Relation of midwall circumferential systolic stress to equatorial midwall fibre shortening in chronic aortic regurgitation

Value as a predictor of postoperative outcome

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SUMMARY Nineteen patients with chronic aortic regurgitation and a large increase in heart size were studied before aortic valve replacement. By relating midwall circumferential systolic stress to midwall circumferential fibre shortening (Cs/Cd) before operation the patients could be divided into two well defined groups. Twelve patients (group 1) had a pronounced decrease in heart size as measured by the cardiothoracic ratio and an excellent clinical outcome six months after operation. Seven patients (group 2) had no significant decrease in heart size and a less good clinical outcome. The ratio of midwall circumferential systolic stress to end systolic volume index was significantly higher in group 1 than in group 2. Group 2 had more severe left ventricular hypertrophy determined by the ratio of the wall thickness to the minor internal radius of the left ventricle (h:r ratio), total left ventricular mass, and left ventricular mass to end diastolic volume ratio. There were no significant differences in any other haemodynamic or angiographic indices between the two groups.

Thus the relation of midwall circumferential systolic stress to fibre shortening is useful in determining the prognosis in individual patients with chronic aortic regurgitation undergoing aortic valve replacement.

The prognosis of individual patients with chronic aortic regurgitation undergoing aortic valve replacement remains one of the more perplexing problems in modern cardiology. In some instances, it seems likely that long standing volume overload leads to irreversible left ventricular dysfunction irrespective of the clinical status.¹ Many studies have analysed preoperative left ventricular function in chronic aortic regurgitation²⁻⁷ in an attempt to identify patients who are at high risk of dying of congestive heart failure during the long-term postoperative course. On the other hand, patients undergoing aortic valve replacement whose cardiac enlargement does not decrease after operation have a less good postoperative outcome than those

who have an appreciable reduction in cardiomegaly.⁸⁻¹⁰

In the present study we used a new approach for the preoperative analysis of patients with chronic aortic regurgitation by applying a stress-strain analysis of left ventricular contraction to identify those patients with irreversible left ventricular dysfunction. We compared the results with the postoperative variations in heart size measured by the cardiothoracic ratio six months after operation.

Patients and methods

PATIENT SELECTION

Fifty five out of 84 patients with pure aortic regurgitation (peak systolic aortic gradient <15 mm Hg) in whom haemodynamic studies had been performed in our laboratory from January 1977 to June 1980 underwent aortic valve replacement. Of these, we selected 19 patients who had pronounced cardiomeg-

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Accepted for publication 15 May 1984

ally on the routine chest x ray film (mean cardiothoracic ratio 0.62; range 0.57–0.70). Of these 19 patients, 18 were clinically evaluated six months after operation; the patient in case 19 died 45 days after the operation as a result of congestive heart failure. We excluded patients who had acute aortic regurgitation on clinical and surgical grounds. Of the 19 patients, six were women and 13 men (mean age 38 (range 24–59) years). Six patients were asymptomatic and 12 had symptoms in NYHA functional class II, of whom five had exertional angina. One patient had symptoms in NYHA functional class IV. All patients showed left ventricular hypertrophy in the electrocardiogram, 16 were in sinus rhythm, three had chronic atrial fibrillation, and two had previously documented transient episodes of atrial fibrillation.

CORONARY ARTERIOGRAPHY

All patients underwent right and left cardiac catheterisation before operation. Coronary arteriography was performed in all male patients aged ≥ 45 years (four patients) and in all patients whose presenting symptom was angina irrespective of sex and age (two patients). Pressures were recorded with external transducers (Stratham P-23 Db). Cardiac output was measured by thermodilution with the aid of a computer (E for M-Lyons TCCO-10). Left ventricular angiography was performed in the right anterior oblique position with the contrast medium (meglumine diatrizoate 66%) injected at a mean speed of 17.5 ml/s through an NIH No 7F or 8F catheter. Films were taken at 50 frames/s, extrasystoles and postextrasystoles were excluded from analysis. In the three cases with chronic atrial fibrillation, the first four beats with good opacification were analysed.

