Estimation Accuracy of a Reduced Lead System During Simulated Ischemia

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Abstract

In this study we investigate the influence of ischemic events on the estimation accuracy of a reduced lead system based on leads I, II, V2 and V5. The estimation accuracy of this lead system has been assessed for generalized and patient specific lead derivation. Assessments have been performed on a piecewise homogenous torso model that has been used to simulated ischemic events of different size and location.

The patient specific lead derivation approach achieved root mean squared error (RMSE) values of 4.7μV, 24.4μV, 4.4μV and 2.6μV for derived leads V1, V3, V4 and V6 when no ischemia was simulated. It was found that RMSE values for patient specific derived leads increased when ischemia was simulated. Median RMSE values calculated for each lead and across all simulated ischemic events were found to be 40.0μV, 126.7μV, 38.2μV and 35.1μV for derived leads V1, V3, V4 and V6. These values are comparable to the median RMSE values 52.7μV, 141.7μV, 46.9μV and 20.3μV for derived leads V1, V3, V4 and V6 that were found when generalized lead derivation was performed for all ischemic events.

1.   Introduction

A commonly applied procedure for the non-invasive diagnosis of heart disease is the 12-lead electrocardiogram (ECG). Recording a 12-lead ECG requires the application of ten electrodes to the patient [1]. In some situations ten electrodes are considered impractical [2] as the recording electrodes and associated cabling can obstruct other diagnostic procedures [3] and emergency interventions [4]. In addition, maintaining ten electrodes during continuous monitoring can also be difficult in some patients [5]. Reduced lead systems (RLS) aim to address the aforementioned practicality issues of the 12-lead ECG in continuous patient monitoring [6]. RLS derive or estimate the 12-lead ECG through the utilisation of a reduced number of recording electrodes [7]. Unrecorded or so-called target leads are thereby derived from the recorded or so-called basis leads [1]. This allows the familiar 12-lead ECG format to be displayed for interpretation whilst streamlining the recording procedure.

The recording electrodes of RLS may be a subset of the 12-lead ECG electrodes [3] or they may be located at other anatomical landmarks [8]. Electrode locations that consider factors such as patient comfort, immunity against motion artefact, interference with diagnostic procedures or clinical interventions in addition to their location on easy to identify landmarks may thus be chosen.

Although, these RLS have been demonstrated as capable of producing a close estimation of the 12-lead ECG [1], differences between derived and recorded 12-lead ECGs do exist [2]. In particular, the negative impact of ischemia on the estimation accuracy has previously been identified [1, 9]. Furthermore, in recognition of the differences between derived and recorded 12-lead ECGs, the AHA/ACC/HRS have released a scientific statement [10] recommending that derived 12-lead ECGs are clearly marked to ensure that they are distinguishable from their recorded 12-lead ECG counterparts.

In this study, we investigate further the impact of ischemic events on the estimation accuracy of RLS. In particular, we investigate how different ischemic events impact upon the similarity between derived and recorded STT segments. This analysis is performed for a commercially available RLS that derives target leads V1, V3, V4 and V6 from recorded basis leads I, II, V2 and V5 [11].

2.   Material and methods

We base our research on the realistically shaped piecewise homogenous human torso model used in ECGSIM [12]. This model is built up from triangulated geometries for the heart, lungs and torso. The heart geometry is defined by 257 nodes. In this model, the body surface potential map (BSPM) is calculated by

multiplication of the transmembrane potential, that is assigned to each heart node, with a forward matrix. The shape of the transmembrane potentials that are used within the ECGSIM model is defined by three parameters namely depolarization time, repolarisation time and action potential amplitude. These parameters can be adjusted to simulate ischemia through alteration of the transmembrane potentials.

The left ventricle of the triangulated heart model was divided into 10 segments. This segmentation was previously described in [13]. The authors in [13] chose this segmentation in consideration of the coronary artery distribution that has previously been defined by Selvester *et al.* [14]. Figure 1 illustrates the 10 left ventricular segments in a 2 dimensional Mercator projection of the left ventricle that is based on [14]. The assignment of heart nodes that make up each of the segments was the same as it was used in [13].
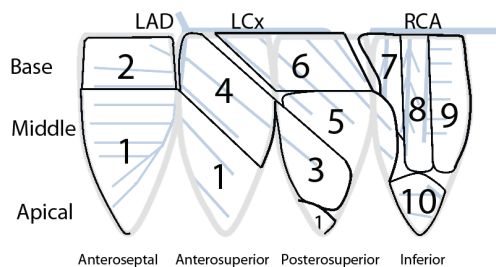

Figure 1: Location of the 10 left ventricular segments that are used in the simulation of the 25 ischemic events. Left anterior descending artery (LAD). Left circumflex artery (LCx). Right coronary artery (RCA).

