Quantification of Athlete's Heartbeats Engaged in Ergometer Exercise: A Detrended Fluctuation Analysis Study Checking the Heart Condition

Toru Yazawa, Albert M. Hutapea, and Yukio Shimoda

Abstract—**Detrended fluctuation analysis (DFA), which was originally developed about three decades ago, was used to check the heartbeats of athletes in the Indonesian Olympic Training Center. We report results of time series analysis of heartbeat on subjects who underwent ergometer exercise. The objective of this research was to determine whether DFA could function as a useful method for the evaluation of the subject's quality of cardiovascular system. Since there are no two individuals that are identical physiologically, we present case studies but novel findings regarding how wellness of subjects can be evaluated by the electrocardiography. Even from the case studies, we can propose a general conclusion that DFA is a new, useful numerical method for quantifying the degree of wellness through the heartbeat recording.**

Index Terms—**exercise, detrended fluctuation analysis, heartbeat, scaling exponent**

I. Introduction

Cardiovascular disease is one of major social health problems. While the default setting is in general good health, there is always an onset of a process of alteration to an anatomical remodeling or a disease that never returns to the default health, in the life time including the period of intra-uterine development. Particularly, in the heart, this onset may result in "silent" angina, and a "silent" heart attack as the worst-case scenario. Early detection of the onset is a crucial solution to combat this pathogenesis, and making prediction of the "onset" is one of the main goals of science.

Cardiac remodeling is typically associated with disease but also occurs in the athlete's heart as an adaptive physiological response. The aim of our work was to detect the onset of a process of a cardiac remodeling, i.e., compensatory gradual changes in structure and function which will be leading the heart to pathogenic condition or adaptation [1]. Bio-medical computation could be very suitable method in investigating how to detect the onset of a cardiac remodeling. Therefore, our ultimate aim is to quantitatively analyze the heart condition, thus making numerical prediction.

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Traditionally, cardiac studies employ heart rate variability (HRV) to detect the onset of cardiac problems, including the disorders of the autonomic nervous system. Problems arise, however, when patients are usually assumed to be healthy before the appearance of symptoms associated with HRV. The detrended fluctuation analysis (DFA) [2] was proposed as a potentially useful method in determining a sign of cardiovascular disease (see ref. [3]), however, it has not yet developed to be a practical medical tool such as the electrocardiogram (EKG). (We prefer the word "EKG," instead of "ECG," with due respect to the inventor, Dutch physiologist, Nobel laureate, Willem Einthoven.)

We recently tested practical usefulness of DFA with using the heart of crustacean-animal models. In the test, we succeeded to show that DFA can distinguish the beating of intact hearts from isolated hearts [4]. In that study, we found that the scaling exponent of the isolated hearts shifted and approached to 0.5 without exception. In turn, the scaling exponent of the intact hearts showed a value about 1.0 without exception. As the results, we realized that DFA was reliable and useful, because DFA probably accurately reflects cardiac and systemic physiology. Unlike HRV, the excelling point for DFA is that it has a baseline value of one (1), like a standard body temperature (37 ºC), a standard blood pH (7.4), and so on. Therefore, DFA was simple as a tool, we hypothesized.

One (1) is nonlinearly determined as "healthy" outcome resulted from complex interactions between structure and function of molecules, cells and organs. Thereby we have a hope that DFA can "numerically" determine the state of health through the quality of the functioning of the heart. DFA seemed to be reflecting the state of not only the heart itself but also its physiological interaction with the nervous system. We considered that DFA might be a tool to detect the onset of cardiac remodeling, including functional changes of the autonomic nervous system.

In this article, we show case studies of heartbeat analysis of subjects who performed exercise on ergometer stationary bicycle to provide empirical evidence for the practical usefulness of DFA and a new EKG amplification device that facilitates automatic DFA computation in practical use. We will show that DFA is a potentially helpful tool for the early identification of physiological disorders, as it reveals information that is not provided by an EKG.

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II. Materials and Method

A. Peak detection of the heartbeat

Interval analysis requires detection of the precise timing of the heartbeat. A consecutive and perfect detection without missing any beat is necessary. According to our preliminary tests, about 2,000 consecutive heartbeats were required for obtaining a reliable computation of scaling exponent. We hypothesized that longer recording of the heartbeats would results in a better diagnosis. However, we found that long recording was not justifiably useful and a recording of about 2,000 consecutive heartbeats is preferable.

