

## Adenoid hypertrophy and its relation with right ventricular function

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**Abstract:** Background: Many studies had previously shown a reduction in pulmonary artery pressure (PAP) post adenoidectomy in children with adenoid hypertrophy (AH) causing upper airway obstruction. However, it is not obvious whether this could significantly be reflected on the right ventricular performance. Aim of the study: to determine if there were any detectable changes in the RV functions post adenoidectomy in children with adenoid hypertrophy. Methods: Thirty children with AH (female/male: 9/21) aged between 2.5 and 12 years (median: five years) were included in this study. Adenoidectomy was performed using adenoid curette and cold instruments. All children had echocardiography one day pre and one month post adenoidectomy. Velocity time integral of tricuspid valve flow (VTItv) and pulmonary valve flow (VTIpa); E/A ratio of tricuspid valve flow; RV end diastolic diameter (RVEDd) and left ventricle fraction shortening (FS) were measured. Heart rate (HR) was also recorded. Results: Preoperatively VTItv, VTIpa, E/A ratio, RVEDd, FS, and HR were 17.4± 3.0 cm, 20.2± 3.1 cm, 1.20± 0.30, 11.5± 2.2 mm, 34.1± 4.1%, and 110± 17, respectively. Postoperatively VTItv, VTIpa, E/A ratio, RVEDd, FS, and HR were 21.7± 2.5 cm, 24.4± 4.3 cm, 1.44± 0.31, 9.3± 2.5 mm, 32.9± 3.1%, and 102± 28, respectively. There were significant differences between preoperative and postoperative VTItv ( $p = 0.03$ ), VTIpa ( $p = 0.01$ ), E/A ratios ( $p = 0.04$ ), and RVEDd ( $p = 0.01$ ). FS and HR were not significantly changed. **Conclusions:** This study concluded that in children suffering from AH, relieving upper airway obstruction by adenoidectomy may result in improvement of RV filling and RV function, associated with the reduction in mPAP.

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### 1.Introduction

Adenoid hypertrophy (AH) is considered one of the most common causes of upper airway obstruction in children (1). Marked upper airway obstruction may result in obstructive sleep apnea and chronic alveolar hypoventilation, which may lead to right sided cardiac dysfunction induced by hypoxemic pulmonary vasoconstriction and the resulting increase in pulmonary vascular resistance (PVR) and pulmonary artery pressure (PAP). Unlike left ventricle, right ventricle (RV) has limited adaptation capacity to pressure overload (2). If RV adaptive mechanisms are exhausted to the limit they cannot compensate for the hemodynamic burden, right ventricle output (RVO) decrease and RV failure (cor pulmonale) may ultimately occur (3).

Although RV failure is considered an uncommon complication of AH, children with borderline cardiopulmonary function may experience severe RV failure if AH happened. Concerns were raised that RV failure became more common as more children survived prematurity. Many of these children continue into childhood with residual subclinical lung disease and/or chronic pulmonary hypertension (PH) caused by bronchopulmonary dysplasia in the neonatal period (1).

Cardiopulmonary changes associated with AH had been described previously in many studies (4). RV dilatation and reduced RV ejection fraction were seen even before clinical signs of cardiac involvement became evident. Many of these reports as well as other more recent systematic studies demonstrated a reduction in PAP post adenoidectomy. However, it is not evident whether this decline in PAP could be reflected on RVO (5).

Aim of work: Was to determine if there were any detectable changes in RV function parameters, especially RVO, post adenoidectomy in children with AH causing marked upper airway obstruction. We assumed that adenoidectomy in such children would result in improving RVO regardless of its effect on PAP.

### 2. Subjects and methods

The study was conducted on children who were scheduled for adenoidectomy in the Department of Otorhinlaryngology, faculty of medicine, Ain Shams University Hospitals, in the period from February 2012 until January 2013. Only children with severe upper airway obstruction caused by AH were chosen. All children scheduled for adenoidectomy in this period (total of 239) were subjected to a detailed clinical history and careful otorhinolaryngological

examination.

