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STROKE VOLUME DYNAMICS IN MALES
WITH DIFFERENT FITNESS LEVEL

Introduction

Stroke volume (SV) is well known as a routine clinical cardiodynamic measure. It represents the amount of blood ejected by the left ventricle in one beat. In a normal subject cardiac output (Q) at rest can be predicted by a model including variables such as heart rate, contractility of the ventricle, afterload and preload. A mild increase of heart rate or contractility, or some afterload reduction will result in increase of cardiac output because of intrinsic adequate preload reserve. During exercise, cardiac output can increase up to five-fold and to even more in trained athletes due to augmentation of all the determinants mentioned (Robergs & Roberts. 1996). During an acute bout of upright progressive exercise in a healthy, untrained person, SV typically rises by 20–30% at low work intensities, then changes little (or 'plateaus') to the point of subject exhaustion (T. W. Rowland, 2005). Highly trained endurance athletes are characterized by their superior cardiac functional capacity, i.e. higher maximal stroke volume and cardiac output, compared with non-athletes (T. W. Rowland & Roti, 2004). It is important to recognise that augmentation of cardiac output is extremely dependent upon preload reserve (Vella & Robergs, 2005). Exercise induced increases in cardiac output are partly explained by the Frank Starling mechanism, but left ventricular filling is more important than myocardial contractility in augmenting stroke volume in normal individuals (Gledhill, Cox & Jamnik, 1994). Clinicians have traditionally used this model to understand the changes of cardiac output in healthy and diseased hearts. This model works extremely well when describing the pathophysiology of primary cardiac problems, such as ischaemic heart disease (Gewillig et al., 2010).

Methods of measurement

Many methods of cardiac output (Q) measurement have been developed, but the number of methods useful for human sports science studies is limited. The »method of choice« for the measurement of cardiac output would be a method that is accurate, precise, operator independent, fast responding, non-invasive, continuous, easy to use, cheap and safe (Geerts, Aarts, & Jansen, 2011). Several methods are available to assess cardiac output (Q) in humans, including invasive techniques such as thermodilution (TD) (Buffington & Nystrom, 2004) ("Thermal dilution cardiac output units," 1988), indocyanine-green dye dilution and the Fick principle; minimally invasive techniques such as trans-oesophageal Doppler (Abrams, Weber, & Holmen, 1989), transpulmonary TD, pulse contour and pulse power analysis (Afshari, Perner, & Bonde, 2006) and non-invasive techniques such as partial CO_2 rebreathing (Agostoni

& Cattadori, 2009), transthoracic Doppler (Akamatsu et al., 2004) and transthoracic electrical bioimpedance cardiography (Albert, 2006). For clinical use, TD has become the standard method in many laboratories for evaluating patients with pulmonary hypertension and severe heart failure unresponsive to pharmacological treatment. TD has, however, disadvantages as it requires right heart catheterization, which potentially can lead to complications such as catheter-related infections, arrhythmias and bleeding (Mathews & Singh, 2008). Furthermore, TD is resource demanding, compared to non-invasive methods (Prentice & Ahrens, 2001). Also, some studies showed that automated cardiac output measurements by ultrasound were inaccurate at high cardiac output (Basdogan et al., 2000). First study undertaken to determine the accuracy of a modification of a single breath method for estimation of cardiac output incorporated a single rebreathing stage followed by a prolonged expiration (Al-Shamma, Hainsworth, & Silverton, 1987) showed that the single breath method is simple to use and sufficiently reliable for use in humans both at rest and during steady states of light exercise. Cardiac output was determined from the O_2 uptake and the instantaneous changes in O_2 and CO_2 in the expired gas during the prolonged expiration. Minimally invasive and non-invasive methods of estimation of cardiac output (Q) were developed to overcome the limitations of invasive nature of pulmonary artery catheterization and direct Fick method used for the measurement of stroke volume (SV). Because gas-exchange measurements during cardiopulmonary exercise testing allow noninvasive measurement of oxygen uptake (VO_2), which is equal to cardiac output (CO) x arteriovenous oxygen content difference [$C(a-vDO_2)$] Q and stroke volume could theoretically be estimated if the $C(a-vDO_2)$ increased in a predictable fashion as a function of % maximum VO_2 ($VO_{2\ max}$) during exercise. Stringer and coworkers concluded that caardiac output (Q) can be accurately estimated from VO_2 during exercise in normal subjects and patients with heart failure by measuring the lactate anaerobic treshold (LAT) or VO_2 peak. From these data and HR, SV can be calculated. This method provided us with a simple and low-cost assessment of cardiac function (Q and SV) in response to exercise that is independent of disturbed lung physiology and acid-base changes during exercise (Stringer, Hansen, & Wasserman, 1997).

