Relationship of Systolic BP to Obstructive Sleep Apnea in Patients With Heart Failure*

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Study objectives: Obstructive sleep apnea (OSA) is an independent risk factor for hypertension in the general population. Hypertension is, in turn, an important risk factor for the development and progression of congestive heart failure (CHF). Our objective was to determine whether OSA would be associated with elevated daytime BP in medically treated patients with CHF.

Design: Cross-sectional study.

Setting: Tertiary care, university-affiliated sleep disorders and heart failure clinics.

Patients: Three hundred one consecutive patients with CHF.

Measurements and results: We measured daytime BP and performed overnight sleep studies to assess for the presence of OSA. Among these patients, OSA was present in 121 patients (40%) and their systolic BP was significantly higher than in patients without OSA. Patients with OSA were 2.89 times (95% confidence interval, 1.25 to 6.73) more likely to have systolic hypertension (ie, BP ≥ 140 mm Hg) than those without OSA after controlling for other risk factors, including obesity. The degree of systolic BP elevation was directly related to the frequency of obstructive apneas and hypopneas.

Conclusions: In medically treated patients with CHF, daytime systolic BP and the prevalence of systolic hypertension are significantly increased in patients with OSA, compared to those without OSA, independent of other potentially confounding factors. OSA may therefore have contributed to the presence of systolic hypertension in some of these patients.

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Hypertension is the most common risk factor for heart failure. It has been demonstrated that exposure of dogs to OSA over several weeks leads not only to the development of nocturnal and daytime hypertension, but also to left ventricular hypertrophy and dysfunction as well, therefore, obstructive apnea-associated elevations in nocturnal and daytime BP could play a role in the development or progression of myocardial hypertrophy and failure. Moreover, once CHF has developed, the detrimental effects of elevated BP are likely to be amplified, since the failing heart is particularly sensitive to the adverse effects of increased left ventricular afterload. Another major concern for patients with CHF is that the pressor effects of OSA are relatively resistant to conventional antihypertensive agents. For example, we have shown that OSA raises nocturnal BP in patients with heart failure, despite treatment with drugs with hypotensive actions; however, it is not yet known whether OSA is associated with daytime hypertension in medically treated patients with CHF. We therefore hypothesized that daytime BP and the prevalence of hypertension would be higher in patients with CHF than in those without OSA, independent of known confounding factors.

Materials and Methods

Study Population

Subjects consisted of 301 consecutive patients with CHF referred to the Sleep Research Laboratory who met our inclusion criteria (see below). Patients were all referred by cardiologists because of clinical suspicion of sleep apnea or refractory symptoms of heart failure despite optimal medical therapy. None were referred because of hypertension. Inclusion criteria were patients with all of the following: (1) diagnosis of CHF of at least 6 months in duration established by a cardiologist on the basis of at least one episode of pulmonary edema and dyspnea, (2) a resting left ventricular ejection fraction (LVEF) of ≤ 40% measured by equilibrium radionuclide angiography or two-dimensional echocardiography within 3 months prior to a diagnostic sleep study, (3) continued dyspnea (New York Heart Association class 2 to 4) despite appropriate medical therapy, and (4) stable clinical status with no medication adjustment for at least 4 weeks prior to polysomnography. Patients with unstable angina or myocardial infarction within 3 months of the study were excluded.

Clinical Data and Outcomes

At the initial examination in the sleep disorders clinic, prior to performance of sleep studies, seated systolic and diastolic BP were measured between 1:30 PM and 4:30 PM after a 15-min rest period using a mercury-column sphygmomanometer and appropriate-sized arm cuff by a single physician within 4 weeks prior to the sleep study. BP recordings from this initial clinic visit were used for analysis. Drug treatment and body mass index (BMI) were also documented during this clinic visit.

