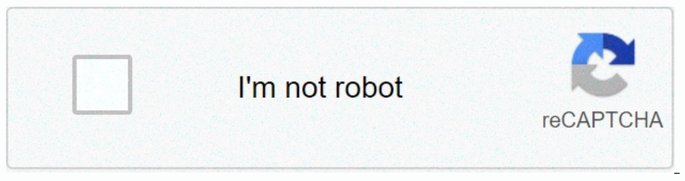

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Aha guidelines 2018 cpr

1Top 10 Take-home Messages For Adult Cardiovascular Life Support On recognition of a cardiac arrest event, layperson simultaneously and immediately activates the emergency response system and initiates cardiopulmonary resuscitation (CPR). High-quality CPR performance includes adequate compression depth and rates while minimizing the pause in compression. Early defibrillation with simultaneous high-quality CPR is critical to survival when sudden cardiac arrest is caused by ventricle fibrillation or ventricle-free tachycardia. The administration of epinephrine with high-quality CPR simultaneously increases survival, especially in patients with inevitable rhythms. Recognition that all cardiac arrest events are not the same is critical for optimal patient outcomes, and specialized management is necessary for many conditions (for example, electrolylite abnormalities, pregnancy, after heart surgery). The opioid epidemic has led to an increase in out-of-hospital cardiac arrest associated with opioids, with permanent care mainstays activating high-quality CPR response systems and high-quality CPR performance. Post-heart capture care is a critical component of the Survival Chain and demands a comprehensive, organized, multi-disciplinary system that requires consistent execution for optimal patient outcomes. The immediate start of targeted temperature management is necessary for all patients who do not comply with the instructions after the return of spontaneous circulation to ensure optimal functional and neurological outcomes. Accurate neurological prognostics in brain-injured cardiac arrest victims are essential to ensure that patients with significant potential for rehabilitation are not destined for certain poor outcomes due to care production. Expectations of rehabilitation and survival plans addressing treatment, surveillance, and rehabilitation should be given to victims of cardiac arrest and their carers at hospital clearances to optimize the transition of care to home and outpatient environments. 2Preamble In 2015, about 350 000 adults in the United States suffered out-of-hospital cardiac arrest (OHCA) attended by emergency medical services staff (EMS).1 About 10.4% of patients with OHCA survived from their initial hospitalization, and 8.2% survived with good functional status. The main driver of successful resuscitation of the OHCA is the saviour of cardiopulmonary resuscitation (CPR) and public use of automatic external defibrillator (AED). Despite recent gains, only 39.2% of adults received CPR initiated by layperson, and the public using AED in just 11.9% of survival rates from OHCA differ dramatically between us territory and the AGENCY EMS.2.3 After significant improvements, survival from OHCA was coated 2012. About 1.2% of U.S. hospitalized adults suffered cardiac arrest in hospital (IHCA).1 Of these patients, 25.8% were discharged from live hospitals, and 82% of the have a good functional status at the time of release. Despite a steady increase in survival rates from the IHCA, many opportunities remain. The Formula of the International Relations Committee on Resuscitation (ILCOR) for Survival emphasizes 3 important components for good resuscitation outcomes: guidelines based on sound resuscitation science, effective education of public providers and resuscitation, and the implementation of the Survival Chain.4 These Guidelines contain recommendations for basic life support (BLS) and advanced life support (ALS) for adult patients and based on the best resuscitation sciences. The Survival Chain, introduced in the Main Concept, is now expanded to emphasize the vital components of survivorship during rehabilitation from cardiac arrest, requiring coordinated efforts from medical professionals in various disciplines and, in the case of OHCA, from layer rescues, emergency dispatchers, and first responders. In addition, specific recommendations on resuscitation provider training are provided in Part 6: Educational Science Resuscitation, and recommendations on the care system are provided in Part 7: Care System. 3Introduction 3.1 Scope The Guidelines are designed primarily for North American healthcare providers who are looking for the latest summary for BLS and ALS for adults as well as for those seeking more in-depth information on resuscitation science and gaps in current knowledge. BLS teenage care follows adult guidelines. This Part of the American Heart Association (AHA) Guidelines 2020 for CPR and Emergency Cardiovascular Care include recommendations for clinical care of adults with cardiac arrest, including those with life-threatening conditions where cardiac arrest will occur, and after successful resuscitation from cardiac arrest. Some suggestions are directly

related to egg-laying rescues that may or may not receive CPR training and who have little or no access to resuscitation equipment. Other recommendations are relevant to people with more advanced resuscitation training, working either with or without access to drugs and resuscitation devices, working either inside or outside the hospital. Some treatment recommendations involve medical care and decide after the return of spontaneous circulation (ROSC) or when the resuscitation is unsuccessful. Importantly, recommendations are given in connection with team debris and systematic feedback to enhance future resuscitation success. 3.2 Compilation of Cardiovascular Life Support Writing Group Adult Life Writing Group includes a diverse group of experts with backgrounds in emergency medicine, critical care, cardiology, toxicology, neurology, EMS, education, research, and public health, along with content experts, staff and senior science editor of AHA. Each proposal has been formally developed and approved by the writing group. AHA has conflict of interest policies and procedures to minimize the risk of improper bias or influence during the development of guidelines. Prior to the appointment, the group's writing revealed all other commercial and potential relationships (including intellectuals) of the conflict. This procedure is fully described in Part 2: Assessment of Evidence and Development of Guidelines. Disclosure information for writing a group member is listed in Appendix 1 (a link opens in a new window). 3.3 Methodology and Evidence This Guidelines are based on extensive assessment of evidence conducted in conjunction with ILCOR and the ILCOR affiliate council. Three different types of evidence reviews (systematic reviews, scoping reviews, and evidence updates) were used in the 2020 process. Each of them resulted in a description of the literature that facilitated the development of guidelines. A more comprehensive description of this method is provided in Part 2: Assessment of Evidence and Development of Guidelines. 3.4Class of Recommendations and Level of Evidence As with all AHA guidelines, each proposal 2020 is given a Proposal Class (COR) based on the strength and consistency of evidence, alternative treatment options, and impacts on patients and communities (Table 1 (link opened in a new window)). The level of Evidence (LOE) is based on the quality, quantity, relevance, and consistency of the evidence available. For each proposal, the writing group discusses and approves the words specific recommendations and assignments of COR and LOE. In determining COR, the writing group considers LOE and other factors, including system issues, economic factors, and ethical factors such as equity, acceptance, and feasibility. This evidence review method, including specific criteria used to determine COR and LOE, is described more fully in Section 2: Assessment of Evidence and Guidelines Development. Members of the Basic And Advanced Adult Life Support Writing Group have the ultimate power over and approve these recommendations officially. Unfortunately, despite improvements in the design and funding support for resuscitation research, the overall certainty of the evidence base for resuscitation science is low. Of the 250 recommendations in these guidelines, only 2 recommendations are supported by Evidence Level A (high quality evidence from more than 1 randomized controlled trial [RCT], or 1 or more RCT supported by high-quality registration studies.) Thirty-seven recommendations are supported by Proof of B-Random Levels (moderate evidence from 1 or more RCTs) and 57 by Level B-Nonrandomized evidence. The majority of recommendations are based on Level C evidence, including those based on limited data (123 recommendations) and expert opinions (31 recommendations). Therefore, the strength of the proposal is weaker than optimal: 78 Class 1 proposals (strong), 57 Class 2a (moderate) recommendations, and 89 Class 2b (weak) recommendations are in these guidelines. In addition, 15 recommendations are set In Class 3: No Benefits, and 11 recommendations are Class 3: Harm. Clinical trials in resuscitation are indispensable. 3.5 StructureGuideline Guidelines 2020 is organized into knowledge lumps, grouped into information discrimination modules on specific topics or management issues.5 Each modular knowledge lump includes a recommendation schedule using AHA tattoos standard COR and LOE. A brief introduction or short synoscope is provided to submit recommendations into context with important background information and comprehensive management or treatment concepts. Specific text of the proposal explains the rationale and major studies data supporting the recommendations. When appropriate, additional flow diagrams or tables are included. Hyperlink references are provided to facilitate quick access and review. 3.6 Review and Approval of Documents Each document guideline 2020 has been submitted for review of blind peers to 5 subject specialists nominated by the AHA. Prior to the appointment, all peer reviewers were required to disclose relations with the industry and any other conflicts of interest, and all disclosures were reviewed by AHA staff. Peer reviewer feedback is available for guidelines in draft format and again in the final format. All guidelines have been reviewed and approved for publication by the AHA Science Advisory and Coordination Committee and the AHA Executive Committee. Exposure information for peer reviewers is listed in Appendix 2 (link opens in a new window). 3.7Abbreviations Abbreviations ACD active compression-decompression ACLS advanced cardiovascular life support ADC clearly automated AED defiant aHA American Heart Association ALS advanced aOR life support coordinated AV ratio of atrioventricular BLS support basic life class COSTR International Consensus Emergency Cardiovascular Care Science and Science With Proposed CPR Cardiopulmonary Resuscitation Treatment CT calculated DWI weighted disposal of ECG electrocardiogram ECPR extraordinary cardiopulmonary resuscitation EEG electronphalogram EMS emergency services dioxide ETI endotracheal intubation GWR gray-white ratio of ICU intensive care unit IHCA in cardiac hospital captures ILCOR International Relations Committee on IO intraosseive RESuscitation ITD impedance device IV intravenous last intravenous system of toxicity system KEYE Level Evidence MAP means pressure neuron-specific enolase OHCA out-of-heart hospital captures Paco2 partial artery carbon dioxide PCI percutaneous coronary intervention PE pulmonary embolism PMCD perimortem cesarean delivery pVT pulseless ventricular tachycardia RCTD perimortem tachycardia RCT return spontaneous circular S100B S100 calcium binding protein SGA SGA SSEP somatosensory waterway evokes STEMI ST-segment height myocardium infaccant SVT supraventricular tachycardia TCA tricyclic antidepressant TOR TTM resuscitation termination at Target temperature management VF ventricular fibrular fibrilation VT ventricular tachycardia 4Major Concepts 4.1Overview Adult Concept Cardiac Arrest Survival and rehabilitation from adult cardiac arrest relies on complex systems working together to get the best results for victims. The main focus in adult cardiac arrest events includes rapid recognition, immediate allocation of CPR, a surprisingly malignant rhythm defibrillation, and post-ROSC supporting care and the treatment of basic causes. This approach acknowledges that the most sudden cardiac arrest in adults is the cause of the heart, especially myocardial infaction and electrical disorders. Major heartless arrests (for example, from respiratory failure, toxic ingestion, pulmonary embolism [PE], or drowning) are also common, however, and in such cases, treatment for reversible fundamental causes is essential for rescuers to consider.1 Some noncardiac etiology may be especially common in hospital. Others, such as opioid doses, increased sharply over the rise in environments outside the hospital.2 For any cardiac arrest, rescuers were instructed to ask for help, perform CPR to restore coronary blood flow and submissions, and apply for AED to directly treat ventricle fibrillation (VF) or ventricle tachycardia (VT), and apply AED to directly treat ventricle fibrillation (VF) or ventricle tachycardia (VT) , and apply for AED to directly treat ventricle fibrilation (VF) or ventricle tachycardia (VF) or ventricle tachycardia (VT), and apply AED to directly treat ventricle fibrilation (VF) or ventricle tachycardia (VF) or ventricle tachycardia (VF)), and apply for AED to directly treat ventricle fibrillation (VF) or ventricle tachycardia (VF) or ventricle tachycardia (VT), and apply the AED to directly treat ventricle fibrillation (VF) or tachy Even if the majority of resuscitation success is achieved with the provision of high quality CPR and defrillation , other specific treatments for possible basic causes may help in some cases. 4.2 Survival Chain Survival Chain The main focus of cardiac arrest management for suppliers is optimisation of all critical measures needed to improve yields. These include activation of emergency response, high-quality CPR preparation and early defibrilation, ALS interventions, effective post-ROSC care including careful pronostication, and support during rehabilitation and survival. All these activities require organizational infrastructure to support education, training, equipment, supplies, and communications that allow every survival. Therefore, we that each aspect of this diverse care contributes to the ultimate survival of cardiac arrest victims. The resuscitation of the causes, processes, and outcomes are very different for OHCA and IHCA, shown in their respective Survival Chains (Fig. 1). At OHCA, victim care depends on community engagement and response. It is important for community members to recognize cardiac arrest, phone 9-1-1 (or local emergency response number), perform CPR (including, for unsourced sailors, compression CPR only), and use AED.3,4 Emergency medical personnel then called to the scene, proceed to resuscitation, and transport patients for stabilization and ultimate management. In In surveillance and prevention are critical aspects of IHCA. When the arrests occurred in hospitals, a strong multidisciplinary approach included a team of medical professionals who responded, provided CPR, immediately defibrillate, embarked on an ALS move, and pursued post-ROSC care. The result of the IHCA as a whole is higher than the OHCA,5 may be due to reduced delays in starting effective resuscitation. OHCA Adults and IHCA Chains of Survival have been updated to better highlight the evolution of the care system and the critical role of recovery and survival with the addition of new links. This Recovery Link highlights the journey of great rehabilitation and survival, from the end of acute treatment for critical illnesses through multimodal rehabilitation (both short-term and long-term), for both victims and families after cardiac arrest. The new link acknowledges the need for a care system to support recovery, discuss expectations, and provides plans that address treatment, surveillance, and rehabilitation for victims of their heart arrests and carers as they shift care from hospital to home and return to social roles and functions. 5Sequence Resuscitation 5.1Recognition of Cardiac Arrest Recommendations for Recognition of Cardiac Arrest COR LOE Recommendations 1 C-LD 1. If the victim is unconscious/unaware, with absent or abnormal breathing (i.e., only gasping), laying saviors should assume the victim is in cardiac arrest. 1 C-LD 2. If the victim is unconscious/unscrupulous, with absent or abnormal breathing (that is, only gasping), the healthcare provider should check the pulse no more than 10 s and, if no pulse is certainly felt, should assume the victim is in heart catch. Synopsis Lay rescuers CPR increased survival from cardiac arrest by 2- to 3-folding.1 Benefits of providing CPR to patients in cardiac arrest beyond any potential risk of giving chest compression to an unconscious but not in cardiac arrest. It has been shown that the risk of injury from CPR is low in these patients.2 Has been indicated earlier that all rescuers may have difficulty detecting the pulse, leading to delays in CPR, or in some cases CPR was not done at all for patients in cardiac arrest.3 Recognition of cardiac arrest by egg-laying rescuers, therefore, determined based on consciousness levels and breathing efforts Recognition of cardiac arrest by healthcare providers includes pulse checks, but the non-prolonged importance of efforts to detect the pulse is stressed. Specialized SupportIng Text Agonal breathing is characterized by slow gasping breathing irregularly ineffective for ventilation. Agonal breathing is described by rescuers laying eggs with various terms including, abnormal breathing, snoring breathing, and gasping breathing.4 Agonals are common, reported to be present in up to 40% to 60% of OHCA.5 Presence of past breathing is cited as reason to put rescuers to be diagnosed patients for not being in cardiac arrest.6 In unresponsive patients, with absent or abnormal breathing, disparaging rescuers should assume the patient is in cardiac arrest, calls for help, and immediately starts CPR. 2 these criteria (patient responsiveness and respiratory evaluation) have been shown to identify the vast majority of patients who are in cardiac arrest, allowing the immediate start of the CPR rescue lay. Further, the onset of chest compression in unconscious patients but not in cardiac arrest is associated with a significant low rate of adverse events.2 Adverse events mentioned include pain in the compression area of the chest (8.7%), fractures (ribs and shellfish) (1.7%)%), and rhabdomyolysis (0.3%), without a visceral injury described.2 Protracted delays in CPR can occur when checking the pulse at the beginning of the resuscitation effort as well as between successive cycles of CPR. Healthcare providers often take too long to check the pulse of 7,8 and have difficulty determining if the pulse is present or absent.7-9 No evidence, however, that examining breathing, coughing, or movement higher than pulse checks to detect circulation.10 Therefore, healthcare providers are instructed to check the pulse quickly and immediately begin the compact ,11 This topic last time received an official evidence review in 2010.3 5.2 Resuscitation Proposal to Restart Resuscitation: Lay Rescuer (Unsupervised or Trained) COR LOE Proposed 1 B-NR 1. All laying rescues should, at least, provide chest compression for victims of cardiac arrest. 1 C-LD 2. After identifying cardiac arrest, lonely responders should activate the emergency response system first and immediately begin CPR. 1 C-LD 3. We recommend that laypersons start CPR for considered cardiac arrest, since the risk of harm to patients is low if the patient is not in cardiac arrest. 2a C-LD 4. For layered rescues trained in CPR using chest compression and ventilation (rescue breath), it is reasonable to provide ventilation (rescue breath) in addition to chest compression for adults in the OHCA Synopsis After cardiac arrest is recognized, the Survival Chain continues with the activation of the emergency response system and the beginning of CPR. The immediate start of CPR is probably the most important intervention to improve survival and nerve outcomes. Ideally, the activation of the emergency response system and the onset of CPR occur simultaneously. In the current era of widespread use and accessibility of mobile devices, lonely responders can activate the emergency response system simultaneously by starting CPR with for help, put the phone on speaker mode to resume communication, and immediately start CPR. In rare circumstances when lonely rescuers must leave victims to dial EMS, priority should immediately activate EMS EMS immediately return to the victim to start CPR. Existing evidence suggests that the potential harm from CPR in patients who have been wrongly identified for having cardiac arrest is low.1 Overall, the initial benefit of CPR in cardiac arrest exceeds the risk of low injury for patients who are not caught heart. The initial phase of resuscitation once cardiac arrest is recognized is the same between conflicting respondents and healthcare providers, with early CPR representing priorities. Layer rescue can provide chest compression– only CPR to simplify the process and promote the onset of CPR, while healthcare providers can provide chest compression and ventilation (Fig. 2-4). CPR Supporting Text Specifics are the single most important intervention for patients in cardiac arrest, and chest compression should be provided immediately. Chest compression is the most critical component of CPR, and the chest compression approach is only suitable if the rescue lay eggs are not trained or do not want to provide breathing. Starting a CPR sequence with compression.2-4 Nationwide chest compression spread-only CPR for lay rescuers is associated with an increasing incidence of survival with encouraging neurological outcomes after OHCAs in Japan, the possibility of an increase in laying rescues providing CPR.5 Chest compression should be provided as soon as possible, without having to withdraw the victim's clothing. The optimal time of CPR startup and activation of the emergency response system were assessed by a systematic review of ILCOR in 2020.1 Observational studies of more than 17 000 OHCA events reporting similar results from either the first call strategy or the first CPR strategy. preferably both calls to activate EMS and CPR startups can occur simultaneously. Four observational studies of7-10s reported results from patients who were not in cardiac arrest and received CPR by laying rescuers. No serious harm from CPR was found in patients when they were later determined not to be in cardiac arrest.1 This is in contrast to the great risk of holding CPR when the patient is in cardiac arrest, making a risk:a strong benefit ratio in favor of providing CPR for considered cardiac arrest. In some observational studies, better results were reminded in cardiac arrest victims who received conventional CPR (compression and ventilation) compared to those receiving chest compressions only.5,11,12 Study others have reported no difference in outcomes for patients receiving conventional compared to compression only CPR.11,13–21 Given the potential for conventional benefits if the fighting rescue is trained accordingly, they should be encouraged to deliver ventilation simultaneously with A comprehensive review of data on compression ratio to ventilation when performing conventional CPR is discussed in Ventilation and and Ratio. The proposal is supported by the ILCOR Consensus 2020 on CPR and Emergency Cardiovascular Care Sciences With Treatment Proposal (CoSTR).1 Proposal to Restart: Healthcare Provider COR LOE Proposed 1 C-LD 1. Lonely healthcare providers should start with chest compression rather than with ventilation. 2a C-LD 2. It is reasonable for healthcare providers to perform chest compression and ventilation for all adult patients in cardiac arrest from either the cause of the heart or noncardiac. Specific Recommendations of the 2010 Guidelines Support Text for CPR and Emergency Cardiovascular Care include major changes for trained rescuers, which are instructed to begin CPR sequences with chest compression rather than by breath (circulation, airways, and breathing versus airways, breathing, and circulation) to minimize time to start this Approach is supported by new literature, summarized in a systematic study of ILCOR 2020 (Table 2).1-4 In recommended massages, once the chest compression has been started, a single trained rescue delivers a breath save through the mouth Manicin studies show that starting with chest compression rather than with ventilation is associated with faster time for chest compression,3,23 breath rescue,4 and the completion of the first CPR cycle.4 Healthcare providers are trained to deliver both compression and ventilation. Breast compression transmission without the help of ventilation for prolonged periods can be less effective than conventional CPR (compression plus ventilation) as arterial oxygen content decreases as the CPR period increases. These concerns are particularly important in setting asphyxic cardiac arrest.11 Healthcare providers, with their training and understanding, can realistically adjust the subsequent sequence of rescue actions to the most likely cause of arrest. This proposal is supported by CoSTR 2020 for BLS.1 Table 2. BLS Adult Massage22 Steps Lay Rescuer Unsupervised Lay Rescuer Trained Health Care Provider 1 Ensures the safety of the scene. Ensuring the safety of the scene. Ensuring the safety of the scene. 2 Check the answers. Check the answers. Check the answers. 3 Breed for nearby assistance. Phone or ask someone to call 9-1-1 (phone or caller with a permanent phone next to the victim, with a phone on speaker mode). Scream for nearby help and activate the emergency response system (9-1-1, emergency response). If someone responds, make sure the phone is on the side of the victim if possible. Shouting for nearby help / activating resuscitation teams; suppliers can activate the resuscitation team at this time or after examining breathing and pulse. 4 Follow the instructions Check no breathing or just gasping; if nothing, start CPR with compression. Check that there is no breathing or just gasping and check the pulse (ideally simultaneously). simultaneously). and retrieval of AED/emergency equipment by a lonely healthcare provider or by a second person sent by a rescue must occur not later than after the inspection for ordinary breathing and no pulse identifies cardiac arrest. 5 Find no breathing or just gasping, on the direction of telecommunications. Answer the sender's questions, and follow the telecommunication instructions. Immediately start CPR, and use AED/defibrillator when available 6 Follow the telecommunication instructions. Send a second person to get an AED, if someone is available. When the second rescue arrived, prepare CPR 2 rescuers and use AED/defibrillator. AED shows automatic external defibrillator; BLS, basic life support; and CPR, cardiopulmonary resuscitation. *Telecommunications and senders are terms that are often used compassionately. 5.3 Opening of Airways Introduction Patent Airways is essential to facilitate proper ventilation and oxygenation. While there is no high-quality evidence favoring one technique for the establishment and maintenance of patient airways, rescuers should be aware of the advantages and disadvantages and maintain efficiency in the skills necessary for each technique. Rescuers should acknowledge that various approaches may be required to establish sufficient airways. Patients should be monitored constantly to verify the patency of airways and adequate ventilation and oxygenation. There are no studies that compare different strategies to opening airways in heart arrest patients. Most of the evidence examining the effectiveness of airway strategies came from radiographic and cadaver studies. Proposal to Open Loe Cor Airway 1 C-EO 1 Proposal. Healthcare providers should use a head-chin-tilted lift maneuver to open the patient's airways when no cervical spine injury is suspected. 1 C-EO 2. Trained laying rescuers who felt confident to do both compression and ventilation should open the airways using a head tilted lift maneuver when no cervical spine injuries were suspected. 2b C-EO 3. The use of adhesives of airways (for example, oropharyngeal and/or nasopharyngeal airways) may be reasonable in unconscious (irresponsible) patients without coughing or gag reflexes to facilitate ventilation transmission with bag mask devices. 2a C-EO 4. In the presence of known or suspected basal skull fractures or severe coagulopathy, oral airways are prioritised compared to nasopharyngeal airways. 3: No C-LD Benefit 5. The use of cricoid stress routines in adult cardiac arrest is not recommended Recommendations Of Supporting Text and 2. The head tilt-chin lift has been shown to be effective in creating airways in noncardiac capture and radiological studies.2-5 No studies have compared head gland lifts maneuvering other airways to set up airways during cardiac arrest. Although there is no evidence checking the effectiveness of its use during cardiac arrest, oropharyngeal and nasopharyngeal nasopharyngeal can be used to maintain patent airways and facilitate appropriate ventilation by preventing the tongue from inclusion of airways. However, incorrect settlements can cause airway barriers by dispensing the tongue backwards of oropharynx.6.7 Oropharyngeal benefits compared to nasopharyngeal airways in the presence of known or suspected or severe skull fractures however, oral airways are prioritised due to the risk of trauma with nasopharyngeal airways. Multiple case reports have observed the intracranial placement of nasopharyngeal airways in patients with basilar skull fractures.8.9 There is no evidence that cricoid pressure facilitates ventilation or reduces the risk of aspiration in cardiac arrest patients. There is some evidence that in non-cardiac arrest patients, cryoid stress can protect against aspiration and gastric insufflation during bag-mask ventilation.10-13 However, cricoid pressure can also prevent ventilation and placement of supraglotic airways (SGA) or intubation,14-20 and increase the risk of airway trauma during intubation.21 This topic last time received a formal evidence review in 2010.22 Specific-Specific Text Healthcare support providers should consider if the spine injury is suspected or cannot be ruled out suppliers should open airways using jaw thrust instead of the disadvantages of head-chin lift.2 Maintaining patent airways and providing adequate ventilation and oxygenation are current priority CPR. If the thrust of the jaw and/or the insertion of the airways is ineffective in opening airways and allows ventilation to occur, the tilted lift of the head may be the only way to open the airways. In this case, this maneuver should be used even in cases of potential spinal injury as the need to open airways outweighs the risk of further spinal damage in cardiac arrest patients. When a spinal injury is suspected or cannot be ruled out, rescuers should maintain manual spinal movement restrictions and not use imobilization devices. Manual stabilization can reduce the movement of the cervical spine during patient care while allowing proper ventilation and airway control.23.24 Spinal immunization devices may make it harder to maintain airway patents25.26 and provide adequate ventilation. This topic was last received an official evidence study in 2010.22 5.4Metrics for high-quality CPR High-Quality Introduction CPR was, along with defibrillation for those with shocked rhythms, the most important life-saving intervention for patients in cardiac arrest. Evidence for what became CPR continue to grow as research emerges. Several key components have been defined for high-quality CPR, including minimizing disruption in chest compression, providing adequate rate compression and depth, avoiding leaning on the chest between compression, and avoiding avoiding avoidance Aeration.1 However, controlled studies were somewhat less, and observational evidence was sometimes contradicted. The effects of individual CPR metrics or interventions are difficult to assess because so many occur simultaneously and can interact with each other in their effects. Compression rates and compression depths, for example, are both associated with better outcomes, however these variables are found to be inversely related to each other so that improving one can worsen other CPR quality interventions often used in packages, making the benefits of any one particular step difficult to ascertain. As more and more EMS centers and systems use feedback devices and collect data on CPR measures such as compression depth and chest compression breakdown, this data will allow continuous updates to these recommendations. Recommendations for Positioning and Location for CPR COR LOE Proposed 1 C-LD 1. When preparing chest compression, rescuers should put the heels of one hand in the middle (center) of the victim's chest (lower half of the sternum) and the other hand heels on the first so that the hands overlap. 1 C-EO 2. Resuscitation generally needs to be done where victims are found, as long as high-quality CPR can be administered safely and effectively at such locations. 2a C-LD 3. It is best to perform CPR on a solid surface and with the victim at the supine position, when feasible. 2b C-LD 4. When the victim cannot be placed in a supine position, it may be reasonable for the rescuer to provide CPR with the victim in a vulnerable position, especially in patients hospitalized with advanced airways. Recommendations Text Support A 2020 systematic study ILCOR identified 3 studies involving 57 total patients investigating the effects of hand positioning on the resuscitation process and results.4 Although no difference in resuscitation outcomes was observed, 2 study found better physiological parameters (peak artery pressure, meaning arterial pressure [MAP], tidal carbon end dioxide [ETCO2]) when compression was performed over the bottom third of the sternum compared to mid-sternum.5 ,6 Third study found no difference.7 Holyrographic studies showed the left ventrika is usually located lower than the internipple line, corresponding to the lower half of the sternum.8 however, Lower hand placement than the internipple line can cause compression over xiphoid.9 Although data from conflicting beaded studies, it does not seem important whether dominant or non-dominant hands are placed in contact with sternum.10 ,11 Key considerations when determining whether the victim should be moved before starting the resuscitation is feasibility and safety location and position where the victim was found. This is a separate question from the decision if or when to patient to with continuous resuscitation. The effectiveness of CPR appears to be maximized with victims in supine positions and rescuers melting beside the victim's chest (for example, outside the hospital) or standing beside a bed (for example, in hospitals).12 It is thought that the best optimal chest compression is delivered with the victim on a sturdy surface.13.14 Manikin studies show a generally acceptable thoracic compression with CPR performed on a hospital mattress. A longer systematic review identified 22 reports of CPR cases being carried out in vulnerable positions (21 in operating rooms, 1 in intensive care unit [ICU]), with 10/22 patients still alive.15 In a series of minor cases of 6 patients with IHCA refractory, positions are exposed to the use of boards with backpacks to compress better haemdy during CPR but do not result in ROSC.16 The effectiveness of CPR in vulnerable positions is not established, but very limited evidence suggests it may be better than providing no CPR, when the patient cannot be placed in a supine position, or so this can be done safely. Recommendations 1, 2, and 3 supported by CoSTR 2020 for BLS.4 Recommendation 4 last received a review of official evidence in 2010.17 Proposal for Compression Breakdown and PAUSE COR LOE Proposition 1 C-LD 1. In adult cardiac arrest, the amount of preshock and postshock pauses in chest compression should be as tired as possible. 