Published in final edited form as:

Sleep Breath. 2016 May ; 20(2): 515–522. doi:10.1007/s11325-015-1219-6.

Automated Detection of Atrial Fibrillation from the Electrocardiogram Channel of Polysomnograms

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Abstract

Purpose—Accurate identification of atrial fibrillation episodes from polysomnograms is important for research purposes, but requires manual review of a large number of long electrocardiographic tracings. As automated assessment of these tracings for atrial fibrillation may improve efficiency, this study aimed to evaluate this approach in polysomnogram-derived electrocardiographic data.

Methods—A previously described algorithm to detect atrial fibrillation from single-lead electrocardiograms was applied to polysomnograms from a large epidemiologic study of obstructive sleep apnea in older men (Osteoporotic Fractures in Men [MrOS] Sleep Study). Atrial fibrillation status during each participant's PSG was determined by independent manual review. Models to predict atrial fibrillation status from a combination of algorithm output and clinical/polysomnographic characteristics were developed and their accuracy was evaluated using standard statistical techniques.

Results—Derivation and validation cohorts each consisted of 1395 individuals; 5% of each group had atrial fibrillation. Model parameters were optimized for the derivation cohort using the Akaike Information Criterion. Application to the validation cohort of these optimized models revealed high sensitivity (85-90%) and specificity (90-95%) as well as good predictive ability, as assessed by the C statistic (> 0.9) and generalized R^2 values (~ 0.6). Addition of cardiovascular or polysomnogram data to the models did not improve their performance.

Conclusions—In a research setting, automated detection of atrial fibrillation from polysomnogram-derived electrocardiographic signals appears feasible and agrees well with

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Conflicts of interest: none

manual identification. Future studies can evaluate the utility of this technique as applied to clinical polysomnograms and ambulatory electrocardiographic monitoring.

Keywords

RR intervals; statistical modeling; atrial fibrillation; polysomnography

Introduction

Over the past decade, the relationship between obstructive sleep apnea (OSA) and atrial fibrillation (AF) has become increasingly well-established, in part due to findings from large-scale epidemiologic studies [1, 2]. The accurate identification of AF cases from polysomnographic (PSG) data is critical to these efforts and is usually carried out by manual review of the EKG signal that is recorded as part of the PSG. As these studies typically enroll thousands of participants, this process can be time-consuming (particularly if the investigation focuses on paroxysmal AF [3]) and could be susceptible to both false-negative and false-positive results. Therefore, an automated method to screen the EKG signal may improve the efficiency and accuracy of AF detection during PSG evaluation.

A recently described algorithm for AF detection from single-lead EKG signals showed encouraging results when tested on annotated tracings from PhysioNet, a publicly available source of physiologic signals [4]. However, the algorithm has not yet been applied to unprocessed data from a research or clinical setting. In addition, the prior study used pre-defined thresholds to dichotomize the algorithm's main outcome parameter in order to assign each record a binary AF status; this methodology does not incorporate all of the available information (as the output of the algorithm is fundamentally continuous) and also does not permit evaluation of the entire range of probabilities of AF occurrence. More specifically related to PSG-related applications, the initial report emphasized analysis of short segments of EKG signals (i.e. 32-128 RR intervals), whereas PSGs contain EKG recordings that are several orders of magnitude greater in length.

Therefore, to gauge the performance of this algorithm and applicability to typical PSG records collected in research (and clinical) studies, we evaluated this promising technique using PSG data from a large-scale epidemiologic study of OSA (Osteoporotic Fractures in Men [MrOS] Sleep Study). Specifically, we sought to (1) determine the effects of variation in user-defined parameters on algorithm performance; (2) evaluate the algorithm's ability to determine the AF status of EKG data obtained from PSGs; and (3) determine whether the algorithm added discriminatory capability to assessment of AF status beyond readily available clinical and polysomnographic data.

Methods

Cohort characteristics and data collection

The MrOS Study is a study of 5994 men, ages 65 and older, initially recruited from six clinical centers in the United States between 2000 and 2002. Study design and recruitment have been published elsewhere [5, 6]. Between 2003 and 2005, the Outcomes of Sleep

Disorders in Older Men (MrOS Sleep) Study, an ancillary study of MrOS, recruited 3135 participants for a comprehensive in-home sleep assessment. The inclusion criteria for MrOS Sleep are similar to the main study (community-dwelling men 65 years or older who were able to walk without assistance and without history of bilateral hip replacement) and the exclusion criterion is current treatment for sleep-disordered breathing. Of the 3135 participants enrolled in the MrOS Sleep Study, 2911 had usable PSG data and 2790 had readily available EKG tracings.

