ORIGINAL RESEARCH ARTICLE

Open Access

Relation between derived cardiovascular indices, body surface area, and blood pressure/ heart rate recovery among active and inactive Nigerian student

Patrick Ayi Ewah[1*] and Adetoyeje Y. Oyeyemi[2]

Abstract

Background: The ease in the computation of derived cardiovascular indices, such as mean arterial pressure (MAP), rate pressure product (RPP), and pulse pressure (PP), makes them attractive for use in making clinical decisions for patient management in resource-deprived environments. This study sought to determine the relationship between these indices and heart rate/blood pressure drops during recovery state among physically active and inactive individuals following submaximal exercise.

Results: This quasi-experimental study conveniently sampled 105 apparently healthy male subjects aged 18–35 years of the University of Maiduguri. Intergroup categorization was executed by IPAQ. The derived indices were calculated using heart rate and blood pressure measurement while the body surface area (BSA) was determined using height and weight. The subjects performed a submaximal exercise test using a bicycle ergometer. Data analysis includes descriptive statistic, Pearson correlation, Student t-test, analysis of covariance, and multiple linear regression. The data was analyzed using SPSS version 25.0 at a significance of $p < 0.05$. The mean BSA and resting PP, MAP, and RPP were 1.84 ± 0.16 m^2, 41.23 ± 7.57 mmHg, 85.92 ± 9 mmHg, and 8266.45 ± 1404.05 respectively. The resting RPP of the physically inactive subjects was significantly higher than that of the active (8742.71 ± 1496.31 Vs 7790.18 ± 1131.59, $p = 0.00$); however, the active subject had a higher resting MAP than the inactive (87.91 ± 7.98 Vs 83.93 ± 9.59, $p = 0.03$). A significant negative relationship was found between the RPP and the Absolute/percent recovery HR ($r = -0.23$, $p = 0.02$ Vs $r = -0.34$, $p = 0.00$). However, for the PP and absolute recovery SBP it was significantly positive ($r = 0.22$, $p = 0.03$) and also that of the MAP and absolute recovery SBP was positive ($r = 0.33$, $p = 0.00$). The best negative predictor of recovery HR was the RPP ($\beta = -0.45$, $p = 0.00$) while the MAP was the best positive predictor of recovery HR and SBP.

Conclusions: Overall, the physically active subjects coped better during the exercise than their inactive counterpart because of lower cardiac work and better blood perfusion to vital body organs. An inverse relationship was found between the RPP and absolute/percent recovery HR at 1 min post-exercise while a positive relationship was found between the PP and absolute drop in SBP/percent drop in DBP 1 min post-exercise. The best predictor of recovery in HR and blood pressure was the RPP.

*Correspondence: payiewah8@yahoo.com

[1] Department of Physiotherapy, Faculty of Allied Medical Sciences, University of Calabar, Calabar, Nigeria
Full list of author information is available at the end of the article

Background

The derived cardiovascular parameters are important clinical tools that complement direct measures such as heart rate (HR) and blood pressure in assessing cardiovascular system status and response to a stimulus. These parameters include pulse pressure (PP), mean arterial pressure (MAP), cardiac index (CI), and rate pressure product (RPP). They are important cardiovascular indices that are derived and are easily computed, making them attractive for use in decision-making for care in resource-challenged environments. The average pressure in the artery obtained in one cardiac cycle is labeled the MAP, and it is an indication of blood perfusion and a function of cardiac output (CO), central venous pressure (CVP), systemic vascular resistance (SVR), and other factors (diameter of blood vessel, nitric oxide NO), endothelin, and baroreceptor reflex) [1]. The MAP is calculated using the equation MAP = (CO. SVR) +CVP or $P_{dias}+1/3$ ($P_{sys} - P_{dias}$), i.e., pressure at diastole plus a third of the difference between the pressure at systole and diastole [1].

An elevated MAP (average value at 60 mmHg) triggers an increase in the shearing forces on the wall of endothelial cells lining the blood vessel which causes NO and other vasodilation compounds to be synthesized with the resultant action of relaxation (vasodilation) of smooth muscle vasculature [2, 3]. On the reverse, a decrease in MAP triggers the release of endothelin with the opposite effect to NO, leading to vasoconstriction [4]. The MAP is also regulated by the autonomic nervous system through the baroreceptor reflex [2]. For example, communication to the solitary nucleus, situated on the dorsolateral area of the medulla oblongata of the brainstem by baroreceptors [2], determines the tone (whether parasympathetic or sympathetic) to lower or increase MAP according to the need of the body [5]. On the reverse side, an increase in baroreceptor stimulation with associated elevation in MAP causes the solitary nucleus to decrease sympathetic output and increase parasympathetic output with a subsequent drop in MAP and a resultant reduction in CO [2].

Likewise, a decrease in the activities of the baroreceptors with an associated decrease in MAP causes the solitary nucleus to reduce parasympathetic tone in favor of sympathetic tone which increases SVR and CO, with the MAP effectively increased [2]. The sympathetic tone is also upregulated during events such as exercise, vascular trauma, and emotional and psychological stress [2].

Furthermore, CO calculated by multiplying the SV and HR is of physiologic importance to the overall organism metabolism [6] and reflects the ability of the body to adjust to metabolism and workload increase. The PP correlates well with the SV and is calculated using the equation, $P_{ulse} = P_{sys} - P_{dias}$, i.e., the change in systolic and diastolic pressure [1].

