The Apex Cardiogram in Left Ventricular Outflow Tract Obstruction

By Emilio Tafur, M.D., Lawrence S. Cohen, M.D., and Harold D. Levine, M.D.

The analysis of the normal apex cardiogram and its temporal relationship to electrical, acoustic, and mechanical cardiac events is the subject of the preceding communication. The “normal pattern” was found to be uniform, reproducible, and clearly correlated with known hemodynamic phenomena.

It was anticipated that different types of obstruction of the outflow tract of the left ventricle, by bringing about abnormal ventricular pressure-volume relationships, might induce changes in the configuration of the apex cardiogram. It is thus the particular purpose of the present report to study the influence of hypertrophic subaortic stenosis and of valvular aortic stenosis upon the several components of the apex cardiogram.

Material and Methods

The patients studied included six with hypertrophic subaortic stenosis, 16 with valvular aortic stenosis, and one with myocardial hypertrophy of undefined etiology.

Hypertrophic Subaortic Stenosis. The patients with this lesion ranged in age from 18 to 39 years: two were men and four were women. In all patients the diagnosis was established by cardiac catheterization. In five patients a moderate to large ventriculo-aortic gradient was present at rest. In patients T.T. and M.J. the diagnosis was not suspected until cardiac catheterization. The former had a ventriculo-aortic gradient of 18 mm. Hg during exercise. In patients M.S., R.G., R.J., and M.P. the diagnosis had been suggested by clinical examination, since these patients presented many of the features characteristic of this condition. Two of these patients had a resting subvalvular pressure gradient ranging from 38 to 92 mm. Hg. In patient R.C. the diagnosis was confirmed at operation and subsequently at autopsy. Selective angiocardiothorax, carried out in two patients, demonstrated systolic narrowing of the outflow tract of the left ventricle. All six patients were in sinus rhythm, five had evidence of left ventricular hypertrophy on the electrocardiogram and had mild to moderate enlargement of the left ventricle on roentgenographic examination. M.J. showed incomplete right bundle-branch block in the electrocardiogram; the heart was not enlarged on roentgenographic examination.

Idiopathic Myocardial Hypertrophy. The patient with this condition was a 42-year-old man without valvular, hypertensive, or coronary artery disease. The diagnosis was made at cardiac catheterization with the aid of angiocardiothorax. At rest there was a 2-mm. Hg ventriculo-aortic gradient but during an infusion of isoproterenol a gradient of 35 mm. Hg developed. The electrocardiogram showed normal sinus rhythm and left ventricular hypertrophy. Roentgenographic examination showed left ventricular enlargement.

Valvular Aortic Stenosis. The 16 patients with this lesion ranged in age from 20 to 64 years: eight were men and eight were women. All were moderately to severely limited in activity as a result of congestive heart failure. All were in sinus rhythm and presented electrocardiographic evidence of left ventricular hypertrophy. Left ventricular enlargement of varying degree was detected on roentgenographic and fluoroscopic examination. The diagnosis was proved in all patients at cardiac catheterization. The systolic mean gradient across the aortic valve ranged from 24 to 44 mm. Hg. In four patients the diagnosis was confirmed at operation and in two of them at postmortem examination.

Methods. The electrocardiogram, phonocardio-
gram, carotid pulse, and apex cardiogram were recorded by means of a four-channel Sanborn Poly-Beam recorder (Model 564). The Sanborn Contact-Microphone (Model 350-1700-C10) was used for recording the heart sounds and the Sanborn Crystal-Microphone (Model 374) was used for the carotid pulse and apex cardiogram. Paper speed was 75 mm. per second. Vertical lines in the tracings represent time intervals of 0.04 second.

Results

Hypertrophic Subaortic Stenosis

"a" Wave and Pre-ejection Components. In four of the six patients with this condition, the "a" wave was uniformly tall and peaked (figs. 1, 2, and 3). This same configuration of the "a" wave was recorded in some patients with valvular aortic stenosis but not in normal subjects studied in this laboratory. In patient T.T. the "a" wave became peaked during isoproterenol infusion (fig. 4). The configuration and duration of the pre-ejection components of the apex cardiogram differed in no way from those seen in normal subjects or in patients with valvular aortic stenosis.

