The Dicrotic Arterial Pulse

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SUMMARY
The study consisted of observations on nine male patients with palpable dicrotic carotid pulses. Average patient age was 37 years. All had advanced myocardial failure as evidenced by cardiomegaly and the presence of prominent atrial and ventricular diastolic gallop sounds. Most of these patients were diagnosed as having primary myocardial disease.

The indirect carotid pulse was characterized by a single systolic wave, a low dicrotic notch, and a large dicrotic wave. The direct brachial arterial pressure pulse had a similar configuration with a shortened ejection time index. The hemodynamic data on these patients was characterized by low cardiac output, low stroke volume, elevated pulmonary arterial wedge pressures, and high total systemic resistance. From these observations we conclude that the presence of a marked dicrotic pulse in afebrile patients at rest may indicate severe functional impairment of the myocardium.

Additional Indexing Words:
Dicrotic notch  Dicrotic wave  Peripheral vascular resistance
Primary myocardial disease  Hemodynamic data

The dicrotic arterial pulse is characterized by two pulsations with each cardiac cycle; the second is due to an accentuated dicrotic wave (fig. 1). Paul Wood wrote, "... the dicrotic pulse is encountered chiefly in patients sick with fever such as typhoid; the peripheral resistance is low, the arteries are lax, and the cardiac output probably normal."

During the past 3 years we have observed a number of patients with a palpable dicrotic arterial pulse. None of them were febrile. It was our clinical impression that these patients had severe impairment of myocardial function. Accordingly, nine of these patients were assessed.

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Methods
Each of the nine male patients studied had a dicrotic carotid pulse that was readily palpable. They ranged in age from 29 to 48 years with an average age of 37 years. Seven had primary myocardial disease, one (Z.C.) had advanced hypertensive cardiovascular disease, and one (C.R.) had coronary artery disease with a large ventricular aneurysm. Two of the patients (Z.C. and H.M.) had associated pulmonary emboli. All patients had cardiomegaly by physical and roentgenographic examination. All had both atrial and ventricular, or summation diastolic gallop sounds on auscultation and phonocardiograms; none had cardiac murmurs. All were in normal sinus rhythm.

Controls for the indirect carotid pulse recordings were obtained from nine normal male subjects who ranged in age from 25 to 38 years with an average age of 29 years. Controls for the hemodynamic measurements and direct brachial arterial tracings were obtained from 15 normal subjects previously catheterized and reported on by Perloff and associates.2 They ranged in age from 16 to 36 years with an average age of 25 years. Differences were analyzed by the two-tailed Student t-test.3

Indirect carotid arterial pulse recordings were made in seven of these patients utilizing an air-filled funnel 2.5 cm in diameter that was connected to a Statham physiologic transducer (model PR 23-2G-300) by a 10-cm polyethylene
Indirectly recorded tracing of the carotid artery pulse showing dicrotic arterial pulse. Note that one impulse is in systole and the second is in diastole. The dicrotic pulse should be differentiated from the bisferiens arterial pulse in which both palpable impulses are systolic. From top to bottom: low-frequency apical phonocardiogram, indirect carotid arterial pulse tracing, and electrocardiogram.

tubing. Recordings were made on an Electronics for Medicine DR 8 recorder at paper speeds of 50 mm/sec or greater. Since the level of the dicrotic notch is influenced by the time-constant of the recording instrument, it is important to note that the recording system used has a time-constant approaching infinity.

Three measurements were made on both the indirect carotid and direct brachial arterial pressure pulse recordings in an effort to quantify the differences in wave form that are obvious when comparing the dicrotic and the normal pulse (fig. 2). The first was to measure the peak height of the dicrotic wave (d) above the level of the dicrotic notch and to express this height as a percentage of the total height of the systolic pulse (t) measured from the onset or foot of the pulse to the peak of the systolic wave (fig. 2). The second was to measure the level of the dicrotic notch (n) above the onset or foot of the systolic wave and to express this distance as a percentage of the total height of the systolic pulse (t). The third was to express the area of the dicrotic wave above the level of the dicrotic notch (fig. 2, black area) as a percentage of the area of the systolic wave (fig. 2, dashed area). Since the duration of ejection appeared short, the ejection time index was also measured. The ejection time index is the systolic ejection time corrected for heart rate by the method of Weissler and associates.4

Right heart catheterization was performed in the standard manner, and pressures were obtained with the use of a Statham 23 Db physiologic transducer and an Electronics for Medicine DR 8 recorder. Left heart catheterization and left ventricular angiograms were obtained in the one patient (C. R.) who had a ventricular aneurysm. Cardiac output was determined in duplicate by either the Fick principle or by the dye-dilution technic, injecting indocyanine-green indicator into the pulmonary artery and sampling from either the brachial or femoral artery.

