의학 박사 학위 논문

소아에서 호흡 주기에 따른 pulse oximeter plethysmographic amplitude 의 변화에 대한 연구

: 접촉 압력에 따른 차이

The effect of sensor contacting force on respiratory variation in pulse oximeter plethysmographic amplitude in children

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이 지 현

Abstract

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Backgrounds: Predicting fluid responsiveness is crucial for adequate fluid management. Respiratory variations in photoplethysmographic waveform amplitude (*Δ*PPG) enable volume status assessment. However, previous studies have been shown inconsistent results regarding the ability to predict fluid responsiveness of ΔPPG when used for children. The contact force between the measurement site and sensor is one of the factors affecting the amplitude of photoplethysmography (PPG). Changes in the contact force can affect the *Δ*PPG value, thereby hindering its indicator in children. We aimed to evaluate contact force effects on *Δ*PPG in children under general anesthesia.

Methods: A two-stage study was conducted. In the first observational study, children aged 3–5 years and scheduled for simple elective surgery were enrolled. After anesthetic induction, mechanical ventilation commenced at a tidal volume of 10 mL/kg. PPG signals were obtained in the supine position from the index finger using a force-sensor–integrated clip-type PPG sensor that increased the contact force from 0–1.4 N for 20 respiratory cycles at each force. The AC amplitude (pulsatile component), DC amplitude (non-pulsatile component), AC/DC ratio, and *Δ*PPG were calculated. In the second study, the changes in the ability of *Δ*PPG as indicator for fluid responsiveness according to the contact force changes were evaluated. Children aged 1 month-5 years and scheduled for major surgery including neurosurgery and cardiac surgery were enrolled. After anesthetic induction, mechanical ventilation commenced with a tidal volume of 10 ml/kg. *Δ*PPG was calculated at five different contacting force level (0–0.3N, 0.3–0.6N, 0.6–0.9N, 0.9–1.2N, and 1.2–1.5N) and individually adjusted contacting force, before volume expansion. Subjects were considered as fluid responders if volume expansion increased the stroke volume index (SVI) by >15%.

Results: In the first study, data from 34 children were analyzed. Seven contact forces at 0.2-N increments were evaluated for each patient. The normalized AC amplitude increased maximally at a contact force of 0.4–0.6 N and decreased with increasing contact force. However, the normalized DC amplitude increased with a contact force exceeding 0.4 N. *Δ*PPG decreased slightly and increased from the point when the AC amplitude started to decrease as contact force increased. In a

0.2–1.2 N contact force range, significant changes in the normalized AC amplitude, normalized DC amplitude, AC/DC ratio, and respiratory variations in photoplethysmography amplitude were observed (all $P < 0.005$). In second study, data from 38 children were analyzed. There was significant difference in ΔPPG between fluid responders and non-responders only at contacting force level of 0.9-1.2N ($P = 0.002$) and individually adjusted contacting force ($P < 0.001$). Additionally, ΔPPG at those contacting force level could predict fluid responsiveness in mechanically ventilated children.

Conclusions: PPG amplitude and ΔPPG changed according to variable contact forces. When contacting force is controlled to an adequate degree, the ability of ΔPPG to predict fluid responsiveness can be improved.

Keywords : Fluid responsiveness; child; photoplethysmography; contacting force

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1. General Introduction

1.1 Photoplethysmography

Photoplethysmography (PPG) is an optically obtained plethysmogram and enables visualization of blood volume changes in the microvascular bed within cardiac cycle (1). PPG can be obtained by simple device consisting of a light source, most commonly an LED, and light detector (photo diode). The detector can be placed either directly across from the light source for transmission plethysmography or next to the light source for reflective plethysmography (2). A commercially available PPG device is the pulse oximeter, which is a highly processed and filtered signal (3). All clinical pulse oximeters have an auto-gain function to maximize the size of PPG waveform displayed.

PPG signals obtained by pulse oximeter comprise pulsatile and non-pulsatile components from arterial pulsation (AC) and venous blood or constant arterial blood (DC), respectively (1). Using 660-nm and 940-nm wavelengths, pulse oximetry calculates the AC/DC ratio and peripheral oxygen saturation.

1.2 PPG waveform analysis and factors affecting PPG waveform

PPG waveform amplitude is a useful plethysmographic features. PPG amplitude contain the information about cardiac cycle, cardiac output, sympathetic tone and vascular compliance. For example, increased PPG amplitude is due to increased

vascular distensibility, increased cardiac output or vasodilation due to anesthesia or vasodilator administration. On the other hand, decreased PPG amplitude is associated with decreased cardiac output or vasoconstriction due to hypovolemia, surgical stress or infusion of vasoconstrictor (2, 4). In addition, the PPG amplitude can be affected by the position or level of measurement site (5, 6) and age (7). The PPG waveform measured at the fingers reflects those effects more sensitively, as the cutaneous vessels are richly innervated by alpha-adrenoreceptors in fingers when compared to other sites.(8)

Moreover, Teng et al. reported that the waveform of PPG signal was affected by the contacting force between the sensor and the measurement site in healthy and normotensive adults (9). In that study, increasing contacting force from 0.2N to 1.8N resulted in the increase in DC amplitude and the increase followed by decrease in AC amplitude and AC/DC ratio. The maximal PPG amplitude could be obtained at the contacting force (9). Changes in PPG amplitude may change *Δ*PPG; thus, contact forces can cause bias when predicting fluid responsiveness using *Δ*PPG.

1.3 Respiratory variations in photoplethysmographic waveform amplitude (*Δ*PPG)

As well as heartbeat signals, respiratory signals can be obtained from PPG (10), by turning off the auto-gain function. Respiratory variations in photoplethysmography

amplitude (*Δ*PPG) can reflect stroke volume variation and predicted fluid responsiveness in mechanically ventilated adult patients (11-13). Fluid management using *Δ*PPG can maintain optimal cardiac output in patients with unstable hemodynamics. However, pediatric *Δ*PPG data are limited. Previously, *Δ*PPG failed to discriminate between children with and without hypovolemia (14). Because many factors can affect the PPG signal, *Δ*PPG may be unreliable when these factors are uncontrolled.

