Apex-Carotis Diagram as Related to Systolic and Diastolic Time Intervals in Mitral Valve Prolapse Syndrome

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SUMMARY

The electrocardiogram, phonocardiogram, carotid pulse tracing and apexcardiogram were simultaneously obtained in 25 patients with mitral valve prolapse syndrome and in 62 normal subjects. This allowed us to measure systolic and diastolic time intervals and to construct the apex-carotis diagram (ACD), a new mechanocardiographic method which integrated the carotid pulse tracing and the apexcardiogram in an orthogonal coordinate system. In mitral valve prolapse syndrome, the ACD showed curve of the segment of slow ventricular ejection (S-AC) to the right, and a decrease of the field of ventricular ejection. The measurement of time intervals gave statistically significant information on Q-I and A2-OS intervals.

Additional Indexing Words:
Mitral valve prolapse Apex-carotis diagram Systolic and diastolic time intervals Cyclic representation of cardiac function

IN 1971, Heuillet and Soulier, in the search for noninvasive methods of studying cardiovascular function, proposed a new approach to the construction of functional diagram of the heart.1,2) The principle of the method consists in integration of apexcardiogram (ACG) and carotid pulse tracing (CP), recorded simultaneously in a single diagram—the apex-carotis diagram or ACD.3–6) They described alterations of the ACD in chronic rheumatic valvulopathy, congenital heart disease, chronic pulmonary heart disease, and various cardiopathies. But there is lack of research about ACD in mitral valve prolapse syndrome.

The ACD provides new ways of interpreting the systolic and diastolic time intervals of left ventricle, with which it is closely associated. All points described in the diagram are elements of the time intervals of cardiac cycle, since the individual segments of the diagram are in fact intervals of systole and diastole of left ventricle. Whereas the systolic and diastolic time intervals

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are linearly related to the duration of each phases of cardiac cycle, the ACD represents the cycle as a whole. It depicts not only the duration but also the path of the curve from point to point. This turn allows conclusion to be drawn regarding the velocity of the different segments.

The purpose of this study was to describe the structure of the ACD in mitral valve prolapse syndrome and to investigate the correlation between ACD features and systolic and diastolic time intervals. Cheng described the prolongation of Q-I and reduction of A2-OS intervals in mitral valve prolapse syndrome.

**Materials and Methods**

Studies were performed on 25 patients with mitral valve prolapse syndrome, 11 males with an average age of 36 years (range 15–57) and 14 females with an average of 37 years (range 16–68). The control group included 62 normal subjects, 42 males with an average age of 31 years (range 16–61) and 20 females with an average age of 34 years (range 17–65). The diagnosis of mitral valve prolapse syndrome was based on history, clinical feature, electrocardiographic change, and phonocardiographic findings according to Barlow and Pocock and Malcolm et al. All patients must have in phonocardiogram nonejection systolic click(s) and mid-late systolic murmur. Patients with isolated systolic click or isolated systolic murmur were not included in this study.

Probable etiology of mitral valve pathology and associated factors in our 25 patients with mitral valve prolapse syndrome are given in Fig. 1. The diagnosis of obstructive cardiomyopathy was confirmed by ventriculography, and that of ruptured chordae tendineae was suspected by echocardiography.

The patients included in this study fulfilled the following criteria: no cardiac failure; no administration of drugs capable of affecting myocardial contractility such as digitalis, catecholamines, atropine, beta-receptor blocking or antiarrhythmic agents; no atrioventricular block, no QRS duration more than 0.1 sec, no hypertension or hypotension.

![Fig. 1. Probable etiology of the mitral valve pathology and associated factors in 25 patients with nonejection click-late systolic murmur.](image-url)
The polygraphic recording was performed by rotating recumbent subjects 30 to 50° toward the left lateral decubitus position. Two piezoelectric transducers were used for ACG and CP tracing (RFT model, GDR) and one piezoelectric microphone for phonocardiogram. During slight expiration simultaneous records were made of the electrocardiogram, phonocardiogram, CP tracing, and ACG on a 6-channel recorder 6 NEK-3, GDR. Paper speed was 100 mm/sec.

