Postextrasystolic Ventricular Pressure Responses

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SUMMARY
The peak postextrasystolic ventricular pressure was examined in tracings obtained during routine catheterization of the right and left ventricles in 252 cases. The invariable response in the apparently "normal" left ventricle was a fall or no change in peak systolic pressure, whereas in the obstructed left ventricle, the failed left ventricle, the severely volume-overloaded left ventricle, and in mitral stenosis the more usual pattern was a rise in postextrasystolic ventricular pressure. On the right side the usual response both in the normal and abnormal was a rise in postextrasystolic pressures. Significant exceptions were found when the right ventricle ejected against the aortic impedance and in constrictive pericarditis.

In the intact circulation the outflow impedance and its rate of decline during the compensatory pause would appear to be the major determinant of the postextrasystolic pressure response. There is some evidence that alterations in the extent and time course of ventricular filling may be a factor, but the effects of variations in the degree of postextrasystolic potentiation based on variations in the prematurity of the extrasystole, which can so easily be demonstrated under controlled conditions, appear to be largely obscured except when fixed outflow obstruction is present. The only practical value of this observation may be the ability to recognize an "abnormal" left ventricle when the postextrasystolic beat shows a higher peak ventricular pressure and to recognize a right ventricle in free communication with the aorta when the peak right ventricular postextrasystolic pressure fails to rise. Conversely, a lower peak left ventricular systolic pressure following a premature beat should not be misinterpreted as being due to a failure of postextrasystolic potentiation and augmentation, as it is in fact a normal phenomenon.

Additional Indexing Words:
Postextrasystolic potentiation Ventricular filling
Peak systolic ventricular pressure First derivative of ventricular pressure

The cardiac cycle following a premature contraction is characterized by a ventricular contraction more forceful than the preceding normal contractions. The mechanisms responsible for the increased force of contraction are complex but in the intact circulation are thought to be due to the combined effects of postextrasystolic potentiation and increased ventricular filling during the compensatory pause. It is fairly generally accepted that the more forceful postextrasystolic contraction in normal individuals manifests itself by higher peak arterial and ventricular systolic pressures, a larger arterial pulse pressure, and a longer duration of ejection. We were therefore somewhat puzzled to observe that in our patients with apparently normal left ventricular hemodynamics the postextrasystolic peak ventricular pressure was always either lower or the same
as that of the preceding normal beats, whereas
in the right ventricle, the obstructed left
ventricle, and in the presence of left ventricu-
lar failure the more usual response was a rise
in peak postextrasystolic ventricular pressure.

It is the purpose of this paper to document
these findings and to discuss the mechanisms
which determine the postextrasystolic ventricu-
lar pressure response. It is also suggested
that a study of the postextrasystolic ventricu-
lar pressure may be of some value in assessing
the hemodynamic and functional state of the
ventricle.

Materials and Methods

Right and left ventricular pressure pulses
recorded at the time of routine studies of left
and right heart catheterization were examined for
the presence of single premature systoles. The
extrasystoles were all of ventricular origin and
either occurred spontaneously or were deliberate-
ly provoked by mechanical stimulation of the
ventricular endocardium with the catheter. Trac-
ings showing atrial fibrillation, multiple extrasyst-
toles, coupling, or interpolated extrasystoles were
excluded, as were the cases where the postextra-
systolic electrocardiogram did not show a normal
sinus beat. The peak systolic and end-diastolic
pressures before and after extrasystole were
measured in all cases. The timing of the
extrasystole was expressed as the coupling index,
i.e. the ratio of the coupling interval to the
previous normal R-R interval. Since the extrasys-
toles were all ventricular, the postextrasystolic
pause was always fully compensatory. In tracings
where fast-speed recordings of 100–200 mm/sec
were made, the maximum rate of rise of the pre-
and postextrasystolic beats was measured directly
from the pressure curve, and in a few cases the first
derivative of the ventricular pressure was
recorded by means of a differentiating circuit.
Simultaneously recorded peripheral arterial trac-
ings were available in some cases, and the change
in peak systolic and pulse pressure was noted in
the postextrasystolic beat.