Sleep Studies

Following the clinic assessment, overnight polysomnography and sleep staging were performed and scored, without knowledge of patients’ BP, using standard techniques. Thoracicabdominal movements and tidal volume were measured by inductance plethysmograph (Respiritrace; Ambulatory Monitoring; Ardsley, NY). Oxyhemoglobin saturation (SaO₂) was measured continuously using a pulse oximeter (Oxyshuttle; SensorMedics; Yorba Linda, CA). Transcutaneous PCO₂ was continuously measured with a transcutaneous capnometer (Kontron Medical; Hoffman-La Roche; Basel, Switzerland). Oxygen saturation during sleep was expressed as follows: (1) mean SaO₂, which represents the mean of the highest and lowest SaO₂ for each 30-s epoch of sleep averaged over the entire sleep period; and (2) lowest SaO₂, which represents the lowest SaO₂ recorded over the entire sleep period. Mean transcutaneous PCO₂ during sleep was calculated as above for mean SaO₂.

Obstructive apneas were defined as the absence of tidal volume excursion for at least 10 s accompanied by paradoxical rib cage and abdominal movements. Obstructive hypopneas were defined as a ≥ 50% reduction in tidal volume from the baseline value, with paradoxical chest wall motion or evidence of flow limitation, persisting for at least 10 s. Central apneas and hypopneas were similarly defined, except that apneas were not accompanied by any rib cage or abdominal movements and there was no paradoxical motion of rib cage and abdomen or evidence of flow limitation during hypopneas. An OSA disorder was defined as the presence of ≥ 10 apneas and hypopneas per hour of sleep, of which ≥ 50% had to be obstructive. A central sleep apnea (CSA) disorder was similarly defined, except that > 50% of the events had to be central. The apnea-hypopnea index (AHI) was defined as the number of apneas and hypopneas per hour of sleep. Patients with an AHI < 10 per hour of sleep were categorized as having no sleep-related breathing disorder (SBD).

Data Analysis

Potential associations between sleep apnea and individual risk factors were assessed for significance with a χ² test for binary variables and one-way analysis of variance with the Tukey post-hoc analysis for continuous variables. To determine whether systolic BP was related to the severity of OSA, linear regression techniques were also applied to the data containing those with OSA and those without any SBD. Systolic and diastolic BP served as the dependent variables and AHI as the primary independent variable. Other covariates were added in a stepwise manner to the crude model. Covariates were included in the final model if the terms changed the risk estimate of the primary independent variable by > 15% or conferred a significant change in the log-likelihood statistic of the reference model. In the final model, we controlled for the effects of obesity, age, gender, and gender. Class of medication had no significant effect on the relationship between BP and severity of OSA and, therefore, was not included in the final model.

In addition, multivariate logistic modeling was used to determine risk factors for hypertension defined as systolic BP ≥ 140 mm Hg or diastolic BP ≥ 90 mm Hg. The reference and comparison groups for the baseline model were patients without any SBD and those with OSA, respectively. Other covariates were added in a stepwise manner to the crude model as described above. Relative odds ratios and 95% confidence intervals (CIs) were constructed using standard methods. Data are presented as mean ± SEM, unless otherwise indicated. Two-tailed p values < 0.05 were considered statistically significant. Standard regression diagnostics were performed to assess model fit and adequacy.
of compliance with the modeling assumptions. All analyses were conducted using SAS, release 6.10 (SAS Institute; Cary, NC).

Results

The causes of CHF were ischemic cardiomyopathy (including hypertensive heart disease) [63%], nonischemic dilated cardiomyopathy (25%), and others (12%), which included valvular and congenital heart disease, and alcoholic cardiomyopathy. Patients with OSA had higher systolic BP (Fig 1), greater BMI, and were more likely to use calcium-channel blockers and angiotensin-converting enzyme (ACE) inhibitors compared to those with CSA or no SBD (Table 1). Patients with OSA also had higher diastolic BP and LVEFs compared to those with CSA; however, these values were not significantly different from those in patients with no SBD (Table 1, Fig 1). In contrast, patients with CSA were older, slimmer, and more likely to be men, but less likely to be receiving β-blockers than those in the other two groups. Patients with OSA had more severe degrees of nocturnal hypoxia than either the no-SBD group or CSA group, as indicated by significantly lower mean and minimum SaO₂ during sleep (Table 2). Patients with CSA had significantly lower mean transcutaneous Pco₂ during sleep than the other two groups.

