Effects of rider position on continuous wave Doppler responses to maximal cycle ergometry

Warren D. Franke PhD, Christopher B. Betz MS and Reed H. Humphrey PhD

Laboratory for Exercise, Sport and Work Physiology, Division of Health, Physical Education and Recreation, Virginia Polytechnic Institute and State University, Blacksburg, Virginia, USA

Using 10 well-trained ($\dot{V}O_{2\text{peak}} = 60.6 \text{ ml kg}^{-1} \text{ min}^{-1}$) college age cyclists and continuous wave Doppler echocardiography, peak acceleration (PkA) and velocity (PkV) of blood flow in the ascending aorta, and the stroke velocity integral (SVI) were assessed to determine if rider position influenced the central haemodynamic responses to graded maximal cycle ergometry. Cyclist position was determined by hand placement on the uprights (UPRI) or drops (DROP) of conventional handlebars or using aerodynamic handlebars (AHB). All subjects consistently achieved a peak workload of 300 W. The Doppler variables did not differ significantly between rider positions at each stage of the maximal exercise tests but did change in response to increasing workloads. PkA was significantly ($P < 0.05$) greater at workloads $\geq 240 \text{ W}$ versus $\leq 120 \text{ W}$. PkV increased significantly ($P < 0.05$) up to 180 W and then reached a plateau. SVI increased to a workload of 120 W and then progressively declined, becoming significantly ($P < 0.05$) less at 300 W. For each stage, neither submaximal $\dot{V}O_2$, $\dot{V}_I$ nor heart rate (HR) differed significantly between each trial. These results suggest that rider position does not affect the physiological response to maximal bicycle ergometry as responses to each position are similar.

Keywords: Peak acceleration, peak velocity, stroke velocity integral, peak oxygen consumption, minute ventilation

In bicycling, optimal performance is dependent upon an interaction between physiological and biomechanical factors. For example, altering the saddle height¹, proper application of force to the pedals² and the use of toe clips all can play a role in efficient cycling. However, air resistance, or drag, is the greatest hindrance to achieving and maintaining high velocities on a bicycle as it constitutes over 90% of the force retarding a cyclist³. While drafting behind other cyclists can decrease oxygen consumption as much as 39%⁴, alternative methods of reducing drag are being constantly sought after.

Recently, an aerodynamic handlebar (AHB) has been developed that permits the cyclist to assume a more tucked position than previously possible with traditional handlebars. As a result, the rider's

aerodynamic profile is reduced and, consequently, his drag. In this position, with the rider's torso nearly parallel to the ground, wind resistance is decreased 20% at racing velocities³. As demonstrated best by Greg LeMond's last-stage victory in the 1989 Tour de France, marked improvements in performance are possible when using these handlebars. However, it is unclear if these performance improvements are due solely to enhanced aerodynamics or if a physiological mechanism is involved.

Research investigating alterations in physiological parameters due to alterations in rider position has yielded inconsistent results. Some studies report significant cardiorespiratory differences with differing rider positions⁵, while others do not⁶. These studies used conventional handlebars. Similar research using AHB has been limited but suggests that no cardiorespiratory differences exist between AHB and conventional handlebars^{7,8}. In all these studies, no measurements were made of central cardiovascular function, e.g. cardiac output or indices of left ventricular function. Thus, due to the few studies done and the limited variables measured in these studies, it remains uncertain if changing rider position results in an altered haemodynamic response to cycle exercise.

The differing haemodynamic responses to upright and supine exercise are detectable with Doppler echocardiography⁹. In determining if the physiological response to cycling is altered with an altered rider position, Doppler echocardiography may therefore be a more sensitive tool than those used to date. Consequently, we investigated the responses of the Doppler echocardiographic indices of central cardiovascular function to altered rider position in cyclists performing graded maximal cycle ergometry. In order to supplement the earlier investigations, we also measured the cardiorespiratory responses to this exercise.

Subjects and methods

Ten men volunteers of mean(s.e.m.) age 22(1.1) years, mean(s.d.) height 178.35(2.13) cm, and mean(s.d.) weight 73(2) kg were used in this study. All were trained cyclists and competitive racers. Subjects were fully informed of the experimental protocols and gave signed informed consent.

