Echocardiography in Chronic Aortic Insufficiency

Is Valve Replacement Too Late When Left Ventricular End-systolic Dimension Reaches 55 mm?

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SUMMARY **To determine whether a ventricular (LV) end-systolic dimension (ESD) ≥ 55 mm and LV left fractional shortening < 25% are risk factors for aortic valve replacement (AVR) in patients with aortic insufficiency, we analyzed the clinical course and M-mode echocardiograms in 47 consecutive patients who underwent AVR for isolated symptomatic AI. Group 1 patients (n = 27) had a preoperative ESD < 55 mm (mean 44 mm, range 30–52 mm) and group 2 patients (n = 20) had a preoperative ESD ≥ 55 mm (mean 62 mm, range 55–85 mm). One patient in group 1 and 10 patients in group 2 had left ventricular fractional shortening < 25%. There were no perioperative or postoperative deaths during an average follow-up of 41 months (range 6–76 months). Five patients had perioperative myocardial infarctions (MIs), three in group 1 and two in group 2. Since myocardial protection with cold potassium cardioplegia was instituted, no patient has suffered a perioperative MI. The average preoperative New York Heart Association functional classification was 2.3 (group 1) and 2.6 (group 2). Postoperatively, it was 1.2 in group 1 and 1.1 in group 2. Thirty-three patients (20 in group 1 and 13 in group 2) had echocardiograms at least 1 year after AVR. Of these, LV-end diastolic dimension decreased from 67 ± 6 to 53 ± 6 mm (mean ± SD) in group 1 ($p < 0.001$) and from 79 ± 3 to 55 ± 6 mm in group 2 ($p < 0.001$). The LVESD also decreased, but this is difficult to interpret because of frequent postoperative abnormal interventricular septal motion. The LV cross-sectional area, an index of LV mass, decreased in group 1 from 25 ± 5 to 20 ± 5 cm^2 ($p < 0.001$) and in group 2 from 32 ± 9 to 20 ± 5 cm^2 ($p < 0.001$). Postoperative end-diastolic dimension and cross-sectional area were not significantly different between the two groups. We concluded that in aortic insufficiency, a preoperative ESD ≥ 55 mm does not preclude successful AVR, as judged by long-term survival, symptomatic relief, and normalization of LV dimensions assessed by echocardiography.**

THE OPTIMAL management of patients with severe aortic insufficiency (AI) is still a matter of debate, especially in asymptomatic or minimally symptomatic patients.[1–3] The recommendation for aortic valve replacement (AVR) in these patients depends, among other factors,[4] on the identification of the degree of left ventricular (LV) dysfunction beyond which the risks of perioperative and postoperative mortality and morbidity are increased.[5]

Henry et al.[6] used M-mode echocardiography to identify a population at high risk for AVR due to AI. In their experience, when the LV end-systolic dimension (ESD) was 55 mm or greater, AVR carried a higher risk of perioperative complications and postoperative death for congestive heart failure. Most of these patients had LV fractional shortening (FS) of less than 25% as well. Other studies have suggested that the end-systolic volume is an important predictor of the postoperative clinical course in patients after AVR for AI.[7]

Other data do not support this,[8, 9] and we have seen several patients in this "high-risk" category with a benign peri- and postoperative course. We therefore performed a retrospective study to test the hypothesis that in patients with symptomatic AI, AVR is too late when the LV FS is less than 25% or ESD is 55 mm.

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Methods

Patients

From January 1972 to December 1980, 70 patients with symptomatic severe AI underwent isolated AVR at our institution. Patients were included in the study if they fulfilled the following criteria: chronic AI, absence of associated aortic stenosis (peak transvalvular gradient less than 20 mm Hg) or other valvular disease, absence of obstructive coronary artery disease and malfunction of the valve prosthesis. Only patients with a high-quality preoperative M-mode echocardiogram were included.

Clinical Studies

Forty-seven of the 70 patients met the criteria and were included in the study. All but one patient underwent preoperative right- and left-heart catheterization, including LV and aortic root cineangiography. In nine patients, angiographic quantification of LV volumes was impossible because the images were unsatisfactory. Selective coronary arteriography was performed in 34 patients: all of those older than age 40 years and in the younger patients with chest pain. Cardiac catheterization was performed within 3 months of surgery.

