CHEST

Improving CPR Performance

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Cardiac arrest continues to represent a public health burden with most patients having dismal outcomes. CPR is a complex set of interventions requiring leadership, coordination, and best practices. Despite the widespread adoption of new evidence in various guidelines, the provision of CPR remains variable with poor adherence to published recommendations. Key steps health-care systems can take to enhance the quality of CPR and, potentially, to improve outcomes, include optimizing chest compressions, avoiding hyperventilation, encouraging intraosseous access, and monitoring capnography. Feedback devices provide instantaneous guidance to the rescuer, improve rescuer technique, and could impact patient outcomes. New technologies promise to improve the resuscitation process: mechanical devices standardize chest compressions, capnography guides resuscitation efforts and signals the return of spontaneous circulation, and intraosseous devices minimize interruptions to gain vascular access. This review aims at identifying a discreet group of interventions that health-care systems can use to raise their standard of cardiac resuscitation. CHEST 2017; ■(■):■-■

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Cardiac arrest affects a significant portion of the US adult population. Over 300,000 out of hospital cardiac arrests (OHCAs) occur yearly in the United States, with only 10% of victims surviving to hospital discharge.[1] Over 200,000 patients suffer an in-hospital cardiac arrest (IHCA) each year, of whom 20% survive.[2] The last decade witnessed a modest improvement in survival from IHCA and OHCA. This progress could be attributed to prompt resuscitative efforts and shocks, improved CPR quality, and better postresuscitation care (including timely angiographic reperfusion).[3] Nevertheless, there is tremendous room for improvement. When health-care institutions are compared, patient outcomes are quite variable, even after accounting for severity of illness and hospital structure.[4,5] This finding suggests that processes of care affect outcomes, with CPR quality a main contributor. The Institute of Medicine, the American Heart Association (AHA), and the International Liaison Committee on Resuscitation place high-quality CPR at the heart of resuscitation.[6] The acknowledgement that CPR quality affects patient outcomes, and varies between institutions, offers a window of opportunity. Novel technologies can provide instantaneous feedback to the

ABBREVIATIONS: AHA = American Heart Association; CPP = coronary perfusion pressure; ERC = European Resuscitation Council; ET-CO_2 = end-tidal CO_2; IHCA = in-hospital cardiac arrest; IO = intraosseous; OHCA = out of hospital cardiac arrest; ROSC = return of spontaneous circulation

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rescuer: a feedback loop that could enhance the provision of CPR and improve outcomes. This article focuses on new key interventions to improve the quality and outcomes after CPR (Table 1), based on the latest evidence in basic and advanced life support.

CPR Physiology

The goal of CPR is to restore blood flow and perfuse vital organs. The two most plausible mechanisms to explain how chest compressions generate blood flow are the cardiac pump and thoracic pump theories. Historically, the cardiac pump theory was considered the prevailing mechanism.[7] By virtue of its location between the sternum and the spine, the heart is squeezed every time the chest is compressed. Because valves limit retrograde flow, extrinsic compression of the heart could act like intrinsic contraction, producing a stroke volume. This mechanism depends on mechanical coupling of the sternum and heart. Echocardiography of animal and human subjects during chest compression shows that the ventricles decrease in size, the aortic valve opens to allow forward flow, and the mitral valve closes to prevent retrograde flow.[8-10]

This theory is attractive, but doubts were raised when cough CPR was described.[11] Patients going into ventricular fibrillation in the angiography suite could maintain consciousness solely by coughing. This clearly happened without chest compression, offering an alternative pumping mechanism: the thoracic pump theory. This theory focused on the rise in intrathoracic pressure, rather than direct cardiac compression, as the generator of forward blood flow from the central vessels and lungs into the systemic circulation. If this mechanism is operative, pressures rise similarly in the pleural space, blood vessels, and heart; atrioventricular valves should be open during compression; and venous valves (or venous compression at the thoracic inlet) prevent retrograde flow of blood from the thorax. Some echocardiography studies reveal the atrioventricular valves to be widely open during compression.[12,13] Direct measurement of thoracic pressures during CPR found that cardiac chamber and central vessel pressures are equal and rise in tandem during compression—this should not be the case according to the cardiac pump theory.[14]