To identify children with severe obstructive disease we applied a standardized questionnaire as proposed by Brouillette et al (6). Only children who scored higher than 3.5 in the standardized questionnaire were enrolled. Scoring higher than 3.5 indicates severe obstruction and is highly predictive of obstructive sleep apnea (6). Exclusion criteria were: (1) other causes of chronic nasal obstruction; (2) scoring 3.5 or less in the questionnaire; (3) presence of cardiopulmonary failure; (4) other systematic illness. Careful anterior rhinoscopy, lateral X-ray nasopharynx, and in some cases endoscopic examination were done to confirm that adenoid is the cause of obstruction. All children were studied after written informed consent about the study and its consequences which was approved by the ethical committee.

The study design was a prospective crossover observational study in which each child served as his/her own control. RV function of the selected children was measured using echocardiography one day pre and in the follow up visit at least one month post adenoidectomy. The scans were recorded. To study the effect of adenoidectomy on right ventricle hemodynamic pre and post operative measurements were subjected to statistical analysis.

Preoperative routine anesthetic evaluation included blood tests and chest X-ray. All subjects underwent adenoidectomy using adenoid curette and cold instruments. All children were operated as inpatient and under general anesthesia. There were no immediate post operative complications that are related to either anesthesia or surgery. The average period of hospital stay was only one day.

All children underwent echocardiographic examination in the pediatric department, faculty of medicine, Ain Shams University, including 2-dimensional, M mode, Doppler, and color Doppler, using device model GE medical system VIVID7 dimension N- 3190 Horten NORWAY. Structural normality of the heart was established on the initial scan. For the purpose of the study, each examination was performed in the following order: Doppler examination of RV inflow across tricuspid valve, Doppler examination of RV outflow across pulmonary valve, and finally M-mode examination of right and left ventricle dimensions.

RV inflow was examined from standard apical four chambers view. Flow across the tricuspid valve was determined by color Doppler and then pulsed Doppler sample volume was placed at the tip of the tricuspid valve in diastole to obtain the tricuspid Doppler flow velocity time signal. RV outflow was examined from standard para-sternal short axis view. Pulmonary flow was determined by color Doppler

and then the pulsed Doppler sample volume was placed in the center of the main pulmonary artery immediately distal to the pulmonary valve to obtain the pulmonary artery Doppler flow velocity time signal. No angle correction was needed for Doppler examination since the angle of insonation was near to zero for both tricuspid and pulmonary flows. The waveform was considered optimal when the highest velocity with a clear distinct envelope was obtained for a minimum of 10–20 consecutive cardiac cycles. M-mode examination was done from the standard parasternal long axis view according to the guidelines of the American Society of Echocardiography for M-mode echocardiography. The M-mode beam was placed perpendicular to the interventricular septum at a level transecting the tips of the mitral leaflets, and measurements were taken by the leading edge methodology.

For the purpose of volumetric assessment of RV inflow and outflow, velocity time integral of tricuspid (VTItv; cm) and pulmonary (VTIpa; cm) Doppler flow velocity waveform were calculated respectively. Since stroke volume (blood flowing in a single heart beat) is equal to the product of VTI and the cross sectional area of the orifice it crosses, changes in VTI will directly reflect changes in stroke volume, as long as the changes in cross sectional area of the orifice of interest is not expected to change. Tricuspid and pulmonary valves in study children were healthy and it was unlikely that their cross sectional areas would change between the two examinations. Consequently, VTItv and VTIpa could be used as relative measures of RV inflow and outflow, respectively.