A logistic regression model was constructed to evaluate the potential relationship between SBDs and the presence of systolic BP ≥ 140 mm Hg or diastolic BP ≥ 90 mm Hg. Systolic BP ≥ 140 mm Hg was found in 13.5% of patients without a SBD, 21.1% of those with CSA, and 35.9% of patients with OSA. Diastolic BP ≥ 90 mm Hg was observed in 13.5% of patients without a SBD, 8.7% of patients with CSA, and 23.1% of patients with OSA. The final multiple logistic regression model, which controlled for BMI (and thus for obesity), age, gender, mean and minimum SaO₂, and LVEF, demonstrated that the presence of OSA was associated with a 2.89 relative odds of having systolic BP ≥ 140 mm Hg compared to patients without any SBD (Table 3). In contrast, there was no significant association between OSA and diastolic BP ≥ 90 mm Hg. There was no significant relationship between arousal index and either systolic or diastolic BP. Age and BMI were also significant determinants of systolic BP ≥ 140 mm Hg, but only BMI was a significant determinant of diastolic BP ≥ 90 mm Hg (Table 3).

Unadjusted relationships between various factors and systolic and diastolic BP are displayed in Table 4. When the potential confounders of obesity (as assessed by BMI), age, gender, mean and minimum SaO₂, and LVEF were controlled for, there was a significant relationship between obstructive AHI and systolic BP (Fig 2). The regression coefficient for obstructive AHI was 0.23, indicating that for every 10-U rise in obstructive AHI, systolic BP increased by 2.3 mm Hg (95% CI, 1.6 to 3.1 mm Hg). Other significant independent factors associated with increments in systolic BP were increasing age, BMI, and LVEF. The β-coefficients for a 1-year increment in age, 1 kg/m² increase in BMI, and 1% increase in LVEF were 0.4, 0.5, and 0.3, respectively. These data indicate that a 10-U increase in obstructive AHI was associated with an increase in systolic BP equivalent to that associated with a 6.3-year increase in age or 4.5 kg/m² increase in BMI. In contrast, there was no significant relationship between AHI and diastolic BP. Among patients with CSA, there was no significant relationship between AHI and either systolic or diastolic BP.

To eliminate any potential confounding influence of obesity on BP, we performed a multivariate linear regression analysis, using the same covariates as for the entire cohort, on the subgroup of non obese patients with OSA (BMI of 22 to 27). The BMI in this subgroup of OSA patients (mean ± SD, 24.8 ± 0.3, n = 24) did not differ significantly from that of the patients without any SBD (24.6 ± 0.3, n = 28). In this non obese sub-

![Figure 1](http://publications.chestnet.org/pdfaccess.ashx?url=data/journals/chest/21993/)

Figure 1. Systolic and diastolic BP stratified according to sleep apnea status. *p < 0.05 compared to both other groups; †p < 0.05 compared to CSA group only.
group, the adjusted regression coefficient for obstructive AHI was 0.29, which indicated that for every 10-U rise in AHI, systolic BP increased by 2.9 mm Hg (95% CI, 0.0 to 6.1 mm Hg).

**DISCUSSION**

The present study is the first to demonstrate, in patients with CHF, that OSA is associated with elevated daytime systolic BP. After controlling for potential confounding factors, including age, sex, and obesity, we found that among 301 medically treated patients with CHF, OSA was associated with increased odds for systolic BP ≥ 140 mm Hg 2.89 times that of patients without any SBD. In addition, when only nonobese patients with OSA were included, the odds for daytime systolic hypertension were even greater: 7.34 times that of nonobese patients without any SBD. Furthermore, there was a direct linear relationship between the severity of OSA and systolic BP: for the entire group, every 10-U increment in the obstructive AHI was accompanied by a 2.3-mm Hg increase in systolic BP, and for nonobese patients, by a 2.9-mm Hg increase.

The independent relationship between OSA and systolic hypertension held even though a higher proportion of the patients with OSA were receiving ACE inhibitors and calcium-channel blockers than in patients without OSA. If these medications had a significant impact on systolic BP, then the strength of the relationship between OSA and elevated systolic BP may have been underestimated. This observation also suggests that BP control with antihypertensive medications may be more difficult to achieve in CHF patients with OSA than in those without it. This concept is consistent with a study in which the great majority of patients with drug-resistant hypertension were found to have OSA. In addition, Tkacova et al showed that CHF patients with OSA experience recurrent apnea-related surges in systolic BP during sleep, despite receiving various combinations of drugs with hypotensive effects. Elimination of OSA by CPAP abolished these apnea-related surges in BP.

