Journal of Electrocardiology 51 (2018) 1029–1034

Contents lists available at ScienceDirect

Journal of Electrocardiology

journal homepage: www.jecgonline.com

ECG attenuation phenomenon with advancing age

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ARTICLE INFO

Available online xxxx

ABSTRACT

With advancing age, the conduction system and heart muscle cells undergo degenerative alterations affecting the electrocardiographic (ECG) parameters. The goal of the study is to determine the effect of age on ECG parameters. Are these changes due to a 'normal aging' or are they indicative to a 'heart disease'?
Italian Longitudinal Study on Aging (ILSA) database has been created to evaluate physiologic and pathologic modifications connected with aging. Standard 12-leads ECG recordings (10 s, 500 Hz) have been taken twice in a period of three years. The database consists of 890 individuals aged 65–84 years.
Analysis of changes has been done also in two groups of individuals in ILSA database – with and without cardiovascular abnormality. This analysis showed a nearly equal age-related change in both groups.
The main changes are reduction in the amplitudes of QRS- and T-waves, referred as 'ECG attenuation phenomenon'. $QRS_{t1} = 0.85$ mV at the time of the first recording t1, and $QRS_{t2} = 0.81$ mV at the time of the second recording t2, the period between the two recordings being three years ($p < 0.0001$). Respectively $T_{t1} = 0.11$ mV, $T_{t2} = 0.10$ mV, $p = 0.0013$.
ECG attenuation phenomenon is more common in males, and it is not significant in females. This attenuation is manifested significantly also in healthy male group, suggesting geriatric changes of "normal aging". Nevertheless, some cardiovascular diseases, such as arrhythmias and hypertension, may have a strong influence on the QRS attenuation, and this supports the open question of the clinical relevance of the phenomenon.
ECG attenuation phenomenon is more manifested in younger individuals and occurs independently of body mass. ECG attenuation phenomenon is a significant fact, and therefore it should be considered in any serial ECG study, for example in the ECG analysis for human identification.

Introduction

Normal aging is associated with a multitude of structural and functional changes in the cardiovascular system. Heart aging is manifested in increased heart's mass and fibrosis of the conduction system. Evidence for outcomes of recommended treatments for prevalent cardiac conditions in the elderly is lacking because generally the trials either included few elderly patients or excluded the elderly completely [1–3].

The occurrence of widespread histologic changes in the conduction system may alter several features of the aging electrocardiogram (ECG), including duration of the PR and QT intervals, orientation of the electrical axis, duration and morphology of the atrial and ventricular complexes, and characteristics of ventricular repolarization [4].

A pioneering study [5] in a group of 3376 apparently healthy males from 3 countries (Italy, Greece and US), aged 20 to 60 years, has found some electrocardiographic age trends, consisting of lowered wave amplitudes and prolonged durations. The authors assert that the magnitudes of the ECG changes are not of "borderline or abnormal" clinical evaluations, they are phenomena common to males; they occur independent of body weight and blood pressure, and possibly independent of the degree of asymptomatic atherosclerosis of the major coronary arteries.

A subsequent study [6] examined the changes in ECG between two serial recordings in a group of 440 healthy males at an age of 23 to 66 years on their first examination (the second was obtained 10 years later). The authors reported longer PR and QT interval duration, shorter QRS-complex duration and smaller T-wave and S-wave amplitude on the second investigation, and also they reported that the S-wave amplitude decreased more in younger age groups. The above mentioned study considered only male population, not providing the opportunity to investigate on gender influence.

In [7], a group of 698 healthy elderly people with normal ECG was studied, observing not significant age-related changes of QRS duration (with higher values in males), whereas the QRS axis shifts leftward as age increases.

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https://doi.org/10.1016/j.jelectrocard.2018.08.019
0022-0736/

In [8], the effect of age and gender were studied in three different populations: 1782 neonates, infants and children, 1555 adult white population and 503 adult Chinese individuals. In the paediatric group, a link has been found between age and QRS duration, which increased linearly from about 1 year of age to adolescence. In the two adult groups, the authors confirmed increased QRS duration in males compared to females in all age groups.

