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School of Medicine

Scholarly Projects Final Report

Title

Temporal Oxygen Sensors in Cardiogenic Shock

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Scholarly Project Final Report

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Project/Research Question

Does the cerebral perfusion correlate with traditional markers of perfusion in cardiogenic shock patients and how do changes in cerebral oxygenation relate to patient mortality?

Type of Project

Research Study

Key words

Cardiogenic Shock, Temporal Oximetry, Microperfusion, Macroperfusion, Oxygenation, Cerebral Oxygentation, Mortality, Microvascular, Macrovascular

Meeting Presentations

N/a

Publications

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Submission to Archive

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N/a

Scholarly Project Final Report

Report*:*

Introduction

Cardiogenic shock patients have significant in-hospital mortality rates, with studies estimating mortality as high as 57%[1]. Current management focuses on improving macrohemodynamics, such as cardiac index, blood pressure and pulmonary capillary wedge pressure, however microcirculation has been demonstrated to be more predictive of patient outcomes[2,3]. Microhemodynamics are monitored via lab values and clinical assessments, such as lactate, capillary refill time and central venous-arterial carbon dioxide difference. The standard practice has been to treat macrovascular hemodynamics as the proxy for microvascular changes, with the assumption that they would change in concert with one another. However, small studies suggest that in patients with cardiogenic shock, microvascular changes may not be reflected in macrovascular trends, leading to inadequate perfusion in patients with normalized macrovascular parameters[4].

Of the microvascular targets, lactate has been demonstrated to have a significant correlation with mortality outcomes, however in practice, these labs can be delayed, difficult to obtain and confounded by other physiological mechanisms such as tourniquet placement[5,6]. Use of microcirculatory monitoring is also not novel, as it has been executed utilizing sublingual orthogonal polarization spectral (OPS) imaging, however this intervention is uncomfortable for patients and intermittent[7]. Importantly, sublingual OPS imaging demonstrated significant alterations in cardiogenic shock patients compared with other hemodynamic measurements, emphasizing the role of capillary monitoring. Given that patients in intensive care units have many invasive monitoring systems that produce continuous monitoring of macrohemodynamics, this data is often preferentially utilized by providers over slow-to-gather data such as lactate or physical exam maneuvers such as capillary refill time, despite its potential for inaccuracies.

Cerebral tissue oximetry would bridge this gap, as it provides close monitoring of capillary beds that are crucial to long term patient function, and often under-monitored. Cerebral tissue oximetry is widely used in cardiac anesthesia and is associated with decreased postoperative cognitive dysfunction[8]. In this study we investigated the use of the ForeSight tissue oximetry sensors in patients with cardiogenic shock. We then compared the correlation of tissue oxygenation (StO_2) with traditional markers of macro and microvascular perfusion such as lactate and cardiac index, and the cohort's mortality rates with trends in measured StO_2.

Methods

A prospective cohort of 37 patients with cardiogenic shock from the Cardiovascular Intensive Care Unit (CVICU) at Oregon Health Science University were recruited for the trial. Patients were only included when they met SCAI cardiogenic shock criteria from Class C, D or E. Patients were defined as having met SCAI criteria when the diagnosis was included in their electronic health record as an active issue by a clinician. Patients or their surrogate decision makers were consented for the trial under Institutional Review Board approval. Patients who were consented and enrolled had the ForeSight cerebral tissue oxygenation sensor applied for the duration of their CVICU stay, or up to a maximum of 7 days. Providers were blinded to cerebral oxygenation (StO2) readings and administered care according to typical protocols.

The Foresight system included two StO_2 sensors placed on the patient's forehead, approximately 10cm above the supraorbital ridge. Each StO_2 sensor collected tissue oximetry data independently and data was collected on an Edwards machine. Data was manually collected from each Edwards machine within 12

hours of sensor detachment and uploaded to the secure study database.

StO_2 data was collected from the ForeSight sensors for the entire duration of their attachment to the patient. Lab data, including lactate, cardiac index, right atrial pressure and blood pressure were collected from the electronic health record, using an approximated time window to sensor attachment. Patient outcomes were also collected, using retrospective chart review. Mortality was defined as in-hospital deaths during the admission during which the Foresight sensor was applied.