Echocardiograms were recorded with the patient in the supine or slight left lateral decubitus position using

standard techniques.[10] To minimize the variability in LV measurements due to body position, serial echocardiograms were obtained in the same position in both the pre- and postoperative studies.

The following echocardiographic measurements were performed: (1) LV end-diastolic dimension (EDD), defined as the distance between the left-sided endocardial surface of the interventricular septum and that of posterior wall at the onset of the Q wave of the ECG: (2) LV ESD, defined as the smallest LV diameter; (3) FS, defined as

$$\frac{\text{EDD} - \text{ESD}}{\text{EDD}} \times 100;$$

(4) LV cross-sectional area (CSA), an index of LV mass,[11] derived from EDD and LV posterior wall thickness (PWth) measurements:

$$\left(\frac{\text{EDD}}{2} + \text{PWth}\right)^2 \pi - \left(\frac{\text{EDD}}{2}\right)^2 \pi.$$

Because of frequent paradoxical interventricular septum motion, ESD and FS were not measured postoperatively. Serial measurements were made with the observer blinded to echocardiographic results.

Patient Groups

Based on their preoperative echocardiogram, patients were divided into two groups (fig. 1). Group 1 patients (n = 27) had an ESD < 55 mm; and group 2 patients (n = 20) had an ESD ⩾ 55 mm. One patient in group 1 and 10 patients in group 2 had a FS of less than 25%. The preoperative clinical, echocardiographic and catheterization data are summarized in tables 1 and 2. By cineangiocardiography, seven patients in group 2 had an ejection fraction of less than 40% (range 24–39%). No patient in group 1 had an ejection fraction of less than 40%. Coronary arteriography was performed in 17 group 1 patients and 17 group 2 patients. In all cases it was normal.

Aortic Valve Replacement

Thirty-six patients (75%) received a Björk-Shiley tilting-disc prosthesis. Among the other patients, six received a Hancock xenograft, two a Starr-Edwards ball-valve prosthesis, two an Angell-Shiley tissue valve and one patient a St. Jude Medical prosthesis. Myocardial protection during surgery was provided by coronary artery perfusion and topical hypothermia in 25 patients (54%) and by cold potassium cardioplegia and topical hypothermia in 22 (46%). The extracorporeal circulation time and the aortic cross-clamping time were similar in both groups.

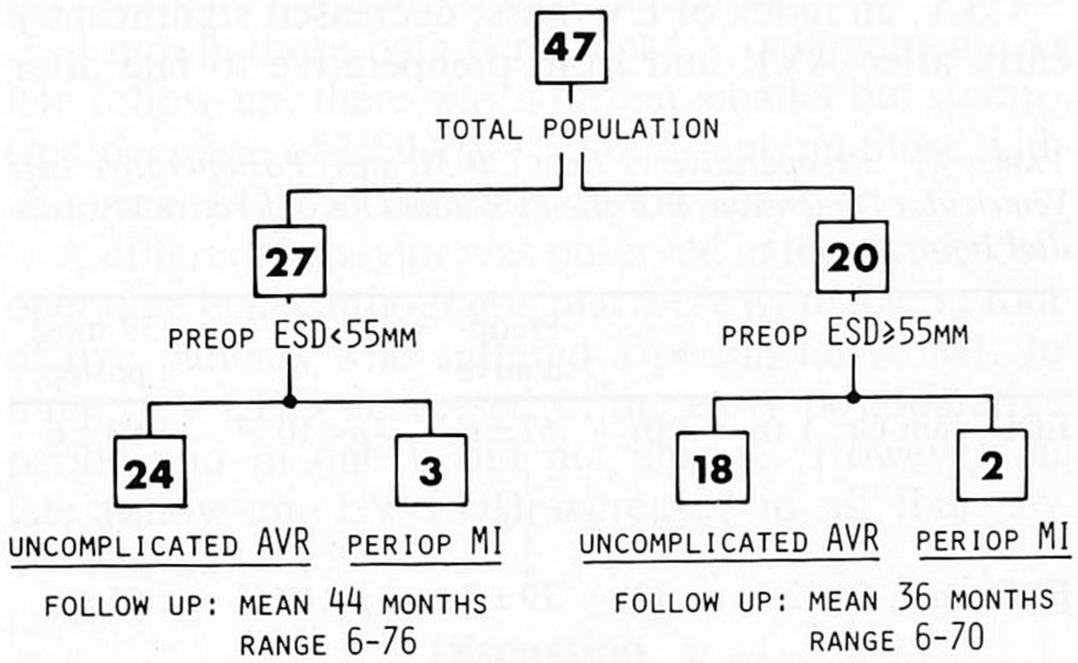

FIGURE 1. *Study population divided according to preoperative end-systolic dimension (ESD) determined echocardiographically. AVR = aortic valve replacement; MI = myocardial infarction.*