1 C-LD 2. Healthcare providers should minimize the time it takes to check the pulse (no more than 10 s) during the rhythmic examination, and if the rescuer is unsure of feeling the pulse, the compression of the chest should be resumed. 2a B-R 3. When 2 or so rescuers are available, it is reasonable to change the chest compressor approximately every 2 minutes (or after about 5 compression cycles and ventilation at a ratio of 30:2) to avoid a decrease in compression quality. 2a B-R 4. It is reasonable to immediately resume chest compression after shock transmission for adults in cardiac arrest in any settings. 2a C-LD 5. For adults in cardiac arrest receiving CPR without advanced airways, it is reasonable to pause the compression to deliver 2 breaths, each given over 1 s. 2b C-LD 6. In adult cardiac arrest, it may be reasonable to perform CPR with a chest compression breakdown of at least 60%. The proposed Text Observation of evidence suggests a better outcome with increased chest compression fractions in patients with a staggering rhythm.18.19 In particular, The study also reported an increase in ROSC with a shorter pause of perishock.22 This proposal is based on the overall principle of minimizing disruption to CPR and maintaining a compression breakdown of the chest at least 60%, which studies reported were associated with better results.18,19,23 chest compression depths began to decrease 90 to 120 seconds of CPR, although the compression rate did not decrease significantly over that period of time.24 Random attempts using manicin found no difference in differences The percentage of high quality compression when rotating every 1 minute compared to every 2 minutes.25 Rotating the chest compressor set every 2 minutes is desirable because this approach maintains the quality of chest compression and takes advantage when CPR will usually be paused for rhythm analysis. Two RCTs that registered over 1000 patients found no improvement in survival when pausing CPR to analyze rhythm after defibrillation.26,27 ObservationAl Studies showed the ROSC declined when chest compression was not resumed immediately after the shock.28,28,28 279 Because the breakdown of chest compression of at least 60% was associated with improved resuscitation results, compression pause for ventilation should be as high as possible.18,19,23 A 2015 systematic review reported significant heterogeneity among the study, with several studies, but not all, reporting a better survival rate to hospital emissions associated with higher chest compression fractions.18,19,23 In 2 studies, higher chest compression fractions are associated with lower odds of survival.2,30 Compression rates and depth and cointerventions such as defrals , airway management, and medications, are also important and can react with a breakdown of chest compression. The prestigious high-level EMS system targets at least 60%, with 80% or higher being a frequent goal. Recommendations 1 and 4 were supported by CoSTR 2020 for BLS.4 Recommendations 2, 3, 5, and 6 last received a formal evidence review in 2015.31 Recommendations for Compression Depth and LOE Rate Proposition 1 B-NR 1. During manual CPR, rescuers need to do chest compression to a depth of at least 2 inches, or 5 cm, for the average adult while avoiding excessive chest compression depths (greater than 2.4 inches, or 6 cm). 2a B-NR 2. In adult victims of cardiac arrest, it is reasonable for rescuers to perform chest compression at a rate of 100 to 120/min. 2a C-LD 3. It can be beneficial for rescuers to avoid leaning on the chest between compressions to allow recoil the complete chest wall for adults in cardiac arrest. 2b C-EO 4. It may be reasonable to do chest compression so that chest compression and recoil/relaxation time is about the same. Recommendations-Specific Text Support A 2020 ILCOR scoping review32 identified 12 studies, including over 12 500 patients, looking at chest compression components. Several studies found better results, including survival to hospital clearance and defibrillation success, when compression depths of at least 5 cm compared to less than 4 cm.3,20,33,34 similar studies identified 13 studies, involving 15 000 patients, who saw compression rates. Results are somewhat inconsistent across studies, with only 3 observational studies in adults showing relevance higher compression rates and yields.1,35,36 The only identified RCT including 292 patients and compared to the rate of 100 to 120 rates, could not find a difference in yields.37 There was no evidence recommended compression rates of 100 to 120/min in adults. Three studies have reported that a deep decline as rates rise, highlighting traps assessing single CPR quality metrics in isolation.1-3 ILCOR Study 32 identified 2 observational studies that provided inconsistent results on the association between the speed of chest compression emissions and survival, With 1 study found no association and others found that faster emission velocity was associated with an increase in survival.38,39 Not allowing complete chest wall recoils to have been linked to increased intrathoracic pressures and a decrease in coronary perfume.41 cycles of CPR duty refers to the proportion of time spent in compression compared to the number of times spent in compression compared to the number of times spent in compression compared to the number of times spent in compression compared to the number of times spent in compression compared to the number of times spent in compression compared to the number of times spent in compression compared to the number of times spent in compression compared to the number of times spent in compression compared to the number of times spent in compression compared to the number of times spent in compression compared to the number of times spent in compression compared to the number of times spent in compression compared to the number of times spent in compression comparison compared to the number of times spent in compression compared to the number of times spent in compression compared to the number of times spent in compression compared to the number of times spent in The 2010 Guidelines recommend a 50% duty cycle, where the time spent in compression and decompression is the same, especially based on the facilities seen to be achieved in practice. Notably, in clinical studies in adults with VF arrests out of hospital (which 43% survived into hospital clearance), the mean duty cycle observed during resuscitation was 39%.42 Studies in children also found the mean duty cycle was 40%, suggest that shorter task cycles may become prevalent in clinical practice.43 Although many animal studies have observed higher blood flow and better results when the task cycle is less than 50%, the optimal task cycle is unknown. Currently, there is insufficient evidence to guarantee changes from existing proposals, which remain a knowledge gap that requires further investigation. Recommendations 1, 2, and 3 supported by CoSTR 2020 for BLS.4 Recommendation 4 last received a review of official evidence in 2010.44 Recommendations for CPR Feedback and CorE Monitoring Proposal 2b B-R 1. It may be reasonable to use the current audiovisual feedback device CPR for real-time optimization of CPR performance. 2b C-LD 2. It may be reasonable to use physiological parameters such as artery blood pressure or neat end co2 when able to monitor and optimize CPR quality. Specific Support Text Recommendations The systematic study of ILCOR 2020 found that most studies have not found a significant association between real-time feedback and better patient outcomes.4 However, no studies have identified significant harm, and some showed clinically significant improvements in survival. An RCT recently reported a 25.6% increase in survival to hospitals from IHCA with audio feedback on compression and recoil depths (54% compared to 28.4%; P&lt.001.45 Data analysis from the Registry of AHA Guidelines shows the possibility of a higher ROSC (probable ratio, 1.22; 95% CI, 1.04–1.34; P=0.017) CPR quality monitored using either ETCO2 or diastolic blood pressure studies in adult patients (IHCA and OHCA) reported that for each increase in compression depth of 10 mm, ETCO2 increased by 1.4 mm Hg.47 A 2018 systematic review ETCO2 as a prognostic indicator for ROSC48 found diversity in cutoff value, but less than 10 mm Hg is generally associated with poor results and more than 20 mm Hg has stronger contact with the ROSC than the value of more than 10 mm Hg. A combination of etcO2 associations higher with ROSC and the finding that increased chest compression depth can increase ETCO2 indicates that targeting compression to a value of at least 10 mm Hg , and ideally 20 mm Hg or larger, may be useful. The authenticity and reliability of ETCO2 in unearthy patients is unsteady. When available, monitoring of invasive artery blood pressure can also help assess and guide CPR efforts. The use of diastelic blood pressure monitoring during cardiac arrest is associated with a higher ROSC,46 but there is insufficient human data to suggest any specific stress. This recommendation is supported by 2020 CoSTRs for BLS and ALS.4.49 5.5 Ventilation and Compression-to-Ventilation Ratio Of Introduction of rescue breath for apneic patients with pulse is important. The relative contribution of assisted ventilation for patients in cardiac arrest is more controversial. There are fears that chest compression transmission without the help of ventilation for prolonged periods can be less effective than conventional CPR (compression plus breath) because the oxygen content of the arteries will decrease as the CPR period increases. These concerns are especially important in setting asphyxia cardiac arrest. Much of the published research involves patients whose arrests are considered cardiac origin and in settings with short EMS response times. It is likely that the time threshold exists beyond where the absence of ventilation may be dangerous, and the consent of discovery to all settings must be carefully considered.1 Once advanced airways have been placed, delivering continuous chest compression increases compression fractions but makes it harder to deliver adequate ventilation. Simultaneous compression and ventilation should be avoided,2 but delivery of chest compression without pause for ventilation seems a reasonable option.3 SGAs use adds to this complexity because ventilation efficiency during cardiac arrest may be worse than when using endotracheal tubes, although this is not incurred in the RCTs.4.5 Proposal was recently published for Fundamental Ventilation During Cardiac Capture COR LOE Proposition 2a C-LD 1. For adults in cardiac arrest receiving ventilation, the total neat amount of about 500 to 600 mL, or enough to produce a visible chest increase, is reasonable. 2a C-EO 2. In patients without advanced airways, it is reasonable to convey breath either by mouth or by using ventilation Bag. 2b C-EO 3. When preparing a breath of rescue, it may be reasonable to give 1 breath over 1 s, take a regular breath (not deep), and give a second rescue breath over 1 s. 3: C-LD Harm 4. 4. should avoid excessive ventilation (too many breaths or too large volume) during CPR. Special Support Text Review Suggestions has reported that sufficient amount of neat tides to cause visible chest increases, or about 500 to 600 mL, providing adequate ventilation while minimizing the risk of overdistension or gastric insufflation.6-9 Both mouth-to-mouth rescue breathing and bagmask ventilation provide oxygen and ventilation to victims.10 To provide mouth-to-mouth rescue breathing, open mouth-to-mouth rescue vessels , pinch of the victim's nose. , make the mouth seal to the most airborne mouth, and give it a breath. Taking regular rather than deep breath prevents rescuers from gaining dizziness or head and prevents the victim's lung overinflation. The most common cause of ventilation difficulties is airways that are not open properly,11 so if the victim's chest does not rise with the first rescue breath, relocate the head by doing a tilted lift-chin head again and then giving a second rescue breath. The 1-second recommendation is to keep pause in CPR as shortly as possible. Excessive ventilation is not necessary and can cause gastric inflation, regurgitation, and aspirations.12,14 excessive ventilation can also be harmful by increasing intrathoracic pressure, reducing venous returns to the heart, and reducing heart output and survival.14 This topic last time received a formal evidence review in 2010.15 Proposals for Ventilation During Cardiac Capture: Special Situations CORE Proposed 2A C-1 It is reasonable for rescuers to use mouth-to-nose ventilation if ventilation through the victim's mouth is impossible or not practical. 2b C-EO 2. For victims with tracheal stoma that require rescue breathing, either mouth to stoma or face mask (pediatric prevailed)– ventilation to-stoma may be reasonable. Recommendations of text mouth-to-nose Ventilation Support may be necessary if ventilation through the victim's mouth is impossible due to trauma, position, or difficulty getting a seal. A series of cases indicate that mouth-to-nose ventilation in adults is feasible, safe, and effective.16 Effective ventilation of patients with tracheal stoma may require ventilation through the stoma, either by using mouth-to-mouth rescue breaths or by using bag mask techniques that create tight seals over stomas with round pediatric face masks. No evidence is published on the safety, effectiveness, or feasibility of mouth ventilation to the stoma. A patient study with laryngectomies showed that paediatric face masks created better peristomal seals than standard ventilation masks.17 This topic last time received a formal evidence review in 2010.15 Ventilation Recommendations in Patients Spontaneous Circle (Respiratory Arrest) CORE 2 If an adult victim with spontaneous circulation (that is, a strong and easy to spend pulse) requires ventilation support, it may be reasonable for health care to breathe rescue at a rate of about 1 breath per 6 s, or about 10 breaths per minute. Specific Support Text Recommendations Since the last study in 2010 rescue breathing in adult patients, there is no evidence to support changes in previous recommendations. A study in critically ill patients who needed ventilatory support found that ventilatory bag mask ventilation at a rate of 10 breath per minute decreased hypoxic events before intubation.18 This topic last time received official evidence reviews in 2010.15 Recommendations for Compression-to-Ventilation Ratio: ALS CORE LO Before the placement of sophisticated airways (supraglottic airways or tracheal tubes), it is reasonable for healthcare providers to perform CPR with a cycle of 30 compressions and 2 breaths. 2b B-R 2. It may be reasonable for EMS suppliers to use the rate of 10 breath per minute (1 breath per 6 s) to provide asynchronous ventilation during continuous chest compression before the placement of advanced airways. 2b C-LD 3. If advanced airways are provided, it may be reasonable for suppliers to deliver 1 breath per 6 s (10 breath/min) while continuous chest compression is being done. 2b C-LD 4. It may be reasonable to initially use minimally disturbed chest compression (that is, ventilation delays) to witness OHCA surprised as part of the care bond. Proposed Specific Support Text Systematic Reviews ILCOR 2017 found a ratio of 30 compressions to 2 breaths linked to better survival than alternative ratios, a proposal that AHA confirmed in 2018.19,20 Most of these studies examined cardiac care packages, making it impossible to know if the increase was due to the compression ratio to the pengudar This ratio is supported by a large OHCA RCT where consumption 30:2 (with a pause in compression less than 5 seconds) is at least as soon as the compression of the chest continues.21 In large trials, survival and survival with encouraging nerve outcomes are the same in a group of patients with OHCA being treated with ventilation at a rate of 10/min compared to the ratio of 30:2 before the intubation.21 A 2017 systematic study identified 1 human study observation and 10 animal studies compared different ventilation rates after the placement of advanced airways.22 No clear benefits from the rate of 10 identified, but no other rates were found to be superior. ILCOR's 2017 systematic review does not identify any new evidence to change this proposal, which has been repeated in its 2017 AHA Focus Update on Adult BLS and CPR Quality: Updates to the AHA Guidelines for CPR and Emergency Cardiovascular Care. 19.20 A 2017 systematic review of ILCOR concluded that despite evidence from an observational study The use of care packages including minimally disturbed chest compression is very low certainty (especially unsynced results), the system already uses using can continue to do so.19 This proposal is supported by 2017 focus updates on BLS quality guidelines and adult CPR.20 5.6 IntroductionDefibrillation Along with CPR, Early defibrillation is critical to survive when sudden cardiac arrest is caused by VF or VT without pulse (pVT).1.2 Defibrillation is most successful when administered as soon as possible after the onset of VF/VT and reasonable immediate treatment when the interval from start to shock is very brief. On the other hand, when VF/VT is more protracted, the compilation of heart energy reserves can compromise on the effectiveness of defibrillation unless refilled by a prescribed period of CPR before rhythm analysis. Minimizing disruption in CPR around the administration of surprise is also a high priority. Currently marketed defibrillators use different forms of proprietary shock waves in their electrical characteristics. This delivers different peak currents even at the same programmed energy settings, making the comparison of the effectiveness of surprises between challenging devices. The energy-setting specifications for cardioversion also differ between defibrillators. Refer to the energy recommended by the device manufacturer for a certain wave form. Technology is now in development to diagnose basic heart rhythms during ongoing CPR and obtain prognostic information from ventricle waves that can help guide patient management. This still requires further testing and verification before routine use. Proposals for Defibrillation Indicators, Type, and Energy COR LOE Proposition 1 B-NR 1. Defibrillators (using biphasic or monophasic waves) are recommended to treat tachyarrhythmias that require surprise. 2a B-R 2. Based on their greater success in termination of arrhythmia, defibrillators use biphasic waves prioritised over monophasic defibrillators for the treatment of tachyarrhythmias. 2a B-NR 3. A single surprise strategy is reasonable in preference to sorting out surprises for defibrillation in the setting of non-monitoraous cardiac arrest. 2a C-LD 4. It is reasonable that the selection remains compared to the growing energy levels for subsequent shocks for shock refractory arrhythmias that are considered to be based on specific manufacturer instructions for the wave. If this is not known, defibrillation at the maximum dose can be considered. 2b B-R 5. If using a defibrillator capable of increasing energy, higher energy for second shocks and in turn can be considered for a shock refractory arrhythmia that is considered a surprise. 2b C-LD 6. In the absence of conclusive evidence that one biphasic wave is superior to the other in the termination of the VF, it is reasonable to use the manufacturer's recommended dose of energy for the first shock. If this is unknown, defibrillation at the maximum dose can be Recommendations of the Text of Cardioversion and defibrillation electrical moisture support are very effective for ending VF/VT and other tachyarrhythmias. No surprise wave has distinguished itself as achieving ROSC rates or higher survival. Biphasic and monophasic shock waves may be equivalent to the effectiveness of their clinical outcomes.3 No surprise waves have proved superior in increasing ROSC rates or survival. However, the biphasic wave form defibrillator (which delivers the opposite polar pulse) exposes patients to lower peak electric current with equivalent or greater effectiveness to end atrium4 and tachyarrh vent Ythmias'tricl of monophasic (single pole) defibrillator do.5-10.13 Potential differences in safety and effectiveness in favor of the use of biphasic defibrillator priorities, when the Biphasic defibrillators available have largely replaced the monophasic surprise defibrillator, which is no longer produced. The rationale for a single surprise strategy, in which CPR immediately resumed after the first shock rather than after the shock sorted serial (if required) was based on some consideration. This included the first surprise high success rate with biphasic waves (reducing the need for consecutive surprises as well), the success of declining second and third serial shocks when the first surprise had failed,14 and lingering disruptions in the CPR needed for a series of shocks sorted. A single surprise strategy resulted in shorter disruptions in CPR and improved survival to hospital admissions and relief (although not 1 year of survival)

compared to serial shocks compiled shocks.15-17 It is not known whether stacked shocks or single shocks were more effective in settings seeing the Heart Capture section after Cardiac Surgery). Regardless of the waves, successful defibrillation requires shock to be sufficient energy to end the VF/VT. In cases where initial shock failed to end VF/VT, subsequent surprises may be effective when recurring at the same or growing energy settings.18.19 Optimal energy settings for first or subsequent biphasic defibrillation, whether fixed or rising, have not been identified, and its selection could be based on defibrillators in particular. There is no definitive evidence of the prominence of one biphasic shock wave over the other for defibrillation.20 Given the diversity in electrical characteristics between proprietary biphasic waves, it is reasonable to use energy settings determined by the manufacturer for that particular device. If the energy settings specified by the manufacturer for defibrillation are not known at the time of intended use, the maximum dosage setting for the device can be considered. Commercially available defibrillators either provide fixed energy settings or allow growing energy settings; both of these approaches are highly effective terminate the optimal VF/VT.18 Energy fixation for first or subsequent biphasic defibrillation, whether fixed or increased, not yet identified and best delayed to defibrillator manufacturers. Randomized attempts compare fixed fixed J biphasic defibrillation with a higher shock spirit increase (200-300-360 J) observed successful defibrillation rates and conversions to planned rhythms after the first shock. However, among patients requiring double shock, the growing shock energy causes a significantly higher conversion rate to organized rhythms, although overall survival does not differ between 2 treatment groups.19 When VF/VT refractory to first shock, equivalent energy settings or higher than first shocks can be considered. However, there is no definitive evidence of the prominence of one biphasic shock wave over the other for defibrillation.20 It is reasonable to use the energy settings determined by the manufacturer for that particular device. If the energy settings specified by the manufacturer for defibrillation are not known at the time of intended use, the maximum dosage setting for the device can be considered. Proposition 1, 2, and 6 last received a review of official evidence in 2015.21 Recommendations 3, 4, and 5 supported by CoSTR 2020 for BLS.22 Recommendation for Pads for Defibrillation COR LOE Recommendation 2a C-LD 1. It is reasonable to put defibrillation paddles or pads in the chest exposed in anterolateral or anteroposterior positions, and use an electrolide diameter pad or pad more than 8 cm in adults. Recommendations of Anterolateral Support Text, anteroposterior, anterior-left infrascapular, and the placement of anterior senior infrascapular electrodes are very effective for treating supraventricular and ventricular arrhythmia. 24-28 Greater pad/paddle size (within a limit of 8-12 cm in diameter) lowered the transthoracic impedance.29.30 self-adhesive towels have largely replaced defibrillation pads in clinical practice. Before the placement of the pad, remove all clothes and jewelry from the chest. The recommendation was supported by a review of the scope of ILCOR 2020, which found no new information to update the recommendations of 2010.22.31 Proposals for Defibrillation Manual-Mode Automatic COR LOE Recommendation 2b C-LD 1. It may be reasonable to use defibrillator in manual mode compared to automatic mode depending on the skill set of the operator. Recommendations of AED Support Text are very accurate in surprising detection of arrhythmias but require a pause in the CPR for automatic rhythm analysis.33 Defibrillation Manual can cause a shorter hands-off period for rhythmic authentication in operators with sufficient skills for a rhythmic interpretation fast and reliable.34.35 This proposal is supported by a scoping review of ILCOR 2020,22 that found no new information to update the proposed 2010.31 Proposal for CPR Before Defibrillation COR LOE Proposal 1 C-LD 1. CPR is recommended until a defibrillator or AED is used. 2a B-R 2. In cardiac arrest not dimonitor, it is reasonable to provide a short period of CPR while the defibrillator is being obtained read for use before initial rhythm analysis and possible defibrillation. 2a C-LD 3. Immediate defibrillation is reasonable for suppliers to witness or monitor short-term VF/pVT when the defibrillator is already in use or available immediately. Recommendations text CPR Support is the single most important intervention for patients in cardiac arrest and should be provided until the defibrillator is used to minimize disruption in compression. When VF/VT was present for more than a few minutes, the myocardial reserves of oxygen and other energy substates were quickly uninsponsed. If repaid by the CPR period before the surprise, defibrillation success improved significantly.1,2,36,37 Because no difference in results was seen in the study comparing short (usually about 30 seconds) with prolonged periods (up to 3 minutes) of CPR ahead of the initial rhythm, Analysis, CPR's brief period analysis while defibrillator is read to be used may be sufficient in non-taxable cardiac capture.38-40 Even in monitored catches, it can take time to attach pads, power on defibrillators, and impose capacity before surprise delivery, where there is good reason to administer the CPR. Early defibrillation increased revenue from cardiac arrest.41-43 When VFs were in a short period, oxygen myocardial reserves and other energy substrates may remain intact. During this initial electricity phase, the most responsive rhythm to defibrillation.44.45 Therefore, if the beginning of the VF is monitored or tested with a defibrillator already used, or that there is immediate access, it is reasonable to administer the shock as soon as possible. Interim CPR should be provided if there are any delays in obtaining or preparing a defibrillator for use. Recommendations 1 and 2 supported by CoSTR 2020 for BLS.22 Recommendation 3 last received a review of official evidence in 2010.46 Recommendations for Defibrillator Anticipatory Charging COR LOE Recommendation 2b C-EO 1. It may be reasonable to impose a manual defibrillator during chest compression either before or after analysis of the scheduled rhythm. Recommendations of Supporting Text There are different approaches to imposing manual defibrillators during resuscitation. It is not uncommon for chest compression paused for rhythm detection and continues to be withheld while the defibrillator is charged and prepared for shock delivery. This approach resulted in a protracted period of hands-off before surprise. Precharging defibrillator during continuous chest compression shortens hands-off chest time around defibrillation, without evidence of harm.47 Although no study directly assesses the effects of self-precharging on heart capture results, shorter perishock pauses (which can be caused by such strategies) are associated with such a better survival is reasonable: either impose a defibrillator before the rhythm checks or resuming compressing compressing after rhythmic examination while charge defibrillator. Whether the approach can reduce the flowless time.49.50 This proposal is supported by CoSTR 2020 for the ALS.51 Proposal for Postshock Rhythm Check CORE Proposition 2b C-LD 1. It may be reasonable to immediately resume chest compression after a shock administration rather than pause CPR to perform a postshock rhythm screening in heart arrest patients. Recommendation-Specific Texts Instant Support reconnection of chest compression after shock results in shorter perishock pauses and increase the entire hand time (chest compression breakdown) during resuscitation, Associated with better survival than the capture of VF.16.48 Despite being successful, defibrillation is often followed by a period of variables (and sometimes protracted) asystole or without pulses, where preparing CPR while waiting for the return of rhythm and pulse is advisable. Whether the resuming of CPR shortly after the surprise might reintroduce the VF/VT is controversial.52-54 This potential concern is not borne by any evidence of worsening survival from such a strategy. If there is physiological evidence of distribution returns such as arterial waves or a sharp rise in ETCO2 after shock, a brief chest compression pause for authentication rhythm analysis can be guaranteed. The recommendation was supported by CoSTR 2020 for BLS.22 Recommendations for Ancillary Defibrillator Technologies COR LOE Proposition 2b C-LD 1. The algorithmic value of artefacts filtration for electrokardiogram rhythm analysis (ECG) during chest compression has not been created. 2b C-LD 2. The analytical value of VF waves to guide the acute management of adults with cardiac arrest has not yet been established. Recommendations Text Support CPR obscures the interpretation of basic rhythms due to the artifacts created by chest compression on the ECG. This difficulty in planning the next step of care and potentially delaying or even abusing drug therapy if given empirically (blind) based on the patient's presumption, but not real, basic rhythm. The time taken for rhythm analysis also interrupts CPR. Filtration of artifacts and other innovative techniques to expose basic rhythms under continuous CPR can overcome these challenges and minimize disruption in chest compression while offering diagnostic advantages for better direct therapy.55-60 Despite the theoretical advantages, no study has assessed this technology in a real-time clinical environment or confirmed their clinical effectiveness compared to current resuscitation strategies. Currently, filtering algorithms are used strictly for visual rhythm interpretations (manual) and not for automatic VF/VT rhythm detection in AED while CPR continues. This potentially added application has yet to be worked out. Recognizing the need for further clinical research, systematic review 2020 recommended against acceptance The algorithm for current rhythm analysis of CPR.51 Writing groups also supports the need for further investigation and clinical confirmation before this technology is adopted into clinical practice. Electrical characteristics of the VF wave are known to change over time.61 VF wave analysis may be valued in predicting the success of defibrillation or other therapies during resuscitation.62-64 Theoretical-based prospects on prognostic analysis of VF waves in real time are an interesting and developing one however, the validity, reliability, and clinical effectiveness of the approach that prompts or withstands shock or other therapies based on prediction analysts are currently uncertain. The only clinical trial that compared standard-first surprise protocols with surprise algorithms guided by wave analysis does not observe no difference in the outcome.65 Consensus writing group is that currently there is insufficient evidence to support the routine use of wave analysis to guide resuscitation care, but it is an area where further research with clinical Recommendation 1 is supported by CoSTR 2020 for ALS.51 Recommendation 2 supported by an update of evidence of ILCOR 2020,51 which found no new information to update the proposed 2010.66 Recommendations for Double Sequential Defibrillation COR LOE Recommendation 2b C-LD 1. Defibrillation uses are double sequence for rhythms that are surprised refractory has not yet been established. Recommendations Text Support There is limited evidence that examines multiple sequenced defibrilation in clinical practice. Some case reports have shown good results in patients receiving double-seeded defibrillation. However, reports of this case are subject to publication tendencies and should not be used to support its effectiveness.67 A handful of observational studies show no difference in outcomes (ROSC, survival, nervous results) with the use of multiple sequentially detached defibrillation compared to standard defibrillation.68-71 These studies should also be interpreted with caution, since the use of double-sequentised defibrillation is not protocol and often used late in resuscitation after standard resuscitation is unsuccessful. The published report also did not distinguish the utilisation of double-sequentially sequenced defibrillation to really shock-refractory (non-stop) VF versus recurring VFs during the CPR period after a successful surprise, which is a more common clinical scenario.3.7 Systematic study of ILCOR 2020 found there was no evidence to support double-sequential defibrillation and recommended against its routine use compared to the defibrillation of standard.51 RCT pilots recently (not included in the systematic study) of 152 patients who remained in the VF after at least 3 higher and ROSC with seceding defibrillation defibrillator pad placement compared to standard defibrillation but not powered for this outcome and did not report patient survival.72 Some unanswered questions remained about double-sequenced defibrillation, including intershock time, pad positioning, technique, and possible harm with increased energy and defibrillator damage.73.74 It is premature for double-seeded defibrillation to be included in routine clinical practice given the lack of evidence. Its uses should be explored in the context of clinical trials. Continuous RCT (NCT04080986) can provide answers to some of these questions. The recommendation was supported by CoSTR 2020 for ALS.51 5.7Other Electric or Pseudo-Electric Therapies for Cardiac Arrest Introduction In addition to defibrillation, some electrical therapy and alternative pseudoelectrics have been explored as possible treatment options during cardiac arrest. Transcutaneous pacing was studied during cardiac arrest with bradyasystolic heart rhythms. The theory is that the heart will respond to electrical stimulation by producing myocardial contraction and generating forward blood movements, but clinical trials show no pacing to improve patient outcomes. Other pseudoelectric therapies, such as CPR cough, fists or percussion pacing, and precordial thump have all been described as seductive measures in selective patients who either periarrest or in the early moments of accomplished cardiac arrest (before losing consciousness in case of cough CPR) when the therapy is finally unavailable. Precordial thump is one, sharp, high-directional effect (or punching) to the middle sternum by a tight aspect of fist ulnar. The force from the precordial thump aims to transmit electricity to the heart, similar to low energy shocks, in the hope of ending the basic tachyarrhythmia. Fist (or percussion) pacing is a serial, rhythmic delivery, a relatively low duplication effect to the sternum by a closed fist.1 Pacing fist is administered in an attempt to stimulate sufficient electrical impulse to cause myocardial depolarization. CPR cough is described as breath in recurrence followed by coughing every few seconds in an attempt to increase aortic and intracardiac pressure, providing temporary hemodynamic support before losing consciousness. Proposal for Electric Beat COR LOE Recommendation 3: No Benefit B-R 1. Electric pacing is not recommended for routine use in established cardiac arrest. Recommendations Text Existing Evidence Support, including observational and quasi-RCT data, suggests that pacing by a transcutaneous, transventous, or transmyocardial approach in cardiac arrest does not increase the possibility of ROSC or survival, regardless of the administrative time of the pacing in the established asistole, the location of the arrest (in hospital or outside or main heart rhythm (asystole, pulseless pulseless activity).2-6 Disruption persists in chest compression while the success of the pacing is assessed can also be detrimental to survival. It is not known whether the start time of the pacing can influence the success of the pacing so that the pacing may be useful at the beginning of the selected cases that are testified, monitored cardiac arrest (see section of Cardiac arrest After Cardiac Surgery). If pacing is driven during cardiac arrest related to the special conditions described above, the supplier cautions against its performance at the expense of high-quality CPR, especially when assessing electrical and mechanical arrests. The topic last underwent an official evidence review in 2010.7 Recommendations for Thump COR LOE Precordial COR LOE 2b B-NR 1 Proposal. Precordial thump can be considered at the onset of a suspected, monitored rescue, unstable ventricular tachyarrhythmia when the defibrillator is not immediately ready to use and is done without delaying CPR or shock delivery. 