BJSM Online First, published on October 3, 2014 as 10.1136/bjsports-2014-093895

The right ventricle following prolonged endurance exercise: are we overlooking the more important side of the heart? A meta-analysis

Adrian D Elliott,[1] Andre La Gerche[2]

[1]School of Medical Sciences, University of Adelaide, Adelaide, South Australia, Australia
[2]St Vincent's Department of Medicine, University of Melbourne, Melbourne, Victoria, Australia

Correspondence to
Adrian D Elliott, School of Medical Sciences, University of Adelaide, Adelaide, SA 5005, Australia; adrian.elliott@adelaide.edu.au

Accepted 26 August 2014

ABSTRACT

Aims Prolonged endurance exercise is associated with elevated biomarkers associated with myocardial damage and modest evidence of left ventricular (LV) dysfunction. Recent studies have reported more profound effects on right ventricular (RV) function following endurance exercise. We performed a meta-analysis of studies reporting RV function pre-endurance and postendurance exercise.

Methods We performed a search of peer-reviewed studies with the criteria for inclusion in the analysis being (1) healthy adult participants; (2) studies examining RV function following an event of at least 90 min duration; (3) studies reporting RV fractional area change (RVFAC), RV strain (S), RV ejection fraction (RVEF) or tricuspid annular plane systolic excursion (TAPSE) and (4) studies evaluating RV function immediately (<1 h) following exercise.

Results Fourteen studies were included with 329 participants. A random-effects meta-analysis revealed significant impairments of RV function when assessed by RVFAC (weighted mean difference (WMD) −5.78%, 95% CI −7.09% to −4.46%), S (WMD 3.71%, 95% CI 2.79% to 4.63%), RVEF (WMD −7.05%, 95% CI −12.3% to −1.8%) and TAPSE (WMD −4.77 mm, 95% CI −8.3 to −1.24 mm). Modest RV dilation was evident in studies reporting RV systolic area postexercise (WMD 1.79 cm^2, 95% CI 0.5 to 3.08 cm^2). In contrast, no postexercise changes in LV systolic function (expressed as LVFAC or LVEF) were observed in the included studies (standardised mean difference 0.03%, 95% CI −0.13% to 0.18%).

Conclusions Intense prolonged exercise is associated with a measurable reduction in RV function while LV function is relatively unaffected. Future studies should examine the potential clinical consequences of repeated prolonged endurance exercise on the right ventricle.

INTRODUCTION

Prolonged endurance exercise is associated with elevations in cardiac biomarkers,[1] but only modest transient depression in left ventricular (LV) function.[2] These changes have been demonstrated following endurance events of varying distance and duration. A majority of research has focused on the left ventricle (LV) and common comorbidities such as coronary vessel disease.[2–4] The right ventricle has been largely overlooked.

Early studies into the phenomenon of 'cardiac fatigue' showed that LV ejection fraction (LVEF) and diastolic filling parameters were mildly impaired in the immediate postexercise period following long-distance triathlon[5–8] and ultra-endurance (>100 km) running.[9 10] However, a number of studies have failed to identify any change in LV measures[11–13] and no study has demonstrated an association between changes in LV function and other markers of myocardial injury.

Changes in right ventricular (RV) function may be significant clinically and may provide a better insight into exercise-induced alterations to myocardial function. However, the magnitude and consistency of studies reporting postendurance exercise changes in RV function have not previously been interrogated. The clinical relevance of postexercise changes in RV function is highlighted by a potential link with chronic RV remodeling and a propensity for ventricular arrhythmias.[14 15]

Assessing RV function is complex and lacks a single, commonly accepted parameter.[16] Evaluation of the RV following endurance exercise typically focused on observing RV fractional area change (RVFAC) and tricuspid annular plane systolic excursion (TAPSE),[17 18] but recent means of assessing systolic function have included strain and strain rate.[13 19] Cardiac MR (CMR) imaging has also been recently employed and enables accurate quantification of biventricular volumes.[4 20] Despite recent advances, variability in the measures used to quantify RV function makes it challenging to draw unifying conclusions as to the extent to which the RV is affected by prolonged endurance exercise.

Owing to the logistical constraints of trying to assess multiple athletes in the immediate postrace window, previous studies have been limited to relatively modest cohort sizes. We report a meta-analysis of all existing studies examining RV function both pre-endurance and postendurance exercise. We hypothesised that an analysis of studies that have assessed RV function in the context of endurance exercise would provide consistent evidence for exercise-induced myocardial impairment that predominantly or exclusively affects the RV.