The effect of altering the arterial resistance on the dicrotic pulse was studied in three patients by distal occlusion of the artery. The brachial or femoral artery was occluded distal to the recording site by placing a blood pressure cuff around the forearm or thigh and inflating it to a pressure above systemic arterial pressure. In one of these three patients, angiotensin was injected through the same Cournand needle from which the pressure pulse was recorded. In another, a vasodilator, tolazoline, was infused in a similar manner. In addition, the effect of amyl nitrite inhalation was recorded in three patients.

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The indirectly recorded carotid pulse. These recordings, two from patients (above) and two from normals (below) were made utilizing an air-filled funnel coupled to a Statham physiologic strain-gauge transducer. Note that the dicrotic pulses are characterized by a single systolic wave, a low dicrotic notch, and a large dicrotic wave. The configuration of the normal indirectly recorded carotid pulse is illustrated below for comparison.

Figure 3

Results

Indirect Carotid Pulse

Two indirect recordings of the carotid arterial pulse from these patients are contrasted with two normal tracings in figure 3. The contour of the systolic wave of the indirect carotid pulse of these patients was characterized by a single peak. This configuration contrasts with the two maxima found in the indirect pulse recordings from normal subjects. The level of the dicrotic notch in the patients was low, averaging 9% of the total pulse height (n/t) in contrast to the average level of 46% in the normal. The dicrotic wave was prominent. The height of the dicrotic wave averaged 49% of the total pulse height (d/t) in these patients compared to 6% in the normal. The area of the dicrotic wave averaged 44% of the area of the systolic wave in the patients in contrast to 2% in normals.

Direct Brachial Arterial Pulse

The brachial arterial pressure pulse was recorded directly in eight of the nine patients. The level of the dicrotic notch was low, averaging 10% of the total height of the recorded pulse compared to the average level of 40% in the normals (fig. 2). The height of the dicrotic wave was increased, averaging 44% of the total height of the pressure pulse in contrast to an average height of 4% in the normals. As in the indirect carotid pulse, the area of the dicrotic wave was increased compared to the area of the systolic wave in the directly recorded brachial arterial pressure pulse; 46% in these patients and 1.3% in the normals. The average ejection time index was 0.04 sec shorter than the normal mean value of 0.42 sec of the controls (P = < 0.001).

Distal occlusion of the artery by a blood pressure cuff or locally increasing the distal constriction by intra-arterial infusion of angiotensin resulted in an accentuation of the dicrotic wave (fig. 4). Inhalation of amyl nitrite resulted in a decrease in the height of the dicrotic wave and either no change or an elevation of the level of the dicrotic notch (fig. 4).

Cardiac Catheterization

The hemodynamic findings are tabulated in table 1. The cardiac output was low, with a mean cardiac index of 1.74 L/min/m². The stroke volume was small, with a mean stroke index of 17 cc/beat/m². The pulmonary arterial wedge or the left ventricular end-diastolic pressures or both, were elevated with a mean pressure of 24.5 mm Hg. The mean total systemic resistance of 2,152 dyne-sec cm⁻⁵ was twice as high as the mean value for the control patients.

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**Figure 4**

From top to bottom: electrocardiogram, direct brachial arterial pulse (BA) with pressure calibrations on the left, and direct femoral arterial pulse (FA) with pressure calibrations on the right. Note that the FA pressure pulse has a smaller dicrotic wave than the simultaneously recorded BA. (Left panel) Control recordings of BA and FA pressure pulses. (Middle panel) The effect of infusion of angiotensin into BA to increase arterial resistance. Note the increase in the height of the dicrotic wave of the BA tracing and the lowered dicrotic notch. (Right panel) BA and FA recordings following inhalation of amyl nitrite. The dicrotic wave is smaller.

