Original Research Article

Ramadan fasting produces changes in the electrocardiogram of healthy young adults

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Abstract

Introduction During the month of Ramadan, Muslims refrain from drinking and eating between sunrise and sunset. **Aim:** This study is aimed to understand the effect of this long intermittent fasting schedule during Ramadan on the electrical activity of the heart in healthy young adults. **Objective:** To prove that fasting has a positive effect on the electrical activity of the heart seen in electrocardiogram (ECG) and thus it decreases the cardiovascular risk. **Method:** Fifty healthy adults were included in the study. They were all tested before and on the 28th day of the Ramadan fast. The following parameters of ECG were thoroughly studied. RR interval, P wave duration , PR interval, QRS complex, QT interval, QTC interval, P axis, QSR axis, Taxis, P(II)volt, S(V1)volt, R(V5)volt. **Results: A** significant increase in the RR interval was observed. Other changes in the ECG found at the end of fasting were positive however not statistically significant. The changes seen were:, an increase in P wave and PR interval, decrease in QRS complex, QT interval, QTC interval, P axis became less positive, QRS axis and T axis has become more positive .P voltage and R voltage too has decreased and S voltage became less negative. Dominance of the sympathetic nervous system (SNS) over the parasympathetic nervous system (PNS) has been shown to be a strong risk factor for cardiovascular disease. **Conclusion:** Fasting is associated with significant improvement in autonomic cardiac modulation through enhancement of parasympathetic modulation and probably reflected in the form of changes seen in ECG of subjects in this study. The effect of fasting on electrical activity of the heart should be further explored to recognize fasting as a potential tool for improving cardiovascular health physiologically.

Keywords: Ramadan fasting, ECG, RR interval, QT interval, QTc interval, T axis.

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Received Date: 02/07/2015 Revised Date: 09/08/2015 Accepted Date: 14/09/2015

Access this article online	
Quick Response Code:	Website: www.medpulse.in
	DOI: 23 September 2015

INTRODUCTION

Ramadan: Fasting during Ramadan is a religious duty for all healthy adult Muslims. Ramadan is the ninth month of the Islamic lunar calendar. Many of the world's great religion recommend a period of fasting or abstinence. The Islamic fast during the month of Ramadan during which a whole month is dedicated to fasting is particular to Islam, and is strictly observed by millions of Muslims worldwide. During Ramadan, the majority of Muslims have two good sized meals; one immediately after sunset and the other just before dawn. They are allowed to eat and drink between sunset and dawn but not after dawn. The month of Ramadan is either 29 or 30 days. From the physiological standpoint, Islamic fasting provides a unique model of fasting.[1] Fasting is defined as a partial or total abstention from all foods or a select abstention from prohibited foods by Trepanowski *et al*[2]. The three most commonly studied fasts are caloric restriction (CR), alternate-day fasting (ADF), and dietary restriction (DR). CR is the reduction of kilocalorie intake by a certain percentage (typically 20 - 40%) of normal consumption. ADF consists of alternating 24-hour periods: during the "feast period," fasters may consume food normally; during the "fast period," food consumption is restricted or halted altogether. Water is allowed during all times. DR is a reduction of one or more components of dietary intake (typically

How to site this article: B Shruthi, B Satyanarayana. Ramadan fasting produces changes in the electrocardiogram of healthy young adults. *MedPulse – International Medical Journal* September 2015; 2(9): 583-588. http://www.medpulse.in (accessed 26 September 2015).

macronutrients) with minimal to no reduction in total kcal intake.

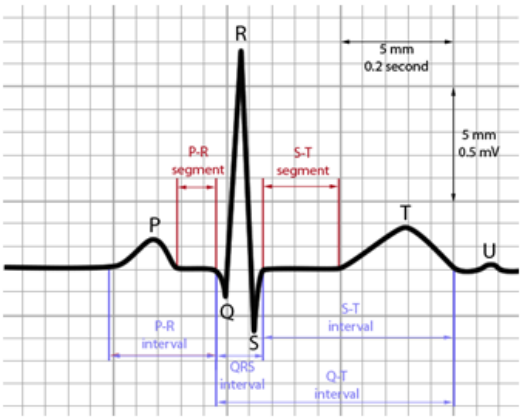


Figure 1

Many studies so far have concentrated on finding the beneficial effect of fasting on different cardiovascular parameters[3] Shruthi *et al*. The objective of the present investigation was to prove fasting as a tool having positive effect on the electrical activity of the heart seen as changes in ECG.

