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Case Report

Arterial and Venous Pressure Monitoring during Cardiopulmonary Resuscitation for Out-of-Hospital Arrests: Four Case Reports

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Abstract: Background: A new physiological monitoring system that simultaneously measures femoral arterial pressure, femoral venous pressure, and cerebral tissue oxygen saturation during CPR was used to evaluate the quality of cardiopulmonary resuscitation. In this case report, we would like to present four representative cases with this physiological monitoring system during CPR. Cases: We invasively measured femoral arterial pressure and femoral venous pressure if catheters were immediately inserted into the femoral artery and femoral vein for potential candidates who required extracorporeal cardiopulmonary resuscitation but did not receive such interventions. We presented several cases, including two cases in which cardiopulmonary resuscitation resulted in higher femoral arterial pressure compared to femoral venous pressure, an upward trend in cerebral tissue oxygen saturation values was observed, and both instances achieved the return of spontaneous circulation. In contrast, we also presented two patients with significant increases in femoral venous pressure and low cerebral tissue oxygen saturation values. In both cases, the return of spontaneous circulation was not achieved. Conclusions: We presented cases in which the femoral venous pressure exceeded the femoral arterial pressure using a simultaneous physiological monitoring system to monitor arterial pressure, venous pressure, and cerebral tissue oxygen saturation during cardiopulmonary resuscitation. Further case accumulations will be necessary to assess the variations in hemodynamic status during cardiopulmonary resuscitation and the association between each hemodynamic status and outcomes after cardiac arrest.

Keywords: out-of-hospital cardiac arrest; physiological monitoring; blood pressure; near-infrared spectroscopy

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1. Introduction

Despite the current advancements in resuscitation science, outcomes following out-of-hospital cardiac arrest (OHCA) continue to be poor [1–4]. Various resuscitation strategies can improve outcomes after cardiac arrest. During cardiac arrest, strategies such as public-access defibrillation and bystander cardiopulmonary resuscitation (CPR) can be beneficial [1–3,5,6]. Post cardiac arrest strategies, including strict control of oxygen and carbon dioxide targets and temperature management, can also improve outcomes [7–12]. High-quality chest compressions are among the most essential components to improving outcomes. The CPR guidelines, updated every five years, specify the depth and rate of chest compressions, among other things, but the fundamental aspects have remained essentially

unchanged for many years [1,2]. This is likely due to the strong support for the forward circulation of oxygenated blood from the heart to the tissues through chest compressions, oxygen administration, and venous blood return to the heart. However, it remains to be seen whether the current method of chest compressions is genuinely effective in generating circulation during resuscitation, as this has yet to be sufficiently elucidated to date.

Assessment and guidance of CPR quality using noninvasive and invasive monitoring techniques have been increasingly recommended in recent years [1,2,13]. Notably, in the 2020 international CPR guideline, the importance of invasive arterial blood pressure monitoring during CPR was mentioned as providing insights into blood pressure responding to chest compressions and pharmacological interventions for pediatric cardiac arrests [14]. However, these statements apply only to pediatric and in-hospital cardiac arrests, and no recommendations exist regarding adults and OHCAs. We started to monitor arterial pressure (AP), venous pressure (VP), and cerebral tissue oxygen saturation ($SctO_2$) during CPR for adult OHCA patients. In this case report, we would like to present four representative cases with this physiological monitoring system during CPR.

2. Case Presentation

The study setting was Kagoshima City Hospital. The data collection started in October 2021. In our emergency department, we immediately insert catheters into the femoral artery and vein for potential candidates who require interventions such as extracorporeal cardiopulmonary resuscitation (ECPR) using extracorporeal membrane oxygenation to ensure prompt treatment after admission for cardiac arrest patients. In this study, we measured femoral arterial pressure (FAP) and femoral venous pressure (FVP) invasively for patients who did not receive interventions such as ECPR and were not subjected to catheterization and used a near-infrared spectroscopy (NIRS) measurement probe on the forehead to measure $SctO_2$ using the NIRO-200NX® device made by Hamamatsu Photonics, irrespective of the presence or absence of FAP and FVP measurements. NIRO-200NX measures SctO2 by 20 Hz, and it can display $SctO_2$ without time lag. Each blood pressure measurement started immediately after cannulations for all patients, and it stopped either when patients achieved ROSC in cases in which it did or at the time of cessation of resuscitation efforts for cases in which it did not. Anticoagulants were not given if EPCR was not performed. These parameters were simultaneously displayed and recorded using a multi-screen recording system (Ciel View™) (Figure 1), which enabled the observation of the resuscitation procedure's external appearance and the physiological responses in real-time. The highest blood pressure (BP) displayed on the multi-parameter monitor was defined as BP at the chest compression phase (CP), and the lowest BP was defined as BP at the chest relaxation phase (RP). The mean arterial pressure (MAP), AP at CP, AP at RP, mean venous pressure (MVP), VP at CP, VP at RP, and $SctO_2$ were extracted every ten seconds for each case. Herein, we present four representative cases for which this monitoring system was utilized during CPR. This study was approved by the institutional review board of Kagoshima City Hospital (Approval No. 2020-56).

