JACC: CARDIOVASCULAR IMAGING, VOL. 10, NO. 4, 2017
APRIL 2017:486–99

of concentric LV hypertrophy, and reduced LV longitudinal strain compared with other players (nonlinemen) (1).

We believe that several points of this investigation deserve to be commented on and clarified. The interesting finding is that heart rate (HR) in linemen did not change during the season, whereas it significantly decreased in nonlinemen. Recently, Lo Iudice et al. (2) reported a strong negative association between HR and LV mechanics in top-level endurance athletes. This could partly explain the results of the current study and lower LV longitudinal strain in linemen. However, Lin et al. (1) showed that HR is not independently associated with LV longitudinal strain, although HR correlated both with LV longitudinal strain before and after the season. From a statistical point of view, the multivariate regression models used in the study have some limitations. First, it is not clear why Lin et al. (1) in the same statistical model included parameters with high collinearity such as systolic and diastolic BP; body mass index and body surface area; and LV mass, LV wall thickness, and LV relative wall thickness. This could be the reason why HR was not independently associated with LV longitudinal strain in the whole study population. Second, inclusion of 8 independent variables in one statistical model in a small study population like this might not be the best solution. Suggested changes of these statistical models possibly would result with other independent predictors of LV longitudinal strain in this population.

The other question that arises is why linemen have higher HR than nonlinemen do. In the previous paper of the same research group the difference in HR was even more obvious (3). Namely, HR in linemen was even higher after the season, whereas trend of HR reduction after season remained in nonlinemen (3).

The intrinsic heart rate mechanisms and vagal tone are predominant over sympathetic tone in professional athletes. However, linemen are exposed to high-volume endurance training during the season, but still have the same (1) or even higher HR (3) than other players do. Is their parasympathetic nervous system deprived or is their sympathetic nervous system excited more than in their teammates? Increased sympathetic activity is associated with elevated BP and increased LV mass as well as higher incidence of LV concentric hypertrophy (4). This might partly explain higher BP in linemen, higher increase in LV mass index in linemen, and higher incidence of concentric LV hypertrophy among linemen (1).

The Lin et al. (1) study opens many topics that remain to be resolved in future investigations. Probably the most important questions concern the cause of LV remodeling and hypertension in linemen players, the reversibility of these changes (complete or incomplete), and the influence of this adaptation on the outcome.

Marijana Tadic, MD, PhD*
Cesare Cuspidi, MD
Branislava Ivanovic, MD, PhD
*Department of Cardiology
Charité-University-Medicine Berlin
Campus Virchow Klinikum (CVK)
Augustenburger Platz 1
13353 Berlin
Germany
E-mail: marijana_tadic@hotmail.com

http://dx.doi.org/10.1016/j.jcmg.2016.12.015

Please note: The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

REFERENCES

1. Lin J, Wang F, Weiner RB, et al. Blood pressure and LV remodeling among American-style football players. J Am Coll Cardiol Img 2016;9:1367–76.

2. Lo Iudice F, Petitto M, Ferrone M, et al. Determinants of myocardial mechanics in top-level endurance athletes: three-dimensional speckle tracking evaluation. Eur Heart J Cardiovasc Imaging 2016 Jun 19 [E-pub ahead of print].

3. Weiner RB, Wang F, Isaacs SK, et al. Blood pressure and left ventricular hypertrophy during American-style football participation. Circulation 2013; 128:524–31.

4. Strand AH, Gudmundsdottir H, Os I, et al. Arterial plasma noradrenaline predicts left ventricular mass independently of blood pressure and body build in men who develop hypertension over 20 years. J Hypertens 2006;24: 905–13.

THE AUTHORS REPLY:

We appreciate the interest of Dr. Tadic and colleagues in our work, specifically our recent paper documenting left ventricular (LV) mechanics among American-style football (ASF) players (1), and the chance to address their inquiries. First, the issue of heart rate and its impact on longitudinal strain has been raised. Specifically, it has been suggested that heart rate may be an explanatory mechanism underlying the observed reductions in LV longitudinal strain among ASF linemen. We are keenly aware of the inverse relationship between heart rate and LV strain, a phenomenon we observed among competitive rowers a decade ago (2). The heart rate-strain correlations in the study were driven exclusively by the nonlineman ASF athletes who engage in considerable isotonic training and experience expected reductions in heart rate. Thus, some of the LV strain increase noted in

