

Mechanisms of Asymmetric Poincaré Plots Obtained by Means of 24-Hour Holter Monitoring in Athletes

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Abstract

We investigated the electrocardiographic mechanisms of asymmetric two-dimensional Poincaré plots (PPs) obtained by 24-hour Holter monitoring (HoM).

Twenty-seven (11 females) young athletes (A) aged 25 ± 4 years and ten healthy age-matched controls (B) underwent HoM using a Spiderview™ recorder. Two-dimensional PPs were constructed and systematically explored regarding the underlying ECG waveforms

Asymmetry (ΔW) was due to: (1) wandering atrial pacemaker activity) (100 %), (2) sudden decreases in cycle lengths (SDC); (60 %), (3) sudden increases in cycle length (SIC) (70 %), and (4) frequent blocked premature atrial depolarizations (10%).

Asymmetry was strongly correlated to instantaneous HRV and, thus, may be a measure of efferent vagal activity.

1. Introduction

An elegant method to assess HRV is a scatter plot of successive R-R intervals versus immediately preceding RR intervals. Plots of such nature were first introduced by Henri Poincaré [1] as a representation of nonlinear system dynamics.

Healthy subjects normally exhibit a comet-like pattern on the two-dimensional Poincaré plot (PP) [2], and the comets' width has been shown to correlate very well with instantaneous heart rate variability (HRV) [3]. Thus, the comet width has been used as an index of efferent vagal cardiac activity [3,4].

In an ongoing study in endurance trained athletes, we observed pronounced asymmetry on the Poincaré plot (Fig.1). In this investigation, we sought to elucidate the mechanisms of PP asymmetry in athletes performing various types of sport.

2. Methods

2.1. Study population

After informed consent twenty-seven (11 females) young healthy athletes (A) aged 25 ± 4 years and ten healthy age-matched controls (C) were included in the study. All subjects included in the study were off any cardiovascular or neurotropic drugs. The athletes performed training sessions on 5 days per week and had a mean training history of 5±2 years. The types of sport performed were running (n=20), soccer (n= 6), and ice hockey (n=1). Control subjects were regularly exercising, but pursuing only leisure time sports.

2.2. Holter and Poincaré plot analysis

Each study participant underwent 24-hour Holter monitoring. ECGs were recorded at a sampling rate of 200 Hz using a Spiderview™ recorder (Ela Medical, Paris 2007). Recordings were analyzed on a Synescope™ system (Elamedical, Paris 2007) regarding the presence of arrhythmias, and the following 24-hour HRV indices were determined: RMSSD, SDNN, LF power, and HF power [5]. RMSSD and HF power were additionally quantified for the sleeping period (0:00h – 4:00h). The respective indices are referred to as RMSSD-N and HF-N.

From the 24-hour RR interval series, two-dimensional PPs with a time lag of one cycle ($\tau = 1$) were generated using the Synescope™. A unique feature of this system, which rendered it perfectly suitable for electrocardiographic PP exploration on a beat-to-beat basis, was that it allowed us to unambiguously reassign an underlying ECG waveform to each of the data points manifest on a given PP. Thus, exact PP exploration was possible with regard to accurate and precise determination of the electrocardiographic rhythm underlying the various parts of the comet patterns manifesting on the Poincaré plots investigated. We previously reported on this method of Poincaré plot exploration in more detail [2]. In brief, a systematic procedure was used to identify a grid of points located both on the external contour of the comet pattern, and of

points located internal to the contour. At each grid point the electrocardiographic waveforms were examined.

Using dedicated software [4], we assessed the symmetry properties of each Poincaré plot at time lags $\tau = 1$ cycle and $\tau = 1000$ cycles, respectively. For that purpose, the Poincaré plot data were represented in the coordinate system (x,y) which was rotated 45° counter clockwise (Fig.1) and given by:

$$x_k = (T_k + T_{k+1}) / \sqrt{2}$$

$$y_k = (T_{k+1} - T_k) / \sqrt{2}$$

where T_k and T_{k+1} are the k th and the $(k+1)$ th RR intervals.

2.3. Definition of Poincaré plot asymmetry

The following quantitative PP indices were used to describe the geometric characteristics of the comet patterns observed: W_R , W_L , W_{max} , and ΔW (Fig.1). The magnitude of the asymmetry (MA) of each comet was quantified as:

$$MA \equiv \Delta W = |W_L - W_R| \quad (1)$$

where W_L represents the maximal width of the data cloud (comet pattern) extending to the left of the Poincaré plot's bisector, and W_R represents the maximal width of the data points mapping to the right of the bisector. Maximal width (W_{max}) of the comet pattern was defined as:

$$W_{max} = |W_L + W_R| \quad (2)$$

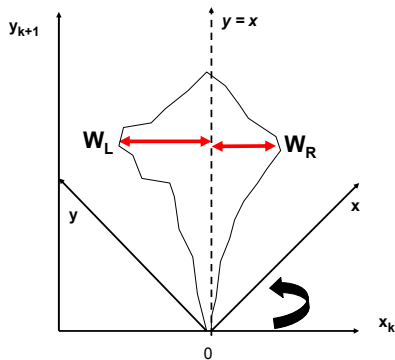


Figure 1: Schematic of an asymmetric comet pattern. W_L : maximal width to the left of bisector ($y=x$); W_R : maximal width to the right of bisector.