An index often used to determine the uptake of oxygen by the myocardium is labeled the RPP and is simply calculated as HR_{rest} multiplied by the SBP [1]. It is also used to determine the energy needed for the proper functioning of the heart and how this energy is expended. The CI is used to show the relationship between how well a person's heart is functioning and the body surface area (BSA); it is also used to show how the CO and BSA are correlated [7–9]. The formula used to calculate the CI is $CI = \frac{CO}{BSA} = \frac{SV \times HR}{BSA}$ [9–11], as shown in the formulae: BSA, body surface area; SV, stroke volume; CO, cardiac output; CI, cardiac index; HR, heart rate and unit of measurement, $L/min/m^2$, i.e., liter per minutes per meter square. Measurement of CI between 2.6 and 4.2 [12] or 2.5 and 4.0 [13] $L/min/m^2$ at baseline has been reported to be normal, and values below and above this range are said to be abnormal [12, 13]. For example, a patient in cardiogenic shock may have a value below $1.8L/min/m^2$ [11]. The CI is, therefore, an essential clinical reading used to assess patients with various kinds of shock [13], seriously sick patients in intensive care [12], and those under the influence of anesthesia.

More so, using the measurement from height and weight, the BSA [meter squared; m^2] is calculated using "DuBois' equation: BSA in meter = (wt in kg) $^{0.425}$ x (ht in cm) $^{0.725}$ x 0.007184" [14].

The recovery HR has also been used to determine cardiovascular fitness and the chances of a person dying within 1 year. For instance, some researchers have reported that "a delayed drop in the HR a minute after stopping an exercise is a strong independent predictor of death within a year" [15, 16]. Similarly, systolic blood pressure recovery has also been utilized by clinicians as an important tool in diagnosing abnormalities in the cardiovascular system [15–17].

The non-invasive method used to estimate the derived cardiovascular parameter has been reported in some literature. For example, Alfie et al. [18] used an impedance cardiograph to determine the SV, MAP, CO, and PP. On the other hand, Marcus et al. [6] used cardiovascular magnetic resonance (similar to

Doppler measurement and bio-impedance), to assess the derived cardiovascular parameter such as CI and SV. The M-mode echocardiograms and Teichholz correction of the cube formula have been utilized to assess SV, interventricular septal/posterior wall thickness, and end-diastolic/systolic volume. Measurement of the SV and LV chamber volumes estimated by M-mode and Teichholz correction method has been shown to have a good correlation with Doppler-echocardiographic volume measurement and other invasive techniques [19–22]. These non-invasive techniques and other invasive methods, although reliable, require that the user should have advanced knowledge on how to use them and are tedious, time-consuming, risky, and expensive procedures. Determination of MAP, PP, and RPP by calculation is also reliable, simple, reproducible, and a less time-consuming method similar to the above-mentioned non-invasive and invasive tedious techniques. For example, assessment of the RPP is a readily available non-invasive means of ascertaining the VO_2max, and it is reliable, simple, and reproducible, serving a similar purpose as expensive non-invasive and invasive methods [23].

Previous studies on the relationship of some derived indices are available in the literature. For example, Marcus et al. [6] report that the CI of the healthy and athletic subjects was not different at rest; however, patients with congestive heart failure had lower CI at rest. de Simone et al. [24] reported that with the elevated size of the body, the CO and SV rate of change was higher in adults compared to children, while Shahraki et al. [25] found that the MAP/PP at rest, during exercise, and after exercise was more in athletic than in the non-athletic female subject and our larger study reported a higher drop in both the HR and blood pressure recovery in the physically active young adults than their sedentary counterpart following a bout of exercise [26]. There appears to be a gap in the existing literature on the relationship between the derived cardiovascular indices (resting PP, MAP, and RPP) and the recovery drop in blood pressure/HR after a removal of the stimulus of exercise and how this relates to BSA among physically active and inactive individuals, since we could not find any study with this report. More so, how this parameter predicts the recovery drop in blood pressure and HR is unexplored.

It is a general belief that the use of derived indices to assess cardiovascular status is not common among clinicians. As most practitioners in low-resource countries such as Nigeria often lament the absence of state-of-the-art instrumentations, the use of derived indices that are easily computed is not widespread. Furthermore, however, the relationship between MAP, PP, and RPP to BSA and the rate of drops in blood pressure and HR after the

removal of exercise stimulus is unexplored. This report is part of a larger study that compares the responses of physically active and physically inactive young adults [26]. Only the data on the derived indices and recovery cardiovascular parameters of HR and SBP are presented in this report. The primary purpose of this study is to determine the relationship between these indices and HR/blood pressure drops in recovery state among physically active and inactive individuals following submaximal exercise. The secondary purpose is to elucidate on some derived cardiovascular indices such as MAP, PP, RPP, and BSA and their clinical implications.

Methods

The methodology for this study is reported in detail elsewhere [26]. Briefly, this study using a quasi-experimental design conveniently sampled roughly 105 young adult male students who were physically active and inactive in the age range 18–35 years. The inclusion criteria were no history of hypertension, cardiac disease, smoking, and alcohol abuse/medication for any cardiac condition while the exclusion criteria were inability to walk, discrepancy length of leg, presence of ankle and knee pain, sprain or strain, or any other orthopedic problem. To estimate the level of physical activity of the subject in the last 7 days, the adapted IPAQ-SF was used and in terms of duration (minute/day) and frequency (day/week), it measures walking activity, sitting, moderate-intensity activity, and vigorous-intensity activity, to classify subject to the intergroup [27]. Using the adapted IPAQ-SF score of lower than 600 MET-minutes/week qualifies the subject as physically inactive while scores above 600 MET-minutes/week qualify them as physically active [28].

An approval to conduct this study was sought for and obtained from the University of Maiduguri Teaching Hospital, Research and Ethical Committee. Consent to participate in the study was also sorted from individual subjects and only those who were willing to partake were recruited. A preliminary instruction on what to do and not to do 24 h before the time when the experiment was conducted include no intake of alcohol, cola nut, cigarette, hard drugs, or energy drinks. The reason why the subjects were not to take these items was explained (the mentioned items are stimulants and can either natively or positively influence their cardiovascular parameters).