Statistical analysis was performed using Statistical Software for the Social Sciences (SPSS). To assess agreement between the two temporal StO_2 sensors, a Bland-Altmann analysis was first conducted to determine the bias and limits of agreements. To identify underlying clinical trends from significant noise associated with continuous data collection, StO_2 data (collected via dual temporal sensors) was combined by taking the mean of the values. This composite value then underwent nonparametric time smoothing with a 501-span centered moving average. This was then validated through residual analysis.

To assess the relationship between StO_2 and non-continuous variables, non-continuous data was temporally aligned, and linear interpolation was performed to estimate time points in between collection points. Smoothed StO_2values were then compared to interpolated non-continuous variables utilizing Pearson's Correlation Coefficient.

StO2 values were compared to mortality outcomes by binary logistic regression. StO_2 nadirs for each patient were extracted, as were mean StO_2 values for the bottom quartile (25th percentile) to compare to extracted mortality metrics.

Results

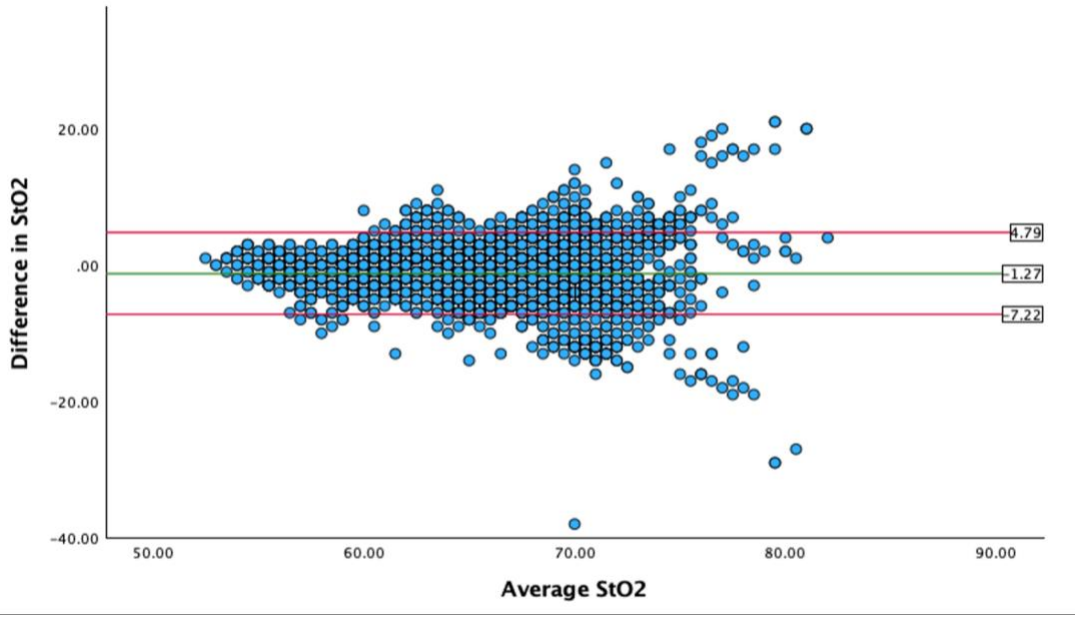

Figure 1. Bland-Altman analysis demonstrating agreement between StO_2 sensors two temporal sensors on a single patient. Each dot represents a difference in StO_2 for each clinical time point. Mean bias of -1.27 with 95% limits of agreement between -7.22 and 4.79. Variability increased at higher saturations suggesting sensors have more agreement at lower tissue saturations.

Sensor Agreement

In order to compare agreement between the two independent StO_2 sensors, a Bland-Altman analysis was performed (Fig 1). There was a mean bias of -1.27% and the 95% limit of agreement was between -7.22 and 4.79. This suggests that there was small variability between StO_2 sensors. The majority of differences between the two sensors remained within the limit of agreement, and there was increased variability at higher tissue saturations. This suggests that at lower StO_2 values, there was less disagreement between the sensors. At higher tissue saturations, the sensor values had more disagreement, suggesting that higher tissue saturation readings were less reliable than critically low values. Several outliers detected in this analysis account for episodes of sensor dropout, where one sensor did not record a value and the other did.

