CHEST

Official publication of the American College of Chest Physicians

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Chest 2005;128;3013-3019
DOI 10.1378/chest.128.4.3013

Use of Pulse Transit Time To Distinguish Respiratory Events From Tidal Breathing in Sleeping Children*

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Study objectives: **Currently, esophageal pressure monitoring is the "gold standard" measure for inspiratory efforts, but its invasive nature necessitates a better tolerated and noninvasive method to be used on children. Pulse transit time (PTT) has demonstrated its potential as a noninvasive surrogate marker for inspiratory efforts. The principle velocity determinant of PTT is the change in stiffness of the arterial wall and is inversely correlated to BP. Moreover, PTT has been shown to identify changes in inspiratory effort via the BP fluctuations induced by negative pleural pressure swings. In this study, the capability of PTT to classify respiratory events during sleep as either central or obstructive in nature was investigated.**

Setting and participants: **PTT measure was used in adjunct to routine overnight polysomnographic studies performed on 33 children (26 boys and 7 girls; mean ± SD age, 6.7 ± 3.9 years). The accuracy of PTT measurements was then evaluated against scored corresponding respiratory events in the polysomnography recordings.**

Results: **Three hundred thirty-four valid respiratory events occurred and were analyzed. One hundred twelve obstructive events (OEs) showed a decrease in mean PTT over a 10-sample window that had a probability of being correctly ranked below the baseline PTT during tidal breathing of 0.92 ($p < 0.005$); 222 central events (CEs) showed a decrease in the variance of PTT over a 10-sample window that had a probability of being ranked below the baseline PTT of 0.94 ($p < 0.005$). This indicates that, at a sensitivity of 0.90, OEs can be detected with a specificity of 0.82 and CEs can be detected with a specificity of 0.80.**

Conclusions: **PTT is able to categorize CEs and OEs accordingly in the absence of motion artifacts, including hypopneas. Hence, PTT shows promise to differentiate respiratory events accordingly and can be an important diagnostic tool in pediatric respiratory sleep studies.**

(CHEST 2005; 128:3013–3019)

Key words: central sleep apnea; hypopnea; noninvasive technique; obstructive sleep apnea; pediatrics; pulse transit time; upper airway

Abbreviations: AHI = apnea-hypopnea index; AUC = area under the receiver operating characteristic curve; CE = central event; HR = heart rate; OE = obstructive event; OSA = obstructive sleep apnea; OSAHS = obstructive sleep apnea/hypopnea syndrome; Pes = esophageal pressure; PTT = pulse transit time; REM = rapid eye movement; RIP = respiratory inductance plethysmography; ROC = receiver operating characteristic; Spo_2 = oxygen saturation measured by pulse oximetry; SDB = sleep-disordered breathing; UARS = upper airway resistance syndrome

Sleep-disordered breathing (SDB) encompasses upper airway resistance syndrome (UARS) and obstructive sleep apnea/hypopnea syndrome (OSAHS). In children, the latter is characterized by recurring episodic upper airway obstructions and may be associated with hypoxemia and hypercapnia.[1] Early detection of these respiratory events then becomes important in the minimization of possible long-term neurobehavioral deficits. The accurate classification of these events as either central or obstructive in nature is critical, as this can determine the appropriate treatment,[2] which can be quite different.[3] Central events (CEs) are defined as a reduction in airflow proportional to breathing efforts, while obstructive events (OEs) are a diminution of airflow in the presence of breathing efforts.[2] Both CEs and OEs can be further subcategorized as apnea and hypopnea. CEs can be common and physiologic in rapid eye movement (REM) sleep. However, the pathogenesis of OEs can be caused by anatomic abnormalities in the upper airway.[4] Increasing evidence suggests that hypopneas or partial pharyngeal airway collapse may be as clinically important as apneas. These nonapneic events are much harder to detect, and sensitive measurements of inspiratory effort changes are needed to classify

them.[5] Furthermore, it is often difficult to distinguish between obstructive and central hypopneas.[2] Currently, polysomnography is the standard procedure for the study of SDB, and it is usually conducted in sleep centers.