Case 1: A patient with acute coronary syndrome as the etiology of cardiac arrest.

The patient collapsed and was transported to our hospital after experiencing chest pain and discomfort for two days. The arrest was not witnessed. A bystander performed CPR. Initial ECG waveform upon contact with the emergency medical team showed cardiac arrest. Although the patient achieved ROSC after administering 2 mg of adrenaline, they experienced pulseless electrical activity (PEA) one minute later. Despite administering an additional 3 mg of adrenaline, the patient was still in PEA upon arrival at our hospital, as shown in Figure 2, which shows the progression of FAP, FVP, and $SctO_2$.

Mechanical chest compressions using the Corpuls cpr® made by Corpuls® were performed from hospital admission, and the initial $SctO_2$ measurement was relatively high, ranging from 45 to 46%. However, the initial AP at CP and MAP measured from the femoral artery were 80 mmHg and 30 mmHg, respectively. Additionally, at approximately 120 s after the start of FVP measurement, the AP at CP and MAP had risen to 90 mmHg and

40 mmHg, respectively, higher than the FVP. Subsequently, while the FAP continued to grow, the FVP remained almost constant, and the VP at CP, the VP at RP, and the MVP never exceeded 25 mmHg. During this period, the $SctO_2$ value increased above 50%, and the patient achieved ROSC at approximately 270 s. $EtCO_2$ level was not evaluated during CPR, and the mean $SctO_2$ was 51.5%. ECPR was not performed because the patient's heart rhythm was restored while ECPR preparations were made. However, this patient returned to cardiac arrest, and resuscitation was discontinued in the emergency department. Total CPR time was 45 min.