In [9], it was noted that there is a highly significant increase in prevalence of ECG abnormalities with advancing age, suggesting strong age dependency.

In [10] two different "normal" databases (112,055 US adult people and 3918 adult Chinese individuals) were studied for age/gender relationship in ECG measurements, showing significant gender differences for QRS duration and QT/QTc interval.

A recent study [3] reported age related QRS widening, QRS increase in amplitude and T-wave amplitude decrease.

In addition, some authors examined the particular group of centenarians [11,12].

In the present study we examined serial ECG recordings in 3-year interval in elderly people in order to study the differences in ECG/VCG measurements. The 3-year interval has been long enough to observe some changes and short enough to limit the influence of concomitant diseases.

Materials

In this study a serial analysis has been done on a set of ECGs from a database, developed in the framework of Italian Longitudinal Study on Aging Project (ILSA). This database has been created to evaluate physiologic and pathologic modifications connected with aging [13–16].

Standard 12 leads ECG recordings (10 s, 500 Hz) were taken at baseline and after three years. The database consists of a random sample of individuals aged 65–84 years, living independently or in institutions, stratified by age and sex using the equal allocation strategy, identified on the demographic list of the registry office of eight Italian municipalities: Genova, Segrate (Milan), Selvazzano-Rubano (Padova), Impruneta (Florence), Fermo (Ascoli Piceno), Naples, Casamassima (Bari) and Catania. The first set of standard 12-leads ECGs was acquired between 1992 and 1993, and the second – between 1995 and 1996.

Of the 903 participants in the ILSA database, 13 were excluded, due to bad quality of ECG either at the time of the first (t1), or second (t2) recording. The 890 patients, involved in the study, were stratified in 4 major groups, according to their status at the time of their first medical examination:

- Patients with a history of cardiovascular disease (group 1): arrhythmia, angina pectoris, myocardial infarction, congestive heart failure (HF), arterial hypertension, peripheral artery disease.
- Patients with diabetes mellitus (group 2)
- Individuals with neurological conditions (group 3): dementia of any type, distal symmetric neuropathy of lower limbs, parkinsonism, stroke
- Healthy subjects (group 4): these subjects were characterized by the absence of any cardiovascular, neurological or chronic pulmonary disease, by the absence of therapy potentially influencing cardiac electrical activity, and by the absence of electrolyte imbalance.

Table 1
Clinical characteristics of ILSA database.

	Healthy	Arrh.	Angina	MI	HF	AH	DM
Healthy	294	0	0	0	0	121	21
Arrh.	0	261	34	32	22	174	35
Angina	0	34	70	24	9	54	6
MI.	0	32	24	75	9	50	8
HF	0	22	9	9	34	22	8
AH	121	174	54	50	22	541	67
DM	21	35	6	8	8	67	91

Arrh. = Arrhythmia; MI = myocardial infarction; HF = heart failure; AH = arterial hypertension; DM = Diabetes mellitus.

Clinical information is presented in Table 1. The neurological group (group 3), not relevant to the current study, is not reported.

Methods

All ECG parameters were automatically measured, thus assuring repeatability of the results and lack of intuitive subjectivism, typical to the manual marking and measurement.

The ECG signals were preprocessed to eliminate or suppress the powerline interference [17], the drift of the baseline [18] and the electromyographic noise [19].

QRS detection was applied [20], and mean P-QRS-T interval was formed by best matching (cross correlation) of successive P-QRS-T intervals (Fig. 1). The use of a mean signal excluded the random selection of an atypical or noise-contaminated signal. Ectopic beats were automatically recognized and were excluded from the mean P-QRS-T.

Fiducial points, such as the beginning and end of the P-, QRS- and T-waves, were allocated automatically on the mean P-QRS-T interval, using algorithms presented in [21,22].

