Ventricular Volume Studies in a Patient with Hypertrophic Subaortic Stenosis

By Charles E. Rackley, M.D., Robert E. Whalen, M.D., and Henry D. McIntosh, M.D.

Since the description of dynamic obstruction of the left ventricular outflow tract by Brock, numerous reports have dealt with the clinical and hemodynamic features of hypertrophic subaortic stenosis. Brachfeld and Gorlin and Braunwald and associates were among the first investigators to report the hemodynamic findings in detail. The hemodynamic responses of such patients to various drugs have been studied in a number of laboratories. Angiographic studies have portrayed certain anatomic features of this disorder, but recently the existence of the chamber obstruction has been questioned.

Although the anatomic features and physiological responses of the left ventricle in hypertrophic subaortic stenosis have been investigated, only recently have the systolic and diastolic volumetric characteristics of the ventricle been examined. The purpose of the present report is to describe changes in left ventricular volume which occurred during the study of a patient with hypertrophic subaortic stenosis. The ventricular volume measurements were obtained utilizing biplane cineangiographic techniques.

Report of Case

The patient was a 33-year-old white female who was first admitted to the Duke Medical Center for cardiac evaluation in 1960. A heart murmur had been detected 2 years earlier after a stab wound to the chest. The patient had noticed shortness of breath with exertion, chest pain, and syncopal episodes for several years, but these symptoms had become more frequent following the stab wound. Hyperventilation occasionally preceded the episodes of syncope.

Examination revealed a well-developed white female. Blood pressure was 120/70. The peripheral arterial pulse was 96 with a bifid impulse. The heart was not enlarged. A systolic thrill was palpable in the third and fourth intercostal spaces. A grade IV/VI harsh systolic ejection type of murmur was heard in the same area and extended widely into the neck, axilla, and back. There was no peripheral edema.

The electrocardiogram showed increased QRS amplitude but was otherwise normal. The chest roentgenograms were normal. At cardiac catheterization a systolic gradient between the body of the left ventricle and the left ventricular outflow tract was present (table 1).

The patient was followed without a change in symptoms until November 1964. The patient became pregnant, had an episode of severe chest pain which suggested a myocardial infarction, and later experienced pulmonary edema which responded to rapid digitalization.* In January 1965, the patient had a total hysterectomy, and 2 months later she underwent open heart surgery. Upon exploration of the left ventricle, a markedly hypertrophied muscle mass was palpated in the left ventricle in the region of the septum. The area was incised, but following closure of the aorta a thrill was palpated high in the right ventricle. It became apparent that a defect had been created in the ventricular septum. The right ventricle was explored and an attempt was made to close a small defect in the ventricular septum.

Postoperatively the patient developed the postcardiomyopathy syndrome. A hydrogen electrode study demonstrated a left-to-right shunt at the ventricular level. The patient has continued taking digoxin and prednisone. Despite the presence of a ventricular septal defect, the patient has improved clinically.

Methods

Right and left heart catheterizations were performed on a number of occasions (table 1) ac-

*Details of these events were supplied by William C. McCurdy, Jr., M.D., McCurdy Memorial Hospital, Purcell, Oklahoma.
Cardiac Catheterization Data

<table>
<thead>
<tr>
<th>Date of catheterization</th>
<th>Conditions</th>
<th>Left ventricle</th>
<th>Infundibular chamber</th>
<th>Aorta</th>
<th>Radial artery</th>
<th>Cardiac output (L/min)</th>
<th>Heart rate</th>
<th>Forward stroke volume (cc/beat)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. 9-14-60</td>
<td>Basal</td>
<td>190/28</td>
<td>116/28</td>
<td>114/64</td>
<td>114/79</td>
<td>4.6</td>
<td>60</td>
<td>77</td>
</tr>
<tr>
<td>2. 3-20-62</td>
<td>Basal</td>
<td>184/15</td>
<td>111/57</td>
<td>105/54</td>
<td>100</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>After isoproterenol*</td>
<td>259/14</td>
<td>105/54</td>
<td>102/51</td>
<td>3.5</td>
<td></td>
<td>70</td>
<td>50</td>
</tr>
<tr>
<td>3. 4-9-62</td>
<td>Two weeks after reserpine† therapy</td>
<td>151/19</td>
<td>102/19</td>
<td>102/51</td>
<td>3.5</td>
<td>70</td>
<td>50</td>
<td></td>
</tr>
<tr>
<td>4. 8-7-62</td>
<td>Four months after reserpine† therapy</td>
<td>158/24</td>
<td>105/24</td>
<td>101/50</td>
<td>4.7</td>
<td>78</td>
<td>60</td>
<td></td>
</tr>
<tr>
<td></td>
<td>After isoproterenol*</td>
<td>200/30</td>
<td>102/50</td>
<td>102/51</td>
<td>3.5</td>
<td></td>
<td>70</td>
<td>50</td>
</tr>
<tr>
<td>5. 11-12-64</td>
<td>Basal</td>
<td>199/20</td>
<td>123/20</td>
<td>123/73</td>
<td>8.4</td>
<td>100</td>
<td>100</td>
<td>84</td>
</tr>
</tbody>
</table>

*Isoproterenol 1.23 μg/min.
†Reserpine 0.25 mg b.i.d.

cording to previously described techniques. The ventricular volume studies were obtained with a biplane cineangiographic unit designed to record 60 motion picture frames per second on 35-mm film. The radial arterial pressure and electrocardiogram were continuously recorded, and the left ventricular pressure was monitored before and after the injection of 35 cc of contrast material into the left ventricle. Biplane filming was continued until the contrast material had been cleared from the ventricular chamber. Following removal of the patient from the laboratory and precise repositioning of the image tubes as in the patient study an aluminum bar, 8 by 2 by 2 cm, was filmed in biplane for correction of magnification in subsequent film projection.

