Ann Ist Super Sanità 2003;39(2):195-203

Prediction of atrial fibrillation from surface ECG: review of methods and algorithms

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Summary. - The study aims to review the mathematical methods developed for the prediction of atrial fibrillation by analysis of surface electrocardiographic records in paroxysmal or post-cardiosurgery patients. A risk stratification based on ECG analysis would be very useful either to optimise the prophylactic anti-arrhythmic treatment in high risk patients, or to limit drugs administration in low risk subjects. The works published so far managed to achieve good results in terms of sensitivity and specificity. However, since these methods are not completely reliable yet, their clinical application is still limited. The present study is divided in sections about time domain, frequency domain, premature complexes detection, heart rate variability, and non linear ECG analysis based methods.

Key words: atrial fibrillation, signal processing, cardiovascular system.

Riassunto *(La predizione della fibrillazione atriale mediante ECG di superficie: review di metodi ed algoritmi).* - Il presente lavoro costituisce una rassegna dei metodi matematici sviluppati per la predizione della fibrillazione atriale mediante analisi di registrazioni elettrocardiografiche di superficie, in pazienti parossistici o post-CABG. La stratificazione del rischio sulla base di analisi elettrocardiografiche contribuirebbe sia ad ottimizzare il trattamento profilattico o antiaritmico in pazienti a rischio, sia a limitare la somministrazione di farmaci in pazienti a basso rischio. Buoni risultati in termini di specificità e sensitività sono già stati ottenuti. Tuttavia poiché tali metodi non risultano essere perfettamente affidabili, la loro applicabilità clinica è limitata. Il lavoro presenta metodi basati su: analisi dell'ECG nel dominio del tempo, della frequenza, individuazione di battiti ectopici, studio della variabilità della frequenza cardiaca, metodi di analisi non lineare.

Parole chiave: fibrillazione atriale, elaborazione di segnali, sistema cardiovascolare.

Introduction

Atrial fibrillation (AF) is the commonest cardiac arrhythmia among population, with an increasing prevalence in the elderly (17% in people above 70 years) [1]. It is also one of the most frequent post-operative complication after cardiothoracic surgery (10-40%) [2], thus contributing to prolongation of hospitalisation and to an increase of the related costs [3].

Although older age alone seems to be the strongest predictor for the development of AF [4, 5], in the last decades several studies have focused on finding algorithms able to predict AF by the analysis of surface electrocardiographic records. The importance of defining possible clinical predictors is even greater for patients undergoing post coronary artery bypass grafting (CABG) or other cardiosurgical operations (e.g. aortic valve replacement). A risk stratification based on preoperative tests would be very useful either to optimise the prophylactic anti-arrhythmic treatment (such as drugs or electrical pacing [6]) in patients prone to develop postoperative AF (shortening patient suffering and reducing costs associated to the hospitalisation), or to limit drugs administration in low risk subjects.

AF is the result of a fractionated atrial electrical activity mainly due to the shortening of atrial refractory period, which allows multiple wavelets pass through the atrial mass. If an obstacle in the conduction path exists, a subsequent phenomenon of reentry of the electrical activation can lead to the arrhythmia. Since loss of atrial muscle and increased fibrous tissue are also consequences of the arrhythmia, AF can probably cause both molecular modifications of electrophysiological activity, and structural, functional, autonomic

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and metabolic alterations. These phenomena are known as atrial remodelling [7]. However, it is unclear whether these changes are the primary conditions to AF onset or if they are a consequence of atrial remodelling due to AF. According to the theory proposed by Scherf *et al*. [8, 9], rapidly firing atrial ectopic foci can result in premature atrial complexes, in episodes of atrial tachycardia or AF, thus acting as starting triggers for the initiation of the arrhythmia on a predisposing abnormal substrate [10, 11].

A significant morbidity is strictly associated to AF. Several concomitant and underlying diseases, such as cardiomyopathy, affect patients with AF and a major risk for strokes or thromboembolic events must be taken into account. Management of patients with AF aims to restoration and maintenance of sinus rhythm, ventricular rate control, stroke prevention and concurrent disorders treatment [11].