These theories are not mutually exclusive and could be complementary. Early after arrest, cardiac valves might still be competent and the ventricles compliant, favoring the cardiac pump theory. As myocardial ischemia persists and the myocardium is stunned, the ventricles become stiff and the valves incompetent, favoring the thoracic pump mechanism.[15] Understanding the prevailing mechanism is important to optimize resuscitation. Hand location and patient anatomy are more relevant for the cardiac pump theory,[16] whereas devices such as the automatic load distributing band and pneumatic vest, which augment changes in thoracic pressure, rely on the thoracic mechanism.[17] Perhaps in support of the thoracic pump mechanism, animal experiments have shown that techniques that amplify thoracic pressure changes can boost systemic perfusion. Nevertheless, none has proved effective for improvement of outcomes in patients.[17]

Regardless of mechanism, and although further research is needed to understand the underlying driving process, the anteroposterior displacement of the chest remains the single most important intervention to increase the chances of successful resuscitation.[18] The rescuer should optimize the delivery of chest compressions, whether it is the rate of compressions, depth, or allowing for chest recoil as will be detailed in the following section.

TABLE 1] Key Steps for Health-Care Systems to Improve Resuscitation

1. Optimize chest compressions
Audit depth and rate, providing feedback to the rescuer
Train leader to recognize rescuer fatigue
2. Avoid hyperventilation
Emphasize compression over ventilation
Consider compression-only CPR, where appropriate
3. Encourage intraosseous access
4. Monitor the capnogram for compression adequacy, to detect return of spontaneous circulation, and to guide the duration of attempted resuscitation

Coronary Perfusion Pressure

Coronary blood flow is governed by coronary perfusion pressure (CPP) (which is the gradient between aortic and right atrial pressure during the relaxation phase of CPR), coronary vascular resistance, and intramyocardial pressure. Its regulation is complex and depends on metabolic needs, neurogenic state, and local humoral factors.[19] CPP has been shown to reflect myocardial blood flow during CPR[20,21] and correlates with successful resuscitation and better patient outcomes. In animal models and human studies, higher CPP is associated with return of spontaneous circulation

(ROSC) and survival at 24 h.[22-24] CPP can also be used to guide termination of resuscitative efforts. In a study involving 100 patients in cardiac arrest, CPP < 15 mm Hg had a negative predictive value of 1.[25]

Therefore, an immediate goal of CPR is to raise the CPP through provision of high-quality CPR, targeting specific depth and rates of compression. Especially important is compression depth, with deeper compressions producing higher CPP and cardiac output.[26] When compression depth is increased in IHCA and OHCA with a shockable rhythm, successful cardioversion, ROSC, and favorable functional outcomes are more likely.[27,28] In swine models, compression to 25% of the anteroposterior diameter of the chest generates CPP > 15 mm Hg, which is sufficient for ROSC.[29] In adult human studies, the most benefit is attained when compression depth is at least 4.5 cm.[30,31] The benefit plateaus between 4.5 and 6 cm,[32] after which deeper compressions could cause complications.[33]

Similarly crucial is the rate of compression. As the rate is increased from 60 to 120 compressions per min, greater CPP is generated,[8,34,35] and the chance of ROSC and survival rise.[36] Maximal benefit is seen in the range of 100 to 120 compressions per min, above or below which ROSC and survival decline (Fig 1).[36,37] The detrimental effect of compressions > 125 per min could be explained by the shallower compressions generated because numerous studies have shown that the compression rate and depth are inversely related.[37,38] Faster rates also reduce the time for chest recoil, venous return to the chest, and cardiac filling, ultimately lowering forward blood flow.[39]

Chest compression is often interrupted in practice when checking for a pulse, delivering rescue breaths, performing endotracheal intubation, obtaining vascular access, administering drugs, or conducting defibrillation. CPP declines dramatically when compressions cease, and only regains adequate levels very slowly once compressions are resumed (Fig 2).[40] The duration of the pause is also significant, with longer pauses being negatively associated with survival to hospital discharge in OHCA.[41] To stress the importance of reducing these interruptions, many investigators analyze the chest compression fraction, which reflects the time of CPR devoted exclusively to chest compression. Irrespective of the etiology of cardiac arrest, a higher chest compression fraction improves patient outcomes.[42,43]