Esophageal pressure (Pes) monitoring is the current reference method of measuring increases in negative intrathoracic pressures and changes in inspiratory efforts.[1,2,6] The invasive nature of Pes monitoring is probably its greatest drawback. As Pes monitoring requires placement of a flexible tube or catheter into the esophagus via the nasal airway, this can cause discomforts to the patient.[3] The concern for Pes monitoring is the confounding effects of the Pes catheter on sleep architecture and the accuracy of the results obtained from polysomnography studies.[2,3,6] However, there are findings that report otherwise.[7] In addition, the presence of a Pes catheter may induce changes in the upper airway dynamics by impairing or modifying pharyngeal reflexes.[2,3] Furthermore, studies have suggested that the use of this technique is also not common in sleep laboratories[2,3] and may require greater patience in setting up.[3,8] Therefore, it is clear that there is a need for a more accommodating alternative technique for assessing changes in inspiratory efforts, including for children.

A noninvasive cardiorespiratory measure, termed *pulse transit time* (PTT), has shown its ability to estimate inspiratory effort changes in adults in response to involuntarily changes in the upper airway[5,9] and recently in children.[1,10] PTT is the time taken for the arterial pulse pressure wave to travel from the left ventricle to a peripheral site, usually on a finger or toe. The principle determinant of the pressure wave is a change in BP, which is in turn affected by the degree of stiffness of the arterial wall. PTT is inversely correlated with the BP and has been shown to predict changes in inspiratory efforts via the BP fluctuations induced by negative pleural pressure swings.[3,5]

In OSAHS events, breathing can be terminated by events of an obstructive nature that can lead to a marked transient increase in BP.[1] These abrupt changes are caused by the pleural pressure swings initiated in response to involuntarily obstructions in the upper airway.[2,3] During these events, both hypoxemia and hypercapnia acting through chemoreflexes escalated sympathetic activities with subsequent increases in the stiffness of arterial wall as well as heart rate (HR).[11,12] With increased inspiratory efforts to restore ventilation, a progressive increase in the inspiratory afterload to the left ventricle can occur, thus impairing its subsequent emptying during expiration.[13] This may lead to an increase in right ventricular preload to produce substantial inspiratory increase in right ventricular end-diastolic pressure. These inspiratory swings in intrathoracic pressure may also contribute to the decreased cardiac output during an OE.[14] Variations in the ventricular filling time and stroke volumes of the heart may be caused by the progressive increase in the size of the heart throughout the occurrence of the OE.[13] Garpestad et al[15] reported that this decrease in stroke volume occurs not only from the onset of the event, but also there is a further decrease immediately after the termination of the event. On the contrary, with the resumption of breathing, there is an increase in venous return and consequently increased cardiac output.[11] These revived inspiratory efforts may increase the vascular pressure around the vessels,[13] causing a severely vasoconstricted peripheral vasculature resulting in surges in BP.[12] As the BP increases, this affects the geometric and mechanical properties of the arterial wall, thus leading to an increase in its stiffness.[2,3] During such episodes, the pulse wave then propagates faster and thereby decreasing the duration of PTT value.[16]

Conversely, events of central respiratory nature are denoted by lesser variations in inspiratory efforts[2] with reduction or possible termination of the entire ventilation process. Katz et al[1] and Argod et al[2] have demonstrated the significant correlations of Pes with PTT, while Smith et al[3] and Pitson and Stradling[9] have established inverse relationships of PTT with BP. Based on these studies, changes in PTT can reflect changes in inspiratory efforts. The objectives of this study were as follows: (1) to determine the use of PTT to detect changes in inspiratory efforts; (2) to assess the capability of PTT to differentiate OEs from CEs, including hypopneas in children during sleep; and (3) to evaluate the potential of PTT to distinguish CEs from tidal breathing.

