium plays crucial roles in maintaining vascular tone and cardiac contractility, and it is likely that significant ionized hypocalcemia could reduce the likelihood of ROSC. Therefore, the committee suggests that measurement of plasma ionized calcium concentrations may be useful during CPR and suggests that substantial ionized hypocalcemia (<0.8 mmol/L) be treated with intravenous calcium.

5.4.5 | Knowledge gaps

A placebo-controlled clinical trial of treatment of hypocalcemia in dogs and cats during CPR is considered by the committee to be a moderate-level knowledge gap. To date, there are no trials in humans or animals investigating the potential outcome benefits of treatment of documented hypocalcemia during CPA.

5.5 | Measuring creatinine after ROSC (MON-03b)

In cats and dogs that have experienced ROSC after CPA (P), does measurement of creatinine (I), as opposed to nonmeasurement (C), improve favorable neurologic outcome, survival to discharge, duration of postarrest hospitalization, or prediction of recurrent CPA (O)?

5.5.1 | Introduction

AKI is common in patients experiencing ROSC as a consequence of prolonged hypoxemia and hypoperfusion, as well as ischemia–reperfusion injury.[150] In both survivors of CPA and many critically ill populations, AKI is considered an adverse outcome that can ultimately affect duration of hospitalization, survival to discharge, and long-term prognosis, independent of underlying cause. This PICO question addressed the utility of postarrest creatinine measurement to improve outcome after ROSC.

5.5.2 | Consensus on science

For the most critical outcome of *favorable neurologic outcome*, 6 observational studies in people (very low quality of evidence, downgraded for very serious indirectness and serious inconsistency) and 1

experimental study in dogs (very low quality of evidence, downgraded for very serious indirectness and serious imprecision) were identified that were relevant to the PICO question.[81,150–154] Four of the 5 observational studies in people documented a lower frequency of favorable neurologic outcome in patients developing AKI in the PCA period. One prospective observational study showed that post-CPA patients with less favorable neurologic outcome had significantly higher rates of AKI than those with favorable neurologic outcome (64% vs 38%).[151] Another observational study of adults in the PCA period showed an OR for poor neurologic outcome of 3.81 (95% CI: 1.98–7.33, $P = 0.0001$) for patients with static or increasing plasma creatinine concentrations 24 hours after ROSC, compared to those whose creatinine decreased during the first 24 hours.[152]

Tamura et al used admission creatinine to estimate glomerular filtration rate in a prospective observational registry study of 5112 people with cardiogenic OHCPA and showed that ORs for favorable neurologic outcome decreased as glomerular filtration rate decreased.[154] Isenschmid et al did a multivariate analysis of admission creatinine concentration and demographic data including co-morbidities in adults with ROSC and found an aOR for poor neurologic outcome of 44.33 (95% CI: 6.99–281.36).[81] In 282 children with ROSC, severe AKI was associated with worse neurologic function in a study evaluating the utility of therapeutic hypothermia in the PCA period.[155] The only observational study in people that did not show an association between AKI and favorable neurologic outcome was a retrospective study of 199 people that showed no difference in neurologic outcomes 3 months post-ROSC between patients with evidence of AKI (85/199, 43%) and those without evidence of AKI.[150] An experimental study in dogs investigated gender-specific differences in 24-hour outcome following ROSC after 9 minutes of untreated cardiac arrest.[153] Neurologic outcome was not significantly different between male and female dogs; however, serum creatinine concentrations progressively increased at 6, 12, and 24 hours after ROSC in female dogs.

For the next most critical outcome of *survival to discharge*, 9 observational studies in people were identified that addressed the PICO question (very low quality of evidence, downgraded for very serious indirectness, serious imprecision, and serious inconsistency).[154–162]

In a clinical trial evaluating the utility of therapeutic hypothermia in children after ROSC, 64% of the children developed AKI, of which 41% developed more severe AKI and 3.5% required renal replacement therapy.[155] Severe AKI was associated with lower survival at 28 days and at 12 months post-ROSC. Several other studies in adults supported these findings, with the majority of patients who developed AKI after ROSC experiencing higher in-hospital mortality and lower survival rates over time after discharge.[150,154,157–161] The incidence of AKI after cardiac arrest was over 40% in 1 retrospective analysis, and these patients displayed severe hemodynamic impairment and required more intensive post-CPA interventions.[150] Another study showed that serum creatinine concentrations at admission following OHCPA were higher in patients who ultimately developed AKI, and 30-day mortality was higher in patients with more advanced (stage 3) AKI compared to those with less severe kidney injury.[161]

There is some evidence in adults that development of AKI (based on the Risk, Injury, and Failure; Loss; and End-stage kidney disease [RIFLE] scoring system) is not independently associated with survival to discharge; however, renal injury is common, with more than one third of patients resuscitated from OHCPA in a large retrospective study developing evidence of renal dysfunction and 19% of those patients meeting the criteria for AKI.[162] Both initial serum creatinine concentrations or low urine output were predictive of development of AKI, and peak serum creatinine concentrations were typically identified during the first 72 hours after ROSC in this population.[162] In this population, the duration of shock after ROSC was related to the development of AKI in the PCA period. The median duration of shock within the first 24 hours after ROSC was 10 minutes (range: 2–59 min) in the non-AKI group compared to 130 minutes (range: 48–250 min) in patients who developed AKI.[162] Other measured parameters such as the time from arrest to ROSC, duration of CPR, and serum lactate concentration immediately after ROSC were not associated with development of AKI.