To detect the timing of the heartbeats, one may assume that common EKG recording is sufficiently useful. However, the problem with conventional EKG was the drifting of the baseline of the recording. Due to the drift and the contamination of unexpected electric power-line noise, recording failures may happen.

Another obstacle arose from the premature ventricular contraction (PVC). Among the "normal" subjects (age over 40 years old), about 60 % of subjects have PVC arrhythmic heartbeats. Normally, this PVC is believed to be benign arrhythmia, and in fact during our recording, we found many healthy-looking individuals exhibited this arrhythmia. However, PVC is an obstacle to a perfect detection of the timing of the heartbeat, because the height of its signal could sometimes vary much. If the baseline of EKG recording could be extremely stable, the heartbeats would automatically be detectable even when irregular beats appeared sporadically. Unfortunately, in commercial EKG recording devices, baseline of the record is not stable.

B. EKG recording with stable baseline

To capture heart beat peaks without missing any detection, we made an EKG amplifier that stabilizes baseline of the recording. Important issue was: we discovered that time-constant for input-stage of recording must be adjusted to an appropriate level.

Having stable baseline recording was an advantage to our DFA research. However, in some cases, inevitable noises would contaminate the recording. In such case, we removed the noises by identifying them visually on PC screen thus making a perfect (without miscounting) heartbeat interval time series. We have already identified how this inconvenience came about. About one-half of these cases were due to the sweat on the skin under the electrodes. We were able to overcome this problem by cleaning the skin with an appropriate solution.

C. DFA: Background

DFA is based on the concepts of "scaling" and "self-similarity" [5]. It can identify "critical" phenomena because systems near critical points exhibit self-similar fluctuations [2] [5] [6], which means that recorded signals and their magnified/contracted copies are statistically similar. Self-similarity is defined as follows: In general, statistical quantities, such as "average" and "variance," of fluctuating signals can be calculated by taking the average of the signal through a certain section; however, the average is not necessarily a simple average. In this study, we took a squared average of the data. The statistical quantity calculation depended on the section size. The signal was self-similar when the statistical quantity was λ^{α} times for a section size magnified by λ. Here, α is the "scaling exponent" and characterizes self-similarity.

Stanley and colleagues considered that scaling property can be detected in biological systems because most of these systems are strongly nonlinear and resemble the systems in nature that exhibit critical phenomena. They applied DFA to DNA arrangement and EKG data in the late 1980s and early 1990s and discovered the usefulness of the scaling property [2][6], and emphasized the potential utility of DFA in the life sciences [6]. Although DFA technology has not progressed to a great extent, nonlinear technology is now widely accepted, and rapid advances are being made in this technology.

D. DFA: Technique

We made our own computation program based on the previous publication [2], which is described in one of the references [7].

E. EKG recording

For EKG recordings, we used a Power Lab System (AD Instruments, Australia). For EKG electrodes, a set of ready-made three AgAgCl electrodes (+, -, and ground; Nihonkoden Co. Ltd. disposable Model Vitrode V) were used. Wires from EKG electrodes were connected to our newly made amplifier. These EKG signals were then connected to a Power Lab System.

F. Volunteers and ethics

EKG recordings were performed at Indonesian National Olympic training center in Bandung. Subjects were selected at random at the Indonesian Olympic Training Center. All subjects were treated as per the ethical control regulations of our universities, Tokyo Metropolitan University, Tokyo Women's Medical University and Universitas Advent Indonesia.

III. Results and Discussion

A. Case study 1

Subject one is a 49-year old healthy looking Indonesian. His resting state heartbeats were recorded for 28 min while the subject was sitting on ergometer, relaxing and answering to several questions made by researchers. Two pre-matured ventricular contractions (so called PVC) can be seen (Fig 1). These PVCs (see also PVC in Fig 3) are a benign type of heartbeat, as classified by guidelines used by medical doctors. It is described that PVCs are observable among 60 % of healthy persons over the age of 40. However, we should acknowledge that a hypothesis associates the occurrence of PVC with sudden death [8].

At the beginning of EKG recording, his heart rate was about 90 beat per min (BPM) (Fig 1, interval 0.6 s, see also Fig 4).

