Comparison of Traditional and Plethysmographic Methods for Measuring Pulsus Paradoxus

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Background: In the evaluation of patients with acute asthma, pulsus paradoxus (PP) is an objective and noninvasive indicator of the severity of airway obstruction. However, in children PP may be difficult or impossible to measure. Indwelling arterial catheters facilitate the measurement of PP, but they are invasive and generally reserved for critically ill patients.

Objective: To determine the utility of the plethysmographic waveform (PP_{pleth}) of the pulse oximeter in measuring PP.

Methods: Patients from the pediatric intensive care unit, emergency department, and inpatient wards of a tertiary care pediatric hospital were eligible for the study. A total of 36 patients (mean age [SD], 11.2 [4.7] years) were enrolled in the study. Pulsus paradoxus was measured using the traditional auscultatory (PP_{ausc}) method with a sphygmomanometer. Pulsus paradoxus was then measured using a blood pressure cuff observing for the disappearance and reappearance of the (PP_{pleth}) on the pulse oximeter. Mean difference and 95% confidence intervals were calculated for each method. The 2 methods were also analyzed for correlation and agreement using the Pearson product moment correlation and a Bland and Altman plot.

Results: Patients with status asthmaticus had higher PP_{ausc} and PP_{pleth} readings compared with nonasthmatic patients. Pulsus paradoxus measured by plethysmography in patients with and without asthma was similar to PP_{ausc} readings (mean difference, 0.6 mm Hg; 95% confidence interval, −0.6 to 2.1 mm Hg). Individual PP_{pleth} readings showed significant correlation and agreement with PP_{ausc} readings in patients both with and without asthma.

Conclusion: Measurement of PP using the pulse oximeter–pulse plethysmographic waveform offers a simple and noninvasive method for evaluating patients with airway obstruction.

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DESPITE ADVANCES IN THE treatment and prevention of asthma, it remains one of the most common reasons for hospitalization in the pediatric age group.[1] Periodic assessment of the severity of airway obstruction is an integral part of the management of status asthmaticus. However, there are limitations to the various subjective and objective factors that are routinely used for assessment. There is a wide variation in how patients perceive and describe their symptoms.[2] The symptoms of an attack such as cough, difficulty breathing, and wheezing frequently do not correlate with severity. In addition, there is a significant interobserver variability among physicians in evaluating physical signs such as the extent of wheezing and magnitude of respiratory distress.[3,4] Some helpful tools in evaluating the severity of asthma are pulse oximetry, blood gas sampling, and bedside spirometry. However, a decrease in oxygen saturation may occur with only very mild airway obstruction or, conversely, may not occur at all even with severe airway obstruction. In addition, hypoxemia may be masked by administration of small amounts of supplemental oxygen, such as with aerosol treatments. Spirometry, such as peak expiratory flow rate, can be useful as an indicator of airway obstruction and as a measure of decreased pulmonary function from baseline in asthmatic individuals who use them regularly. Despite this, the utility of the peak expiratory flow rate is limited in a child with severe respiratory distress. Measurement of the partial pressure of arterial carbon dioxide ($PaCO_2$) is a useful tool in evaluating alveolar ventilation. In mild and moderate asthma the $PaCO_2$ is usually low. "Normalization" or significant elevations in $PaCO_2$ are encountered only when air-

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way obstruction is severe enough to result in overall alveolar hypoventilation. In addition, measurement of Pa_{CO_2} requires blood sampling.

Pulsus paradoxus (PP) reflects an exaggeration of the normal decrease in systolic blood pressure (SBP) during inspiration and is a quantifiable indicator of airway obstruction. It has been shown to correlate well with other objective indicators of airway obstruction such as Pa_{CO_2} and peak expiratory flow rate[5,6]; however, it is noninvasive and does not require cooperation with pulmonary function tests. Pulsus paradoxus may be a useful adjunct in assessing the severity of airway obstruction at a given time, as well as documenting response to therapy, especially in situations in which more invasive tests are not readily available, such as an office setting. Timing of PP measurement to inspiration and expiration can be difficult in asthmatic children who are tachypneic, tachycardic, and likely to be uncooperative. Therefore, instead of attempting to correlate SBP with phases of respiration, measurement of PP using variations in SBP has been proposed in children.[7] Nevertheless, such a measurement is cumbersome and subject to considerable interrater and intrarater variability as well as observer bias. Improvements in the method of determining PP may allow it to be a more useful tool in evaluating airway obstruction.

