

Change of the monitor T wave per mmol/L of potassium within patients: an analysis in emergency department patients with potassium below 6 mmol/L

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Research Article

Keywords: electrocardiography, potassium, hyperkalaemia, T wave, emergency department, patient monitoring, repeatability

Posted Date: October 20th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-11412868/v1>

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Additional Declarations: The authors declare no competing interests.

Abstract

Background.

Potassium results arrive an hour or more after the order, while a lead II monitor shows the T wave continuously. How much the monitor T amplitude changes with potassium within a patient, in a mostly normal range, is unclear.

Methods.

Retrospective within-patient analysis of MC-MED (one emergency department). The analysis was fixed in a written specification and script before the primary analysis was run. We paired consecutive serum potassium values of one visit, 1 to 12 hours apart, and measured the QRS-relative T amplitude (lead II, in mm) with an automated algorithm near each sample. Primary pairs: initial potassium below 6.0 mmol/L, predominantly regular rhythm, no treatment indication. Slopes were estimated with a patient-cluster bootstrap.

Results.

Of 5,585 pairs, 216 (3.9%) were primary pairs (about 200 patients). T amplitude changed by 0.26 mm per mmol/L (95% CI -0.04 to 0.63 mm). The smallest slope detectable with 80% power was 0.40 mm per mmol/L. The estimate depended on 11 influential pairs (Huber estimator 0.07 mm, without them 0.04 mm). Post hoc, the algorithm measured smaller T amplitudes at these pairs than the author did on reading them. T-wave steepness (secondary, no reference reading) had a positive slope (0.75 mV/s per mmol/L; CI 0.21 to 1.37) that was not stable.

Conclusion.

In this selected group of emergency department patients (initial potassium below 6 mmol/L, predominantly regular rhythm, no treatment indication), the mean change in measured T amplitude per mmol/L potassium was small and uncertain (0.26 mm; 95% CI -0.04 to 0.63 mm) and depended on a few pairs. Slopes above 0.63 mm per mmol/L lie outside the 95% interval; smaller slopes are not excluded. An association is neither established nor excluded. This is a group finding, not a statement on individual patients or treated hyperkalaemia.

Key message

What is already known on this topic. The ECG is an unreliable marker of potassium at a single time point, and within patients T-wave changes have been followed, for example, during haemodialysis, where potassium changes substantially.

What this study adds. In 216 within-patient pairs with potassium below 6 mmol/L and no treatment indication, the monitor T amplitude changed by 0.26 mm per mmol/L (95% CI -0.04 to 0.63 mm), a small

and imprecise result that depended on 11 influential pairs; an association is neither established nor excluded.

How this study might affect research, practice or policy. The data are compatible with a mean change from slightly negative to about 0.6 mm per mmol/L (upper limits of sensitivity analyses up to 1.0 mm); no recommendation follows, and studies with larger potassium changes, documented sampling times and an independent reference reading are needed.

Introduction

Patients with a possible potassium disorder are usually on a continuous lead II monitor in the emergency department, while the potassium result arrives later (in the pairs analysed here, a median of 62 to 84 minutes after the monitor segment nearest to the order). The physiological basis of potassium effects on repolarisation is well described,[1,2] which raises the question whether the monitor T wave moves with potassium between two samples.

At a single time point the ECG is an unreliable marker. In 90 patients with a potassium of 6 mmol/L or more, the ECG was insensitive and no diagnostic threshold for T-wave amplitude could be found.[3] Two physicians detected potassium above 5.0 mmol/L from the ECG with sensitivities of 34% and 43%.[4] In acute kidney injury, T-wave amplitude was a poor predictor of serum potassium,[5] and in 330 analysable routine ECGs the correlation coefficients of T amplitude, T-wave slope and a normalised feature with potassium were small (absolute values of 0.22 or less).[6] A deep-learning model using two ECG leads detected hyperkalaemia in chronic kidney disease with an area under the curve of 0.85 to 0.88.[7] Within patients, T-wave amplitude and downslope were used to estimate potassium during haemodialysis, with a mean absolute error of 0.46 mmol/L.[8]

Most of this work concerns hyperkalaemia or a single time point. Our data differ: potassium was mostly in or near the normal range (Results), there was no potassium-directed treatment indication, and the potassium change was small (SD 0.70 mmol/L). We asked whether the lead II T amplitude changes with potassium between two samples in this range, and with what precision.