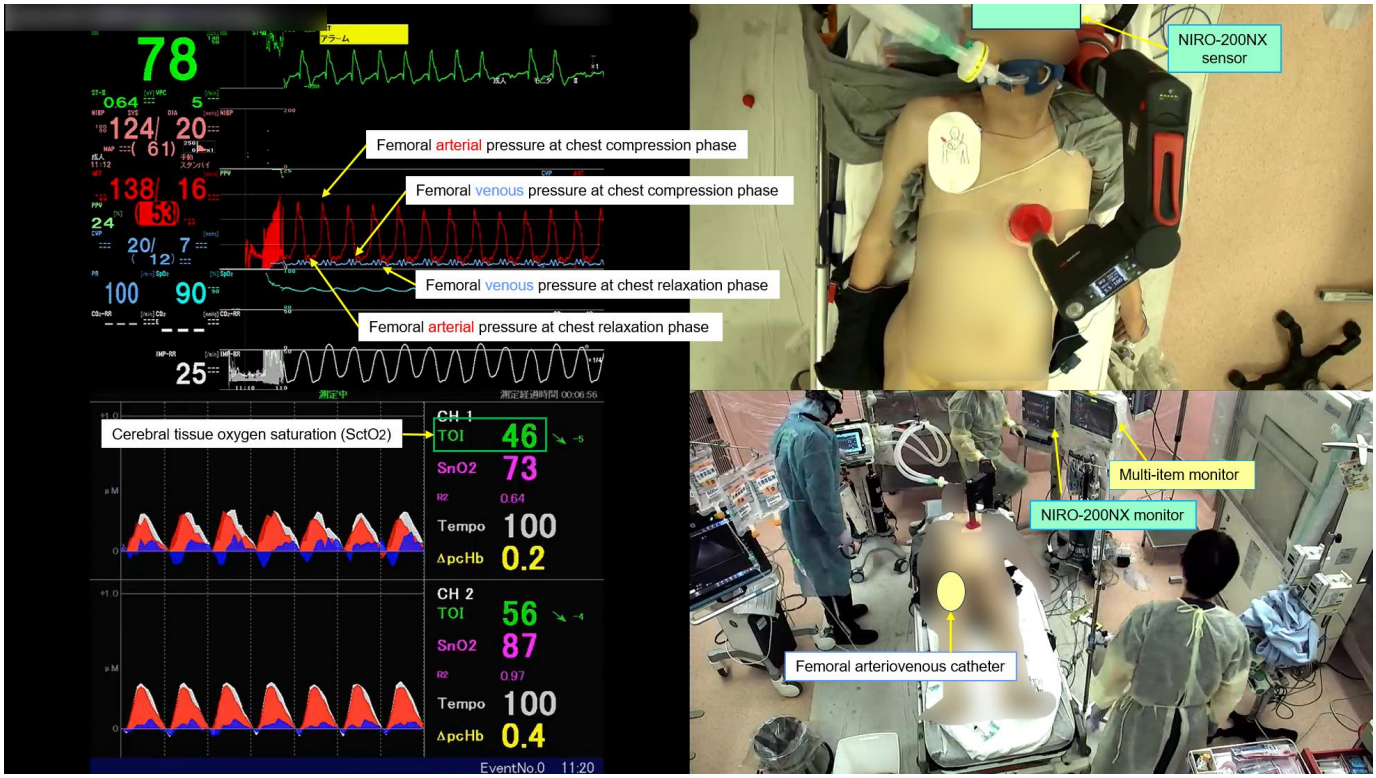

Figure 1. Multi-screen recording system (Ciel View^{TM}). (**Left upper**) NIHON KOHDEN Multi-item monitor (BSM6701). (**Right upper**) Ceiling-suspended mobile camera monitor. (**Left lower**) NIRO200NX monitor. (**Right lower**) Fixed surveillance monitor.

Case 2: A patient with asphyxia as the etiology of cardiac arrest.

The patient had experienced a loss of appetite two weeks prior but was being monitored. After going to bed, the patient's family found vomit next to the pillow, and the patient was found not breathing, prompting an emergency call. The arrest was unwitnessed, and a bystander did not perform CPR. The initial ECG waveform taken by the ambulance crew showed asystole, and the patient was transported to our hospital while airway management and cardiopulmonary resuscitation were performed. Figure 3 shows the changes in FAP, FVP, and $SctO_2$.

At first, manual chest compressions were performed from hospital admission, followed by mechanical chest compressions using the Corpuls cpr® after five minutes. Measurement of FAP and FVP started six minutes after arrival, and both were initially low. Although FAP began to rise around 120 s after measurement began, FVP remained almost unchanged. With each administration of adrenaline, AP at CP gradually increased, exceeding 140 mmHg at about 1000 s. In contrast, $SctO_2$ increased with the rise in FAP, fluctuating between 40 and 45%, and ultimately ROSC was achieved. The $EtCO_2$ level was around 25 mmHg during CPR, and the mean $SctO_2$ was 38.7%. Initially, there was consideration of implementing ECPR, but it was not performed as this patient achieved ROSC. However,

this patient returned to cardiac arrest, and resuscitation was discontinued in the emergency department. Total CPR time was 75 min.