Electrocardiogram: Electrical activity is a basic characteristic of the heart and is the stimulus for cardiac contraction.

The genesis of the electrocardiogram

The sinus node is situated in the right atrium close to the entrance of the superior vena cava. The atrioventricular node lies in the right atrial wall immediately above the tricuspid valve. The fibres of the AV bundle (of His) arise from the atrioventricular node and run along the posterior border of the septum between the ventricles. On reaching the muscular part of the septum, they split into right and left bundle branches and then spread out in the subendocardium of the ventricles as the Purkinje system. The right bundle is a slender, compact structure. The left bundle soon splits into two or more divisions or fascicles, one of which proceeds anteriorly, sharing the same blood supply as the right bundle, and another is directed posteriorly. In the usual sequence of events, the electrical impulse arises in the sinus node and spreads across the atria to reach the atrioventricular node. It can then only reach the ventricles by passing into the rapidly conducting atrioventricular bundle and its branches. The first part of the ventricles to be activated is the septum, followed by the endocardium. Finally, the impulse spreads outwards to the epicardium. The spread of the cardiac impulse gives rise to the main deflections of the electrocardiogram: P, QRS and T waves, as shown in figure 1

- The P wave represents atrial depolarization.
- The PR interval represents the time taken for the cardiac impulse to spread over the atrium and through the AV node and His–Purkinje system.
- The QRS complex represents ventricular depolarization.
- The T wave represents ventricular repolarization.

Waveforms and Intervals[4]

P Wave: Best viewed in Lead II or V1, Upright in Lead II, biphasic in V1, Max height should be less than 2.5 mm and duration should be less than 0.12 sec.

PR interval: Includes the P wave and the PR segment from the beginning of P wave to the beginning of the next QRS.PR interval should be between 0.12 and 0.2seconds.Normally it is < 0.2 seconds. If it is >0 .2 seconds, it suggests first degree block.

QRS Complex: Q wave is the first deflection below isoelectric line it should be less than one box (0.04 sec wide) and less than 1/4 the height of R wave R Wave is any deflection above the isoelectric line, S wave is any deflection below the isoelectric line that is not a Q wave. Entire QRS complex should be less than 0.1 secs, QRS interval (beginning of Q to the end of the S wave) should be < 120msecs.

ST segment: Begins at the junction or J point (end of QRS complex), to the start of T wave. Gives information regarding Myocardial Injury

T wave: It represents ventricular repolarization. The period Beginning of QRS to apex of T wave is under absolute refractory period. Last half of T wave is under relative refractory period. It follows the direction of the QRS complex.

QT interval: Indicates how fast the ventricles are repolarized. How fast they are ready for the next cardiac cycle. The QT interval (QTi) is rate related. QTi gets shorter as the heart rate increases. QTi can be prolonged in the presence of ischemia or electrolyte imbalances. Prolongation of the QTi can lead to Torsades de Pointes or Ventricular fibrillation. It is determined in the electrocardiogram from the beginning of the QRS to the point at which the T wave (or the U wave, if present) returns to the isoelectric line. Thus, it includes the duration of ventricular depolarization and repolarization, and corresponds to the action potential duration.

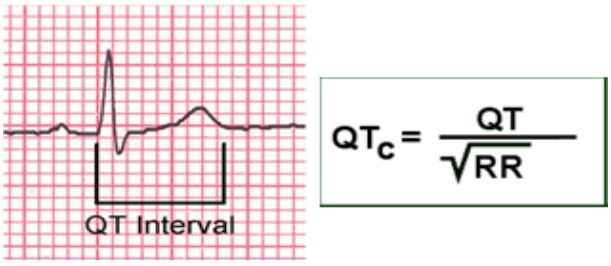


Figure 2

Corrected QT interval (QTc)

It can be calculated as, QTc men < 450 msec, QTc women < 470 msec, QT interval (beginning of QRS to end of T wave) should be less than half of the preceding RR interval - this varies with the rate. For normal rates, QT < .4 seconds (2 large boxes). "QT prolongation" (too long) can lead to a refractory form of ventricular tachycardia called torsades de pointes according to Whitsel.