No studies were identified that directly addressed the impact of serum creatinine measurement on the *prediction of recurrent CPA* or the *duration of postarrest hospitalization*.

5.5.3 | Treatment recommendation

We recommend measuring serum creatinine concentrations, as an indicator of AKI, as soon as feasible in the PCA period and subsequently no less often than every 24 hours during hospitalization in dogs and cats that achieve ROSC (strong recommendation, very low quality of evidence).

5.5.4 | Justification of treatment recommendation

There is consensus in human medicine that PCA AKI is associated with worse outcomes, including increased risk of death and decreased frequency of favorable neurologic outcome. Therefore, identification of AKI in dogs and cats in the PCA period, especially if the kidney injury is progressive, may have prognostic value. Despite the frequent measurement of renal values in clinical practice, there is a paucity of literature on serum creatinine measurement and AKI in dogs and cats experiencing ROSC after CPA. Two experimental studies were reviewed, 1 in pigs and 1 in dogs, which provided support for measurement of serum creatinine concentration in animals after CPR and ROSC.[97,163] In an experimental pig model of cardiac arrest, serum creatinine was higher for subjects that experienced CPA when compared to the control group of pigs who did not experience CPA.[163] Similarly, in a canine model of cardiac arrest, serum creatinine concentrations were significantly higher both during CPR and after ROSC, with creatinine increasing approximately 11% and 29% from baseline, respectively.[97] Neither study addressed the outcomes evaluated by this particular question, and there was no comparison of measurement versus no measurement in study participants.

There have been no studies of the utility of management of AKI guided by serial measurement of creatinine concentration to improve outcomes in the PCA period, but monitoring for evidence of development or progression of AKI in the PCA period and adjusting management strategies to address kidney injury are likely to be of benefit to these patients.

5.5.5 | Knowledge gaps

Currently, there is no evidence in dogs and cats that serum creatinine concentration increases in dogs and cats in the PCA period or that increased serum creatinine during CPR or after ROSC is associated with decreased survival to discharge or poor neurologic outcomes. Studies to identify patient factors associated with increased risk of AKI in dogs and cats following ROSC, optimal frequency of serum creatinine measurement, and a determination of whether severity and duration of postarrest kidney injury has an effect on outcome of these patients are warranted.

6 | MONITORING PATIENTS AT RISK OF CPA

With a knowledge of the common conditions that predispose to CPA, the identification of specific monitoring parameters in at-risk individuals may alert caregivers to impending CPA or may speed response time in the event of CPA. Because vital monitors are generally available in veterinary clinics, this series of PICO questions was designed to investigate and inform their place in the monitoring of at-risk patients.

6.1 | ECG monitoring in patients at risk of CPA (MON-06)

In dogs and cats at risk of CPA (eg, under anesthesia, in shock, in respiratory distress, post-ROSC) (P), does ECG monitoring (I) compared to no ECG monitoring (C) improve favorable neurologic outcome, survival to discharge, ROSC, time to start CPR, or time to identification of CPA (O)?

6.1.1 | Introduction

Continuous ECG monitoring may be observed directly or remotely and can alert nurses and doctors to the presence of arrhythmias that may be associated with CPA. Because ECG devices and monitoring capability can be limited in some hospitals, this query was designed to determine if ECG monitoring in patients at risk of CPA affected the outcomes of interest.

6.1.2 | Consensus on science

For the most critical outcome of *favorable neurologic outcome*, 1 retrospective, registry-based observational study in people was identified (very low quality of evidence, downgraded for very serious indirectness and serious imprecision).[164] In 6789 patients who developed CPA due to a shockable arrest rhythm, the aOR for favorable neurologic outcome among patients defibrillated more than 2 minutes after developing CPA was 0.74 (95% CI: 0.56–0.96), suggesting that more rapid time to defibrillation is important. The OR for delayed defibrillation in patients managed in an inpatient unit with telemetry was 0.47 (95% CI: 0.41–0.53), suggesting that patients with continuous telemetry-based ECG monitoring were more likely to have prompt defibrillation and, by association, improved favorable neurologic outcome.

For the next most critical outcome of *survival to discharge*, 6 observational studies in people were identified that addressed the PICO question (very low quality of evidence, downgraded for very serious indirectness and serious imprecision).[164–169] In human hospitals, hospitalization in an unmonitored ward is associated with lower frequency of survival to discharge after IHCPA.[164,165] One particular at-risk group that experienced IHCPA secondary to aspiration pneumonia had a lower frequency of survival to discharge when CPA occurred in an unmonitored area.[165] The primary factor leading to the decision to implement continuous ECG monitoring in human patients was ongoing myocardial infarction, which may limit the applicability of these findings to dogs and cats.[166] These studies did not directly compare ECG monitoring to no ECG monitoring, however, and most patients with ECG monitoring were housed in wards with nurses with more expertise caring for higher acuity patients, which may have influenced success of CPR interventions. Other studies in people support an increased chance of survival to hospital discharge following IHCPA and resuscitation in patients housed in areas that are better monitored (including, but not limited to, continuous ECG) compared to IHCPA in locations where the level of monitoring was lower and continuous ECG monitoring was absent.[166–168] Two observational studies found that continuous ECG monitoring by a central station did not improve survival to discharge following IHCPA.[169,170] The Mohammed study specifically evaluated the use of continuous telemetry monitoring in noncritically ill patients.[169] The variability of quality of CPR is not directly addressed in these studies, although the location of many monitored patients in higher acuity wards likely results in some bias regarding familiarity of the attending nurses and physicians with CPR protocol.