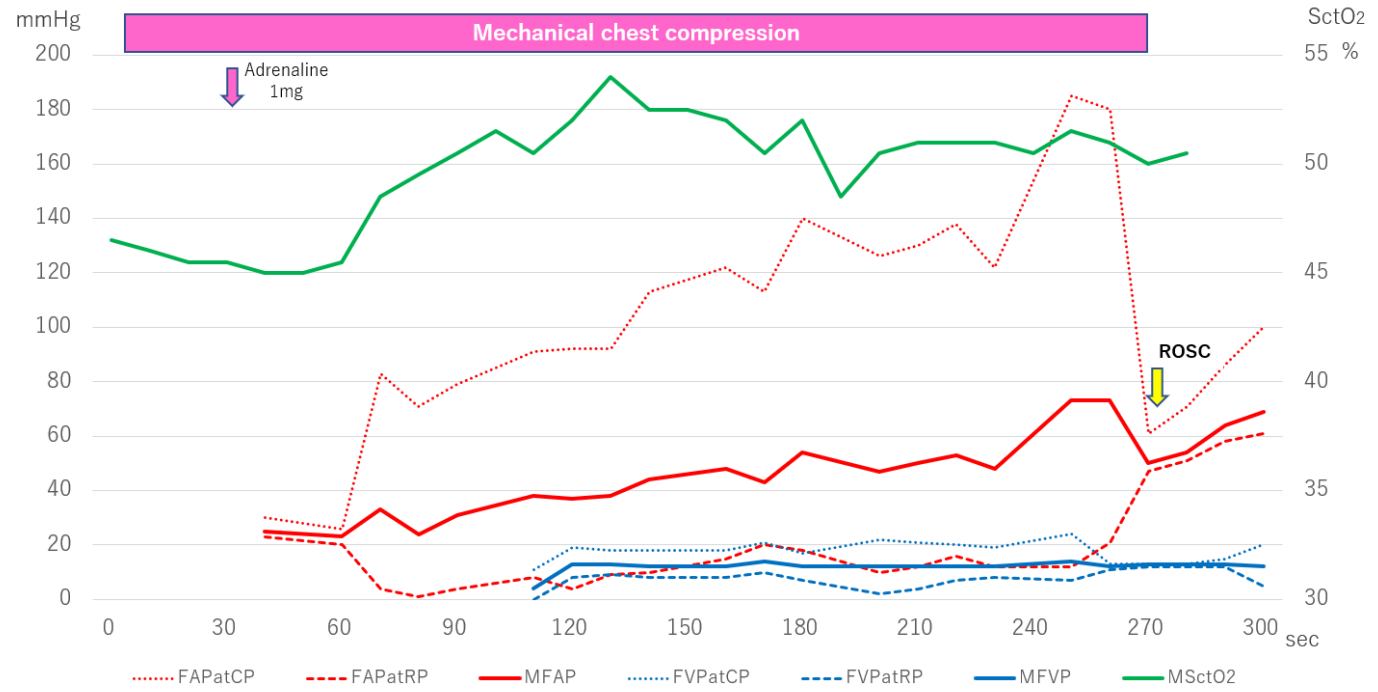

Figure 2. Femoral arteriovenous pressure and $SctO_2$ in case 1. FAP indicates femoral arterial pressure; CP, chest compression phase; RP, chest relaxation phase; FVP, femoral venous pressure; $MSctO_2$, mean cerebral tissue oxygen saturation; ROSC, return of spontaneous circulation.