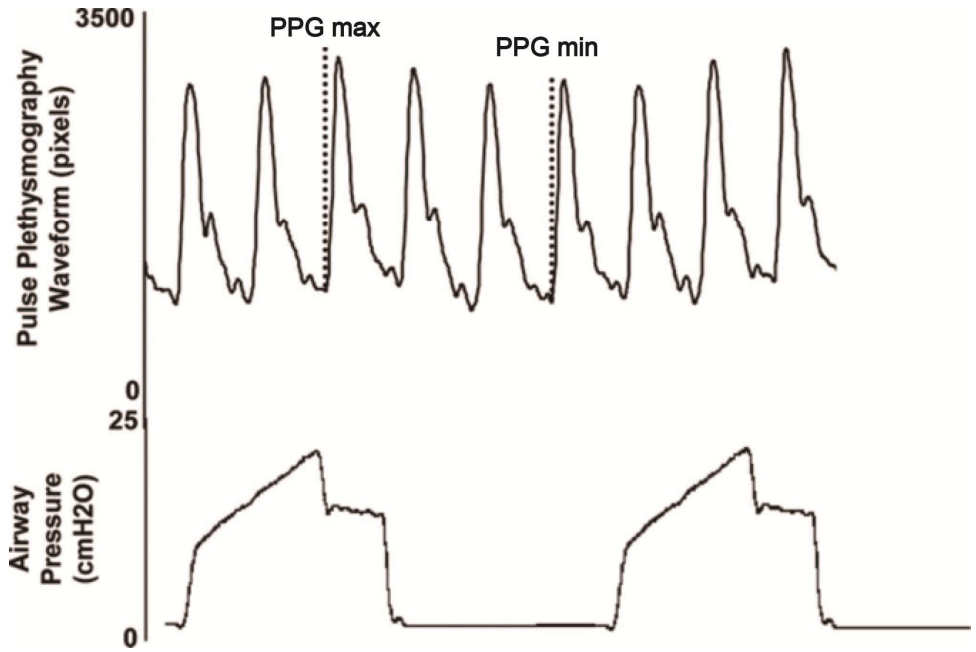

Figure 1. PPG waveform and airway pressure recordings in an illustrative patient. PPG max and PPGmin were defined as maximum and minimum PPG amplitude, respectively. *Δ*PPG was then calculated as (PPGmax – PPGmin)/[(PPGmax + PPGmin)/2].(11)

1.4 Necessity and importance of this research

PPG signal can be obtained only by commercial pulse oximeter device. For the monitoring of pulse oximeter, clip-type or adhesive band type sensor can be used. Regarding adhesive band-type sensors, it may be tightly wrapped at the finger, resulting in poor signal quality with lower PPG amplitude, especially in pediatric patients. This may occur in daily clinical practice and in such cases, we cannot obtain the accurate information from PPG signals such as *Δ*PPG.

Therefore, we conducted a two-stage study. In the first observational study, the effect of contact force on PPG amplitude and *Δ*PPG in anesthetized, mechanically ventilated children undergoing simple surgery. In the second experimental study, we evaluated the influence of contacting force on the ability of *Δ*PPG to predict fluid responsiveness in children undergoing major surgery. In this study, we attempted to determine the optimal contacting force in which the reliability of *Δ*PPG was maximized.

2. First study protocol and results

2.1 Methods

Study population

This prospective, non-randomized interventional study was approved by the Institutional Review Board of Seoul National University Hospital (H1609-067-791) and registered at https://ClinicalTrials.gov (number: NCT02940938; principal investigator: K.H.S.; date of registration: 9 October 2016). After obtaining written informed consent from the children's parents or guardians, children aged between 3 and 5 years undergoing elective surgery including ophthalmic, orthopedic, and urologic surgery under general anesthesia from December 2016 to March 2017 were enrolled. Patients excluded from the study were those with any cardiovascular disease with arrhythmia or ventricular dysfunction, cyanosis, cerebrovascular disease, obstructive or restrictive lung disease with decreased respiratory compliance, or unstable hemodynamics due to bleeding or sepsis, which could affect the perfusion index.

Study protocol and data collection

All children underwent 8 hours of fasting before the procedure. After monitoring of electrocardiography and non-invasive blood pressure, and pulse oximetry had commenced, anesthesia was induced with atropine (0.02 mg/kg),

thiopental sodium (5 mg/kg), and rocuronium (0.6 mg/kg), and maintained with sevoflurane. Each child lay in the supine position. After tracheal intubation, mechanical ventilation was started with a constant tidal volume of 10 mL/kg and the respiratory rate was adjusted to obtain an end-tidal carbon dioxide (E_TCO_2) value of 30–35 mmHg. The anesthetic concentrations were controlled to maintain the bispectral index between 40 and 60.

Five minutes after anesthetic induction without surgical stimuli, a PPG signal was obtained for at least 20 respiratory cycles from 0–1.4 N of contact force at 0.2-N increments.

Acquisition of PPG data and the testing apparatus

To assess PPG amplitude and *Δ*PPG with varying contact forces, a clip-type force-sensor–integrated PPG monitoring device was developed (Figure 2). A pair of light-emitting diodes (LEDs) (SM1206NHC, Bivar Inc., USA) and a silicon photodiode (SFH2430, OSRAM, Germany) were used to measure PPG in reflectance mode. The LED was red in color with a peak wavelength of 625 mm and driven by a continuous current of 20 mA. The distance between the center of the photodiode and the LED was 4 mm. A curved structure was designed and placed on the photodiode-LED pair to prevent the LED light from being absorbed directly by the photodiode. The current from the photodiode was converted to a voltage using a trans-impedance amplifier and then band-pass filtered between 0.1–

20 Hz and amplified.

In addition to the PPG sensor, a force-sensing resistor was used (FSR 408, Interlink Electronics, USA) to measure the contact force between the PPG sensor and measurement site. A force-sensing resistor is a robust polymer thick film (PTF) device that exhibits changes in resistance when force is applied to the active area of the sensor. The force sensor was placed directly opposite from the photodiode-LED pair, and an elastomer was placed under the sensor so that the force was evenly focused on the active area of the sensor. The varying resistance of the force sensor was calculated by measuring the voltage applied to the sensor. All analogue signals were digitized using a 24-bit delta-sigma analogue-to-digital converter (ADS1298, Texas Instruments, USA) at a sampling rate of 250 Hz. In all patient, this self-manufactured sensor was placed at the index finger for data acquisition.

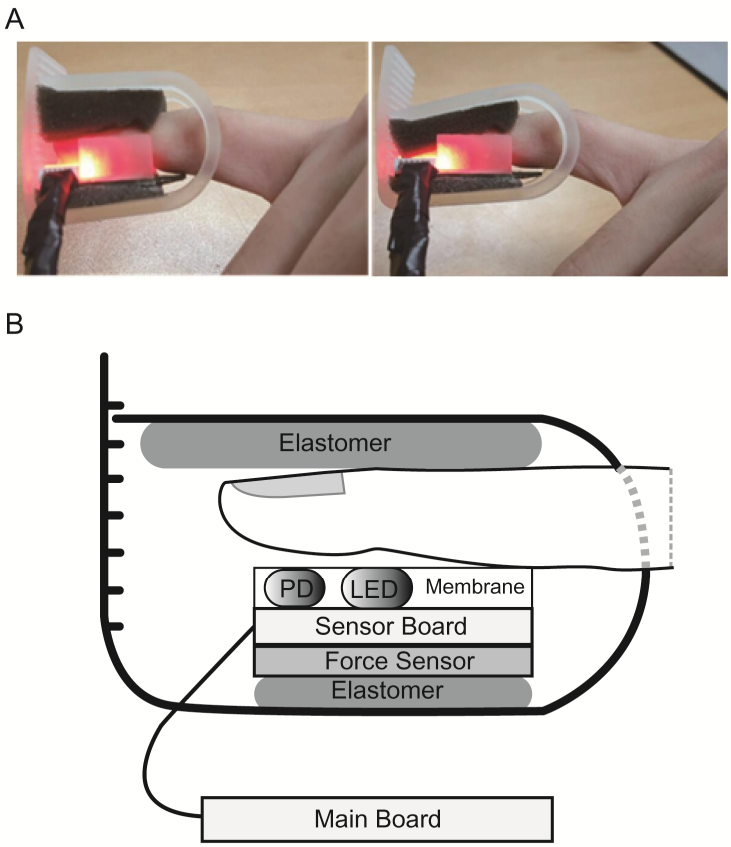

Figure 2. The clip-type PPG sensor. A, The application of the PPG sensor in clinical practice for the study. B, The layout of the sensor comprised of a light-emitting diode (LED), a photodetector (PD), and a force sensor