The experiment was carried out at the University of Maiduguri Teaching Hospital, in the department of physiotherapy gymnasium. A room within the gymnasium maintained at a temperature of 25° C was secured for this experiment. Before commencement of the experiment, the subjects were told to rest for 30 min in a sitting position, and 5–10 min into resting, measurement of baseline values such as blood pressure, HR, height, and

weight was obtained. The instrument used in data collection includes an electronic device for measuring blood pressure and HR (Model UB-512 by Life Source); a scale for weight and height made in Great Britain, Hospibrand model ZT 160, with a maximum weight capacity of 160kg was used to measure the weight and height of subjects; and a bicycle ergometer (mode/art; Din. EN 957/ 0783-700, max weight 110kg, Germany Kettler: HKS-selection ergometer EX1) for the submaximal exercise testing. A data capture form was utilized for recording the subject's data and a blackberry phone was employed to record time. Anthropometric variables were measured as per the guideline of WHO. The MAP was estimated from baseline measurement of blood pressure using the formula MAP$= P_{dias}+1/3$ $(P_{sys} - P_{dias})$, while the pulse pressure was calculated using the equation $P_{ulse} = P_{sys} - P_{dias}$, and the BSA and the RPP were estimated using DuBois' equation [14] and RPP= HRrest *SBP respectively.

After the baseline measurement and calculation, the subject then proceeds for the "submaximal exercise" test using a stationary bicycle. This test was carried out from 6 am up to 8 pm and subjects were at liberty to choose a suitable time within this period. Prior to the experiment, all subjects were dully instructed on how to carry out the test. The experiment was conducted in a stepwise manner; thus, there was an initial period of warm-up at 25-watt resistance for 2 min maintained 50–60 revolutions in cycle per minute. This was immediately progressed by increasing the resistance by 25 watts after every other minute to the exertion point of the individual subject (70% target age-predicted HRmax). The equation of Tanka et al. ("HR$_{max}$ = "208 minus (0.7 x age") was used to determine the HRmax [29]. The HR at exhaustion was labeled the peak HR. After this time, the HR was then monitored up to about 5 min after the removal of the exercise stimulus to determine the relative/absolute drops of blood pressure and HR.

The equations "(peak-HR – post-HR) / peak-HR x 100" and "(peak-BP – post-BP) / peak-BP x100" were used for this purpose [30–32]. The recovery derived indices were however, calculated by estimating the difference between the peak-and post-value or the percent of the value. For example, the recovery MAP was estimated using the eqation; (MAP$_{peak}$ − MAP$_{post}$) and (MAP$_{peak}$ − MAP$_{post}$) / MAP$_{peak}$ ×100) for the absolute and percent recovery respectively. The Borge scale was also used to monitor the hardness of the exercise on the subjects. Data analysis was done using SPSS version 25, and statistics of description such as frequency and mean were utilized to analyze the physical parameters of the subjects. The relationship between the MAP, RPP, PP, and recovery drops in HR and blood pressure to the BSA were analyzed using the Pearson correlation coefficient (data were normally distributed). The Student independent t-test was also used to ascertain any difference in the MAP, BSA, RPP, and PP among the physically active and inactive subjects. Factors that influence the blood pressure and HR recovery 1 min after removal of exercise stimulus were also determined using multiple linear regression. To adjust for pre-exercise differences in MAP and RPP among the intergroup, an analysis of covariance (ANCOVA) test was conducted. A $p < 0.05$ was used as a significant level.

Results

The subject's physical characteristics

The physical characteristics such the age, height, weight, blood pressure, HR, recovery blood pressure, and HR were part of a larger study reported elsewhere [26]. One hundred and two (102) apparently healthy "physically active" and inactive students in the age range 18–35 participated in this study. Twenty-three of the subjects (22.5%) were of age range between 18 and 22 years, 53.9% ($n = 55$) were of age range 23–27 years, and 23.5% ($n = 24$) were of age range 28–32 years. About 12.7% ($n = 13$) were underweight, 75.5% ($n = 77$) were normal weight, 9.8% ($n = 10$) were overweight, and 2% ($n = 2$) were obese. The mean $\pm$ SD of the BSA was 1.84 $\pm$ 0.16 m^2 (range, 1.5–2.33), while the mean PP was 41.23 $\pm$ 7.57 mmHg (range, 25–61). Also, the mean MAP and RPP were 85.92 $\pm$ 9 mmHg (range, 67–108 mmHg) and 8266.45 $\pm$ 1404.05 (range, 5411.24–12401.01) respectively.

Comparison of derived cardiovascular indices and body surface area among the physically active and inactive subjects

There was no significant difference ($p = 0.36$) between the BSA of the physically active (1.86 $\pm$ 0.16 m^2) and inactive (1.83 $\pm$ 0.16 m^2) subjects, albeit the BSA of the physically active was higher than their inactive counterparts. Similar findings were seen between the resting PP of physically active and inactive subjects (41.59 $\pm$ 7.88 mmHg, $p = -0.68$ Vs 40.87 $\pm$ 7.30 mmHg, $p = 0.63$). The resting RPP of the physically inactive (8742.71 $\pm$ 1496.31) subjects was significantly ($p = 0.00$) higher than that of active (7790.18 $\pm$ 1131.59) subjects, suggestive perhaps that the physically active group is coping better with the exercise with relatively lower cardiac work output compared to the inactive group. The resting MAP of the physically active subject was significantly higher than that of their inactive counterpart (87.91 $\pm$ 7.98 Vs 83.93 $\pm$ 9.59, $p = 0.03$), suggesting that the blood perfusion of tissues is better for the active group than the inactive group; the results for PP, MAP, and RPP at the peak of exercise and 1, 3, and 5 min post-exercise are also presented in Table 1.