Single-lead EKG tracings from the in-home PSGs were manually reviewed by a single registered polysomnologist with EKG training. A board-certified critical care physician confirmed occurrences of AF and questionable cases were arbitrated by a cardiologist. Based on these reviews, each PSG study was evaluated for the presence or absence of AF; this assignment was considered the 'gold standard' against which the algorithm's performance was measured. Studies with paroxysmal AF (i.e. discrete episodes of AF occurring within a background of predominantly sinus rhythm) were included in the AF group, but were analyzed separately as well. Studies with atrial flutter (AFL) were excluded.

Sleep-related co-variables used in the present study include apnea-hypopnea index (AHI), central apnea index (CAI), and percentage of total sleep time with oxygen saturation less than either of two thresholds of hypoxemic stress (90% and 80%). AHI and CAI are measures of the severity of sleep-disordered breathing; AHI is the number of all apneas (obstructive and central) and hypopneas (accompanied by $\geq 4\%$ desaturation) that occurred per hour of sleep and CAI is a similar metric that measures only central apneas. The details of PSG data collection and respiratory event scoring have been described previously [2]. Information regarding the cardiovascular co-variables of hypertension, heart failure, and coronary heart disease (myocardial infarction and/or either percutaneous or surgical revascularization) was obtained from questionnaire data.

Pre-analysis EKG signal processing

During the PSG, the single-lead EKG signal was acquired at a sampling rate (f_s) of 256 Hz. For each PSG, the sample number of each R wave was automatically identified from the EKG tracings using dedicated software (Somte – CompuMedics; Abbotsford, Victoria, Australia). Series of RR intervals were constructed using the number of samples between consecutive R waves and the known sampling frequency ($RR_i = [1/f_s] * [R_{i+1} - R_i]$). RR intervals outside of a reasonable physiologic range (i.e. < 250 ms or > 2000 ms) were removed as they likely represented artifact; excluded RR intervals were uncommon ($0.3 \pm 0.5\%$ per participant) and were predominantly short (< 250 ms).

AF detection algorithm and RR series analysis

The AF detection algorithm [4] constructs a plot from a segment of consecutive RR intervals (of a given window length 'n', where n = the number of RR intervals in that segment; Figure 1a) that consists of current RR interval duration (RR_i) on the horizontal axis and change in RR interval duration ($dRR_i = RR_i - RR_{i-1}$) on the vertical axis (Figure 1b). Each (dRR , RR) pair represents a point on a grid, comprised of squares (cells) of user-specified size (representing temporal resolution). The key output of the algorithm is the number of non-

empty cells (NEC), defined as the number of squares on the grid occupied by at least one point from a given (dRR, RR) series. Qualitatively, for a grid composed of cells of a given size, a series consisting of sinus rhythm (with a narrow range of RR intervals) would have relatively few NECs and a series with AF would be expected to have a relatively large NEC count due to the inherent irregularity of AF. The algorithm assigns a classification of AF or non-AF to an RR series based on the NEC count for that series in comparison to a user-defined threshold. A NEC count greater than the threshold leads to that series being labeled as 'AF', whereas a NEC count below the threshold results in a designation for that series as 'non-AF'.

For the present study, the detection algorithm was adapted from its original description with several modifications to the analytic approach. We used NEC percentage (NECp: defined as $[\text{NEC count}/\text{window length}] \times 100$) as the key algorithm output rather than absolute NEC count due to the ranges of window lengths and cell sizes that were evaluated (see below). To minimize the effects of discretizing a continuous measure in our main analysis, we formulated models of AF probability as a function of NEC percentage rather than using a threshold to assign a binary AF status. For completeness, standard receiver-operating curve (ROC) analysis was performed as well.