Data analysis

For each patient, PPG measurements were acquired for at least 20 respiratory cycles for each contact force. The contact force was varied from 0 to 1.4 N in 0.2-N increments. All data were equally subjected to moving average digital filtering using a 0.1-s window size for smoothing. For each cardiac cycle, the maximum and minimum points of the PPG AC signals were used to calculate the PPG AC amplitude for each beat. Any beats with an excessively large or small AC amplitude due to artefacts caused by movement of the hand or movement of the operating table were excluded. Next, the mean AC amplitude for each contact force was calculated. The low-frequency component of the PPG (< 0.1 Hz) was defined as the PPG DC component, and the magnitude of the DC component for each contact force was calculated as the DC amplitude. The beat-to-beat DC amplitude was not calculated because it did not change significantly for each trial. As the AC and DC amplitude of PPG signals varied widely across patients, the AC and DC amplitudes for each contact force were normalized for each patient. This was performed by dividing the measured values by the maximum AC and DC amplitudes observed in the respective patients (9). Lastly, to calculate ΔPPG, the respiratory cycle was first divided by analyzing the PPG AC amplitude variation. Next, the maximum and minimum PPG AC amplitudes within a respiratory cycle (PPG_{max} and PPG_{min}, respectively) were determined. Thereafter, ΔPPG was calculated as follows: $\Delta PPG(\%) = 100 \times [PPG_{max} - PPG_{min}]/[(PPG_{max} + PPG_{min}) / 2]$. Then, ΔPPG was averaged over three consecutive respiratory cycles. All data

processing was conducted using MATLAB 2016 (The MathWorks, Inc).

Statistical analysis

The primary and secondary outcomes of this study were to evaluate changes in PPG amplitude and *Δ*PPG according to changes in contact force, respectively. Because there were no previous data, we used Cohen's F-test with a moderate effect size of 0.25 to calculate the sample size (15). The required sample was 36 patients with an alpha of 0.05, a power of 90%, and a dropout rate of 20%.

Statistical analyses were performed using SPSS ver. 22 (IBM Corp., Armonk, NY, USA). Continuous variables are expressed as means (standard deviations [SDs]) or medians and interquartile ranges after performing the Kolmogorov–Smirnov test. A linear mixed effect model was used to evaluate changes in PPG amplitudes and *Δ*PPG according to increasing contact force. Age and sex were also included as fixed effects. The paired t-test was used for comparisons between parameters obtained before and after PPG measurements. A correlation analysis was performed to find the relationship between the PPG amplitude and *Δ*PPG. All P values are two-sided; $P < 0.05$ was considered significant.

2.2 Results

A total of 36 children were enrolled from December 2016 to March 2017, and two patients were excluded because of sensor failure. Therefore, data from 34 patients were analyzed (Figure 3). Table 1 shows the baseline characteristics of the 34 children. The mean measurement duration was 6.2 ± 0.7 minutes and there was no significant change in heart rate, blood pressure, or sevoflurane concentration during PPG measurement (Table 2).

We divided the contact force into seven levels from 0 N to 1.4 N. Table 3 shows the number of children from whom PPG signals could be obtained and the mean actual contact force at each level. The normalized AC amplitudes, DC amplitudes, AC/DC ratios, and *Δ*PPG values at each contact force level are shown in Figure 4. The normalized AC amplitude was increased to its maximal value at a contact force of 0.4–0.6 N and decreased as the contact force increased. On the other hand, the normalized DC amplitude increased with contact forces exceeding 0.4 N. The AC/DC ratio showed a similar pattern to that of the normalized AC amplitude according to the contact force. *Δ*PPG was lowest at contact forces of 0.4–0.6 N and gradually increased with increasing contact force. In the range of contact forces from 0.2–1.2 N, significant changes in normalized AC amplitude, normalized DC amplitude, AC/DC ratio, and *Δ*PPG were observed ($P < 0.001$ for normalized AC amplitude, normalized DC amplitude and AC/DC ratio; $P = 0.002$ for *Δ*PPG). Age and sex did not seem to induce between-subject variability. *Δ*PPG was negatively correlated with the normalized AC amplitude (Pearson's correlation

coefficient, r = -0.71; 95% confidence interval [CI], -0.78 to -0.62; *P* = 0.000) and the AC/DC ratio (r = -0.69; 95% CI, -0.76 to -0.60; *P* = 0.000).