Materials and Methods

Polysomnography

Routine overnight polysomnography was performed in the sleep laboratory with monitoring that included EEG (electrodes C3-A2 and O2-A1); left and right electro-oculography (LE-A2

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Manuscript received January 8, 2005; revision accepted April 9, 2005.

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and RE-A1); oronasal airflow tracing (via pressure transducer); alternating current-coupled respiratory inductance plethysmography (RIP) of chest and abdominal movement (Respitrace; Ambulatory Monitoring; Ardsley, NY); infrared photoplethysmography; HR; and oxygen saturation using pulse oximetry (Spo_2) [Novametrix Medical Systems; Wallingford, CT]. The studies were continuously observed by experienced polysomnography personnel, and all readings were recorded by a commercial computerized polysomnography system (Uniquant System; La-Mont Medical; Madison, WI). An episode of obstructive sleep apnea (OSA) is defined as a complete cessation of airflow with the presence of chest and/or abdominal wall movement. An obstructive hypopnea has similar characteristics, except that airflow is reduced by at least 50% from its baseline.[1,2] In this study, both are classified as OEs. Central hypopnea events are defined as a reduction of $\geq$ 50% in airflow proportional to the decrease in chest wall movement.[2] For central sleep apnea, it is the complete cessation in both airflow and respiratory drive.[17] Similarly, they are regarded as CEs in the context of this study. Apneas or hypopneas are considered mixed when they contained both obstructive and central components.[18] For the purpose of this study, they are classified as OEs to simplify the comparison with the corresponding polysomnography scorings.

PTT Measurements

A standalone PTT system using a microcontroller to continuously acquire physiologic data from a single-lead ECG machine (S&W Medico; Teknik; Greve, Denmark) and photoplethysmography signals derived from the polysomnography pulse oximetry was developed. This system has an accuracy of 1 ms for all measurements. The ECG signal is sampled at a 1-ms interval, and a slope detection algorithm is used to determine the initial upstroke of the R wave. A differentiator in firmware then detects the peak, and a timer is initiated at this point. The photoplethysmography signal is also sampled at 1-ms sampling period, and a moving threshold detector is used to minimize its baseline instability. The corresponding 25% of peak-to-peak amplitude is derived as suggested by Katz et al[1] and Smith et al[3] in their similar studies to detect the arrival of the pulse wave at the periphery. At this point, the timer is terminated, and its count is stored as the PTT value. This system then outputs an analog voltage signal to the polysomnography system so as to allow comparison of PTT with all other measured parameters. The algorithm of this PTT detection system is given in Figure 1.

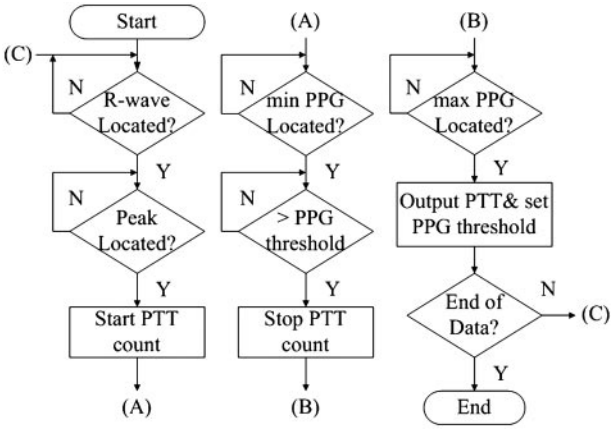

FIGURE 1. The algorithm used in the standalone system to estimate PTT calculation. (A), (B), and (C) indicate interlinks within the flowchart; N = no; Y = yes; PPT = photoplethysmography; min = minimum; max = maximum.

