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Stroke work progressively increased until maximal exercise in long distance runners, but plateaued at submaximal exercise in sprinters

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Abstract The possible difference in the pattern of cardiac work and stroke work response during exercise between long-distance runners and sprinters remains unclarified. The present study was conducted to elucidate the difference in these responses during exercise between long-distance runners and sprinters. A total of 11 female college athletes (5 long-distance runners and 6 sprinters) performed incremental bicycle exercise testing with respiratory gas exchange measurement. Cardiac index and stroke volume index were continuously measured during exercise by a thoracic impedance method (Physioflow). Cardiac work index (CWI) and stroke work index (SWI) were calculated as the product of cardiac index and mean blood pressure, and the product of stroke volume index and mean blood pressure, respectively. CWI progressively increased from rest to maximal exercise both in long-distance runners and sprinters. SWI progressively increased until maximal exercise in long-distance runners (at 150W: 89.5 ± 3.3 vs. at maximal exercise: 109.3 ± 6.8 g·m·m⁻², $p < 0.005$), but plateaued at 150W in sprinters (at 150W: 107.9 ± 7.6 vs. at maximal exercise: 109.8 ± 6.7 g·m·m⁻², $p = \text{N.S.}$). In conclusion, SWI progressively increased until maximal exercise in long-distance runners, but plateaued at submaximal exercise in sprinters. Judging from SWI response during exercise, long-distance runners might have a superior cardiac function compared to sprinters.

Keywords : arterio-venous oxygen content difference, stroke work, endurance athlete, exercise

Introduction

Cardiac output and stroke volume are indices of cardiac reserve. Endurance sports athletes, such as long-distance runners, have been reported to have an increased heart volume and superior cardiac function compared to healthy controls¹⁻⁴). Although several previous studies measured cardiac output and/or stroke volume during exercise in long-distance runners⁵⁻⁹), there has only been one study which measured cardiac output during exercise in both long-distance runners and sprinters⁷). In that study, only cardiac output at 20, 40 and 60% of exercise intensity was measured; and thus, it is still unknown whether cardiac output and stroke volume at intensities above 60% maximal oxygen uptake ($\dot{V}O_2 \text{ max}$) differ between long-

distance runners and sprinters.

Since cardiac work, the triple product of stroke volume, heart rate (HR), and mean blood pressure, is the rate of doing work by the ventricle, cardiac work represents the power of the ventricle. On the other hand, another heart mechanical parameter is stroke work¹⁰), which is a function of both stroke volume and mean blood pressure, expressing the external work of the left ventricle¹¹). Since cardiac work and stroke work reflect afterload, cardiac work and stroke work can serve as more clinically useful indices of cardiac performance than cardiac output and stroke volume, respectively¹²). Although cardiac output and stroke volume during exercise have been previously investigated, only several studies have measured cardiac work and/or stroke work during exercise in healthy sub-

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Acronyms: $\dot{V}O_2$ = oxygen uptake, $C(a-v)O_2$ = arterio-venous oxygen content difference, CWI = cardiac work index, SWI = stroke work index, HR = heart rate

jects^{4,13-16}), in patients with heart failure^{13,17,18}), and those with ischemic heart diseases¹⁹). As for athletes, there has only been one study, and it only measured cardiac work at 45% and 70% $\dot{V}O_2$ max in endurance trained long-distance bicyclists and runners¹⁴). Therefore, in athletes, it remains unknown whether cardiac work and/or stroke work progressively increase until maximal exercise or not, and whether cardiac work and/or stroke work during exercise differ between athletes of different sports.

There is conflicting evidence as to whether stroke volume progressively increases until maximal exercise or not in healthy subjects, and athletes^{4-6,20}). Several reports indicated that the stroke volume of endurance trained athletes progressively increased until maximal exercise, and that the stroke volume plateaued at submaximal exercise in untrained subjects^{5,6}). Endurance training can induce cardiac adaptations, such as increased diastolic filling⁵), and changes in left ventricular cavity dimensions²⁰), leading to a progressive increase in stroke volume until maximal exercise in endurance-trained athletes^{5,20}). However, stroke work, but not stroke volume, defines the area within the pressure-volume loop and directly reflects cardiac function¹²). It has been hypothesized that endurance-trained athletes can use the Frank-Starling mechanism to a greater extent than non-athletes during incremental exercise⁵). Therefore, stroke work might increase progressively until maximal exercise in athletes who have superior cardiac function.

We hypothesized that stroke work progressively increases until maximal exercise in long-distance runners, but that it progressively increases until submaximal exercise, and then plateaus in sprinters. The present study was conducted to elucidate whether stroke work during incremental exercise until maximal exercise differs between long-distance runners and sprinters.

