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Background and Purpose—The goals of this study were to compare the ability of statewide and institutional models of stroke risk after coronary artery bypass (CAB) to predict institution-specific results and to examine the potential additive stroke risk of combined CAB and carotid endarterectomy (CEA) with these predictive models.

Methods—An institution-specific model of stroke risk after CAB was developed from 1975 consecutive patients who underwent nonemergent CAB from 1994 to 1999 in whom severe carotid stenosis was excluded by preoperative duplex screening. Variables recorded in the New York State Cardiac Surgery Program database were analyzed. This model (model I) was compared with a published model (model II) derived from analysis of the same variables using New York statewide data from 1995. Predicted and observed stroke risks were compared. These formulas were applied to 154 consecutive combined CAB/CEA patients operated on between 1994 and 1999 to determine the predicted stroke risk from CAB alone and thereby deduce the maximal added risk imputed to CEA.

Results—Risk factors common to both models included age, peripheral vascular disease, cardiopulmonary bypass time, and calcified aorta. Additional risk factors in model I also included left ventricular hypertrophy and hypertension. Risk factors exclusive to model II included diabetes, renal failure, smoking, and prior cerebrovascular disease. Our observed stroke rate for isolated CAB was 1.7% compared with a rate predicted with model II (statewide data) of 1.56%. The observed stroke rate for combined CEA/CAB was 3.9%. When the Stony Brook model (model I) based on patients without carotid stenosis was used, the predicted stroke rate was 2.8%. When the statewide model (model II), which included some patients with extracranial vascular disease, was used, the predicted stroke rate was 3.4%. The differences between observed and predicted stroke rates were not statistically significant.

Conclusions—Estimation of stroke risk after CAB was similar whether statewide data or institution-specific data were used. The statewide model was applicable to institution-specific data collected over several years. Common risk factors included age, aortic calcification, and peripheral vascular disease. The observed differences in the predicted stroke rates between models I and II may be due to the fact that carotid stenosis was specifically excluded by duplex ultrasound from the patient population used to develop model I. Modeling stroke risk after CAB is possible. When these models were applied to patients undergoing combined CAB/CEA, no additional stroke risk could be ascribed to the addition of CEA. Such models may be used to identify groups at increased risk for stroke after both CAB and combined CAB/CEA. The ultimate place for CEA in patients undergoing CAB will be defined by prospective randomized trials. **(*Stroke*. 2003; 34:1212-1217.)**

Key Words: bypass surgery ■ carotid endarterectomy ■ stroke

Rates of stroke after coronary artery bypass (CAB) surgery have varied widely in the literature. In a review by Gardner et al,[1] stroke rates ranged from 0.8% to 7%. The variation in reported stroke rates among different series may reflect differences in patient demographics, selection bias, or definitions of stroke. It is clear, however, that postoperative strokes contribute significantly to increased perioperative mortality, costs, and lengths of stay.[2,3]

In an attempt to identify patients at high risk for postoperative stroke, models of stroke risk after CAB based on demographics and perioperative variables have been proposed. John et al[4] developed a multivariable logistic regres-

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The Appendix is available online at http://stroke.ahajournals.org.

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sion equation incorporating preoperative and intraoperative factors to predict stroke based on 19 244 patients who underwent CAB in New York State during 1995. The purposes of the present study were (1) to create our own institutional model to predict postoperative stroke after CAB based on preoperative variables; (2) to compare the previously published statewide model and our institutional model and to test the ability of the published model to predict stroke after CAB in a specific institution over time; and (3) to apply both models to our series of consecutive combined CAB and carotid endarterectomy (CEA) patients to assess whether the addition of CEA to CAB affected the postoperative stroke rate in our institution.

