Statistical differences between event and control patients for selected features

Feature	Event	Control	P
RR interval average length (ms)	790 ± 181	895 ± 161	.0420
PR interval standard deviation (ms)	52.3 ± 12.1	37.8 ± 8.1	<.0001
Standard deviation of peak of P-wave to peak of Q-wave (ms)	44.6 ± 14.3	30.2 ± 7.3	<.0001
QRS complex standard deviation (ms)	40.8 ± 10.2	35.8 ± 5.1	.0266
QT interval average length (ms)	1061 ± 965	651 ± 177	.0277
QT interval standard deviation (ms)	5317 ± 4185	3643 ± 1320	.0464
Start of Q wave to start of T wave (ms)	1141 ± 1008	739 ± 226	.0403
Start of Q wave to peak of T wave (ms)	1185 ± 1008	778 ± 232	.0385
P-wave average elevation (mV)	20.32 ± 24.88	42.46 ± 29.66	.0122

Values are reported as mean ± SD.

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Is heart rate a better risk predictor than heart rate variability?

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Although some publications in the past showed the good predictive power of the heart rate at rest, it did by far not achieve the attention compared with the parameters of heart rate variability.

In addition, it is commonly believed that heart rate and heart rate variability are statistically independent parameters. Particularly the latter seems not to be true, as investigations show findings that will be presented here. The observable range of the standard deviation of heart rate variability decreases with increasing heart rate at rest as well for a larger ensemble of patients who are undergoing a stress test. Thus, even if the correlation between heart rate and heart rate variability is poor, it is a fact that at high heart rates, the possible standard deviation of heart rate variability becomes low. In other words, heart rate variability is not independent of heart rate. Thus, an increased risk for patients with high heart rate means automatically that they have a low standard deviation of the heart rate variability.

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Normal limits of the ECG in African blacks

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Introduction: Racial variation in the electrocardiogram (ECG) is well known. This study aimed to derive normal limits of the ECG from a large population of healthy individuals living in Nigeria.

Methods: Twelve-lead ECGs were recorded using a Burdick Atria 6100 electrocardiograph in and around Ilorin, Nigeria. Volunteers were recruited from the University of Ilorin and from surrounding villages. Each was medically examined by a physician, and a detailed medical history was obtained. Data were gathered locally on a PC and sent to Glasgow for further analysis. Electrocardiograms were reviewed to exclude any that were technically unsatisfactory and others that had an unexpected abnormality.

The ECG measurements underwent statistical analysis using SAS v9.1 (SAS, Cary, NC). Plots and summary statistics were used to assess, informally, the relationship with age and sex. Regression techniques addressed formal relationships. Reference ranges were established by splitting the data into age-sex subgroups and by calculating the 96th percentile range within each subgroup.

Results: The study included 782 men and 479 women, all apparently healthy, with a relatively even spread of ages between 20 and 87 years.

Heart rate was higher in women than men in all age groups and increased with age. The mean (SD) heart rate was 81 (14) beats/min in women and 75 (13) beats/min in men ($P < .0001$). Mean (SD) QRS duration was 87.8 (9.4) milliseconds in men and 83.4 (7.7) milliseconds in women ($P < .0001$). The upper limit of normal QTc (Hodges) increased with age, whereas the mean (SD) QTc was 393 (16) milliseconds in men and 407 (17) milliseconds in women ($P < .0001$).

The Cornell product was higher in all male age groups compared with corresponding female groups. It decreased with increasing age in men, but the reverse was true in women.

There was a clear trend for STj amplitude to be lower in older individuals and to be higher in men compared to women. This was particularly true for V_2 and V_3.

T-wave inversion in V_2 was more prevalent in women and was not present in any men older than 30 years. This was also the case for V_3.

Conclusion: This is the first large study of automated ECG recording in healthy blacks living in West Africa. Many of the age- and sex-related changes were similar to those seen in whites, but there were some unexpected findings that require further study.

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Dispersion of APD restitution quantified from the surface ECG

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Dependence of repolarization duration on heart rate has been shown to provide relevant information for arrhythmic risk stratification. A way of characterizing the relationship between the action potential duration (APD) and the RR interval is by the APD restitution (APDR) curve. Heterogeneities in ventricular myocardium make the APDR curve present spatial variations, and some studies propose transmural dispersion of restitution to act as a potent arrhythmogenic substrate. Also, increments in restitution dispersion have been associated with greater propensity for ventricular tachycardia/fibrillation.