We found 128 left ventricular and 104 right
ventricular tracings suitable for analysis. The left
ventricular material was grouped according to the
type of hemodynamic abnormality into the
following categories:

(1) "Normal" Left Ventricle. These in-
cluded patients with atrial septal defect, pulmo-
mary stenosis, and pulmonary hypertension;
patients with angina pectoris in whom the heart
size, resting electrocardiogram, cardiac output,
left ventricular end-diastolic pressure, and ven-
tricular wall movement during ventriculography
were all normal; and patients investigated for
difficult murmurs or chest pain in whom no
structural or hemodynamic abnormality of any
kind was found (30 cases).

(2) Outflow Obstruction. Cases with valvar
or subvalvar aortic stenosis and hypertrophic sub-
aortic stenosis (21 cases).

(3) Myocardial Disease. Cases with ischemic
muscle damage or idiopathic congestive cardio-
myopathy (21 cases).

(4) Volume Overload Mild or Compensated.
Cases with ventricular septal defect, patent
ductus, or mitral or aortic insufficiency with left
ventricular end-diastolic pressures of less than
15 mm Hg (23 cases).

(5) Volume Overload Severe or in Failure.
The same defects as in (4) but with left
ventricular end-diastolic pressures of 15 mm Hg
or more (15 cases).

(6) Inflow Obstruction. Cases with mitral
stenosis (six cases).

(7) Increased Systemic Resistance. Cases
with idiopathic hypertension, coarctation, or
aortic arteritis (nine cases).

(8) Constrictive or Restrictive Defects. Con-
strictive pericarditis (three cases).

The tracings obtained from the right ventricle
were divided into four groups: (1) a large mixed
group including normal hearts, atrial septal
defect, pulmonary stenosis, pulmonary hyperten-
sion, and myocardial disease (70 cases); (2)
cases of tetralogy of Fallot, transposition, and
pulmonary atresia where the right ventricle
effectively ejected into the aorta (15 cases); (3)
cases with equal or near equal pulmonary and
systemic pressures due to large ventricular septal
defect or patent ductus (eight cases); and (4)
cases with constrictive pericarditis (10 cases).

Results

The premature beats examined were all of
ventricular origin followed by a fully compen-
satory period, but atrial premature beats
appeared to have similar effects. In two cases,
premature beats were provoked by both left
and right ventricular stimulation without an
apparent change in the type of postextrasysto-
lic pressure responses. The simultaneously
recorded electrocardiogram often showed S-T
segment and T-wave changes in the postextra-
systolic beat particularly in abnormal left
ventricles. These changes consisted of flattening
or inversion of T waves, depressed S-T
segments, and prolongation of QT periods;
they were, however, inconsistent and bore no
The type 1 postextrasystolic pressure response. The left ventricular pressure following the premature beat has a lower peak systolic pressure, and subsequent beats show a gradual rise back to the control level. Despite the lower peak pressure the $dp/dt$ of the postextrasystolic beat is increased.

Figure 1

The type 2 postextrasystolic pressure response. The right ventricular pressure in a case of tetralogy of Fallot shows no change in peak pressure although the $dp/dt$ is increased.

Figure 2

The timing of the premature beat varied considerably from early (coupling index 0.38) to very late (index 0.93), the average for all premature beats being 0.64, and the mean values of coupling index for all types of
The type 3 postextrasystolic pressure response. Both the peak systolic pressure and the $dp/dt$ of the postextrasystolic beat are greater than the control levels.

The pressure response were similar. In one patient with a normal type 1 pressure response, premature beats occurring at coupling indices varying between 0.45 and 0.71 were not observed to have an effect on the magnitude of the ventricular pressure response (fig. 5), but, in another with severe pulmonary stenosis and premature systoles occurring at coupling indices of 0.48 to 0.93, higher postextrasystolic pressures were observed after the earlier premature beats (fig. 6). However, variations in timing of the premature beat were never observed to change the pressure response from one type to another. Whatever the effect on peak postextrasystolic pressure the influence of the timing of the premature beat could clearly be observed on the first derivative of the postextrasystolic ventricular pressure: the earlier the premature beat the greater the rise in $dp/dt$ (fig. 5 and 6).

