Brazilian Journal of Otorhinolaryngology 2021;**87(5)**:497–504

Brazilian Journal of
OTORHINOLARYNGOLOGY

www.bjorl.org

ORIGINAL ARTICLE

The systolic pulmonary artery pressure and the E/e' ratio decrease after septoplasty in patients with grade 2 and 3 pure nasal septal deviation

Deniz Avcı [a,*], **Ayşegül Hartoka Sevinç** [b], **Sabri Güler** [a]

[a] *Nevsehir State Hospital, Department of Otorhinolaryngology, Nevsehir, Turkey*
[b] *Nevsehir State Hospital, Department of Cardiology, Nevsehir, Turkey*

Received 29 July 2020; accepted 16 October 2020
Available online 9 November 2020

KEYWORDS
2D echocardiography;
Nasal septum deviation;
Septoplasty;
Systolic pulmonary artery pressure

Abstract
Introduction: Nasal septal deviation may contribute to a wide range of symptoms including nasal obstruction, headache, increased secretion, crusting, mucosal damage, and loss of taste and smell. Excessive increase in the respiratory resistance, as seen in nasal septal deviation, results in reduced lung ventilation, thereby potentially leading to hypoxia, hypercapnia, pulmonary vasoconstriction. The deformities in the nasal cavity can be associated with major respiratory and circulatory system diseases.
Objective: To investigate cardiovascular effects of septoplasty by comparing pre- and postoperative transthoracic echocardiography findings in nasal septal deviation patients undergoing septoplasty.
Methods: The prospective study included 35 patients with moderate and severe nasal septal deviation (mean age, 23.91 ± 7.01) who underwent septoplasty. The Turkish version of the nasal obstruction symptom evaluation, NOSE questionnaire, was administered to each participant both pre- and postoperatively in order to assess their views on the severity of nasal septal deviation, the effect of nasal obstruction, and the effectiveness of surgical outcomes. A comprehensive transthoracic echocardiography examination was performed both preoperatively and at three months postoperatively for each patient and the findings were compared among patients.
Results: Mean preoperative NOSE score was 17.34 ± 1.62 and the mean postoperative score was 2.62 ± 1.68 ($p = 0.00$). Mean preoperative systolic pulmonary artery pressure value was 22.34 ± 4.31 mmHg and postoperative value was 18.90 ± 3.77 mmHg ($p = 0.00$). Mean E/e' ratio was 5.33 ± 1.00 preoperatively and was 5.01 ± 0.90 postoperatively ($p = 0.01$). The NOSE scores, systolic pulmonary artery pressure values, and the E/e' ratios decreased significantly after septoplasty ($p < 0.05$ for all), whereas no significant difference was found in other transthoracic echocardiography parameters ($p > 0.05$).

* Corresponding author.
E-mail: deniz.avci@hotmail.com (D. Avcı).
Peer Review under the responsibility of Associação Brasileira de Otorrinolaringologia e Cirurgia Cérvico-Facial.

https://doi.org/10.1016/j.bjorl.2020.10.006

Conclusion: The decrease in NOSE scores following septoplasty indicated that the satisfaction levels of the patients were increased. Upper airway obstruction secondary to nasal septal deviation may be a cardiovascular risk factor and may affect transthoracic echocardiography measurements. Moreover, the significant decrease in the systolic pulmonary artery pressure value and E/e's ratio following septoplasty indicated that negative echocardiographic findings may be prevented by this surgery.

Introduction

The nasal septum separates the nasal cavity into two symmetric halves. Nasal septal deviation (NSD) results from congenital or traumatic deformities including dislocation, deviation, and bending of the cartilage and/or bone that form the roof of the nasal cavity.[1] NSD has a reported incidence of 14.1%–90.4%.[2] A particular trauma's severity and direction creates the size and shape of a deviated nasal septum. NSD may have a wide range of symptom manifestations including nasal obstruction, headache, increased mucosal secretion, crusting, mucosal damage, and loss of taste and smell.[3] Septoplasty is a operation commonly used in the otorhinolaryngology practice for treating NSD, which is a major cause of upper respiratory tract obstruction.[4]

The nose constitutes approximately one half of the total respiratory resistance to airflow.[5] Excessive increase in the respiratory resistance, as seen in NSD, results in reduced lung ventilation, thereby potentially leading to hypoxia, hypercapnia, pulmonary vasoconstriction, and increased intrathoracic pressure. All of these factors have a potential to affect sympathetic and parasympathetic activation and cardiovascular autonomic response and thereby to increase pulmonary artery pressure, thus possibly causing cor pulmonale, pulmonary edema, ventricular hypertrophy, cardiomegaly, cardiac arrhythmia, and ultimately heart failure. Despite the lack of sufficient evidence on their mechanisms, cardiovascular complications associated with NSD are often caused by hypoxia and hypercarbia.[6–8]