Newer pulse oximeters are equipped with a visual display of the pulse plethysmographic waveform (PP_{pleth}). We proposed that the PP_{pleth} of the pulse oximeter would be an acceptable substitute for Korotkoff sounds for the measurement of PP. Our hypothesis was PP measured by the pulse oximetry PP_{pleth} would be greater in asthmatic children compared with nonasthmatic children. Furthermore, PP measured using the pulse oximeter plethysmograph would have good agreement with the value obtained by the traditional method of auscultation of Korotkoff sounds.

METHODS

Thirty-six patients from the pediatric intensive care unit, emergency department, and inpatient wards of the Children's Hospital of Michigan, Detroit, were enrolled over a 4-month period. Both asthmatic and nonasthmatic patients were evaluated. All asthmatic patients were at the time of evaluation diagnosed as having status asthmaticus, which was defined as the acute onset of symptomatic expiratory airflow obstruction without significant improvement after administration of aerosolized β-agonist treatments. Verbal consent was obtained by the primary investigator (J.A.C.) from the guardian or patient and documented in the patient's medical record. This study was approved by the Wayne State University Human Investigations Committee. The patients were chosen by their ability to cooperate with study requirements (ie, remain still) from the emergency department and inpatient populations (generally aged from 0 to 18 years).

All demographic data and PP measurements were taken by a single investigator (J.A.C.). Age, sex, and underlying disease were recorded for all patients. Rhythm strip showed all patients to be in sinus rhythm with a stable rate. Blood pressure was obtained manually using an aneroid sphygmomanometer (Welch-Allyn, Skaneateles Falls, NY). Pulsus paradoxus was determined using the traditional auscultatory (PP_{ausc}) method with the sphygmomanometer. With the patient sitting with the arm at the side, an appropriately sized arm cuff was inflated until all Korotkoff sounds disappeared. The cuff was deflated approximately 2 mm Hg per second until a few intermittent Korotkoff I sounds were heard. This pressure was assumed to be the SBP during exhalation (P_{ex}). The cuff pressure was similarly decreased until all Korotkoff I sounds were heard. This pressure was assumed to be the SBP during inspiration (P_{in}). The difference between P_{in} and P_{ex} was the PP_{ausc}. If P_{ex} and P_{in} were not well defined during the initial deflation, the cuff was reinflated once and an attempt at measurement was repeated. If Korotkoff I sounds were again not well heard, the cuff was deflated completely, and this process was repeated after a short wait, a maximum of 3 times in total. The first PP that was clearly heard was recorded as PP_{ausc}. If PP was not clearly discernible by auscultation, the patient was excluded from the study.

Pulsus paradoxus was then determined in the same patient using the PP_{pleth} from a standard patient transport monitor (Marquette Electronics, Milwaukee, Wis). The pulse oximeter probe (Nellcor, Pleasanton, Calif) was placed on the patient's second or third finger of the same arm used for PP_{ausc} and an adequate signal quality was ascertained by comparison of the displayed PP_{pleths} to the palpable pulse. The appropriate-sized cuff was placed on the arm and inflated until the PP_{pleth} disappeared from the monitor. The cuff pressure was then decreased at approximately 2 mm Hg per second in a similar fashion to PP_{ausc} determination, until intermittent PP_{pleths} appeared and this pressure was assumed to be the SBP during P_{ex}. The cuff pressure was lowered further until all PP_{pleths} were displayed. This pressure was the SBP during P_{in}. The difference between P_{ex} and P_{in} was the PP_{pleth}.

Pulsus paradoxus by auscultation and PP_{pleth} means (SDs) were calculated, as well as the mean difference between the 2 methods with 95% confidence intervals. Pearson product moment correlation was used to compare values obtained by the different methods. In addition, because of concerns regarding the use of the product moment correlation for the comparison of different methods of measurement, we also compared the readings using a Bland and Altman plot, giving an estimation of agreement.

