Monitor the quality of cardiopulmonary resuscitation in 2020

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Purpose of review
The current review will give an overview of different possibilities to monitor quality of cardiopulmonary resuscitation (CPR) from a physiologic and a process point of view and how these two approaches can/should overlap.

Recent findings
Technology is evolving fast with a lot of opportunities to improve the CPR quality. The role of smartphones and wearables are step-by-step identified as also the possibilities to perform patient tailored CPR based on physiologic parameters. The first steps have been taken, but more are to be expected. In this context, the limits of what is possible with human providers will become more and more clear.

Summary
To perform high-quality CPR, at first, one should optimize rate, depth and pause duration supported by process monitoring tools. Second, the evolving technological evolution gives opportunities to measure physiologic parameters in real-time which will open the way for patient-tailored CPR. The role of ultrasound, cerebral saturation and end-tidal CO_2 in measuring the quality of CPR needs to be further investigated as well as the possible ways of influencing these measured parameters to improve neurological outcome and survival.

Keywords
cardiopulmonary resuscitation, feedback devices, monitoring, physiologic monitoring, quality

INTRODUCTION

Out-of-hospital cardiac arrest (OHCA) is a major cause of health problems and death worldwide [1,2]. The outcome after cardiac arrest improved slowly during the last decades with current survival rates to hospital discharge of 10%. However, survival rates differ between regions with survival rates between 2.2 and 12.0% [3]. Many different factors have an effect on the outcome and among which is quality of the administered cardiopulmonary resuscitation (CPR).

The main goal of CPR is to achieve return of spontaneous circulation (ROSC) as quickly as possible secondary to maintaining sufficient cerebral and myocardial perfusion to preserve the prearrest neurological state. However, the mechanical goal of CPR is to circulate blood in the absence of cardiac output (CO) to perfuse the heart and brain until defibrillation is possible or ROSC is achieved. To achieve this goal, CPR currently exists out of chest compressions, ventilation and resuscitation drugs. If we pursue high-quality CPR, defined as the optimal CPR for a person to achieve the highest chance of survival, it is necessary to measure physiologic parameters to follow the effect of a given treatment. Measuring chest compression depth and rate is a beginning but it is also only measuring to what extent the evidence-based guidelines are followed [4].

The continuous evolution in technology has a constant impact on the possibilities for measuring physiologic parameters and can improve in an indirect manner the quality of CPR and therefore also the outcome of cardiac arrest patients. If CPR is based on invasive measured physiologic parameters, higher 24 h rate of favourable outcome is observed

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KEY POINTS

- Measuring quality of CPR can improve the administered CPR quality.
- Higher CPR quality is associated with better outcome.
- New technology can and should be used to measure and improve the quality of CPR.
- The next challenge is to identify the best physiologic monitor(s) to use during CPR.

in animal models [5,6]. Monitoring the quality of CPR is not only the first step before we can improve the quality of CPR but also a step in the direction of an individualized patient care. If there is real time feedback about the quality of CPR, it will be possible to adapt our resuscitation strategies to improve the quality of CPR, the exact specifications of which may differ between individual patients. The key for this future strategy is reliable, real time monitor(s), which give us insight into the ongoing physiologic status of the cardiac arrest patient.

The current review will give an overview of different possibilities to monitor quality of CPR from a physiologic and a process point of view and how these two approaches can/should overlap.

MEASURING CARDIOPULMONARY RESUSCITATION PERFORMANCE

CPR is foremost a mechanical process that can be characterized, and therefore evaluated, by many process parameters. The fundamental mechanical unit of CPR is a single chest compression, which begins with application of force to the sternum, such that the sternal surface displaces inward toward the spine [7]. Compression of the thoracic space in this manner increases the intrathoracic pressure and (in)directly facilitates ejection of blood from the ventricles. A number of parameters characterize the downstroke, including the peak force, velocity, depth (displacement) and initial surface position. Some of these parameters are thought to be highly influential in the optimization of forward blood flow during CPR, although historically chest compression depth is supported by the most substantial evidence [8]. Chest compression depth can be measured or inferred through a number of sensory modalities, the most common of which is currently accelerometry, conducted alone or in combination with supplementary sensors [9–11]. The largest human observational studies support absolute depth as a key process measure related to resuscitation outcomes, a finding that is echoed in laboratory resuscitation models [12–14]. Little human data are available supporting proportional depth in the setting of OHCA, although it is intuitive that larger chest diameters might benefit from deeper chest compressions [15▪].