Methods

Subjects. Eleven female college athletes (5 long-distance runners and 6 sprinters) performed symptom-limited bicycle exercise testing. Their age was 20.9 ± 0.3 years old (long-distance runners: 20.6 ± 0.5 vs. sprinters: 21.2 ± 0.4 years old, $p = \text{N.S.}$, $1-\beta = 0.12$), body height was 160.8 ± 1.4 cm (long-distance runners: 158.5 ± 1.4 vs. sprinters: 162.8 ± 2.0 cm, $p = 0.13$, $1-\beta = 0.33$), body weight was 53.6 ± 1.7 kg (long-distance runners: 51.0 ± 1.8 vs. sprinters: 55.9 ± 2.5 kg, $p = 0.16$, $1-\beta = 0.28$), body surface area was 1.55 ± 0.03 m² (long-distance runners: 1.50 ± 0.02 vs. sprinters: 1.59 ± 0.04 m², $p = 0.10$, $1-\beta = 0.38$), and body mass index was 20.7 ± 0.5 kg · m⁻² (long-distance runners: 20.3 ± 0.8 vs. sprinters: 21.1 ± 0.7 kg · m⁻², $p = \text{N.S.}$, $1-\beta = 0.10$). They were not elite athletes, but participated in club activities five days a week and 3-4 hours a day. The 100 m best track records of those sprinters were about 12.1-12.7 seconds. They had no diseases including pectus excavatum. This study was approved by the Ethical Committee of Japan Women's College of Physical Education (Approved

No. 2008-20) and written informed consent was obtained from all subjects before participation in the study.

Exercise protocol and expired gas analysis. Before the study, subjects were asked to perform a familiarization exercise test with expired gas analysis, and then an incremental exercise test using a ramp protocol on an electromagnetically braked upright cycle ergometer (Aerobike 800, Combi, Tokyo, Japan) at least 2 hours after a meal. The exercise protocol and gas exchange analysis were performed according to our modified methods, as was previously described in detail²¹⁻²³). In brief, after a 2-minutes rest on the cycle ergometer, exercise was started at 20W for a 4-minute warm up and work rate was then increased in 1-W increments every 3 seconds. Blood pressure was measured by a doctor using a mercury sphygmomanometer (Yamasu No. 620, Kenzmedico, Saitama, Japan) every minute. Expired gases were measured continuously in all subjects on a breath-by-breath basis using an expired gas analyzer (AE-280, Minato Medical Science, Osaka, Japan). Ventilatory parameters, including oxygen uptake ($\dot{V}O_2$), carbon dioxide output, minute ventilation, the ratio of minute ventilation to carbon dioxide output and end-tidal carbon dioxide pressure were calculated. The subjects were encouraged to continue when leg fatigue or dyspnea set in order to ensure that all subjects reached maximum exertion. The $\dot{V}O_2$ max was defined as the highest $\dot{V}O_2$ value at attainment of a plateau of $\dot{V}O_2$ with increasing intensity, and respiratory exchange ratio >1.10 ²⁴).

Cardiac output measurement. During cardiopulmonary exercise testing, stroke volume and cardiac output were measured in all subjects by the impedance method, as previously described elsewhere in detail²⁵⁻²⁸). In brief, an impedance cardiography device (Physioflow type PF05L1, Manatec Biomedical, Paris, France) was used to determine HR, stroke volume, and cardiac output at rest and during exercise. Two sets of electrodes, one electrode transmitting and the other sensing, were applied above the supraclavicular fossa at the left base of the neck and along the xiphoid, respectively. Another set of two electrodes was used to monitor a single electrocardiogram (V1/V6 position). With this impedance device, a first evaluation of stroke volume was calculated during a calibration procedure based on 30 consecutive heart beats, recorded in the resting condition. The Physioflow measures changes in transthoracic impedance in response to an administered electrical current during cardiac ejection to calculate stroke volume. During the data-acquisition phase, the variation of parameters was analyzed and compared with those obtained during the calibration procedure. The Physioflow calculated cardiac output and stroke volume continuously at every heart beat, and these calculations made with Physioflow over a 30-second period were averaged. We previously investigated the reproducibility of stroke volume during exercise testing by the same method

in 3 healthy male subjects (Age: 22.0 ± 1.5 years, Body height: 173.3 ± 2.7 cm, Body weight: 69.0 ± 3.1 kg, Body surface area: 1.83 ± 0.05 m², Body mass index: 22.9 ± 0.6 kg·m⁻²), and the coefficient of variation was 1.8% (unpublished data).

