Dicrotic Pulse After Open Heart Surgery

ROBERT CHARLES ORCHARD, M.D., AND ERNEST CRAIGE, M.D.

SUMMARY Pre- and postoperative echocardiograms (EPGs) and preoperative hemodynamic data of 108 patients who underwent valve replacement were reviewed to establish the frequency and significance of a dicrotic pulse (DP) postoperatively. DP occurred almost exclusively in patients who underwent valve replacement for regurgitant lesions (20 of 28 with aortic regurgitation, nine of 25 with mitral regurgitation, and four of six with both aortic and mitral regurgitation). These patients were divided into dicrotic and nondicrotic groups. Preoperatively, the dicrotic group had significantly larger end-diastolic volumes (p < 0.01) and end-systolic volumes (p < 0.01) and significantly lower ejection fractions (p < 0.01). Echocardiographically, the dicrotic group had larger left ventricular dimensions, both systolic (p < 0.01) and diastolic (p < 0.05), reduced percentage fractional shortening of the left ventricular cavity (p < 0.01) and poor thickening properties of the left ventricular posterior wall (%ΔTH-LVPW) (p < 0.01). Postoperatively the dicrotic group had a slightly larger end-diastolic dimension (p = NS) and markedly depressed %ΔTH-LVPW (p < 0.001) compared with the nondicrotic group.

On follow-up EPG the persistence of a DP correlated with continued left ventricular dysfunction by echocardiographic and hemodynamic studies and an extremely poor clinical course. DP after valve replacement is therefore an important prognostic sign.

A DICROTIC PULSE is characterized by two palpable pulsations (fig. 1). As opposed to anacrotic and bisferiens pulses, in which the two pulsations are systolic in timing, the second pulsation of the dicrotic pulse is diastolic, immediately after the dicrotic notch.

The dicrotic pulse has long been recognized, particularly as a feature of febrile states such as typhoid fever. More recently the dicrotic pulse has been associated with a variety of cardiac diseases that have in common a low cardiac output. These include cardiomyopathies, pericardial tamponade, constrictive pericarditis and pulmonary embolism. There is only one report, however, of dicrotism after open heart surgery, and the significance of this finding is unknown. At our institution, pre- and postoperative echocardiograms are routinely performed on most adult patients who undergo cardiac surgery, so we had the opportunity to observe the frequent, although not uniform, presence of a dicrotic pulse after open heart surgery. The present study was undertaken to establish the frequency and significance of this clinical sign in patients who undergo cardiac surgery for valvular disease.

Methods

Subjects

The patients were identified by a computerized data retrieval system as having undergone pre- and postoperative echocardiograms for valvular surgery between January 1973 and December 1978. The patients studied were all age 45 years or younger, and any patient who developed prosthetic malfunction was excluded. After technically inadequate carotid pulse tracings were excluded, 108 patients remained. Postoperative tracings were performed 7–10 days after surgery.

Echocardiography

Phonocardiograms were obtained from the four standard precordial areas using either an Irex 101 or a Cambridge MC IV multichannel recorder and Leatham suction microphones. Indirect carotid artery pulse recordings were made with an air-filled funnel.
2.5 cm in diameter that was connected to a pulse microphone by a 10-cm polyethylene tubing. Because the level of the dicrotic notch is influenced by the time constant of the apparatus, the transducer-recording systems must have time constants of longer than 3 seconds.5,6 Carotid artery recordings were performed with the patient in the supine position, with a pillow under the shoulders. The head was slightly extended but not turned.

Echocardiograms were recorded with either a Smith Kline Ekoline 20-A ultrasonoscope interfaced with the Cambridge recorder or an Irex 101 ultrasonoscope interfaced with the Irex recorder. Echoes of the interventricular septum, left ventricular posterior wall and mitral valve or mitral prosthesis were obtained by scanning methods previously described.7 A lead II of the ECG was recorded simultaneously with the echophonocardiogram. Paper speed was set at 100 mm/sec for phono and pulse recordings. All records were processed by a photographic method to ensure clarity of detail.