2.4. Statistical analysis

Data are given as arithmetic means and pertinent standard deviations. Correlations were performed using Pearson's method. The U – test was used to test for differences. A p value ≤ 0.05 was considered the threshold of statistical significance.

3. Results

The athletes exhibited a significantly larger PP asymmetry (i.e. ΔW) than the controls: 187 ± 121 ms (A) vs. 27 ± 15 ms (C), $p \leq .001$.

The following rhythm changes contributed to Poincaré plot asymmetry, if they induced a cycle length change of at least 30%:

- (1) Wandering atrial pacemaker activity (WAP), present in 100% of asymmetric PPs (APPs);
- (2) Sudden sustained increases in cycle lengths (SIC), present in 60 % of APPs;
- (3) Sudden sustained decreases in cycle lengths (SDC), observed in 70 % of APPs;
- (4) Blocked premature atrial depolarizations (APDs), seen in 10% of APPs.

Neither the athletes nor the controls exhibited ventricular arrhythmias. In each group, ΔW , W_L , and W_{max} showed a very strong positive correlation with RMSSD, RMSSD-N, and a fairly strong correlation with HF and HF-N, respectively. In contrast, ΔW was very weakly correlated to SDNN, and negatively to LF power. Interestingly, W_R exhibited only a weak correlation with 24-hour RMSSD, but was virtually uncorrelated to night time RMSSD and night time HF power (Table I and II).

Table I: Correlations between Poincaré plot measures & HRV time domain indices (n=37).

	RMSSD	RMSSD-N	SDNN
W_L	0.89	0.730	0.53
W_R	0.44	-.004	0.73
ΔW	0.75	0.820	0.21
W_{max}	0.86	0.560	0.69

Table II: Correlations between Poincaré plot measures & HRV frequency domain indices (n=37).

	HF (n.u.)	HF-N (n.u.)	LF (n.u.)
W_L	0.68	0.51	-0.78
W_R	0.30	0.07	-0.34
ΔW	0.59	0.52	-0.68
W_{max}	0.65	0.42	-0.74

In the athletes, the asymmetric comet pattern present on the unlagged PPs ($\tau=1$ cycle) disappeared on the PP lagged at $\tau=1000$ cycles, and more triangular patterns with irregular boundaries emerged instead. In contrast, the controls exhibited a pure discoid pattern at $\tau = 1000$ cycles without any bulging on the boundaries.



Figure 2: Sudden (arrow) sustained increase in cycle lengths (SIC) causes left-sided PP asymmetry

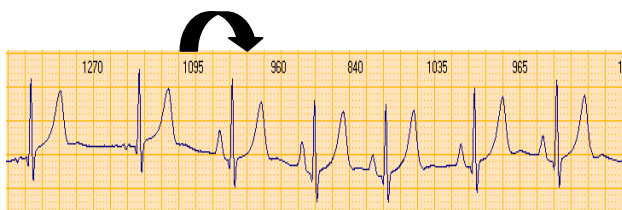


Figure 3: Sudden (arrow) sustained decrease in cycle lengths (SDC) causes right-sided PP asymmetry.

4. Discussion and conclusions

In this study, we were able to demonstrate that endurance trained athletes exhibited a conspicuously large asymmetry on both the unlagged ($\tau=1$) and lagged ($\tau=1000$) two-dimensional Poincaré plots. In contrast the controls showed either a significantly smaller or no PP asymmetry. Also, in the control group, no asymmetry was observed on the PPs with time lag $\tau = 1000$ cycles.

4.1. Mechanisms of asymmetry

Electrocardiographic exploration of the Poincaré plots showed that the asymmetries were brought about by

rhythm changes that were characterized by sudden changes of cycle lengths. Basically two mechanisms contributed to the genesis of a comet's asymmetry:

- (1) sudden sustained cycle length increases (SIC),
- (2) sudden sustained cycle length decreases (SCD)

The fact that in each of the asymmetric comet patterns W_L was significantly greater than W_R indicates that there were significantly more SIC than SCD episodes present on the ECG. In other words, what caused an asymmetry to arise were very frequently occurring episodes with a sudden cycle length prolongation. However, that an asymmetry could arise at all was not merely due to the presence of SIC episodes. More important was that the cycle length increase lasted not only for one single cycle, but was rather sustained affecting a series of ensuing RR intervals .