Address for correspondence: Warren D. Franke PhD, 246 PEB, Iowa State University, Ames, Iowa 50011, USA

© 1994 Butterworth-Heinemann Ltd
0306-3674/94/010038-05

Protocols

Subjects underwent three bouts of graded exercise using a mechanically braked bicycle ergometer (Bodyguard 990, Oglænd DBS AS, Sandnes, Norway); each bout was separated by a minimum of 48 h. During each bout, the subject initiated exercise at a workload of 60 W using a cadence of 60 r.p.m. The workload was increased by 60 W every 3 min until the subject was unable to maintain the appropriate cadence. The highest oxygen consumption observed was considered to be the peak $\dot{V}O_2$ ($\dot{V}O_{2peak}$). Total and minute inspiratory ($\dot{V}_I$) ventilations were measured with a Parkinson-Cowan meter (Instrumentation Associates, New York, New York, USA). Gas fractions were determined using Applied Electrochemistry S-3A and CD-3A analysers (Ametek, Pittsburgh, Philadelphia, USA) for oxygen and carbon dioxide, respectively. Before and after each test, the Parkinson-Cowan meter was checked for accuracy, while the analysers were calibrated using commercially available standardized gases.

Using a CM₅ lead configuration, heart rate (HR) was monitored continuously via an oscilloscope. Blood pressure (BP) was measured using the auscultatory method. The peak acceleration (PkA) and peak velocity (PkV) of blood ejected from the left ventricle into the ascending aorta were measured using a stand-alone continuous wave Doppler echocardiograph operating at 3 MHz (EXERDOP, Quinton Instruments, Seattle, Washington, USA). Sampled every 5 ms, signals are band-pass filtered between 480 Hz and 11.5 kHz; blood flow is then measured as modal velocity with PkA calculated internally as the greatest increase between consecutive measures. With this device, PkV can be measured in the range of 0.2 to 2.5 m sec⁻¹ while PkA is determined in the range of 2 to 99 m s⁻². This device has been described in detail elsewhere^{10,11}. The Doppler measure of stroke velocity integral (SVI) was also determined. An index of cardiac output (RelQ) was calculated as the product of HR and SVI. This measure has been shown to be representative of relative changes in Q within, but not between, subjects^{12,13}.

During the maximal exercise bouts, HR, gas fractions (F_eO₂, F_eCO₂) and $\dot{V}_I$ were recorded at the end of each minute. At 1 min 30 s into each 3-min stage, the technician positioned the nonimaging Doppler transducer at the suprasternal notch with the ultrasonic beam interrogating the ascending aorta. Blood flow velocity data were then collected from 1 min 45 s to 2 min 05 s. Blood pressure and an overall rating of perceived exertion were measured in the last minute of each stage.

Each of the three bouts of exercise differed only in the posture assumed by the rider. In one bout, the subject placed his hands on the upper portion, or uprights, of conventional handlebars (UPRI). In another session, the subject placed his hands on the lower portion, or drops, of the handlebars (DROP). In both these positions, the subject was required to maintain elbow extension throughout the test. In the third bout, the subject assumed a more tucked or crouched position using the AHB (Scott USA, Sun Valley, Idaho, USA) (AERO). With the AHB, the

subject placed his elbows on the cushioned uprights of the handlebars with his forearms directed in front of him. The order of these three bouts was randomized.

To standardize these rider positions, the handlebar uprights were consistently placed 2 inches below a plane level with the top of the saddle. Before the beginning of the first exercise bout, individual saddle heights were determined and kept constant throughout the study.

Statistics

Doppler data for each stage and each subject were considered acceptable when greater than 60% of the pulses measured were within limits based upon a floating average. For each pulse, this floating average was derived by an internal microprocessor from the mean of the preceding eight accepted beats. Only Doppler data meeting these guidelines were analysed statistically. Supine Doppler measures were obtained before each of the three bouts. The mean value from these three measures was used in comparisons with the resting values obtained when the cyclist was seated on the cycle ergometer. Differences between supine resting, and resting measures obtained when the subjects were in each of the three cycle positions, were analysed using one-way analysis of variance (ANOVA). If significant differences were identified, they were located using the Tukey *post hoc* procedure. Trial effects were analysed using a two-way (trial by workload) repeated measures ANOVA. An α level of 0.05 was chosen *a priori* as the level of significance. All data are presented as mean(s.e.m.).

Results

Time-to-test termination (mean(s.e.m.) = 14.6(0.2 min)), and $\dot{V}O_{2peak}$ (mean(s.e.m.) = 60.6(0.2) ml kg⁻¹ min⁻¹) did not differ significantly ($P > 0.05$) between trials. Neither $\dot{V}_I$ nor submaximal $\dot{V}O_2$, expressed as the percentage of $\dot{V}O_{2peak}$ achieved in the second minute of each stage, differed significantly between trials. Neither HR nor RelQ presented significant intertrial differences. Mean arterial pressure (estimated as (diastolic BP + one-third pulse pressure)) also did not differ between trials.