Every effort should be done to augment the CPP. This dictates the many steps of CPR and supports the various resuscitation guidelines. Based on the current evidence, the AHA and the European Resuscitation Council (ERC) recommend that compression rates be between 100 and 120 compressions per min and compression depth be least 5 cm (2 in) but not greater than 6 cm (2.4 in). Emphasis is placed on minimizing interruptions and the duration of pauses.[44,45]

Cardiopulmonary Interactions

By virtue of their shared anatomic location, the heart and lungs are mechanically coupled. Positive pressure ventilation, chest compression, and chest recoil all have meaningful consequences for cardiac preload, thoracic

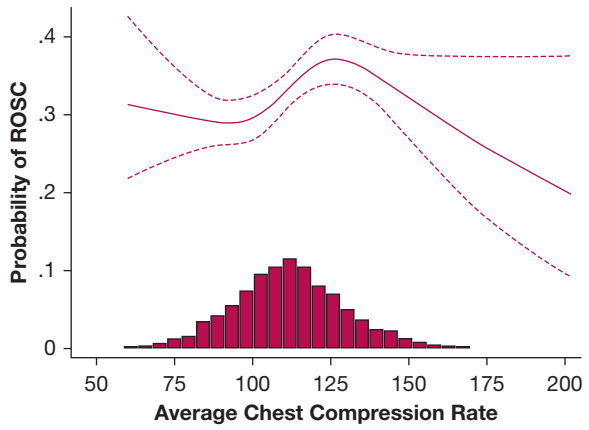

Figure 1 – *Adjusted cubic spline of the relationship between chest compression rates and the probability of ROSC. Dashed lines show 95% CIs. ROSC = return of spontaneous circulation. (Reprinted with permission from Idris et al.[36])*

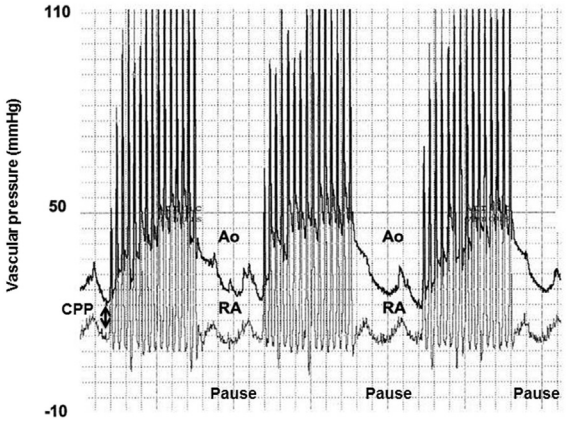

Figure 2 – *The CPP is the gradient between the Ao and RA pressures during the relaxation phase. The CPP decreases when chest compressions are stopped. The CPP does not immediately return to prior levels once compressions are resumed, but requires a few compressions to build up. The x axis reflects the time in seconds. The y axis reflects the vascular pressure in millimeters of mercury. Ao = aortic; CPP = coronary perfusion pressure; RA = right atrial. (Reprinted with permission from Berg et al.[40])*

venous return, and CPP (because the downstream pressure is the right atrial pressure).

Much attention has centered on the role of ventilation. Positive pressure ventilation raises the mean pleural pressure, impeding venous return. Hyperventilation amplifies this, especially in patients with obstructive lung diseases who are subject to air trapping. Although relevant in all mechanically ventilated patients, this downside is more critical for patients in cardiac arrest.[46] During resuscitation, reduced pulmonary blood flow requires low minute ventilation for optimal matching of ventilation and perfusion. In practice, the opposite is often observed, with excessive rates and volumes being delivered.[47] In addition to the detrimental effects on intrathoracic pressure, excessive ventilation results in respiratory alkalosis, inducing cerebral vasoconstriction in an already compromised brain. Alkalosis also shifts the oxyhemoglobin curve to the left, possibly reducing oxygen off-loading to tissues.[48]

The goals of ventilation during CPR are threefold: (1) deliver oxygen, (2) eliminate carbon dioxide, and (3) minimize the impedance to systemic perfusion. Oxygen transport depends more heavily on blood flow than on high partial pressures of oxygen in the arterial blood, with most of cardiac arrests being caused by cardiac pathology (and not asphyxial in nature).[49] In the early stages of cardiac arrest, oxygen content is often sufficient, whereas oxygen consumption is minimal. In animal models of ventricular fibrillation using compression-only CPR (no ventilation), oxygen saturation remained $>$ 70% for 10 min.[50] Mathematical modeling reveals that increasing the chest compression to ventilation ratio to up to 60:2 improves overall oxygen delivery.[51]