Typical age-related changes of the ECG parameters are shown in Fig. 2, where visual attenuation of all P-, QRS- and T-waves can be seen.

Morphology parameters: Principle Component Analysis (PCA) through singular value decomposition was applied to QRS and T-wave

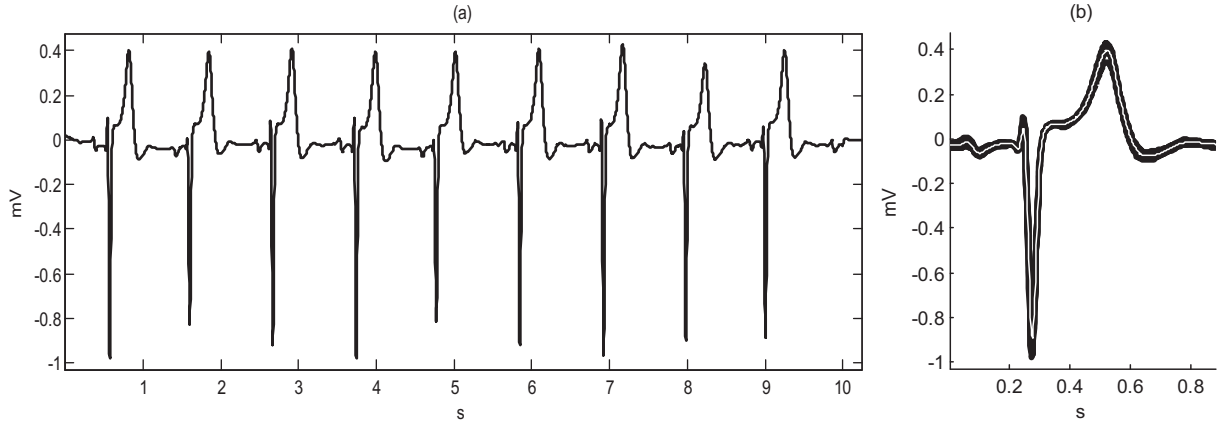

Fig. 1. (a). The amplitudes of the negative peaks of the QRSs vary from −0.96 to −0.79 mV. (b) Overlaying of all P-QRS-T intervals (black traces) and their mean signal (white trace). The amplitude of the negative peak of the mean QRS is −0.89.