Studies investigating other causes of upper respiratory tract obstruction such as adenotonsillar hypertrophy, nasal polyps, obstructive sleep apnea syndrome (OSAS), and retrosternal goiter and investigating the effects of NSD on cardiopulmonary system have revealed that the surgical relief of upper respiratory tract obstruction led to improvement in right ventricular (RV) function and a significant reduction in pulmonary artery pressure.[6,9–14]

Septoplasty outcomes can be evaluated both objectively and subjectively. Accordingly, reliable methods are needed in otolaryngology practice to approve the accuracy of indications for septal surgery. Nevertheless, the use of objective methods for this aim is controversial and there is no consensus on an objective assessment tool for NSD.[15,16] For these reasons, in the present study, the Turkish version of the NOSE questionnaire was administered to each patient both preoperatively and at three months postoperatively. The NOSE questionnaire consists of five items, including: (1) nasal congestion or stuffiness; (2) nasal blockage or obstruction; (3) trouble breathing through my nose; (4) trouble sleeping; (5) inablity to get enough air through my nose during exercise or exertion. Each item is scored using the 5-point Likert scale with the range from 0 to 4 (0: no problem; 1: very mild; 2: moderate; 3: fairly bad; and 4: severe). The raw score ranges from 0 (no nasal obstruction) to 20 (the most severe nasal obstruction). The raw scores are added whereby higher scores indicate more severe nasal obstruction.[17]

To our knowledge, there has been no comprehensive study specifically investigating cardiovascular effects of septoplasty in patients with pure NSD. Accordingly, based on the hypothesis that the deformities in the nasal cavity could be associated with major respiratory and circulatory system diseases, the present study aimed to conduct an extensive evaluation of the cardiovascular effects of septoplasty via transthoracic echocardiography (TTE). Moreover, the study also aimed to evaluate multiple cardiovascular parameters, particularly including systolic pulmonary artery pressure (sPAP) and E/e' ratio (the ratio between peak velocity of early diastolic transmitral flow and peak velocity of early diastolic mitral annular motion as determined by pulsed wave Doppler), through a holistic approach and to examine the success of septoplasty on TTE.

Methods

The prospective study included 35 patients who had suffered from nasal obstruction for at least six months, were diagnosed with NSD and subsequently underwent septoplasty operation in our clinic between September 2019 and March 2020.

The study was approved by the local ethics committee (Approval date: August 21, 2019; nº 2019.14.108). The study protocol was conducted in accordance with the Helsinki Declaration and ethical standards and an informed written consent was obtained from each patient prior to the initiation of the research.

A clinical history was supplied from each participant. Subsequently, an extensive otorhinolaryngological examination was made by an experienced otorhinolaryngologist and an extensive cardiac examination was made by an experienced cardiologist for each patient.

NSD along with its type and indications were diagnosed via anterior rhinoscopy, nasal endoscopy, and paranasal sinus computed tomography (PCT).

The NSD types were classified based on the classification proposed by Dreher et al.[18]: Grade I — mild deviation (deviation less than half of the total distance from midline septum to the lateral wall), Grade II — moderate deviation (deviation greater than half of the total distance from midline septum to the lateral wall but not touching it), Grade III — severe deviation (deviation touching the lateral wall).

Patients who had a Grades II and III septal deviation (i.e. greater than half of the total distance), had a normal otorhinolaryngological and cardiac examination as well as a normal body mass index, normal heart rate, normal peripheral oxygen saturation (SpO_2), normal diastolic and systolic blood pressure, patients with NOSE score greater than 15 and no health problems other than NSD between the ages of 18–47 were included in the study.

Endoscopic oropharyngeal, nasopharyngeal, and laryngeal examinations were completed before the operation. In these examinations, no pathological findings were determined that could lead to obstruction in terms of OSAS. There was no obesity in patients. In addition, the patients were asked whether there was morning fatigue, daytime sleepiness, restlessness, shortness of breath, sweating, and apnea during sleep. There was no finding to point out OSAS in patients. Therefore, there was no cause for polysomnography test.

To evaluate the success of septoplasty, the Turkish version of the Nasal Obstruction Symptom Evaluation (NOSE) questionnaire was administered to each participant both pre- and postoperatively in order to assess their views on the severity of NSD, the effect of nasal obstruction, and the effectiveness of surgical outcomes.[17] Patients with NOSE score between 15-20 points were included in the study. Prior to surgery, a routine examination including blood tests, chest radiography, and electrocardiogram was performed in each patient.

