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Original Paper

Obstructive Sleep Apnea Is Closely Related to Cardiovascular Risk Factors, but Not to Clinical Recurrence of Atrial Fibrillation after Catheter Ablation: An Analysis of Atrial Fibrillation Patients

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Keywords
Cardiovascular risk · Obstructive sleep apnea · Atrial fibrillation recurrence

Abstract
Background: Obstructive sleep apnea (OSA) is a well-known predictor of atrial fibrillation (AF). However, OSA usually accompanies other risk factors of AF. We tried to investigate whether OSA is related to AF recurrence after catheter ablation. ***Methods:*** A total of 378 patients (mean age 59.9 ± 10.7 years, 72.5% male) who underwent catheter ablation of AF were enrolled and underwent overnight ambulatory polysomnography before the ablation procedure. These patients were examined once every 3 months at the outpatient clinic to determine AF recurrence. ***Results:*** Based on the apnea-hypopnea index (AHI), we divided the study subjects into 3 groups defined as mild (AHI <10), moderate (10 <AHI <30), or severe (AHI >30) OSA. Patients with severe OSA had a higher prevalence of hypertension, diabetes mellitus, and coronary artery disease (CAD). However, AF recurrence was not different between the three groups. The Kaplan-Meier analysis also showed no significant difference in AF recurrence according to the degree of severity of OSA. Multivariate logistic regression analysis revealed that OSA might be a predictor of CAD; however, Cox regression analysis showed that only early recurrence is closely related to AF recurrence after catheter ablation, rather than the severity of OSA. ***Conclusion:*** This study shows that the severity of OSA is not associated with the recurrence of AF after catheter ablation in Korean patients. Treatment of OSA for the sole indication of lowering AF recurrence may need to be reconsidered.

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Introduction

Obstructive sleep apnea (OSA) is a chronic disorder characterized by repeated cessation of air flow during sleep despite respiratory efforts [1]. It is relatively common with a prevalence of 2~7% in the general population [2]. OSA causes various physiologic changes such as sympathetic activation, endothelial dysfunction, vascular oxidative stress, and inflammation, which are all related to cardiovascular disease including atrial fibrillation (AF) [3]. Recent studies showed that continuous positive airway pressure (CPAP) therapy for OSA raises the efficacy of radiofrequency catheter ablation (RFCA) in patients with severe OSA [4–6]. However, these studies should be interpreted with caution because their study populations were relatively small and their diagnosis of OSA was insufficient. In addition, patients with OSA usually have associated other cardiovascular risk factors. Therefore, in this study, we investigated the sole impact of OSA on the recurrence of AF after RFCA in Korean patients with AF.

Methods

Study Population

All patients gave informed consent for inclusion in the Yonsei AF Ablation Cohort Database. This study population consisted of 378 patients with AF who underwent RFCA at our institution from December 2011 through May 2014. The study exclusion criteria were as follows: (1) permanent AF, refractory to electrical cardioversion, (2) AF with valvular disease ≥grade 2, (3) associated structural heart disease other than left ventricular hypertrophy, (4) a history of prior RFCA or cardiac surgery, and (5) patients who did not have sleep apnea assessments. In order to visualize the anatomy of the pulmonary vein (PV) and the left atrium (LA), 3D-cardiac CT images were acquired in all patients. All antiarrhythmic drugs were discontinued for a minimum period of 5 half-lives, and amiodarone was stopped at least 4 weeks before the procedure. This study received prior approval from the Institutional Review Board at the Yonsei University Health System (IRB No. 4-2014-1080).

