Validation of a point-of-care capillary lactate measuring device (Lactate Pro 2).

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Original research

Keywords: lactate, point-of-care, shock.

DOI: https://doi.org/10.21203/rs.3.rs-21680/v2

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Abstract

Background: The measurement of lactate in emergency medical services has the potential for earlier detection of shock and can be performed with a point-of-care handheld device. Validation of a point-of-care handheld device is required for prehospital implementation.

Aim: The primary aim was to validate the accuracy of Lactate Pro 2 in healthy volunteers and in haemodynamically compromised intensive care patients. The secondary aim was to evaluate which sample site, fingertip or earlobe, is most accurate compared to arterial lactate.

Methods: Arterial, venous and capillary blood samples from fingertips and earlobes were collected from intensive care patients and healthy volunteers. Arterial and venous blood lactate samples were analysed on a stationary hospital blood gas analyser (ABL800 Flex) as the reference device and compared to the Lactate Pro 2. We used the Bland-Altman method to calculate the limits of agreement and used mixed effect models to compare instruments and sample sites. A total of 49 intensive care patients with elevated lactate and 11 healthy volunteers with elevated lactate were included.

Results: There was no significant difference in measured lactate between Lactate Pro 2 and the reference method using arterial blood in either the healthy volunteers or the intensive care patients. Capillary lactate measurement in the fingertip and earlobe of intensive care patients was 47% (95% CI (29% to 68%), p<0.001) and 27% (95% CI (11% to 45%), p<0.001) higher, respectively, than the corresponding arterial blood lactate. In the healthy volunteers, we found that capillary blood lactate in the fingertip was 14% higher than arterial blood lactate (95% CI (4% to 24%), p= 0.003) and no significant difference between capillary blood lactate in the earlobe and arterial blood lactate.

Conclusion: Our results showed that the handheld Lactate Pro 2 had good agreement with the reference method using arterial blood in both intensive care patients and healthy volunteers. However, we found that the agreement was poorer using venous blood in both groups. Furthermore, the earlobe may be a better sample site than the fingertip in intensive care patients.

Background

Circulatory shock is usually divided in categories according to the etiology that causes circulatory failure. The cause of circulatory shock among traumatic patients is often hypovolemia due to blood loss, while the etiology of non-traumatic shock may be more complex. Circulatory failure and shock develop as the perfusion and oxygen delivery to the organs and tissues decrease and is insufficient to meet the metabolic demand. When oxygen delivery is below a critical level, shock occurs with the accumulation of oxygen debt. Occult shock is the state of early hypoperfusion causing metabolic acidosis, that may occur in patients prior to detectable changes in vital signs. Due to insufficient oxygen delivery to the tissues, anaerobic metabolism leads to the production of lactate (1, 2).
Circulatory failure following both non-traumatic conditions and trauma is common in the prehospital setting, and the mortality rates in patients presenting with shock in the emergency departments are high. Correct initial assessment and identification of shock followed by early resuscitation is important to improve survival of these patients (3, 4).

Prehospital monitoring of vital signs such as systolic blood pressure (SBP) and heart rate (HR) are the main indicators used to identify shock. However, such vital signs often do not change until a patient is near a critical stage, and therefore often fail to predict shock at an early stage (5-7). Identification of patients in early stages of shock in the field is difficult. Blood lactate monitoring is widely accepted in-hospital as an indirect marker of tissue hypoxia in critically ill patients. The Surviving Sepsis Campaign Guidelines use a cut-off value of greater than 4 mmol/L lactate for early resuscitation therapy to treat sepsis (8). Furthermore, lactate levels are commonly measured during resuscitation to predict mortality and to evaluate and guide treatment in both the emergency department (ED) and the intensive care unit (ICU). However, lactate levels are infrequently measured in the prehospital setting (9, 10).

Recent prehospital studies on lactate measurement suggest that lactate may be more sensitive than SBP and HR in identifying haemorrhagic shock patients who may benefit from early blood transfusion, and suggest that lactate may be independent of conventional vital signs in identifying these patients (10-14). Implementation of point of care lactate monitors in emergency medical services (EMS) may contribute to the evaluation of patients in the early stages of shock, thereby establishing a trigger for resuscitation (15, 16). However, so far there is little evidence on the prehospital use of point-of-care lactate monitors in the field.

