Predicting Acute Hypotensive Episodes from Mean Arterial Pressure

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Abstract

Acute hypotensive episodes (AHE) are serious clinical events in intensive care units. We present an algorithm for automated computer prediction of AHE in patients using mean arterial pressure (MAP). We defined an AHE index based on the observation that patients with documented AHE experienced more transient reductions in MAP compared to those without AHE. The algorithm was developed and tested using the PhysioNet/Computers in Cardiology Challenge 2009 data sets. The algorithm, which classifies records with at least one occurrence of a reduction in MAP to 65 mmHg for at least 75% of a 30 minute window as AHE positive, correctly classified 8 out of 10 records in test set A and 28/40 records in test set B.

Using MAP alone the algorithm achieved modest accuracy for prediction of AHE in patients. The algorithm could be improved by taking account of temporal changes in MAP.

1. Introduction

Acute hypotensive episodes (AHE), defined here as incidences of mean arterial pressure (MAP) falling below 60 mmHg for at least 90% of any 30 minute period, are serious clinical events in intensive care units [1]. Several clinical studies have proved that AHE could result in multiple organ failure and they are strongly linked to morbidity and mortality [2]. Identifying patients with AHE would be an important step for clinicians or physicians to respond in a timely manner and perform necessary patient-specific therapeutic intervention. It is known that characteristics of patients and their clinical states are associated with an increased risk for AHE. Based on the relevant variables, the applications of computer-based predictive models like logistic regression models and artificial neural networks are potential predictors of AHE [3, 4].
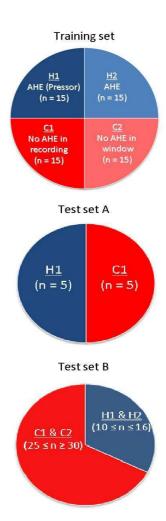

Figure 1. Schematic representation of the Challenge data sets. Blue represents patient data with AHE within the forecast window and red represents patient data without AHE in the forecast window.

But, the performance of these models highly depends on the numbers of parameters inputted. Since hemodynamic parameters such as blood pressure and heart rate are the obvious signs of hypotension, some researchers proposed to use heart rate variability to classify patients with AHE [5]. Our aim was to design algorithms using only MAP for identifying patients susceptible to AHE.

2.   Methods

Data was provided by the 2009 PhysioNet/Computers in Cardiology Challenge and comprised training and test sets as illustrated in figure 1 [6]. Further details of the Challenge are provided in [7]. Our focus was to design, using the training set, an algorithm to differentiate patients with documented AHE events occurring within a 1 h forecast window from those with no documented AHE events, and to test the algorithm against the test set (test set A). Additionally, we tested the algorithm's ability to differentiate patients with AHE events in the forecast window from those without events in the forecast window but who may or may not have documented AHE events using a second test set (test set B). The test set record classifications were unknown until after the algorithm had been tested on the test set data.

Based on a visual inspection of the MAP data from the training set we observed that, compared with patients without AHE, patients with AHE appeared to have i) lower average MAP and ii) more episodes of transient reductions in MAP, including episodes where MAP did not reduce sufficiently or for long enough to meet the criteria (< 60 mmHg for 90% of any 30 minute period) for AHE events. Accordingly we defined two indices:

i) aMAP, the average MAP across the recording of the patient;

ii) AHE index, the proportion of MAP in a 30 minute sliding window falling below a threshold pressure. The index was calculated at two thresholds, 65 and 70 mmHg. Hence, this index sought to quantify events in which the reduction in MAP was not sufficient in amplitude or duration to be classified as an AHE event.