Four observational studies in people were identified that informed the answer to this PICO question for *ROSC* (very low quality of evidence, downgraded for very serious indirectness and serious imprecision).[164,165,169,170] Two observational studies in people specifically evaluated ECG monitoring and ROSC for patients with IHCPA, finding that continuous ECG monitoring was associated with an increased frequency of ROSC.[164,170] In addition, 1 observational study in people evaluating patients with aspiration pneumonia compared to patients with respiratory failure due to other causes found that the latter group was more frequently monitored with continuous ECG and had a higher frequency of ROSC than patients with aspiration pneumonia.[165] Only 1 observational study in noncritically ill patients found no effect of continuous monitoring with telemetry on ROSC.[169]

Because substantial evidence was available to inform an answer to this PICO question for the 3 most critical outcomes, only a cursory evaluation of the literature for the 2 least critical outcomes (*time to start CPR*, *time to identify CPA*) was done. Many of the studies cited

above also demonstrated decreased time to diagnosing CPA and initiating BLS and ALS interventions in patients with continuous ECG monitoring.[164,165,167] In pediatric populations, however, ECG monitoring may not reliably identify all CPA events. In 1 study, children with an arrest rhythm of PEA experienced a significant delay from onset of CPA to start of CPR compared to infants with ECG diagnoses of either bradycardia or ventricular fibrillation.[171,172] In experimental studies in dogs subjected to uncoupling of electrical and mechanical heart function and pigs subjected to asphyxial arrest, the ECG indicated either no change or bradyarrhythmias for a period of time (4–6 min) without a perfusing rhythm, highlighting the major problem with sole reliance on ECG monitoring to diagnose CPA in certain scenarios.[173,174] In a study of pediatric CPA, other monitored parameters such as blood pressure and pulse oximetry more accurately diagnosed CPA than the ECG.[171] However, another study showed that diagnosis of bradycardia in preterm infants was slower with a pulse oximeter than an ECG.[175]

6.1.3 | Treatment recommendation

We recommend continuous ECG monitoring in dogs and cats at risk of CPA (eg, under anesthesia, in shock, in respiratory distress, post-ROSC, aspiration risk) (strong recommendation, very low quality of evidence).

6.1.4 | Justification of treatment recommendation

Although much of it is indirect, there is substantial evidence of better outcomes in patients with IHCPA who have continuous ECG monitoring in place at the time of the CPA. However, ECG monitoring alone is not adequate to diagnose CPA in patients with PEA or pulseless ventricular tachycardia, and other monitoring (such as ABP, pulse oximetry, and $ETCO_2$ monitoring) may provide important additional information to increase the sensitivity of the monitoring plan for CPA.

6.1.5 | Knowledge gaps

There are no veterinary studies in hospitalized dogs and cats investigating the utility of ECG monitoring for the diagnosis of CPA in at-risk populations. In addition, there are limited data available to identify patient subsets that would benefit most from continuous ECG monitoring.

6.2 | ABP monitoring in patients at risk of CPA (MON-04)

In dogs and cats at risk of CPA (eg, under anesthesia, in shock, in respiratory distress, post-ROSC) (P), does blood pressure monitoring (I) compared to no blood pressure monitoring (C) improve favorable neurologic outcome, survival to discharge, ROSC, time to identify CPA, or time to start CPR (O)?

6.2.1 | Introduction

Systemic ABP monitoring is frequently performed in veterinary and human hospitals. Serial blood pressure measurements allow early recognition of clinical deterioration and provide the clinician with important patient care information that may ultimately affect the quality of care provided. This PICO question investigated the use of blood pressure monitoring in patients at risk of CPA to improve outcomes.

6.2.2 | Consensus on science

For the most critical outcome of *favorable neurologic outcome*, 1 observational study in people who achieved ROSC after OHCPA was identified (very low quality of evidence, downgraded for very serious indirectness and serious imprecision).[176] This study included 11,352 patients with OHCPA who achieved ROSC and were admitted to the hospital. Of those, 7706 had indirect SBP measured in the prehospital setting. Both hypotension and hypertension were associated with decreased favorable neurologic outcome in this cohort. Relative risks for poor neurologic outcome were 1.069 (95% CI: 1.033–1.105) for SBP of 80–99 mm Hg, 1.203 (95% CI: 1.158–1.243) for SBP of <80 mm Hg, 1.076 (95% CI: 1.043–1.110) for SBP of 130–160 mm Hg, and 1.168 (95% CI: 1.126–1.208) for SBP of >160 mm Hg.