Left ventricular volume was calculated by the area-length method.¹¹ Magnification for volume and mass was corrected using a calibrated grid placed 10 cm above the plane of the table.¹² Ejection fraction (EF) was calculated from the corrected volumes as follows: $EF = EDV - ESV / EDV$ where EDV is end diastolic volume and ESV end systolic volume. The regurgitant fraction (RF) was calculated as follows: $RF = (\text{angiographic stroke volume minus anterograde thermodilution stroke volume}) / \text{angiographic stroke volume}$. Left ventricular mass was calculated using the method of Trenouth *et al.*¹³ Left ventricular free wall was drawn in the middle third of the cardiac shadow at end diastole, and the resulting rectangle was measured by planimetry. The area divided by the length and corrected for radiographic magnification was taken as the mean end diastolic wall thickness (hd). On the basis that the left ventricular mass remains constant during cardiac contraction end systolic wall thickness (hs) was calculated as proposed by Hugenholz *et al.*¹⁴ Left ventricular internal

diameters at end diastole (Dd) and end systole (Ds) were calculated by the area-length method.

In all cases, midwall circumferential systolic stress (MWCS) was determined using the equation of Mirsky¹⁵: $MWCS = pb/h \times (1 - h/2b - b^2/2a^2)$, where h is end systolic wall thickness, a end systolic midwall major semi-axis, b end systolic midwall minor semi-axis, and p intraventricular peak systolic pressure, measured as the mean value of the 10 beats immediately preceding left ventricular angiography. We calculated the circumferential shortening of the equatorial midwall fibre as Cs/Cd, Cs being = Ds+hs and Cd being = Dd+hd.

AORTIC VALVE REPLACEMENT

All patients were operated on within two months of cardiac catheterisation. Ten patients had a Hancock porcine heterograft and nine a Björk-Shiley prosthesis implanted. All patients underwent a complete clinical evaluation, which included a standard posteroanterior chest radiograph six months after operation.

STATISTICAL ANALYSIS

Values are expressed as means and standard deviations (SD). The least squares method for regression equations was used. The statistical analysis of the regression lines was performed by covariance analysis. Means of two samples were compared by Wilcoxon's rank sum test.

Results

HAEMODYNAMIC MEASUREMENTS

The individual values of midwall circumferential sys-

Figure 1 Relation of midwall circumferential systolic stress to circumferential midwall fibre shortening (Cs/Cd) in patients in group 1 (●) and group 2 (○).

Table 1 Values for the midwall circumferential systolic stress (MWCS), midwall circumferential shortening (Cs/Cd) and MWCS/end systolic volume index (MWCS/ESVI) in 19 patients with chronic aortic regurgitation

Case No	MWCS (dyn/cm ² × 10 ⁻³)	MWCS/ESVI	Cs/Cd
<i>Group 1</i>			
1	319	6.25	0.79
2	458	4.62	0.89
3	390	2.54	0.90
4	225	2.88	0.76
5	438	3.56	0.85
6	367	2.54	0.88
7	483	4.09	0.90
8	319	2.38	0.82
9	422	2.18	0.89
10	319	4.08	0.82
11	349	2.49	0.88
12	285	5.48	0.82
Mean(SD)	364(76)	3.59(1.33)	0.85(0.045)
<i>Group 2</i>			
13	230	3.33	0.82
14	265	2.10	0.85
15	235	2.21	0.84
16	227	2.60	0.88
17	217	3.28	0.83
18	302	1.97	0.87
19	342	1.44	0.94
Mean(SD)	259(44)	2.41(0.69)	0.86(0.039)
Significance	p < 0.005	p < 0.025	NS

tolic stress and midwall circumferential fibre shortening were plotted, giving two different groups of data (Figure); 12 patients fell within the 95% confidence limits of the linear relation between the two measurements (group 1) and seven patients fell outside these confidence limits (group 2) indicating significantly lower midwall circumferential systolic stress for the same degree of midwall circumferential fibre shortening. A regression line was fitted to each group of data to give the following regression equations: $Y_1 = MWCS_1 = 1362(Cs/Cd)_1 - 793$; ($r = 0.8389$; $p < 0.001$) and $Y_2 = MWCS_2 = 936(Cs/Cd)_2 - 547$; ($r = 0.8178$; $p < 0.05$). A covariance analysis of these regression lines showed a highly significant difference between them ($Y_1 \neq Y_2$; $p < 0.001$). Although the slopes of both regression lines were different, this difference did not reach the level of statistical significance ($0.05 < p < 0.1$).