A pharmacologic approach is usually pursued by administration of antiarrhythmic or anticoagulation drugs. Since each patient has a specific and peculiar drug-response it is difficult to predict which agent will be the most effective. A starting standard treatment is thus prescribed while selected therapy is established subsequently. A non-pharmacologic approach is recommended in patients showing drug resistance or intolerance but its modalities and options can differ depending on the arrhythmia conditions and severity.

In CABG patients AF generally occurs 1-5 days after surgery with a peak incidence on day 2 (Table1). It is usually self-limiting as its symptoms and effects fade within the first few days or weeks after operation. Patients developing post-CABG AF usually show no previous history of AF episodes. This probably means that the anatomic stresses developed as a result of surgery can trigger post-CABG AF on predisposing conditions [12, 13].

All the methods further described are mainly research matters and their clinical application is still limited. A possible explanation could be that although most of these methods show high predictive values in terms of specificity and sensitivity, none of them seem to be completely reliable yet. Because of the lack of a standard definition of P wave duration, time domain techniques cannot be easily compared. The other methods presented hereby do not need the localization of P wave boundaries, but their application led to less significant results.

Note that no specific discrimination based on preoperative patient disease, surgery procedures details or drug administration has been made in the present review, however further details can be found in the original works the text refers to.

Time domain analysis

An abnormal prolongation of P wave duration on the surface ECG reflects the presence of intra-atrial conduction defects [14]. A slowed conduction is a prerequisite for development of a reentrant arrhythmia, as the shortening of refractory period makes the atrial tissue variably sensible to atrial premature depolarisation. This means that significant and consistent information can be extracted evaluating the extent of the abnormal atrial activation through time domain P wave features, such as P wave duration, dispersion, spatial velocity, P terminal force and isoelectric interval length. Predictive values of all these parameter are summarised in Table 2.

P wave duration

The crucial problem in P wave duration measurement consists in the definition of the fiducial points. Starting and ending of the P wave are detected either manually or automatically. In some cases they are defined as the points corresponding respectively to the first (*onset*) and last (*offset*) deflection from the baseline [15-18]; in other cases onset and offset are chosen as the points where signal amplitude rises above a fixed level or falls below the same level [19, 20]. Depending on the lead used to record the ECG signal, a different temporal length can be observed. The maximum P wave duration

Table 1. - Post CABG atrial fibrillation incidence

Authors	Number of patients enrolled in post-CABG studies	Number of patients who developed AF (no other arrhythmias documented)	Mean day of occurrence of AF
Buxton *et al.*, 1981 [14]	99	19 (19%)	3
Klein *et al.*, 1995 [20].	54	16 (36%)	2 to 4
Steinberg *et al.*, 1993 [12]	130	33 (25%)	2.6 ± 2.0
Chang *et al.*, 1999 [18]	120	37 (31%)	5 ± 2
Stafford *et al.*,1997 [19]	189	51 (27%)	2 (0.5 to 7)
Passman *et al.*, 2001 [29]	152	64 (42%)	-