Ejection Component. This was distinctive and clearly differed from the normal apex cardiogram and from that of valvular aortic stenosis. During early ejection the apex cardiogram inscribed its normal descent. In five of the patients with subaortic stenosis, however, this descent was abruptly interrupted by a second sharp positive wave (figs. 1, 2, and 3). This wave coincided with both the peak of the diamond-shaped systolic ejection murmur and with the trough between the

![Diagram](image-url)

**Figure 1**

A. Electrocardiogram, carotid pulse (CP), phonocardiogram (PCG), and apex cardiogram (ACG) in a normal subject. a, atrial wave; PEC-I and II, pre-ejection components; EC, ejection component; PD, protodiastole; ESS, end-systolic shoulder; RFW, rapid filling wave; SFW, slow filling wave.

B. Patient R.G. Hypertrophic subaortic stenosis. Large a wave. A second systolic wave (SSW) is inscribed in the ACG at mid-ejection coinciding with the peak of the diamond-shaped systolic murmur. Time intervals of PEC-I and II are within normal limits. End-systolic shoulder (ESS) marking the onset of protodiastole (PD) occurs before A2, which coincides with the notch N2 of the apex cardiogram and follows P2 (reversed split).
“percussion” and “tidal” waves of the carotid pulse. In one patient a second systolic wave was recorded on one occasion, not recorded on another. From the end of this second systolic wave a normal curve, including a normal bulge or an end-systolic shoulder, was inscribed (figs. 1, 2, and 3). It must be emphasized that the second systolic wave occurring in mid-ejection, here described, is quite distinct from the “late systolic bulge” described in, and considered to be characteristic of, this condition. Figure 3 illustrates a late systolic bulge recorded in a normal subject. This bulge is a normal finding and as such does not differentiate patients with hypertrophic subaortic stenosis from normal subjects. In summary, the apex cardiogram of patients with subaortic stenosis generally shows two distinct systolic waves plus either a normal late systolic bulge or an end-systolic shoulder. This proved too refined a distinction to be made from physical examination. In a few patients in whom a double early systolic wave was suspected on palpation, this was not confirmed in the apex cardiogram which showed instead a prominent “a” wave or possibly a prominent late systolic bulge. Proto-diastole, the rapid filling wave, and the slow filling wave did not differ from those in normal subjects (fig. 1).

In six of the seven patients with functional obstruction of the left ventricular outflow tract, the end-systolic shoulder of the apex cardiogram preceded the onset of the aortic component of the second sound (A₂) as it did in the 25 normal subjects studied in this laboratory. In the single exception this shoulder followed A₂ by 0.02 second. This was in patient H.P. (fig. 2A) in whom cardiac catheterization demonstrated mitral regurgitation in addition to hypertrophic subaortic stenosis. Preliminary observations in this laboratory demonstrated that in many patients with mitral regurgitation the endsystolic shoulder of the apex cardiogram may follow the onset of A₂. This finding can be explained by the early closure of the aortic valve described in cases of mitral insufficiency.

The use of isoproterenol has been de-
Figure 3
A. Apex cardiogram. Normal subject. A late systolic bulge (B) occurs at end-systole shortly before A2. B. Apex cardiogram in patient M.S. with hypertrophic subaortic stenosis. The second systolic wave (SSW), characteristic of this condition, is inscribed in mid-ejection well before the late systolic bulge (B) or end-systolic shoulder.

Figure 4
Effect of isoproterenol in the apex cardiogram of patient T.T. with hypertrophic subaortic stenosis. A. Normal apex cardiogram at rest. B. During isoproterenol infusion a peaked a wave and a second systolic wave (SSW) appear.
Figure 5

Effect of isoproterenol in patient J.L. with idiopathic myocardial hypertrophy. A. Normal apex cardiogram at rest. B. Appearance of a second systolic wave (SSW) during isoproterenol infusion.

scribed as a provocative test for the diagnosis of hypertrophic subaortic stenosis. The parallel observation of a second systolic positive wave may likewise be induced in the apex cardiogram only under the special condition of such a test. Patient T.T., for example, was found to have muscular subaortic stenosis at cardiac catheterization. At rest the gradient across the left ventricular outflow tract measured 40 mm. Hg. During the infusion of isoproterenol the left ventricular pressure rose, the brachial artery pressure fell, and the gradient increased to 80 mm. Hg. On the following day an apex cardiogram was recorded (fig. 4). At rest this showed no significant difference from normal but an infusion of isoproterenol induced the characteristic changes of subaortic stenosis described above; a second systolic wave developed coinciding with the peak of the diamond of the systolic ejection murmur. At the same time the intensity of the murmur increased and the carotid pulse became characteristic of that described in hypertrophic subaortic stenosis.

