Received: 22 March 2024 | Accepted: 25 April 2024

DOI: 10.1111/vec.13387

SPECIAL ARTICLE

Journal of Veterinary Emergency and Critical Care | WILEY

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

Kate Hopper BVSc, PhD, DACVECC[1] | Steven E. Epstein DVM, DACVECC[1] | Jamie M. Burkitt-Creedon DVM, DACVECC[1] | Daniel J. Fletcher PhD, DVM, DACVECC[2] | Manuel Boller Dr med vet, MTR, DACVECC[3,4] | Erik D. Fausak MSLIS, RVT[5] | Kim Mears MLIS, AHIP[6] | Molly Crews MLS[7] | the RECOVER Basic Life Support Domain Evidence Evaluators

[1]Department of Veterinary Surgical and Radiological Sciences, School of Veterinary Medicine, University of California, Davis, Davis, California, USA

[2]Department of Clinical Sciences, College of Veterinary Medicine, Cornell University, Ithaca, New York, USA

[3]VCA Canada Central Victoria Veterinary Hospital, Victoria, British Columbia, Canada

[4]Department of Veterinary Clinical and Diagnostic Sciences, Faculty of Veterinary Medicine, University of Calgary, Calgary, Alberta, Canada

[5]University Library, University of California, Davis, Davis, California, USA

[6]Robertson Library, University of Prince Edward Island, Charlottetown, Prince Edward Island, Canada

[7]Department of Small animal Clinical Sciences, College of Veterinary Medicine & Biomedical Sciences, Texas A&M University, College Station, Texas, USA

Correspondence
Kate Hopper, Department of Veterinary Surgical and Radiological Sciences, School of Veterinary Medicine, University of California, Davis, 1 Shields Ave, Davis, CA 95616, USA.
Email: khopper@ucdavis.edu

Funding information
Zoetis; Boehringer Ingelheim Animal Health

Abstract

Objective: To systematically review evidence and devise treatment recommendations for basic life support (BLS) in dogs and cats and to identify critical knowledge gaps.

Design: Standardized, systematic evaluation of literature pertinent to BLS following Grading of Recommendations, Assessment, Development, and Evaluation (GRADE) methodology. Prioritized questions were each reviewed by 2 Evidence Evaluators, and findings were reconciled by BLS Domain Chairs and Reassessment Campaign on Veterinary Resuscitation (RECOVER) Co-Chairs to arrive at treatment recommendations commensurate to quality of evidence, risk to benefit relationship, and clinical feasibility. This process was implemented using an Evidence Profile Worksheet for each question that included an introduction, consensus on science, treatment recommendations, justification for these recommendations, and important knowledge gaps. A

Abbreviations: ACD, active compression–decompression; A–P, anterior–posterior; BLS, basic life support; C:V, compression-to-ventilation; CI, confidence interval; CoPP, coronary perfusion pressure; CPA, cardiopulmonary arrest; CPC, Cerebral Performance Category; EE, Evidence Evaluator; GRADE, Grading of Recommendations, Assessment, Development, and Evaluation; IHCPA, in-hospital cardiopulmonary arrest; I-time, inspiratory time; OHCPA, out-of-hospital cardiopulmonary arrest; OR, odds ratio; PEEP, positive end-expiratory pressure; PICO, Population, Intervention, Comparator, and Outcome; PIP, peak inspiratory pressure; RECOVER, Reassessment Campaign on Veterinary Resuscitation; ROSC, return of spontaneous circulation; RR, respiratory rate; TV, tidal volume; VF, ventricular fibrillation.

Kate Hopper and Steven E. Epstein contributed equally to this work.

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: Twenty questions regarding animal position, chest compression point and technique, ventilation strategies, as well as the duration of CPR cycles and chest compression pauses were examined, and 32 treatment recommendations were formulated. Out of these, 25 addressed chest compressions and 7 informed ventilation during CPR. The recommendations were founded predominantly on very low quality of evidence and expert opinion. These new treatment recommendations continue to emphasize the critical importance of high-quality, uninterrupted chest compressions, with a modification suggested for the chest compression technique in wide-chested dogs. When intubation is not possible, bag–mask ventilation using a tight-fitting facemask with oxygen supplementation is recommended rather than mouth-to-nose ventilation.

Conclusions: These updated RECOVER BLS treatment recommendations emphasize continuous chest compressions, conformation-specific chest compression techniques, and ventilation for all animals. Very low quality of evidence due to absence of clinical data in dogs and cats consistently compromised the certainty of recommendations, emphasizing the need for more veterinary research in this area.

KEYWORDS

canine, cardiopulmonary resuscitation, clinical trials, consensus guidelines, critical care, evidence-based medicine, feline

1 | INTRODUCTION

Basic life support (BLS) in veterinary CPR of adult dogs and cats includes recognition of cardiopulmonary arrest (CPA), airway management, ventilation, and chest compressions. CPR can be initiated by either medical professionals or pet owners, although some recommendations presented will apply to the hospital environment only. Initiation of early, high-quality BLS has been associated with improved return of spontaneous circulation (ROSC), survival to discharge, and favorable neurologic outcome in animals and people.[1,2]

The RECOVER (Reassessment Campaign on Veterinary Resuscitation) initiative first released veterinary CPR guidelines in 2012 (2012 RECOVER CPR Guidelines). Those guidelines investigated 17 PICO (Population, Intervention, Comparator, and Outcome) questions concerning BLS.[3] Of the 20 BLS PICO questions evaluated in the current version, 15 questions were re-evaluated from the 2012 RECOVER CPR Guidelines and 5 new questions were added.

2 | METHODS

Full explanation of the methods used to generate the BLS treatment recommendations is available in a companion paper.[4] What follows here is an overview. This BLS Domain Paper and the associated RECOVER CPR Guidelines[5] were generated using a modified version of the GRADE (Grading of Recommendations, Assessment, Development, and Evaluation) system for guidelines generation in health care.[6]

The RECOVER Co-Chairs assigned content experts to serve as chairs for the BLS domain (SE, KH). These Domain Chairs generated research questions in the PICO format including multiple relevant outcomes for each PICO question. PICO questions were rated as high priority, moderate priority, or lower priority. Thirty PICO questions were developed for evidence evaluation for BLS; 10 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 20 high-priority BLS PICO questions evaluated, 15 questions were re-evaluated from the 2012 RECOVER CPR Guidelines and 5 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

BOX 1: Major updates

- Chest compression depth:
  - Compress one-third to one-half of the thoracic width if patient in lateral recumbency.
  - Compress one-fourth of the thoracic depth if patient in dorsal recumbency.
- Only interrupt chest compression cycle if strong objective evidence of return of spontaneous circulation.
- Limit pauses between chest compression cycles to <10 seconds.
- Recommend using the peak inspiratory pressure (PIP) required to see a visible chest rise.
- When performing CPR in patients undergoing mechanical ventilation, recommend converting to manual ventilation.
- When endotracheal intubation is not possible, a tight-fitting facemask with oxygen supplementation should be used to provide positive pressure breaths rather than mouth-to-nose ventilation.

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.[7] Once potentially relevant articles were identified, 2 Evidence Evaluators (EEs) (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 EEs.

A purpose-developed, web-based evaluation system was used to guide EEs 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. EEs 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; EEs 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 were 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 BLS PICO question appears below, and the additional study evaluation notes appear in the full Evidence Profile Worksheets[a].

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 | CHEST COMPRESSIONS

High-quality chest compressions are required to generate blood flow during closed-chest CPR. As external chest compressions do not result in normal levels of cardiac output, it is essential to optimize their delivery to maximize blood flow. There are multiple factors that affect the blood flow generated by chest compressions, and this section reviews the timing, delivery, and cycle duration of chest compressions. While recommendations for most aspects of chest compressions (eg, rate, depth, cycle length) are consistent across all dogs and cats, recommendations for the specific location on the chest at which compression force should be focused (the compression point) vary between chest shapes, and the PICO questions in this section address several special circumstances. It should be noted that chest conformations and mechanical characteristics vary along a spectrum across individuals, and these compression point recommendations should be taken as a starting point. If objective measures of chest compression quality, such as $ETCO_2$, suggest inadequate chest compression efficacy, it may be necessary to adjust the compression point if all other aspects of chest compressions have been optimized.

3.1 | Ventilation-first versus compressions-first CPR (BLS-11)

In nonintubated cats and dogs in CPA (P), does the use of ventilation-first CPR (ABC) (I), compared to compressions-first CPR (CAB) (C), improve favorable neurologic outcome, survival to discharge, ROSC, or time to completion of first CPR cycle (O)?

3.1.1 | Introduction

High-quality chest compressions are considered essential for positive CPR outcomes. There has been an emphasis on shortening the time to commencement of chest compressions in CPR guidelines.[8] The current adult human CPR guidelines acknowledge a lack of evidence on which to make a recommendation whether to start CPR with airway and breathing or with chest compressions first, and they suggest starting CAB in adult out-of-hospital cardiopulmonary arrest (OHCPA).[9] This suggestion is based on the fact that most OHCPA in adults is cardiac in cause in addition to data on manikin-based studies that show decreased time to initiation of chest compressions with the CAB approach. In contrast, current human pediatric CPR guidelines consider the evidence so limited that no recommendation can be made but acknowledge that most pediatric arrests are asphyxial in nature, which suggests that early ventilation is paramount.[2] Likewise, registry data suggest that only 8% of dogs and cats undergoing CPR experienced CPA due to a cardiac arrhythmia, with the suspected cause for most CPAs being respiratory failure, heart failure, trauma, hemorrhage, or metabolic/electrolyte derangements.[10]

3.1.2 | Consensus on science

For the most critical outcomes of favorable neurologic outcome, survival to discharge, and ROSC, we identified no studies addressing the PICO question.

For the important outcome of time to completion of the first CPR cycle, we found 2 experimental, simulator-based studies (very low level of evidence, downgraded for very serious indirectness) that addressed the PICO question.[11,12] Both studies found that CPR using the CAB approach had a shorter time to completion of the first chest compression cycle compared to CPR using the ABC approach. This evidence suggests that CAB shortens the time for the completion of the first CPR cycle when compared to ABC.

3.1.3 | Treatment recommendations

For multi-rescuer CPR in dogs and cats, we recommend that chest compressions be initiated without delay to assess airway and gain airway access (strong recommendation, very low quality of evidence).

For multi-rescuer CPR in dogs and cats, we recommend that the airway be evaluated and the animal endotracheally intubated as soon as possible after initiation of chest compressions (strong recommendation, expert opinion).

For single-rescuer CPR in dogs and cats, prior to initiation of chest compressions, we recommend that an airway evaluation be performed during the initial patient assessment (shake & shout) prior to initiation of chest compressions (strong recommendation, expert opinion).

3.1.4 | Justification of treatment recommendations

Considering the critical importance of circulation in delivering oxygen to tissues, we believe that high-quality chest compressions should not be delayed in nonresponsive, nonintubated, apneic dogs and cats, even in cases of asphyxial arrest. Practically, the period of time required to assess and secure an airway, even in the hospital setting, is too long a period to wait to initiate chest compressions in our opinion. However, we recognize the importance of oxygenation and ventilation in asphyxial arrest scenarios and believe that the airway should be assessed, and ventilation should be provided as quickly as possible, as long as this does not preclude the immediate provision of high-quality compressions.

These recommendations vary based on the number of rescuers. For single-rescuer CPR, airway evaluation should occur during the initial patient assessment steps, with chest compressions started immediately thereafter; we believe this is a reasonable approach in that it delays ventilation by only 15 seconds. We considered this 15-second delay acceptable even in the face of asphyxial arrest to promote consistency in guidelines and at presumed low risk for individual patients. For multi-rescuer CPR, we recommend against a delay in chest compressions since airway evaluation, intubation, and compressions can occur simultaneously.

3.1.5 | Knowledge gaps

Studies in dogs and cats comparing these 2 approaches (CAB vs ABC) are needed, ideally with evaluation of critical outcomes of favorable neurologic outcome, survival to discharge, and ROSC.

3.2 | Compression point in round-chested dogs (BLS-02)

In medium- and large-sized, round-chested dogs in CPA (P), does placing hands over the heart for chest compressions (I), compared to placing hands over the widest point of the thorax for chest compressions (C), improve favorable neurologic outcome, survival to discharge, ROSC, or surrogate markers of perfusion (O)?

3.2.1 | Introduction

In dogs in lateral recumbency, it is assumed that chest compressions performed directly over the anatomic location of the heart are more likely to directly compress the heart (cardiac pump theory), while chest compressions over the widest portion of the chest are more likely to increase the overall intrathoracic pressure (thoracic pump theory).[13] Previous RECOVER guidelines suggest hand placement over the widest part of the chest in medium- to large-sized, round-chested dogs. This question investigates whether chest

compressions in medium- to large-sized, round-chested dogs are better performed with hands placed over the widest part of the thorax or over the heart.

3.2.2 | Consensus on science

For the most critical outcomes of favorable neurologic outcome, survival to discharge, and ROSC, we identified no studies addressing the PICO question. For the important outcome of *surrogate markers of perfusion*, we found 5 experimental animal studies (very low-quality of evidence, downgraded for very serious indirectness and serious imprecision) that addressed the PICO question.[14–18] No studies were found that directly compare chest compressions performed with hands placed over the heart versus hands placed on the widest point of the thorax. No studies were identified that directly compare how each of these methods affects intrathoracic pressure and blood flow. Five experimental studies were identified with some relevance for development of clinical guidelines. Four canine studies and 1 porcine study reported markers of perfusion using different approaches to chest compressions during CPR.[14–18] A large degree of indirectness is present in all of these studies.

Direct cardiac compression has been shown to generate blood flow during CPR. One swine study documented that left ventricular position changes during chest compressions and that the proportion of the left ventricle that is being compressed correlates positively with cardiac output.[15] Three of the identified studies showed that increases in intrathoracic pressure result in blood flow during CPR.[14,16,18] One canine model of sternal chest compressions suggests that the thoracic pump mechanism is at play in these dogs and that intracardiac and intravascular pressures depend on fluctuations in intrathoracic pressure and not direct compression of the heart.[14] A study of manual CPR in dogs in supine position demonstrated that the thoracic pump mechanism prevails in this scenario (rather than direct compression of the heart) and that blood flow is generated in response to intrathoracic pressure changes.[16] Another canine study showed that intrathoracic pressure changes can lead to closure of the mitral valve.[18]

A difference in the predominant mechanism of blood flow with different chest compression points during lateral chest compressions was supported by an experimental dog study evaluating mitral valve motion using transthoracic and transesophageal echocardiography in dogs weighing 18–26 kg.[17] With hands placed over the widest portion of the chest, the thoracic pump mechanism with the heart acting as a passive conduit seemed to prevail as demonstrated by nonopposing mitral valve leaflets, while with compressions directly over the heart, the mitral valve leaflets fully closed, supporting the cardiac pump theory.[17] The chest conformation of the dogs in this study is not well described.

Moreover, this evidence suggests that blood flow in CPR can occur due to both changes in intrathoracic pressure and direct cardiac compression, and that hand placement over the thorax can impact which of these mechanisms predominates during chest compressions.

3.2.3 | Treatment recommendation

We suggest performing chest compressions with hand placement over the widest part of the thorax in medium- to large-sized, round-chested dogs (weak recommendation, very low quality of evidence).

3.2.4 | Justification of treatment recommendation

Although no studies were identified to directly address this PICO question, evidence suggests that hands placed directly over the heart during chest compressions in lateral recumbency create a cardiac pump mechanism of blood flow and that when hands are placed in other locations, a thoracic pump mechanism predominates. In medium- to large-sized, round-chested dogs, there exists the clinical concern that hand placement over the heart does not create sufficient compression of the thorax to create a cardiac pump mechanism of flow; thus, hand placement over the widest part of the chest may be preferable in these dogs.

3.2.5 | Knowledge gaps

There are no studies that evaluate outcomes among different hand placements for lateral chest compressions in medium- to large-sized, round-chested dogs.

3.3 | Compression point in keel-chested dogs (BLS-03)

In medium- and large-sized, keel-chested dogs in CPA (P), does placing hands over the widest point of the thorax (I), compared to placing hands over the heart for chest compressions (C), improve favorable neurologic outcome, survival to discharge, ROSC, or surrogate markers of perfusion (O)?

3.3.1 | Introduction

In dogs in lateral recumbency, it is assumed that chest compressions performed directly over the anatomic location of the heart are more likely to directly compress the heart (cardiac pump theory), while chest compressions over the widest portion of the chest are more likely to increase the overall intrathoracic pressure (thoracic pump theory). Previous veterinary guidelines suggest hand placement directly over the heart in medium- and large-sized, keel-chested dogs.[19] This question investigates whether chest compressions in medium- to large-sized, keel-chested dogs are better performed with hands placed over the widest part of the thorax or over the heart.