The 35-mm biplane films were reduced to 16 mm, and the projector-to-screen distance adjusted so that the image of the bar coincided with the actual bar. Calculations of the left ventricular volume were made by the Dodge technique, which is based on the premise that the left ventricle can be considered an ellipsoid. Each calculated volume was corrected by a factor obtained from a regression line determined from volume observations of barium-filled left ventricles of human postmortem hearts. The individual volume observations were plotted in a sequential manner and related to simultaneous radial artery pressure and electrocardiogram.

Results

Catheterization Data

The cardiac catheterization data from each study (table 1) demonstrated a significant systolic gradient between the left ventricular chamber and the subvalvular region. All observations of left ventricular end-diastolic pressure were found to be abnormally elevated, although the patient had no evidence of edema or fluid retention during any of these studies. Premature ventricular beats were followed by beats which had an increased systolic gradient between the left ventricle and radial artery and a diminished peripheral arterial pulse pressure when this pulse pressure was compared to that associated with other normally conducted beats. Indicator-dilution curves sampled from the left atrium after injection into the left ventricle demonstrated mitral regurgitation.

Left Ventricular Volume Data

Since the determinations of ventricular volume performed during the catheterization on November 12, 1964, are based on measurement of the opacified left ventricular chamber, selected systolic and diastolic biplane images during the first normal sinus beat following the premature ventricular contraction (post PVC beat) and the subsequent normal sinus beat are illustrated. Figure 1 illustrates anteroposterior and lateral ventricular cineangiographic frames during end-systole and end-diastole during the first normally conducted beat following the premature ventricular contraction. Figure 2 illustrates selected end-systolic and end-diastolic frames obtained from the second sinus rhythm beat after the premature ventricular contraction. The presence of mitral insufficiency can be appreciated in both illustrations, and the left atrium appears slightly more opacified in the
first beat than the second beat following the PVC.

The sequential determinations of ventricular volume are shown in figure 3 and are correlated with the simultaneously recorded radial artery pressure and electrocardiogram. It is evident that a premature beat occurred during the injection of contrast material, and the earliest ventricular images that could be traced were those occurring at the end of the diastolic period following the premature ventricular contraction. This end-diastolic period is considered the initial phase of the subsequent cardiac cycle.

The first beat following the PVC had an end-diastolic volume (EDV) of 123 cc, an end-systolic volume (ESV) of 22 cc, a left ventricular stroke volume (LVSV) of 101 cc, and an ejection fraction of 83%; the second beat had an EDV of 119 cc, an ESV of 30 cc, an LVSV of 89 cc, and an ejection fraction of 75%; and the third beat had an EDV of 111 cc, an ESV of 27 cc, an LVSV of 84 cc, and an ejection fraction of 76%. The EDV for the fourth cycle was 120 cc, but ventricular opacification did not permit tracing additional volumes. Although the radial arterial systolic pressure (fig. 3) in the first cycle following the PVC was less than the systolic pressure in the subsequent beats, the LVSV was greater in the post PVC beat. Simultaneous left ventricular pressure was not recorded.
during the filming since the contrast material had been injected through the catheter.

**Discussion**

Ventricular volume studies by the thermodilution technique in patients with hypertrophic subaortic stenosis have revealed decreased end-diastolic volumes. However, cardiac arrhythmias and mitral insufficiency introduce errors in the measurement of ventricular volume by this method. Although angiographic studies have described changes in the configuration of the left ventricular chamber in hypertrophic subaortic stenosis, the left ventricular chamber in the patient of this report did not demonstrate a definite area of subvalvular narrowing nor did the contour of the ventricle differ from that observed in other forms of ventricular hypertrophy. Therefore, it was felt that the application of an ellipsoid reference figure for calculation of the angiographic left ventricular volumes in this patient would be valid.

In this patient with hypertrophic subaortic stenosis, the end-diastolic volumes were normal, but the decreased end-systolic volumes resulted in an increased ejection fraction. The left ventricular stroke volume was greater in the post PVC beat (101 cc) than in the subsequent sinus beats (89 and 84 cc). The larger stroke volume was accompanied by a smaller rise in the systolic arterial pressure,
Sequential left ventricular volumes are related to the simultaneous electrocardiogram and radial artery pressure. It is apparent that a premature ventricular contraction occurred with the injection of the contrast material. The radial artery systolic pressure did not increase during the post PVC beat. The end-diastolic volume of the post PVC beat is not significantly larger than the end-diastolic volume of subsequent normal sinus beats.

Summary

Left ventricular volume studies were performed on a patient with hypertrophic subaortic stenosis utilizing cineangiocardiography. During the injection of contrast material the patient had a premature ventricular contraction followed by sinus rhythm. The ventricular volume measurements were made during the post PVC beat and subsequent sinus beats. The volume studies revealed normal end-diastolic volumes and small end-systolic volumes with an increased ejection fraction. Although the left ventricular stroke volume was greater during the post PVC beat than during subsequent beats, the simultaneous arterial systolic and pulse pressure did not rise. The elevated diastolic filling pressure, the normal end-diastolic volumes, and the failure of the end-diastolic volume to increase with a
longer diastolic filling period are compatible with diastolic restriction in hypertrophic stenosis.

References
Ventricular Volume Studies in a Patient with Hypertrophic Subaortic Stenosis
CHARLES E. RACKLEY, ROBERT E. WHALEN and HENRY D. MCINTOSH

Circulation. 1966;34:579-584
doi: 10.1161/01.CIR.34.4.579

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1966 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/34/4/579

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/