RESULTS

During a 4-month period, 36 patients were enrolled. Their ages ranged from 27 months to 20 years, with a mean (SD) age of 11.2 (4.7) years. Twenty patients were male, and 16 were female. Eleven were admitted having a diagnosis of status asthmaticus; the remaining 25 patients had various diagnoses none of which was expected to generate a significant PP (**Table 1**). Five patients were excluded after consent owing to the inability to measure PP_{ausc} because of the patient's inability to remain still during PP measurement. In all patients whose PP_{ausc} was discernible, PP_{pleth} was able to be measured as well.

The PP was significantly higher in the asthmatic group compared with the nonasthmatic group when measured by either auscultation or plethysmography (**Table 2**). When PP_{pleth} readings were compared with PP_{ausc} readings, no statistically significant difference was found (Table 2).

Pearson product moment correlation was used to determine the correlation between the values for PP determined by the 2 methods (**Figure 1**). The PP_{ausc} and the PP_{pleth} method showed significant correlation among both asthmatic patients (r^2=0.76, P<.01) and nonasth-

Table 1. Patient Diagnoses

Diagnosis	No. of Patients
Status asthmaticus	11
Sickle cell anemia	4
Gastroenteritis	3
Malignancy	3
Pneumonia	2
Intussusception	1
Ventriculoperitoneal shunt malfunction	1
Seizure	1
Congenital heart disease	1
Varicella	1
Bone marrow transplantation	1
Diabetic ketoacidosis	1
Pharyngitis	1
Bladder augmentation	1
Chronic renal failure	1
Lung abscess	1
Adjustment disorder	1
Decubitus ulcer	1

Table 2. Pulsus Paradoxus Values for Asthmatic and Nonasthmatic Patients

Variable	No. of Asthmatic Patients (n = 11)	No. of Nonasthmatic Patients (n = 25)
PP_{ausc}, mean (SD), mm Hg	10.9 (2.6)	5.6 (1.7)
PP_{pleth}, mean (SD), mm Hg	10.2 (2.2)	6.0 (1.7)
Mean difference, $PP_{ausc}-PP_{pleth}$	0.6	−0.4
95% CI	−0.6 to 2.1	−1.2 to 0.4

Abbreviations: CI, confidence interval; PP_{ausc}, pulsus paradoxus measured by auscultation; PP_{pleth}, pulsus paradoxus measured by plethysmographic waveform method.

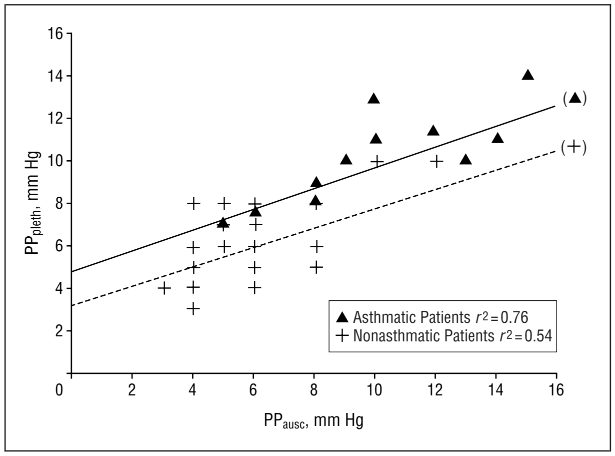

Figure 1. Correlation between pulsus paradoxus measured by auscultation (PP_{ausc}) and pulsus paradoxus measured by the plethysmographic waveform method (PP_{pleth}).

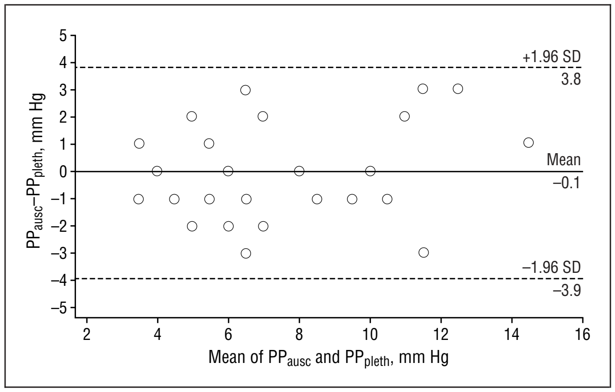

Figure 2. Bland and Altman plot for all patients. PP_{ausc} indicates pulsus paradoxus measured by auscultation; PP_{pleth}, pulsus paradoxus measured by the plethysmographic waveform method.

matic patients (r^2=0.54, $P<.01$). In addition, a Bland and Altman plot (**Figure 2**) shows good agreement between the 2 methods, with all differences being 1 SD or less from the mean.

