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ORIGINAL ARTICLE

Non-invasive haemodynamic assessments using Innocor™ during standard graded exercise tests

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Abstract Cardiac output (Q) and stroke volume (V_S) represent primary determinants of cardiovascular performance and should therefore be determined for performance diagnostics purposes. Since it is unknown, whether measurements of Q and V_S can be performed by means of Innocor™ during standard graded exercise tests (GXTs), and whether current GXT stages are sufficiently long for the measurements to take place, we determined Q and V_S at an early and late point in time on submaximal 2 min GXT stages. 16 male cyclists (age 25.4 ± 2.9 years, body mass 71.2 ± 5.0 kg) performed three GXTs and we determined Q and V_S after 46 and 103 s at 69, 77, and 85% peak power. We found that the rebreathings could easily be incorporated into the GXTs and that Q and V_S remained unchanged between the two points in time on the same GXT stage (69% peak power, Q: 18.1 ± 2.1 vs. 18.2 ± 2.3 l min^{-1}, V_S: 126 ± 18 vs. 123 ± 21 ml; 77% peak power, Q: 20.7 ± 2.6 vs. 21.0 ± 2.3 l min^{-1}, V_S: 132 ± 18 vs. 131 ± 18 ml; 85% peak power, Q: 21.6 ± 2.4 vs. 21.8 ± 2.7 l min^{-1}, V_S: 131 ± 17 vs. 131 ± 22 ml). We conclude that Innocor™ may be a useful device for assessing Q and V_S during GXTs, and that the adaptation of Q and V_S to exercise-to-exercise transitions at moderate to high submaximal power outputs is fast enough for 1 and 2 min GXT stage durations.

Keywords Performance diagnostics · Cardiac function · Haemodynamics · Steady state · Graded exercise tests · Inert gas rebreathing

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Introduction

Conventional cardiovascular performance diagnostics relies on heart rate, blood lactate concentration, perceived exertion, respiratory (oxygen consumption, carbon dioxide output), and ventilatory (breathing frequency, tidal volume) parameters. Changes in these parameters over time, in particular, with respect to maximal oxygen consumption, are used to determine performance improvements after training. Nevertheless, (maximal) oxygen consumption does not constitute the primary determinant of cardiovascular performance. In fact, it is believed that in healthy humans, a high maximal oxygen consumption primarily depends on maximal oxygen delivery to the exercising muscles (Saltin and Calbet 2006), and therefore on a high, maximal cardiac output (Calbet et al. 2004). Consequently, we propose that for performance diagnostics purposes, cardiac output and stroke volume should standardly be determined under exercise conditions and subsequently related to the conventional respiratory, ventilatory, and metabolic parameters.

However, cardiac output and stroke volume determinations must meet several requirements in order to be viable for incorporation into standard exercise tests. A central requirement is that the employed measurement method is valid, reliable, and safe. Furthermore, it should not pose any major discomfort to the participants. In addition, the

employed method should be easily applicable to standard graded exercise test (GXT) protocols (Bentley et al. 2007). One promising non-invasive approach having the potential to fulfil these requirements is nitrous oxide (inert gas) rebreathing, which can be performed using the recently introduced Innocor™ system (Innovision, Odense, Denmark). Validation of the Innocor™ cardiac output measurement against direct Fick showed a good accordance (Agostoni et al. 2005; Gabrielsen et al. 2002; Peyton and Thompson 2004). In addition, we recently found that cardiac output determinations using Innocor™ are reliable (Fontana et al. 2009). While it was shown that cardiac output determinations by Innocor™ are valid and reliable, it is still unknown whether the measurement point in time during a typical GXT stage (i.e. 2 min) affects the outcome of the cardiac output measurement in healthy participants, i.e. whether cardiac output differs if measured within the first minute after the exercise-to-exercise transitions compared to when measured at the end of the 2 min stages. Evidence for quick adjustments of cardiac output to increased power comes from the examination of haemodynamics during rest-to-exercise transitions in healthy participants by determining cardiac function on a beat-by-beat basis, using Doppler ultrasound (De Cort et al. 1991) or the model flow method (Lador et al. 2006, 2008). These studies showed that when increasing power from rest to 50, 80, or 100 W (i.e. "rest-to-exercise" transitions), a steady state for cardiac output is quickly reached (e.g. within the first minute). However, all power outputs were not higher than 100 W, and only rest-to-exercise, but not exercise-to-exercise transitions were examined. Furthermore, these studies do not provide any information about intra-stage haemodynamics, i.e. the evolvement of cardiac output, stroke volume, and heart rate on the single stages of moderate and high submaximal power during standard GXTs.