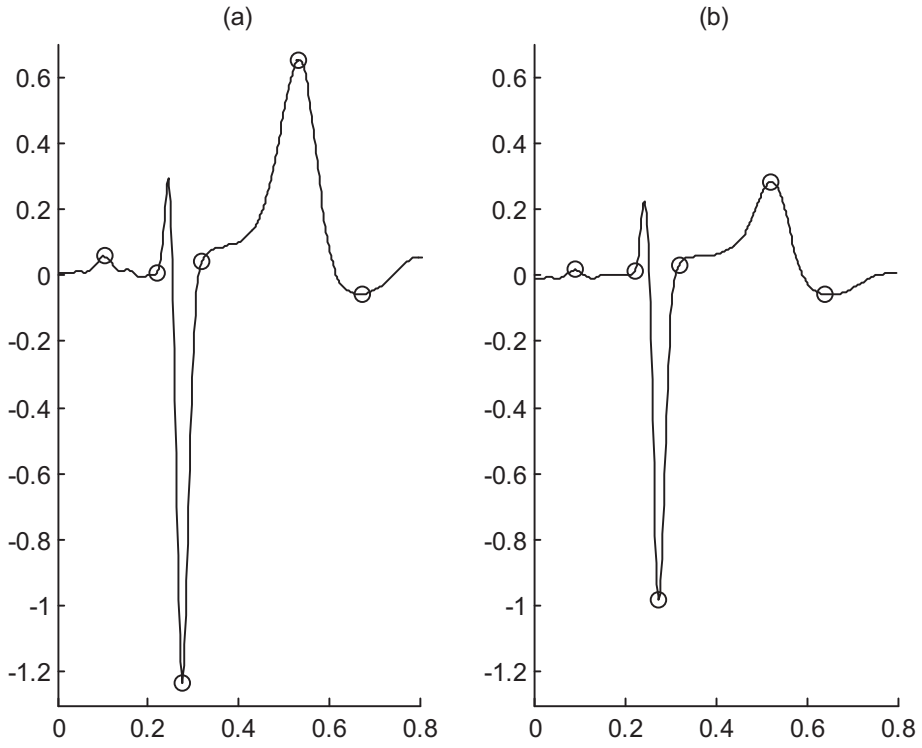

Fig. 2. (a). Mean P-QRS-T of V2-lead, recorded at a time t1. (b) Mean P-QRS-T of V2-lead, of the same person, recorded three year later. Typical attenuation of all waves can be observed.

intervals of all ECG leads. Usually the first three components of the eigenvectors provide the total variation/information of the original data, and the first component gives the greatest contribution. For this study, the ratio between the second and the first Eigen values (complexity index) was used:

$$\text{QRS PCA} : \lambda_{2(\text{QRS})}/\lambda_{1(\text{QRS})}$$

$$\text{T PCA} : \lambda_{2(\text{T})}/\lambda_{1(\text{T})}$$

which is related to the measure of the width of the QRS or T-wave loop.

Orthogonal Frank X, Y and Z leads were derived from the standard 12-leads, using the transfer matrix of Dower [23]. Several vectorcardiographic 3-D and frontal 2-D parameters were measured: maximal vector of QRS- and T-loops, area of the loops, angle between the maximal vectors, etc.

Ratio of maximum to mean vector magnitudes was calculated, characterizing the loop as either elongated (narrow) or rounded:

$$\text{Vector ratio} = \text{Vmax}/\text{mean}(\text{Vn}),$$

where *Vmax* is the magnitude of the maximal vector of the loop, and *Vn* is the magnitude of the spatial vector at a sample *n*.

The magnitudes of the vectors were calculated by:

$$V = \sqrt{V_x^2 + V_y^2 + V_z^2}$$

The vector ratio (VR) parameter was adopted from [24], and was used further by us for myocardial infarction and ischemia

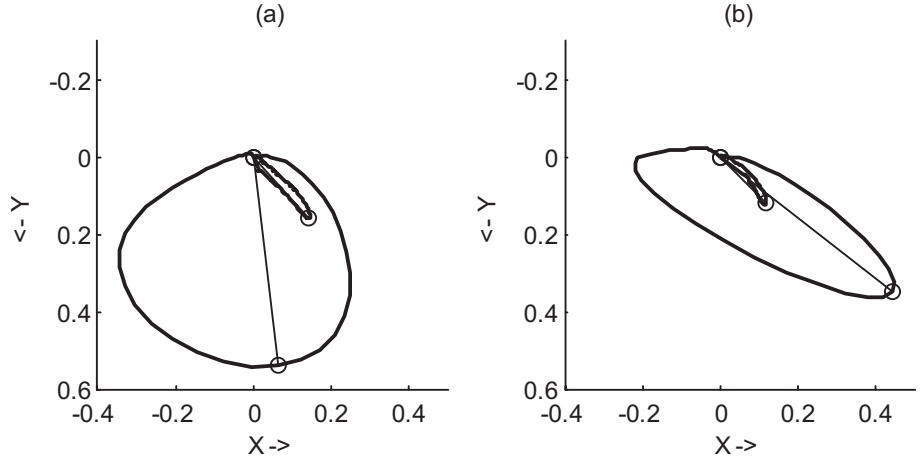

Fig. 3. (a). Vectorcardiographic QRS- and T-loops at a time t1. (b) Vectorcardiographic loops of the same person, recorded three year later. As an effect of aging, the QRS-loop area has decreased from 0.25 mV^2 to 0.12 mV^2, while the form of QRS-loop has become more elongated. The QRS-loop vector ratio has changed from 2.6 mV to 3.3 mV, and the QRS roundness index has changed from 0.85 mV to 0.38 mV at almost no change of the QRS amplitude of the maximal vector.