COMMENT

Pulsus paradoxus is a phasic variation in SBP with the lowest pressure occurring during P_{in} and the highest during P_{ex}. The physiologic mechanisms for this are thought to be related to changes in left ventricular stroke volume and afterload during the respiratory cycle.[8,9] During P_{in}, the reduction in pleural pressure causes a decrease in intrathoracic pressure, and increases the pressure gradient from the systemic venous reservoir to the right ventricle. This increases venous return to the right ventricle. Since there is no increase in the gradient from the pulmonary veins to the left ventricle, a concurrent increase in left ventricular filling does not occur. Therefore, the interventricular septum at end-diastole is displaced to the left, and left ventricular end-diastolic volume decreases, with a resultant drop in left ventricular stroke volume. In addition, an increase in negative intrathoracic pressure increases the pressure gradient against which the left ventricle has to pump (left ventricular afterload), which decreases left ventricular stroke volume. As left ventricular stroke volume falls during P_{in}, SBP decreases. During P_{ex}, these physiologic changes are reversed, resulting in an increase in preload and a decrease in afterload of the left ventricle, leading to an increase in stroke volume and an increase in the SBP. With airway obstruction, such as that seen in asthma, these changes are magnified, resulting in a greater drop in SBP during P_{in}, and an increase in PP. The magnitude of PP has been shown by Galant et al[6] to correlate with other objective indicators of airway obstruction, such as an increase in Pa_{CO_2}, and a decrease in forced expiratory volume in the first second and peak expiratory flow rate in asthmatic children.

Because PP provides an objective measure of airway obstruction, the National Heart, Lung, and Blood Institute has recommended its measurement as one estimate of the severity of an asthma exacerbation.[3,5] The recommendations of the National Heart, Lung, and Blood Institute for management of an acute asthma exacerbation are based on severity, so PP provides an objective measure to guide treatment. The National Heart, Lung, and Blood Institute recommendations for measuring PP_{ausc} in children are to determine the difference in systolic blood pressure between the pressure at which an observer first hears sporadic, faint pulse sounds and the pressure at which he or she hears all sounds. No attempt should be made to correlate pulsus paradoxus with the phase of respiration in small children.[7(p508)]

Despite this, relative tachypnea, tachycardia, small size, and inability of children to remain still make the measurement of PP difficult or impossible. Indeed, it was necessary to exclude 5 patients from our study because of an inability to determine PP_{ausc}. The use of indwelling arterial catheters has made the measurement of PP easier, but their use is invasive and not without risk, and is generally reserved for critically ill patients.

Newer pulse oximeters are equipped with a visual display of the (PP_{pleth}). The generation of the PP_{pleth} is based on the fact that the pulsatile absorbance between the light source and the detector of the pulse oximeter probe is from arterial blood. A PP_{pleth} is generated by separating the pulsatile component of the absorption from the nonpulsatile component. The nonpulsatile component represents absorption from the tissue bed, including the venous, capillary, and nonpulsatile arterial blood. The amount of "pulse-added" absorption is calculated by the pulse oximeter and a PP_{pleth} is generated that corresponds to pulsatile flow through the tissue.[10] To our knowledge, the use of measuring PP using the pulse oximeter PP_{pleth} has not been examined.

Potential pitfalls in the method used here are that a single observer (J.A.C.) obtained all data, and PP readings were not taken randomly. Both of these issues introduce the possibility of observer bias. Because PP is present under normal conditions, our design was meant to demonstrate the utility of using the pulse oximeter to measure PP. However, most patients included in this study had mild asthma, and it is possible that agreement between the PP_{ausc} and PP_{pleth} methods may yield results that do not agree when PP is further exaggerated. Additional study into this is warranted, possibly including comparison to PP measured by intra-arterial pressure monitoring.