RV diastolic function was assessed using RV early to atrial (E/A) filling velocity ratio of tricuspid valve flow. The lower the E/A ratio the more the impairment of the RV diastolic function. To assess for RV dilatation, RV end diastolic diameter (RVEDd) from M-mode examination, was measured. Left ventricular global systolic function was assessed using left ventricle fraction shortening (FS) from M-mode examination. Heart rate was also recorded. From the pulmonary artery Doppler flow tracing time to peak velocity of pulmonary flow (TPV) was measured and mean pulmonary artery pressure (mPAP) was calculated using the formula: mPAP [mmHg] = 73 (0.42TPV). For each parameter, the average of five consecutive cardiac cycles was taken (7).

Data were expressed as mean and standard deviation, or median and range if not normally distributed. Wilk test was applied to test for normal distribution of the data. Paired sample t test was used to compare hemodynamic parameters pre and post adenoidectomy. To test for the relationship between PAP and RV outflow, the relationship between TPV

and VTIp<sub>a</sub> was tested by linear regression analysis taking TPV as the independent variable. This was repeated for pre and post adenoidectomy. Data were analyzed with PC-based statistics package (SPSS for Windows, SPSS, Chicago, Ill). Statistical significance was taken as a value of two tailed  $p < 0.05$ .

### 3. Results

Thirty children had fulfilled the clinical criteria for the diagnosis of adenoid hypertrophy; they were 9 girls (30%), and 21 boys (70%) had completed the follow up echocardiographic examination. Clinical criteria, demographic data and timing of examination of children who completed the follow-up examination after adenoidectomy are shown in table 1.

The results of our study are summarized in table 2. Individual responses of study group as regard parameters of RV function are shown in figure 1. Comparing RV function parameters pre and post adenoidectomy showed significant increase in both VTItv and VTIp<sub>a</sub>, reflecting improvement of RV in

and outflows. VTItv and VTIp<sub>a</sub> were elevated post adenoidectomy in 18 and 23 children, respectively. RVEDd diminished significantly post adenoidectomy. E/A ratio revealed a significant increase post adenoidectomy. There were no significant differences noted in fraction shortening or heart rate.

Post adenoidectomy, mean TPV was significantly longer indicating a significant decrease in mPAP. Eighteen children in our study showed an increase in TPV (and hence a reduction in mPAP), two showed no change and in 10 children mPAP increased. Improvement in VTIp<sub>a</sub> was not associated with a decrease in mPAP in all cases. Out of 23 children who showed an improvement in their VTIp<sub>a</sub>, 13 children showed a decrease in mPAP as shown in figure 2.

Weak positive correlation between TPV and VTIp<sub>a</sub> pre and post adenoidectomy was noted. Both cases did not reach statistical significance. However, an upward shift of the regression line post adenoidectomy indicating higher VTIp<sub>a</sub> for the same PAP was detected as shown in figure 3.

Table 1: Clinical criteria, demographic data and timings of examination (n = 30).

	Median	Range
Age (years)	5	2.5–12
Period of obstructive symptoms (years)	2.2	1.2–9
Timing of pre adenoidectomy data (days)	1	1–7
Timing of post adenoidectomy data (days) Males (21 children= 70 %) Females (9 children = 30%)	36	30–52

Table 2: Doppler echocardiographic parameters pre and post adenoidectomy (n=30).

Doppler echo parameters (mean SD)	Preoperative		Postoperative		p value
Velocity time integral of tricuspid flow (VTItv, cm)	17.4	± 3.0	21.7	± 2.5	0.03
Velocity time integral of pulmonary flow (VTIp <sub>a</sub> , cm)	20.2	± 3.1	24.4	± 4.3	0.01
Right ventricle early to atrial filling velocity ratio (E/A)	1.20	± 0.30	1.44	± 0.31	0.04
Right ventricle end-diastolic diameter (RVEDd, mm)	11.5	± 2.2	9.3	± 2.5	0.01
Fraction shortening (FS, %)	34.1	± 4.1	32.9	± 3.4	0.25
Heart rate (bpm)	110	± 17	102	± 28	0.16
Time to peak velocity of pulmonary artery flow (TPV, ms)	100	± 21	110	± 16	0.007
Calculated mean pulmonary artery pressure (mPAP, mmHg)	31.6	± 8.6	27.2	± 6.8	0.007

