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Correlation between arterial dP/dt measured by supra-systolic oscillometric central blood pressure (cBP) and Smith-Madigan inotropy index (SMII) for bedside estimation of inotropy

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Abstract

***Introduction*: Shock, which, if not treated adequately, will increase the risk of mortality in children. Non-invasive hemodynamic monitoring is one of the keys to reducing child mortality due to shock. The Smith-Madigan inotropy index (SMII) has been tested as a tool for rapid and non-invasive estimation of myocardial contractility measures. This study evaluated the correlations between arterial dP/dt measured by supra-systolic oscillometric central blood pressure (cBP) and SMII to identify the clinical utility of this non-invasive method as an alternative.**

***Methods*: This cross-sectional study was conducted among a healthy child population from four elementary schools in two provinces in Indonesia, aged 8-14 years. Individuals who met the inclusion criteria were performed on a physical examination (weight and height) as well as cBP and arterial dP/dt max measurements using BP+ USCOM and SMII measurements using USCOM 1A simultaneously. Measurements using BP+ USCOM and USCOM 1A were performed simultaneously. Based on the previous study, left ventricle (LV) dP/dt = 1.25 (arterial dP/dt), so in this study, after obtaining arterial dP/dt data, the calculation of LV dP/dt was continued, and then evaluated the correlations between 1.25 arterial dP/dt (LV dP/dt) and SMII.**

***Results*: Between August 2023 and January 2024, 283 children were enrolled as research subjects, with a median age of 11 years (ranging from 7 to 14 years). The cohort comprised 146 boys (51.6%) and 137 girls (48.4%). The median body weight recorded was 34.9 kg. The predominant nutritional status observed was classified as good nutrition (55.8%), while the prevalent height status among the subjects was categorized as normal height. In this study, a significant correlation was found between 1.25 arterial dP/dt (LV dP/dt) and SMII contractility parameters of kinetic energy (KE) with r=0.34, 95% confidence interval (CI) 0.22-0.44, p<0.0001.**

***Conclusion*: This study obtained results indicating a significant relationship between arterial dP/dt max and SMII contractility parameters. The LV dP/dt max value, calculated from the measurement of arterial dP/dt max using a non-invasive method, can be used as an alternative to assess LV contractility.**

Keywords: Arterial dP/dt, supra-systolic oscillometric central blood pressure, Smith-Madigan inotropy index, ultrasound cardiac output monitoring.

Introduction

Shock or hemodynamic disorders, if not treated adequately, will increase the risk of mortality in children. (1,2) One crucial factor for effectively managing shock or hemodynamic disorders is the utilization of hemodynamic monitoring. The ability of hemodynamic monitoring to precisely identify issues in patients related to preload, contractility, or afterload is essential. Hence, a profound understanding of hemodynamic physiology is imperative. (3-6)

Hemodynamic understanding is closely related to ventriculo-arterial coupling (VAC), which results

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from a complex and constant interaction between the cardiac pump and the vascular system downstream. VAC serves to regulate the hemodynamic system in the body by considering and providing a physiological understanding of the overall and comprehensive function of the heart and blood vessels. Although the heart and systemic vascular tissue possess distinct inherent characteristics, they share a common hemodynamic role: to ensure sufficient organ perfusion. (7-9) VAC delineates the connection between the contractile function of both the right and left ventricles and the arterial resistance to the blood flow propelled by the heart (afterload). The left ventricle is responsible for pumping oxygenated blood into the systemic circulatory system, while the right ventricle pumps unoxygenated blood into the pulmonary circulation. Arterial load signifies the impedance encountered by the ventricles during the propulsion of blood into the circulatory system. Hence, assessing cardiac contractility while considering afterload is imperative. (10)