Subjects

This study included 33 children (26 boys and 7 girls; mean ± SD age, 6.7 ± 3.9 years; range, 1 to 14 years; height, 115.4 ± 29.4 cm; weight, 30.0 ± 22.1 kg). The children were scheduled to undergo routine overnight polysomnography for investigation of UARS or OSAHS. It is worth noting that the study population included five children with comorbidities: congenital craniofacial disorders (n = 2) and neurologic and/or neuromuscular disorders (n = 3). The remaining 28 children had a history or symptoms suggestive of UARS or OSAHS and were part of the study population. They were referred by their local general practitioners for nocturnal polysomnography studies for suspected UARS or OSAHS. The caregiver(s) and the children were given the study purposes and procedures verbally. Informed consent and institutional ethical approval were obtained before the commencement of this study.

Experimental Protocol

The two exclusion criteria in this study were age and coexisting cardiac diseases. For the former, the method which respiratory events were categorized with polysomnography scorings may be inadequate for newborns. Particularly, the contribution of the chest to breathing may be limited in newborns due to its shape, high compliance, and deformability. A study[19] has shown that by the age of 1 year, this contribution is similar to that of the adolescent. In order to minimize complications with results, infants (< 12 month old) were excluded in this study. For the latter criteria, cardiac diseases may affect the isometric contraction time of the left ventricle that forms a major part of the measured PTT. The effects on PTT measurement can be similar to when there were changes in inspiratory efforts in response to arousals. Furthermore, there was limited information about the behavior of PTT in these pathologic states.[3] Likewise, in order to keep the PTT validation process simple, children in both the categories were excluded in this study.

The accuracy of PTT measurements in this study was verified against the corresponding readings of both the RIP and oronasal airflow measure. These were the two measurements that were commonly used to quantify the occurrences of respiratory events in standard polysomnography scorings, and this procedure has been set by the American Thoracic Society.[20] The RIP method usually detected increased inspiratory efforts through the desynchronization of chest and abdominal movements,[21,22] while oronasal airflow measurement was determined by the temperature changes in relation to inspiration and expiration. In order to assess the accuracy of PTT measurement, only respiratory events that fulfilled a predefined selection criterion were considered in this study. Particularly, events that satisfied all the following conditions were used: (1) valid event identified in the corresponding polysomnography scorings by two blinded observers who were unaware of the purposes of this study, (2) duration > 10 PTT readings, and (3) no apparent motion artifacts. The latter criterion was achieved by monitoring the baseline stability of photoplethysmography signals throughout the duration of each recording of the present study.

Data Analysis

For each valid respiratory event, the PTT data were extracted for the duration of the event and for an equal duration of tidal breathing directly prior to the event. This gave an equal amount

of data for both the tidal breathing events and the respiratory events. Next, the mean PTT of all the tidal breathing events for each subject was subtracted from all of the event data for that subject. This effectively removes intersubject variation in PTT values and gives a mean PTT of zero for all tidal breathing events.

PTT is a semiquantitative inspiratory effort measure, and therefore individual PTT values cannot be quantified. PTT examination needs to be determined over several breaths by observing the relative fluctuations from its baseline history value.[3,9] In this study, the classifications of respiratory events are by using the mean and variance of 10 consecutive PTT samples associated with each event. In practice, this process can be viewed as a 10-sample "sliding window" on the data, with respiratory events being distinguished from normal tidal breathing based on deviations in the mean and/or variance in this window. In this way, events with duration longer than the length of analysis window (in this case, 10 samples) contributed multiple estimates for the mean and variance of the PTT during that event.

It is recommended to use the sensitivity and specificity when comparing two methods of clinical measurement.[23] Further, these measures can be made independent of decision threshold by plotting a receiver operating characteristic (ROC) curve. The area under the ROC curve (AUC) is a useful overall measure of classifier performance, as it represents the probability that a randomly chosen positive example is correctly ranked with a larger classification score than a randomly selected negative example, that is $P(xp > xn)$.[24] Moreover, this probability is the same quantity estimated by the nonparametric Wilcoxon statistic (also often referred to as the Mann-Whitney *U* test). Therefore, AUC can be used to test the (null) hypothesis that two samples come from identical populations without having to make any assumptions about the shape of those distributions. It is worth noting that for this study, a negative event relates to a period of tidal breathing while a positive event relates to respiratory event as either OE or CE.