3: No C-LD Benefit 2. Precordial thump cannot be used routinely for established cardiac arrest. Specific Support Text Suggestions and 2. The precordial thump intent is to transmit the mechanical force of thump to the heart as an analogic electricity to a pacing boost or very low energy shock (depending on its force) and referred to as electromistic transduction.1 There is no evidence that the use of pre-season thump during the card Routine Catch care outside the hospital or in settings in hospitals increases the ROSC rate or survival for hospital discharge.8-12 It may be beneficial only in the early start of VT when arrhythmia is most vulnerable to low energy termination as in a state of care, monitored, or in a controlled laboratory environment, but although it is rarely effective.13 Despite reports of success cases without evidence of harm from a practical thump ,9,14,15 if deliberately administered on the electric beat part of the planned rhythm (wave T), thump (such as unsynchronized shock) risk of acceleration or conversion of rhythm to VF ,16-19 analogy to the commotio cordis.20 although thump may be useful as a single brief intervention in certain circumstances (that is, when cardiac arrest is detected by responders and certified monitors due to VF/VT and defibrillator is not available for use), it cannot slow down CPR or defibrillator use. The recommendation is supported by CoSTR 2020 for the Recommendation of BLS.21 for Fists/Percussion Pacing COR LOE Recommendations 2b C-LD 1. Fists (percussion) pacing can be considered a seductive measure in unusual circumstances such as being tested, monitored arrests in hospitals (for example, cardiac catheterization laboratories) for bradyasystole before losing consciousness and if done without delaying the ultimate therapy. Special Supporting Text Puller Suggestions, or percussion, pacing with the goal of stimulating sufficient electrical impulses to cause depolarization and myocardial contraction, resulting in a pulse. There are several case reports and a series of cases that examined the use of fist pacing during asystolic or bradycardic incidents that threatened the lives of events1,22-25 showed good results for survival2 and ROSC.23 None of these studies, however, controlled or compared, and it is not known if the use of the fist pacing itself increases the rate of ROSC or survival There is no role for pacing pullers in patients in cardiac arrest. The recommendation is supported by CoSTR 2020 for the Recommendation of BLS.21 for Cough CPR COR LOE Recommendation 2b C-LD 1. CPR Cough can be considered a tempting step to be tested, monitored the onset of tachyarrhythmia or significant bradyarrhythmia before losing consciousness without delaying the ultimate therapy. Recommendations of Text Support It is important to stress that although CPR cough by definition should not be used for unconscious patients, it can be harmful in any setting if diverting time, effort, and attention from implementing high-quality CPR. CPR cough is described as an in-depth inspiration repeatedly followed by cough every few seconds before missing consciousness. It can only be implemented at the onset of essential haemy arrhythmia in cooperatives, conscious patients who have been ideally directed on its performance, and as a bridge to ultimate care. There are no studies that compare CPR cough to standard resuscitation care. Limited evidence from case reports and case series shows a temporary increase in aortic and intracardiac pressures with the use of CPR cough at the onset of tachyarrhythmias or bradyarrhythmias in patients conscious.10,26-28 This study experienced a large selection bias and lack of comparative groups, and did not control the confusing effects of other treatments, making them difficult to control other treatments, making them difficult to control the effects of confusing treatment, making them difficult to control other treatments, making them difficult to control the effects of confusing treatment, making them difficult to control other treatments, making them difficult to control the effects of confusing treatment, making them difficult to control other treatments, making them difficult to control the effects of confusing treatment, making them difficult to control other treatments, making them difficult to control the effects of confusing other treatments, making them difficult to control the effects of confusing treatment, making them difficult to control other treatments, making them difficult to control the effects of confusing treatment, making them difficult to control other treatments, making them difficult to control the effects of confusing treatment, making them difficult to control other treatments, making them difficult to control The recommendation was supported by CoSTR 2020 for BLS.21 5.8Vaskular Access Recommendations for Vascular Access in Cardiac Arrest Management COR LOE Recommendations 2a B-NR 1. It is reasonable for suppliers to first attempt to create intravenous access to drug administration in cardiac arrest. 2b B-NR 2. Intraosseous access can be considered if attempts at intravenous access are unsuccessful or unacceptable. 2b C-LD 3. In appropriately trained suppliers, venous central access can be considered if attempts to create intravenous and intraosseous access are unsuccessful or non-feasible. 2b C-LD 4. The administration of endotracheal drugs may be considered when other access routes are unavailable. Synoxis The traditional approach to giving emergency pharmacotherapy is with a periferal IV route. However, gaining IV access under circumstances appears to be challenging based on patient characteristics and operator experience leading to delays Treatment. Alternatives to IV access for acute drug administration include IO, venous center, intracardiac, and endotracheal routes. The administration of intracardiac drugs is not encouraged in the 2000 AHA Guidelines for CPR and Emergency Cardiovascular Care given the highly specialized skill sets, morbidity potential, and other options available for access.1,2 Endotracheal drug administration resulted in low blood concentrations and unpredictable pharmacological effects and also largely fallen into use Central venous access is primarily used in the hospital environment as it requires appropriate training to acquire and maintain the necessary skill sets. IO access has grown in popularity given the relative ease and speed for which it can be achieved, higher placement rates are successful compared to IV insulation, and relatively low procedural risks. However, the effectiveness of IV versus the administration of IO drugs in permanent cardiac arrest will be refined. The recommendations of periperal IV Route Support Text have become a traditional approach to vascular access for emergency drugs and fluid administration during resuscitation. The nature of pharmacodinetics, acute effects, and clinical efficacy of emergency medications has been particularly described when given intravenously.3-6 Route IV has priority, is usually accessible, and is capable of a potentially more predictable drug response, making it a reasonable initial approach to vascular access. The dishonesty of information about the effectiveness of the current IO drug administration of CPR was acknowledged in 2010, but since then the IO route has grown in popularity. IO access is increasingly being implemented as the first-line approach to emerging vascular access. A systematic review of ILCOR 20207 compared to IV versus IO (mainly pre-defense placement) of drug administration during cardiac arrest found that IV routes were associated with better clinical outcomes compared to IO in 5 retrospective studies.8-12 There were significant concerns for bias, mainly due to the fact that the need for IO settlements may indicate Subgroups IV analysis against IO routes from 2 RCTs is also included in this systematic study. In this regard, no significant statistical impact modifications by administrative routes have been identified. Eye estimates favor IV access except for ROSC results in the PARAMEDIC2 trial, where the effects of epinephrine are the same regardless of route.13.14 Site Specifications can also be an issue with the IO administration, since IO access is almost always pretibial in the study. Based on these results, the writing group concluded that setting up peripheral IV remains a reasonable initial approach, but IO access can be considered IV was unsuccessful or feasible. Further research is needed to assess the effectiveness of drugs transmitted intravenously compared to intraosseously (tibial and and The administration of drugs by venous center access (by internal jugular or sub-productive veins) achieves higher peak concentrations and faster circulation times than drugs administered by the periction of IV do,15-17 but currently there is no data comparing clinical outcomes between these access routes. Central access is associated with higher morbidity, takes time to perform, and may also require CPR outages. The current use of this approach is largely in hospital and can be considered by skilled suppliers when IV and IO access is unsuccessful or feasible. The administration of endotracheal drugs is considered the least prioritised route of drug administration because it is associated with unpredictable drug concentrations (but generally low) drug concentrations18-20 and lower rosc rates and survival rates.21 Proposals 1 and 2 supported by 202 CoSTR for ALS.22 Recommendations 3 and 4 finally received official evidence reviews in 2010.20 5.9Vasopressor Medications During the Proposed Cardiac Arrest for Vasopressor Management in Cardiac Arrest COR LOE Proposed 1 B-R 1. We recommend that epinephrine be administered for patients in cardiac arrest. 2a B-R 2. Based on the protocol used in clinical trials, it is reasonable to administer epinephrine 1 mg every 3 to 5 minutes for cardiac arrest. 2a C-LD 3. With regard to time, for cardiac arrest with an unresolved rhythm, it is reasonable to administer the epinephrine as soon as possible. 2b C-LD 4. With regard to time, for cardiac arrest with a shocking rhythm, it may be reasonable to administer epinephrine after an initial defibrillation attempt has failed. 2b C-LD 5. Vasopressin alone or vasopressin in combination with epinephrine can be considered in cardiac arrest but offers no advantage as a substitute for epinephrine in cardiac arrest. 3: No Benefit B-R 6. High dose epinephrine is not recommended for routine use in cardiac arrest. Epinephrine synopsis has hypothesized to have a beneficial effect during cardiac arrest mainly because of the α-adrenergic effect, leading to increased pressure on coronary perfume and sting during CPR. On the other hand, β-adrenergic effects can increase the demand for myocardial oxygen, reduce subendocardial perfume, and possibly proarrhythmic. Two randomized, placebo-controlled trials, enrolled over 8500 patients, assessing the effectiveness of epinephrine for OHCA.1.2 Systematic and meta-analysis studies and other studies3 concluded that epinephrine significantly increases the ROSC and survival to Epinephrine hospitals does not lead to increased survival with neurological outcomes although these two results occurred slightly more often in the group epinephrine.2 Data suggests better outcomes when epinephrine is given early, and low survival with encouraging neurological outcomes in existing trials may be due in part to the median time of 21 minutes from the catch to receive an epinephrine. Delay this time a consistent issue in the OHCA trial. The time for drugs at IHCA is usually much shorter, and the effects of epinephrine on results in the IHCA population may vary. No attempts so far have found any benefits of either higher dose epinephrine or other vasopressors over standard dose epinephrine during CPR. Recommendations Text Specifics Support Proposals to administer epinephrine have been reinforced to recommendations based on systematic and meta-analysis review,3 which includes the results of 2 randomized epinephrine tests for OHCA, 1 of which included over 8000 patients,1,2 showing that epinephrine rose ROSC and epinephrine increased ROSC and survival. At 3 months, the point of time felt most meaningful for nervous rehabilitation, there was an intangible increase in survivors with both favorable and unfavorable neurological outcomes in the epinephrine.2 Any drug that increases the ROSC rate and survival, but given after a few minutes of downtime, will likely increase both possible neurological outcomes Therefore, continuing to use the drugs that have been shown to improve survival, while focusing our broader efforts on shortening time for drugs for all patients so that more survivors will have favorable neurological outcomes, apparently the most beneficial approach. Existing tests have been using 1 mg protocol every 3 to 5 minutes. In terms of operation, administering the epinephrine of each second cycle of CPR, after the initial dose, may also be reasonable. Of the 16 observational studies on time in a recent systematic review, all found associations between previous epinephrine and ROSC for patients with unresolved rhythms, although an increase in survival was not seen universally.3 For shocked rhythms, trial protocols have ordered that epinephrine be given after a third shock. Literature supports prioritizing defibrillation and CPR initially and giving epinephrine if initial attempts with CPR and defibrillation were unsuccessful.3 Recent systematic reviews found no difference in outcomes in trials that compared vasopressin alone or vasopressin combined with epinephrine to epinephrine although this study is combined with epinephrine for epinephrine alone for cardiac arrest, although this study is combined with epinephrine for epinephrine alone for cardiac arrest , although this study is combined with epinephrine for epinephrine alone for cardiac arrest, although this study is combined with epinephrine for epinephrine alone for cardiac arrest, although this study is with epinephrine for epinephrine alone for cardiac arrest, although this study is combined with epinephrine for epinephrine alone for cardiac arrest, Although this study is combined with epinephrine for epinephrine alone for cardiac arrest, although this study is combined with epinephrine for epinephrine alone for cardiac arrest, although this study is combined with epinephrine for epinephrine alone for cardiac arrest, wa Various RCTs have compared high doses with standards , and although some show higher ROSC rates with high dose epinephrine, none showed an increase in survival to perform or any long-term outcome.4-11 This proposal is supported by the 2019 AHA Focused Update on Advanced Cardiovascular Life Support: Use of Advanced Airways, Vasopressors, and CPR Extracorporeal During Cardiac Capture: Updates to AHA Guidelines for CPR and Cardiovascular Care 12 5.10Nonvasopressor Medications During The Proposed Cardiac Arrest for Nonvasopressor COR LOE Medications Proposed 2b B-R 1. Amiodarone or or can be considered for VF/pVT that is not responsible for defibrillation. 2b C-LD 2. For patients with OHCA, current steroid use of CPR is an uncertain benefit. 3: No Benefit B-NR 3. Routine administration of calcium for cardiac arrest treatment is not recommended. 3: No Benefit B-R 4. Use of sodium biconbonate routines is not recommended for patients in cardiac arrest. 3: No Benefit B-R 5. The use of magnesium routines for cardiac arrest is not recommended. Cardiac capture pharmacological treatment is usually deployed when CPR with or without attempted defibrillation failed to reach the ROSC. This may include vasopressor agents such as epinephrine (discussed in Vasopressor Drugs During Cardiac Arrest) as well as medications without direct (non-pressing) hemodynamic effects such as antiarrhythmic drugs, magnesium, sodium bikarbonate, calcium, steroids or steroids (discussed here). Although theoretically interesting and some of the proven benefits in animal studies, no last therapy has been proven definitively to improve overall survival after cardiac arrest, although some may have the possible benefits in selected populations and/or special conditions. Recommendations for the treatment of cardiac arrest due to hypercalemia, including the use of calcium and sodium bikarbonates, are presented in electrolyte Abnormalities. Recommendations for the management of torsades de pointes are also presented at Torsades de Pointes. Specifically-Specific Text Administration Amiodarone or lidocaine support to patients with OHCA was last formally reviewed in 20181 and showed better survival for hospital admissions but did not increase overall survival to hospital discharge or survival with good neurological outcomes.1,2 However, amiodarone and lidocaine each increased survival to hospitals in a subgroup of patients determined by the arrests found bystanders, potentially arguing for the benefit of time and groups for which these drugs may be more useful. Other antiarrhythmic agents have not been specifically addressed in the review of the most recent evidence and further merit assessment. This includes a bretylium tosylate, which was recently reintroduced in the United States for immediate treatment of life-threatening ventricle arrhythmia but without any new information about its effectiveness or safety.3 Sotalol requires administration as a slow infusion, cause it is impractical to use in cardiac arrest.4 The same limitation also applies to procainamide, although it has been given by rapid infusion as a second-row agent in cardiac arrest, with volatile benefits.5 The effectiveness of antiarrhythmic drugs when given in combination for cardiac arrest is not systematically and remains a knowledge gap The role of prophylactic antiarrhythmic drugs on the ROSC after a successful defibrillation is also volatile. Although not linked to better survival for hospital relief, lidocaine declines repeatedly when prophylactically administered after successful defibrillation and ROSC.6 2018 AHA Focused Update on Advanced Cardiovascular Life Support of Current Antiarrhythmic Drugs and Immediately After Capture Heart: Updates to the AHA Guidelines for CPR and Emergency Cardiovascular Care1 conclude that the use of lidocaine may be considered in certain circumstances (such as during ems transportation) when recurrent VF/pVT treatments may be affected. There is no evidence addressing the use of antiarrhythmic drugs others for these specific indicators. Two randomized tests from the same center reported increased survival and neurology results when steroids were combined in combination with vasopressin and epinephrine during cardiac arrest and also administered after the success of resuscitation from cardiac arrest.7.8 However, The unenforced study of strict corticosteroid administration, in addition to standard resuscitation, shows mixed results.9.10 Due to the only study that suggested benefits from a single center with combined interventions, and observational data that had conflicting results, whether that was beneficial during cardiac arrest remains unclear. At least 1 attempt attempt to verify the discovery of Mentzelopoulos et al is in progress (NCT03640949). Since the last time dealt with by the 2010 Guidelines, a 2013 systematic review found little evidence to support the use of calcium routines in accidental cardiac arrest, although the evidence is very weak calcium due to being the last resort drug in cardiac refractory arrest.11 Calcium administration in special conditions such as hypercalemia and doses of calcium blockers dealt with in Abnormalities and Electrolyte Toxicity: β-Adrenergic Blockers and Calcium Channelers Clinical trials and observational studies since the 2010 Guidelines have not produced new evidence that the routine administration of sodium bikarbonate increases the result of accidental arrest and heart evidence suggests that it can worsen survival and nerve rehabilitation.12-14 Sodium consumption of bikarbonate sodium in special conditions such as hypercalemia and medicines addressed in Abnormalities and Electrolyte Toxicity: Sodium Channel Blockers, Including the role of Tricyclic Antidepressants Magnesium as the last antiarrhythmic agent addressed by a 2018 update focused on advanced cardiovascular life support (ACLS) guidelines as a result of survival or neurology regardless of the rhythm of cardiac arrest delivers,15-18 or useful for VT.19 monomorphic There are anecdoal reports and a series of minor cases that show the effectiveness of magnesium in the treatment of torsades de pointes (See Torsades de Pointes). Recommendations 1 and 5 supported by 2018 focus updates on ACLS.1 Recommendations 2 last received a formal proof review 2015.20 Recommendations 3 and 4 last received a formal evidence review in 2010.21 5.11Adjuncts to the CPR Recommendations CPR Adja to CPR COR LOE Proposition 2b C-LD 1. If an experienced sonographer is present and the use of ultrasound does not interfere with the standard cardiac arrest treatment protocol, then the ultrasound can be considered an adjatic to the assessment of standard patients, although its use is not yet established. 2b C-LD 2. When additional oxygen is available, it may be reasonable to use maximum-inspired oxygen concentrations during CPR. 2b C-LD 3. A sharp rise in the final co2 could be used to detect the ROSC during compression or when a rhythmic inspection reveals a planned rhythm. 2b C-EO 4. Routine measurement of artery blood gas during CPR has volatile value. 2b C-EO 5. Arterial pressure monitoring by the artery line can be used to detect the ROSC during chest compression or when rhythmic inspection reveals the organized rhythm. Synopsis Although the vast majority of cardiac arrest attempts have been conducted in the OHCA, the IHCA consists of nearly half of the catches that occur in the United States each year, and many OHCA resuscitation goes straight to the emergency department. IHCA patients often have invasive monitoring devices in places such as venous lines or central arteries, and staff to perform advanced procedures such as artery blood gas analysis or ultrasound care points are often present. Advanced monitoring such as ETCO2 monitoring is increasingly in use. Determining physiological monitoring utilities or diagnostic procedures is important. High-quality CPR, defibrillation when appropriate, vasopressors and/or antiarrhythmics, and airway management remains the basis of cardiac arrest resuscitation, but some emerging data suggest that combining patient-specific imaging and physiological data into our approach to resuscitation holds some promises. See Metrics for High Quality CPR for current physiological monitoring recommendations of CPR. More research in this area is clearly needed. Suggestions-Specific Point-of-care Text Support cardiac ultrasound can identify cardiac tamponade or other potentially reversible cardiac arrest and identify cardiac movement in electrical activity without heartbeat.1.2 However, Heart ultrasounds were also associated with longer disorders in single small RCT compression found no increase in results with the use of heart ultrasounds during CPR.4 No adult human studies directly compared inspired levels of oxygen concentrations during CPR. A small number of studies have shown that pao2 higher during CPR is associated with the ROSC, but this may be due to differences in patients or quality resuscitation.5-7 ObservationAl Studies have found that an increase in ETCO2 over 10 mm Hg may indicate the ROSC, although no specific cutoff value indicates the ROSC has been identified.8 Arterial Value depending on the output and ventilation of the heart and therefore depending on both the characteristics of the patient and the quality of the CPR. A small study found widespread in blood gas between venous samples and mixed arteries during CPR and concludes that artery samples are inaccurate during resuscitation.9 If the artery line is in place, a sharp increase in diastolic pressure or the presence of artery waves during rhythmic examination indicates a planned rhythm may be ROSC. Proposal 1, 3, and 5 last received an official evidence review in 2015.10 Recommendation 2 last received an official evidence review in 2015.10 with a proof update completed in 2020.11 Recommendation 4 last received a formal evidence review in 2010.12 5.12Mination of Resuscitation Recommendations for Termination of Resuscitation COR LOE Recommendation 1 B-NR 1. If termination of resuscitation (TOR) is being considered, the BLS EMS provider must use the termination of the BLS resuscitation rules in which alS is unavailable or may be significantly delayed. 2a B-NR 2. It is reasonable for prehospital ALS providers to use adult ALS TOR rules to end on-field resuscitation efforts for OHCA adult victims. 2a B-NR 3. In the ALS- system- and progressive BLS providers, the use of TOR BLS regulations can avoid confusion at the scene of cardiac arrest without compromising diagnostic accuracy. 2b C-LD 4. In in patients inseminated, the failure to reach co2 end neat ends is greater than 10 mm Hg by wave capnography after 20 minutes of ALS resuscitation can be considered a component of a multimodal approach to deciding when to end resuscitation efforts, but it cannot be used in isolation. 3: No C-LD Benefit 5. We propose against the use of ultrasound care points for current prognostication of CPR. 3: Harm C-EO 6. In uneven patients, the value of a specific CO2 end cutoff at any time during CPR should not be used as an indicator to end the resuscitation effort. OHCA synoxis is the resource-intensive state most often associated with low survival rates. It is important for EMS providers to be able to distinguish patients where continuous resuscitation is pointless from patients with the opportunity to survive which should receive continuous resuscitation and transportation to hospital. This will help in the use of resources and optimize patient opportunities for survival. Using certified TOR rules will help ensure accuracy in determining cynical patients (Fig. 5 and 6). Futility is often defined as less than 1% chance of survival,1 suggesting that for legitimate TOR rules it should show high accuracy to predict futility with a lower confidence limit of greater than 99% on external authentication. Recommendations Text Support Regulation BLS TOR recommends TOR when all the following criteria apply before moving to an ambulance for transportation: (1) arrests are not detected by the first EMS provider or responder; (2) no ROSC is obtained; and (3) no surprise In recent meta-analysis 7 published studies (33 795 patients), only 0.13% (95% CI, 0.03%– 0.58%) patients who meet BLS BLS termination criteria to hospital discharge.3 ALS TOR regulations recommend TOR when all the following criteria apply before moving to an ambulance for transportation: (1) arrests are not to be found; (2) no bystander CPR is provided; (3) no ROSC after full ALS care in the field; and (4) no AED surprises were delivered. In recent meta-analysis 2 studies published (10 178 patients), only 0.01% (95% CI, 0.00%–0.07%) patients who meet the ALS termination criteria survived from hospital clearance.3 TOR regulations, otherwise known as universal TOR rules (arrests are not detected by EMS providers; no surprises delivered; no ROSC), has been confirmed in a combination of BLS and ALS.4 systems Although the rules do not have sufficient specifications after 6 minutes of resuscitation it achieved better than 99% of specifications after about 15 minutes of resuscitation attempts, while still reducing half transportation. Retrospective analysis found that the use of universal TORs on 20 minutes of resuscitation could predict futility, identifying more than 99% of survivors and patients with good neurological outcomes.5 In patients sown, ETCO2 measurements of less than 10 mm Hg showed low to blood flow. Several small studies provide evidence suggesting that ETCO2 is less than 10 mm Hg after 20 minutes of strong ALS resuscitation but not perfect futility forecast.6-9 This small observational study experiences a high risk of bias. Alternative ETCO2 thresholds and time points have been proposed. The use of ETCO2 alone to predict patient outcomes needs to be confirmed in large prospective studies. A recent systematic study found that no sonographic findings have consistently high sensitivity to clinical outcomes to be used as a single criterion for ending cardiac arrest resuscitation.10 Although some findings show a higher range of sensitivity and/or specifications, Studies studies examining the use of ultrasound care points during cardiac arrest showed varying results and there were many heterogenes between studies in terms of time and ultrasound use of care points as well as inconsistent definitions and terms in terms of heart movement. Further there is little research examining the interrater reliability of ultrasound discovery during cardiac arrest.11.12 In addition, see Adjung to CPR for ultrasound in addition to CPR. No studies have found that specific examination of ETCO2 use in cardiac arrest patients without advanced airways. It is not known whether the value of ETCO2 during the ventilation of the bag mask is as reliable as those with advanced airways in place. Due to the lack of evidence, there is nothing to support using any etco2 cutoff value to about TOR in unereafiable patients. Recommendations 1, 2, 3, and 5 supported by EPF 2020 for BLS and ALS.13,14 Recommendations 4 and 6 last received formal evidence review in 6 Advanced Techniques and Devices for Resuscitation 6.1Advanced Airway Placement Identification of Airway management during cardiac arrest usually begins with basic strategies such as ventilation of bag masks. In addition, it may be useful for suppliers to master advanced airway strategies as well as a second (backup) strategy to use if they are unable to establish first-choice airway adjaculation. Because the placement of advanced airways can lead to chest compression disorders, malposition devices, or unwanted hyperventilation, suppliers should carefully weigh these risks to potential benefits of advanced airways. A 2019 focus update on ACLS guidelines addresses the use of advanced airways in cardiac arrest and states that either bag mask ventilation or advanced airway strategies can be considered during CPR for adult cardiac arrest in any advanced airways and ventilation of bag masks heavily dependent on the set of skills and experience of suppliers (Figure 7). Therefore, the ultimate decision of consumption, type, and time of advanced airways will require consideration of various characteristics of patients and suppliers who are not easily defined in global recommendations. An important consideration to determine airway management strategies is the skills and experience of supplier airway management, regular retraining for suppliers, and continuous quality improvement to minimize airway management complications. Recommendation of Advanced Air Intervention During Cardiac Arrest COR LOE Proposes 2b B-R 1. Whether ventilated bag masks or advanced airway strategies can be considered during CPR for adult cardiac arrest in any settings depending on the condition and skill set of suppliers. Recommendations of The Large RCT Support Text in the OHCA compare bag mask ventilation with endotracheal intubation (ETI) in the doctor-based EMS system showing no significant benefits for either techniques for survival or 28-day survival with encouraging neurological outcomes.2 ETI success rates in this study are 8%, suggests a fairly optimal environment for potential ETI success as an intervention. Further research is needed to determine equality or prominence between 2 approaches to acute airway management. This proposal is supported by a 2019 focus update on the ACLS.1 Recommendations-Specific Text Support 2, and 3. An RCT in OHCA compares SGA (with iGel) to ETI in non-doctor-based EMS systems (ETI success, 69%) did not find a difference in survival or survival with encouraging neurological outcomes at the hospital discharged.3 second RCT in OHCA compared the SGA (with laryngeal tubes) with ETI in the non-medical EMS system (ETI success, 52%) find both better survival improved hospitalisation and survival for hospital clearance with good neurological outcomes in SGA-managed patients.4 These results are challenging for contextuality as they are allowed for supplier irregularities from protocols based on clinical considerations. In addition, the exact threshold for high or low tracheal intubation success rates has not been identified, although guidance can be taken from existing clinical trials. Therefore, it is difficult to understand the potential benefits (or harm), each individual, which prompts the decision to put certain advanced airway devices. Decisions on the placement of advanced airways require an understanding of the characteristics of patients and suppliers that are not easily defined in global proposals. Because of the paucity of studies on airway management advanced for IHCA, IHCA's proposals were extradited from OHCA data. Based on these issues, there is a need for further research specifically on the interface between patient factors and experience, training, tools, and supplier skills. Given these reasons, the recommendations for the SGA in priorities to the ETI would be premature. The proposal is supported by a 2019 update focused on the ACLS.1 Proposal guidelines for Advanced Air Placement Consideration 1 B-NR 1. Frequent experience or regular retraining is recommended for providers who perform endotracheal intubation. 1 C-LD 2. If the placement of advanced airways will interfere with chest compression, the supplier may consider delaying airways insertion until the patient fails to respond to early CPR and attempts at defibrillation or obtaining the ROSC. 1 C-LD 3. Continuous wave kapnagraphy is recommended in addition to clinical assessment as the most reliable method of verifying and monitoring the correct placement of endotracheal tubes. 1 C-EO 4. EMS systems that perform prehospital intubation should provide a continuous quality improvement program to minimize complications and track the entire supraglotic airways and the success rate of endotracheal tube placements. Specific Support Text Recommendations To maintain supplier skills from early training, regular reinsuring is important.5.6 However, future research needs to address certain types, volumes, and duration between training experiences. Although advanced airways can be placed without disturbing chest compression,7 unfortunately, such disruptions still occur. Therefore, suppliers should weigh the potential benefits of advanced airways with the benefit of maintaining a high chest compression breakdown.8-10 In small clinical trials and some observational studies, Wave form capnography is 100% specifically to verify the position of the endotracheal tube during cardiac capture.11-13 Sensitivity of the wave form decreases after prolonged cardiac arrest.11-13 The use of wave form capnography to assess the placement of other advanced airways (for example , Combitube, laryngeal mask airways) have not been studied. Rationale for tracking overall success rates for the ETI is to make the right decision on the fact that the practice should justify the ETI, move towards the SGA, or simply use the masks of the for patients in cardiac arrest; suggestions will vary depending on the overall success rate in a particular system. This recommendation is supported by updates focused on the guidelines ACLS 2019.1 6.2 Techniques and CPR Devices Introduction Many alternatives and advenges to conventional CPR have been developed. These include mechanical CPR, impedance threshold devices (ITD), active compression CPR (ACD), and connected stomach compression CPR. Most of these techniques and devices require specialized equipment and training. Mechanical