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In three min, the heart rate was approaching over 100 BPM after the start of the recording (Fig 4). He seemed to be nervous because the measurement equipment as well as the foreigner researcher doing the recording were new to him. During the period for 28 min of resting stage measurement, he got relaxed by conversations with us, and his heart rate gradually decreased (Fig 1, interval about 0.7 s, see also Fig 4).

At the start of the exercise stage his heart rate immediately increased to over 100 BPM, and kept slow increment until reaching a plateau before the end of the exercise (Figs 3 and 4).

After 20 min of the exercise he mentioned to us that he was not tired yet. He also mentioned at the end of exercise session that he felt he could continue with this low load (50 watt) aerobic exercise for another 30 min. One PVC appeared at near the end of exercise (Figs 3 and 4) as was seen at rest condition (Fig 1).

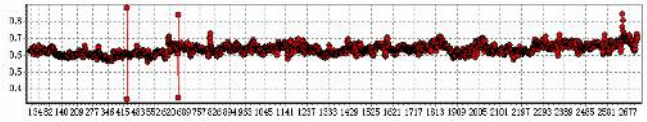

Fig 1. Heartbeat recording from *subject one* at rest. Y axis, Heartbeat intervals (s). X axis, beat number. Figure shows 2719 beats in total during 28 min, which were 100 % accurately detected, as shown in next Fig 2.

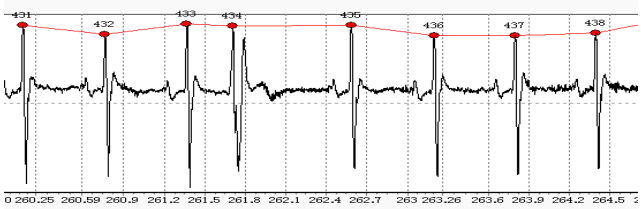

Fig 2. Example recording of heartbeats and accurate peak detections. The heartbeat number 434 is PVC. A time period 260~264 sec from start of recording is shown. The figure shows that our amplifier produced a steady baseline EKG even though subject was freely moving. (Here, subject was answering to question).

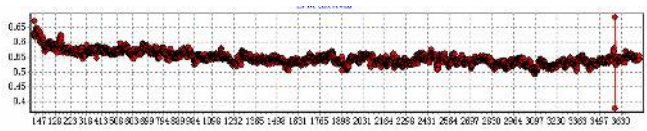

Fig 3. The same subject shown in Fig. 1 and 2. Y axis, Heartbeat interval (s). X axis, beat number. Continuous recording from Fig. 1. He started ergometer exercise, at the heartbeat number 1 (one) with a 50watt load strength and a 96 rpm speed, lasting for 32 min. 1 to 3746 beats shown.

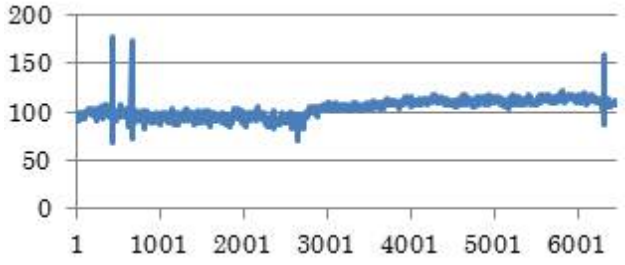

Fig 4. Entire time series of *subject one*. Connected data of Fig 1 and Fig 3. Y axis, heart rate (BPM). X axis, beat number.

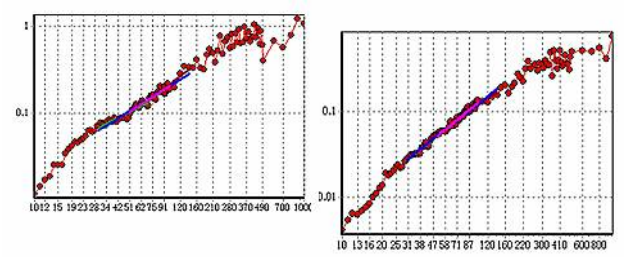

Fig 5. DFA computation. Left graph, at rest. Right graph, at exercise. For the same subject shown in Figures 1, 2, 3 and 4. Y axis, variance. X axis, box size, from 10 to 1000. Log scale in both axis (see original article [2] for the basics of DFA). The slope of the graph gives the scaling exponent, which is calculated at various "windows," i.e., "box size," as shown in Table 1.