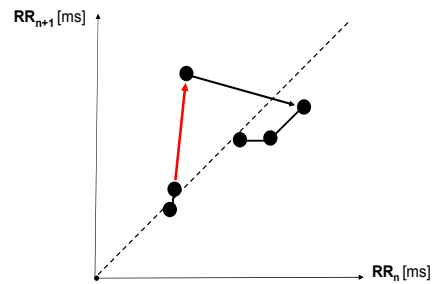


Figure 4: Mapping behavior of SIC episode (see Fig.2)

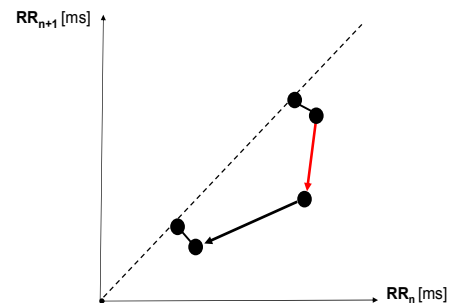


Figure 5: Mapping behavior of SDC episode (see Fig.3)

A significant change (i.e. $\geq 30\%$) in cycle length caused the pertinent data point on the PP to map away from the bisector, while the ensuing RR interval prolongations caused the data points to map back in regions close to the bisector, albeit at longer mean cycle lengths (Fig.4). However, if the sudden cycle length increase would have affected only one single RR interval with the ensuing RR intervals showing cycle lengths similar to the mean cycle lengths prevailing before the occurrence of the rhythm change, no asymmetry would have arisen, but instead a perfectly symmetric pattern would have manifested on the Poincaré plot. Overall, the SIC mechanism resulted in an increase of W_L , while it did not affect W_R .

The second mechanism resulting in PP asymmetry consisted in a sudden and sustained decrease of cycle lengths (SDC). However, in contrast to the SIC mechanism, episodes of decreasing cycle lengths caused the data points on the Poincaré plot to map asymmetrically to the right of the bisector (Fig.5), thus causing an increase in W_R without affecting W_L .

Since, in the individuals with an asymmetric comet pattern, we always observed predominant left sided asymmetry ($|W_L| > |W_R|$), it is concluded that, in these cases, the number of SIC episodes significantly exceeded that of SCD episodes. In other words, in the subjects exhibiting left-sided PP asymmetry, rhythm changes with a concomitant increase in cycle lengths were significantly more prevalent than rhythm alterations resulting in decreased cycle lengths.

While all the athletes showed predominant left-sided PP asymmetry, a few control subjects exhibited a symmetric comet pattern (SCP) with $|W_L| = |W_R|$. In this SCP subgroup, the number of SDC episodes equalled that of SIC events.

4.2. Physiological considerations

From the results it can be inferred that MA is related to increased and / or predominant efferent vagal activity (EVA) leading to frequent SIC episodes. This is in accordance with the observation that, in athletes, rhythm changes resulting in cycle length increases, such as sinus bradycardia or WAP, are caused by a net increase in EVA [5]. It is intriguing to speculate that W_R , a measure of right-sided PP asymmetry that did not correlate with instantaneous HRV, may represent episodes of decreased EVA and/or increased efferent sympathetic activity.

5. Conclusions

Our results demonstrate that in athletes and a subgroup of subjects performing leisure sports the asymmetry of

the comet pattern is due to sudden rhythm changes that result in sustained increases of RR-interval duration. The data also suggest that the comet's asymmetry (ΔW) may be a more specific indicator of EVA than W_{max} . Thus, ΔW may be a useful marker to monitor exercise induced autonomic fitness in athletes.

Acknowledgements

The authors appreciate the support of Sorin Group Deutschland GmbH, Munich, Germany and D Marangoni, Verona, Italy. The authors wish to thank Julia C. Graney, Chicago, IL, for her excellent technical assistance.

References

- [1] Poincaré H. La Science et l'Hypothèse, Flammarion, Paris, 1902.
- [2] Esperer HD, Esperer C, Cohen RJ. Cardiac arrhythmias imprint specific signatures on two-dimensional Lorenz plots. *Ann Noninvasive Electrocardiol.* 2008 Jan;13(1):44-60.
- [3] Brennan M, Palaniswami M, Keman P. Poincaré plot Interpretation using a physiological model of HRV based on a network of oscillators. *Am J Physiol Heart Circ Physiol.* 2002; 283:H1873-1886.
- [4] Esperer HD, Esperer C, Chernyak YB. Risk stratification in advanced heart failure using Lorenz plot indices of heart rate variability. *Comput Cardiol* 2004; 31:209-212.
- [5] Task Force of ESC & NASPE. *Circulation* 1996; 93: 1043-1065.
- [6] Hottenrott K, Hoos O, Esperer HD. Heart rate variability and physical exercise. Current status. *Herz* 2006;31:544-52.

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