**Table 1**

Hemodynamic Data from Nine Patients with Dicrotic Arterial Pulses

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>Diagnosis</th>
<th>BSA (m²)</th>
<th>Mean BP (mm Hg)</th>
<th>CI (L/min/m²)</th>
<th>SI (cc/beat/m²)</th>
<th>TSR (dyne·sec·cm⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Z.C.</td>
<td>33</td>
<td>HCVD</td>
<td>2.20</td>
<td>BA 120, RA 7, Wedge 28</td>
<td>1.68</td>
<td>20</td>
<td>2,591</td>
</tr>
<tr>
<td>R.L.</td>
<td>32</td>
<td>PMD</td>
<td>1.38</td>
<td>BA 73, RA 11, Wedge 18</td>
<td>1.91</td>
<td>15</td>
<td>2,209</td>
</tr>
<tr>
<td>J.W.</td>
<td>33</td>
<td>PMD</td>
<td>1.64</td>
<td>BA 75, RA 15, Wedge 18</td>
<td>2.24</td>
<td>20</td>
<td>1,628</td>
</tr>
<tr>
<td>R.B.</td>
<td>35</td>
<td>PMD</td>
<td>1.85</td>
<td>BA 84, RA 8, Wedge 25</td>
<td>2.54</td>
<td>26</td>
<td>1,423</td>
</tr>
<tr>
<td>L.E.</td>
<td>29</td>
<td>PMD</td>
<td>2.15</td>
<td>BA 80, RA 20, Wedge 32</td>
<td>1.18</td>
<td>12</td>
<td>2,507</td>
</tr>
<tr>
<td>C.R.</td>
<td>46</td>
<td>CAD-VA</td>
<td>1.68</td>
<td>BA 68, Wedge 27</td>
<td>1.19</td>
<td>12</td>
<td>2,717</td>
</tr>
<tr>
<td>L.L.</td>
<td>38</td>
<td>PMD</td>
<td>1.97</td>
<td>BA 83, Wedge 27</td>
<td>1.77</td>
<td>19</td>
<td>1,906</td>
</tr>
<tr>
<td>E.H.</td>
<td>38</td>
<td>PMD</td>
<td>2.02</td>
<td>BA 87, Wedge 24</td>
<td>1.85</td>
<td>15</td>
<td>1,854</td>
</tr>
<tr>
<td>H.M.</td>
<td>48</td>
<td>PMD</td>
<td>2.15</td>
<td>BA 89, Wedge 15</td>
<td>1.32</td>
<td>13</td>
<td>2,530</td>
</tr>
</tbody>
</table>

**Average:**

| Patients | 37 | 1.89 | 84 | 12 | 24.5 | 1.74 | 17 | 2,152 |
| sd       | ±0.28 | ±15 | ±4.1 | ±5.5 | ±0.46 | ±4.7 | ±466 |
| Normals  | 25 | 1.79 | 88 | 5 | 12 | 3.79 | 51 | 1,099 |
| sd       | ±0.32 | ±13 | ±404 |
| P        | 0.5 | N.S. | 0.001 | 0.001 | 0.001 |

Abbreviations: BSA = body surface area; m = meter; BP = blood pressure; BA = brachial artery; RA = right atrium; wedge = pulmonary arterial wedge; CI = cardiac index; SI = stroke index; TSR = total systemic resistance; HCVD = hypertensive cardiovascular disease; PMD = primary myocardial disease; CAD-VA = coronary artery disease with ventricular aneurysm.

Normal values were calculated from the data obtained by Perloff and associates from 15 subjects. Normal values were not obtained from the control subjects. The values listed represent the upper limits of normal for our laboratory.
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Discussion

The English literature has few references to the dicrotic arterial pulse. Textbooks of cardiology\(^1,6\) refer to the work of Sir James MacKenzie. In 1902, MacKenzie published his classic book, The Study of the Pulse, Arterial, Venous and Hepatic and of the Movement of the Heart, in which he wrote: "When the ventricular contraction is weak the systolic wave is low, the dicrotic notch is deep, reaching often to the base line, while the dicrotic wave is much increased in size, indicating a remarkable fall in arterial pressure." He illustrated the dicrotic pulse in a patient with typhoid fever and attributed the marked increase in the dicrotic wave to the fact that "... the arterial relaxation was much greater in the typhoid case."\(^7\) Paul Wood also found this type of arterial pulsation in patients with infectious or toxic reactions. He wrote, "... a good sign of vascular relaxation is a markedly dicrotic pulse and although not necessarily serious should put the physician on guard."\(^1\)

Our studies indicate that the dicrotic pulse occurs in young patients who have elevated peripheral vascular resistance and a low stroke volume. Although these observations appear to contrast with those in the literature,\(^1,6,7\) it is tempting to speculate that those earlier observations were made on patients who had either typhoid myocarditis or a low stroke volume secondary to severe dehydration from typhoid diarrhea.