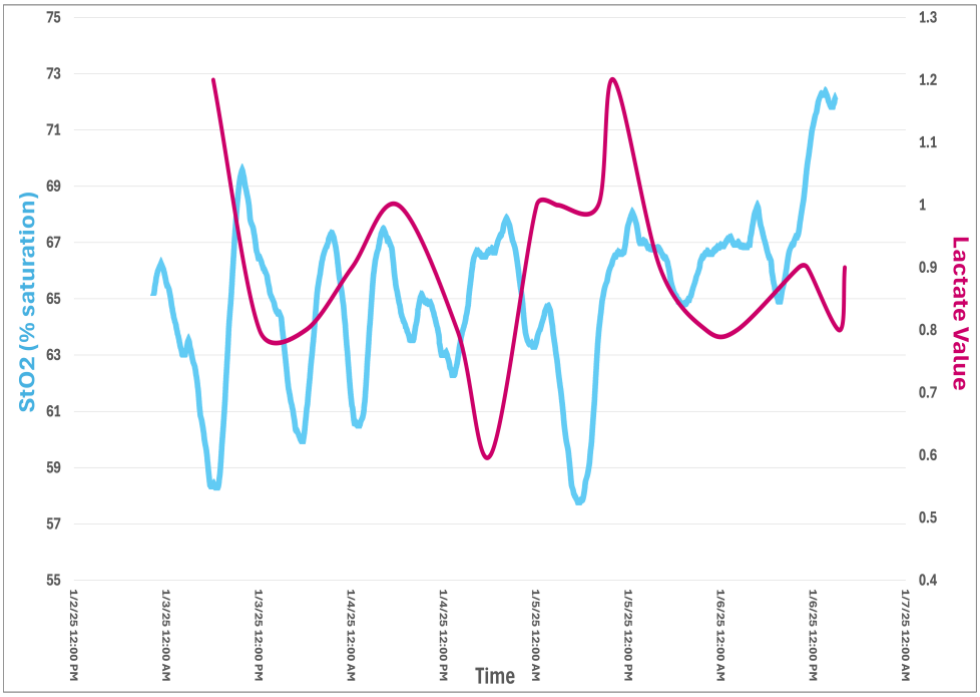

Figure 2. Graph demonstrating temporal saturation (StO_2) as measured by the mean of temporal sensors compared to lactate values over time of sensor attachment. Increases in lactate values are time offset from decreases in StO_2, notably on 1/5/2025 suggesting that temporal oximetry is more sensitive to changes in microcirculation than lactate.

Lactate

To assess the relationship of StO_2 with lactate, StO_2 was compared over time of sensor attachment to interpolated lactate values over the same period of collection. Increases in lactate appeared to occur after significant decreases in StO_2, as demonstrated on 1/5. There was more variability noted in StO_2 as well, due to continuous nature of the data collected and likely increased sensitivity compared to lactate sampling. Pearson analysis reveals a moderate inverse relationship between lactate and StO_2, with a correlation coefficient of -0.317 (p-value <0.001) (Table 1). Lactate and cardiac index had the strongest relationship of all variables tested, with a Pearson correlation coefficient of -.384 (p-value <0.001) suggesting a moderate inverse relationship. Lactate and continuous cardiac index did not have this same relationship, as the calculated Pearson coefficient was only -1.90 (p<0.001) representing a weak inverse relationship.