Table 2. - Time domain methods: main results

Authors	Predictive parameter and respective value	Sensitivity (%)	Specificity (%)	Positive predictive value (%)	Negative predictive value (%)
Steinberg *et al.* [12]	140 ms (P dur SAECG)	77	55	37	87
Chang *et al.* [18]	100 ms (P dur SAECG lead II)	-	-	37	83
Buxton *et al.* [14]	110 ms (P tot)	83	43	38	86
Buxton *et al.* [14]	110 ms (P tot)+IEI≥10ms	66	70	48	83
Klein *et al.* [20]	155 ms (P dur SAECG)	69	79	65	82
Klein *et al.* [20]	140 ms (P dur SAECG)	31	69	71	29
Stafford *et al.* [19]	141 ms (P dur SAECG)	73	48	34	83
Fukunami *et al.* [24]	120 ms (P dur SAECG)	95	48	70	-
Passman *et al.* [29]	110 ms (P dur lead V1)	-	-	-	-
Dilaveris *et al.* [17]	110 ms (P max)	88	75	84	-
Aytemir *et al.* [15]	106 ms (P max)	83	72	79	-
Andrikopoulos *et al.* [16]	110 ms (P max)	88	75	-	-
Dilaveris *et al.* [17]	40 ms (PWD)	83	85	89	-
Aytemir *et al.* [15]	36 ms (PWD)	77	82	85	-
Andrikopoulos *et al.* [16]	40 ms (PWD)	83	85	-	-
Dilaveris *et al.* [17]	110+40 ms (P max + PWD)	75	92	95	-
Aytemir *et al.* [15]	106+36 ms (P max + PWD)	70	92	92	-
Andrikopoulos *et al.* [16]	120 ms^2 (P var)	80	74	-	-
Passman *et al.* [29]	180 ms (PR)	-	-	-	-
Fukunami *et al.* [24]	3.5 μV (LP_{20})	95	54	73	-
Fukunami *et al.* [24]	120 ms + 3.5 μV	91	76	83	-

P dur: P wave duration; SAECG: signal averaged ECG; P tot: total P wave duration; IEI: isoelectric interval; P max: maximum P wave duration; PWD: P wave dispersion; P var: variance of the P wave; PR: duration of the PR interval.

detectable in each lead is thus usually measured. Buxton and Josephson [14] defined a total P wave duration too, as the interval between the first onset and the last offset of the P wave in the three standard leads they recorded (Fig. 1). The importance of such a measure stands in the circumstance that atrial depolarisation measured through endocardial electrodes precedes the first deflection detectable in any of the surface ECG leads [21-23]. A cut point must then be defined to compare maximum or total P wave durations between patients and controls. Buxton and Josephson followed the New York Heart Association criteria to define an intra-atrial conduction defect as a single standard lead P wave duration greater than 110 ms. Other works adopted different limits depending on the highest sensitivity and specificity that could be achieved.

Isoelectric interval

The isoelectric interval (IEI) is defined as the difference between total P wave duration and maximum P wave duration [14] (Fig. 1). A higher value is present in patients developing AF than in control subjects. The actual meaning of this indicator is not well established, yet Buxton and Josephson proposed some possible explanations. The most likely of all seems to be that a prolongation in the isoelectric interval is the result of the asynchronous atrial activation due to anatomic and electrophysiologic alterations predisposing to AF. However a prolongation of the IEI is not a good predictor for the arrhythmia, unless used in combination with P wave duration.

Signal-averaged P wave duration

In this case P wave duration is computed as the interval between the start and the end of the P wave obtained through an averaging of several beats (usually at least 100, according to the noise level achieved by the averaging technique) (Fig. 2). P wave endpoints are fixed as explained above. In order to align the different P waves collected a trigger is needed. Some works are based on a QRS complex trigger [12], other on a P wave trigger [24, 20]. In the first case a 300 ms window is chosen for the averaging. However the fiducial point is shifted at the extreme right side of the window so that P wave and PR segment are exposed. In the second case the trigger is chosen within a specially filtered P wave [24] as the first peak of the wave itself. The P wave triggered signal averaging shows a significantly shorter duration and a higher amplitude of the peak when compared to the standard

R wave triggered averaging. This is mainly due to the PQ interval variability and to the inclusion of ectopic beats in the R wave triggered averaging operation.

The leads recorded can be the orthogonal X, Y and Z leads, or any standard ones. In the first case the vector magnitude must be computed according to the formula $(X^2+Y^2+Z^2)^{1/2}$. To remove artifacts (as atrial ectopics) a template comparison is usually performed so that P waves not matching the template with the desired degree of correlation are automatically rejected. Specificity and sensitivity of this method in distinguishing patients at risk of AF are reported in Table 1 for different authors. Positive and negative predicted values are also shown.

Since changes in waves morphology can be observed during ECG recordings, Stafford *et al*. introduced an automatic algorithm to select the averaging template as the most frequently occurring P wave morphology for each patient. P waves showing a high correlation degree with this template are used to perform the averaging procedure, while the others are rejected, thus assuring a high fidelity of the resultant averaged waveform [25]. This method also improves the recovery of P wave energy after the averaging [26].