The 334 respiratory events were separated into 112 OEs and 222 CEs. Next, ROC curves were generated for the mean and variance of the PTT for both the 112 OEs and 222 CEs against all 334 tidal breathing events. Finally, the AUC and its associated SE were calculated for each of the ROC curves. Finally, a large-sample *z* test was performed on the sampling distribution of AUC specifically to test whether the measured AUC is a by-chance result from a random-choice classifier (AUC of 0.5; that is, a ROC curve in which sensitivity equals specificity for all decision thresholds). The results of all these calculations are shown in Table 1.

Results

Polysomnography Scorings

From the study population, the recorded mean total sleep time was 6.58 ± 1.49 h, with a mean sleep efficiency of 86.4 ± 7.2%. The distribution of sleep was as follows: REM, 1.77 ± 0.81 h; non-REM, 4.81 ± 1.26 h. Dividing the number of apneas and hypopneas (combing both OE and CE) by the hours of sleep, the mean apnea-hypopnea index (AHI) was found to be 4.58 ± 4.04 events/h (range, 0.3 to 16.1 events/h). From polysomnography results, 23 of the subjects were considered normal with an AHI of ≤ 5 events/h, 4 other children had an AHI from 5 to 10 events/h, and the remaining 6 children had significant symptoms with an AHI of ≥ 10 events/h. The total number of respiratory events (for both CE and OE) scored in the polysomnography recordings were 993.

Table 1—*AUC Results of Respiratory Events*

Events	No.	AUC	SE	p Value
OEs	112			
Mean PTT		0.923	0.011	< 0.005
Variance PTT		0.823	0.016	< 0.005
CEs	222			
Mean PTT		0.515	0.017	> 0.05
Variance PTT		0.940	0.008	< 0.005

PTT Measurement

From the 993 identified polysomnography respiratory events, only 334 were considered as valid events to compare PTT measurement with the polysomnography scorings. The exclusion of the remaining events was due to one of the following reasons: (1) motion artifacts including events ending with a sigh; (2) events less than two breaths apart (the proximity of these events may affect the accuracy of PTT computation used in this study); or (3) scorings discrepancies between the two blinded observers. Moreover, it is common for arousals to follow the termination of an OE, and this can cause artifacts on noninvasive measurements.[25] Hence, the number of valid respiratory events is generally restricted by this factor.

In Figure 2, it can be observed that the consequence of an OE is a significant decrease in PTT. For CE, variations in PTT become less prominent with a general reduction in breathing efforts, as shown in Figure 3. The rib cage and abdominal component of the RIP in this example shows a cessation of breathing efforts as well as the termination of airflow. Without the ongoing breathing efforts, the normative PTT variations diminished. Furthermore, it can be seen that the Spo_2 value of the child deteriorated with the progression of the prolonged CE. These illustrative examples support the ROC curves, generated using the mean and variance of the PTT in the 10-sample sliding window, for OE and CE, shown in Figures 4 and 5 respectively. In particular, Figure 4 shows that both the mean and variance of PTT have discriminative power for OE, with mean PTT being superior to variance PTT ($p < 0.005$). Figure 5 shows that only the variance of PTT has discriminative power for CE, the ROC curve for mean PTT being indicative of a random-choice classifier. Table 1 shows that for OE, both of these AUC results are very highly significant ($p < 0.005$), while for CE only the variance of PTT shows an AUC significantly ($p < 0.005$) different to the by-chance AUC of 0.50.