3.3.2 | Consensus on science

For the most critical outcomes of favorable neurologic outcome, survival to discharge, and ROSC, we identified no studies addressing the

PICO question. For the important outcome of *surrogate markers of perfusion*, we found 5 experimental animal studies (very low-quality of evidence, downgraded for very serious indirectness and serious imprecision) that addressed the PICO question.[14–18] No studies were found that directly compare chest compressions performed with hands placed over the heart versus hands placed on the widest point of the thorax. No studies were identified that directly compare how each of these methods affects intrathoracic pressure and blood flow. Five experimental studies were identified with some relevance for development of clinical guidelines. Four canine studies and 1 porcine study reported markers of perfusion using different approaches to chest compressions during CPR.[14–18] A large degree of indirectness is present in all of these studies.

Direct cardiac compression has been shown to generate blood flow during CPR. One swine study documented that left ventricular position changes during chest compressions and that the proportion of the left ventricle that is being compressed correlates positively with cardiac output.[15] Three of the identified studies showed that increases in intrathoracic pressure result in blood flow during CPR.[14,16,18] One canine model of sternal chest compressions suggests that the thoracic pump mechanism is at play in these dogs and that intracardiac and intravascular pressures depend on fluctuations in intrathoracic pressure and not direct compression of the heart.[17] A study of manual CPR in dogs in supine position demonstrated that the thoracic pump mechanism prevails in this scenario (rather than direct compression of the heart), and that blood flow is generated in response to intrathoracic pressure changes.[16] Another canine study showed that intrathoracic pressure changes can lead to closure of the mitral valve.[18]

A difference in the predominant mechanism of blood flow with different chest compression points during lateral chest compressions was supported by an experimental dog study evaluating mitral valve motion using transthoracic and transesophageal echocardiography in dogs weighing 18–26 kg.[17] With hands placed over the widest portion of the chest, the thoracic pump mechanism with the heart acting as a passive conduit seemed to prevail as demonstrated by nonopposing mitral valve leaflets, while with compressions directly over the heart, the mitral valve leaflets fully closed, supporting the cardiac pump theory.[17] The chest conformation of the dogs in this study is not well described.

This evidence suggests that blood flow in CPR can occur due to both changes in intrathoracic pressure and direct cardiac compression, and that hand placement over the thorax can impact which of these mechanisms predominates during chest compressions.

3.3.3 | Treatment recommendation

We recommend performing chest compressions with hand placement over the heart in medium- to large-sized, keel-chested dogs (strong recommendation, very low quality of evidence).

3.3.4 | Justification of treatment recommendation

Although no studies were identified to directly address this PICO question, evidence suggests that hands placed directly over the heart during chest compressions in lateral recumbency create a cardiac pump mechanism of blood flow and that when hands are placed in other locations, a thoracic pump mechanism predominates. In medium- to large-sized, keel-chested dogs, it is generally assumed based on conformation that hand placement directly over the heart creates sufficient compression of the thorax to create a cardiac pump mechanism of flow; thus, hand placement over the heart is likely preferable in these dogs.

3.3.5 | Knowledge gaps

There are no studies that evaluate outcomes among different hand placements for lateral chest compressions in medium- to large-sized, keel-chested dogs.

3.4 | Patient positioning for non-wide-chested dogs (BLS-04)

In non-wide-chested dogs and in cats in CPA (P), does performing chest compressions with the animal in dorsal recumbency (I), compared to lateral recumbency (C), improve favorable neurologic outcome, survival to discharge, ROSC, or surrogate markers of perfusion (O)?

3.4.1 | Introduction

In clinical veterinary patients, chest compressions during CPR are most commonly performed with animals in lateral recumbency, in contrast to experimental animal and clinical human studies, in which dorsal recumbency is most common. Dorsal recumbency for chest compressions in non-wide-chested dogs is challenging without restraint or additional equipment, given their conformation. The previous veterinary guidelines make no comment regarding the role of sternal compressions in dorsal recumbency for non-wide-chested dogs.[19]

3.4.2 | Consensus on science

For the most critical outcomes of favorable neurologic outcome and survival to discharge, we identified no studies addressing the PICO question. For the important outcome of *ROSC*, we found 1 experimental animal study (very low quality of evidence, downgraded for very serious indirectness) and 1 observational study (very low quality of evidence, downgraded for serious risk of bias and serious indirectness, upgraded for effect despite confounding).[20,21] One experimental study in cats with an asphyxiation model of CPA failed to demonstrate a difference between chest compressions in dorsal versus lateral

recumbency.[20] One 2009 observational study in a veterinary teaching hospital documented that dogs in CPA receiving chest compressions in lateral recumbency had higher odds of ROSC (odds ratio [OR]: 46.6, 95% confidence interval [CI]: 4.1–535.6) in multivariate logistic regression. Body conformation was not reported in this study.[21]

For the important outcome of *surrogate markers of perfusion*, we identified 1 experimental study in dogs (low quality of evidence, downgraded for serious imprecision) that addressed the PICO question.[22] This study included 24 "mongrel dogs" weighing 25–35 kg whose conformations were not described and found that manual compressions administered in lateral recumbency resulted in higher left ventricular pressure and aortic flow than those delivered with the dog in supine position. Comparative numerical values were not reported, and no statistical analyses appear to have been performed.

3.4.3 | Treatment recommendation

We recommend performing chest compressions in lateral recumbency in non-wide-chested dogs (strong recommendation, very low quality of evidence).

3.4.4 | Justification of treatment recommendation

There are no studies directly comparing outcomes of dorsal versus lateral body position for chest compressions in dogs of any conformation. Despite the very low quality of evidence, this strong treatment recommendation is based on the logistical challenges of performing sternal chest compressions in non-wide-chested dogs in dorsal recumbency and the high compliance of the chest wall of many dogs in lateral recumbency. This recommendation is supported by 1 veterinary observational study that reported a higher odds of ROSC and 1 experimental study in dogs that found improved left ventricular pressure and aortic flow when chest compressions were performed with dogs in lateral recumbency compared to dorsal recumbency.

3.4.5 | Knowledge gaps

There are no studies that clearly report on any critical outcome for chest compressions performed in dorsal versus lateral recumbency in non-wide-chested dogs.

3.5 | Patient positioning for wide-chested dogs (BLS-05)

In wide-chested dogs in CPA (P), does performing chest compressions with the dog in lateral recumbency (I), compared to dorsal recumbency (C), improve favorable neurologic outcome, survival to discharge, ROSC, or surrogate markers of perfusion (O)?

3.5.1 | Introduction

In clinical veterinary patients, chest compressions during CPR are most commonly performed with animals in lateral recumbency, in contrast to experimental animal and clinical human studies where dorsal recumbency is most common. Wide-chested dogs such as Bulldogs often can be placed in dorsal recumbency without restraint, making sternal chest compressions feasible clinically in these animals. The previous veterinary guidelines suggest that sternal compressions in dorsal recumbency for wide-chested dogs may be considered.[19]

3.5.2 | Consensus on science

For the most critical outcomes of favorable neurologic outcome and survival to discharge, we identified no studies addressing the PICO question. For the next most important outcome of *ROSC*, we found 1 experimental animal study (very low quality of evidence, downgraded for very serious indirectness) and 1 observational study (very low quality of evidence, downgraded for serious risk of bias and serious indirectness, upgraded for effect despite confounding).[20,21] One experimental study in cats with an asphyxial model of CPA failed to demonstrate a difference between chest compressions in dorsal versus lateral recumbency.[20] One 2009 observational study in a veterinary teaching hospital documented that dogs in CPA receiving chest compressions in lateral recumbency had higher odds of ROSC (OR: 46.6, 95% CI: 4.1–535.6) in multivariate logistic regression. Body conformation was not reported in this study.[21]

For the important outcome of surrogate markers of perfusion, we identified no studies addressing the PICO question.

3.5.3 | Treatment recommendations

We suggest lateral chest compressions focused over the widest part of the chest in wide-chested dogs until an endotracheal tube is placed and secured (weak recommendation, expert opinion).

In wide-chested dogs that are positionally stable in dorsal recumbency, we suggest moving the dog to dorsal recumbency during an intercycle pause and performing chest compressions over the sternum directly over the heart once an endotracheal tube is in place (weak recommendation, expert opinion).

3.5.4 | Justification of treatment recommendations

Despite the lack of clear evidence, the working group believed that the unique thoracic conformation of some wide-chested dogs warrants special consideration when approaching chest compressions. We believe that anatomical constraints imposed by this conformation likely favor the chest compression approach used in people, in whom chest compressions are done with the patient in dorsal

recumbency, focused directly over the heart in the mid-sternal region. This patient positioning and compression point allow the rescuer to compress the heart directly between the sternum and the spine. However, given the importance of ventilation in these patients, most of which are brachycephalic, the working group recommended starting chest compressions in lateral recumbency using the approach for round-chested dogs to facilitate endotracheal tube placement. When intubation in lateral recumbency is difficult, the critical task of correct intubation in these brachycephalic animals may require cessation of chest compressions and intubation in sternal recumbency. Once the endotracheal tube is in place, rescuers can consider placing the dog in dorsal recumbency and using the sternal compression approach. Any elective shift in patient position should be done during a planned pause between chest compression cycles, should not delay the resumption of chest compressions, and should be aborted immediately if the dog is not positionally stable in the chosen recumbency.

3.5.5 | Knowledge gaps

There are no studies that directly evaluate the efficacy of chest compressions performed with the animal positioned in dorsal versus lateral recumbency in wide-chested dogs.

3.6 | Circumferential versus lateral chest compressions in cats and small dogs (BLS-12)

In cats and small dogs in CPA (P), do 2-handed circumferential ("2-thumb technique") chest compressions (I), compared to lateral chest compressions (C), improve favorable neurological outcome, survival to discharge, ROSC, surrogate markers of perfusion, or complications(O)?

3.6.1 | Introduction

The optimal chest compression technique in cats and small dogs is unknown. Lateral chest compression can be achieved in these small-sized animals with 2-finger, 1-handed, or 2-handed techniques. Alternatively, 2 hands can be used to encircle the chest, which allows the rescuer to squeeze the thorax during compressions. The current pediatric human guidelines state there is insufficient evidence to make a recommendation regarding compression technique and suggest either a 1-handed or 2-handed technique can be used.[2]

3.6.2 | Consensus on science

For the most critical outcomes of favorable neurologic outcome, survival to discharge, and ROSC, we identified no studies addressing the PICO question.

For the important outcome of *surrogate markers of perfusion*, we found 2 manikin-based experimental studies (very low level of evidence, downgraded for very serious indirectness).[23,24] These 2 studies found that a circumferential, 2-thumb (eg, 2-handed) technique generates higher pressure in a simulated arterial system compared to a 2-finger technique.[23,24]

For the less important outcome of *complications*, we identified no studies addressing this PICO question.

3.6.3 | Treatment recommendation

We recommend that chest compressions in cats and small dogs be performed using 1 of the following 3 methods, based on a combination of compressor preference and real-time markers of perfusion (eg, $ETCO_2$, direct blood pressure monitoring):

a. Using a circumferential 2-thumb chest compression technique with the animal in lateral recumbency and both of the thumbs directly over the heart (strong recommendation, very low quality of evidence).
b. Using a 1-handed technique with the dominant hand wrapped around the sternum at the level of the heart performing compressions between the flat portion of the fingers and the flat portion of the thumb (strong recommendation, expert opinion).
c. Using a 1-handed technique with heel of the dominant hand compressing one-third to one-half of the chest width over the area of the heart with the animal in lateral recumbency while the nondominant hand supports the dorsal thorax (strong recommendation, expert opinion).

3.6.4 | Justification of treatment recommendation

Given there is very little evidence to inform a recommendation regarding ideal compression technique in cats and small dogs, we believe it is appropriate to provide a variety of options, integrating recommendations from multiple sources. The circumferential 2-thumb technique is recommended based on findings that suggest it may be superior in human infants, while lateral recumbency is suggested based on chest conformation. The limited available evidence suggests that a circumferential 2-thumb chest compression technique may produce higher intrathoracic pressures than a 2-finger compression technique in human infants. This may translate to improvement in critical outcomes and supports methods (a) and (b), above. Additionally, anecdotal experience suggests that compression depth appears adequate in these animals when high-quality, 1-handed chest compressions are administered in lateral recumbency in cats and small dogs, which supports method (c), above.

3.6.5 | Knowledge gaps

There are no studies of cats or small dogs or in animal models in lateral recumbency evaluating different chest compression techniques for any outcome of interest.

3.7 | Chest compression rate (BLS-07)

In cats or dogs in CPA (P), does the use of any other specific rate for external chest compressions (I), compared to external chest compression rate of 100–120/min (C), improve favorable neurologic outcome, survival to discharge, ROSC, or surrogate markers of perfusion (O)?

3.7.1 | Introduction

The current human and previous veterinary guidelines recommend a chest compression rate of 100–120/min based primarily on experimental and human data.[9,19] This PICO question investigated whether other compression rates are superior to 100–120/min in dogs and cats during CPR.

3.7.2 | Consensus on science

For the most critical outcome of *favorable neurological outcome*, we identified 2 experimental studies (very low quality of evidence, downgraded for risk of bias, serious indirectness, and serious imprecision, and upgraded for effect found despite confounding) and 3 observational studies (very low quality of evidence, downgraded for very serious risk of bias and serious indirectness) that addressed the PICO question.[25–29] Two experimental animal studies, including 1 in dogs, found that chest compression rates substantially lower than 100–120/min (60 and 80/min) were associated with poorer neurological outcome than a chest compression rate of 100.[25,26] Two observational studies in adults supported a compression rate of 100–120/min.[27,28] One pediatric study found that a compression rate of 80–99/min had a more favorable neurological outcome in a very small number of children.[29] Due to confounding factors in that study, its authors recommended against changing guidelines based on their findings.

For the next critical outcome of *survival to discharge*, we identified 4 observational studies in people (very low quality of evidence, downgraded for very serious risk of bias, very serious indirectness, and serious imprecision) that addressed the PICO question.[29–32] The same pediatric study cited above[29] also documented that a chest compression rate of <100/min in a very small portion of patients was associated with greater survival to discharge.[29] Authors of that study recommended against changing compression recommendations from existing guidelines. Two studies (1 adult and 1 pediatric) found no difference between intervention and comparator group.[30,31] One large-scale adult human study showed that the nontarget compression rate groups (<100 and >120/min) were associated with worse survival to discharge rates.[32]

For the critical outcome of *ROSC*, we identified 3 experimental studies (very low quality of evidence, downgraded for serious risk of bias and serious imprecision) and 5 observational studies (very low quality of evidence, downgraded for very serious risk of bias, serious indirectness, and serious imprecision).[25,26,28–30,32–34]

Of the 3 experimental studies, 1 canine[25] and 1 swine study[26] found lower ROSC rates with chest compression rates <100/min, while 1 piglet study identified no difference between a compression rate of 90 and 120/min.[33] Of 5 observational studies, 2 studies in human pediatric patients found no difference in ROSC with chest compression rates outside the range of 100–120/min.[29,30] One adult human study of in-hospital cardiopulmonary arrest (IHCPA) found higher ROSC rates with chest compression rates of 121–140/min compared to 100–120/min or >140/min.[28] In a large OHCPA adult human study, there was no difference in ROSC with a chest compression rate of 80–99/min or >120/min when compared to 100–119/min.[32] In a prospective, observational veterinary study, there was no difference in the median chest compression rate between patients with and without ROSC.[34]

Evidence was not summarized for "Outcome 4: Surrogate markers of perfusion" because of the evidence available for the more critical outcomes above.

3.7.3 | Treatment recommendation

We recommend using a chest compression rate of 100–120/min during CPR in dogs and cats (strong recommendation, very low quality of evidence).

3.7.4 | Justification of treatment recommendation

The preponderance of evidence supports chest compression rates of 100–120/min to optimize the most critical outcomes of favorable neurological outcome and survival to discharge. Although some studies are neutral as to the impact on these outcomes, others show improvement in the less critical outcome of ROSC when higher chest compression rates outside this range are used. However, there is no evidence that a chest compression rate other than 100–120/min improves any critical outcome in dogs and cats, and a recent study demonstrated that at a compression rate of 150/min, rescuers were unable to maintain adequate chest compression depth in a round-chested dog mannequin.[35]

3.7.5 | Knowledge gaps

There is no clinical evidence to support a specific chest compression rate in dogs or cats.

3.8 | Chest wall recoil (BLS-01)

In cats and dogs in CPA (P), does incomplete chest wall recoil (I), compared to allowing complete chest wall recoil (via 50:50 duty cycle, decreasing fatigue and leaning) (C), improve favorable neurologic outcome, survival to discharge, ROSC, or surrogate markers of perfusion (O)?

3.8.1 | Introduction

Incomplete chest wall recoil during CPR may increase intrathoracic pressure between chest compressions and thereby decrease venous return. Leaning on the chest between compressions will impair chest wall recoil and has been found to occur commonly in human clinical studies. The current human and previous veterinary guidelines recommend allowing for complete chest wall recoil between compressions based primarily on experimental data. This question investigates whether impaired chest wall recoil affects outcome in dogs and cats undergoing CPR.