RR series derived from PSGs differ from the standardized data sets that were used in the algorithm's initial evaluation in that they are considerably longer and may contain a more heterogeneous distribution of RR interval durations (and thus dRR durations). As a result, the performance of the algorithm may be more dependent on cell size and/or window length. Therefore, for each RR series, we evaluated combinations of grid sizes (10 ms, 25 ms, 100 ms) and window lengths (100 RR intervals, 1000 RR intervals, 10000 RR intervals, entire series). For each cell size, the RR series was divided into segments each consisting of the specified number of RR intervals and a distribution of NECp was generated (one NECp for each segment). We chose the 9th decile of NECp to summarize each RR series as this was the least redundant in formal analyses. When an entire RR series was analyzed as a single segment (i.e. window length = total number of RR intervals in the series), only one NECp was generated for that series.

Statistical analysis

The cohort was randomly divided in half to generate a derivation group and validation group. For the derivation group, logistic regression models were fit to assess the ability of NECp to predict AF status (using log odds of AF). Initially, 4 models were fit for each combination of window length and cell size. These include: (1) age and sleep-related variables alone; (2) NECp as a linear term; (3) NECp as a linear term + NECp as a non-linear term; and (4) age/sleep-related variables + NEC as a linear term + NECp as a non-linear term. The Akaike Information Criterion (AIC) was used to compare the relative predictive ability of these models for a given window length and cell size. The C index and generalized R^2 were used to compare the performance of the models across the various combinations of window length and cell size.

Based on the results from the derivation cohort, the best-performing models (i.e. those with the lowest AIC and the highest C index and/or R^2 values) were applied to the validation

cohort. Probability of AF, as determined by the models, was compared between the non-AF and AF groups. For completeness, standard ROC analyses were performed as well by generating sensitivity and specificity using varying thresholds of NECp.

Comparisons of continuous variables were performed using the Wilcoxon rank-sum test and comparisons of categorical variables were made using the Pearson chi-squared test; p values < 0.05 were considered significant. Analyses were performed using the R statistical package (version 3.0.1; R Foundation for Statistical Computing – Vienna, Austria).

Results

Cohort characteristics

Cohort demographics and sleep characteristics stratified by sub-group assignment (derivation vs validation) are shown in Table 1. There was no difference in RDI or CAI between the groups. While there were statistically significant differences between the groups in age as well as percentage of sleep time with oxygen saturation below 90% and 80%, the clinical relevance of these absolute differences is likely small.

NECp as a function of cell size and window length

Figure 2a shows the relationship between NECp and both cell size and window length for the derivation cohort. For a given cell size, longer windows are associated with decreased NECp; similarly, for a given window length, larger cell size (decreased temporal resolution of the grid) results in a lower NECp as well. Of note, the range of NECp (i.e. maximum-minimum NECp) was greatest for 4 combinations of cell size/window length: 10 ms/1000 intervals, 10 ms/10000 intervals, 25 ms/1000 intervals, and 100 ms/100 intervals (Figure 2b).

Comparison of NECp to demographic and sleep characteristics as predictors of AF status

For each combination of cell size and window length, Table 2 displays the AIC for the models containing age and sleep co-variables, NECp, and their combination. The highest AIC, by far, is for the model containing age and sleep-related co-variables alone; this decreased with the addition of any NECp term. For the majority of combinations of window length and cell size, the addition of a non-linear NECp term resulted in a lower or minimally increased AIC, suggesting a non-linear component of the relationship between probability of AF and NECp. Interestingly, for all but 1 combination of window length and cell size, the addition of age/sleep co-variables to a model containing only NECp terms lead to an increased AIC, suggesting a detrimental effect in terms of predictive capability. Substitution of cardiovascular co-variables for sleep-related variables in the above modeling approach yielded similar results.

Effect of cell size and window length on model performance

Table 3 shows several indicators of model performance as a function of cell size and window length. As assessed by the C statistic, all combinations of cell size and window length yielded good model fits. The effect of adding clinical co-variables to NECp-based models was essentially negligible. Models using the entire RR series as a single segment performed

modestly worse than those containing shorter segment lengths. The R^2 analysis suggests that the 4 cell size and window length combinations that resulted in the largest range of NECp (10 ms/1000 intervals, 10 ms/10000 intervals, 25 ms/1000 intervals, and 100 ms/100 intervals) gave results that explained a larger percentage of the variability in AF status than other combinations. The relationship between probability of AF and NECp for these combinations is displayed graphically in Figure 3. The distribution of NECp is shown along the horizontal axis. In each of these models, there is a reasonably uniform distribution of NECp across the range of possible values, consistent with the relatively large ranges shown in Figure 2b.