this group may indeed be related to intrinsic changes in heart rate. The same cannot be true for the ASF lineman. This subgroup of athletes had no change in resting heart rate, an indirect marker of unchanged cardiorespiratory fitness, and developed relative hypertension and concentric LV hypertrophy. Thus, the observed reductions in LV longitudinal strain are in no way analogous to the myocardial mechanics we and others have described among endurance athletes but are more consistent with early-stage hypertensive remodeling. Second, there were several issues regarding our statistical analyses, including variable selection and the number of independent variables used in our regression modeling. Regarding variable selection, specifically the potential for high collinearity, it is important to consider the heterogeneous nature of the study cohort. ASF populations comprise athletes spanning the full spectrums of body size, sport physiology, and LV remodeling, making collinearity less of an issue than it would be in a more homogenous study population. While opinions regarding the appropriate number of independent variables for use in a multivariate analysis differ, we chose a standard and rather conventional approach in which we limited variable inclusion to no more than 1 per 10 subjects. Finally, potential mechanisms underlying higher heart rates among linemen versus nonlinemen are considered. We concur that ASF linemen may very well have an autonomic balance favoring sympathetic over parasympathetic activity, particularly in comparison to nonlinemen, but think it is unlikely that this is driven by a deprived parasympathetic nervous system. The statement that "linemen are exposed to high-volume endurance training during the season, but have still the same or even higher heart rates than other players do" is inaccurate, and we encourage Tadic and colleagues to rethink field position–specific physiology underlying the ASF participation. In reality, linemen get very little endurance training but rather engage repetitively in activities characterized by nearly pure isometric physiology. In addition, ASF linemen often concomitantly gain weight, eat high-sodium diets, use high quantities of nonsteroidal anti-inflammatories, and experience increases in resting blood pressure. One need not complicate the pathophysiologic picture by invoking intrinsic deficiencies in autonomic nervous system function when these simple physiological explanations are such low-hanging fruit.

Jeffrey Lin, MD*
Francis Wang, MD
Rory B. Weiner, MD
James R. DeLuca, BA
Meagan M. Wasfy, MD
Brant Berkstresser, MS, ATC
Gregory D. Lewis, MD
Adolph M. Hutter, Jr., MD
Michael H. Picard, MD
Aaron L. Baggish, MD

*Cardiovascular Performance Program
Massachusetts General Hospital
55 Fruit Street
Boston, Massachusetts
E-mail: jlin7@partners.org

http://dx.doi.org/10.1016/j.jcmg.2017.01.011

Please note: The original manuscript was supported by research grants from the National Institutes of Health/National Heart Lung & Blood Institute (HL RO1 125869 to Dr. Baggish) and the American Heart Association (FTF220328 to Dr. Baggish). Neither funding source played any role in the design and conduct of the study, in the collection, analysis, and interpretation of the data, and in the preparation, review, or approval of the manuscript. The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

REFERENCES

1. Lin J, Wang F, Weiner RB, et al. Blood pressure and LV remodeling among American-style football players. J Am Coll Cardiol Img 2016;9:1367–76.

2. Baggish AL, Yared K, Wang F, et al. The impact of endurance exercise training on left ventricular systolic mechanics. Am J Physiol Heart Circ Physiol 2008;295:H1109–16.

FFR-Derived From Coronary CT Angiography Using Workstation-Based Approaches

Computed tomography (CT) angiography–derived fractional flow reserve (FFR) is a potentially disruptive technology in its ability to noninvasively predict coronary FFR values. Promising initial results have engendered vigorous discussions about this particular image analysis pathway as a gatekeeper to the catheterization laboratory. There is evidence to suggest that CT-derived FFR can significantly improve the detection of obstructive coronary artery disease (CAD) and avoid unnecessary invasive testing, with beneficial economic and quality-of-life outcomes.

Hence, we have read the article by Ko et al. (1) with great interest. As discussed by the authors, one major limitation of the currently commercialized, Food and Drug Administration–approved solution for CT-based FFR derivation consists in the need for transferring patient image data to an external core laboratory. The calculation and transfer process remains time consuming (around 1 to 4 h) and is less suitable for prompt clinical decision making, which obviously limits the practical utility. As a result, less computationally demanding approaches residing on a regular workstation have been developed and implemented, involving, for instance, reduced-order computational