3.8.2 | Consensus on science

For the most critical outcomes of favorable neurologic outcome, survival to discharge, and ROSC, we identified no studies that addressed the PICO question. For the important outcome of *surrogate markers of perfusion*, 4 experimental studies (moderate level of evidence, downgraded for serious indirectness) in a porcine ventricular fibrillation (VF) model of CPA were identified, 4 of which document that leaning decreases cardiac index and left ventricular myocardial index.[36,37] Two studies evaluated different duty cycles and documented that decreased duty cycle or maximizing the recoil phase increased cerebral or myocardial perfusion pressure; we considered adequate time in noncompression to be important for left ventricular filling during the recoil phase.[38,39] Cerebral perfusion pressure and aortic pressure were optimized by a 50:50 duty cycle of compression:noncompression in 8-week-old piglets.[39]

3.8.3 | Treatment recommendations

We recommend allowing full chest wall recoil between chest compressions in dogs and cats undergoing CPR (strong recommendation, moderate quality of evidence).

We recommend targeting a duty cycle of 50:50 for compression:noncompression during CPR in dogs and cats (strong recommendation, moderate quality of evidence).

3.8.4 | Justification of treatment recommendations

All pertinent evidence to date shows improved surrogate markers of perfusion when there is adequate opportunity for full chest wall recoil.

3.8.5 | Knowledge gaps

Whether allowing full chest recoil during CPR in dogs and cats improves neurologic outcome, survival to discharge, or ROSC is unknown.

There is no supporting evidence to address the PICO question in human clinical trials, nor in dogs and cats in any setting.

Whether veterinary healthcare providers can maintain adequate chest compression technique without leaning between compressions for a full 2-minute BLS cycle during CPR in dogs and cats of variable size and conformations is unknown.

It is unclear whether healthcare providers are able to assess themselves and others for leaning during the recoil phase of CPR in dogs and cats.

3.9 | Chest compression depth (BLS-18)

In cats and dogs in CPA (P), does any other specific compression depth (I), as opposed to one-third to one-half of the width of the thorax (C), improve favorable neurologic outcome, survival to discharge, ROSC, surrogate markers of perfusion, or complications (O)?

3.9.1 | Introduction

Chest compressions during CPR aim to generate blood flow through either direct compression of the heart or secondary to global increases in intrathoracic pressure. The depth of chest compressions is likely to have some relationship with cardiac output during CPR, but this benefit needs to be weighed against the potential for harm with increasing compression depth. The current human CPR guidelines recommend a chest compression depth of approximately 5 cm while avoiding excessive compression depths (greater than 6 cm).[9] The previous veterinary guidelines suggested that a compression depth of one-third to one-half of the width of the thorax was reasonable.[19]

3.9.2 | Consensus on science

For the most critical outcome of *favorable neurologic outcome*, we identified 2 observational studies of OHCPA in people (very low quality of evidence, downgraded for serious risk of bias, very serious indirectness, and serious imprecision) that addressed the PICO question.[40,41] One before-and-after study of 593 adults comparing chest compression depth prior to and after institution of a comprehensive CPR quality improvement initiative found that each 5-mm increase in mean chest compression depth significantly increased the odds of survival with favorable functional outcome with an adjusted OR of 1.30 (95% CI: 1.00–1.70).[41] In another before-and-after study of 32 people with OHCPA, the use of a real-time audiovisual feedback device increased chest compression depth from 38.8 ± 11.5 to 48.0 ± 9.2 mm, while no change was noted in chest compression depth when no feedback was provided. No difference in favorable neurologic outcome was found between the 2 groups of 16 people each.[40]

For the next critical outcome of *survival to discharge*, we identified 3 observational studies (very low quality of evidence, downgraded for very serious indirectness and serious imprecision) that addressed the PICO question.[40–42] In a large observational study of 9136 adults with OHCPA, the adjusted OR for survival to discharge was

1.04 (95% CI: 1.00–1.08) for each 5-mm increment in compression depth, 1.45 (95% CI: 1.20–1.76) for cases with a depth range of >38 mm, and 1.05 (95% CI: 1.03–1.08) for percentage of minutes within depth range. Covariate-adjusted spline curves revealed that the maximum survival in these adult people is at a depth of 45.6 mm (15-mm interval with highest survival between 40.3 and 55.3 mm); no differences were found between male and female patients.[42] A smaller before-and-after study of 593 adults comparing chest compression depth prior to and after institution of a comprehensive CPR quality improvement initiative found that each 5-mm increase in mean chest compression depth increased the odds of survival to discharge with an adjusted OR of 1.29 (95% CI: 1.00–1.65).[41] A very small before-and-after study of 32 people with OHCPA found that the use of a real-time audiovisual feedback device increased chest compression depth from 38.8 ± 11.5 to 48.0 ± 9.2 mm, while no change was noted in chest compression depth when no feedback was provided. No difference in survival was found between the 2 groups of 16 people each.[40]

For the critical outcome of *ROSC*, we identified 4 observational studies in people (very low quality of evidence, downgraded for serious risk of bias, very serious indirectness, serious imprecision, and serious inconsistency)[40,42–44] and 2 experimental swine studies (very low quality of evidence, downgraded for very serious indirectness, serious imprecision, and serious inconsistency).[45,46] In a large observational study of 9136 adults with OHCPA, the adjusted OR for ROSC was 1.06 (95% CI: 1.04–1.08) for each 5-mm increment in compression depth.[42] The remaining 3 studies are much smaller, 2 supporting these findings and 1 finding no difference. In a before-and-after, observational study of 284 OHCPA events comparing the use of an automated feedback system to no feedback, the feedback group had greater compression depth (38 ± 6 vs 34 ± 9 mm); logistic regression found that the average compression depth (per millimeter increase) had an OR of 1.05 (95% CI: 1.01–1.09) for ROSC.[43] In an observational study in 60 people with either IHCPA or OHCPA, logistic regression analysis demonstrated that successful defibrillation was associated with higher mean compression depth during the 30 seconds of CPR preceding the preshock pause with an adjusted OR of 1.99 (95% CI: 1.08–3.66) for every 5-mm increase.[44] A very small before-and-after study of 32 people with OHCPA found that the use of a real-time audiovisual feedback device increased chest compression depth from 38.8 ± 11.5 to 48.0 ± 9.2 mm, while no change was noted in chest compression depth when no feedback was provided. No difference in ROSC was found between the 2 groups of 16 people each.[40]

An experimental swine VF model compared optimal chest compression depth (25% = 6 cm) in an anterior–posterior (A–P) direction with a chest compression depth of 4.2 cm (70% of optimal = ~17% chest diameter); this study found that greater A–P chest compression depth was associated with ROSC.[45] In an experimental VF model in which swine underwent chest compression in dorsal recumbency, delivering chest compression to a depth of 35.2–57.0 mm (rescuer targeting 50 mm or 25% of the A–P diameter of the chest) resulted in no difference in ROSC than delivery of chest compression to a depth of 19.0–38.5 mm.[46]

For the important outcome of *surrogate markers of perfusion*, we identified 6 experimental studies (very low quality of evidence, downgraded for very serious indirectness and serious imprecision) that addressed the PICO question.[45–50] In dogs weighing 6–12 kg positioned in dorsal recumbency and receiving 62 compressions per minute with varied chest compression depths, cardiac output varied with chest displacement. Mean chest compression depth of 1.8 ± 0.85 cm was required to achieve mean arterial pressure (MAP) > 0 mm Hg. Increasing MAP was associated with increasing compression depth.[48] A swine experimental model comparing 20% to <14% A–P compression depth showed higher systolic arterial pressure, coronary perfusion pressure (CoPP), $ETCO_2$, and central venous O_2 for the 20% compression group.[47] In a swine experimental model, compressions to a depth of 35.2–57.0 mm (rescuer targeting a depth of 50 mm or 25% of the A–P diameter of the chest) resulted in a significantly higher CoPP than compressions to a depth of 19.0–38.5 mm (rescuer targeting 70% of "good" CPR depth, equivalent to a depth of 35 mm or ~17% of the A–P diameter of the chest) in dorsal recumbency.[46] In arrested piglets receiving 3-cm compressions versus 5-cm compressions, diastolic arterial pressure and CoPP were significantly higher in piglets receiving 5-cm compressions.[50] Another swine study found that CoPP was better with 5 cm chest compression depth than with 3 cm chest compression depth.[49] In a swine experimental VF model comparing optimal A–P chest compression depth (25% = 6 cm) with conventional depth (4.2 cm—70% of optimal = ~17%), CoPP and $ETCO_2$ were both significantly higher with greater depth of chest compression.[45]

For the important outcome of *complications*, we identified 1 observational (very low quality of evidence, downgraded for serious indirectness)[51] and 3 experimental studies (very low quality of evidence, downgraded for serious indirectness and serious imprecision) that addressed the PICO question.[45,46,52] Among male human patients, CPR-related injuries were associated with deeper mean and peak compression depths. No such association was observed in women. The frequency of injuries was not different in mean compression depth categories <5, 5–6, and >6 cm (28%, 27%, and 49%, respectively).[51] In a 1-2-week-old swine model, there was a significantly higher incidence of epicardial hemorrhage in the intervention group ($ETCO_2$-guided CPR, resulting in deeper chest compression) compared to the control group (standard CPR).[52] In an experimental swine VF model, chest compression to a depth of 35.2–57.0 mm (rescuer targeting a depth of 50 mm or 25% of the A–P diameter of the chest) found no rib fractures were evident in any pig.[46] In an experimental swine study comparing optimal chest compression depth (25% = 6 cm) in the A–P direction with conventional depth (4.2 cm—70% of optimal = ~17%), no evidence of CPR-related injury was found on necropsy in any animal.[45]

3.9.3 | Treatment recommendations

In dogs and cats that are positioned in lateral recumbency, we recommend providing chest compressions to a depth of one-third to one-half of the lateral diameter of the chest at the compression point (strong recommendation, very low quality of evidence).

In dogs and cats that are positioned in dorsal recumbency, we recommend providing chest compressions to a depth of one-quarter the A–P diameter of the chest at the compression point (strong recommendation, very low quality of evidence).

3.9.4 | Justification of treatment recommendations

Available evidence shows that in people receiving A–P chest compressions, the ideal compression depth is approximately 20%–25% of the depth of the A–P diameter at the compression point. Considering that the spine and epaxial musculature is thicker in most animals than the lateral body wall and considering that the main compressible anatomical part of the thorax is the lung, we estimate that the degree of thoracic space reduction achieved by 33%–50% compression of the lateral diameter of the chest would achieve the same degree of thoracic space reduction as a 20%–25% compression depth in the A–P orientation. Unfortunately, all of this evidence is based on human or animal studies in dorsal recumbency receiving sternal compressions, including the single canine study identified, which leaves open the question of specifically how deep compressions should be in the target species delivered in the presumed optimal state of lateral recumbency.

3.9.5 | Knowledge gaps

There are no direct studies evaluating the optimal compression depth in dogs and cats of varying size and conformation in lateral recumbency, and there is limited information for dorsal recumbency.

A maximum safe compression depth in dogs and cats is unknown, and the relationship between compression depth and risk of complications in animals in lateral recumbency is likely different than that in the most studied populations (people and pigs in dorsal recumbency).

Further, the risk of complications associated with hands over the heart (cardiac pump) versus hands over the widest part of the chest (thoracic pump) may be different and are unknown.

3.10 | Chest compression interruptions (BLS-08)

In cats and dogs in CPA (P), does the use of any other specific timing for interruptions to chest compressions to diagnose the heart rhythm (I), compared to ECG check every 2 minutes (C), improve favorable neurologic outcome, survival to discharge, ROSC, or surrogate markers of perfusion (O)?

3.10.1 | Introduction

During CPR, every pause of chest compressions will lead to cessation of vital organ blood flow. Even when the pause of chest compressions is short, it takes a substantial amount of time after resumption of chest compressions until maximum perfusion pressures are reestablished.[53] Therefore, even a very short pause can have a significant hemodynamic impact. Current human and previous veterinary CPR guidelines have recommended a pause for ECG evaluation every 2 minutes in an effort to minimize the number of chest compression pauses and in consideration of rescuer fatigue.[1,3] The ideal length of continuous chest compression cycle for dogs and cats is unknown.

3.10.2 | Consensus on science

For the most critical outcomes of favorable neurological outcome, survival to discharge, ROSC, and surrogate markers of perfusion, we identified no studies that addressed the PICO question.

There are currently no studies that directly addressed the question of specific timing for interruptions to chest compressions to diagnose the heart rhythm for any outcome of interest. There is evidence that the frequency and duration of interruptions to chest compressions can impact the outcome following CPR. In experimental pig studies, it has been shown that it takes approximately 60 seconds of continuous chest compressions to generate and maintain maximal CoPP, and pauses in chest compressions are associated with immediate decrease in CoPP.[53,54] There is some evidence from human clinical trials that a period of BLS CPR is beneficial prior to performing a rhythm check. In a prospective, observational analysis of witnessed human arrests, providing 2-minute (200 chest compressions at 100 compressions/min) blocks of uninterrupted chest compressions, pausing only to perform a rhythm check ± defibrillation, was associated with significant improvement in survival and neurological function when compared to a cohort of patients treated using the 2000 American Heart Association guidelines in which chest compressions were frequently interrupted.[55] Mosier et al. reported similar results in a retrospective analysis of the use of the same protocol.[56]

3.10.3 | Treatment recommendation

In intubated dogs and cats undergoing CPR, we recommend delivering CPR in 2-minute cycles of continuous high-quality chest compressions (strong recommendation, expert opinion).

3.10.4 | Justification of treatment recommendation

Recommendations for the 2-minute interval follow from the evidence that more frequent interruptions of chest compressions have been associated with worse outcome, while personnel may become fatigued when performing manual chest compressions for longer than 2 minutes (see BLS-15). As a pause is needed for changing of compressors, it is a logical time to check the ECG.

3.10.5 | Knowledge gaps

The optimal cycle duration of continuous high-quality chest compressions during CPR in dogs and cats is unknown.

It is unknown whether the 2-minute chest compression cycle should be paused for patient and ECG evaluation when there is a marked increase in $ETCO_2$ or another compelling sign of ROSC.

3.11 | Timing of chest compression cycles (BLS-15)

In cats and dogs in CPA (P), does performing chest compression cycles for an extended period of time (e.g., 5 minutes) (I), compared to 2-minute cycles (C), improve favorable neurologic outcome, survival to discharge, ROSC, surrogate markers of perfusion, or chest compression quality (rate, depth, leaning) (O)?

3.11.1 | Introduction

High-quality chest compressions are considered essential to successful CPR. The quality of chest compressions is impacted by the depth of compression, compression rate, and leaning during the recoil phase. There is evidence that the quality of chest compressions can deteriorate over time due to rescuer fatigue, and the current human and previous veterinary CPR guidelines recommend alternating rescuers every 2 minutes.[9,19]

3.11.2 | Consensus on science

For the most critical outcomes of favorable neurologic outcome, survival to discharge, ROSC, and surrogate markers of perfusion, we identified no studies addressing the PICO question.

For the important outcome of *chest compression quality*, we found 5 experimental studies and 1 observational study that addressed the PICO question.[57–62] One observational study of CPR in adults with IHCPA (very low quality of evidence, downgraded for serious indirectness and serious imprecision, upgraded for dose–response effect) found chest compression rate was consistent with a single rescuer for up to 3 minutes of chest compressions; however, chest compression depth significantly diminished at both 2 and 3 minutes. There was a significant linear decrease in depth of 6.6 ± 4.9 mm from 90 to 180 seconds, representing a 12.1% decay in compression depth during that time. Five experimental manikin studies were identified (low quality of evidence, downgraded for serious indirectness and serious imprecision, upgraded for dose–response effect); 2 of these studies found the quality of chest compressions after 1 minute was better than after 5 minutes. One study found that the quality of chest compressions after 1 minute was better than at 2 minutes and 1 study showed continuous deterioration in chest compression quality over 10 minutes of continuous compressions, with the mean percent of adequate chest compressions per 30-second interval being <70% at the 2-minute time point. In contrast, 1 manikin study found no difference in chest compression quality over 5 min of continuous chest compressions.

3.11.3 | Treatment recommendations

We recommend the cycles of chest compressions delivered by an individual rescuer not extend beyond 2 minutes in intubated dogs and cats undergoing CPR (strong recommendation, low quality of evidence).

We recommend that if a rescuer perceives they are becoming fatigued, or if other rescuers perceive inadequate chest compression quality, it is reasonable to change compressors during a cycle while minimizing interruption in chest compressions (<1 second) (strong recommendation, expert opinion).

3.11.4 | Justification of treatment recommendations

Several experimental studies show diminished chest compression quality between minute 1 and minutes 2–3 of chest compressions in a manikin model, and quality of compressions diminishes greatly when a single rescuer performs 5 or more minutes of continuous chest compressions. While there is also a decline in compression quality from 1 to 2 minutes, data show that compressions must be delivered for 60 seconds before reaching maximal arterial pressures during CPR.[53] Thus, the risk of decline in compression quality must be weighed against the potential for longer hands-off time that could result from pausing to change the compressor more frequently.

3.11.5 | Knowledge gaps

The ideal duration of manual, continuous chest compressions before switching rescuers in dogs and cats undergoing CPR is unknown.

The onset of rescuer fatigue when performing chest compressions in dogs and cats may be different than that documented in human manikin models and may differ depending on patient size and chest conformation.