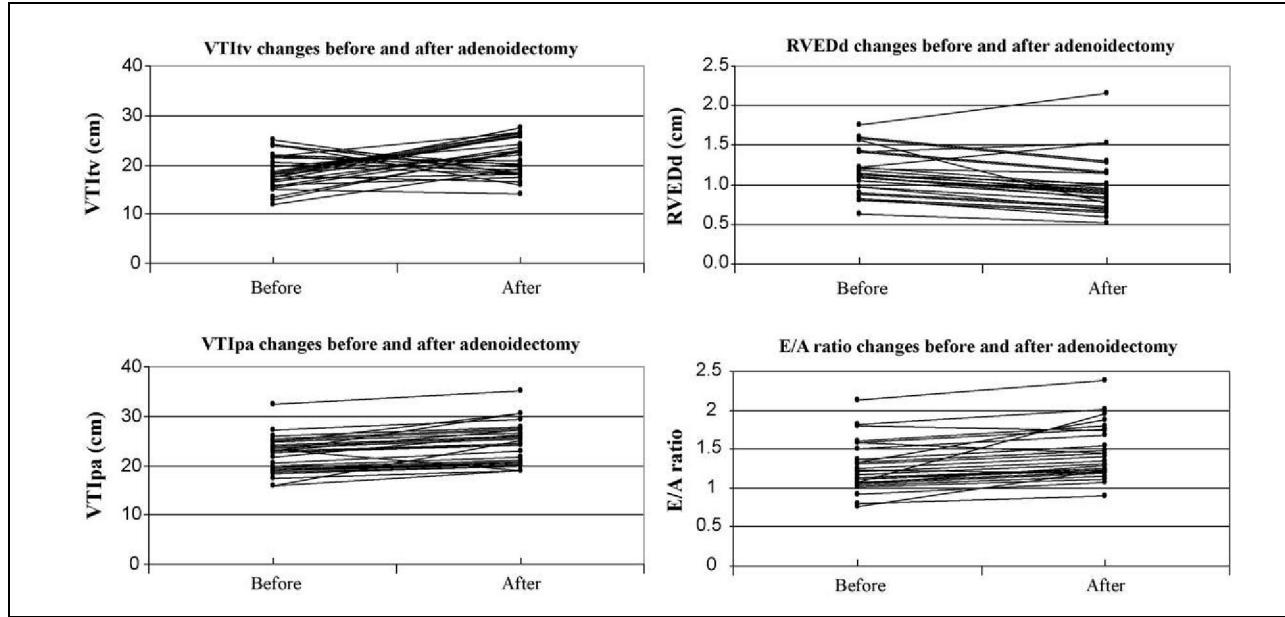


Figure 1: Scatter plots showing effects on parameters of right ventricle function from immediately pre to one month post adenoideotomy in study group (VTI tv, velocity time integral of tricuspid flow; VTI pa, velocity time integral of pulmonary flow; RVEDd, right ventricle end diastolic diameter; E/A, early to atrial filling velocity ratio of tricuspid valve flow).