Measurements

Diagnosis of a Dicrotic Pulse

In figure 1, a normal carotid pulse tracing is compared with a dicrotic pulse. On the dicrotic pulse, the dicrotic notch reaches almost to the level of the baseline and is followed by a large diastolic wave. Establishing a standard for the diagnosis of dicrotism is difficult, but for purposes of this study, a dicrotic pulse was defined as one in which the nadir of the dicrotic notch falls at or below 20% of the height of the pulse wave (d/t ≤ 20%) and in which the dicrotic wave is greater than 20% of t (d/t ≥ 20%). This is in keeping with previous work by Meadows et al.8 Systolic time intervals were measured from the carotid pulse recording and corrected for pulse rate by the method of Weissler and Garrard.9

The left ventricular echocardiographic dimensions were measured during the left ventricular volume study at a level just below the free edge of the mitral leaflets. The end-systolic dimension (ESD) was measured at the point of maximal anterior excursion of the left ventricular posterior wall (LVPW) during systole and the end-diastolic dimension (EDD) was measured at the peak of the R wave of the ECG. Left ventricular posterior wall thickness (Th-LVPW) was measured at end-diastole from the leading edge of the epicardial echo to the leading edge of the endocardial echo. The septal thickness (Th-S) was measured from the leading edge of the right septal echoes to the leading edge of the left septal echoes.

The percent fractional shortening of the left ventricular cavity (%ΔD) was calculated as

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

The fractional increase in thickness of both the septum and posterior wall (%ΔTh) was calculated as:

$$\frac{\text{Th(es)} - \text{Th(ed)}}{\text{Th(ed)}} \times 100$$

where Th(ed) is wall thickness at end-diastole and Th(es) is wall thickness at end-systole.

The interventricular septal motion was considered to be normal if the septum moved posteriorly more than 2 mm from end-diastole to end-systole, paradoxical if it moved anteriorly more than 2 mm from end-diastole to end-systole and hypokinetic if it moved less than 2 mm in either direction from end-diastole to end-systole.9 All echocardiographic measurements were made independently by two trained echocardiographers.

Cardiac Catheterization and Angiography

Right- and left-heart catheterization had been performed on all patients, following the routine of this hospital. Left ventricular and pulmonary artery wedge pressures were recorded with fluid-filled catheters. Cardiac output was measured by the Fick method. Left ventriculography was performed using either biplane 35-mm cineangiography (posteroanterior and lateral) or single-plane 35-mm cineangiography in the right anterior oblique projection. Left ventricular volumes were determined by planimetry using the area-length method of Dodge and co-workers10 and Kasser and Kennedy.11

Statistical Methods

A generalized two-way analysis of variance (using indicator variables for the lesion type and carotid fac-
tors in a multiple regression linear model) was used to assess the differences between the dicrotic and nondicrotic carotid groups. Differences between these two groups with respect to both sex and age distributions, were adjusted for when appropriate (by including these factors as covariates in the above model). Also, preliminary models, which indicated a lesion by carotid interaction term, were used to test the significance of these interactions.

Results

Patients were divided into two groups: those who developed a dicrotic pulse postoperatively (dicrotic group) and those who did not (nondicrotic group). As the hemodynamic and echocardiographic variables of the dicrotic group were statistically similar regardless of whether the initial diagnosis was aortic regurgitation, mitral regurgitation or combined aortic and mitral regurgitation (the lesion type of carotid interaction was not significant $[p > 0.10]$ for each variable), these diagnostic subgroups are merged in the subsequent discussion for brevity. This was also done for the nondicrotic group.

Incidence of Dicrotism

The incidence of postoperative dicrotism in various conditions subjected to valve replacement is given in figure 2. The development of a dicrotic pulse was confined almost exclusively to patients who underwent valve replacement for regurgitant lesions. Aortic regurgitation was the most common preoperative condition that led to this result: 20 of 28 patients who underwent aortic valve replacement developed a dicrotic pulse. Mitral regurgitation (nine of 25) and mitral plus aortic regurgitation (four of six) were also associated with dicrotism after valve replacement. Patients who underwent valve replacement for stenotic lesions, whether mitral or aortic, rarely developed a dicrotic pulse after surgery. One patient developed dicrotism after mitral commissurotomy.