Diagnosis of OSA

Before catheter ablation, polysomnography was performed with a portable device (Embletta X-100; Embla Systems Inc., Broomfield, CO, USA), which is a level 2 sleep monitoring system, according to the recommendations of the European Respiratory Society and the European Society of Hypertension [7]. None of the patients used CPAP at baseline. The device recorded data including the electroencephalogram, electromyogram, electrocardiogram (ECG) or heart rate, airflow, respiratory effort, and oxygen saturation. Polysomnographic data were manually scored by an experienced specialist in accordance with the definition of OSA syndrome by the American Academy of Sleep Medicine [8]. Apnea was defined as a >90% decrease in airflow for >10 s in the presence of thoracoabdominal ventilatory efforts, and hypopnea was defined as a >50% reduction in airflow with desaturation of >3% or arousal for >10 s, or >30% reduction in airflow with desaturation of >4% for >10 s in the presence of thoracoabdominal ventilatory efforts. The apnea-hypopnea index (AHI) was calculated by dividing the total duration of apnea and hypopnea by the total sleep time. Patients were grouped according to OSA severity, which was classified as previously described [4]: mild OSA (AHI <10), moderate OSA (10 ≤AHI <30), severe OSA (AHI ≥30). Desaturation was defined as an oxygen saturation of ≤90%, and the desaturation index was defined as the number of desaturation episodes per hour of sleep. The Pittsburgh Sleep Quality Index (SQI) and the Epworth Sleepiness Scale (ESS) data were collected from participants before polysomnography.

Electrophysiologic Mapping and RFCA

The details of the electrophysiologic mapping and RFCA technique and strategy were as described in previous studies [9, 10]. Briefly, RF energy was delivered using an open irrigated-tip catheter (Celsius, Johnson and Johnson Inc., Diamond Bar, CA, USA; Coolflex, St. Jude Medical Inc., Minnetonka, MN, USA; 30–35 W; 47 °C). All patients initially underwent circumferential PV isolation and bidirectional block of the cavotricuspid isthmus. For the patients with persistent AF, we added a roof line, a posterior inferior line, and an anterior line as the standard lesion set. The operator could choose to perform additional ablations in the

superior vena cava or non-PV foci, or conduct complex fractionated electrograms at his discretion. The procedure was complete when there was no immediate recurrence of AF after cardioversion with isoproterenol infusion (5 μg/min). If there were mappable AF triggers or atrial premature beats, we carefully mapped and ablated those non-PV foci as much as possible. All RFCA procedures were conducted by 2 operators with over 10 years of experience, and according to the specific protocol listed above.

Follow-Up after Ablation

All patients were given no anti-arrhythmic drugs after RFCA. Patients were asked to visit follow-up appointments at 1, 3, 6, 9, and 12 months after RFCA, and every 6 months thereafter in an outpatient clinic. An ECG was obtained at every visit, and additional ECGs were recorded when patients reported symptoms suggestive of AF. A Holter ECG (24 or 48 h) and/or an event recorder were worn at 3, 6, 12, 18, and 24 months after RFCA according to the 2012 HRS/EHRA/ECAS Expert Consensus Statement guidelines [11]. Recurrence of AF was defined as any episode of AF or atrial tachycardia lasting longer than 30 s. AF episodes within the 3-month blanking period during follow-up were regarded as an early recurrence. Any ECG documentation of AF recurrence after 3 months was diagnosed as clinical recurrence, which was analyzed as endpoint in this study.

Statistical Analysis

Continuous variables were expressed as mean ± SD and were compared using a one-way analysis of variance (ANOVA) or an independent *t* test. Categorical variables were summarized as numbers and percentages of the group total. Pearson's χ^2 test was performed to compare categorical variables. The association between the severity of OSA and coronary artery disease (CAD) was rated using multiple logistic regression analysis. AF recurrence-free survival curves for each group were presented as Kaplan-Meier plots and compared by log-rank test. Univariate and multivariate Cox regression analyses were conducted to identify the predictor of AF recurrence. All statistical analyses were performed using SPSS version 20.0 for Windows (Statistical Package for the Social Sciences Inc., Chicago, IL, USA). A p value of <0.05 was considered statistically significant.

Results

OSA as a Risk Factor for CAD in Patients with AF

Table 1 shows baseline characteristics of the study participants according to the severity of their OSA. Overall, the patients were middle aged (59.9 ± 10.7 years old), overweight (body mass index [BMI] 25.1 ± 3), and had normal cardiac function (left ventricular ejection fraction 63 ± 7.9%). Anthropometric data revealed that weight and BMI had a tendency to be higher in the moderate and severe OSA groups; however, this trend did not reach statistical significance. Notably, neck diameter and waist circumference were significantly higher in the severe OSA group compared to the mild OSA group. Patients with moderate and severe OSA were older and had higher incidences of diabetes mellitus (DM) and hypertension than those with mild OSA. Although left ventricular function was not different among the groups, LA volume was larger in the moderate and severe OSA groups. Notably, the prevalence of CAD was significantly higher with increasing severity of OSA ($p < 0.001$).