Table 1. Comparison between at rest and during exercise.

	Rest			Exercise	
Box Size		DFA (α)	Box Size		DFA (α)
51 ~ 100		1.01	51 ~ 100		1.32
30 ~ 140		0.99	30 ~ 140		1.25

Table 2. Our preliminary guideline (see [7])

DFA (α)	
0.5~0.89	Stressfulness, PVC, Alternans, Naturally dying
0.9~1.19	Healthy, 1/f fluctuation
1.2~1.5	This heart is at resk of catastrophic circulation stoppage.

After obtaining heartbeat-interval time series, we proceeded to next step, calculating the scaling exponents (α) by our computing method the DFA (Table 1). One can see that exercise increased α, which can be seen in overall slops (see Fig 5). It means that the scaling exponents were pushed up by exercise.

At rest, this 49 years old man exhibited healthy scaling exponents nearly 1.0 (see the windows, both 51-100 and 30-140 in Table 1 and corresponding graphs of Fig 5). From our previous studies we determined a guideline in interpreting the scaling exponents to define whether an individual is healthy or unhealthy (Table 2). We were surprised to discover that an apparently healthy person (athlete's heart) did get risky high value of the scaling exponent "during exercise" (Table 1). Interestingly, we also discovered that the same exercise induced an increase in the scaling exponents of the other subject, a swimmer girl (see below).

From the results from *subject one*, we started to consider that "athlete's heart," which is a remodeling heart, may not be so healthy than we first believed. In the class room, general physiology teaches that athlete's hearts undergoes physiological cardiac hypertrophy instead of pathological hypertrophy, e.g., caused by hypertension. However, a literature pointed out difficulties associated with distinguishing the athlete's heart from hypertrophic cardiomyopathy (HCM). HCM is the leading cause of sudden cardiac death in young athletes [1].

The EKGs of the other three subjects shown in this study were recorded at rest and while engaging in exercise in the

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same room at the same environment, temperature of 25 ºC, together with the *subject one* aforementioned (Figs 1 to 5).

B. Case study 2

Subject two, age 28, an Indonesian female swimmer. Her average-heart-rate at resting state is rather high with unknown reason (Figs 6 and 7). According to the doctor's guideline: the heart rate above 100 bpm is referred to as tachycardia. However, we did not investigate the reason for this seemingly abnormal condition.

After starting ergometer exercise, heart rate quickly increased (see *A in Fig 7). Heart rate soon attained a steady state, about 160 BPM. Apparently a high rate was maintained over the period of exercise. This ability indicates that she has athlete's heart, i.e., her heart seems to have adapted to the physiological demand of a long distance swimming.

Her heart rate quickly decreased at the end of exercise (see *B in Fig 7). A significant characteristic, "maintained" rate during exercise, were seen in both *subject one* (Fig 1) and *two* (Fig 6).Therefore, we can conclude that both subjects *one* and *two*, Case studies 1 and 2, show characteristics of "athlete's heart."

From Fig 7, one can clearly see an exponential rise (*A) and decay (*B). This exponential behavior is due to the changes of the cardio-inhibitory nerve activity, i.e., parasympathetic nerve activity, was switched-off and switched-on (respectively, at *A and *B in Fig 7). We have already described a mathematical model for this exponential function of neurotransmitter release regarding to cardio-inhibitory nerve control [9].

Figures 6 and 7 indicate that fluctuation of the heartbeat interval, i.e., variability in rate, is greater at rest than during exercise. This must be due to a change of vagal tone governing the heart: We can interpret that during exercise the heart received a decreased discharge frequency in the inhibitory autonomic nerve fibers innervating the heart. In other words, acceleration-dominant-state was induced by exercise. From neurophysiological consideration, this acceleration-dominant-state can be explained by reduction of inhibitory influence that caused the acceleration (*A in Fig 7), and thereafter getting dis-inhibition (*B in Fig 7). This consideration was experimentally proven: We have already demonstrated real EKG data and mathematical model in crustacean heart [9].