CPR devices convey automatic chest compression, thereingly eliminating the need for manual chest compression. There are 2 different types of mechanical CPR devices: load distribution compression bands that compress the entire thorax circumference and pneumatic piston device that compresses the chest in the direction of anteroposterior. A recent systematic review of 11 RCTs (in total moderate to low certainty of evidence) found no evidence of better survival with good neurological outcomes with mechanical CPR compared to manual CPR either OHCA or IHCA.1 Given the perceived logistics advantages associated with limited staff and safety during patient transport, mechanically ACD-CPR is done using a portable device with a suction cup used on midsternum, actively lifting the chest during decompression, thereatingly increasing the negative intrathoracic pressures produced by the recoil chest and increasing the return of venous and heart output during the next chest compression. ITD is a sensitive pressure valve attached to advanced airways or face masks that limit air entry into the lungs during the CPR decompression phase, increasing the negative intrathoracic pressure produced during chest wall recoil and increases venous returns and heart output during CPR. There are many alternative CPR techniques used, and many are not proven. For example, there is insufficient evidence of cardiac arrest bonding with the entry of CPR heads-ups to provide recommendations on its use.2 Further investigation in this and other alternative CPR techniques best explored in the context of formal controlled clinical research proposals for Formal Metchanical CPR Devices COR LOE Proposition 2b-LD. The use of mechanical CPR devices can be considered in certain settings in which high-quality manual compression delivery may be challenging or dangerous for suppliers, as long as rescuers limit disruption in CPR during device usage and removal. 3: No Benefit B-R 2. Routine use of mechanical CPR devices is not recommended. Specific Support Text Suggestions and 2. Mechanical CPR device studies show no benefit when compared to manual CPR, with recommendations of more severe neurological outcomes in some studies. ASPIRE trial (1071 patients), use of load distributor band devices are associated with the same possibility of living for hospital clearance (adjusted odds ratio [aOR], 0.56; CI, 0.31–1.00; P=0.06), and worse survival with good neurological outcomes (3.1% compared to 7.5%; P=0.006), compared with CPR.3 manual In CIRC trial (n=4231), the use of load-CPR distribution bands resulted in statistical survival rates equivalent to hospital clearance (aOR, 1.06; CI, 0.83–1.37) and survival with good neurological outcomes (aOR, 0.80; CI, 0.47–1.37).4 In paraMEDIC trials (n=4470), the use of mechanical piston devices produces the same 30-day survival rate (aOR, 0.86; CI, 0.64–1.15), and worse survival with good neurological outcomes (aOR, 0.72; CI, 0.52–0.99), compared with CPR.5 manual In LINC trials (n=2589), survival with good neurological outcomes was similar in both groups (8.3% compared to 7.8%; risk differences, 0.55%; 95% CI, –1.5% to 2.6%).6 Acknowledge this data, the use of mechanical CPR devices by trained personnel can be beneficial in settings where high-quality manual compression is unlikely or may cause risk to staff (i.e. , limited staff, mobile ambulances, angiographic suites, prolonged resuscitation, or with concerns for the exposure of infectious diseases). This topic was last received an official evidence review in 2015.7 Proposal for Active Compression CPR And Impedance Threshold Device COR LOE Proposed 2b B-NR 1. The effectiveness of active compression-decompression CPR is uncertain. Active compression CPR may be considered for use when suppliers are adequately trained and monitored. 2b C-LD 2. The combination of active compression CPR and impedance threshold devices may be reasonable in settings with available equipment and trained personnel. 3: No Benefit A 3. Routine use of impedans threshold devices in addition to current conventional CPR is not recommended. Recommendations Text Support A 2013 Cochrane Review 10 attempt to compare ACD-CPR with standard CPR found no difference in the functioning of death and neurology in adults with OHCA or IHCA.8 Important additional considerations with this modality is that increased fatigue of the rescue, which can affect the overall quality of CPR. ACD-CPR and ITD can act synergistically to increase venous returns during chest decompression and increase blood flow to vital organs during CPR. ResQTrial points out that ACD plus ITD is associated with better survival for hospital emissions with encouraging neurological functions for OHCA compared to standard CPR, although this study is limited by blind shortages, different CPR feedback elements between the study weapons (i.e., cointervention), lack of CPR quality assessment, and early TOR.9 CPR and Emergency Cardiovascular Care7 evaluate this topic and state that despite the large RCT its use, additional attempts are needed to verify results due to limitations of the study are mentioned. Therefore, ACD-CPR plus ITD is not recommended in previous versions of the AHA Guidelines. However, in settings where equipment and trained personnel are available, ACD-CPR and ITD can be an alternative to standard CPR. In the PRIMED study (n=8178), the use of ITD (as opposed to sham devices) did not significantly increase survival to hospital discharge or survival with good neurological function in patients with OHCA.11 Despite the addition of post-hoc analysis of PRIMED trials for ITD,12 itD routine use in addition This topic was last received an official evidence review in 2015.7 Recommendations for Alternative CPR Techniques COR LOE Recommendation 2b B-NR 1. CPR compression of the connected stomach can be considered during resuscitation in hospital when adequate staff trained in their use are available. Specific Recommendations Text Support Interposed stomach compression CPR is a 3-savior technique that includes conventional chest compression combined with intermittent stomach compression. Dedicated rescuers providing manual stomach compression will compress the stomach between xiphoids and umbilicus during the chest compression relaxation phase. The topic was last reviewed in 2010 and identified 2 random tests, a CPR compression abdominal interposed conducted by trained rescues increasing short-term survival of13 and survival to hospital relief,14 compared to conventional CPR for adult IHCA. An adult RCT OHCA15 shows no live advantages for the abdominal interposed compression of CPR. More assessments are needed to determine the routine use of this technique. This topic was last received an official evidence review in 2010.16 6.3Extracorporeal CPR Synopsis ECPR referred to the onset of cardiopulmonary bypass during patient resuscitation in cardiac arrest. This involves insulation of large veins and arteries and the beginning of extracorporeal venoarterial circulation and oxygenation membrane (ECMO) (Figure 8). The goal of the ECPR is to support final organ confusion while potentially reversible conditions are addressed. The ECPR is a complex intervention that requires highly trained teams, specialized equipment, and multi-disciplinary support in the healthcare system. A 2019 update focused on ACLS1 guidelines addressing the use of ECPR for cardiac arrest and noting that there is insufficient evidence to recommend the use of ECPR routines in cardiac arrest. However, the ECPR could be considered if there is a potentially reversible cause of arrest that will benefit from temporary cardiorespiratory support. An important consideration is the selection of patients for the ECPR and further research is needed to determine the the most will benefit from intervention. Furthermore, the intensity of the resources needed to start and maintain the ECPR program should be considered in the context of strengthening the link in Survival Chain. Additional investigations are necessary to assess the cost effectiveness, allocation of resources, and ethics surrounding the routine use of the ECPR in resuscitation. Recommendations of Text Support No RCTs use ECPR for OHCA or IHCA. Fifteen observational studies have been identified for OHCA that vary in entry criteria, ECPR settings, and study design, with the majority of studies reporting better neurological outcomes associated with the use of ECPR.2 For ECPR in the hospital environment, all studies have been assessed as having a very serious bias risk (mainly due to confounding) and the overall certainty of evidence is rated as very low for all outcomes.2 In 3 studies, The ECPR is not associated with beneficial effects for short-term or long-term neurological outcomes, 3-5 while 1 study6 reports related to short-term and long-term although many studies reported good results with the use of the ECPR, the vast majority of studies were from single centers with various entry criteria and settings, with the decision to implement the ECPR made on a case-by-case basis. Although there is currently no evidence to clearly determine what should be selective patients, most of the analyzed studies include younger patients with less comorbidities. More clear data is needed from higher methodological quality studies, including randomized tests. The recommendation was supported by a 2019 update focusing on the guidelines of ACLS.1 7Specific Arrhythmia Management 7.1Wide-Kompleks Tachycardia Proposal for High Complex Hemodynamic Pharmacology Management Tachycardia COR LOE Proposed 2b B-NRR 1. In stable hemodynamic patients, IV adenosine can be considered for treatment and helps diagnose rhythmics when the cause of normal rhythm, monomorphic cannot be determined. 2b B-R 2. Administration of IV amiodarone, procainamide, or sotalol can be considered for the treatment of extensive tachycardia. 3: Harm B-NR 3. Verapamil cannot be administered for any vast complex tachycardia unless it is known to come from supraventricular and is not operated by an accessory pathway. 3: C-LD Harm 4. Adenosine should not be administered for unstable, unstable, unregular hemodinal, or vast polymorphic complex tachycardias. The vast Synopsis of Tachycardia is defined as a rapid rhythm (generally 150 bran/min or more when associated with arrhythmias) with a QRS period of 0.12 seconds or more. It can represent any supraventricular tachycardia (SVT), including a paroxysmal SVT caused by re-entry of atrioventricular (AV), aberrantly fibrilation atrium, atrial flutter, or tachycardia atrium ectopic. Extensive tachycardia can also be caused by any of these supraventricular arrhythmias when operated by accessory routes pre-spelled arrhythmias). Conversely, the extensive tachycardia can also be caused by VT or rapidly rapidly rhythm paced in patients with pacemaker. The initial management of complex tachycardia requires a rapid assessment of the patient's hemodynamic stability. Unstable patients need immediate electric cardioversion. If haemynamics are stable, the diagnosis of presumptive rhythm should be tried by getting a 12-lead ECG to assess the characteristics of tachycardia. This includes identifying waves P and their relationship with the QRS complex and (in the case of patients with pacemaker) pacing spikes ahead of the vast QRS. Tachycardia complex can be common or unregular and have a uniform (monomorphic) or different (polymorphic) complex QRS from beat to beat. Each of these features can also be useful in making a diagnosis of presumptive rhythm. Inregrated tachycardia with monomorphic QRS complex suggests atrial fibrillation with aberrancy, while pre-excited atrial fibrillation or polymorphic VT may be when the QRS complex changes in their configuration from beat to beat. On the other hand, common wide-width complex tachycardia can represent monomorphic VT or aberrant-operated paroxysmal SVT, ectopic atrial tachycardia, or atrial flutter. Distinguishing between the etiology of this rhythm is the key to proper selection of drugs for treatment. Although stable haemorrhydy provides an opportunity for pharmacological assessment and treatment, the need for immediate electrical cardioversion should be expected if arrhythmias prove unresponsive to these measures or rapid commegation occurs. A more detailed approach to rhythm management is found elsewhere.1-3 Recommendations Text Support Before starting empirical drug therapy, getting ECG 12-lead and/or seeking specialist consultation for diagnosis is encouraged, if any. If the usual widespread tachycardia is suspected as a paroxysmal SVT, vagal maneuvers can be considered before starting pharmacological therapy (see Common Narrow Complex Tachycardia). Adenosine is an effective short ultra-acting drug in ending regular tachycardias when caused by an AV entry Adenosine usually will not end atrial arrhythmia (such as atrial flutter or atrial tachycardia) but will intentionally slow the ventricle rate by blocking the flow of waves P via AV nodes, capable of Although ineffective in ending ventricle arrhythmia, the relatively short life effect on blood pressure makes it less likely to weaken VT monomorphy in stable hemodynamic patients. These characteristics make adenosine relatively safe to treat stable, common hemodynamics, monomorphic width complex tachycardia of unknown types4 and as an aid in rhythmic diagnosis, although its consumption is not entirely without risk.5.6 IV antiarrhythmic drugs can be considered in stable patients with complex thycardia, especially suspected as VT VT having failed adenosine. Because of their longer duration of action, antiarrhythmic agents may also be useful to prevent recurrence of complex tachycardia. Lidocaine is not included as a treatment option for extensive unintentional complex tachycardia because it is a relatively narrow-spectrum remedy that is not effective for SVT, perhaps because its kinetic properties are less effective for VT at hemodynamic rates received from amiodarone, procainamide, or sotalol is .7-10 Different, amiodarone, procainamide, and sotalol is a broader spectrum of antiarrhythmics than lidocaine and both can treat , but they can cause hypotension. Since the 2010 Guidelines, amiodarone's new branded bioequivalent formulation has been available for IV infusions with less hipotensive effects than older generic formulations.11 There have been some direct comparisons of effectiveness between amiodarones, procainamide, and sotalol itself,12 whose writing group felt was not enough to favor one of these other medications, in addition to being careful about its use in patients with long QT, amiodarone in suspected pre-excited arrhythmia , or give these medications in combination without consultation of a specialist first. Any of these drugs may also worsen the complex tachycardia, converting it to faster arrhythmias, less hemodynamic stable, or more malignant, until the availability of defibrillators is encouraged when these drugs are administered.13 Verapamil is a calcium channel blocking agents that delay the flow of AV nodes, shorten the refractory period of accessory pathways, and act as negative inotpero and vasodators. Its effects are between different mechanisms and longer survive than adenosine. Although effective for treating extensive complex tachycardia known as supraventricular origins and does not involve removal of accessory pathways, Inotropic effects and negative hypotensive verapamil may affect VT14 and accelerate pre-excited atrial fibrillation and flutter.15 Similar concerns can also be used for other drugs commonly used to treat SVTs, such as diltiazem and β-adrenergic blockers, which are not handled in these recommendations and require evidence. The combination of slow-term AV flowing nodes, shortens refractory on myocardial pathways and accessories, and the hypotensive effect makes it inappropriate to unstable hemodynamic patients and to treat unregular tachycardias and extensive polymorphics. Adenosine only slows down rhythms of illaries, such as atrial fibrillation, causing it to be inappropriate for their management. Hypotensive and refractoriness-shortening drug tissue effects can accelerate ventricular rates in polymorphic VT and, when atrip or flutter fibrilation is carried out by accessory pathways, deterioration to VF.16 Therefore, drugs are not recommended in unstable hemodynamic patients or to treat regularly or tachycardias of a spacious polymorphic complex. This topic last received an official evidence review in 2010.17 of the Hemodynamics Core Electrical Management Recommendations Broad Tachycardia COR LOE Proposition 2a C-LD 1. If pharmacological therapy is unsuccessful for the treatment of vast complex hemodinal tachycardia, cardioversion or seeking an immediate specialist consultation is reasonable. Specific Recommendations Text Support When available, specialist consultations can assist in the diagnosis and management of tachycardia complexes of treatment-refractory. Electric cardioversion can be useful either as a first line treatment or for the complex tachycardia refractory of recent drugs due to entry rhythms (such as atrial fibrillation, atrial flutter, AV entry, and VT). However, electric cardioversion may be ineffective for automatic tachycardias (such as ectopic atrial tachycardias), requires risks associated with the drug edasi, and does not prevent the recurrence of complex tachycardia. Primarily, when the QRS complex is uniform morphology, shocks synchronized to QRS are encouraged as this minimizes the risk of triggering VF by the wrong surprise during the exposed period of the heart cycle (T waves).18 Different, polymorphic complex tachycardias cannot be synchronized resistant to it due to the different characteristics of each QRS complex, and requires high energy defibrillation.19 This topic last received an official evidence review in 2010.17 7.2Torsades de Pointes Synopsis of the original ventricle tachycardia with a different configuration of the QRS complex from the beat to Beat. However, the most critical feature in polymorphic VT diagnosis and treatment is not rhythmic morphology but what is known (or suspected) about the patient's basic QT interval. Torsades de pointes is a form of polymorphic VT associated with prolonged heart rate of QT intervals when rhythm is normal and VT is absent. The risk of developing torsades increased as the corrected QT interval was greater than 500 milliseconds and accompanied by bradycardia.1 Torsades can be caused by inherited genetic abnormalities and can also be caused by medications and imbalances electrolytes that cause extending QT interval.3 Conver vT polymorphics not linked to long QT are most frequently caused by acute myocardial ischimia.4 ,5 Other potential causes including polymorphic catecholaminergic VT , genetic abnormalities in which polymorphic VTs are promised by exercise or emotion without the prolongation of QT6; Short QT syndrome, a form of polymorphic VT linked to the incredible short QT interval (QT interval is corrected less than 330-370 mignon)7.8 ; and VT dwaksitions seen in digitalist toxicity where alternative QRS complex axes shift by 180 degrees.9 support data for acute pharmacological treatment VT polymorphics, with without length The QT interval, largely based on case reports and a series of cases, as no RCTs exist. Recommendation for Electrical Treatment Polymorphic VT COR LOE Recommendation 1 B-NR 1. Defibrillation is immediately recommended for sustainable, hemodynamic VT unstable. Special Support Text Recommendations Regardless of the basic QT interval, all forms of polymorphic VT tend to be hemodynamics and electricity are unstable. They can recur recur and send spontaneously, be sustainable, or deteriorate to VF, for which electric shocks may be required. When the QRS VT complex is a uniform morphology, electric cardioversion with shock synchronized to QRS minimizes the risk of triggering VF by wrong shock during the exposed period of the heart cycle (T Waves).10 Different, VT polymorphics cannot be synchronized for sure because different characteristics of each QRS complex and require defibrillation not to be synchronized high energy.11 While effective in finishing polymorphic VT, electric shocks may not prevent its recurrence , for which pharmacological therapy is often required and the main focus of subsequent recommendations This topic was last received a review of official evidence in 2010.12 Recommendations for Pharmacological Treatment of VT-Related Polymorphics With Long QT Intervals (Torsades De Pointes) COR LOE Proposal 2b-CD. Magnesium can be considered for polymorphic VT treatments associated with long QT intervals (torsades de pointes). Suggestions Text Support Torsades de pointes usually present in recurrent patterns of self-termination, polymorphic VT haemordinates that are unstable in the context of known or suspected long QT abnormalities, often with related bradycardia. Immediate defibrillation is an optional treatment when torsades are maintained or deteriorated to VF. However, the termination of torsades by surprise does not prevent its recurrence, which requires additional measures. In a series of minor cases, IV magnesium has been effective in suppressing and preventing recurrence of torsades.13-16 Magnesium is believed to block early afterwards, which fluctuate in potential miocardous actions that can trigger VT salvos seen in torsades.17 Correct any abnormalities Torsades are not treated with antiarrhythmic drugs, which can prolong the QT interval and promote arrhythmia. When given acutely, β adrenergic blockers can also prosecute torsades by causing or aggravating bradycardia. In patients with bradycardia or paused presiding torsades, specialist consultations are best sought for additional measures such as overdrive pacing or isoproterenol,18-20 if needed. The use of magnesium in torsades de pointes was addressed by the 2010 Guidelines and updated in its 2018 focus update on ACLS,21 guidelines with an interim evidence review identifying no new information previous backup. This topic was last received in the study of official evidence in 2010.12 Reserves for The Treatment of Polymorphic Pharmacology VT Not Associated With Long QT Hose COR LOE Reserve 2b C-LD 1. IV lidocaine, amiodarone, and measures to treat myocardial ischemia may be considered to treat polymorphic VT without prolonged QT hoses. 3: No Benefit C-LD 2. We do not recommended regular use of magnesium for the treatment of polymorphic VT with regular QT hoses. Syor-Specific Vt Polymorphic Support Texts that are not associated with QT lengthening are often triggered by acute myocardial ischemia and infaction,4,5 often rapidly degenerate into VF, and are treated similarly to other ventricular arrhythmias (VT and VF). However, the embedding of polymorphic VT with defibrillation may not prevent recurrence, which often requires additional measures. No RCTs have been performed to determine the best practices for the management of polymorphic VT pharmacology. However, measures to treat myocardial ischemia (e.g., β-adrenergic barriers or coronary cruelty interventions) as well as lidocaine and amiodarone may be effective22-29 together with defibrillation when arrhythmias are maintained. β-Adrenergic blockers have also been shown to reduce the incidence of ventricular arrhythmias in acute coronary syndrome.30,31 Expert negotiations are advised when other polymorphic VT results are suspected, where the β-adrenergical and antiarrhythmics barriers may also have usefulness.6,32 This topic was last addressed by the 2010 Guidelines, with the current pack of interim evidence that identified no new information that would have changed the previous reserve. Newer diagnostic entities are estimated to lead to the assessment of future evidence of polymorphic VT. In the absence of long QT, magnesium was not shown to be effective in the polymorphic treatment of VT 13 or for benefits in the acute management of other ventricular tachyarrhythmias.16 This reserve supported by the current pack of 2018 which focused on the ACLS guidelines.21 7.3Regular Narrow-Complex Tachycardia Introduction to SVTs Management is the subject of a recent joint treatment guidelines from the AHA, The American College of Cardiology, and the Heart Rhythm Society.1 Tachycardia's narrow complex represents a variety of tachyarrhythmias derived from a litar or pedesent involving atria or AV nod. The doctor must determine whether tachycardia is a complex or extensive tachycardia and if it has an ordinary or irregular rhythm. For patients with sinus tachycardia (heart rate greater than 100/min, P wave), no special drug treatment is required, and the doctor must focus on the identification and treatment of tachycardia basic stem (fever, dehydration, pain). If the patient is present with SVT, the main goal of treatment is to quickly identify and treat patients who are unstable hemodynamics (ischemic chest pain, altered mentality, surprises, surprises, acute heart failure) or symptoms due to arrhythmia. Cardioversion or synchronized medications or both can be used to control unstable tachycardia or common symptoms of narrow complexes. The available evidence shows that there is no available difference in success or the rate of major adverse events between calcium channel blockers and adenosine.2 In patients with narrow complex tachycardia that refractory to the measures described, this may indicate a more complicated rhythm of abnormalities for which expert consultations may be advised. Recommendations for Electrical Therapy for Tachycardia COR LOE Common Complex Proposal 1 B-NR 1. Synchronized cardioversion is recommended for acute treatment in patients with unstable hemodynamic SVT. 1 B-NR 2. Synchronized cardioversion is recommended for acute treatment in patients with stable hemodynamic SVT when vagal maneuvering and pharmacological therapy are ineffective or contraindicated. Specific Support Text Suggestions and 2. Unstable hemodynamic patient management with SVT must begin with an immediate restoration of sinus rhythm through cardioversion use. Cardioversion has been shown to be safe and effective in a prejudice setting for unstable hemodinal patients with SVT who have failed to respond to vagal maneuvers and pharmacological therapy IV.3 Cardioversion advised in patients present with hypotension, acutely altered mental status, shock signs, to the even if rare, cardioversion may also be The most stable patients with SVT have a high conversion success rate of 80% to 98% with pharmacological management (for example, adenosin, diltiazem).4.5 however, if the drugs fail to restore sinus rhythms, cardioversion is safe and effective for patients who are stable after adequate sedation and anaesthesia. The proposal is supported by the 2015 ACC/AHA/HRS Guidelines for Adult Patient Management With SVT: American College of Cardiology Reports/ AHA Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. 6 Recommendations for Pharmacological Therapy for Tachycardia COR LOE Common Narrow Complex Proposal 1 B-R 1. Vagal maneuvers are recommended for acute treatment in patients with SVT at a fixed rate. 1 B-R 2. Adenosine is recommended for acute treatment in patients with SVT at a fixed rate. 2a B-R 3. IV diltiazem or verapamil can be effective for acute treatment in patients with stable hemodynamic SVT at a fixed rate. 2a C-LD 4. IV β-adrenergic treatment is reasonable for acute treatment in patients with stable hemodynamic SVT at a fixed rate. Specific Support Text Rate Success for Valsalva's maneuver in terminating SVT ranges from 19% to 54%.7 Increasing Valsalva's maneuver with passive foot increase is more effective.8 Warnings are advised when using massage in older patients given the potential thromboembolic risks. 2015 2015 The Guidelines of the College Rhythm Society, AHA, and Cardiac Rhythm are evaluated and recommended adenosine as the first line treatment for a regular SVT due to its effectiveness, very short, and profitable side effect profiles.6 Systematic Study of Cochrane 7 RCTs (622 patients) found similar conversion rates to sinus rhythms with adenosine channel blockers and no significant difference in hypotension.2 Adenosine may have a profound effect in post-heart transplant patients and can cause severe bronchospasm in asthmatics. The treatment of stable haemylynamic patients with IV diltiazem or verapamil has been shown to convert SVT into normal sinus rhythms in 64% to 98% of patients.98% patients.4.9–11 These agents are very useful in patients who cannot tolerate β-adrenergic blockers or who have SV Warnings should be taken to administer these drugs slowly to reduce the potential for hypotension.11 Diltiazem and verapamil inappropriate in setting suspected systolic heart failure.6 Evidence for β-adrenergic blockers in terminating SVT is limited. In an experiment that compares esmolol with diltiazem, diltiazem is more effective in ending SVT.5 however, β-adrenergic blockers are generally safe, and it is reasonable to use it to end SVT in stable hemodynamic patients.6 This proposal is supported by 2015 American College, AHA, and The Heart Rhythm Association's Guidelines for Adult Patient Management With SVT.6 7.4Atrial Fibrillation or Flutter With Rapid Ventricle Response Introduction Fibrillation Atrium is an SVT consisting of unorthodected atrial electrical activation and contraction of uncoordinated atrial atrials. Atrial flutter is an SVT with a macroreentrant circuit that results in rapid atrimin activation but intermittent ventricular response. These arrhythmias are common and often coexist, and the recommendations of their treatment are the same. The treatment of atrial fibrilation/flutter depends on the stability of the patient's hemodynamics as well as the previous history of arrhythmia, comorbidities, and responsiveness to medications. Hemodynamic patients are unstable and those with rate-related ischemia should receive immediate electrical cardioversion. Stable haemyphylic patients can be treated with a rate control strategy or rhythm control. Rate control is more common in emergency settings, using the administration of IV calcium channels nondihydropyridine antagonists (for example, diltiazem, verapamil) or β-adrenergic blockers (for example, metoprolol, esmolol). Although amiodarone is usually considered a rhythm control agent, it can effectively reduce ventricle rates with potential consumption in patients with congestion heart failure where β-adrenergic may not be and nondihydropyridine calcium antagonist channels are contraindicated. Long-term anoaguulation may be necessary for patients who are at risk for thromboembolic thromboembolic events at their CHA2 DS2 - VASC SCORE. Anticovilization options are beyond the scope of these guidelines. Rhythm control strategies (sometimes called chemical cardioversion) include antiarrhythmic drugs given to convert rhythms to sinuses and/or prevent fibrillation/flutter recurrence of atrials (Table 3). The selection of patients, evaluation, time, drug selection, and anticommunition for patients undergoing rhythm control are beyond the scope of these guidelines and are presented elsewhere.1.2 Patient management with preexcitation syndrome (aka Wolff-Parkinson-White) is protected in the Tachycardia Section of Broad Complex. Recommendations for Electrical Therapy for Atrial Fibrilation / Flutter COR LOE Proposition 1 C-LD 1. Hemodynamic patients are unstable with atrib fibredomy or atrip flutter with a quick ventricle response should receive electric cardioversion. 