Derived parameters. Arterio-venous oxygen content difference ($C[a-v]O_2$), the difference between arterial oxygen content and venous oxygen content was calculated using the Fick principle: $C(a-v)O_2 = \dot{V}O_2 \cdot \text{cardiac output}^{-1}$.

The cardiac index (CI) and stroke volume index (SVI) were calculated using the equation: cardiac index (CI) = cardiac output (CO) · body surface area⁻¹ (BSA⁻¹), and stroke volume index (SVI) = stroke volume (SV) · body surface area⁻¹ (BSA⁻¹), respectively. Body surface area (BSA) was calculated by the formula of Haycock: Body surface area = $0.024265 \cdot \text{body weight}^{0.5378} \cdot \text{body height}^{0.3964}$.

Cardiac work index (CWI) was calculated using the equation: $CWI = \text{cardiac index} \cdot \text{mean blood pressure} \cdot 451^{-1}$, where mean blood pressure is (systolic blood pressure + 2 · diastolic blood pressure) · 3⁻¹ (17). Stroke work index (SWI) was calculated using the equation: $SWI =$

$0.0136 \cdot \text{stroke volume index} \cdot \text{mean blood pressure}^{12}$.

Statistics. Differences between the means at each time point in the two groups were performed by two-way analysis of variance for repeated measures followed by Tukey's (honest significant difference) tests. Comparison for the variation in a parameter in relation to another parameter was performed by paired t-test. A value of probability <0.05 was considered significant. All data were shown as the mean ± standard error. Statistical analyses were performed with JMP 7.0.1 software (SAS Institute, Cary, NC, USA). Also, statistical power (1-β error probability) analysis was performed with G*Power 3²⁹.

Results

The maximal work load was higher in long-distance runners than in sprinters (long-distance runners: 236 ± 10 vs. sprinters: 197 ± 6 W, $p < 0.01$, $1-\beta = 0.87$). $\dot{V}O_2$ at rest was not significantly different both in long-distance runners and sprinters (long-distance runners: 4.6 ± 0.1 vs. sprinters: 4.5 ± 0.3 ml·kg⁻¹·min⁻¹, $p = \text{N.S.}$, $1-\beta = 0.06$, Table 1), but $\dot{V}O_2$ max was higher in long-distance run-

Table 1. Summary of Hemodynamic Variables and Oxygen Uptake in Long Distance Runners and Sprinters at Rest and Maximal Exercise

	Long distance runners		Sprinters	
	Rest	Maximal Ex	Rest	Maximal Ex
$\dot{V}O_2$ (ml·kg ⁻¹ ·min ⁻¹)	4.6±0.1	49.9±1.5***	4.5±0.3	40.1±1.6
$C(a-v)O_2$ (ml·100ml ⁻¹)	4.9±0.1	14.0±0.7*	4.6±0.3	11.5±0.6
CI (l·min ⁻¹ ·m ⁻²)	3.2±0.2	12.2±0.6	3.5±0.2	12.3±0.8
SVI (ml·m ⁻²)	48±3	68±4	48±2	68±4
HR (beats·min ⁻¹)	68±4	179±3	72±1	181±4
DP	7184±608	30857±970	7613±330	31766±727
TPR (dyn·s·cm ⁻⁵)	1360±96	531±37	1176±39	509±42
O ₂ pulse (ml)	3.49±0.25	14.16±0.73*	3.47±0.18	12.17±0.46
SWI (g·m·m ⁻²)	51.7±3.2	109.3±6.8	52.4±2.8	109.8±6.7
CWI (W·m ⁻²)	0.57±0.04	3.19±0.19	0.62±0.04	3.23±0.19

$\dot{V}O_2$ indicates oxygen uptake; $C(a-v)O_2$, arterio-venous oxygen content difference; CI, cardiac index; SVI, stroke volume index; HR, heart rate; DP, double product; TPR, total peripheral resistance; SWI, stroke work index; CWI, cardiac work index; Ex, exercise. Values are mean ± SE. * $p < 0.05$, *** $p < 0.005$ vs. sprinters.

ners than in sprinters (long-distance runners: 49.9 ± 1.5 $\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, range 44.8–53.3 vs. sprinters: 40.1 ± 1.6 $\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, range 34.3–44.6, $p < 0.005$, $1-\beta = 0.99$). Both in long-distance runners and sprinters, there was a linear increase in $\dot{V}\text{O}_2$ as a function of work rate (Fig. 1a). $\dot{V}\text{O}_2$ was not significantly different during submaximal exercise in both groups.