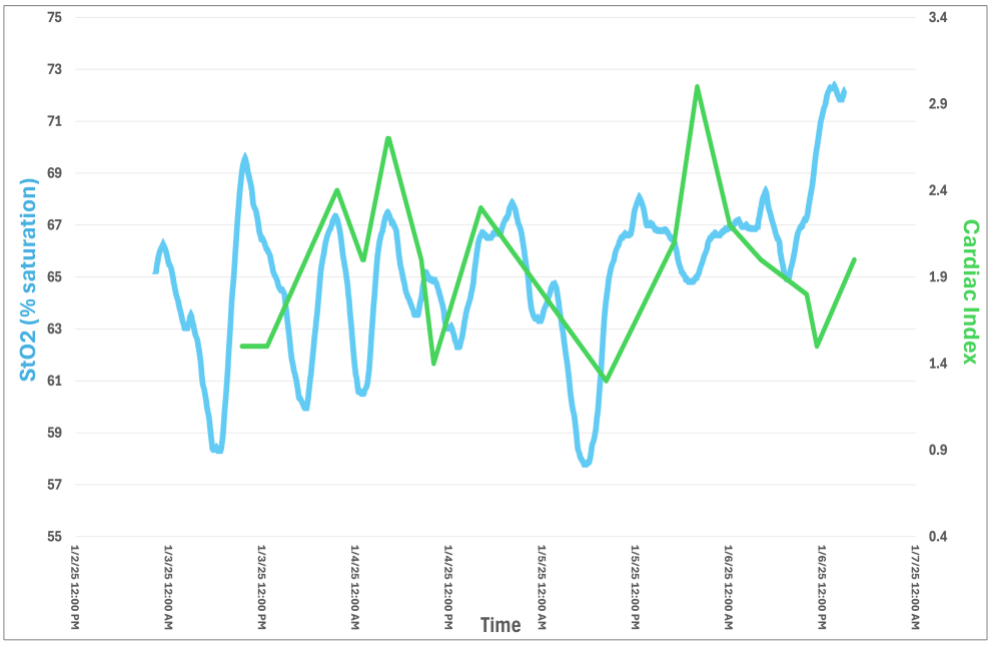

Figure 3. Graph of mean temporal saturation (StO2) as measured by two temporal sensors compared to measured cardiac index over time of sensor attachment. Decreases in temporal oximetry are expected to align with decreases in cardiac index.

Cardiac Index

Cardiac index was also directly compared to changes in StO_2, in the same manner as lactate. As StO_2 decreased, so did cardiac index, and temporal alignment appeared to be more consistent similar than with lactate, however there were also more cardiac index values taken from which to create the linear interpolation. The correlation between cardiac index and StO_2 had a Pearson Coefficient of 0.91 (p-value <0.001), when compared utilizing the linear interpolation values (Table 1). This coefficient suggests a weak relationship between StO_2 and Cardiac Index. Cardiac index and continuous cardiac index also had a Pearson Coefficient of 0.162 (p-value <0.001), suggesting a weak positive relationship.

		StO2	Lactate	Cardiac Index	Cont Cardiac Index
StO2	Pearson Correlation	1	-.317**	.091**	.272**
	Sig. (2-tailed)		<.001	<.001	<.001
	N	15424	14259	13654	15424
Lactate	Pearson Correlation	-.317**	1	-.384**	-.190**
	Sig. (2-tailed)	<.001		<.001	<.001
	N	14259	14921	14316	14921
Cardiac Index	Pearson Correlation	.091**	-.384**	1	.162**
	Sig. (2-tailed)	<.001	<.001		<.001
	N	13654	14316	14326	14326
Continuous Cardiac	Pearson Correlation	.272**	-.190**	.162**	1

Index					
	Sig. (2-tailed)	<.001	<.001	<.001	
	N	15424	14921	14326	17056
**. Correlation is significant at the 0.01 level (2-tailed).					

Table 1. Table of Pearson correlation of mean StO_2 value compared to linear interpolation of lactate values, cardiac index and continuous cardiac index values. Lactate and StO_2 demonstrate a moderate inverse relationship, and continuous cardiac index demonstrates a weak positive relationship, and cardiac index demonstrates a very weak, almost negligible relationship. All p values are significant, suggesting that these relationships are not by error. Cardiac index and continuous cardiac index do not have a significant relationship with a low Pearson correlation coefficient, but lactate and cardiac index are the most related with a moderate inverse relationship.