Significant differences were found between supine resting and resting Doppler variables obtained when the subjects were positioned on the ergometer. PkV, PkA and SVI were significantly less in the UPRI position than in the supine position. When in the DROP position, only PkA was significantly less. None of the three Doppler variables differed significantly between supine and AERO rest positions. These resting data are presented in Figure 1.

Nonsignificant ($P > 0.05$) intertrial differences were noted for PkA, PkV, and SVI. However, these variables did change significantly with the increase in workload. At or above 240 W, PkA was significantly higher than at workloads less than 180 W. PkV increased significantly up to a workload of 180 W, then reached a plateau, and did not differ significantly for the remainder of the tests. SVI increased up to 120 W and then declined progressively. These mean results are depicted in Figures 2–4.

Figure 1. Alterations in the resting Doppler variables peak acceleration (PkA), peak velocity (PkV), and stroke velocity integral (SVI) with changes in cyclist position. □, supine; ▨, upright; ▤, drop; ▩, aero (mean(s.e.m.)) * $P < 0.05$; + $P < 0.06$; two-way analysis of variance)

Figure 2. Alterations in peak acceleration (PkA) due to varying rider position and increasing workload. □, supine; ▨, upright; ▤, drop; ▩, aero. Workloads with different letters differ significantly ($P < 0.05$; two-way analysis of variance)

Figure 3. Changes in peak velocity (PkV) due to varying rider position and increasing workload. Symbols are as in Figure 2

Figure 4. Changes in stroke velocity integral (SVI) due to varying rider position and increasing workload. Symbols are as in Figure 2

Discussion

The lack of any significant intertrial differences in the Doppler echocardiographic variable responses strongly suggests that rider position, using either conventional handlebars or AHB, does not affect the haemodynamic responses to bicycling. The cardiorespiratory variables studied here did not differ significantly between trials which supports this contention.

It has long been recognized that stroke volume and cardiac output are lower at rest in the upright rather than the supine posture¹⁴. These differences are usually attributed to an enhanced preload in the supine position. Our Doppler echocardiographic results support this finding of differences in central cardiovascular function at rest. A comparison of the supine rest to the UPRI rest data indicates a significantly ($P < 0.05$) higher PkA, PkV and SVI

response in the former. These three measures approached significance ($P < 0.06$) during supine rest compared to DROP rest.

Our resting findings are similar to others in that Daley and co-workers⁹ noted higher PkV and mean acceleration during supine rest compared with standing rest. Manyari and Kostuk¹⁵ noted smaller left ventricular end-systolic volumes in the sitting rather than supine position. As PkA¹⁶ and PkV¹⁷ are insensitive to preload, our findings suggest that a greater inotropic response is seen in the supine posture rather than in the upright. Note that none of the Doppler variables differed significantly between AERO rest and supine rest. Therefore, at least during rest, the central cardiovascular response to the AERO position most closely resembles that found in the supine position.

Workload-induced alterations in PkA, PkV and SVI were consistent across trials. PkA was significantly ($P < 0.05$) greater at workloads at or above 240 W compared to workloads less than 180 W. PkA at 180 W was significantly greater than PkA at 60 and 120 W. These results are consistent with findings of a decreased end-systolic volume at higher exercise intensities^{18,19}. This decrease is probably due to an increased inotropic state, as PkA is sensitive to inotropic changes^{9,16}.

Following increases at the lower exercise intensities, SVI began to decline once the workload reached 180 W. At 50–60% $\dot{V}O_{2peak}$, SV is considered to have reached a plateau²⁰ and, therefore, chronotropic increases are responsible for further increases in $\dot{Q}$. For our subjects, exercise at 180 W was 55% $\dot{V}O_{2peak}$. Thus, the SVI decline seen at exercise workloads at or above 180 W probably reflects a maximized stroke volume. This SVI decline with increasing exercise intensities, at higher intensities, has been demonstrated elsewhere²¹.

PkV showed a significant stage-by-stage increase up to a workload of 180 W after which it essentially levelled off. Other researchers found that PkV continues to increase up to peak exercise intensities with maximal supine cycle ergometry¹³ and maximal exercise using a treadmill²². PkV is a sensitive indicator of overall left ventricular function under varied preload, HR and inotropic conditions¹⁷. As SVI peaked at about the same time PkV did, our results support the notion that the Frank–Starling mechanism plays a lesser role than the chronotropic response in maintaining $\dot{Q}$ at higher exercise intensities¹⁸.