Idiopathic Myocardial Hypertrophy

In the patient with this condition, no obstruction was detected at rest across the left ventricular outflow tract during cardiac catheterization. During an infusion of isoproterenol, however, a gradient of 35 mm. Hg developed. An apex cardiogram performed at rest was normal but infusion of isoproterenol, similarly performed, induced the characteristic changes of muscular obstruction of the outflow tract (fig. 5).

Valvular Aortic Stenosis

With one exception the apex cardiogram in 16 consecutive patients with valvular aortic stenosis, observed in the course of 1 year, reflected obstruction of the outflow tract from the very moment of the onset of left ventricular ejection (fig. 2). Instead of a rapid descent from the maximal systolic peak, there was a gradual, sloping plateau that remained.
APEX CARDIOGRAM

Figure 6

Effect of isoproterenol in the apex cardiogram of patient M.N. with valvular aortic stenosis. A. The resting apex cardiogram shows a sloping plateau during ejection. B. Isoproterenol does not induce a second systolic wave.

elevated through most of the ejection period. It is in this respect that the apex cardiogram of valvular aortic stenosis differed from that of hypertrophic subaortic stenosis. The single exception was a 49-year-old man with mixed valvular aortic stenosis and regurgitation, and no subaortic stenosis. A clear-cut second systolic wave was recorded in the apex cardiogram.

The effect of isoproterenol was studied in one of the patients with valvular aortic stenosis. Patient M.M. with rheumatic aortic stenosis showed, at rest, an apex cardiogram characteristic of this condition (fig. 6). During an infusion of isoproterenol, which accelerated the heart rate and accentuated the murmur, a slight change developed in the configuration of the ejection component of the apex cardiogram but a second systolic wave did not appear.

Effect of Isoproterenol upon the Normal Apex Cardiogram

As seen above, patients with mild or moderate subaortic stenosis may have a normal apex cardiogram at rest but when such patients are given an infusion of isoproterenol these tracings may exhibit the characteristic changes associated with this condition (figs. 4 and 8). This raised the question of the effect of this agent upon individuals without subaortic stenosis. When tested in three normal individuals, the drug did not induce this effect. Figure 7 recorded in one of us, for example, shows a normal apex cardiogram and the changes that occurred during the infusion of isoproterenol. Although the heart rate doubled, a second systolic wave in mid-ejection was not induced. It would seem, therefore, that the second systolic wave induced in some patients with hypertrophic subaortic stenosis was an effect not of the drug, but of the underlying disease.

Discussion

Characteristic carotid tracings have been an aid to the diagnosis of valvular aortic stenosis and hypertrophic subaortic stenosis. The configuration of the pulse wave reflects the nature of the obstruction of the left ventricular outflow tract. In valvular aortic stenosis the carotid pulse generally shows a prolonged ejection time, is characteristically small, develops slowly, and often has an ana-

Figure 7

Effect of isoproterenol in the apex cardiogram of a normal subject. A. Normal apex cardiogram at rest. B. During isoproterenol infusion the heart rate doubles and a late systolic bulge (B) develops but a second systolic wave does not appear.
Effect of isoproterenol in the apex cardiogram of patient M.J. with hypertrophic subaortic stenosis. A. Normal apex cardiogram at rest. B. Appearance of a second systolic wave (SSW) in mid-ejection during isoproterenol infusion. This second systolic wave occurs between the initial descent of the ejection component of the apex cardiogram and the late systolic bulge (B). Compare with figure 7.

In hypertrophic subaortic stenosis the carotid pulse usually shows a prolonged ejection time but, by contrast, there is a rapid "percu-
sion wave" followed by a "secondary tidal peak." The percussion wave reflects the period of unobstructed left ventricular ejection; the secondary tidal wave is inscribed during the period when the hypertrophied septum actually obstructs the left ventricular outflow tract. In other words, early ejection is not compromised; obstruction develops in the latter phases of ventricular systole as contraction proceeds.