Patients and Methods

Patient Population

From 1994 to 1997, we adopted a uniform policy of carotid duplex screening of all CAB patients before CAB. The only patients not screened (n=12) were those who went to surgery immediately from the cardiac catheterization laboratory for hemodynamic instability. During this time period, all patients with 80% to 99% carotid stenosis by duplex ultrasound underwent digital subtraction or MR angiography to confirm the ultrasound findings. Patients with confirmed carotid bifurcation stenosis >80% were subjected to combined CAB/CEA. Patients requiring concomitant noncoronary surgery (eg, valve surgery, ventricular aneurysmectomy) were excluded. This resulted in a homogeneous population of patients subjected to isolated CAB in whom carotid stenosis >80% was excluded preoperatively by ultrasound screening. These 1975 patients were prospectively entered into the New York State Cardiac Database, which contained 35 variables (see the Appendix, which can be found online at http://stroke.ahajournals.org). This population of patients was used to construct our institutional model for stroke after isolated CAB. Our evaluation protocol resulted in a second patient population consisting of 154 consecutive patients who underwent combined CAB/CEA for carotid stenosis >80%. This concurrent population, in whom the same risk factors were prospectively collected, was evaluated for postoperative stroke risk with our institution-specific model and the model developed by John et al.[4]

Postoperative Stroke

Postoperative stroke was defined as any new focal neurological deficit occurring after surgery but before hospital discharge. The possibility of postoperative stroke was identified by the medical or surgical team (cardiologist, surgeon) caring for the patient and confirmed by an independent neurological evaluation. Prospective preoperative and postoperative neurological evaluation was not performed in this study group. Neuropsychiatric testing was not performed in this cohort. The diagnosis of stroke was confirmed with brain imaging studies; however, routine postoperative brain imaging was not performed in these patients.

Risk Factors

Demographics and risk factors were identified from our cardiac surgical database, which conforms to the New York State Cardiac Surgical Database maintained by the New York State Department of Health, Bureau of Hospital and Primary Care Services–Cardiac Services Program. A total of 35 demographic and preoperative variables were evaluated (see the Appendix). These variables encompassed patient demographics, comorbid medical conditions, perioperative events, and anatomic variables. These were the same variables used by John et al[4] to develop their stroke model.

Statistical Analysis

Demographics and preoperative variables were compared between the 2 populations (CAB versus CAB/CEA) through the use of Student's t test for continuous data and χ^2 for discrete variables. Univariate analysis of demographics and variables to identify potential stroke risk factors was performed by the Student's t test, Wilcoxon's rank-sum test, or the χ^2 test. Multivariate determinants of stroke were obtained by stepwise multivariate logistic regression analysis. Candidate variables with a value of $P \leq 0.20$ were entered into a logistic model. Regression coefficients that are significantly different from 1.00 ($P \leq 0.05$) in the completed model are considered associated with stroke. Odds ratios and 95% confidence limits, as well as probability values, are reported. Statistical analysis was performed with the SPSS software (SPSS Corp). Application of these equations allowed us to predict stroke rates for the 2 patient groups (CAB alone and CAB/CEA) operated on in our institution during the study period. With the methodology developed for the New York State Cardiac Database, these results are displayed as both expected stroke rate and risk-adjusted ratio. The risk-adjusted stroke rate is normalized to the characteristics of the patient population from which the original formula was developed. This risk adjustment takes into account the differences between the original population used for the equation and the population under study. This risk-adjusted ratio is calculated by dividing the observed stroke rate by the expected stroke rate and then multiplying by the stroke rate in the reference population (which was used to calculate the stroke risk formula). These data are presented with 95% confidence limits.

Results

Description of Patient Population

Between 1994 and 1997, 1987 patients underwent isolated CAB. Twelve patients had emergent CAB in which carotid duplex was not performed and are excluded from subsequent analysis. In the remaining 1975 patients, carotid stenosis >80% was excluded by preoperative duplex ultrasound. Their mean age was 63.5 years; 73% of patients were male, and 27% were female. Of the patient population, 65.1% had a history of hypertension, 27.2% were diabetic, 14.8% had a history of peripheral vascular disease, 5.6% had a prior stroke, and 15.6% had a history of smoking. Table 1 gives a complete overview of the patient demographics and preoperative variables for patients who underwent isolated CAB.