METHODS

Search strategy and inclusion criteria

We performed a search of all peer-reviewed studies published prior to February 2014 in the English language. We searched PubMed and MEDLINE databases using keywords from an initial limited search of the literature. Keywords and phrases used in the search included 'right ventricular function', 'cardiac fatigue', 'ventricular dysfunction', 'ventricular damage' and 'prolonged exercise', 'endurance exercise', 'marathon', or 'triathlon'. Reference

To cite: Elliott AD, La Gerche A. *Br J Sports Med* Published Online First: [*please include* Day Month Year] doi:10.1136/bjsports-2014-093895

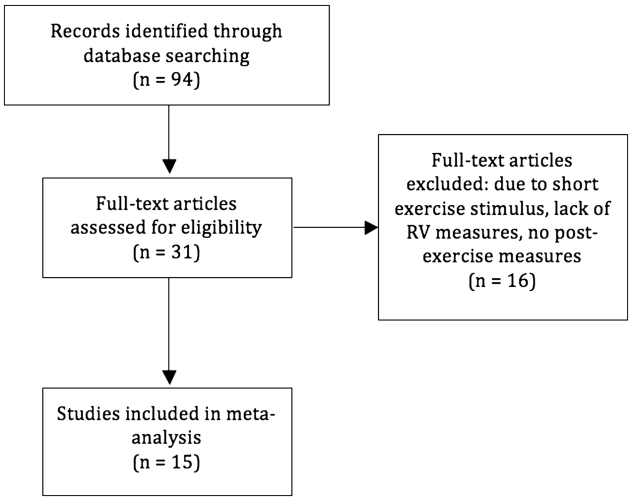

Figure 1 Flow diagram depicting study search and exclusion process.

lists from published papers were searched for any additional studies missed in the online search.

The inclusion criteria for the meta-analysis were (1) studies recruiting healthy participants aged 18 years and over; (2) studies examining RV function following an exercise event of at least 90 min in duration; (3) studies assessing RV function using two-dimensional (2D), 2D guided M-mode, or 3D echocardiography, tissue Doppler or CMR; (4) studies reporting either RVFAC, RV strain (S), RV ejection fraction (RVEF) or TAPSE; and (5) studies recording RV function immediately (<1 h) following endurance exercise.

Study review and data extraction

The search strategy identified 96 articles, of which 31 were retrieved in full-text. After further review, 15 studies were deemed to meet the inclusion criteria (figure 1). From each included study, the age and gender of participants, total exercise time and the modality of exercise were recorded while measures of RV function and chamber size were extracted where reported. Where the data were reported as subgroups, the average mean value across groups was calculated and a common SD determined using pooled variances.

As indicators of RV systolic function, RVFACs, RVEF, global RV strain (S) and/or TAPSE were included for the analysis of RV function pre-exercise and postexercise. Global strain was determined by averaging segmental strain in studies reporting data per segment. In addition, RV dilation was recorded using RV area during systole and diastole, respectively.

Statistical analysis

A random-effects meta-analysis was used to determine the weighted mean difference (WMD) and 95% CIs of RV measures between pre-exercise and postexercise. All statistical analyses were performed using Review Manager V.5.2 (Cochrane Centre, Copenhagen, Denmark). A forest plot for each outcome was used to portray the differences between pre-exercise and postexercise. Statistical heterogeneity was assessed using the χ^2 and I^2 statistic. For all analyses where statistical heterogeneity was observed and the number of included studies was greater than 10, we performed a subgroup analysis based on exercise duration for further investigation.

RESULTS

Eligible studies

All eligible studies for the meta-analysis are shown in table 1. Fifteen studies, resulting in a total of 354 participants, were included. The mean duration of exercise ranged from 130 to 1472 min. Eleven (73%) studies used running as the exclusive mode of exercise. Two studies (13%) evaluated participants following an ironman triathlon and one study (7%) used a long-distance cycling event. One study included four events ranging from a marathon to ironman triathlon. The mean age of participants ranged from 28 to 55 years. To characterise baseline cardiac structure and function, the pre-exercise end-diastolic area and volume measurements are reported alongside pre-exercise measures of ejection fraction in table 2. In all parameters assessed, mean values were below the upper limits of normal reported for the athlete's heart.[21]