$\text{C(a-v)}\text{O}_2$ at rest was not significantly different in both long-distance runners and sprinters, but $\text{C(a-v)}\text{O}_2$ at maximal exercise was higher in long-distance runners than in sprinters (long-distance runners: 14.0 ± 0.7 vs. sprinters: 11.5 ± 0.6 $\text{ml} \cdot 100\text{ml}^{-1}$, $p < 0.05$, $1-\beta = 0.71$). Also, $\text{C(a-v)}\text{O}_2$ during submaximal exercise tended to be higher in long-distance runners than in sprinters when compared at the same work rate, although it did not reach statistical significance (at 100 W, long-distance runners: 11.8 ± 0.8

vs. sprinters: 9.3 ± 0.8 $\text{ml} \cdot 100\text{ml}^{-1}$, $p = 0.052$, $1-\beta = 0.52$; at 150 W, long-distance runners: 13.3 ± 0.9 vs. sprinters: 10.7 ± 1.0 $\text{ml} \cdot 100\text{ml}^{-1}$, $p = 0.082$, $1-\beta = 0.42$; Fig. 1b).

The cardiac index (CI) progressively increased from rest to maximal work rate in long-distance runners (at rest vs. at 20 W, $p < 0.005$, $1-\beta = 1.00$; at 20 W vs. at 50 W, $p < 0.001$, $1-\beta = 1.00$; at 50 W vs. at 100 W, $p < 0.001$, $1-\beta = 1.00$; at 100 W vs. at 150 W, $p < 0.005$, $1-\beta = 1.00$; at 150 W vs. at maximal exercise, $p < 0.001$, $1-\beta = 1.00$) and in sprinters (at rest vs. at 20 W, $p < 0.001$, $1-\beta = 1.00$; at 20 W vs. at 50 W, $p < 0.001$, $1-\beta = 1.00$; at 50 W vs. at 100 W, $p < 0.001$, $1-\beta = 1.00$; at 100 W vs. at 150 W, $p < 0.001$, $1-\beta = 1.00$; at 150 W vs. at maximal exercise, $p < 0.05$, $1-\beta = 0.89$; Fig. 1c). Cardiac index at rest was not significantly different in long-distance runners and sprinters. Although cardiac index at 150 W was lower in

Fig. 1 Oxygen uptake (a), arterio-venous oxygen content difference (b) and cardiac index (c) at rest, 20W, 50W, 100W, 150W and maximal exercise in relation to work rate in long-distance runners and sprinters. Bars show the mean value $\pm$ standard error (SE). * $p < 0.05$, *** $p < 0.005$ vs. sprinters.

- (a) a main effect of group, $p = 0.19$; a main effect of work rate, $p < 0.001$; group $\times$ work rate interaction, $p < 0.001$.
 (b) a main effect of group; $p = \text{N.S.}$, a main effect of work rate; $p < 0.001$, group $\times$ work rate interaction; $p < 0.05$.
 (c) a main effect of group; $p = 0.11$, a main effect of work rate; $p < 0.001$, group $\times$ work rate interaction; $p < 0.01$.

long-distance runners than in sprinters, cardiac index at maximal exercise was not significantly different between the two groups (long-distance runners: 12.2 ± 0.6 vs. sprinters: 12.3 ± 0.8 $\text{l} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$, $p = \text{N.S.}$, $1-\beta = 0.05$).

The stroke volume index (SVI) almost progressively increased from rest to maximal exercise in long-distance runners (at rest vs. at 20 W, $p < 0.001$, $1-\beta > 0.99$; at 20 W vs. at 50 W, $p < 0.01$, $1-\beta = 0.85$; at 50 W vs. at 100 W, $p = 0.076$, $1-\beta = 0.85$; at 100 W vs. at 150 W, $p = \text{N.S.}$, $1-\beta = 0.08$; at 150 W vs. at maximal exercise, $p < 0.05$, $1-\beta > 0.99$). On the other hand, stroke volume index progressively increased from rest to 150 W and plateaued after that in sprinters (at rest vs. at 20 W, $p < 0.001$, $1-\beta = 1.00$; at 20 W vs. at 50 W, $p < 0.005$, $1-\beta > 0.99$; at 50 W vs. at 100 W, $p < 0.01$, $1-\beta > 0.99$; at 100 W vs. at 150 W, $p < 0.01$, $1-\beta = 0.92$; at 150 W vs. at maximal exercise, $p = \text{N.S.}$, $1-\beta = 0.18$; Fig. 2a). Stroke volume index (SVI) at rest was not significantly different in both long-distance runners and sprinters. Stroke volume index during exercise was not significantly different between the two groups either (at maximal exercise, long-distance runners: 68 ± 4 vs. sprinters: 68 ± 4 $\text{ml} \cdot \text{m}^{-2}$, $p = \text{N.S.}$, $1-\beta = 0.05$).