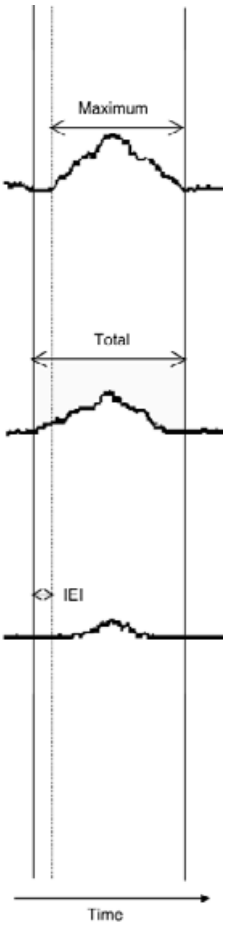

Fig. 1. - Definition of maximum and total P wave duration and isoelectric interval (see text for details).

P wave dispersion

Dilaveris *et al*. presented a novel predictor for AF after CABG that they named P wave dispersion (PWD) [17]. It is defined as the difference between the maximum and the minimum P wave duration detected in a 12-lead standard ECG. A lead-variable P wave duration is an indicator of the site-dependent inhomogeneous variability of the atrial conduction delay that is considered as one of the predisposing condition to AF [27]. This condition can be easily identified through observation of differently oriented ECG leads recordings. This means that P wave dispersion can be used as a marker of this alteration, thus indicating patients at risk for developing AF. Predictive ability of this test in different clinical models is shown in Table 1.

P wave variance

It is defined as the square of the standard deviation of P waves durations and calculated as:

$$P_{var} = \frac{n\Sigma x_i^{\,2} - (\Sigma x_i)^2}{n^2}$$

where n is the number of P waves and x_i represents the P wave duration at the instant i. Like P dispersion it is an indicator of the conduction variability but it is less dependent on P wave morphology and more reproducible [16].

P terminal force and spatial velocity

P terminal force is a standard electrocardiography index and it is usually used as an indicator of left atrial abnormality [28]. As atrial enlargement is often a concomitant AF disease, Stafford *et al*. tested this parameter effectiveness as an ECG marker of atrial alteration predisposing to AF [13]. It is defined as the duration (in seconds) of the terminal part (negative) of the P wave in lead V1 multiplied by its depth in millimetres (Fig. 3). If the P wave terminal part is positive, then the interval extending from the first notch to the wave end must be considered.

The spatial velocity is the rate of change of the P wave voltage with respect to time.

PR interval

Passman *et al*. showed that a prolonged PR interval measured from lead V1 is a risk factor for post-CABG AF [29]. More information about conduction delay can be obtained through a V1 lead recording because of its spatial orientation: its terminal portion reflects the posterior atrial potential that cannot be recorded through a limb lead.

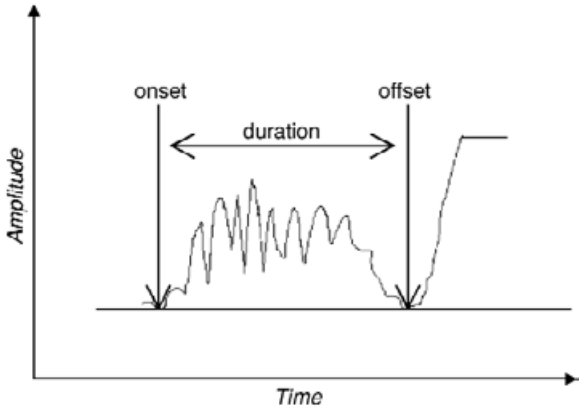

Fig. 2. - Example of a signal-averaged ECG recording. Onset, offset and duration of the resulting P wave are shown.