TABLE 1. *Clinical Preoperative Data*

		Group 1	Group 2
No. of pts		27	20
Age (years) (mean, range)		45 (22–75)	49 (22–65)
Sex (M/F)		16/11	13/7
Etiology			
Rheumatic		8	4
Endocarditis		6	5
Marfan		1	1
Lues		—	2
Ankylosing spondylitis		—	1
Unknown		12	7
Functional class	I	—	—
	II	20	7
	III	5	13
	IV	2	—

Group 1 — preoperative end-systolic dimension < 55 mm; group 2 — preoperative end-systolic dimension ⩾ 55 mm.

Follow-up

Follow-up data were obtained by serial outpatient clinic visits. All patients had at least one good-quality echocardiogram postoperatively. In 27 patients, an

TABLE 2. *Preoperative Echocardiographic and Catheterization Data (mean ± SD)*

	Group 1	Group 2	*p* (paired *t* test)
Echocardiographic data			
EDD (mm)	67 ± 7	82 ± 6	$<10^{-9}$
ESD (mm)	44 ± 6	62 ± 7	$<10^{-10}$
FS (%)	33 ± 6	24 ± 6	$<10^{-5}$
CSA (cm^2)	24 ± 6	32 ± 8	<0.001
Catheterization data			
LVEDP (mm Hg)	19 ± 8	18 ± 8	NS
LVEDVI (ml/m^2)	147 ± 43	247 ± 42	$<10^{-5}$
LVESVI (ml/m^2)	68 ± 26	141 ± 48	$<10^{-6}$
LVEF (%)	54 ± 7	42 ± 9	$<10^{-4}$
LVMI (g/m^2)	215 ± 77	354 ± 151	$<10^{-3}$

Abbreviations: EDD = left ventricular (LV) end-diastolic dimension; ESD = LV end-systolic dimension; FS = LV fractional shortening; CSA = LV cross-sectional area; LVEDP = LV end-diastolic pressure; LVEDVI = LV end-diastolic volume index; LVESVI = LV end-systolic volume index; LVEF = LV ejection fraction; LVMI = LV mass index.

echocardiogram was performed early postoperatively (average 3 months, range 2–6 months) and late postoperatively (mean 36 months, range 12–67 months). Six others had only late postoperative echocardiograms.

Data Analysis

Analysis of variance was performed to analyze serial changes in echocardiographic dimensions in patients with three observations. A paired *t* test was used to compare preoperative with early and late postoperative measurements and an unpaired *t* test to compare the values between the two groups.

Results

No patient died during follow-up (average 41 months, range 6–76 months).

Perioperative Myocardial Infarctions

Five patients (10%) had a perioperative myocardial infarction (MI). The diagnosis was based on the development of pathologic Q waves. Three of the MIs were in group 1 and two in group 2; therefore, the incidence of perioperative MI was the same in both groups. There was no significant difference between the five patients with perioperative MI and those with uncomplicated AVR with respect to preoperative echocardiographic, hemodynamic and angiographic values. All patients with MI had myocardial protection with coronary perfusion. Since we started myocardial protection with cold potassium cardioplegia, no further MI occurred in this series of patients. Aortic cross-clamping time and extracorporeal circulation time were similar in the five patients with MI and those without MI.

Subjective Results

The subjective results of surgery were evaluated using the New York Heart Association (NYHA) functional classification (fig. 2). Of the five patients with perioperative MI, three were in NYHA functional class II preoperatively and two in class III. During an average follow-up of 43 months (range 21–55 months), all five were alive; three were in class I and two were in class II.