1 C-LD 2. Direct current cardioversion immediately fibrillation of new atrials in setting acute coronary syndrome is recommended for patients with hemodynamic compromises, persistent ischaemics, or inadequate rate control. 2a C-LD 3. For cardioversion synchronized atrial fibrillation using biphasic energy, the initial energy of 120 to 200 J is reasonable, depending on the specific biphasic defibrillator used. 2b C-LD 4. For cardioversion synchronized flutter atrium using biphasic energy, the initial energy of 50 to 100 J may be reasonable, depending on the specific biphasic defibrillator used. Specific Support Text Suggestions and 2. Uncontrolled tachycardia can affect ventricle filling, heart output, and coronary perfuter while increasing the demand for myocardial oxygen. Although attempted medications and/or accelerated liquids may be appropriate in some cases, unstable patients or patients with persistent heart ischemia with atrial fibrillation or atrial flutter should be immediately contained.1-3 When deciding for cardioversion, one should also consider whether arrhythmia is the potential for rapid ventricle response hunting by secondary causes (for example, sepsis) should be considered and can inform an initial attempt at stabilization of hemodynamics with pharmacotherapy. There is some data addressing this strategy in hemodineal patients unstable. However, studies showing the benefits of successful cardioversion hemodynamics have been published.4.5 In addition, Hypothermic and hypophosical risks with negative inotrope consumption have been indicated even in normotensive patients.6-8 Unstable Hemodynamic patients and those with persistent heart iscopy may benefit from better haemdy status associated with sinus rhythm restoration and avoid hypotension caused by alternative pharmacological therapy. Depending on the clinical scenario, patients dedicated to atrial fibredration or atrial flutter 48 or longer is the candidate for anticoagulants. Granules Anticommunition selection is available elsewhere.2 and 4. The electricity needed to succeed cardiovert patients from atrial fibrillation or atrial flutter to sinus rhythms varies and generally less in patients with new arrhythmia, thin body habits, and when biphasic wave shocks are delivered.9-15 Obese patients may need greater energy.16 If early cardioversion is not successful Less energy is usually required for atrial flutter than atrial fibrillation.11 Energy higher 200 J or more associated with better first shock success and decreased energy delivery volume. In addition, retrospective analysis found that lower energy shocks were associated with a higher risk of VF.17 Guidelines caused by cardioversion including comparison of monophasic and biphasic waves. The proposal now focuses primarily on biphasic waves. The recommended energy levels vary with different devices, reducing the validity of general recommendations. This topic requires further research with a comprehensive systematic review to better understand the optimal dose of electricity with the current device. The assessment of the LOE writing group as A C-LD is consistent with limited evidence using modern devices and energy waves. The proposal is supported by the AHA/ACC/HRS 2014 Guidelines for Patient Management With Atrial Fibrilation: The American College of Cardiology Report/ AHA Task Force on the Practice Guidelines and Heart Rhy of the Athletic Association 2018 as well as focus updates on guidelines published in 2019.2 Recommendations for Medical Therapy for Medical Therapies for Medical Facilities for Atrial Fibrilation/Flutter IV administration of β-adrenergic blocker or nondihydropyridine calcium antagonist channel is recommended to slow the ventricle heart rate in acute settings in patients with atrial fibrilation or atrial flutter with a rapid ventricle response without preexcitation. 2a B-NR 2. IV amiodarone can be useful for rate control in critically ill patients with atrial fibrillation with a rapid ventricle response without preexcitation. 3: C-LD Harm 3. In patients with atrial fibredregies and atrial flutters in preexcitation settings, digoxin, nondihydropyridine calcium tract antagonists, β-adrenergic blockers, and IV amiodarone cannot be administered because they can increase ventrical response and cause 3: Harm C-EO 4. Calcium nondihydropyridine channel antagonists and IV blockers β-adrenergic should not be used in patients with left ventricle systolic dysfunction and disconnected heart failure as this could lead to further hemodynamic compromises. Specific Support Text Suggestions and 2. Clinical trial evidence suggests that the antagonists of the nondihydropyridine calcium channel (for example, diltiazem, adrenergic drug β (for example, esmolol, propranolol), amiodarone, and digoxin are all effective for rate control with atrial fibrilation/flutter.6-8.19-23 Calcium channel blockers may be more effective than amiodarone, and cause more hypotension.6 Digoxin rarely used in acute environments because of the slow start of the effect.1.2 Based on limited case reports and small cases series, There are fears that patients with preexcitation and atrial fibrolation or atrial flutter can develop VF in response to an accelerated ventricle response after the administration of AV nodes blocked agents such as digoxin, nondihydropyridine calcium tract antagonists, β adrenergic blockers, or IV amiodarone.24-27 In this setting, cardioversion is recommended as the most appropriate management. Because of their negative inotropic effects, nondihydropyridine calcium tract antagonists (for example, diltiazem, verapamil) can decay patients with left ventricular cystolic dysfunction and heart failure symptoms. They can be used in patients with heart failure with a breakdown of preserved mocking. β adrenergic blockers can be used in compensation patients with cardiomyopathy; However, they should be used with caution or avoidance altogether in patients with disconnected heart failure. This recommendation is based on expert consensus and rationale for pathophysiologic.2,18,28 β-Adrenergic blockers can be used in patients with chronic obstructive chone disease as some studies have shown no negative effects.29 This proposal is supported by AHA 2014, American College of Cardiology, and Heart Rhythm Society Guideline for Management of Patients With Atrial Fibrillation18 as well as guidelines focused on guidelines published in 2019.2 7.5Bradycardia Introduction Bradycardia is generally defined as a heart rate of less than 60/min. Bradycardia can be a common discovery, especially for athletes or during sleep. When bradycardia occurs secondary to pathological causes, it can lead to a decrease in heart output with the resulting hypotension and tissue hypoperfusion. Clinical manifestations of bradycardia can range from the absence of symptoms to bradycardia symptoms (bradycardia associated with acutely altered mental status, ischemic chest discomfort, acute heart failure, hypotension, or other signs of persistent shock even if the cause of bradycardia can determine the severity of the show. For example, patients with severe hypoxia and upcoming respiratory failure can suddenly develop deep bradycardia that leads to cardiac arrest if not handled immediately. On the other hand, patients developing third-degree heart blocks but otherwise good compensation may suffer relatively low blood pressure but otherwise stable. Therefore, the management of bradycardia depends on both the underlying cause and the severity of the clinical presentation. In 2018, AHA, American College of and the Heart Rhythm Society publishes a broad guideline on stable assessment and management and bradycardia.2 These guidelines focus exclusively on bradycardia symptoms in ACLS settings and maintain consistency with the 2018 guidelines. Proposal for Bradycardia COR Early Management LOE Proposition 1 C-EO 1. In patients present with bradycardia acute symptoms, evaluation and treatment of reversible causes are recommended. 2a B-NR 2. In patients with acute bradycardia linked to hemodynamic compromises, the administration of atropine is reasonable to increase heart rate. 2b C-LD 3. If bradycardia is not responsible for atropine, IV adrenergic agonists with effects accelerate rates (for example, epinephrine) or transcutaneous pacing may be effective while patients are ready to temporarily pacing survival if needed. 2b C-EO 4. Immediate pacing can be considered in unstable patients with high degree AV blocks when IV/IO access is unavailable. Bradycardia Symptom Support Text Recommendations may be caused by a number of potentially reversible or treatable causes, including structural heart disease, increased vagal tones, hypoxemia, myocardial ischaemia, or drugs.2 Bradycardia may be difficult to settle until the underlying cause is treated, makes the foundation's assessment Atropine has proven effective for bradycardia symptom treatment in both observational studies and in 1 limited RCT.3-7 If atropine is ineffective, either an alternative agent for increasing heart rate and blood pressure or transcutaneous pacing are reasonable next steps. For the medical management of periarrest patients, epinephrine has gained popularity, including IV absorption and use of push-dose administration for acute bradycardia and hypotension. Studies on push-dose epinephrine for bradycardia in particular are less, although limited data support its use for hypotension.8 Use of push-dose vasopressors requires careful attention to correct the dose. Drug errors leading to adverse effects have been reported.9 Dopamine Infusions may also increase heart rate.10 There are limited studies that compare drugs to transcutaneous pacing for bradycardia treatment. A random feasibility study in patients failed atropine versus dopamine to transcutaneous pacing and found no difference in survival for discharge.10 Whether for transcutaneous pacing attempts, epinephrine, dopamine, or other vasoactive agents will likely rely on clinician experiences and available resources. For bradycardia severe symptoms cause shock, if no IV or IO access is available, immediate transcutaneous pacing while access is being run can be carried out. A 2006 systematic review involving 7 transcutaneous pacing studies for bradycardia symptoms and arrest bradyasystolic in a prehospital atmosphere found no benefit from pacing compared to standard ACLS, although subgrope analysis of 1 attempt suggests Benefits in patients with bradycardia symptoms.11 This proposal is supported by 2018 ACC/AHA/HRS Guidelines on Assessment and Patient Management With Bradycardia and Heart Conduction Delays: Cardiology College Report / AHA Task Force America on Clinical Practice Guidelines and The Heart Rhythm Society. 2 Proposal for Transvenous Pacing for Bradycardia COR LOE Proposition 2a C-LD 1. In patients with continuous haemorrhydia refractory to medical therapy, transvenous pacing while it is reasonable to increase heart rate and improve symptoms. Specific Support Text Recommendations When bradycardia refractory to medical management and results in severe symptoms, the next reasonable step is the placement of temporary catheter pacing for transvenous pacing. Limited evidence for this intervention consists of an observational study, many of which have focused on relatively high indicators and complication rates (including blood flow infections and pneumothorax, among others).12-14 However, when the heart rate does not improve with medication and continuous shock, Transvenous pacing can increase heart rate and symptoms until more definitive treatment (correction of base cause or permanent pacemaker placement) can be implemented this recommendation is supported by the Guidelines of the American College of Cardiology 2018, AHA, and Heart Rhythm The Society on patient assessment and management with bradycardia and cardiac flow delays.2 7.6Care After Rosc Postresuscitation Care Introduction Post-heart care arrest is a critical component of the Survival chain. What defines optimal hospital care for patients with ROSC after cardiac arrest is not fully known, but there is a growing interest in identifying and optimizing practices that might improve outcomes. The systemic effects of ischaemic injuries caused by cardiac arrest and subsequent resuscitation require post-cardiac arrest care to support the affected multi-organ system. After initial stabilization, the care of critically ill posarrest patients rallied on hemodynamic support, mechanical ventilation, temperature management, diagnosis and treatment of underlying causes, diagnosis and treatment of seizures, fumigation for and treatment of infections, and the management of critical conditions of the patient. Many patients who catch the heart survivors of the initial incident will eventually die due to lifelong withdrawal of treatment in the setting of a nerve injury. The cause of this death is very prominent in those with OHCA but also frequently after IHCA.1.2 Therefore, many posarrest care focuses on reducing injuries to the brain. Possible contributors to this goal include fiber perfume pressure optimization, oxygen and carbon dioxide level management, temperature control core, and detection and treatment of seizures (Fig. 9). 9). arrests resulted in heterogeneous injuries; therefore, death can also be caused by multiorgan dysfunction or shock. Given the complexity of posarrest patients, multi-disciplinary teams with expertise in cardiac arrest care are prioritised, and the development of multi-disciplinary protocols is critical to optimizing the results of survival and neurology. Key topics in postresuscitation care were not covered in this section, but discussed later, targeted temperature management (TTM) (Targeted Temperature Management), quantity coronary intervention (PCI) in cardiac arrest (PCI After Cardiac Arrest), neuroprognostication (Neuroprognostication), and rehabilitation (Rehabilitation). Recommendations For Consideration during the Initial Postressusitation Period COR LOE Proposition 1 B-NR 1. A comprehensive, organized, multi-disciplinary care system should be implemented consistently for the treatment of post-cardiac arrest patients. 1 B-NR 2. A 12-lead ECG should be obtained as soon as possible after the ROSC to determine whether the acute ST segment height is present. 2a C-EO 3. To prevent hypoxia in adults with ROSC during the immediate posarrest period, it is reasonable to use the highest concentration of oxygen until the saturation of the arteries oxyhemoglobin or partial pressure of artery oxygen can be measured remarkably. Specific Support Text Studies Observations assessed the utility of cardiac acceptance centers indicating that a strong care system could represent a logical clinical relationship between successful resuscitation and ultimate survival.3 Although data is limited, taken along with experiences from regional approaches to other emergencies such as trauma, stroke, and st-height infa Patients with 12-lead identification of myocardial infaction st-segment (STEMI) should have coronary anography for possible PCI, highlighting the importance of obtaining the ECG for diagnostic purposes.4 However, Various studies have reported that the absence of ST-segment height does not rule out coronary lesions that can intervene.5-7 Some RCTs have compared the oxygen administration's titrated approach to the approach of administering 100% oxygen in the first 1 to 2 hours after the ROSC.8-10 All this carried out prejudice settings. However, these tests are only titrated oxygen once saturated oxygen can be measured with pulse oxymeter. No studies have investigated oxygen titration in patients whose oxygen saturation (by pulse oxymeter) or semi-oxygen pressure in the blood (by arterial blood gas) cannot be measured. Recommendations for administering 100% oxygen until the measurement of this important sign is possible based on physiology and expert opinion that hypoxia can worsen the damage of the final organ and should be avoided. Recommendation 1 supported Updates focused on the ACLS.3 Recommendation 2 guidelines last received an official evidence review in 2015.4 Recommendation 3 supported by CoSTR 2020 for ALS.11 Blood pressure management recommendations after the ROSC COR LOE Recommendation 2a B-NR 1. It is best to avoid hypotension by maintaining sicsollic blood pressure of at least 90 mm Hg and mean arterial pressure of at least 65 mm Hg during the period of postresuscitation. Hypotension Text-Specific Recommendations can worsen the brain and other organ injuries after cardiac arrest by reducing oxygen transmission to tissues. The optimal PETA target after the ROSC, however, is unclear. The topic was previously reviewed by ILCOR in 2015.12 and a detailed evidence update was conducted by the Australian and New Zealand Resuscitation Council on behalf of ILCOR for 2020.11 Some Observational studies have found that postresuscitation hypotension is associated with worse survival and neurological outcomes.13-19 A study found no association between higher PETA during treatment and TTM results , although the current shock of admission is associated with poor results.20 Hypotension definition varies between studies, with systolic blood pressure of 90 mm Hg and MAP 65 mm Hg commonly used. Two RCT conducted since 2015 compared to low blood pressure targets (standard care or MAP greater than 65 mm Hg in one study and MAP 65-75 mm Hg on the other side) with higher targets (MAP 85-100 in one study and MAP 80-100 mm Hg in another).21,22 Both studies failed to detect any differences in survival or survival with encouraging neurological outcomes , although insane studies are not appropriately enforced for this outcome. An experiment has found an increase in cerebral oxygen with a higher MAP,21 which is a proposed mechanism for the effects of higher MAP benefits in hypoxic ischaemic encephalopathy. A recent observational study compared results in patients with MAP 70 to 90 mm Hg to those with MAPs larger than 90 mm Hg also found that higher PETA was associated with better neurological outcomes.23 Although some of these data suggested targeting MAPs of 80 mm Hg or higher in those at risk for cardiac neurological injuries may be beneficial , this remains undeterred. The recommendations are supported by the Updated Guidelines 24 2015 and the 2020.11 evidence updates of proposals for Oxygenation and Ventilation After ROSC COR LOE Proposed 1 B-NR 1. We recommend avoiding hypoxemia in all patients who remain comatose after ROSC. 2b B-R 2. Once a reliable measurement of periperal blood oxygen saturation is available, avoiding hyperoxemia by typing in inspired oxygen breakdown to target oxygen saturation 92% to 98% may be reasonable on which remains comatose after the ROSC. 2b B-R 3. Maintaining partial pressure of carbon dioxide arteries (Paco2) in the usual physiological range (generally 35-45 mm Hg) may be reasonable in patients who remain comatose after ROSC. Recommendations of Recommendations Text In a systematic study of ILCOR 2020.11 observational studies reported that hypoxemia after circulation returns were associated with worse outcomes.25 This was not seen in other studies,26-28 and all high-risk biased studies. Therefore, these recommendations are based primarily on the physiological rationale that hypoxia increases the risk of final organ damage, and the fact that hypoxemia is the best surrogate available for hypoxia. There are some physiological and preclinical data for hyperoxemia leading to increased inflammation and exaceraging brain injuries in posarrest.29 A 2020 ILCOR systematic study11 identifies 5 meddling RCTs compare titrated or lower oxygen administration strategies with regular care or higher oxygen administration strategies in posarrest patients: 3 in prehospital environments and 2 in ICU settings.8-10,30,31 Overall , these tests found no difference in clinical outcomes, but all were less leasing for these outcomes. Large RCT recently compared regular care with aggressive eggplant hyperoxemia in mechanical ventilation critical patients and found no difference between groups in overall cohorts but increased survival on the arm intervened in subgroups 164 patient posts.32 Observation data is inconsistent and very limited by confounding.11 Three RCTs on this topic under progress (NCT03138005, NCT03653325, NCT03141099). The proposed range of 92% to 98% is meant as a practical estimate of the normal range. Two RCTs compare strategies targeting Highnormal Paco2 (44-46 mmHg) with one targeting low normal Paco2 (33–35 mmHg)31 and hi strategy targets Moderatecapnia (Paco2 50- 55 mmHg) compared to normocapnia (Paco2 35-45 mmHg).33 No attempt finds a difference in any clinical outcome. Results across

6 observational studies were inconsistent, and all studies were limited by the risk of bias.25,34-38 There was a huge continuous RCT addressing this question (NCT03114033). This recommendation is supported by CoSTR 2020 for ALS.11 Recommendations for Seizure Diagnosis and Management COR LOE Proposed 1 C-LD 1. We recommend clinically significant seizure treatment in the arrest of post-heart adults of survivors. 1 C-LD 2. We recommend immediately doing and interpreting electromencephalograms (EEG) for seizure diagnosis in all comatose patients after the ROSC. 2b C-LD 3. Non-convulsive seizure treatments (diagnosed by EEG only) can be considered. 2b C-LD 4. The same anticonvulsant regimen used for seizure treatments caused by other etiology can be considered for seizures detected after cardiac arrest. 3: No Benefit B-R 5. Prophylaxis seizure in the arrest of post-hearted adults of victims is not recommended. Recommendations of Support Text A 2020 ILCOR systematic study11 There are definitely no controlled studies that compare seizure treatment without the treatment of seizures in this population. Despite lack of evidence, untreated Significant seizure activity is considered potentially harmful to the brain, and seizure treatment is recommended in other settings39 and may also be warranted after cardiac arrest. The writing group acknowledged there was no direct evidence that the EEG to detect unconvulsive seizures increased yields. This recommendation is based on the fact that non-convulsive seizures are common in posarrest patients and the presence of seizures may be progressively important, although whether unconvulsive seizure treatment affects the outcome in these settings remains uncertain. Systematic reviews of ILCOR conducted for 2020 do not specifically address the time and methods of obtaining EEGs in posarrest patients who are still unscpulous. Data on the relative benefits of continuous versus intermittent EEG are limited. A study found there was no difference in survival with good nerve results at 3 months in patients monitored with routine (one to two 20 minutes EEGs over 24 hours) compared to continuing (for 18-24 hours) Unconvulsive seizures were common after cardiac arrest. Whether the treatment of seizure activity on EEG is not associated with seizures that are clinically affecting the outcome is currently unknown. A random trial investigating this question is progressing (NCT02056236). CoSTR 2020 recommends that seizures be treated when diagnosed in posarrest.11 No particular agent is recommended. However, CoSTR described 2 retrospective studies suggesting valproate, levetiracetam, and phosphenytoin can all be effective, with phosphenytoin found to be linked to more hypotension in 1 study.41,41 41 42 Common sedatives such as propofol and midazolam were also found to be effective in suppressing seizure activities after cardiac arrest.45 A 2020 systematic review of ILCOR 11 identified 2 RCTs comparing seizure prophylaxis without seizure prophylaxis in comatosecas ,47 Not learning to find any difference in convulsions or survival with encouraging neurological outcomes between groups. The recommendation was supported by CoSTR 2020 for ALS.11 Recommendations Text Support A 2020 ILCOR systematic review of no controlled studies that compared seizure treatment without seizure treatment in this population. 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The effectiveness of agents to reduce neurological injuries in patients who remain comatose after ROSC is uncertain. 2b B-R 4. The use of steroid routines for patients with shock after ROSC is volatile. Recommendations text One small RCT Support from 2007,48 found no difference in survival between tight and medium glucose control. In the absence of other evidence dedicated to cardiac arrest, it seems reasonable to manage blood glucose levels in posarrest patients with the same approach applied to the general critical population, i.e. using insulin therapy when needed to maintain blood glucose 150 to 180 mg/dL.49 Systematic study of ILCOR 2020 found 2 RCTs and a small number of observational studies assessed the effects of prophylactic antibio Naski on outcomes in postal patientsrest.11,50 RCTs found no difference in survival or nerve outcomes.51.52 One RCT51 found lower inflammation of the early lungs in those receiving prophylactic antibiotics, but this did not translate to in other results. When data from 2 RCTs is collected, there is no difference in the entire infection.51.52 The last neuroprotective agent topics reviewed in 2010. Various agents, including magnesium, coenzyme Q10 (ubiquinol), exanatide, gas xenon, methylphenidate, and amantadine, have been considered as possible agents to either reduce nerve injuries or facilitate the rise of patients. The work has been largely observational,53-57 although random testing was carried out on Q10 coenzyme, xenon gas, and exanatide.58-60 Small trials on the effects of Q10 coenzyme reported better survival on those receiving coenzyme Q10, but there was no significant difference in the outcome of the encouraging neurology and these findings have yet to be confirmed.58 An additional coenzyme Q10 trial has yet to be completed but the results have not yet been completed but the results have not yet been confirmed.58 An additional coenzyme Q10 trial has yet to be completed but the results have not yet been confirmed.58 An additional coenzyme Q10 trial has yet to be completed but the results have not yet been completed but the results have not yet been confirmed.58 An additional coenzyme Q10 trial has yet to be completed but the results have not yet been completed but the results have not yet been confirmed.58 An additional coenzyme Q10 trial has yet to be completed but the results have not yet been completed but the results have not yet been confirmed.58 One additional coenzyme Q10 trial has yet to be completed but the results have not yet been completed but the results have not yet been confirmed.58 An additional coenzyme Q10 trial None of the other studies identified have been able to show differences in any clinical outcomes using any agent studied. Since this topic was last updated in detail in 2015, at least 2 random tests have been settled on the effects of steroids on shock and other outcomes after the ROSC, only 1 have been published so far.61 In this study, shock reversals and other outcomes do not differ between groups. A large retrospective observation study found that steroid use after cardiac arrest was associated with survival.62 Steroid use for septic shock was widely assessed, with a recent trial of more than 1200 patients found better survival in those treated with steroids.63 Trials enrolled 3800 patients found no death benefits, although time for emissions from the ICU and time to surprise reversals both shorter in the steroids group.64 Taken together , there is no definitive evidence of the benefits of steroids after the ROSC. However, data in sepsis suggests that some patients with severe shocks can benefit from steroids and that incidents of sepsis and cardiac arrest are essential to consider. The final recommendation was received a formal evidence review in 2010 and supported by the Insulin Infusion Usage Guidelines for Hyperglycemia Management in Critically Ill Patients from the Critical Care Medicine Association.49 Recommendation 2 supported by 2 CoSTR for ALS.11 Recommendations 3 and 4 last received formal evidence checks in 2015.24 7.7 Introduction of TTM Temperature Management between 32°C and 36°C for at least 24 hours recommended for all heart rhythms in both OHCA and IHCA. Various randomized tests were conducted in various TTM domains and were summarised in a systematic review published in 2015.1 After the 2015 proposal, additional randomized attempts have rated TTM for inevitable rhythms as well as the duration of TTM. Most have been reviewed in evidence updates provided in KOSR 2020 for ALS.2 Many in permanent TTM topics, including whether the temperature should vary based on the characteristics of the patient, how long TTM should be maintained, and how quickly it should start. Latest systematic On several aspects of this important topic are needed once the clinical trials in progress have been completed. Recommendation for Recommendation for TTM COR LOE 1 B-R 1. We recommend TTM for adults who do not follow the instructions after ROSC from OHCA with any initial rhythm. 1 B-R 2. We recommend TTM for adults who don't follow the instructions after ROSC from IHCA with an inevitable initial rhythm. 1 B-NR 3. We recommend TTM for adults who do not follow the instructions after the ROSC from IHCA with a surprising initial rhythm. Recommendations Text Support Two RCT patients with OHCA with a surprising rhythm published in 2002 reportedly benefited from mild hypothermia when compared to no temperature management.1,3,4 More recent attempts compared temperatures targets of 33°C to 37°C in patients (IHCA and OHCA) with undetected initial rhythms also found better results in those treated with a temperature of 33°C.5 Large trials were conducting TTM tests compared to normothermia (NCT03114033). RCT published in 2019 compared to TTM at 33°C to 37°C for patients who did not follow the instructions after the ROSC from cardiac arrest with an undetectable initial rhythm. Survival with favorable neurological outcomes (Cerebral Performance Category 1-2) was higher in groups treated with 33°C.5 This trial included both OHCA and IHCA and was the first randomized attempt at TTM after cardiac arrest to include IHCA patients. In the subgroup analysis, TTM's benefits did not appear to be significantly different by the IHCA/OHCA subgroup. No TTM RCTs have included IHCA patients with surprising initial rhythms, and these recommendations are largely based on extrapolation from OHCA studies and patient studies with initially undetected rhythms that include IHCA patients. An observational study on TTM for IHCA with any initial rhythm has reported mixed results. Two studies that included patients enrolled in AHA Get With The GuidelinesResuscitation registry reported whether there were no worse benefits or outcomes than TTM.6.7 Both capped by very low overall TTM use in registration and the lack of data on comma presence, making it difficult to determine whether TTM was indicated for IHCA patients given. This topic was last received an official evidence review in 2015.8 with evidence updates conducted for CoSTR 2020 for the ALS.2 Proposal for TTM COR LOE 1 B-R 1 Proposal Performance. We recommend choosing and maintaining a continuous temperature of between 32°C and 36°C during TTM. 2a B-NR 2. It is reasonable that TTM maintains at least 24 h after reaching the target temperature. 