HR at rest was not significantly different in both long-distance runners and sprinters (long-distance runners: 68 ± 4 vs. sprinters: 72 ± 1 $\text{beats} \cdot \text{min}^{-1}$, $p = \text{N.S.}$, $1-\beta = 0.06$). HR was lower in long-distance runners than in sprinters at 100 W and 150 W, but maximal HR was not significantly different in the two groups (long-distance runners: 179 ± 3 vs. sprinters: 181 ± 4 $\text{beats} \cdot \text{min}^{-1}$, $p = \text{N.S.}$, $1-\beta = 0.06$, Fig. 2b).

Systolic blood pressure (SBP) and mean blood pressure

(MBP) at rest, 20 W, 50 W, 100 W and maximal exercise were not significantly different in long-distance runners and sprinters, but those at 150 W were lower in long-distance runners than in sprinters (systolic blood pressure [SBP], long-distance runners: 153 ± 4 vs. sprinters: 165 ± 2 mmHg, $p < 0.05$, $1-\beta = 0.61$; mean blood pressure [MBP], long-distance runners: 108 ± 3 vs. sprinters: 117 ± 2 mmHg, $p < 0.05$, $1-\beta = 0.59$; Table 2). Diastolic blood pressure (DBP) at rest and during exercise was not significantly different in long-distance runners and sprinters.

CWI progressively increased until maximal exercise in long-distance runners (at rest vs. at 20 W, $p < 0.005$, $1-\beta = 1.00$; at 20 W vs. at 50 W, $p < 0.001$, $1-\beta = 1.00$; at 50 W vs. at 100 W, $p < 0.001$, $1-\beta = 1.00$; at 100 W vs. at 150 W, $p < 0.001$, $1-\beta = 1.00$; at 150 W vs. at maximal exercise, $p < 0.001$, $1-\beta = 1.00$). In sprinters, CWI also progressively increased until maximal exercise (at rest vs. at 20 W, $p < 0.001$, $1-\beta = 1.00$; at 20 W vs. at 50 W, $p < 0.001$, $1-\beta = 1.00$; at 50 W vs. at 100 W, $p < 0.001$, $1-\beta = 1.00$; at 100 W vs. at 150 W, $p < 0.001$, $1-\beta = 1.00$; at 150 W vs. at maximal exercise, $p < 0.05$, $1-\beta > 0.99$; Fig. 3a). CWI at rest was not significantly different in long-distance runners and sprinters. CWI at 50 W, 100 W, and 150 W was lower in long-distance runners than in sprinters (at 50 W, long-distance runners: 1.02 ± 0.07 vs. sprinters: 1.21 ± 0.06 $\text{W} \cdot \text{m}^{-2}$, $p < 0.05$, $1-\beta = 0.45$; at 100 W, long-distance runners: 1.46 ± 0.06 vs. sprinters: 1.96 ± 0.14 $\text{W} \cdot \text{m}^{-2}$, $p < 0.05$, $1-\beta = 0.77$; at 150 W, long-distance runners: 2.11 ± 0.07 vs. sprinters: 2.87 ± 0.18 $\text{W} \cdot \text{m}^{-2}$, $p < 0.01$, $1-\beta = 0.91$), but CWI at maximal exercise was not significantly different between the two groups (long-distance runners:

Fig. 2 Stroke volume index (a) and heart rate (b) at rest, 20W, 50W, 100W, 150W and maximal exercise in relation to work rate in long-distance runners and sprinters. Bars show the mean value $\pm$ SE. * $p < 0.05$ vs. sprinters.

(a) a main effect of group; $p = \text{N.S.}$, a main effect of work rate; $p < 0.001$, group $\times$ work rate interaction; $p = 0.17$.

(b) a main effect of group; $p = 0.15$, a main effect of work rate; $p < 0.001$, group $\times$ work rate interaction; $p < 0.05$.