Exclusion criteria were as follows: chronic lung disease, age less than 18 years and over 50 years, cardiac diseases, laryngeal disorders, morbid obesity, OSAS, cigarette smoking and alcohol consumption, prior nasal surgery, grade 3 or 4 tonsillar hypertrophy according to Brodsky scale, history of intranasal drug use, and nasal obstruction caused by other factors.

A comprehensive TTE examination was performed both preoperatively and at three months postoperatively in the Cardiology department and the results were recorded for each patient.

Septoplasty operation and postoperative period

All the operative procedures were performed by two surgeons using the same technique for all the patients at our clinic. The operation was made using the conventional closed technique under general anesthesia. After sterile draping, 1%–2% lidocaine with 1/100,000 epinephrine was injected into the subperichondrial plane along the septum using a dental-tip injector. Afterwards, a Killian incision was made on the side of the deviation. The mucoperichondrial and mucoperiosteal flaps were elevated and the deviated segment was dissected from adjacent structures via posterior and inferior chondrotomy; the septum was then fixed to the midline nasal spine. The L-strut structure was preserved by leaving an adequate strut (1 cm) in superior and anterior septal cartilages. The bone support was also preserved. Septal mucosa was transfixed with 3/0 Vicryl suture. Following bleeding control, the patency of both nasal passages was evaluated endoscopically in each patient. A septal silicone splint was inserted in both nasal passages to prevent bleeding and septal hematoma. The process was completed without complications. Mean operation time ranged between 20 and 30 min. Amoxicillin/clavulanic acid, analgesic, nasal cream, and nasal wash were suggested to patients. All the patients were discharged 24 h after operation. The septal silicone splints were removed, and the nasal passages were cleansed at postoperative day 6. Patients were controlled at week 2 and at 1 and 3 months. No intranasal steroids and nasal decongestants were used postoperatively. In the postoperative period, a complete nasal passage opening in the nose was evaluated by another otolaryngologist as an evidence of whether the operation was successful or not and no residual NSD (i.e. uncorrected NSD) was detected.

Transthoracic echocardiography examination and assessment

M-mode and 2-dimensional (2D) echocardiographic images and spectral and color Doppler images were obtained using the GE VIVID S60 Ultrasound System (General Electric, Chicago, Illinois, USA) with a 3.5-MHz probe. In line with the American Society of Echocardiography's Guidelines and Standards, parasternal long-axis and short-axis views and apical 2- and 4-chamber views were evaluated with the patient in the left lateral decubitus position.[19] From apical 4-chamber views, transmitral pulsed wave Doppler velocities were registered with the sample volume placed within the valve orifice near the leaflet tips and by analyzing three consecutive cycles. Left atrium diameter (LAD), aortic root (Ao), ascending aorta diameter (AoAscD), right atrium diameter (RAD), RV diameter (RVD), pulmonary artery diameter (PAD), RV outflow tract (RVOT), sPAP, interventricular septum thickness at end-systole (IVSs), interventricular septum thickness at end-diastole (IVSd), ejection fraction (EF), stroke volume (SV), left ventricular internal dimension at end-systole (LVIDs), left ventricular internal dimension at end-diastole (LVIDd), fractional shortening (FS), left ventricular posterior wall thickness at end-systole (LVPWs), left ventricular posterior wall thickness at end-diastole (LVPWd), peak velocity of early diastolic transmitral flow (E), peak velocity of early diastolic mitral annular motion as determined by pulsed wave doppler (e'), pulmonary maximum velocity (PVmax), aortic maximum velocity (AVmax) and the E/e' ratio were measured and then recorded.

sPAP was calculated from the continuous-wave Doppler peak tricuspid regurgitation velocity. All the TTE processes were performed both preoperatively and at three months postoperatively by the same experienced cardiologist who was blinded to the clinical and surgical characteristics of the patients, using the same ultrasound device.

Statistical analysis

Descriptive were expressed as mean ± standard deviation. Categorical variables were compared using Chi-square test. Normal distribution of continuous variables was analyzed using Shapiro Wilk test and Q–Q Plots. Then, differences between pre- and post-operative TTE findings were compared. Variables with normal distribution were compared using Paired sample *t*-test and variables nonnormal distribution were compared using Wilcoxon signed-rank test. A *p*-value of <0.05 was considered significant.

Results

The 35 patients were comprised of 16 (45.7%) men and 19 (54.3%) women with a mean age of 23.91 ± 7.01 (range, 18–47) years. Unilateral NSD was present in 31 (88.5%) and bilateral NSD (S type) was present in 4 (11.4%) patients. Mean time for nasal obstruction was 5.34 ± 3.24 years. According to Dreher's classification, 21 (60%) patients had Grade II and 14 (40%) patients had Grade III pure NSD. According to NSD etiology, 32 of the patients had congenital etiology. Only 3 patients had a traumatic status. On preoperative PCT, no clinical condition other than NSD was detected as a cause of nasal obstruction in any patient. Table 1 presents the demographic characteristics of the patients.