The ejection of blood flow from the left ventricle (LV) into the arterial system generates aortic pressure, which is reliant on both ventricular and vascular elements. The increased blood flow resulting from increased left ventricular contractility (LV dP/dt max) leads to a rapid increase in aortic pressure. It is thus postulated that a fundamental correlation exists between the maximum rate of aortic pressure (Ao dP/dt max) and left ventricular pressure (LV dP/dt max), depending on mechanical properties and vascular load. (11) LV contractility is one of the most important parameters in determining cardiac performance and function, consequently exerting a direct influence on the hemodynamic state of patients on a global scale. Clinical conditions that can cause left ventricular contractility disorders, such as septic shock and cardiogenic shock, can increase mortality and require intensive care in pediatric patients. Therefore, there is a clinical need to perform bedside measurements in determining patient contractility. (12) One of the non-invasive techniques available for evaluating cardiac contractility is the Smith-Madigan inotropy index (SMII), which is measured by ultrasound cardiac output monitoring (USCOM). The method of measuring cardiac contractility using SMII has been proven to be valid. SMII describes a novel approach to assessing the status of inotropy or cardiac contractility. The approach to calculating contractility utilizing SMII parameters deviates from the conventional inotropy measurements commonly employed in physiological investigations, thereby positioning SMII as a more efficient and pragmatic measurement tool applicable in clinical settings. (13)

Several studies have demonstrated a correlation between arterial dP/dt and the left ventricle's performance. Arterial dP/dt max can be derived from the arterial pressure waveform obtained using a supra-systolic oscillometric central blood pressure (BP+ USCOM) device. (12) According to findings from a perioperative investigation, there is a significant association between arterial dP/dt max and LV dP/dt max, suggesting that alterations in arterial dP/dt max can effectively indicate variations in left ventricular contractility. (14) In other studies, a significant relationship between LV dP/dt and arterial dP/dt, as expressed by the equation LV dP/dt = 1.25 x (arterial dP/dt). (12) Therefore, we wanted to investigate the relationship between arterial dP/dt measured by supra-systolic oscillometric central blood pressure (cBP), as a determinant of LV dP/dt and the SMII, assessed using a non-invasive approach known as USCOM. (13) The primary aim of this study was to evaluate the correlations between arterial dP/dt measured by supra-systolic oscillometric central blood pressure and SMII to identify the clinical utility of this non-invasive method as an alternative.

Materials and methods

This study was a cross-sectional investigation aimed at evaluating the correlations between arterial dP/dt, as measured by supra-systolic oscillometric central blood pressure, and SMII to identify the clinical utility of this non-invasive method as an alternative. (13) This study was conducted on a healthy child population in Lumajang, East Java, and Tasikmalaya, West Java, aged 8-14 years. Data were collected from August 2023 to January 2024 and filtered to be registered as a research sample. The exclusion criteria in this study were children with congenital heart disease, heart valve problems, hypertension, congestive heart failure, kidney failure, and arrhythmias or abnormal heart rhythms. (15)

Sampling was conducted using consequential sampling until the specified time limit was reached. The independent variable was arterial dP/dt max, and the bound variable was SMII. The tools used in this study were USCOM 1A (USCOM Pty Ltd, Coffs Harbour, NSW, Australia) and USCOM BP+ (USCOM Ltd., Sydney, NSW 2000, Australia). Individuals who met the inclusion criteria were performed on a physical examination (weight and height) as well as cBP and arterial dP/dt max measurements using BP+ USCOM and SMII measurements using USCOM 1A. Measurements using BP+ USCOM and USCOM 1A were performed simultaneously. Measurements using BP+ USCOM and

USCOM 1A were conducted by pediatricians and residents who had performed blind tests on simultaneous data using two data recorders (to assess interobserver reliability). To mitigate potential bias, the USCOM 1A measurement protocol required the child to attain a state of calmness, ensuring that the recorded heart rate remains unaffected by any external stimuli that may confound the study outcomes. A pediatric cardiologist conducted echocardiographic assessments on all participants in the study. Individuals with congenital heart anomalies, valve irregularities, and cardiac arrhythmias were not included as study subjects.

Based on the previous study, LV dP/dt = 1.25 (arterial dP/dt), so in this study, after obtaining arterial dP/dt data, the calculation of LV dP/dt was continued. (12) Contractility parameters evaluated through USCOM utilized the SMII. The assessment of contractility in SMII was based on power, which denoted the rate of energy transfer, gauged by the potential energy (PE) and kinetic energy (KE). (13) The contractile performance of the LV was notably influenced by kinetic energy (KE) and force (F). Hence, in addition to the overall SMII quantification, this study also implemented SMII calculation, focusing on the kinetic energy (KE) components. The determination of KE was derived from the formula (KE/(TO + PE)) x SMII. Given that SMII equaled SMI/body surface area, for enhanced precision in outcomes, LV dP/dt was additionally normalized by the body surface area (BSA).