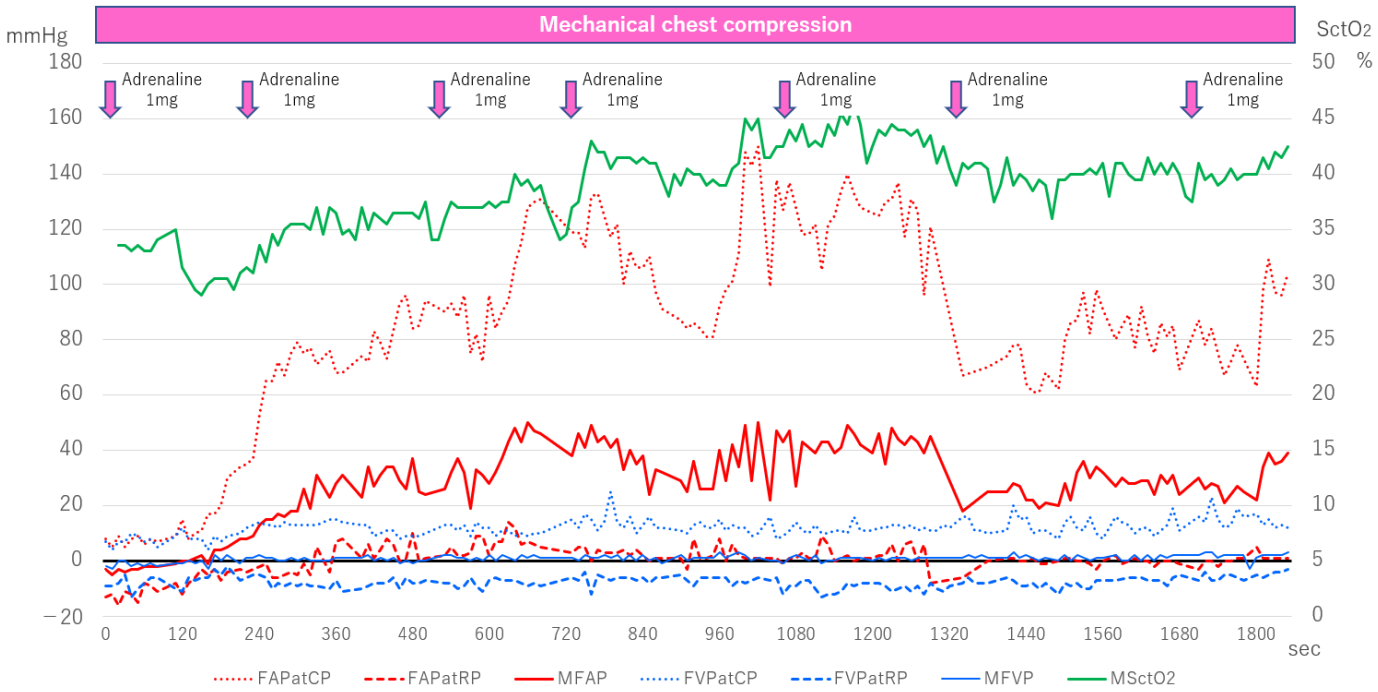

Figure 3. Femoral arteriovenous pressure and $SctO_2$ in case 2. FAP indicates femoral arterial pressure; CP, chest compression phase; RP, chest relaxation phase; FVP, femoral venous pressure; $MSctO_2$, mean cerebral tissue oxygen saturation; ROSC, return of spontaneous circulation.

Case 3: A patient with acute aortic dissection as the etiology of cardiac arrest.

The patient was discovered collapsed at home. The arrest was unwitnessed, and bystander CPR was present. Upon contact with the emergency medical service, the initial electrocardiogram (ECG) waveform indicated cardiac arrest. The patient was transported to our emergency department without return of spontaneous circulation (ROSC). Upon arrival, the patient remained in cardiac arrest, and echocardiography revealed a small amount of pericardial effusion, subsequently increasing. Therefore, pericardiocentesis and pericardiotomy were performed, and a total of 5 mg of adrenaline was administered. However, ROSC could not be achieved, and resuscitation was discontinued. ECPR was not performed because pericardial effusion was present, which gradually increased, leading to the suspicion of aortic dissection. Figure 4 shows the changes in FAP, FVP, and $SctO_2$.

