Physiol. Res. 72 (Suppl. 2): S127-S135, 2023 *https://doi.org/10.33549/physiolres.934952*

REVIEW

Sex Related Differences in Electrocardiography

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Received July 25, 2022
Accepted November 8, 2022

Summary

Since its implementation into the clinical medicine by Willem Einthoven electrocardiography had become one of crucial diagnostic method in cardiology. In spite of this fact effects of gender differences on parameters of electrocardiographic recordings started to be studied only recently. Sex related differences in physiological ECG are only minimal in childhood but there are developing during adolescence reflecting rapidly evolving differences particularly in hormonal secretion and activity of an autonomic nervous system. The heart rate is approximately 7 % higher in women than in men, PQ and QRS intervals are longer in men while QT interval is longer in women. The ST segment in females is flatter but generally the sex-related differences in ST-T waveform patterns are relatively very small with higher level of ST segment and taller T wave in men. The effects of sex-related differences, including sex hormones, on cardiac cell injury and death and their influence in determining rhythmogenesis and action potential configuration and conduction play an important role in clinics. Women have a higher prevalence of sick sinus syndrome, inappropriate sinus tachycardia, atrioventricular nodal reentry tachycardia, idiopathic right ventricular tachycardia, and arrhythmic events in the long-QT syndrome. In contrast, men have a higher prevalence of atrioventricular block, carotid sinus syndrome, atrial fibrillation, supraventricular tachycardia due to accessory pathways, Wolff-Parkinson-White syndrome, reentrant ventricular tachycardia, ventricular fibrillation and sudden death, and the Brugada syndrome.

Key words

Electrocardiography • Sex-related difference • Arrhythmias

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Introduction

Cardiovascular diseases remain the primary cause of death worldwide. Gender disparity plays an important role in cardiovascular disease prevalence and burden with significant gender related differences reported in cardiovascular clinical presentation mortality, morbidity and risk factors profiles [1]. The higher incidence of cardiovascular disease in men than in women of similar age, and the menopause-associated increase in cardiovascular disease in women, has led to speculations both about physiological background of these differences and about more specific gender-related approach to diagnostic decision making. Since its implementation into the clinical medicine by Willem Einthoven electrocardiography had become one of crucial diagnostic method in cardiology. In spite of this fact effects of gender differences on parameters of electrocardiographic recordings started to be studied only recently. It is also necessary to mention that sex related differences in electrocardiography (ECG) are closely dependent on age [2], being usually identical in both sexes during childhood (5-12 years) and starting to be different only during adolescence (12-19 years) or even in young adulthood (20-29 years). The vast majority of the data dealing with these phenomena comes from epidemiological studies focused mostly on gender-related predictive value of ECG [3,4]. But we need to reveal basic patterns of ECG differences between women and men in order to suggest the best forecast, diagnosis, treatment and post-therapeutic prognosis what corresponds to the up to date approach of personalized medicine. Therefore, the aim of this review is to describe so far known main characteristics of the male and female ECG recordings.

Physiological 12-leads ECG

Sex related differences in ECG are only minimal in childhood but there are developing during adolescence reflecting rapidly evolving differences particularly in hormonal secretion and activity of an autonomic nervous system. Two principle mechanisms could explain these differences between the sexes: hormonal effects on the expression distribution and function of ion channels or, conversely, sex related differences in autonomic tone. It is also well possible that a combination of these two mechanisms may be involved.

Heart rate

The heart rate is approximately 7 % higher in women than in men starting from the adolescence and persisting in young adulthood but then gradually diminishing in magnitude with age (Fig. 1). It reflects particularly shorter QT interval in men than in women (see chapter 1.2.3 and Fig. 4). Both testosterone [5] and estrogen [6] were proved to decrease the heart rate, but testosterone seems to be more efficient in this action. Also the heart rate variability is in adult premenopausal women higher than in men, but whereas the difference between men and women in the case of the heart rate is influenced predominantly by sex hormones, the heart rate variability is increased in women by combination of endocrine effects and higher tone of parasympaticus [7]. Higher vagal tone in women in comparison to men can be explained by effect of oxytocin that is moreover potentiated by estrogen: Higher oxytocin levels are namely associated with increased vagal activity [8], likely through oxytocin-type neurons from the paraventricular nucleus communicating with cardiovagal neurons in the nucleus tractus solitarius, the dorsal motor nucleus of the vagus, and the nucleus ambiguous [9]. Another possible mechanism that can contribute to the higher intrinsic heart rate in women is sex difference in exercise capacity that was proved to be a significant predictor of sinus cycle length [10].