The individual data for each patient are shown in Table 1. The mean value of midwall circumferential systolic stress was significantly higher in group 1 than in group 2 ($p < 0.005$). The mean value of Cs/Cd was similar in both groups. The mean value of midwall circumferential systolic stress to end systolic volume index (MWCS/ESVI) for group 1 was higher than that for group 2 ($p < 0.025$). There was, however, a high degree of overlap between both groups for this value.

Table 2 Clinical features in 19 patients with chronic aortic regurgitation

Case No	Age (yrs)	Sex	Cardiac rhythm	Preoperative symptoms	NYHA class	
					Before operation	After operation
Group 1						
1	24	F	Sinus	None	I	I
2	32	M	Sinus	None	I	I
3	47	M	Sinus	POE, angina	II	I
4	25	M	Sinus	DOE	II	I
5	27	F	Sinus	DOE, POE	II	I
6	25	M	Sinus	None	I	I
7	59	M	Sinus	DOE, angina	II	I
8	16	M	Sinus	DOE	II	I
9	39	M	Sinus	DOE	II	I
10	38	M	Sinus	None	I	I
11	55	F	Sinus	DOE, POE	II	I
12	45	F	Sinus	None	I	I
Group 2						
13	38	M	AF(T)	DOE, angina	II	I*
14	43	M	AF(C)	DOE, POE	II	I†
15	51	M	Sinus	Angina	II	I
16	35	M	AF(C)	None	I	I
17	43	F	Sinus	Angina	II	I
18	38	F	AF(C)	DOE	II	I
19	49	M	AF(T)	DOE, PND, angina	IV	IV‡

AF, atrial fibrillation (C, chronic; T, transient); DOE, dyspnoea on exertion; PND, paroxysmal nocturnal dyspnoea; POE, palpitations on exertion.

*Acute pulmonary oedema 42 months after operation.

†Died two years after operation.

‡Died 45 days after operation.

Table 3 Changes in the cardiothoracic ratio six months after operation in 19 patients with chronic aortic regurgitation

Case No	Cardiothoracic ratio	
	Before operation	After operation
Group 1		
1	0.61	0.43
2	0.60	0.52
3	0.69	0.60
4	0.57	0.50
5	0.58	0.40
6	0.69	0.47
7	0.62	0.52
8	0.63	0.51
9	0.65	0.55
10	0.57	0.49
11	0.62	0.48
12	0.61	0.53
Mean(SD)	0.62(0.038)*	0.50(0.051)‡
Significance	p < 0.001	
Group 2		
13	0.63	0.61
14	0.57	0.56
15	0.59	0.59
16	0.65	0.65
17	0.65	0.58
18	0.70	0.70
19	0.62	—
Mean(SD)	0.63(0.042)†	0.61(0.051)§
Significance	NS	

*Compared with †, NS.

‡Compared with §, p<0.001.

CLINICAL DATA

Table 2 shows the clinical data for both groups of patients. Mean age in both groups was not significantly different (36 (13) years *vs* 42 (5.9) years; $p>0.05$). There were seven symptomatic patients (58%) in group 1 and six in group 2 (85%). All patients were asymptomatic six months after operation, except one patient in group 2 whose symptoms remained in NYHA functional class IV after operation and who died 45 days later. All patients in group 1 were in sinus rhythm, whereas three patients in group 2 had chronic atrial fibrillation and two others transient episodes of atrial fibrillation.

POSTOPERATIVE VARIATION IN HEART SIZE

There was a pronounced difference between the two groups in the reduction of the cardiothoracic ratio after operation (Table 3). There was no difference between both sets of preoperative values. Group 1 showed a pronounced reduction in cardiothoracic ratio six months after operation giving a normal value in all patients but one (case 3), who had a 13% reduction, which did not reach the normal value of 0.55. The mean postoperative ratio in group 1 was significantly less than the mean preoperative value ($p<0.001$). The mean decrease was 15% for this group as a whole.

In contrast, patients in group 2 did not show any significant change in ratio six months after operation.

Only the patient in case 17 had a decrease of 10%, which was less than any of those in group 1 and not enough to normalise the cardiothoracic ratio.