For the next most critical outcome of *survival to discharge*, 1 observational study in people was identified that informed an answer to the PICO question (very low quality of evidence, downgraded for very serious indirectness and serious imprecision).[177] This retrospective registry study evaluated the association between SAP on arrival to the hospital and survival to discharge in 3620 adult people surviving OHCPA. Among the subjects, 14% were hypotensive at hospital admission. In the patients with an initial shockable rhythm, hypotension was associated with less frequent survival to discharge. In these patients, SAP <90 mm Hg was associated with significantly decreased aOR of survival to discharge: 80–89 mm Hg aOR = 0.49 (95% CI: 0.24–0.95); <80 mm Hg aOR = 0.24 (95% CI: 0.10–0.61); unrecordable aOR = 0.10 (95% CI: 0.04–0.30). However, in patients with initial nonshockable rhythms, SAP was not associated with survival to discharge.

For the next most critical outcome of *ROSC*, no relevant studies were identified to inform an answer to the PICO question.

For the next important outcome of *time to identification of CPA*, 1 clinical trial of simulated anesthetic events in people (very low quality of evidence, downgraded for very serious indirectness and serious imprecision) and 1 observational study in people were identified (very low quality of evidence, downgraded for very serious indirectness).[178,179] The clinical trial included 58 senior anesthesiology residents randomized to interpret either invasive blood pressure monitoring or noninvasive blood pressure monitoring information during a simulated CPA. The group with access to invasive blood pressure monitoring palpated for pulses in the patient 6.5 seconds sooner and initiated chest compressions 17 seconds sooner than the group with indirect blood pressure monitoring. The observational study evaluated 269,956 adults admitted to hospital wards, 422 of which developed

CPA.[179] The analysis showed that the lowest SAP and DBP measured in the 4 hours prior to CPA were lower than those measured in patients who did not experience CPA, but no specific blood pressure cutoffs were reported.

For the outcome of *time to start CPR*, 1 clinical trial of simulated anesthetic events in people (very low quality of evidence, downgraded for very serious indirectness and serious imprecision) addressed the PICO question.[178] As described above, the group with access to invasive blood pressure monitoring information initiated chest compressions 17 seconds sooner than the group with only indirect blood pressure monitoring.

6.2.3 | Treatment recommendations

We recommend frequent or continuous ABP monitoring in patients at risk of CPA, including patients under anesthesia, in shock, and in the PCA period (strong recommendation, very low quality of evidence).

We suggest the use of continuous, direct ABP monitoring if feasible in patients at risk of CPA (weak recommendation, very low quality of evidence).

6.2.4 | Justification of treatment recommendations

The reviewed evidence supports the utility of blood pressure monitoring to rapidly diagnose CPA in high-risk patients. Direct, continuous ABP monitoring allows faster recognition of CPA and initiation of CPR, and so is preferred over intermittent noninvasive monitoring if feasible.[178]

A single veterinary study was evaluated in which the relationship between Doppler blood pressure (DopBP) and survival or response to treatment in critically ill cats was investigated.[180] This was a retrospective study of 83 cats, and there was a significantly higher frequency of mortality in hypotensive cats.[180] In this report, hypotensive critically ill cats with an increase in DopBP of at least 20 mm Hg during hospitalization were more likely to survive to discharge when compared to cats in which the difference in DopBP was <20 mm Hg (survival rates 69% and 17%, respectively). No data were available related to the relationship between severity of hypotension, frequency of monitoring, or incidence of CPA, but the study provides some additional justification for blood pressure monitoring in critically ill cats.

A retrospective observational study evaluated the association of blood pressure monitoring with adverse outcomes in people admitted to an emergency department.[181] Worsening of clinical signs was associated with decreases in SAP and DBP values; however, there was no association between BP and length of hospital stay, repeat admission to the hospital, or CPA.

Human studies have also used the Modified Early Warning Score to identify high-risk ward patients and trigger rapid response teams in cardiac arrest.[179,182] The Modified Early Warning Score system incorporates SAP measurements, as well as heart rate (HR), respiratory rate (RR), body temperature, and level of consciousness to provide a warning score for clinical deterioration. Use of this scoring system has provided some evidence that patients with lower SAP and DBP have a higher risk of CPA.[179]

6.2.5 | Knowledge gaps

There have been no studies in dogs or cats examining the utility of blood pressure monitoring in patients that ultimately develop CPA. Given the minimal risk of blood pressure monitoring and the likelihood that early identification of hypotension in patients at risk of CPA is useful, the committee suggests that further studies of blood pressure monitoring in these scenarios are a low priority.

6.3 | Pulse oximetry monitoring in patients at risk of CPA (MON-05)

In dogs and cats at risk of CPA (eg, under anesthesia, in shock, in respiratory distress, post-ROSC) (P), does pulse oximetry monitoring (I) compared to no pulse oximetry monitoring (C) improve favorable neurologic outcome, survival to discharge, ROSC, time to identification of CPA, or time to start CPR (O)?

6.3.1 | Introduction

Pulse oximetry is used to report arterial oxygen saturation of hemoglobin and thus estimate Pa_{O_2} in dogs and cats. Most pulse oximeters also provide an auditory signal indicating pulse detection. Because hypoxemia, resultant desaturation, and diminishing perfusion can precipitate CPA, pulse oximetry monitoring of at-risk animals may prompt caregivers to intervene prior to the occurrence of CPA. This PICO question was designed to determine whether monitoring dogs and cats at risk of CPA with pulse oximetry could improve outcome.