This begs the question of whether ventilation is warranted during CPR. Although some observational studies in OHCAs from a cardiac cause demonstrated that compression-only CPR is at least equivalent to standard CPR (ie, including rescue breaths),[52,53] a larger randomized trial did not support this finding.[54]

Some health-care institutions have adopted compression-only CPR as their standard resuscitation technique for OHCAs. There are multiple additional benefits to compression-only CPR: airway management accounts for significant interruptions in CPR; endotracheal intubation can cause serious complications (eg, esophageal intubation); even after an airway is secured, providing rescue breaths leads to frequent interruptions in chest compression[55]; and bystanders are more inclined to begin CPR when only compressions are required.

In light of the conflicting data, we think chest compressions should remain prioritized. In the event of a witnessed cardiac arrest (where the etiology is believed to be cardiac), or when a lay bystander is performing CPR, continuous chest compressions are appropriate. For asphyxic cardiac arrest, especially if CPR is provided by a trained rescuer, chest compressions interrupted by rescue breaths are appropriate.

When CPR is conducted with ventilation for OHCAs, endotracheal intubation is used most commonly; however, results are mixed.[56,57] Alternatives include bag-mask ventilation and supraglottic airways. Retrospective studies show that bag-mask ventilation is associated with better outcomes than endotracheal intubation; however, this might reflect a less sick patient population.[58] There is no conclusive evidence that early intubation improves patient outcomes; therefore, some rescuers intubate patients after ROSC.

Ventilation seems relatively unimportant regarding carbon dioxide elimination. The partial pressure for carbon dioxide rises only modestly during arrest, and its impact does not seem pronounced.[59]

Chest recoil, through rib cage and lung expansion, creates a negative intrathoracic pressure. This lowers right atrial pressure, which increases venous return to the right heart. Leaning on the patient's chest (as often happens with rescuer fatigue), prevents full recoil, blunting the negative intrathoracic pressure and slowing blood flow. Similarly, leaning on the chest raises intrathoracic and right atrial pressures, which decreases CPP and myocardial blood flow,[60] with deleterious effects on survival.[61]

Understanding cardiopulmonary interactions and the impact on venous return and cardiac output has been used to develop several devices to optimize cardiac resuscitation. The impedance threshold device limits air entry during chest recoil, maintaining the negative intrathoracic gradient. Interposed abdominal compressions during chest relaxation improve organ perfusion.[62] This is accomplished by increasing the intrathoracic pressure and increasing venous return to the heart.[63]

There has been an evolving recognition of the importance of chest compression over ventilation. The 2010 AHA guidelines endorsed a dramatic shift in resuscitation sequence from airway-breathing-circulation to circulation-airway-breathing.[64] Similarly, the

compression to ventilation ratio was increased from 15:2 to 30:2. In the event that the patient does have an advanced airway (endotracheal tube or a supraglottic device), a breath should be delivered only every 6 s. The 2015 AHA guidelines suggest three cycles of 200 continuous compressions before positive pressure ventilation for patients undergoing OHCA with a shockable rhythm.[65] Both the AHA and ERC recommend allowing for complete chest recoil between compressions, and avoiding leaning on the patient's chest.[44,45]

Tools to Improve the Quality of CPR

Real-time Feedback

Despite the evidence behind these essential components of high-quality CPR and robust published guidelines, numerous studies have demonstrated poor compliance with the recommended targets and wide variability in the quality of CPR in clinical practice.[66,67] This disparity between guidelines and practice is multifactorial: rescuers could have inadequate knowledge or poor retention after training; resuscitation is a complex situation, with the rescuer expected to manage various time-sensitive tasks; and rescuer fatigue is common, manifesting as early as 1 min after starting CPR.[68]

When CPR metrics are audited, compression rates are frequently outside of the recommended range.[37,69] Pauses are frequent, and some studies report compressions only one-half of the code duration.[66] Compression depth does not reach the target in one-half of cases.[31] Rescuers inadvertently impede chest recoil during the decompression phase.[70] Patients are commonly hyperventilated, with respiratory rates double what is recommended.[61]