Our data show that the PP_{pleth} of the pulse oximeter can be used to measure PP. This may be helpful in situations in which auscultation is difficult, such as a noisy emergency department, or with a crying child. Because PP can be useful in quantifying the severity of an asthma exacerbation, such data may be helpful in directing therapy or determining the need for continued observation. Nevertheless, technical difficulties still exist. Although it may be easier to measure PP using a visual PP_{pleth}, patient cooperation may still be necessary because movement artifact can result in inadequate pulse oximeter waveforms.[11] Advances in pulse oximeter technology that help remove artifact from the waveform are being evaluated and may alleviate this problem.[11,12] In addition, standardization of the PP_{pleth} to actual arterial pressures would simplify PP measurements further. This would make PP measurement similar to that of PP measurement based on the difference in heights of the P_{in} and P_{ex} SBP waveforms from indwelling arterial catheters but without the invasive arterial catheter placement. Unfortunately, this technology is not widely available. Until such time, we recommend the use of the PP_{pleth} for the measurement of PP when PP_{ausc} may be difficult to obtain.

What This Study Adds

Pulsus paradoxus has been shown to be one of the few noninvasive and objective indicators of airway obstruction. However, in children, PP can often be difficult or impossible to measure. Therefore, any technique that would facilitate its measurement would enhance our ability to objectively assess asthma severity in children.

It is apparent from the existing literature that the reappearance of the pulse oximeter PP_{pleth} during arm cuff deflation correlates with the return of pulsatile blood flow as measured from a radial artery catheter. It has not been demonstrated that the reappearance of the pulse oximeter PP_{pleth} can be used to measure PP. From our data, we believe that PP_{pleth} offers an easier yet accurate alternative to PP_{ausc}.

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REFERENCES

1. Centers for Disease Control (CDC) National Asthma control program. asthma control programs and activities related to children and adolescents: reducing costs and improving quality of life. Available at: http://www.cdc.gov/nceh/airpollution/asthma/children.htm. Accessed April 1, 2002.
2. Li JTC, O'Connell E. Clinical evaluation of asthma. *Ann Allergy Asthma Immunol.* 1996;76:1-10.
3. National Heart, Lung, and Blood Institute (NHLBI). *National Asthma Education and Prevention Program Expert Panel Report 2: Guidelines for the Diagnosis and Management of Asthma.* Washington,DC: National Heart, Lung, and Blood Institute; 1997. NIH publication No. 97-4051. Also available at: http://www.nhlbi.nih.gov/guidelines/asthma/asthgdln.htm. Accessed April 1, 2002.
4. Shim CS, Williams MH. Evaluation of the severity of asthma: patients versus physicians. *Am J Med.* 1980;68:11-13.
5. Martell JA, Lopez JG, Harker JE. Pulsus paradoxus in acute asthma in children. *J Asthma.* 1992;29:349-352.
6. Gallant SP, Groncy CE, Shaw KC. The value of pulsus paradoxus in assessing the child with status asthmaticus. *Pediatrics.* 1978;61:46-51.
7. National Heart, Lung, and Blood Institute (NHLBI). National Asthma Education Program Expert Panel Report: guideline for the diagnosis and management of asthma. *J Allergy Clin Immunol.* 1991;88(pt 2):425-534.
8. McGregor M. Pulsus paradoxus. *N Engl J Med.* 1979;301:480-482.
9. Blaustien AS, Risser TA, Weiss JW, Parker JA, Holman BL, McFadden ER. Mechanisms of pulsus paradoxus during resistive respiratory loading and asthma. *J Am Coll Cardiol.* 1986;8:529-536.
10. Schnapp LM, Cohen NH. Pulse oximetry: uses and abuses. *Chest.* 1990;98:1244-1250.
11. Lindberg LG, Lennmarken C, Vegfors M. Pulse oximetry: clinical implications and recent technical developments. *Acta Anaesthesiol Scand.* 1995;39:279-287.
12. Scuderi PE, Bowton DL, Anderson RL, Prough DS. Pulse oximetry: would further technical alterations improve patient outcome. *Anesth Analg.* 1992;74:177-180.