Therefore, we aimed at determining cardiac output and stroke volume using inert gas rebreathing by means of Innocor™, as well as heart rate, during standard GXTs after 46 and 103 s at 69, 77, and 85% peak power in healthy male participants. We hypothesised that these measurements are feasible for stage durations of 2 min, and that on each test stage no differences with respect to cardiac output and stroke volume exist between the two points in time (46 and 103 s).

Methods

Participants

We recruited 16 recreationally trained male cyclists. The participants' individual values are presented in Table 1. All participants were healthy and non-smoking. In order to avoid fatigue-related effects on test performance, no strenuous physical activity was allowed for 48 h prior to the tests. Starting 2 weeks before the examinations, the participants recorded their physical activity in a training log and continued to do so until the end of the study. Based on the training log, we observed that the training volume was similar throughout the study. We informed the participants about all procedures involved and about the

Table 1 Values of body mass, height, age, peak oxygen consumption, and peak power for 16 male cyclists

Participant	Body mass (kg)	Height (m)	Age (years)	Peak oxygen consumption (l min^{-1})	Peak power (W)
1	62.5	1.611	25.3	4.48	360
2	71.6	1.750	22.1	4.40	390
3	76.0	1.877	22.2	4.39	390
4	69.5	1.775	26.5	4.60	390
5	84.4	1.902	22.3	4.68	390
6	74.7	1.820	26.1	3.98	370
7	70.7	1.830	22.1	4.40	400
8	70.2	1.778	26.9	4.11	400
9	70.8	1.815	22.4	4.71	430
10	66.9	1.792	25.4	4.54	370
11	74.8	1.795	26.9	4.38	373
12	73.9	1.866	28.3	4.45	400
13	65.8	1.745	26.9	4.05	348
14	71.2	1.795	30.4	4.43	350
15	68.0	1.785	22.5	4.47	403
16	68.9	1.880	30.1	4.36	375
Mean ± SD	71.2 ± 5.0	1.801 ± 0.069	25.4 ± 2.9	4.40 ± 0.21	384 ± 22

SD standard deviation

associated risks. After the completion of a routine health questionnaire, the participants gave written informed consent. The study was approved by the human ethics committee of the ETH Zurich.

Study design and experimental setup

All participants performed three GXTs to volitional exhaustion on a cycle ergometer (Ergoselect 200 K, Ergoline, Bitz, Germany). In all participants, test 1 (increments of 30 W every 2 min until volitional exhaustion) served for determining peak power and maximal gas exchange (Innocor^{TM}, Innovision, Odense, Denmark). During test 1, we also determined the freely chosen pedalling rate ($\geq$70 min^{-1}), which was then held constant throughout all tests. In tests 2 and 3, we non-invasively determined cardiac output by inert gas rebreathing (Innocor^{TM}, Innovision, Odense, Denmark) at 69, 77, and 85% peak power attained in test 1. The respective rebreathings were performed in a randomised order, either during the first or the second minute on each stage (either in test 2 or 3). Throughout tests 2 and 3, we continuously recorded heart rate and oxygen consumption. All tests took place within a time frame of 2 weeks, except for subject number 9, who was tested 5 weeks apart, due to a common cold.