Table 2
Changing of ECG parameters caused by aging.

Parameter	ECG or VCG	Mean ± Standard deviation		Paired *t*-test (p)
		t1	t2 = t1 + 3 years	
QRS amplitude [mV]	ECG	0.85 ± 0.34	0.81 ± 0.33	<0.0001
QRS area [mV ∗ mV]	VCG	0.117 ± 0.135	0.121 ± 0.127	ns
QRS vector, max [mV]	VCG	0.73 ± 0.27	0.72 ± 0.27	ns
QRS angle [°]	VCG	274.7 ± 120.3	269.9 ± 126.4	ns
QRS vector ratio [number]	VCG	3.39 ± 0.70	3.34 ± 0.69	0.012
QRS PCA [number]	ECG	0.26 ± 0.29	0.25 ± 0.19	ns
QRS duration [ms]	ECG	118 ± 19.0	119 ± 21.3	ns
QRS roundness index [number]	VCG	0.248 ± 0.22	0.250 ± 0.22	ns
QT interval duration [ms]	ECG	428 ± 41.7	427 ± 44.5	ns
QT dispersion [ms]	ECG	39.6 ± 12.2	38.7 ± 19.6	ns
ST elevation [mV]	ECG	0.060 ± 0.13	0.066 ± 0.16	0.029
T ampl [mV]	ECG	0.11 ± 0.08	0.10 ± 0.08	0.0013
T area [mV ∗ mV]	VCG	0.0053 ± 0.0063	0.0052 ± 0.0067	ns
T vector, max [mV]	VCG	0.22 ± 0.10	0.21 ± 0.10	0.028
T angle [°]	VCG	315. ± 74.1	308 ± 84.4	0.025
T vector ratio [number]	VCG	2.56 ± 0.45	2.54 ± 0.43	ns
T PCA[number]	ECG	0.10 ± 0.11	0.11 ± 0.12	0.015
T roundness index [number]	VCG	0.117 ± 0.13	0.120 ± 0.12	ns
HR [bpm]	ECG	68.2 ± 11.6	68.9 ± 12.1	ns

characterization of T-loop morphology in VCG [25], and for longitudinal characterization of the VCG loops [26].

Another parameter, characterizing the roundness of the loop, is the roundness index (RI), measured as the ratio of the area of the loop to the square of the maximum vector [27]:

$$RI = Area/Vmax^2$$

Changes of VCG parameters, caused by aging, are shown in Fig. 3. Visual attenuation of the QRS-area in the frontal XY plane can be seen.

Results

The age-related changes of ECG parameters are presented in Table 2. Statistically significant changes were reported in the following:

- Decrease of QRS amplitude in ECG, from 0.85 ± 0.34 mV to 0.81 ± 0.33; $p < 0.0001$;
- Decrease of the QRS vector ratio in VCG, from 3.39 ± 0.70 mV to 3.34 ± 0.69; $p = 0.012$;
- Increase of the ST elevation in ECG, from 0.060 ± 0.13 mV to 0.066 ± 0.16 mV; $p = 0.029$;
- Decrease of T-wave amplitude in ECG, from 0.11 ± 0.08 mV to 0.10 ± 0.08; $p < 0.0013$;
- Decrease of the maximal vector of the T-loop in VCG from 0.22 ± 0.10 to 0.21 ± 0.10; $p = 0.028$;
- Decrease of the angle (clockwise shift) of the maximal vector of the T-loop in VCG from 315 ± 74.1 to 308 ± 84.4; $p = 0.025$;
- Increase of the PCA value (morphology change) of the T-wave in ECG, from 0.10 ± 0.11 to 0.11 ± 0.12; $p = 0.015$.