All postextrasystolic beats tended to have the following features in common: the end-diastolic pressure was higher in 80% of all cases. In constrictive pericarditis, however, an increase in end-diastolic pressure never occurred. Overloaded or failed ventricles and cases with mitral stenosis had larger increases in end-diastolic pressure than "normals." The maximum rate of rise of the postextrasystolic ventricular pressure was increased by 17–49% even in cases where the peak systolic pressure was reduced. In two cases the recorded first derivative showed little change in peak amplitude probably because ejection commenced at a lower aortic diastolic pressure, before the peak value could be reached.

Simultaneous tracings recorded from a peripheral artery always showed a larger pulse pressure except in some cases of hypertrophic obstructive cardiomyopathy, but the peak systolic pressure of the peripheral pulse did not necessarily reflect the peak pressure of the ventricular or central aortic tracing. Not infrequently there was a rise in peak postex-
The relationship of the timing of the extrasystole expressed as the coupling index, i.e., the ratio of the coupling interval to the previous normal R-R interval to the percent change in peak left ventricular systolic pressure (solid circles) and dp/dt (crosses) in a case with the type I response. The timing of the premature beat has no effect on the peak systolic pressure of the postextrasystolic beat, but the earlier the extrasystole the greater the rise in dp/dt.

The relative frequency of the four types of postextrasystolic pressure response for each of the hemodynamic categories is given in tables 1 and 2. Mean values for the change in systolic pressure, end-diastolic pressure, coupling index, and percent change in dp/dt are given.

When the left ventricular material is considered, the striking fact that emerges from this analysis is that all "normal" ventricles showed a type 1 or 2 postextrasystolic response; on the other hand, 95% of cases with outflow obstruction showed a type 3 or 4 response. The one exception was in a case of mild aortic stenosis with a systolic gradient of 20 mm Hg in whom the end-diastolic pressure before the postextrasystolic beat fell rather than rose. In cases with myocardial disease, 76% showed a type 3 or 4 response. Only five cases (24%) showed a type 1 or 2 response, and these had normal cardiac indices and only moderate degrees of cardiomegaly, but similar elevations of end-diastolic pressure when compared with the cases showing the type 3 or 4 response. With volume-overloaded ventricles there was an increase in the frequency of type 3 and 4 responses as the severity of the lesion increased; 9% in the mild, and 53% in severe cases. Systemic hypertension was not a frequent cause of the type 3 or 4 response unless heart failure also was present. Four out of 6 cases with mitral stenosis showed the type 3 response, and the three cases with constrictive pericarditis had a type 1 response.
Simultaneous recordings of left ventricular and femoral arterial pressure following a ventricular premature beat shows no change in peak systolic ventricular pressure, i.e., a type 2 response, but the femoral arterial tracing shows both an increase in peak systolic and pulse pressure.

In the right ventricle the pattern was different. Taken overall, the vast majority including “normals” showed a type 3 response.

An important exception to this finding occurred in the group where the right ventricle effectively ejected against the aortic impedance. In this situation, 81% showed the pattern usually associated with the normal left ventricle, i.e., the type 1 or 2 response. In the group with large ventricular septal defects and patent ductus where the pulmonary pressure approached or equaled systemic, 37% showed a type 1 or 2 response. In constrictive pericarditis, 50% failed to show a rise in systolic pressure in the postextrasystolic beat.

