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Case Report

Acute response and chronic stimulus for cardiac structural and functional adaptation in a professional boxer

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The individual response to acute and chronic changes in cardiac structure and function to intense exercise training is not fully understood and therefore evidence in this setting may help to improve the timing and interpretation of pre-participation cardiac screening. The following case report highlights an acute increase in right ventricular (RV) size and a reduction in left ventricular (LV) basal radial function with concomitant increase at the mid-level in response to a week's increase in training volume in a professional boxer. These adaptations settle by the second week; however, chronic physiological adaptation occurs over a 12-week period. Electrocardiographic findings demonstrate an acute lateral T-wave inversion at 1 week, which revert to baseline for the duration of training. It appears that a change in training intensity and volume generates an acute response within the RV that acts as a stimulus for chronic adaptation in this professional boxer.

INTRODUCTION

Pre-participation cardiac screening of the athlete using both electrocardiography (ECG) and echocardiography is important in reducing the rate of sudden cardiac death from undiagnosed inherited cardiac disease [1], yet the influence of acute and chronic intense training on cardiac structure, function and ECG findings is not fully understood. Individual response to training may provide important information for the screening practitioner with regard to the timing and interpretation of these investigatory findings.

CASE REPORT

A 32-year-old Caucasian professional boxer with no history of cardiovascular disease was training for an average of 10 h per week prior to the start of an intensive 3 months preparation (average of 32 h per week) leading up to an international fight. 12-Lead ECG and echocardiographic data were acquired at the same time of day (early morning prior to training) at periodic intervals (baseline prior to increase in training intensity

and then at Weeks 1–3 of training followed by a final data collection prior to the fight at 12 weeks). Echocardiography included standard 2D, Doppler and tissue Doppler imaging (TDI) as well as myocardial speckle tracking (MST) derived indices of deformation from the left and right ventricles. Dual-energy X-ray absorptiometry (DXA) scans were also acquired as well as a full account of training and nutritional information. The athlete typically trained two to three times per day 5–6 days per week with training sessions consisting of fasted morning steady-state (i.e. 70% $\text{VO}_{2\text{max}}$) and/or high-intensity intermittent (encompassing repeated 3-min intervals at 90% $\text{VO}_{2\text{max}}$) running (8–9 a.m.), boxing-specific training (1–4 p.m.) and strength and conditioning (7–8 p.m.). During the 12-week-training period, the athlete adhered to a daily diet approximately equivalent to resting metabolic rate (2100 kcal: 2.5 g/kg body mass carbohydrate, 2 g/kg protein, 1 g/kg fat) and total body mass loss equated to 7.8 kg. DXA scans were performed in the morning fasted state at regular 3–4 week intervals and data confirmed significant alterations to body composition (e.g. at Weeks 1 and 11, percent body fat equated to 13.9 and 8.8%, respectively).