Between 1994 and 1999, 154 consecutive patients underwent combined CAB/CEA for symptomatic coronary artery disease and carotid bifurcation stenosis of 80% to 99%. Although data from Asymptomatic Carotid Atherosclerosis Study (ACAS) suggest benefit of CEA for stenoses >60%, these data were for isolated CEA in patients without severe coronary artery disease. The concern that combined CAB/CEA would lead to higher stroke rates than seen in ACAS caused us to restrict combined operations to patients with more severe (≥80%) stenoses. No patient in this group was judged unfit for surgery. The average age was 68 years; 70% of the patients were male, and 30% were female. Seventy-two percent of patients had a history of hypertension, 28% were diabetic, 33% had a history of peripheral vascular disease, 14% had a prior stroke, and 26% had a history of smoking. Twenty-one patients (13.6%) had a prior history of stroke, although no patient experienced a fixed neurological deficit within 1 month of surgery. It was not possible to accurately determine the cause or laterality of the stroke in these patients from retrospective chart review. Most patients had asymptomatic carotid stenosis. Table 1 gives a complete overview of the patient demographics and preoperative variables for patients who underwent combined CAB/CEA.

TABLE 1. Patient Demographics and Risk Factors

Variable	CAB Patients, n=1987 (%)	Combined CAB/CEA, n=154 (%)	*P* Value
Age, average y	63.5	68	0.01*
Male	1457 (73)	108 (70)	
Female	530 (27)	46 (30)	0.443
Hypertension	1293 (65.1)	111 (72)	0.094
Diabetes	541 (27.2)	43 (28)	0.539
Smoking history	309 (15.6)	40 (26)	0.05*
Previous MI	387 (19.5)	26 (17)	0.544
Aortoiliac disease	173 (8.7)	30 (19)	<0.001*
Femeropopliteal disease	123 (6.1)	22 (14)	<0.001*
History of stroke	112 (5.6)	21 (14)	<0.001*
EKG evidence of LVH	160 (8.0)	14 (9)	0.387
COPD	206 (10.4)	19 (12)	0.377
Renal failure	29 (1.5)	6 (4)	0.169
IABP preoperatively	458 (23.0)	22 (14)	0.138
Extensively calcified ascending aorta	143 (7.2)	19 (12.3)	0.030*
Previous cardiac surgery	85 (4.3)	6 (4)	1.0
History of CHF	185 (9.3)	20 (13)	0.394
Malignant ventricular arrhythmia	109 (5.5)	3 (2)	1.0

MI indicates myocardial infarction; IABP, intra-aortic balloon pump; CHF, congestive heart failure.

Patients who underwent CAB alone were compared with the combined CAB/CEA patients. The combined CAB/CEA patients were significantly older, with a significantly higher percentage of patients reporting a history of smoking, peripheral vascular disease, and prior stroke. A significantly higher percentage of patients in the combined CAB/CEA group were found to have extensively calcified ascending aortas compared with the CAB group. The operating cardiac surgeon determined aortic calcification clinically.

Determination of Stroke Risk

Univariate analysis of patient demographics and preoperative variables was performed for CAB patients without significant carotid bifurcation stenosis (ie, >80%) to identify preoperative risk factors for the development of postoperative stroke. Those CAB patients who experienced a postoperative stroke were significantly older and experienced a prolonged cardiopulmonary bypass (CPB) time, as well as a higher incidence of hypertension, aortoiliac disease, ECG evidence of left ventricular hypertrophy (LVH), chronic obstructed pulmonary disease (COPD), and an extensively calcified ascending aorta (Table 2).

Multivariate logistic regression analysis of patient demographics and preoperative risk factors for postoperative stroke in CAB patients was performed. The following were found to be significant risk factors for the development of postoperative stroke: age, CPB time, aortoiliac disease, ECG evidence of LVH, and an extensively calcified ascending aorta (Table 3).

Based on the independent predictors of stroke identified from the multivariate logistic analysis, an institutional model to predict stroke after CAB was developed. The following formula was constructed: probability of postoperative stroke=$1/1+e(11.232-0.84\times age-0.01\times CPB-0.875\times aortoiliac-0.929\times LVH-1.021\times calcified\ aorta)$. When this model was applied to the 154 consecutive combined CAB/CEA patients, their expected postoperative stroke rate was 2.83% and their risk-adjusted stroke rate was 2.29% (range, 0.84% to 4.99%). The Columbia University New York Statewide model for stroke risk[4] was then applied to both the 1975 CAB patients without significant carotid stenosis and the 154 combined CAB/CEA patients with resultant predicted postoperative stroke rates of 1.56% for CAB alone and 3.41% for the combined CEA/CAB group. The risk-adjusted stroke rates were 1.49% (range, 1.09% to 2.22%) and 1.59% (range, 0.67% to 3.94%), respectively. The observed stroke rates in these 2 populations were 1.7% for CAB patients and 3.9% for the combined CAB/CEA patients ($P=0.105$) (Table 4). Although history of prior stroke was not a significant predictor of outcome in our institution-based model, we did compare results of CAB/CEA in the 21 patients with history of prior stroke in the CAB/CEA group with results from 133 patients with no prior neurological symptoms. Two patients (9.5%) with prior stroke suffered perioperative stroke, and an