Only two patients, both of whom had mitral valve replacement performed for mitral regurgitation, were considered by our criteria to have a dicrotic pulse preoperatively. In each case, the dicrotic pulse was more marked postoperatively. Dicrotism was rare after valve replacement for stenotic lesions (fig. 2), so the following discussion is confined to patients who underwent valve replacement for regurgitant lesions.

Hemodynamic Studies

The preoperative hemodynamic studies revealed a striking difference in the left ventricular angiographic volumes between the dicrotic and nondicrotic groups (fig. 3, table 1). The difference was manifest in both the end-systolic volumes (dicrotic group $98.0 \pm 37.5$ ml/m² vs nondicrotic group $44.6 \pm 23.1$ ml/m²) ($p < 0.01$) as well as the end-diastolic volumes (dicrotic group $217 \pm 69.1$ ml/m² vs nondicrotic group $128.4 \pm 49.2$ ml/m²) ($p < 0.01$). Of the other preoperative hemodynamic measurements, the left ventricular ejection fraction was also different in the two groups. It was significantly lower in the dicrotic group ($53.5 \pm 12.2\%$ vs $65.7 \pm 12.4\%$) ($p < 0.01$).

Echocardiographic Results

Echocardiograms were reviewed in all patients in the dicrotic and nondicrotic groups (fig. 4, table 1). The ESD of the dicrotic group was considerably larger preoperatively ($2.78 \pm 0.50$ cm/m² vs $2.07 \pm 0.25$ cm/m²) ($p < 0.01$), as was the EDD ($4.05 \pm 0.60$ cm/m² vs $3.49 \pm 0.55$ cm/m²) ($p < 0.05$). Postoperatively the EDD was slightly larger in the dicrotic group, but not significantly so. The fractional shortening of the ventricular cavity ($\%\Delta D$) was depressed in the dicrotic group preoperatively ($p < 0.01$). The $\%\Delta D$ and ESD could not be measured in either group in the immediate postoperative period because of the high incidence of paradoxical septal motion. The fractional increase in thickening of the LVPW ($\%\Delta Th$) was lower in the dicrotic group preoperatively.
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TABLE 1. Preoperative Hemodynamic and Pre- and Postoperative Echocardiographic Variables for Dicrotic and Nondicrotic Groups

<table>
<thead>
<tr>
<th></th>
<th>Preop dicrotic</th>
<th>Preop nondicrotic</th>
<th>Postop dicrotic</th>
<th>Postop nondicrotic</th>
</tr>
</thead>
<tbody>
<tr>
<td>E/e (cpm/ml)</td>
<td>23.0 ± 11.1</td>
<td>33.0 ± 11.1</td>
<td>23.0 ± 11.1</td>
<td>33.0 ± 11.1</td>
</tr>
<tr>
<td>EF (%)</td>
<td>89.0 ± 37.5</td>
<td>53.5 ± 12.2</td>
<td>89.0 ± 37.5</td>
<td>53.5 ± 12.2</td>
</tr>
<tr>
<td>E/DV (cm/m2)</td>
<td>27.0 ± 11.1</td>
<td>128.4 ± 32.3</td>
<td>27.0 ± 11.1</td>
<td>128.4 ± 32.3</td>
</tr>
<tr>
<td>ESV (%)</td>
<td>19.0 ± 11.1</td>
<td>19.0 ± 11.1</td>
<td>19.0 ± 11.1</td>
<td>19.0 ± 11.1</td>
</tr>
<tr>
<td>IVC (cm/m2)</td>
<td>1.0 ± 1.0</td>
<td>1.0 ± 1.0</td>
<td>1.0 ± 1.0</td>
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*p < 0.01
**p < 0.001

EDV = end-diastolic volume; ESV = end-systolic volume; EF = ejection fraction; IVC = inferior vena cava diameter; E/e = ratio of early to late mitral inflow velocities; E/DV = E/e corrected for body surface area (BSA).