In order to evaluate the role of OSA as a predictor of CAD, multivariate logistic regression analysis was performed (online suppl. Table 1; for all online suppl. material, see www.karger.com/doi/10.1159/000489854). This analysis revealed that severe OSA was a powerful risk factor for CAD, even though it was not statistically significant in the regression model adjusted for both DM and hypertension as a confounding factor.

Relationship between OSA and the Recurrence of AF

After ablation of AF, the mean duration of the follow-up period was 20.2 ± 9.7 months, and the time to clinical recurrence was 11.8 ± 8.1 months. With respect to the presence or

Table 1. Clinical characteristics according to the severity of OSA

	Mild OSA (*n* = 190)	Moderate OSA (*n* = 131)	Severe OSA (*n* = 57)	*p* value
Age, years	55.7±11.5	61.7±8.9	63±8.8	**<0.001**
Male	123 (64.7)	107 (81.7)	44 (77.2)	**0.006**
Type of AF				0.542
Persistent	69 (36.3)	50 (38.2)	17 (29.8)	
Paroxysmal	121 (63.7)	81 (61.8)	40 (70.2)	
Hypertension	70 (36.8)	80 (61.1)	38 (66.7)	**<0.001**
DM	23 (12.1)	20 (15.3)	14 (24.6)	**0.029**
Prior stoke or TIA	24 (12.6)	24 (18.3)	11 (19.3)	0.131
CHA2DS2-VASc	1.5±1.3	2±1.5	2.3±1.7	**<0.001**
Coronary artery disease	15 (7.9)	26 (19.8)	16 (28.1)	**<0.001**
Valvular heart disease	18 (9.5)	10 (7.6)	3 (5.3)	0.292
Systolic blood pressure, mm Hg	118.5±14.8	119.0±10.7	122.1±13.1	0.581
Diastolic blood pressure, mm Hg	75.4±10.8	73.5±9.3	75.7±5.4	0.725
LA dimension, mm	41±5.8	42.7±5.6	42.9±7.2	**0.016**
LA volume index, mL/m^2	30.8±15	34.9±14.6	34.2±15.7	**0.038**
LVEF, %	63.4±7	62.5±8.2	63±9.9	0.606
E/E'	10.5±4.5	10.8±4.7	10.3±4	0.75
LV mass index, g/m^2	68.9±42.4	75.3±43	75.1±47.3	0.372
LVEDD, mm	49.4±4.4	49.8±4.3	50.1±5.6	0.596
LVESD, mm	33.4±5.7	33.6±5.6	34.1±6.7	0.772
Height, cm	166.2±9.1	164.7±16.4	168±9.2	0.219
Weight, kg	68.7±11.8	69.8±9.9	72.6±12.5	0.072
BMI	24.8±3.1	25.3±2.6	25.6±3.2	0.093
Neck diameter, cm	36.8±3.2	37.5±2.6	38.1±3.2	**0.008**
Waist circumference, cm	89.3±11.2	91.6±8.9	95.3±8.5	**<0.001**
hsCRP, mg/dL	3±8.3	1.6±2.9	2±2.5	0.138
AHI, apnea + hypopnea/h	3.8±3	17.8±5.7	44.5±13	**<0.001**
ESS	6.8±4.1	6.8±4.6	6.3±4	0.675
SQI	5.2±3.1	4.9±3	5.5±3.5	0.393
Test time, min	406.8±89.4	437.6±86.9	435.3±96.2	**0.005**
Mean duration of OSA, s	11.3±9.6	21±6.8	24.9±6.2	**<0.001**
Mean duration of hypopnea, s	20.1±7.2	23.6±4.9	24.7±5.5	**<0.001**
Minimum oxygen saturation, %	89.7±3.6	85.2±4.3	81.5±4.4	**<0.001**
Desaturation index, event/h	3.4±2.8	15.5±6.1	39.3±14	**<0.001**
Early recurrence	58 (30.5)	42 (32.1)	14 (24.6)	0.581
Antiarrhythmic drug after procedure	31 (16.3)	27 (20.6)	7 (12.3)	0.342

p values <0.05 are marked in bold. Data are presented as *n* (%) or mean ± SD. AHI, apnea-hypopnea index; AF, atrial fibrillation; BMI, body mass index; DM, diabetes mellitus; ESS, Epworth sleepiness scale; hsCRP, high sensitive C reactive protein; LA, left atrium; LV, left ventricle; LVEDD, LV end diastolic dimension; LVEF, LV ejection fraction; LVESD, LV end systolic dimension; OSA, obstructive sleep apnea; SQI, sleep quality index; TIA, transient ischemic attack.

