End QRS Notching or Slurring in the Electrocardiogram

Influence on the Definition of “Early Repolarization”

To the Editor: The transition from the QRS complex to the ST-segment on the 12-lead electrocardiogram (ECG) has been conventionally termed the ST junction (STj). Occasionally, the terminal portion of the QRS complex manifests a notch or slur when transiting into the ST-segment. This has been described as a normal variant in healthy individuals (1) in whom STj is very often elevated. The term “early repolarization” has been used to describe the latter phenomenon with or without end QRS notching or slurring (2).

Until recently, end QRS notching or slurring has generally been considered benign. However, Haïssaguerre et al. (3) reported a higher frequency of the early repolarization pattern in 206 survivors of idiopathic ventricular fibrillation compared with 412 healthy controls matched for age, sex, race, and level of physical activity. More recently, a “horizontal” ST-segment with or without STj elevation, but with end QRS notching or slurring, has been shown to have an important prognostic role (4,5).

Although numerous studies have investigated the benign and pathological significance of end QRS slurring or notching, there are many inconsistencies in definitions. The goal of our study was to explore the prevalence and demographic characteristics of QRS end notching or slurring and of associated STj amplitude in an apparently healthy white population.

ECG recordings were obtained from 1,496 adults as previously described (6). There were 859 males and 637 females, with a mean age of 37.4 ± 12.6 years (range 18 to 82 years) overall (39.2 ± 12.1 years [range 18 to 82 years] for males and 35.1 ± 12.9 years [range 18 to 71 years] for females). ECGs were analyzed with the widely used University of Glasgow ECG Analysis Program (7). Average beats were calculated automatically and reviewed manually. STj amplitude was measured in the inferior limb (II, III, and aVF), lateral limb (I and aVL), and lateral precordial (V4, V5, and V6) leads using the program.

Different interpretations of “J-point elevation” were considered and classified as shown in Figure 1. In the following definitions, the amplitude of the peak of an end QRS notch is denoted pkQRSn and the amplitude at the onset of a QRS slur is denoted onQRSs. In this study, neither of these is regarded as QRS end (STj).

- Type 1 pkQRSn ≥0.1 mV and STj ≥0.1 mV and ST-segment upward sloping.
- Type 2 pkQRSn ≥0.1 mV and STj <0.1 mV.
- Type 3 onQRSs ≥0.1 mV and STj ≥0.1 mV and ST-segment upward sloping.
- Type 4 onQRSs ≥0.1 mV and STj <0.1 mV.
- Type 5 No QRS notching or slurring and STj ≥0.1 mV and ST-segment upward sloping.

The presence of these patterns in 2 contiguous inferior or lateral leads was evaluated manually by 2 observers, who used the automated STj measurement. V1 to V3 were excluded from the analysis. A mixed type was defined by a mixture of types 1 to 4 with STj not exceeding 0.1 mV in 2 contiguous leads (i.e., a combination of types 1 and 3 was not part of the mixed group).

All variations of J-point elevation in 2 contiguous leads other than V1 to V3 occurred in 438 (29.3%) subjects. Table 1 shows the prevalence of J-point elevation. It was most prevalent in the inferior leads whereas type 4 was the most frequently occurring. J-point elevation was more prevalent in inferior than in lateral limb and precordial leads (p < 0.0001).

Across all age groups, J-point elevation patterns were more prevalent in males (35.0% [301 of 859]) than females (21.5% [137

Figure 1
Classification of Waveforms With J-Point Elevation on the 12-Lead Resting ECG

ECG = electrocardiogram; onQRSs = amplitude at the onset of a QRS slur; pkQRSn = amplitude of the peak of an end QRS notch; STj = ST junction.
of 637]) (p < 0.0001). This pattern persisted even when considering only those with associated ST-segment elevation: 0.5% (3 of 637) of females, 3.3% (28 of 859) of males (p < 0.0001). In male patients, there was a trend toward a lower prevalence of J-point elevation patterns with increasing age. In female patients, the reverse was true, although those with associated ST-segment elevation were present only in the group aged 18 to 29 years.

Our results demonstrate that the prevalence of what has been termed J-point elevation differs significantly according to the inclusion (2.1%) or exclusion (29.3%) of STj elevation as defined in this study. These data are strikingly similar to the results of Uberoi et al. (8) in a slightly older VA population. Previous reports on early repolarization that included ST-segment elevation as a criterion found similar figures of 1% to 5% in the population (e.g.,2). However, recent reports (e.g., 3) found a larger proportion of healthy control subjects, ranging from 3% to 33%, manifesting end-QRS notching or slurring, which compares with our 29.3% with all types of J-point elevation.

The prognostic value of J-point elevation in an apparently healthy individual remains uncertain, as a significant proportion of healthy control subjects from various studies exhibit this morphology. Our results suggest that the exclusion of STj elevation from J-point elevation may further decrease its specificity in healthy individuals.

We have presented data on the prevalence of J-point elevation with or without STj elevation in an apparently healthy white population. These results have implications for studies looking at the significance of J-point elevation and/or what is currently termed early repolarization. In short, agreed definitions of J-point elevation, STj, and ultimately what has been termed malignant early repolarization (4) are required.

Sijie Jacob Heng, BSc, MBChB
Elaine N. Clark, MA
*Peter W. Macfarlane, DSc

*Electrocardiology Section
Level 1, University Tower
Royal Infirmary
10 Alexandra Parade
Glasgow G31 2ER
United Kingdom
E-mail: peter.macfarlane@glasgow.ac.uk

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REFERENCES


Potential Treatment Influences and Mechanisms Related to Asymmetric Dimethylarginine Control in Heart Failure

We found the fascinating work presented by Shao et al. (1) to be particularly compelling, as it highlights the physiological impact—and potential clinical importance—of the endogenous nitric oxide synthase inhibitor asymmetric dimethylarginine (ADMA) and dimethylarginine dimethylaminohydrolases (DDAHs) in patients with congestive heart failure. Ongoing research has provided ample evidence for the prognostic value of ADMA in the setting of cardiovascular disease, and the enormous breadth of disorders with endothelial dysfunction as a central component suggests a prominent role for ADMA in vascular disease progression. By scrutinizing ADMA production and metabolism paralleling vascular dysfunction with subsequent heart failure, Shao et al. underscore the role of arginine dysregulation and elevated ADMA in cardiovascular disease.

There are a few observations in their study (1), however, that warrant further scrutiny. The authors report a significant treatment difference between those with congestive heart failure and those with advanced decompensated heart failure; the former exhibits high angiotensin-converting enzyme inhibitor (ACEI) and/or angiotensin receptor