ECG intervals

The sex-related difference and age-related differences in ECG intervals belonged among parameters studied on a large cohort of almost 80 000 subjects in order to define new reference ranges for ECG intervals [11] appropriate to modern technologies and contemporary general demographic status.

PQ interval

The extensive analysis of Mason and coworkers [11] has registered a progressive increase in PQ interval with aging as well as higher values in men compared to women starting form young adulthood (Fig. 2). Consistently, the same tendency was proved also for the P wave duration.

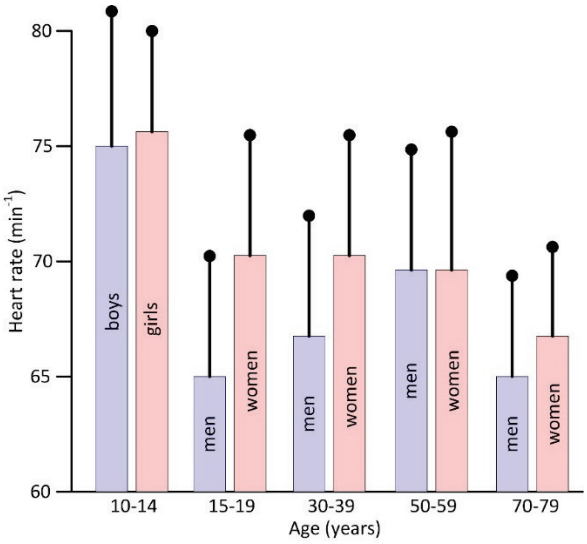

Fig. 1. Sex-related differences in heart rate (mean ± SD) according to age (derived from data of [11])

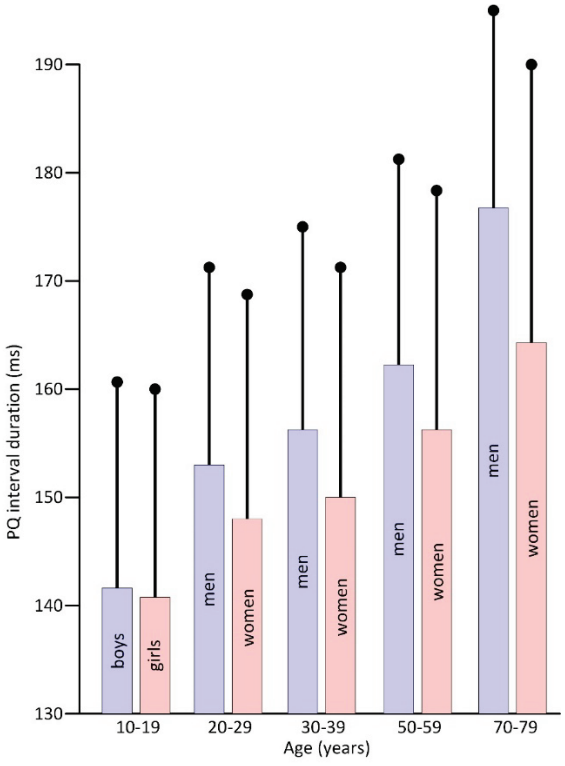

Fig. 2. Sex-related differences in PQ interval (mean ± SD) according to age (derived from data of [10])

QRS complex

The sex-related difference in QRS duration starts to be significant during adolescence, when the QRS duration was found to be 6 ms shorter in women resulting from lower left ventricular mass [12]. This difference persists during the whole adulthood (Fig. 3). The difference between men and women is resulting from different left ventricular mass. The standard reference values of normal left ventricular myocardial thickness (LVMT) assessed by cardiac magnetic resonance were defined by Kawel *et al.* [13]. According to their findings the average of the maximum/minimum LVMT at the mid-cavity is 7/5 mm in women and 9/6 mm in men, respectively. For any segment observed on long axis images, the maximum/minimum LVMT is on average 9/4 mm in women and 11/4 mm in men.