Hypertrophic subaortic stenosis was associated with characteristic changes in the ejection component of the apex cardiogram. Why these were at variance with the apex cardiogram of normal patients or of patients with valvular aortic stenosis may be explained in the following way. At the moment of aortic valve opening and the onset of left ventricu-
lar ejection the apex cardiogram inscribed, as in normal individuals, a sharp negative deflec-
tion. As the hypertrophied myocardium impinged upon the outflow tract a second systolic wave was inscribed in mid-ejection. In other words, "hypertrophy" developed into "stenosis" as systole progressed. In valvular aortic stenosis, by contrast, obstruction was reflected in the apex cardiogram from the very moment of onset of ejection (fig. 2). The second systolic wave of subaortic stenosis, here described, was quite distinct from the late systolic bulge mentioned above. A late systolic bulge was inscribed in seven of 25 consecutive apex cardiograms recorded in normal subjects (fig. 3). This bulge occurred later in the ejection component of the apex cardiogram than the second systolic wave.

The dynamic nature of subaortic stenosis has been documented recently. At rest with the heart initiating contraction from a normal end-diastolic volume, no obstruction to outflow need be apparent. Any stimulus, e.g., isoproterenol, causing the heart to initiate contraction from a smaller end-diastolic volume, would favor the intrusion of the hypertrophied septum upon the outflow tract. Whereas no gradient may be measured across the outflow tract at rest, one may thus be detected under the stimulus of this agent.

Idiopathic myocardial hypertrophy may also result in subaortic stenosis. Although the muscular elements of the heart are hypertrophied, the chambers are often diminished in size. This set of circumstances predisposes to obstruction of the outflow tract. Isoproterenol may therefore transform a potential gradient into a real gradient. Although this drug causes tachycardia, it appears that no changes similar to those seen in hypertrophic subaortic stenosis or myocardial hypertrophy are observed in the apex cardiogram when this agent is administered to individuals with normal hearts or patients with valvular aortic stenosis (figs. 6 and 7).

The prominent "a" waves recorded both in patients with subaortic stenosis and valvular
APEX CARDIOGRAM

399

aortic stenosis are not diagnostic of obstruction of the outflow tract of the left ventricle. It has been suggested that in instances of left ventricular hypertrophy the passive filling of the ventricle is diminished due to the resistance offered by the ventricle; the contribution of atrial systole is therefore augmented to compensate for the diminished filling of the left ventricle during early diastole. In short, the prominent “a” waves recorded at the apex cardiogram are a nonspecific effect of left ventricular hypertrophy.

Summary

The use of the apex cardiogram offers a simple yet valuable aid in the diagnosis of obstruction of the left ventricular outflow tract. Characteristic changes in the ejection component of the apex cardiogram distinguish obstruction due to hypertrophic subaortic stenosis from obstruction due to valvular aortic stenosis. A second systolic wave is inscribed in the mid-ejection period of the apex cardiogram in hypertrophic subaortic stenosis. This wave reflects the development of the obstruction of the outflow tract of the left ventricle by the hypertrophied septum. This obstruction is not apparent in the apex cardiogram at the onset of ejection but becomes manifest as contraction progresses. Although the apex cardiogram in patients with subaortic stenosis may appear normal at rest, isoproterenol may act as a provocative agent inducing the characteristic changes of this condition.

In valvular stenosis obstruction is reflected in the apex cardiogram from the very onset of ejection and is manifest throughout the total ejection phase as a sustained gradual sloping plateau.

References


Science + the Scientist

The scientist should be a man willing to listen to every suggestion, but determined to judge for himself. He should not be biased by appearances; have no favorite hypothesis; be of no school; in doctrine have no master. He should not be a respecter of persons, but of things. Truth should be his primary object. If to these qualities be added industry, he may indeed hope to walk within the veil of the temple of nature.—MICHAEL FARADAY.

Circulation, Volume XXX, September 1964
The Apex Cardiogram in Left Ventricular Outflow Tract Obstruction
EMILIO TAFURR, LAWRENCE S. COHEN and HAROLD D. LEVINE

Circulation. 1964;30:392-399
doi: 10.1161/01.CIR.30.3.392
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1964 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/30/3/392

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/