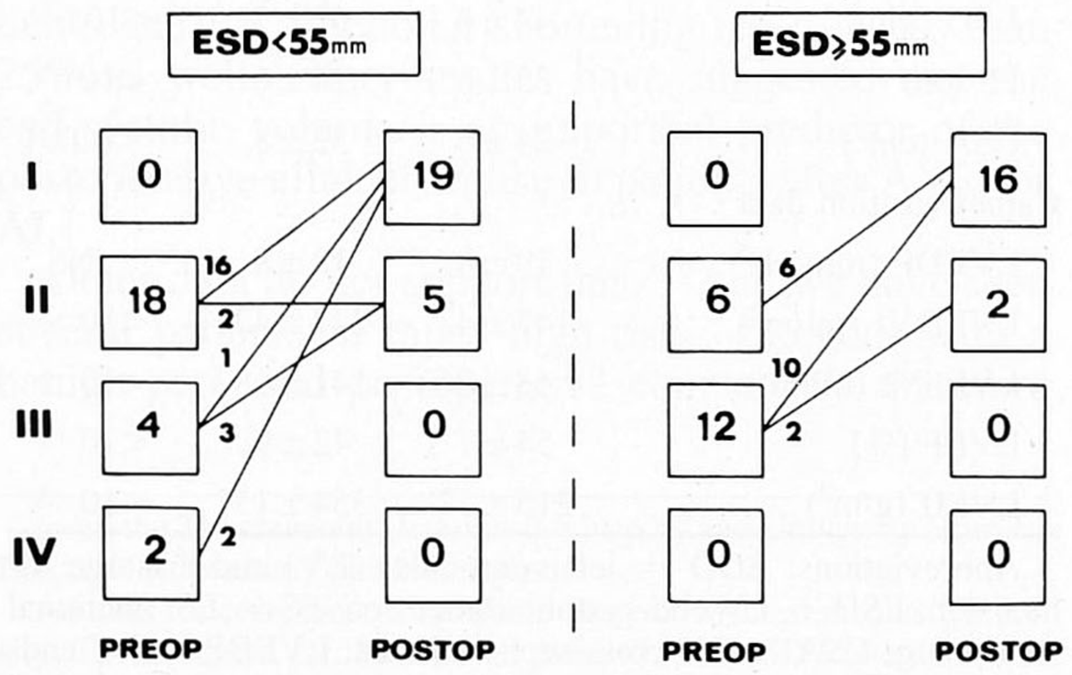

FIGURE 2. *Pre- and postoperative functional class (New York Heart Association classification) in patients without perioperative myocardial infarction. ESD = end-systolic dimension.*

TABLE 3. *Preoperative vs Early (3 Months) Postoperative Left Ventricular Dimensions in Patients Without Perioperative Myocardial Infarction*

	Preop		3 mos. postop
Group 1 EDD (mm) (n = 21)	66 ± 6	$p<10^{-6}$	53 ± 8
	$p<10^{-7}$		$p<10^{-3}$
Group 2 EDD (mm) (n = 15)	88 ± 6	$p<10^{-6}$	63 ± 7
Group 1 CSA (cm^2) (n = 21)	24.3 ± 4.7	$p<0.007$	21.2 ± 6.8
	$p<0.02$		NS
Group 2 CSA (cm^2) (n = 15)	32.2 ± 9.1	$p<0.003$	22.7 ± 3.7

Preoperataive vs Postoperative Echocardiographic Evaluation

Echocardiographic data for the five patients suffering a perioperative MI were not included in the analysis of the other patients so as not to obscure the independent role of preoperative LV dimensions on postoperative results.

In 36 out of the 42 patients without perioperative MI, a good-quality echocardiogram was performed in the "early" postoperative period (average 3 months, range 2–6 months). Of these, 21 were in group 1 and 15 in group 2 (table 3). In both groups, EDD decreased significantly after AVR. Both preoperative and postoperative EDDs were significantly higher in group 2 than in group 1.

In 33 patients, "late" postoperative echocardiograms (mean 39 months, range 12–75 months) were performed. Late postoperative EDDs were compared in the two groups and were not statistically different (table 4). This lack of significant difference in the late postoperative LV EDD may have been due to the lack of late postoperative echocardiograms in some patients with very large preoperative EDDs in group 2. However, all of these patients showed a sizable decrease in LV EDD at the early postoperative study (table 5).

CSA, an index of LV mass, decreased significantly early after AVR and from preoperative to late after

TABLE 4. *Preoperative vs Late (39 Months) Postoperative Left Ventricular Dimensions in Patients Without Perioperative Myocardial Infarction*

	Preoperative		39 mos. postop
EDD, mm Gr. 1 (n = 20)	67 ± 6	$p<10^{-7}$	53 ± 6
	$p<10^{-5}$		NS
EDD, mm, Gr. 2 (n = 13)	79 ± 3	$p<10^{-7}$	55 ± 6
CSA, cm^2, Gr. 1 (n = 20)	25.2 ± 5.0	$p<10^{-4}$	19.9 ± 5.3
	$p<0.02$		NS
CSA, cm^2, Gr. 2 (n = 13)	32.1 ± 9.8	$p<10^{-3}$	20.0 ± 5.5

Values are mean ± SD.