Novel tools are now available to address these deficiencies in CPR quality and minimize interruptions. New feedback devices track compression metrics (rate, depth, chest recoil, and pauses), display them in real time, and provide instantaneous audiovisual guidance (Fig 3). Studies have shown that these devices bring CPR metrics closer to the established guidelines.[27,71] This seems important progress, but patient-centered outcomes are not clearly better.[72] This discrepancy could be caused by multiple reasons. There is a learning curve to optimize the benefit from this new technology. These devices might need to be refined to suit the user: for example, rescuers might not be able to respond to multiple recommendations (push faster and deeper), and the frequency of recommendations might need to be adjusted as well (too many orders might hamper any progress and too little might not be enough). Alternatively, the gains obtained might not be sufficient to increase the chances of successful resuscitation. To date, there are only a handful of studies with a small number of patients. We anticipate more robust research to study the impact of these devices.

Mechanical Chest Compression

Given the importance of uninterrupted high-quality compressions, mechanical devices are an attractive option for replacing fallible human rescuers. These devices (eg, LUCAS device; Jolife AB, a part of Physio-Control, Inc) deliver uninterrupted mechanical compressions at a fixed rate and depth, and guarantee active chest decompression. Physiologic animal data show that cerebral blood flow is greater with mechanical than with manual compression.[73] However, subsequent studies in patients have not demonstrated an improvement in outcomes when these mechanical compression devices are used.[74,75] This could be because of the time needed to deploy them (it takes 1-2 mins to appropriately place them on the patient) or only a small beneficial increment over manual CPR. Despite the current lack of evidence, these devices may be beneficial when CPR quality is challenged by circumstances, such as during patient transport, throughout percutaneous coronary intervention, or when cardioversion is attempted. The AHA suggests using them during transport or in situations where prolonged resuscitation is being done and rescuer fatigue is anticipated.[65]

Timely Vascular Access

Although the role of IV pharmacologic therapy in patients undergoing cardiac arrest is controversial and requires further research, antiarrhythmics and vasoconstrictors remain part of advanced cardiovascular life support.[76] Securing vascular access is essential for drug administration, fluid resuscitation, infusing blood products, and obtaining blood for laboratory studies. Compressions are compromised, and precious time is wasted attempting to gain vascular access. Rescuers spend an average of 3.6 to 5.8 min attempting to insert a peripheral IV catheter, with a first attempt success of only 43%.[77,78] Similarly, central venous catheters can require $>$ 10 min, with a success rate of 60%.[79]

Intraosseous (IO) access offers an invaluable alternative to peripheral and central venous catheters. The IO route is noncollapsible and ensures access to systemic circulation through the highly vascular medullary plexus. Drugs administered through this route have