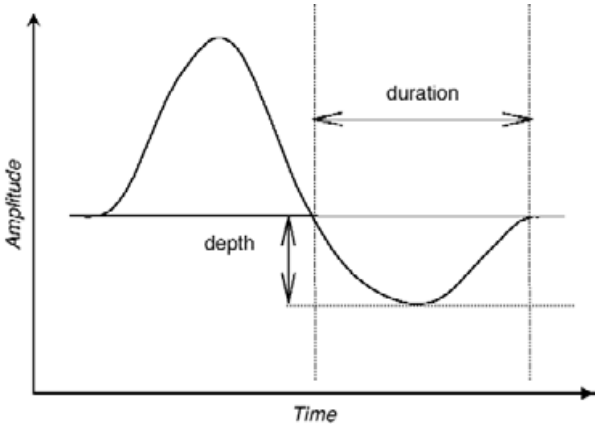

Fig. 3. - P terminal force is defined as the duration of the negative part of the P wave in lead V1 multiplied by its depth in millimetres.

Atrial late potentials

Some studies have tried to find out if low amplitude atrial late potentials can predict AF, as they can reflect the late depolarisation of tissue when a reentrant circuit exists. Engel *et al*. [30] made signal averaged X-, Y-, Z- leads recording (both high and low frequency). They defined as atrial low amplitude signal (and probably late potential) the difference between the two P wave durations recorded at two different filter settings. However, from this kind of analysis, they didn't find any significant information for the AF risk prediction. This is probably due to the circumstance that atrial signals amplitude are quite low in amplitude and the area of the atrial reentry could not have sufficient mass to be detected.

Fukunami *et al*. [24] analysed the terminal portion of signal averaged P wave. They found that in patients with AF the terminal portion is lower in amplitude and longer in duration than it is in control subjects. These findings were obtained by measuring the root mean square voltages (RMS) for the last 10, 20, 30 ms (LP_{10}, LP_{20}, LP_{30}) of the filtered P wave.

Frequency domain analysis

Bollman *et al*. have recently evaluated the frequency content of the fibrillatory baseline in patients with both paroxysmal (PAF) and persistent AF [33, 34]. They found that the fibrillatory frequency (obtained through a QRST complex removal and a subsequent FFT analysis on the remaining signal) increases within 5 minutes after the onset and decreases prior to termination. Since a clear relationship thus exists between the arrhythmia and the fibrillatory rate it is presumably possible to find further information about the risk for AF development in susceptible patients through a frequency analysis which has recently been proposed as an alternative to time domain analysis. Moreover, since the detection of ventricular late potential activity (due to the inhomogeneous ventricular activation in damaged myocardium) is successfully performed through a frequency analysis of the terminal QRS complex, several studies have recently analysed if fractionated atrial activation during PAF could produce low amplitude potential detectable as high frequency components in a spectrum analysis of the P wave [25]. Stafford *et al*. compared the results of the entire P wave spectrum analysis to those achieved through a conventional terminal P wave frequency domain processing. They investigated the performance of these two techniques at discriminating PAF patients from controls and their robustness to P wave endpoints and duration variations [25]. In a conventional analysis they analysed the energy content of the last 100 ms of the P wave. A DC correction is first performed and a windowing procedure is needed to minimise spectral leakage due to waveform truncation. In an entire P wave analysis the baseline wander in the P wave must be removed. Stafford *et al*. generated a ramp signal extending from the beginning to the end of the P wave that was then subtracted from the original signal. Windowing is not necessary in this case because of the natural limited extension of the original waveform. In both methods the absolute powers of the frequency bands extending from F = 20, 30, 40, 60, 80 Hz to 150 Hz are calculated and then algebraically summed to give the total power found between each starting frequency and 150 Hz (P20, P30…etc.). Ratios of high frequency (from F to 150 Hz) to low frequency

(extending from 10 Hz to F) power are usually calculated. As a percentage they are expressed as:

$$PR(F) = \frac{\sum_{f=F}^{150Hz} P}{\sum_{f=10}^{FHz} P}$$

Stafford *et al*. showed that an increase in the high frequency part of the P wave third quarter is usually an indicator of higher risk for AF [35].