FIGURE 3. Preoperative angiographic left ventricular volumes for dicrotic (closed circles) and nondicrotic (open circles) groups. The mean of each group is given. EDV = end-diastolic volume; ESV = end-systolic volume.

(p < 0.01). Postoperatively, the values in the dicrotic group were markedly depressed (28.6 ± 7.2%) and the values of the nondicrotic group were in the normal range (63.1 ± 13.1%) (p < 0.001) (fig. 5).

Paradoxical septal motion after surgery was present.

FIGURE 4. Preoperative echocardiographic left ventricular dimensions of dicrotic (closed circles) and nondicrotic (open circles) groups. The mean of each group is given. EDD = end-diastolic dimension; ESD = end-systolic dimension, corrected for body surface area (BSA).
in the majority of patients in each group (dicrotic 89%, nondicrotic 76%). The heart rate was not significantly different between the two groups postoperatively.

The patients in the dicrotic group were younger (25.3 ± 8.7 years vs 31.2 ± 8.5 years) and had a higher proportion of males (19 of 32 vs 10 of 25) than the nondicrotic group. Systolic time intervals postoperatively revealed that the left ventricular ejection time index was similar, with the pre-ejection period/left ventricular ejection time ratio slightly but not significantly lower in the dicrotic group (0.48 ± 0.13 vs 0.57 ± 0.13) (p > 0.10).

Follow-up tracings were obtained for 16 patients at various intervals (2 months to 3½ years) after surgery. Figure 6 shows a sequence of carotid pulse patterns on a patient undergoing mitral valve replacement for mitral regurgitation. Preoperatively, the carotid tracing was normal, but immediately postoperatively a marked dicrotic pulse was present. On follow-up 1 year later, the carotid pattern had returned to normal.

Figure 7 shows the results of follow-up studies with %ΔTh-LVPW, which was used as an index of myocardial contractility. Five patients were initially nondicrotic and remained nondicrotic on follow-up. Seven patients who were initially dicrotic reverted to normal on follow-up. In each case this was associated with an improvement of the %ΔTh. Four patients had persistent dicrotism and continued to display poor thickening properties and left ventricular dilatation, and had an extremely poor clinical course. Repeat catheterization on two of these patients confirmed extremely poor

![Figure 5](https://example.com/figure5.png)

**Figure 5.** Pre- and postoperative percentage thickening of the left ventricular posterior wall (%ΔTh-LVPW) by echocardiography for dicrotic (closed circles) and nondicrotic (open circles) groups. The mean of each group is indicated.

![Figure 6](https://example.com/figure6.png)

**Figure 6.** A series of carotid pulse tracings on one patient who underwent mitral valve replacement for mitral regurgitation. A markedly dicrotic pulse present immediately postoperatively had reverted to normal at follow-up at 1 year. PCG-PA = phonocardiogram pulmonary area; PCG-MA = phonocardiogram mitral area.
left ventricular function. Three of these four patients have died of congestive heart failure.

**Discussion**

The dicrotic arterial pulse has been recognized by clinicians since 1863 with the development of Marey’s sphygmograph. In 1881, Flemming suggested that conditions necessary for the development of a dicrotic pulse included a fast pulse, elastic vessels, feeble tension in the vessels and a small amount of fluid injected with each ventricular systole. In 1902, MacKenzie reiterated that dicrotism was usually present when ventricular contraction was weak and the vessels were relaxed. One of his illustrations showed a dicrotic pulse in a patient with typhoid fever. Paul Wood also noted dicrotic pulses in patients with infectious or toxic conditions and considered it a sign of vascular relaxation. More recently, dicrotism has been associated with cardiomyopathy. Ewy et al. in 1969 described nine patients with advanced myocardial failure, principally attributed to cardiomyopathy, who displayed a dicrotic pulse on physical examination that was confirmed by direct and indirect arterial recordings. In 1972, Meadows et al. described a large series of patients with dicrotic pulses demonstrated by direct brachial artery recordings. Most of these patients suffered from cardiomyopathy. Barner et al. were the first to study dicrotism after open heart surgery, and found that a dicrotic pulse frequently appeared after correction of aortic regurgitation (34% vs 0.5% for aortic stenosis). They suggested that there might have been some accommodation of the arterial system to aortic regurgitation such that its reservoir function would have been enhanced. Correction of aortic regurgitation could result in a dicrotic pulse.