The collected data were processed and analyzed using SPSS software for Windows 23.0 (IBM, Armonk, NY, USA). The data was analyzed descriptively, by calculating the frequency distribution and proportion to determine the characteristics of the research subjects. The Pearson correlation test was used when the distribution was normal, and the Spearman correlation test was used when the distribution was not normal. (16)

Results

The participants of the investigation comprised 311 children, with 26 of them being omitted from the study subsequent to evaluation by a pediatric cardiologist, revealing conditions such as congenital heart disease, heart valve abnormalities, hypertension, arrhythmias, and irregular heart rhythms. Two children were further excluded from the research due to incomplete data documentation (**Figure 1**). The examination was conducted on 283 patients, presenting a median age of 11 years (ranging from 7 to 14 years), encompassing 146 boys (51.6%) and 137 girls (48.4%). The median body weight recorded was 34.9 kg. The predominant nutritional status observed was classified as good nutrition (55.8%), while the prevalent height status among the subjects was categorized as normal height (84.1%) (**Table 1**).

In this study, a significant correlation was obtained between LV dP/dt and SMII contractility parameters with r=0.195, 95% confidence interval (CI) 0.093-0.296, p<0.001 (**Figure 2**), and a significant correlation of LV dP/dt and SMII contractility parameters of kinetic energy (KE) with r=0.34, 95%CI 0.22-0.44, p<0.0001 (**Figure 3**).

Discussion

The determination of LV contractility is imperative in clinical settings as a crucial hemodynamic parameter. Various techniques for assessing contractility in the LV have been extensively documented; however, several of these methods pose challenges for routine clinical application due to their reliance on invasive procedures, expensive equipment, and restriction to specialized medical facilities. (7,17) The community health centers and referral hospitals frequently encounter patients with hemodynamic irregularities, yet lack precise tools for evaluating patients' hemodynamic condition. This investigation aimed to examine the correlations between arterial dP/dt, as measured by supra-systolic oscillometric central blood pressure, and SMII to determine the clinical utility of this non-invasive method as an alternative. Our study employed a non-invasive technique that could be feasibly integrated into daily clinical routines. An earlier examination that evaluated hemodynamic status in healthy children was conducted invasively through catheterization in 1967, involving 29 research participants. (18) Furthermore, there has been limited research utilizing healthy pediatric subjects to evaluate hemodynamic status, particularly in regions such as Indonesia. Through our investigation involving healthy pediatric participants, we aimed to establish a comprehensive understanding of children's hemodynamic status in the most physiologically accurate manner.

The effect of nutritional status and hemodynamics in children

The subjects of this study consisted of 55.8% with good nutritional status, 8.5% who were overweight, and 14.5% who were obese. The hemodynamics of children were impacted by their nutritional status. A separate investigation revealed that children with an overweight nutritional status were at a 2.19-fold higher risk of developing hypertension. (19) While 8.5% of the participants in our research were overweight and 14.5% were obese, individuals with hypertension were deliberately excluded from the analysis to prevent it from confounding the results. Merely, 3.2% of the subjects in the study were identified as having short stature, whereas 84.1% were considered to have normal stature, and 12.4% were classified as tall. Accurate determination of participants' height is crucial, as previous findings indicated that short stature could influence hemodynamic conditions. A study has shown that short stature has an impact on aortic stiffness, increases the pulsatile effort of the left ventricle, and can accelerate the heart rate. (20)

The small percentage of subjects with short stature was expected to have no effect on the results of our study. To minimize bias in our study related to nutritional status, LV dP/dt max was divided by BSA. It was therefore hoped that this calculation would minimize the bias due to nutritional status. In addition, all children included in this study underwent echocardiography examinations to screen for congenital heart abnormalities and heart valve abnormalities. The study participants were also evaluated for cardiac arrhythmia. If the study participants were found to have congenital cardiac abnormalities, valvular defects, and cardiac rhythm disorders, they would be excluded from the study. This measure was implemented to maintain the validity of this research.