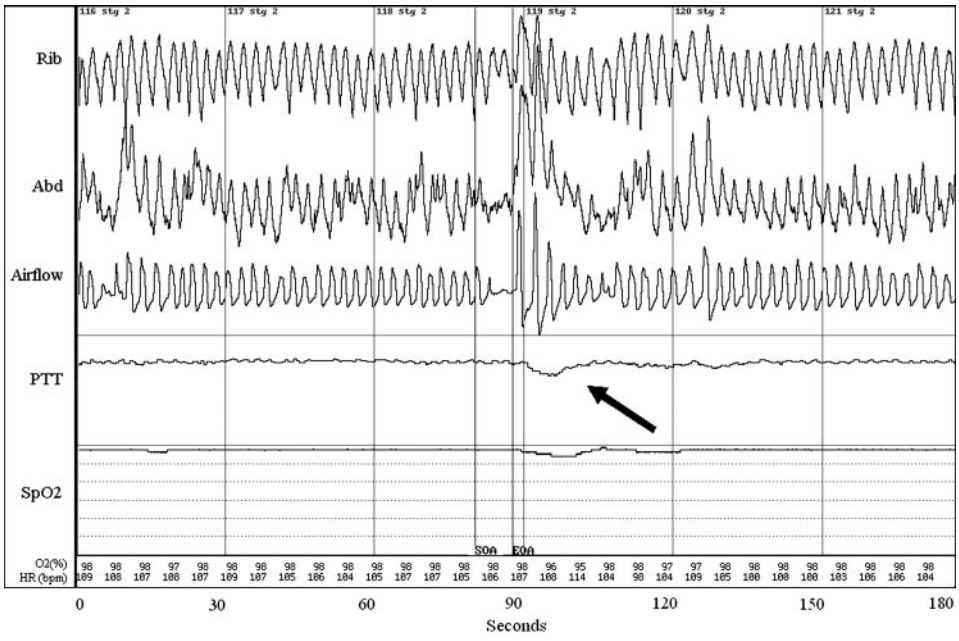

FIGURE 2. In this 180-s polysomnography recording, a 9-year-old girl with an obstructive apneic event that led to significant PTT variations when compared to prior tidal breathing PTT baseline (indicated by arrow) can be observed. Rib = rib cage; Abd = abdomen; bpm = beats per minute; SOA = start of obstructive apnea; EOA = end of obstructive apnea.

DISCUSSION

From this study, PTT measurement can be used as a simple supportive technique in assessing inspiratory effort changes in children during sleep because it has shown its ability to differentiate respiratory events accordingly. PTT fluctuations have shown their correlation with abnormal changes in inspiratory efforts