6.3.2 | Consensus on science

For the most critical outcome of *favorable neurologic outcome*, we identified 1 observational study in people[176] (very low quality of evidence, downgraded for very serious indirectness and serious imprecision) and 2 experimental studies in dogs[183,184] (very low quality of evidence, downgraded for very serious indirectness and serious imprecision) that addressed the PICO question.

A single registry-based observational study of OHCPA in 9405 adults showed that in people who achieved ROSC and were monitored with Spo_2 (oxygen saturation percentage as measured by pulse oximetry) in the prehospital PCA phase, Spo_2 <94% was associated with worse favorable neurologic outcome at 30 days (RR: 1.108, 95% CI: 1.069–1.147) and having an Spo_2 of 99%–100% did not appear to be harmful (RR 0.9851, 95% CI: 0.956–1.015). Multivariate analysis of 4897 subjects confirmed that Spo_2 <94% portended a poorer

favorable neurologic outcome prognosis (aOR: 1.39, 95% CI: 1.02–1.89) and that SpO_2 >98% was not a risk factor for poor favorable neurologic outcome (aOR: 0.85, 95% CI: 0.70–1.03).[176]

An experimental model of fibrillatory arrest in dogs showed that titrating oxygen supplementation following ROSC to an SpO_2 of 94%–96% for 1 hour resulted in improved favorable neurologic outcome and fewer histopathologic brain lesions at 24 hours when compared to supplementation of a fractional inspired oxygen (FiO_2) of 1.0 without titration.[184] A study with similar methodology evaluated brain histopathology at 24 hours post-ROSC and found more severe histopathologic lesions in the animals that had not received oximetry-directed oxygen titration following ROSC.[183] These experimental studies suggest that pulse oximetry monitoring may be useful in the postarrest period to help avoid hyperoxemia, which may improve favorable neurologic outcome.

For the next most critical outcome of *survival to discharge*, we identified 1 clinical trial[185] (very low quality of evidence, downgraded for serious risk of bias and very serious indirectness) and 5 observational studies[186–190] (very low quality of evidence, downgraded for very serious risk of bias and serious indirectness).

One randomized, nonblinded clinical trial designed specifically to evaluate the utility of pulse oximetry for influencing the frequency of ICU transfer from a 33-bed post-cardiothoracic surgery care ward evaluated 1219 adults and found no difference in survival to discharge whether people were monitored continuously with pulse oximetry following surgery or not, although patients with continuous SpO_2 monitoring were likely to be transferred sooner after surgery than those with standard monitoring.[185]

One retrospective case–control study in 237 dogs and 181 cats showed that absence of recording SpO_2 values during general anesthesia in cats was associated with death within 7 days; the same was not found for dogs.[186] Another retrospective case–control study in 635 dogs found no association between the use of a pulse oximeter for anesthetic monitoring and occurrence of death during or within 48 hours of the anesthetic procedure.[187] A retrospective case–control study in 730 cats found that cats that had "pulse or pulse oximetry" monitoring during the procedure were 3–4 times less likely to die than those that did not (OR: 0.2, 95% CI: 0.1–0.4).[188] Oximetry monitoring was used in conjunction with other vital signs monitoring in all of these studies.

A large retrospective cohort study in 96,512 people showed that differences in various vital signs including oxygen saturation were independently associated with 1-day mortality (aOR: 5.2, 95% CI: 3.1–9.0 for short-term death in patients with SpO_2 <90% despite supplemental oxygen therapy compared to those with SpO_2 >95%). However, in this report, bradypnea (aOR: 18.1, 95% CI: 2.1–155.5) and tachypnea (aOR: 4.9, 95% CI: 3.4–7.3) had stronger or nearly as strong associations with 1-day mortality as SpO_2.[189] Similar findings were noted regarding 30-day mortality (aOR: 3.7, 95% CI: 2.8–5.0 for patients with SpO_2 <90% receiving supplemental O_2 compared to those with SpO_2 >95%; aOR: 3.1, 95% CI: 2.6–3.6 for tachypnea).[189]

A smaller retrospective study of 358 people experiencing IHCPA in a single hospital showed that the National Early Warning Score (NEWS), which includes pulse oximetry monitoring, could be used to predict survival at 30 days following CPA (medium NEWS value [OR: 4.43, 95% CI: 1.81–10.83] or high NEWS value [OR: 9.88 95% CI: 2.77–35.26] for nonsurvival at 30 days compared to patients with a low NEWS value).[190] Oximetry monitoring was used in conjunction with other vital signs monitoring in this study as well.

No studies were identified for the critical outcome of *ROSC*.

For the next critical outcome of *time to identification of CPA*, 2 clinical trials[185,191] (very low quality of evidence, downgraded for very serious indirectness and serious inconsistency), 6 observational studies[192–197] (very low quality of evidence, downgraded for very serious indirectness and serious inconsistency), and 5 experimental studies[198–202] (very low quality of evidence, downgraded for very serious indirectness) were identified that addressed the PICO question.