2b C-LD 3. It may be reasonable to actively prevent fever in comatose patients TTM. 3: No Benefit A 4. We do not recommend the use of routine use of rapid absorption of cold liquid IV for the cooling of prehospital patients after Rosc Recommendation-Specific Text Support In 2013, an attempt of more than 900 patients compared to TTM at 33°C to 36°C for patients with OHCA and anywhere rhythm, excludes unexpected asystole, and found that 33°C was not higher than 36°C.9 Newer trials compared to 33°C to 37°C for patients with ROSC after an unresolved initial rhythm and found better survival of nerve outcomes that were encouraging in the group treated with 3°C.5 There were reports of better survival of nerve yields in groups treated with 3°C.5. , with one hypothesis that some doctors interpret the entry of 36°C as the target temperature as a normothermia equivalent, or no strict temperature control.10 Systematic studies are required at the question of which target temperature is most beneficial. However, based on the available evidence, TTM at a temp between 32°C and 36°C remains a Class 1 proposal. An RCT including 355 patients found no difference in outcomes between TTM for 24 and 48 hours.11 This study may have been under authority to detect differences in clinical outcomes. An initial 2002 trial cooled patients for 123 and 24 hours4 while a 2013 trial was used 28 hours.9 A larger, adaptive clinical trials were underway investigating a variety of different hypothermia periods from 6 to 72 hours, using a target temperature of 33°C for all patients registered (NCT0421751). There is no clear best approach to rewarming after TTM, although the 0.5°C per hour protocol followed in the 2013.9 Optimal rate of rewarming, and in particular whether the slower rates were beneficial, were the knowledge gap, and at least 1 attempt was in progress (NCT02555254). Fever after THE ROSC was associated with poor nerve outcomes in patients who were not treated with TTM, although these findings were reportedly less consistent in patients treated with TTM.12-20 It has not yet been established either fever treatments linked to increased yields, but treatment or fever prevention appears to be a reasonable approach. A 2015 systematic study found that prehospital cooling with certain methods of rapid absorption of cold IV fluids was associated with higher pulmonary edema and a higher risk of back.1 Since the study, some RCTs on prehospital cooling were carried out. An attempt to compare prehospital hypothermia induction with any method (including ice packs and cold IV liquids) without prehospital cooling, and found higher acceptance at TTM hospitals in those with prespital starts. The trial found there had been no increase in adverse events in those treated with prehospital cooling.21 Other prehospital cooling methods, such as esofaj or nose devices, had also been investigated; whether the outcome that affects this is the knowledge gap. This topic was last received an official evidence review in 2015.8 with evidence updates conducted for CoSTR 2020 for ALS.2 7.8PCI After Cardiac Arrest for PCI After Cardiac Arrest COR LOE Reserve 1 B-NR 1. Coronary angography needs to be done by appearing for all cardiac arrest patients with suspected cardiac stem captured and st-segment st-segments on the ECG. 2a B-NR 2. The coronary estimate over appears it is reasonable to choose (for example, an electrical adult patient or an unstable hemodinal) whose commatosa after OHCA comes from a suspected heart but without the height of ST-segment on the ECG. 2a C-LD 3. Free from the patient's mental status, coronary anography is reasonable in all post-heart arrest patients whose coronary graphics are otherwise indicated. Coronary artery disease Synopsis (CAD) is rampant in cardiac arrest setting.1-4 Patients with cardiac arrest because the surprising rhythm has shown a very high CAD rate: up to 96% of patients with STEMI on their postresusitation of the ECG,2.5 up to 42% for patients without ST-segment height,2,5-7 and 85% of severe VF/VT hospital arrest patients have a CAD.8 CAD role in cardiac arrest with unresolved rhythms unresolved. When an important CAD is observed during the post-ROSC coronary anography, revascularization can be safely achieved in most cases.5,7.9 More, Successful PCI was associated with improved survival in various observational studies.2,6,7,10,11 Additional benefits of assessment in cardiac catheterization laboratories including the discovery of anomalous coronary anatomy, opportunities to assess the functioning of left ventricles and hemodynamic status, and potential for the insertion of anomalous circulation devices The 2015 Guidelines Update recommends coronary anography that appears for patients with ST-segment heights at post-ROSC ECG. Emerging coronary anniography and PCI have also been linked to better neurological outcomes in patients without STEMI on their post-ROSC resuscitation ECG.4.12 However, a large randomized attempt found no improvement in survival in patients being restructured from OHCA with a surprising early rhythm where no st-segment height or signs were present. It will still be tested whether patients with signs of shock benefit from coronary canography appear and PCI. Recommendations Text SpecificAtion Support Some observational studies have shown better survival of lucrative neurology when early coronary holography is performed followed by PCI in patients with cardiac arrest having STEMI.5,14-17 This led to the proposed Class 1 in Guidelines Update 2015 that was not conflicted by any other recent study. This recommendation is in line with global recommendations for all patients with STEMI. Various observational studies have shown an association between emerging coronary angiography and PCI and better neurological outcomes in patients without st-segment height.5,7,14,15,18 A meta-analysis also supports the use of early coronary anggraphy in patients without a large randomized trial st-segment found no improvement in living in patients reordered from OHCA with shocked early rhythms where there is no height of the ST segment shock signs were present.20 In addition, while coronary artery disease was found at 65% of patients undergoing coronary canopy, only 5% of patients had acute thrombotic coronary occlusions. Various RCT is underway, but the role of coronary anniography and PCI that appears in patients without ST-elevation but with fixed shock signs will be tested. The use of coronary canography in patients with haemdy or electrical instability is consistent with guidelines for non-STEMI patients.21-23 Optimal treatment of hemodydia and electrical patients stable without ST-segment height remains unclear. The area was last systematically reviewed in 2015 and requires additional systematic review after completion of active testing (NCT03119571, NCT02309151, NCT02387398, NCT02641626, NCT02750462, NCT02876458). Evidence suggests that patients with commatosa after ROSC benefit from invasive sophistication, when indicated, as indicated by patients awake.4,14,18 therefore, invasive coronary anography is reasonably free of neurological status. This topic was last received an official evidence study in 2015.24 7.9Neuroprognostication General Considerations for Neuroprognostication Introduction Of Hypoxic-ischemic Brain Injury was the leading cause of death and death in victims the survivors of the OHCA and account for a fraction but significant results after resuscitation of the IHCA.1.2 Most of the deaths caused by posarrest brain injuries were due to the active production of living treatments based on predicted poor neurological outcomes. Accurate neurological prognostics are essential to avoid the withdrawal of inappropriate treatments in patients' lives that may otherwise achieve meaningful neurological rehabilitation and also to avoid ineffective treatment when poor results are inevitable (Figure 10).3 Recommendations for General Consideration for Neuroprognostication COR LOE Proposal 1 B-NR 1. In patients who remain comatose after cardiac arrest, we recommend that neuroprognostication involve a multimodal approach and are not based on any single findings. 1 B-NR 2. In patients who remain computerized after cardiac arrest, we recommend that neuroprognostication be delayed until sufficient time has passed to ensure avoiding confounding by the effects of medications or persistent adverse examinations during the initial postinjury period. 1 C-EO 3. We recommend that teams looking after comatose heart capture victims have regular and transparent multidisciplinary discussions with substitutes about expected time courses for and uncertainty surrounding neuroprognostication. 2a B-NR 4. In patients who remain comatose after cardiac arrest, it is reasonable to perform multimodal neuroprognostication 72 hours after normothermia, although individual prognostic tests can be obtained earlier than this. The Neuroprognostication synoxis depends on interpreting the results of the diagnostic test and associating the results with Given that false-positive tests for poor neurological outcomes can lead to inappropriate withdrawal of life support from patients who would otherwise recover, the most important test feature is specification. Many of the tests being considered are subject to errors due to the effects of medications, organ dysfunction, and temperature. Furthermore, many research studies have methodological limitations including small sample size, one center design, lack of blindness, potential for self-fulfilling prophecies, and the use of revenue at hospital clearance rather than time points related to maximum recovery (usually 3-6 months after arrest).3 Because of any single method of neuroprognostication has a rate of intrinsic errors and may be subject to confounding various modalities should be used to improve decision-making Recommendations-Specific Texts The overall certainty support in evidence of neurological progressive studies is low because of bias that limits the internal validity of studies as well as reliability issues that limit their external legitimistry. Therefore, confidence in the prognostic diagnostic tests studied was also low. Neuroprognostication that uses multimodal tests felt better to predict results than relying on single test results to predict poor prognosis.3.4 Sedatives and neuromuscular blockers could be metabolized more slowly in post-heart catch patients, and injured brains may be more sensitive to depressant waste or paralysis eradication can confuse the accuracy of clinical examination.5 Prognostication of nerve rehabilitation is complex and limited by uncertainty in most cases. Contradictions in care goals between doctors and families/surrogates have been reported in more than 25% of critically ill patients.6 Lack of adequate communication are one of the important factors, and regular multidisciplinary conversations can help reduce this. In terms of operation, the time for prognostication is usually at least 5 days after the ROSC for patients treated with TTM (i.e. about 72 hours after normothermia) and should be carried out under conditions that minimize the confounding effects of sedating medications. Individual test modalities can be obtained earlier and integrated results into multimodality assessments are synthesized at least 72 hours after normothermia. In some instances, prognostication and withdrawal of life support may occur earlier because of non-reneurologic disease, brain herniation, patient goals and wishes, or clearly inevitable situations. The proposal was supported by CoSTR 2020 for ALS,4 which added the last comprehensive study of this topic conducted in 2015.7 Use of Clinical Examinations in Neuroprognostication Proposals for Clinical Examinations Neuroprognostication COR LOE Reserve 2b B-NR 1. When done with other prognostics it may be reasonable to consider the light reflexes of two-way pupils absent at 72 h or more after cardiac arrest to support poor neurological outcome prognosis in patients who remain comatose. 2b B-NR 2. When performed with other prognostic tests, it may be reasonable to consider quantitative pupils at 72 h or more after cardiac arrest to support poor neurological outcome prognosis in patients who remain comatose. 2b B-NR 3. When done with other prognostic tests, it may be reasonable to consider bilateral corneal reflexes at 72 h or more after cardiac arrest to support poor neurological outcome prognosis in patients who remain comatose. 2b B-NR 4. When performed with other prognostic tests, it may be reasonable to consider the status of myoclonus that occurs in 72 h after cardiac arrest to support poor neurological outcome prognosis. 2b B-NR 5. We recommend recording the EEG in the presence of myoclonus to determine if there is a relevant horror correlation. 3: Harm B-NR 6. The presence of accidental myoclonic movements after cardiac arrest should not be used to support poor neurological prognosis. 3: Harm B-NR 7. We recommend that the discovery of the best motor reactions at the top that either absent or extensor movements are not used alone due to predicting poor neurological outcomes in patients who remain comatose after cardiac arrest. The findings of Synopsis clinical examination were related to poor outcomes but also subjected to confounding by TTM and medications, and prior studies had methodological limitations. In addition to assessing the level of awareness and conducting basic neurological examinations, clinical examination elements may include pupils' light reflexes, pupils, corneal reflexes, myoclonus, and myoclonus status when assessed within 1 week of cardiac arrest. The systematic review of ILCOR included studies regardless of TTM status, and findings were associated with nervous results at the time of the point between hospital emissions to 12 months after the arrest.4 Quantitative pupils were automatic assessment of pupils' reactivity, measured by a reduction in the percentage of pupil size and reported levels of reactivity as Benefits this method was standard assessment Myoclonus status is usually defined as spontaneous or sound sensitive, commuting, illustrated brief jerks on both faces and limbs present most days within 24 hours after cardiac arrest.8 Myoclonus status is different from the myoclonic status of epilepticus; the status of myoclonic epilepticus is defined as the status of epilepticus with the physical manifestation of continuous myoclonic movements and is considered a subtype of epilepticus status for these guidelines. Recommendations for Supporting Text 17 study, 9-25 absent light reflexes of absent students were assessed from immediately after ROSC until 7 days after arrest predicting weak neurological results with specification specifications from 48% to 100%. Specifications vary on a time-by-time basis, with the highest specifications seen at 72 hours or so after the arrest. Three studies assessed the light of quantitative pupils reflex15,26,27 and 3 studies assessing the index of neurological pupils15,28,29 at a time between 24 to 72 hours after being arrested. Pupil reflexes absent as evaluated by quantitative pupils (that is, reflexes of reflexive quantitative pupils=0%) is an objective finding and, in 1 study of 271 patients, had high specifications for poor outcomes when assessed at 72 hours after the arrest.15 The Neurological pupil index was unspecified and may be affected by medication; Thus, the absolute neurological pupil index cutoff and certain thresholds that predict poor prognosis are unknown.15,28,29 Eleven observational studies of observation9–11,14,14,1 16,17,19,21,22,30,31 assessed the absence of corneal reflexes at the time of the point between shortly after the ROSC to 7 days after being arrested. Poor yield specifications were between 25% to 100% and an increase in the study assessed corneal reflex at 72 hours or more after capture (between 89% to 100%). As with the findings of other inspections, corneal reflexes are subject to coconination by drugs, and some specific studies assess the potential effects of waste drugs. In 2 studies involving 347 patients,21,32 attendance of myoclonus status within 72 hours predicted poor neurological outcomes from hospital discharge to 6 months, with specifications rangeing from 97% to 100%. Getting an EEG in myoclonus status is essential for rejecting basic aistal activity. In addition, myoclonus status may have an uncaly ictal EEG correlation but may have prognostic meanings, and additional research is needed to outline this pattern. Some EEG-related status patterns of myoclonus may have poor prognosis, but there may also be more benign subtypes of myoclonus with EEG associating.33,34 Six observational studies of 16,19,30,35-37 assessing the presence of myoclonus within 96 hours after being caught with specifications for weak results between 77.8% to 97.4%. There are methodological limitations in all studies, including lack of standard definition, lack of blindness, incomplete data about EEG attrimating, and the inability to distinguish myoclonus subtypes. Literature is so accurate to make it potentially dangerous if unrepentant myoclonus is used as a prognostic marker. Historically, the best motor inspections at the top have been used as prognostic tools, with extensors or absent movements linked to poor results. Previous literature is limited by methodological concerns, including around inadequate controls for the effects of TTM and self-fulfilling drugs and prophecies, and there are more false-positive rates from acceptable (10% to 15%).7 Motor examination performance was not assessed in the systematic review of ILCOR 2020. Updates made to the Recommendations are based on concerns that motor inspections are subject to misleading and have unacceptable false-positive rates and, therefore, should not be used as a prognostic tool or as a screen for the next test. The proposal was supported by CoSTR 2020 for ALS,4 which added the last comprehensive study of this topic conducted in 2015.7 Use of Serum Biomarkers for Neuroprognostication Proposals for Serum Biomarkers for Neuroprognostication COR LOE Proposed 2b B-NR 1. When done in combination with other prognostic tests, it may be reasonable to consider the high serum value of neuronal-specific enolase (NSE) in 72 h after cardiac arrest to support poor neurological outcome prognosis in patients who remain comatose. 2b C-LD 2. The use of protein binding calcium S100 (S100B), Tau, neurofilament-based light chain, and glial fibrillary proteins in neuroprognostication are uncertain Synopsis Serum biomarkers are blood-based tests that measure the concentration of proteins commonly found in the central nervous system (CNS). These proteins are instiled into the blood in the setting of nerve injuries, and their serum levels reflect the degree of brain injury. Limitations to their prognostic utilities include diversity in testing methods based on site and laboratory, among laboratory inconsistencies at level, tendency to additional uncertainty due to hemolysis, and potential protein excise sources. The NSE and S100B are the 2 most commonly studied markers, but others are included in the study as well. The ILCOR 2020 systematic assessment study obtained serum biomarkers within the first 7 days after the arrest and concentration of serum biomarker related to nerve outcomes. Other tests of serum biomarkers, including test levels on serial time points after capture, were not assessed. A large observational cohold study investigating this and other serum biomarkers of novels and their performance as biognostic markers would be of high clinical significance. Recommendations Text Specifics Twelve observational studies rated the NSE was collected within 72 hours after being caught.10,13,21,23,38–45 The maximum level associated with poor results rangeed from 33 to 120 μg/L with specifications for weak yields of 75% to 100%. The evidence is limited due to blind shortages, laboratory inconsistencies, various thresholds needed to reach 100% specifications, and imprecision. Therefore, the NSE's absolute value cutoff that predicts poor prognosis is unknown, although very high NSE levels can be used as part of multimodal prognostics. There was research interest in evaluating serial measures throughout the first day after being caught as a prognostic tool rather than using the absolute value of a single.10,46 Three observational studies40,47,48 assessing S100B levels during the period the first hours after being arrested. Maximum levels associated with poor revolve around broadly depending on the study and time when it is measured after captivity. At the value reported to have reached a 100% specification, test sensitivity was between 2.8% to 77.6%. The evidence was limited by the number of minor studies and various thresholds across the studies needed to achieve 100% specification. The ILCOR study also evaluated 1 study each assessing protein-oriented glial fibrillary proteins44 and Tau49 and 2 studies assessing the neurofilament light chain.50.51 Given the low number of studies, LOE was low, and these serum biomarkers could not be recommended for clinical practice. The proposal was supported by CoSTR 2020 for ALS,4 which added the last comprehensive study of this topic conducted in 2015.7 Use of Electrophysiology Tests for Neuroprognostication Proposals for Electrophysiology for Neuroprognostication COR LOE Proposed 2b B-NR 1. When assessed with other prognostic tests, the prognostic value of seizures in patients who remained comatose after cardiac arrest was uncertain. 2b B-NR 2. When done with other prognostic tests, it may be reasonable to consider the continuous status of epilepticus 72 h or more after cardiac arrest to support poor neurological outcome prognosis. 2b B-NR 3. When performed with other prognostic tests, it may be reasonable to consider breaking suppression on the EEG in the absence of sedating drugs at 72 h or more after capture to support poor neurological outcome prognosis. 2b B-NR 4. When done with other prognostic tests, it may be reasonable to consider two-way absent N20 somatosensory evaking potential (SSEP) waves more than 24 h after cardiac arrest to support poor neurological result prognosis. 2b B-NR 5. When assessed with other prognostic tests after capture, the use of rhythmic periodic emissions to support poor neurological outcome prognosis cannot be ascertained. 3: No Benefit b-NR 6. We recommend that the absence of EEG reactivity within 72 h after arrest is not used alone to support poor neurological prognosis. Electroencephalography synopsis is widely used in clinical practice to assess cortical brain activity and diagnostic seizures. Its use as a promising neuroprognostic tool, but literature is limited by a number of factors: lack of terms and standard definitions, relatively small sample sizes, single central study design, blind deficiency, subjectivity in interpretation, and lack of accounting for the effects of medications. There is also inconsistencies in the definition used to explain specific findings and patterns. EEG patterns assessed in the systematic review of ILCOR 2020 included inactive EEG, epileptiform emissions, seizures, epilepticus status, broken suppression, and highly malignant EEG. Unfortunately, different studies Highly malignant EEG is differently or improper, using these findings unexpectedly. SSEPs are obtained by stimulate median nerves and for the presence of cortical N20 waves. Two-way absent N20 SSEP waves have been linked to poor prognosis, but the reliability of this modality is limited by requiring appropriate operator skills and care to prevent electrical interruption from muscle artifacts or from the ICU environment. One benefit to SSEPs is that they are subject to less interference than drugs than other modalities. Recommendations Text Support Five observational studies observations35,52-55 assess the role of electrographic and/or convulsive seizures in neuroprognostication. The study focuses on electrographic seizures, although some studies also include convulsive seizures. Although the specifications of seizures in the study included in the systematic study of ILCOR were 100%, The sensitivity of these findings was poor (0.6% to 26.8%), and other studies not included in the study found patients with posarrest seizures who had good results.36,56,57 Additional methodological concerns including selection bias for which patients undergo EEG monitoring and inconsistent definitions of seizure terms encompassing the broad pathological spectrum from one brief electrograph seizure to epilepticus refractory status, and this imprecision messaging of more limited recommendations. Six observational studies of21,55,58–61 were assessed epilepticus status within 5 days after being arrested and assessed results at a time between from hospital discharge to 6 months after being arrested. The specifications of the epilepticus status for weak yields spanned between 82.6% to 100%. Interestingly, although the status of epilepticus is a severe form of seizures, the specifications of the status of epilepticus for adverse outcomes are less than reported in the study examining seizures as a whole (as above). Additional concerns include the definition of inconsistent epilepticism status, blind shortages, and the use of epileptic status to justify the withdrawal of therapy that maintains lives leading to self-potential fulfilling prophecies. Six studies of21,35,54,59,62,63 rated suppression broke out within 120 hours of being arrested. An additional study64 divided suppression burst into sync compared to heterogeneous patterns. The definition of fracking suppression varies or is not specified. Specifications are between 90.7% to 100%, and sensitivity is 1.1% to 51%. Lack of standard definition, the potential for self-fulfilling prophecies, and the lack of controls for drug effects limits the ability to make stronger recommendations, despite a high overall specification. Additional focus on identifying subtypes of fracking suppression, such as synchronous subtypes (which seem very specific in one study), should be investigated further. Fracking suppression can be caused by medications, so it is very important that suppliers have knowledge of the potential effects on this prognostic device. Fourteen The study9,13,15–17,23,59,64-70 assessed the two-way N20 SSEP wave within 96 hours of being caught and attripting the findings with the result at the time of the eye between hospital clearances to 6 months after being arrested. Specifications are between 50% to 100%. Three studies have specifications below 100%, and additional methodological limitations include blindness and potential for self-fulfilling prophecies. While studies assessed SSEPs obtained at any time starting immediately after the arrest, there was a high likelihood of potentially misleading factors early after the arrest, leading to suggestions that SSEPs should only be obtained more than 24 hours after the arrest. The discharge on the EEG is divided into 2 types: rhythmic/periodic and nonrhythmic/periodic. Nine observational studies assessed rhythm/periodic emissions.16,45,52–54,61,63,66,69 Rhythmic/periodic emissions specifications between 66.7% to 100%, with poor sensitivity (2.4%–50.8%). The study assessed rhythmic/periodic emissions inconsistent in the definition of emissions. Most did not take into account the effects of medications, and some studies found low unacceptable specifications. However, as the time from cardiac arrest increases, the specifications of rhythmic/periodic emissions for poor results improve. There is an opportunity to develop these EEG findings as a prognostic tool. Five observational studies were conducted,53,64,66,69 rated nonrhythmic/periodic discharge. Specifications for poor results were low during the period of post-cardiac arrest assessed in the study. Ten observational studies of16,30,53–55,62,65,71-73 reported on the value of inactive EEG prognostics. Specifications were between 41.7% to 100% and below 90% in most studies. There is inconsistency in the definition and stimulus used for EEG reactivity. Studies also do not take into account the effects of temperature and medications. Therefore, the overall certainty of the evidence has been rated as very low. The proposal was supported by CoSTR 2020 for ALS,4 which added the last comprehensive study of this topic conducted in 2015.7 Use of Neuroimaging for Neuroprognostic Proposals for Neuroimaging for Neuroprognostication COR LOE Proposed 2b B-NR 1. When performed with other prognostic tests, it may be reasonable to consider the reduced white grey ratio (GWR) on brain-computerized topography (CT) after cardiac arrest to support poor neurological outcome prognosis in patients who remain comatose. 2b B-NR 2. When performed with other prognostic tests, it may be reasonable to consider a wide area of limited diffuser on the brain MRI at 2 to 7 days after cardiac arrest to support poor neurological outcome prognosis in patients who remain comatose. 2b B-NR 3. When performed with other prognostic tests, it may be reasonable to large area of reduced suction (ADC) in brain MRI at 2 to 7 days after cardiac arrest to support poor poor prognosis results in patients who remain comatose. Synopsis Neuroimaging can help after arrest for detecting and measuring structural brain injury. CT and MRI are the 2 most common modalities. On CT, brain edema can be quantified as GWR, defined as a ratio between density (measured as hounsfield units) of grey matter and white matter. The usual brain has a GWR of about 1.3, and this amount decreases with edema. At AN MRI, cytotoxic injuries can be measured as a limited diffusion-weighted imaging (DWI) and can be quantified by the ADC. DWI/ADC is a measure of sensitive injury, with normal values between 700 and 800×10^{-6} mm^2/s and reduced value with injury. The findings of CT and MRI brain injuries develop in the first few days after the arrest, so the time of the study imaging the importance is very important because it is related to prognosis. Recommendations Text Support Twelve studies23,24,31,38,66,74-79 evaluate GWR on heads of CT. GWR across the brain (average GWR) and GWR in certain areas were evaluated. Specifications were 85% to 100%, and only 1 study reported specifications that were not 100%. Many studies assessed the CT of the head obtained within the first 24 hours of being caught, although several studies including CT heads were obtained up to 72 hours after being caught. There are methodological limitations, including selection tendencies, multidisparency risks, and heterogeneous measurement techniques, such as anatomical sites and calculation methods. Thus, certain GWR thresholds that predict poor prognosis with 100% specification are unknown. In addition, the optimal time to get a CT head after the arrest to optimize GWR as a prognostic tool is unknown. Five observational studies of11,23,74,80,81 investigated DWI changes on the MRI within 5 days after being arrested. Studies qualitatively assess MRI for high signal intensity and positive findings, but the definition of positive findings varies between studies and, in some studies, only studying specific areas of the brain. Specifications are 55.7% to 100%. The definition of imprecise and short-term outcomes in some studies led to significant uncertainty over how to use DWI MRI to predict poor prognosis. In a proper setting, the burden of DWI's MRI findings or DWI MRI findings in certain areas of interest may be related to poor prognosis, but broader recommendations are unsupportable. Three observation studies82-84 investigated the ADC on MRI within 7 days after being arrested. The study was designed to determine the threshold that achieved 100% specification, although the ADC threshold and the amount of brain needed to reach the specifications varied widely. Although quantitative ADC measurements are promising tools, their extensive use is limited by feasibility concerns. In addition, slightly, and every other imaging feature, there is a heterogeneity measurement technique, including in the site and the calculation method. Certain ADCs that predicts poor prognosis is unknown. These recommendations are supported by CoSTR 2020 for ALS,4 which added the last comprehensive study of this topic conducted in 2015.7 8Recovery 8.1Recovery and Survivorship After The Proposed Cardiac Arrest for Rehabilitation and Survivorship After Cardiac Arrest CORE Proposed 1 B-NR 1. We recommend structured assessments for their anxiety, depression, posttraumatic stress, and fatigue for victims of their heart arrest and carers. 1 C-LD 2. We recommend that survivors of the cardiac arrest have multimodal rehabilitation assessment and treatment for physical, neurological, cardiopulmonary, and cognitive impairment before being discharged from the hospital. 1 C-LD 3. We recommend that survivors and their guardians receive comprehensive multi-disciplinary clearance planning, including medical and rehabilitation treatment recommendations and return to work activities/expectations. 2b C-LD 4. Debriefings and referrals for follow-up for emotional support for laying egg rescues, EMS providers, and hospital-based healthcare workers after a cardiac arrest event may be beneficial. Cardiac Synopsis arrests survivors, such as many victims of critical illnesses, often suffering from a spectrum of physical, neurological, cognitive, emotional, or social issues, some of which may not become clear until after the hospital's release. Survivorship after cardiac arrest is a journey through rehabilitation and rehabilitation and highlights the far-right impact on patients, families, healthcare partners, and communities (Figure 11).1-3 Approach to care systems for cardiac arrest including communities and healthcare response to cardiac arrest. However, with more people surviving cardiac arrest, there is a need to organize long-term emissions planning and rehabilitation care resources. Survivorship plans that address treatment, surveillance, and rehabilitation should be provided at hospital clearance to optimize the transition of care to the outpatient environment. For many patients and families, these plans and resources are perhaps most important to improve quality of life after cardiac arrest. The survivorship plan helps guide patients, carers, and primary care providers and includes summary of inpatient courses, recommended follow-up appointments, and expectations of post-discharge recovery (Figure 12). Victims of heart arrest, their families, and non-rescue families may be strong supporters for community response to cardiac arrest and patient-centered outcomes. Improving survival and rehabilitation after cardiac arrest should be a systematic priority, in line with the recommendations of treatment for surviving patients with stroke, cancer, and other critical illnesses.3-5 Recommendations-Specific Text Support About one from heart arrest victims suffer from anxiety, depression, or posttraumatic stress.6-9 Fatigue is also common and may be caused by physical, cognitive, or impairment deterioration. Family or guardians can experienced significant pressure and benefited from therapy.10-17 Cognitive impairment after cardiac arrest including difficulties with memory, attention, and executive function.18-22 Physical impairment, neurology, and cardiopulmonary are also common.3 early assessments for cardiac and physical rehabilitation, occupation, and speech language therapy may be useful to develop strategies to recover from, cope, or adapt to deterioration.3,23-25 Community Resyntheration and return to work or other activities may be slow and dependent on social support and relationships.29 Patients require driving and upon return to intimate.30,31 Rescue may experience posttraumatic anxiety or stress about providing or not providing BLS.23.32 hospital-based care providers may also experience the emotional or psychological effects of keeping patients with heart detention.34 Debriefings teams can allow a review of quality improvement) as well as recognition of natural stress related to caring for patients near death : Scientific Statement From AHA. 3 9Special State Resuscitation 9.1Accidental Hypothermia Recommendation for Hypothermia COR LOE Proposition 1 C-LD 1. Full resuscitation measures, including extracorporeal rewarming when available, are recommended for all accidental hypothermia victims without characteristics assuming they are unlikely to survive and without any fatal traumatic injuries. 1 C-EO 2. Accidental hypothermia victims should not be considered dead before rewarming has been prepared unless there are clear signs of death. 2b C-LD 3. It may be reasonable to perform defibrillation attempts according to the standard BLS algorithm simultaneously with the rewarming strategy. 2b C-LD 4. It may be reasonable to consider the administration of epinephrine during cardiac arrest according to standard ACLS algorithms simultaneously with rewarming strategies. Severe environmental hypothermia synopsis (body temperature is less than 30°C [86°F]) resulting in a significant decrease in both heart rate and respiratory rate and may make it difficult to determine whether the patient is really in cardiac arrest. A victim may also appear clinically dead due to very low body temperature effects. Life-saving procedures, including standard BLS and ACLS, are therefore crucial to continue until the patient resumes unless the victim clearly dies (for example, rigor mortis or inevitable traumatic injury). Aggressive rewarming, perhaps including invasive techniques, may be required and may require transportation to the hospital earlier than would have been done in other OHCA conditions.1 Patient-specific care that falls victim to avalanche is not included in these guidelines but is available in place Patient Support Specifications with accidental hypothermia are often present in these guidelines but are available elsewhere.2 Patient Support SpecificAtions with accidental hypothermia are often present in these guidelines but are available elsewhere.2 Suggestions-Specific Patient Support with accidental hypothermia is often present in these guidelines but is available elsewhere.2 Patient Support Specific-Specifics with accidental hypothermia are often present in these guidelines but are available elsewhere.2 Patient Support Suggestions with accidental hypothermia is often present in these guidelines but can be found elsewhere.2 Patient Support Suggestions with accidental hypothermia marked CNS and cardiovascular depression and the emergence of near death or death, requiring the need for full resuscitation measures immediately unless there are clear signs of death. Along with providing standard BLS and ALS treatments, the next steps include preventing eroding heat loss by removing wet clothing and protecting victims from further environmental exposure. For patients with severe hypothermia (less than 30°C [86°F]) with confusing rhythms, core rewarming is often used. Techniques include warm damp oxygen administration, warm IV fluid, and intrathoracic or intraperitoneal warm water lavage.3-5 For patients with severe hypothermia and cardiac arrest, Extraordinary renovations allow most rapid rewarming when available.6-11 Severe Hyperkalemia and very low core temperatures can also predict resuscitation futility.12.13 When the victim is hypothermic, Pulse and breathing