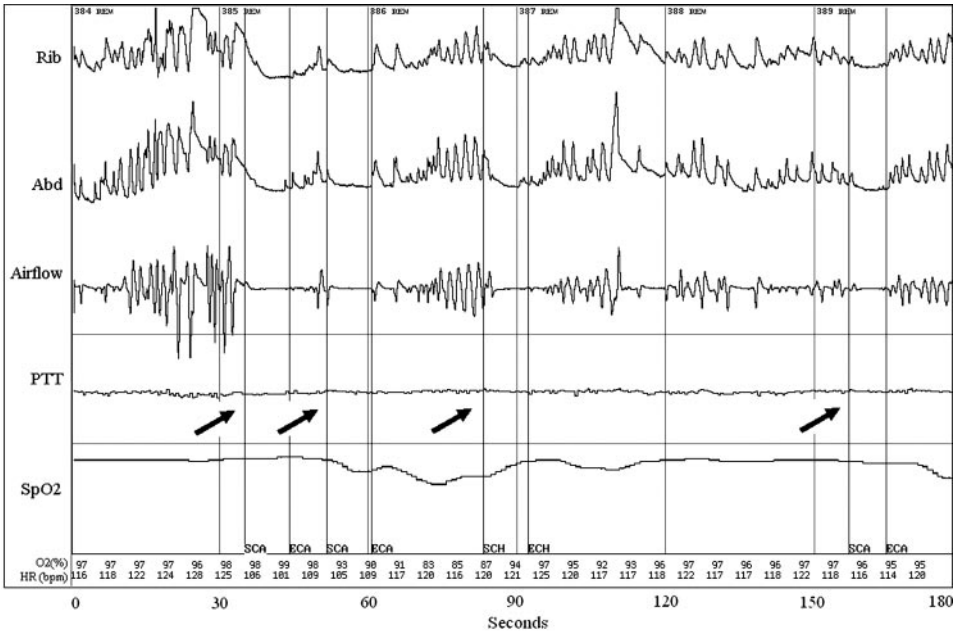

FIGURE 3. A 3-year-old boy with mixture of central apneic and hypopneic events with relatively minimal PTT variations (indicated by arrows) that can be seen in this 180-s polysomnography recording. SCA = start of central apnea; ECA = end of central apnea; SCH = start of central hypopnea; ECH = end of central hypopnea. See Figure 2 legend for expansion of abbreviations.

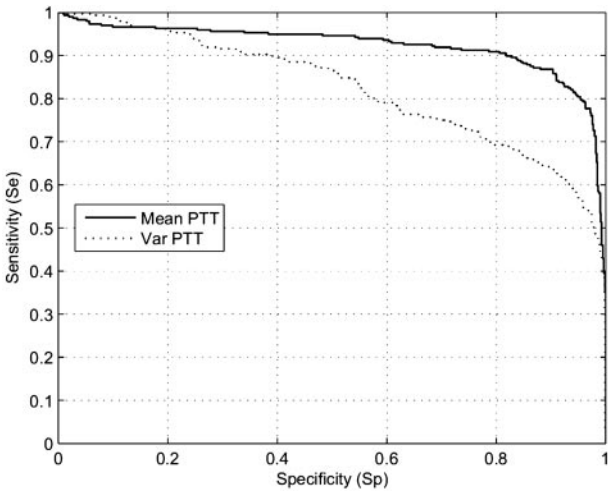

FIGURE 4. ROC curves generated using the mean and variance (Var) of PTT in a 10-sample sliding window for all 112 OEs.

detected by both RIP and oronasal airflow. During normal sleep, it can be observed that there were marginal PTT fluctuations, and this can be dependent on their BP fluctuations during tidal breathing. Trinder et al[26] reported that these breath-based BP changes may be due to the mechanical effects of changing intrathoracic pressure and lung volume, thereby affecting the cardiac preload and afterload. Furthermore, they suggested that oscillations of venous return and cardiac output caused by periodic rises and falls in ventilation and pleural pressure during tidal breathing may also lead to BP fluctuations.

From the results obtained, the marginal PTT fluctuations observed during tidal breathing signifies the ability of PTT to monitor even small changes in inspiratory efforts. PTT not only has the capability to monitor changes in inspiratory efforts, but also shows the potential to differentiate a CE from an OE, an OE from tidal breathing, and potentially a CE from tidal breathing. For example, a decrease in mean PTT together with an increase in the variance of PTT is indicative of an OE, while a reduction in the variance of PTT with no change in mean PTT is indicative of a CE.

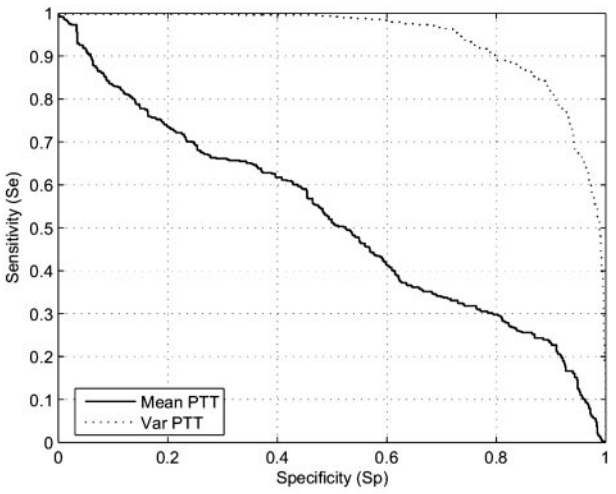

FIGURE 5. ROC curves generated using the mean and variance of PTT in a 10-sample sliding window for all 222 CEs. See Figure 4 legend for expansion of abbreviation.