HAEMODYNAMIC AND ANGIOGRAPHIC DATA

Table 4 shows the haemodynamic and angiographic data of both groups. Aortic pressures did not show any significant difference. Left ventricular end diastolic pressure (LVEDP) was increased in both groups to a similar extent and showed a wide scattering of values. The mean values for cardiac output and cardiac index were slightly higher in group 1 than in group 2, the differences not being significant. The mean values for end diastolic volume and end systolic volume were appreciably increased in both groups, with no significant differences. The regurgitant fraction was smaller in group 1, but the difference was not significant. The ejection fraction showed a mean value which was equally depressed in both groups.

LEFT VENTRICULAR HYPERTROPHY

Table 5 shows the values for the ratio of the wall thickness to the minor internal radius of the left ventricle (h:r ratio) and left ventricular mass to end diastolic volume ratio. Both were significantly higher in group 2 ($p<0.005$) and ($p<0.005$). The same was true for the total left ventricular mass and left ventricular mass index, both values being greater in group 2 ($p<0.025$) than in group 1 ($p<0.025$).

MORTALITY DATA

The mean follow up period after operation was similar for both groups of patients (23.9 months in group 1 *vs* 22.7 months in group 2). During follow up, there were no deaths in group 1 and two in group 2. The patient in case 14 (group 2) had several hospital admissions after operation because of ventricular arrhythmias; he died suddenly two years after operation. The patient in case 19 died 45 days after operation because of congestive heart failure. The patient in case 13 showed a pronounced clinical improvement six months after operation, remaining in stable clinical condition for 42 months, after which he was readmitted because of acute pulmonary oedema. Catheterisation at that time showed a normally functioning aortic prosthesis with no improvement in left ventricular systolic function and no decrease in end diastolic volume.

Discussion

The postoperative outcome of patients with chronic aortic regurgitation depends mainly on the existence, or absence, of irreversible preoperative left ventricular dysfunction. The preoperative diagnosis of this condition is still a major problem. The patient's clinical

Table 4 Haemodynamic and angiographic data in 19 patients with chronic aortic regurgitation

Case No	BS (m ²)	AoP (mm Hg)	LVEDP (mm Hg)	CO (l/min)	CI (l/min per m ²)	RF	EDV (ml)	ESV (ml)	EF
Group 1									
1	1.45	108/40	43	2.90	2.0	0.66	164	74	0.55
2	1.62	165/73	5	7.55	4.6	0.21	251	161	0.35
3	1.68	185/36	6	6.16	3.6	0.42	408	258	0.36
4	1.85	112/42	22	4.88	2.6	0.68	371	146	0.60
5	1.52	160/60	1	4.63	3.0	0.50	303	187	0.38
6	1.92	106/40	15	4.80	2.5	0.60	424	278	0.34
7	2.04	158/54	44	—	—	—	321	241	0.24
8	1.40	128/50	30	3.10	2.2	0.74	328	188	0.42
9	1.83	144/52	7	4.40	2.4	0.62	495	354	0.28
10	1.78	135/55	9	—	—	—	285	140	0.50
11	1.81	108/63	25	5.80	3.2	0.48	378	254	0.33
12	1.56	203/75	7	3.39	2.1	0.37	172	82	0.52
Mean(SD)		142(32)/53(11)	17.8(14)	4.76(1.45)	2.8(0.8)	0.52(0.16)	325(98)	196(83)	0.40(0.11)
Group 2									
13	1.96	130/80	10	3.77	1.9	0.69	266	136	0.48
14	1.80	150/70	16	4.50	2.5	0.68	408	228	0.44
15	1.62	180/50	28	3.90	2.4	0.68	368	173	0.53
16	1.69	104/40	8	4.40	2.6	0.32	238	148	0.37
17	1.60	124/54	3	4.00	2.5	0.43	198	106	0.46
18	1.47	127/47	12	3.80	2.5	0.53	336	225	0.33
19	1.58	180/70	45	4.00	2.5	0.77	520	375	0.28
Mean(SD)		142(29)/58(14)	17.4(14)	4.05(0.28)	2.4(0.23)	0.58(0.16)	333(110)	198(89)	0.41(0.08)
Significance		NS/NS	NS	NS	NS	NS	NS	NS	NS

BS, body surface; AoP, aortic pressure; CO, cardiac output; CI, cardiac index; RF, regurgitation fraction; EDV, end diastolic volume; ESV, end systolic volume; EF, ejection fraction.