Table 1 Details of included studies

Study	Event	Sample size (M/F)	Mean (SD) duration, min	Mean (SD) age, years
Douglas *et al*[17]	IM triathlon	41 (22/19)	Not reported	38 (10)
Dávila-Román *et al*[22]	161 km UM	14 (14/0)	Not reported	
Oxborough *et al*[23]	Marathon	35 (29/6)	230 (42)	30 (8)
Neilan *et al*[13]	Marathon	60 (41/19)	245 (range 175–355)	41 (11)
Neilan *et al*[24]	Marathon	20 (10/10)	243 (34)	34 (10)
La Gerche *et al*[25]	IM triathlon	27 (20/7)	650 (75)	Median 32 (range 22–54)
Mousavi *et al*[4]	Marathon	14 (8/6)	245 (68)	33 (6)
Banks *et al*[26]	150 min running @ 80% VO_{2max}	18 (12/6)	150	28 (4)
Oxborough *et al*[18]	Marathon	17 (17/0)	209 (19)	33 (6)
Trivax *et al*[27]	Marathon	12 (12/13)	256 (44)	39 (9)
Oxborough *et al*[19]	161 km UM	16 (12/4)	1472 (200)	42 (8)
Oomah *et al*[28]	Half-marathon	15 (7/8)	130 (24)	32 (6)
Karlstedt *et al*[29]	Marathon	25 (21/4)	252 (33)	55 (4)
La Gerche *et al*[30]	1. Marathon, n=7; 2. Half-IM triathlon, n=11; 3. Alpine cycling, n=9; 4. IM triathlon, n=13	40 (36/4)	1. 179 (30) 2. 324 (25) 3. 485 (43) 4. 652 (76)	37(8)
Claessen *et al*[20]	150 km cycling race	14 (14/0)	Not reported	36 (6)

F, female; IM, ironman; M, male; UM, ultramarathon.

Table 2 Baseline cardiac structure and function reported from pre-exercise data in the included studies

	Structural				Functional	
Study	RVEDA (cm^2)	LVEDA (cm^2)	LVEDV (mL)	RVEDV (mL)	RVEF (%)	LVEF (%)
Douglas *et al*[17]	21.4 (5.3)	37.7 (6.9)				
Dávila-Román *et al*[22]	18.5 (3.6)	28.7 (4)	89.9 (19.9)			56.6
Oxborough *et al*[23]	19.2 (2.2)	36.2 (4.5)				
Neilan *et al*[13]	17 (4)		110 (20)			60 (6)
Neilan *et al*[24]	17 (3)		106 (20)			59 (6)
La Gerche *et al*[25]	28.9 (5.7)					60.4 (4.7)
Mousavi *et al*[4]				160.6 (19.7)		
Banks *et al*[26]			110 (4.5)			56.4 (3.5)
Oxborough *et al*[18]						66 (5.1)
Trivax *et al**[27]			185.1 (32.4)	144 (24.9)	53.6 (7.1)	57.7 (4.1)
Oxborough *et al*[19]	23 (4)		129 (19.2)			65 (4)
Oomah *et al*[28]	13 (4)		115 (17)	125 (26)	59 (4)	64 (3)
La Gerche *et al*[30]			150 (23)	170 (30)	51 (3.6)	56.4 (5.2)
Karlstedt *et al*[29]			114 (12)			63 (4)
Claessen *et al**[20]			229 (20)	237 (26)	60.9 (4.4)	62.9 (2.8)
Normal athlete's heart, male			*180–340*	*200–390*	*40–58*	*41–77*
Normal athlete's heart, female			*140–260*	*150–290*	*40–67*	*44–76*

Normal data taken from Prior and La Gerche.[21] Data are presented as mean (SD). Normal data are presented as a range. Data not reported indicate not available in the original manuscript.
*Indicates studies where measurements were obtained by cardiac magnetic resonance rather than echocardiography. Italics are the summary rows.
LVEDA, left ventricular end-diastolic area; LVEDV, LVED volume; LVEF, left ventricular ejection fraction; RVEDA, right ventricular end-diastolic area; RVEDV, RVED volume; RVEF, right ventricular ejection fraction.

RV systolic function

All included studies incorporated RVFAC, RVEF, global strain or TAPSE as a measure of RV systolic function. RVFAC was the most commonly reported parameter (12 studies, n=312). In five studies, strain was reported (n=154); TAPSE was reported in six studies (n=175), while four studies included RVEF (n=94). Meta-analysis of RVFAC revealed WMD of −6.8% (95% CI −8.95% to −4.66%) from pre-exercise to postexercise (figure 2). Statistical heterogeneity was observed between studies (χ^2=144.76; I^2=92%, p<0.001). The WMD of S from pre-exercise to postexercise was 3.71% (95% CI 2.79% to 4.63%; figure 3). A lack of statistical heterogeneity was observed between studies reporting S following endurance exercise (χ^2=7.26; I^2=45%; p=0.12). For TAPSE, the WMD was −5.52 mm (95% CI−9.07 to −1.97 mm; figure 4), while for RVEF the WMD was −7.05 (95% CI−12.3 to −1.8; figure 5). Statistical heterogeneity was observed for both TASPSE and RVEF (I^2=95, 90% for TAPSE, RVEF, respectively; p<0.001). We did not observe any evidence of publication bias on visual inspection of funnel plots for any outcome (data not shown). Subgroup analysis of RVFAC based on exercise duration revealed similar significant decreases in RVFAC pre-exercise versus post-exercise in the <6 h exercise group and the >6 h group (figure 6). Of all studies, only one reported a postexercise depression in LV function.[19] Meta-analysis of LV systolic function, expressed as either LVEF or LVFAC, revealed no significant difference between pre-exercise and postexercise (standardised mean difference 0.01, 95% CI −0.14 to 0.16).