Methods

Design and data

This is a retrospective within-patient analysis of MC-MED version 1.0.1 (118,385 adult visits to one academic emergency department, Stanford, 2020 to 2022; continuous lead II ECG, laboratory results and orders).[9-11] We followed STROBE and RECORD.[12,13] The analysis was fixed in a written specification and a script, and both were locked on 16 October 2026 before the primary analysis was run; the lock is documented by a lock file held by the author, without an independent time stamp. Two further questions of the specification (QRS duration, persistent hyperkalaemia) are in the Supplement. A feasibility check on the training split before locking looked at counts, signal quality and spread, not at any relation

between potassium and the ECG. The expected slope (0.21 mm per mmol/L) came from an unpublished, non-peer-reviewed analysis by the author of an intraoperative database (VitalDB[14,15]) and was therefore not blind.

Pairs and treatment

A pair was two consecutive serum potassium values of one visit, 1 to 12 hours apart; the potassium change (ΔK) is the second minus the first value. We searched orders and medication records for treatment that lowers potassium or acts on the ECG (Supplement). Pairs with documented or possible treatment, and pairs with an initial potassium of 6.0 mmol/L or more, were excluded from the primary analysis. The remaining pairs have “no treatment indication”, which is not the same as untreated, because 34% of medication orders lack an administration time.

ECG measurement

For each end of a pair we took the 10-second ECG segment of monitor lead II nearest to the order time, within a window around the order and result times. An automated algorithm measured a 60-second window around that segment. The algorithm (500 Hz; filtering and R-peak detection with NeuroKit2[16]) forms a median beat aligned to the R peak and measures the T amplitude from the PR baseline to the T peak, signed relative to the main QRS deflection (positive: the T wave points the same way as the QRS). A window is valid if the rhythm is predominantly regular (at most 30% irregular beats) and at least 20 seconds of good beats remain; pairs with an invalid window at either end drop out. Amplitudes are given in mm at 10 mm per mV. We also measured the steepness of the descending T limb (mV/s).

Statistical analysis

The primary estimate is the slope β of the change in T amplitude (second minus first) on ΔK , with a free intercept and the change in a spline of the monitor heart rate as covariate, fitted by least squares; the model is linear across the potassium range. The 95% confidence interval comes from 2,000 patient-cluster bootstrap draws. As a precision measure we report the smallest slope detectable with 80% power, from the pre-specified planning formula (2.8 times the standard error, with the observed spread of ΔK and residual SD).

The pre-specified repeatability limit is $1.96 \times \sqrt{2} \times$ the within-sample SD of two non-overlapping 60-second windows near the same sample (21,055 samples).[17,18] The repeat differences have heavy tails, so we also report a robust limit based on a robust SD (post hoc); it does not correspond to the 95th percentile of the differences. Both limits describe how far apart two single measurements of the same sample lie. They are context for single measurements, not a yardstick for a group mean, and not a detection limit for a potassium change. Repeatability is reported according to GRRAS.[19] Pre-specified

sensitivity analyses and three controls are in the Supplement; intervals are not adjusted for multiple analyses, and no clinical margin was set.

Reference reading

An ECG-experienced reader, the author, who specified the algorithm and knew the hypothesis, measured the T amplitude on 100 ten-second strips, blind to potassium, treatment and the algorithm's values, after the analysis was locked and before the main analysis was run. We compared 70 readings of more than 60 strips with a value from both reader and algorithm. This check is not independent.

Reader check of influential pairs (post hoc)

After the results were known, the same reader measured both ends of the 11 influential pairs (pairs whose removal changes the slope by more than expected, $|DFBETAS|$ above $2/\sqrt{n}$, a pre-specified rule) and of 11 randomly chosen comparison pairs, in random order and blind to potassium. Comparison pairs were not matched on T amplitude, so blinding to the origin of the strips was partial. This post hoc check examines the measurement; a re-estimation of the slope with the reader's values is in the Supplement.