One clinical trial in people demonstrated that rescue events decreased from 3.4 ± 2.2 (95% CI: 1.89–4.85) to 1.2 ± 0.94 (95% CI: 0.53–1.88) per 1000 patient discharges after implementation of continuous pulse oximetry monitoring of postoperative patients in a general surgery ward.[191] Another randomized, nonblinded clinical trial designed specifically to evaluate the utility of continuous pulse oximetry for postoperative monitoring in 1219 adults in a postsurgical unit showed no difference in transfer frequency from the unit into the ICU whether patients had SpO_2 monitoring or not.[185] An observational study of hospitalized people found that 3816 out of 13,115 (29.1%) patients experienced SpO_2 <80% in the 1 hour prior to IHCPA.[195] Likewise, in a hospitalized pediatric population, oxygen desaturation was noted in 32% of 3647 medical emergency team events, with 6.1% (223 events) progressing to acute respiratory compromise and 0.5% (17 events) progressing to CPA. In this report, details of the cases that progressed to CPA were not specified.[194]

One observational study of 2179 people seen in an emergency department showed that of 551 people who were "up-triaged" due to worsening health status, 489 (88.7%) had an increased RR, and 539 (97.8%) had an increased RR or HR. Only 12 cases (2.2%) had normal RR and HR and were up-triaged only due to abnormal SpO_2. This observational, descriptive study concluded that RR and HR were more impactful than SpO_2 in determining patient status.[196] One retrospective study in 1980 hospitalized people similarly found that SpO_2 values recorded in the emergency department did not distinguish which patients would require rapid response team activation in the first 72 hours of hospitalization, while RR (aOR: 1.92, 95% CI: 1.38–2.67) was the variable most strongly associated with rapid response team activation.[192]

An observational study in 50 people following major abdominal surgery found that continuous SpO_2 monitoring was superior for detection of desaturation events compared to intermittent SpO_2 evaluation. In this study, events of SpO_2 <92% with a duration of more than 60 minutes were observed in 58% of patients.[197] These results were mirrored by a retrospective study of 833 postoperative human patients, which found that although 37% experienced prolonged (≥1 h) episodes of SpO_2 <90% and 11% experienced at least 1 episode lasting ≥6 h, clinical hypoxemia was estimated to have occurred in only 5% of patients per the every 12-hour medical record.[193]

In a human study of experimentally induced upper airway obstruction, SpO_2 was unreliable in reflecting upper airway airflow limitation; instead, RR, a visual analogue scale, and a dyspnea scale were statistically correlated with upper airway airflow limitation.[202]

One experimental rabbit study that evaluated the SpO_2 during progressive graded blood loss documented normal-appearing SpO_2 tracings that were present until the MAP had dropped to 44 mm Hg, indicating that SpO_2 is not a reliable indicator of hypotension in this setting.[198] Similarly, an experimental study in swine showed increased bias as well as poor accuracy and precision of pulse oximetry during periarrest hypoxemia.[201]

Another experimental swine study found that SpO_2 monitoring failed to detect 3 minutes of complete upper airway obstruction in pigs pretreated with 100% oxygen;[200] however, an experimental study in dogs spontaneously breathing room air demonstrated that pulse oximetry is useful in this setting to detect tracheostomy tube obstruction greater than 25%.[199] This study also determined that the greater the degree of obstruction, the shorter the time required for SpO_2 decline.[199]

No studies were identified that addressed the endpoint of pulse oximetry monitoring on the *time to start CPR*.

6.3.3 | Treatment recommendations

In dogs and cats at risk of CPA (eg, under anesthesia, in shock, in respiratory distress, post-ROSC), we recommend against monitoring only with a pulse oximeter (strong recommendation, very low quality of evidence).

In dogs and cats at risk of CPA (eg, under anesthesia, in shock, in respiratory distress, post-ROSC), we suggest continuous pulse oximetry monitoring in conjunction with continuous or frequent monitoring of other vital parameters such as RR, HR and rhythm, and ABP (weak recommendation, very low quality of evidence).

In cats under general anesthesia, we recommend continuous monitoring of pulse oximetry or pulse quality (strong recommendation, very low quality of evidence).

In dogs and cats in which a pulse oximetry reading cannot be obtained and patient movement and nonpatient factors are ruled out as the cause, we recommend assessment of perfusion status by other means (eg, pulse palpation, blood pressure measurement, ECG monitoring, apnea monitoring, plasma lactate concentration measurement, point-of-care cardiac ultrasound) (strong recommendation, expert opinion).

6.3.4 | Justification of treatment recommendations

There is variable evidence in hospitalized people that pulse oximetry can be used to alert rescue teams to potentially critical situations. Continuous pulse oximetry can alert caregivers to the presence of brady- or tachyarrhythmias, although ECG monitoring is likely superior for this indication (and potentially less likely to be displaced than a pulse oximeter probe in veterinary patients). In human and veterinary patients receiving supplemental oxygen, meaningful respiratory events (eg, upper airway obstruction, apnea) may be missed with a pulse oximeter while PaO_2 is still adequate; however, it is able to more rapidly identify desaturation in patients breathing room air. Pulse oximetry is not a good indicator of hypotension, and so other monitoring should be used in patients where this is likely to be of concern.