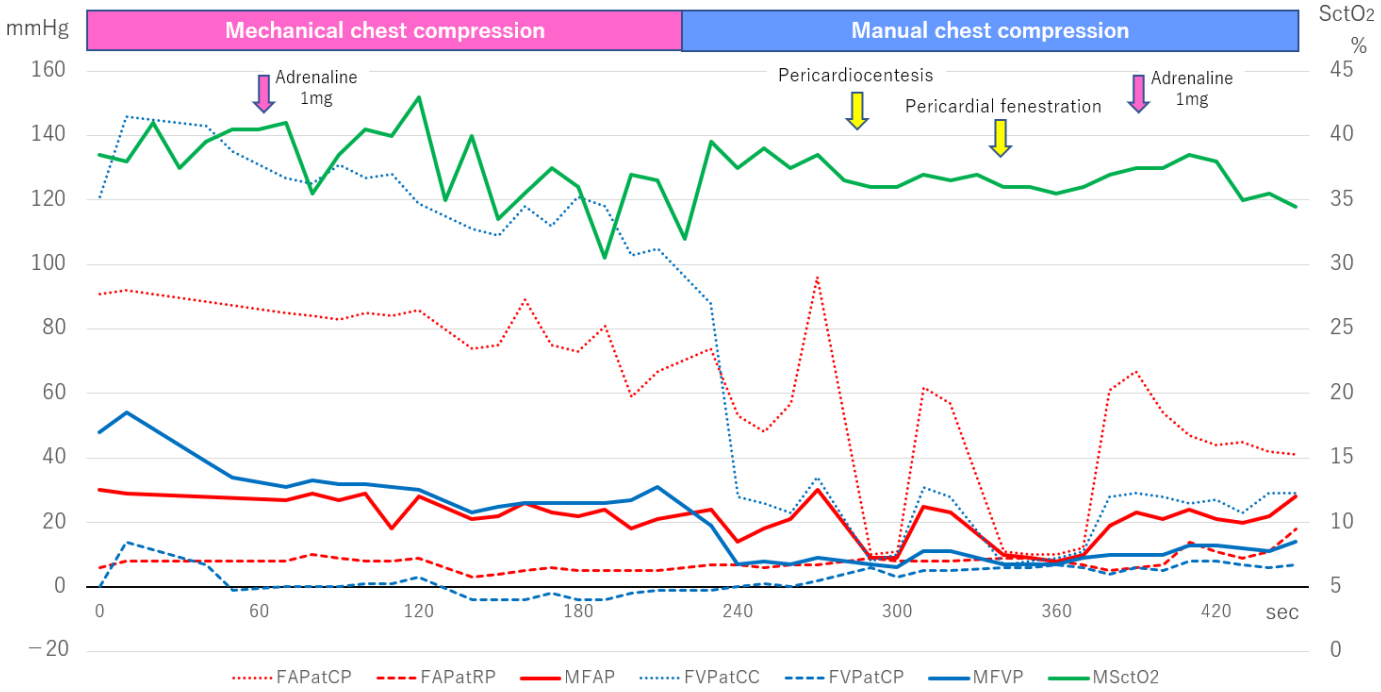

Figure 4. Femoral arteriovenous pressure and $SctO_2$ in Case 3. FAP indicates femoral arterial pressure; CP, chest compression phase; RP, chest relaxation phase; FVP, femoral venous pressure; $MSctO_2$, mean cerebral tissue oxygen saturation.

FAP and FVP measurement was initiated six minutes after arrival, during mechanical chest compressions using the AutoPulse® model 100 V1.5 made by ZOLL®, and the FVP was higher than the FAP during both the compression and mean phases. In particular, the FVP at CP exceeded 140 mmHg, and $SctO_2$ ranged from 40% to the low 30 s. Manual chest compressions were performed at approximately 240 s, the FVP at CP rapidly decreased, and the FAP and FVP reversed. During pericardiocentesis and pericardiotomy, chest compressions were temporarily suspended. Still, upon resumption of chest compressions, the AP at CP exceeded the VP at CP, and $SctO_2$ slightly increased and ranged in the high 30 s. Total CPR time was 80 min. The $EtCO_2$ level was around 10 mmHg during CPR, and the mean $SctO_2$ was 37.3%.

Case 4: A patient with presumed cardiac etiology of arrest.

The patient was witnessed sinking while taking a bath in a public bathhouse, and emergency services were called. A bystander performed CPR. Upon initial contact with the ambulance team, the electrocardiogram showed asystole and remained so upon arrival at our emergency medical center. Although 5 mg of adrenaline was administered, resuscitation was discontinued due to a lack of return of ROSC. ECPR was not performed as there was a high probability of the patient having hypoxic-ischemic encephalopathy. Figure 5 shows the changes in the FAP, FVP, and $SctO_2$. When the FAP and FVP measurements were started 6 min after arrival, the patient underwent manual chest compressions, and the VP at

CP was higher than the AP at CP, but this reversed around 150 s. The MAP and MVP were at the same level during those times, and $SctO_2$ fluctuated around 35 to 38%. Subsequently, mechanical chest compressions using the AutoPulse® model 100 V1.5 were initiated around 270 s, and the FVP rose rapidly. In particular, the VP at CP increased to 120 to 140 mmHg, while the AP at CP remained around 50 to 60 mmHg, with the VP consistently higher than the AP. Furthermore, when manual chest compressions were resumed around 660 s, the FAP and FVP decreased rapidly. When the mechanical chest compressions were resumed again around 750 s, the VP at CP quickly increased to 120 to 140 mmHg as before, but the AP at CP only rose to 50 mmHg, and $SctO_2$ fluctuated around 35%. Total CPR time was 62 min. The $EtCO_2$ level was 11 to 13 mmHg during CPR, and the mean $SctO_2$ was 35.9%.