Patient and public involvement

Patients and the public were not involved in the design, conduct, reporting or dissemination of this study, which is a secondary analysis of de-identified data.

Use of generative AI

The author used generative AI tools (Anthropic Claude and OpenAI Codex) to assist with analysis code, literature searches, data analysis workflows, drafting and editing of text, and figure preparation. AI-assisted output was reviewed, corrected where necessary and approved by the author, who takes full responsibility for the content. Patient-level analyses were run locally. AI tools are not listed as authors.

Results

Pairs

Of 5,661 consecutive pairs, 5,585 had usable result times and uncensored values. An ECG segment at both ends within the window existed in 970 (17.4%), a valid measurement at both ends in 592, and the same clear polarity in 522. After excluding overlapping windows, missing heart rate, and documented or possible treatment, 324 pairs remained, of which 216 had an initial potassium below 6.0 mmol/L: 3.9% of 5,585 pairs, from about 200 patients (Supplement Table S1). Of the 216 pairs, 139 had an initial potassium of 3.5 to 5.0 mmol/L, 53 above 5.0 and 24 below 3.5 mmol/L. The SD of ΔK was 0.70

mmol/L, about half of the 1.34 mmol/L planned; fewer than 10 pairs rose by 1 mmol/L or more. Table 2 compares the primary pairs with other pairs without a usable measurement (post hoc).

Table 2

Primary pairs compared with other pairs without a usable measurement (post hoc, descriptive)

Characteristic	A: primary pairs	B0: other pairs without a usable measurement
Pairs (n)	216	3,090 (rounded down)
Patients (n, rounded down)	about 200	2,520
Age, years: median (Q1 to Q3)	60 (39 to 73)	64 (49 to 77)
Male sex	61%	52%
Initial potassium, mmol/L: median (Q1 to Q3)	4.2 (3.8 to 5.1)	4.2 (3.9 to 4.7)
Potassium change, mmol/L: median (Q1 to Q3); SD	-0.20 (-0.53 to 0.10); 0.70	-0.10 (-0.40 to 0.20); 0.61
Interval between samples, hours: median (Q1 to Q3)	2.6 (1.7 to 3.8)	4.7 (2.9 to 6.9)
Triage level 1 (resuscitation)	< 5%	2%
Triage level 2 (emergent)	45%	39%
Triage level 3 (urgent)	50%	57%
Triage level 4 or 5, or missing	< 5%	1%
Discharged home	35%	12%
Admitted to a ward	40%	59%
Intensive care	10%	9%
Observation or missing	20%	21%
ED length of stay (unit of the source file, consistent with hours): median (Q1 to Q3)	7.7 (6.0 to 9.5)	10.6 (7.6 to 16.7)

Post hoc, descriptive; one row per patient (first pair). B0: valid pairs outside the primary selection with initial potassium below 6.0 mmol/L and no documented treatment (unclear treatment included) that lacked a usable ECG measurement at both ends. Q1 to Q3: quartiles. Percentages as in the source table; for group A, triage and disposition are rounded to 5%. Full table: Supplement Tables S11 to S14.

Primary result

T amplitude changed by 0.26 mm per mmol/L potassium (95% CI -0.04 to 0.63 mm). The pre-specified category was “imprecise” (the interval includes zero and the margin of 0.1 mm per mmol/L). Slopes above 0.63 mm per mmol/L lie outside the 95% interval; smaller slopes, including zero, do not. With the

observed spread of ΔK (SD 0.70 mmol/L against 1.34 planned) and residual SD (1.4 mm), the smallest slope detectable with 80% power was 0.40 mm per mmol/L, about twice the expected 0.21 mm. The repeatability limits of single measurements are in Table 1.