A pronounced and significant reduction of QRS and T wave amplitudes (ECG attenuation) can be observed. The effect of gender on the ECG attenuation, with advancing age, is presented in Table 3. The results show clearly that the phenomenon of aging-related ECG attenuation is significant only for males ($p < 0.0001$ for QRS and $p = 0.008$ for T-wave) and not significant for females, for both QRS- and T-waves.

To analyse the effect of age on the ECG attenuation, the entire group of 890 patients was divided into 3 age groups (≤69 years, 70 ÷ 75 years, and >75 years), Table 4.

The results indicated clearly that the youngest group (65 ÷ 69 years) showed the most significant decrease of QRS and T wave amplitudes, ($p < 0.0001$ and $p = 0.0013$). Less decrease was observed in the middle group (70 ÷ 75), while the oldest group (76 ÷ 84 years) had no significant changes.

Analysis of ECG attenuation according to cardiovascular diseases and risk factors was performed in a relatively balanced population of males (476) and females (414), Table 5.

In females the presence of cardiovascular diseases and risk factors had no significant effect on the magnitude of ECG attenuation.

The significant QRS attenuation in the male group was also present in subgroups with arterial hypertension ($p < 0.0001$), arrhythmia ($p < 0.01$), normal blood pressure ($p = 0.004$), and healthy group ($p < 0.001$).

Attenuation of the T-wave amplitude in males was present in subgroups with arterial hypertension ($p = 0.032$), normal blood pressure ($p = 0.011$), and healthy group ($p = 0.0014$).

We analysed the effect of weight gain/loss between the two ECG-acquisition time-points, because modification of body impedance may influence ECG amplitudes. Table 6 reports the change in body mass index (BMI) according to gender and presence or absence of cardiovascular abnormalities. The analysis showed that BMI does not change significantly during follow-up.

Discussions

In the present study we found an age-related ECG attenuation in males, but not in females, analyzing the large ILSA database. This finding was not associated with any specific cardiovascular disease, and was also present in the healthy subgroup.

The reported QRS and T-wave attenuation caused by aging, is in accordance with other studies of age-related ECG changes [5,6], and in part agrees with the geriatric lowering of T-waves amplitudes, but conflicts with the increase of QRS amplitudes reported in [3].

Our results showed a non-significant age related QRS widening (118 ms at t1, and 119 ms at t2). The latter was proven as a negative prognostic factor [28]. There are conflicting results in the literature concerning QRS duration with aging. A decrease in QRS duration was reported in [6], while an increasing in QRS width was found in [3,8].

The differences in the results reported in the literature are probably due to the structure of the databases used. It is important whether ECG parameters of the same person are compared over a certain period of time (as it is in the current study and in [6]), or whether different age groups are compared. It is known that left ventricular hypertrophy results in an increase in QRS amplitude. Therefore, echocardiographic data should be accounted for in studies analysing the effects of aging on QRS amplitude. A limitation of the present study is that ILSA database does not include echocardiographic data. Nevertheless, we have data about blood pressure values, and they remain similar during the follow-up.

Table 3
Effect of gender on the ECG attenuation.

Clusters	n	QRS ampl [mV]			T ampl [mV]		
		t1	t2	p	t1	t2	p
All subjects	890	0.85 ± 0.34	0.81 ± 0.33	< 0.0001	0.11 ± 0.08	0.10 ± 0.08	0.0013
Males	476	0.90 ± 0.35	0.84 ± 0.35	< 0.0001	0.13 ± 0.09	0.11 ± 0.09	0.008
Females	414	0.79 ± 0.31	0.78 ± 0.30	ns	0.10 ± 0.07	0.10 ± 0.07	ns

Table 4
Effect of age on the ECG attenuation.