Table 1 Comparison of derived cardiovascular indices and body surface area among physically inactive and physically active subjects

Variables	Physically inactive (*n*=51)	Physically active (*n*=51)	*t*-value	*F*-value	*p*-value
BSA	1.83±0.16 m²	1.86±0.16	−0.92		0.36
Rest PP	40.87±7.30 mmHg	41.59±7.88 mmHg	−0.48		0.63
Peak PP	48.18±12.33mmHg	55.33±15.04 mmHg		6.96	0.01
Rec PP 1	42.59±8.53 mmHg	46.94±10.33 mmHg	−2.23		0.02
Rec PP 2	42.80±10.12mmHg	45.45±13.36 mmHg	−1.13		0.26
Rec PP3	40.73±7.69 mmHg	43.02±11.18 mmHg	−1.21		0.23
Rest MAP	83.93±9.59 mmHg	87.91±7.98 mmHg	−2.28		0.03
Peak MAP	90.73±12.99mmHg	97.21±11.33 mmHg		2.78	0.09
Rec MAP 1	85.47±12.77 mmHg	89.98±10.01 mmHg	−1.98		0.05
Rec MAP 2	81.21±11.87 mmHg	85.60±7.89 mmHg	−2.20		0.03
Rec MAP 3	80.52±9.23 mmHg	84.46±8.83 mmHg	−2.20		0.03
Rest RPP	8742.71±1496.31	7790.18±1131.59	3.63		0.00
Peak RPP	13983.78±2904.78	16558.35±2972.10		42.77	0.00
Rec RPP 1	11022.65±2306.70	11167.90±2313.85	−0.32		0.75
Rec RPP 2	10229.55±1948.09	10166.73±1816.80	0.17		0.86
Rec RPP 3	9679.02±1595.94	9380.43±1529.97	0.96		0.34

Rest PP Resting pulse pressure, *peak PP* Peak pulse pressure, *rest MAP* Resting mean arterial pressure, *peak MAP* Peak mean arterial pressure, *rec PP1, 2, and 3,* pulse pressure 1, 3, and 5 min after exercise; *rec MAP 1, 2, 3,* mean arterial pressure 1, 3, and 5 min after exercise; *rec RPP 1, 2, 3,* rate pressure product 1, 3 and 5 min after exercise; NS, *p* > 0.05 not significant; *, *p* < 0.05 significant; peak PP, MAP, and RPP are based on analysis of covariance test

Table 2 Relationship between body surface area, derived cardiovascular indices, and recovery heart rate/blood pressure 1 min after removal of exercise stimulus

Variables	ARHR1		ARSBP1		ARDBP1		% RHR1		% SBP1		% DBP1	
	r	*p*-value	*r*	*p*-value	*r*	*p*-value	*r*	*p*-value	*r*	*p*-value	*r*	*p*-value
BSA	0.09	0.37	0.04	0.67	−0.12	0.22	0.17	0.09	−0.02	0.81	−0.09	0.33
RPP	**−0.23***	**0.02**	0.03	0.79	−0.10	0.32	**−0.34****	**0.00**	0.01	0.92	−0.05	0.62
PP	0.05	0.59	**0.22***	**0.03**	0.19	0.05	0.12	0.23	0.14	0.16	**0.20***	**0.04**
MAP	0.18	0.07	**0.33****	**0.00**	0.02	0.84	0.16	0.10	**0.28****	**0.00**	0.07	0.46

BSA Body surface area, *RPP* Rate pressure product, *PP* Pulse pressure, *MAP* Mean arterial pressure, *ARHR1* Absolute recovery heart rate at 1 min, *ARSBP1* Absolute recovery systolic blood pressure at 1 min, *ARDBP1* Absolute recovery diastolic blood pressure at 1 min, *%RHR1* Percent recovery heart rate at 1 min; *%RSBP1* Percent recovery systolic blood pressure at 1 min; *%RDBP1,* percent recovery diastolic blood pressure at 1 min; **correlation significant at 0.01; *correlation significant at 0.05

Relationships between the derived cardiovascular indices, body surface area, and absolute/percentage recovery heart rate/ blood pressure

Table 2 presents a negatively significant but tenuous relationship between the RPP and the absolute recovery HR at 1 min ($r = −0.23^*$, $p = 0.02$) and percent recovery HR at 1 min after exercise ($r = −0.34^{**}$, $p = 0.00$). There was also a tenuous positive significant relationship between the PP and absolute recovery SBP ($r = 0.22^*$, $p = 0.03$) and percent recovery DBP ($r = 0.20^*$, $p = 0.04$) at 1 min post-exercise. There was a tenuous positive significant relationship between the MAP ($r = 0.33^{**}$, $p = 0.00$) and percent recovery SBP (0.28^{**}, $p = 0.00$). The BSA showed no significant relationship with absolute recovery HR/ percent recovery HR, absolute recovery SBP/percent recovery SBP, and absolute recovery DBP/percent recovery DBP as shown in Table 2.

In Table 3, there was again a tenuous negative significant relationship between the RPP and absolute recovery HR3/percent recovery HR3 ($r = −0.20$, $p = 0.04$ Vs $r = −0.33$, $p = 0.00$) at 3 min post-exercise. Interestingly, there was no significant relationship between the PP and the recovery BP and HR at 3 min post-exercise. However, there was again a tenuous positive significant relationship between the MAP and absolute recovery SBP/percent recovery SBP at 3 min following the removal of exercise stimulus ($r = 0.25$, $p = 0.01$ Vs $r = 0.20$, $p = 0.05$).