Our aim in present study was to evaluate, both, the stroke volume dynamics in males with different fitness level and the relationship between maximal SV value measured during graded exercise testing (GXT) on treadmill and $VO_{2\ max}$ / HR_{max}, respectively.

Methods and study design

One hundred (100) male participants of age $43,06 \pm 9.03$ years, height 175 ± 8.25 cm, body mass 76.94 ± 15.42 kg, BSA $2.01 \pm 0,15$ m^2 and peak oxygen consumption 46.55 ± 7.03 ml kg^{-1} min^{-1} volunteered to participate in this study. All participants were healthy and non-smoking. In order to avoid fatigue-related effects on the test outcomes, we allowed no strenuous physical activity for 48 h prior to the measurements. We informed the participants about all procedures involved and about the associated risks. Subsequently, participants completed a routine health

questionnaire and gave their written informed consent. The experimental protocol was approved by the Ethics Committee (University of Ljubljana - Faculty of Sport) and performed in accordance with the ethical standards laid down in the Declaration of Helsinki for human experimentation. All tests were performed in Exercise Physiology Laboratory (University of Ljubljana, Faculty of Sport) with Cosmed K4b^2 portable "breath by breath" system for spiroergometry on Woodway EG6 motor driven treadmill. Special GXT protocol for recreational runners were used. Maximum oxygen uptake (VO$_{2 max}$) is widely recognised as the best single measure of aerobic fitness (Wasserman, Hansen, Sue, et al,, 2005). According to different level of fitness participans were divided into two groups:

a) expirienced recreational runners   (average VO$_{2max}$ 47.28 ± 6.22 ml · min^{-1} · kg^{-1})
b) sedentary persons or less trained recreational runners (average VO$_{2max}$ 36.6 ± 5.47 ml · min^{-1} · kg^{-1})

Cardiac output (Q) was calculated according to method previously described by Stringer and coworkers (Stringer, Hansen, & Wasserman, 1997):

$$Q = O_{2\,uptake} / [5,721 + (0,1047 \ x \ \% \ VO_{2max})]$$

Stroke volume (SV: ml · utrip^{-1}) and stroke volume index (SI: ml · utrip^{-1} · m^{-2}) were calculated according to formulas below:

$$SV = Q / HF \text{ (heart frequency; beats · min}^{-1})$$

$$SI = SV / BSA \text{ (body surface area; m}^2)$$

Stroke volume dynamics were recorded during every test and analysed according to previously published recommendation (T. Rowland, 2009). Statistical calculations were performed by the use of SPSS Statistics 17.0. The results are presented as mean ± SD. The Student t-test was used to evalaute the possible differences between the two groups. Probabilities of less than 0.05 were accepted as significant.

Results

Most impotant findings of our study showed that expirienced recreational runners significantly differed from sedentary persons or less trained recreational runners. They have higher Q; 20.92 ± 4.57 vs 16,90 ± 4,34 l · min^{-1} (p ≤ 0,001), bigger SV: 136,43 ± 34,59 vs 112,04 ± 30,11 ml · beat $^{-1}$ (p ≤ 0,001), higher SI: 73,41 ± 13,42 vs 60,66 ± 11,58 ml · beat $^{-1}$ · m^{-2} (p ≤ 0,001). Interestingly, both groups reached maximal SV at practically identical level of test (58.62 ± 17.81 % vs 59,85 ± 14,27 % VO$_{2max}$). Also, the pattern of SV fall according to VO$_{2max}$ in both groups tested, were similar (12.08 ± 6.26 % vs 11,33 ± 6,23 %).