Validation cohort

NECp data generated from the validation cohort using the 4 ‘best-fit’ combinations of cell size and window length from the derivation cohort were subsequently used to test the model's ability to predict AF status. The results of the C statistic and R^2 analyses in the validation cohort were similar to the derivation cohort. For each of these combinations, Table 4 shows the predicted probability of AF, stratified by independently adjudicated AF status. Although the mean and median absolute values of the probabilities for the AF group are $< 50\%$, each combination of grid size and window length demonstrates an ability to discriminate between non-AF and AF, as the probabilities for the non-AF group are extremely low ($\sim 1\text{-}2\%$). The sensitivity and specificity analysis for each combination of grid size and window length is shown in Table 5; the NECp thresholds are evaluated in increments of 10%. An ‘optimal point’ on the related ROC curve (i.e. the threshold that yields the maximum sum of sensitivity and specificity) seems to coincide with the beginning of the rise of the corresponding curves from Figure 3, suggesting that most of the discriminatory power of NECp may lie in this region.

Discussion

Addition to current knowledge and application to research/clinical work

This analysis demonstrates that the previously described algorithm [4] can reasonably classify the AF status of a given series of RR intervals extracted from a full overnight PSG. The current work adds to the fundamental description of the algorithm by (1) validating its performance in a large number of records obtained from in-home PSGs (i.e. non-standardized data sets); (2) using the continuous metric of NECp rather than an arbitrary threshold of absolute NEC; and (3) defining the probability of AF in terms of NECp. The shape of the curves that characterize the relationship between probability of AF and NECp can allow for tailoring of the definition of AF, as designated by the algorithm, to a variety of scenarios.

For example, in a research setting, a high-sensitivity ‘screening’ technique may be desirable when searching a large cohort for cases of AF. In clinical polysomnography, identification of AF in patients without a known diagnosis would be important, but implementation of a robust procedure for identifying these arrhythmias may be challenging given the focus on respiratory scoring; therefore, a similar rationale for automated ‘screening’ could be extended to a high-volume clinical Sleep Laboratory as well. If AF can be identified (or

excluded) in a timely and accurate manner, inclusion of this information in a clinical PSG report could be valuable to referring clinicians (potentially leading to medication adjustments and/or referral to a Cardiologist), at no extra cost to the patient. Application to ambulatory monitoring would be less directly translatable as there is likely to be a lesser degree of artifact during sleep than in patients that are awake and, presumably, more active.

This study focuses on application of the previously described algorithm to EKG data derived from PSGs, rather than on comparison of methods for automated AF detection. However, the algorithm's original description addresses its advantages relative to other techniques [4].

Expected impact of cell size and window length on NECp

NECp varied with cell size and window length in an expected fashion. As cell size increases (and temporal resolution of the grid decreases), the total number of cells decreases exponentially and thus the absolute NEC decreases as well. If the window length remains constant, the NECp would decrease as well as there is the same number of RR intervals occupying fewer cells; as cell size increases, NECp approaches its minimum of $(1/\text{window length}) \times 100$. For a given cell size, any given (dRR, RR) pair will occupy the same space on the grid regardless of window length; with increasing window length, the absolute NEC count doesn't change, but NECp decreases. Only points at the 'transitions' (i.e. the beginnings/ends of segments) will be affected by changes in window length; the frequency of these transitions is $\sim (1/\text{window length}) \times 100$. Thus, even at the smallest window length used in this analysis (100 intervals), transitions make up only 1% of the data sets.

Application to paroxysmal AF and challenges with AFL

A NECp-based approach for discerning AF from PSG-related EKG signals could be particularly attractive for cases of paroxysmal AF. The window length could be optimized as to not miss short episodes of AF. In the present cohort, there were only 8 cases of paroxysmal AF and 6 of those were included in the derivation sample. Of the 2 remaining cases, the algorithm assigned a relatively high probability of AF to both. However, more cases need to be evaluated before the algorithm's utility in this situation can be more confidently commented upon. Likewise, the algorithm's ability to discern AFL was not evaluated, primarily due to the small number of AFL cases in this cohort. Furthermore, AFL may be interpreted as sinus rhythm by the algorithm, unless variable atrio-ventricular block is present.