Of course, what goes down must come up, and so each downstroke is followed by a corresponding upstroke whereby downward force is withdrawn from the sternum, such that it can return or 'recoil' to a resting, uncompressed position. At the start of recoil, the deformation of the thorax and corresponding redistribution of its contents creates a negative pressure gradient between the compressed space and the surrounding compartments. The net effect on the circulatory system is to draw blood back towards the heart [16]. Extent of recoil and recoil or release velocity are two parameters of interest in the upstroke phase of the chest compression, with some evidence suggesting that both may have implications for resuscitation outcomes [17–19]. Incomplete recoil is a practical consequence of the provider leaning on the patient and is relatively common [20]. Release velocity is the speed at which the chest returns to resting level, and is a function of the speed at which the provider is upstroking, the present constitution of the chest, and likely other innate patient factors [21▪]. Measurement of both can be accomplished with the same technologies used to determine peak compression depth, where force sensing provides a direct assessment of leaning, unconfounded by chest deformations that may alter resting level.

CPR is a cyclic mechanical process, and so chest compressions are delivered in series, with the chest oscillating between the target compression depth and resting level. The period of this oscillation, more frequently taken in inverse as the rate of chest compressions, is a well characterized parameter of CPR delivery associated with resuscitation outcomes [22,23▪]. While chest compression depth might be considered analogous to cardiac contraction, compression rate is conceptually analogous to heart rate, with similar theoretical implications for optimization, including considerations for adequate ventricular filling time and contribution to CO [24]. Measurement of chest compression rate is possible with a more diverse array of sensors than depth. Transthoracic impedance signal available in nearly all modern prehospital defibrillator-monitors allows for adequate rate measurement under many circumstances, although the impedance waveform is susceptible [25,26]. Rate can also be accurately determined through analysis of the same sensors used to determine depth, either through peak analysis or spectral analysis methods [27,28].

As chest compressions are delivered over time, process interruptions may occur, breaking the continuity of CPR. Algorithmically, this could be related to pulse checks, defibrillation events or planned provider changes for prolonged CPR. There is a body of literature on the effects of planned pauses, most specifically and extensively on the peri-shock pause interval, supporting the minimization of pause-related no-flow periods [29,30]. Theoretically, the same sensory modalities that facilitate rate and depth measurement can detect individual prolonged pauses. In the same manner, the cumulative impact of pauses within any given bout of CPR can be assessed through derivation of the chest compression fraction, the proportion of time when chest compressions are happening over the total analysed time period [31]. When accounting for important confounders that obscure the cause and impact of pauses contributing to low chest compression fraction, significant observational evidence lends support for monitoring of this measure [18,32].

CPR process quality assessment is most widely conducted with the aid of clinical monitors equipped with the necessary sensors. Real-time process quality is frequently operationalized as audiovisual feedback, and some evidence supports the use of monitor-based real-time feedback for controlling CPR process quality, although evidence is not overwhelming [33]. The same real-time feedback may be achievable with independent, portable technologies deployed in smartphones or standalone quality measurement units [34,35,36▪,37,38▪]. Furthermore, computer vision methods offer an additional future avenue for measuring CPR process quality, deriving process parameter measurements from analysis of video imagery of CPR [39,40].

One of the challenges of the pursuit of high-quality CPR process is determining which parameters are both influential on outcomes and translatable into practice for human care providers [41▪]. As advances are made in medical robotics, it is to be expected that advanced mechanical chest compression devices will incorporate performance parameters that are not practical for humans to self-adjust, the limits of which may be relatively narrow [42▪]. For now, those parameters that are most intuitive human performers – rate, depth and pause duration – are also those emphasized by the guidelines, and should be the focus of CPR process quality control efforts, supported by process monitoring tools [8]. One of the most consistent findings from large human resuscitation trials is that, despite these recommendations, provider CPR performance varies greatly, reflecting a need for greater work to ensure high-quality CPR delivery [13,22,43▪].

MONITORING THE PHYSIOLOGICAL RESPONSE OF CARDIOPULMONARY RESUSCITATION

Current resuscitation guidelines are mainly focused on the performance of the CPR-provider, as described above, which in theory should vary little from patient to patient. Over recent years, patient-tailored resuscitation has been recommended, if feasible, during which the physiological/haemodynamic response to resuscitative efforts should be monitored. Figure 1 gives an overview of the monitoring possibilities.