The primary aim of this study was to validate the handheld device Lactate Pro 2 (LP2) in two different groups: healthy volunteers and critically ill patients. The secondary aims were to compare capillary blood lactate in the fingertip and earlobe with arterial blood lactate to investigate whether capillary lactate reflects the actual arterial levels.

**Methods**

*Study design*

This is a prospective observational study. The study was performed with two groups: haemodynamically compromised intensive care patients and healthy volunteers performing a maximal oxygen consumption test (VO2 max test). The healthy volunteer group had a longitudinal study design with repeated measurements over a short period of time.

*Setting*

The study took place in the Intensive Care Unit, Haukeland University Hospital, Norway. The healthy volunteers were tested at the VO2 test laboratory at the same hospital. The study was conducted from September 2016 to February 2018.
Participants

Patients were enrolled from the ICU at Haukeland University Hospital. Inclusion criteria for intensive care patients were (i) adults at least 18 years of age, (ii) an arterial line and a central venous catheter, and (iii) informed consent from the next of kin or the patient him- or herself if awake and competent to give consent. Healthy volunteers were enrolled from the medical faculty in Bergens sports team. Inclusion criteria for the healthy volunteers included were (i) adults at least 18 years of age and (ii) informed consent.

Sampling

Blood gases from the arterial and central venous lines of the ICU patients were sampled in heparinized syringes by the ICU nurses. They were analysed on both the LP2 and the ABL800 Flex (ABL) immediately. Capillary blood was drawn from the fingertip and earlobe and analysed on the LP2. Two LP2 instruments, denoted LP2.1 and LP2.2, were used for quality assessment: the first drop of blood was measured on the LP2.1 and the second on LP2.2. In one ICU patient only one arterial and one venous sample was drawn. Two capillary samples each from fingertip and earlobe were sampled. All capillary samples were measured by the same researcher (A.R). The timing of sampling with respect to day of ICU treatment was not registered, but the patients were included fairly early as the ICU nurses called A.R. when an eligible patient was admitted to the ICU. The healthy volunteers underwent a VO2-max test on treadmill (Modified Bruce Protocol) with an arterial line and a peripheral venous catheter during the test (17). Anaesthesiologists conducted the arterial cannulation. Repeated measurements of arterial, venous and capillary blood lactate in this group were recorded at rest, directly after the VO2-max test and at 3, 5, 10 and 20 min after the test was completed. Capillary blood samples were drawn from the fingertip and earlobe. The arterial and venous blood samples were drawn in heparin tubes, stored on ice and analysed on the ABL within 30 min.

Materials

Lactate Pro 2 (AKRAY Europe B.V. Prof J.H Bavincklaan 5 1183 AT, Amstelveen, the Netherlands) is a handheld point-of care analyser that operates by enzymatic amperometric detection. Blood lactate reacts with the reagent on the test strip, which produces a small electrical current proportional to the concentration of blood lactate. The meter measures this current and calculates the blood lactate level. It requires 0.3 microliters of a whole-blood sample and 15 seconds to measure the lactate value. LP2 has a measurement range between 0.5-25.0 mmol/L. If “Hi” or “Lo” appears on the display it means that the blood lactate level is above 25.0 mmol/L or below 0.5 mmol/L, respectively. Therefore, in this study only lactate values between 0.5 and 25 were included.

ABL800 Flex (Radiometer Medical ApS, Åkandevej 21, DK-2700 Brønshøj, Denmark) is the standard blood gas machine used in the ICU at Haukeland University Hospital. It uses an amperometric method to measure the lactate value. The enzyme lactate oxidase converts lactate to H2O2 and the oxidation of H2O2 produces an electrical current that is directly related to the amount of lactate. The analyser thereby automatically calculates the lactate concentration in the sample.
**Statistical methods**

Logarithmic transformation was performed for lactate and the inverse transformation was used to obtain interpretable estimated differences, since lactate has a skewed distribution and logarithmic transformation is closer to normal distribution. Due to the repeated measurements in each individual in the healthy volunteer group, a mixed effects model including instrument and time as variables was used. First, we estimated a model with the same difference between instruments at all time points and then a model when this was not assumed. For ICU patients, mixed effects models were used for the measurements at different sites and with different instruments, and Bland-Altman plots were used to compare different instruments and different sites (18). Calculations were performed with R (R Foundation for Statistical Computing, Vienna, Austria) using the R package nlme for mixed effects analysis (19).