absence of clinical recurrence, LA dimension, type of AF, and early recurrence were significantly different between each group (Table 2). The use of antiarrhythmic drug after catheter ablation was more frequent in patients with clinical recurrence. The average times to recurrence were 11.3 ± 7.4 months in the mild OSA group, 13.3 ± 9.3 months in the moderate OSA group, and 7.9 ± 3.4 months in the severe OSA group. However, clinical recurrence rate and duration were not statistically different between the groups. There was no difference in the use of antiarrhythmic drug according to the severity of OSA (Table 1).

Table 2. Clinical characteristics according to the recurrence of atrial fibrillation

	No recurrence (n = 313)	Recurrence (n = 65)	p value
Age, years	58.7±10.8	59.8±10.7	0.443
Male	223 (71)	52 (80)	0.14
Type of AF			**0.006**
Persistent	103 (32.8)	33 (50.8)	
Paroxysmal	211 (67.2)	32 (49.2)	
Hypertension	156 (49.7)	33 (50.8)	0.873
DM	46 (14.6)	11 (16.9)	0.641
Prior stoke or TIA	49 (15.6)	10 (15.4)	0.964
CHA2DS2-VASc	1.8±1.5	1.7±1.4	0.817
Coronary artery disease	49 (15.6)	8 (12.3)	0.498
Valvular heart disease	25 (8)	6 (9.2)	0.734
Systolic blood pressure, mm Hg	119.2±14.8	118.4±9.3	0.772
Diastolic blood pressure, mm Hg	74.7±10.8	75.5±7.7	0.743
LA dimension, mm	41.6±6	43.2±5.8	**0.046**
LA volume index, mL/m^2	32.3±15.1	34.3±15.5	0.323
LVEF, %	63.1±7.9	62.4±7.9	0.512
E/E'	10.6±4.4	10.4±4.8	0.812
LV mass index, g/m^2	72±43.8	72.4±41.4	0.95
LVEDD, mm	49.5±4.6	50.1±4.4	0.334
LVESD, mm	33.5±6	34±4.6	0.482
Height, cm	166.4±8.8	163.9±22.2	0.132
Weight, kg	69.6±11.3	69.7±11.8	0.951
BMI	25.1±3	25±3.1	0.965
Neck diameter, cm	37.2±3.1	37.4±2.6	0.651
Waist circumference, cm	1±0.7	1±0.5	0.576
hsCRP, mg/dL	2.5±6.7	1.6±1.8	0.052
AHI, apnea + hypopnea/h	15.0±16.0	13.6±12.6	0.508
ESS	6.6±4.2	7.2±4.4	0.508
SQI	5.1±3.2	5.3±2.6	0.348
Test time, min	420.6±92.6	427±79.7	0.602
Mean duration of OSA, s	16.8±9.8	15.9±10.9	0.520
Mean duration of hypopnea, s	21.9±6.5	22.1±6.7	0.827
Minimum oxygen saturation, %	86.6±5.1	88.2±4	**0.01**
Desaturation index	13.2±14.7	12.1±10.8	0.568
Early recurrence	81 (25.8)	33 (50.8)	**<0.001**
Antiarrhythmic drug after procedure	28 (8.9)	13 (20.3)	**<0.001**

p values <0.05 are marked in bold. Data are presented as n (%) or mean ± SD. Abbreviations are explained in Table 1.

Table 3 shows the results of the Cox regression analysis for risk factors of the recurrence of AF. In the univariate model, LA diameter, persistent AF, and early recurrence were significantly related to the clinical recurrence of AF; however, the severity of OSA was not associated with clinical recurrence. Multivariate Cox regression analysis revealed that only early recurrence was a significant risk factor for clinical recurrence after adjustment for other confounding factors. However, the severity of OSA was not a predictor of clinical recurrence (Table 3; Fig. 1).