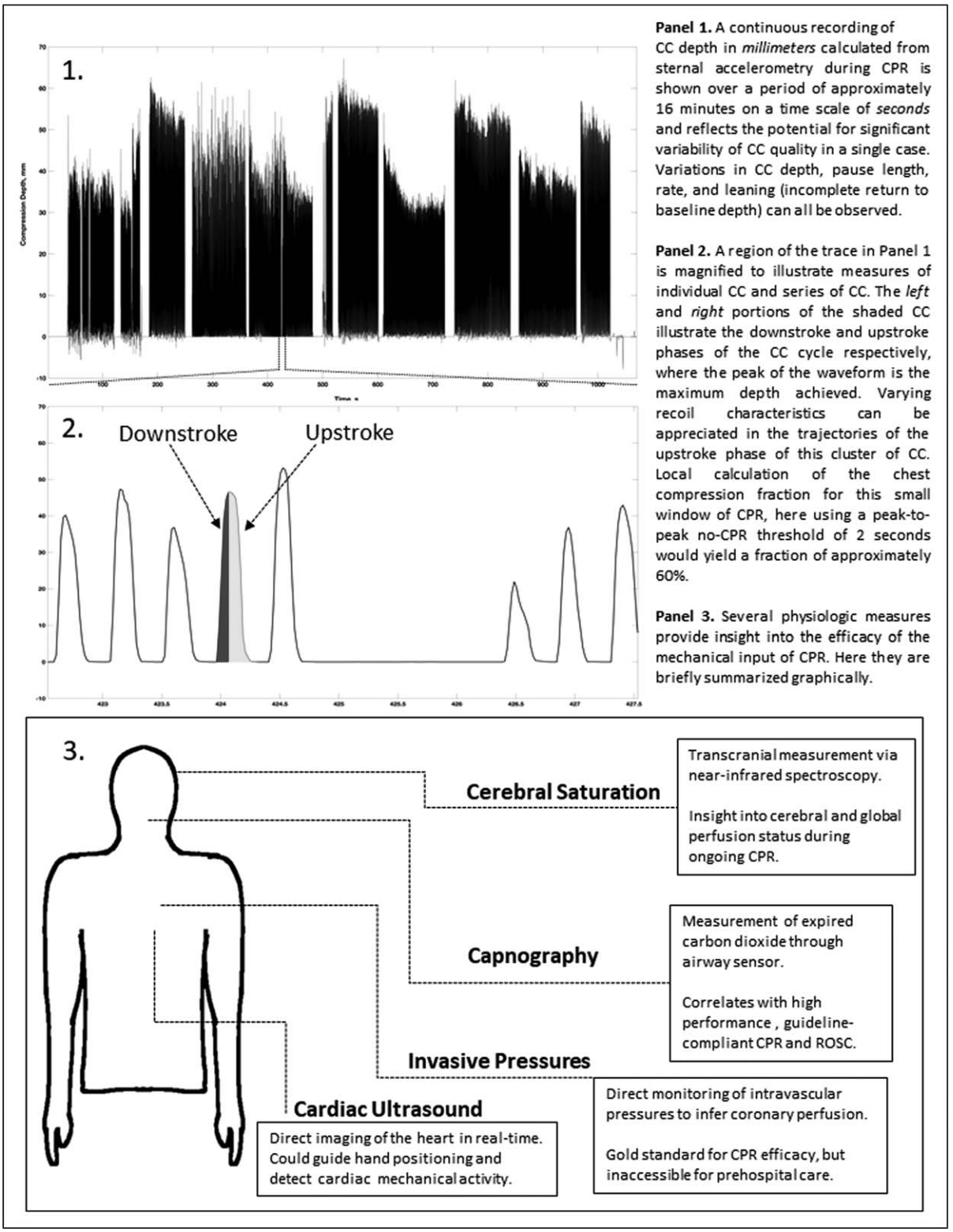
CORONARY PERFUSION PRESSURE AND DBP

The coronary perfusion pressure (CPP), defined as the aortic-to-right atrial pressure gradient during the relaxation period of cardiac decompression, is the primary determinant of myocardial perfusion and oxygen delivery during resuscitation [16,44,45]. The greater the CPP, the higher the resulting myocardial blood flow. Therefore, augmenting CPP during CPR by providing compressions with an optimal compression depth (4.5–5 cm) and rate (100–120/min) is pivotal to achieve ROSC, but limiting chest interruptions to the minimum is equally important to avoid declining of CPP [12,46–49].