There is now strong epidemiologic evidence that OSA is a cause of hypertension, which is the most common risk factor for heart failure. In addition, exposure of dogs to experimental OSA induces sustained daytime hypertension and left ventricular dysfunction. Moreover, epidemiologic data indicate that the presence of OSA is associated with increased odds of having CHF. It is therefore likely that in many of our patients with CHF, OSA, and hypertension preceded the onset of their cardiac failure, although this information was not available to us. Because patients with hypertension who acquire

### Table 1—Characteristics of the Subjects*

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>No SBD</th>
<th>CSA</th>
<th>OSA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients, No. (%)</td>
<td>75 (25)</td>
<td>105 (35)</td>
<td>121 (40)</td>
</tr>
<tr>
<td>Age, yr</td>
<td>56.9 ± 1.6</td>
<td>67.2 ± 0.9†‡</td>
<td>59.4 ± 1.1</td>
</tr>
<tr>
<td>Male gender, %</td>
<td>82.7</td>
<td>93.3‡</td>
<td>86.0</td>
</tr>
<tr>
<td>BMI, mg/kg</td>
<td>28.4 ± 0.7</td>
<td>26.1 ± 0.4‡</td>
<td>32.7 ± 0.2‡</td>
</tr>
<tr>
<td>LVEF, %</td>
<td>26.0 ± 1.7</td>
<td>23.6 ± 1.5</td>
<td>30.4 ± 1.5‡</td>
</tr>
<tr>
<td>Ischemic cardiomyopathy, %</td>
<td>55.4</td>
<td>74.8‡</td>
<td>60.3</td>
</tr>
<tr>
<td>Nonischemic cardiomyopathy, %</td>
<td>32.0</td>
<td>17.1‡</td>
<td>27.3</td>
</tr>
<tr>
<td>ACE inhibitors, %</td>
<td>78.7</td>
<td>73.3</td>
<td>85.8‡</td>
</tr>
<tr>
<td>Digitalis, %</td>
<td>73.3</td>
<td>68.6</td>
<td>64.2</td>
</tr>
<tr>
<td>Calcium-channel blocker, %</td>
<td>13.3</td>
<td>15.4‡</td>
<td>23.3‡</td>
</tr>
<tr>
<td>ß-Blockers, %</td>
<td>21.3</td>
<td>11.5</td>
<td>22.7</td>
</tr>
<tr>
<td>Diuretics, %</td>
<td>82.3</td>
<td>82.9</td>
<td>79.1</td>
</tr>
<tr>
<td>History of snoring, %</td>
<td>49.2</td>
<td>41.1</td>
<td>79.11‡</td>
</tr>
</tbody>
</table>

*Data are presented as mean ± SEM unless otherwise indicated.
†Significantly different from the OSA group (p < 0.05).
‡Significantly different from CSA group (p < 0.05).
§Significantly different from no-SBD group (p < 0.05).

### Table 2—Sleep Data*

<table>
<thead>
<tr>
<th>Variables</th>
<th>No SBD</th>
<th>CSA</th>
<th>OSA</th>
</tr>
</thead>
<tbody>
<tr>
<td>AHI, No./h</td>
<td>4.2 ± 0.3</td>
<td>40.3 ± 2.0†</td>
<td>34.1 ± 1.9†</td>
</tr>
<tr>
<td>Arousal index, arousals/h of sleep</td>
<td>18.3 ± 3.0</td>
<td>37.8 ± 3.1†</td>
<td>37.8 ± 2.3†</td>
</tr>
<tr>
<td>Mean transcutaneous PCO2 during sleep, mm Hg</td>
<td>43.4 ± 5.8†</td>
<td>38.2 ± 6.8</td>
<td>44.7 ± 6.7†</td>
</tr>
<tr>
<td>Mean Sao2 during sleep, %</td>
<td>93.5 ± 2.6</td>
<td>93.3 ± 1.9</td>
<td>92.5 ± 3.2‡</td>
</tr>
<tr>
<td>Lowest Sao2 during sleep, %</td>
<td>86.1 ± 7.1</td>
<td>84.1 ± 5.6</td>
<td>78.5 ± 9.7†</td>
</tr>
</tbody>
</table>