In Table 4, the RPP shows significant tenuous negative relation only with the percent recovery HR at 5 min into the removal of exercise stimulus ($r = −0.28$, $p = 0.00$)

Table 3 Relationship between body surface area, derived cardiovascular indices, and recovery heart rate/blood pressure 3 min after removal of exercise stimulus

Variables	ARHR3		ARSBP3		ARDBP3		% RHR3		% SBP3		% DBP3	
	r	*p*-value	*r*	*p*-value	*r*	*p*-value	*r*	*p*-value	*r*	*p*-value	*r*	*p*-value
BSA	0.04	0.73	0.02	0.84	−0.08	0.44	0.14	0.18	−0.06	0.57	−0.09	0.54
RPP	**−0.20***	**0.04**	−0.11	0.27	−0.07	0.47	**−0.33****	**0.00**	−0.13	0.18	−0.06	0.54
PP	0.05	0.64	0.15	0.14	0.17	0.08	0.01	0.94	0.05	0.60	0.17	0.09
MAP	0.13	0.19	**0.25***	**0.01**	0.06	0.55	0.139	0.16	**0.20***	**0.05**	0.06	0.55

BSA Body surface area, *RPP* Rate pressure product, *PP* Pulse pressure, *MAP* Mean arterial pressure, *ARHR1* Absolute recovery heart rate at 3 min, *ARSBP3* Absolute recovery systolic blood pressure at 3 min, *ARDBP3* Absolute recovery diastolic blood pressure at 3 min; *%RHR3* Percent recovery heart rate at 3 min, *%RSBP3* Percent recovery systolic blood pressure at 3 min, *%RDBP3* Percent recovery diastolic blood pressure at 3 min; **correlation significant at 0.01; *correlation significant at 0.05

Table 4 Relationship between body surface area, derived cardiovascular indices, and blood pressure/heart rate recovery 5 min after removal of exercise stimulus

Variables	ARHR5		ARSBP5		ARDBP5		% RHR5		% SBP5		% DBP5	
	r	*p*-value	*r*	*p*-value	*r*	*p*-value	*r*	*p*-value	*r*	*p*-value	*r*	*p*-value
BSA	0.06	0.55	0.03	0.77	−0.10	0.34	0.17	0.09	−0.03	0.77	−0.10	0.30
RPP	−0.16	0.10	−0.01	0.95	−0.01	0.89	**−0.28****	**0.00**	−0.08	0.43	−0.04	0.70
PP	−0.06	0.53	0.18	0.06	0.07	0.48	−0.01	0.90	0.07	0.46	0.08	0.43
MAP	**0.21***	**0.03**	**0.23***	**0.02**	0.20	0.84	**0.26****	**0.01**	0.12	0.24	−0.01	0.95

BSA Body surface area, *RPP* Rate pressure product, *PP* Pulse pressure, *MAP* Mean arterial pressure, *ARHR5* Absolute recovery heart rate at 5 min, *ARSBP5* Absolute recovery systolic blood pressure at 5 min, *ARDBP5* Absolute recovery diastolic blood pressure at 5 min, *%RHR5* Percent recovery heart rate at 5 min, *%RSBP5* Percent recovery systolic blood pressure at 5 min, *%RDBP5* Percent recovery diastolic blood pressure at 5 min; **correlation significant at 0.01; *correlation significant at 0.05

Table 5 Influence of derived indexes on heart rate/blood pressure recovery

Variables	ARHR1		ARSBP1		ARDBP1		% RHR1		% SBP1		% DBP1	
	β	*p*-value	*β*	*p*-value	*β*	*p*-value	*β*	*p*-value	*β*	*p*-value	*β*	*p*-value
BSA	−0.01	0.92	−0.08	0.41	−0.20	0.05	0.04	0.62	−0.13	0.198	−0.18	0.08
RPP	**−0.45****	**0.00**	**−0.25***	**0.03**	−0.26	0.03	**−0.62****	**0.00**	−0.23	0.05	−0.22	0.06
PP	0.12	0.24	**0.22***	**0.03**	**0.29****	**0.01**	**0.23***	**0.02**	0.15	0.15	**0.28****	**0.01**
MAP	**0.38****	**0.00**	**0.41****	**0.00**	0.12	0.31	**0.40****	**0.00**	**0.39****	**0.00**	0.15	0.19

BSA Body surface area, *RPP* Rate pressure product, *PP* Pulse pressure, *MAP* Mean arterial pressure, *ARHR1* Absolute recovery heart rate at 1 min, *ARSBP1* Absolute recovery systolic blood pressure at 1 min, *ARDBP1* Absolute recovery diastolic blood pressure at 1 min, *%RHR1* Percent recovery heart rate at 1 min, *%RSBP1* Percent recovery systolic blood pressure at 1 min, *%RDBP1* Percent recovery diastolic blood pressure at 1 min; **correlation significant at 0.01; *correlation significant at 0.05

but not with other variables. However, there was a significant and also tenuous relationship between the MAP and the absolute recovery HR, absolute recovery SBP, and percent recovery HR: $r = 0.21$, $p = 0.03$; $r = 0.23$, $p = 0.02$, and $r = 0.26$, $p = 0.01$ respectively.

Predictors of recovery heart rate and blood pressure 1 min after exercise

Table 5 presents the factors that predict recovery, 1 min after the removal of the exercise stimulus.

For the absolute recovery HR1, the model was significant, F-ratio = 5.18, $p = 0.01$, and $R = 0.42$; hence, the model was 42% of the variance in absolute recovery HR1. For absolute recovery SBP, the model was significant, F-ratio = 5.06, $p = 0.01$, and $R = 0.42$; hence, the model was 42% of the variance in absolute recovery SBP. For the absolute recovery DBP, the model was significant, with F-ratio = 2.94, $p = 0.02$, and $R = 0.33$; hence, the model explained 33% of the variance in absolute recovery DBP.

For percent recovery HR1, the model was significant, F-ratio = 11.00, $p = 0.00$, and $R = 0.56$; hence, the model is 56% of the variance in percent recovery HR. For the percent recovery SBP, the model was significant, with F-ratio = 3.63, $p = 0.01$, and $R = 0.36$. The model is 36% of the variance in percent recovery SBP.

For the percent recovery DBP, the model was significant, F-ratio $= 2.56$, $p = 0.04$, and $R = 0.39$. The model is 39% of the variance in percent recovery DBP.