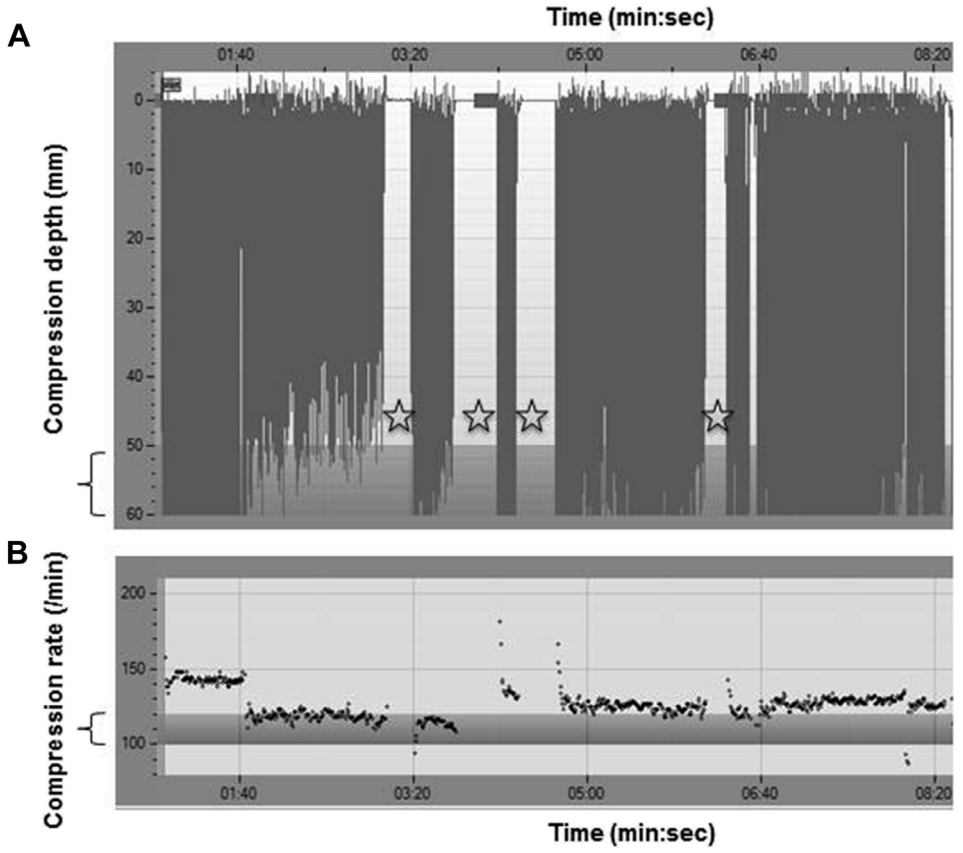

Figure 3 – *A, B, Actual CPR tracing from a feedback device: compression depth (lower limit of 0 mm, upper limit of 60 mm) (A), and the compression rate (lower limit of 80 compressions per min and upper limit of 200 compressions per min) (B). The brackets represent the recommended compression depth and rate (50-60 mm and 100-120 compressions per min, respectively). The stars represent pauses during CPR. In this example, the compression depth is reached throughout the code; however, the compression rate is often times above the recommended target. At 1:40, there is a decline in compression depth and rate, suggestive of rescuer fatigue. The chest is allowed to recoil (to 0-mm depth) for most compressions.*

similar pharmacokinetics and absorption as peripherally given medications.[80-83] Insertion requires less training than a central venous catheter, has a higher success rate (> 80%), and can be achieved rapidly (from 20 s to approximately 2 min).[77,79] IO access is also more reliable for drug delivery than endotracheal, sublingual, or intramascular routes, all of which have unpredictable absorption. IO access can be obtained through the sternum, the humerus, or the tibia. The sternal route achieves time to peak drug blood concentration comparable with a central venous catheter.[82] The risk of injury to the mediastinal organs and withholding compressions for insertion make this route less favorable. Despite the seemingly higher infusion rate achieved through the humeral route, the tibia is more preferred because it does not interfere with chest compressions, has a higher success rate of insertion, and has a shorter insertion time.[77,84,85] Both the AHA and ERC recommend IO access when vascular access is not available.[44,45]

Capnography

During non-steady-state conditions (eg, during CPR, with ROSC), pulmonary blood flow is a major contributor to end-tidal CO_2 (ET-CO_2). As such, ET-CO_2 reflects the cardiac output generated during chest compressions.[86] Capnography can be used to assess the effectiveness of chest compressions: a drop in the ET-CO_2 is an indicator of inefficient compressions (as would happen with rescuer fatigue).[87] ET-CO_2 also correlates with CPP, which has prognostic value: an ET-CO_2 value < 10 mm Hg after 20 min of CPR reflects very little myocardial perfusion and is associated with a very high mortality.[88] On restoration of a perfusing rhythm, the ET-CO_2 surges, an indicator of brisk pulmonary blood flow (Fig 4).[89] By signaling ROSC, capnography can be used to minimize unnecessary interruptions to check for a pulse. Although most of the evidence stems from the use of endotracheal tubes, the ensuing physiology and recommendations hold true when supraglottic airways are used.[90]