Hiraki *et al*. [36] also calculated the area ratios of the whole P wave by dividing the power spectrum area in a fixed interval (0-10 Hz, 0-20 Hz, 0-30 Hz) by that in the remaining interval extending to 100 Hz (10-100 Hz, 20-100 Hz, 30-100 Hz) AR_{10}, AR_{20}, AR_{30}, respectively. They found that AR_{10}, AR_{20}, AR_{30} values in patients with Paroxysmal AF are in some leads significantly higher that in control. Moreover high frequency components in patients were decreased in the 20-50 Hz range as compared with the control.

Premature complexes detection

Kolb *et al*. analysed 297 spontaneous episodes of AF in 33 patients [37]. They found that atrial premature complexes (APC) initiated 93% of them. This cause-effect mechanism is even more evident when considering atrial premature complexes with an aberrant P wave morphology [38]. Vikman *et al*. also found that the number of ectopic beats seems to increase prior to the arrhythmia onset [39]. This phenomenon is known as being the trigger for the arrhythmia on a predisposing substrate. Several works have then tried to find a correlation between premature complexes rate and shape and AF onset. Most of them have been presented at the Computers in Cardiology 2001 challenge. In this case the database available consisted in a learning set and a training set. The first one was used to better understand the characteristics of ECG recordings prior to AF episodes, during paroxysmal AF episodes or distant from them. The second database was used to assess the reliability of the predictive algorithm developed.

The first aim is to detect ectopic beats from an ECG recording. Atrial and ventricular premature complexes can be easily identified through an analysis of the temporal series of the RR intervals tachogram). When a premature atrial complex occurs a sudden reduction in the tachogram is present. The following sinus beat, however, has a normal distance from the ectopic one. A comparison of the RR intervals with respect to the mean of the preceding RR can be implemented to automatically detect APC. Ventricular premature complexes (VPC), instead, are characterised by a prolonged RR interval immediately following the ectopic beat (compensatory pause) (Fig. 4). Langley *et al*. [37] algorithm identifies as possible ectopics those beats whose RR interval falls below the moving average of RR less 20%. Among these, the beats followed by beat introducing a ± 10% variation from the mean RR are classified as atrial; those followed by RR value exceeding the moving RR average of ± 30 % as ventricular. Zong *et al*. made also a shape comparison between possible ectopic beats and sinus ones [40]. The atrial ectopic beats must have a QRS complex shape similar to that of the dominant (sinus) beats. Schreier *et al*., for example, hypothesised that a P wave showing an inverse signal morphology when compared to the neighbouring P waves might be a potential trigger for the onset of paroxysmal AF [41]. Patients showing a higher number of atrial ectopic beats should thus be at higher risk for AF. Primary importance seems to stand in classical isolated APC, rather than aberrantly conducted or consecutive APCs sequences [37]. Once the classification is complete a symbolic analysis can be performed to detect any hidden pattern helpful for risk stratification. Yang *et al*. applied a "footprint" analysis to RR sequences to classify heart rate through a few states, no changes, acceleration, deceleration, in order to achieve a symbolic mapping of a clinical condition [42].