Determination of hemodynamic status by various non-invasive methods

The hemodynamic status on a global scale is influenced by cardiac function, particularly the efficacy of the LV. LV contractility is one of the most important parameters that determine the performance of the LV. The clinical condition of patients with LV contractility disorders is often present in cases of septic shock, which is closely related to infection, which can affect outcomes and even increase child mortality, especially in developing countries such as Indonesia. (21) Therefore, there is a necessity for a straightforward assessment tool that can be easily utilized by all healthcare professionals to determine LV contractility. Nevertheless, the evaluation of LV contractility in routine clinical practice remains notably constrained. This is primarily due to the indispensable nature of invasive procedures and the specialized skills required to ensure precise outcomes. (22) Estimating LV contractility in a non-invasive manner can be achieved through echocardiography. However, implementing this method in developing nations poses challenges, as its widespread adoption across all healthcare facilities remains arduous. Echocardiography is predominantly used by referral hospitals as a non-invasive method for determining hemodynamics. The limitations faced by health centers or referral hospitals in employing echocardiography stem from insufficient resources, encompassing both financial constraints and a shortage of personnel proficient in conducting echocardiographic examinations. (23)

The mean maximum increase in left ventricular pressure during ventricular contraction (LV dP/dt max) can be used as one of the parameters of left ventricular inotropy and contractility. A study conducted by Ostadal et al. found that there was a significant relationship between arterial dP/dt max and LV dP/dt max as determined by echocardiography ($r=0.70$, 95% CI 0.51-0.82, $p<0.0001$). In linear regression, LV dP/dt max = 1.25 x arterial dP/dt max was obtained. In addition to having a significant relationship with LV dP/dt max, arterial dP/dt max also has a significant relationship with stroke volume (SV) and cardiac output (CO). (12) We conducted a study by measuring arterial dP/dt max to determine LV dP/dt max calculated from the waveform of arterial pressure measured with a BP+ USCOM device, which employed the supra-systolic recording method to estimate central aortic pressure (CAP). A supra-systolic recording of the oscillometric pulse waveform was made by applying cuff pressure above the systolic blood pressure (SBP) so that the brachial artery was completely blocked. CAP measurements can provide useful clinical information as a hemodynamic monitoring tool, one of which is arterial dP/dt max. (24) Central blood pressure measurement using BP+USCOM compared to the gold standard for direct aortic pressure measurement has good validity. (8)

Arterial dP/dt max, LV dP/dt max, and cardiac contractility

This study aimed to establish the correlation between LV dP/dt max and cardiac contractility determined by SMII measured with USCOM 1A. The LV dP/dt max was derived using the formula 1.25 multiplied by the arterial dP/dt max, which was acquired from the outcomes of measurements conducted with the BP+ USCOM instrument. (12) In the quantitative assessment of LV contractility, one may assess it by quantifying the levels of KE and force (F). KE is delineated as the work performed by the LV in propelling the mass (Msv) from a position of rest or zero velocity to the maximum velocity (PV). The following is an equation related to LV contractility:

$$\text{Kinetic energy} = \tfrac{1}{2}\,(\text{Msv}).(\text{PV})^2$$

$$\text{Msv} = \rho\ .\ \text{SV}$$

$$\text{Force} = \text{Msv}\ .\ \text{MA}$$

$$\text{MA} = \text{PV}/\text{FTp}$$

*ρ=blood density; SV=stroke volume; MA=mean acceleration; FTp=flow time to peak velocity. (25)

The ventricular contractility parameters measured by USCOM 1A are determined by SMII. The calculation of contractility in SMII is based on power. Power is the rate at which energy is transferred. In the context of LV contractility, power is the total power delivered to the aorta. The formula will be:

$$\text{Power} = \text{PE}/\text{flow time} + \text{KE}/\text{flow time}$$

$$\text{SMI} = (\text{BP mean x SV x } 10^{-3}/7.5 \text{ x FT}) + (\text{SV x } 10^{-6} \text{ x } \rho \text{ x Vmean}^2/2 \text{ x FT})$$

$$\text{SMII} = \text{SMI}/\text{BSA}$$

*BP mean=mean arterial pressure – central venous pressure (mmHg); SV=stroke volume (ml); r=density (kg.m^{-3}); Vmean=mean velocity (m.s^{-1}); FT=systolic flow time (ms); PE=potential energy; KE=kinetic energy.

