# Cardiac output but not stroke volume is similar in a Wingate and (V) over dot O-2Peak test in young men



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SHORT COMMUNICATION

# Cardiac output but not stroke volume is similar in a Wingate and $\dot{V}O_{2peak}$ test in young men

**Piero Fontana · Katharina Betschon · Urs Boutellier · Marco Toigo**

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**Abstract** Wingate test (WT) training programmes lasting 2–3 weeks lead to improved peak oxygen consumption. If a single 30 s WT was capable of significantly increasing stroke volume and cardiac output, the increase in peak oxygen consumption could possibly be explained by improved oxygen delivery. Thus, we investigated whether a single WT increases stroke volume and cardiac output to similar levels than those obtained at peak exercise during a graded cycling exercise test (GXT) to exhaustion. Fifteen healthy young men (peak oxygen consumption 45.0 ± 5.3 ml $kg^{-1}$ $min^{-1}$) performed one WT and one GXT on separate days in randomised order. During the tests, we estimated cardiac output using inert gas rebreathing (nitrous oxide and sulphur hexafluoride) and subsequently calculated stroke volume. We found that cardiac output was similar (18.2 ± 3.3 vs. 17.9 ± 2.6 l $min^{-1}$; $P = 0.744$), stroke volume was higher (127 ± 37 vs. 94 ± 15 ml; $P < 0.001$), and heart rate was lower (149 ± 26 vs. 190 ± 12 beats $min^{-1}$; $P < 0.001$) at the end (27 ± 2 s) of a WT as compared to peak exercise during a GXT. Our results suggest that a single WT produces a haemodynamic response which is characterised by similar cardiac output, higher stroke volume and lower heart rate as compared to peak exercise during a GXT.

**Keywords** Cardiac output · Stroke volume · Wingate test · Sprint interval training · High-intensity interval training

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## Introduction

Sprint interval training (SIT) is a novel type of high-intensity interval training consisting of 4–6 repeated all-out 30 s Wingate tests (WTs) separated by 4 min recovery. It has been demonstrated that 2–3 weeks (3 training sessions per week) of SIT increases peak oxygen consumption (Burgomaster et al. 2008; Hazell et al. 2010). However, the underlying adaptations in oxygen delivery and extraction possibly mediating the increase in peak oxygen consumption following SIT are not yet clear.

Peak oxygen consumption is bounded by the parametric limits of the Fick equation (oxygen consumption = cardiac output × arterio-venous oxygen difference) (Fick 1870). If the increase in peak oxygen consumption after SIT was mediated by an increase in peak cardiac output, it should be expected that a single WT is capable of increasing cardiac output to near-maximal values. In fact, high-intensity aerobic training (HIT) is more effective in mediating increases in cardiac output and stroke volume than continuous training at a power corresponding to lactate threshold (Daussin et al. 2007; Helgerud et al. 2007). HIT is characterised by 4–5 blocks of short (1–4 min) training intervals at a power output corresponding to, e.g. 90–100% peak power attained during a GXT, separated by 3–4 min

at a power corresponding to 50–60% peak GXT power. Daussin et al. (2007) concluded that the near-maximal cardiac output attained during HIT mediates the observed long-term adaptations in peak cardiac output and stroke volume which, in turn, underlay the observed increase in peak oxygen consumption after several weeks of HIT.

One study compared cardiac output and stroke volume between a GXT and a WT by means of echocardiographic measurements (Sagiv et al. 2000). It was concluded that stroke volume is increased only with GXT, and that the increase of cardiac output is significantly less for WT than for GXT. This conclusion implies that a WT cannot provide a sufficiently high stimulus for triggering cardiocirculatory adaptation, and consequently that the observed increase in peak oxygen consumption after a WT training programme (Burgomaster et al. 2008; Hazell et al. 2010) cannot originate from an increase in peak stroke volume and cardiac output. As echocardiographic measurements are not feasible when the upper body is moving, the participants' backs had to be strapped to the wall in the study of Sagiv et al. (2000). As an obvious consequence, the participants could not generate as much power as during a real WT with freely moving upper body. In this study, we thus wanted to re-investigate the cardiac output response during WT using an inert gas (nitrous oxide and sulphur hexafluoride) rebreathing technique, which does not restrict the participants' position (and thus exercise capacity).