Postoperative nasal examination revealed no complications such as bleeding, septal perforation, hematoma, infection, residual NSD, and synechia in any patient.

Mean preoperative NOSE score was 17.34 ± 1.62 and the mean postoperative score was 2.62 ± 1.68 and a significant reduction was observed in postoperative NOSE points compared to preoperative scores ($p = 0.00$).

Mean preoperative sPAP value was 22.34 ± 4.31 mmHg and the mean postoperative value was 18.90 ± 3.77 mmHg ($p = 0.00$) (Fig. 1). In 32 of 35 patients, there was a decrease in the sPAP value compared with the preoperative period. Preoperatively, 10 (28.5%) patients had an sPAP value of >25 mmHg, which decreased to below 25 mmHg postoperatively in eight patients. In the remaining two patients, it decreased remarkably postoperatively although it remained above 25 mmHg. In only three patients, a minimal increase was observed in postoperative sPAP value compared to its preoperative value. Mean E/e' ratio was 5.33 ± 1.00 preoperatively and was 5.01 ± 0.90 postoperatively ($p = 0.01$) (Fig. 2). Both the sPAP values and E/e' ratios decreased significantly after surgery, indicating clinical improvement in our patients. On the other hand, although the LAD, AoAscD, RAD, RVD, PAD, RVOT, EF, SV, LVIDs, LVIDd, FS, LVPWs, LVPWd, PVmax, and AVmax values decreased after surgery, no significant difference was established in any parameter ($p > 0.05$) (Table 2).

Table 1 Demographic data of patients with nasal septum deviation.

Variables	Mean ± SD
Age, years	23.91 ± 7.01
Male, n (%)	16 (45.7)
Female, n (%)	19 (54.3)
Body Mass Index, kg/m^2	
Preoperative	22.08 ± 2.10
Postoperative	22.09 ± 2.10
p	0.95

SD, standard deviation.

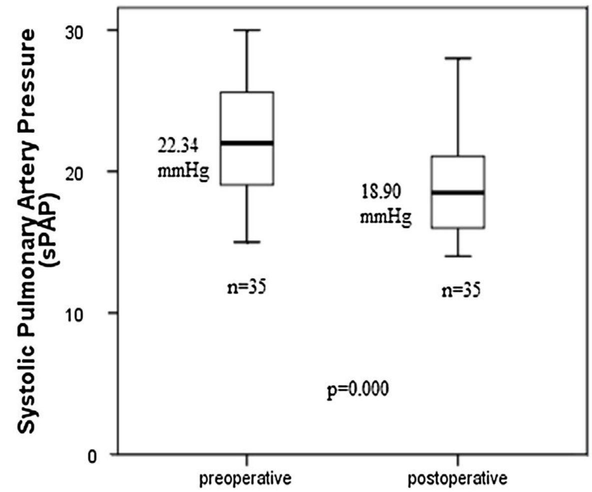

Figure 1 Distribution of preoperative and postoperative systolic pulmonary artery pressure (sPAP) values on box plot graph.

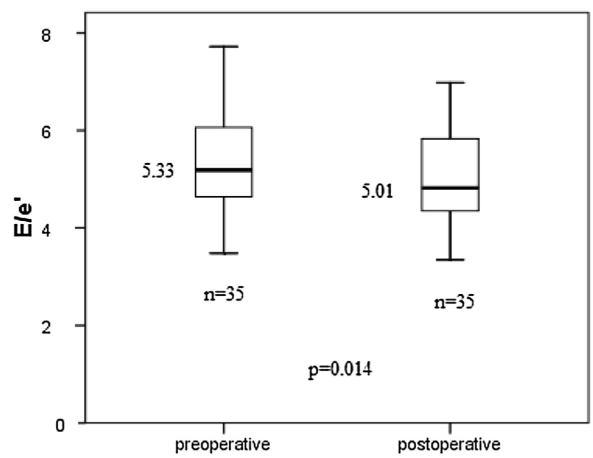

Figure 2 Distribution of preoperative and postoperative E/e' values on box plot graph.

Discussion

Nasal airflow is the primary criterion for successful septoplasty. The nasal septum is the main support of the nasal roof and also plays key role in regulating nasal airflow.[1] NSD is considered as the leading cause of numerous clinical conditions associated with nasal obstruction including concha bullosa, lower concha hypertrophies, and nasal polyposis.[20] Septoplasty is the mainstay for surgical treatment of NSD.[4] According to our clinical experience, this technique is mostly performed under general anesthesia and provides short healing time and patients are mostly discharged 24 h after the procedure.