On the first examination day, we informed the participants about the upcoming procedures. Then, they performed test 1. Test 2 was either carried out 1 h after test 1 or on day 2. Test 3 was always conducted on day 2, either 1 h after test 2 or as the only test performed. Before tests 2 and 3, we equipped the participants with a heart rate monitor (S610i, Polar Electro, Kempele, Finland), a facemask (Hans Rudolph, Shawnee, KS, USA), and an antibacterial filter (PALL PF30-S, Pall, East Hills, NY, USA). We then connected the participants to the Innocor^{TM} device. Before test 2, the participants practiced the rebreathing procedure with ambient air during three attempts, as described earlier (Fontana et al. 2009). Subsequently, we measured cardiac output at rest. Two minutes after the rebreathing under resting conditions, cycling started at 54% peak power for 2 min ("warm-up"). Then, we increased power to 69% peak power for 2 min. Afterwards, power was raised twice by 8% peak power for 2 min. Cardiac output determinations were performed at 69, 77, and 85% peak power. All measurements were performed by the same investigator.

Determination of cardiac output

Rebreathings were performed in a closed system, which consisted of a three-way respiratory valve connecting a facemask, an anti-static rubber bag, and an infrared photoacoustic gas analyser (Clemensen et al. 1994). The participants rebreathed a gas mixture of nitrous oxide (0.5%) and sulphur hexafluoride (0.1%) in oxygen, diluted with atmospheric air, from an anaesthesia bag of size 3–6 l, depending on the individual participant's predicted vital capacity (Quanjer et al. 1993). At rest, the settings of the Innocor^{TM} were a 20% bolus volume (volume of gas mixture, consisting of nitrous oxide, sulphur hexafluoride, and oxygen), a rebreathing frequency of 20 min^{-1}, and a total gas mixture volume of 40% of the predicted vital capacity (Quanjer et al. 1993). The Innocor^{TM} software adjusted these settings dynamically under exercise conditions, considering a maximal bolus volume of 40%, a minimal oxygen content of 13%, and a maximal carbon dioxide content of 15%, taking into account the participants' individual breathing frequencies. Rebreathing was typically performed over 5–8 breaths, of which the last 2–3 breaths were used for the calculation of pulmonary blood flow and cardiac output, taking into account estimated shunt flow (Gabrielsen et al. 2002). The calculation of cardiac output was performed according to the manufacturer. For detecting the single breaths during the rebreathings, we standardly used end-tidal carbon dioxide concentrations. This procedure has been shown to give reliable measurements of pulmonary blood flow and cardiac output, even at peak exercise (Fontana et al. 2009). However, during some rebreathings, the detection of the breaths was facilitated using end-tidal oxygen concentrations. Employing this procedure, all rebreathings could be used in order to calculate cardiac output. Each rebreathing lasted between 8 and 10 s. Since the mean transit time of a dye marker after 60–120 s of high intensity exercise appears to be in the range of 12 s (Krustrup et al. 2009), it is unlikely that recirculation of nitrous oxide occurred. Additionally, we checked online breath-by-breath rebreathing gas concentrations during each cardiac output measurement in order to detect possible elevations in nitrous oxide concentrations due to recirculating gas. We observed no nitrous oxide gas recirculation during all measurements. Thus, a recirculation-based measurement bias should be excluded.

Calculations

For each participant, we calculated peak power as "power on the last completed stage + t(s)/120 s $\times$ 30 W". For peak oxygen consumption, we took the highest mean over 30 s achieved in test 1. During tests 2 and 3, the exact rebreathing points in time relative to test stage duration were calculated by determining the point in time of the valve opening and adding the time elapsed until complete mixing of the rebreathing gases was achieved. Therefore, rebreathing points in time were on average 46 or 103 s either in test 2 or 3 on the three test stages (Table 2). We

Table 2 Individual rebreathing points in time (s) after exercise-to-exercise transitions at 69, 77, and 85% peak power for 16 male cyclists

Participant	46 s	103 s
1	38	119
2	52	104
3	36	97
4	36	96
5	37	98
6	46	97
7	45	104
8	52	107
9	45	104
10	64	101
11	46	106
12	50	103
13	50	104
14	50	104
15	49	105
16	48	98
Average ± SD	46 ± 7	103 ± 5

SD standard deviation

calculated stroke volume by dividing cardiac output by heart rate. For heart rate, we took the 10 s mean recorded right after the rebreathings.