There was a modest negative significant influence of RPP on the absolute/percent recovery HR ($\beta = -0.45$, $= 0.00$ Vs $\beta = -0.62$, $p = 0.00$). Hence, the highest significant negative predictor of recovery HR at 1 min post-exercise was the RPP. This means that higher resting RPP leads to a low percent/absolute recovery HR (slower drops in HR) at 1 min after exercise. However, the RPP showed a tenuous negative significant influence on the absolute recovery SBP/DBP ($\beta = -0.25$; $p = 0.03$ Vs $\beta = -0.26$; $p = 0.03$), but not on the percent recovery blood pressure ($p = 0.05$ Vs $p = 0.06$; SBP/DBP). This indicates again that higher resting RPP was associated with slower recovery of blood pressure (slow drop in blood pressure) but not the percent recovery of blood pressure.

The PP showed a significant tenuous positive influence on absolute recovery SBP/DBP, percent recovery HR, and percent recovery DBP: $\beta = 0.22$, $p = 0.03$; $\beta = 0.29$, $p = 0.01$; $\beta = 0.23$, $p = 0.02$ and $\beta = 0.28$, $p = 0.01$ respectively, but not on the absolute recovery HR and percent recovery SBP ($p > 0.05$). This means that the higher resting PP was associated with faster recovery (faster drops) in the absolute recovery blood pressure and percent recovery HR/DBP at 1 min post-exercise.

Also, the MAP showed a modest positive influence on the absolute recovery HR/SBP, percent recovery HR/SBP: $\beta = 0.38$, $p = 0.00$; $\beta = 0.41$, $p = 0.00$; $\beta = 0.40$, $p = 0.00$ and $\beta = 0.39$, $p = 0.00$ respectively, but no influence on the recovery DBP ($p > 0.05$). No significant influence was found by the BSA on recovery drop in blood pressure and HR ($p > 0.05$). Hence, the MAP was the highest significant positive predictor of recovery HR and SBP. This indicates that higher MAP was associated with faster drops in recovery HR/SBP.

Discussion

The derived cardiovascular parameter such as the MAP, RPP, and PP are important measures used to determine the perfusion of blood to all the body tissue, oxygen uptake/energy requirement by the myocardium, and the body's ability to adjust to increasing workload/metabolism. The ease in the computation of these indices makes them very attractive for use in decision-making for patient management in the resource-deprived environment such as seen in countries with a low-middle level of income. Determining these indices by calculation is a reliable, simple, reproducible, and cheap method similar to very expensive and other non-invasive and risky invasive methods often employed to monitor and manage a patient in high-tech clinical settings.

The average BSA, resting RPP, resting PP, and resting MAP for this cohort of subjects were considered normal. The normal value of BSA for males and females are 1.90 m^2 and 1.60 m^2 respectively. The BSA is important because it is useful in drug dosage calculation (cytotoxic agent) and to determine derived cardiovascular indicators [33, 34]. The RPP is a major indicator of the energy demand of the heart, a normal value falls between 5000 and 10,000, and values above this put an individual at risk of developing heart disease [35–37]. The MAP is a major indicator of blood perfusion to the key organs like the kidney and heart, with normal values ranging from 70 to 100mmHg.

The BSA of the physically inactive subjects and that of the active subjects were similar in the present study. Similarities in resting PP among the intergroup as seen in this study contradict findings by Shahraki et al. with a report of significantly higher PP in the athletic than in the non-athletic subjects [25]. However, the baseline MAP was significantly more in the physically active than in the inactive subjects. These findings were consistent with that of Shahraki et al. [25]. However, the present study recruited age-matched male subjects, but the report of Shahraki et al. [25] was on female subjects.

Also, the resting RPP of the physically inactive subjects was significantly more than that of the active subject, and this indicates that the physically inactive subjects were at higher risk of developing heart disease. Values greater than 10,000 put a person at a greater risk of coming down with heart disease [35–37]. Normal baseline values for RPP have also been reported to fall between 7000 and 9000 [23].

Reduce resting RPP as seen in the physically active subjects in the present study indicates an increase in parasympathetic nervous activity and tone believed to be cardio-protective [38]. This means that the physically active subjects are safer with enhanced parasympathetic mediated cardioprotection than the physically inactive group [23].

This study also assessed the relationship between the derived indices (BSA, RPP, PP, and MAP) and the absolute/percent recovery of HR and blood pressure at 1, 3, and 5 min after the removal of the exercise stimulus.

There was a negative significant relationship between the resting RPP and absolute/percent recovery HR, and this indicates a relationship in the inverse direction between the resting RPP and the recovery HR 1 min after the removal of exercise stimulus but not the recovery blood pressure. A similar finding was seen between the resting RPP and the HR and blood pressure recovery 3 min post-removal of exercise stimulus. The resting RPP showed an inverse relationship again with the percent recovery HR at 5 min post-exercise; however, resting RPP

was not significantly related to the absolute recovery HR/blood pressure.

There was a positive significant relationship between the resting PP and absolute recovery SBP/percent recovery DBP 1 min post-exercise, but not on the recovery HR or percent recovery SBP or absolute recovery DBP. This indicates that the higher the resting PP, the higher the absolute recovery SBP/percent recovery DBP. However, there was no significant relationship between the PP and the recovery HR/blood pressure at 3–5 min post-exercise.

The resting MAP showed a significant positive relationship with the absolute/percent recovery SPB but not on the recovery HR or DBP at 1 to 3 min post-exercise. This also indicates that the higher the MAP, the higher the recovery SBP. Interestingly, we found a significant positive relationship between the MAP and the recovery HR at 5 min post-exercise.