Evidence concerning the importance of CPP during CPR is mainly derived from animal studies, although human data also confirm that high CPPs are key to establish ROSC [50–52]. In this way, CPP provides unique feedback concerning the performance of the CPR-provider. Although there is no consensus about an optimal CPP, successful resuscitation becomes more likely when CPPs above 20 mmHg are being targeted. Animal data even suggest to aim for CPPs between 30 and 40 mmHg [52]. In recent pig studies, CPP-targeted resuscitation was found to be superior to guideline-provided care in terms of short-term and long-term survival [6,53,54]. Especially due to the complexity of measuring CPP in the clinical field, undeniable evidence from human studies to corroborate these experimental data is currently lacking.

During CPR, arterial DBPs approximate aortic diastolic pressures through which they can serve as surrogate for CPP [55]. Although there is hardly any clinical evidence, animal data demonstrated that invasively measured DBPs are excellent discriminators for survival. Failure to maintain diastolic pressures above 30–35 mmHg unlikely resulted in ROSC [50,56,57].

Based on the available clinical and animal data, an American expert panel endorsed to use CPP as the primary physiological target (i.e. CPP > 20 mmHg)



Panel 1. A continuous recording of CC depth in *millimeters* calculated from sternal accelerometry during CPR is shown over a period of approximately 16 minutes on a time scale of *seconds* and reflects the potential for significant variability of CC quality in a single case. Variations in CC depth, pause length, rate, and leaning (incomplete return to baseline depth) can all be observed.

Panel 2. A region of the trace in Panel 1 is magnified to illustrate measures of individual CC and series of CC. The *left* and *right* portions of the shaded CC illustrate the downstroke and upstroke phases of the CC cycle respectively, where the peak of the waveform is the maximum depth achieved. Varying recoil characteristics can be appreciated in the trajectories of the upstroke phase of this cluster of CC. Local calculation of the chest compression fraction for this small window of CPR, here using a peak-to-peak no-CPR threshold of 2 seconds would yield a fraction of approximately 60%.

Panel 3. Several physiologic measures provide insight into the efficacy of the mechanical input of CPR. Here they are briefly summarized graphically.

FIGURE 1. Monitoring possibilities. Panel (a) a continuous recording of chest compression (CC) depth in *millimetres* calculated from sternal accelerometry during cardiopulmonary resuscitation is shown over a period of approximately 16 min on a time scale of *seconds* and reflects the potential for significant variability of CC quality in a single case. Variations in CC depth, pause length, rate and leaning (incomplete return to baseline depth) can all be observed. Panel (b) a region of the trace in panel (a) is magnified to illustrate measures of individual CC and series of CC. The *left* and *right* portions of the shaded CC illustrate the downstroke and upstroke phases of the CC cycle, respectively, where the peak of the waveform is the maximum

when both arterial and central venous catheters are in situ at the time of arrest, and to aim for DBPs above 25 mmHg in case only an arterial line is in place [58]. In patients suffering from an in-hospital cardiac arrest, invasive haemodynamic monitoring is often at hand, and consequently haemodynamic-targeted CPR becomes feasible and highly recommendable. On the contrary, in an out-of-hospital cardiac arrest setting, placing this type of invasive monitoring is rather difficult through which noninvasive alternatives like end-tidal CO_2 ($ETCO_2$) monitoring or cerebral oximetry need to be considered.

CAPNOGRAPHY

End-tidal CO_2, measured during CPR, is primarily reliant on pulmonary blood flow, and therefore can serve as a surrogate for CO and coronary perfusion. Aside from confirming endotracheal tube positioning, capnography can have a dual role during resuscitation [59,60]. First, it can guide the rescuer in providing high-quality CPR since $ETCO_2$ is positively correlated with chest compression depth and CO [61,62]. Failure to preserve $ETCO_2$ concentrations above 10 mmHg has been associated with death [63]. In case no invasive haemodynamic monitoring is in place during CPR, a consensus statement was made to aim for $ETCO_2$ concentrations above 20 mmHg without providing excessive ventilation [58]. Second, an abrupt, but sustained rise in $ETCO_2$ appears to be an early indicator of ROSC, through which capnography might be used to justify the decision to terminate or continue with resuscitation efforts. Available data suggest that, under constant ventilation, an $ETCO_2$ increase of more than 10 mmHg strongly predicts ROSC [64▪,65–67]. However, it should be noted that an invasive airway management is necessary to perform capnography. In addition, $ETCO_2$ is influenced by the ventilation rate which can lead to a misinterpretation of the measured values.

