Berheim “a” wave: obstructed right ventricular inflow or atrial cross talk?

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Abstract

Objective—To study the possible mechanisms underlying the dominant “a” wave in the jugular venous pulse seen in patients with left ventricular hypertrophy (Bernheim “a” wave).

Design—Prospective examination of the left ventricular transverse and longitudinal axes, transmitral and tricuspid flows, and jugular venous pulse recordings.

Setting—Tertiary referral centre for cardiac disease.

Subjects—23 patients with left ventricular hypertrophy of various aetiologies and a dominant “a” wave in the jugular venous pulse. Controls were 21 normal volunteers.

Results—Early diastolic filling of the right ventricle was normal. During right atrial systole the (mean(SD)) tricuspid ring motion was exaggerated (1.2(0.4) v 0.8(0.2) cm, p < 0.001) and Doppler A wave velocity slightly increased (0.3(0.1) v 0.2(0.08) m/s, p < 0.01), although the E wave remained dominant. By contrast left ventricular isovolumic relaxation time was longer than normal (70(20) v 55(10) ms, p < 0.001) with wall motion incoordinate in the septal long axis, 15% (9-5%) v 6-6%/ (3%) total excursion occurring before mitral valve opening. During early filling the extent of long axis motion was decreased to 0-6(0.5) cm from 1-1(0.2) cm, (p < 0.001) and 0.5(0.2) cm from 0.9(0.2) cm, (p < 0.0001) at the left and septal sites, and similarly its peak lengthening rate reduced to 5-4(2.5) cm/s from 10(3) cm, (p < 0.001) and 4-3(2.2) cm/s from 8(2) cm, (p < 0.001).

The atrial component of long axis lengthening was increased to 43%/ (18%) from 29% (6%) (p < 0.01) and 55% (15%) from 33% (8%) of the total excursion (p < 0.0001). Left ventricular E/A ratio was less than normal (0.9(0.8) v 1.4(0.4), p < 0.05).

Conclusions—There is no evidence of obstruction or any other disturbance of early diastolic right ventricular inflow in Bernheim's syndrome. It is possible that the haemodynamically appropriate increase in left atrial activity is mirrored on the right side due to shared interatrial myocardial fibres. This could represent a form of atrial interaction.

An “a” wave is commonly seen in the jugular venous pulse in patients with left ventricular hypertrophy, and is traditionally referred to as being part of the Bernheim syndrome. It has been ascribed to distortion of the right ventricular cavity by a bulging interventricular septum,1 or to abnormal diastolic function of the left ventricle affecting that of the right.2 Wood, while commenting on this association between left sided hypertrophy and a right sided “a” wave, pointed out that although necropsy evidence of obstruction to the right sided inflow was suggestive, definite proof for this mechanism was lacking.3 It therefore seemed of interest to reinvestigate the question with echocardiographic and Doppler techniques, as these approaches have proved useful in studying other disturbances of ventricular filling.

Patients and methods

PATIENTS

M mode echocardiograms, Doppler traces, and jugular venous pulses were recorded in 23 patients with a dominant “a” wave in the venous pulse and left ventricular hypertrophy. We excluded patients with clinically apparent coronary artery disease, organic mitral valve disease, increased overall venous pressure, or more than mild aortic regurgitation causing left ventricular cavity enlargement. Six patients had left ventricular hypertrophy due to hypertrophic cardiomyopathy, 13 had aortic valve stenosis, two had undergone prothetic aortic valve replacement, and two had systemic hypertension. All had a normal left ventricular cavity size. There were 15 men and eight women aged 21 to 82 years (mean (SD) 62 (18)). Twenty one additional healthy volunteers, 13 men and eight women of mean (SD) age 51 (11) years without clinical, electrocardiographic, or echocardiographic evidence of heart disease served as normal controls.

METHODS

M mode and cross sectional echocardiograms were taken with a Hewlett Packard Sonos 1000 echocardiograph and a 2-5 MHz phased array transducer as the patient lay in the partial left lateral position. Standard M mode echocardiograms of the left ventricular minor axis were recorded just below the level of the mitral valve leaflets showing clear continuous echoes from both surfaces of the septum and posterior wall.
Septal and posterior wall thicknesses were measured from their leading edges at the time of the onset of the Q wave of the electrocardiogram (end diastole). The timing of aortic valve closure was taken as the onset of the first high frequency vibration of the aortic component of the second heart sound, and isovolumic relaxation as the time interval between this and the onset of separation of mitral valve leaflets.