**Discussion**

In experimentally controlled situations, potentiation of the postextrasystolic beat can be shown to occur independently of changes in fiber length or afterload. The degree of potentiation increases with the prematurity of the extrasystole and the number of extrasystoles but decreases if the postextrasystolic pause is too short for full restitution of the contractile process to occur. The potentiating effect may persist with diminishing intensity for several successive beats. Before the rediscovery of postextrasystolic potentiation by Cattell and Gold in 1941, most investigators considered that the increased force of the postextrasystolic contraction was due to increased ventricular filling during the compensatory pause. More recently, since the interest in paired and coupled pacing techniques, the tendency has been to regard postectopic potentiation as a major factor in determining the behavior of the postextrasystolic beat, even when it occurs spontaneously in the intact circulation, and attempts have been made to explain the occasional fall in postextrasystolic ventricular pressure on the basis of the lateness of a premature beat. For several reasons it seems unlikely that variations in the degree of potentiation play an important role in determining the postextrasystolic pressor response in the clinical setting. Most inotropic interventions while having a marked effect on the velocity aspects of ventricular contraction tend to have little or no effect on the peak systolic pressure developed in the intact normal circulation.
Secondly, most of the premature beats studied occurred rather late so that the degree of potentiation might be expected to be much less and the timing of the premature beat less critical than occurs with paired electrical stimulation with the coupling interval set at or near its optimal value. In this study the degree of potentiation as assessed from the increase in $dp/dt$ might well be an overestimation because of the simultaneous increase in ventricular filling.

In our material, variations in potentiation produced by variations in the timing of the ectopic did not appear to have any influence in determining the type of ventricular pressure response elicited—the mean values for the coupling index were similar for all groups and variations of the coupling index in the same patient were not observed to influence the type of postextrasystolic ventricular pressure response. However, in the presence of outflow obstruction the timing of the premature beat did appear to influence the magnitude of pressure response (fig. 6).

A major determinant of the tension developed by a muscle is the afterload against which it has to contract. In the intact normal circulation, spontaneously occurring premature beats are followed by a compensatory pause during which systemic and pulmonary vascular beds empty progressively, and the pressure falls in an exponential fashion so that during the postextrasystolic beat the ventricle ejects against a decreased resistance. On the other hand, when a fixed outflow obstruction is present most of the resistance to ejection resides in the area of the stenosis which does not diminish during the compensatory pause. The importance of the outflow impedance in determining the magnitude of the postextrasystolic pressure rise is well recognized. Thus, the exaggerated postextrasystolic rise in pressure in cases with pulmonary and aortic valve stenosis\(^6\) and the reduced postextrasystolic pressor response in cases with Fallot's tetralogy as opposed to cases with an intact ventricular septum\(^7\) have previously been observed. The difficulty in demonstrating much change in peak systolic pressure in the intact nonfailing heart during paired pacing or even after a single premature beat compared with the obvious changes seen in an isovolumic preparation have been attributed to the different afterloads in the two situations.\(^6\)

In the present analysis, the almost complete separation of normal from obstructed left ventricles, according to whether the postextrasystolic peak pressure fails to rise (type 1 or 2) or rises (type 3 or 4) is strong evidence that the outflow impedance is a major determinant of the postextrasystolic ventricular pressure. It would thus appear that in the normal left ventricle, following a spontaneous extrasystole, the degree of augmentation and potentiation are insufficient to offset the simultaneous reduction in aortic impedance, and the peak pressure developed by the postextrasystolic beat is lower or unchanged. In the severely obstructed vehicle, however, similar degrees of augmentation and potentiation meet an unchanged outflow impedance with a consequent rise in peak postextrasystolic pressure. As well as accounting for the different responses seen in normal and obstructed left ventricles, the variation in outflow impedance probably also accounts for the different responses seen in the normal right and left ventricles. Previous studies on the effects of paired pacing on the left and right ventricular pressures have consistently shown larger increases in right than left ventricular pressure.\(^6\) This has been explained by the different impedance characteristics of the pulmonary and systemic vascular beds, the pulmonary impedance increasing as a function of ejection velocity more markedly than does the systemic. A second factor may be the different rate and extent of pressure decline in the two systems during the long diastolic pause, the aortic pressure falling to relatively lower levels than the pulmonary. In the normal heart then, even in the absence of any differences in end-diastolic volume or degree of potentiation in the two ventricles, the right ventricular systolic pressure will rise, whereas the left will fall during the postextrasystolic beat, purely due to the different impedances.
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<th>Hemodynamic category</th>
<th>Type of postextrasystolic pressure response</th>
<th>No. of cases</th>
<th>% of cases</th>
<th>Mean change in syst pressure (mm Hg)</th>
<th>Change in diast pressure (mm Hg)</th>
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### Table 2

**Postextrastolic Pressure Response in the Right Ventricle**

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<th>% of cases</th>
<th>Mean change in syst pressure (mm Hg)</th>
<th>Change in diast pressure (mm Hg)</th>
<th>Heart rate (beats/min)</th>
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<td>+8</td>
<td>+1.2</td>
<td>0-3</td>
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<tr>
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<td>Type 4</td>
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<tr>
<td>VSD and ductus with equal pressures</td>
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Abbreviation: VSD = ventricular septal defect
of the pulmonary and systemic vascular beds.