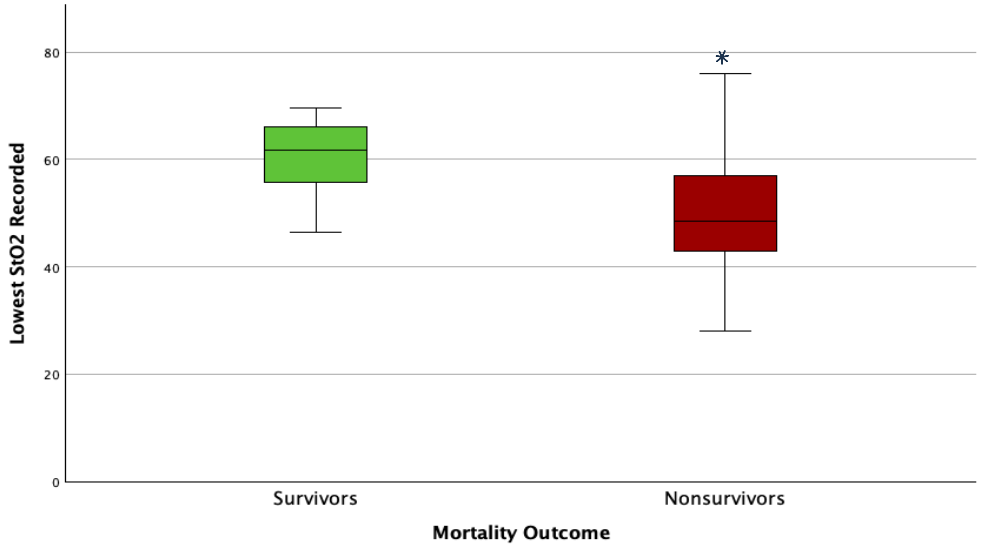

Figure 4. Box-Whisker plot demonstrating the relationship between StO_2 nadir recorded and mortality outcome among all patients (n=37) included in the study. Non-survivors (n=20) had statistically significantly lower mean StO_2 nadirs than survivors (n=17), however, it is important to note the variability present among the non-survivor population as demonstrated by the error bars.

Variable	Mean - Survivors	Mean – Non-survivors	P –value (sig <0.5)	Odds Radio (95% CI)
Lowest StO_2 Recorded	**59.9**	**47**	**0.017***	**0.901 (0.827-0.982)**
StO_2 from Bottom Quartile	**66.56**	**62.52**	**0.884**	**.993 (0.901-1.094)**

Table 2. Table of mean StO_2 nadirs and StO_2 bottom quartiles from survivors and non-survivors and statistical significance as calculated by binary logistical regression. Average nadir of all patients noted as 54.7%, nadir of non-survivors 47% and of survivors 59%. Logistic regression revealed p value of 0.017 and OR of 0.917 (95% CI 0.827–0.982). Bottom 25^{th} percentile was not significantly related to mortality outcomes.

Mortality

The mortality of all patients included in the study was also investigated by comparing the lowest StO_2 value recorded to the patient outcome, as well as the StO_2 value of the lowest quartile and the patient outcome. Non-survivors had significantly lower StO_2 nadirs compared to survivors, with the mean StO_2 for a non-survivor being 47% and for a survivor, 59.9%, p value 0.017 (95% CI 0.827-0.982). Comparatively, non-survivors and survivors did not have significantly different mean bottom quartile StO_2 values, 62.52% and 66.56% respectively with p value 0.884 (95% CI 0.901-1.094).

Discussion

This first analysis was conducted in one of the patients from the study from whom the most lactate and cardiac index assessments were performed; this was intentionally chosen to guide statistical analysis and decrease possible error associated with linear interpolation.

Inter-sensor agreement was analyzed with the Bland-Altman analysis, demonstrating agreement at critical low values and divergence at higher tissue saturations. Of note, temporal oximetry sensors have already been validated for clinical use by various studies including Benni et al, so re-validation was not performed in this study.[9] During cardiac surgery, the threshold for intervention is below 50% or more than 20% below the patient's baseline, making it essential for monitors to adequately assess perfusion continuously[10]. With this analysis, the inter-monitor reliability of the system was demonstrated at low saturations, suggesting that the use of a single monitor is likely acceptable for patient assessment and reassuring against outliers that could have been created from sensor attachment issues causing abnormally low readings. This is crucial given factors such as patient comfortability and adhesive longevity in intensive care unit settings, where shock patients may be diaphoretic and staff often experience issues maintaining continuous sensor contact. This was seen in several patients in whom only one sensor was able to be applied, or in whom otherwise no data was able to be acquired due to sensor intolerance.