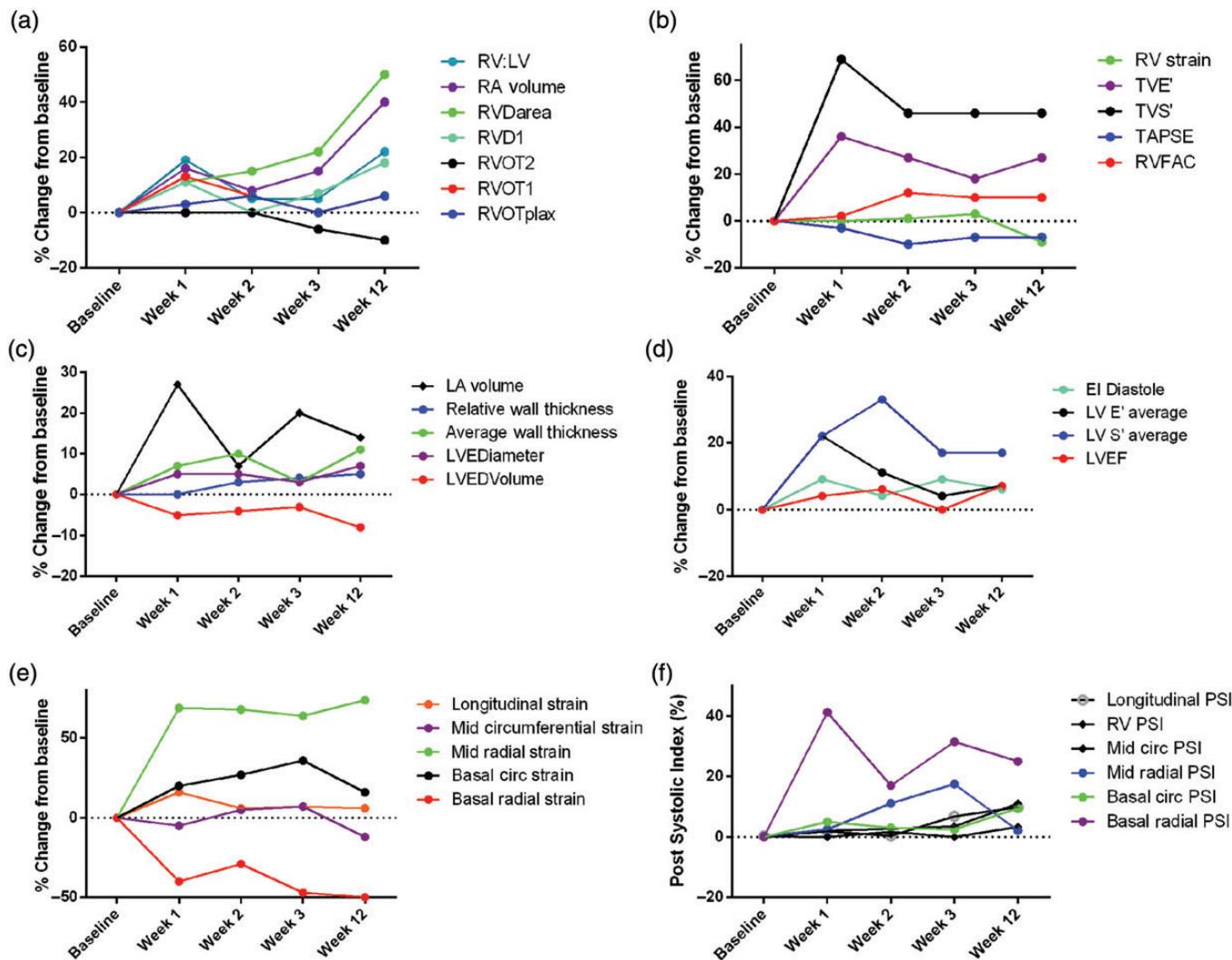


Figure 1: Echocardiographic findings at baseline, Week 1–3 and Week 12 of an intensive training programme. (a) RV structural parameters, (b) RV functional parameters, (c) LV structural parameters, (d) LV functional parameters, (e) LV strain parameters, (f) LV post-systolic index.

Following the first week of training, an acute response was observed with marked right ventricular (RV) outflow/inflow and left atrial (LA) enlargement with a parallel negative impact on basal left ventricular (LV) radial strain and an increased post-systolic index. A concomitant increase in LV mid-wall radial function was observed. RV function was unchanged when measured using strain while TDI velocities increased. The baseline 12-lead ECG was unremarkable, however, following the first week of training new lateral T-wave inversion in leads V5 and V6 were noted. Echocardiographic data from Week 2 of training provided evidence of RV structure and function returning closer to baseline with a reduction in LV radial post-systolic shortening. The RV inflow diameters and LA volume were increased over baseline at the final data collection point while LV structure and function was less effected with exception of the basal segments which continued with depressed radial strain/increased post-systolic index and increased mid-radial strain compared with baseline (see Fig. 1).

The lateral T-wave changes subsequently reverted to normal for the remainder of the training period (see Fig. 2).

DISCUSSION

The acute effects of prolonged strenuous exercise on RV function have previously been documented [2, 3] with evidence of dilatation and dysfunction. This phenomenon has been attributed to a relatively higher increase in exercise-induced RV wall stress [4] which appears transient in nature with a rapid resolution [5]. In fitting with the previous work, the findings in this case highlight marked acute changes in RV size from both the outflow and inflow (main body of the RV) following a transition from moderate training (10 h per week) to intensive dynamic and static training (32 h per week) while uniquely highlighting an impact on LV radial function. It is well established that RV pressure/volume overload impacts on septal



Figure 2: Resting 12-lead electrocardiograms at Week 1 and 2 of an intensive training programme. Circles highlight T-wave changes in the lateral leads V5 and V6.

geometry with post-systolic movement [6]; however, this has never been highlighted following an acute change in training intensity. The reduction in basal strain appears to be compensated by an increase at mid-level and; therefore, overall LV function is not compromised. The 12-lead ECG changes observed following the first week are intriguing with the acute development of lateral T-wave inversion. It is well established that athletic conditioning induces modifications which can include T-wave inversion, however; the presence of lateral T-wave inversion is considered pathological [7]. To the best of our knowledge, this is the first case to report this finding and in association with the marked structural and mechanical changes it is clear that this finding requires further exploration.

There is strong evidence to support chronic eccentric hypertrophy of the RV in athletes who engage in a high volume of combination (dynamic and static) training such as cyclists, rowers and boxers [8–10] with the RV inflow dimensions having the greatest degree of remodelling. This case supports this finding over a short period of time (3 months) and furthermore highlights the transition from an acute response to gradual chronic adaptation. Interestingly, the LV functional change in mechanics, a dominant mid-level radial function, persist throughout the training period possibly highlighting a shift in cardiac functional adaptation which prompts further

exploration. The lateral T-wave inversion was transient as all subsequent recordings were normal. This phenomenon is difficult to explain particularly with the lack of any LV structural or functional adaptation including no evidence of hypertrabeculation. The impact of an acute bout of exercise on electrical repolarization has received some attention with evidence of changes to the QT interval and T-wave axis (vector loops) [11] post-exercise with alterations persisting 1 day post-exercise. There is also a growing evidence base supporting the association of early repolarization to fitness levels as documented in a longitudinal training study [12]. Although both these studies highlight a transient and physiological alteration in ventricular repolarization in response to exercise training, there is no literary evidence of a transient T-wave inversion in the presence of a sustained training stimulus, as demonstrated in this case report. It is unlikely that the early repolarization data offers significant insight into the mechanisms for T-wave inversion in this setting; however, the mechanisms underpinning changes to the QT interval and T-wave dispersion following prolonged exercise may be more relevant. Although transient changes in ion-channel function and (or) blood electrolyte content may play a role there is some speculation that the electrical alterations may be secondary to the mechanical stress and interaction between the right and left ventricles

[10]. In view of our additional mechanical findings, this case report supports this theory and furthermore highlights the acute impact of a change in training intensity and volume on the electromechanical components of cardiac structure and function.

CONCLUSION

This case highlights an acute response to a significant change in training volume with RV dilatation and a shift in LV mechanical function, likely as a consequence of parallel impact from the RV. Acute changes return close to baseline, however, maintenance of the training volume results in chronic structural adaptation of the RV with a persistent change in LV radial mechanics. These findings may have an impact on pre-participation screening generating a particular consideration to acute and chronic adaptation of cardiac structure, function and ECG findings.

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