Cross sectionally guided M mode echocardiograms of the left and right ventricular long axes were then taken after longitudinal placement of an M mode cursor from the apex through the atrioventricular ring at the three standard sites; left, septal (central fibrous body), and right, visualised on an apical four chamber cross sectional view. Minor and long axis echocardiograms were digitised.

DOPPLER
Transmitral and tricuspid forward flow velocities were recorded at cusp tip level with Hewlett Packard or Doppler equipment in pulsed mode (2.0 or 3.5 MHz transducer).

VENOUS PULSE
The jugular venous pulse was recorded from the internal jugular vein on the right side of the neck with the patient lying supine in the optimal position, often less than 45 degrees, as overall venous pressure was not significantly raised in any patient. A Cambridge pulse transducer was used with a time constant of 4 s.

All records were made photographically at a paper speed of 10 cm/s with a Honeywell (Ecoline) strip chart recorder with simultaneous electrocardiogram (lead II) and phonocardiogram.

MEASUREMENTS
From the original traces, we measured left ventricular end diastolic and end systolic dimensions, septal thickness, and isovolumic relaxation time. Peak rate of increase of transverse dimension was derived by digitisation. On the pulsed Doppler traces of the transmitral and tricuspid flow we measured peak E and A wave velocities and found the corresponding E/A ratio.

From the long axis traces (fig 2), we measured the overall amplitude of excursion and any change in dimension during isovolumic relaxation. Early diastolic lengthening was taken as the increase in length from the minimum value to that at the onset of diastasis; and atrial contraction was the further backward displacement of the atrioventricular ring toward the atrium after the P wave. From the digitised traces, the peak rate of early diastolic lengthening was measured at each of the three sites of the long axis.

Jugular venous pressure traces were assessed with reference to the presence of a “x” or a “y” descent accompanying the “a” waves. An “x” descent was taken as a fall in venous pressure, of amplitude one quarter that of the “a” wave that was complete before P2, (the pulmonary component of the second heart sound) on the phonocardiogram; a “y” descent was one of similar amplitude occurring after P2.

RESULTS
RIGHT VENTRICLE
Tables 1–4 show the results. Right ventricular filling pattern was effectively normal, showing a dominant E wave on the transtricuspid Doppler trace. The amplitude of the “a” wave was slightly although consistently larger than normal (p < 0.01). Early diastolic excursion of the ring was normal in both absolute and relative terms, as was peak lengthening rate. The excursion during atrial systole was consistently but modestly increased (p < 0.01). The jugular venous pulse showed a dominant “a” wave in all patients, by definition. Additional small “x” descents were present in 22 patients and a “y” descent in one.

(fig 1)

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Minor axis measurements (mean [SD])</th>
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<tbody>
<tr>
<td>Variable</td>
<td>Normal</td>
</tr>
<tr>
<td>Minor axis excursion (cm)</td>
<td>1-7 (0-3)</td>
</tr>
<tr>
<td>End diastolic diameter (cm)</td>
<td>4-9 (0-5)</td>
</tr>
<tr>
<td>End systolic diameter (cm)</td>
<td>3-3 (0-5)</td>
</tr>
<tr>
<td>Shortening fraction</td>
<td>0-3 (0-1)</td>
</tr>
<tr>
<td>Septal thickness (cm)</td>
<td>1-0 (0-1)</td>
</tr>
<tr>
<td>Posterior wall thickness (cm)</td>
<td>1-0 (0-1)</td>
</tr>
<tr>
<td>Peak thinning rate (cm/s)</td>
<td>11 (2-7)</td>
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</tbody>
</table>

*Significantly different by definition.

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Long axis measurements (mean [SD])</th>
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</thead>
<tbody>
<tr>
<td>Variable</td>
<td>Normal</td>
</tr>
<tr>
<td>Total excursion (cm)</td>
<td>L: 1-5 (0-3)</td>
</tr>
<tr>
<td>Total excursion (cm)</td>
<td>S: 1-4 (0-2)</td>
</tr>
<tr>
<td>Total excursion (cm)</td>
<td>R: 2-5 (0-4)</td>
</tr>
<tr>
<td>Early diastole (cm)</td>
<td>L: 1-1 (0-2)</td>
</tr>
<tr>
<td>Early diastole (cm)</td>
<td>S: 0-9 (0-2)</td>
</tr>
<tr>
<td>Early diastole (cm)</td>
<td>R: 1-6 (0-3)</td>
</tr>
<tr>
<td>Atrial A wave (cm)</td>
<td>L: 0-4 (0-1)</td>
</tr>
<tr>
<td>Atrial A wave (cm)</td>
<td>S: 0-5 (0-2)</td>
</tr>
<tr>
<td>Atrial A wave (cm)</td>
<td>R: 0-8 (0-2)</td>
</tr>
<tr>
<td>Atrial-total excursion (%L)</td>
<td>29 (6)</td>
</tr>
<tr>
<td>Atrial-total excursion (%S)</td>
<td>33 (8)</td>
</tr>
<tr>
<td>Atrial-total excursion (%R)</td>
<td>35 (5)</td>
</tr>
<tr>
<td>Peak thinning rate (cm/s)</td>
<td>L: 10 (3)</td>
</tr>
<tr>
<td>Peak thinning rate (cm/s)</td>
<td>S: 8 (2)</td>
</tr>
<tr>
<td>Peak thinning rate (cm/s)</td>
<td>R: 10 (2)</td>
</tr>
</tbody>
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L, left; S, septal; R, right sites of atrioventricular ring motion.