Discussion

It is commonly accepted that, during incremental, upright exercise to maximum, stroke volume increases from rest to certain level of exercise and plateaus at 40–50% of $VO_{2\ max}$ (Astrand, Cuddy, Saltin, & Stenberg, 1964). On the other hand, some researchers in the same decade, reported that stroke volume progressively increased during treadmill exercise at workloads of 40–80 % of $VO_{2\ max}$ and at $VO_{2\ max}$ in nine of 13 well trained athletes (Ekblom & Hermansen, 1968). The theory of a stroke volume plateau developed from early research based on a few subjects during two or three exercise intensities, (Bevegard, Holmgren, & Jonsson, 1963) (Grimby, Nilsson, & Saltin, 1966) with the latter characteristic limiting the number of data points used to describe the stroke volume response to exercise. The concept of a plateau in stroke volume was attributed mainly to a decrease in the diastolic filling time that occurs during exercise of increasing intensity (Higginbotham et al., 1986). Unfortunately, these findings were largely ignored and it became accepted that stroke volume plateaus during exercise of increasing intensity. More recent investigations have reported that stroke volume progressively increases in certain people (Gledhill, Cox, & Jamnik, 1994; Krip, Gledhill, Jamnik, & Warburton, 1997; Martino, Gledhill, & Jamnik, 2002). The mechanisms for the continual increase in stroke volume are not completely understood. Same authors proposed that enhanced diastolic filling and subsequent enhanced contractility are responsible for the increased stroke volume in trained subjects (Gledhill, et al., 1994). However, an increase in stroke volume with an increase in exercise intensity has also been reported in untrained subjects (Krip, et al., 1997; Martino, et al., 2002).

Conclusion

Finally, our study showed certain misconception in previous findings that SV is reaching its maximal value during first stages of test (i.e. lower % VO_{2max}). Current research indicates that there is a range of responses in stroke volume to reflect the range of a subject's training status. In addition, training may not be the only factor affecting the stroke volume response to exercise (Schlader et al., 2010). Four main types of stroke volume responses to exercise have been reported in the literature: plateau, (Astrand, et al., 1964; Higginbotham, et al., 1986; McLaren, Nurhayati, & Boutcher, 1997; Proctor et al., 1998; Rivera et al., 1989; Spina et al., 1993), plateau with a drop (Ogawa et al., 1992; Proctor, et al., 1998; Spina, et al., 1993; Spina et al., 1992), plateau with a secondary increase (Chapman, Fisher, & Sproule, 1960; Ferguson, Gledhill, Jamnik, Wiebe, & Payne, 2001; Wiebe, Gledhill, Jamnik, & Ferguson, 1999) and progressive increase (Ekblom & Hermansen, 1968; Gledhill, et al., 1994; Krip, et al., 1997; Martino, et al., 2002; Proctor, et al., 1998; Rivera, et al., 1989; Warburton, Gledhill, Jamnik, Krip, & Card, 1999; Zhou et al., 2001). The implications of a progressive increase in stroke volume to $VO_{2\ max}$ have yet to be completely understood.

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SUMMARY

Stroke volume (SV) is well known as a routine clinical cardiodynamic measure. It represents the amount of blood ejected by the left ventricle in one beat. Our aim in present study was to evaluate, both, the stroke volume dynamics in males with different fitness level and the relationship between maximal SV value measured during graded exercise testing (GXT) on treadmill and $VO_{2\ max}$. One hundred male participants of age 43,06 ± 9.03 years, height 175 ± 8.25 cm, body mass 76.94 ± 15.42 kg, BSA 2.01 ± 0,15 m^2 and peak oxygen consumption 46.55 ± 7.03 ml kg^{-1} min^{-1} volunteered to participate in this study. All tests were performed in Exercise Physiology Laboratory (University of Ljubljana, Faculty of Sport), The group of expirienced recreational runners differed from sedentary or less trained recreational runners in

higher cardiac output (Q); 20.92 ± 4.57 vs 16,90 ± 4,34 l · min^{-1} (p ≤ 0,001), bigger SV: 136,43 ± 34,59 vs 112,04 ± 30,11 ml · $beat^{-1}$ (p ≤ 0,001) and higher SI: 73,41 ± 13,42 vs 60,66 ± 11,58 ml · $beat^{-1}$ · m^{-2} (p ≤ 0,001). Interestingly, both groups reached maximal SV at practically identical level of test (58.62 ± 17.81 % vs 59,85 ± 14,27 % VO_{2max}). Also, the pattern of SV fall according to VO_{2max} in both groups tested, were similar (12.08 ± 6.26 % vs 11,33 ± 6,23 %).

Key words: *stroke volume, cardiac output, running, fitness*