QT interval

The difference in QT duration between men and women is developing during adolescence when the QT interval is shortened in boys while it remains unchanged in girls and the difference thus created then remains through adulthood with gradual growth of values with age in both sexes (Fig. 4). At the beginning of adulthood the sex-related difference in QTc intervals represents 18 ms. The QTc duration is then increasing almost linearly through adulthood in both genders (Fig. 5). This increase is more expressed in men what causes a gradual decrease in the difference between genders. Nevertheless the difference remains significant up to old age [14].

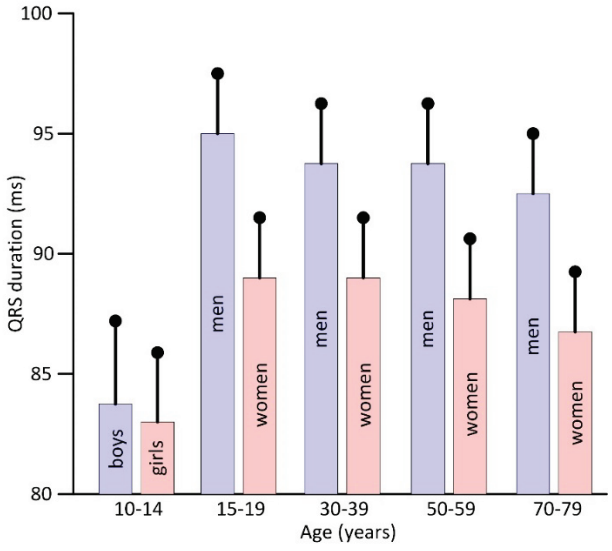

Fig. 3. Sex-related differences in QRS duration (mean ± SD) according to age (derived from data of [11])

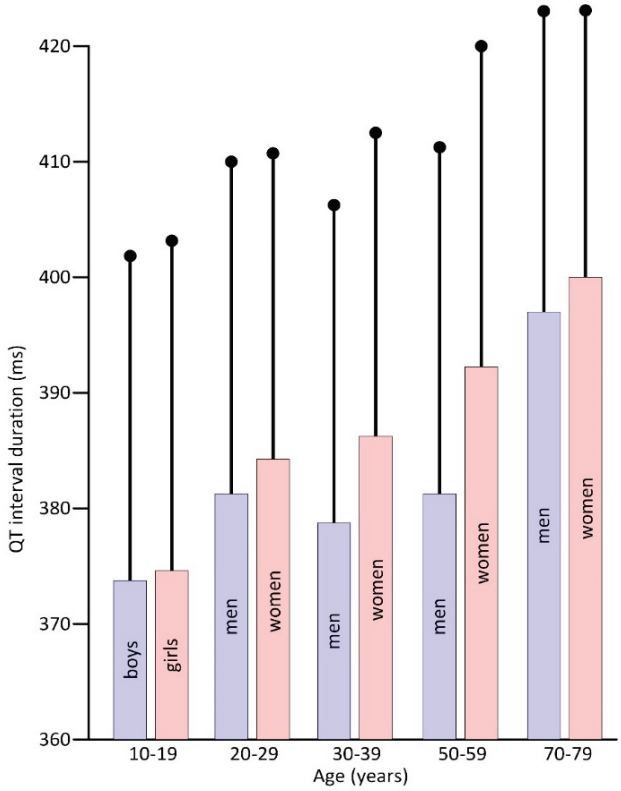

Fig. 4. Sex-related differences in QT interval (mean ± SD) according to age (derived from data of [34])