TABLE 5. *Preoperative and Postoperative Echocardiographic Measurements after Aortic Valve Replacement in Nine Patients without Preoperative Myocardial Infarction in Whom Only An Early (2–6 Months) Postoperative Echocardiogram Was Performed*

	Preoperative			Early postop		Postop NYHA class	Follow-up (months)
	ESD (mm)	FS (%)	EDD (mm)	EDD (mm)	ΔEDD (mm)		
Group 1	45	30	65	50	15	II	41
	40	38	60	55	5	I	9
	50	28	70	42	28	II	41
	52	23	68	55	13	I	6
Group 2	60	29	85	50	35	I	42
	60	26	82	70	12	I	9
	85	15	100	68	32	II	6
	68	24	90	58	32	I	6
	63	28	88	70	18	I	6

Abbreviations: ESD = end-systolic dimension; FS = fractional shortening; EDD = end-diastolic dimension; ΔEDD = decrease in EDD after aortic valve replacement; NYHA = New York Heart Association functional classification.

AVR (table 3 and 4). Preoperatively, the CSA was significantly higher in group 2, but postoperatively there was no difference between the two groups. In the 33 patients who had late postoperative echocardiograms, the postoperative change in CSA correlated with preoperative values ($r = 0.75$, $p < 0.0001$).

In nine of the 42 patients without perioperative MIs, only an early postoperative echocardiogram was performed (four in group 1 and five in group 2) (table 5). The patient with the smallest preoperative EDD (60 mm) showed the smallest postoperative decrease (5 mm), but in the others EDD decreased 12–35 mm.

Serial Postoperative Echocardiographic Measurements (fig. 3).

Early and late postoperative echocardiograms were available in 27 patients. We divided these patients into two subgroups: 11 who normalized their EDD ($\leq$ 55 mm) in the early postoperative phase and 16 who showed a persistent LV enlargement. LV EDD decreased significantly in both groups in the early postoperative period, from 66 ± 7 to 48 ± 5 mm in patients who normalized EDD and from 73 ± 7 to 63 ± 4 mm in those with persistent LV enlargement. At late follow-up, there was a further smaller but significant decrease of EDD ($p < 0.002$) only in those with abnormal EDD in the early follow-up.

A different behavior was observed in the serial postoperative echocardiograms that were available in four of five patients who suffered a perioperative MI. In three, LV EDD decreased in the early postoperative period and in one it did not change. However, at late follow-up, LV EDD increased in all four, by 10–18 mm.

Discussion

If M-mode echocardiography allowed identification of a high-risk subgroup of syptomatic patients with AI, it could also be used to determine the timing for AVR in patients with asymptomatic AI. Henry et al.[6] demonstrated that a preoperative ESD $\geq$ 55 mm carried a significantly higher risk of perioperative death, myocardial damage or late death from congestive heart failure. An LV FS of less than 25% was an additional risk factor in these patients.

Our results do not support these findings. In our series, no patient died perioperatively or postoperatively and the number of patients and the duration of postoperative follow-up were similar to those in the study of Henry et al.[6]

The 0% mortality in our series is probably a chance statistical variation. Such a variation must not be considered exceptional, as larger series of AVRs without perioperative deaths have been reported.[13] Furthermore, of the 58 consecutive isolated AVRs for aortic stenosis, AI or a mixed lesion performed at our institution in 1981 (after the completion of the present study),