3.12 | Timing of pauses in chest compressions (BLS-16)

In cats and dogs in CPA (P), does taking a longer pause (e.g., 30 s) (I), compared to minimizing pauses between compression cycles (e.g., <10 s) (C), improve favorable neurologic outcome, survival to discharge, ROSC, or surrogate markers of perfusion (O)?

3.12.1 | Introduction

Maintaining high-quality chest compressions is considered essential for successful CPR but interruptions to chest compressions are required to perform ECG rhythm checks, defibrillation, and other clinical interventions. Pauses in chest compressions can lead to reductions in coronary and cerebral blood flow and lead to worse survival

outcomes from CPR.[63] The current human CPR guidelines emphasize minimizing the hands-off time and limiting perishock pauses to less than 10 seconds.[9] There were no specific recommendations for duration of pause in chest compressions in the previous veterinary guidelines.[19]

3.12.2 | Consensus on science

For the most critical outcome of *favorable neurologic outcome*, we identified no studies that addressed the PICO question.

For the critical outcome of *survival to discharge*, we found 1 observational study and 1 experimental study that addressed the PICO question. An observational study in people, (low quality of evidence, downgraded for very serious indirectness, upgraded for large magnitude of effect and for dose–response effect) addressed the PICO question.[64] Using log-linear modeling, this study in people with OHCPA and a shockable rhythm showed a decrease in survival to hospital discharge of 14% for every 5-second increase in length of perishock pause up to 50 seconds.[64] In an experimental rodent study (very low quality of evidence, downgraded for very serious indirectness and serious imprecision, upgraded for large magnitude of effect), 25 rats underwent 4 minutes of fibrillatory arrest followed by 6 minutes of precordial compressions. Compressions were stopped and followed by a 0-, 10-, 20-, 30-, or 40-second pause before electrical defibrillation. Survival to 24 hours was 80% for rats with immediate defibrillation, 40% for those with a 10-second pause prior to defibrillation, and 0% ($P < 0.05$ compared to immediate defibrillation) for those with 20-, 30-, or 40-second pauses prior to defibrillation; no significant difference in 48-hour survival was found among groups.[65]

For the critical outcome of *ROSC*, we identified 2 observational and 2 experimental studies that addressed the PICO question. The 2 observational studies were in adult people experiencing OHCPA with shockable rhythms[64,66] (very low quality of evidence, downgraded for very serious indirectness, upgraded for large magnitude of effect). In 35 adult people experiencing VF, multivariate logistic regression analysis showed an adjusted OR for ROSC of 13.07 (95% CI: 3.42–49.94) with a preshock interval of <3 seconds and postshock interval of <6 seconds (total pause <9 s) compared to a total pause of ≥9 seconds.[66] Using log-linear modeling, another study in 815 people with OHCPA and a shockable rhythm showed that the OR of ROSC was 0.52 (95% CI: 0.27–0.97) with a perishock pause of ≥40 seconds compared to a perishock pause of <20 seconds.[64] Two experimental studies were identified, 1 in rodents and 1 in swine[65,67] (low quality of evidence, downgraded for very serious indirectness and serious imprecision, upgraded for large magnitude of effect and for dose–response effect). In 1 study, 25 rats underwent 4 minutes of fibrillatory arrest followed by 6 minutes of precordial compressions. Compressions were stopped and followed by a 0-, 10-, 20-, 30-, or 40-second pause before electrical defibrillation. Following defibrillation, ROSC was achieved in 100%, 60%, 60%, 20% ($P < 0.05$ compared to immediate defibrillation), and 0% ($P < 0.01$ compared to immediate defibrillation) animals, respectively.[65] In an experimental study including 60 pigs with a fibrillatory arrest model, a perishock pause in compressions of 40 seconds was associated with significantly worse ROSC than was a perishock pause between 0 and 20 seconds.[67]

Evidence was not summarized for "Outcome 4: Surrogate markers of perfusion" because of the evidence available for the more critical outcomes above.

3.12.3 | Treatment recommendation

We recommend minimizing pauses between compression cycles (<10 s) in dogs and cats during CPR (strong recommendation, low quality of evidence).

3.12.4 | Justification of treatment recommendation

Evidence in multiple species in different settings show that the longer the duration of pause in chest compressions, the less likely it is to achieve survival to discharge or ROSC. It should be noted that all available data are in VF arrest scenarios; however, the physiology occurring during pauses that would worsen outcome is likely similar regardless of ECG diagnosis and support minimizing pause duration. In addition, higher chest compression fractions during CPR (ie, minimizing hands-off time—not specifically addressed in this PICO question) have been associated with improved outcomes.[53] Minimizing pause duration increases chest compression fraction, which lends additional support to the treatment recommendation.

3.12.5 | Knowledge gaps

The ideal chest compression pause duration during which to evaluate the ECG during CPR in dogs and cats is unknown.

3.13 | Interrupting chest compression cycles (BLS-17)

In cats and dogs in CPA (P), does interrupting a 2-minute cycle of chest compressions if ROSC is suspected (I), compared to not interrupting the 2-minute cycle (C), improve favorable neurologic outcome, survival to discharge, ROSC, or complications (O)?

3.13.1 | Introduction

Minimizing interruptions in chest compressions has been associated with improved outcomes (see BLS-16). A 2-minute chest compression cycle is recommended to optimize cardiac output but still allow pauses for patient assessment (see BLS-15). If ROSC is suspected during a 2-minute chest compression cycle, the rescuers have the option of pausing chest compressions to determine if ROSC is present, or to

complete the cycle and assess the patient at the scheduled pause. In human CPR guidelines for children and adults, no evidence was identified to assess this question as of 2020 and the treatment recommendation was to minimize interruptions in chest compressions and avoid pausing to evaluate circulation, without strong suspicion of ROSC.[9]

3.13.2 | Consensus on science

For the most critical outcomes of favorable neurologic outcome, survival to discharge, and ROSC, we identified no studies addressing the PICO question.

3.13.3 | Treatment recommendations

We suggest interrupting a 2-minute chest compression cycle only when ROSC is suspected based on a combination of (1) a sudden and persistent increase in $ETCO_2$ of great magnitude (eg, by ≥10 mm Hg to reach a value that is ≥35 mm Hg) and (2) evidence of an arterial pulse distinct from chest compressions (weak recommendation, expert opinion).

In the absence of capnography data, we recommend against interruption of a 2-minute chest compression cycle even if ROSC is suspected (strong recommendation, expert opinion).

3.13.4 | Justification of treatment recommendations

Harm to patients undergoing CPR when not actually in CPA has been shown to be minimal,[19] and hands-off time has been associated with nonsurvival. Additionally, a study in adults with nontraumatic OHCPA found that a rise in $ETCO_2$ of ≥10 mm Hg was highly specific (97%, 95% CI: 91%–99%), though poorly sensitive for ROSC (33%, 95% CI: 22%–47%); specificity was 100% for people with noncardiac causes of CPA (eg, nonshockable rhythms).[68] There is evidence in experimental pigs of significant and potentially clinically meaningful impairment of perfusion (arterial pressure, CoPP) when chest compressions are administered to animals with a spontaneous heartbeat.[50]

3.13.5 | Knowledge gaps

There is no evidence regarding the interruption of 2-minute chest compression cycles in dogs and cats when ROSC is suspected.

3.14 | Chest-compression-only CPR (BLS-10)

In nonintubated cats and dogs in CPA or during single-rescuer CPR in cats and dogs (P), does chest-compression-only CPR (I) when compared to conventional CPR (C) improve favorable neurologic outcome, survival to discharge, $PaCO_2$, oxygenation, ROSC, or surrogate markers of perfusion (O)?

3.14.1 | Introduction

Chest compressions are recognized as an essential component of BLS. When relying on lay persons for CPR, there is some debate about whether it is preferable to focus on chest compressions only (no ventilation) to make it easier to teach, remember, and perform. However, there is concern that compression-only CPR may be less effective because of inadequate oxygenation and ventilation. Ventilation during CPR is considered to have particular importance in asphyxial CPAs, which are believed to be the most common type of CPA in dogs and cats. The current human guidelines for adults with OHCPA recommend chest compressions be performed for all patients and suggest that rescue breaths be provided if the rescuer is trained and willing.[9] In human infants and children in whom the cause of CPA is most likely to be asphyxial in nature, there is more emphasis on ventilation during CPR, and current guidelines recommend CPR with chest compressions and rescue breaths for this population.[69]

3.14.2 | Consensus on science

For the most critical outcome of *favorable neurological outcome*, we identified 28 studies including 1 clinical trial in adult human beings (low quality of evidence, downgraded for very serious indirectness),[70] 10 experimental swine trials (very low quality of evidence, downgraded for serious risk of bias, serious indirectness, and serious inconsistency),[54,71–79] and 17 observational studies in people (very low quality of evidence, downgraded for serious risk of bias, very serious indirectness, and serious inconsistency)[79–96] that addressed the PICO question. One randomized, multicenter clinical trial of adults with OHCPA receiving dispatcher-instructed bystander CPR found no difference in favorable neurologic outcome between compression-only CPR and conventional CPR.[70] Four experimental swine studies, 2 of which were asphyxial arrest models, found that conventional CPR was superior for favorable neurologic outcome,[71,73,74,76] whereas 6 experimental swine studies found no difference between compression only CPR and conventional CPR.[54,73,75,77–79] Five observational studies in people found conventional CPR was associated with a better neurologic outcome[85,86,89,91,96] when compared to compression-only CPR; however, 12 human clinical observational studies showed no difference in this outcome with compression-only CPR versus conventional CPR.[80–84,87,88,90,92–95]

For the next critical outcome of *survival to discharge*, we identified 3 clinical trials (low quality of evidence, downgraded for very serious indirectness)[70,97,98] and 5 observational studies (very low quality of evidence, downgraded for very serious indirectness and serious inconsistency)[83,89–91,94] that addressed the PICO question. Three randomized human clinical trials found no difference in survival between compression-only and conventional CPR.[70,97,98] Four human clinical

observational studies found no difference in survival to discharge with compression-only CPR compared to conventional CPR.[83,89,90,94] However, 1 study in people with OHCPA found compression-only CPR to be superior to conventional for survival to hospital discharge.[91]

For the important outcome of $PaCO_2$, we identified 15 experimental animal trials addressing the PICO question (very low quality of evidence, downgraded for very serious risk of bias, very serious risk of indirectness, and serious imprecision, and upgraded for large effect).[54,71–74,76–78,99–105] Thirteen experimental swine studies found $PaCO_2$ to be significantly higher with compression-only CPR when compared to conventional CPR.[54,71–74,76,78,99–102,104] Two experimental animal studies (1 dog, 1 swine) found no difference in $PaCO_2$ between CPR types.[103,105]

For the important outcome of *oxygenation*, we identified 14 experimental studies (very low quality of evidence, downgraded for serious risk of bias, very serious indirectness, and very serious imprecision, and upgraded for large effect) that addressed the PICO question.[54,71–74,76–78,99–103,105] Thirteen experimental animal studies (12 in swine, 1 in dogs) found oxygenation to be significantly decreased with compression-only CPR when compared to conventional CPR.[54,71–74,76–78,99–103,105] One swine study found no difference in oxygenation between CPR types.[78]

The outcomes of ROSC and surrogate markers of perfusion were not evaluated since a recommendation could be made based on these 4 more critical outcomes.

3.14.3 | Treatment recommendations

In nonintubated dogs and cats undergoing CPR or during single-rescuer CPR, we recommend provision of rescue breaths if feasible and safe during pauses in chest compressions (strong recommendation, very low quality of evidence).

In nonintubated dogs and cats undergoing CPR, we recommend the use of a tight-fitting facemask and a manual resuscitator to deliver rescue breaths (strong recommendation, very low quality of evidence).

In nonintubated dogs and cats undergoing CPR that pose minimal risk to the rescuer (eg, due to potential for zoonotic disease or narcotics exposure), when a tight-fitting facemask and manual resuscitator are not available, we recommend provision of rescue breaths via the mouth-to-nose (mouth-to-snout) technique (strong recommendation, very low quality of evidence).

In nonintubated dogs and cats that may pose risk to the rescuer (eg, due to potential for zoonotic disease or narcotics exposure), when a tight-fitting facemask and manual resuscitator are not available, we recommend chest-compression-only CPR (strong recommendation, expert opinion).

3.14.4 | Justification of treatment recommendations

The recommendation for provision of rescue breaths to nonintubated dogs and cats undergoing CPR is based on the evidence that compression-only CPR was generally found to be inferior or equivalent to conventional CPR for the critical outcomes of favorable neurologic outcome and survival. Because most CPA in cats and dogs is believed to be asphyxial (noncardiac) in nature, rescue breathing during CPR may have greater importance in these animals than it does in most experimental and human clinical populations, in which cardiac causes of CPA predominate. Thus, we also based the decision to recommend rescue breaths in our nonintubated populations on the convincing evidence (large treatment effect) of improved $PaCO_2$ and oxygenation seen in experimental studies of CPR when rescue breaths are delivered during CPR. Recent data have shown that both oxygenation and ventilation are improved in dogs undergoing CPR when manual breaths are delivered via the mouth-to-nose technique or a tight-fitting facemask and manual resuscitator compared to dogs receiving chest-compression-only CPR.[105]

Given the possibility of risks to rescuers, and the apparent equivalence of these 2 techniques for delivery of rescue breaths, we recommend the use of a tight-fitting facemask and manual resuscitator if available. In environments in which zoonotic disease or narcotics exposure potential is high and a tight-fitting mask and manual resuscitator are not available, we recommend that compression-only CPR be administered to minimize risk to rescuers. Conversion to ventilation with face mask and resuscitator or by endotracheal intubation should occur as early as possible.

3.14.5 | Knowledge gaps

Direct comparisons of compression-only CPR to conventional CPR in cats and dogs are lacking for the most critical outcomes of favorable neurologic outcome, survival to discharge, and ROSC. As the predominant cause of CPA is different in human medicine, there is limited translational relevance of human clinical trials to veterinary medicine, and future veterinary clinical trials are needed.

The efficacy of rescue breathing in nonintubated cats and dogs as well as the pause in chest compressions associated with rescue breathing have not been fully evaluated.

The ability and willingness of lay persons to perform CPR with rescue breathing in animals are unknown.

3.15 | Active compression–decompression (BLS-06)

In cats and dogs in CPA (P), does active compression–decompression (ACD) (I), compared to active compression/passive decompression chest compressions (C), improve favorable neurologic outcome, survival to discharge, ROSC, or surrogate markers of perfusion (O)?

3.15.1 | Introduction

ACD-CPR uses a handheld device with a suction cup applied to the midsternal region in human patients to allow active lifting of the chest wall during the chest decompression phase of CPR. This enhances

negative intrathoracic pressure generated during chest recoil, augmenting venous return. This PICO question investigated the utility of ACD-CPR in dogs and cats.

3.15.2 | Consensus on science

For the critical outcome of *favorable neurologic outcome*, we identified 7 human clinical trials (very low quality of evidence, downgraded for serious risk of bias and very serious indirectness) and 1 observational study (very low quality of evidence, downgraded for serious risk of bias and very serious indirectness) that addressed the PICO question.[106–113] One clinical trial showed improved favorable neurologic outcome with ACD, but this was not repeatable in the other trials.[106,108–113] The observational study failed to demonstrate a difference.[107]

For the next critical outcome of *survival to discharge*, we identified 12 human clinical trials (very low quality of evidence, downgraded for serious risk of bias and very serious indirectness) and 1 observational study (very low quality of evidence, downgraded for serious risk of bias and serious indirectness) that addressed the PICO question.[107,109,110,112–118] Of the relevant human clinical trials using ACD-CPR compared to standard CPR, none found a significant difference in survival to discharge.[109,110,112–119] The observational study failed to demonstrate a difference.[107]

For the important outcome of *ROSC*, we identified 11 human clinical trials (very low quality of evidence, downgraded for serious risk of bias and very serious indirectness), 3 experimental studies (very low quality of evidence, downgraded for serious risk of bias and serious indirectness), and 1 observational study (very low quality of evidence, downgraded for serious risk of bias and serious indirectness).[109,110,112–122] Of the relevant human clinical trials evaluating the use of ACD-CPR compared to standard CPR, these studies yielded mixed results but overall do not support that ACD-CPR improves ROSC.[106,108,110–114,116–118,123] There are no relevant veterinary studies.

For the important outcome of *surrogate markers of perfusion*, we identified 15 experimental studies (very low quality of evidence, downgraded for serious risk of bias and serious indirectness).[120–122,124–135] An experimental dog study provides evidence that ACD increases left ventricular pressure time product, coronary perfusion, cardiac output, and pulmonary artery flow.[128] Increased minute ventilation with ACD has also been shown.[125] A study of 8 beagle dogs showed increased cerebral and pulmonary blood flow in dogs during ACD-CPR compared to conventional CPR.[127] Several porcine experimental models show improvements in surrogate markers (cerebral, carotid, renal, or myocardial blood flow, cardiac output, or blood pressure) with ACD-CPR.[120–122,124,126,129–135]

3.15.3 | Treatment recommendation

We recommend against the use of ACD-CPR in dogs and cats (strong recommendation, expert opinion).