Table 2. Summary of Blood Pressure in Long Distance Runners and Sprinters at Rest, 20 W, 50 W, 100 W, 150 W and Maximal Exercise

	Long distance runners			Sprinters		
	Systolic	Diastolic	Mean	Systolic	Diastolic	Mean
Rest	106±4	68±4	80±4	105±2	68±3	80±3
20 W	114±4	70±1	85±2	113±4	73±2	86±3
50 W	124±5	72±3	90±4	128±4	76±4	94±3
100 W	142±4	73±5	96±4	145±5	84±4	105±4
150 W	153±4*	86±3	108±3*	165±2	93±2	117±2
Maximal Ex	174±5	90±3	118±3	178±3	90±4	119±3

Ex, exercise. Values are mean ± SE. * $p < 0.05$ vs. sprinters.

Fig. 3 Cardiac work index (a) and stroke work index (b) at rest, 20W, 50W, 100W, 150W and maximal exercise in relation to work rate in long-distance runners and sprinters. Bars show the mean value ± SE. * $p < 0.05$, ** $p < 0.01$ vs. sprinters. (a) a main effect of group; $p = 0.05$, a main effect of work rate; $p < 0.001$, group × work rate interaction; $p < 0.001$. (b) a main effect of group; $p = \text{N.S.}$, a main effect of work rate; $p < 0.001$, group × work rate interaction; $p < 0.05$.

3.19 ± 0.19 vs. sprinters: $3.23 \pm 0.19 \text{ W} \cdot \text{m}^{-2}$, $p = \text{N.S.}$, $1-\beta = 0.06$). The variation in CWI in relation to work rate from rest to 150 W ($[\text{CWI}_{150 \text{ W}} - \text{CWI}_{\text{rest}}] \cdot 150^{-1}$) was lower in long-distance runners than in sprinters (long-distance runners: 0.0103 ± 0.0002 vs. sprinters: $0.0150 \pm 0.0010 \text{ m}^{-2}$, $p < 0.005$, $1-\beta = 0.95$). The variation in CWI in relation to work rate from 150 W to maximal exercise ($[\text{CWI}_{\text{max}} - \text{CWI}_{150 \text{ W}}] \cdot [\text{W}_{\text{max}} - 150]^{-1}$) tended to be higher in long-distance runners than in sprinters (long-distance runners: 0.0126 ± 0.0013 vs. sprinters: $0.0075 \pm 0.0017 \text{ m}^{-2}$, $p = 0.059$, $1-\beta = 0.53$). The ratio of the variation

in CWI from 150 W to maximal exercise in relation to that from rest to 150 W ($[\text{CWI}_{\text{max}} - \text{CWI}_{150 \text{ W}}] \cdot [\text{W}_{\text{max}} - 150]^{-1} \cdot [\text{CWI}_{150 \text{ W}} - \text{CWI}_{\text{rest}}] \cdot 150$) was higher in long-distance runners than in sprinters (long-distance runners: 1.22 ± 0.13 vs. sprinters: 0.51 ± 0.13 , $p < 0.005$, $1-\beta = 0.93$).

SWI progressively increased until maximal exercise in long-distance runners (at rest vs. at 20 W, $p < 0.005$, $1-\beta = 1.00$; at 20 W vs. at 50 W, $p < 0.005$, $1-\beta = 1.00$; at 50 W vs. at 100 W, $p < 0.05$, $1-\beta = 1.00$; at 100 W vs. at 150 W, $p < 0.005$, $1-\beta = 1.00$; at 150 W vs. at maximal exercise,

$p < 0.005$, $1-\beta = 1.00$). On the other hand, SWI increased until 150 W, and after that it plateaued in sprinters (at rest vs. at 20 W, $p < 0.001$, $1-\beta = 1.00$; at 20 W vs. at 50 W, $p < 0.001$, $1-\beta = 1.00$; at 50 W vs. at 100 W, $p < 0.005$, $1-\beta = 1.00$; at 100 W vs. at 150 W, $p < 0.001$, $1-\beta = 1.00$; at 150 W vs. at maximal exercise, $p = \text{N.S.}$, $1-\beta = 0.05$; Fig. 3b). SWI at rest and during exercise was not significantly different in long-distance runners and sprinters (at maximal exercise, long-distance runners: 109.3 ± 6.8 vs. sprinters: $109.8 \pm 6.7 \text{ g} \cdot \text{m} \cdot \text{m}^{-2}$, $p = \text{N.S.}$, $1-\beta = 0.05$). The variation in SWI in relation to work rate from rest to 150 W ($[\text{SWI}_{150 \text{ W}} - \text{SWI}_{\text{rest}}] \cdot 150^{-1}$) was lower in long-distance runners than in sprinters (long-distance runners: 0.253 ± 0.011 vs. sprinters: $0.370 \pm 0.037 \text{ g} \cdot \text{W}^{-1} \cdot \text{m}^{-1}$, $p < 0.05$, $1-\beta = 0.70$). The variation in SWI in relation to work rate from 150 W to maximal exercise ($[\text{SWI}_{\text{max}} - \text{SWI}_{150 \text{ W}}] \cdot [\text{W}_{\text{max}} - 150]^{-1}$) was higher in long-distance runners than in sprinters (long-distance runners: 0.226 ± 0.060 vs. sprinters: $0.019 \pm 0.068 \text{ g} \cdot \text{W}^{-1} \cdot \text{m}^{-1}$, $p < 0.05$, $1-\beta = 0.52$). The ratio of the variation in SWI from 150 W to maximal exercise in relation to that from rest to 150 W ($[\text{SWI}_{\text{max}} - \text{SWI}_{150 \text{ W}}] \cdot [\text{W}_{\text{max}} - 150]^{-1} \cdot [\text{SWI}_{150 \text{ W}} - \text{SWI}_{\text{rest}}]^{-1} \cdot 150$) was higher in long-distance runners than in sprinters (long-distance runners: 0.91 ± 0.24 vs. sprinters: 0.09 ± 0.18 , $p < 0.05$, $1-\beta = 0.70$).