Table 3. Univariate and multivariate Cox regression analyses for the recurrence of atrial fibrillation

Predictor	Univariate		Multivariate	
	hazard ratio	*p* value	hazard ratio	*p* value
Age	1.01 (0.98–1.03)	0.541	1.00 (0.98–1.03)	0.722
Male gender	1.53 (0.83–2.8)	0.173	1.50 (0.8–2.84)	0.210
LA diameter	1.04 (1–1.09)	**0.037**	1.02 (0.97–1.07)	0.445
LVEF	0.99 (0.96–1.02)	0.361	0.99 (0.96–1.02)	0.448
Hypertension	1.03 (0.63–1.68)	0.899	0.95 (0.55–1.64)	0.865
DM	1.13 (0.59–2.15)	0.722	1.02 (0.5–2.07)	0.962
BMI	1.00 (0.92–1.09)	0.962	1.00 (0.91–1.1)	0.920
Persistent AF	1.85 (1.14-3.01)	**0.013**	1.39 (0.79–2.43)	0.254
Early recurrence	2.71 (1.66–4.41)	**<0.001**	2.63 (1.59–4.37)	**<0.001**
ESS	1.04 (0.98–1.1)	0.232	NI	NI
SQI	1.02 (0.95–1.1)	0.585	NI	NI
AHI	1.00 (0.98–1.01)	0.739	NI	NI
Mean duration of OSA	0.99 (0.97–1.02)	0.553	NI	NI
Mean duration of hypopnea	1.00 (0.96–1.04)	0.924	NI	NI
Desaturation index	1.00 (0.98–1.02)	0.815	NI	NI
Mild OSA	–	–	–	–
Moderate OSA	1.39 (0.83–2.33)	0.216	1.08 (0.61–1.91)	0.785
Severe OSA	0.83 (0.36–1.88)	0.649	0.73 (0.31–1.73)	0.477

p values <0.05 are marked in bold. Abbreviations are explained in Table 1. NI, not included.

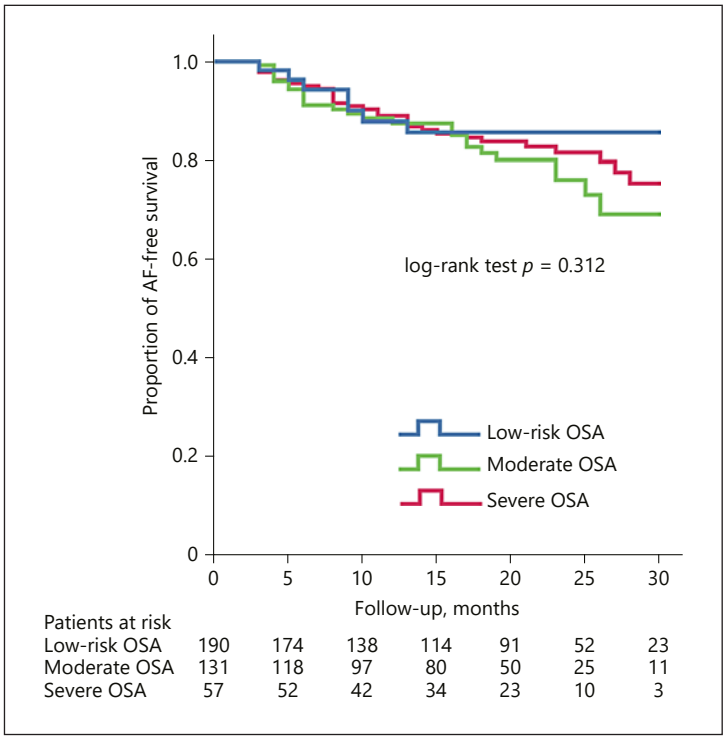

Fig. 1. Kaplan-Meier curve for AF-free survival after catheter ablation according to the severity of OSA.