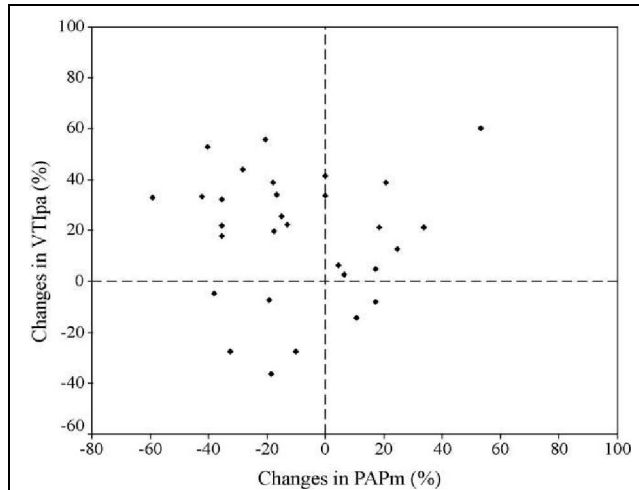


Figure 2: The relationship between changes in VTI pa and that of mPAP from pre to post adenoideotomy expressed in percentage (VTI pa, velocity time integral of pulmonary flow; mPAP, calculated mean pulmonary artery pressure).

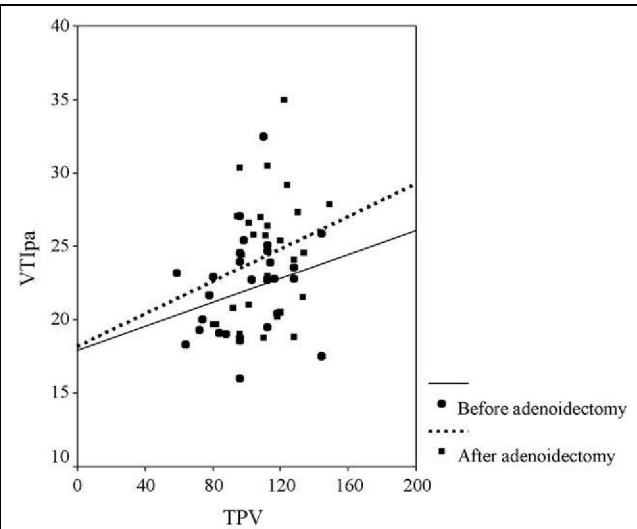


Figure 3: The relationship between VTI pa and TPV pre adenoideotomy (solid line,  $r = 0.24$ ,  $p = 0.19$ ) and post adenoideotomy (dotted line,  $r = 0.22$ ,  $p = 0.0.24$ ) plotted together (VTI pa, velocity time integral of pulmonary flow; TPV, time to peak velocity of pulmonary flow Doppler tracing).

#### 4. Discussion

Obstructive hypoventilation that leads to alveolar hypoxia is the main stimulus leading to pulmonary vasoconstriction; this is in accordance to the most accepted theory of hemodynamic changes and classification of PH revised by the WHO (8). This happens through the effect of mediators or through the direct presser effect of hypoxemia. This will elevate the pulmonary vascular resistance

leading to increased RV after load and consequently decreases the RVO. So, reduction in RVO occurs due to a direct effect of elevated RV after load and indirect effect through the right ventricular adaptation to the stress of increased after load. This adaptation occurs by hypertrophy and dilatation of RV. While RV dilatation diminishes its preload reserve with resultant tricuspid regurgitation, RV hypertrophy may predispose to RV hypo-perfusion and ischemia.

Both effects will result in more reduction in RV (3). RVO should be an important parameter to measure during searching for the possible effects of chronic hypoxia on the cardiopulmonary functions.

In our study we reported an improvement in RVO that happened after relieving the upper airway obstruction by adenoidectomy. Previous studies had evaluated the effects of upper airway obstruction on cardiopulmonary functions, assessment of RV functions was done through parameters that measure; RV diastolic filling, as E/A ratio; RV dimensions, as RVEDd ; RV downstream impedance, as PAP and/or RV global systolic function, as RV ejection fraction. Those parameters were affected by factors other than RVO and none of them is considered a direct measure of RVO. In spite of that they were used to assess the improvement in RVO after the relief of upper airway obstruction (9).