In this study, the relationship of macro and microvascular measures of perfusion were compared to StO_2 trends, to assess their correlation. Lactate, a measure of microvascular perfusion, was first analyzed with a moderate inverse relationship noted. The direction of the relationship was expected, as increasing lactate values are markers of poor perfusion as cell metabolism shifts to anaerobic energy utilization, and this would correspond to low tissue saturation. The strength of the relationship may be explained by several factors, the first of which is the limited number of lactate samples that were taken during the testing time. The statistical analysis performed attempts to limit this; however, it is inexact, and limited. An increased rate of sampling may demonstrate similar peaks and valleys to those seen in the temporal oximetry, however it is difficult to know. This limitation reverberates throughout the study, as manual cardiac index measurements are also taken at limited intervals and is one of the reasons that the temporal oximetry poses an advantage in minute-to-minute monitoring. Another factor affecting the correlation strength is

the temporal relationship of lactate sampling to fluctuations in perfusion. Lactate is a byproduct of hypoperfusion, therefore by the time lactate has been produced, anerobic metabolism has been occurring. Oximetry measures oxygen saturation in the blood, and assess the delivery of oxygen to the tissue, rather than tissue utilization of the oxygen. There was more variability in the StO_2 measured over time, suggesting that it may be a more sensitive tool for detection of hypoperfusion than lactate. Of interest, it appears that there was an instance of significant hypoperfusion that preceded a lactate increase, suggesting that there may be a predictive element with a lag time to this relationship, however it requires further investigation.

Macrovascular perfusion, represented here as cardiac index, had a weaker relationship to StO_2 than microvascular perfusion. As others have demonstrated, measures of cardiac performance do not always correspond to measures of tissue perfusion in the cardiogenic shock state, which was reflected in this data. Interestingly, although visually it appears that increases in cardiac index correspond to increases in StO_2, the relationship was statistically weak. Continuous cardiac index, a measure that is not often used by clinicians, had a stronger positive relationship with StO_2, however it remained an overall weak relationship. The difference between continuous cardiac index and cardiac index suggests that more (non-statistically acquired) data points likely does affect the strength of a relationship, as demonstrated here, increasing it. Overall, however, the macrovascular markers were simply less likely to reflect the oxygen saturation in small vascular beds such as those measured by cerebral oximetry.

Differences in perfusion were also noted between survivors and non-survivors in a cohort-wide analysis. StO_2 nadir was significantly correlated to non-survival, while the mean StO_2 of the lowest quartile was not correlated with mortality. This suggests that events of significant hypoperfusion may lead to poor survival outcomes, rather than sustained relatively low perfusion. It may be postulated that maintenance at low perfusion states would be preferred to significant drops in perfusion, which may preclude any kind of physiological compensation for decreased oxygenation. Future investigation will need to delineate the time spent at nadir from short interval drops in perfusion, to help better identify guidelines for clinical intervention. Given the significant amount of data collected with continuous monitoring, clear thresholds must be included to be implemented into the complicated care stream for cardiogenic shock patients.

Finally, this study will require further investigation to compare these initial findings to those for other patients included in the study. Duration of sensor attachment, and incidence of lab sampling may create barriers for future analysis, however these will be attenuated by simply increasing the sample pool. Other opportunities for investigation will also include comparison of StO_2 and other measures of perfusion, assessment of the impact of mechanical circulatory devices and possible correlations of StO_2 to patient outcome markers such as intensive care unit length of stay.

Conclusions

Cerebral tissue oximetry, reported as tissue saturation or StO_2 is demonstrated to have a moderately strong inverse relationship with lactate, a marker of microvascular perfusion, and a weak positive relationship with cardiac index, a marker of macrovascular perfusion. StO_2 nadir was also found to have a statistically significant relationship with patient mortality outcome, with non-survivors having lower StO_2 nadirs. This suggests that cerebral tissue oximetry may be a reliable tool to assess perfusion in patients with cardiogenic shock and help guide clinical decision making.

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