CEREBRAL SATURATION

During the last decade, the use of near-infrared spectroscopy (NIRS) to measure cerebral oximetry during cardiac arrest has gained a lot of interest. This noninvasive technique measures cerebral saturation (rSO_2) in real-time, and in contrast to pulse oximetry independent of a pulsatile signal. Not unimportantly, it has been shown that measuring rSO_2 using NIRS technology is feasible during CPR [68▪]. However, so far, only one undersized clinical study compared the effect of low-quality versus high-quality CPR on rSO_2 and did not show any difference [69]. However, the periods of low-quality CPR were short (30 s until 6 min) and the time to allow changes in rSO_2 was rather limited. Together with the small cohort size, a possible delay in effect on rSO_2 could be one of the reasons for this neutral observation. However, higher rSO_2 values have been measured using a mechanical compression device compared with manual compression [70]. Multiple studies demonstrated that increases in rSO_2 and higher mean values during CPR are associated with ROSC, though, with present evidence, they do not reflect real-time quality of CPR [68▪,70–74]. In the ideal world, it would be possible to use cut-off values to predict outcome on short and long-term as also to indicate the quality of CPR. Different cut-off values are proposed both for initial measured rSO_2 values as for mean rSO_2 and absolute increase in rSO_2. Due to the heterogeneity of the used study protocols, population and devices (which have different rSO_2 ranges) and the rather small study populations it is not yet possible to confirm usable cut-off values. An interesting finding demonstrating the potential role of rSO_2 during CPR is the observed dip in rSO_2 every 2 min reflecting rhythm control [75]. On the contrary, this finding has not yet been confirmed by others.

Currently, the NICA trial (Impact of NIRS-guided Cardiopulmonary Resuscitation After Cardiac Arrest on Resuscitation Rate) is recruiting and we can expect results by the end of 2021 [76]. This trial will compare NIRS-guided CPR (target $rSO_2 > 40\%$ after 10 min of Advanced Life Support (ALS)) to guideline-provided CPR on ROSC rate and short and long-time neurological outcome.

In animal studies, contradicting results have been published comparing rSO_2 with invasively measured physiologic parameters such as brain oxygen tension [77–80]. Correlations are observed during CPR between rSO_2 and coronary blood flow, Mean Arterial Pressure (MAP) and CO measured as pulmonary blood flow [77,80]. A correlation between rSO_2 and pulmonary blood flow has been observed, if aimed for a preset target, 30, 50 or 70%

depth achieved. Varying recoil characteristics can be appreciated in the trajectories of the upstroke phase of this cluster of CC. Local calculation of the chest compression fraction for this small window of cardiopulmonary resuscitation, here using a peak-to-peak no-cardiopulmonary resuscitation threshold of 2 s would yield a fraction of approximately 60%. Panel (c) several physiologic measures provide insight into the efficacy of the mechanical input of cardiopulmonary resuscitation. Here, they are briefly summarized graphically.

[80]. This implies if a certain rSO_2 percentage/value is pursued, higher quality CPR will be performed. As in the human studies, no animal study investigated yet the effect on hemodynamic variables if a NIRS-guided CPR protocol is being followed. It is hard to compare rSO_2 with invasive oxygen tension measurements or cerebral blood flow as they measure something else. Tension is not necessary directly correlated with saturation, which is highly dependent of oxygen delivery and extraction, and with flow.

Probably NIRS measurements reflect more global perfusion than only rSO_2. Even if this is the case, information about the global perfusion status of the patient is still very valuable, especially taken into account that is associated with ROSC.

CARDIAC ULTRASOUND

In 2001, Salen *et al.* [81] was one of the first investigating the role of cardiac ultrasound during resuscitation. Although at that time they could not show an added role for cardiac ultrasound to capnography for predicting ROSC, cardiac ultrasound had just found its way into the resuscitation setting. In addition to its role in excluding the reversible causes of cardiac arrest and guidance of procedures during resuscitation, other applications have been explored during the last decade [82,83]. Cardiac ultrasound can for example differentiate between asystole, pulseless electrical activity (PEA) and pseudo-PEA [84▪].