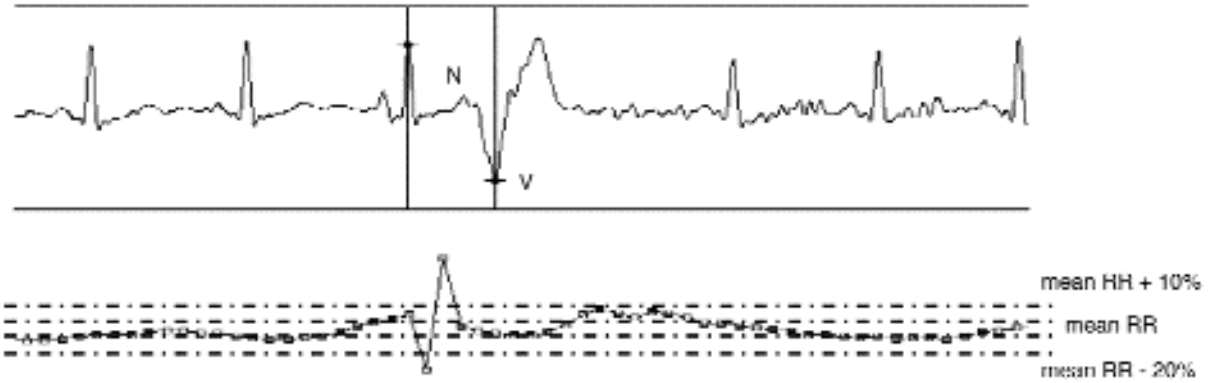

Fig. 4. - Example of an RR series where a ventricular ectopic beat can be easily identified.

Heart rate variability based analysis

Several prediction methods are based on the application of techniques that describe heart rate variability (HRV). Time domain features are mainly those quantifying HRV magnitude, such as standard deviation (SD) of RR intervals (ectopic beats included), of consecutive normal beats (SDNN), the number of successive pairs of normal intervals that differ more than 50 ms (pNN50) and the square root of the mean of the summed squares of differences between adjacent NN intervals (RMSSD). Maier *et al*. performed separate analysis on interval series containing ectopics and on series consisting only of normal beats [43]. Concerning the time domain features they found a reduced sinus HRV in PAF-patients and with a SDNN ≥ 27ms (standard deviation between successive beats of normal origin) they reached a sensitivity and specificity of 71% in separating groups of patients with and without paroxysmal AF. However, the best results are obtained using time domain features on the total RR intervals, as they are obviously influenced by the presence of ectopic beats, which are AF triggers. Vikman *et al* analysed HRV frequency domain features by computing an RR interval spectrum over 20-minute periods [39]. They found that both LF (0.04 to 0.15 Hz) and HF (0.15 to 0.40 Hz) components decrease before AF but the LF/HF ratio remains unchanged.

Vikman *et al*. calculated the approximated entropy (ApEn) of RR interval series, an index that indicates the presence of repetitive patterns, and the short term scaling exponent α_1 which has been previously found to be a predictor of arrhythmias [43, 44]. Higher values of ApEn are found when repetitive patterns exist, while α_1 measures the strength of the short time correlation properties of the RR interval data. They found that a reduced complexity of RR interval dynamics and altered fractal properties usually precede the onset of AF episodes as indicated by decreasing values for ApEn and α_1 [43, 45]. This behaviour was observed on RR series in both cases including or excluding ectopic beats. The reduced RR interval complexity may by itself serve as a trigger of the onset of AF as it is a marker of both altered regulation of sinus node behaviour and an increase of atrial firing ectopics, that both predispose to the spontaneous onset of AF.

Other methods have been developed for the Computers in Cardiology 2001 challenge. Most of them investigated several features at the same time. They then tried to find an optimal decision rule or a good classifier, according to data presented in the test set and data set. De Chazal *et al*. [46] analysed the power spectrum density (PSD) of RR series, some time domain measures from RR series, amplitude measures of P wave and frequency representation of the P wave area. In response to this set of input features the output of the classifier is a set of numbers representing the probability estimate of each class. Linear discriminants partition the feature space into the different classes using a set of hyperplanes. They found that the best set is the one represented by the PSD of the RR series feature group.

Discussion

The limitations of all methods based on P wave duration are that no accepted definition of duration exists and, consequently, no universal cut point has been proposed yet [15]. Although the highest accuracy could be achieved detecting P wave endpoints manually, intraobserver and interobserver measurement errors still affect these methods [16]. The use of different endpoint definition for P waves makes the comparison of the results achieved by various groups difficult. The automatic P wave detection methods usually assure a higher reliability and reproducibility, yet P wave delineation is more difficult to implement than QRS complex one, mainly owing to the low signal-to-noise ratio and the shape variability of the P wave.

In order to increase the accuracy and reproducibility of P wave duration measurements Andrikopoulos *et al*. [16] proposed a semiautomatic algorithm for evaluating this parameter from standard ECG formats. A schematic summary of this method is reported in Fig. 5. A comparison between automated and manual measurements showed that interobserver and intraobserver errors are significantly lower using the first method than by use of the standard manual method (for P dispersion estimation they achieved interobserver relative errors of 29.6 ± 16.4 % (manual) *vs* 14.4 ± 6.4 % (automated)).