Table 1
Precision and measurement noise

Quantity	Value
Slope of T amplitude per mmol/L potassium (primary)	0.26 mm (95% CI -0.04 to 0.63)
Smallest slope detectable with 80% power (observed spread)	0.40 mm per mmol/L
SD of the potassium change	0.70 mmol/L
SD of the T-amplitude change not explained by potassium	1.4 mm
Within-sample SD of the measurement	0.63 mm
Repeatability limit, pre-specified (classical)	1.73 mm
Repeatability limit, robust (post hoc; not a 95th percentile)	0.53 mm

CI: confidence interval (patient-cluster bootstrap, 2,000 draws). 216 pairs. The limits describe the difference between two single measurements of the same sample and are context, not a yardstick for the slope.

Dependence on influential pairs (pre-specified)

The estimate depended on a small group of pairs. With a robust (Huber) estimator it was 0.07 mm per mmol/L (-0.11 to 0.29), and without 11 influential pairs (5% of the pairs) it was 0.04 mm (-0.10 to 0.23).

Reader check of the influential pairs (post hoc)

The reader measured both ends of the influential pairs and of the comparison pairs (most ends in each group were readable). At the influential ends the algorithm measured smaller T amplitudes than the reader (mean 0.09 vs 0.23 mV, that is, flat T waves by the algorithm's measurement); the mean difference was 0.13 mV (95% CI 0.02 to 0.25), the median difference 0.02 mV and the ICC 0.62. At the comparison ends the mean difference was 0.05 mV (0.01 to 0.08), the median difference 0.02 mV and the ICC 0.73. The mean difference at the influential ends was driven by a few large differences (largest 0.76 mV, against 0.21 mV at the comparison ends). For about 20 pairs with both ends read, the limits of agreement between reader and algorithm for the change in T amplitude were - 0.46 to + 0.57 mV (ICC 0.49), wider than the SD of the observed change not explained by potassium (0.14 mV). The slope depends on the change within a pair, which is measured less precisely than single values. Re-estimating the slope with the reader's values (Supplement) lowers it by construction, because only the pairs that drove the estimate were replaced.

Individual changes and noise

For potassium falls of 1 mmol/L or more (33 pairs), the median change in T amplitude was – 0.08 mm (interquartile range – 0.70 to 0.27). For falls of 0.5 to 1 mmol/L (28 pairs) it was – 0.01 mm (-0.96 to 0.41), and where potassium changed by less than 0.5 mmol/L (132 pairs) it was – 0.09 mm (-0.67 to 0.41; Fig. 1). The interquartile ranges were 1.0 to 1.4 mm wide in all three categories. The SD of the change not explained by potassium was 1.4 mm, against 0.9 mm expected from two windows of the same sample: between two samples the T amplitude varied more than within one.

Sensitivity analyses, other populations and controls

Of 22 pre-specified sensitivity analyses with an estimate (Supplement Table S4), point estimates ranged from – 0.26 mm per mmol/L (potassium rises only) to 0.51 mm, and upper confidence limits from 0.10 to 1.02 mm. Only pairs with a heart-rate change below 10 per minute (142 pairs) had an interval that excluded zero (0.51 mm; 0.09 to 0.97). The 105 pairs with documented treatment (slope 0.01 mm; -0.61 to 0.44) are not a group of treated hyperkalaemia: their median initial potassium was 3.7 mmol/L, half had a potassium rise, and about 60% received potassium and about 60% a potassium-lowering drug class (Supplement Table S13). Adding 108 pairs with an initial potassium of 6.0 mmol/L or more and no treatment indication gave – 0.01 mm (-0.12 to 0.10) in all 324 pairs. In these 108 pairs potassium almost always fell (median change – 1.9 mmol/L) over a short interval (median 1.7 hours); the cause is unexamined, and the slope in the added pairs alone was not calculated.

Three controls did not clearly differ from zero. ECG windows shifted 2 to 6 hours away from the order time (118 pairs) gave – 0.26 mm per mmol/L (-0.64 to 0.02), the same size as the main estimate with the opposite sign; pairing ECG and potassium changes of different patients gave a permutation P of 0.084; sodium gave – 0.05 mm per 5 mmol/L (-0.37 to 0.23). They were too imprecise to show that the algorithm returns no signal where none is expected, so they neither support nor refute the main estimate.