TABLE 2. Univariate Analysis of Risk Factors for Stroke in CAB Patients

Variable	No Stroke	Stroke	*P* Value
Demographics			
No. of patients	1953	34	
Age, y	63.5	71.9	<0.001*
Male	1437	20	
Female	516	14	0.076
Preoperative Risk Factors			
Hypertension	1265 (64.8%)	28 (82.4%)	0.044*
Diabetes	532 (27.2%)	9 (26.5%)	1.000
Smoking history	305 (15.6%)	4 (11.8%)	0.810
Previous MI	384 (19.7%)	3 (8.8%)	0.130
Aortoiliac disease	164 (8.4%)	9 (26.5%)	0.002*
Femeropopliteal disease	120 (6.1%)	3 (8.8%)	0.464
History of stroke	109 (5.6%)	3 (8.8%)	0.436
EKG evidence of LVH	152 (7.8%)	8 (23.5%)	0.004*
COPD	198 (10.1%)	8 (23.5%)	0.020*
Dialysis dependent	28 (1.4%)	1 (2.9%)	0.396
IABP preoperatively	449 (23.0%)	9 (26.5%)	0.681
Extensively calcified ascending aorta	132 (6.8%)	11 (32.4%)	<0.001*
Previous cardiac surgery	82 (4.2%)	3 (8.8%)	0.176
History of CHF	179 (9.2%)	6 (17.6%)	0.125
CHF on admission	261 (13.4%)	6 (17.6%)	0.446
Malignant ventricular arrhythmia	109 (5.6%)	0	0.257
Hemodynamically unstable	136 (7.0%)	4 (11.8%)	0.296
Emergency CAB after PTCA	11 (0.6%)	1 (2.9%)	0.188
Cerebrovascular disease	229 (12%)	8 (23.5%)	0.055

MI indicates myocardial infarction; IABP, intra-aortic balloon pump; CHF, congestive heart failure; PTCA, percutaneous transluminal coronary angiography.

TABLE 3. Multivariate Logistic Regression Analysis of Risk Factors for Stroke in CAB Patients

Risk Factor	Variable Estimate	Standard Error	*P* Value	Odds Ratio	95% CI
1. Age	0.084	0.023	0.0002	1.09	1.040–1.136
2. Aortoiliac disease	0.875	0.419	0.037	2.40	1.055–5.451
3. EKG evidence of LVH	0.929	0.429	0.030	2.53	1.093–5.872
4. Extensively calcified ascending aorta	1.021	0.413	0.013	2.78	1.237–6.231
5. Bypass time	0.01	0.005	0.043	1.01	1.000–1.019

additional patient (4.8%), who was without neurological deficit, died. Although stroke rates exceeded those in the asymptomatic group (4 of 133=3%), the numbers of events were too small to be statistically significant.

Discussion

Our database is unique in a number of ways. Over a period of 4 years, we adopted a uniform practice of carotid duplex screening of all nonemergency CAB patients. In addition, we had a uniform policy of combined CAB/CEA in patients with >80% carotid stenosis. Demographic data, intraoperative variables, and postoperative results were prospectively collected in these patients in accordance with the New York State Department of Health requirements. This resulted in a prospectively collected database with 2 distinct populations: 1 proven not to have carotid stenosis >80% who underwent CAB alone, and 1 with carotid stenosis >80% (identified by duplex and confirmed by MR angiography or contrast angiography) who underwent combined CAB/CEA. This population allowed us to develop a stroke model in CAB patients known to be free of significant (>80%) carotid stenosis and compare it with both a statewide model (John et al[4]) that included all patients with CAB alone (carotid status unknown) and our population of combined CAB/CEA.