rates may be slow or difficult to detect,13,14 and the ECG may also show asystole, making it important to perform life-saving interventions until the victim is warm and/or clearly dead. Because severe hypothermia is often affected by other disorders (for example, the overdose of drugs, alcohol consumption, trauma), it is advisable to find and treat this basic condition and at the same time treat the hypothermia. The hypothermic heart may not be responsible for cardiovascular drugs, pacemaker stimulation, and defibrillation; However, the data to support this is essentially theoretical.15 If VT or VF continues after a single shock, the value delays subsequent defibrillations until the target temperature is reached volatile. There is no evidence to benefit from deviating from standard BLS protocols for defibrillation. Evidence in human effects of vasopressors or other medications during cardiac arrest in hypothermia setting consists of case-only reports.11,16,17 Systematic studies of several animal studies concluded that the use of vasopressors during hypothermic cardiac arrest increases ROSC.18 No evidence identified at the time of review including vasopressor medications, during hypothermic cardiac arrest. This topic last time received an official evidence review in 2010.1 9.2 IntroductionAnaphylaxis Between 1.6% and 5.1% of US adults have experienced anaphylaxis.1 About 200 Americans die from anaphylaxis each year, mostly from adverse reactions to medications.2 Although anaphylaxis is a multisystem disease, life-threatening manifestations most often involve respiratory tract (edema, bronchospasm) and/or circulatory system (vasodilatory shock). Epinephrine is the basis of treatment for anaphylaxis.3-5 Recommendations for Heart Of Anaphylaxis COR LOE Syor 1 C-LD 1. In secondary cardiac arrest to anaphylaxis, standard resuscitation measures and the immediate administration of epinephrine should take priority. Special Support Backup Backup No RCTs assess alternative treatment algorithms for cardiac arrest because of anaphylaxis. Evidence is limited to case reports and extrapolations from non-face cases, pathophysiological interpretations, and consensus opinions. Immediate support of airways, breathing, and blood circulation is essential in suspected anaphylactic reactions. Due to limited evidence, the secondary cardiac arrest management base to anaphylaxis is standard BLS and ACLS, including early airway management and epinephrine. There are no proven benefits from the use of antihistamines, inhaled beta agonists, and IV corticosteroids during cardiac arrest caused by anaphylaxis. Recommendations for Anaphylaxis Without Cardiac Capture COR LOE Proposition 1 C-LD 1. Epinephrine should be administered early by intramuscular injections (or autoinjector) to all patients with signs of systemic allergic reactions, especially hypotensions, swelling of airways, or breathing difficulties. 1 C-LD 2. The recommended dose of epinephrine in anaphylaxis is 0.2 to 0.5 mg (1:1000) intramuscular, which will be repeated every 5 to 15 minutes as needed. 1 C-LD 3. In patients with anaphylactic shock, close hemodynamic monitoring is recommended. 1 C-LD 4. Given the potential for rapid development of oropharyngeal or laryngeal edema, an immediate reference to health professionals with expertise in advanced airway placement, including surgical airway management, is recommended. 2a C-LD 5. When the IV line is provided, it is reasonable to consider the IV route for epinephrine in anaphylactic shock, at doses 0.05 to 0.1 mg (0.1 mg/mL, aka 1:10 000). 2a C-LD 6. IV absorption of epinephrine is a reasonable alternative to IV boluses for the treatment of anaphlaxis in patients who are not in cardiac arrest. 2b C-LD 7. IV absorption of epinephrine can be considered for shock after catch in patients with anaphylaxis. Recommendations Of Supporting All patients with anaphylaxis evidence require early treatment with epinephrine. Severe anaphylaxis can cause complete obstruction of airways and/or cardiovasculars collapsing from vasogenic shock. The administration of epinephrine may save lives.6 Intramuscular is the early route of choice due to the convenience of administration, effectiveness, and safety.7 Injections of epinephrine into the lateral aspect of the thigh produces the rapid concentration of epinephrine plasma.7 Autoinjector intramuscular epinephrine and the paediatric epinephrine autoinjector intramuscular will deliver 0.15 mg of epinephrine. Many patients will need an additional dose, with recurrence of symptoms after 5 to 15 minutes reported.8 Patients in anaphylactic shock critical pain, and cardiovascular and respiratory status can change quickly, making close monitoring of imperative.9 When anaphylaxis produces edema obstructive air management, rapid air management In some cases, emergency cricothyroidotomy or tracheostomy may be required.10,11 required.10,11 epinephrine is a suitable alternative to intramuscular administration in anaphylactic shock when IV is prepared. The dose of IV 0.05 to 0.1 mg (5% to 10% of the dose of epinephrine used routinely in cardiac arrest) was used successfully for anaphylactic shock.9 Although not specifically studied by this path in anaphylaxis, IO epinephrine may also be effective at comparison In anaphylactic shock canine models, the constant absorption of epinephrine is more effective for treating hypotension than no treatment or treatment of epinephrine bolus is.12 If the shock recurrences after early treatment, infusion IV (5-15 μg/min) can also allow caution to titration and avoid overdose Although specific data for patients with ROSC after cardiac arrest from anaphylaxis is not identified, an anaphylactic shock observation study suggests that IV infusion of epinephrine (5-15 μg/min), along with other resuscitation measures such as volume resuscitation, can be successful in the treatment of anaphylactic shock.13 Because of its role in the treatment of anaphylaxis , epinephrine is a logical option for posarrest shock treatment in this environment. This topic was last received an official evidence review in 2010.14 9.3 ArrestCardiac Due to Asthma Proposal for Cardiac Arrest Management Due to Asthma COR LOE Proposition 1 C-LD 1. For asthma patients with cardiac arrest, sudden height in peak inspirational pressure or ventilation difficulties should immediately evaluate for pneumothorax tension. 2a C-LD 2. Due to the potential effects of positive intrinsic pressure expired (auto-PEEP) and the risk of barotrauma in asthma patients with cardiac arrest, the ventilation strategy of low respiratory rates and neat volume is reasonable. 2a C-LD 3. If an auto-PEEP increase or sudden decrease in blood pressure is included in asthma that receives assisted ventilation in periarrest conditions, brief cuts from bag masks or ventilators with chest wall compression to relieve air trapping can be effective. Severe asthma attacks can lead to deep respiratory distress, carbon dioxide retention, and air trapping, causing acute respiratory acidosis and high intrathoracic pressure. Death from acute asthma has decreased in the United States, but asthma continues to be a cause of acute death for over 3500 adults a year.1.2 Patients with respiratory arrest from asthma develop acute respiratory acidosis life threatening.3 Both deep acidemia and reduced venous return to heart from high intrathoracic stress may be caused The care of any patient with cardiac arrest in the setting of asthma acute hunting begins with standard BLS. There are also no specific changes to ACLS for with cardiac arrest from asthma, despite airway management and increased significant ventilation given the possibility of causes of respiratory arrest. Acute asthma management was reviewed in detail in the 2010.4 Guidelines for 2020, the writing group focused on additional considerations of ACLS specifically for asthmatics during the immediate periarrest period. Text Support Pneumothorax strain is a rare complication of life-threatening asthma and the cause of potentially reversible arrest.5 Although usually occurs in patients receiving mechanical ventilation, cases of spontaneous respiratory patients have reported.5-7 High peak airway pressure resulting from ventilation While difficulty in narrative asthma patients in extremmis is more likely to be caused by high hyperinflation and intrathoracic stress, the assessment for tension pneumothorax remains important. Acute respiratory failure that can claim cardiac arrest in asthma patients is characterized by severe obstacles leading to air trapping. Due to limitations in incredible airflow, large tidal volume transmission at higher respiratory rates can lead to progressive air trapping and effective decrease in ventilation. Approach using lower neat volume, lower respiratory rates, and increased expiration times can minimize the risk of auto-PEEP and barotrauma.8 Breath compiling asthma patients with limited ability to sniff can lead to increased intrathoracic pressure, Decrease in venous returns and coronary perfume pressure, and cardiac arrest.9-11 This can be real due to increased difficulty in ventilators, alarms of high airway pressure on ventilators, or sudden drops in blood pressure. Brief cuts from ventilators or pauses in the ventilation of bag masks and thorax compression to help refinement can relieve hyperinflation. This topic was last received an official evidence review in 2010.4 9.4 ArrestCardiac After Proposed Cardiac Surgery for Cardiac Arrest After Cardiac Surgery COR LOE Proposed 1 B-NR 1. External chest compression should be done if emergency resternotomi is not immediately available. 1 C-LD 2. In the arrests testified by a trained provider of post-heart surgery patients, an immediate defibrillation for VF/VT should be done. CPR should start if defibrillation is unsuccessful within 1 min. In the arrests faxed by a trained supplier of post-cardiac surgery patients where pacer wires are already available, we recommend immediate pacing in asystolic or bradycardic arrests. CPR should start if the pacing is unsuccessful within 1 min. 2a B-NR 4. For patients with cardiac arrest after cardiac surgery, it is reasonable to perform an early resternotomi in an ICU that is on duty and equipped Appropriate. 2a C-LD 5. Open chest CPR can be useful if cardiac arrest develops during surgery when the chest or stomach is already open, or in the postoperative period early after cardiotoporation surgery. 2b C-LD 6. In post-heart surgery patients who are to standard resuscitation procedures, mechanical blood circulation support may be effective in increasing yield. Synopsis Cardiac Arrest occurs after 1% to 8% of cases of heart surgery.1-8 Etiologies including tachyarrhythmias such as VT or VF, bradyarrhythmias such as heart blocks or asystole, obstructive causes such as tamponade or pneumothorax, technical factors such as new valve dysfunction, inclusion of grown arteries, or bleeding. Like all patients with cardiac arrest, the immediate goal is the recovery of perfuber with CPR, the onset of ACLS, and rapid identification and correction of the cause of cardiac arrest. Unlike most other cardiac arrests, these patients usually develop cardiac arrest in highly monitored environments such as ICU, with highly trained staff available to perform rescue therapies. These guidelines are not intended to be comprehensive. A recent consensus statement on the topic was published by the Thoracic Surgeons Association.9 The Specific Support Text Case Report has rarely described damage to the heart due to the compression of the external chest.10-14 However, other case series did not report such damage,8 and external chest compression remained the only way of preparing perfume in some form. In this case, the risk of compression of the external chest is much greater than that of a certain death in the absence of perfumery. VF is a presentation rhythm in 25% to 50% of cases of cardiac arrest after heart surgery. Immediate defibrillation by trained suppliers presents different advantages in these patients, while morbidity associated with external chest compression or resternotomy can have a significant recovery effect. Sparse data has been published addressing this question. Limited data is available from defibrillator threshold tests with backup transthoracic defibrillation, using variable wave forms and energy doses.15-17 First shock success of more than 90% has been observed in most of these studies, although the results collected from 15 studies found a success rate of 78% defibrillation for the first surprise, 35% for the second, and 14% for the third shock.18 Association of Thoracic Surgeons Task Force on Resuscitation After Heart Surgery9 and the European Society for Cardio-Thoracic18 Surgery recommended 3 compiled defibrillation in 1 minute , before starting CPR. Departure from the ACLS of this standard may be warranted in post-heart surgical settings due to highly monitored settings and unique risks of compression and resternotomi. In post-heart surgery patients with asistole or bradycardic arrests in the ICU with on-site pacing indicators, pacing can be started immediately by a trained supplier. Haemdy monitoring modalities available in conjunction with manual pulse detection opportunity to confirm the myocardial capture and adequate functioning of the heart. When the pacing attempt was not immediately successful, standard ACLS including CPR was shown. This protocol is supported by society,9,18 although no data is available to support its use. No resternotomi time RCTs have been done. However, good results were observed with rapid resternotomi protocols when performed by experienced suppliers in ICU.1,4,8,19–25 Other studies were neutral or showed no benefits of resternotomi compared to standard therapy.3,6,26,27 Resternot done outside the ICU decision in poor results.1.3 The Thoracic Surgeons Association recommends that resternotomi be the standard part of the resuscitation protocol for at least 10 days after the surgery.9 No random RCTs have been performed comparing open chests with external CPR. Two minor studies have shown an increase in the effects of open chest CPR when compared to external chest compression in heart surgery patients.3.4 A series of double cases have shown potential benefits from mercaran circulation accompulsion of support including ECMO and cardiopulmonary bypass in patients refractory to standard resuscitation procedures.24,28–34 No RCT has been performed to date. This topic was last received an official evidence review in 2010.35 this proposal plus a 2017 review published by the Thoracic Surgeons Association.9 9.5Drowning Proposals to Drown COR LOE Proposition 1 C-LD 1. Rescuers should provide CPR, including rescue breathing, once the victim is unsclapulously submerged removed from the water. 1 C-LD 2. All drowning victims requiring any form of resuscitation (including rescue breathing only) should be transported to hospitals for evaluation and monitoring, despite appearing to be on alert and showing effective cardiorespiratory functions at the scene. 2b C-LD 3. Mouth-to-mouth ventilation in the water may be useful when administered by trained rescuers if it does not compromise safety. 3: No Benefit b-NR 4. Cervical spine routine stabilization without conditions suggesting spinal injuries are not recommended. Synopsis Every year, drowning is responsible for about 0.7% of deaths worldwide, or more than 500 000 deaths a year.1.2 Recent studies using data from the United States reported a survival rate of 13% after cardiac arrest related to drowning.3 People who were at high risk of drowning including children, those with seizure disorders, and those who were intoxicated with alcohol or other medications.1 Even though survival was unusual , successful resuscitation has been reported.4-9 For this reason, scene resuscitation should be initiated and the victim is transported to hospital unless there are clear signs of death. Standard BLS and ACLS are the basis of treatment, with airway management and ventilation very important due to the cause of respiratory arrest. Evidence for this proposal was last studied carefully in 2010. Recommendations of Duration Support Text and the severity of the hypoxia maintained as drowning is the single most important determinant of the outcome.10.11 With the result, once the unscpulous submerged victim is removed from the water, rescuers should provide CPR, with rescue breathing, if trained accordingly. The immediate start of rescue breathing increases the chances of victims to survive.12 Double observational assessments, especially in pediatric patients, have shown that decompensation after drowning of fresh water or salt can occur in the first 4 to 6 hours after the events.13,14 This supports transporting all victims to medical facilities for monitoring at least 4 to 6 hours if possible The immediate cause of death drowning is hypoxemia. Based on rescue training, and only if the safety of the scene can be maintained for rescuers, sometimes ventilation can be provided in water (underwater resuscitation), which can lead to better patient outcomes compared to ventilation delays until the victim is out of the water.8 Incidents of cervical spinal injuries reported in drowning victims are low 15.16 Routine stabilization of the cervical spine in the absence of a condition that suggests spinal injuries are unlikely to be beneficial to patients and possibly the necessary delays in resuscitation.16,17 This proposal incorporates the results of CoSTR ILCOR 2020, which focuses on prognostic factors in drowning.18 Otherwise, this topic last time received a formal evidence review in 2010.19 These guidelines are added to the Wilderness Medical Society. Clinical Practice Guidelines for Drowning Treatment and Prevention: Update 2019. 20 9.6 Abnormalities Proposal for Electrolite Abnormalities in Cardiac Catch COR LOE Proposition 1 C-LD 1. For cardiac arrest with known or suspected hypercaemia, in addition to standard ACLS care, calcium IV should be administered. 1 C-LD 2. For cardiotoxicity and cardiac arrest from severe hypomagnesemia, in addition to standard ACLS care, magnesium IV is recommended. 2b C-EO 3. For cardiac arrest with known or suspected hypermagnesemia, in addition to standard ACLS care, it may be reasonable to administer empirical calcium IV. 3: C-LD Harm 4. IV potent administrative bolus for cardiac arrest in suspected hypocalypia is not recommended. Synopsis Electrolyte abnormalities may cause or contribute to cardiac arrest, hinder resuscitation efforts, and affect hemodynamic rehabilitation after cardiac arrest. In addition to standard ACLS, certain interventions may save lives for cases of hypercaemia and hypermagnesemia. Hyperkalemia is usually caused by kidney failure and can precide cardiac arrhythmia and cardiac arrest. Clinical signs associated with hypercalemia severe (more than 6.5 mmol/L) including complicate paralysis, paresthesia, depressed tendon reflexes, or shortness of breath.1-3 Early signs of electrocydicics including T-escalation waves on the ECG followed by flat or absent T waves, prolonged PR PS intervals, widening QRS deep S waves, and the merger of S and T.4.5 waves As hypercalymia progresses, ECG can develop idioventricular rhythms, forming spinal wave patterns, and developing into severe asthmocolic cardiac arrest.4.5 Hypokalemia is less common but can occur in the setting of gastrointestinal or kidney losses and can lead to life-threatening ventricle arrhythmia.6–8 Severe hypermagnesemia is likely to occur in an obstetric environment in patients being treated with magnesium IV At a very high level, hypermagnesemia can lead to altered awareness, bradycardia or ventricle arrhythmias, and cardiac arrest.9,10 Hypomagnesemia can occur in the setting of gastrointestinal or malnutrition diseases, among other causes, and, when significant, can lead to both atrial and arrhythmia ventricles.11 Recommendations-Specific Support Texts Other than ACLS standard, some therapies have long been recommended to treat life-threatening hypercaemia.12 This includes the administration of IV calcium and/or bikarbonate, insulin with glucoseosa, and/or parental Calcium alhal can stabilize the myocardial insert membrane and therefore most likely to be useful during cardiac arrest and can be given by route IV or IO. The usual dose is 5 to 10 mL of 10% calcium chloride solution, or 15 to 30 mL solution 10% gluconate calcium, administered via IV or IO lines over 2 to 5 minutes.12 The use of sodium polystyrena standards (Kayexalate) is now not restored due to poor efficacy and risk of intestinal complications. Haemodialysis of cumulation in hospital environments remains the ultimate treatment for life-threatening hypercalemia. Although the magnesium IV administration has not been found to be beneficial for VF/VT in the absence of a prolonged QT, consideration of its use for cardiac arrest in patients with prolonged QT advised.13 Hypomagnesemia may cause or aggravate prolonged QT, linked to various arrhythmias, and can precide cardiac arrest.11 This provides a physiological rationale for normal level rehabilitation, although the ACLS remains the basis of treatment. Recommendations for treatment of torsades de pointes are provided in the Tachycardia Section of the Broad Complex. The administration of IV or CALCIUM IO, in the recommended dose for hypercalemia, can increase haemdy in severe magnesium toxicity, supports its use in cardiac arrest despite direct evidence of less.14 potassium-controlled administration of IV for ventricle arrhythmias because severe hypocalemia can be used, but case reports generally include potassium absorption and not bolus dose.15 Bolus dose without adverse heart effects reported in at least 1 series of minor cases of heart surgery patients where it is administered but this effectiveness for cardiac arrest is unknown, and security concerns remain.16 This topic last received official evidence in 2010.12 9.7Opioid Overdose The continuous introduction of the opioid epidemic resulted in an increase in opioid-related OHCA, Leads to about 115 deaths per day in the United States and mostly affect patients from 25 to 65 years.1-3 Initially, isolated opioid toxicity is associated with CNS and protracted respiratory depression to respiratory arrest followed by cardiac arrest. Most opioid-related deaths also involve coding various drugs or medicines and mental health comorbidities.4-7 In creating these recommendations, the writing group considers the difficulty in distinguishing opioid-related resuscitation emergencies from other causes of cardiac arrest and breathing. An opioid-related resuscitation emergency is defined by the presence of cardiac arrest, respiratory arrest, or severe life-threatening instability (such as severe CNS or respiratory depression, hypotension, or cardiac arrhythmia) suspected of being caused by opioid toxicity. In these situations, the primary care remains the initial recognition of the emergency followed by the activation of the emergency response system (Figure 13 and 14). Opioid doses slumped to cardiopulmonary arrests due to the loss of airway patentage and lack of respiratory deficiencies; therefore, dealing with airways and ventilation in periarrest patients is the highest priority. The next steps in care, including CPR performance and naloxone administration, are discussed in detail below. Additional recommendations on excessive opioid response education are provided in Part 6: Educational Sciences Resuscitation. Recommendations for Acute Management Opioid Overdose COR LOE Proposed 1 C-LD 1. For patients in respiratory arrest, rescue or ventilation of bag masks should be maintained until the return of spontaneous breathing, and standard measures of BLS and/or ACLS should continue if the spontaneous respiratory return does not occur. 1 C-EO 2. For patients known or suspected to be in cardiac arrest, in the absence of proven benefits from the use of naloxons, standard resuscitation measures should take priority over the administration of naloxsons, with a focus on high-quality CPR (compression plus ventilation). 1 C-EO 3. Respondents laying and trained should not delay activating the emergency response system while waiting for the patient's response to naloxone or other interventions. 2a B-NR 4. For patients suspected of excessive opioids who have a definite pulse but no common breathing or just gasping (i.e. respiratory arrest), in addition to providing standard BLS care and/or ACLS, it is reasonable for respondents to administer naloxons. The proposed early Management Support Text should focus on supporting airways and respiratory patients. This starts by opening air followed by a breath-rescue delivery, ideally with the use of bag-masks or blocking devices.8-10 ACLS provisions should continue if returns breathing does not occur. Because there are no studies showing improvements in patient outcomes from naloxone administration during cardiac arrest, CPR preparation should be the focus of early care.3 Naloxone can be administered along with standard ACLS care if it does not slow down high-quality CPR components. Early activation of the emergency response system is critical for patients suspected of overdose of opioids. Rescuers cannot ensure that a person's clinical condition is caused by respiratory depression caused by opioids only. This is especially true in first aid and BLS, where the determination of the presence of the pulse is unreliable.11,12 Naloxone is ineffective in other medical conditions, including excessive doses involving nonopioids and cardiac arrest of any cause. Secondly, patients who respond to naloxone administration can develop recurrent CNS and/or respiratory depression and require longer periods of observation before safe release.13-16 Twelve studies study the use of naloxone in respiratory capture, where 5 compared to intramuscular, intravenous routes, and/or intransal administration of naloxone (2 RCT,17.18 3 non-RCT19-21) and 9 assess the safety of naloxone use or naloxone use observation studies.22-30 This study reports that naloxone is safe and effective treatment of respiratory depression caused by opioids and complications Recommendations for Opioid Overdose Postsuscitation Management COR LOE Proposed 1 C-LD 1. After the return of spontaneous respiratory, patients should be observed in the healthcare environment until the risk of recurrent opioid toxicity is low and the level of awareness of patients and important signs have been normal. 2a C-LD 2. If recurrent opioid toxicity develops, a small dose of recurrence or absorption of naloxone can be beneficial. Patient Suggestions Text Support that respond to naloxon administration can develop recurrent CNS and/or respiratory depression. Although the abbreviation observation period may be sufficient for patients with fentanyl, morphine, or excessive heroin,28,30-34 longer observation periods may be required to release patients with life-threatening opioid overdoses that long act or persistently.13-15 Prehospital providers faced with the challenges of patients who refuse transportation after treatment for life-threatening overdoses are advised to follow protocols and practices Because the duration of naloxone action may be shorter than the effects of opioid respiratory depression, especially old acting formulations, repeated doses of naloxone, or naloxone infusion may be required.13-15 This proposal is supported by AHA 2020 scientific statements about OHCA.3 9.8Cardiac Pregnancy About 1 in 12 000 shipments for shipping in the United States Although it remains a rare event, the incidence has increased.2 The survival rates of mothers and fetuses/neonatals reported varying often, the best outcome for both the mother and the fetus is through successful mother resuscitation. Common causes of maternal cardiac arrest are bleeding, heart failure, amniotic fluid embolism, sepsis, aspirational pneumonitis, venous trombembolism, preeclampsia/eclampsia, and anesthesia complications.4,4,6 Literature during largely observations, and some treatment results are based primarily on pregnancy physiology and extrapolation from non-cancerous pregnancy states.9 High quality resuscitation and therapeutic interventions targeting the most likely causes of cardiac arrest are paramountly perimortem (PMCD) on or more than 20 weeks of uterine size, sometimes referred to as hysterotomy resuscitation, apparently increasing the result of the mother's cardiac arrest when quick resuscitation resulted in the ROSC (Figure 15).10–14 More, a shorter interval than capture to delivery appears to lead to better mother and neonatal results.14. 15 However, clinical decision to implement PMCD— and time with respect to the mother's cardiac — due to the diversity of team practitioner levels and training , patient factors (for example, arrest etiology, pregnancy age), and system resources. Finally, case reports and a series of cases using the ECMO in mother cardiac arrest patients reported a good mother survival.16 Cardiac arrest treatment in late pregnancy represents a major scientific divide. Recommendations for Planning and Preparation of Cardiac Arrest in Pregnancy COR LOE Proposition 1 C-LD 1. Team planning for cardiac arrest during pregnancy should be done in collaboration with obstetrics, neonatal, emergency, anesthesiology, intensive care, and cardiac arrest services. 1 C-LD 2. Because the ROSC is immediately unreachable, local resources for the delivery of perimortem cesareans should be sumvied as soon as cardiac arrest in a woman in the second half of pregnancy is recognized. 1 C-EO 3. Protocols for OHCA management during pregnancy should be developed to facilitate timely transportation to the center with the capacity to immediately perform perimortem cesarean delivery while providing continuous resuscitation. Recommendations for SupportIng Texts To ensure successful mother resuscitation, all potential stakeholders should engage in planning and training for cardiac arrest during pregnancy, including possible requirements for PMCD. Based on the same but critical interventions time, planning, simulation training and olek emergencies will assist in the confidentiality of the facility.17-21 Since the initial efforts to resuscitate the mother may be unsuccessful, preparation for the PMCD should begin early in the because lowering time to PMCD is associated with better mother and fetal yield.8 In cases of previous mother arrest, previously, directly to the ease of resuscitation of PMCD and neonatal, with the initial activation of adult resuscitation ease, obstetrics, and neonatal resuscitation forces, providing the best chance for successful results. Reserves for Cardiac Arrest Resuscitation in Pregnancy COR LOE Reserve 1 C-LD 1. Priorities for pregnant women in cardiac arrest should include the provision of high-quality CPR and the relief of aortocaval mampatan through the left lateral uterine disease. 1 C-LD 2. Because pregnant patients are more likely to hypoxia, oxygenation and airway management should take precedence over resuscitation rather than cardiac arrest during pregnancy. 1 C-EO 3. Due to a potential disorder with maternal resuscitation, fetal monitoring cannot be performed during cardiac arrest during pregnancy. 1 C-EO 4. We are monitoring the temperature management that is in place for pregnant women who remain comatose after resuscitation from cardiac arrest. 1 C-EO 5. During temperature management targeted at pregnant patients, it is suggested that the fetus is monitored continuously for bradycardia as a potential complication, and obstetric and neonatal negotiations should be sought. Syor-Specific Text Support The fat uterus can compress lower venous cava, prevent venous returns, thereorish reducing the number of strokes and cardiac output. In supine position, aortocaval compression can apply to singleton pregnancies starting at approximately 20 weeks of gestational age or when the height of funds is at or above the umbilicus paras.22 Manual left lateral uterine acid relieves aortocaval pressure in patients with hypotension (Rajah 16).23,23 airing, and oxygenation is very important in the determination of pregnancy because the increase in maternal metabolism and the capacity of rizab function decreases due to the uterus rumbling , making pregnant patients more likely to hypoxia. In addition, fetal hypoxia has been known to have adverse effects. Both of these considerations support advanced airways management for pregnant patients. Resuscitation of pregnant women, including PMCD when indicated, is the first priority as it can lead to increased survival of both women and the fetus.9 Fetal monitoring does not achieve this goal and may distract from maternal resuscitation efforts, especially defibrillation and abdominal provision for PMCD. There is no rawak test of the use of TTM while pregnant. However, there are some reports of cases of good maternal and fetal outcomes with the use of TTM after cardiac arrest.24,25 After the success of maternal resuscitation, the indeter certain fetus remains destant to the effects of hypothermia, acidosis, hypoxemia, and hypotension, all of which can be occurred in post-ROSC determination In addition, deterioration of fetal status may be a warning sign of early deterioration of the mother. Reserves for Cardiac Arrest and COR LOE Proposal 1 C-LD 1. During cardiac arrest, if the woman is pregnant with the height of funds on or above the umbilicus does not reach the ROSC with the usual resuscitation measures plus the left lateral uterine displacement manual, it is advisable to prepare to empty the uterus while resuscitation continues. 1 C-LD 2. In situations such as inevitable mother trauma or prolonged pulselessness discomfort, where the mother's resuscitation efforts are considered in vain, there is no reason to delay doing perimortem cesarean transmission in the appropriate patient. 