**Results**

Forty-nine ICU patients and 11 healthy volunteers were included. Of the healthy volunteers there were 4 women and 7 men. The mean age was 24.5 years. In the ICU patients there were 41 missing values (6.3%), including 34 values above the detection limit. In the healthy volunteers, there were 10 missing values (2.5%), including 4 values above the detection limit.

**Instrument comparisons**

In the ICU group (n=49), we found no significant difference in measured lactate between the LP2 and the ABL using arterial blood (Table 2, Figure 1). We found significantly higher values with LP2 than ABL using central venous blood (Table 2, Figure 2).

In the healthy volunteer group (n=11), we found no significant difference between LP2 and ABL using arterial blood but significantly higher values for LP2 when using peripheral venous blood (Table 2). We found no statistically significant difference between LP2.2 and LP2.1 in both arterial and venous blood. In a mixed effects model where the instrument discrepancy could vary during follow-up, LP2 measured lower values in arterial blood compared to ABL at “rest” (data not shown), but we found no significant difference between LP2 and ABL at the other time points.

**Sample site comparisons**

In the ICU group, we found significant differences in both fingertip and earlobe compared to arterial blood using the LP2.1. Capillary blood lactate in the fingertip and earlobe was 47% (95% CI (29% to 68%), p<0.001) and 27% (95% CI (11% to 45%), p<0.001) higher than in arterial blood, respectively (Table 3). When comparing fingertip to earlobe we found that capillary blood lactate in the fingertip was 16% higher than in the earlobe (95% CI (2% to 32%), p=0.029). Bland Altman plots for comparison between capillary blood lactate in fingertip with arterial blood lactate on both handheld instruments (LP2.1 and LP2.2) in the ICU group are presented in Figure 3. We observed a rise in the lactate values in the fingertip from the first blood drop measured on LP2.1 to the second blood drop measured on LP2.2. We did not observe the same effect in the earlobe.
In the healthy volunteer group (n=11), we found that capillary blood lactate in the fingertip was 14% higher than arterial blood lactate (95% CI (4% to 24%), p=0.003) (Table 3). We found no significant difference between capillary blood lactate in the earlobe and arterial blood lactate (Table 3).

**Discussion**

The LP2 and ABL agreed well in arterial blood taken from both healthy volunteers and intensive care patients. Capillary blood lactate values in the fingertip were significantly higher than the corresponding arterial values in both ICU patients and healthy volunteers. Capillary blood lactate values in the earlobe were significantly higher than arterial blood lactate values in the ICU group but not in the healthy volunteer group.

The earlobe seems to be a better sample site than the fingertip, possibly because it is more central than the fingertip. However, more importantly, it is less sensitive to the variation between two measurements taken consecutively. Contenti et al. used the earlobe as a sample site for capillary blood and found that capillary blood lactate was higher than both venous and arterial lactate in patients in an ED, while in our study, ICU patients had higher capillary blood lactate than arterial blood lactate but almost the same as venous blood lactate (20).

Studies performed with other handheld devices in ICU populations also report good agreement between capillary and arterial lactate, but the devices were validated in lower lactate ranges (21-23).

Collange et al. is the only other study that compared both capillary and arterial blood samples on a handheld device instead of comparing capillary blood measured on a handheld device with arterial blood measured on a reference method. Like us, they found that capillary lactate was higher than arterial blood lactate. However, in contrast to our findings, they reported that the handheld instrument measured slightly lower lactate values than the reference instrument using arterial blood (22).

Interpretations of capillary blood lactate levels in patients suffering from shock may be challenging because of discrepancies between capillary and arterial blood due to changes in peripheral perfusion following vasopressor use. Vasopressors may also increase lactate production via stimulation of skeletal muscle beta-2 adrenergic receptors, and reduce lactate clearance in peripheral tissues, and thereby increase the difference between arterial, venous and capillary lactate values (24). Hence, vasopressors may also influence the difference in capillary blood lactate between the fingertip and earlobe. Pattharanitima et al. found no evidence of norepinephrine use effecting the relationship between capillary or arterial lactate (23). We did not adjust for vasopressor as a confounder in the ICU group, and therefore our results may not be directly applicable to the prehospital patients who do not receive vasopressor.