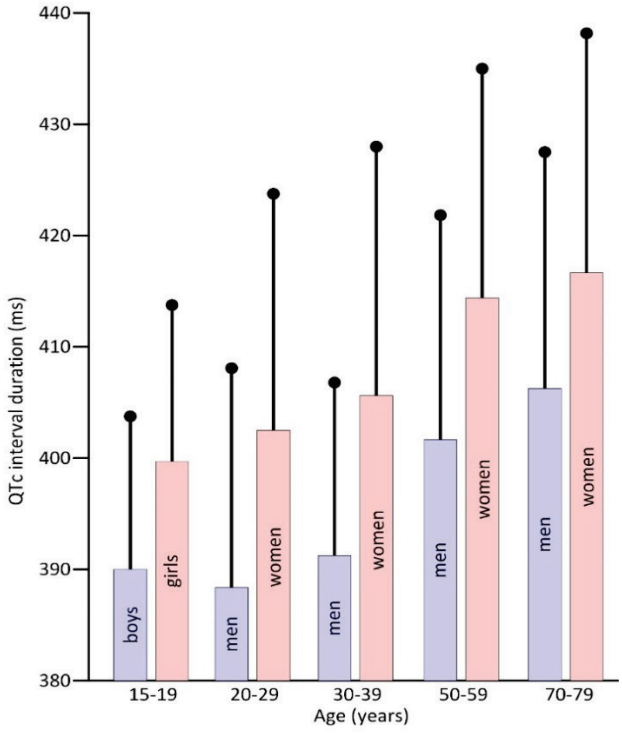

Fig. 5. Sex-related differences in QTc interval (mean ± SD) according to age (derived from data of [11])

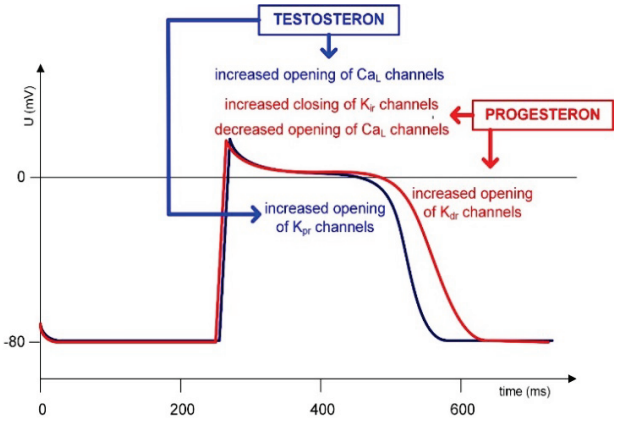

Fig. 6. Effect of sex hormones on the repolarization patterns in men and women

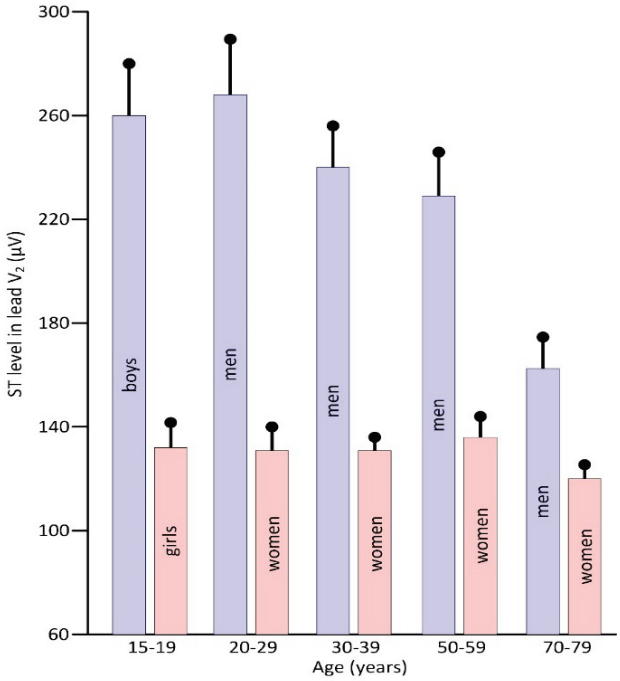

Fig. 7. Sex-related differences in ST level in V_2 (mean ± SD) according to age (derived from data of [17])