There are some potential confounders in this analysis. Some of the data (ie, aortic calcification, peripheral vascular disease) were not objectively documented (eg, epiaortic imaging, ankle brachial indexes). However, the criterion for identifying these conditions was defined by the New York State Cardiac Database and was applied uniformly throughout the study period. Assessment of these variables, which was prospective, can be assumed to be standard across both groups (CAB and CAB/CEA) of our patients in the study period. Data from the New York State Cardiac Database and similar cardiac databases have been used to predict risk and were the basis for the risk model of John et al.[4] Similarly, each patient was not evaluated preoperatively and postoperatively by a neurologist, and postoperative neurological deficits were identified by the medical or surgical team before being confirmed by a neurologist. Although this procedure may have resulted in an overall underestimation of neurological events, if anything, such an underestimation would have been lower in the combined CAB/CEA group in whom sensitivity to neurological status of the patient and frequency of neurological consultation was greater. Therefore, although the absolute incidence of risk factors (ie, aortic calcification, peripheral vascular disease) and outcomes such as neurological events may be less than if a prospective objective evaluation was performed, it is not likely that the incidence differed significantly across the groups studied. More definitive analysis will require a prospective study in which all patients have both preoperative and postoperative neurological evaluation. However, our data set offers an additional advantage by allowing direct comparison with the work of John et al, who used the same New York State Cardiac Database. This allows us to analyze the capabilities of the published model to predict institution-specific results compiled over a period of several years.

The analysis of demographics and preoperative variables of our patients undergoing CAB in this study has shown several significant risk factors associated with postoperative stroke. Univariate analysis of our data revealed that increasing age, ECG evidence of LVH, CPB time, an extensively calcified ascending aorta, and a history of hypertension, aortoiliac disease, or COPD was associated with an increased risk of postoperative stroke after isolated CAB. Multivariate logistic regression analysis identified 5 factors predictive of postoperative stroke: age, CPB time, aortoiliac occlusive disease, LVH, and calcification of the aorta. Although other series have variations in the type and frequency of risk factors associated with post-CAB stroke, all of the significant risk factors identified in this study have been cited by others to contribute to increased post-CAB stroke.[3–8]

TABLE 4. Observed and Predicted Stroke Rates

		Stony Brook Model		Columbia Statewide Model	
	Observed	Expected	Risk Adjusted*	Expected	Risk Adjusted*
CAB (n=1987)	1.7%	N/A		1.56%	1.49% (1.09–2.22)
Combined CAB and CEA (n=154)	3.9%	2.83%	2.29% (0.84–4.99)	3.41%	1.59% (0.67–3.94)

Risk-adjusted stroke rates=observed/expected×stroke rate of reference population. In the case of model I this was 1.7%. In model II this was 1.4%.

Our institutional model for predicting post-CAB stroke differs from the statewide model reported from Columbia University. The Stony Brook model found that ECG evidence of LVH was a significant risk factor for stroke, which was not seen in the Columbia model. The Columbia model included risk factors such as renal failure, diabetes, smoking, and prior cerebral vascular disease, which were not significant in the analysis of our patients.[4] Some of these differences can be ascribed to sample size and homogeneity of the study sets. The absence of cerebrovascular disease as a risk factor in our analysis is expected because patients with carotid stenosis >80% were excluded from CAB alone by our clinical protocol. Our data are insufficient to determine the impact of prior stroke on the outcome of CAB/CEA. Although the stroke risk was increased in this group, the number of events was small, and the relationship of stroke to carotid stenosis could not be determined in all cases. Our data support the impression shared by others that patients with prior neurological symptoms are a high-risk group. However, quantification of this risk awaits a larger prospective study. The fact that 14% of our CAB/CEA group had prior neurological symptoms emphasizes the importance of developing a strategy to deal with these patients. Both models identified age, CPB time, peripheral vascular disease, and a calcified aorta as significant risk factors. A major difference between our institutional model (model I) and the statewide model (model II) is the specific exclusion of patients with known severe carotid bifurcation disease in model I, whereas model II clearly included some of those patients. As a consequence, cerebral vascular disease was not a risk factor in model I because carotid stenosis was excluded by duplex ultrasound. This is a likely reason for the lower estimated stroke risks in model I, which reflected a more homogeneous patient population without carotid stenosis.