When expressed as a percentage of rest, the PkV values derived here are between those cited for supine and upright exercise. Supine exercise to 125 W was associated with a 137% increase¹³ while Daley and co-workers⁹ found a PkV increase of 149% with supine cycle ergometry. In the latter study, the subjects achieved an increase to 181% that of rest with treadmill walking. This figure is similar to the 173% found by Mehta and colleagues²¹. In this study the increase was 156%.

The exercise Doppler data presented here are similar to those data found elsewhere for exercise in the upright position. Two groups^{21,23} have evaluated

the Doppler responses to maximal treadmill walking. Both groups noted that PkV plateaued at about 70% estimated $\dot{V}O_{2peak}$. PkA also increased linearly with HR, while SVI increased initially and then reached a plateau²³ or declined²¹ once HR exceeded about 130 beats min^{-1} .

Daley and colleagues⁹ investigated the PkA, SVI and mean acceleration responses to supine bicycle ergometer exercise and treadmill walking. They noted significant increases in all three variables with exercise in either position. However, values obtained at peak exercise did not differ significantly between exercise modes. Their results are similar to ours and, due to the greater difference in postures assumed by the subjects, supports further the lack of difference in aortic blood flow velocity responses in differing cycling positions.

Research conducted on subjects in the semisupine posture may be more applicable to the present study. Weiss and colleagues²⁴ noted that subjects exercising to a maximum of 150 W, while in a 20°-head-up tilt, experienced a significant increase in left ventricular end-diastolic dimension (LVEDD) only at peak exercise while left ventricular end-systolic dimension (LVESD) did not change. Elsewhere, subjects capable of exercising to 250 W, also in a 20°-head-up tilt, experienced no change in LVEDD but a 15% decrease in LVESV²⁵. This posture probably compares most closely to the AERO position used in the present study.

Exercise in a 60°-upright position may be more representative of exercise in the DROP position. During graded maximal exercise in this position, LVEDV did not decline significantly while LVESV decreased significantly compared with rest²⁶. Kelbaek and colleagues²⁷ found LVEDV to increase slightly at lower workloads and return to near-rest values as the subjects approached maximal exercise. In their study, LVESV decreased significantly with increasing work. Using subjects in a 70°-upright position, other researchers found a significant decline between resting and immediate post-exercise LVEDV but no change in LVESV²⁸.

The present study and the cited literature present evidence strongly suggesting that body position has no real haemodynamic effect on bicycling performance. Neither cardiorespiratory nor continuous wave Doppler measures of aortic blood flow velocity are affected as a result of altering rider position. The literature cited suggests that neither LVEDV nor LVESV are strongly influenced by differences in body position under moderate-to-high intensity exercise conditions. Moreover, this absence of an effect is reflected in the response of the Doppler variables PkA, PkV or SVI measured under exercise conditions. Thus, the present study suggests that improved performances associated with the use of AHB must be due solely to an improved aerodynamic efficiency. Perhaps as important, the present study suggests that AHB do not hinder the physiological performance of the rider. Future research in this area should, consequently, be directed towards perfecting the AHB in order to optimize this component of bicycling.

Acknowledgements

The authors thank Matthew S. Hickey and J. Shane Curren for their technical assistance. Parts of this manuscript have appeared previously in abstract form (*Med Sci Sports Exerc* 1990; 22: S101).