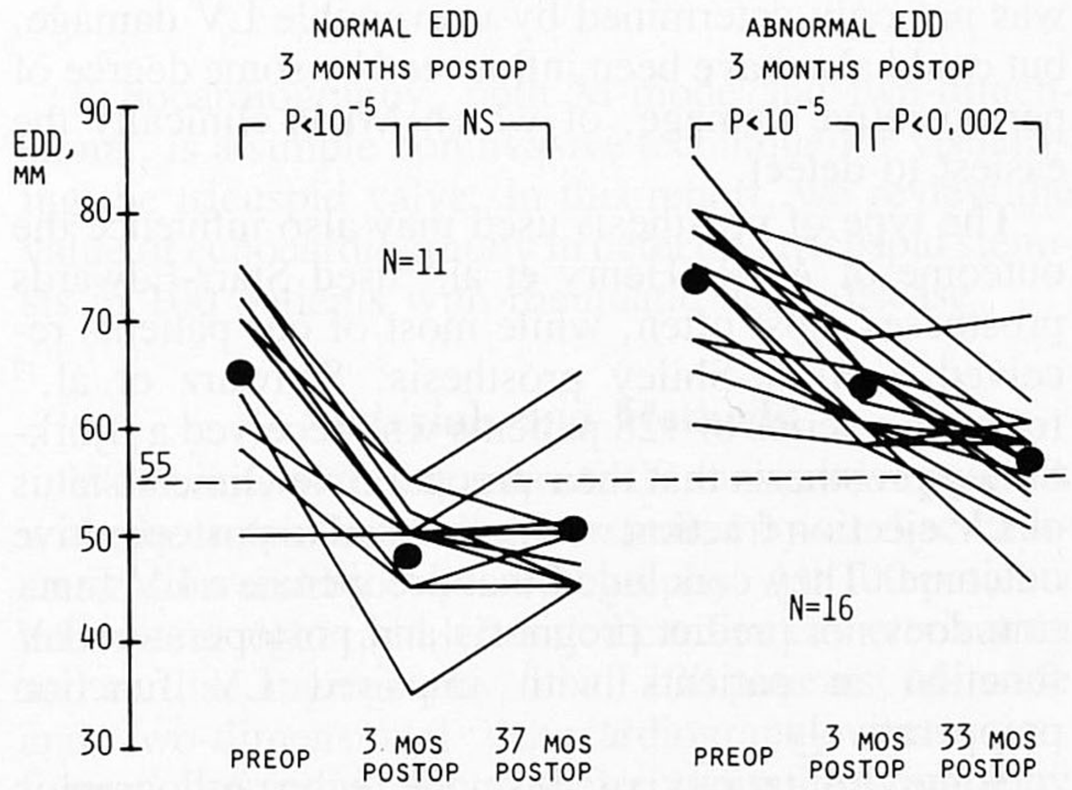

FIGURE 3. *Pre- and serial postoperative left ventricular end-diastolic dimension (EDD) in patients who normalized ($\leq$ 55 mm) EDD in the early postoperative phase and in those with persistent early postoperative left ventricular enlargement. Although EDD did not further change in the long term postoperative phase in the first group, it further significantly decreased in those with persistent early postoperative left ventricular enlargement.*

only one patient died perioperatively (within 1 month after surgery).

Perioperative MI occurred in 10% of our patients, but its distribution was not different in patients in group 1 or 2 (fig. 1). The high-risk subgroup in our series defined by using the criteria proposed by Henry et al.[6] did not have a bad prognosis: no patient died and two of 20 had a perioperative MI. Three factors might explain this difference: the relatively high prevalence of coronary artery disease in the series of Henry et al.; the intraoperative myocardial protection provided by cold potassium cardioplegia in half of our cases; and the different types of prostheses used in the two series.

None of our patients had significant coronary artery disease, but 20% of the patients reported by Henry et al.[6] did. However, our data do not indicate whether the presence of coronary artery disease plays a major prognostic role in AVR.[3, 12]

There is little doubt that cold potassium cardioplegia decreases the risk of perioperative myocardial damage.[13, 14] Better myocardial preservation may explain the good results of AVR in our series. This is especially important in patients with hypertrophied ventricles, who have a higher risk of perioperative myocardial damage. None of the patients in the series reported by Henry et al.[6] had surgery with cold potassium cardioplegia, while 46% of our patients did. Furthermore, our patients were operated upon more recently, and improved surgical techniques may have influenced the results.[15]

The mechanism of perioperative MI is most likely ischemic damage, and hypertrophied ventricles are most vulnerable.[16] All five of our perioperative infarcts occurred in patients who underwent AVR before cold potassium cardioplegia was introduced, and this did not occur in any of our patients afterwards. This leads us to suggest that the outcome observed by Henry et al. was not only determined by irreversible LV damage, but could also have been influenced by some degree of perioperative damage, of which MI is clinically the easiest to detect.