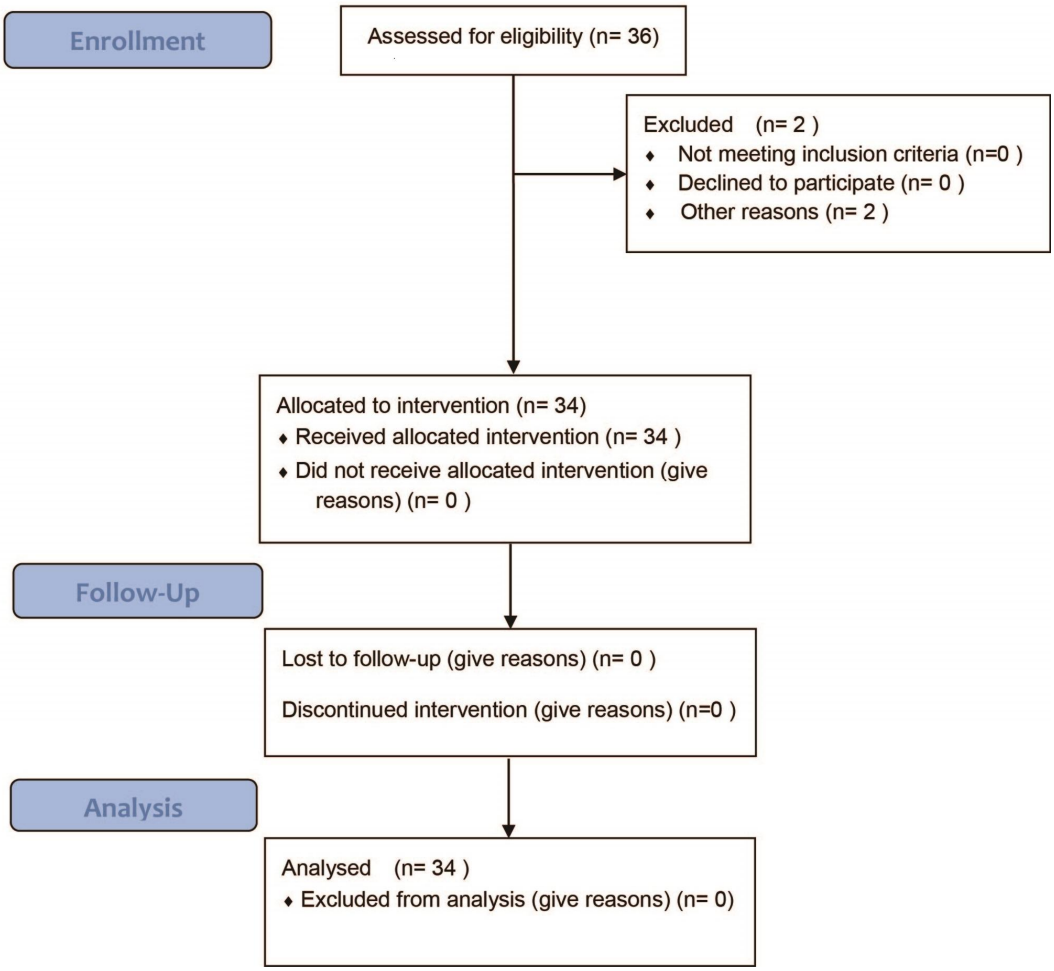

Figure 3. Recruitment flowchart

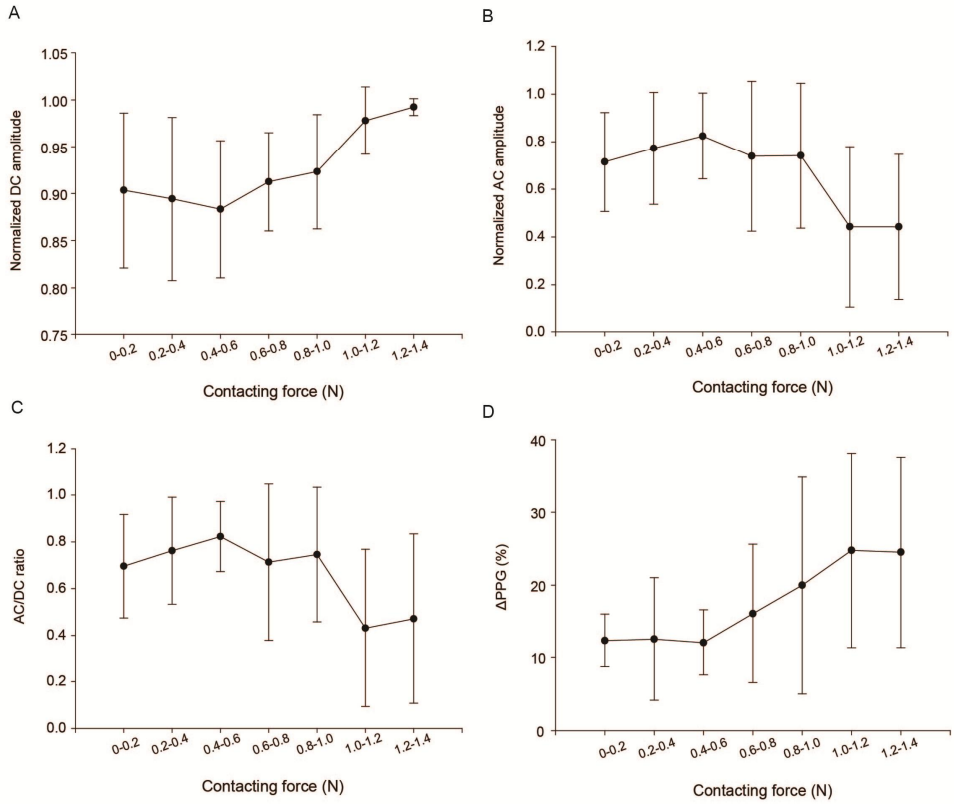

Figure 4. The changes in the normalized DC amplitude (A), AC amplitude (B), AC/DC ratio (C), and respiratory variations in pulse oximeter photoplethysmography amplitudes (ΔPPG, D)

Table 1. Baseline characteristics of all patients

Variables	Values
Age (yr)	4.1 ± 0.65
Height (cm)	108.0 ± 8.9
Weight (kg)	18.5 ± 3.9
Sex (M/F)	20/14
Operations	
Opthalmic surgery	15
Orthopaedic surgery	7
Urologic surgery	12

Data are presented as mean ± SD or number

Table 2. Vital signs before and after photoplethysmographic measurement in 34 patients

Parameters	Before measurement	After measurement	95% CI for differences	P value
Heart rate (/min)	98 ± 9	99 ± 9	-0.9 to 1.1	0.856
Systolic blood pressure (mmHg)	89.8 ± 11.3	89.3 ± 12.4	--0.6 to 0.4	0.547
Diastolic blood pressure (mmHg)	50.6 ± 8.8	48.9 ± 9.7	-1.6 to 1.4	0.625
Mean blood pressure (mmHg)	62.1 ± 9.3	61.5 ± 8.9	-0.5 to 0.7	0.722
Body temperature (°C)	36.3 ± 1.2	36.3 ± 1.2	-0.01 to 0.01	1.0
Sevoflurane concentration (vol%)	2.0 ± 0.6	1.9 ± 0.7	-0.1 to 0.1	0.683

Data are presented as mean ± SD.

CI; confidence interval

3. Second study protocol and results

3.1 Methods

This study was approved by the Institutional Review Board of Seoul National University Hospital (H1609-066-791) and registered at ClinicalTrials.gov (NCT 02952651). Written informed consent was obtained from all parents. Children aged between 1 month and 5 years who were scheduled for congenital cardiac surgery or neurosurgery and expected large fluid shifting were enrolled. Patients with a single ventricle, right heart failure, pulmonary hypertension, any hepatic, renal or pulmonary disease, any cardiac arrhythmia or valvular heart disease, or signs of increased intracranial pressure were excluded.

Anesthetic protocol

Induction of general anesthesia was performed using 0.02 mg/kg atropine and 5 mg/kg thiopental sodium. Rocuronium (0.6 mg/kg) and (if required) fentanyl (5-10 μg/kg) were used for endotracheal intubation. Mechanical ventilation was controlled to obtain a $PaCO_2$ of 35 – 40 mmHg, using the volume-controlled mode to maintain a constant tidal volume of 10 mL/kg during surgery.

After induction of anesthesia, a peripheral arterial catheter was placed at the right or left radial artery. A central venous catheter was inserted into the right internal jugular vein under the ultrasound guidance. Anesthetic maintenance was

performed at the discretion of the attending anesthesiologist using inhalational or intravenous anesthetic agents. The anesthetic depth was adjusted to maintain Bispectral Index value between 40 and 60.

Experimental protocol and test apparatus

The experiment was performed after the patients were weaned from cardiopulmonary bypass successfully and sternum was closed during cardiac surgery, or anytime when suspicious hypovolemia was exist and no active bleeding during neurosurgery. Before experiment, the anesthesiologists confirmed that there was no residual anatomical defect or abnormality using transthoracic echocardiography (TEE, Philips iE33 system, Philips Healthcare, Andover, MA, USA) in patients undergoing cardiac surgery.

Baseline hemodynamic data including arterial blood pressure (ABP) and central venous pressure (CVP), and echocardiographic data including stroke volume were obtained. Pulse pressure variation (PPV) was calculated using the following formula: PPV (%) = 200 × (PP_{max}-PP_{min}) / (PP_{max}+PP_{min}) in one respiratory cycle (PP_{max} and PP_{min}; maximum and minimum systolic pressure). The mean value of the three consecutive respiratory cycles was used for analysis. Some patients received varying degrees of inotropic support, the vasoactive – inotropic score during the experiment was calculated as follows: dopamine dose (μg/kg/min) + dobutamine dose (μg/kg/min) + (epinephrine dose × 100 [μg/kg/min]) +

(milrinone dose × 100 [μg/kg/min]) + (vasopressin dose × 10,000 [U/kg/min]) + norepinephrine dose × 100 [μg/kg/min]) (16).