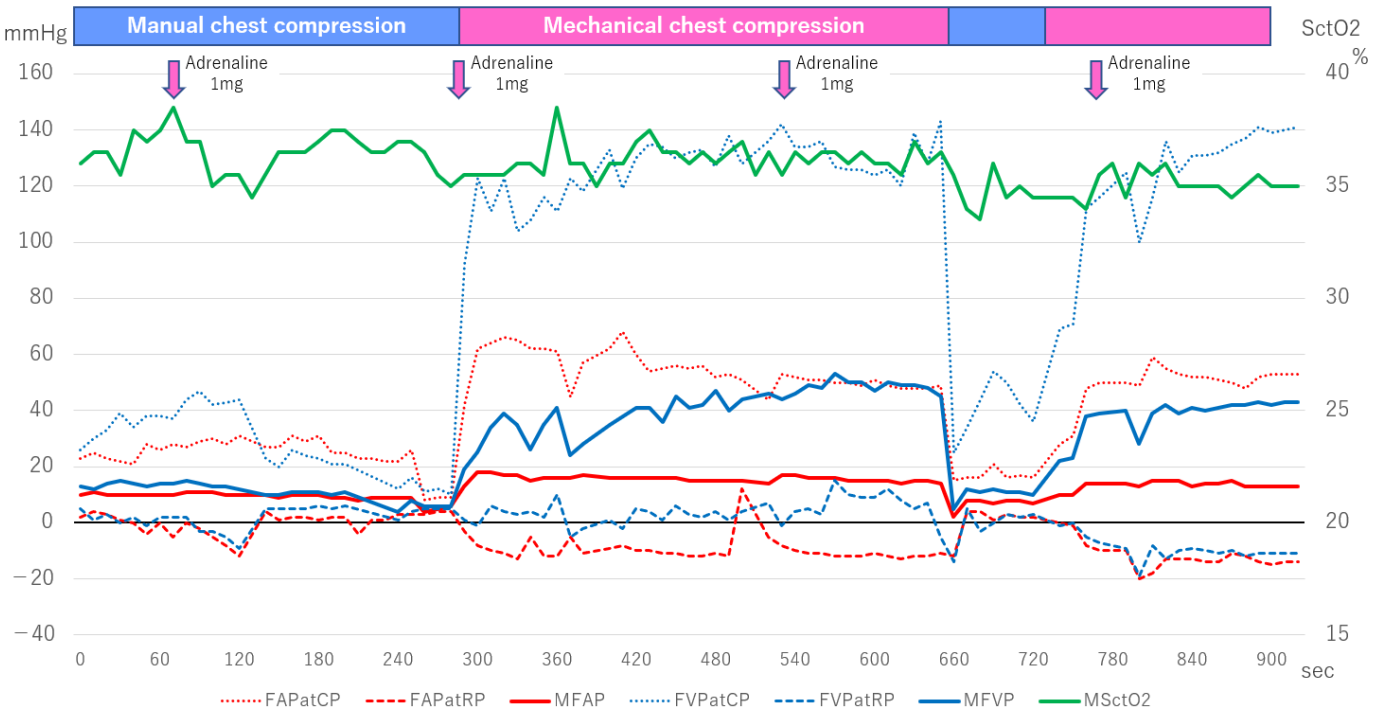

Figure 5. Femoral arteriovenous pressure and $SctO_2$ in Case 4. FAP indicates femoral arterial pressure; CP, chest compression phase; RP, chest relaxation phase; FVP, femoral venous pressure; $MSctO_2$, mean cerebral tissue oxygen saturation.

3. Discussion

In this study, we presented a simultaneous physiological monitoring system capable of monitoring AP, VP, and $SctO_2$. We also presented several cases, including two cases in which CPR resulted in higher FAP than FVP, and an upward trend in $SctO_2$ values was observed. In contrast, we also showed two patients with significant increases in FVP and low $SctO_2$ values.

Recently, there has been an increasing focus on measuring the quality of CPR. Notably, in the 2020 international CPR guideline, a single cut-off point for diastolic BP during the first ten minutes was introduced (at least 25 mmHg in infants and at least 30 mmHg in children) based on a retrospective analysis of arterial waveforms [14,15]. However, there is limited evidence to guide the target BP for adults and OHCAs, constituting most cardiac arrest cases. In contrast to the previous study, our protocol included the measurement of VP. It revealed that several OHCA patients exhibited predominantly retrograde flow during CPR, underscoring the importance of simultaneous measurement of AP and VP to monitor the quality of CPR.

There are two circulation mechanisms for closed-chest compressions: the heart pump theory and the chest pump theory [16]. The former posits that direct compression of the heart (mainly the ventricle) induces cardiac output, while the latter suggests that an