Despite that PAP as well as RVO showed a significant improvement after adenoidectomy, the correlation between TPV and VTIp<sub>a</sub> was insignificant pre and post adenoidectomy. Beside this, not all children with improved RVO, showed a reduction in PAP and vice versa. It is known that PAP is the product of RVO multiplied by PVR. So, the change in PAP could reflect the changes that occurred either in RVO, PVR or a combination of both. Accordingly, the increase in RVO associated with reduction in PVR, may keep PAP constant. On the other side, PAP may remain constant in spite of the reduction in RVO in the situations where PVR increases (5).

In this study, regression line of the relation between VTIp<sub>a</sub> and TPV had shifted upward post adenoidectomy that indicates a higher RVO for the same PAP. This probably happened due to a reduction in PVR post adenoidectomy. So, measuring the changes in PAP alone could be misleading if the changes in RVO is to be investigated. This may incorrectly underestimate or even miss the diagnosis of right ventricular dysfunction if we depend only on it as a parameter to detect the effects of upper airway obstruction on cardiopulmonary function. This finding was similar to the finding of weak correlation between left ventricular output and systemic blood pressure. Also, it is similar to the finding that critically ill infants systemic BP is a poor indicator of systemic blood flow (10).

The relief of upper airway obstruction post adenoidectomy will improve the state of chronic alveolar hypoxia, and thus, reversing hypoxic pulmonary vasospasm and decreasing PVR. Our results showed a decline in PAP after relieving severe upper airway obstruction. However, the degree of this decline was minimal; reaching about 4 mmHg in our study and about 6 mmHg in previous ones (9). This

minimal difference does not explain the marked improvement in the clinical condition noticed after the relief of upper airway obstruction (11).

The right ventricle is a thin walled chamber that acts as a volume pump rather than a pressure pump. RV adapts more efficiently to the preload changes than to the after load changes. When the after load increases, the RV will increase its systolic pressure to keep the gradient. At a certain point, further increase in PAP will bring significant RV dilation, an increase in RV end-diastolic pressure, and circulatory collapse. In accordance to previous studies (5, 9), we recorded reductions in RVEDd and E/A ratio post adenoidectomy that indicated resolution of RV dilatation and improvement of RV diastolic filling. Both led to improved RV reserve post adenoidectomy.

The pulmonary vascular bed is a dynamic system. It is subjected to many mechanical, neural and biochemical influences as changes in left atrial pressure, oxygen tension and catecholamine levels (12). We reported that, improvement of RVO due to possible changes in those factors caused by adenoidectomy could not be excluded. Another factor implicated in inducing RV dysfunction during pulmonary hypertension states is the decrease in left ventricular output caused by decreased left ventricular compliance due to RV dilatation (ventricular interdependence phenomena) (13) and as a consequence of decreased RVO. Consequently, decreased left ventricular output will reduce coronary blood flow and induces more RV ischemia, and hence, RV dysfunction. In the current study, left ventricular fraction shortening (FS), a widely used echocardiographic index of global left ventricular function, showed no change. Although many studies showed that FS could rise after removal of obstruction (based on elimination of the ventricular interdependency effect) (14), others did not (5, 9). Although fraction shortening is considered one of the most important measurements of LV function, it has got some inherent limitations. It is obtained from a single dimensional view, and hence, carries the assumption that the left ventricle is regular as an ellipse and contracts homogeneously. So, errors could happen due to distortion of the left ventricle and abnormal septal motion associated with RV dilation (15).

To avoid errors in calculating the cross sectional area of pulmonary and tricuspid valves, VTIp<sub>a</sub> and VTIt<sub>v</sub> were used in our study as indicators of RV out and inflows, respectively. We followed the recommendations of many studies on non invasive measuring of blood flow (7). In accordance to these studies the commonest error in Doppler estimation of blood flow results from vessel diameter

measurement, and to avoid large interobserver variability during serial measurements of blood flow, vessel diameter could be assumed to remain unchanged during the period of studying. So, VTI may be used as a relative measure of blood flow.