Table 5 Left ventricular hypertrophy data in 19 patients with chronic aortic regurgitation

Case No	h:r ratio	LVM (g)	LVM/EDV (g/ml)
Group 1			
1	0.18	89	0.54
2	0.28	211	0.84
3	0.31	405	0.99
4	0.29	270	0.73
5	0.23	210	0.69
6	0.18	240	0.56
7	0.25	252	0.78
8	0.24	251	0.76
9	0.30	469	0.94
10	0.25	215	0.75
11	0.22	259	0.68
12	0.35	200	1.16
Mean(SD)	0.25(0.05)	225(97)	0.78(0.17)
Group 2			
13	0.31	273	1.02
14	0.33	444	1.08
15	0.38	475	1.29
16	0.31	236	0.99
17	0.35	225	1.13
18	0.28	303	0.90
19	0.38	663	1.27
Mean(SD)	0.33(0.03)	374(160)	1.09(0.14)
Significance	p<0.005	p<0.025	p<0.005

EDV, end diastolic volume; h, wall thickness of the left ventricle; LVM, left ventricular mass; r, minor internal radius of the left ventricle.

cal status is of no help. Not all of those with heart failure before operation have a poor outcome after operation. On the other hand, the absence of symptoms does not automatically rule out the existence of left ventricular dysfunction, as several authors have shown.¹⁶⁻¹⁸ By definition, the postoperative outcome

after successful operation is an excellent guide to the previous existence, or absence, of irreversible ventricular dysfunction. Several investigators⁸⁻¹⁰ have found that the persistence of left ventricular dilatation, as measured by echocardiography, is associated with a higher risk of developing heart failure after operation, even if the haemodynamic disturbance has been corrected successfully. There is also general agreement, with the exception of the observations of Toussaint *et al*,¹⁹ that the reduction in heart size takes place early, from two weeks to two months after operation with very slight changes up to 63 months later.^{8-10 20 21} These studies validated earlier observations on the prognostic value of the change in the cardiothoracic ratio after operation,^{22 23} which related the lack of reduction to a poorer prognostic outcome. The present study supports these previous reports. Of a total of 19 patients, 12 (group 1) had an important reduction in cardiothoracic ratio six months after operation, and six (group 2) showed no significant change in this value. The different behaviour of the cardiothoracic ratio was associated with the poorer clinical outcome in group 2.

Once these different clinical outcomes had been established, the purpose of the present study was to identify any factor in the preoperative haemodynamic study that would be able to predict the reduction in heart size after operation. The usual haemodynamic and angiographic indices were poor predictors. There was no difference between both groups in left ventricular end diastolic pressure, cardiac output, cardiac index, or regurgitant fraction. The mean value of end

diastolic volume and end systolic volume was greatly increased, and the ejection fraction depressed, showing a similar degree of left ventricular systolic dysfunction in both groups. It is usually believed that patients with normal left ventricular systolic function have an excellent postoperative outcome.¹ Conversely, those with systolic dysfunction preoperatively have an unpredictable and uncertain outcome.⁴ It has recently been suggested that the end systolic diameter measured with echocardiography is of prognostic value in determining the development of congestive heart failure in the long term after aortic valve replacement.^{8 24-26} Not all patients within this high risk group, however, have a poor postoperative outcome, as is shown by our results and those of other authors.²⁷ It is fundamental, therefore, to establish preoperatively which of those patients in the high risk group have a reversible type of left ventricular dysfunction after operation.

Recently, Borow *et al* found that end systolic stress and percentage fractional shortening determined by non-invasive methods are inversely and linearly related and that this relation was highly sensitive to alterations in left ventricular inotropic state.²⁸ Based on these findings, they suggested that this relation may be potentially useful in assessing intrinsic left ventricular contractile impairment in patients with valvular disease. We have tested this hypothesis in patients with chronic aortic regurgitation, although with some methodological differences when compared with Borow's study.