Discussion

There have only been a few studies which investigated stroke volume (SV) during exercise in endurance-trained athletes, indicating that the stroke volume of endurance-trained athletes progressively increased until maximal exercise^{5,6}. However, no reports have examined stroke volume during exercise in both long-distance runners and sprinters. The present study showed that stroke volume almost progressively increased until maximal exercise in long-distance runners, but it increased until submaximal exercise (150 W), and then plateaued in sprinters.

Stroke work reflects the work done by the left ventricle to eject a volume of blood (i.e., stroke volume) into the aorta, and is used to assess ventricular function¹². The use of stroke work rather than stroke volume, has several advantages to evaluate cardiac function: 1. stroke work defines the area within the pressure-volume loop. 2. stroke work includes measurements of both systolic and diastolic performance. 3. stroke work contains the major variables that alter cardiac performance, such as HR, preload, and afterload¹². In the present study, SWI progressively increased from rest to maximal work rate during incremental exercise in long-distance runners, but SWI progressively increased from rest to submaximal exercise (150 W) and plateaued after that in sprinters. This discrepancy of the response of SWI might be due to differences in cardiac function during exercise. Endurance-trained athletes such as long-distance runners develop eccentric hypertrophy², and have compliant and

distensible ventricles which enable a steeper slope of the Frank-Starling curve³ and lower left ventricular filling pressure to stroke volume during exercise⁴. In endurance-trained athletes, an enhanced diastolic function allows for a more complete filling during the later stages of vigorous exercise⁵. Stickland et al.⁴ demonstrated, in a study with a small number of subjects, that not only stroke volume, but also stroke work progressively increased until maximal exercise in those with high $\dot{V}\text{O}_2 \text{ max}$, but not in those with low $\dot{V}\text{O}_2 \text{ max}$. Thus, the continuous increase in SWI until exhaustion of long-distance runners in the present study could be explained by superior left ventricular function, such as greater left ventricular filling, which allows them to increasingly use the Frank-Starling mechanism throughout incremental exercise.

Cardiac work is the product of stroke work and HR, which is the equivalent of the triple product of stroke volume, mean blood pressure and HR. Because cardiac work is the rate of doing work (work per unit time) by the ventricle, it represents the power of the ventricle. In subjects with extremely high blood pressure during exercise and in subjects with slightly high blood pressure, when cardiac output is the same between the two, cardiac work in the former is greater than that in the latter. In patients with heart failure, it was reported that peak cardiac work measured during exercise had an association with adverse outcomes, and that peak cardiac work added independent prognostic information to peak $\dot{V}\text{O}_2$ ³⁰. Thus, cardiac work is a more clinically important physiological marker than cardiac output. The present study for the first time showed that CWI progressively increased until maximal exercise in long-distance runners and in sprinters, but the increase progressively diminished from 150 W to maximal exercise in sprinters. This finding suggested that long-distance runners might have superior cardiac function compared to sprinters.

$\dot{V}\text{O}_2 \text{ max}$ was higher in long-distance runners than in sprinters in the present study, which was in accordance with previous reports^{7,31}. Because $\dot{V}\text{O}_2$ is the product of cardiac output and $\text{C(a-v)}\text{O}_2$, the finding of the higher $\dot{V}\text{O}_2 \text{ max}$ in long-distance runners of the present study, compared to that in sprinters, was not due to increased cardiac output, but rather to increased $\text{C(a-v)}\text{O}_2$ at maximal exercise.