The generation of potential energy arises from the ventricular work involved in producing arterial pressure, resulting from alterations in volume and pressure, whereas kinetic energy is attributed to the ventricle's role in propelling blood circulation. Kinetic energy emerges from the product of mass and velocity. (13) Based on the previous explanation that several factors that are quite strong in influencing LV contractility (that are KE and F), in this study, the contractility calculation used not only total SMII, but also SMII based on KE aspects. In this study, a significant correlation ($p<0.001$) was obtained from LV dP/dt max (1.25 x arterial dP/dt max) and SMII contractility parameters measured by USCOM 1A, with $r=0.195$. There was a significant correlation ($p<0.0001$) between LV dP/dt max and SMII contractility parameters based on KE aspects, with a stronger correlation compared to the total SMII parameter ($r=0.34$). Ventricular dP/dt max is commonly utilized to evaluate ventricular contractility; however, its measurement traditionally involved invasive procedures with intraventricular catheters, posing challenges in clinical application. The non-invasive method employed in this study to assess LV dP/dt max by measuring arterial dP/dt max using the supra-systolic oscillometric central blood pressure method proved to be uncomplicated, rapid, reproducible, easily learned, and clinically feasible. (11)

The relationship between LV dP/dt max and SMII based on kinetic energy aspects

The flow of blood expelled from the LV into the arterial system produces aortic pressure that depends on the interaction between the ventricles and the vascular component. Increased blood flow is induced by a rapid increase in aortic pressure and LV contractility. Therefore, it is very possible that the maximum rate of aortic pressure (aortic dP/dt max) and LV dP/dt max have an intrinsic correlation that depends on mechanical properties and vascular loading. (11) The correlation strength between LV dP/dt max (1.25 x arterial dP/dt max) and SMII contractility parameters in this study was only with $r=0.195$. This was likely because the determination of arterial dP/dt max using the supra-systolic technique of oscillometric pulse waves by applying strong cuff pressure above SBP still had weaknesses. Among other things, total damming of the brachial artery caused a reflection effect of blood flow in the brachial artery towards the aorta, thereby affecting the arterial dP/dt max. The reflected flow due to the brachial artery dam will reduce the forward flow from the aorta. This will certainly affect arterial dP/dt max. (26) However, another result in this study showed a significant correlation ($p<0.0001$) between LV dP/dt max (1.25 × arterial dP/dt max) and SMII contractility parameters based on KE aspects. The results showed a stronger correlation compared to the total SMII parameter, with a correlation coefficient of $r=0.34$. Power is the rate of energy transfer determined by 2 energies, namely PE and KE. (13) The contractility of LV is strongly determined by KE and F. This may explain why the correlation between LV dP/dt max and SMII in terms of kinetic energy was stronger than that of total SMII. (13)

The end-systolic elastance and arterial elastance factors present in the subjects of this study would also impact arterial dP/dt max. Should further investigations be conducted in subsequent studies, these two components need to be duly considered. Comprehension of vascular elastance necessitates an understanding of VAC. VAC is the end result of a complex and constant interaction between the cardiac pump and the vascular system downstream. The coordination of ventriculo-arterial coupling involved regulating the body's hemodynamic system by integrating the functions of the heart and blood vessels holistically, rather than in isolation. This coupling mechanism significantly contributed to a thorough physiological grasp of cardiovascular and vascular functionality. Despite the distinct intrinsic characteristics of the heart and systemic vascular tissue, they both serve the common hemodynamic purpose of ensuring adequate organ perfusion. (7-9)

Ventriculo-arterial coupling

Ventricular performance can be assessed from the end-systolic elastance (Ees), which reflects the ability of the ventricles to pump blood (stroke volume) through variations in systolic final pressure. This state reflects the ventricle's ability to adapt to variations in afterload. Meanwhile, the performance and properties of the vascular system are determined by vascular elastance (Ea). Vascular elastance reflects resistance and compliance in the main arteries. The relationship between Ees and Ea can be explained through the VAC index, which is the Ea/Ees ratio, which reflects the systolic performance of the left ventricle in dealing with arterial compliance. Based on several studies, the value of the VAC index is between 0.6-1.2 or 0.5-1.3. A VAC index value of more than 1.3 indicates a disorder of the work balance of the heart and blood vessels that requires medical intervention. (9,27,28) The calculation of the VAC index is based on the calculation in the following equation:

Ees = (DBP-(End(est) x SBP x 0.9))/End(est) x SV

Ea = (SBP x 0.9)/SV

*DBP=diastolic blood pressure; SBP=systolic blood pressure; End(est)=the estimated normalized ventricular elastance at the onset of ejection; SV=stroke volume.