Septoplasty studies with short control periods (6 weeks–6 months) have revealed that the symptoms were relieved in 45%–92% of the patients following surgery.[21] In our study, since the patients maintained adequate nasal airflow, their NOSE scores improved significantly at three months postoperatively (decreased by 84.9%), which indicates a high success rate for septoplasty. We think the improvement in the NOSE scale scores are enhanced with the improvement

Table 2 Comparison of preoperative and postoperative clinic findings in patients with nasal septum deviation (paired sample *t*-test was used).

Parameters	Preoperative (n = 35) Mean ± SD	Postoperative (n = 35) Mean ± SD	*p*-Value
LAD, cm	3.16 ± 0.42	3.09 ± 0.47	0.11
Ao, cm	2.35 ± 0.25	2.40 ± 0.28	0.26
AoAscD, cm	2.82 ± 0.37	2.76 ± 0.32	0.07
RAD, cm	3.13 ± 0.38	3.07 ± 0.36	0.10
RVD, cm	3.17 ± 0.35	3.12 ± 0.38	0.22
PAD, cm	1.94 ± 0.21	1.90 ± 0.16	0.15
RVOT, cm	2.21 ± 0.21	2.20 ± 0.23	0.91
IVSs, cm	1.07 ± 0.15	1.08 ± 0.11	0.47
IVSd, cm	0.78 ± 0.11	0.80 ± 0.11	0.22
EF, %	68.74 ± 4.10	68.40 ± 2.96	0.64
SV, mL	62.30 ± 10.72	60.77 ± 9.71	0.27
LVIDs, cm	2.66 ± 0.31	2.65 ± 0.30	0.75
LVIDd, cm	4.37 ± 0.39	4.35 ± 0.27	0.73
FS, %	40.39 ± 3.79	39.39 ± 3.21	0.10
LVPWs, cm	1.06 ± 0.12	1.04 ± 0.12	0.51
LVPWd, cm	0.79 ± 0.11	0.78 ± 0.11	0.66
PVmax, m/s	1.11 ± 0.17	1.09 ± 0.17	0.46
AVmax, m/s	1.26 ± 0.18	1.24 ± 0.14	0.39
E/e’	5.33 ± 1.00	5.01 ± 0.90	0.01[a]
sPAP, mm Hg	22.34 ± 4.31	18.90 ± 3.77	0.00[a]
NOSE score	17.34 ± 1.62	2.62 ± 1.68	0.00[a]

SD, standard deviation; LAD, left atrium diameter; Ao, aortic root; AoAscD, ascending aorta diameter; RAD, right atrium diameter; RVD, right ventricular diameter; PAD, pulmonary artery diameter; RVOT, right ventricular outflow tract; sPAP, systolic pulmonary artery pressure; IVSs, interventricular septum thickness at end-systole; IVSd, interventricular septum thickness at end-diastole; EF, ejection fraction; SV, stroke volume; LVIDs, left ventricular internal dimension at end-systole; LVIDd, left ventricular internal dimension at end-diastole; FS, fractional shortening; LVPWs, left ventricular posterior wall thickness at end-systole; LVPWd, left ventricular posterior wall thickness at end-diastole; E, peak velocity of early diastolic transmitral flow; E’, peak velocity of early diastolic mitral annular motion as determined by pulsed wave Doppler; PVmax, pulmonary maximum velocity; AVmax, aortic maximum velocity; NOSE, nasal obstruction symptom evaluation.

[a] Statistically significant.

in sPAP and E/e’ ratio. We claim that this idea increases the importance of our study.

Relationship between NSD and cardiovascular diseases and the cardiovascular complications of upper airway obstruction (right heart failure, cor pulmonale, and sudden death) has recently become a major concern among researchers.[6,9–11,22,23]

Caglar et al. expressed that pulmonary artery pressure (PAP) is a parameter easily measured on TTE. The authors found that the PAP values and RVD were significantly higher in NSD patients and also noted that NSD affected not only the RV function but also the left ventricular (LV) function in early life. Based on these findings, the authors suggested that prompt correction of NSD may prevent cardiopulmonary complications.[22]

Fidan et al.[9] found that the mean pulmonary artery pressure (mPAP) was significantly higher in patients with marked NSD compared to control subjects. It was also found that mPAP values of NSD patients decreased significantly after surgery. The authors developed an emergency treatment plan for septoplasty to prevent and reverse possible adverse alterations in the cardiopulmonary system in patients with high mPAP values. Following surgery, the mPAP values were normalized in 16 (72.7%) out of 22 patients and decreased remarkably in the remaining six patients.