ACG and CP tracing were recorded simultaneously by the usual methods described in the monographs of phonomechanocardiography.7),10) The problem of calibration and standardization of these two curves has not yet a satisfactory solution. The author of this method provisionally equalized the maximum amplitudes of the 2 curves, but an improved method should provide stable curve recording for several cardiac cycles.

To draw the ACD it was necessary to measure the height from basic line of each mechanogram every 20 msec. Thus we have 2 values for each time. We plot the values of the ACG on the horizontal axis (abscissa) and those of the CP

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**Fig. 2.** Construction of apex-carotid diagram and its main elements in normal subjects.
tracing on the vertical axis (ordinate). Connection of the different points in a chronological order forms a diagram, i.e. ACD. On the diagram it is possible to identify the different phases of cardiac cycle, which represent its main elements:

Points: OM—opening of the mitral valve; MC—mitral closure; OA—opening of the aortic cusps; S—peak of ventricular ejection; AC—closure of the aortic cusps.

Lines: OM-MC is the mitral line and AC-OA is the aortic line. Segments: MC-OA corresponds to isovolumetric contraction; AO-S to rapid ventricular ejection; S-AC to slow ventricular ejection; AC-OM to isovolumetric relaxation and OM-MC is a diastolic segment or segment of ventricular filling. Field: The field of ventricular filling is enclosed with, the segment of ventricular filling and with the mitral line. The field of ventricular contraction is defined by the mitral and aortic lines on the one hand, and the segment of isovolumetric contraction and relaxation on the other hand. The field of ventricular ejection is enclosed with the aortic lines and with the segments OA-S and S-AC. Construction of ACD and its main elements are given in Fig. 2. Fig. 3 depicts the intervals used in this investigation. Rapid ventricular ejection time (r LVET) is a time required for reaching maximum height of CP tracing i.e. from the onset of CP upstroke to its maximum height. Slow ventricular ejection (a LVET) elapses from maximum height of CP tracing to the onset of the dicrotic notch. All intervals were corrected for heart rate by Bazett’s formula.

![Fig. 3. Systolic and diastolic time intervals.](image-url)
RESULTS

The structure of ACD in mitral valve prolapse syndrome presents some characteristic changes. The segment of slow ventricular ejection (S-AC) is extended and instead of almost linear descendent from point S to point AC in normal subjects, it showed curve (twist) to the right with formation of ‘bay’, sometime reaching to the segment of rapid ventricular ejection (OA-S). See Fig. 4. This characteristic change in the ACD was found in 23 of the 25 patients (92%), but in none of the control group (p<0.001).

The field of ventricular ejection is decreased in relation to the total surface area of the diagram. In the patients with mitral valve prolapse syndrome its value amounted to 18±7% (p<0.001). Fig. 4 showed the ACD of a patient with mitral valve prolapse syndrome constructed by hand on a polygraphic tracing.

Of 11 systolic and diastolic time intervals examined, only 2 were found

![Fig. 4. Apex-carotis diagram in a patient with mitral valve prolapse. The apex-carotis diagram showed curve of the segment of slow ventricular ejection (S-AC) to the right and a decrease of the field of ventricular ejection.](image)

<p>| Table I. Some of the Systolic and Diastolic Time Intervals in Mitral Valve Prolapse Syndrome |
|-----------------------------------------------|----------------|----------------|----------------|</p>
<table>
<thead>
<tr>
<th>Q-I</th>
<th>rLVET</th>
<th>sLVET</th>
<th>A2-OS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal subjects  n=62</td>
<td>58±12</td>
<td>84±14</td>
<td>210±15</td>
</tr>
<tr>
<td>Mitral valve prolapse n=25</td>
<td>72±13</td>
<td>87±15</td>
<td>217±16</td>
</tr>
<tr>
<td>p</td>
<td>&lt;0.02</td>
<td>&gt;0.10</td>
<td>&gt;0.10</td>
</tr>
</tbody>
</table>

All intervals are expressed in msec. rLVET is rapid left ventricular ejection time and sLVET is slow left ventricular ejection time.
to be modified in mitral valve prolapse syndrome. The prolongation of Q-I and reduction of A2-OS were the only significant findings (Table I).