The causes of a dicrotic pulse are not completely understood. Apparently, however, important among the factors involved are the contractile state of the ventricle, the competence of the aortic valve and the state of the systemic vascular tree. The normal dicrotic wave has been ascribed to the rebound of blood against a closed aortic valve. A competent aortic valve appears to be necessary for its development, as suggested by the fact that one patient in the study by Barner et al. and two patients in our study lost their initially large dicrotic waves when a paravalvular aortic leak developed. Age also appears to be an important factor in the genesis of the dicrotic pulse. Meadows et al. found that most of their patients who had dicrotism were younger than 45 years of age. Thus, elastic arterial walls are apparently necessary for the development of a large dicrotic wave, so the patients in our study were restricted to those younger than 45 years. Meadows et al. considered tachycardia an important factor. Ewy et al. reported that the dicrotic pulse was found in the presence of raised systemic vascular resistance. They suggested that earlier observations regarding the association of dicrotism and conditions usually thought to be associated with arterial relaxation, such as typhoid fever, could have been made during a phase of the dis-
ease complicated by myocarditis or a low stroke volume secondary to dehydration.

The contractile state of the myocardium also appears to be important in the genesis of a dicrotic pulse. Post premature beats, in the absence of hypertrophic subaortic stenosis, are less dicrotic than control beats. In the presence of pulsus alternans, the weaker beats are more dicrotic than the stronger beats. Dicrotism has been described in pericardial tamponade and thought to be on the basis of a low stroke volume.2

The similarity in the pulse-wave pattern in cardiomyopathy and other conditions mentioned above characterized by poor ventricular function, and our patients whose surgery had been necessitated by volume overload situations suggests that the common denominator in these apparently dissimilar groups might lie in indexes of myocardial performance. A dicrotic wave after open heart surgery was present almost exclusively in patients who underwent valve replacement for regurgitant lesions and rarely after valve replacement for stenotic lesions. This observation is consistent with the previous studies of Kennedy et al., who found that patients with aortic valve obstruction and a low ejection fraction may be expected to improve after surgery, while those with aortic regurgitation and low ejection fraction may show less improvement after surgery.18 Several other studies in man have emphasized that left ventricular dilatation or hypertrophy resulting from volume overload imposed by mitral or aortic regurgitation may be slow to regress after valve surgery or may fail to improve.18 Gault et al. showed that in five patients after correction of free aortic regurgitation, there was no improvement in myocardial contractility as measured by tension-velocity-length indexes.23 Papadimtriou et al. found that in dogs with left ventricular volume overload and failure induced by aorta-to-inferior vena cava fistulas, dilatation and profound ultrastructural changes persisted in the left ventricular muscle for at least 3 months after correction.18

Hemodynamic measurements that appear to predict development of a dicrotic pulse in the postoperative period include end-systolic volume, end-diastolic volume and percent ejection fraction. Grossman et al.,19 in a series of patients evaluated by pressure-volume relationships, suggested that end-systolic volume showed good discrimination of normal from poor contractile function as reflected in the ejection fraction. Mitchell et al.20 found that at a constant inotropic state, the left ventricle always appeared to return to the same ESD regardless of the EDD. Borow et al.21 observed that end-systolic volume was unique as a predictor of postoperative left ventricular function in volume overload from valvular regurgitation.

Schuler et al.22 showed that in patients who undergo mitral valve replacement for chronic mitral regurgitation, the presence preoperatively of a low-normal or depressed shortening fraction, in association with large increases in EDD (greater than 7.0 cm) and ESD (greater than 5.0 cm) portended progressive deterioration of left ventricular function and no decrease in chamber size after surgery.