End systolic stress is calculated through the use of the end systolic pressure and the end systolic volume. The end systolic pressure is difficult to determine, requiring the simultaneous recording of left ventricular pressure and volume. This necessitates the use of high fidelity angi catheters and this complex methodology would exclude this analysis from everyday use. One of our main purposes was to establish a method that could be used in ordinary practice. In addition, several investigators^{24 29-31} have previously shown that the relation of end systolic pressure to end systolic volume behaves very similarly to that of peak systolic pressure and end systolic volume in the clinical setting, in such a way that the use of peak systolic pressure introduces numerical rather than fundamental differences. Thus it seems reasonable that the same holds true for the relation of end systolic stress and end systolic volume and that of the midwall circumferential systolic stress and end systolic volume because in the equation to calculate the stress all indices are the same, with the exception of pressure being substituted by end systolic pressure in one case and by peak systolic pressure in the other. In our stress-shortening analysis, we referred shortening to the midwall equatorial fibre of the left ventricle. With left

ventricular contraction the myocardium not only shortens but also thickens, and several models have been described for the thickening-shortening relation.^{32 33} The midwall fibre is a theoretical construction which reflects the fibre shortening better than the internal left ventricular diameters. We normalised the end systolic circumference (Cs) by dividing it by the end diastolic circumference (Cd). The resulting index (Cs/Cd) is dimensionless and has the characteristics of a strain, with the general formula L_f/L_o , where L_f is final length and L_o initial length. In addition, this index maintains a very simple relation with the most usual percentage fractional shortening ($(Cd - Cs)/Cd = 1 - Cs/Cd$).

With this stress-strain analysis, we were able to separate preoperatively the patients into two groups. Group 2 showed the same Cs/Cd value as group 1 for significantly lower left ventricular afterload (MWCS) levels. This finding suggests that patients in this group had a relatively poorer intrinsic shortening ability than those in group 1, this fact being closely related to a poorer outcome after operation. In addition, the midwall circumferential stress was linearly and inversely related in both groups with the left ventricular midwall percentage fractional shortening $(1 - Cs/Cd)$. These findings are in close conceptual agreement with those of Borow *et al* in normal subjects²⁸ and suggest that the midwall circumferential stress to Cs/Cd relation reflects the left ventricular intrinsic contractile state in chronic aortic regurgitation.

A controversial point in our results is the different degree of left ventricular hypertrophy in both groups of patients. Our data disagree with those of Gaasch *et al*,⁹ referring to the concept of "inadequate hypertrophy" in end stage chronic aortic regurgitation. These authors found that this ratio, as determined by echocardiography, was significantly lower in patients with chronic aortic regurgitation and a poor outcome after aortic valve replacement. These authors, as well as others,³² suggest that as the left ventricle dilates and, therefore, wall stress increases cardiac hypertrophy progresses to maintain wall stress within certain limits. In some cases, the ventricle dilates without an adequate hypertrophy, with a subsequent increase in stress and with the probability of developing irreversible left ventricular dysfunction.

In our view, this concept is questionable. Data from several authors^{7 33-35} show that the end systolic stress to end systolic volume index ratio is significantly higher in those patients with a better postoperative outcome, even though they relate to different pathological conditions. It is obvious that if two ventricles are indistinguishable with regard to end systolic volume and end systolic pressure, in order for the end systolic stress/end systolic volume index ratio

to be higher in one patient, the h:r ratio must be less in that same patient. On the other hand, other investigators have found no correlation between this last index and the postoperative course in chronic aortic regurgitation.^{1,25} In addition, there is some evidence suggesting that the postoperative course in chronic aortic regurgitation depends on total left ventricular mass rather than on relative hypertrophy, as measured by the h:r ratio; Krayenbuehl *et al* found that patients with chronic aortic regurgitation and a left ventricular mass >180 g/m² were not able to normalise the left ventricular systolic function after successful aortic valve replacement, in contrast with those with a left ventricular mass <180 g/m², who normalised their left ventricular systolic function after aortic valve replacement.³⁶ Our results agree with these observations, suggesting that a very high degree of left ventricular hypertrophy precludes a good postoperative evolution in patients with chronic aortic regurgitation undergoing successful aortic valve replacement. We believe, however, that further studies are needed to resolve this controversial point.

The data presented in this study refer to a small group of patients and must, therefore, be regarded with caution. Future applications of this analysis to a larger number of patients should allow us to define its sensitivity and specificity with a higher degree of accuracy.

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