Discussion

The findings of this study are as follows: (1) OSA is closely related with cardiovascular disease and/or risk factors such as age, DM, and abdominal obesity among the patients with AF; (2) however, the severity and the existence of OSA are not predictors of the recurrence of AF after ablation; (3) after adjustment of possible confounding factors, early recurrence of AF is the only predictor of the clinical success of ablation for the treatment of AF.

Hypertension is well known to be related with sleep apnea [12]. According to previous research, it appears that 50% of patients with OSA are hypertensive and 30% of hypertensive patients have OSA [13, 14]. Furthermore, patients with moderate or severe OSA have a 3-fold elevated risk of CAD compared to patients without OSA, and it has been reported that CPAP reduces the risk of CAD in patients with OSA [15, 16]. Our data are partially in agreement with these previous studies. The proportion of DM and hypertension in patients with severe OSA is nearly 2-fold compared to those in patients with mild OSA. Moreover, the prevalence of CAD is higher with increased severity of OSA. Interestingly, logistic regression analysis showed that severe OSA was no longer significantly associated with CAD after adjustment for both DM and hypertension, which are major risk factors for CAD. This is probably because severe OSA patients were more likely to have DM or hypertension. Therefore, we conclude that, when studying OSA, the interplay between OSA and other clinical factors should be considered carefully.

The close relationship between OSA and AF has been widely studied and supported for a long time [4–6, 17]. However, our data demonstrate that the severity of OSA cannot be a predictor of the recurrence of AF after catheter ablation. There are various possible reasons for the discrepancy between the results of our study and other studies. One reason may be the different amounts of energy used for AF ablation, leading to different ablation efficacies. The more energy gets delivered to the tissue, the better the outcome of the treatment [18]. According to our study protocol, we used 35 W, which is a relatively high power. Another possibility for the clinical recurrence is that the LA diameter, which is the major substrate for development of AF, did not differ depending on the severity of OSA. The recurrence of AF after catheter ablation may occur because the electrical activity of AF was not blocked sufficiently by the procedure. The probability of recurrence was significantly higher in patients with larger LA size [19]. Furthermore, our study was conducted in the Korean population which has lower BMI even at a similar rate of hypertension, DM, and dyslipidemia compared to the western countries [20]. Our study population had lower BMI compared to the study by Fein et al. [5] and by Matiello et al. [4], which included patients with severe OSA but relatively low BMI. These demographic differences may be an important factor for the contrast between our results and other studies supporting the relationship between OSA and the recurrence of AF.

Nevertheless, there have been studies indicating that OSA is not related to AF. Mehra et al. [21] showed that OSA was closely associated with ventricular ectopy but not AF after adjustment of confounding factors, although they did not evaluate the recurrence of AF. Our study also adjusted for other cardiovascular risk factors coexisting with OSA, and the severity of OSA was not related to clinical recurrence. Chilukuri et al. [22] showed that obesity, rather than OSA, is an independent predictor of procedural failure after catheter ablation of AF. In addition, according to Tang et al. [23], the severity of OSA is not associated with the recurrence of AF. However, in those studies, OSA was diagnosed using the Berlin questionnaire, which is a major limitation. Our study used portable polysomnography, which has proven to be an effective tool for the diagnosis of breathing disorders during sleep [24].

The present study has some limitations. First, because this is an observational study, the heterogeneity of the individual OSA groups might be problematic. Second, the results may not be similar to those of other studies due to ethnic differences. Third, portable polysomnography

was used for evaluating the severity of OSA, and it was performed only one time at enrollment. Therefore, the diagnosis and classification of OSA may lack accuracy to some degree. This is because portable polysomnography is likely to overestimate the severity of OSA in patients with a mild sleep disorder and to underestimate AHI in those with more severe OSA, compared to in-lab polysomnography [25, 26]. Fourth, variables not included in this study might serve as confounding factors. For example, the duration of AF is known to be an independent predictor of maintenance of sinus rhythm [27], but was not included in this analysis.

In conclusion, this study shows that the severity of OSA is closely related with well-known cardiovascular risk factors and CAD, but it is not a predictor of the recurrence of AF after catheter ablation in Korean patients. Therefore, the relationship between OSA and AF should be further investigated under well-defined conditions. Treatment of OSA for the sole purpose of lowering AF recurrence may need to be reconsidered.

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Disclosure Statement

The authors have nothing to declare.

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