Steepness

T-wave steepness, a secondary outcome without a reference reading, had a positive slope of 0.75 mV/s per mmol/L (95% CI 0.21 to 1.37). In 19 post hoc sensitivity analyses the slope was positive in all (0.02 to 1.27 mV/s per mmol/L) and its interval excluded zero in 14, but the size was not stable: the Huber estimate was 0.48, and in the training partition alone (177 pairs) the interval included zero. The lower interval limit varied between 0.17 and 0.25 across 44 repeats of the bootstrap with 2,000 draws each (Supplement). Without the influential pairs the slope was 0.58 mV/s; the reader check did not cover steepness.

Reference reading

On 70 readings of more than 60 strips, the reader's T amplitude was higher by 0.04 mV (0.4 mm; 95% CI 0.02 to 0.06 mV), with limits of agreement of -0.16 to + 0.24 mV (-1.6 to + 2.4 mm). The algorithm was invalid in 19 of the 89 strips that the reader could measure (21%).

Discussion

Across 216 within-patient pairs, a potassium change of 1 mmol/L was accompanied by a mean change in the measured T amplitude of about a quarter of a millimetre (95% CI -0.04 to 0.63 mm). The result is imprecise, since the smallest slope detectable with 80% power was 0.40 mm per mmol/L (0.47 with the bootstrap standard error), and it depended on 11 influential pairs. In a post hoc reading by the author at these pairs, the algorithm measured smaller amplitudes than the reader (mean difference 0.13 mV at the ends). The check covers only the pairs that drove the estimate, was made by the author and does not show that the algorithm is biased elsewhere; replacing values only there lowers the slope by construction. The primary estimate is compatible with the 0.21 mm (95% CI 0.10 to 0.33) expected from an unpublished, non-peer-reviewed analysis by the author of an intraoperative cohort (Supplement S1b); that setting (anaesthesia) differs.

Studies in routine care found weak or no correlation between T-wave features and potassium,[5, 6] and in hyperkalaemia no diagnostic threshold for T-wave amplitude.[3] This is in line with our description between patients (-0.03 mm per mmol/L, 6,000 patients; Supplement). The dialysis study followed the large changes in potassium during treatment.[8] Estimating the slope precisely needs larger changes, which occur mostly under treatment or dialysis. Our data contain no such group: the 105 pairs with documented treatment had a median initial potassium of 3.7 mmol/L, and in the 108 pairs with initial potassium of 6.0 mmol/L or more, potassium almost always fell for reasons we did not examine.

Steepness had a positive slope in every sensitivity analysis, yet its size was unstable, the controls were imprecise and there is no reference reading; we treat it as a hypothesis.

What this means for practice

In patients like those analysed, the data are compatible with a mean change in T amplitude from slightly negative to about 0.6 mm per mmol/L potassium (upper limits of sensitivity analyses up to 1.0 mm); between two samples of the same patient the unexplained change had an SD of 1.4 mm. No recommendation follows. The analysis describes groups, gives no detection limit for individual patients and says nothing about what a clinician can see on the monitor. As a post hoc observation, at the ends of the influential pairs the algorithm gave lower amplitudes than the reader where its amplitude was small. The findings do not apply to potassium of 6.0 mmol/L or more, to treated hyperkalaemia, to irregular rhythm such as atrial fibrillation, or to the 12-lead ECG.

Limitations

The primary pairs are 3.9% of the pairs. They were selected by monitoring at both ends, predominantly regular rhythm, initial potassium below 6.0 mmol/L and no treatment indication; repeat tests are often triggered by an abnormal first value, which invites regression to the mean. Compared with other pairs without a usable measurement, the primary pairs were younger (median 60 vs 64 years), more often male (61% vs 52%), had shorter intervals between samples (median 2.6 vs 4.7 hours), were admitted

less often (about 40% vs 59%) and discharged more often (about 35% vs 12%); initial potassium was the same (median 4.2 mmol/L). Because these patients were discharged more often and had shorter stays than those in the other pairs, the findings may not apply to sicker patients, and whether this selection changes the slope was not examined. Sampling times are not recorded, so the ECG nearest to the order is only an approximation of the sample time, and this misclassification probably pulls the slope towards zero. The model is linear and adjusts for heart rate only; calcium, drugs, ischaemia and QRS widening were not adjusted for. One lead was used, and amplitude does not capture T-wave shape. No positive control for the algorithm was performed.