Before volume infusion, a PPG signal was obtained for at 20 respiratory cycles from 0-1.5N of contact force at 0.3-N increments. The PPG sensor used in the first study was also used in this second study. After obtaining PPG signal ended, Volulyte® (6% hydroxyethyl starch 130/0.4) of 10 mL/kg was administered for 20 min. After volume administration, hemodynamic and echocardiographic measurements were repeated.

Echocardiographic measurement

All echocardiography data were measured by a single expert using TEE or transthoracic echocardiography (TTE) before and after fluid loading.

1) *Stroke volume (SV), stoke volume index (SVI), cardiac output and cardiac index*

The aortic annulus diameter (D) was measured during the systolic phase in the mid-esophageal aortic valve long axis view by TEE or parasternal long axis view by TTE. The aortic blood flow waveform at the level of the aortic annulus was obtained using pulsed-wave Doppler in the deep-transgastric long-axis view of

TEE or apical five chamber view of TTE. The mean velocity time integral (VTI) was calculated from three consecutive waves at the end of the expiratory period.

Variables were calculated as follows:

$$SV = VTI \times 3.14 (D/2)^2$$

Stoke volume index (SVI) = SV/body surface area

Cardiac output = SV × heart rate

Cardiac index = Cardiac output/ body surface area

2) ⊿Vpeak

The maximum and minimum Vpeak during one respiratory cycle were measured at the aortic valve level in the same view for VTI. ⊿Vpeak was calculated as follows: ⊿Vpeak (%) = 200 × ($Vpeak_{max}$ – $Vpeak_{min}$) /($Vpeak_{max}$ + $Vpeak_{min}$). The mean ⊿Vpeak was calculated using three consecutive respiratory cycles.

Statistical analysis

The purpose of this study was to investigate whether the predictive ability

of *Δ*POP changed by different contact force and to find the optimal contact force for utilizing *Δ*POP to guide fluid management in children under general anesthesia. For this, we planned to estimate area under the ROC curve (AUC) of *Δ*POP for discriminating between fluid responder and non-responder at each level of contact force, and evaluate the differences in AUC among the contact force levels. Furthermore, given the observation from our previous study (registered at www.ClinicalTrials.gov;NCT02940938; principal investigator: K.H.S.; date of registration: 9 October 2016) that changes of *Δ*POP by contacting force were significantly different between subjects and the greater amplitude of POP may give more reliability on *Δ*POP, we attempted to evaluate AUC of *Δ*POP at individually different contacting force at which amplitude of POP was maximized.

According to previous study performed in children undergoing, the ratio between fluid responders and non-responders was approximately 1:1, and the area under the receiver operating characteristic (ROC) curve for pleth variability index (PVI) (Masimo Corp., Irvine, CA, USA), which were automatically calculated *Δ*PPG on the basis of the perfusion index (ratio of pulsatile and non-pulsatile component), was 0.767 (17). Therefore, we postulated that *Δ*POP at certain contact force would predict fluid responsiveness with an area under the ROC curve of 0.7 with precision of 0.15 (18). The ratio of fluid responders and non-responders was postulated as 1:1. The required sample size was between 38 and 55, depending on the AUC with an estimated attrition rate of 10%. Therefore, 43 patients finally enrolled in this study.

We divided the patients into two groups following fluid challenge: the "fluid responder group", comprising patients in whom fluid administration increased the stroke volume index (SVI) by > 15%, and the "non-responder group", comprising the remaining patients, as in previous studies (11, 14, 19).

Statistical analyses were performed using SPSS (ver. 22; SPSS, Chicago, IL, USA), MedCalc (ver. 12.7.7; MedCalc, Ostend, Belgium) software and MATLAB 2014 (The Mathworks, Natick, MA, USA). Student's t-test or the Mann-Whitney U-test was used to evaluate the group differences. Paired t-test was used to evaluate the differences in parameters between before and after fluid loading. To determine the ability of ΔPOP to predict fluid responsiveness, ROC curves were generated for each contact force level. AUC at each pressure was calculated and compared among different contact force level. A value of $P < 0.05$ was considered to indicate statistical significance.

3.2 Results

This study enrolled total 43 patients and 5 patients were excluded due to limited echocardiographic measurement. Therefore, 38 pediatric patients who had undergone simple cardiac surgery including atrial septal defect or ventricular septal defect, and neurosurgery were included. Table 3 shows the baseline characteristics of all enrolled patients. There were 20 volume responders and 18 non-responders, and no significant group difference was observed in clinical characteristics.

Changes in hemodynamic parameters after volume expansion

Before Volume expansion, the responder group showed a significant lower cardiac index, SVI and ΔVpeak than the non-responder group (P = 0.03, 0.008, and 0.015, respectively). Volume expansion reduced heart rate and increased central venous pressure in both groups, while cardiac index, SVI and ΔVpeak were significantly changed in only the responder group (all $P < 0.001$) (Table 4). No significant complications such as pulmonary edema or cardiac dysfunction associated with volume expansion were observed in both groups

Predictability of ΔPPG for fluid responsiveness at different contacting force

We determined the 5 contacting forces level as follows; 0 – 0.3N, 0.3 – 0.6N, 0.6 – 0.9N, 0.9 – 1.2N, and 1.2 – 1.5N. Table 3 shows ΔPOP value for fluid responders and non-responders at each contacting force levels. Significant difference was

found in ⊿POP between the responders and non-responders at 0.9 – 1.2N contacting force (P = 0.002), while ⊿POP at other contacting force groups did not show significant differences between the responder and non-responder group (Figure 5).

The mean and standard deviation of individual contacting forces that induce maximum amplitude of POP was 0.9 ± 0.3 N. When ⊿PPG was used at the individual contacting force, the responder group were more clearly discriminated against non-responder group than when using ⊿PPG at the same contacting force for all subjects. According to ROC curve analysis, the AUC Of ⊿PPG at contacting force of 0.9-1.2N and individual contacting force were 0.815 (95% CI, 0.674-0.956; *P* = 0.001) and 0.847 (95% CI, 0.716-0.978; *P* <0.001). The ⊿PPG ≥12.7% at individual contacting force could predict a 15% increase in SVI with a sensitivity of 74% and a specificity of 88%, and ⊿PPG ≥22% at contacting force of 0.9 ~1.2N could predict a 15% increase in SVI with a sensitivity of 59% and a specificity of 94% (Figure 6 a. and Table 5).