RV dilation

Eight studies reported pre-exercise versus postexercise changes in RV area. Meta-analysis of RV systolic area revealed a WMD of 1.79 cm^2 (95% CI 0.5 to 3.08). Analysis of RV area during diastole revealed a WMD of 1.36 cm^2 (95% CI −0.08 to 2.81). Statistical heterogeneity was noted for both variables (I^2>75%; p<0.0001).

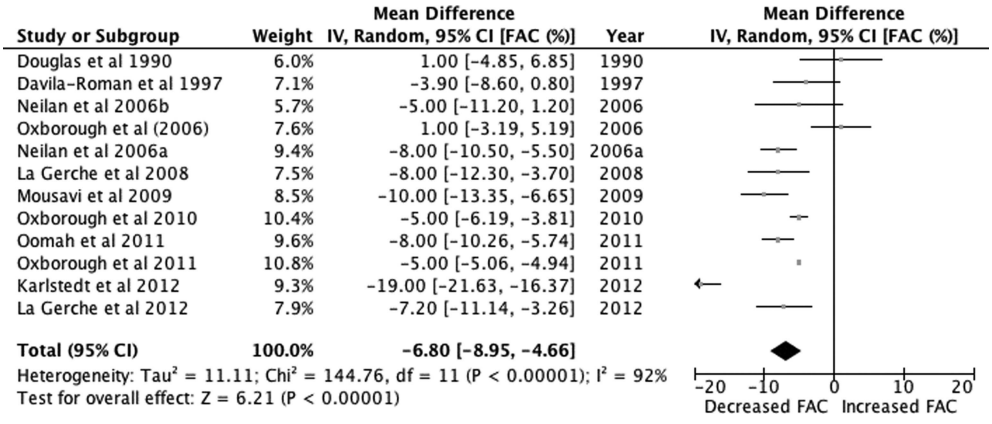

Figure 2 Forest plot comparing right ventricular fractional area change (FAC) pre-endurance versus postendurance exercise. Mean differences are calculated by random-effects meta-analysis and weighted by estimated precision of the effect.

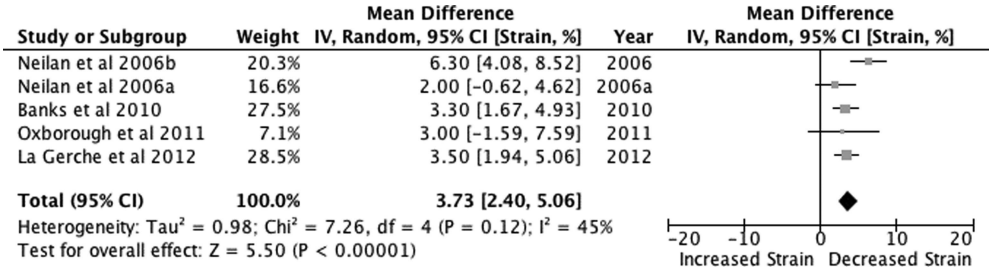

Figure 3 Forest plot comparing right ventricular strain pre-endurance versus postendurance exercise. Mean differences are calculated by random-effects meta-analysis and weighted by estimated precision of the effect.

DISCUSSION

Our meta-analysis of all studies reporting RV function pre-endurance and postendurance exercise indicates that RV function is reduced following endurance exercise despite no apparent alteration to LV function in the same studies. All included measures of RV function showed evidence of a depression in RV systolic function on completion of exercise.

Biological rationale: underpinning mechanism

The cause of a decline in RV function following endurance exercise is unclear; the most likely candidate is the relatively greater increase in load placed on the RV during exercise,[31] potentially making it more susceptible to exercise-induced fatigue or injury. Exercise leads to significant increases in pulmonary artery pressure thus increasing RV end-systolic wall stress.[32–34] The greater work required to sustain a high cardiac output throughout exercise may lead to greater depression in RV function, which persists into the postexercise recovery period. This premise is supported by the methodology of Claessen *et al*[20] in which they studied RV function *during* exercise. As compared with prerace measures, RV functional reserve (the ability to increase RVEF during intense exercise) was attenuated after 150 km of intense cycling. Thus, it may be that the increased wall stress and work of exercise cause relative RV dysfunction, which is most evident under the haemodynamic stressors of exercise.