Statistical analysis

We checked the data for normality by *Q*–*Q* plot, and analysed it for statistical significance ($P < 0.05$) using SPSS 16.0 (SPSS, Chicago, IL, USA). For comparisons of cardiac output, stroke volume, and heart rate between 46 and 103 s on each single stage, we used Student's paired *t* test. For comparisons between stages, ANOVA repeated measures with Bonferroni correction were used. Since we did not perform multiple comparisons, the occurrence of a type I error is unlikely and the aforementioned analyses were chosen instead of a two-way ANOVA (time × stage).

Results

Cardiac output

Cardiac output measurements by Innocor™ inert gas rebreathing could be performed at 69, 77, and 85% peak power at both, 46 and 103 s after the stage transitions. Furthermore, cardiac output for the 16 participants did not differ between the early (46 s) and late (103 s) rebreathing points in time on all three test stages (69, 77, and 85% peak power; Table 3; Fig. 1), and the adjustments of cardiac output between 46 and 103 s were the same after increments of 15 (from 54 to 69% peak power) and 8% peak power (from 69 to 77% and from 77 to 85% peak power; Table 3). Additionally, cardiac output increased significantly from rest to 69% of peak power at 46 and 103 s (Table 3). Between 69 and 77% peak power, cardiac output further increased significantly with respect to both measurement points in time, whereas between 77 and 85% peak power, cardiac output was the same (Table 3).

Stroke volume

Stroke volume was not significantly different between the two measuring points in time at 69, 77, and 85% peak power (Table 3). Furthermore, stroke volume significantly increased when transitioning from rest to 69% peak power, both when measured at 46 and 103 s (Table 3). At 77 and 85% peak power, stroke volume was not different than at 69% peak power (Table 3).

Heart rate

Heart rate was significantly higher after 103 than after 46 s at 69% peak power (Table 3). Both, at 77 and 85% peak power, heart rate was not different between 46 and 103 s (Table 3). Heart rate rose from rest to 69, 77, and 85% peak power with respect to both measurement points in time (Table 3).

Discussion

In this study, we have shown that it is feasible to non-invasively determine cardiac output by inert gas rebreathing at 46 and 103 s on moderate and high intensity submaximal GXT stages, using the Innocor™ device. This study also shows that cardiac output of recreationally trained cyclists does not change between these two points in time at 69, 77, and 85% peak power (Fig. 2). While stroke volume, too, is not different when determined at 46 and 103 s after power stage transitions, heart rate is higher after 103 than after 46 s at 69% peak power, but is unchanged between the two measurement points in time at both 77 and 85% peak power. Our finding that at different power stages of standard graded cycling exercise tests, non-invasive measurements by inert gas rebreathing using the Innocor™ device can easily be performed in healthy, recreationally trained male cyclists, might offer a new dimension to current cardiovascular performance diagnostics. Since peak oxygen consumption is mainly determined by peak cardiac output (Fick 1870), and the latter mainly depends on a large stroke volume (Levine 2008), central haemodynamic improvements after training could be

Table 3 Cardiac output, stroke volume, heart rate, and oxygen consumption at rest, 69, 77, and 85% peak power