Hassanpour et al. also found higher mPAP values in patients with marked NSD undergoing septorhinoplasty.[24] Wiegand and Zwillich suggested that hypoxia is a leading cause of cardiac mortality in patients with OSAS and that NSD may lead to arrhythmia by producing changes in cardiac autonomic functions.[25]

Yilmaz et al. reported that adenotonsillar hypertrophy that causes airway obstruction increases PAP values and patients with adenotonsillar hypertrophy have significantly reduced PAP values following adenotonsillectomy.[10] In a 2016 study, Ozkececi et al. compared 25 NSD patients with control subjects and reported that the NSD patients had higher PAP values and also had worse RV function compared to controls. In these patients, the RV function improved and the mean PAP value decreased following septoplasty.[26] Ghazipour et al. evaluated 35 NSD patients and found that the mPAP values of the patients decreased after surgery (from 23.8 to 19.8 mmHg).[27] As consistent with the literature, the sPAP values of our patients also decreased following septoplasty.

The normal range of sPAP value is 18–25 mmHg.[28] In routine practice, RV dysfunction is commonly assessed by sPAP value.[23] RV function is accepted as the main predictor of prognosis and symptoms in patients with pulmonary hypertension.[29] Simsek and Simsek evaluated RV function in patients with nasal obstruction associated with NSD using TTE and reported that the tissue Doppler based measurements of RV systolic function improved, the sPAP values decreased (from 32.54 ± 5.24 to 24.22 ± 4.55 mmHg), and the SpO_2 levels increased in NSD patients following septoplasty. Based on these findings, the authors proposed that chronic hypoxia could be treated by septoplasty.[23]

Kayapinar et al. found that the mean atrial electromechanical delay times and PAP values in NSD patients decreased after septoplasty and suggested that NSD may result in increased pulmonary vascular resistance and mPAP values, thereby leading to an adverse effect on RV function.[30]

Ogura et al. reported that the nasopharyngeal reflex created with the aid of bronchial muscles compromised the pulmonary compliance associated with nasal obstruction and also decreased the elevated pulmonary resistance.[31] This phenomenon could be explained by the reduced sPAP in our patients following septoplasty. Sahin et al. evaluated pre- and postoperative tricuspid annular-plane systolic excursion (TAPSE), RVD, E/A ratio, and mPAP values and reported that the TAPSE and mPAP values were significantly improved following surgery. However, in a similar way to our study, the authors found no significant change in RVD.[32]

To our knowledge, there has been no comprehensive study specifically investigating the cardiovascular effects of septoplasty and most of the studies evaluating NSD patients have only compared PAP values. In the present study, we evaluated multiple cardiovascular parameters through a holistic approach using TTE, which is a noninvasive, practical, and effective technique.

The E/e' ratio can be practically measured by TTE and a lower E/e' ratio indicates improved diastolic functions.[33] On the other hand, the E/e' ratio has been shown to be a prognostic predictor of survival in patients with congestive heart failure, hypertension, and acute myocardial infarction.[34–36] An average E/e ́ratio of >12 is indicative of diastolic dysfunction, a ratio of 8–12 denotes possible diastolic dysfunction, and a ratio of <8 is accepted normal.[37]

Zhou et al. suggested that the E/e' ratio is a noninvasive marker of LV filling pressure and a strong noninvasive indicator of cardiac events in patients with preserved LV ejection fraction and revealed that a 1-unit rise in the E/e' ratio increased the risk of cardiac events by 17%. In the same study, the mean E/e' ratio was found to be 10.3 ± 2.8 in patients with no cardiac events as opposed to 14.5 ± 5.3 in patients with cardiac events and the difference was statistically significant. The authors also noted that the E/e' ratio could play an early and effective role in the classification of cardiovascular risk in patients with hypertension.[38] In our study, depending on its important role in cardiac events, the E/e' ratio was also evaluated in our patients and, to our knowledge, the present study is the first of its kind to evaluate the E/e' ratio in NSD patients.

In the present study, sPAP and E/e' ratio decreased significantly after septoplasty, suggesting that septoplasty may improve some cardiac disorders and also may reduce the cardiovascular risk. Many studies are needed to confirm this idea.

The strength of our research was that it had a prospective project and a high follow up rate (100%), patients with a higher probability of surgical success were selected, and TTE parameters that had not been appraised in previous researches were examined in the research. Moreover, possible biases were eliminated by blinding the surgeon to the TTE records of the patients.

Our study was limited since the TTE findings were not confirmed by cardiac catheterization, the evaluations were made based on short-term TTE data, the research had a relatively small number of patients, the patency of nasal passages was not approved by objective techniques such as rhinomanometry, exclusion of patients with high pulmonary artery pressure, the absence of polysomnography (for OSAS exclusion) and there was no control group. The lack of a control group was due to the fact that most of the patients were selected for surgical treatment of NSD. However, there is not another method or treatment with proven efficacy for NSD other than operation. This situation influenced the use of a control group.