Lactate monitoring in EMS may help the provider in detecting shock at an earlier stage and may be used as an early trigger for blood transfusion in haemorrhaging patients or for fluid therapy in septic patients (14). In general, arterial blood is preferred. Capillary lactate should only be measured when rapid measurement is necessary or when arterial blood is not available. Since patients in the prehospital setting
in most cases do not receive an arterial line, a handheld capillary blood lactate measuring device may be well suited for EMS services. Measuring lactate values in the field is quick and should not delay other treatments or interventions on-scene. Our results show that LP2 also has the potential to overestimate the lactate values, which may lead to overtreatment or overtriage of patients. This may, however, be of less importance than the potential consequences of under-triage in situations where EMS providers fail to detect the early stages of shock.

Despite overestimation of the actual values in a single reading, this value may be helpful as an adjunct to the other vital signs during assessment of these patients. Further, multiple lactate readings may provide even more information to the EMS provider because a lactate trend may reflect the clinical course of shock and the potential effect of resuscitation. This may be of value for clinical decision making and for guiding treatment. When interpreting the lactate values, one must consider both the instrumental bias of the LP2 and the physiological discrepancy between arterial and capillary blood lactate values in different sample sites and different haemodynamical states.

Strengths and limitations

The strength of this study lies in the standardized procedure for collecting blood and analysing lactate. In contrast to other studies comparing capillary and arterial blood lactate we measured both capillary and arterial/venous blood on the same instrument. Most other studies compare the capillary lactate value measured on the handheld device with the arterial value measured by the reference method. We believe our method is more correct because it separates the instrument agreement from the difference in the different blood samples. Another strength is that we validated LP2 in a wide range of lactate values (from 1-25 mmol/l) in both healthy volunteers and intensive care patients, which shows the differences in lactate distribution in capillary and arterial/venous blood in these two haemodynamically different groups. This study has some limitations of note. We have previously observed that cold LP2 strips may influence the measured value randomly; therefore, we made sure that the LP2 and the strips were stored at room temperature. This limitation in the instrument may have caused a bias in patients with low body temperature. We are considering further research on this issue. The manufacturer states that the device must be used between 5 and 40 degrees Celsius and should be adjusted to the surroundings for at least 20 minutes. In the pre-hospital environment, the temperature may exceed these limits. This requires the device to be stored in a temperature-controlled environment, e.g. in an isolated casing.

Regarding capillary measurements, the disinfecting agent can cause haemolysis, which may increase the lactate concentration. This study includes few study subjects, which limits the generalizability of the study findings. The number of healthy volunteers included is low due to the ethical aspect of arterial cannulation and the associated risk of complication. The number of missing values in our data set, partly due to the detection limits of the LP2 also constitutes a limitation.

Conclusion
We found that Lactate Pro 2 had good agreement with the reference method using arterial blood but poorer agreement using venous blood. Our results show the potential for overestimation of the lactate values in haemodynamically compromised patients. The levels of lactate in capillary blood from the fingertip and earlobe were 47% and 27% higher, respectively, than in arterial lactate in intensive care patients. The earlobe may be a better sample site than the fingertip in haemodynamically compromised patients.

Declarations

Ethics approval and consent to participate

All patients’ next of kin and all volunteers involved signed an informed consent form. This study was approved by the Regional Ethics Committee (REK-Vest number 2016/815 and 2017/162).

Consent for publication

Not applicable.

Availability of data and materials

Data are available upon reasonable request.

Competing interests

The authors declare no competing interests.

Funding

We received funding from the Regional Akuttmedisinsk Kompetansesenter (RAKOS Helse Vest).

Authors’ contributions

A.R. was the lead investigator and contributed to all aspects of the study. J-K.H. contributed to all aspects of the study. G.S. contributed to the development of the study design, data interpretation and critical revision. B.B. contributed to the development of the study design, data analysis, data interpretation and critical revision. C.B., R.K. and H.S.E. contributed to the development of the study design and data collection. T.W-L. participated in planning and performing the statistical analyses and preparation of the data for statistical analysis together with the first author. All authors participated in critical revision.

Acknowledgements

All nurses and doctors at the ICU at Haukeland University Hospital for help with data sampling. Thank you Professor Anne Berit Guttormsen for support and guidance throughout the study period.