In Fig. 5 some of the systolic and diastolic time intervals in normal subjects (broken line) and in patients with mitral valve prolapse syndrome (continuous line) were superposed. If the complete cardiac cycle lasts 1 sec, then 10 msec corresponds to 3.6°. Inside this circle we have constructed the ACD of normal subjects (broken line) and that of patients with mitral valve prolapse syndrome (continuous line). One can see that deformation of ACD coincides with nonejection click and onset of mid (late) systolic murmur.

Combining the time intervals with the ACD one can describe the durations and velocities of the various cardiac events. For the same interval of time, a greater length of the ACD segment will correspond to a greater velocity. The A2-OS interval of the diagram is recorded with a greater velocity, and Q-I interval with a slower velocity in patients with mitral valve prolapse syndrome than in normal subjects. If the ACG and carotid pulse tracing are plotted continuously and simultaneously as a X-Y plot on a memory oscilloscope, the segment of slow ventricular ejection (S-AC) will be represented with a greater interval between dash dots. In the same time there is no statistically significant difference between the slow LVETs in control group and in patients with mitral valve prolapse syndrome (p>0.10).

Fig. 5. Combination of apex-carotis diagram and systolic and diastolic time intervals in cyclic representation. Q-Q wave of electrocardiogram; I-mitral component of the first heart sound; C -click; SM-systolic murmur. The direction of the cardiac cycle is marked with arrows.
DISCUSSION

Numerous studies over the past 16 years have shown that systolic non-ejection click and associated mid-late systolic murmur are due to an abnormality of the mitral valve.\(^7\)\(^-\)\(^9\),\(^11\)\(^-\)\(^18\)

For the first time Kesteloot and VanHoute\(^11\) and Willems et al\(^12\) described downslope or retraction of the systolic plateau of ACG in patients with mitral valve prolapse syndrome. Similar changes in ACG were found by several investigators.\(^8\),\(^9\),\(^13\)\(^-\)\(^17\) Willems et al\(^12\) found a retraction or a more steep downslope of the systolic wave of CP tracing in patients with mitral valve prolapse syndrome, and later this finding was confirmed by many authors.\(^7\),\(^14\),\(^15\),\(^18\) This downslope of the systolic plateau of ACG and systolic retraction of CP tracing exactly coincide with the nonejection click or with the onset of the mid (late) systolic murmur.\(^8\),\(^9\),\(^11\)\(^-\)\(^18\) These abnormalities of ACG and CP tracing are thought to be a reflection of the altered hemodynamics as a consequence of the prolapsing mitral valve. Thus Willems et al\(^12\) obtained marked difference between the right and left ventricular pressure curves. In the left ventricular curve they found an inflection of the downslope, which coincided with the systolic deep retraction on simultaneously recorded CP tracing, and with systolic click and onset of mid (late) systolic murmur. Barlow and Pocock\(^8\) support the hypothesis that the systolic retraction results from tugging of chordae of papillary muscles at the time of peak prolapse of the leaflet. Spencer et al\(^16\) suppose that the systolic retraction on the ACG suggests abnormal contraction due to the systolic decompression of the left ventricle by the prolapsing mitral leaflets and represents a 'checking' phenomenon associated with the abrupt halt of blood and that this phenomenon represents an abrupt change in the systolic impedance to left ventricular ejection as the suddenly taut mitral leaflets halt the transfer of blood beneath them directing the ejection of the remaining stroke volume exclusively into the aorta against a higher impedance. The ACD, integrated in itself of 2 curves reflecting hemodynamics, potentiates the changes of each component curves.

In conclusion, our findings relative to the changes of systolic and diastolic time intervals in mitral valve prolapse syndrome are in agreement with Cheng.\(^19\) The ACD as related to the systolic and diastolic time intervals provides more complete information than classical mechanogram and opens a new way of investigating the cardiac cycle and cardiac function.
REFERENCES