The principle mechanism that have been proposed to explain the difference between the sexes consists in hormonal effects on the expression or function of ion channels (Fig. 6): Sex hormones could regulate the expression of cardiac ion channels, for instance progesterone decreases inward rectifier K^+ current and increases delayed rectifier K^+ current through the nitric oxide production pathway. In spite of the progesterone effect female delayed rectifier K^+ current is lower in comparison to men what explains slower repolarization in women. Progesterone also prevents cyclic adenosine monophosphate enhancement of L-type Ca^{2+} current [15].

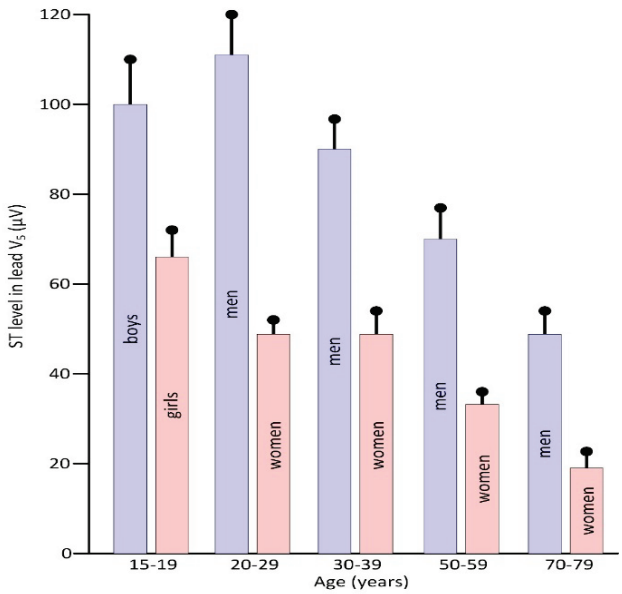

Fig. 8. Sex-related differences in ST level in V_5 (mean ± SD) according to age (derived from data of [17])

Moreover, myocardial ion channels function is modified also by testosterone. Bidoggia *et al.* [16] described prolonged QT interval in castrated men and shorter QT interval in women with hyperandrogenism. Restoration to the normal length after therapeutic testosterone administration was later proved on hypogonadic men by Charbit *et al.* [17]. This effect of testosterone is realized by functional modulation of L-type Ca^{2+} channels and primary repolarizing K^+ channels.

ST segment

The ST segment in females is flatter but generally the sex related differences in ST-T waveform patterns are relatively very small [14]. The physiological ST level (measured in the middle of the ST segment) is identical in both sexes only in childhood. Whereas in women it is almost unchanged during the whole life, in men it increases progressively during adolescence and thereafter it decreases gradually with age [18]. These patterns of sex-related differences changed with age are similar in leads V_2 a V_5, which are representative of the right and left ventricle, respectively (Fig. 7 and 8). The study of Ezaki *et al.* (2010) has also found that androgen-deprivation therapy significantly lowered ST levels in both leads and that they closely resembled the ST levels in age-matched control females. These results suggest that the ST segment level is modulated by testosterone. Generally, ST segment configuration is modified particularly by the transient outward K^+ current (I_{Kto}) triggered by opening of transient outward K^+ channels

after reaching of maximal transpolarization. As this current is significantly enhanced in men particularly due to higher density of transient outward K^+ channels in epicardium in men, the characteristic male ST segment including J point has higher potential level in comparison to women. Enhanced activation of delayed rectifier K^+ channels in males promotes faster repolarization resulting in u-shaped configuration of ST segment and asymmetric and tall T wave on the male ECG recording.