References

- Nordeen-Snyder K. The effect of bicycle seat height variation upon oxygen consumption and lower limb kinematics. *Med Sci Sports Exerc* 1977; 9: 113–7.
- Ericson MO, Nisell R. Efficiency of pedal forces during ergometer cycling. *Int J Sports Med* 1988; 9: 118–22.
- Kyle CR, Burke E. Improving the racing bicycle. *Mechanical Engineering* 1984; 106: 34–45.
- McCole SD, Anderson R, Conte JC, Claney K, Hagberg JM. Interaction between aerodynamics and energy expenditure in competitive cycling. *Med Sci Sports Exerc* 1989; 21(Suppl.): S-9 (abstract).
- Faria I, Dix C, Frazer C. Effect of body position during cycling on heart rate, pulmonary ventilation, oxygen uptake and work output. *J Sports Med* 1978; 18: 49–56.
- Dal Monte A, Severini V, Angella F. Studio di alcuni valori spirografici in relazione alle posizioni fipiche del ciclismo agonistico. *Medicini Dello Sport* 1966; 6: 744.
- Dorsa PB, Brilla LR. Physiological responses in cyclists to drop versus aero handlebars. *Med Sci Sports Exerc* 1989; 21(Suppl.): S-51 (abstract).
- Shapiro AR, Otto RM, Wygand J, Shaughnessy S, St. George K. The effect of handlebar position upon cycling efficiency. *Med Sci Sports Exerc* 1989; 21(Suppl.): S-50 (abstract).
- Daley PJ, Sagar KB, Wann LS. Doppler echocardiographic measurement of flow velocity in the ascending aorta during supine and upright exercise. *Br Heart J* 1985; 54: 562–7.
- Labovitz AJ, Pearson AC, Chaitman BR. Doppler and two-dimensional echocardiographic assessment of left ventricular function before and after dipyridamole stress testing for detection of coronary artery disease. *Am J Cardiol* 1988; 62: 1180–5.
- Sabbah HN, Przybylski J, Albert DE, Stein PD. Peak aortic blood acceleration reflects the extent of left ventricular ischemic mass at risk. *Am Heart J* 1987; 113: 885–90.
- Elkayam U, Gardin JM, Berkley R, Hughes CA, Henry WL. The use of Doppler flow velocity measurement to assess the hemodynamic response to vasodilators in patients with heart failure. *Circulation* 1983; 67: 377–83.
- Gardin JM, Kozlowski J, Dabestani A, Murphy M, et al. Studies of Doppler aortic flow velocity during supine bicycle exercise. *Am J Cardiol* 1986; 57: 327–32.
- Stenberg J, Astrand P-O, Ekblom B, Royce J, Saltin B. Hemodynamic response to work with different muscle groups, sitting and supine. *J Appl Physiol* 1967; 22: 61–70.
- Manyari DE, Kostuk WJ. Left and right ventricular function at rest and during bicycle exercise in the supine and sitting positions in normal subjects and patients with coronary artery disease. *Am J Cardiol* 1983; 51: 36–42.
- Noble MIM, Trenchard D, Guz A. Left ventricular ejection in conscious dogs: measurement and significance of the maximum acceleration of blood from the left ventricle. *Circ Res* 1966; 19: 139–47.
- Wallmeyer K, Wann LS, Sagar KB, Kalbfleisch J, Klopfenstein HS. The influence of preload and heart rate on Doppler echocardiographic indexes of left ventricular performance: comparison with invasive indexes in an experimental preparation. *Circulation* 1986; 74: 181–6.
- Plotnick GD, Becker LC, Fisher ML, Gerstenblith G, et al. Use of the Frank-Starling mechanism during submaximal versus maximal upright exercise. *Am J Physiol* 1986; 251: H1101–H1105.
- Stein RA, Michielli D, Diamond J, Horwitz B, Krasnow N. The cardiac response to exercise training: echocardiographic analysis at rest and during exercise. *Am J Cardiol* 1980; 46: 219–25.
- Astrand P-O, Cuddy TE, Saltin B, Stenberg J. Cardiac output during submaximal and maximal work. *J Appl Physiol* 1964; 19: 268–74.
- Mehta N, Boyle G, Bennett D, Gilmour S, et al. Hemodynamic response to treadmill exercise in normal volunteers: an assessment by Doppler ultrasonic measurement of ascending aortic blood velocity and acceleration. *Am Heart J* 1988; 116: 1298–307.
- Bryg RJ, Labovitz AJ, Mehdirad AA, Williams GA, Chaitman BR. Effect of coronary artery disease on Doppler-derived parameters of aortic flow during upright exercise. *Am J Cardiol* 1986; 58: 14–19.
- Harrison MR, Smith MD, Nissen SE, Grayburn PA, DeMaria AN. Use of exercise Doppler echocardiography to evaluate cardiac drugs: effects of propranolol and verapamil on aortic blood flow velocity and acceleration. *J Am Coll Cardiol* 1988; 11: 1002–9.
- Weiss JL, Weisfeldt ML, Mason SJ, Garrison JB, Livengood SV, Fortuin NJ. Evidence of Frank-Starling effect in man during severe semisupine exercise. *Circulation* 1979; 59: 655–61.
- Anderson K, Vik-Mo H. Role of the Frank-Starling mechanism during maximal semisupine exercise after oral atenolol. *Br Heart J* 1982; 48: 149–155.
- Wyns W, Melin JA, Vanbutsels RJ, DeCoster PM, Steels M, Piret L, Detry J-M. R. Assessment of right and left ventricular volumes during upright exercise in normal men. *Eur Heart J* 1982; 3: 529–36.
- Kelbaek H, Christensen NJ, Godtfredsen J. Left ventricular volumes during graded upright exercise in healthy untrained subjects. *Clin Phys* 1988; 8: 51–6.
- Thompson WR, Shapiro J, Thompson DL, Bulawa B. Comparison of ventricular volumes in normal and post-myocardial infarction subjects. *Med Sci Sports Exerc* 1987; 19: 430–5.