There was no clear evidence in awake veterinary species regarding pulse oximetry monitoring in pre- or postoperative patients with regard to the PICO question. In anesthetized cats, however, record of pulse oximetric monitoring was associated with a survival benefit compared to no such documentation. While this was not seen in a similar survey in dogs, no harm was associated with pulse oximeter usage in the canine population, and the low barrier to application of the pulse oximeter may be useful in either species.

6.3.5 | Knowledge gaps

The utility of continuous or intermittent pulse oximetry monitoring for identification of impending or occurring CPA in dogs and cats in a clinical setting is unknown.

The design of the currently available pulse oximeter probes does not make them amenable to continuous monitoring of awake veterinary patients; development of probes that can be used in a continuous manner in veterinary species is encouraged.

7 | DISCUSSION

The continuous measurement of $ETCO_2$ continues to be a strong recommendation during CPR efforts. In addition to an increase in the targeted $ETCO_2$ value that is associated with ROSC (18 mm Hg), other uses for this monitoring device are highlighted. $ETCO_2$ is unaffected by patient motion (unlike ECG monitoring), and so it can be used interactively during CPR to alert rescuers to either successful or less effective CPR interventions. A number of studies were reviewed that indicated that $ETCO_2$- guided CPR was superior to CPR quality assessed by more traditional means. A strong recommendation despite very low quality of evidence was produced for a targeted $ETCO_2$ of no less than 18 mm Hg in dogs and cats undergoing CPR. A lower $ETCO_2$ despite apparently good quality chest compressions should prompt urgent conversations about additional therapeutics that may be required to treat hypovolemic, distributive, or obstructive shock (eg, administration of IV or IO fluids or vasopressors; open-chest CPR).

We also investigated the utility of $ETCO_2$ to confirm proper ETT placement at initiation of CPR. Evidence suggests that while $ETCO_2$ presence is consistent with correct endotracheal intubation, lack of CO_2 in the low blood flow state of CPR does not necessarily mean the ETT is misplaced. While an $ETCO_2$ of $\geq$12 mm Hg likely indicates proper ETT placement, an $ETCO_2$ of <12 mm Hg should lead the rescuer to confirm tracheal intubation by other means, such as by direct laryngeal visualization or auscultation during a breath in the planned pause

between 2-minute chest compression cycles. If at any time during CPR the $ETCO_2$ is 0 or very low (eg, <5 mm Hg) despite high-quality chest compressions, rescuers should confirm proper intubation by other means.

Although electrolyte disturbances are common in critically ill patients, there was no definitive evidence regarding the impact of their measurement, or that of other point-of-care chemistry analytes, during CPR. We deemed the rapid treatment of hypo- or hyperkalemia clinically relevant due to the impact of blood potassium concentration on cardiac conduction. To identify potential potassium abnormalities, we suggest measuring potassium concentrations in all dogs and cats during CPR; however, this recommendation is stronger if potassium abnormalities are thought to be a contributing factor to the CPA event. While very limited evidence was found regarding calcium use during CPR, we suggest measurement of blood ionized calcium concentration during CPR and suggest administration of a calcium-containing solution during CPR only in patients with documented hypocalcemia. Similar to human guidelines, the routine administration of calcium during CPR in the absence of documented hypocalcemia or other specific indications is not recommended. Although there was no evidence regarding the measurement of blood sodium concentration during CPR, instances of severe hyper- or hyponatremia should prompt thoughtful fluid choices in the periarrest period.

Considering PCA care and monitoring, recommendations for measurement of lactate, creatinine, and glucose were reviewed. Due to the association of persistently elevated blood lactate concentration with poor outcome, we strongly recommended performing serial measurements of blood lactate in the PCA period and adjusting therapy to effect rapid normalization of the lactate concentration. Likewise, increased serum creatinine concentration was consistent with the development of AKI in the PCA period, which is associated with morbidity and mortality. We strongly recommend serial measurement of serum creatinine in the PCA period in addition to other relevant monitoring and treatment to identify, prevent, or treat AKI. Both hypo- and hyperglycemia in the PCA period were negatively associated with long-term survival or favorable neurologic outcome in people, and thus we made a strong recommendation for BG measurement in the PCA period if glucose abnormalities were suspected. We suggest measuring BG in all PCA patients, and although therapeutic interventions were not evaluated in the Monitoring Domain, hypoglycemic patients should receive dextrose support. The most beneficial therapeutic approach to hyperglycemia is less clear, and caution is warranted because iatrogenic hypoglycemia may be associated with poor outcome.

It is preferable to prevent CPA whenever possible, and therefore patients at risk of arrest or re-arrest should be hospitalized in an environment with frequent, close monitoring. Monitoring of patients using continuous ECG can identify tachycardia, bradycardia, and arrhythmias that may precede CPA, and we strongly recommend continuous ECG monitoring in patients at risk of CPA. Changes in vasomotor tone resulting in hypo- or hypertension may not be reflected in the ECG tracing, and evidence led to a strong recommendation for blood pressure monitoring in at-risk patients. Specifically, we recommend frequent or continuous ABP monitoring in patients at risk of CPA, including patients under anesthesia, in shock, and in the PCA period. In addition, we suggest continuous direct ABP monitoring when possible in these patients. While direct ABP monitoring may provide more accurate moment-to-moment values for SAP and DBP, no studies were identified that compared continuous direct ABP monitoring to frequent, indirect ABP monitoring, which generally is more feasible in clinical practice. Direct measurement of DBP, however, appears to be a good surrogate for monitoring CPR efficacy, and if an arterial catheter is in place, it is reasonable to use it to monitor DBP as long as it does not detract from CPR efforts.