*Data are presented as mean ± SEM.
†Significantly different from CSA group (p < 0.05).
‡Significantly different from no-SBD group (p < 0.05).
CHF usually do so secondary to ischemic heart disease, such patients would have been classified under ischemic cardiomyopathy in Table 1. Once the myocardium begins to fail, persistent elevations in systemic BP are associated with accelerated deterioration in cardiac function.\textsuperscript{10,11} Aggressive control of BP is therefore recommended as an important component of heart failure management.\textsuperscript{18} Even so, many of the CHF patients with OSA in our study had elevated daytime systolic BP despite receiving multiple antihypertensive medications. Our data therefore raise the possibility that OSA, through hypertension, could both predispose to CHF and contribute to its progression.\textsuperscript{9,10}

Cardiovascular risk is greater for systolic than for diastolic hypertension, probably because of its greater impact on left ventricular wall tension. Accordingly, the critical importance of controlling systolic hypertension, especially in patients with cardiovascular diseases, such as CHF, has recently been emphasized.\textsuperscript{19} Moreover, OSA prevents the normal fall in systolic BP at the onset of sleep.\textsuperscript{13,20,21} Thus, for a given level of daytime BP, the systolic BP burden over the full 24-h period is likely to be greater in those patients with than in those without OSA.\textsuperscript{20,21} It might therefore be particularly important to initiate specific therapy for OSA in such patients, because of the potential to lower both nocturnal and daytime BP.\textsuperscript{6,13} In fact, we have previously shown that abolition of OSA by CPAP in patients with CHF causes remarkable improvements in both LVEF and symptoms of heart failure.\textsuperscript{22} These beneficial effects were likely due, in part, to lowering of BP and afterload.\textsuperscript{13}

Systolic, but not diastolic BP was elevated in our patients with OSA. These findings are consistent with those of a study of patients without CHF. In that study, Logan and associates\textsuperscript{12} found that among patients with hypertension refractory to medical therapy, those with OSA had predominantly systolic hypertension, while those without any SBD had predominantly diastolic hypertension. The reason for this is not clear, but a possible explanation is a somewhat different pathophysiology of systolic and diastolic hypertension.\textsuperscript{23}

Mechanisms by which OSA could raise daytime systolic BP are not completely understood. However, intermittent apnea-related hypoxia seems to play an important role in the causation of acute nocturnal BP elevations, and subsequent development of daytime hypertension.\textsuperscript{8,24} These events increase BP acutely by raising sympathetic nervous system activity.\textsuperscript{3} In theory, they could chronically raise BP by resetting the baroreflex or by up-regulating chemoreflex control of sympathetic vasocostractor discharge.\textsuperscript{25–27} For example, exposure of humans to intermittent hypoxia leads to an increase in sympathetic vasocostractor activity that lasts for at least 20 min after return to normoxia.\textsuperscript{28} In addition, overnight exposure of humans to hypoxia leads to a sustained increase in BP that carries over into wakefulness.\textsuperscript{29} and rats subjected to intermittent hypoxia for 8 h/d acquire sustained hypertension.\textsuperscript{24} Moreover, carbon dioxide also stimulates sympathetic vasocostractor activity that is additive to the effects of hypoxia.\textsuperscript{30} In this regard, patients with OSA had more severe nocturnal hypoxia than either the no-SBD group or the CSA group (Table 2). They also had higher Pco\textsubscript{2}.

### Table 3—Adjusted Risk Factors for Systolic and Diastolic Hypertension

<table>
<thead>
<tr>
<th>Variables</th>
<th>Systolic BP ≥ 140 mm Hg</th>
<th>Diastolic BP ≥ 90 mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Odds Ratio</td>
<td>95% CI</td>
</tr>
<tr>
<td>OSA†</td>
<td>2.89</td>
<td>1.23–6.73</td>
</tr>
<tr>
<td>BMI‡</td>
<td>1.09</td>
<td>1.04–1.15</td>
</tr>
<tr>
<td>Age, yr</td>
<td>1.05</td>
<td>1.02–1.09</td>
</tr>
<tr>
<td>Male gender§</td>
<td>0.40</td>
<td>0.18–1.09</td>
</tr>
</tbody>
</table>

*Odds ratios are adjusted simultaneously for all the variables on the list.
†Compared to those without any SBD.
‡Reflects 1-U increase.
§Compared to women.