Classification of respiratory events is clinically important for future pathologic studies and their distinct treatment.[3] It has been established by other studies[1–3] that there is an inverse correlation between changes in BP and PTT values. Based on these studies, a parallel relationship of PTT variations with changes in inspiratory efforts can be drawn. The results herein show that PTT can discriminate respiratory events accordingly and may therefore be useful clinical tool for children in the respiratory sleep studies. However, in order for PTT to assess changes in inspiratory effort, comparison to its baseline history values is required. It can be seen from Figures 4 and 5 that, at a sensitivity of 0.90, OEs can be detected with a specificity of 0.82 and CEs can be detected with a specificity of 0.80.

Despite its intrusive nature, Pes monitoring is still the reference method for measuring increases in negative intrathoracic pressures and inspiratory efforts.[6,7] However, this measure was not included in the present study. As Pes measurement was not part of the standard protocol used in the polysomnography study,[20] ethics approval for its use was unobtainable. The concern for Pes monitoring is that there is very limited published information about the effect of the Pes catheter on the sleep architecture.[6] Previous studies[2,3,6] also affirmed that Pes monitoring can affect the accuracy of results obtained from polysomnography studies. In addition to the lack of acceptance by patients, the presence of a Pes catheter may also induce changes in the upper airway dynamics by impairing or modifying pharyngeal reflexes.[3,8] Due to the complexity of the Pes method, it requires greater technician management, thus adding to the cost of the sleep study.[3] Moreover, evidences in the present literature have suggested that there is a significant correlation between Pes and PTT.[1–3,5] In view of these reasons, Pes monitoring was therefore not included in this study.

It is recognized that PTT has some limitations. Firstly, as there can be differences in BP and vascular compliance in children,[27] their corresponding transit time can vary.[28,29] Hence, the PTT value may only be useful when it is observed over a period of time on an individual basis. Secondly, PTT may not reflect true variations of inspiratory efforts in children with coexisting cardiac diseases. These diseases can affect the isometric contraction time in response to changes in inspiratory efforts causing false changes in PTT measurement.[3] Thirdly and most importantly, the major

limitation of PTT is the effects of motion artifacts. This can be caused mainly by the movements at the periphery interfering with the integrity of the photoplethysmography signals. These may cause a shift in PTT baseline and may be incorrectly regarded as an occurrence of abnormal variations in breathing efforts. This can be reflected by the high number of respiratory events excluded in the present study due to artifacts, including those following a sigh. However, artifacts of this nature can be detected or minimized with appropriate technological applications.[30,31] Lastly, it is recognized that detection of a CE from tidal breathing may not be as straightforward as the distinction from an OE. However, an analysis of the variance on the obtained readings can provide quantitative information between the two, as demonstrated in this study.

Conclusions

The invasive nature of Pes monitoring warrants the need for a noninvasive measure for inspiratory efforts in the monitoring of SDB in children. In this study, PTT shows not only the ability to detect changes in inspiratory efforts, but it is also able to differentiate respiratory events as central or obstructive in nature in the absence of motion artifacts. Furthermore, PTT has showed its capability to monitor marginal BP fluctuations during tidal breathing. It is relatively easy to implement, it is not intrusive, and it has the potential to be included in a simplified screening package for domiciliary SDB studies. Further experiences with PTT and development of the technology to reduce motion artifact are required to determine the suitability and utility in childhood clinical practice.

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Use of Pulse Transit Time To Distinguish Respiratory Events From Tidal Breathing in Sleeping Children*

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Chest 2005;128; 3013-3019
DOI 10.1378/chest.128.4.3013