European Heart Journal - Cardiovascular Imaging (2020) **21**, 1246–1247
doi:10.1093/ehjci/jeaa186

EDITORIAL

Electro-mechanics or mechano-electrics, an intricate interplay

Kristina H. Haugaa [1,2]* and Ida S. Leren [3]

[1]Department of Cardiology, Oslo University Hospital, Rikshospitalet, PO Box 4950 Nydalen, NO-0424 Oslo, Norway; [2]Institute for Clinical Medicine, University of Oslo, Boks 1072 Blindern, 0316 Oslo, Norway; and [3]Department of Internal Medicine, Diakonhjemmet Hospital, Diakonveien 12, 0370 Oslo, Norway

Online publish-ahead-of-print 24 June 2020

This editorial refers to 'Left ventricular myocardial deformation pattern, mechanical dispersion and their relation with ECG markers in the large population-based STANISLAS cohort: insights into electro-mechanical coupling' by M. Verdugo-Marchese *et al.*, pp. 1237–1245.

Excitation–contraction coupling, describing how electrical signals translate to mechanical activity, is extensively studied both in animal models and in patients with electrical diseases, cardiomyopathies, and heart failure. However, translation to clinical practice is not straightforward. Lessons learnt from imaging studies in cardiac resynchronization therapy (CRT) candidates have highlighted that understanding the field of electro-mechanical interactions is complicated.

Typical measures of myocardial electrical delay and inhomogeneity are increased QRS duration and QT duration on electrocardiogram (ECG), representing delay in intraventricular conduction and electrical repolarization. Prolonged QRS duration affects the electro-mechanical interplay, as clearly illustrated in patients with left bundle branch block and echocardiographic finding of dyssynchronous contractions. Strain echocardiography is superior in detecting regional differences in contraction magnitude and timing, and can accurately visualize dyssynchrony in ventricular segments.[1] Mechanical dispersion is a measure of inhomogeneous myocardial contractions and is increasingly recognized as a marker of ventricular arrhythmias.[2] Improving electrical dyssynchrony by CRT improves mechanical synchrony in patients responding to this therapy and thereby increase the systolic cardiac function. Unfortunately, attempts to use echocardiography to select patients for CRT has failed in large, randomized trials despite the initial optimism and promise of this technique.[3,4]

Prolonged QT duration on ECG also translate to mechanical consequences. Previous studies have indicated that patients with long QT syndrome (LQTS), an inherited electrical disorder with increased risk of ventricular arrhythmias, have mechanical alterations detectable by strain echocardiography.[5] These patients have prolonged contraction duration and increased mechanical dispersion, reflecting mechanical consequences of electrical disease, and subsequent increased risk of ventricular arrhythmias.[6] Furthermore, this assumed purely electrical disorder leads to altered myocardial relaxation, influencing systolic and diastolic cardiac function.[7]

The full spectrum of electro-mechanical interactions in healthy individuals with normal cardiac function is less extensively studied. In this issue of the *European Heart Journal – Cardiovascular Imaging*, Verdugo-Marchese *et al.* add to existing knowledge by presenting electro-mechanical data from a large French cohort of healthy individuals.[8] The authors compared the electrical parameters QRS and QTc duration with strain echocardiographic data including left ventricular function by global longitudinal strain (GLS) and mechanical dispersion. The individuals with the longest QRS duration had the lowest GLS, probably reflecting non-optimal contractions induced by electrical delay. Mechanical dispersion was not affected by longer QRS duration, likely because QRS duration was still within normal and physiological ranges. In contrast, longer QTc was associated with increased mechanical dispersion. The authors concluded that physiological variability in QRS and QT duration were associated with subtle, but significant changes in myocardial deformation patterns, which may improve our understanding of electro-mechanical coupling in the healthy heart.

This study adds to previous knowledge by reporting electro-mechanics in a large number of healthy subjects. The results largely support previous studies in patients with long QT syndrome (LQTS) showing that prolonged QT duration increase mechanical dispersion.[5–7] This finding is also in accordance with the fact that both prolonged QT and increased mechanical dispersion are markers of increased risk of ventricular arrhythmias. The current study reports better GLS values in the quartile of patients with longest QT duration. This is in contrast to previous reports from LQTS patients, showing worse GLS in those with longest QTc.[7] However, the QT

The opinions expressed in this article are not necessarily those of the Editors of *EHJCI*, the European Heart Rhythm Association or the European Society of Cardiology.