MATERIAL AND METHODS

This study was done in the research lab of Department of Physiology Bhaskar Medical College during the month of Ramadan in the year 2011(Aug and Sept).This study has been approved by the institutional Ethics Committee. The study population was among the students and interns of Bhaskar Medical College and General Hospital who were practicing the fast during Ramadan.50 young adults in the age group 18-24yrs were included in the study after taking consent. Only normal adults with no medical history were included in the study.6 could not come back towards the end of the fast due to personal reasons. Data was collected from 44 men and women Each subject was studied twice for each of the parameters mentioned below firstly before the fasting and next on the 28th day of the fasting[5]. Morning hours were chosen for all the recordings. After entering the lab the subjects were made comfortable and then the ECG was recorded. A 12 lead Schiller's ECG machine as shown in Fig-2 was used to record the electrocardiogram. The parameters recorded were heart rate, RR interval, P wave duration PR interval, QRS complex, QT interval, QTC interval, P axis, QSR axis, Taxis, P(II)volt, S(V1)volt, R(V5)volt as shown in Fig-3

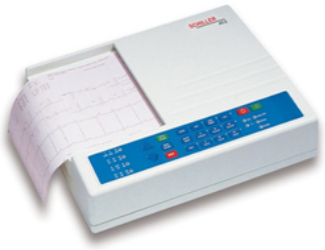


Figure 3

Figure 4

The data was processed through SPSS software. Paired sample statistics of t test was used to calculate the mean and Standard error of mean and the significance stated. All the participants were healthy with no history of any medical illness. They all had been fasting every year following the regimen of Ramadan fasting.

OBSERVATION AND RESULTS

At the end of the fasting RR interval increased significantly from 756.5 to 802.68(p<0.001) as shown in the Figure -1 Rest all other findings are even though not significant statistically but show a positive trend towards a good effect of fasting shown on heart. The duration of P wave and PR interval has increased as shown in Figure-2 QRS complex, QT interval, QTC interval has decreased. P axis became less positive, QRS axis and T axis has become more positive. P voltage and R voltage too has decreased. S voltage became less negative

Table 1: Shows the ECG findings before and towards the end of the fasting

ECG parameters	Baseline	28th day	*P* value
RR	756.50± 22.107	802.68± 20.321	<0.001
P	98.64 ±2.617	99.09± 2.400	0.870
PR	130.86 ±2.920	133.00 ±3.352	0.451
QRS	90.41 ± 5.201	84.27 ±1.448	0.237
QT	361.64 ±3.453	360.32 ±3.112	0.715
QTC	421.07 ±5.933	413.68 ±7.425	0.359
Paxis	49.73 ±3.518	46.45± 2.365	0.303
QRS axis	56.00 ±3.455	56.43 ±3.233	0.883
T axis	34.48 ±2.075	36.86 ±2.086	0.262
P(II)volt	0.1236 ±.00557	0.1164 ±.00468	0.079
S(V1)volt	-3.0477± 1.81393	-1.3800 ±.09362	0.367
R(V5)volt	0.8849 ±.09006	0.841 ±.0938	0.639

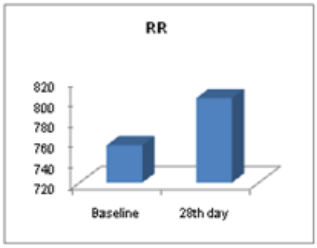


Figure 5

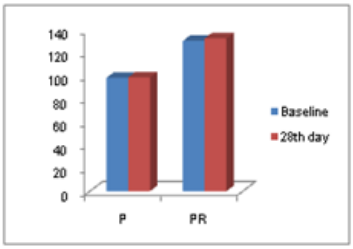
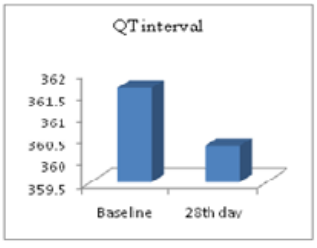


Figure 6

Figure 7



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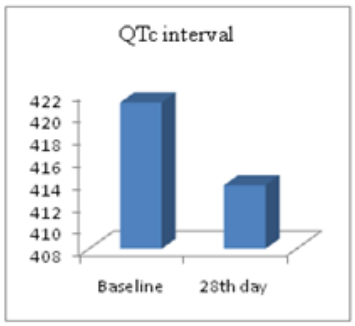

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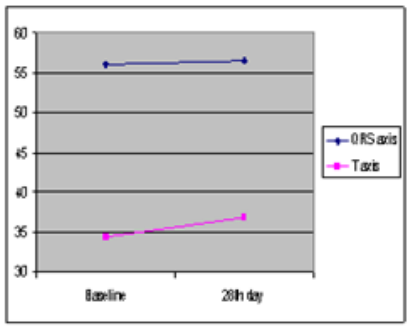