Ability of hemodynamic parameters for prediction of fluid responsiveness

Among hemodynamic parameters aside from ⊿PPG, only ⊿Vpeak predicted a 15% increase in SVI (*P* = 0.008) whereas fluid responsiveness was not predicted by heart rate, central venous pressure and PPV. The optimal cut-off value of ⊿Vpeak was 11.3%, with a sensitivity of 78.9% and specificity of 63.2%. The AUC of heart rate, central venous pressure, PPV, and ⊿Vpeak was 0.512 [95%

confidence interval (CI), 0.345 – 0.678], 0.506 (95% CI, 0.301 – 0.710), 0.621 (95% CI, 0.433 – 0.786) and 0.726 (95% CI 0.557 – 0.858), respectively. When hemodynamic parameters were compared in terms of AUC, ΔPOP at both individual contacting force and 0.9 ~1.2N range was better than all other hemodynamic parameters including ΔVpeak in discriminating responder and non-responder group (Figure 6 b)

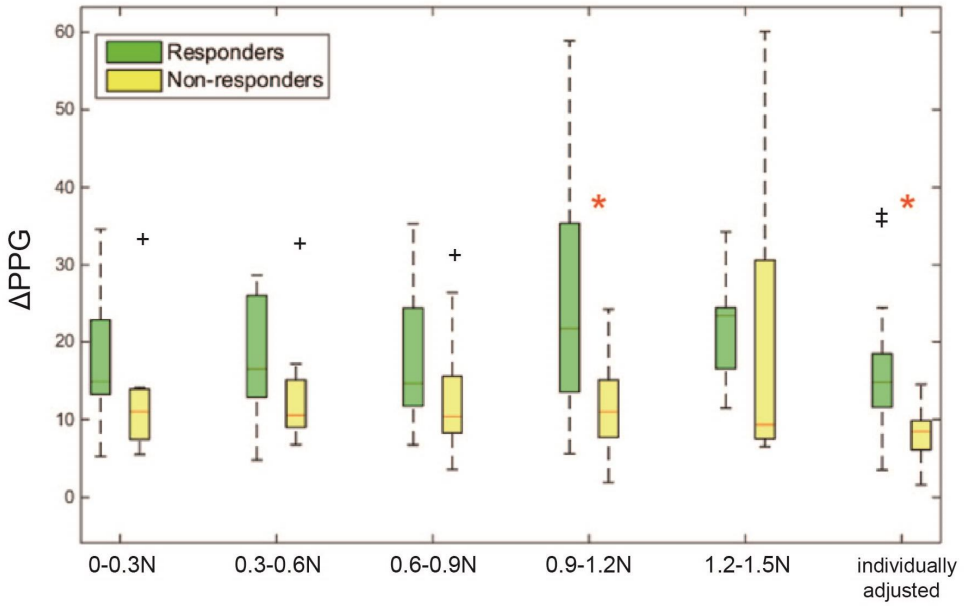

Figure 5. Box plots of the *Δ*PPG values for fluid responders and non-responders at different contacting force levels. Significant difference between the responders and non-responders was found in *Δ*PPG at 0.9 – 1.2N contacting force and individual contacting force. Cross symbol represents extreme values. An asterisk represents $P < 0.05$ between responders and non-responders.

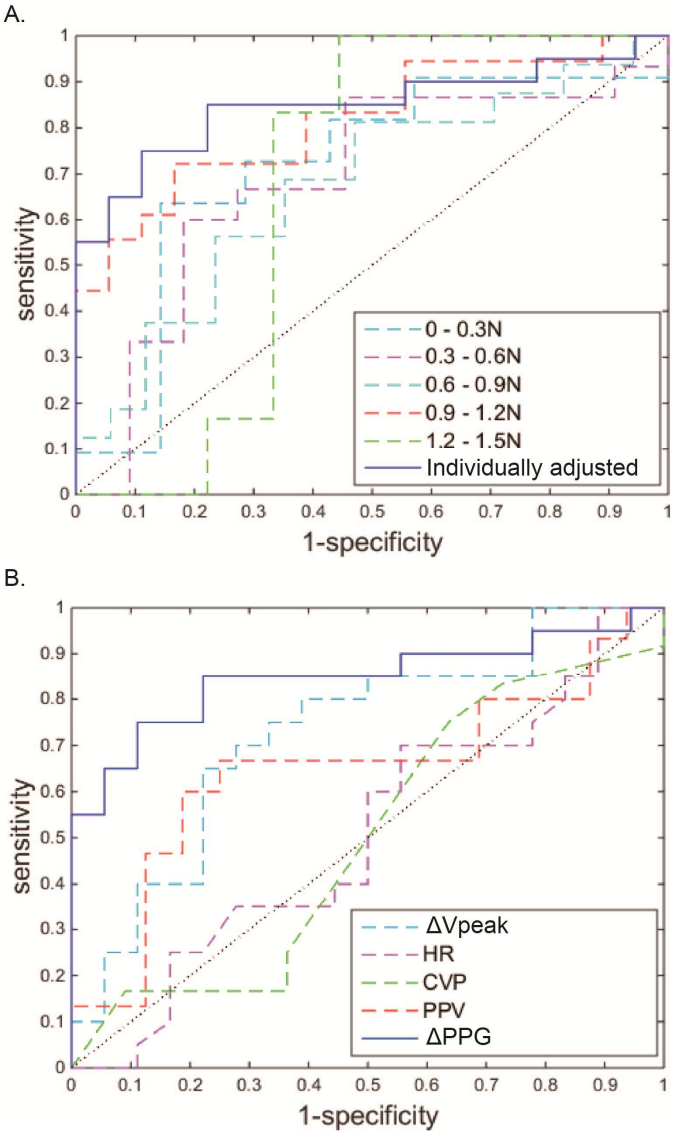

Figure 6. Comparison of the predictive ability of *Δ*PPG at the individually adjusted contacting force. A, with *Δ*PPG at the five contacting force level. B, with other hemodynamic and respiratory variables using areas under the receiver operating characteristic curves.

Table 3. Baseline characteristics of patients and intraoperative variables

Values	Responder (*n*= 20)	Non-responder (*n*=18)
Age (years)	3.2 (1.7; range 0.3 – 5)	3.3 (1.7; range 0.2 – 5)
ASA physical status (I/II/III/IV/V)	18/2/0/0	18/2/0/0
Height (cm)	95.2 (16.6)	97.2 (17.5)
Weight (kg)	15.9 (6.6)	15.8 (5.0)
Sex (male/female)	8/12	11/7
Operations		
Cardiac surgery	9	8
Neurosurgery	11	10
Body temperature (°C)	36.1 (0.8)	36.2 (0.9)
Vasoactive-inotropic score	3.1 (3.7)	4.0 (4.2)

Values are presented as mean (standard deviation) or number

ASA, American society of anesthesiologists

Table 4. Hemodynamic variables before and after volume expansion

Variable	Responder (*n*=20)		Non-responder (*n*= 18)	
	Before VE	**After VE**	**Before VE**	**After VE**
Heart rate (beats/min)	116.9 (4.4)	112.3 (17.8)*	115.9 (21.9)	105.3 (25.8)*
Systolic arterial pressure (mmHg)	95.3 (13.2)	98.0 (17.8)*	93.0 (15.7)	97.9 (15.6)
Diastolic arterial pressure (mmHg)	47.5 (7.7)	53.7 (7.0)*	52.6 (9.9)	55.1 (11.3)
Mean arterial pressure (mmHg)	62.8 (9.9)	70.3 (10.6)*	67.2 (11.9)	69.7 (13.6)
Central venous pressure (mmHg)	7.0 (2.7)	9.0 (2.4)*	6.9 (3.2)	8.9 (3.0)*
Peak airway pressure (cmH_2O)	15.7 (2.9)	16.2 (3.5)	14.3 (1.3)	14.8 (1.7)
Cardiac index ($L/min/m^2$) †	2.5 (0.6)	3.2 (0.9)*	3.2 (1.3)	3.1 (1.5)
Stroke volume index (ml/m^2) †	22.4 (5.7)	28.8 (7.4)*	27.7 (11.2)	29.2 (11.10)
Pulse pressure variation (%)	15.3 (5.1)	14.7 (5.7)	15.0 (6.6)	13.2 (4.6)
ΔVpeak (%)†	14.7 (3.6)	9.5 (4.3)*	11.3 (3.4)	9.8 (4.0)

Variables are expressed as mean (SD).