increase in intrathoracic pressure resulting from compression of all tissues and organs in the thoracic cavity leads to cardiac output. Both mechanisms are currently believed to be at work, but compressing the sternum affects the ventricle, the atrium, and the superior vena cava in the thoracic cavity [16]. Tokuda et al. reported that during the chest compressions, both aortic and central venous pressures increased almost simultaneously, and the essential flaw of closed-chest compressions is that it causes venous reflux due to the compression of not only the ventricle but also the atrium [17]. Furthermore, Mackenzie et al. reported that during the chest compressions, right atrial pressure increased to 88 to 116 mmHg [18]. The discussion shifted to how to prevent venous reflux from maintaining circulation during resuscitation [19], with proposals such as the early closure theory of intrathoracic veins (system) due to increased intrathoracic pressure, and the function of internal jugular vein valves, subclavian vein valves, or venous valve-like structures within the superior vena cava [20]. These valves and systems can withstand up to approximately 60 mmHg pressures and prevent reflux from the heart [20]. Still, to date, no reports have measured venous pressure outside the thoracic cavity, leaving it unclear whether reflux is indeed suppressed. While central venous pressure and right atrial pressure were not measured in the present study, the fact that the pressure in the femoral vein during the chest compression phase exceeded 100 mmHg suggests that early closure due to increased intrathoracic pressure or venous valve structures in the superior vena cava may not have been able to prevent venous reflux. This might result in suboptimal perfusion of peripheral tissues during CPR.

$SctO_2$ provided by NIRS is a highly anticipated noninvasive physiological monitor for assessing the quality of CPR and predicting outcomes [21]. Prior studies indicated that higher $SctO_2$ values are associated with a greater likelihood of achieving ROSC and favorable neurological outcomes [21–25]. Our investigation revealed that in Cases 1 and 2, which had higher MAP, ROSC was reached after increased $SctO_2$ values. In contrast, in Cases 3 and 4, where MVP exceeded AP, lower $SctO_2$ values were observed, and ROSC was not achieved. It should be noted, however, that this study does not establish any causal relationship between BP and $SctO_2$, let alone between BP and prognosis. In addition, the femoral venous pressure increased during mechanical chest compressions in cases 3 and 4. The possibility that it is caused by individual differences, such as the position of the right atrium and right ventricle, differences in the compression site, and differences in the depth of compression, may be related. However, the small number of cases does not prove a causal relationship between the difference in mechanical and manual chest compressions or the difference in equipment and the cause of the increased venous pressure. Nevertheless, our findings have the potential to provide insight into the prediction of systemic and cerebral circulation during CPR, and further accumulation of cases is warranted to generate evidence regarding the relationship between AP, VP, and $SctO_2$ values, as well as their association with prognosis.

While we believe that our protocol for invasive BP monitoring in OHCA patients is feasible and that more cases can be accumulated in the future, it is impossible to perform invasive monitoring for all cardiac arrest patients. Therefore, a noninvasive and easy-to-use physiological monitor should be established based on our results. Additionally, since the optimal method of CPR may vary from person to person, future randomized controlled trials will be necessary to assess the impact of individually changing the CPR method based on real-time physiological feedback (i.e., physiologically guided resuscitation) instead of the current uniform CPR method for all individuals.

This case report has several limitations.

First, we only demonstrated the four cases, so we can not demonstrate an association between FAP, FVP, and SctO2 or outcome by adjusting for other important variables. Therefore, further case accumulation is warranted.

Second, we lack several other important variables associated with CPR quality and outcomes.

Third, the hemodynamic status in the early stages of cardiac arrest, including during the prehospital period, was unavailable.

Fourth, higher values on NIRS have been reported to be associated with improved prognosis, but RCTs have not proven causality.

4. Conclusions

We presented four representative cases evaluated with a simultaneous physiological monitoring system that can monitor AP, VP, and $SctO_2$. We have also presented cases in which venous pressure exceeded arterial pressure during CPR. Further case accumulations will be necessary to assess the variations in hemodynamic status during CPR and the association between each hemodynamic status and outcomes after cardiac arrest.

Author Contributions: H.O.: Conceptualization, Methodology, Writing—original draft preparation. T.M.: Writing—original draft preparation. Y.Y.: Investigation, Writing—review and editing. D.I.: Data curation, Writing—review and editing. T.K.: Investigation, Resources, Writing—review and editing. M.N.: Resources, Visualization, Writing—review and editing. Y.I.: Methodology, Resources, Visualization. H.K.: Project administration, Formal analysis, Supervision, Visualization, Writing—review and editing. All authors have read and agreed to the published version of the manuscript.

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Institutional Review Board Statement: This study was approved by the institutional review board of Kagoshima City Hospital (Approval No. 2020-56).

Informed Consent Statement: Patient consent was waived due to the observational study and de-identification of personal data.

Data Availability Statement: Not applicable.

Conflicts of Interest: The authors declare no conflict of interest.

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