The determinants of the dicrotic wave are not completely understood. The dicrotic wave of the normal arterial pressure pulse has been ascribed to the rebound of arterial blood against a closed aortic valve. However, peripheral factors contribute materially to its formation.\(^8-12\) There is a decrease or loss of the dicrotic wave with age, hypertension, generalized atherosclerosis, and diabetes.\(^5,10,13\) Our patients were relatively young, with a mean age of 37 years. It may be that the arterial resiliency of the young is necessary for the generation of the dicrotic pulse.

What other factors contribute to the formation of the dicrotic pulse? Increasing local arterial resistance in our patients heightened the dicrotic wave. In the one patient studied, local injection of an arterial dilator resulted in a decrease of the dicrotic wave. These observations confirm the importance of peripheral factors in the genesis of the dicrotic wave. Three other observations bear on the genesis of the dicrotic pulse: The first is that the post-premature beats were less dicrotic than the control beats (fig. 5). In the absence of muscular subaortic stenosis, the more forceful contraction of the post-premature beat results in an increased stroke volume. Second, in our patients with pulsus alternans, the alternate beats with a higher pressure had a smaller dicrotic wave and a higher dicrotic notch than the beats with lower pressure. The third observation was in a patient who had pericardial tamponade and a dicrotic arterial pulse only on inspiration (fig. 6A). In tamponade it is known that the stroke volume decreases with inspiration.\(^14\) In addition, the ejection time index (an indirect measurement of stroke volume) of the dicrotic beats was 0.04 sec shorter than that of the more normal beats (fig. 6B). The dicrotic contour of the brachial arterial pulse was not present following the removal of 500 ml of pericardial fluid. These observations seem to indicate that a small stroke volume or a weaker force of

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**Figure 5**

Electrocardiogram (E.C.G.) and direct recording of the brachial arterial pulse (B.A.). Note that the post-premature beat has a more rapid upstroke velocity, a larger systolic wave, a higher dicrotic notch, and a smaller dicrotic wave than the control beat occurring before the extrasystole.

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Effect of respiration on brachial arterial pulse contour in a uremic patient with pericardial tamponade.

(A) During tamponade. From top to bottom: electrocardiogram, direct brachial arterial pressure pulse (BA), intrapericardial pressure recording (IP) and respiration: inspiration (in), giving a downward deflection, and expiration (ex), giving an upward deflection. Note the marked dicrotic pulse (arrows) on inspiration, when stroke volume would be small and the almost normal contour of the pulse during expiration when the stroke volume would be larger. The intrapericardial pressure is 20 mm Hg.

(B) Following removal of 500 ml of pericardial fluid by technic previously reported.\(^{15}\) From top to bottom: electrocardiogram, direct recording of brachial arterial pressure pulse (BA), respiration, and intrapericardial pressure (IP). Following the relief of tamponade, note the absence of the dicrotic pulse even during inspiration. The intrapericardial pressure decreased to a mean of 5 mm Hg with respiratory fluctuations between 11 and -1 mm Hg.

The dicrotic arterial pulse appears to be determined by the presence of certain hemodynamic factors in relatively young patients. Therefore, the dicrotic pulse is not necessarily related to a specific disease entity as reported by Meadows and co-workers.\(^{16}\) We have observed the dicrotic arterial pulse in a variety of disease states, including primary myocardial disease, hypertensive cardiovascular disease, ischemic heart disease, acute myocardial infarction, primary pulmonary hypertension, and intermittently in a patient with pericardi-
al tamponade. A dicrotic arterial pulse has been reported in constrictive pericarditis.\textsuperscript{17}

In view of these studies, whenever a dicrotic pulse is palpated in an afibrile patient, we are oriented to think of severe functional impairment of the myocardium.

Acknowledgment

We are indebted to Dr. A. C. deLeon for making the hemodynamic data on the normal patients reported by Perloff and associates\textsuperscript{2} available for analysis. Dr. Nayab Ali made the recording used in figure 6. Drs. A. C. deLeon, J. K. Perloff, James Ronan, and R. A. Massumi kindly reviewed the manuscript. The authors appreciate the help of Dr. George Just of Mainz, Germany, for referring us to the work of Dr. Kurt Wezler and to Volker Sonntag for the German translations. The secretarial assistance of Mrs. Yvonne McIlwain is gratefully appreciated.

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