3.15.4 | Justification of treatment recommendation

The majority of the evidence evaluated did not support a benefit of ACD during CPR in human patients, despite the fairly consistent improvement in surrogate markers of perfusion found in experimental animal studies. In addition, adherence of the ACD device's suction cup to the thoracic wall, and thus the applicability to dogs and cats in the clinical setting (eg, those with full haircoats), is limited.

3.15.5 | Knowledge gaps

Evaluation of prioritized outcomes (favorable neurological outcome, survival to discharge) for ACD-CPR versus conventional CPR in dogs and cats is needed to help determine whether clipping fur during CPR to apply an ACD would be worthwhile. Alternatively, development and evaluation of safe, clinically applicable ACD equipment in dogs and cats would be needed.

4 | AIRWAY AND VENTILATION MANAGEMENT

Establishing a patent airway enables the provision of ventilation during CPR, which allows for gas exchange at the level of the alveolus. This section reviews ventilatory management in CPR.

4.1 | Inspiratory time and tidal volume (BLS-13)

In cats and dogs in CPA (P), does providing ventilation with other inspiratory times (I-times) and tidal volumes (TVs) (I), compared to a 1-second I-time and TV of about 10 mL/kg (C), improve favorable neurologic outcome, survival to discharge, ROSC, $PaCO_2$, oxygenation, or surrogate markers of perfusion (O)?

4.1.1 | Introduction

The optimal TV and I-time during ventilation in CPR would generate an appropriate $PaCO_2$ and oxygenation while minimizing the negative cardiovascular effects of positive pressure ventilation. The current human guidelines for CPR generally recommend a TV of 6–7 mL/kg (up to 10 mL/kg in some circumstances) and an I-time of 1 second.[9] The previous veterinary guidelines recommend a TV of 10 mL/kg and an I-time of 1 second.[19] These recommendations were based on a limited body of evidence and equipoise remains.

4.1.2 | Consensus on science

For the most critical outcome of *favorable neurologic outcome*, we identified 1 experimental VF study in swine that addressed the PICO question (very low quality of evidence, downgraded for very serious

indirectness and serious imprecision).[136] This study found no difference in neurological outcome at 24 hours when comparing a TV of 10 mL/kg at 10 bpm to a TV of 20 mL/kg at 35 bpm; I-time was not described.[136]

For the next critical outcome of *survival to discharge*, we identified 1 experimental VF study in swine that addressed the PICO question (very low quality of evidence, downgraded for very serious indirectness and serious imprecision).[136] This study found no difference in survival at 24 hours when comparing a TV of 10 mL/kg at 10 bpm to a TV of 20 mL/kg at 35 bpm; I-time was not described.[136]

For the next critical outcome of *ROSC*, we identified 3 experimental VF studies in swine that addressed the PICO question (very low quality of evidence, downgraded for very serious indirectness and serious imprecision).[136–138] One study compared a TV of 10 mL/kg at 10 bpm to a TV of 20 mL/kg at 35 bpm (I-time not described)[136]; 1 study compared a TV of 10 mL/kg at baseline respiratory rate (RR) ~17 bpm to a TV of 7 mL/kg at 10 bpm, both with a 2-second I-time; and the final study compared a TV of 8–9 mL/kg at 10 bpm to a TV of 2–3 mL/kg at 50 bpm (I-time not described).[138] No study found a difference in ROSC between respective treatment groups.[136–138]

For the important outcome of $PaCO_2$, we identified 3 experimental VF studies in swine that addressed the PICO question (very low quality of evidence, downgraded for very serious indirectness and serious imprecision).[136–138] One study compared a TV of 10 mL/kg at 10 bpm to a TV of 20 mL/kg at 35 bpm (I-time not described)[136]; 1 study compared a TV of 10 mL/kg at baseline RR (~17 bpm) to a TV of 7 mL/kg at 10 bpm, both with a 2-second I-time[137]; and the final study compared a TV of 8–9 mL/kg at 10 bpm to a TV of 2–3 mL/kg at 50 bpm (I-time not described).[138] No study found a difference in $PaCO_2$ between respective treatment groups.[136–138]

For the important outcome of *oxygenation*, we identified 3 experimental VF studies in swine that addressed the PICO question (very low quality of evidence, downgraded for very serious indirectness and serious imprecision).[136–138] One study found PaO_2 was higher with a 10-mL/kg TV delivered at a baseline RR (~17 bpm) versus a 7-mL/kg TV at 10 bpm, both with 2-second I-times.[137] The remaining 2 studies found no difference in PaO_2 when comparing a TV of 10 mL/kg at 10 bpm to a TV of 20 mL/kg at 35 bpm (I-time not described)[136] or a TV of 8–9 mL/kg at 10 bpm to a TV of 2–3 mL/kg at 50 bpm (I-time not described).[138]

For the important outcome of *surrogate markers of perfusion*, we identified 3 experimental VF studies in swine that addressed the PICO question (very low quality of evidence, downgraded for very serious indirectness and serious imprecision).[136–138] One study compared a TV of 10 mL/kg at 10 bpm to a TV of 20 mL/kg at 35 bpm (I-time not described)[136]; 1 study compared a TV of 10 mL/kg at baseline RR (~17 bpm) to a TV of 7 mL/kg at 10 bpm, both with a 2-second I-time[137]; and the final study compared a TV of 8–9 mL/kg at 10 bpm to a TV of 2–3 mL/kg at 50 bpm (I-time not described).[138] No study found a difference in hemodynamic parameters between respective treatment groups.[136–138]

4.1.3 | Treatment recommendation

We recommend administering positive pressure ventilation at a TV of 10 mL/kg and a 1-second I-time during CPR in intubated dogs and cats (strong recommendation, very low quality of evidence).

4.1.4 | Justification of treatment recommendation

The evidence identified found a TV of ~10 mL/kg had similar outcomes to several other TVs evaluated. Overall, this treatment recommendation is based on a very low level of evidence that failed to identify a superior option. I-time is very poorly evaluated in the literature, and the treatment recommendation is based on common clinical practice and previous consensus statements.

4.1.5 | Knowledge gaps

The ideal I-time and TV in intubated dogs and cats during CPR are unknown.

4.2 | Ventilation rate (BLS-14)

In cats and dogs in CPA (P), does any other ventilation rate (I), as opposed to a ventilation rate of 10 bpm (C), improve favorable neurologic outcome, survival to discharge, ROSC, surrogate markers of perfusion, $PaCO_2$, or oxygenation (O)?

4.2.1 | Introduction

Providing rescue breaths during CPR has been shown to improve oxygenation, ventilation, and outcomes, but these benefits must be weighed against the potential negative cardiovascular consequences of positive pressure breaths. Increases in intrathoracic pressure associated with positive pressure breaths can reduce venous return and cardiovascular performance during chest compressions.[139] In addition, it has long been known that hyperventilation can lead to hypocapnia-associated cerebral vasoconstriction that may worsen neurologic outcomes.[140] The current human and previous veterinary CPR guidelines recommend a ventilation rate of 10 bpm.[9,19]

4.2.2 | Consensus on science

For the most critical outcome of *favorable neurologic outcome*, we found 3 observational studies in people (very low quality of evidence, downgraded for very serious indirectness, serious imprecision, and serious inconsistency) and 1 experimental study in swine (very low quality of evidence, downgraded for very serious indirectness

and serious imprecision) that addressed the PICO question.[136,141–143] In an observational study directly comparing CPR ventilation rates (mean ± SD) in 285 adult survivors of OHCPA with a better Cerebral Performance Category (CPC) of 1–2 to rates in those with worse CPC of 3–5, CPR ventilation rates had been higher (12.7 ± 6.1 vs 7.3 ± 3.5 bpm) in patients who achieved a better CPC. Additionally, in multivariate analysis, increasing ventilation rate was associated with a favorable neurologic outcome (CPC 1–2 vs 3–5) (OR: 3.795, 95% CI: 1.507–9.557).[142] Another study in 337 adults with OHCPA compared ventilation rates of <10 bpm to rates >10 bpm and found no difference between groups in favorable neurologic outcome at 1 year.[141] In 18 children experiencing IHCPA, a ventilation rate of >30 bpm in <1-year-olds or >25 bpm in 1- to 17-year-olds had an OR of 4.73 (95% CI: 1.17–19.13) for favorable neurologic outcome compared to lower rates.[143] An experimental swine study using a fibrillatory arrest model compared favorable neurologic outcome at 24 hours in pigs receiving a ventilation rate of 10 bpm at 10 mL/kg to those receiving a rate of 35 bpm at 20 mL/kg; no difference in favorable neurologic outcome was found.[136]

For the critical outcome of *survival to discharge*, we found 2 observational studies[141,143] (very low quality of evidence, downgraded for very serious indirectness, serious imprecision, and serious inconsistency) and 1 experimental animal trial[136] (very low quality of evidence, downgraded for very serious indirectness and serious imprecision) that addressed the PICO question. In 47 children experiencing IHCPA, a ventilation rate of >30 bpm in <1-year-olds or >25 bpm in 1- to 17-year-olds had an OR of 4.73 (95% CI: 1.32–16.27) for improved survival to discharge compared to patients in whom lower RRs were used.[143] Another study in 337 adults with OHCPA compared ventilation rates of <10 bpm to rates >10 bpm and found no difference between groups in likelihood of survival to discharge.[141] An experimental swine study using a fibrillatory arrest model compared survival at 24 hours in pigs receiving a ventilation rate of 10 bpm at 10 mL/kg to those receiving a rate of 35 bpm at 20 mL/kg; no difference in 24-hour survival was found.[136]

For the critical outcome of *ROSC*, we identified 2 observational studies[141,143] (very low quality of evidence, downgraded for very serious indirectness, serious imprecision, and serious inconsistency) and 4 experimental studies[136,141,144–146] (very low quality of evidence, downgraded for serious indirectness, serious imprecision, and serious inconsistency) that addressed the PICO question. One study in 337 adults with OHCPA compared ventilation rates of <10 bpm to rates >10 bpm and found no difference between groups in likelihood of ROSC.[141] In 47 children experiencing 52 events of IHCPA, a ventilation rate of >30 bpm in <1-year-olds or >25 bpm in 1- to 17-year-olds had an OR of 4.64 (95% CI: 1.17–19.13) for increased incidence of ROSC compared to events in which lower RRs were used.[143] An experimental swine study using a fibrillatory arrest model compared ROSC in pigs receiving a ventilation rate of 10 bpm at 10 mL/kg to those receiving a rate of 35 bpm at 20 mL/kg; no difference in ROSC was found.[136] In a second swine study of fibrillatory CPA, an RR of 10 versus 33 bpm at TVs of both 6 and 18 mL/kg had no statistical association with ROSC.[144] An experimental piglet study of asphyxial arrest compared ventilation rates of 10, 20, and 30 bpm and found no difference in ROSC.[145] A final experimental swine study compared CPR using 12 or 30 bpm; ROSC was significantly greater (6/7) in pigs receiving 12 bpm compared to those (1/7) receiving 30 bpm.[146]

For the important outcome of *surrogate markers of perfusion*, we found 1 observational study[143] (very low quality of evidence, downgraded for very serious indirectness and serious imprecision) and 8 experimental animal studies[136,143–150] (very low quality of evidence, downgraded for very serious indirectness, serious imprecision, and serious inconsistency) that addressed the PICO question. In 47 children experiencing 52 events of IHCPA, ventilation rate was not associated with arterial blood pressure.[143] In an experimental swine study, an RR of 10 versus 33 bpm at TVs of both 6 and 18 mL/kg was not associated with aortic pressure, right atrial pressure, carotid blood flow, CoPP, or cerebral perfusion pressure. $ETCO_2$ was lower with higher ventilation rates and TVs.[144] A fibrillatory swine study found that an RR of 2 bpm resulted in lower brain oxygen tension, carotid blood flow, right atrial systolic pressure, and intracranial systolic pressure, than did an RR of 10 bpm.[150] In a fibrillatory canine model comparing an RR of 10 bpm (not synchronized) with an RR of 30 bpm (synchronized), dogs ventilated at 30 bpm (synchronized) had higher right atrial pressure, higher carotid artery pressure, higher jugular vein pressure, and higher carotid artery-to-right atrial pressure gradient (an approximation of cerebral perfusion pressure). Left carotid artery flow was also significantly higher in the 30 bpm (synchronized) group.[148] An additional 5 experimental swine studies showed no difference in any surrogate marker of perfusion measured with variable ventilation rates.[136,145–147,149]

For the important outcome of $PaCO_2$, we found 4 experimental animal studies (very low quality of evidence, downgraded for very serious indirectness, serious imprecision, and serious inconsistency) that addressed the PICO question.[136,145,146,148] An experimental swine study of fibrillatory arrest compared ventilation patterns of 10 bpm at 10 mL/kg versus 35 bpm at 20 mL/kg and showed that the 35 bpm group had an inappropriately low $PaCO_2$, while the 10 bpm group had mean $PaCO_2$ in the low 30s.[136] An experimental swine study comparing 12 versus 30 bpm showed that the 30 bpm group had a lower $PaCO_2$.[146] In an asphyxial arrest model in 1- to 2-month-old piglets comparing 10 versus 20 versus 30 bpm, no differences in $PaCO_2$ at 3, 8, 18, and 24 minutes of CPR were found.[145] In an experimental canine study comparing an RR of 10 bpm (not synchronized) with 30 bpm (synchronized), no difference in $PaCO_2$ at 7 min of CPR was found.[148]

For the important outcome of *oxygenation*, we found 5 experimental studies (very low quality of evidence, downgraded for very serious indirectness and serious imprecision) that addressed the PICO question.[136,144,145,148,149] All 5 experimental studies (4 swine and 1 canine) found no differences in oxygenation with different ventilation rates used.[136,144,145,148,149]

4.2.3 | Treatment recommendation

In intubated dogs and cats undergoing CPR, we recommend an RR of 10 bpm (strong recommendation, very low quality of evidence).

4.2.4 | Justification of treatment recommendation

For the outcome of survival to discharge and favorable neurological outcome, there are mixed findings. The studies suggesting a benefit of higher ventilation rates were all of a very low quality for our population. However, for the outcomes of ROSC, surrogate markers of perfusion, and $PaCO_2$, a preponderance of the evidence supports the use of lower ventilation rates. The committee selected a ventilation rate of 10 per minute to maintain consistency for the purpose of $ETCO_2$ monitoring and for ease of rescuer performance.

4.2.5 | Knowledge gaps

The ideal ventilation rate during CPR for dogs and cats is unknown.

4.3 | Peak inspiratory pressure (BLS-19)

In cats and dogs in CPA (P), does the use of any other specific peak inspiratory pressure (PIP) (I), compared to 40 cm H_2O PIP (C), improve favorable neurologic outcome, survival to discharge, ROSC, or complications (O)?

4.3.1 | Introduction

Ventilation during CPR can be performed manually or with a mechanical ventilator. Chest compressions increase intrathoracic and airway pressures, necessitating application of higher than normal (for that patient) PIP to facilitate ventilation. The optimal PIP during CPR has not been defined in human or veterinary medicine and is likely to vary between patients depending on factors such as thoracic conformation, respiratory pathology, and endotracheal tube size. The current human CPR guidelines recommend ventilation force be sufficient to generate a visible chest rise.[9]

4.3.2 | Consensus on science

For the most critical outcomes of favorable neurological outcome, survival to discharge, ROSC, and complications, we identified no studies that addressed the PICO question.

4.3.3 | Treatment recommendations

We recommend that a PIP be applied that creates visible but not excessive chest rise (strong recommendation, expert opinion).

We recommend against the routine use of a PIP that exceeds 40 cm H_2O (strong recommendation, expert opinion).

4.3.4 | Justification of treatment recommendations

Changes in chest wall compliance due to chest compressions result in variable TVs being delivered when a static PIP is applied in the setting of closed-chest CPR. As TV is a major determinant of $PaCO_2$ and given the variability of PIP required to generate adequate TVs during CPR, we believe that the parameter of chest rise is more physiologically relevant to adequate ventilation in this setting than PIP.

Excessive airway pressures can be harmful. Thus, the committee decided to recommend a maximum of no more than 40 cm H_2O PIP, consistent with the release valve limits commonly used on commercial rebreathing bags[c]. It should be noted that animals with lung disease that severely limits pulmonary compliance may require PIP in excess of 40 cm H_2O to achieve adequate TVs while receiving external chest compressions.

4.3.5 | Knowledge gaps

No evidence is available regarding effective and safe PIPs for manual ventilation during chest compressions in dogs and cats.

4.4 | Ventilation technique in patients undergoing mechanical ventilation (BLS-20)

In cats and dogs in CPA already on a mechanical ventilator (P), does continuing mechanical ventilation (I) compared to switching to manual ventilation (C) improve survival to discharge, ROSC, surrogate markers of perfusion, $PaCO_2$, or oxygenation (O)?

4.4.1 | Introduction

Providing rescue breaths during CPR is considered important to maintain oxygenation and to optimize $PaCO_2$. The use of a mechanical ventilator during CPR could be beneficial if it can provide appropriate respiratory support and alleviate the need for personnel to be dedicated to provision of rescue breaths. There are concerns that the positive intrathoracic pressure generated by chest compressions could interfere with the operation of a mechanical ventilator and lead to inadequate ventilation. There are no recommendations in the current human or previous veterinary CPR guidelines on the best option for providing rescue breaths to patients who develop CPA while on mechanical ventilation.[9,19]

4.4.2 | Consensus on science

For the most critical outcome of favorable neurologic outcome or survival to discharge, we identified no studies that addressed the PICO question.