Abbreviations
SBP: Systolic blood pressure  
HR: Heart rate  
ED: Emergency Department  
ICU: Intensive Care Unit  
EMS: Emergency medical services  
LP2: Lactate Pro 2  
VO2: maximal oxygen consumption test  
ABL: ABL800 Flex  
CI: Confidence interval

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Tables

Table 1: Demographic data of ICU patients.
| Gender          | N (%)       |
|-----------------|-------------|
| Male            | 28 (57.0 %) |
| Female          | 16 (33.0 %) |
| NA              | 5 (10.0 %)  |

| Mean age        | 62.0 ± 17.1 years |

| Diagnosis              | N (%)       |
|------------------------|-------------|
| Sepsis                 | 15 (30.6 %) |
| Hypovolemic shock      | 4 (8.2 %)   |
| Severe burn injury     | 3 (6.1 %)   |
| Respiratory failure    | 3 (6.1 %)   |
| Traumatic brain injury | 2 (4.1 %)   |
| Cardiac arrest         | 2 (4.1 %)   |
| Cardiogenic shock      | 2 (4.1 %)   |
| Thoracic trauma        | 2 (4.1 %)   |
| Liver failure          | 2 (4.1 %)   |
| Pancreatitis           | 1 (2.0 %)   |
| Metformin intoxication | 1 (2.0 %)   |
| Other                  | 7 (14.3 %)  |
| NA                     | 5 (10.2 %)  |

**Table 2:** Results of instrument comparisons with arterial and venous blood in ICU patients and healthy volunteers.

| ICU patients | Estimate (Ratio) | 95 % CI          | p-value |
|--------------|------------------|------------------|---------|
| Arterial     |                  |                  |         |
| LP2.1 vs ABL | 1.03             | 0.99 to 1.08     | 0.140   |
| LP2.2 vs ABL | 1.04             | 0.99 to 1.09     | 0.102   |
| LP2.2 vs LP2.1 | 1.004       | 0.96 to 1.05     | 0.871   |
| Venous       |                  |                  |         |
| LP2.1 vs ABL | 1.29             | 1.24 to 1.35     | <0.001  |
| LP2.2 vs ABL | 1.29             | 1.23 to 1.35     | <0.001  |
| LP2.2 vs LP2.1 | 0.998       | 0.96 to 1.04     | 0.938   |

| Healthy volunteers | Estimate (Ratio) | 95 % CI          | p-value |
|--------------------|------------------|------------------|---------|
| Arterial, LP2 vs ABL | 0.96             | 0.93 to 1.002    | 0.063   |
| Venous, LP2 vs ABL  | 1.07             | 1.03 to 1.11     | 0.001   |

**Table 3:** Results of sample site comparisons in ICU patients and healthy volunteers.
| ICU patients                          | Estimate (Ratio) | 95 % CI      | p-value |
|--------------------------------------|------------------|--------------|---------|
| Finger LP2.1 vs Arterial LP2.1       | 1.47             | 1.29 to 1.68 | <0.001  |
| Earlobe LP2.1 vs Arterial LP2.1      | 1.27             | 1.11 to 1.45 | <0.001  |
| Finger LP2.2 vs Arterial LP2.2       | 1.85             | 1.55 to 2.21 | <0.001  |
| Earlobe LP2.2 vs Arterial LP2.2      | 1.22             | 1.03 to 1.46 | 0.024   |
| Finger LP2.1 vs Venous LP2.1         | 1.14             | 1.01 to 1.30 | 0.040   |
| Earlobe LP2.1 vs Venous LP2.1        | 0.99             | 0.87 to 1.12 | 0.871   |
| Finger LP2.2 vs Venous LP2.2         | 1.43             | 1.20 to 1.69 | <0.001  |
| Earlobe LP2.2 vs Venous LP2.2        | 0.95             | 0.80 to 1.13 | 0.569   |
| Healthy volunteers                   | Estimate (Ratio) | 95 % CI      | p-value |
| Finger vs arterial                   | 1.14             | 1.04 to 1.24 | 0.003   |
| Earlobe vs arterial                  | 0.98             | 0.90 to 1.06 | 0.568   |

**Figures**
Figure 1

Bland-Altman plot for instrument comparison of LP2 and ABL in arterial blood from ICU patients, based on log transformed lactate.
Figure 2

Bland-Altman plot for instrument comparison of LP2 and ABL in venous blood from ICU patients, based on log transformed lactate.
Figure 3

Bland-Altman plots for comparison between lactate in fingertip with arterial lactate in ICU patients, based on log transformed lactate.

Supplementary Files

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- SupplementaryTables.docx