This calculation uses echocardiography. The relationship between the concepts of VAC and arterial dP/dt max can be understood through mathematical models. The model consists of afferent pathways of arterial and cardiopulmonary receptors, efferent sympathetic pathways, and parasympathetic activity, as well as responses from several different effectors, including cardiac contractility, peripheral resistance, blood volume, and heart rate. In our research, the findings were in accordance with the concept of VAC and arterial dP/dt. So, factors such as blood vessel resistance and compliance, blood volume, and heart rate will influence the relationship between arterial dP/dt and contractility. We attempted to ensure that the heart rate did not affect the validity of our results by conducting the USCOM 1A and BP+ USCOM examinations. The subject was waited until the heart rate was stable before the examination was carried out. Baroreceptors are located in the aortic arch and carotid sinuses, positions above the heart. The pressure input heading into the afferent path is essentially a heart-high aortic pressure adjusted to the hydrostatic column. The afferent arterial pathway of the baroreceptor can be described in the following formula:

$$\tau_z \frac{dP_{sa}}{dt} + (P_{sa} - \rho gHsin(\alpha)) = \tau_p \frac{d\overline{P}}{dt} + \overline{P}$$

*τp and τz are the time constants for the real pole and the zero pole. Psa is the pressure on the large artery, ρgHsin is the hydrostatic pressure. dPsa/dt is a change in pressure in a large artery. $\overline{P}$ is the output variable of the linear dynamic block.

When analyzed, the change in pressure in the large artery is influenced by many factors, one of which is hydrostatic pressure. Hydrostatic pressure is determined by ρgHsin, where ρ is the viscosity of the blood, g is the acceleration of gravity, and α is the angle of inclination of the supine position. Therefore, arterial dP/dt is also influenced by various factors, including hydrostatic pressure, blood viscosity, gravity, and others. (29) In this study, we did not take blood samples for hemoglobin and hematocrit examination. This may still have an effect on our research. If further research is conducted, the research subject can undergo a complete blood test to check hemoglobin and hematocrit, ensuring the sample is more homogeneous. In the children's research subjects, the nutritional status and height of the child are related to their hemoglobin and hematocrit levels. This is evidenced by a study conducted in Indonesia with children under five as subjects, which found differences in hemoglobin and hematocrit levels between children with short stature and those with normal stature. In our study, 3.5% of the subjects were short in stature. This may affect hematocrit and blood viscosity as well as arterial dP/dt max. (30)

Limitations of the study

The limitations present in this study pertain to the varying nutritional statuses and heights exhibited by the research participants. Notably, the hemoglobin and hematocrit levels of patients are influenced by these factors. The interplay between hemoglobin, hematocrit, and blood viscosity can impact both hydrostatic pressure and arterial dP/dt max. Should further investigation be conducted, it is imperative to not only document and assess the nutritional and height profiles of children but also to carefully select groups with matching characteristics in these aspects. Furthermore, the research subjects must undergo assessments for hemoglobin and hematocrit levels. The determination of arterial dP/dt max in this research employs a supra-systolic method involving oscillometric pulse waves and the application of cuff pressure exceeding the SBP. Applying significant pressure on the brachial artery results in complete occlusion, leading to a reverberating motion of blood flow from the brachial artery towards the aorta. Consequently, this may have an impact on arterial dP/dt max. The retrograde flow caused by the occlusion of the brachial artery hinders the forward flow originating from the aorta. When conducting further research on this subject, it is crucial to consider this potential confounding variable by assessing the influence of reflected flow due to occlusions in the brachial artery on arterial dP/dt max measurements, particularly when using a BP+ USCOM device.

Conclusion

This study obtained results indicating a significant relationship between arterial dP/dt max and SMII contractility parameters. The LV dP/dt max value, calculated from the measurement of arterial dP/dt max using a non-invasive method, could be used as an alternative to assess LV contractility.