For the critical outcome of *ROSC*, we identified 1 experimental study (low quality of evidence, downgraded for serious indirectness) that

addressed the PICO question.[151] In a newborn piglet model of hypoxic CPA, no difference was found in ROSC among groups treated with 3 different types of respiratory support during CPR (T-piece, self-inflating bag, or mechanical ventilator).[151]

For the important outcome of *surrogate markers of perfusion*, we found 2 experimental studies (low quality of evidence, downgraded for serious indirectness).[151,152] In a newborn piglet model of hypoxic CPA, no difference was found in measured hemodynamic parameters among groups treated with 3 different types of respiratory support during CPR (T-piece, self-inflating bag, or mechanical ventilator).[151] In another newborn piglet study using an asystolic CPA model, there was no difference in plasma lactate concentration among groups treated with 3 different types of respiratory support (T-piece vs rebreathing bag vs ventilator).[152]

For the important outcome of $PaCO_2$, we found 2 experimental studies (low quality of evidence, downgraded for serious indirectness).[151,152] In a newborn piglet model of hypoxic CPA, no difference was found in $PaCO_2$ among groups treated with 3 different types of respiratory support during CPR (T-piece, self-inflating bag, or mechanical ventilator).[151] In another newborn piglet study using an asystolic CPA model, there was no difference in $PaCO_2$ among groups treated with 3 different types of respiratory support (T-piece vs rebreathing bag vs ventilator).[152]

For the important outcome of *oxygenation*, we found 2 experimental studies (low quality of evidence, downgraded for serious indirectness).[151,152] In a newborn piglet model of hypoxic CPA, no difference was found in oxygenation among groups treated with 3 different types of respiratory support during CPR (T-piece, self-inflating bag, or mechanical ventilator).[151] In another newborn piglet study using an asystolic CPA model, there was no difference in oxygenation among groups treated with 3 different types of respiratory support (T-piece vs rebreathing bag vs ventilator).[152]

4.4.3 | Treatment recommendations

In dogs and cats that experience CPA while undergoing mechanical ventilation, we suggest switching to manual ventilation (weak recommendation, expert opinion).

If delivering breaths by mechanical ventilator during CPR in dogs and cats, ventilator settings should be adjusted to assure breaths are delivered (eg, volume control mode; TV 10 mL/kg; ventilation rate 10/min; positive end-expiratory pressure [PEEP] 0 cm H_2O; pressure limit 40 cm H_2O; and a trigger sensitivity least likely to detect a breath [e.g., −10 cm H_2O]) (strong recommendation, very low quality of evidence).

4.4.4 | Justification of treatment recommendations

The studies identified, both in newborn piglets, found no differences in the critical and important outcomes evaluated, whether ventilation was provided with a mechanical ventilator or with manual ventilation. Given the time delay and possible errors introduced in adjusting a mechanical ventilator adequately for breath delivery during CPR, we believe that switching to a manual ventilatory device may be easier and more efficient. In addition, a study in pigs found that using clinically relevant patient trigger variables during CPR resulted in hyperventilation, lower minute ventilation, and poorer oxygenation compared to those achieved by disabling the trigger sensitivity or using a trigger setting of −20 cm H_2O.[153] These results suggest that it may be necessary to adjust ventilator settings to ensure appropriate respiratory support is provided during CPR. It is important to note that the application of PEEP during CPR could have negative hemodynamic effects, and the optimal settings for PEEP during CPR are unknown. Also, animals with lung disease that severely limits pulmonary compliance may require PIP in excess of 40 cm H_2O to achieve adequate TVs while receiving external chest compressions.

4.4.5 | Knowledge gaps

Whether mechanical ventilation during CPR is superior or inferior to manual ventilation in dogs and cats is unknown.

The optimal mechanical ventilator settings for use during CPR are unknown, including the use of PEEP.

4.5 | Compression:ventilation ratio (BLS-09)

In nonintubated cats and dogs in CPA or during single-rescuer CPR in cats and dogs (P), does the use of another specific compression-to-ventilation (C:V) ratio (I), compared to a C:V ratio of 30:2 (C), improve favorable neurologic outcome, survival to discharge, ROSC, $PaCO_2$, oxygenation, or surrogate marker(s) of perfusion (O)?

4.5.1 | Introduction

When providing CPR to animals without an advanced airway such as an endotracheal tube, or during single-rescuer CPR, it is necessary to pause chest compressions in order to provide ventilation. The current human and previous veterinary CPR guidelines recommend a C:V ratio of 30:2.[9,19]

4.5.2 | Consensus on science

For the critical outcome of *favorable neurologic outcome*, we identified 2 experimental studies in swine (low quality of evidence, downgraded for serious indirectness) and 1 observational study in people (very low quality of evidence, downgraded for very serious risk of bias and very serious indirectness) that addressed the PICO question.[76,79,154] One experimental study in 42 swine compared neurologic outcome at 24 hours in pigs treated with either chest-compression-only CPR, CPR at a ratio of 30 compressions:1 room air ventilation, or CPR at a ratio of 30 compressions:2 room air ventilations.[79] This study found that pigs undergoing CPR at a C:V ratio of 30:1 had significantly better favorable neurologic outcome at 24 hours than those in

either the chest-compression-only group or those receiving C:V at a ratio of 30:2. Another experimental study in 40 pigs undergoing CPR compared favorable neurologic outcome among 4 C:V ratios: 15:2, chest-compression-only CPR, 50:5, or 100:2.[76] Ventilated gas was simulated exhaled gas with 4% CO_2 and 17% O_2. This study showed that pigs treated with a C:V of 100:2 had significantly better favorable neurologic outcome at 24 hours than those treated with either 15:2 or chest-compression-only CPR. One observational study (very low quality of evidence, downgraded for very serious risk of bias and very serious indirectness) in adult human beings with OHCPA found no difference in favorable neurologic outcome in survivors between patients treated in the field with a C:V ratio of 30:2 versus those treated with a C:V ratio of 15:2.[154]

For the critical outcome of *survival to discharge*, we identified 4 experimental studies (very low quality of evidence, downgraded for serious risk of bias and very serious indirectness) and 2 observational studies (very low quality of evidence, downgraded for very serious risk of bias and serious indirectness) that addressed the PICO question.[76,79,154–157] Two experimental studies in swine showed no difference in 24-hour survival with C:V of 30:2 versus 30:1.[76,79] One experimental study in 20 pigs showed no difference in 24-hour survival when pigs underwent either compression-only CPR, C:V of 15:1, or C:V of 5:1.[156] This study included other interventions that likely yield its results less valuable for this PICO question.[156] The single study in 30 dogs monitored subjects that achieved ROSC and found no difference in 2-hour survival among dogs treated with 15:1, 15:2, or 30:2 C:V ratios.[157] Two human clinical observational studies showed improved survival to discharge with C:V of 30:2 when compared to 15:2; both studies included many additional interventions in combination with the change in C:V.[154,155]

For the critical outcome of *ROSC*, we identified 5 experimental studies (very low quality of evidence, downgraded for serious risk of bias, serious indirectness, and serious imprecision)[79,100,102,156,157] and 2 observational studies (very low quality of evidence, downgraded for very serious risk of bias and very serious indirectness)[154,155] that addressed the PICO question. Of the 5 experimental studies identified, a C:V ratio of 30:2 was shown to be no different than 5:1,[156] 15:1,[157] 15:2,[100,156,157] 30:1,[79] or 100:5.[102] While 4 of these studies were in pigs, 1[157] was in a dog model. Two human observational studies support the idea that implementation of C:V of 30:2 improves ROSC compared to a ratio of 15:2; both studies included many additional interventions in combination with the change in C:V.[154,155]

The less critical outcomes of $PaCO_2$, oxygenation, and surrogate markers of perfusion were not fully summarized since a recommendation could be made based on the most critical 3 outcomes.

4.5.3 | Treatment recommendation

We recommend a C:V ratio of 30 chest compressions: 2 breaths (30:2) in nonintubated dogs and cats undergoing CPR (strong recommendation, very low quality of evidence).

4.5.4 | Justification of treatment recommendation

There is evidence that a C:V ratio of 30:2 is superior to both lower C:V ratios (ratios that involve fewer consecutive chest compressions for each ventilation break) and to compression-only CPR for the outcome of favorable neurologic outcome in experimental studies. Additionally, there is evidence in observational studies in people that a C:V ratio of 30:2 is superior to the lower C:V ratio of 15:2 for the outcomes of survival to discharge and ROSC. There is some evidence in experimental studies that a C:V ratio as high as 100:2 is still superior to the lower C:V ratio of 15:2. Additionally, 1 experimental study in pigs showed superiority of favorable neurologic outcome with a C:V ratio of 30:1 compared to 30:2; however, this is a VF model rather than a model of asphyxial arrest, the more common cause of CPA in dogs and cats.

Taken together, these findings suggest that at least 30 consecutive chest compressions should be administered during CPR in nonintubated dogs and cats, and that while ventilation is important, it is possible that it could be de-emphasized to a C:V ratio of 30:1 without meaningfully worse outcome, and possibly with improvement.

4.5.5 | Knowledge gaps

In nonintubated dogs and cats, the ideal C:V ratio has not been established for critical outcomes of favorable neurologic outcome, survival to discharge, or ROSC.

It is unknown whether there may be a benefit to C:V of 30:1 (rather than 30:2) in nonintubated dogs and cats undergoing CPR.

5 | DISCUSSION

In this update of BLS treatment recommendations for dogs and cats, most of the systematic reviews performed were for PICO questions initially evaluated in 2012. For the 2012 RECOVER CPR Guidelines, evidence evaluation was based on single worksheet author-driven literature searches, which had limitations. For the 2024 RECOVER CPR Guidelines, we used a standardized approach based on the GRADE system to evaluate the evidence and develop treatment recommendations, aiming to provide more standardized, reproducible, and scientifically justifiable treatment recommendations.[4] Despite the difference in methodology, the treatment recommendations for BLS in dogs and cats have not changed substantially from the 2012 RECOVER CPR Guidelines. For PICO questions that are similar to those asked by the International Liaison Committee on Resuscitation regarding optimal CPR practices in people, the RECOVER initiative drew on similar evidence to generate treatment recommendations. For PICO questions unique to dogs and cats—for instance those regarding ideal patient or hand positioning given different thoracic conformations—there remain significant knowledge gaps due to a paucity of available evidence in pertinent species. As such, the writing group relied heavily on expert opinion in both the 2012 and the current RECOVER

CPR treatment recommendations. We made treatment recommendations despite lack of evidence in many cases because of the need for clear, consistent standards for critical BLS interventions. We expect that some treatment recommendations will change as more evidence becomes available.

For this BLS update, the writing group asked 4 distinct questions regarding 2-minute chest compression cycles, planned pauses between cycles, and cycle interruption; our findings underscored the importance of uninterrupted 2-minute chest compression cycles and the value of shortening the planned pause between cycles. The only indication for interrupting a 2-minute chest compression cycle is objective substantiation of ROSC such as a persistent increase in $ETCO_2$ and/or palpation of an arterial pulse unrelated to chest compressions. Pauses between 2-minute CPR cycles are required to evaluate the ECG, but the pauses should be as brief as possible to minimize hands-off time. These recommendations are supported by evidence in people that the greater the percentage of CPR time spent in active chest compressions, the more likely victims are to survive.[158,159]

The 2012 RECOVER CPR Guidelines did not address ACD-CPR. The 2020 American Heart Association CPR guidelines state that ACD-CPR can be considered in people when providers are adequately trained and patients are appropriately monitored.[9] The evidence that the use of ACD improves survival in people is limited, and given the technical challenges of performing ACD-CPR in dogs and cats, we do not recommend its use.

We asked whether patients who experience CPA while undergoing mechanical ventilation should remain connected to the ventilator during CPR or be disconnected for manual breath delivery. Evidence regarding this issue was very limited and thus we recommended disconnection and manual ventilation in most cases due to ease of manual resuscitator use compared to the challenge of making multiple ventilator adjustments while working to initiate high-quality CPR.

We recommend that PIP not exceed 40 cm H_2O when ventilation is being delivered simultaneously with external chest compressions. This recommendation was based on very little evidence, but was selected to help avoid barotrauma that could occur from higher applied airway pressures, and because the recommendation is easy to follow using commonly available equipment[c]. In animals undergoing CPR while connected to a mechanical ventilator, we recommend that breaths be delivered in volume-controlled ventilation mode with a TV setting of 10 mL/kg; in these patients, we recommend a pressure limit of no greater than 40 cm H_2O similar to approaches in human CPR.[160–162] It should be noted that these pressure limits are high because the patients in question are undergoing closed-chest CPR with external chest compressions. Animals with lung disease that markedly compromises lung compliance may require pressure limit settings that exceed 40 cm H_2O during closed-chest CPR in order to achieve adequate TVs.

A major aim of the RECOVER initiative is to stimulate research in veterinary CPR, and although there have been several studies performed since the 2012 RECOVER CPR Guidelines were published, few addressed the knowledge gaps identified in the BLS domain. These knowledge gaps have been further detailed in this update and highlight the need for investigation of CPR techniques specific to dogs and cats.

AUTHOR CONTRIBUTIONS

Kate Hopper and Steven Epstein: Conceptualization; data curation; formal analysis; investigation; methodology; supervision; writing—original draft; writing—review and editing. **Manuel Boller**: Conceptualization; data curation; formal analysis; funding acquisition; investigation; methodology; writing—original draft; writing—review and editing. **Kim Mears**: Data curation; writing—review and editing. **Molly Crews**: Data curation; writing—review and editing.

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. 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. (Supporting Information).

CONFLICT OF INTEREST STATEMENT

Drs. Burkitt-Creedon and Epstein are editors of the Journal but only participated in review process as authors. The authors declare no other conflicts of interest.

REPRINTS

Reprints will not be available.

ORCID

Steven E. Epstein DVM, DACVECC https://orcid.org/0000-0001-6126-6695

Jamie M. Burkitt-Creedon DVM, DACVECC https://orcid.org/0000-0003-3726-0706

Daniel J. Fletcher PhD, DVM, DACVECC https://orcid.org/0000-0001-8382-5910

Molly Crews MLS https://orcid.org/0000-0002-4960-5591

ENDNOTES

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

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

[c] https://www.ambu.com/emergency-care-and-training/resuscitators/product/ambu-spur-ii (accessed on February 12, 2024).