The scaling exponents of the *subject two* are very low at rest (see Table 3) as can be seen in the left graph where slope is less steep (Fig 8) and the scaling exponents ranges around 0.6 – 0.8 at rest (Table 3). We already know that if one has perfect health condition, the slope must exhibit 45 degree, i.e., the scaling exponent is one (1) at relaxed condition. This means that her general health condition and especially heart condition is not perfect, although DFA cannot tell the physiological reason(s) that contributes to this (see our guideline Table 2).

When she was engaged in exercise, the DFA-slope became steeper in almost entire ranges of window size (Fig 8) and thus computed scaling exponents were significantly increased. The values are astonishingly high, ranging from 1.3 to 1.5 (see Table 3). This significant increase of the scaling exponents during exercise was also found in other athlete's heart, *subject one* (see Case study 1). According to our guideline (Table 2), we may interpret that this high values during exercise may indicate that their hearts are at a risky state. If this consideration would be proven in the future investigations, we must conclude that athlete's heart that is remodeled heart is not normal. It is intriguing that we have already found that a high scaling exponent is associated with a subject who has ischemic heart, such as those received stent placement and/or bypass implantation [10].

There are a lot of elements in the body such as molecules, cells, tissues and organs. Nonlinear interactions between the elements of athlete's body must be contributing to this scaling behavior of the heartbeat.

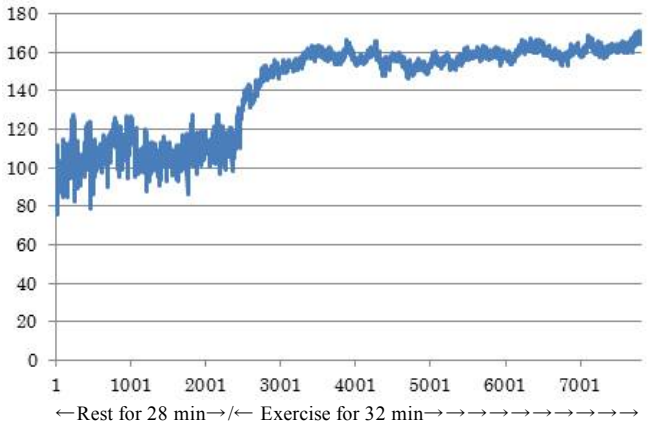

Fig 6. Time series, 2471 beats for 28 min resting state, and 5341 beats for 32 min exercise state. Y axis, heart rate. X axis, beat number. Ergometer exercise session started at the heartbeat number 2472 with a 50 watt load strength and a 96 rpm speed, lasting for 32 min, identical strength in the case study 1.

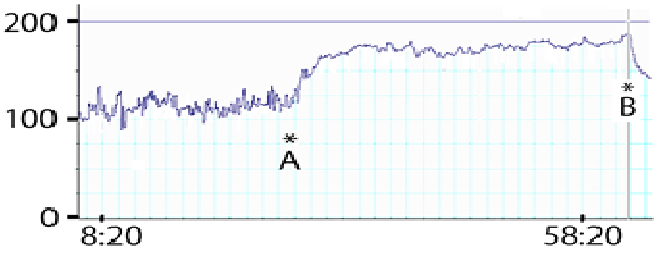

Fig 7. Exponential rise and decay in heart rate. The same record as in Figure 5 but X axis is shown in time (min) instead of beat number (see Fig 6). *A, exercise started. *B, exercise stopped. Y axis, heart rate.

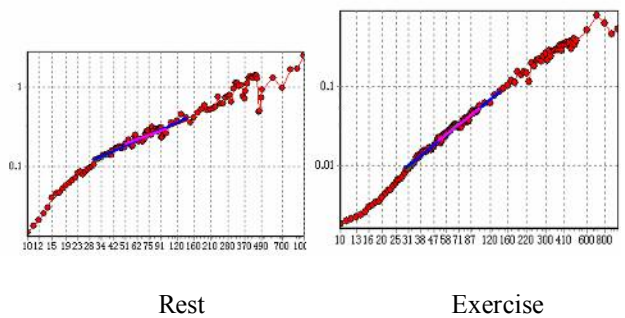

Fig 8. DFA computation. Left graph shows results at rest and right graph at exercise. The same subject shown in Figs 6 and 7. Y axis, variance. X axis, the Box Size from 10 to 1000. Log scale in both axis.