The reader check of influential pairs was post hoc, covered 11 influential and 11 comparison pairs (small numbers), used comparison pairs not matched on T amplitude and replaced values only at pairs selected for their influence. The reference reading and the reader check were made by one reader, the author, who specified the algorithm and knew the hypothesis. There was no independent second reader and no repeat reading after an interval, and the reference reading rests on 70 readings of more than 60 strips. The limits of agreement of about 2 mm exceed the slope per mmol/L, agreement for the change within a pair is poorer than for single values, and the readings cover amplitude only. The repeatability limit depends on how heavy tails are handled (1.73 mm pre-specified, 0.53 mm robust and post hoc) and covers variation within minutes, not over hours. Intervals are not adjusted for multiple analyses, the sensitivity analyses share the same pairs, the expected slope was not blind, the random splits of the database are not independent, and the data come from one emergency department. Replication in an independent cohort is missing.

Studies with larger potassium changes, documented sampling times, a positive control and an independent reference reading are needed to answer the question this analysis leaves open.

Declarations

Code availability

Analysis scripts are available at <https://github.com/leoprobst/ecg-auswertung-code> (release v1.3.0, folder s3_mcmed; MIT licence); the frozen LoopLens snapshot in that repository is under PolyForm Noncommercial 1.0.0 and was not used in this study. This follows clause 8 of the PhysioNet Credentialed Health Data License 1.5.0. Aggregate results are provided in the Supplementary Material.

Data availability statement

MC-MED (version 1.0.1; Kansal et al.; <https://doi.org/10.13026/wvyw-g663>) is available to credentialed users on PhysioNet under the PhysioNet Credentialed Health Data License 1.5.0.[9,10] VitalDB (version 1.0.0; doi:10.13026/czw8-9p62; CC BY 4.0) is open.[14,15] The standard citation of the PhysioNet resource is given in reference [11]. Derived patient-level data cannot be shared under the data use agreement. Aggregate results are provided in the Supplementary Material. No individual signals or identifiers are redistributed.

Ethical Approval and Consent to participate

This study is a secondary analysis of a de-identified research database. The creation and release of MC-MED was approved by the Stanford University Institutional Review Board and Secondary Data Use Committee with a waiver of consent.[9,10] Access to the credentialed MC-MED data was obtained under the PhysioNet Credentialed Health Data Use Agreement after completion of the required Collaborative Institutional Training Initiative (CITI) training. The database was de-identified by its providers before release; it was analysed as anonymous data, and no attempt was made to re-identify individuals. Additional ethics approval or informed consent was not sought, because the data are de-identified and the analysis follows the terms of the PhysioNet data use agreement, consistent with published secondary analyses of these resources.

Consent for publication

Not applicable. The manuscript contains no individual patient data, images or identifiers.

Competing interests

The author is the developer of LoopLens (non-financial interest). LoopLens was not used in this study; the measurement algorithm and the analysis are separate code of the author, to be released with this work, and use the NeuroKit2 toolbox.[16] The author has no financial interest in LoopLens or in any related company or product, and no other competing interests.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors. The work was self-funded by the author.

Authors' contributions

L.P. conceived the study, developed the software, curated the data, performed the analyses, prepared the figures and wrote the manuscript. L.P. approved the final version.

Acknowledgements

The author thanks the PhysioNet team and the investigators who created and maintain MC-MED.

Use of generative AI

The author used generative AI tools (Anthropic Claude and OpenAI Codex) to assist with analysis code, literature searches, data analysis workflows, drafting and editing of text, and figure preparation. AI-assisted output was reviewed, corrected where necessary and approved by the author, who takes full responsibility for the content. Patient-level analyses were run locally. AI tools are not listed as authors.