$\text{C(a-v)}\text{O}_2$ during exercise mainly reflects the oxygen uptake of exercising muscles during exercise, because most of the blood is delivered to working muscles during exercise. Endurance training can cause not only an increase in activated muscle volume, especially slow type (type I)³², but also an increase in oxygen diffusion capacitance in muscle tissue³³, mitochondria density³⁴ and oxidative enzyme activity³⁵, and a significant increase in capillary density and the percentage of type I fibers⁷. These mechanisms could induce a further increase in muscle oxygen consumption, resulting in a greater degree of decrease in the mixed venous oxygen pressure and a greater increase

in $C(a-v)O_2$ during exercise in long-distance runners, which was observed in the present study. Furthermore, 15-day cessation of training in middle-distance runners can cause a decrease in $\dot{V}O_2$ max and a similar percentage decrease in $C(a-v)O_2$ at maximal exercise⁹, which is consistent with the findings in the present study.

The cardiac output, determined by the impedance method of Physioflow, was validated against the direct Fick method during exercise in healthy subjects³⁶ and patients²⁵. Charloux et al.²⁵ showed that cardiac output measured by this impedance method was as accurate as that obtained with the Fick method during exercise in patients with sleep apnea syndrome and chronic obstructive pulmonary disease. Also, the stroke volume values obtained by the Physioflow were reproducible, and the coefficient of variation was 1.8% in our previous findings and 3.6% in other reports³⁷. Furthermore, the superior aspect of the Physioflow is that it calculates stroke volume independently from baseline impedance values^{25,37}, and that sweating and dehydration cannot affect stroke volume³⁷. Therefore, the measurement of cardiac output and stroke volume by this impedance method is a useful noninvasive method to assess the hemodynamic profile of subjects during exercise, and there have been many studies which evaluated cardiac output and stroke volume during exercise using this method^{24-26,36-38}.

Limitations for the present study are as follows. Firstly, although cycling is not specific for runners, a bicycle exercise test was used in the present study, because bicycle exercise is suitable to quantify the external work of subjects, and because blood pressure can be more accurately measured by bicycle exercise test than treadmill test. Secondly, the subjects of the present study were not elite athletes, and their $\dot{V}O_2$ max was relatively low. Thus, if those were elite athletes, cardiac work and/or stroke work at maximal exercise might become higher in long-distance runners than those in sprinters. Thirdly, the sample size was relatively small to compare cardiac work and stroke work during exercise between long-distance runners and sprinters. Despite these limitations, we found that the patterns of CWI, and especially SWI during exercise, were different between long-distance runners and sprinters. Fourthly, the protocol of the present study was ramp incremental exercise of 1-W increments every 3 seconds, which is used in many investigations^{21-23,27,28,39}. By using this protocol, the subtle difference of $\dot{V}O_2$ max and maximal exercise intensity can be detected. On the other hand, a steady state of respiratory and cardiac parameters is not obtained.

Manual auscultatory methods by an experienced doctor or investigators have been the most popular and most widely accepted method of blood pressure measurement during exercise⁴⁰, and have been employed in many studies investigating exercise blood pressure responses⁴¹⁻⁴³. Although intra-arterial blood pressure measurements are more accurate during exercise testing than manual

auscultatory methods, intra-arterial blood pressure measurements have seldom been used because of technical difficulties and risks to subjects⁴⁴. Also, during exercise testing, an automated blood pressure measurement system is more affected by movement artifacts than the manual auscultatory method^{40,45}. Therefore, we used the manual auscultatory method by an experienced doctor in the present study. However, if blood pressure is measured using a manual auscultatory method or automated blood pressure measurement during exercise, it requires 10-20 seconds to measure blood pressure. Therefore, it happens that the timing to measure systolic blood pressure becomes faster than that of measuring diastolic blood pressure. In all subjects of the present study, the value of systolic blood pressure during exercise may probably be 1-2 mmHg lower than the true value, and thus, this phenomenon should be considered in the present study.

From the findings of the present study, it is useful to measure the pattern of SWI and CWI during exercise in order to examine whether cardiac function is superior or inferior in athletes, especially track and field athletes. When SWI and/or CWI progressively increase until maximal exercise in athletes, it may mean they have superior cardiac function. Thus, it may be possible to apply for exercise training, such as an incremental bicycle exercise test, as an exercise prescription in track and field and other athletes.

Taken together, in the present study, SWI progressively increased until maximal exercise in long-distance runners, but not in sprinters, suggesting that long-distance runners might have superior cardiac function compared to sprinters.

Conflict of Interests

The authors declare that they have no conflict of interests.

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