Ethics approval

This study was approved by the Ethics Commission for Medical and Health Research, Faculty of Medicine, Universitas Brawijaya, Saiful Anwar General Hospital, Malang. The approval number was 258/EC/KEPK/1/2022, and the approval date was November 22, 2022.

Competing interests

The authors declare that they have no competing interests.

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Table 1. Baseline subjects' characteristics

Variable	n=283
Age (years), median (min-max)	11 (7-14)
Gender, n (%) - Male - Female	 146 (51.6) 137 (48.4)
Body weight (kg), median (min-max)	34.9 (13.9-91.4)
Body height, n (%) - Short stature - Normal - High stature	 10 (3.5) 238 (84.1) 35 (12.4)
Nutritional status, n (%) - Malnutrition - Underweight - Normal - Overweight - Obese	 14 (4.9) 46 (16.3) 158 (55.8) 24 (8.5) 41 (14.5)
Heart rate (beats/min), (mean±SD)	93.3±14.3
Systolic blood pressure measured by BP+ USCOM (mmHg), median (min-max)	102 (65-135)
Diastolic blood pressure measured by BP+ USCOM (mmHg), mean±SD	68.7±10.4
Pulse pressure (mmHg), median (min-max)	34 (19-57)
Stroke volume measured by echocardiography (ml), median (min-max)	45 (19.6-58.3)
SMII measured by USCOM 1A (Wm^{-2}), median (min-max)	1.70 (1-5.8)
Ejection fraction (%), median (min-max)	69 (57.4-88)
Arterial dP/dt max (mmHg/sec), median (min-max)	932 (545-1987)

Legend: SD=standard deviation; SMII=Smith-Madigan inotropy index.