The main limitation on the usability of APDR dispersion is that its quantification requires invasive procedures. We have developed a method to estimate dispersion of the APDR curves by making only use of the surface electrocardiogram (ECG, specifically based on the dynamics of the distance from T-wave peak to T-wave end (Tpe). The underlying hypothesis of this work is that changes of Tpe reflect changes in spatial dispersion of repolarization, in some studies related to transmural dispersion.

We measure differences in Tpe normalized by differences in the RR interval, that is, $\Delta Tpe/\Delta RR$, between 2 different stationary states separated by a transient heart rate change as in a tilt test trial. If we restrict spatial to transmural dispersion of APD restitution, then it is possible to estimate the dispersion increase as $\Delta\alpha = (\alpha_{mid} - \alpha_{epi})$, where α_{mid} and α_{epi} denote the midmyocardial and the epicardial slopes of the restitution curves, respectively, and are computed for a specific RR range as: $\alpha_{mid} = \Delta APD_{mid}/\Delta RR$ and $\alpha_{epi} = \Delta APD_{epi}/\Delta RR$. Noting that in this study, ΔTpe represents $\Delta APD_{mid} - \Delta APD_{epi}$, we propose that $\Delta\alpha$ can be estimated as: $\Delta\alpha = \Delta Tpe/\Delta RR$.

We have evaluated our index on ECG recordings of healthy subjects performing the tilt test, by using selected ECG segments presenting stable heart rate. In our study, mean and SD values of $\Delta Tpe/\Delta RR$ are 0.0371 ± 0.0327 ms/ms, which are in very good agreement with differences in dynamic APDR slopes evaluated for the same RR range using an electrophysiologically detailed human ventricular model (ten Tusscher 2006), where $\Delta\alpha$ is 0.0364 ± 0.0217. The mean ± SD value of the individual differences is −7.3E − 4 ± 0.0256, where SD quantifies the intrasubject variability. This comparison considers the spatial dispersion represented by Tpe to be transmural, and then modelling including connections and a more complete geometry will be the next step to evaluate the extrapolation of these results to more realistic conditions.

In brief, the method proposed in this study allows estimating dispersion of the dynamic APD restitution slopes along the ventricular wall by

making only use of the surface ECG. A potential application of this method is to identify drugs that alter restitution curves in such a way that they increase dispersion of restitution and lead to cardiotoxicity.

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Gender differences in regional action potential durations and repolarization times

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Longer QT in women than in men despite shorter QRS is thought to predispose women to adverse effects of cardioactive agents. We used a novel repolarization model in 4992 normal men and women with normal ventricular conduction to estimate regional repolarization times (RTs) and action potential durations (APDs), with the objective to elucidate their possible role as a mechanism for generating the sex differences in global QT. The model uses global rate-adjusted QTpeak time for RT_{epi} and QRpeak time (QRp) for excitation time (ET_{epi}) of the left lateral wall, with specific adjustments for other regions. Endocardial RT was determined by the algorithm: $RT_{endo} = RT_{epi} - \lambda * (ET_{endo} - ET_{epi})$, where the regression coefficient $\lambda = -0.25$ was obtained by an iterative procedure to maintain the normal left ventricular repolarization reversed and to keep APD gradients within normal limits.

Parameter estimates for repolarization times and action potential durations by sex*

	RT_{epi}	APD_{epi}	APD_{endo}	APD_{grad}	Te-Tp	QT	QRSd	APD_{ter}
Men	320	275	316	41	95	415	95	320
	(21)	(21)	(21)	(7.4)	(19)	(22)	(9)	(21)
Women	337	294	334	40	88	426	90	336
	(20)	(20)	(21)	(6.6)	(19)	(21)	(9)	(21)
Men− women	−18†	−19†	−17†	2NS	7†	−11†	5†	−16†

*Parameter estimates (in milliseconds) are all rate adjusted, with rate-adjusted QTpeak interval being a common reference.
† $p < .001$.
NS = non-significant for gender differences.
Subscripts "epi" and "endo" refer to epicardial and endocardial, respectively; RT_{epi}, rate-adjusted QTpeak interval; $APD_{grad} = (APD_{endo} - APD_{epi})$; Te-Tp, Tend-Tpeak interval; APD_{ter}, APD of the last region repolarized.

The data, listed above for left lateral wall, were closely similar for other regions and showed that APDs were 16 to 19 milliseconds shorter and rate-adjusted QT was 11 milliseconds shorter in men than in women ($P < .001$), but sex differences in transmural RT gradients were minimal. Tend-Tpeak interval was rate invariant. Shorter APDs and earlier onset of RT_{epi} in men during repolarization initial half-period are the primary determinant of sex differences in QT interval. More localized rather than transmural RT gradients remain a plausible mechanism for cardiovulnerability in women.

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