## Methods

### Participants

Fifteen male participants of age 26.7 ± 5.6 years, height 1.81 ± 0.05 m, body mass 73.6 ± 7.0 kg and peak oxygen consumption 45.0 ± 5.3 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 of the ETH Zurich and was performed in accordance with the ethical standards laid down in the Declaration of Helsinki for human experimentation.

### Study design and experimental setup

Each participant performed one GXT on a cycle ergometer (Ergoselect 200 K, Ergoline, Bitz, Germany) and one WT (Ergomedic 894E Peak Bike, Monark, Vansbro, Sweden). The two tests were separated by at least 48 h and were performed in a randomised order. Each participant performed the two tests at the same daytime. During GXT and WT, cardiac output and stroke volume were estimated by inert gas rebreathing (Innocor™, Innovision, Odense, Denmark). In addition, heart rate was continuously recorded (S610i, Polar Electro, Kempele, Finland). Before the tests, we informed the participants about the upcoming procedures and they practiced the rebreathing technique with ambient air (Fontana et al. 2009). No additional familiarisation trials were performed prior to GXT and WT.

Prior to the start of the GXT, participants rested for 2 min on the cycle ergometer. Subsequently, they started pedalling at 100 W using a self-selected pedalling rate [70–90 revolutions per minute (rpm)], which was held constant (±5 rpm) throughout the test. Power was increased by 30 W for every 2 min until participants stopped cycling or were unable to maintain pedalling rate within the given limits for more than 5 s (mean GXT duration 12.4 ± 2.5 min). We previously used the same incremental protocol to show that test–retest reliability of cardiac output and stroke volume during a GXT is high (Fontana et al. 2009). Furthermore, we demonstrated that at 46 and 103 s after a power stage transition (54–69, 69–77 and 77–85% peak power) both cardiac output and stroke volume are similar (Fontana et al. 2010), indicating that less than 46 s are needed to reach a steady state for cardiac output and stroke volume. For WT, participants started unloaded pedalling as fast as possible. As soon as they reached 130 rpm, the computer interfaced with the test bike automatically loaded the ergometer with 7.5% of the participant's body weight (Burgomaster et al. 2005). Participants were verbally encouraged to continue to pedal as fast as possible.

### Estimation of cardiac output

The rebreathing manoeuvres during GXT were performed in accordance with our previously published protocol (Fontana et al. 2009). We manually set the rebreathing parameters to 3 l rebreathing volume and 10% bolus volume (volume of gas mixture, consisting of nitrous oxide, sulphur hexafluoride and oxygen). During the rebreathing manoeuvres, participants continued to spontaneously breathe, as their breathing frequency was already high enough while cycling. Breathing frequency was monitored online by the examinator and participants were given verbal feedback to maintain a constant breathing frequency. All participants cycled until the software of the Innocor™ indicated the end of the rebreathing. For WT, the exact point in time (averaged over all participants) of the first breath, which we used for fitting the regression line

(calculation of cardiac output, represented by complete gas mixing), was 27 ± 2 s (range 24–32 s) after the beginning of the sprint bout (after loading). Given that the rebreathing lasted ~3 s, a WT lasted 27–35 s. Using the end tidal nitrous oxide concentrations of at least two breaths, cardiac output could be estimated in all study participants during the final phase of WT, as well as at peak GXT. The coefficient of variation (CV) over repeated preliminary measurements of cardiac output during the final phase of a WT for the investigator was 8%. This is in agreement with values obtained for a GXT (95% confidence interval: 5.5–9%, Fontana et al. 2009).

### Calculations

#### *GXT*

We calculated stroke volume at peak exercise by dividing cardiac output by the heart rate measured during the rebreathing (10 s average), corresponding to the highest 10 s average throughout the GXT (i.e. peak heart rate).

#### *WT*

WT stroke volume during the WT was obtained by dividing cardiac output by heart rate. Here, we used a 5 instead of a 10 s mean because of the shorter test duration. The corresponding heart rate measurement started as soon as the valve to the rebreathing bag opened during cardiac output determination.

### Statistical analysis

Statistical analyses were performed with SPSS software (version 16.0, SPSS, Chicago, IL, USA). Normality of ln-transformed data was ascertained by Q–Q plots. For comparisons of cardiac output, stroke volume and heart rate between GXT and the WT, we used Student's paired *t* test. Results are presented as mean ± standard deviation. Statistical significance was set at $P < 0.05$.