The dominating influence of the aortic impedance on the behavior of the right ventricular postextrasystolic pressure in tetralogy of Fallot, pulmonary atresia, transposition, and to a lesser extent in large high-pressure ventricular septal defects and patent ductus is such that a high incidence of type 1 or 2 response is seen following a premature beat in patients with these defects. During cardiac catheterization of the hypertensive right ventricle, failure of the pressure to rise after a premature beat may be taken as a definite indication of the presence of a free communication between this chamber and the left ventricle or aorta.

Several factors may be responsible for the high incidence of type 3 or 4 response seen in the presence of left ventricular failure. Calculated peripheral resistance is increased, and the peripheral arterioles become increasingly stiff and fail to dilate normally after vasodilator stimuli, possibly due to sympathetic arteriolar constriction and also to sodium retention by the arterial wall. Stroke volume and ejection velocity are decreased so that the amount of elastic recoil during diastole may be reduced. These factors would tend to reduce the aortic runoff during diastole, and the potentiated and augmented postextrasystolic beat would eject against an aortic impedance which has not decreased with time to the same extent as in health. It is well known that the hemodynamic effects of paced pacing are more easily demonstrable in heart failure than in normal hearts, but whether this is due to a greater degree of potentiation obtained in the failure situation or to the potentiating effect simply being more easily demonstrable hemodynamically, as with digitalis, is uncertain. The third possibility which ought to be considered is that the increment in end-diastolic volume following a premature beat is greater in failing than in normal hearts. At first sight this would appear unlikely because failed ventricles operate on the steep part of their passive pressure volume curve and also at the plateau of their ventricular function curve, so that during the compensatory pause even large increments in filling pressure would produce little change in diastolic volume, and even large increases in end-diastolic volume would produce little further increase in stroke volume. There is, however, fairly convincing evidence that during paired pacing or even after a single premature beat ventricular compliance is increased. Our data indicate that the increment in end-diastolic pressure following the premature beat is about three times greater in heart failure than in normal cases, and in the presence of a postextrasystolic increase in compliance this may well represent better ventricular filling.

Variations in the degree of ventricular filling dependent on the length of diastole in the intact heart have been noted to influence the postextrasystolic ventricular pressure, particularly when the premature beat is late or interpolated without a compensatory pause. The atrial contraction may also be important for adequate filling of the first postextrasystolic beat. All variations in filling due to too short a diastolic pause or to lack of an atrial contribution have been excluded from our material by selection. There is some evidence, however, that changes in the extent and time course of ventricular filling in cases with mitral stenosis and constrictive pericarditis may be important determinants of the postextrasystolic ventricular pressure. In severe mitral stenosis, the dynamics of ventricular filling are altered from the normal pattern to an almost continuous but exponentially declining inflow with a reduced atrial contribution making ventricular filling much more time-dependent than in the normal heart. Four of the six cases with isolated mitral stenosis showed a postextrasystolic ventricular pressure rise, and all cases were noted to show a larger than normal increment in end-diastolic pressure after the compensatory pause. The increasing incidence of type 3 and 4 pressure responses in volume-overloaded left ventricles suggests that increased ventricular filling during the compensatory pause might be the responsible factor. In the presence of severe
volume overload as defined in this analysis there must be considerable overlap with the heart-failure situation, and the high incidence of type 3 and 4 pressure responses is not unexpected. In constrictive pericarditis filling occurs rapidly early in diastole and is then abruptly halted by the rigid pericardium. Filling is restricted but far less time-dependent than in normal hearts. Five of 10 cases with constrictive pericarditis failed to show the normal rise in right ventricular pressure after a premature beat, and none showed any increase in diastolic pressure after the compensatory pause.

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