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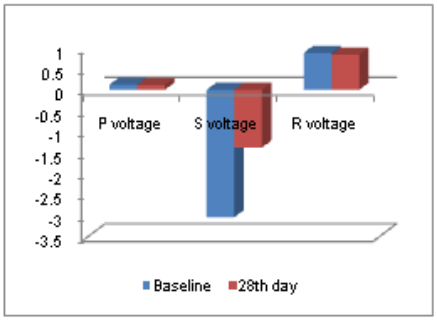

Figure 11

Legend

Figure 5: Significant increase in RR interval in msec; **Figure 6:** Increase in P wave and PR interval
Figure 7: Shows decrease in QRS interval in msec; **Figure 8:** decrease in QT interval in msec
Figure 9: Decrease in QTc interval in msec; **Figure 10:** QRS axis and T axis (in degrees); **Figure 11:** P, S, R voltages in mV

DISCUSSION

The cardiac autonomic nervous system (ANS) consists of 2 branches—the sympathetic and the parasympathetic systems—that work in a delicately tuned, yet opposing fashion in the heart. This extrinsic control mechanism can dominate intrinsic regulatory mechanisms that modulate heart rate and cardiac output. These branches differ in their neurotransmitters (norepinephrine and acetylcholine) and exert stimulatory or inhibitory effects on target tissue via adrenergic and muscarinic receptors.[6] The SA node has an intrinsic rate of firing, but ordinarily this is modified by the autonomic nervous system. The decision-making for autonomic functions occur in the medulla in the brain stem and the hypothalamus. Instructions from these centers are communicated via SNS and PSN which both innervate the heart. The SNS increases the rate of SA node firing (hence raising heart rate) and also innervates the myocardium itself, increasing the propagation speed of the depolarization wave, mainly through the AV node, and increasing the strength of mechanical contractions.[7] The PNS major effect is on heart rate and the velocity of propagation of the action potential through the AV node. Most organs are innervated by both the sympathetic and the parasympathetic branches of the ANS and the balance between these competing effects determines function. The sympathetic and parasympathetic systems are rarely totally off or on; instead, the body adjusts their levels of activation, known as tone, as is appropriate to its needs. Therefore, for normal subjects at rest, the effects of the heart rate's "brake" are greater than the effects of the "accelerator," although it is the balance of both systems that dictates the heart rate. The body's normal reaction when vagal tone is increased (the brake) is to simultaneously reduce sympathetic tone (the accelerator). Similarly, when sympathetic tone is increased, parasympathetic tone is usually withdrawn.[8] Shouldice R. *et al* explained that the PP or RR intervals of the surface lead ECG are an indicator of autonomic activity at the SA node – the dominant pacemaker under normal conditions. The secondary site in the heart under direct neural control is the AV node, through which all normally conducted beats pass. In addition to being directly affected by autonomic neural activity, the parasympathetic innervation for the SA and AV nodes originates in the medulla and is conveyed to the heart by way of the vagus nerve. Under normal conditions, the parasympathetic effect is the dominant neural influence on the heart.[9] Heneghan *et al* explained that ANS regulates 2 processes; firstly the overall cycle length and hence heart rate assessed by RR interval, secondly the speed of conduction of the electrical activity through the heart including the AV node. Changes in the cycle length or AV conduction delay usually involve a reciprocal action of the two divisions of ANS. Shortened cycle length/AV conduction delay are produced by a diminution of parasympathetic and simultaneous increase in sympathetic activity. Increased cycle lengths or AV conduction delay are usually achieved by opposite mechanism. Both the above two studies suggest SAN is predominantly affected by right vagus whereas AVN is predominantly affected by left vagus. Ishmail *et al*[10] found the effects of fasting on heart rate can be attributed to an increase in vagal tone. Poirier *et al*[11] studied that weight loss is associated with significant improvement in autonomic cardiac modulation through enhancement of parasympathetic modulation, which clinically translates into a decrease in heart rate. Our study confirms this finding with the significant increase in the RR interval showing that there is an increase in the cycle length after fasting due to vagal dominance as shown in Figure-1. Dominance of the SNS over the PNS has been shown to be a strong risk factor for cardiovascular disease. Obesity and aging are associated with increased SNS activity, and