Registration and related work

The study was not prospectively registered. The specification was fixed and hashed before the primary analysis was run (Supplement S1); this is not prospective registration. This manuscript is posted as a preprint on Research Square. The author's related manuscripts use other databases.

Provenance and peer review

Not commissioned. This preprint has not been peer reviewed.

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Figures

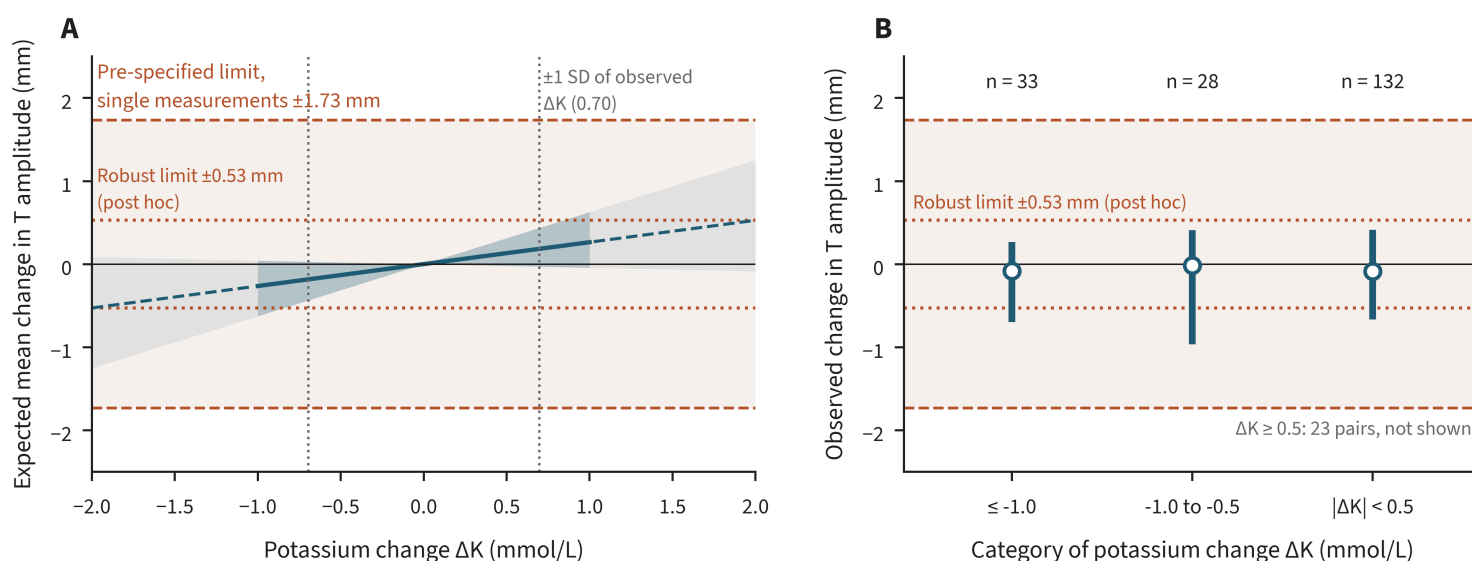


Figure 1

Expected and observed change in T amplitude per potassium change. A: Expected mean change (line) from the primary slope with its 95% confidence interval (band; the interval of the slope is projected over the potassium change and does not include the intercept), for a potassium change from -2 to +2 mmol/L. Dotted vertical lines mark plus and minus one SD of the observed potassium change (0.70 mmol/L); beyond about 1 mmol/L the line is extrapolation, and fewer than 10 pairs rose by 1 mmol/L or more. B: Observed median change (point) and interquartile range (bar) in the three categories of potassium change with at least 10 pairs each; n above each bar. Pairs with a rise of 0.5 mmol/L or more

(23 pairs) are not shown. The dashed lines mark the pre-specified repeatability limit (plus and minus 1.73 mm) and the dotted horizontal lines the robust limit calculated after the results were known (plus and minus 0.53 mm), the limits for the difference between two single measurements of the same sample. They are context for single measurements, not a yardstick for the slope. Bars show the middle half of the pairs.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [ProbstmonitorECGpotassiumsupplement.pdf](#)