3.   Results

The hypotensive patients in the training set had significantly lower aMAP than the non-hypotensive
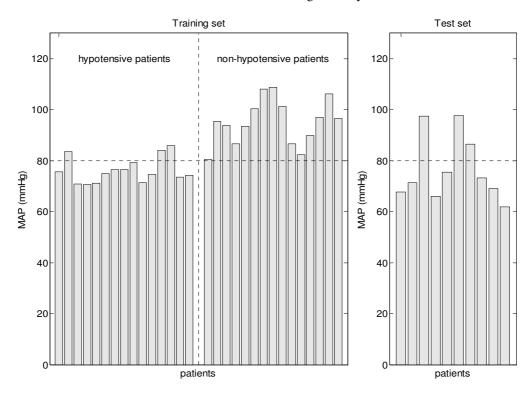

Figure 2. aMAP for all patients in the training set and test set A. In the training set all the non-hypotensive patients have aMAP greater than 80 mmHg. However, in the test set there are only three patients with aMAP above this level.

patients (76 (71-81) mmHg vs 95 (84-104) mmHg, mean (95% CI) p < 0.001). In the non-hypotensive group aMAP was greater than 80 mmHg for all patients and only 20% (3/15) of the hypotensive group had aMAP greater than 80 mmHg. On this basis we used aMAP < 80 mmHg as a predictor of AHE. However, in test set A only 30% (3/10) of cases had aMAPs greater than 80 mmHg so we were unable to classify the 5 patients with and without hypotension using this index. Nonetheless, cases above the threshold were likely candidates for the non-hypotensive group. Figure 2 compares the aMAP of the hypotensive and non-hypotensive members of the training set along with the aMAP for members of the test set. It was later confirmed that the 5 records with the lowest aMAP were the correct AHE classifications, indicating that aMAP is a sensitive predictor with a threshold of 72 mmHg.

In the training set, all of the hypotensive patients had episodes in which the AHE index exceeded 0.75 (in other words, more than 75% of the 30 minute window was lower than the threshold) for both thresholds of 65 and 70 mmHg with on average 24 (range 5 to 51) and 35 (range 10 to 71) episodes per patient respectively. None of the

non-hypotensive patients had AHE index exceeding 0.75 for a pressure threshold of 65 mmHg, but for a threshold of 70 mmHg, there were two episodes in this group. Hence, we considered AHE index > 0.75 with a threshold of 65 mmHg to be a positive index of patients susceptible to AHE.

Figure 3 illustrates the variability in AHE index across the MAP recordings for each patient in test set A. 5 patients in the test set had AHE index > 0.75 so were classified as hypotentive and the remainder classified as non-hypotensive. Two of these classifications were incorrect: record 102bn was classified as non-hypotensive and record 105bn was classified as hypotensive.

When applied to test set B the algorithm achieved 28 (70%) correct classifications.

4. Discussion and conclusions

Our algorithms have demonstrated the feasibility of developing algorithms for automatic prediction of AHE in the ICU using blood pressure only. We achieved modest accuracy of predicting AHE events with algorithms based on our observations that patients who have AHE have lower average MAP and more transient
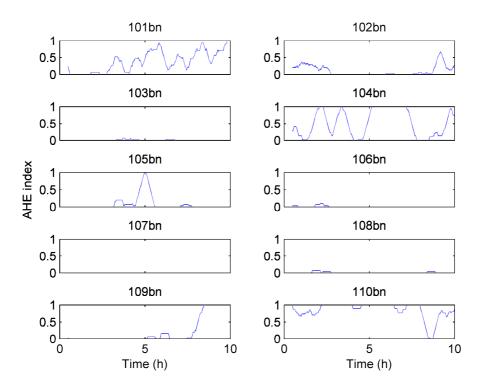

Figure 3. AHE index indicating the proportion of MAP in a 30 minute sliding window falling below 65 mmHg for patients in the test set. The data is for the 10 hours preceding the forecast window. Records 101bn, 104bn, 105bn, 109bn, 110bn exceeded the threshold so were classified as hypotensive by our algorithm. Records 102bn and 105bn were incorrectly classified by the algorithm.

reductions in MAP compared to those who do not have AHE. The algorithms could be further developed to increase accuracy by combining the algorithms so that both average MAP and AHE index contribute to the prediction. For example, a patient with low average MAP, experiencing large transient reductions in MAP is more likely to experience an AHE than a patient with low average MAP and no transient reduction in MAP. Additionally the temporal dynamics of these parameters might by used to increase predictive accuracy.

Acknowledgements

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