A significant negative influence of the resting RPP on the absolute/percent recovery HR and absolute recovery SBP/DBP means that a higher RPP reduces the chances of the subject's HR and blood pressure recovering faster (lower change in recovery) 1 min after a bout of exercise. Higher values in the RPP put a person at risk of sustaining a heart disease [35–37], and lower/slower recovery in HR 1 min after the cessation of exercise has been reported to put an individual at risk of dying within 1 year [15, 16]. A positive significant influence of the resting PP on the absolute recovery SBP/DBP and percent recovery HR/DBP indicates that higher PP increases the absolute recovery SBP/DBP and percent recovery HR/DBP (higher change in recovery). Also, a positive significant influence of the resting MAP on the recovery of HR and SBP means that higher MAP increases the chances of the HR and SBP to recover faster 1 min after the removal exercise stimulus. Elevated MAP leads to an increase in shearing force on the blood vessel wall, and this favors the synthesis of NO with a net effect of relaxation (vasodilation) of the blood vessels perfusing the smooth muscle [2, 3]. The faster (higher change in recovery) the rate of recovery of HR and blood 1 min post-exercise removal signifies fitness. On the reverse side, it has been hypothesized by some researchers that slower drop-in blood and HR 1 min after a bout of exercise is an independent and powerful risk of predicting death in the space of 1 year and a useful marker during prognosis [15, 16].

Limitations and strengths of the study
Interpreting the result of this study should be done considering some limitations. First, the measurement of BSA, MAP, PP, and RPP was calculated using the measurement of blood pressure and HR and was not direct measures. Second, the inability to measure other derived indexes such as the CI, SV, and CO could not allow us to determine a relationship and influence of physical activity status on these measures on the recovery of blood pressure and HR. We recommend that further studies be carried out to determine the relationship and influence of these measures on the recovery drop in blood pressure and HR. Nevertheless, this study provided some insight into the relationship and influence of some derived cardiovascular parameters on the recovery of blood pressure and HR post-removal of exercise stimulus.

Conclusions
The present study found that the RPP at rest was significantly more in the physically inactive subject than in the active subject. Although no difference was found between the resting PP and BSA among the intergroup, there was a significantly higher difference in the MAP at rest in the physically active subjects than in their sedentary counterpart. There was also an inverse relationship between the RPP and absolute/percent recovery HR at 1 min post-exercise removal. Contrary-wise, a significant positive relationship was seen between the resting PP and absolute recovery SBP/percent recovery DBP 1 min post-exercise. The resting MAP also showed a significant relationship with the absolute/percent recovery SBP 1 min post-exercise. The highest negative predictor of absolute/percent recovery HR and absolute recovery SBP/DBP was the RPP while the highest positive predictor of recovery HR and SBP was the MAP.

Consent for publication

This is not applicable for this study.

Competing interests

The authors declare that they have no competing interests.

Author details

[1]Department of Physiotherapy, Faculty of Allied Medical Sciences, University of Calabar, Calabar, Nigeria. [2]Department of Medical Rehabilitation (physiotherapy), Faculty of Allied Health Sciences University of Maiduguri, Maiduguri, Nigeria.

References

1. Klabunde R. Cardiovascular physiology concepts. 2nd ed. Philadelphia': Lippincott Williams & Wilkins/Wolters Kluwer; 2012. p. 93–4.
2. DeMers D, Wachs D. Physiology, mean arterial pressure. (2021): [Updated 2021 Apr 21]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan Available from:https://www.ncbi.nlm.nih.gov/books/NBK538226/
3. Lundberg JO, Weitzberg E, Gladwin MT. The nitrate-nitrite-nitric oxide pathway in physiology and therapeutics. Nat Rev Drug Discov. 2008;7(2):156–67 [PubMed].
4. Trindade M, Oigman W, Fritsch NM. Potential role of endothelin in early vascular aging. Curr Hypertens Rev. 2017;13(1):33–40 [PubMed].
5. Andresen MC, Doyle MW, Jin YH, Bailey TW. (2001): Cellular mechanisms of baroreceptor integration at the nucleus tractus solitarius. Ann N Y Acad Sci. 2001;940:132–41 [PubMed].
6. Marcus C, Andersson R, Bloch KM, Steding-Ehrenborg K, Mosén H, Stahlberg F, et al. Cardiac output and cardiac index measured with cardiovascular magnetic resonance in healthy subjects, elite athletes, and patients with congestive heart failure. J Cardiovasc Magnet Resonance. 2012;14:51 http://www.jcmr-online.com/content/14/1/51.
7. Guyton AC, Hall JE. Textbook of medical physiology: 11th Ed. Philadelphia: Elsevier Saunders, Amsterdam; 2006. 106-116, 161- 232, 1060-1066
8. Robert SP, Justin LK. Cardiac catheterization: cardiovascular tests and procedure. In: Michael JS, editor. The Merck manual. 19th ed. Whitehouse Station; 2009.
9. Kovacs G, Avian A, Olschewski A, Olschewski H. Zero reference level for right heart catheterisation. Eur Respir J. 2013;42(6):1586–94. https://doi.org/10.1183/09031936.00050713.
10. Swan HJ, Ganz W, Forrester J, Marcus H, Diamond G, Chonette D. Catheterization of the heart in man with use of a flow-directed balloon-tipped catheter. New Engl J Med. 1970;283(9):447–51. https://doi.org/10.1056/NEJM197008272830902.
11. Reynolds HR, Hochman JS. Cardiogenic shock: current concepts and improving outcomes. Circulation. 2008;117(5):686–97. https://doi.org/10.1161/CIRCULATIONAHA.106.613596.
12. Barratt-Boyes BG, Wood EH. Cardiac output and related measurements and pressure values in the heart and associated vessels, together with an analysis of the hemodynamic response to the inhalation of high oxygen mixtures in healthy subjects. J Lab Clin Med. 1958;51:72–90.
13. Patel N, Durland J, Makaryus AN. Physiology, cardiac index. [Updated 2021 Sep 28]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK539905/
14. Du Bois D, Du Bois EF. A formula to estimate the approximate surface area if height and weight be known. Nutrition. 1989;5(5):303–13.
15. Cole CR, Blackstone EH, Pashkow FJ, Snader CE, Lauer MS. Heart-rate recovery immediately after exercise as a predictor of mortality. New Engl J Med. 1999;341(18):1351–7. https://doi.org/10.1056/NEJM199910283411804.
16. Jouven X, Empana JP, Schwartz PJ, Desnosw M, Courbon D, Ducimetiere P. (2005): Heart rate profile during exercise as a predictor of sudden death. New England. J Med. 2005;352:1951–8.
17. Levy WC, Cerqueira MD, Harp GD, Johannessen KA, Abrass IB, Schwartz RS, et al. (1998): Effect of endurance exercise training on heart rate