* Corresponding author. Tel: +47 2 3071393; Fax: +47 2 3073530. E-mail: kristina.haugaa@medisin.uio.no

intervals in the current study on healthy individuals were within normal range. One may hypothesize that the optimal QT interval needed for maximum GLS values lies in the upper normal limit, and that both longer and shorter QT duration will result in worse GLS. The optimal interaction between electrical and mechanical systole has been studied previously[9] showing that the end of the ECG T wave should coincide with aortic valve closure in healthy individuals. Any deviation from this rule, as seen in patients with long QT syndrome and short QT syndrome, increase arrhythmic risk and probably leads to non-optimal contractions and GLS. It is important to emphasize that the data presented in the current study are from healthy individuals, and consequently differences will be small as measurements are within normal limits. This will inevitably result in less variability in the results. The clinical value of these data may seem hard to interpret and, certainly, the link to ventricular arrhythmias cannot be explored. Nevertheless, the data from Verdugo-Marchese *et al*. come from a large cohort, which increase the reliability of data and provide new insight to this intricate field.

The individuals in the current study were relatively young (49 years). Previous age-specific studies in healthy individuals have indicated an increase in mechanical dispersion by age.[10] Whether electro-mechanical interactions change by age, or have clinical implications may be investigated in future studies.

Elucidating electro-mechanical interactions in healthy individuals is important to improve understanding of the delicate interplay between electrical signals and myocardial contractions and may add information on how this intricate system is meant to work. In CRT treatment, the aim is to re-establish electrical synchronization and thereby improve mechanical dyssynchrony and cardiac function. Prediction of response is notoriously difficult, and the search for prognostic parameters beyond QRS duration and left ventricular ejection fraction is extensive. Better understanding of the healthy electro-mechanical interplay may be an important stepping-stone for this field of research.

Conflict of interest: K.H.H. has licensed a patent on mechanical dispersion to GE. The other author declared no conflict of interest.

References

1. Edvardsen T, Haugaa KH. Imaging assessment of ventricular mechanics. *Heart* 2011;**97**:1349–56.
2. Haugaa KH, Grenne BL, Eek CH, Ersbøll M, Valeur N, Svendsen JH *et al*. Strain echocardiography improves risk prediction of ventricular arrhythmias after myocardial infarction. *JACC Cardiovasc Imaging* 2013;**6**:841–50.
3. Chung ES, Leon AR, Tavazzi L, Sun JP, Nihoyannopoulos P, Merlino J *et al*. Results of the predictors of response to CRT (PROSPECT) trial. *Circulation* 2008; **117**:2608–16.
4. Ruschitzka F, Abraham WT, Singh JP, Bax JJ, Borer JS, Brugada J *et al*. Cardiac-resynchronization therapy in heart failure with a narrow QRS complex. *N Engl J Med* 2013;**369**:1395–405.
5. Haugaa KH, Amlie JP, Berge KE, Leren TP, Smiseth OA, Edvardsen T. Transmural differences in myocardial contraction in long-QT syndrome: mechanical consequences of ion channel dysfunction. *Circulation* 2010;**122**:1355–63.
6. Haugaa KH, Edvardsen T, Leren TP, Gran JM, Smiseth OA, Amlie JP. Left ventricular mechanical dispersion by tissue Doppler imaging: a novel approach for identifying high-risk individuals with long QT syndrome. *Eur Heart J* 2008;**30**: 330–7.
7. Leren IS, Hasselberg NE, Saberniak J, Haland TF, Kongsgard E, Smiseth OA *et al*. Cardiac mechanical alterations and genotype specific differences in subjects with long QT syndrome. *JACC Cardiovasc Imaging* 2015;**8**:501–10.
8. Verdugo-Marchese M, Coiro S, Selton-Suty C, Kobayashi M, Bozec E, Lamiral Z *et al*. Left ventricular myocardial deformation pattern, mechanical dispersion and their relation with ECG markers in the large population-based STANISLAS cohort: insights into electro-mechanical coupling. *Eur Heart J Cardiovasc Imaging* 2020;**21**:1237–45.
9. ter Bekke RM, Haugaa KH, van den Wijngaard A, Bos JM, Ackerman MJ, Edvardsen T *et al*. Electromechanical window negativity in genotyped long-QT syndrome patients: relation to arrhythmia risk. *Eur Heart J* 2015;**36**:179–86.
10. Rodriguez-Zanella H, Haugaa K, Boccalini F, Secco E, Edvardsen T, Badano LP *et al*. Physiological determinants of left ventricular mechanical dispersion: a 2-dimensional speckle tracking echocardiographic study in healthy volunteers. *JACC Cardiovasc Imaging* 2018;**11**:650–1.