When comparing our population of CAB patients who did not have significant carotid stenosis with patients with combined CAB/CEA patients, all of whom had severe carotid stenosis, we noted several differences between the 2 groups. The combined CAB/CEA patients were older and more often had extensively calcified ascending aortas and peripheral vascular disease. These 3 variables represent 3 of the 4 risk factors found to be significant in our multivariate regression analysis and are incorporated into our stroke risk model. The Columbia University model identifies history of smoking and prior stroke as significant risk factors for stroke. Both of these risk factors were also significantly more common in our population of combined CAB/CEA patients.

Our observed stroke rate in the CAB alone patients was equivalent to the expected and risk-adjusted stroke rates calculated using the Columbia model (1.7% observed versus 1.56% expected and 1.4% risk adjusted). This speaks to the ability of the Columbia model to predict stroke rates in a specific institution over a period of several years.

When the Stony Brook and Columbia models of stroke risk for isolated CAB were applied to our population of 154 consecutive patients who underwent combined CAB/CEA, our observed stroke rate (3.9%) was similar to that predicted by the Columbia Model (3.41%) but greater than that predicted by model I (2.83%), although the difference was not statistically significant. After adjustment of the observed stroke rate of 3.9% for risk factors, the risk-adjusted stroke rates were not statistically different from the reference population stroke rates from both predictive models. Thus, using 2 separate calculations, we were unable to demonstrate any increased risk of adding CEA to CAB in patients with surgical lesions of both the carotid and coronary circulations. One can draw inferences but not conclusions from these observations. It is important to remember that in model I extracranial occlusive disease was excluded. Performing CEA at the time of CAB does not impart predicted stroke risk as low as that in CAB patients without carotid stenosis. It is tempting to speculate that this difference is attributable to the risk of CEA itself. At the same time, however, it seems clear that the addition of CEA in these 154 patients did not significantly increase the risk of postoperative stroke predicted by either model. This suggests that the addition of CEA to CAB does not synergistically increase stroke risk over what one would expect from patient risk factors. Although our data confirm that patients who undergo combined CAB/CEA have an increased rate of postoperative stroke (an observation supported by many observational studies), our analysis suggests that this risk is due primarily to factors (eg, age, aortic calcification) other than the performance of a CEA. This offers some degree of equipoise to those contemplating a randomized study of the effects of CEA in CAB patients.

Our data do not address the influence of neurological status on the outcomes of combined CAB/CEA or the management of the neurologically symptomatic patient in need of CAB. It has been our observation that most CAB patients with carotid stenosis are neurologically asymptomatic, as is true of most patients with carotid stenosis in general. In our study, no patient gave a history of a fixed neurological deficit within 1 month of combined surgery. However, our data are insufficient to determine the frequency of more subtle or transient neurological symptoms or the incidence of more remote (eg, 2 to 12 month) neurological events. The true frequency of such patients in the CAB population and their proper management await the results of a prospective study.

The ability to predict stroke after CAB based on preoperative variables is of great significance. It will allow the prediction of risk-adjusted stroke rates for institutions similar to the risk-adjusted mortality rates currently in use. This should eventually allow improvements in stroke morbidity after CAB similar to those seen in mortality rates after this operation in the past. Preoperative risk adjustment will also be important in comparing treatment options such as staged versus combined CEA/CAB or even carotid angioplasty before CAB in patients with combined coronary and carotid disease.

In summary, analysis of CAB and combined CAB/CEA patients can identify significant risk factors that contribute to the risk of postoperative stroke. These risk factors will vary, depending on institutional patient demographics and preoperative variables. Three of these risk factors (age, aortic calcification, peripheral vascular disease) may be reflective of an overall increase of atherosclerotic burden in these patients. Modeling stroke risk after CAB based on multivariate regres-

sion analysis of patient demographics and preoperative variables is possible. Using this method to evaluate results in patients who underwent combined CAB/CEA at our institution revealed no statistically significant increase in stroke risk above the predicted stroke risk associated with the addition of CEA to CAB. Although the role of CEA in reducing stroke in CAB patients will be determined by a prospective randomized trial, these results suggest that combined surgery can be performed without increasing postoperative stroke. Efforts to develop such a prospective study are currently in progress.

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