## Results

Cardiac output at (or immediately before) the end of WT and GXT was similar (Table 1). Furthermore, stroke volume was higher (34 ± 31%), whereas heart rate (−22 ± 12%) was lower at the end of WT compared to GXT. Power (460 ± 54 vs. 286 ± 38 W; $P < 0.001$) and pedalling rate (89 ± 9 vs. 77 ± 5 rpm; $P = 0.002$) during WT rebreathings were significantly higher than during GXT rebreathings.

**Table 1** Cardiac output, stroke volume and heart rate in 15 young healthy men at the end of a Wingate test (WT), as well as at the point in time of volitional exhaustion during a graded cycling exercise test (GXT)

| | Wingate test (WT) | Graded cycling exercise test (GXT) | *P* |
|---|---|---|---|
| Cardiac output (l $min^{-1}$) | 18.2 ± 3.3 | 17.9 ± 2.6 | 0.744 |
| Stroke volume (ml) | 127 ± 37 | 94 ± 15 | <0.001 |
| Heart rate (beats $min^{-1}$) | 149 ± 26 | 190 ± 12 | <0.001 |

## Discussion

In the present study, we found that cardiac output is similar at the end of WT compared to the point in time of volitional exhaustion during GXT in healthy young men. In contrary to cardiac output, stroke volume was significantly higher and heart rate was significantly lower for WT than GXT. Our results contrast with the data of Sagiv et al. (2000), who found that both cardiac output and stroke volume were significantly higher for GXT than WT. We ascribe the divergent results to the unnatural (i.e. fixed upper body) WT condition used by Sagiv et al. (2000).

Having shown that a single WT is capable of increasing cardiac output to maximal levels, we have provided novel, scientific insight into the mechanistic bases possibly underlying the increase in peak oxygen consumption which is observed after 2–3 weeks of WT training programmes (Burgomaster et al. 2008; Hazell et al. 2010). In fact, our results suggest that the observed increase in peak oxygen consumption after a WT training programme (Burgomaster et al. 2008; Hazell et al. 2010) might originate from an increase in peak cardiac output.

There are a few considerations pertinent to the estimation of cardiac output that are warranted. First, the absorption of nitrous oxide by the blood, and hence the calculation of pulmonary blood flow (equal to cardiac output when there is no pulmonary shunt flow), can be affected not only by ventilation inhomogeneity but also by flow inhomogeneity. If breathing frequency during the rebreathing manoeuvres is not constant, the slope of the regression line through end-tidal gas concentrations could yield a wrong estimate of cardiac output. Only in 5 out of 30 rebreathing manoeuvres participants could not maintain a constant breathing frequency. For those rebreathings, we manually moved the regression line to 2–3 breaths with a stable pulmonary blood flow to correctly estimate cardiac output.

Second, pulmonary blood flow equals cardiac output only if there is no significant pulmonary shunt flow. Eldridge et al. (2004) showed that arterio-venous intrapulmonary shunts develop at about 59% peak oxygen consumption and persist at all subsequent power stages.

Thus, in both exercise modalities, arterio-venous intrapulmonary shunts may have developed. Assuming for each participant a blood haemoglobin concentration of 15 g $dl^{-1}$ and considering the respective arterial oxygen saturations (data not shown), we found that during a GXT, the estimated arterio-venous intrapulmonary shunt fraction tended to be higher (6.0 ± 2.7%) than during a WT (1.9 ± 2.7%). However, even when considering these possible differences in estimated arterio-venous intrapulmonary shunt flow, our findings presented in Table 1 do not significantly change.

Finally, we want to address the issue that maximal stroke volume might have been underestimated during the GXT. It appears that during a GXT with constant power increments, stroke volume reaches maximal values already at 64% peak power and remains at this level until exhaustion (Calbet et al. 2007; Fontana et al. 2010). In contrast, for GXT protocols with varying power increments it has been shown that stroke volume is maximal at 80% and subsequently decreases to reach a significantly lower value at exhaustion (Mortensen et al. 2005). Besides differences in exercise protocols, levels of exertion, exercise mode, sex, age and training status also might lead to different responses in stroke volume. Given the fact that we used a GXT with constant power increments and that the participants' sex, age as well as training status was similar in the study by Calbet et al. (2007), we believe that we report (near-)maximal values for stroke volume in the present study.

We conclude that a single, unrestricted WT produces a haemodynamic response which is characterised by similar cardiac output, higher stroke volume and lower heart rate compared to peak exercise during a GXT in young men.

**Conflict of interest** None.

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