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Table 3. Comparison between at rest and during exercise.(a female swimmer)

Rest			Exercise		
Box Size		DFA (α)	Box Size		DFA (α)
51 ~ 100		0.62	51 ~ 100		1.36
30 ~ 140		0.8	30 ~ 140		1.42

C. *Case study 3*

Subject three, age 29, an Indonesian male basketball player (Fig 9 and Table 4). It should be noted that here the load was 75 watt instead of 50 watt in the case of other three subjects, which are Case studies 1, 2, and 4.

It took long time before reaching a plateau phase due to higher load (Fig 9). Figure 9 shows that plateau started at about 5500 in heartbeat number and lasted until the end of exercise. He (*subject three*) mentioned to us that, at the end of exercise, he was tired and he wished very much to stop the exercise session.

Here, one can see again that fluctuation during exercise is smaller than that at rest. As is in the *subject two* (Table 3), again the scaling exponents during exercise were very high (Table 4). His scaling exponent at rest is low (Table 4) which characteristics are similar to the recording from the s*ubject two* (Table 3). We conclude that his (*subject three*) heart system at rest is not perfect in terms of DFA analysis.

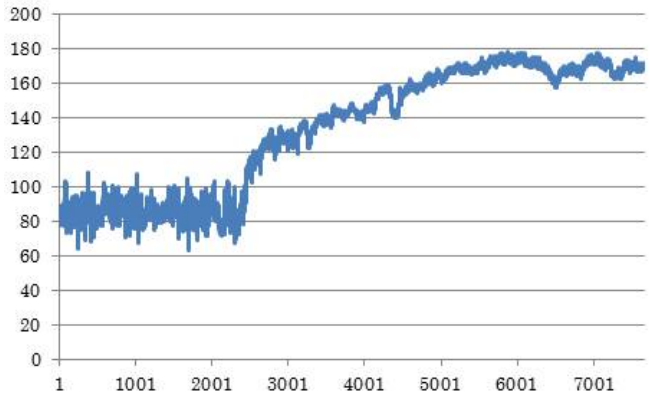

Fig 9. Time series of *subject three*, 2438 beats for 28 min resting state, and 5224 beats for 32 min exercise state. Y axis, heart rate. X axis, beat number. Ergometer exercise was started at the heartbeat number 2439 with a 75 watt load and a 96 rpm speed, lasting for 32 min.

Table 4. Comparison between at rest and during exercise.

Rest			Exercise		
Box Size		DFA (α)	Box Size		DFA (α)
51 ~ 100		0.76	51 ~ 100		1.62
30 ~ 140		0.81	30 ~ 140		1.52

D. *Case study 4*

Subject four, age 27, male, an Indonesian futsal player (Spanish Futbol de Salon). In this subject, the scaling exponents were increased by exercise as those shown by the rest of the three cases in this study. However, his heart stayed in a healthy range of the scaling exponent during exercise (see Table 5). He mentioned that, at the end of exercise, he was not tired at all with 50 watt and 96 rpm. We may conclude that he is the most appropriate sports-aspiring person among four subjects in this case study, because he has no risky value, even in terms of DFA. During exercise, his autonomic nervous system was still capable to send inhibitory command to the heart, which is observable as sporadically occurring "slowing down" in heart rate (see enlargement of time series, Fig 10). This "slowing down" in heart rate is derived from inhibitory discharge in the autonomic nerves, i.e., the parasympathetic nerve or the vagus nerve. Figure 10 shows that his vagus nerve still regulates the heart properly. He might be able to have much high load exercise though we have not yet tested. His exercise period was not "up to the chin" condition. That is why his scaling exponent shows nearly one (1), which means his heartbeat can behave dynamically, that is, responding dynamically to internal demands. The ability to meet demands is a good condition of the heart that can respond properly and dynamically to the internal and external environment. However, his heart condition at rest shows that he might have a stress in terms of DFA analysis. This fact is similar to that of both *subjects two* and *three* in this study.

Athlete who has a healthy scaling exponent at rest in the present report, is only *subject one* (Case Study 1). The subject who has a healthy scaling exponent at exercise, is only *subject four* (Case Study 4).