#### Conclusion:

We showed that the relief of upper airway obstruction by adenoidectomy in children with adenoid hypertrophy improved RVO. It is concluded that, RVO should be used instead of PAP in the assessment of the effect of upper airway obstruction on cardiopulmonary function. RVO measurement through Doppler echocardiography may also be useful in the assessment of the correlation between hypoxia and RV performance in patients with obstructive sleep apnea.

#### References:

- (1) Xu Z, Cheuk D K and Lee S L, Clinical evaluation in predicting childhood obstructive sleep apnea, *Chest* 130 (2006) 1765–1771.
- (2) Park M K and Troxler R G, *Pulmonary Hypertension in Pediatric Cardiology for Practitioners*, 4th ed., Mosby Inc., St Louis, MO, 2002, pp. 417–426.
- (3) Chemla D, Castelain V, Herve' P, Lecarpentier Y and Brimiouille S, Hemodynamic evaluation of pulmonary hypertension, *Eur. Respir. J.* 20 (2002) 1314–1331.
- (4) Ramakrishna S, Ingle V S, Patel S, Bhat P, Dada J E, Shah F A, et al., Reversible cardio-pulmonary changes due to adenotonsillar hypertrophy, *Int. J. Pediatr. Otorhinolaryngol.* 55 (2000) 203–206.
- (5) Yilmaz M D, Onrat E, Altuntas A, Kaya D, Kahveci O K, Ozel O, et al., The effects of tonsillectomy and adenoidectomy on pulmonary arterial pressure in children, *Am. J. Otolaryngol.* 26 (2005) 18–21.
- (6) Brouillette R, Hanson D, David R, Klemka L, Szatkowski A, Fernbach S, et al., A diagnostic approach to suspected obstructive sleep apnea in children, *J. Pediatr.* 105 (1984) 10–14.
- (7) Gullberg N, Winberg P and Sellden H, Changes in stroke volume cause change in cardiac output in neonates and infants when mean airway pressure is altered, *Acta Anaesth. Scand.* 43 (1999) 999–1004.
- (8) Rich S, Executive Summary From the World Symposium on Primary Pulmonary Hypertension, Evian, France, September 6–10, 1998 co-sponsored by the World Health Organization. Date updated: June 2002 <http://www.who.int/ncd/cvd/pph.html>.
- (9) Tezer M S, Karanfil A and Aktas D, Association between adenoidal-nasopharyngeal ratio and right ventricular diastolic functions in children with adenoid hyper-trophy causing upper airway obstruction, *Int. J. Pediatr. Otorhinolaryngol.* 69 (2005) 1169–1173.
- (10) Pladys P, Wodey E, Beuchee A, Branger B and Betremieux P, Left ventricular output and mean arterial blood pressure in preterm infants during the 1st day of life, *Eur. J. Pediatr.* 158 (1999) 817–824.
- (11) Williams III E F, Woo P, Miller R and Kellman R M, The effects of adenotonsillectomy on growth in young children, *Otolaryngol. Head Neck Surg.* 104 (1991) 509–516.
- (12) Dabestani A, Mahan G, Gardin J M, Takenaka K, Burn C, Allfie A, et al., Evaluation of pulmonary artery pressure and resistance by pulsed Doppler echocardiography, *Am. J. Cardiol.* 59 (1987) 662–668.
- (13) Robotham J L, Peters J, Takata M and Wetzel R C, Cardiorespiratory Interactions, in: M.C. Rogers (Ed.), *Textbook of Pediatric Intensive Care*, 3rd ed., Williams and Wilkins, Philadelphia, PA, 1996, pp. 369–396.
- (14) Tal A, Leiberman A, Margulis G and Sofer S, Ventricular dysfunction in children with obstructive sleep apnea: radionuclide assessment, *Pediatr. Pulmonol.* 4 (1988) 139–143.
- (15) Colan S D, Borrow K M and Neumann A, Left ventricular end-systolic wall stress-velocity of fiber shortening relation: a load-independent index of myocardial contractility, *JACC* 4 (1984) 715–724.

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