weight loss and/or exercise seem to have positive effects on this balance. Weight loss improved SNS/PNS balance especially when CR is combined with exercise as explained by[12] Lilian *et al*. The changes which are observed in our study in the form of decreased QRS complex and decreased amplitude of P, S, and R waves may reflect the effect of decreased SNS effect after fasting as seen in Figures 3 and 7. The increased cycle length and AV conduction delay that is a characteristic of PNS also explains the lengthening of P wave and PR interval in our study seen after fasting as shown in the Figure-2 The PNS control over the ventricles is less than the SNS and fasting decreases the SNS dominance that probably explains the decrease in QRS complex, QT interval, QTc interval in our study as seen in Figures-4 and 5. Similar findings of decreased amplitude of QRS complex and T wave and right axis deviation was seen by studies of Azizi in prolonged fasting[13]. Theorell T found significantly decreased ratios between QRS and T wave amplitudes in leads I and II with decrease in body weight after fasting[14] Mshui *et al*[15] study results show that obesity per se causes both prolongation of QTc interval and an increase in QTc dispersion, and that weight reduction improves the prolonged QTc interval observed in obese patients. Pietrobelli[16] found decrease in QTand QTC interval in obese after fasting. The QTc interval normalized in three subjects with prolonged fasting that suggest improved cardiac repolarization with weight loss, as also is seen in our study.[17] Whitsel studied that the QT interval includes the duration of ventricular depolarization and repolarization, and corresponds to the action potential duration Autonomic tone influences RR interval variation (RRV) and the heart rate-corrected QT interval index (QTI). Together, QTI and RRV may improve characterization of sympathovagal control and estimation of risk of primary cardiac arrest. Combination of a high QTI and low RRV is associated with an increased risk of primary cardiac arrest among persons without clinically recognized heart disease QTc prolongation suggest the presence of autonomic dysfunction, i.e. increased sympathetic and decreased parasympathetic activity.[18] Javier found changes in T wave morphology and QT interval prolongation to be associated with an increased risk of sudden death and of cardiovascular death, probably because they could be markers of ventricular hypertrophy, left ventricular dysfunction or myocardial ischemia. Acute myocardial ischemia has been shown to modify the duration of the QT interval, increase repolarization heterogeneity (expressed as an increase in QT dispersion) and prolong the duration of the maximum electrocardiographic QT interval. Several mechanisms have been proposed to be involved in the prolongation of the QT interval secondary to acute myocardial ischemia: changes in the myocardial response to catecholamines or to cholinergic stimulation, perturbation of calcium or potassium ion channels, or induction of changes in the intracellular hydrogen concentration. Several studies have shown T axis as a predictive index for cardiac events.[19] Sabin *et al* demonstrated that an abnormal T-wave axis (i.e., falling between either -15° and-180°, or +105° and +180°) was a statistically significant predictor of Sudden Cardiac Death(SCD) Deodato Assanelli, *et al*[20] found the presence of a significant shift in the T-wave axis as the most predictive ECG index, both for fatal and non fatal events. The value of T-wave axis in the frontal plane was categorized in three groups as follows: Normal: $15° \leq TDev \leq 75°$ - Borderline: $75° < TDev \leq 105°$ or $-15° \leq TDev < 15°$ - Abnormal: $-180° \leq TDev < -15°$ or $105° < TDev \leq 180$ T-wave axis deviation reflects abnormal ventricular repolarization which is indicative of subclinical myocardial pathology. This parameter has been associated with an increased risk of coronary heart disease (CHD) and total mortality. In our study we have observed that T axis is in the normal limits but a shift towards the right as shown in Figure-6 probably shows a decreased risk for CHD and SCD after fasting.[21] Cole *et al* confirmed that the increased vagal activity has been associated with a reduction in the risk of death.[22] Amatruda *et al* assures us that vigorous supplementation of a hypocaloric diet prevents cardiac arrhythmias and mineral depletion.[23] As per Meyer *et al* Caloric restriction has cardiac-specific effects that ameliorate aging-associated changes in Diastolic functions. Castello even proved that fasting protects the rats against systemic inflammation and myocardial fibrosis[24]

CONCLUSIONS

Fasting does produce a change in the electrical activity of the heart as a result of the increase in vagal tone. There is further scope for similar studies in a larger group of fasting individuals. Apart from having beneficial effects on different systems Ramadan fasting brings about promising changes in the electrical activity of the heart which may decrease the risk towards CHD and SCD. Very few studies have chosen to prove the effects of fasting on electrical activity of the heart. So this study is special in its own way, as upcoming studies can choose to investigate ECG in fasting, further.

ACKNOWLEDGEMENT

The authors are grateful to the participants of this study for their full cooperation. They are also thankful to the biostatistician Sri. Balakrishna for his assistance in the statistics. Thanks also to the lab assistant Mr. Arun and the attenders Ms. Devi and Mr. Rajender for their help.

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Source of Support: None Declared
Conflict of Interest: None Declared