We extended the segmentation of the triangulated heart model to the right ventricular free wall. The right ventricular free wall was divided into the 8 distinct segments that are shown in Figure 2. This segmentation was chosen taking into consideration of the right coronary artery distribution.
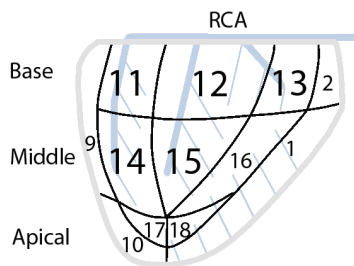

Figure 2: Location of the 8 left ventricular segments that are used in the simulation of the 25 ischemic events. Right coronary artery (RCA).

The 18 regions identified in Figure 1 and Figure 2 were combined and used to simulate 25 transmural ischemic events of different sizes (Table 1). Ischemic events 1 to 23 of Table 1 were previously defined in [13]. Ischemic events 24 and 25 were included in order to simulate involvement of the right ventricular free wall.

Simulation of ischemia was based upon changes to the default parameter values of the nodes that lay inside an ischemic area. Depolarization time for nodes within an ischemic region was delayed for 15ms [13]. Action potential duration and action potential amplitude were reduced to 80% of their normal values [13]. BSPMs were then calculated for each of the 25 ischemic events and when no ischemia was presented. This resulted in a total of 26 BSPMs. Basis leads I, II, V2 and V5 and Mason Likar (ML) 12-lead ECGs were extracted from these BSPMs. The basis leads were then used to derive target leads V1, V3, V4 and V6. Both patient specific and generalized lead derivation were assessed.

Table 1: Simulated ischemic events with ventricular segments as illustrated in Figure 1 and Figure 2.

Ischemic Event #	Ventricular segments	Ischemic Event #	Ventricular segments
1	1	14	5&6&7
2	10	15	5&6
3	1&2&10	16	5
4	1&2&3&4&10	17	7&8&9&10
5	1&2&3&4	18	7&8
6	1&3&4&10	19	7
7	1&3&4	20	8&9&10
8	3&4	21	8&9
9	1&2	22	8
10	5&6&7&8&10	23	9
11	5&6&7&8	24	9&10&17&11&14
12	5&6&7&8&9&10	25	9&10&11&12& 14&15&17
13	5&6&7&8&9		

Patient specific transformation coefficients were developed by multiple linear regression analysis over the entire QRST of the normal (non-ischemic) ECG. Transformation coefficients published in [15] were used for generalized lead derivation.

Root mean squared error (RMSE) was used to assess the similarity between derived and actual target leads. This assessment was performed for the STT segment.

The diagnostic classifications of both the actual and derived ML 12-lead ECGs were obtained using the ESC/ACCF/AHA/WHF ECG criteria [16] for diagnosis of acute myocardial ischemia. The diagnostic classification was analyzed for ischemic events that were identified by the actual ML 12-lead ECGs and missed by the derived ML 12-lead ECGs.

3. Results

In Figure 3 and Figure 4 RMSE values are presented that have been calculated over the STT segment between the derived and the recorded target lead V4. RMSE values are shown for the ischemic events described in Table 1. Ischemic events have been arranged, in accordance to their corresponding RMSE, from low (left) to high (right). Figure 3 details the RMSE values based

upon patient specific lead derivation. RMSE values based on generalized lead derivation are shown in Figure 4. The solid line in Figure 3 and Figure 4, illustrates the RMSE value that has been obtained for the normal ECG (in the absence of ischemia). It can be seen, that this value is lower in the patient specific lead derivation approach (Figure 3).
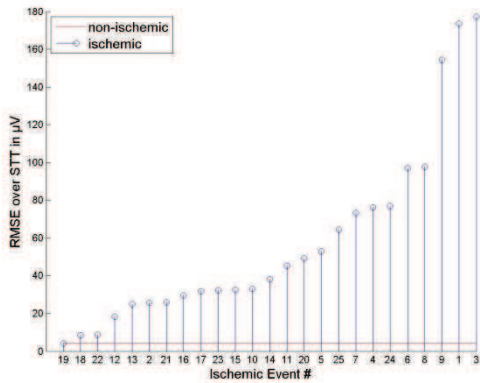