Clusters	n	QRS ampl [mV]			T ampl [mV]		
		t1	t2	p	t1	t2	p
All subjects	890	0.85 ± 0.34	0.81 ± 0.33	<0.0001	0.11 ± 0.08	0.10 ± 0.08	0.0013
Age ≤ 69	268	0.87 ± 0.34	0.80 ± 0.33	<0.0001	0.13 ± 0.09	0.11 ± 0.08	0.00009
Age 70 ÷ 75	294	0.83 ± 0.33	0.78 ± 0.29	0.002	0.11 ± 0.08	0.10 ± 0.07	0.016
Age > 75	328	0.86 ± 0.35	0.84 ± 0.35	ns	0.10 ± 0.07	0.10 ± 0.08	ns

T-wave age-related attenuation was more pronounced in males, and insignificant in females, and this point was in agreement with previous studies [5,29].

Two main hypotheses are available for explanation of age-related ECG changes:

1) Altered topography of the heart in relation to thorax and diaphragm with advancing age [30].
2) Age-related degenerative structural modifications in the conduction system and heart muscle cells affecting ECG parameters [3].

The question remains, if these changes could be attributed to a 'normal aging' process?

The significant decrease of the QRS amplitude also in the healthy male group suggests geriatric changes to "normal aging". Nevertheless, some cardiovascular diseases, such as arrhythmias and hypertension, may have a strong influence on the QRS attenuation, and this supports the open question of the clinical relevance of the phenomenon.

Limitations

The time interval of serial ECG recordings is of importance in age related studies. We used the ILSA database with a time-interval of 3 years, which is not long enough to observe significant age-related changes in most of the ECG and VCG parameters. Exceptions are only the QRS and T-wave amplitudes. The interval of 10 years used in [6] may give enough time for subtle changes in ECG parameters to become statistically significant, but with the limitations of a probability for occurrence of concomitant heart diseases during the long period of observation. Another limitation in our study group is the advanced age at baseline (65–84 years).

Conclusions

Geriatric changes of the ECG are observed and reported in the literature. The analysis of serial changes, 3 years apart, in a group of individuals with/without cardiovascular diseases of the ILSA database showed significant age-related changes.

The main ECG alteration that we observed was a reduction in the amplitudes of the QRS- and T-waves, referred as 'ECG attenuation phenomenon'. This phenomenon was present in males and was insignificant in females. Significant ECG attenuation in male healthy group suggests geriatric changes to "normal aging", although some cardiovascular diseases may have a strong influence on the QRS attenuation. In the age range of the ILSA contingent, the ECG attenuation was more manifested in the younger group. ECG attenuation phenomenon with advancing age, however, is a significant fact, and should be considered in any serial ECG study, for example when using ECG for human identification.

Acknowledgements

This study was supported by a joint project between the Italian National Research Council (Institute of Neuroscience IN-CNR) and the Bulgarian Academy of Sciences (Institute of Biophysics and Biomedical Engineering).

The authors acknowledge the Working Group of the Italian Longitudinal Study on Aging, The ILSA Working Group: E. Scafato, G. Farchi, L. Galluzzo, C. Gandin, Istituto Superiore di Sanitá, Rome; A. Capurso, F. Panza, V. Solfrizzi, V. Lepore, P. Livrea, University of Bari; L. Motta, G. Carnazzo, M. Motta, P. Bentivegna, University of Catania; S. Bonaiuto, G. Cruciani, F. Fini, A. Vesprini, D. Postacchini, Italian National Research Centre on Aging (INRCA), Fermo; D. Inzitari, L. Amaducci, University of Florence; A. Di Carlo, M. Baldereschi, Institute of Neuroscience, National

Table 5
ECG attenuation according to cardiovascular and metabolic diseases and gender.