Limitations

There are several additional limitations of this study; most are related to the data sets used to evaluate the algorithm's performance. While NECp emerged as a much stronger predictor of AF status than the demographic and PSG-related co-variables evaluated in these analyses, it is possible that other clinical and sleep-based co-variables may have contributed more predictive value than those examined here. To avoid overfitting, the number of co-variables was constrained by the number of AF cases. This cohort was relatively homogenous regarding the key co-variables of gender (all male) and age (exclusively > 65 years old), so the discriminatory capability of these traditionally powerful factors may not have been adequately assessed. Strictly speaking, it also follows that these results cannot be

extrapolated to younger individuals or women, as these groups were not included in the study. Standard measures of sleep-disordered breathing severity and oxygen desaturation, thought to contribute to the pathogenesis of OSA-related AF [7], seemed reasonable choices for co-variables, although CAI and oxygen saturation < 80% did not vary much over this cohort. Data on common indicators of cardiovascular status (hypertension, coronary artery disease, heart failure) were included here, but may not always be available in sleep laboratory settings.

In summary, this analytic approach shows promise in the automated detection of AF from PSG-derived EKG signals. A next step will include a prospective evaluation of the algorithm's performance in comparison to the standard EKG interpretation that currently accompanies clinically-indicated PSG studies.

Acknowledgments

The authors thank Lori Daniels, M.D. for her thoughtful review of the manuscript.

The Osteoporotic Fractures in Men (MrOS) Study is supported by National Institutes of Health funding. The following institutes provide support: the National Institute on Aging (NIA), the National Institute of Arthritis and Musculoskeletal and Skin Diseases (NIAMS), the National Center for Advancing Translational Sciences (NCATS), and NIH Roadmap for Medical Research under the following grant numbers: U01 AG027810, U01 AG042124, U01 AG042139, U01 AG042140, U01 AG042143, U01 AG042145, U01 AG042168, U01 AR066160, and UL1 TR000128. The National Heart, Lung, and Blood Institute (NHLBI) provides funding for the MrOS Sleep ancillary study "Outcomes of Sleep Disorders in Older Men" under the following grant numbers: R01 HL071194, R01 HL070848, R01 HL070847, R01 HL070842, R01 HL070841, R01 HL070837, R01 HL070838, and R01 HL070839.

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Fig 1. Schematic representation of Cell Size and Window Length

(a) Representative series of RR intervals. The entire series contains 'n' intervals and thus has a window length of 'n'. The top of the panel depicts splitting the series into 2 smaller windows, each containing 'n/2' intervals and therefore each having a window length of 'n/2'. (b) Grid depicting the change in RR interval duration (dRR) as a function of current RR interval duration. The dimension of a representative square cell is illustrated in the lower left corner; this dimension is the cell size. The dots represent individual dRR/RR data points. The non-empty cell percentage of the data set shown here is 80% (8 non-empty cells/window length of the data set, which is 10 RR intervals in this case)

A **B**

Fig 2. Non-empty cell percentage as a Function of Cell Size and Window Length (Derivation Cohort)

(a) The percentage of non-empty cells (NECp) decreases with increasing cell size and with increasing window length. (b) The range of NECp (maximum – minimum) is greatest for the following combinations of cell size and window length: 10 ms/1000 intervals, 10 ms/10000 intervals, 25 ms/1000 intervals, and 100 ms/100 intervals. Data represent the median value for the derivation cohort of the 9th decile of NECp for a given cell size and window length

Fig 3. Model-based Probability of Atrial Fibrillation based on Percentage of Non-Empty Cells (Derivation Cohort)

(a) Cell size 10 ms, Window Length 1000 RR intervals; (b) Cell size 10 ms, Window Length 10000 RR intervals; (c) Cell size 25 ms, Window Length 1000 RR intervals; (d) Cell size 100 ms, Window Length 100 RR intervals. For these combinations of window length and cells size, the distribution of NECp (shown on the horizontal axis) is relatively uniform and covers a wide range of possible values