The type of prosthesis used may also influence the outcome of AVR. Henry et al.[6] used Starr-Edwards prostheses most often, while most of our patients received a Björk-Shiley prosthesis. Schwarz et al.[17] found in a series of 128 patients who received a Björk-Shiley prosthesis that their preoperative clinical status or LV ejection fraction was not related to postoperative outcome. They concluded that preoperative LV function does not predict prognosis and postoperative LV function in patients with impaired LV function preoperatively.

Some limitations of M-mode echocardiographic measurements in patients with severe AI[2, 18, 19] must be discussed, especially when the measurements are used to choose the optimal moment for operation in asymptomatic or minimally symptomatic patients. Preoperative echocardiographic measurements of the left ventricle in severe AI must be interpreted with caution. LV dimensions may vary by as much as 20%, depending on angulation of the transducer and the position of the patient.[2] A day-to-day variability is inherent to any technique. We studied the measurement-to-measurement variability in a series of 89 normal subjects by repeating an M-mode echocardiogram on the following day. The standard deviation of the measurement variability (ie., within-patient) was 2 mm for LV EDD and of the same magnitude for LV ESD. It is unlikely that day-to-day variability of the LV measurements significantly influenced our conclusions.

Despite agreement between echocardiographic and angiographic measurements,[19] we found a large standard error of the estimate, probably because of LV shape abnormalities in AI patients. Our data are consistent with those from earlier studies from our laboratory.[20] For the series reported here, the correlation coefficient between preoperative LV end-diastolic volume calculated from right anterior oblique cineangiocardiograms and LV EDD from M-mode echocardiograms was $r = 0.62$ ($p < 0.001$) and the SEE was 116 ml, representing a very large variability, up to 30% of the mean angiographic end-diastolic volume.

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Value of Two-dimensional Echocardiography in Detecting Tricuspid Stenosis

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SUMMARY **We reviewed the M-mode and two-dimensional echocardiograms of 100 consecutive patients with rheumatic heart disease. All were subsequently studied by cardiac catheterization and angiography. In four patients, cardiac catheterization showed tricuspid stenosis (average mean diastolic gradient 6.2 mm Hg), which was confirmed during cardiac surgery. M-mode echocardiography showed a diminished EF slope in 12 patients (mean 26 mm/sec), including the four patients with tricuspid stenosis. Seven of the eight patients without tricuspid stenosis had significant pulmonary hypertension; the reasons for the diminished EF slope in the other patient could not be identified. Tricuspid stenosis was diagnosed in four patients from two-dimensional echocardiograms on the basis of diastolic doming and restricted leaflet motion of the tricuspid valve. These four patients were the same patients in whom tricuspid stenosis was diagnosed by cardiac catheterization. We conclude that two-dimensional echocardiography is useful in the diagnosis of tricuspid stenosis.**

TRICUSPID STENOSIS may be a difficult diagnosis and can be easily missed on clinical examination[1–5] and on cardiac catheterization. It is most often caused by rheumatic heart disease[2, 3, 6] though it has been described in systemic lupus erythematosus,[3] carcinoid syndrome,[7] Loeffler's endocarditis,[8] metastatic melanoma[9] and as a congenital anomaly.[10] The incidence of significant tricuspid stenosis in rheumatic heart disease has been estimated to be about 3–5% on the basis of data from autopsy and cardiac catheterization studies.[11, 12] The diagnosis of triscupid stenosis is important because undetected and uncorrected tricuspid stenosis increases operative morbidity and mortality of cardiac surgery for left-sided cardiac valvular disease and worsens the prognosis for those who survive.[5, 13, 14]

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Echocardiography, both M-mode and two-dimensional, is a simple noninvasive technique for visualizing the tricuspid valve. In this report, we review the value of echocardiography in detecting tricuspid stenosis in 100 patients with rheumatic heart disease.

Materials and Methods

We reviewed the records of 100 consecutive patients with rheumatic heart disease studied by cardiac catheterization and by echocardiography. All 100 patients underwent right- and left-sided cardiac catheterization and cardiac angiography. In all 100 patients, M-mode and two-dimensional echocardiography were performed before cardiac catheterization. Angiography was performed in the usual manner.[4] Simultaneous pressure across the tricuspid valve was obtained through a double-lumen catheter. Pullback pressure tracings were also obtained across the tricuspid valve. The mean pressure gradient across the tricuspid valve was calculated by planimetry.

M-mode echocardiography was performed using an Ekoline 20-A recorder. A 2.25-MHz transducer was