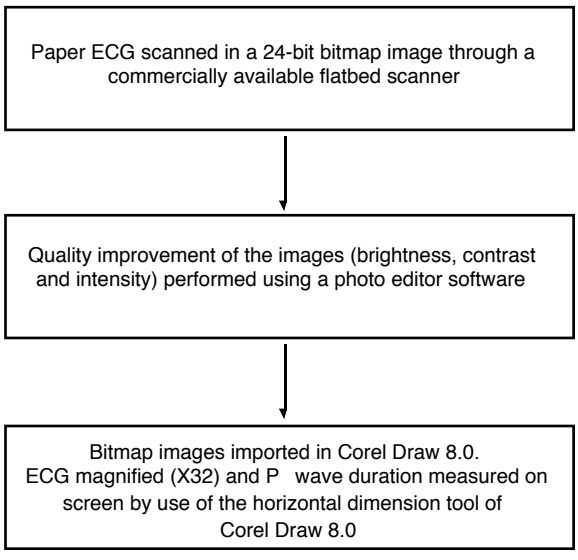

Fig. 4. - A semiautomatic algorithm to measure P wave duration from standard ECG formats (Andrikopoulos, 2000).

Clavier *et al*. presented a method for automatic analysis of the P wave based on the use of a hidden Markov model (HMM) and wavelets transformation [31]. ECG segmentation is performed using the coefficient of an ECG wavelet transform as observation of the HMM. They managed to isolate and delineate the P wave and thus extract parameters for discrimination of patients with AF. They evaluated the P wave duration, shape parameters (to detect symmetrically shaped P waves, P waves with slowly descending values, P waves with slowly ascending values, bimodal P waves and diphasic), spectral parameters and wavelet entropy parameters.

All the techniques mentioned above show high predictive values, however a discriminating test must also be reproducible when performed under similar conditions [19]. To assess reproducibility between paired signal averages of identical digital recordings, Stafford *et al*. used the coefficient of reproducibility, expressed as a percentage of the mean value of the variable being examined [19, 32]:

$$\%CR_{k1,k2} = \frac{2 \times SD(k_1 - k_2)}{Mean(k_1 - k_2)} \times 100$$

where k_1 and k_2 are the two measurements obtained by recordings performed after each other. A lack of reproducibility can induce differences in P wave duration estimation of about 10%, but affordable values for discrimination between patients and controls can be of about 11 ms. This could explain why the diagnostic accuracy of these parameters has not proved to be completely reliable. Anyway, they found that SAPWD computed as described above is a reproducible measurement, however not all the numerical measures derived from a signal averaging P wave analysis are always reproducible. Frequency analysis and spatial velocity are less repeatable because of the considerable changes in P wave morphology with time.

The results obtained using more complex signal processing tools, such as frequency domain analysis or non-linear techniques did not yield indexes as useful as those obtained by time-domain analysis. Since these methods do not require the accurate localisation of P-wave onset and offset, they may overcome the limitations suffered by the time domain techniques. However, the full potentialities of these new approaches are not fully exploited and deserve further investigations.

Conclusions

This study aimed to review the algorithms developed in the last years about the AF prediction. Considerable progresses have been made recently in AF prediction, however no reliable method has been proposed yet. This is mainly due to the lack of an automated P wave detection method. Thus the performances of each proposed non-invasive AF predictor cannot be well evaluated yet. Nevertheless the results obtained using these time domain indexes are of clinical interest. So far, frequency domain and heart rate variability methods did not yield useful predictors.

If patients at higher risk for AF could be identified then prophylactic antiarrhythmic treatment could be performed in those subjects showing predisposition to the arrhythmia onset, thus contributing to reduce patient's suffering and costs associated to the hospitalisation. The encouraging results obtained prompt for further scientific efforts in this field.

Received on 4 February 2003.
Accepted on 17 April 2003.

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