2a C-EO 3. To achieve early delivery, preferably within 5 minutes after the time of arrest, it is reasonable to immediately prepare for the delivery of perimortem cesareans while the initial interventions of BLS and ACLS are being done. Suggestions-Specific Text Support Fat uterine transplants relieve aortocaval compression and can increase the likelihood of ROSC.10-14 In the second half of pregnancy, PMCD can be considered as part of the mother's resuscitation, regardless of the viability of the fetus.26 Early delivery associated with better mother and neonatal survival.15 In situations Early delivery of the fetus can also improve neonatal survival.26 Optimal time for PMCD's performance is unsteady and must logically vary based on the set of supplier skills and available resources as well as the arrest characteristics of patients and/or heart. A systematic review of literature assessed all reports of cardiac arrest cases during pregnancy about PMCD time, but various cases of heterogeneity and reporting bias did not allow the conclusion.15 Survival of the mother was reported up to 39 minutes after the onset of the mother's cardiac arrest.4,10,27–29 In a systematic study of the literature published 19 The median time from the mother's cardiac arrest to delivery was 9 minutes in surviving mothers and 20 minutes in unwavering mothers.15 In the same study, the median time for PMCD was 10 minutes in life and 20 minutes in unwavering neonates. The time for delivery is within 4 minutes in just 4/57 (7%) Reported cases.15 In the UK coholor study, 4 median times from collapse to PMCD were 3 minutes in women who survived compared to 12 minutes in non-flight. In the study, 24/25 babies survived when PMCD occurred within 5 minutes after the mother's cardiac arrest compared to 7/10 babies when PMCD occurred more than 5 minutes after cardiac arrest. Neonatal survival has been documented with the PMCD done up to 30 minutes after the onset of the mother's cardiac arrest.10 Specialist recommendations for time for PMCD in cardiac arrest less than 5 minutes remained an important goal, although rarely achieved.9 No evidence for a particular survival threshold at 4 minutes.8 This proposal by Cardiac Arrest during Pregnancy: Scientific Statements Of AHA9 and current pack evidence 2020.30 9.9Pulmonary Embolism Embolism for Pulmonary Embolism COR LOE Proposition 2a C-LD 1. In patients with pulmonary embolism certified as precipitant cardiac arrest, trombical, embolectomical surgery, and mechanical embolectomy are reasonable emergency treatment options. 2b C-LD 2. Thrombolysis can be considered when cardiac arrest is suspected of being caused by pulmonary embolism. Synoxis This topic was reviewed in the systematic review of ILCOR for 2020.1 PE was the cause of potentially reverse shock and cardiac arrest. The acute increase in right ventricle pressure due to pulmonary artery barriers and vasoactive mediator emissions resulted in cardiogenic shocks that might rapidly grow to cardiovascular collapse. Pe acute management is determined by the severity of the disease.2 Fulminant PE, characterized by cardiac arrest or severe hemodynamic instability, defines a massive PE subset that is the focus of this proposal. Anonymous electrical activity is a presentation rhythm in 36% to 53% of PE-related cardiac arrest, while the surprised main rhythm is the incredible systemic anticoagulant.3-5 systemic procedural is generally indicated for patients with massive and submasive PE to prevent freezing and endogenic anticoagulant support alone is not sufficient for patients with massive and submasive PE Pharmacological and mechanical therapy to reverse the entry of pulmonary arteries quickly and restore adequate pulmonary and systemic circulation has emerged as a major therapy for mass PE, includes current advanced treatment options including thrombolysis systemic, surgical or mechanical surgery embolectomy, and PR Recommendation-Specific Text Support In a systematic review of ILCOR 2020, no randomized tests were identified addressing cardiac arrest treatments caused by PE certified A fibrinolytic therapy observation study for PE suspected of having a large bias and showed mixed results in terms of improvements in yields.3,7–10 Two series of cases totaling 21 patients with PE underwent CPR undergoing embolectomy surgery reporting a 30-day survival rate of 12.5% and 7% 11,12 Series of cases of patients with pe-related cardiac arrest reported the ROSC in 6 out of 7 patients (86%) treated with mechanical thrombectomy percutaneous.13 In terms of potential adverse effects, clinical trials and some observational studies show that the risk of large bleeding in patients receiving thrombosis and CPR is relatively low.7-9 Despite the uncertainty of benefits, the risk of death from cardiac arrest exceeds the risk of bleeding from thrombism and/or mechanical or surgical risks Because there are no obvious benefits to another, thrombisiss options or surgery or mechanical thrombectomy will depend on the time and expertise available. cardiac arrest when PE is suspected but not confirmed less obvious, given that misdiagnosis can lay off at risk for interest-free bleeding. Recent evidence, however, suggests that the risk of major bleeding is not much higher in cardiac arrest patients receiving thrombolysis.8 PE is difficult to diagnose in an intra-catch environment, and when the ROSC was not obtained and PE strongly suspected, the evidence supports thrombolysis consideration.1 This proposal is supported by a systematic review of ILCOR 2020.1 9.10Toxicity: Recommendations of Benzodiazepines for Benzodiazepine Overdose COR LOE 3: Harm B-R 1. The administration of flumazenil to patients with coma that is not based on risk and is not recommended. Excessive doses of Simopsis Benzodiazepine cause CNS and respiratory depression and, especially when taken with other sedatives (for example, opioids), can cause respiratory arrest and cardiac arrest. Flumazenil, certain benzodiazepine antagonists, restores awareness, protective airway reflexes, and respiratory drives but can have significant side effects including seizures and arrhythmia.1 This risk increases in patients with benzodiazepine dependence and with coingestion of cyclic antidepressants Half life flumazenil is shorter than many benzodiazepines, requiring close monitoring after the administration of flumazenil.2 Alternatives to the flumazenil administration are breathing support with ventilation of bag masks followed by ETI and mechanical ventilation until benzodiazepine has been metabolized. Recent Meta-analysis Text Recommendations 13 RCTs (990 patients who can be assessed) found that adverse events and serious adverse events were more common in patients who randomly received flumazenil from placebo (the numbers needed to harm: 5.5 for all incidents , intermediate, aggressive behavior); Serious adverse incidents were reported to include tachycardia, supraventricular arrhythmias, premature ventricle complexes, seizures, and hypotensions. While no patient died in this clinical trial, rare cases of death associated with the flumazenil administration have been reported.3.4 Flumazenil administration to patients with accidental overdoses can provide unnecessary risks to patients, making the focus to provide best approach supportive care. This topic was last received an official evidence review in 2010.5 9.11Toxicity: β-Adrenergic Blockers and Calcium Channel Blockers Introduction β-Adrenergic recep Antagonist antagonists (β-adrenergic blockers) and L-type calcium channel antagonists (calcium tract blockers) are common antihypertensive and heart rate control drugs. Because receptors β-adrenergic regulate the activities of L.1 excess calcium tract these drugs present equally, causing hypotension life and/or bradycardia that may be refractory to standard treatment such as vasopressor infusions.2.3 For patients with refractory with refractory to standard treatment such as vasopressor infusions.2.3 For patients with refractory refractory Stability, therapical options including the administration of high-dose insulin, calcium IV, or glucagon, and consultations with medical toxicologists or regional poison centers can help determine optimal therapy. Resuscitation from cardiac arrest caused by β adrenergic blockers or calcium tract blockers excessively according to standard resuscitation guidelines. Proposal to β-Adrenergic Blocker Overdose COR LOE Proposed 2a C-LD 1. In patients with β-adrenergic overdose that are in refractory shock, the administration of high dose insulin with glucose is reasonable. 2a C-LD 2. In patients with β adrenergic blockers who are in refractory shock, the administration of IV glucagon is reasonable. 2b C-LD 3. In patients with β adrenergic blockers who are in refractory shock, calcium administration can be considered. 2b C-LD 4. In patients with β adrenergic blockers who are in the shock refractory to pharmacological therapy, ECMO may be considered. Specific Recommendations for Text Support Animal Studies, case reports, and a series of cases have reported improved heart rate and better haemodynamics after the administration of high dose insulin for β-adrenergic blockers toxicity.4-6 Common insulin doses used in this study are 1 U/kg bolus, followed by an absorption of 1 U/kg per hour trated for effect; infusion dextrose and potassium administered.2.7 No controlled studies on this topic have been identified. Although there are no controlled studies, some case reports and a series of minor cases have reported an increase in bradycardia and hypotension after the administration of glucagon.8-10 Limited animal data and rare case reports suggest a possible calcium utility to increase heart rate and hypotension in the β-adrenergic blocker toxicity.. 2 11-13 Case reports and at least 1 retrospective observational study were published on survival after the ECMO in patients present with refractory shocks from β-adrenergic blocker overdose.14.15 Evidence for ECMO for any cardiac arrest is very limited, but refractory shock from reversible causes such as drug inflammation may be a situation when the ECMO can deliver benefits. The recommendations are supported by the Guidelines of the American College Rhythm Society 2018, AHA, and heart Rhythm Society on patient assessment and management with bradycardia and cardiac conduction delays.16 Proposals for Calcium Channel Blocker Overdose COR LOE Proposition 2a C-LD 1. In patients with excess calcium channel blockers that are in refractory shock, calcium administration is reasonable. 2a C-LD 2. In patients with excessive calcium channel blockers that are in refractory shock, the administration of high dose insulin with glucose is reasonable. 2b C-LD 3. In patients with excessive calcium channel blockers that are in refractory shock, administration IV can be considered. 2b C-LD 4. In patients with excessive calcium channel barrier who are in shock refractory to pharmacological pharmacology The ECMO may be considered. Recommendations Text Specifics Support No controlled studies examined the effects of calcium IV for calcium channel toxicity blocker.16 Series of cases and case reports have reported the effectiveness of variables with low incidence of adverse effects. A systematic review states consistent benefits in animal studies but inconsistent results in human reports.17-21 A 2017 consensus statement experts recommend calcium as the first line treatment for catecholamine-refractory surprises from calcium tract blockers, Acknowledged the very low testimony to this intervention.22 Two systematic reviews have identified case reports, and human observation studies that have reported improved heart rate and better haemdy after the administration of high doses for the toxicity of calcium channel blockers.4,16,21,23,24 Like β blocks The usual dose of insulin used in the study was a 1 U/kg bolus, followed by an absorption of 1 U/kg per hour classified into clinical effects; dextrose and potassium infusions administered.2,4,7,21 Findings in both animal studies and human case reports/series cases on the effects of glucagon in toxicity of calcium channel blockers have been inconsistent, with some heart rate increase reporting and some reporting no effects.21 At least 1 retrospective study on the use of the ECMO for patients with cardiac arrest or refractory shock in drug inflammatory fixing has reported better results.14 As with all retrospective studies, the risk of bias is high because of other considerations in deciding which patients will be treated The risk of bias is high because of other considerations in determining which patients will be treated with all retrospective studies, the risk of bias is high because other considerations in deciding a consensus statement recently supported the use of the ECMO for refractory shocks from refurbishable causes such as drug toxicity.22 This proposal is supported by the American College of Cardiology 2018 , AHA, and Cardiac Rhythm Society Guidelines on patient assessment and management with bradycardia and delayed heart removal.16 9.12Toxicity: Cocaine Recommendations for Toxicity COR LOE Proposition 2a B-NR 1. For patients with high blood pressure caused by cocaine, tachycardia, agitation, or chest discomfort, benzodiazepines, alpha blockers, calcium channel blockers, nitroglycerin, and/or morphine can be beneficial. 2b C-LD 2. Although conflicting evidence exists, it may be reasonable to avoid the use of β-adrenergic drug genuine in setting cocaine toxicity. Synoxis cocaine toxicity can cause adverse effects on the cardiovascular system, including disrhythmia, high blood pressure, tachycardia and coronary artery, and heart flow delay. These effects can also claim acute coronary syndrome and stroke. Human experimental data suggests that benzodiazepines (diazepam, lorazepam), alpha blockers (phentolamine), calcium tract blockers (verapamil), morphine, and nitroglycerine are all safe and potentially beneficial in cocaine-drunk patients; no data available compares this approach.1-5 to this.1-5 Data surrounding the β-adrenergic blockers.6-8 Patients with cocaine density can deteriorate rapidly depending on the amount and time of ingestion. If cardiac arrest develops as a result of cocaine toxicity, there is no evidence to suggest irregularities from standard BLS and ALS guidelines, with certain treatment strategies used in the post-cardiac phase as needed if there is evidence of severe cardiotoxicity or neurotoxicity. Once the ROSC is reached, urgent consultation with a medical toxicologist or regional poison center is proposed. Specific Support Text Suggestions No large RCT assesses different treatment strategies for patients with acute cocaine inflammation exists. A systematic study of literature identified 5 minor trials, 3 retrospective studies, and several case reports and a series of cases with conflicting results. Some literature reported good results while others reported significant adverse events.9 Well-conducted human trials showed that the propranolol administration reduced coronary blood flow in patients with cocaine exposure.8 Despite recent systematic reviews indicates that the use of β-adrenergic barrier may be harmless,6.7 safe This topic last time received an official evidence review in 2010.10 9.13Toxicity : Local Anaesthetic Proposal for Overdose Cor LOE Local Proposed 2b C-LD 1. It may be reasonable to administer lipid IV emulsion, along with standard resuscitation care, to patients with systemic toxicity of local anaesthetics (LAST), and especially to patients with neurotoxicity premonitory or cardiac arrest due to bupivacaine toxicity. Synopthesis of local anaesthetic dosages (also known as systemic toxicity of local anaesthetics, or LAST) is a life-threatening emergency that can be present with neurotoxicity or cardiovascular fulminant collapse.1.2 The most commonly reported agents associated with LAST are bupivacaine, lidocaine, and ropivacaine.2 By definition, LAST is a special condition in which an alternative approach should be considered in addition to BLS standards and ALS. Case reports and animal data have suggested that IV lipid emulsions may be beneficial.2-5 END resulting in deep inhibition of the voltage-fenced channels (especially sodium transduction) in the cell membrane. Potential mechanism of lipid IV emulsion action includes active blindness of local anaesthetic drugs away from the heart and brain, increased cardiac contractivity, vasoconstriction, and cardioprotective effects.1 Last reported incidents range from 0 to 2 per 1000 nervous blocks but appears to decrease as a result of increased awareness of toxicity and better techniques.1 Proposal-Specific,6 some detailed systematic reviews of literature and practice advice America and Pain Medicine have published.1-5 There is still no RCT or study published in comparison with standard resuscitation care. Human data comes from about 100 case reports published until 2014.6 with an additional 47 separate cases in 35 articles between 2014 and November 2016, although patients only in 10 of these 47 cases receive any CPR.2 In identified cases, the results cannot be easily interpreted or associated with IV lipid emulsions given the lack of comparison groups. The administration of lipid IV emulsion is considered fairly benign, although pancreatitis and acute respiratory distress syndrome have been linked to its use.7 This topic last time received a formal evidence review in 2015.6 9.14Recommendations for Cardiac Capture Because of Sodium Channelers, Includes Tricyclic Antidepressant Proposals for Cardiac Arrest Due to Sodium Channel Blockers COR LOE Proposition 2a C-LD 1. The administration of sodium bikarbonate for cardiac arrest or delayed life-threatening cardiac flow (that is, the prolongation of QRS of more than 120 ms) due to sodium blocker/tricyclic antidepressant channels (TCA) can be beneficial. 2b C-LD 2. Use of the ECMO for cardiac arrest or refractory shock because sodium barrier/inflammatory TCA channels can be considered. Synopsis Overdose sodium-blocking drugs, such as TCAs and other medications (for example, cocaine, flecainide, amlopram), can cause hypotension, dysrhythmia, and death by the restriction of sodium heart channels, among other mechanisms. ECG findings feature include tachycardia and prolongation of QRS with senior bundle branch patterns.1.2 toxicity TCA can mimic ECG patterns of standard types of Brugada.3 therapies for hypotension or cardiotoxicity from sodium channel blocker poisoning consists of sodium boluses and alkaline serums, usually achieved through the administration of sodium bikarbonate boluses. This approach is supported by animal studies and human case reports and has recently been systematically reviewed.4

Clinical trials examined the administration of magnesium in addition to sodium bikarbonate for patients with TCA-induced hypotension, acidosis, and/or QRS prolongation.5 Although the overall outcome is better in the magnesium group, no significant statistical effects are found in death, magnesium patients are far less ill than control on the inclusion of the study, and the methodological weakness makes this work early. Although case reports describe good results after the use of lipid emulsion therapy ECMO6 and IV lipid therapy7-10 for sodium channels severe cardiotoxicity blocker, no controlled human studies are available, and limited animal data does not support the effectiveness of lipid emulsion.11 No human-controlled studies found evaluation dog.12 Recommendations-Specific Hypertonic Administrative Support Text (8.4%, 1 mEq / mL) sodium bikarbonate solution for Sodium channel restrictions caused by TCAs and other toxics are supported by human observation studies13,14 and animal experiments.12,15-22 This literature has recently been systematically reviewed.4 Although studies seeking doses are not available, initial dose of 1 to 2 mEq/kg (1-2 mL/kg 1 mEq/mL [8.4%]) sodium bikarbonate, recurring as necessary to achieve clinical stability while avoiding hypernatremia or extreme alkalemia) has a history of being recommended and looking effective. Case reports supporting the use of the ECMO for patients with refractory shock due to keraxicity TCA.23.24 Although the overall evidence for the ECPR to increase outcomes is limited, since inflammation of TCA is the reverse cause of cardiogenic shock/cardiac arrest, the use of ECPR/ECMO in patients with life-threatening inflammatory refractory to other therapies is logical This topic last time received an official evidence review in 2010.25 9.15Toxicity: Carbon Monoxide, Digoxin, and Cyanide's Proposal for Carbon Monoxide, Digoxin, and Cyanide Poisoning 1 B-R 1. Antidigoxin Fab antibodies should be administered to patients with severe heart glycopic toxicity. 2b B-R 2. Hyperbaric oxygen therapy can help in the treatment of acute carbon monoxide poisoning in patients with severe poisoning. 2a C-LD 3. Hydroxocobalamin and 100% oxygen, with or without sodium thiosulfate, can be beneficial to cyanide poisoning. Digoxin Synopsis poisoning can cause severe bradycardia, AV nodal restrictions, and life-threatening ventricle arrhythmia. Poisoning from other heart glycosides, such as oleander, foxglove, and digitoxin, has similar effects. Immediate treatment of heart glycoside toxicity is essential to prevent or treat life-threatening arrhythmia. Carbon monoxide poisoning reduces the ability of hemoglobin to deliver oxygen and also causes direct cellular damage to the brain and myocardium, leading to death or long-term risk of nerve and myocardial injuries. While cardiac arrest due to carbon monoxide poisoning is almost always fatal, studies on nerve sequels from less severe carbon monoxide poisoning may be relevant. Cyanide toxicity is mostly caused by aerobic cell metabolism cleaners. Cyanide reversely binds the cytochrome oxidase ferric ion in mitochondria and stops cellular breathing and adenosine triphosphate production. Cyanide poisoning may be caused by smoke inhalation, industrial exposure, self-poisoning, violence, or administration of sodium nitroprusside. Symptoms usually occur within minutes, and the findings may include arrhythmia, apnea, hypotension with bradycardia, seizures, and cardiovascular acidosis collapse.1 Lactic acidosis is a sensitive and specific finding.2.3 Instant antidotes including hydrocococculamines and nitrites; However, the container has a profile better. Sodium thiosulfate increases the effectiveness of nitrite by increasing cyanide detoxification, despite its role in patients treated with hydroxocobalamin hydroxocobalamin less certain.4 Antidote novels are under development. Recommendations of Supporting Text There is no data that assesses the use of antidotes to be digoxin excessively specifically in the setting of cardiac arrest. Data from 1 RCT5 and 4 cases of series6-9 conclude that Fab's antidigoxin fragments are safe and effective for the treatment of serious heart arrhythmia caused by digitalists and other glycoside glycoside doses of excess heart. Several patients who develop cardiac arrest from carbon monoxide poisoning survive for hospital clearance, regardless of treatment administered after the ROSC, although a rare good outcome has been described.10-12 Clinical trials of hyperbaric oxygen therapy to prevent neurological injuries from carbon monoxide poisoning patients with cardiac arrest excluded from all trials.13,14 Hyperbaric oxygen therapy has an incidence Several studies have shown that patients with known or suspected cyanide toxicity are present with cardiovascular instability or cardiac arrest undergoing immediate treatment with IV hydroxocobalamin, scavenger cyanide,2,15-19 can have a life-threatening toxicity reversal. Whether the addition of sodium thiosulfate, cocooctor for ciida metabolism, increases the antidotal effects of hydroxocobalamin is controversial. Four studies in animals20-23 and 2 studies in humans2,24 show increased efficacy of hydroxocobalamin when sodium thiosulfate is administered, although this did not happen in other models.4 This topic last time received a formal evidence review in 2010.25 10Knowledge Gaps and Research Preferences As part of the overall work for the development of these guidelines, the writing group was able to review large amounts of literature on the management of adult cardiac arrest. One challenge expected to face through this process is the lack of data in many areas of cardiac arrest research. The challenge was faced in both the 2010 Guidelines and the 2015 Guidelines Update process, where only a small percentage of guideline proposals (1%) based on high grade LOE (A) and nearly three quarters are based on low-grade loe (C).1 Similar challenges faced in the 2020 Guidelines process, where some critical knowledge gaps have been identified in the management of adult cardiac arrest. These topics are identified as not only areas where no information is identified but also where continuous research outcomes can directly affect recommendations. Throughout the specific text of the proposal, the need for specific research is identified to facilitate the next steps in the evolution of these questions. The critical knowledge gap is summarized in Table 4. Table 4. Adult Guidelines 2020 Critical Knowledge Gap Massage Restructuring Restructuring What is the strategy to improve the performance of CPR rescuers? Metrics for high-quality CPR What is optimal for the CPR task cycle (the proportion of time spent in compression is relative to compression-plus-decompression cycle time)? Metrics for high-quality CPR What is the validity and reliability of ETCO2 in disadvantaged patients? Metrics for high-quality CPR For patients with artery lines in place, does targeting CPR to certain blood pressure increase outcomes? Metrics for high-quality CPR How is the performance of the integrated team, as opposed to the performance of individual resuscitation skills, affecting resuscitation results? Defibrillation Is there an ideal time in the CPR cycle for defibrillator charging? Defibrillation May artifacts censor algorithms for ECG rhythm analysis during CPR in real-time clinical environments reduce pause in chest compression and improve yield? Defibrillation Does preshock wave analysis lead to better results? Defibrillation Does double-seeded defibrillation and/or alternative defibrillator pad positions affect the outcome in cardiac arrest with a surprising rhythm? Vascular access Does the IO path of drug administration be safe and efficient in cardiac arrest, and does effectiveness vary according to IO sites? Vasopressor medications during cardiac arrest Are epinephrine, when administered early after cardiac arrest, increasing survival with encouraging neurological outcomes? Non-penal drugs during cardiac arrest Are antiarrhythmic drugs, when given in combination for cardiac arrest, increasing the result of cardiac arrest with a surprising rhythm? Non-detention drugs during cardiac arrest Are antiarrhythmic prophylactic drugs on the ROSC after defibrillation managed to reduce recurrence of arrhythmia and increase yield? Nonvasopressor medications during cardiac arrest do steroids increase shock or other outcomes in patients who remain hypotensive after ROSC? Adjung to CPR Does the use of cardiac ultrasound care points during cardiac arrest increase yield? Adjung to CPR Targeting certain ETCO2 values during beneficial CPR, and what level of increase in ETCO2 shows rosc? Termination of resuscitation Can ETCO2 be used for intra-capture prognostics, in combination with other metrics? Termination of resuscitation May point-of-care the ultrasound heart, along with other factors, inform the termination of resuscitation? Advanced Techniques and Devices for Advanced AirWay Placement Resuscitation What is the optimal approach to extended airway management for IHCA? Advanced airway placement There is a need for further research specifically on the interface between patient factors and experience, training, tools, and supplier skills when choosing an approach to airway management. Advanced airway placement What kind, volume, and specific interval between airway management training experience to maintain efficiency? Techniques alternative CPR devices Whose residents are most likely to benefit from ECPR? Special Arrhythmia Management Atrial fibrillation or flutter with rapid ventricular response Is optimum energy necessary for atrial cardioversion and atrial flutter? Bradycardia What is the optimal approach, vasopressor or transcutaneous beat, in managing bradycardia symptoms? Post-care Postresuscitation ROSC Does avoid hyperoxia during the posarrest period lead to better outcomes? Postresuscitation care What are the effects of hypocarbia or hypercarbia on outcomes after cardiac arrest? Post-postsuscitation care Is seizure treatment unconvulsive, common in posarrest patients, improving patient outcomes? Postresuscitation Care What is the optimal pharmacological treatment regimen for the management of posarrest seizures? Postresuscitation care Does neuroprotective agents increase encouraging neurological outcomes after capture? Postresuscitation Care What is the most efficient management approach to posarrest cardiogenic shock, including pharmacological intervention, catheter, or devices that cannot be compressed? Postresuscitation care Is there a role for prophylactic antiarrhythmics after the ROSC? Targeted temperature management Are targeted temperature management, compared to strict normothermia, boost yields? Targeted temperature management What are the optimal temperature goals for targeted temperature management? Targeted temperature management What is the optimal period for targeted temperature management before swearing? Targeted temperature management What is the best approach to re-scanning post-postgraduate patients after treatment with targeted temperature management? PCI after cardiac arrest Does PCI appear for patients with ROSC after VF/VT cardiac arrest and no STEMI but with signs of shock or electrical instability improving yield? Neuroprognostication What is the interrater agreement for physical examination findings such as pupil light reflexes, corneal reflexes, and myoclonus/myoclonus status? Can we identify consistent NSE and S100B thresholds to predict poor neurological outcomes after cardiac arrest? Neuroprognostication Does the NSE and S100B help when checked later than 72 h after the ROSC? Neuroprognostication Is protein acidic fibrilari glial, protein tau serum, and precious light chain neurofilament for neuroprognostication? Neuroprognostication More uniform definitions for epilepticus status, malignant EEG patterns, and other EEG patterns are required to be able to compare prognostic values across studies. Neuroprognostication What is the optimal time for CT heads for prognostication? Neuroprognostication Is there a consistent threshold value for prognostication for GWR or ADC? Standardization of Neuroprognostication methods for measuring GWR and ADC will be useful. Rehabilitation and survival after cardiac arrest What are the survivors of cardiac arrest survival effects look like, and how they differ which current generic or clinician measures are obtained? Rehabilitation and survival after cardiac arrest Are there interventions in hospitals that can reduce or prevent physical impairment after cardiac arrest? Heart? and survival after cardiac arrest Which patients with disorders affect/psychological well-being after cardiac arrest, and are they treatable/preventable/recoverable? Rehabilitation and survival after cardiac arrest Is the planning of discharge of hospital-based protocols for cardiac arrest victims increasing access to/referrals to rehabilitation services or patient outcomes? Special Conditions of Accidental Hypothermia Resuscitation What are the combination of characteristics that can identify patients without the opportunity to survive, even promised? Accidental hypothermia If severe hypothermic patients receive intubation and mechanical ventilation or simply warm damp oxygen? Accidental hypothermia Should hypothermic patients in VF who fail early defibrillation attempts receive additional defibrillation? Accidental hypothermia In case of severe hypothermic patients in cardiac arrest receive epinephrine or other resuscitation medications? If so, what dosage and schedule should be used? Drowning In what situations tries to resuscitate victims of tropical drowning? Drowning How long after mild drowning events should patients be observed for late-onset respiratory effects? Electrolyte abnormalities What is the optimal treatment for hypercalemia with life-threatening arrhythmia or cardiac arrest? Excessive opioid What is the minimum safe observation period after a reversal of respiratory depression from excess opioids with naloxone? Does this vary based on the opioids involved? Excessive opioids Are there benefits to nalocson administration in patients with opioid-related cardiac arrest receiving CPR with ventilation? Excessive Opioids What is the ideal initial dose of naloxone in an environment where fentanyl and fentanyl analog are responsible for most of the excessive opioids? Excessive opioids In cases of suspected opioid doses are managed by non-healthcare providers who cannot be trusted to check the pulse, is the beginning of CPR beneficial? Pregnancy What is the ideal time for PMCD for a pregnant woman in cardiac arrest? Which pulmonary embolism patients with cardiac arrest on suspicion of pulmonary embolism benefit from anxiety thrombolysis during resuscitation? Toxicity: β-adrenergic blockers and calcium channel blockers What is the ideal sequencing of modalities (traditional vasopressors, calcium, glucagon, high-dose insulin) for refractory shocks because of β-adrenergic blockers or excessive calcium channel blockers? Toxicity: Local anaesthetic What is the ideal dose and formulation of lipid IV emulsion therapy? Toxicity: carbon monoxide, digoxin, and which patient cyanide has the benefits of cyanide poisoning from antidotal therapy? Toxicity: carbon monoxide, digoxin, and essence Do sodium thiosulfate provide additional benefits to patients cyanide poisoning treated with hydroxocobalamin? ADC shows a significant multiplication of perceives; CPR, cardiopulmonary resuscitation; CT, computer tomography; Ecg Ecg ECPR, cardiopulmonary resuscitation is outstanding; EEG, electroencephalogram; ETCO2, the ultimate tidal carbon dioxide; GWR, gray-and-white ratio; IHCA, cardiac arrest in hospital; IO, intraosseous; IV, intravenous; NSE, neuron-specific enolase; PCI, the intervention of the perkutgan coronary; PMCD, delivery of cesarean perimortem; ROSC, spontaneous circular repatriation; S100B, S100 calcium binding protein; STEMI, ST-segment myocardial infarah height; and VF, ventricular fibrillation. 11 The Weapons Proclamation of the American Heart Association requested that this document be referred to as follows: Panchal AR, Bartos JA, Cabañas JG, Donnino MW, Drennan IR, Hirsch KG, Kudenchuk PJ, Kurz MC, Lavonas EJ, Morley PT, O'Neil BJ, Peberdy MA, Rittenberger JC, Rodriguez AJ, Sawyer KN, Berg KM; for the Adult Basic and Advanced Life Support Writing Group. Part 3: basic and advanced adult life support: American Heart Association Guidelines 2020 for Cardiopulmonary Resuscitation and Cardiovascular Anxiety Care. Circulation. 2020;142(suppl 2):S366-S468. doi: 10.1161/CIR.0000000000000916 11.1Authors Ashish R. Panchal, MD, PhD, Chairman Jason A. Bartos, MD, PhD José G. Cabañas, MD, MPH Michael W. Donnino, MD Ian R. Drennan, ACP, PhD(C) Karen G. Hirsch, MD Peter J. Kudenchuk, MD Michael C. Kurz, MD, MS Eric J. Lavonas, MD, MS Peter T. Morley, MBBS Brian J. O'Neil, MD Mary Ann Peberdy, MD Jon C. Rittenberger, MD, MS Amber J. Rodriguez, PhD Kelly N. Sawyer, MD, MS Katherine M. Berg, MD, Vice Chair of the Basic and Advanced Adult Support Writing Group 11.2A acknowledges the following admits donor writing groups: Julie Arafeh, RN, MSN; Justin L. Benoit, MD, MS; Maureen Chase; MD, MPH; Antonio Fernandez; Edison Ferreira de Paiva, MD, PhD; Bryan L. Fischberg, NRP; Gustavo E. 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