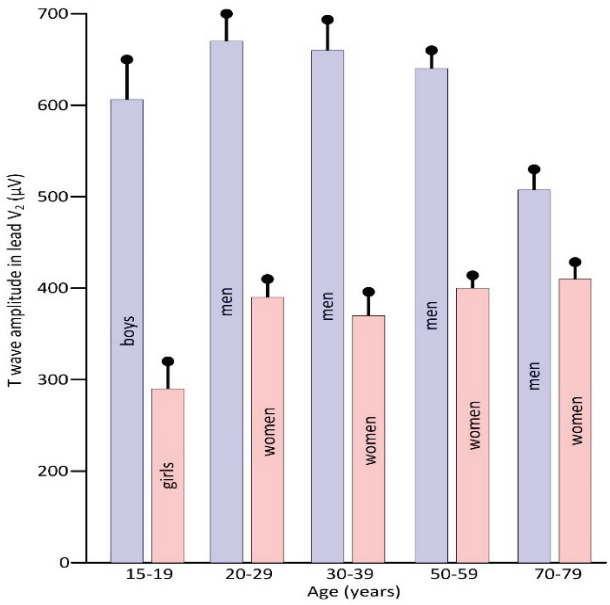

Fig. 9. Sex-related differences in T wave amplitude in lead V_2 (mean ± SD) according to age (derived from data of [17])

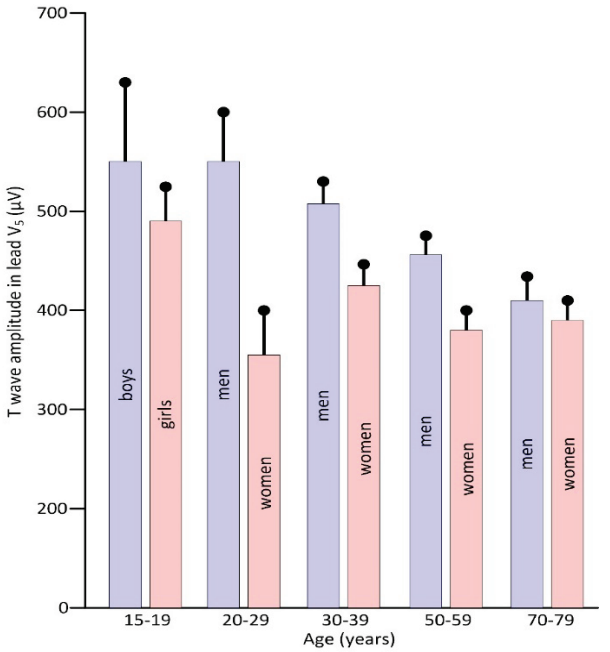

Fig. 10. Sex-related differences in T wave amplitude in lead V_5 (mean ± SD) according to age (derived from data of [17])

T wave

The T wave amplitude in lead V_2 (corresponding to the right ventricle) starts to be different between men and women also during adolescence and the difference decreases gradually with age [18], whereas the difference in the left precordium (V_5) starts to be significant only in adulthood at the age 20-29 years and then decreases. In comparison with the right precordium it stops to be significant in old age (Fig. 9 and 10). The possible mechanisms are endocrine-dependent different distributions of ion channels between both sexes, as female hearts show decreased expression of a number of repolarizing ion-channels

Pathological 12-leads ECG

The effects of gender differences, including sex hormones, on cardiac cell injury and death started to be studied only recently. Also studies focused on influence of sex hormones in determining rhythmogenesis and action potential configuration and conduction are very rare. On the other hand, hormones are important but not unique actors in this issue, further genetic and epigenetic determinants being involved [19]. Women have a higher prevalence of sick sinus syndrome, inappropriate sinus tachycardia, atrioventricular nodal reentry tachycardia, idiopathic right ventricular tachycardia, and arrhythmic events in the long-QT syndrome. In contrast, men have a higher prevalence of atrioventricular block, carotid sinus syndrome, atrial fibrillation, supraventricular tachycardia due to accessory pathways, Wolff-Parkinson-White syndrome, reentrant ventricular tachycardia, ventricular fibrillation and sudden death, and the Brugada syndrome [20].

Supraventricular arrhythmias

Different occurrence of supraventricular arrhythmias (SVT) in men and women is caused most likely by sex hormones. A cyclic variation in SVT occurrence in premenopausal women was described by Rosano *et al.* [15] demonstrating a higher incidence of SVT episodes during the luteal phase of the menstrual cycle. This finding suggests that progesterone can enhance vulnerability to SVT either directly or by increasing sympathetic activity. It corresponds also to a fivefold higher risk for SVT during pregnancy [21].