Figure 3: Root mean square error values in µV. Calculated over the STT segment between patient specific derived and recorded target lead V4. Solid line RMSE based on the non-ischemic case. Vertical bars RMSE based on the 25 simulated ischemic events.
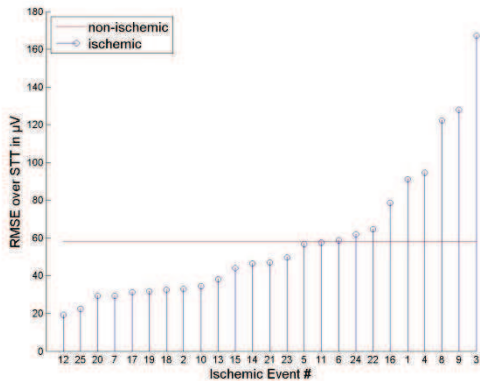

Figure 4: Root mean square error values in µV. Calculated over the STT segment between derived and recorded target lead V4. RMSE values shown are based upon generalized lead derivation. Solid line RMSE based on the non-ischemic case. Vertical bars RMSE based on the 25 simulated ischemic events.

Although space does not permit illustration of the performance for each of the derived precordial leads it was found that the RMSE values of target leads V1, V3 and V6 had similar overall profiles to that illustrated in Figures 3 and 4 for V4. Nevertheless, the order of the ischemic events on the x-axis and their corresponding RMSE values did differ.

Table 2 details a summary of the findings obtained for all target leads. Column 1 of Table 2 indicates the derivation method (pd = patient specific derived, gd = generalized derived) and the derived lead. Results obtained using personalized and generalized derivation of target lead V1 e.g. are shown in rows 1 (pdV1) and 2 (gdV1) respectively. RMSE values for the non-ischemic case are shown in column 2 of Table 2. Minimum, median and maximum RMSE values (calculated over all 25 ischemic events of table 1) are shown in columns 3 to 5. The six ischemic events that are associated with the largest RMSE values (circa the 4th quartile of the RMSE values) are shown in column 6 of Table 2.

Table 2: RMSE values between derived and recorded target leads (V1, V3, V4 and V6) as calculated over the STT segment. Also listed are the ischemic events that are associated with the 4th quartile of the RMSE values for each lead.

Derivation method / Derived lead	RMSE [µV] Non-Ischemic	RMSE [µV] Ischemic			Ischemic Events
		min	med	max	
pdV1	4.7	9.3	40.0	176.9	6;9;25;5;3;4
gdV1	55.0	18.6	52.7	220.8	6;25;9;5;3;4
pdV3	24.4	16.1	126.7	508.5	4;7;3;5;1;9
gdV3	22.1	13.7	141.7	481.0	4;7;3;5;1;9
pdV4	4.4	4.0	38.2	177.1	24;6;8;9;1;3
gdV4	58.2	19.2	46.9	167.1	16;1;4;8;9;3
pdV6	2.6	4.6	35.1	90.6	9;25;24;6;4;3
gdV6	13.6	6.2	20.3	72.5	20;6;25;4;3;24

Ischemic events 1, 3 and 6 of Table 1 were missed when the ESC/ACCF/AHA/WHF ECG criteria [15] for diagnosis of acute myocardial ischemia was applied to derived ML 12-lead ECGs. These ischemic events were missed for both, the patient specific derived and the generalized derived leads. The ischemic events, were however identified when the ECG criteria [15] was applied to the actual ML 12-lead ECGs.

4. Discussion

The results have shown that, in the absence of ischemia, lower RMSE values are obtained when patient specific lead derivation is employed. This finding has been reported in previous studies where real patient data has been used [17]. When ischemic events are simulated the RMSE values during patient specific lead derivation increase to a level that is comparable to that obtained during generalised lead derivation.

Ischemic events 1, 3 and 6 were missed, when the ESC/ACCF/AHA/WHF ECG criteria [16] was applied to the derived ML 12-lead ECGs. However, measurements taken from the derived ECGs were only marginally short of the threshold dictated by the criteria. This would be obvious to a human observer but may not be detected by a computerized algorithm.

The greatest RMSE differences (calculated over the STT segment) between derived and actual precordial leads were found for acute anterior myocardial infarction. Similar findings based on real patient data have been reported in [1]. This provides confidence in the modelling approach that has been applied.

However, the findings presented in this study are based solely on simulations and therefore require further validation with real patient data. Furthermore, the effects of different torso geometries and heart orientations have not yet been investigated.

5.   Conclusion

Our simulations have shown that, in the absence of ischemic events (pathological changes), patient specific derivation is superior to generalised derivation. We have shown that the superiority of the patient specific approach is largely compromised in the presence of ischemic events.

This finding raises questions about the superiority of the patient specific lead derivation approach for continuous monitoring. It also highlights the importance of evaluation of such systems on data that reflect pathological changes. It is the likelihood of such changes that necessitates recording/monitoring in the first instance. Further research on recorded 12-lead ECGs with ischemic episodes is required to compare the simulation based results with real patient data.

Acknowledgements

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