We observed a trend for increasing RV end-diastolic area following prolonged exercise. This observation supports the view that changes in RV function cannot be solely attributed to postexercise alterations in volume status. Moreover, postexercise elevations in end-diastolic area should improve RV function, according to the Frank-Starling mechanism, assuming maintained RV contractility. Decreased RV strain in the presence of increased end-diastolic areas reported in three studies[4 28 30] suggests a true functional impairment of the RV.

Obtaining objective, quantitative measures of RV function is particularly challenging. Difficulties in imaging the RV are largely due to its geometry, location and trabeculation. Determination of RVFAC is commonly considered to be cumbersome with high interobserver variability.[31] In the studies analysed, RVFAC at baseline ranged from 25%[17] to 51.9%,[23] thus supporting the view of RVFAC as a technique with considerable variability. To further examine the possible causes of heterogeneity between studies for RVFAC, we grouped the studies according to the duration of exercise employed. There were no apparent differences between the decline in RVFAC in studies using exercise of less than or greater than 6 h.

We also included studies where TAPSE, RV strain and/or RVEF were used to represent RV function. In the six studies reporting TAPSE following exercise, a significant reduction was observed. RV strain may be a more representative parameter of true contraction and relaxation. In our analysis, RV strain was reported by five studies and showed evidence of statistical homogeneity, which was absent in the other measures. RV strain decreased significantly from pre-exercise to postexercise, in agreement with other measures of RV function. While strain remains a load-dependent measure,[16] its measurement is relatively independent of ventricular morphology and global cardiac motion.[33]

In our analysis, we chose to include 'global' RV strain. Longitudinal strain, obtained from tissue velocity measurements, is commonly divided into basal, mid and apical segments with a reverse basoapical gradient. While some studies included in this analysis reported strain values per segment using this approach,[13 24] others reported only global longitudinal strain determined by 2D echocardiography. This may be an important consideration; Neilan *et al*[13 24] noted greater decreases in strain in the apical segments following marathon. It is, therefore, possible that studies reporting global longitudinal strain may underestimate the true impact of endurance exercise in the regional segments of the RV.

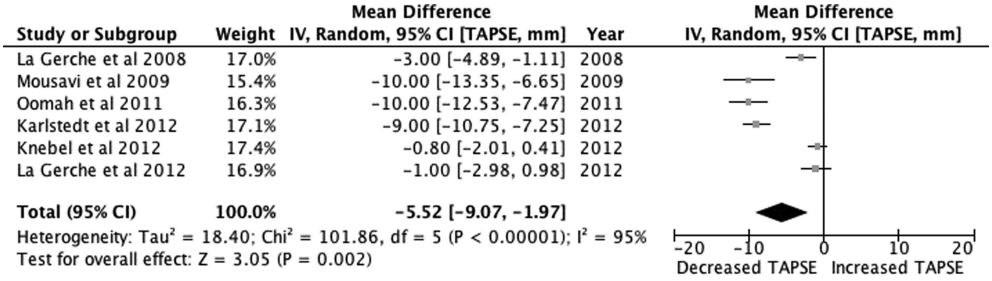

Figure 4 Forest plot comparing tricuspid annular plane systolic excursion (TAPSE) pre-endurance versus postendurance exercise. Mean differences are calculated by random-effects meta-analysis and weighted by estimated precision of the effect.

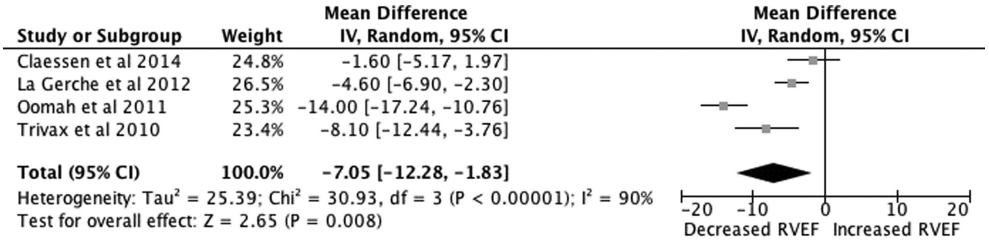

Figure 5 Forest plot comparing right ventricular ejection fraction (RVEF) pre-endurance versus postendurance exercise. Mean differences are calculated by random-effects meta-analysis and weighted by estimated precision of the effect.