Reentrant supraventricular tachycardias

Atrioventricular nodal reentry tachycardia

(AVNRT) and atrioventricular reentrant tachycardia (ART) are the most common types of paroxysmal supraventricular reentrant tachycardia [22]. AVRNT has higher prevalence in women in comparison with men in a ratio 2:1 but in the case of ART the ratio is quite opposite: it is diagnosed twice as often in men as in women [20]. The higher incidence of AVNRT in women may be explained by a shorter refractory period of the atrioventricular pathway in women whereas higher incidence of ART in men corresponds to a higher occurrence of accessory atrioventricular pathways in men.

Atrial fibrillation

The prospective Framingham Heart Study found that men have a 1.5times higher risk of developing atrial fibrillation (AF) that women [23]. Atrial fibrillation occurs more frequently in ageing men but does not change in ageing women. The absolute number of women with AF, however, is higher than men because of their longer life expectancy [22] as there is almost twice the number of women as men over 75 years of age, where the percentage of AF is the highest. Higher incidence of AF in women at this age could be also related to their higher incidence of type 2 diabetes mellitus that is associated with an increased risk of AF [24]. According to the findings of Hnatkova *et al.* [25] women with paroxysmal AF have longer episodes and faster ventricular rates.

Ventricular arrhythmias

Gender differences have been observed in the epidemiology, pathogenesis and clinical presentation of various ventricular arrhythmias [26, 27]. In general, ST segment changes and abnormal T waves were found to be more prevalent in women consistently in all age groups (Fig. 11) whereas arrhythmias, bundle branch blocks, left axis deviation, and left ventricular hypertrophy were dominating in men [3]. Women are particularly less likely to experience ventricular tachycardia/ventricular fibrillation (VT/VF), and have fewer VT/VF episodes than men. We can only speculate that the underlying mechanism is an effect of sex hormones on the expression and function of ion channels that control cardiac cell excitation and repolarization as well as on key proteins that regulate Ca^{2+} dynamics at the cellular level. These mechanisms could explain already proved role of sex hormones in modifying the dynamic spatiotemporal (regional and transmural) heterogeneities in action potential duration, e.g., the arrhythmogenic substrate [28].

Long-QT syndrome

Sex differences in cardiac repolarization and the arrhythmogenic risk of patients with inherited and acquired long-QT syndromes (LQTS) are well appreciated clinically. Female sex exhibits longer QTc and is thus a risk factor for both congenital and acquired LQTS and uses to be associated with higher incidence of arrhythmic events, particularly torsade de pointes, in women [29, 30]. Women also exhibit higher risk of LQTS and related cardiac events in the period after giving birth.

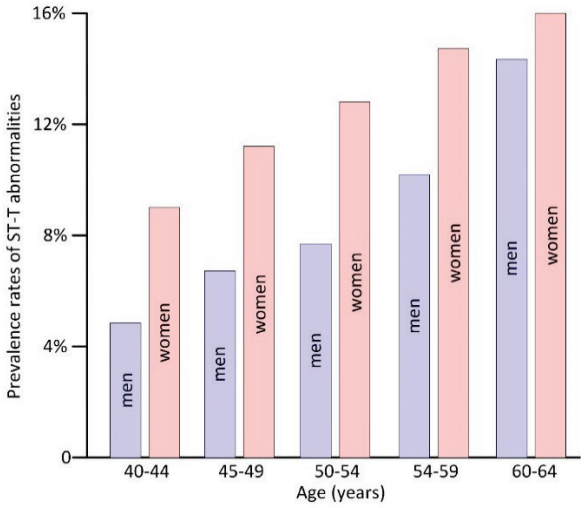

Fig. 11. Significant ST-T abnormalities in men and women according to age (derived from data of [2]).