<table>
<thead>
<tr>
<th>Table 3</th>
<th>Isovolumic relaxation measurements (mean [SD])</th>
</tr>
</thead>
<tbody>
<tr>
<td>Variable</td>
<td>Normal</td>
</tr>
<tr>
<td>Awave-mitral valve opening (ms)</td>
<td>55 (10)</td>
</tr>
<tr>
<td>Dimension changes during IVR(%)</td>
<td>8-6 (5)</td>
</tr>
<tr>
<td>Minor axis</td>
<td>10 (7-5)</td>
</tr>
<tr>
<td>Long axis L</td>
<td>10 (7-5)</td>
</tr>
<tr>
<td>Long axis S</td>
<td>6-6 (3)</td>
</tr>
<tr>
<td>Long axis R</td>
<td>11 (9)</td>
</tr>
</tbody>
</table>

IVR, Isovolumic relaxation; other abbreviations as for table 2.

<table>
<thead>
<tr>
<th>Table 4</th>
<th>Pulsed Doppler measurements (mean [SD])</th>
</tr>
</thead>
<tbody>
<tr>
<td>Variable</td>
<td>Normal</td>
</tr>
<tr>
<td>Mitral E wave velocity (m/s)</td>
<td>0-7 (0-1)</td>
</tr>
<tr>
<td>Mitral A wave velocity (m/s)</td>
<td>0-5 (0-1)</td>
</tr>
<tr>
<td>Mitral E/A ratio</td>
<td>1-4 (0-4)</td>
</tr>
<tr>
<td>Tricuspid E wave velocity (m/s)</td>
<td>0 (0-15)</td>
</tr>
<tr>
<td>Tricuspid A wave velocity (m/s)</td>
<td>0 (0-08)</td>
</tr>
<tr>
<td>Tricuspid E/A ratio</td>
<td>1-9 (0-4)</td>
</tr>
</tbody>
</table>

E, early diastolic; A, late diastolic.
Note the simultaneous (ECG) diastolic excursion aortic and phonocardiogram. Note the dominant "a" wave and "x" descent.

LEFT VENTRICLE

On M mode, left ventricular transverse dimensions and shortening fraction were all normal. Isovolumic relaxation time, however, was prolonged (p < 0.001) and the peak rate of increase of transverse dimension was reduced (p < 0.01). Long axis motion was also abnormal. Its overall excursion was reduced due to a significant reduction in the early diastolic component, affecting both free wall and septum (both p < 0.001). This was accompanied by a decrease in peak rate of early diastolic lengthening at both sites (p < 0.001). The amplitude of the early diastolic component included the lengthening that occurred during isovolumic relaxation. This was significantly increased at the septum (p < 0.01); so that the lengthening that occurred during filling itself—that is, after mitral valve opening—was even further reduced here in the patients with left ventricular hypertrophy (fig 2).

On the transmitral flow velocity trace, E wave velocities were reduced and A wave velocities significantly increased (both p < 0.05) with respect to normal, so that the E/A ratio was correspondingly low (p < 0.05, fig 3)

Discussion

Our patients fulfilled the accepted criteria for a Bernheim "a" wave as they appear in recent publications. All had left ventricular hypertrophy with a dominant presystolic wave in the jugular venous pulse. We found no evidence of separate right sided disease, which might have provided an alternative explanation, and although we did not undertake invasive studies, a normal or prolonged left ventricular isovolumic relaxation time strongly suggests that mean left atrial pressure was normal. The presystolic wave in the venous pulse corresponded with the atrial component of tricuspid flow, which, in all but one patient, could be clearly dissociated from the early diastolic component or E wave. Finally, the amplitude of tricuspid ring motion and transtricuspid flow velocities, although normal in early diastole, were both increased above normal during atrial systole,

Figure 1 Jugular venous pulse (JVP) recording with simultaneous electrocardiogram (ECG) and phonocardiogram (PCG) from a patient with left ventricular hypertrophy. Note the dominant "a" wave and "x" descent.