### Table 4—Unadjusted Relationship Between Various Factors and Systolic or Diastolic BP

<table>
<thead>
<tr>
<th>Variables</th>
<th>Systolic BP</th>
<th>Diastolic BP</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>β-Coefficient</td>
<td>95% CI</td>
</tr>
<tr>
<td>BMI*</td>
<td>0.95</td>
<td>0.58 to 1.33</td>
</tr>
<tr>
<td>Age, yr*</td>
<td>0.32</td>
<td>0.12 to 0.53</td>
</tr>
<tr>
<td>LVEF, %*</td>
<td>0.53</td>
<td>0.36 to 0.69</td>
</tr>
<tr>
<td>Male gender†</td>
<td>10.70</td>
<td>10.42 to 11.00</td>
</tr>
<tr>
<td>Mean Low SaO₂ during sleep, %*</td>
<td>0.93</td>
<td>0.24 to 1.61</td>
</tr>
<tr>
<td>Obstructive AHI, No./h*</td>
<td>0.17</td>
<td>0.06 to 0.28</td>
</tr>
</tbody>
</table>

*Reflects 1-U increase.
†Compared to women.
during sleep than patients with CSA. Greater degrees of vasoconstrictor chemostimulation may therefore have contributed to the higher systolic BP in patients with OSA than in those with CSA, despite similar AHI. There were slight differences in medication use among the three groups. However, data from Kraiczi et al indicate that a wide variety of antihypertensive medications have no effect on the severity of OSA. It is therefore very unlikely that greater antihypertensive medication use in the OSA group induced OSA in our study subjects.

Only a minority of patients in our study were receiving β-blockers. This is because the results of large randomized trials demonstrating the usefulness of β-blockers in the therapy of CHF only became available after our study was completed. Moreover, it usually takes several years before clinical trial results are implemented in community medical practice. It is possible that increased use of β-blockers could provide additional BP-lowering effects in CHF patients with OSA. However, even after controlling for medication use, including β-blockers, the relationship between OSA and elevated BP held.

Another interesting observation in our study was that patients with CSA did not have elevations in either daytime systolic or diastolic BP compared to patients without any SBD or those with OSA. This may be because of lesser degrees of nocturnal chemostimulation than patients with OSA, as discussed above. Another possibility is that differences in BP may be related to the differing pathophysiologies of OSA and CSA. Whereas OSA likely contributes to the development of CHF, partly through hypertension, CSA appears to be a consequence of CHF, and may therefore not have the same effects on daytime BP as does OSA. Moreover, because of more severe left ventricular systolic dysfunction, patients with CSA (Table 1) may have had insufficient contractile force to generate as high BP as patients with OSA. However, LVEF in patients with OSA did not differ significantly from that in patients with no SBD. In addition, after controlling for differences in LVEF, multiple regression analysis indicated OSA was independently associated with systolic hypertension (Table 3, Fig 2).
In view of the poor prognostic implications of uncontrolled BP in patients with CHF, and of the clinical benefits of lowering BP in such patients, it is important to identify potentially reversible causes of hypertension.\textsuperscript{10,11} Our data suggest that, in patients with CHF, OSA should be considered as a possible contributing factor to drug-resistant hypertension.\textsuperscript{12}

Indeed, recent evidence suggests that treatment of OSA by CPAP can lower both nocturnal\textsuperscript{13} and daytime BP.\textsuperscript{6} More importantly, there is mounting evidence that treatment of OSA by CPAP in patients with CHF improves left ventricular function and symptoms of CHF beyond that due to standard medical therapy.\textsuperscript{22,33,34} Consequently, it seems very likely that OSA is contributing to the development or progression of CHF in a significant proportion of patients, partially through the intermediary step of hypertension. Therefore, larger trials are now required to determine whether the treatment of OSA in medically treated patients with CHF reduces BP and improves cardiac function.

\textbf{REFERENCES}


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