	Rest	69% peak power			77% peak power			85% peak power			Rest versus 69% peak power		69 versus 77% peak power		77 versus 85% peak power	
		46 s	103 s	*P* (46 vs. 103 s)	46 s	103 s	*P* (46 vs. 103 s)	46 s	103 s	*P* (46 vs. 103 s)	*P* (46 s)	*P* (103 s)	*P* (46 s)	*P* (103 s)	*P* (46 s)	*P* (103 s)
Cardiac output (l min^{-1})	6.4 ± 1.2	18.1 ± 2.1	18.2 ± 2.3	0.794	20.7 ± 2.6	21.0 ± 2.3	0.356	21.6 ± 2.4	21.8 ± 2.7	0.678	<0.001	<0.001	<0.001	<0.001	0.070	0.263
Stroke volume (ml)	91 ± 23	126 ± 18	123 ± 21	0.343	132 ± 18	131 ± 18	0.874	131 ± 17	131 ± 22	0.999	<0.001	<0.001	0.137	0.052	1.000	1.000
Heart rate (beats min^{-1})	72 ± 8	145 ± 7	149 ± 9	0.005	158 ± 7	161 ± 7	0.169	166 ± 9	168 ± 11	0.276	<0.001	<0.001	<0.001	<0.001	0.003	0.002
Oxygen consumption (l min^{-1})	0.43 ± 0.08	3.33 ± 0.30	3.62 ± 0.27	<0.001	3.77 ± 0.28	3.90 ± 0.28	0.071	4.07 ± 0.27	4.13 ± 0.33	0.430	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001

During exercise, measurements were performed at 46 and 103 s after the exercise-to-exercise transition. Data are mean ± standard deviation for 16 male cyclists. We compared cardiac output, stroke volume, heart rate, and oxygen consumption between 46 and 103 s on each stage [*P* (46 vs. 103 s)], as well as between stages [*P* (rest vs. 69% peak power), *P* (69 vs. 77% peak power), and *P* (77 vs. 85% peak power)]

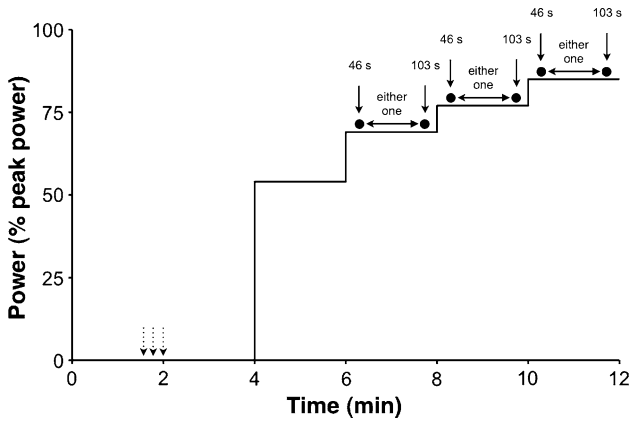

Fig. 1 Cardiac output measurement protocol (tests 2 and 3). Rebreathings were performed either at 46 or 103 s after the power stage transitions at 69, 77, and 85% peak power, as assessed during test 1. On each stage, the measurement point in time (46 or 103 s) was randomised (*horizontal black arrows*). *Vertical black arrows* cardiac output measurements, *vertical dashed arrows* learning trials (rebreathings with ambient air, only before test 2)

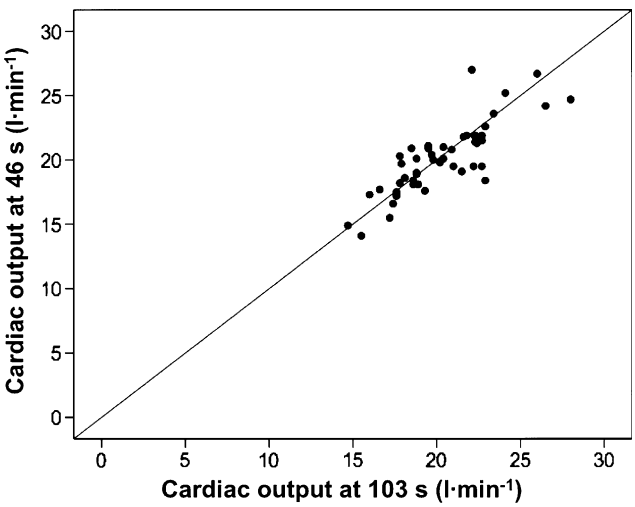

Fig. 2 Individual values of cardiac output at 46 and 103 s at 69, 77, and 85% peak power for 16 male cyclists. The *solid line* indicates the line of identity

detected in parallel with gas exchange in future standard cardiovascular diagnostic tests. This possibility might prove useful in better defining the link between changes in metabolic demand and cardiovascular control following exercise and/or inactivity, and help physiologists as well as coaches to improve the supervision of athletic performance.