*$P < 0.05$ before VE vs after VE

†$P < 0.05$ between responders and non-responders before VE.

VE, volume expansion

Table 5. The *Δ*PPG values of fluid responders and non-responders at different contacting force levels.

Contacting force levels	*Δ*PPG of Responders	*Δ*PPG of Non-responders	*P* value*	AUC(95% CI)
0 – 0.3N	14.9 (13.2-22.8) (n=11)	11.0 (7.5-13.9) (n=7)	0.151	0.714(0.451-0.978)
0.3 – 0.6N	16.5 (12.9-26.1 (n=15)	10.6 (9.1-15.1) (n=11)	0.121	0.685(0.465-0.904)
0.6 – 0.9N	14.7 (11.7-24.3) (n=16)	10.4 (8.3-15.6) (n=17)	0.094	0.673(0.486-0.860)
0.9 – 1.2N	21.7 (13.5-35.3) (n=18)	11.0 (7.7-15.1) (n=18)	0.001	0.815(0.674-0.956) ‡
1.2 – 1.5N	23.4 (16.5-24.4) (n=6)	9.4 (7.5-30.6) (n=9)	0.328	0.667(0.373-0.960)
Optimal contacting force†	14.8 (11.6-18.5) (n=19)	8.4 (6.1-9.8) (n=19)	0.000	0.847(0.716-0.978) ‡

Data were presented as median (interquartile ranges)

**P* value from comparison between responders and non-responders using Mann-Whitney U test.

†Optimal contacting force was defined as the contacting force where the maximal PPG amplitude was derived in each patients.

‡$P < 0.05$ for ROC curve analysis

PPG, pulse oximeter photoplethysmography

4. Discussion

4.1 Discussion of the first study

We observed that contact force changes induced PPG amplitude changes and thus respiratory variations in PPG amplitudes and *Δ*PPG in anesthetized children. The maximal PPG amplitude was shown at contact forces between 0.4–0.6 N, whereas the value of *Δ*PPG was lowest at these contact forces. To our knowledge, this is the first study to investigate contact force effects on PPG-derived parameters in children.

Acute changes in cardiac output are usually observed during surgery and the optimization of volume status is an important factor for maintaining cardiac output. Assessment of volume status using routine monitoring is difficult, especially in pediatric patients (20). *Δ*PPG has been proposed as a non-invasive predictor of fluid responsiveness (11, 13). However, PPG amplitudes can be affected by many factors including vasomotor tone, level of sedation, presence of nociceptive input, and body temperature. Additionally, the contact force between a sensor and a measurement site can be an important factor. A previous report emphasized that pulse oximeter sensor should be carefully placed to avoid restrictive taping, which can limit arterial blood flow (21). Changes in PPG amplitude by the contact force can result in changes in *Δ*PPG. The cut-off value for *Δ*PPG that discriminated between fluid responders and non-responders was 13–14% in adults (11, 12) and can be changed according to the contact force.

The PPG AC amplitude is mainly determined by vascular compliance (the distensibility of arterioles) (4). Amplitude-based compliance, the change in arterial volume over that in the corresponding arterial pulse pressure, was maximal when the transmural pressure (the difference between intra-arterial pressure and external pressure) was zero (22). Previously, significant changes in PPG amplitude were shown with changing transmural pressure (23). In this study, transmural pressure might be close to zero at contact forces between 0.4 and 0.6 N, which was the maximal AC amplitude. Increases in contact force resulted in decreases in transmural pressure, arterial distensibility, and PPG AC amplitude.

The results of this study, which showed a negative correlation between ΔPPG and the AC amplitude, suggested that ΔPPG would change as transmural pressure changed. This is presumably because in the ΔPPG calculation, when the AC amplitude increases, the average of the maximal and minimal PPG amplitudes, which is the denominator, is more influential than the difference between maximal and minimal PPG amplitudes, which is the numerator. The effect of transmural pressure on PPG components other than AC amplitude might be negligible after the ΔPPG calculation.

Arterial distensibility could vary depending on microvascular tone changes induced by temperature, sympathetic system activity (24), and external compression. Previous studies demonstrated that in decreasing arterial compliance, such as vasoconstriction induced by sympathetic activity or norepinephrine release after pneumoperitoneum, ΔPPG increased (25, 26). Similarly, changes in arterial

compliance induced by external compression resulted in an increased *Δ*PPG in children while the AC amplitude decreased.

However, the DC amplitude tended to increase when contact force increased. This result was in accordance with Teng et al.'s study (9), which demonstrated that the DC amplitude increased from 1.54 ± 0.19 V to 1.82 ± 0.15 V with increasing contact forces from 0.21–0.80 N at the finger in adults. According to Mascaro et al., when contact force at the fingertip exceeded 0.3 N, venous return was restricted and resulted in pooling of the arterial blood in the capillaries beneath the fingernail (27). Therefore, the portion of the non-pulsatile DC component increased with the contact force.

Some limitations should be mentioned. First, we measured core temperature, not local temperature, during the study. Although all patients showed normothermia, there might be bias from peripheral temperature variations because it could affect the finger's vasculature and plethysmography amplitude (28). Second, although contact forces were the same, there might be individual pressure variability from differences in finger dimensions. The results regarding contact force associated with the maximal PPG amplitude or *Δ*PPG may not be applicable to infants or older children. Additionally, we could not measure the vascular resistance and cardiac output which can affect the AC and DC amplitude. Although we attempted to maintain stable anesthetic depth, bias from any factors affecting cardiac output and vascular tone should be considered. Finally, we did not evaluate which contact force was associated with the optimal *Δ*PPG to predict fluid

responsiveness. Further study is required to evaluate the ability of *Δ*PPG to detect fluid responsiveness at each force level in children.

In summary, PPG amplitudes and respiratory variations in PPG amplitudes were affected by the contact force between the measurement site and sensor in children under general anesthesia. The AC amplitude and the AC/DC ratio were maximal in the force range of 0.4–0.6 N and decreased as force increased. *Δ*PPG also changed according to variable contact forces, implying that it may not reflect stroke volume variation induced by respiration. Additionally, the previously known cut-off value for *Δ*PPG to discriminate between volume responders and non-responders should be changed according to the contact force. Therefore, contact force should be considered for PPG signal analysis and when utilizing *Δ*PPG to predict fluid responsiveness.

4.2 Discussion of the second study

In the second study, we found that the ability to predict fluid responsiveness of *Δ*PPG was affected by contacting force between the pulse oximeter sensor and measurement site; Among 5 contacting force levels, in only one contacting force level, 0.9 – 1.2N, the fluid responsiveness was statistically predicted. In addition, when we found that *Δ*PPG at the individually different contacting force level which induced maximal POP amplitude among 5 contacting force levels showed the best predictability of fluid responsiveness prediction in children under mechanical ventilation.