REFERENCES

1. Berg KM, Bray JE, Ng KC, et al. 2023 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations: summary from the basic life support; advanced life support; pediatric life support; neonatal life support; education, implementation, and teams; and first aid task forces. *Circulation*. 2023;148(24):e187-e280.
2. Topjian AA, Raymond TT, Atkins D, et al. Part 4: Pediatric Basic and Advanced Life Support: 2020 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2020;142(16_suppl_2):S469-S523.
3. Hopper K, Epstein SE, Fletcher DJ, Boller M. RECOVER evidence and knowledge gap analysis on veterinary CPR. Part 3: basic life support. *J Vet Emerg Crit Care*. 2012;22(s1):S26-S43.
4. Fletcher DJ. *RECOVER Part 1 - Methods*. JVECC; 2024.
5. Burkitt-Creedon J. *RECOVER Part 5 - Guidelines*. JVECC; 2024.
6. Guyatt GH, Oxman AD, Kunz R, et al. Going from evidence to recommendations. *BMJ*. 2008;336(7652):1049-1051.
7. McGowan J, Sampson M, Salzwedel DM, et al. PRESS Peer Review of Electronic Search Strategies: 2015 Guideline Statement. *J Clin Epidemiol*. 2016;75:40-46.
8. Bobrow BJ, Panczyk M. Time to compress the time to first compression. *J Am Heart Assoc*. 2018;7(9):e009247.
9. Panchal AR, Bartos JA, Cabañas JG, et al. Part 3: Adult Basic and Advanced Life Support: 2020 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2020;142(16_suppl_2):S366-S468.
10. Hoehne SN, Balakrishnan A, Silverstein DC, et al. Reassessment Campaign on Veterinary Resuscitation (RECOVER) Initiative small animal CPR registry report 2016–2021. *J Vet Emerg Crit Care*. 2023;33(2):143-155.
11. Lubrano R, Cecchetti C, Bellelli E, et al. Comparison of times of intervention during pediatric CPR maneuvers using ABC and CAB sequences: a randomized trial. *Resuscitation*. 2012;83(12):1473-1477.
12. Marsch S, Tschan F, Semmer NK, et al. ABC versus CAB for cardiopulmonary resuscitation: a prospective, randomized simulator-based trial. *Swiss Med Wkly*. 2013;143:w13856.
13. Cipani S, Bartolozzi C, Ballo P, Sarti A. Blood flow maintenance by cardiac massage during cardiopulmonary resuscitation: classical theories, newer hypotheses, and clinical utility of mechanical devices. *J Intensive Care Soc*. 2019;20(1):2-10.
14. Rudikoff MT, Maughan WL, Effron M, Freund P, Weisfeldt ML. Mechanisms of blood flow during cardiopulmonary resuscitation. *Circulation*. 1980;61(2):345-352.
15. Jung YH, Jeung KW, Lee DH, et al. Relationship between left ventricle position and haemodynamic parameters during cardiopulmonary resuscitation in a pig model. *Heart Lung Circ*. 2018;27(12):1489-1497.
16. Halperin HR, Tsitlik JE, Guerci AD, et al. Determinants of blood flow to vital organs during cardiopulmonary resuscitation in dogs. *Circulation*. 1986;73(3):539-550.
17. Feneley MP, Maier GW, Gaynor JW, et al. Sequence of mitral valve motion and transmitral blood flow during manual cardiopulmonary resuscitation in dogs. *Circulation*. 1987;76(2):363-375.
18. Halperin HR, Weiss JL, Guerci AD, et al. Cyclic elevation of intrathoracic pressure can close the mitral valve during cardiac arrest in dogs. *Circulation*. 1988;78(3 I):754-760.
19. Fletcher DJ, Boller M, Brainard BM, et al. RECOVER evidence and knowledge gap analysis on veterinary CPR. Part 7: clinical guidelines: RECOVER clinical guidelines. *J Vet Emerg Crit Care*. 2012;22(s1):S102-S131.
20. Henik RA, Wingfield WE, Angleton GM, Porter RE. Effects of body position and ventilation/compression ratios during cardiopulmonary resuscitation in cats. *Am J Vet Res*. 1987;48(11):1603-1606.
21. Hofmeister EH, Brainard BM, Egger CM, Kang SW. Prognostic indicators for dogs and cats with cardiopulmonary arrest treated by cardiopulmonary cerebral resuscitation at a university teaching hospital. *J Am Vet Med Assoc*. 2009;235(1):50-57.
22. Maier GW, Tyson Jr GS, Olsen CO, et al. The physiology of external cardiac massage: high-impulse cardiopulmonary resuscitation. *Circulation*. 1984;70(1):86-101.
23. Smereka J, Szarpak L, Rodríguez-Núñez A, et al. A randomized comparison of three chest compression techniques and associated hemodynamic effect during infant CPR: a randomized manikin study. *Am J Emerg Med*. 2017;35(10):1420-1425.
24. Dorfsman ML, Menegazzi JJ, Wadas RJ, Auble TE. Two-thumb vs two-finger chest compression in an infant model of prolonged cardiopulmonary resuscitation. *Acad Emerg Med*. 2000;7(10):1077-1082.
25. Feneley MP, Maier GW, Kern KB, et al. Influence of compression rate on initial success of resuscitation and 24 hour survival after prolonged manual cardiopulmonary resuscitation in dogs. *Circulation*. 1988;77(1):240-250.
26. Wu JY, Li CS, Liu ZX, Wu CJ, Zhang GC. A comparison of 2 types of chest compressions in a porcine model of cardiac arrest. *Am J Emerg Med*. 2009;27(7):823-829.
27. Duval S, Pepe PE, Aufderheide TP, et al. Optimal combination of compression rate and depth during cardiopulmonary resuscitation for functionally favorable survival. *JAMA Cardiol*. 2019;4(9):900-908.
28. Kilgannon JH, Kirchhoff M, Pierce L, et al. Association between chest compression rates and clinical outcomes following in-hospital cardiac arrest at an academic tertiary hospital. *Resuscitation*. 2017;110:154-161.
29. Sutton RM, Reeder RW, Landis W, et al. Chest compression rates and pediatric in-hospital cardiac arrest survival outcomes. *Resuscitation*. 2018;130:159-166.
30. Sutton RM, Case E, Brown SP, et al. A quantitative analysis of out-of-hospital pediatric and adolescent resuscitation quality – a report from the ROC epistry-cardiac arrest. *Resuscitation*. 2015;93:150-157.
31. Idris AH, Guffey D, Aufderheide TP, et al. Relationship between chest compression rates and outcomes from cardiac arrest. *Circulation*. 2012;125(24):3004-3012.
32. Idris AH, Guffey D, Pepe PE, et al. Chest compression rates and survival following out-of-hospital cardiac arrest. *Crit Care Med*. 2015;43(4):840-848.
33. Li ES, Cheung PY, Lee TF, et al. Return of spontaneous circulation is not affected by different chest compression rates superimposed with sustained inflations during cardiopulmonary resuscitation in newborn piglets. *PLoS ONE*. 2016;11(6):e0157249.
34. McIntyre RL, Hopper K, Epstein SE. Assessment of cardiopulmonary resuscitation in 121 dogs and 30 cats at a university teaching hospital (2009-2012). *J Vet Emerg Crit Care San Antonio*. 2014;24(6):693-704.
35. Reyes-Martínez M, Herrería-Bustillo VJ. Evaluation of compressor fatigue at 150 compressions per minute during cardiopulmonary resuscitation using a large dog manikin. *J Vet Emerg Crit Care*. 2023;33(5):495-500.
36. Yannopoulos D, McKnite S, Aufderheide TP, et al. Effects of incomplete chest wall decompression during cardiopulmonary resuscitation on coronary and cerebral perfusion pressures in a porcine model of cardiac arrest. *Resuscitation*. 2005;64(3):363-372.
37. Zuercher M, Hilwig RW, Ranger-Moore J, et al. Leaning during chest compressions impairs cardiac output and left ventricular myocardial blood flow in piglet cardiac arrest. *Crit Care Med*. 2010;38(4):1141-1146.
38. Dean JM, Koehler RC, Schleien CL, et al. Age-related effects of compression rate and duration in cardiopulmonary resuscitation. *J Appl Physiol*. 1990;68(2):554-560.

39. Dean JM, Koehler RC, Schleien CL, et al. Improved blood flow during prolonged cardiopulmonary resuscitation with 30% duty cycle in infant pigs. *Circulation*. 1991;84(2):896-904.
40. Riyapan S, Naulnark T, Ruangsomboon O, et al. Improving quality of chest compression in thai emergency department by using real-time audio-visual feedback cardio-pulmonary resuscitation monitoring. *J Med Assoc Thai*. 2019;102(3):245-251.
41. Vadeboncoeur T, Stolz U, Panchal A, et al. Chest compression depth and survival in out-of-hospital cardiac arrest. *Resuscitation*. 2014;85(2):182-188.
42. Stiell IG, Brown SP, Nichol G, et al. What is the optimal chest compression depth during out-of-hospital cardiac arrest resuscitation of adult patients? *Circulation*. 2014;130(22):1962-1970.
43. Kramer-Johansen J, Myklebust H, Wik L, et al. Quality of out-of-hospital cardiopulmonary resuscitation with real time automated feedback: a prospective interventional study. *Resuscitation*. 2006;71(3):283-292.
44. Edelson DP, Abella BS, Kramer-Johansen J, et al. Effects of compression depth and pre-shock pauses predict defibrillation failure during cardiac arrest. *Resuscitation*. 2006;71(2):137-145.
45. Ristagno G, Tang W, Chang YT, et al. The quality of chest compressions during cardiopulmonary resuscitation overrides importance of timing of defibrillation. *Chest*. 2007;132(1):70-75.
46. Li H, Zhang L, Yang Z, et al. Even four minutes of poor quality of CPR compromises outcome in a porcine model of prolonged cardiac arrest. *Biomed Res Int*. 2013;2013:171862.
47. Ryu SJ, Lee SJ, Park CH, et al. Arterial pressure, end-tidal carbon dioxide, and central venous oxygen saturation in reflecting compression depth. *Acta Anaesthesiol Scand*. 2016;60(7):1012-1023.
48. Babbs CF, Voorhees WD, Fitzgerald KR, Holmes HR, Geddes LA. Relationship of blood pressure and flow during CPR to chest compression amplitude: evidence for an effective compression threshold. *Ann Emerg Med*. 1983;12(9):527-532.
49. Xu J, Li C, Zheng L, et al. Pulse oximetry: a non-invasive, novel marker for the quality of chest compressions in porcine models of cardiac arrest. *PLoS ONE*. 2015;10(10):e0139707.
50. Xu J, Li C, Li Y, et al. Influence of chest compressions on circulation during the peri-cardiac arrest period in porcine models. *PLoS ONE*. 2016;11(5):e0155212.
51. Hellevuo H, Sainio M, Nevalainen R, et al. Deeper chest compression - more complications for cardiac arrest patients? *Resuscitation*. 2013;84(6):760-765.
52. Hamrick JT, Hamrick JL, Bhalala U, et al. End-tidal CO2-guided chest compression delivery improves survival in a neonatal asphyxial cardiac arrest model. *Pediatr Crit Care Med*. 2017;18(11):e575-e584.
53. Berg RA, Sanders AB, Kern KB, et al. Adverse hemodynamic effects of interrupting chest compressions for rescue breathing during cardiopulmonary resuscitation for ventricular fibrillation cardiac arrest. *Circulation*. 2001;104(20):2465-2470.
54. Kern KB, Hilwig RW, Berg RA, Ewy GA. Efficacy of chest compression-only BLS CPR in the presence of an occluded airway. *Resuscitation*. 1998;39(3):179-188.
55. Kellum MJ, Kennedy KW, Barney R, et al. Cardiocerebral resuscitation improves neurologically intact survival of patients with out-of-hospital cardiac arrest. *Ann Emerg Med*. 2008;52(3):244-252.
56. Mosier J, Itty A, Sanders A, et al. Cardiocerebral resuscitation is associated with improved survival and neurologic outcome from out-of-hospital cardiac arrest in elders. *Acad Emerg Med*. 2010;17(3):269-275.
57. Ock SM, Kim YM, Chung JH, Kim SH. Influence of physical fitness on the performance of 5-minute continuous chest compression. *Eur J Emerg Med*. 2011;18(5):251-256.
58. Gianotto-Oliveira R, Gianotto-Oliveira G, Gonzalez MM, et al. Quality of continuous chest compressions performed for one or two minutes. *Clin Sao Paulo*. 2015;70(3):190-195.
59. Badaki-Makun O, Nadel F, Donoghue A, et al. Chest compression quality over time in pediatric resuscitations. *Pediatrics*. 2013;131(3):e797-e804.
60. McDonald CH, Heggie J, Jones CM, Thorne CJ, Hulme J. Rescuer fatigue under the 2010 ERC guidelines, and its effect on cardiopulmonary resuscitation (CPR) performance. *Emerg Med J EMJ*. 2013;30(8):623-627.
61. Odegaard S, Saether E, Steen PA, Wik L. Quality of lay person CPR performance with compression: ventilation ratios 15:2, 30:2 or continuous chest compressions without ventilations on manikins. *Resuscitation*. 2006;71(3):335-340.
62. Sugerman NT, Edelson DP, Leary M, et al. Rescuer fatigue during actual in-hospital cardiopulmonary resuscitation with audio-visual feedback: a prospective multicenter study. *Resuscitation*. 2009;80(9):981-984.
63. Brouwer TF, Walker RG, Chapman FW, Koster RW. Association between chest compression interruptions and clinical outcomes of ventricular fibrillation out-of-hospital cardiac arrest. *Circulation*. 2015;132(11):1030-1037.
64. Cheskes S, Schmicker RH, Christenson J, et al. Perishock pause: an independent predictor of survival from out-of-hospital shockable cardiac arrest. *Circulation*. 2011;124(1):58-66.
65. Sato Y, Weil MH, Sun S, et al. Adverse effects of interrupting precordial compression during cardiopulmonary resuscitation. *Crit Care Med*. 1997;25(5):733-736.
66. Sell RE, Sarno R, Lawrence B, et al. Minimizing pre- and post-defibrillation pauses increases the likelihood of return of spontaneous circulation (ROSC). *Resuscitation*. 2010;81(7):822-825.
67. Walcott GP, Melnick SB, Walker RG, et al. Effect of timing and duration of a single chest compression pause on short-term survival following prolonged ventricular fibrillation. *Resuscitation*. 2009;80(4):458-462.
68. Lui CT, Poon KM, Tsui KL. Abrupt rise of end tidal carbon dioxide level was a specific but non-sensitive marker of return of spontaneous circulation in patient with out-of-hospital cardiac arrest. *Resuscitation*. 2016;104:53-58.
69. Wyckoff MH, Greif R, Morley PT, et al. 2022 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations: summary from the basic life support; advanced life support; pediatric life support; neonatal life support; education, implementation, and teams; and first aid task forces. *Circulation*. 2022;146(25):e483-e557.
70. Rea TD, Fahrenbruch C, Culley L, et al. CPR with chest compression alone or with rescue breathing. *N Engl J Med*. 2010;363(5):423-433.
71. Berg RA, Kern KB, Sanders AB, et al. Bystander cardiopulmonary resuscitation: is ventilation necessary? *Circulation*. 1993;88(4):1907-1915.
72. Berg RA, Kern KB, Hilwig RW, Ewy GA. Assisted ventilation during "bystander" CPR in a swine acute myocardial infarction model does not improve outcome. *Circulation*. 1997;96(12):4364-4371.
73. Berg RA, Kern KB, Hilwig RW, et al. Assisted ventilation does not improve outcome in a porcine model of single-rescuer bystander cardiopulmonary resuscitation. *Circulation*. 1997;95(6):1635-1641.
74. Berg RA, Hilwig RW, Kern KB, Babar I, Ewy GA. Simulated mouth-to-mouth ventilation and chest compressions (bystander cardiopulmonary resuscitation) improves outcome in a swine model of prehospital pediatric asphyxial cardiac arrest. *Crit Care Med*. 1999;27(9):1893-1899.
75. Berg RA, Hilwig RW, Kern KB, Ewy GA. "Bystander" chest compressions and assisted ventilation independently improve outcome from piglet asphyxial pulseless "cardiac arrest". *Circulation*. 2000;101(14):1743-1748.
76. Sanders AB, Kern KB, Berg RA, et al. Survival and neurologic outcome after cardiopulmonary resuscitation with four different chest compression-ventilation ratios. *Ann Emerg Med*. 2002;40(6):553-562.