18. variability at rest in healthy young and older men. Am J Cardiol. 1998;82:1236–41.
19. Alfie J, Galarza C, Waisman G. Noninvasive hemodynamic assessment of the effect of mean arterial pressure on the amplitude of pulse pressure. AJH–February 2005–VOL. 18, NO. 2, Part 2. 2005. https://doi.org/10.1016/j.amjhyper.2004.11.026.
19. Teichholz LE, Kreulen T, Herman MV, Gorlin R. Problems in echocardiographic volume determination: echocardiographic-angiographic correlations in the presence or absence of asynergy. Am J Cardiol. 1976;37:7–12 Crossref. PubMed.
20. Kronik G, Slany J, Mosslacher H. Comparative value of eight M-mode echocardiographic formulas for determining left ventricular stroke volume: a correlative study with thermodilution and left ventricular single plane cineangiography. Circulation. 1979;60:1308–16 Crossref. PubMed.
21. Asanoi H, Sasayama S, Kameyama T. Ventriculoarterial coupling in normal and failing heart in humans. Circ Res. 1989;65:483–93 Crossref. PubMed.
22. Wallerson DC, Ganau A, Roman MJ, Devereux RB. (1990): Measurement of cardiac output by M-mode and two-dimensional echocardiography: application to patients with hypertension. Eur Heart J. 1990;11(suppl 1):67–78.
23. Sembulingam P, Sembulingam K, Ilango S, Sridevi G. Rate pressure product as a determinant of physical fitness in normal young adults. J Dent Med Sci. 2015;14(4):08–12 www.iosrjournals.org. (IOSR-JDMS) e-ISSN: 2279-0853, p-ISSN: 2279-0861.
24. de Simone G, Devereux RB, Kimball TR, Mureddu GF, Roman MJ, Contaldo F, et al. Interaction between body size and cardiac workload influence on left ventricular mass during body growth and adulthood. Hypertension. 1998;31:1077–82. https://doi.org/10.1161/01.HYP.31.5.1077 Originally published1 May 1998.
25. Shahraki MR, Mirshekari H, Shahraki AR, Shahraki E, Naroi M. Arterial blood pressure in female students before, during, and after exercise. ARYA atherosclerosis. 2012;8(1):12–5.
26. Oyeyemi A Y, Ewah P A & Oyeyemi A L. Comparison of recovery responses of young physically active and sedentary Nigerian undergraduates following exercise testing. Int J Phys Educ Sports Health. 2015;2(2):60–5. P-ISSN: 2394-1685.
27. Oyeyemi A, Oyeyemi AY, Adegoke BO, Oyetoke FO, Aliyu HN, Aliyu SU, et al. The short international physical activity questionnaire cross-cultural adaptation, validation, and reliability of the Hausa language version in Nigeria. BMC Med Res Methodol. 2011;11:156.
28. International physical activity Questionnaire, IPAQ (2005): Guidelines for the data processing and analysis of the International Physical Activity Questionnaire. www.ipaq.ki.se, 13/12/12, 6:30 pm.
29. Tanaka H, Monahan KD, Seals DR. Age-predicted maximal heart rate revisited. J Am College Cardiol. 2001;*37*(1):153–6. https://doi.org/10.1016/s0735-1097(00)01054-8.
30. Haroonrashid MH, Shaheenkousar HH, Salim AD, Kusal KD. Recovery heart rate response in a sedentary and physically active young healthy adult of Bijapur, Karnataka, India. Basic Sci Med. 2012;1(5):30–3. https://doi.org/10.5923/j.medicine.20120105.02.
31. Du N, Bai S, Oguri K, Kato Y, Matsumoto I, Kawase H, et al. Heart rate recovery after exercise and neural regulation of heart rate variability in 30-40-year-old female marathon runners. J Sports Sci Med. 2005;4:9–17.
32. Dimpka U, Ugwu AC. Determination of systolic blood pressure recovery time after exercise in apparently healthy, normotensive, non-athletic adults and the effects of age, gender and exercise intensity. Int J Exer Sci. 2009;2(2):115–30 http://www.intjexersci.com/.
33. US department of health and human service. (2017): National Institutes of Health. Assessing your health and weight risk. Retrieved on 7/18/17 from: https://www.nhlbi.nih.gov/health/education/lose_wt/BMI/bmicalc.htm
34. Up toDate: obesity in adults: prevalence, screening and eval.retrived on 17/18/17 from https://www.uptodate.com/contents/obesity-in-adult-prevalence-screening-and-evaluation? Source=search_result&search=bmi&selected Tile=1-150
35. Golbel FL, Norstrom LS, Nelson RR, Jorgensen CR, Wang Y. (1978): The rate pressure product as an index of myocardial oxygen consumption during exercise in patients with angina pectoris. Circulation. 1978;57(3):549–56.
36. Dodd NS, KellyS CPW, Engel L. A pressure-time product, work rate & endurance during breathing in human. J Appl Physiol. 1985;64(4):1397–404.

37. Forintano LD, Godoy MF. Increased RPP as predictor for absence of significant obstructive coronary artery disease in patient with positive exercise test. Arq Bras Cardiol. 2016;86:138–44.28.
38. Figueroa Michael A, Demeersman RE, Manning J. The autonomic and rate pressure product responses of Tai Chi practitioners. N Am J Med Sci. 2012;4(6):270–5.

Publisher's Note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.