Echocardiographically, the larger preoperative dimensions of the dicrotic group (fig. 4, table 1) correlate with their larger volumes by angiography. Other indexes of left ventricular function by echocardiography, including %ΔTh and %ΔD, were significantly poorer in the dicrotic group on the preoperative echocardiogram (fig. 4). Postoperatively the end-diastolic volume by echocardiography was slightly larger in the dicrotic group. Abnormal septal motion made calculation of ESD and %ΔD in the immediate postoperative period unreliable. The paradoxical septal motion, however, does not invalidate measurement of %ΔTh of the LVPW postoperatively. This index indicated the severe depression of myocardial contractility in the dicrotic group (fig. 5, table 1).

Serial follow-up of 16 patients (fig. 7) revealed that those who returned to a normal carotid pattern after being initially dicrotic on immediate postoperative tracing (seven patients) had improved %ΔTh LVPW. However, four patients displayed a persistent dicrotic pulse and demonstrated continued left ventricular dysfunction on echocardiographic and hemodynamic studies, as well as an extremely poor clinical course. Three of these four patients died of congestive heart failure.

We conclude that a dicrotic arterial pulse after open heart surgery tends to be confined to patients who manifest regurgitant lesions preoperatively with a significant increase in left ventricular dimension and a decreased ejection fraction on preoperative hemodynamic studies. Such patients have more marked left ventricular failure after surgery. Echocardiographic studies performed both pre- and postoperatively confirm this finding, indicating depressed left ventricular function at the time of appearance of the dicrotic pulse. Patients who show improved left ventricular function by echocardiography on follow-up revert to a normal carotid pulse pattern. A continued dicrotic pulse appears to correlate with continued left ventricular dilatation by echocardiographic and hemodynamic studies and is therefore an important prognostic sign.

References

10. Dodge HT, Sandler H, Ballen DW, Lord JD Jr: The use of
Enhancement of Tactile Perception in Palpation

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SUMMARY We studied tactile perception in palpation of the precordium to determine the frequency response of the hand and to improve, if possible, the sensitivity of the hand as a transducer for precordial movement. The threshold of tactile sensation was determined for 10 subjects by manipulating the amplitude of movement of an impulse generator at each of a series of frequency settings in the subaudible range (1-40 Hz.) Relatively gross movements were necessary to achieve threshold in the lowest frequencies. A more than fourfold increase in sensitivity was obtained by restraining the fingers with the application of a light but unyielding disc to their dorsal surface. Clinical application of this device permitted the easy perception of a systolic thrust as well as a rapid filling wave in normal adult subjects over the right ventricle at the left sternal edge, an area generally considered to be motionless by conventional palpation.

PALPATION of the precordial movement has been a part of the physical examination of the heart for over 130 years. The information derived from this procedure includes an estimate of heart size, rhythm, evidence of left or right hypertrophy and detection of thrills. For more than a century, graphic records have been widely used to depict the events involved in precordial movement, although the exact relationship between a graphic tracing and one's subjective sense of movement remains controversial. Tactile perception in the art of palpation, however, has not been extensively studied in terms of the frequency response of the hand or with respect to how the sensitivity of the hand might be improved.

Physiologists have shown that the human hand is endowed with certain neurons that are primarily sensitive to positional change and other neurons sensitive to the time rate of positional change or velocity. Therefore, the sensation appreciated by the palpating hand could be derived from a combination of both positional and velocity factors.

To clarify the physical features that may be important in palpation of precordial movement, we undertook an in vitro study in which the amplitude and frequency of a sinusoidal wave form were independently varied to determine the threshold of tactile perception.

Method

To provide a moving stimulus to the palpating fingers, a device capable of delivering a sinusoidal impulse was constructed by mounting a plexiglass disc on the plunger arm of an electromechanical transducer (fig. 1). The disc was 38 mm in diameter and suitable for the light application of the subject's fingers. The forearm was supported in a comfortable position to reduce muscle fatigue during the experiment.

The transducer was driven by a sinusoidal voltage

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