The recent inclusion of CMR imaging for the determination of RV volume and RVEF following exercise is a valuable addition to the field. Two studies[20 27] have incorporated CMR-derived RVEF into their assessment of RV function. In addition, Oomah *et al*[28] and La Gerche *et al*[30] determined RVEF by 3D echocardiography. We observed a significant reduction in RVEF following exercise that showed statistical heterogeneity (figure 5). This observation may be due to the three varying durations of exercise, which ranged from a half marathon to an ironman triathlon. Moreover, the use of 3D echocardiography for determining RVEF is still an emerging field that may be open to variation in repeated measurements.

Time course of depressed RV function

Despite the immediate postexercise depression of RV function, there is strong evidence suggesting this to be a transient, fully reversible phenomenon. Although some studies reported RV function at a follow-up timepoint, we did not include these measures in the current meta-analysis because the number of studies was small, the measures inconsistent and the time period to assessment was highly variable. However, it is notable that in each of these studies most measures of RV function returned to normal.[18 24 28 30] The hypothesis that postexercise RV dysfunction, repeated over many years, may provide a proarrhythmogenic substrate is worthy of further investigation. Heidbüchel *et al*[14] identified RV arrhythmogenic involvement in almost 60% of high-level endurance athletes with ventricular arrhythmias, thus indicating the possible clinical significance of repeated endurance exercise.

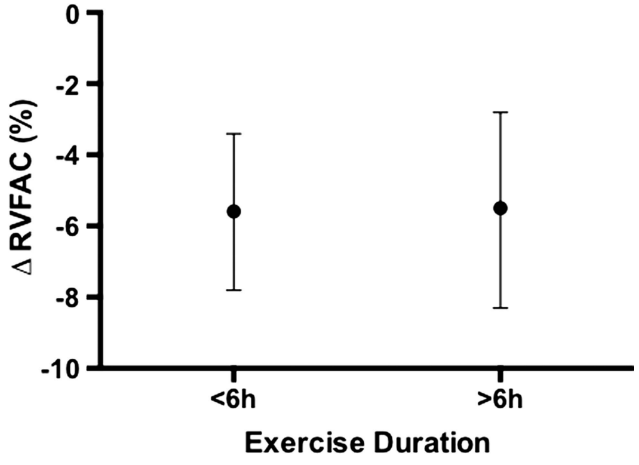

Figure 6 Subgroup analysis showing WMD and 95% CI of the difference in right ventricular fractional area change (RVFAC) pre-exercise versus postexercise.

Interestingly, Ector *et al*[35] studied 22 athletes with ventricular arrhythmias showing that these athletes had a significantly lower RVEF than athletes without arrhythmias. These studies show remarkable similarities with preclinical data. Benito *et al*[36] exposed young rats to a strenuous 18-week treadmill-running regime that, the authors argued, approximated 10 years of endurance exercise training in humans. As compared with the sedentary control rats, the 'marathon rats' demonstrated an increase in atrial and RV inflammation/fibrosis, whereas the LV was spared. Perhaps most importantly, this predominant RV remodeling was associated with a greater potential for inducible ventricular arrhythmias (42% versus 6%, p=0.05). Together, these studies lend weight to the hypothesis that repeated endurance exercise may provide a proarrhythmogenic substrate in athletes and highlights the need for prospective studies assessing long-term clinical outcomes.

Limitations

Although this study provides the first meta-analysis on the effect of prolonged endurance exercise on RV function, there are several limitations worth noting; first, the variability in the techniques used to determine RV function may have influenced the outcomes of this study. Second, many of the studies did not blind assessors of RV function, thus potentially leading to observer bias. Third, this analysis included a total pool of 354 participants, which is still relatively limited for the assessment of small postexercise changes. Furthermore, the number of participants analysed for any given measure of RV function was even smaller, thus further reducing statistical power. Additionally, there is no standardised definition of what constitutes an athlete and, similarly, there is inconsistency in the literature as to whether it is the least or most trained particiants who incur the most profound cardiac injury.[37] Finally, although we reported LV function, we included only studies that reported both LV and RV function. This is not a thorough review of the LV literature; we provide insight into concomitant changes in RV and LV function following endurance exercise.

Summary

In summary, despite heterogeneity in the measures employed, the existing literature consistently reports that prolonged endurance exercise depresses RV systolic function. Prolonged exercise modestly increases the size of the RV and minimally impacts the LV. These findings are in line with the observation of postexercise increases in cardiac biomarkers and potentially indicate a short-term, reversible phenomenon that is purely physiological. Nonetheless, the long-term health consequences of prolonged, strenuous exercise require further investigation. These findings should also be discussed at the next meeting of the Seattle Criteria group[38] (Drezner 2013S) and included in future iterations of that important consensus document.