Table 1
Cohort characteristics

	Derivation cohort (n = 1395)	Validation cohort (n = 1395)	P-value
Age (years)	75.8 ± 5.3	76.6 ± 5.6	< 0.001
Hypertension (%)	51	48	0.2
Heart failure (%)	5	5	1
MI/PCI/CABG (%)	29	28	0.3
SpO2 < 90% (% TST)	3.6 ± 8.3	4.7 ± 10.6	< 0.001
SpO2 < 80% (% TST)	0.06 ± 0.36	0.12 ± 0.62	0.003
AHI (events/hour)	16.4 ± 13.4	17.8 ± 15.3	0.2
CAI (events/hour)	1.3 ± 3.9	1.1 ± 3.7	0.3
Number of AF cases	74	70	-

Values represent the mean ± standard deviation

Comparisons made using the Wilcoxon rank-sum test for continuous variables and the Pearson chi-squared test for categorical variables

AF: atrial fibrillation

AHI: apnea-hypopnea index

CABG: coronary artery bypass graft surgery

CAI: central apnea index

MI: myocardial infarction

PCI: percutaneous coronary intervention

SpO2: oxygen saturation

Table 2
Akaike Information Criterion as a Function of Cell Size and Window Length

Cell size (ms)	Window length (RR intervals)	Age + sleep variables only	NECp linear	NECp linear + NECp non-linear	Age/sleep + NECp linear + NECp non-linear
10	100	562	330	314	322
10	1000	562	278	261	269
10	10000	562	283	284	290
10	Entire series	562	329	318	323
25	100	562	275	252	261
25	1000	562	257	258	264
25	10000	562	297	295	299
25	Entire series	562	366	352	353
100	100	562	257	259	266
100	1000	562	295	294	297
100	10000	562	338	336	337
100	Entire series	562	448	420	419

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Table 3
Model performance as a Function of Cell Size and Window Length

Cell size (ms)	Window length (RR intervals)	NECp linear		NECp linear + NECp non-linear		Demographics + NECp linear + NECp non-linear	
		C	R ²	C	R ²	C	R ²
10	100	0.94	0.48	0.93	0.51	0.93	0.51
10	1000	0.94	0.57	0.92	0.58	0.93	0.58
10	10000	0.94	0.56	0.94	0.56	0.94	0.57
10	Entire series	0.93	0.48	0.93	0.48	0.93	0.49
25	100	0.95	0.57	0.94	0.59	0.93	0.59
25	1000	0.95	0.60	0.95	0.60	0.95	0.61
25	10000	0.94	0.53	0.93	0.53	0.94	0.54
25	Entire series	0.92	0.41	0.92	0.41	0.92	0.43
100	100	0.95	0.60	0.95	0.60	0.95	0.61
100	1000	0.94	0.54	0.94	0.54	0.94	0.55
100	10000	0.93	0.46	0.93	0.46	0.93	0.48
100	Entire series	0.87	0.25	0.88	0.28	0.88	0.30

Table 4
Predicted Probability of Atrial Fibrillation in the Validation Cohort

Cell size (ms)	Window length (RR intervals)	Atrial fibrillation (-) (n = 1325)	Atrial fibrillation (+) (n = 70)
25	1000	0.01 ± 0.04 (0.002)	0.32 ± 0.24 (0.28)
100	100	0.01 ± 0.04 (0.003)	0.29 ± 0.24 (0.24)
10	10000	0.02 ± 0.04 (0.004)	0.29 ± 0.24 (0.22)
10	1000	0.01 ± 0.05 (0.0005)	0.38 ± 0.17 (0.39)

Data are mean ± standard deviation (median)

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Table 5
Model Sensitivity and Specificity based on Window Length and Cell Size (Validation Cohort)

NECp	10 ms/1000 RRs		10 ms/10000 RRs		25 ms/1000 RRs		100 ms/100 RRs	
	Sens	Spec	Sens	Spec	Sens	Spec	Sens	Spec
10	1	0.01	1	0.55	1	0.35	1	0.38
20	1	0.15	0.99	0.85	1	0.75	1	0.78
30	1	0.44	0.88	0.96	0.97	0.90	0.93	0.93
40	1	0.66	0.49	0.99	0.86	0.97	0.60	0.98
50	1	0.80	0.21	1	0.54	0.99	0.31	1
60	1	0.89	0.03	1	0.29	1	0.08	1
70	0.97	0.95	0	1	0.06	1	0	1
80	0.78	0.98	0	1	0	1	0	1
90	0.25	1	0	1	0	1	0	1