The *Δ*PPG has been suggested as promising dynamic indicator of fluid responsiveness for its easy access in clinical situation and non-invasive feature compared to PPV and *Δ*Vpeak which are invasive or not easily available (11, 13). Despite its advantage, several previous studies using plethysmography have had conflict results regarding reliability of respiratory variations in PPG amplitude in assuming the preload status during surgery in mechanically-ventilated children (14, 17, 29). Pereira de Souza Neto et al.(14) found that neither *Δ*PPG nor pleth variability index (PVI) could predict response to volume expansion in children undergoing neurosurgery. However, Byon et al.(17) and Renner et al.(29) reported that PVI was a useful parameter to predict fluid responsiveness in children undergoing neurosurgery and cardiac surgery. On the contrary, we observed both positive and negative ability of *Δ*POP in discriminating between responder and non-responder depending on the contacting force which is inevitably exerted

between the finger and the sensor. According to our results, ⊿POP is only reliable when contacting force is controlled, which is possibly the reason other previous studies have shown the negative ability of ⊿PPG as a fluid responsiveness predicting parameter.

In the present study, the reliability of ⊿PPG was maximized when contacting force at which PPG amplitude was maximum was applied individually. The mean value of individually adjusted contacting force was about 0.9, which falls within the controlled contacting force level (0.9 ~ 1.2N). Taking the results of the first and second studies together, contacting force should be controlled inducing transmural pressure close to zero for reliability of ⊿PPG. When transmural pressure is close to zero, vascular compliance increases resulting in maximal AC amplitude and other vessel variations such as respiratory variation can be intensified. Our result is in accord with the earlier study of Broch et al. (30), who reported that the accuracy of PVI was improved in high perfusion index (>4%), which corresponds to greater amplitude of PPG and argued PVI has to be used with caution, depending on perfusion index (30).

Maximal AC amplitude was obtained at contacting force between 0.4 and 0.6 N in the first study while 0.9-1.2 N in the second study. The possible reason was that the age range was different between the two studies. Additionally, considering the great standard deviation of contacting force that induce maximum PPG amplitude, the individual control rather than applying mean value is desirable for utilizing PPG-derived parameters.

Along with ⊿PVI, PPV has been suggested as predictors for increase in cardiac output after volume expansion in adults (11, 31). However, in pediatric population, ⊿Vpeak has been extensively studied and shown as the only predictive parameter with cut-off values ranging from 7% to 20% (14, 17, 29, 32). In agreement with the previous studies, ⊿Vpeak could predict fluid responsiveness in the present study with cut off value of 11.3% while PPV could not. The predictability of ⊿Vpeak may be explained by the fact that the ⊿Vpeak was measured at aortic valve level at which blood flow pass the relatively large area and the diameter was unchanged, and thus the velocity might be correlated directly to amount of stroke volume.

Several limitation should be mentioned. First, we did not measure the perfusion index, which had to be maintained stable during obtaining PPG signal. However, the duration of data acquisition was short and no acute bleeding, and significant changes in hemodynamics and anesthetic depth were observed during measurement of PPG. Second, it can be viewed impractical in clinical situation to measure the contacting force and find out the optimal contacting force in which maximum amplitude of POP can be measured prior to volume expansion. However, if the optimal pressure is automatically set within a short period by an automatic feedback system consisting of a pulse oximeter sensor and automatic contacting force controller, which should be dealt with in a future study, more reliable prediction with ⊿PPG can be achieved in a practical way. Third, the differences in compliance of individual arterial system was not considered in the present study.

Finally, we measured the force, not the pressure which is the force per area. Therefore, the optimal contacting force range from this study may not be applied to older children or adolescents. However, if PPG can be measured using optimal contacting force individually, clinicians can obtain more accurate, useful information and improve the hemodynamic management in critically ill children.

In summary, the present study demonstrates the different predictability of ΔPPG in predicting fluid responsiveness depending on the contacting force exerted between the sensor and measurement site in mechanically ventilated children. In only controlled contacting force which maximizes the amplitude of PPG, ΔPPG can predict fluid responsiveness.

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국문 초록

배경: 적절한 수액 요법을 위해 수액 반응성을 예측하는 것은 매우 중요하다. 호흡에 따른 photoplethysmography (PPG) 파형 높이의 변화(*Δ*PPG)는 체내 혈류 상태를 반영한다. 그러나 소아에서 수액 반응성 예측을 위한 *Δ*PPG의 유용성에 대한 과거 연구들은 서로 상충되는 결과를 보여주고 있다. PPG 측정 센서와 측정 부위의 접촉 압력은 PPG 파형 높이와 *Δ*PPG 값에 영향을 미칠 수 있다. 따라서 본 연구에서는 접촉 압력이 PPG 파형과 *Δ*PPG의 신뢰성에 미치는 영향을 알아보고자 한다.

방법: 2단계 연구가 수행되었다. 첫 번째 관찰연구에는 3-5세의 간단한 수술을 받는 소아 환자들을 대상으로 연구가 진행되었다. 마취 유도 후 일회 호흡량 10 mL/kg로 기계 환기를 시작하였다. 앙와위 자세에서, 클립식 PPG 센서를 검지 손가락에 부착하고 접촉 압력을 0에서 1.4N까지 0.2N씩 증가시키며 각 압력에서 20번의 호흡 주기 동안 PPG 파형을 얻었다. AC, DC 진폭, AC/DC 비 및 *Δ*PPG를 계산하였다. 두 번째 연구에서는 접촉 압력 변화에 따른 수액 반응성의 지표로서 *Δ*PPG의 예측력의 신뢰성을 평가하였다. 신경외과 및 흉부외과 수술 등 대 수술을 받는 1개월-5세 사이의 소아를 대상으로, 다섯 단계의 접촉 압력 (0-0.3N, 0.3-0.6N, 0.6-0.9N, 0.9-1.2N 및 1.2-1.5N) 및 개별 조정된 접촉 압력에서 수액 투여 전 *Δ*PPG를 측정하였다. 수액 투여 후 일회 박출 지수가 15% 이상 증가

한 경우 반응군으로 정의하였다.

결과: 총 34명의 데이터가 첫 번째 연구에서 분석되었다. Normalized AC 진폭은 접촉 압력 0.4-0.6N에서 최대였으며 접촉 압력이 증가함에 따라 감소하였다. 반면 normalized DC 진폭은 접촉 압력 0.4N 이상에서 증가하였다. ⊿PPG는 약간 감소하다가 AC 진폭이 감소하기 시작한 지점에서 꾸준히 증가하였다. 전체적으로 0.2-1.2N 사이에서 각 접촉 압력 단계의 AC, DC 진폭, AC/DC 비 및 ⊿PPG는 유의하게 변화하였다. 두 번째 연구에서는 총 38명의 데이터가 분석되었다. 접촉 압력 0.9-1.2N과, PPG 파형을 최대화시키는 접촉 압력 (0.9±0.3N)에서 수액 반응군과 비반응군 사이의 ⊿PPG에 유의한 차이가 있었다. 또한 이 두 접촉 압력 범위에서의 ⊿PPG가 수액 반응성을 예측할 수 있었다.

결론: 기계 환기를 받는 소아에서 PPG 센서와 측정 부위의 접촉 압력에 따라 PPG 파형 진폭 및 ⊿PPG가 유의하게 변화하였다. 접촉 압력이 적절하게 조절된다면, 소아에서 ⊿PPG의 수액 반응성에 대한 예측력을 개선시킬 수 있을 것이다.

주요어: 수액 반응성; 소아; photoplethysmography; 접촉 압력

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