What are the new findings?

- Multiple studies have consistently demonstrated relative right ventricular (RV) dysfunction following prolonged exercise of more than 90 min duration.
- This impairment is consistent regardless of the parameter used to quantify RV systolic function and occurs in the absence of significant left ventricular dysfunction.
- There is a significant increase in postexercise RV area during systole and a trend towards an increase in RV area during diastole, indicating RV dilation.

How might it impact on clinical practice in the near future?

- A decrease in right ventricular (RV) function is observed following prolonged endurance exercise, although there is little evidence relating to the clinical consequences of this change in cardiac function.
- The short-term consequences of transient relative RV dysfunction require assessment. Potential issues include: the time required for recovery, the completeness of recovery and whether transient RV impairment impacts on cardiac performance during repeated exercise bouts.
- Future human studies should investigate the long-term clinical consequences of prolonged endurance exercise.

Contributors ADE was responsible for the first draft of the manuscript, which was subsequently revised by ALG. Both authors were involved in all stages of the study and agreed the final version of the manuscript.

Funding University of Adelaide.

Competing interests None.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

1 Shave R, Baggish A, George K, *et al*. Exercise-induced cardiac troponin elevation: evidence, mechanisms, and implications. *J Am Coll Cardiol* 2010;56:169–76.
2 Middleton N, Shave R, George K, *et al*. Left ventricular function immediately following prolonged exercise: a meta-analysis. *Med Sci Sports Exerc* 2006;38:681–7.
3 Möhlenkamp S, Lehmann N, Breuckmann F, *et al*. Running: the risk of coronary events: prevalence and prognostic relevance of coronary atherosclerosis in marathon runners. *Eur Heart J* 2008;29:1903–10.
4 Mousavi N, Czarnecki A, Kumar K, *et al*. Relation of biomarkers and cardiac magnetic resonance imaging after marathon running. *Am J Cardiol* 2009;103:1467–72.
5 Rifai N, Douglas PS, O'Toole M, *et al*. Cardiac troponin T and I, echocardiographic [correction of electrocardiographic] wall motion analyses, and ejection fractions in athletes participating in the Hawaii ironman triathlon. *Am J Cardiol* 1999;83:1085–9.
6 Whyte GP, George K, Sharma S, *et al*. Cardiac fatigue following prolonged endurance exercise of differing distances. *Med Sci Sports Exerc* 2000;32:1067–72.
7 Shave R, Dawson E, Whyte G, *et al*. Altered cardiac function and minimal cardiac damage during prolonged exercise. *Med Sci Sports Exerc* 2004;36:1098–103.
8 Tulloh L, Robinson D, Patel A, *et al*. Raised troponin T and echocardiographic abnormalities after prolonged strenuous exercise—the Australian ironman triathlon. *Br J Sports Med* 2006;40:605–9.
9 Shave RE, Dawson E, Whyte G, *et al*. Evidence of exercise-induced cardiac dysfunction and elevated ctnt in separate cohorts competing in an ultra-endurance mountain marathon race. *Int J Sports Med* 2002;23:489–94.
10 Scott JM, Esch BTA, Shave R, *et al*. Cardiovascular consequences of completing a 160-km ultramarathon. *Med Sci Sports Exerc* 2009;41:26–34.
11 George K, Whyte G, Stephenson C, *et al*. Postexercise left ventricular function and ctnt in recreational marathon runners. *Med Sci Sports Exerc* 2004;36:1709–15.
12 Whyte G, George K, Shave R, *et al*. Impact of marathon running on cardiac structure and function in recreational runners. *Clin Sci (Lond)* 2005;108:73–80.
13 Neilan TG, Januzzi JL, Lee-Lewandrowski E, *et al*. Myocardial injury and ventricular dysfunction related to training levels among nonelite participants in the Boston marathon. *Circulation* 2006;114:2325–33.
14 Heidbüchel H, Hoogsteen J, Fagard R, *et al*. High prevalence of right ventricular involvement in endurance athletes with ventricular arrhythmias. Role of an electrophysiologic study in risk stratification. *Eur Heart J* 2003;24:1473–80.
15 Heidbuchel H, Prior DL, La Gerche A. Ventricular arrhythmias associated with long-term endurance sports: what is the evidence? *Br J Sports Med* 2012;46(Suppl 1):i44–50.
16 Mertens LL, Friedberg MK. Imaging the right ventricle—current state of the art. *Nat Rev Cardiol* 2010;7:551–63.