Our result that at 69, 77, and 85% peak power, cardiac output in healthy male cyclists is the same at 46 and 103 s after reaching the power stage (Fig. 2) lends further credence to earlier studies investigating the haemodynamic response to rest-to-exercise transitions. In these studies, it

was shown that a steady state for cardiac output is attained between 30 (Lador et al. 2006, 2008) and 60 s (De Cort et al. 1991). Also, Davies et al. (1972) found a rapid adjustment of cardiac output after increasing power (i.e. walking velocity and gradient) during treadmill exercise, using a modified indirect Fick method. However, the observed halftime for cardiac output adaptation after increasing power from moderate (~40% maximum aerobic power) to heavy exercise (~80% maximum aerobic power) was 20 s. In our study, the respective halftime seems to be in the range of 12 s, indicating slightly faster cardiac output kinetics than in Davies et al. (1972), although more measurement points in time would be required to obtain comprehensive kinetic data.

We also observed that cardiac output in healthy male cyclists is the same at 46 and 103 s after reaching the power stage, irrespective of the size of the preceding power increment (up to 15% peak power) between stages (15% from 54 to 69% peak power, 8% from 69 to 77%, and 8% from 77 to 85%). Therefore, it seems that small power increments (up to 15% peak power) between two stages may not influence the evolution of cardiac output on the higher stage, although we did not explicitly examine cardiac output on stages with equal power after increments of different sizes. Furthermore, the evolution of cardiac output after increasing power during a GXT seems not to be influenced by a levelling off of cardiac output, which in our experiments occurred between 77 and 85% peak power. Our observed flattening of the cardiac output–power relationship at high submaximal intensities is consistent with the findings of Calbet et al. (2007, dye-dilution) and Mortensen et al. (2005, Fick principle), who found cardiac output to level off after 84 and 80% peak power, respectively.

Evidence for rapid adjustments of stroke volume after stage transitions is that stroke volume was not significantly different between 46 and 103 s at 69, 77, and 85% peak power. Furthermore, stroke volume at 69% peak power tended to be higher after 46 s than at 103 s. This finding was accompanied by a significantly lower heart rate. Since stroke volume was calculated by dividing cardiac output by heart rate, the lack of statistical significance concerning the potentially lower stroke volume at 103 s might be a statistical issue. This assumption may be supported by the data of Davis et al. (2005), who estimated stroke volume during GXTs with different increment durations, i.e. 1 and 4 min. They found that stroke volume at submaximal power was higher for the 1-min than for the 4-min protocol. However, the rapid inter-stage adjustment of stroke volume was not compromised by the levelling off of stroke volume between 69 and 85% peak power. In fact, in our experiments, stroke volume increased significantly when transitioning from rest to 69% peak power, but was the same at 69, 77, and 85% peak power. These findings are consistent with those of Calbet et al. (2007), who reported stroke volume to rise after the transition from unloaded pedalling to submaximal exercise, and then reach maximal values beyond 64% peak power. Furthermore, our data confirm the findings of Davies et al. (1972), who found stroke volume to be similar between 40 and 80% of maximal aerobic power. However, according to our results, stroke volume might reach a plateau as late as at 77% peak power, since stroke volume tended to be lower at 69 compared to 77% of peak power.