Conclusion

The decrease in NOSE scores following operation indicated that the satisfaction levels of the patients were increased, and that septoplasty had a high success rate. Upper airway obstruction secondary to NSD may be a cardiovascular risk factor and may affect TTE measurements. Moreover, the significant decrease in the sPAP value and E/e's ratio following septoplasty indicated that negative echocardiographic findings and cardiovascular risks may be prevented by this corrective nasal obstruction surgery.

Research involving human participants

All procedures performed in studies involving human participants were in accordance with the ethical standards of the Ethics Committee of Nevşehir Hacı Bektaş Veli University Scientific Research (Approval date: August 21, 2019; nº 2019.14.108) and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent

Informed written consent was obtained from each participant prior to the initiation of the study.

Conflict of interest

The authors declare no conflicts of interest.

References

1. Brain DJ. The nasal septum. In: Kerr AG, Gleeson M, editors. Scott-Brown's otolaryngology. 2nd ed. Oxford: Reed Educational and Professional Publishing Ltd.; 1997. p. 1–25.
2. Shokri A, Faradmal MJ, Hekmat B. Correlations between anatomical variations of the nasal cavity and ethmoidal sinuses on cone-beam computed tomography scans. Imaging Sci Dent. 2019;49:103–13.
3. Güzelküçük Akay H, Bayar Muluk N, Inal M, Şimşek G, Kiliç R. Evaluation of olfactory sensation, acoustic rhinometry, and quality of life of the patients with nasal septal deviation. J Craniofac Surg. 2019;30:1221–7.
4. Most SP, Rudy SF. Septoplasty: basic and advanced techniques. Facial Plast Surg Clin North Am. 2017;25:161–9.
5. Proctor DF. The upper airways. I. Nasal physiology and defense of the lungs. Am Rev Respir Dis. 1977;115:97–129.
6. Naiboglu B, Deveci S, Duman D, Kaya KS, Toros S, Kinis V, et al. Effect of upper airway obstruction on pulmonary arterial pressure in children. Int J Pediatr Otorhinolaryngol. 2008;72:1425–9.
7. Olsen KD, Kern EB, Westbrook PR. Sleep and breathing disturbance secondary to nasal obstruction. Otolaryngol Head Neck Surg. 1981;89:804–10.
8. Luke MJ, Mehrizi A, Folger GM Jr, Rowe RD. Chronic nasopharyngeal obstruction as a cause of cardiomegaly, cor pulmonale, and pulmonary edema. Pediatrics. 1966;37:762–8.
9. Fidan V, Aksakal E. Impact of septoplasty on pulmonary artery pressure in patients with markedly deviated septum. J Craniofac Surg. 2011;22:1591–3.
10. Yilmaz MD, Onrat E, Altuntaş A, Kaya D, Kahveci OK, Ozel O, et al. The effects of tonsillectomy and adenoidectomy on pulmonary arterial pressure in children. Am J Otolaryngol. 2005;26:18–21.
11. McNicholas WT. Chronic obstructive pulmonary disease and obstructive sleep apnea: overlaps in pathophysiology, systemic inflammation, and cardiovascular disease. Am J Respir Crit Care Med. 2009;180:692–700.
12. Sommaruga L, Del Rio P, Accardi FG, Cademartiri F, Arcuri MF, Sianesi M. Severe obstruction of upper airway by cervicomediastinal goitre in patients with cardiopulmonary insufficiency. G Chir. 2008;29:354–8.
13. Koc S, Aytekin M, Kalay N, Ozcetin M, Burucu T, Ozbek K, et al. The effect of adenotonsillectomy on right ventricle function and pulmonary artery pressure in children with adenotonsillar hypertrophy. Int J Pediatr Otorhinolaryngol. 2012;76: 45–8.
14. Şimşek E, Şimşek Z, Taş MH, Kucur C, Günay E, Üçüncü H. Evaluation of right ventricular functions in patients with nasal polyposis: an observational study. Anadolu Kardiyol Derg. 2013;13:251–6.
15. André RF, Vuyk HD, Ahmed A, Graamans K, Nolst Trenité GJ. Correlation between subjective and objective evaluation of the nasal airway. A systematic review of the highest level of evidence. Clin Otolaryngol. 2009;34:518–25.
16. Konstantinidis I, Triaridis S, Triaridis A, Karagiannidis K, Kontzoglou G. Long term results following nasal septal surgery. Focus on patients' satisfaction. Auris Nasus Larynx. 2005;32: 369–74.
17. Onerci Celebi O, Araz Server E, Yigit O, Longur ES. Adaptation and validation of the turkish version of the nasal obstruction symptom evaluation scale. Int Forum Allergy Rhinol. 2018;8:72–6.