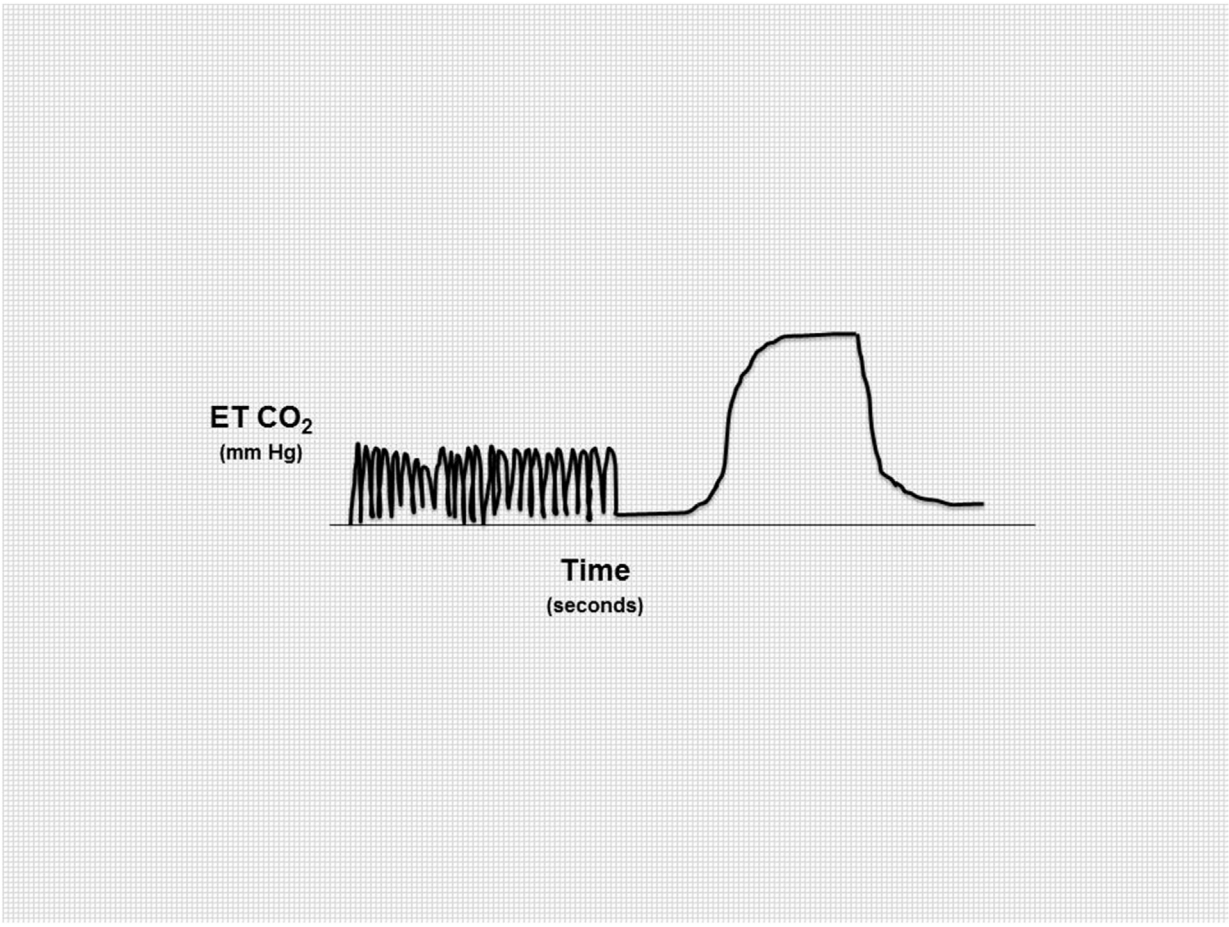

Figure 4 – *A sketch representing a typical capnographic pattern during CPR. A sustained increase in ET CO_2 signals return of spontaneous circulation. ET CO_2 = end-tidal CO_2.*

The AHA recommends achieving ET-CO_2 of at least 20 mm Hg, using capnography to check for ROSC and including it when deciding on terminating resuscitative efforts.[44,45] Continuous waveform capnography, by detecting alveolar CO_2, is also an excellent tool to check for the proper tracheal location of an endotracheal tube or a supraglottic airway device.[44]

Conclusions

Improving the chances for successful resuscitation relies on optimizing compression depth and rate, while minimizing the potentially harmful effect of ventilation. To minimize interruptions in CPR, modified approaches to ventilation, vascular access, and monitoring for ROSC are recommended (Table 1). Guidelines from the AHA and International Liaison Committee on Resuscitation are based on best evidence, but real-world performance often falls short. Institutions committed to improving CPR processes can implement new feedback devices to improve rescuer performance. Combined with supervision and feedback, these offer hope to improve patient outcomes.

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