Clusters	Gen-der	n	QRS ampl [mV]			T ampl [mV]		
			t1	t2	p	t1	t2	p
All subjects	f	414	0.79 ± 0.32	0.78 ± 0.30	ns	0.10 ± 0.07	0.09 ± 0.07	ns
	m	476	0.90 ± 0.35	0.84 ± 0.35	<0.0001	0.13 ± 0.09	0.11 ± 0.09	0.008
Diabetes mellitus	f	47	0.72 ± 0.29	0.73 ± 0.30	ns	0.08 ± 0.05	0.09 ± 0.07	ns
	m	44	0.90 ± 0.46	0.82 ± 0.32	ns	0.11 ± 0.08	0.11 ± 0.08	ns
Angina Pectoris	f	28	0.84 ± 0.43	0.80 ± 0.30	ns	0.08 ± 0.05	0.08 ± 0.09	ns
	m	42	0.89 ± 0.31	0.89 ± 0.34	ns	0.08 ± 0.07	0.10 ± 0.08	ns
Arterial hypertension	f	277	0.82 ± 0.34	0.80 ± 0.32	ns	0.10 ± 0.07	0.09 ± 0.07	ns
	m	264	0.94 ± 0.36	0.86 ± 0.38	<0.0001	0.12 ± 0.09	0.11 ± 0.09	0.032
Normal blood presser	f	131	0.73 ± 0.26	0.73 ± 0.27	ns	0.10 ± 0.07	0.09 ± 0.07	ns
	m	209	0.87 ± 0.33	0.81 ± 0.30	0.004	0.13 ± 0.08	0.11 ± 0.08	0.011
Myocardial infarction	f	19	0.77 ± 0.39	0.78 ± 0.37	ns	0.08 ± 0.08	0.08 ± 0.06	ns
	m	56	0.79 ± 0.29	0.77 ± 0.34	ns	0.08 ± 0.06	0.06 ± 0.06	ns
Arrhythmia	f	97	0.86 ± 0.38	0.80 ± 0.37	ns (0.06)	0.09 ± 0.07	0.08 ± 0.07	ns
	m	164	0.89 ± 0.37	0.83 ± 0.38	0.0096	0.10 ± 0.09	0.10 ± 0.08	ns
Hearth failure	f	19	0.88 ± 0.33	0.87 ± 0.30	ns	0.07 ± 0.07	0.07 ± 0.09	ns
	m	15	0.80 ± 0.17	0.74 ± 0.31	ns	0.06 ± 0.05	0.09 ± 0.09	ns
Peripheral artery disease	f	21	0.85 ± 0.44	0.80 ± 0.30	ns	0.09 ± 0.09	0.08 ± 0.07	ns
	m	33	0.85 ± 0.26	0.84 ± 0.35	ns	0.10 ± 0.07	0.09 ± 0.08	ns
Healthy	f	130	0.75 ± 0.29	0.75 ± 0.28	ns	0.11 ± 0.07	0.10 ± 0.07	ns
	m	164	0.92 ± 0.34	0.85 ± 0.31	0.0006	0.15 ± 0.09	0.12 ± 0.09	0.0014

Table 6
Body mass index changes during follow-up.

Clusters	n	Body mass index [kg/m^2]		
		t1	t2	p
All subjects	756	26.6 ± 4.0	26.7 ± 4.1	ns
Males	427	26.2 ± 3.6	26.3 ± 3.7	ns
Females	329	27.2 ± 4.5	27.0 ± 4.5	ns
Cardiovascular disease	493	26.8 ± 4.1	26.9 ± 4.2	ns
Healthy	263	26.2 ± 3.9	26.1 ± 3.7	ns

Research Council of Italy (CNR), Florence; A. Ghetti, R. Vergassola, Health Area 10, Florence; C. Gandolfo, M. Conti, University of Genoa; N. Canal, M. Franceschi, San Raffaele Institute, Milan; G. Scarlato, L. Candelise, E. Scarpini, University of Milan; F. Rengo, P. Abete, F. Cacciatore, F. Covelluzzi, University of Naples; G. Enzi, L. Battistin, G. Sergi, G. Crepaldi, M. Bressan, University of Padua; G.Bortolan, National Research Council of Italy (CNR), Padova; S. Maggi, N. Minicuci, M. Noale, National Research Council of Italy (CNR), Aging Section, Padua; F. Grigoletto, E. Perissinotto, Institute of Hygiene, University of Padua; P. Carbonin, Universitá Cattolica del Sacro Cuore, Rome.

The authors are grateful to the helpful reviewers' comments.

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