Figure 1. Flow chart of recruitment of research subjects

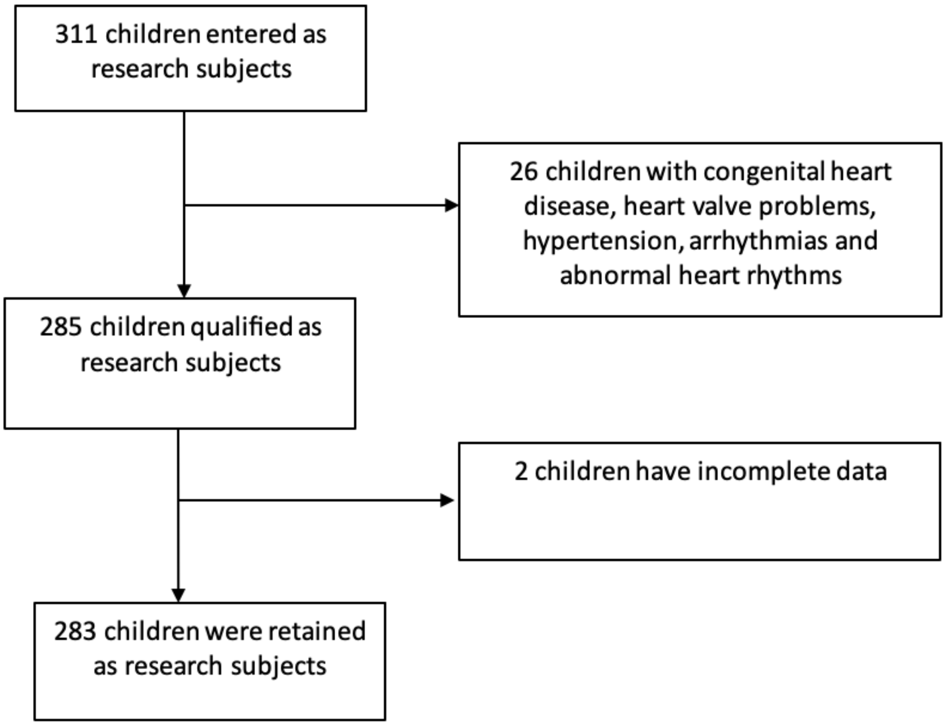
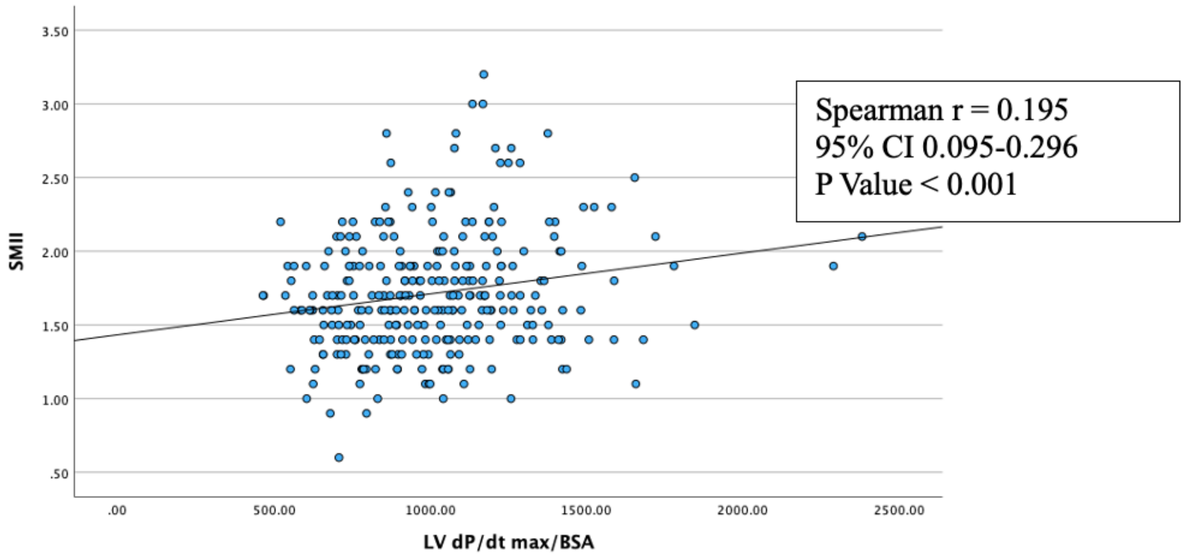
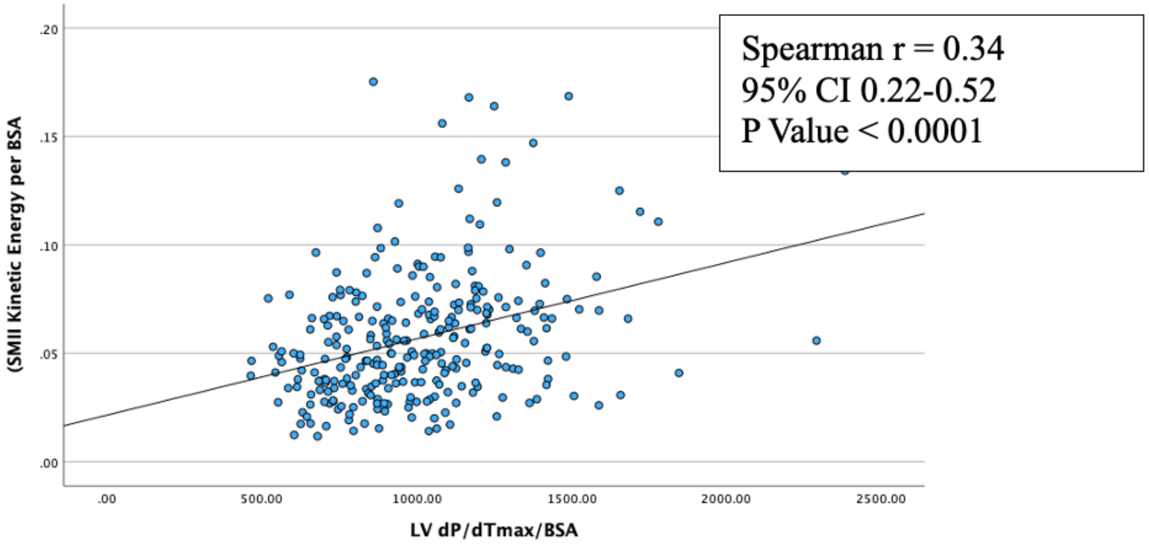


Figure 2. Relationship between LV dP/dt max/BSA and SMII



Legend: LV=left ventricle; BSA=body surface area; SMII=Smith-Madigan inotropy index; CI=confidence interval.

Figure 3. Relationship between LV dP/dt max/BSA and SMII kinetic energy



Legend: LV=left ventricle; BSA=body surface area; SMII=Smith-Madigan inotropy index; CI=confidence interval.

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