17 Douglas PS, O'Toole ML, Hiller WD, *et al*. Different effects of prolonged exercise on the right and left ventricles. *J Am Coll Cardiol* 1990;15:64–9.
18 Oxborough D, Whyte G, Wilson M, *et al*. A depression in left ventricular diastolic filling following prolonged strenuous exercise is associated with changes in left atrial mechanics. *J Am Soc Echocardiogr* 2010;23:968–76.
19 Oxborough D, Shave R, Warburton D, *et al*. Dilatation and dysfunction of the right ventricle immediately after ultraendurance exercise: exploratory insights from conventional two-dimensional and speckle tracking echocardiography. *Circ Cardiovasc Imaging* 2011;4:253–63.
20 Claessen G, Claus P, Ghysels S, *et al*. Right ventricular fatigue developing during endurance exercise: an exercise CMR study. *Med Sci Sports Exerc* 2014;46:1717–26.
21 Prior DL, La Gerche A. The athlete's heart. *Heart* 2012;98:947–55.
22 Dávila-Román VG, Guest TM, Tuteur PG, *et al*. Transient right but not left ventricular dysfunction after strenuous exercise at high altitude. *J Am Coll Cardiol* 1997;30:468–73.
23 Oxborough D, Shave R, Middleton N, *et al*. The impact of marathon running upon ventricular function as assessed by 2D, Doppler, and tissue-Doppler echocardiography. *Echocardiography* 2006;23:635–41.
24 Neilan TG, Yoerger DM, Douglas PS, *et al*. Persistent and reversible cardiac dysfunction among amateur marathon runners. *Eur Heart J* 2006;27:1079–84.
25 La Gerche A, Connelly KA, Mooney DJ, *et al*. Biochemical and functional abnormalities of left and right ventricular function after ultra-endurance exercise. *Heart* 2008;94:860–6.
26 Banks L, Sasson Z, Busato M, *et al*. Impaired left and right ventricular function following prolonged exercise in young athletes: influence of exercise intensity and responses to dobutamine stress. *J Appl Physiol* 2010;108:112–19.
27 Trivax JE, Franklin BA, Goldstein JA, *et al*. Acute cardiac effects of marathon running. *J Appl Physiol* 2010;108:1148–53.
28 Oomah SR, Mousavi N, Bhullar N, *et al*. The role of three-dimensional echocardiography in the assessment of right ventricular dysfunction after a half marathon: comparison with cardiac magnetic resonance imaging. *J Am Soc Echocardiogr* 2011;24:207–13.
29 Karlstedt E, Chelvanathan A, Da Silva M, *et al*. The impact of repeated marathon running on cardiovascular function in the aging population. *J Cardiovasc Magn Reson* 2012;14:58.
30 La Gerche A, Burns AT, Mooney DJ, *et al*. Exercise-induced right ventricular dysfunction and structural remodelling in endurance athletes. *Eur Heart J* 2012;33:998–1006.
31 La Gerche A, Macisaac AI, Prior DL. Should pre-participation cardiovascular screening for competitive athletes be introduced in Australia? A timely debate in a sport-loving nation. *Heart Lung Circ* 2011;20:629–33.
32 Argiento P, Chesler N, Mulè M, *et al*. Exercise stress echocardiography for the study of the pulmonary circulation. *Eur Respir J* 2010;35:1273–8.
33 La Gerche A, Prior DL, Heidbüchel H. Clinical consequences of intense endurance exercise must include assessment of the right ventricle. *J Am Coll Cardiol* 2010;56:1263; author reply 1263–4.
34 Lewis GD, Bossone E, Naeije R, *et al*. Pulmonary vascular hemodynamic response to exercise in cardiopulmonary diseases. *Circulation* 2013;128:1470–9.
35 Ector J, Ganame J, van der Merwe N, *et al*. Reduced right ventricular ejection fraction in endurance athletes presenting with ventricular arrhythmias: a quantitative angiographic assessment. *Eur Heart J* 2007;28:345–53.
36 Benito B, Gay-Jordi G, Serrano-Mollar A, *et al*. Cardiac arrhythmogenic remodeling in a rat model of long-term intensive exercise training. *Circulation* 2011;123:13–22.
37 Schwabe K, Schwellnus M, Derman W, *et al*. Medical complications and deaths in 21 and 56km road race runners: a 4-year prospective study in 65 865 runners—safer study I. *Br J Sports Med* 2014;48:912–18.
38 Drezner JA, Ackerman MJ, Anderson J, *et al*. Electrocardiographic interpretation in athletes: The 'Seattle criteria'. *Br J Sports Med* 2014;47:122–4.

The right ventricle following prolonged endurance exercise: are we overlooking the more important side of the heart? A meta-analysis

Adrian D Elliott and Andre La Gerche

Br J Sports Med published online October 3, 2014
doi: 10.1136/bjsports-2014-093895