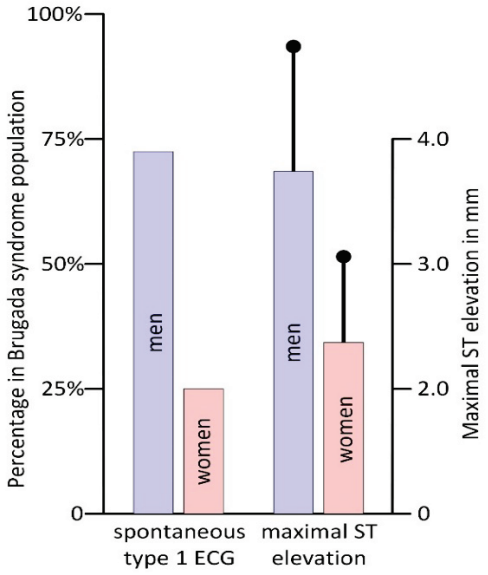

Fig. 12. Sex-related differences in ECG characteristics (mean ± SD) of patients with seriously symptomatic Brugada syndrome (derived from data of [31]).

Right ventricular outflow tract tachycardia (RVOT-VT)

Idiopathic monomorphic ventricular tachycardia originates most commonly from the right ventricular outflow tract (RVOT-VT) and is more common in women than in men [31]. Studies have identified triggers of RVOT-VT and found women tended to have frequent VT initiation during hormonal flux in the premenstrual state [32], whereas in men RVOT-VT is more commonly initiated by exercise or stress.

Brugada syndrome

In spite of fact that mutations in the SCN5A gene (responsible for a subset of patients with Brugada syndrome) are supposed to be inherited equally in both sexes, men are affected by Brugada syndrome more commonly than women. Moreover male sex is a risk factor of life-threatening arrhythmias in Brugada syndrome population. Electrocardiographic characteristics of patients with severely symptomatic Brugada syndrome significantly different according to gender [33] are summarized in Fig. 12.

Ventricular tachycardia and ventricular fibrillation

Women have been proved to have lower inducibility rate for ventricular tachycardia or ventricular fibrillation (VT/VF) at baseline electrophysiology testing than men. Albert CM *et al.* [34] identified in their study in survivors of cardiac arrest that 46 % of the women versus 27 % of the men had no inducible arrhythmia at baseline testing, but it must be admitted, that the inducibility of VT/VF during electrophysiological studies is not very reliable having low both sensitivity and specificity. On the other hand VT/VF are resulting from underlying cardiac disease, predominantly from coronary artery disease (CAD). As epidemiological data indicate that females are affected by CAD approximately 10 years later than males [35] it can be simply concluded that this fact can explain higher incidence of VT/VF in men. But a large multi-centric study has proved that while females with CAD had in this trial worse clinical status than males, they experienced fewer episodes of VT/VF [36]. Moreover, the authors of this study showed that ventricular tachycardia can be predicted by increased beat-to-beat QT variability in men but not in women what corresponds to the prognostic significance of changes in heart rate and QT interval in arrhythmogenesis [37]. These results suggest that men with CAD have a more vulnerable myocardial repolarization and thus a higher risk of VT/VF.

Conclusion

Sex differences suggest that biomedical principles, learned from the study of males, may not apply equally to females. Recognizing the differences between the sexes with respect to cardiac electrophysiology facilitates understanding of the mechanisms whereby homeostasis can be achieved using different contributions or components of the living system. Significant differences exist in the cardiac electrophysiology between men and women including gender differences in the incidence of arrhythmias. From a clinical standpoint, gender differences related to potentially life-threatening arrhythmias are the most relevant. Enhancing our knowledge of the mechanisms underlying the gender-related differences is critical to improve our personalized diagnostic decision making as well as therapeutic strategies for preventing severe cardiac arrhythmias and particularly sudden cardiac death.

Conflict of Interest

There is no conflict of interest.

Acknowledgements

Supported by the project National Institute for Research of Metabolic and Cardiovascular Diseases (Program EXCELES, ID Project No. LX22NPO5104) - Funded by the European Union – Next Generation EU.

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