Giving chest compressions at an optimal depth and rate is one of the pillars of high-quality CPR. The position of the hands is of equal importance as they should be on the lower half of the sternum to achieve the highest hemodynamic response [59]. In practice, often not the right ventricle is compressed but the aorta or left ventricle outflow tract inducing significant less circulating blood [85]. Ultrasound, preferably transoesophageal ultrasound (TEE), can improve hand positioning and compression depth during chest compression in real-time and consequently improve the resulting blood propulsion [86,87]. TEE also has the advantage that it can be used in all body types. Pulse checks within the predetermined 10 s have a poor sensitivity and specificity which can be improved by using ultrasound [87–89]. Another potential use of cardiac ultrasound in monitoring and improving the quality of CPR is the ability to assess the presence of cardiac activity during asystole and PEA [90]. The differentiation between pseudo and true PEA gives the opportunity to treat these entities differently and increase the opportunity to achieve ROSC.

On the contrary, the use of ultrasound during cardiac arrest requires an experienced performer and preferably a member of the ALS team who is only dedicated to perform ultrasound at time of resuscitation. Another limitation is the possible lengthening of the hands-off time during CPR, however this can be prevented by positioning the ultrasound transducer before the pulse check and a verbal clock during pulse checks as shown by the Cardiac Arrest Sonographic Assessment protocol, that could reduce the pulse check time from 19.8 to 15.8 s [91▪]. However, no validation of this protocol has yet been published.

BLOOD GAS ANALYSIS DURING CARDIOPULMONARY RESUSCITATION

Despite the availability of blood sample analyses during in-hospital CPR, they are not often used. Nonetheless, there is some data showing a potential benefit of the use of blood gas analysis during CPR rather as prediction tool for outcome then a role for monitoring CPR [92–97]. In a prehospital, blood analyses are often not available, although some point of care blood sample tests (venous, arterial, intraosseous) could be made available in the prehospital setting [98].

Resulting from immediate ischaemia, lactate is being formed as one of the end products of anaerobic glycolysis during cardiac arrest. Blood lactate concentrations measured at admission have proven to be associated with the duration of no-flow and low-flow status during CPR [44,99,100]. Although both could be important factors in determining outcome following cardiac arrest, they cannot always be reliably estimated, especially not in unwitnessed arrests [101]. Even in witnessed arrests, where CPR is provided by a bystander, the quality of CPR can be questionable either, through which low-flow time becomes a meaningless variable [102]. Lactate levels, measured during CPR, reflect the ischemic status directly on a cellular level, and consequently might serve as a better estimator of low-flow time or could perhaps indicate the quality of (bystander) CPR [100,103,104]. On the contrary, the evidence to use lactate or even other values derived from blood gas analysis (e.g. pH, PaO_2, $PaCO_2$ and HCO_3 levels) as an indicator for the quality of CPR is scarce. Therefore, the effect of qualitative CPR on serial blood measurements could be an interesting topic for future research.

So far, more is known about the role of blood gas measurements and the association with outcome. For example, serum lactate levels measured during CPR seem to correlate with survival, and some now even suggest to initiate extracorporeal CPR more rapidly in In-Hospital Cardiac Arrest (IHCA) patients with high lactate levels since these patients have a

rather low probability of achieving rapid [92,94]. Moreover, pH levels above 6.8, measured during the first minutes after the initiation of CPR, have been associated with good neurological outcome. If the potassium level is above 8.5 meq/l or if the PaO_2 level is below 60 mmHg during the first minutes of CPR, favourable neurological outcome becomes unlikely [93,95–97].

CONCLUSION

To perform high-quality CPR, at first, one should optimize rate, depth and pause duration supported by process monitoring tools. Second, the evolving technological evolution gives opportunities to measure physiologic parameters in real-time which will open the way for patient tailored CPR. The role of ultrasound, rSO_2 and $ETCO_2$ in measuring the quality of CPR needs to be further investigated as well as the possible ways of influencing these measured parameters to improve neurological outcome and survival.

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Conflicts of interest

There are no conflicts of interest.

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- ■ of special interest
- ■■ of outstanding interest

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