18. Dreher A, de la Chaux R, Klemens C, Werner R, Baker F, Barthlen G, et al. Correlation between otorhinolaryngologic evaluation and severity of obstructive sleep apnea syndrome in snorers. Arch Otolaryngol Head Neck Surg. 2005;131: 95–8.
19. Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Pellikka PA, et al. Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. J Am Soc Echocardiogr. 2005;18: 1440–63.
20. Acar B, Yavuz B, Karabulut H, Gunbey E, Babademez MA, Yalcin AA, et al. Parasympathetic overactivity in patients with nasal septum deformities. Eur Arch Otorhinolaryngol. 2010;267: 73–6.
21. Pedersen L, Schiöler L, Holmberg K, Emanuelsson CA, Hellgren J. Age and unplanned postoperative visits predict outcome after septoplasty: a national Swedish register study. Int J Otolaryngol. 2018;2018:2379536.
22. Caglar O, Aksit E. Evaluation of heart functions with detailed echocardiogram in patients with septum deviation. J Craniofac Surg. 2018;29:2148–52.
23. Simsek Z, Simsek E. Does nasal surgery affect right ventricular myocardial functions at the tissue level in patients with nasal septum deviation? J Clin Med. 2018;7:186.
24. Hassanpour SE, Moosavizadeh SM, Araghi FM, Eshraghi B. Pulmonary artery pressure in patients with markedly deviated septum candidate for septorhinoplasty. World J Plast Surg. 2014;3:119–21.
25. Wiegand L, Zwillich CW. Obstructive sleep apnea. Dis Mon. 1994;40:197–252.
26. Ozkececi G, Akci O, Bucak A, Ulu S, Yalım Z, Aycicek A, et al. The effect of septoplasty on pulmonary artery pressure and right ventricular function in nasal septum deviation. Eur Arch Otorhinolaryngol. 2016;273:3747–52.
27. Ghazipour A, Noori SM, Madjidi S, Nikakhlagh S, Saki N, Rekabi H. Determination of the effect of nasal septoplasty on pulmonary artery pressure in patients with deviated nasal septum. Jentashapir J Health Res. 2014;5:e21890.
28. Nauser TD, Stites SW. Diagnosis and treatment of pulmonary hypertension. Am Fam Physician. 2001;63:1789–98.
29. Sandoval J, Bauerle O, Palomar A, Gómez A, Martínez-Guerra ML, Beltrán M, et al. Survival in primary pulmonary hypertension. Validation of a prognostic equation. Circulation. 1994;89:1733–44.
30. Kayapinar O, Kaya A, Ozde C, Cebeci D, Unlu I. Evaluating the effect of nasal septoplasty on atrial electromechanical features. Am J Otolaryngol. 2019;40:179–82.
31. Ogura JH, Unno T, Nelson JR. Baseline values in pulmonary mechanics for physiologic surgery of the nose. Preliminary report. Ann Otol Rhinol Laryngol. 1968;77:367–97.
32. Sahin MS, Ozmen OA, Yalcin MU. Does septoplasty improve heart functions of patients with septum deviation in terms of echocardiography findings? J Craniofac Surg. 2017;28:1803–5.
33. Hadi S, Tabrizi MT, Separham A, Hoseinalizade M, Sadeghi M, Parizad R. Prevalence and predictors of left ventricle regional wall motion abnormality 6 weeks after primary percutaneous intervention in patients with first acute anterior myocardial infarction. Crescent J Med Biol Sci. 2020;7:116–23.
34. Acil T, Wichter T, Stypmann J, Janssen F, Paul M, Grude M, et al. Prognostic value of tissue Doppler imaging in patients with chronic congestive heart failure. Int J Cardiol. 2005;103:175–81.
35. Sharp ASP, Tapp RJ, Thom SAM, Francis DP, Hughes AD, Stanton AV, et al. Tissue Doppler E/E' ratio is a powerful predictor of primary cardiac events in a hypertensive population: an ASCOT substudy. Eur Heart J. 2010;31:747–52.
36. Hillis GS, Møller JE, Pellikka PA, Gersh BJ, Wright RS, Ommen SR, et al. Noninvasive estimation of left ventricular filling pres-

sure by E/e' is a powerful predictor of survival after acute myocardial infarction. J Am Coll Cardiol. 2004;43:360—7.

37. Arques S, Roux E, Luccioni R. Current clinical applications of spectral tissue Doppler echocardiography (E/E' ratio) as a non-invasive surrogate for left ventricular diastolic pressures in the diagnosis of heart failure with preserved left ventricular systolic function. Cardiovasc Ultrasound. 2007;5:16.

38. Zhou D, Huang Y, Fu M, Cai A, Tang S, Feng Y. Prognostic value of tissue Doppler E/e' ratio in hypertension patients with preserved left ventricular ejection fraction. Clin Exp Hypertens. 2018;40:554—9.