IV. Concluding Remarks

The techniques and experimental results in the present study are new as far as we know. In the present observations with DFA computation we tried to find any apparent correlations between the scaling exponent and the state of heart during exercise. While data of the heartbeat time series were obtained from subjects who were healthy looking individuals, three of four subjects (Case Studies 1, 2, and 3) exhibited surprising results: exercise brings them to a risky state in terms of the scaling exponents. We would like to suggest that their state of heartbeats during exercise is the state that the heart is ready to stop any time, as demonstrated before in animal model experiments and human ischemic cardiac conditions [10].

Athlete's heart is believed to be a benign adaptation [1]. Four subjects in this study have obviously different genomic structure from each other. However, outcome of control system commanding the heart performance was all identical: exercise increases the scaling exponents, that is, to a normal level in one subject and to an alarming level in the rest of three subjects in this study. This is probably normal function of healthy subjects who have complex internal nonlinear physiological interactive pathways. However, it is important to know that there has been some debate over whether the athlete's heart is a truly physiological phenomenon or whether long-term, chronic exercise training is maladaptive and leads to heart disease or sudden cardiac death [11]. This investigation may cause a stir in the debate.

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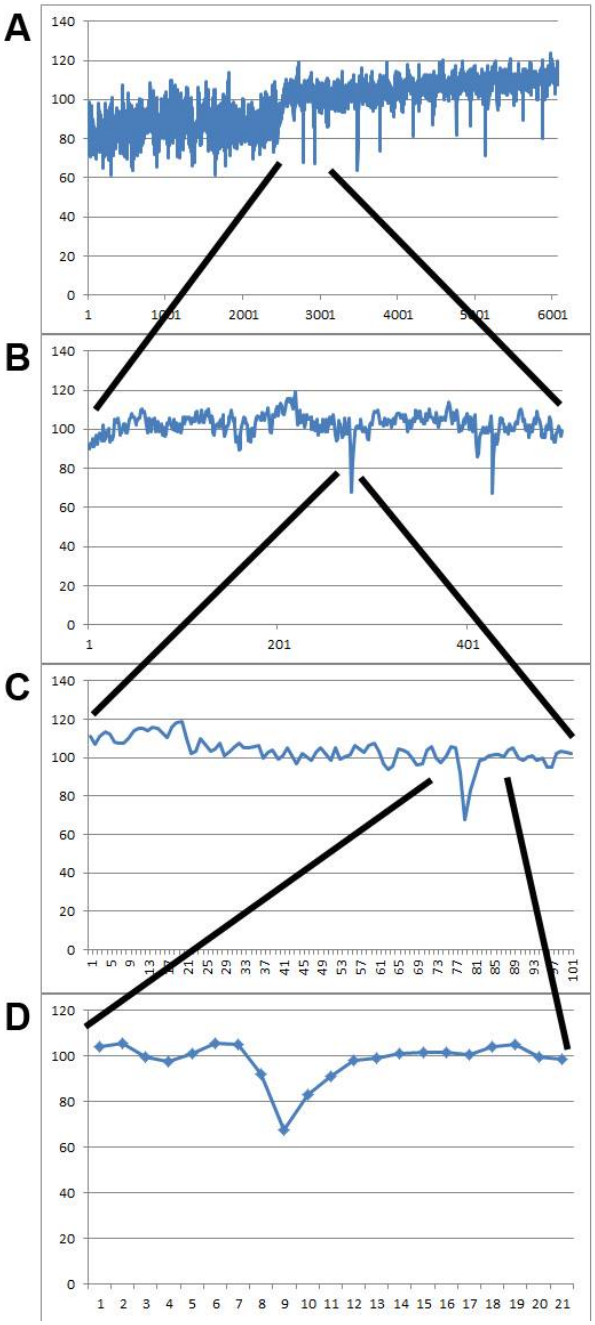

Fig 10. Heartbeat-interval time series of *subject four*. A, 6072 beats in total. B, C, D, partially enlarged to show detail of the time series. B, Beat number 2500-3000. C, 2700-2800. D, 2770-2790

Table 5. Comparison between at rest and during exercise.

	Rest			Exercise	
Box Size		DFA (α)	Box Size		DFA (α)
51 ~ 100		0.85	51 ~ 100		1.15
30 ~ 140		0.89	30 ~ 140		1.12

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