The encountered haemodynamic pattern leads us to speculate that at 69% peak power, maximal stroke volume may not be fully exploited. Thus, at this moderate intensity, stroke volume may still possess a reserve for further increases that could serve to quickly adjust cardiac output after increasing power by 15% from 54 to 69% peak power. The fact that at 69% peak power heart rate was different between 46 and 103 s may be further indicative of stroke volume's role in mediating the rapid adjustment of cardiac output after the stage transition from 54 to 69% peak power. Further support for our speculation that stroke volume might mediate the initial increase in cardiac output comes from De Cort et al. (1991), who reported that stroke volume quickly reacts to increases of power, and then tends to decrease after 90–120 s of constant power exercise, with cardiac output remaining unchanged. However, at exercise intensities of 77 and 85% peak power, peak stroke volume seems to be fully exploited. Accordingly, stroke volume at these intensities might not be further increased to raise cardiac output after power stage transitions. Therefore, cardiac output adjustments after increasing power from 77 to 85% peak power may solely rely on the increase in heart rate. Indeed, our data indicate that heart rate at 77 and 85% peak power is the same at 46 and 103 s. The quicker heart rate adjustment at high intensities may possibly be facilitated by the pronounced increase of sympathetic nervous system activity above 60–70% peak oxygen consumption (Nakamura et al. 1993; Saito and Nakamura 1995; Yamamoto et al. 1991) with concomitant pronounced increases of norepinephrine and epinephrine concentrations (Escourrou et al. 1984; Nakamura et al. 1993; Orizio et al. 1988). This positive inotropic effect of the increased sympathetic drive to the heart is likely not to further raise stroke volume, since at 77 and 85% peak power maximal stroke volume was reached.

Methodological considerations

Three methodological issues must be considered. The first one concerns the rebreathing points in time (46 ± 7 and 103 ± 5 s). They result from averaging the individual points in time (Table 2) across all participants, and are

explained by the fact that the point in time of the first breath, of which the end-tidal nitrous oxide concentration was used for fitting the regression line, slightly varied between participants as well as within participants between the tests. Two reasons explain this variation. The first reason is that the point in time of complete gas mixing (respiratory tracts, anaesthesia bag, mask, and valves) during inert gas rebreathing by Innocor^{TM} slightly differed within the tests (within and between the participants). In our study, complete gas mixing was typically achieved within the first 3–5 breaths after the start of the rebreathing procedure. Complete gas mixing is required for reliably calculating pulmonary blood flow (from which cardiac output is determined), since the calculation of pulmonary blood flow is based on a regression line through end-tidal nitrous oxide concentrations versus time. The second reason for the variation of the individual rebreathing points in time is that depending on the participants' breathing pattern, the start of the rebreathing procedure varied by 1–5 breaths (within and between the participants). Hence, based on these individual measurement delays, the rebreathing points in time (averaged within and between the participants) were 46 and 103 s. The second methodological issue concerns a possible measurement bias which might be caused by recirculation of nitrous oxide. In our experiments, the rebreathing manoeuvres at 103 s did not lead to nitrous oxide gas recirculation at 46 s. Thus, time intervals of approximately 60 s between measurements point in time (and thus stage durations lasting 60 s) seem to be safe. However, for time intervals below 60 s, recirculation of nitrous oxide cannot be excluded. The third methodological consideration concerns the power stages, at which we performed the rebreathings (69, 77, and 85% peak power). These three power stages, corresponding to moderate to high exercise intensity levels before and after a hypothetic levelling off of cardiac output (Mortensen et al. 2005), were chosen because we speculated that a levelling off could influence the intra-stage adaptation of cardiac output and stroke volume. Furthermore, increments corresponding to 8% peak power were chosen because this value represents approximately 30 W increments in GXTs performed by experienced male road cyclists (Amann et al. 2004).

We conclude that during standard GXTs, the determination of cardiac output and stroke volume by Innocor^{TM} inert gas rebreathing at moderate to high submaximal power outputs in healthy, recreationally trained male cyclists is easily feasible for performance diagnostics purposes. Furthermore, our results indicate that during standard GXTs the haemodynamic response to power stage transitions from 54 to 69%, 69 to 77%, and 77 to 85% peak power is fast enough to ensure same values of cardiac output and stroke volume at 46 and 103 s on the higher stage. Due to this rapid adjustment of cardiac output, stage durations lasting less than 60 s are possible. Finally, since power increment sizes of 8 and 15% at submaximal levels seem not to affect intra-stage adjustments of cardiac output on the higher stages, these power increments may be well suited to assess submaximal cardiac output during GXTs.

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