77. Ewy GA, Hilwig RW, Zuercher M, et al. Continuous chest compression resuscitation in arrested swine with upper airway inspiratory obstruction. *Resuscitation*. 2010;81(5):585-590.
78. Wang S, Li C, Ji X, et al. Effect of continuous compressions and 30:2 cardiopulmonary resuscitation on global ventilation/perfusion values during resuscitation in a porcine model. *Crit Care Med*. 2010;38(10):2024-2030.
79. Kim YW, Kim HI, Hwang SO, et al. Single ventilation during cardiopulmonary resuscitation results in better neurological outcomes in a porcine model of cardiac arrest. *Yonsei Med J*. 2018;59(10):1232-1239.
80. Fukuda T, Fukuda-Ohashi N, Doi K, Matsubara T, Yahagi N. Effective pre-hospital care for out-of-hospital cardiac arrest caused by respiratory disease. *Heart Lung Circ*. 2015;24(3):241-249.
81. Maeda T, Kamikura T, Tanaka Y, et al. Impact of bystander-performed ventilation on functional outcomes after cardiac arrest and factors associated with ventilation-only cardiopulmonary resuscitation: a large observational study. *Resuscitation*. 2015;91:122-130.
82. Takei Y, Kamikura T, Nishi T, et al. Recruitments of trained citizen volunteering for conventional cardiopulmonary resuscitation are necessary to improve the outcome after out-of-hospital cardiac arrests in remote time-distance area: a nationwide population-based study. *Resuscitation*. 2016;105:100-108.
83. Naim MY, Burke RV, McNally BF, et al. Association of bystander cardiopulmonary resuscitation with overall and neurologically favorable survival after pediatric out-of-hospital cardiac arrest in the united states: a report from the cardiac arrest registry to enhance survival surveillance registry. *JAMA Pediatr*. 2017;171(2):133-141.
84. Fukuda T, Ohashi-Fukuda N, Kobayashi H, et al. Conventional versus compression-only versus no-bystander cardiopulmonary resuscitation for pediatric out-of-hospital cardiac arrest. *Circulation*. 2016;134(25):2060-2070.
85. Kaneko H, Hara M, Mizutani K, et al. Improving outcomes of witnessed out-of-hospital cardiac arrest after implementation of International Liaison Committee on Resuscitation 2010 Consensus: a nationwide prospective observational population-based study. *J Am Heart Assoc*. 2017;6(8):e004959.
86. Goto Y, Funada A, Goto Y. Conventional versus chest-compression-only cardiopulmonary resuscitation by bystanders for children with out-of-hospital cardiac arrest. *Resuscitation*. 2018;122:126-134.
87. Zhang X, Zhang W, Wang C, et al. Chest-compression-only versus conventional cardiopulmonary resuscitation by bystanders for children with out-of-hospital cardiac arrest: a systematic review and meta-analysis. *Resuscitation*. 2019;134:81-90.
88. Fukuda T, Ohashi-Fukuda N, Hayashida K, Kondo Y, Kukita I. Bystander-initiated conventional vs compression-only cardiopulmonary resuscitation and outcomes after out-of-hospital cardiac arrest due to drowning. *Resuscitation*. 2019;145:166-174.
89. Tobin JM, Ramos WD, Greenshields J, et al. Outcome of conventional bystander cardiopulmonary resuscitation in cardiac arrest following drowning. *Prehosp Disaster Med*. 2020;35(2):141-147.
90. Olasveengen TM, Wik L, Steen PA. Standard basic life support vs. continuous chest compressions only in out-of-hospital cardiac arrest. *Acta Anaesthesiol Scand*. 2008;52(7):914-919.
91. Bobrow BJ, Spaite DW, Berg RA, et al. Chest compression-only CPR by lay rescuers and survival from out-of-hospital cardiac arrest. *JAMA*. 2010;304(13):1447-1454.
92. Iwami T, Kawamura T, Hiraide A, et al. Effectiveness of bystander-initiated cardiac-only resuscitation for patients with out-of-hospital cardiac arrest. *Circulation*. 2007;116(25):2900-2907.
93. SOS-KANTO study group. Cardiopulmonary resuscitation by bystanders with chest compression only (SOS-KANTO): an observational study. *Lancet*. 2007;369(9565):920-926.
94. Ong MEH, Ng FSP, Anushia P, et al. Comparison of chest compression only and standard cardiopulmonary resuscitation for out-of-hospital cardiac arrest in Singapore. *Resuscitation*. 2008;78(2):119-126.
95. Goto Y, Maeda T, Goto Y. Impact of dispatcher-assisted bystander cardiopulmonary resuscitation on neurological outcomes in children with out-of-hospital cardiac arrests: a prospective, nationwide, population-based cohort study. *J Am Heart Assoc*. 2014;3(3):e000499.
96. Iwami T, Kitamura T, Kiyohara K, Kawamura T. Dissemination of chest compression-only cardiopulmonary resuscitation and survival after out-of-hospital cardiac arrest. *Circulation*. 2015;132(5):415-422.
97. Svensson L, Bohm K, Castrèn M, et al. Compression-only CPR or standard CPR in out-of-hospital cardiac arrest. *N Engl J Med*. 2010;363(5):434-442.
98. Hallstrom A, Cobb L, Johnson E, Copass M. Cardiopulmonary resuscitation by chest compression alone or with mouth-to-mouth ventilation. *N Engl J Med*. 2000;342(21):1546-1553.
99. Dorph E, Wik L, Strømme TA, Eriksen M, Steen PA. Oxygen delivery and return of spontaneous circulation with ventilation: compression ratio 2:30 versus chest compressions only CPR in pigs. *Resuscitation*. 2004;60(3):309-318.
100. Cavus E, Meybohm P, Bein B, et al. Impact of different compression-ventilation ratios during basic life support cardiopulmonary resuscitation. *Resuscitation*. 2008;79(1):118-124.
101. Markstaller K, Rudolph A, Karmrodt J, et al. Effect of chest compressions only during experimental basic life support on alveolar collapse and recruitment. *Resuscitation*. 2008;79(1):125-132.
102. Kill C, Torossian A, Freisburger C, et al. Basic life support with four different compression/ventilation ratios in a pig model: the need for ventilation. *Resuscitation*. 2009;80(9):1060-1065.
103. Botran M, Lopez-Herce J, Urbano J, et al. Chest compressions versus ventilation plus chest compressions: a randomized trial in a pediatric asphyxial cardiac arrest animal model. *Intensive Care Med*. 2011;37(11):1873-1880.
104. Xanthos T, Karatzas T, Stroumpoulis K, et al. Continuous chest compressions improve survival and neurologic outcome in a swine model of prolonged ventricular fibrillation. *Am J Emerg Med*. 2012;30(8):1389-1394.
105. Hopper K, Rezende ML, Borchers A, Epstein SE. Efficacy of manual ventilation techniques during cardiopulmonary resuscitation in dogs. *Front Vet Sci*. 2018;5:239.
106. Plaisance P, Lurie KG, Vicaut E, et al. A comparison of standard cardiopulmonary resuscitation and active compression–decompression resuscitation for out-of-hospital cardiac arrest. *N Engl J Med*. 1999;341(8):569-575.
107. Schwab TM, Callaham ML, Madsen CD, Utecht TA. A randomized clinical trial of active compression-decompression CPR vs standard CPR in out-of-hospital cardiac arrest in two cities. *JAMA*. 1995;273(16):1261-1268.
108. Tucker KJ, Galli F, Savitt MA, et al. Active compression-decompression resuscitation: effect on resuscitation success after in-hospital cardiac arrest. *J Am Coll Cardiol*. 1994;24(1):201-209.
109. Stiell IG, Hébert PC, Wells GA, et al. The Ontario trial of active compression-decompression cardiopulmonary resuscitation for in-hospital and prehospital cardiac arrest. *JAMA*. 1996;275(18):1417-1423.
110. Plaisance P, Adnet F de´ ric, Vicaut E, et al. Benefit of active compression-decompression cardiopulmonary resuscitation as a prehospital advanced cardiac life support: a randomized multicenter study. *Circulation*. 1997;95(4):955-961.
111. Nolan J, Smith G, Evans R, et al. The United Kingdom pre-hospital study of active compression-decompression resuscitation. *Resuscitation*. 1998;37(2):119-125.

112. Mauer D, Schneider T, Dick W, et al. Active compression-decompression resuscitation: a prospective, randomized study in a two-tiered EMS system with physicians in the field. *Resuscitation*. 1996;33(2):125-134.
113. Lurie KG, Shultz JJ, Callaham ML, et al. Evaluation of active compression-decompression CPR in victims of out-of-hospital cardiac arrest. *JAMA*. 1994;271(18):1405-1411.
114. Skogvoll E, Wik L. Active compression-decompression cardiopulmonary resuscitation: a population-based, prospective randomised clinical trial in out-of-hospital cardiac arrest. *Resuscitation*. 1999;42(3):163-172.
115. Mauer D, Schneider T, Elich D, Dick W. Carbon dioxide levels during pre-hospital active compression–decompression versus standard cardiopulmonary resuscitation. *Resuscitation*. 1998;39(1-2):67-74.
116. Luiz T, Ellinger K, Denz C. Active compression-decompression cardiopulmonary resuscitation does not improve survival in patients with prehospital cardiac arrest in a physician-manned emergency medical system. *J Cardiothorac Vasc Anesth*. 1996;10(2):178-186.
117. Aufderheide TP, Frascone RJ, Wayne MA, et al. Standard cardiopulmonary resuscitation versus active compression-decompression cardiopulmonary resuscitation with augmentation of negative intrathoracic pressure for out-of-hospital cardiac arrest: a randomised trial. *Lancet*. 2011;377(9762):301-311.
118. Gunaydin YK, Cekmen B, Akilli NB, et al. Comparative effectiveness of standard CPR vs active compression-decompression CPR with CardioPump for treatment of cardiac arrest. *Am J Emerg Med*. 2016;34(3):542-547.
119. Callaham ML. A randomized prospective trial of active compression-decompression CPR versus manuel CPR in prehospital cardiac arrest. *Am Emerg Med*. 1993;22:174.
120. Kern KB, Figge G, Hilwig RW, et al. Active compression-decompression versus standard cardiopulmonary resuscitation in a porcine model: no improvement in outcome. *Am Heart J*. 1996;132(6):1156-1162.
121. Bahlmann L, Klaus S, Baumeier W, et al. Brain metabolism during cardiopulmonary resuscitation assessed with microdialysis. *Resuscitation*. 2003;59(2):255-260.
122. Udassi JP, Udassi S, Shih A, et al. Novel adhesive glove device (AGD) for active compression-decompression (ACD) CPR results in improved carotid blood flow and coronary perfusion pressure in piglet model of cardiac arrest. *Resuscitation*. 2012;83(6):750-754.
123. Frascone RJ, Wayne MA, Swor RA, et al. Treatment of non-traumatic out-of-hospital cardiac arrest with active compression decompression cardiopulmonary resuscitation plus an impedance threshold device. *Resuscitation*. 2013;84(9):1214-1222.
124. Sunde K, Wik L, Naess PA, et al. Effect of different compression-decompression cycles on haemodynamics during ACD-CPR in pigs. *Resuscitation*. 1998;36(2):123-131.
125. Tucker KJ, Khan JH, Savitt MA. Active compression-decompression resuscitation: effects on pulmonary ventilation. *Resuscitation*. 1993;26(2):125-131.
126. Wik L, Naess PA, Ilebekk A, Steen PA. Simultaneous active compression-decompression and abdominal binding increase carotid blood flow additively during cardiopulmonary resuscitation (CPR) in pigs. *Resuscitation*. 1994;28(1):55-64.
127. Chang MW, Coffeen P, Lurie KG, et al. Active compression-decompression CPR improves vital organ perfusion in a dog model of ventricular fibrillation. *Chest*. 1994;106(4):1250-1259.
128. Tucker KJ, Khan J, Idris A, Savitt MA. The biphasic mechanism of blood flow during cardiopulmonary resuscitation: a physiologic comparison of active compression-decompression and high-impulse manual external cardiac massage. *Ann Emerg Med*. 1994;24(5):895-906.
129. Langhelle A, Stromme T, Sunde K, et al. Inspiratory impedance threshold valve during CPR. *Resuscitation*. 2002;52(1):39-48.
130. Voelckel WG, Lurie KG, Sweeney M, et al. Effects of active compression-decompression cardiopulmonary resuscitation with the inspiratory threshold valve in a young porcine model of cardiac arrest. *Pediatr Res*. 2002;51(4):523-527.
131. Raedler C, Voelckel WG, Wenzel V, et al. Vasopressor response in a porcine model of hypothermic cardiac arrest is improved with active compression-decompression cardiopulmonary resuscitation using the inspiratory impedance threshold valve. *Anesth Analg*. 2002;95(6):1496-1502, table of contents.
132. Metzger AK, Herman M, McKnite S, Tang W, Yannopoulos D. Improved cerebral perfusion pressures and 24-hr neurological survival in a porcine model of cardiac arrest with active compression-decompression cardiopulmonary resuscitation and augmentation of negative intrathoracic pressure. *Crit Care Med*. 2012;40(6):1851-1856.
133. Shih A, Udassi S, Porvasnik SL, et al. Use of impedance threshold device in conjunction with our novel adhesive glove device for ACD-CPR does not result in additional chest decompression. *Resuscitation*. 2013;84(10):1433-1438.
134. Kwon Y, Debaty G, Puertas L, et al. Effect of regulating airway pressure on intrathoracic pressure and vital organ perfusion pressure during cardiopulmonary resuscitation: a non-randomized interventional cross-over study. *Scand J Trauma Resusc Emerg Med*. 2015;23:83.
135. Steinberg MT, Olsen JA, Eriksen M, et al. Haemodynamic outcomes during piston-based mechanical CPR with or without active decompression in a porcine model of cardiac arrest. *Scand J Trauma Resusc Emerg Med*. 2018;26(1):31.
136. Hayes MM, Ewy GA, Anavy ND, et al. Continuous passive oxygen insufflation results in a similar outcome to positive pressure ventilation in a swine model of out-of-hospital ventricular fibrillation. *Resuscitation*. 2007;74(2):357-365.
137. Tan D, Sun J, Geng P, et al. Duration of cardiac arrest requires different ventilation volumes during cardiopulmonary resuscitation in a pig model. *J Clin Monit Comput*. 2020;34(3):525-533.
138. Ruemmler R, Ziebart A, Moellmann C, et al. Ultra-low tidal volume ventilation-A novel and effective ventilation strategy during experimental cardiopulmonary resuscitation. *Resuscitation*. 2018;132:56-62.
139. Corp A, Thomas C, Adlam M. The cardiovascular effects of positive pressure ventilation. *BJA Educ*. 2021;21(6):202-209.
140. Raichle ME, Plum F. Hyperventilation and Cerebral Blood Flow. *Stroke*. 1972;3(5):566-575.
141. Vissers G, Duchatelet C, Huybrechts SA, et al. The effect of ventilation rate on outcome in adults receiving cardiopulmonary resuscitation. *Resuscitation*. 2019;138:243-249.
142. Sanson G, Ristagno G, Caggegi GD, et al. Impact of "synchronous" and "asynchronous" CPR modality on quality bundles and outcome in out-of-hospital cardiac arrest patients. *Intern Emerg Med*. 2019;14(7):1129-1137.
143. Sutton RM, Reeder RW, Landis WP, et al. Ventilation rates and pediatric in-hospital cardiac arrest survival outcomes. *Crit Care Med*. 2019;47(11):1627-1636.
144. Gazmuri RJ, Ayoub IM, Radhakrishnan J, Motl J, Upadhyaya MP. Clinically plausible hyperventilation does not exert adverse hemodynamic effects during CPR but markedly reduces end-tidal PCO(2). *Resuscitation*. 2012;83(2):259-264.
145. López J, Fernández SN, González R, et al. Different respiratory rates during resuscitation in a pediatric animal model of asphyxial cardiac arrest. *PLoS ONE*. 2016;11(9):e0162185.
146. Aufderheide TP, Lurie KG. Death by hyperventilation: a common and life-threatening problem during cardiopulmonary resuscitation. *Crit Care Med*. 2004;32(9 Suppl):S345-S351.
147. Xu J, Yu X, Zhang L, et al. Modified volumetric capnography-derived parameter: a potentially stable indicator in monitoring

cardiopulmonary resuscitation efficacy in a porcine model. *Resuscitation*. 2020;150:94-101.
148. Luce JM, Ross BK, O'Quin RJ, et al. Regional blood flow during cardiopulmonary resuscitation in dogs using simultaneous and nonsimultaneous compression and ventilation. *Circulation*. 1983;67(2):258-265.
149. Aufderheide TP, Sigurdsson G, Pirrallo RG, et al. Hyperventilation-induced hypotension during cardiopulmonary resuscitation. *Circulation*. 2004;109(16):1960-1965.
150. Lurie KG, Yannopoulos D, McKnite SH, et al. Comparison of a 10-breaths-per-minute versus a 2-breaths-per-minute strategy during cardiopulmonary resuscitation in a porcine model of cardiac arrest. *Respir Care*. 2008;53(7):862-870.
151. Mendler MR, Weber C, Hassan MA, et al. Effect of different respiratory modes on return of spontaneous circulation in a newborn piglet model of hypoxic cardiac arrest. *Neonatology*. 2016;109(1):22-30.
152. Mendler MR, Maurer M, Hassan MA, et al. Different techniques of respiratory support do not significantly affect gas exchange during cardiopulmonary resuscitation in a newborn piglet model. *Neonatology*. 2015;108(1):73-80.
153. Tan D, Xu J, Shao S, et al. Comparison of different inspiratory triggering settings in automated ventilators during cardiopulmonary resuscitation in a porcine model. *PLoS ONE*. 2017;12(2):e0171869.
154. Hinchey PR, Myers JB, Lewis R, et al. Improved out-of-hospital cardiac arrest survival after the sequential implementation of 2005 AHA guidelines for compressions, ventilations, and induced hypothermia: the Wake County experience. *Ann Emerg Med*. 2010;56(4):348-357.
155. Sayre MR, Cantrell SA, White LJ, et al. Impact of the 2005 American Heart Association cardiopulmonary resuscitation and emergency cardiovascular care guidelines on out-of-hospital cardiac arrest survival. *Prehosp Emerg Care*. 2009;13(4):469-477.
156. Kern KB, Hilwig RW, Berg RA, Schock RB, Ewy GA. Optimizing ventilation in conjunction with phased chest and abdominal compression-decompression (Lifestick) resuscitation. *Resuscitation*. 2002;52(1)91-100.
157. Hwang SO, Kim SH, Kim H, et al. Comparison of 15:1, 15:2, and 30:2 compression-to-ventilation ratios for cardiopulmonary resuscitation in a canine model of a simulated, witnessed cardiac arrest. *Acad Emerg Med*. 2008;15(2):183-189.
158. Christenson J, Andrusiek D, Everson-Stewart S, et al. Chest compression fraction determines survival in patients with out-of-hospital ventricular fibrillation. *Circulation*. 2009;120(13):1241-1247.
159. Vaillancourt C, Petersen A, Meier EN, et al. The impact of increased chest compression fraction on survival for out-of-hospital cardiac arrest patients with a non-shockable initial rhythm. *Resuscitation*. 2020;154:93-100.
160. Gabrielli A, Layon AJ, Idris AH. Physiology of ventilation during cardiac arrest. In: Ornato JP, Peberdy MA, eds. *Cardiopulmonary Resuscitation*. Humana Press; 2005:39-94.
161. Jamshed N, Sahu AK, Timilsina G, Mathew R. "Six-dial strategy" - mechanical ventilation during cardiopulmonary resuscitation. *Indian J Crit Care Med*. 2020;24(6):487-489.
162. Tangpaisarn T, Tosibphanom J, Sata R, et al. The effects of mechanical versus bag-valve ventilation on gas exchange during cardiopulmonary resuscitation in emergency department patients: a randomized controlled trial (CPR-VENT). *Resuscitation*. 2023;193:109966.

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: Hopper K, Epstein SE, Burkitt-Creedon JM, et al. 2024 RECOVER Guidelines: Basic Life Support. Evidence and knowledge gap analysis with treatment recommendations for small animal CPR. *J Vet Emerg Crit Care*. 2024;34(Suppl 1):16–43. https://doi.org/10.1111/vec.13387