Pulse oximetry is available in many veterinary clinics, and most monitors give both an audible pulse tone and a surrogate value for PaO_2, which gives pulse oximetry the potential to alert veterinary staff to low-perfusion states or oxygen desaturation that may precede CPA. Desaturation events may be due to hypoventilation or due to acute anatomic changes (eg, pneumothorax, upper airway obstruction) that occur in the periarrest period, although there may be a meaningful delay before the event affects the pulse oximetry value if the animal is receiving supplemental oxygen. While evidence supports the use of pulse oximetry to monitor at-risk patients in conjunction with other vital monitors (eg, ECG, ABP monitoring), evidence is lacking that pulse oximetry alone can be used as a reliable monitor in awake patients at risk of CPA. In anesthetized cats, the use of pulse oximetry or pulse monitoring was associated with a decreased incidence of anesthetic death, and we provided a strong recommendation for the use of pulse oximetry specifically in this context. We suspect that the reported association between a record of pulse (palpation or oximetry) monitoring and anesthetic survival in cats reflects these healthcare teams' abilities to obtain a reading and record the value—in other words, the cats in which monitoring was possible had detectable pulsatile blood flow. In this context, continuous pulse oximetry measurement in the anesthetized cat may be very helpful: if a reading can be attained, this likely indicates adequate perfusion; and if the SpO_2 value itself is $\geq$96%, this likely indicates adequate oxygenation to those perfused tissues. Unfortunately, pulse oximetry was not found to be as obviously useful in anesthetized dogs. As with any monitoring device, all pulse oximetry alerts should be verified through cross-reference with a physical examination and other monitoring devices to exclude machine malfunction or detachment as a cause of the alert.

There are inherent limitations to determining the impact of any monitoring measure on outcome. Users must decide to perform a monitoring intervention, conduct and interpret the test correctly, and then make clinical decisions based on that interpretation before the clinical outcome occurs. The evidence reviewed to generate the recommendations often required extrapolation from human clinical investigations or experimental animal models. While these data are relevant and important, very few clinical veterinary studies were found, introducing the possibility of bias or nonapplicability to the species or clinical scenarios in question. Ultimately, this indirectness compromises the certainty with which treatment recommendations for dogs and cats can be made. Further prospective or retrospective studies in clinical veterinary patients are required to verify the applicability of the recommendations to clinical medicine.

AUTHOR CONTRIBUTIONS

Benjamin Brainard: Conceptualiztion; formal analysis; investigation; methodology; writing—original draft; writing—review and editing. **Selena Lane**: Conceptualization; formal analysis; investigation; methodology; writing—original draft; writing—review and editing. **Jamie Burkitt-Creedon**: Conceptualization; formal analysis; investigation; methodology; writing—original draft; writing—review and editing. **Manuel Boller**: Conceptualization; formal analysis; funding acquisition; investigation; methodology; writing—original draft; writing—review and editing. **Daniel Fletcher**: Conceptualization; formal analysis; funding acquisition; investigation; methodology; writing—original draft; writing—review and editing. **Molly Crews**: Data curation; investigation; writing—review and editing. **Erik Faustak**: Data curation; investigation; writing—review and editing. Evidence evaluators: formal analysis; investigation; writing—original draft.

ACKNOWLEDGMENTS

The authors would like to acknowledge the following individuals and organizations, without whom an endeavor of this size would have been impossible. Peter Morley, Vinay Nadkarni, and the International Liaison Committee on Resuscitation provided invaluable mentorship, guidance, and a wealth of experience to inform the RECOVER guidelines process. RECOVER as an organization grew out of the American College of Veterinary Emergency and Critical Care and the Veterinary Emergency and Critical Care Society, and we are grateful for the ongoing support of both organizations as we work to produce guidelines, educational content, and research to improve the care of critically ill and injured animals. We appreciate the tireless efforts of Jamie Holms, who provided ongoing administrative support through the process of developing these guidelines. And finally, Emma Fralin and Samantha Moya, veterinary students at Cornell University, provided support for the extensive reference management needed for this project (Supporting Information). This project support was received from Boehringer Ingelheim Animal Health and Zoetis Animal Health, both of which helped to fund the purpose-developed, web-based system used for evidence evaluation.

CONFLICT OF INTEREST STATEMENT

Drs. Brainard and Burkitt-Creedon are editors of the Journal but did not participate in review process other than as authors. The authors declare no other conflicts of interest.

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ENDNOTES

[a] Search strategies and other primary documents, Open Science Framework: http://osf.io/DB2AM.

[b] www.RECOVERinitiative.org (accessed on March 19, 2024).

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

How to cite this article: Brainard BM, Lane SL, Burkitt-Creedon JM, et al. 2024 RECOVER Guidelines: Monitoring. Evidence and knowledge gap analysis with treatment recommendations for small animal CPR. *J Vet Emerg Crit Care*. 2024;34(Suppl 1):76–103. https://doi.org/10.1111/vec.13390