Figure 2 (A) Long axis M mode recordings from a normal person, taken at the left (L), septal (S), and right (R) sites of atrioventricular ring motion showing the normal relations between early (E), late (A), and total (T) diastolic excursions. (B) Long axis M mode recording from a patient with aortic stenosis and left ventricular hypertrophy. Note the decrease in early diastolic excursion and rate of lengthening, and also the relative increase in A at all three sites. Both recordings are made with simultaneous electrocardiogram (ECG) and phonocardiogram (PCG). Vertical line corresponds to the timing of aortic closure (A2). Vertical scale is in cm.
Figure 3  (A) Pulsed wave Doppler transmitral flow velocity trace from a patient with aortic stenosis and left ventricular hyper trophy. Note the dominant late diastolic A wave and small E/A ratio. (B) Pulsed wave Doppler trace of the tricuspid flow velocities from the same patient. Note the normal E wave and E/A ratio. Abbreviations as for Fig 2.

providing further evidence for abnormal mechanical activity of the right atrium. We conclude, therefore, that a true “a” wave can occur in the jugular venous pulse of patients with left ventricular hypertrophy. Its mechanism differs fundamentally from the presystolic wave seen in those with dilated cardiomyopathy or severe pulmonary hypertension, where a very short filling time allows only a single summation flow pulse across the valve.

The name of Bernheim is associated with the syndrome under study because he suggested that left ventricular disease might directly cause right ventricular stenosis, thereby causing “l’asystole veineuse.” Bernheim himself made no objective observations of the venous pulse, though he noted that the neck veins were engorged in his patients. The idea of venous asystole is no longer current, but a few years before Bernheim, Mackenzie had ascribed the “v” wave of severe ventricular disease to right atrial paralysis. The exact nature of the “stenose” of the right heart produced by bulging of interventricular septum has never been clearly delineated. On general grounds its effect on ventricular filling might manifest itself as physical obstruction of the right ventricular inflow tract, interference with the mechanisms underlying rapid right ventricular filling, or as increased right ventricular passive stiffness. We found no evidence of any of these. Although overall venous pressure was not raised, early diastolic E wave velocities were normal on transtricuspid Doppler. This finding excluded both physical obstruction to the inflow and significant impairment of early diastolic filling mechanisms, whereas a normal venous pressure until the end of diastasis would seem to exclude any clinically important loss of ventricular compliance. In our patients, therefore, we found the rather unexpected combination of enhanced right atrial activity with normal early diastolic filling.

The unusual state of affairs on the right side of the heart is highlighted by comparing it with that on the left. Although left ventricular cavity size and shortening fraction were both normal, isovolumic relaxation time was prolonged and wall motion before mitral valve opening was incoordinate. We have previously shown that both of these abnormalities independently reduce the amplitude of the E wave on the transmitral Doppler trace. At the same time, the absolute extent of early diastolic lengthening of the long axis fell as did the increase in the peak rate of transverse and long axes. As a result of these disturbances to early diastolic filling, A wave flow velocities were increased as was the relative increase in the long axis during atrial systole. By obvious contrast to events on the right side of the heart, therefore, there was clear evidence on the left that isovolumic relaxation and early diastolic filling were both abnormal, with a corresponding increase in ventricular volume during atrial systole.

The setting in which increased atrial activity occurred thus differed considerably on the two sides of the heart. On the left, it seemed homeostatic, the increased atrial contribution compensating for the reduced early diastolic flow. This allowed stroke volume to be maintained at normal filling pressure by mechanisms that are well understood. Increased right atrial activity cannot be so easily explained. Early diastolic flow velocity was normal. The atrial component of the transtricuspid flow was small and the increased pressure “a” wave would, if anything, reduce
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venous return. It must thus be asked whether the increased right atrial activity was patho-
logical rather than homeostatic. The theoretical possibility of an inappropriate increase in
the force of atrial contraction has been little
explored. The transfer function between the
force of atrial contraction and the nature and
extent of ventricular disease is poorly under-
stood. It might have been disturbed in our
patients, in some unspecified way, perhaps by
the septal hypertrophy. An alternative expla-
nation might depend on the muscular anatomy
of the atria being very simple com-
pared with that of the ventricles. Even right
or left ventricular hypertrophy cannot exist in
isolation due to the common interventricular
septum. As well as muscle bundles confined
to one or other atrium, additional muscular-
tissue is described as running continuously
around the whole atrial cavity, unrelated to
the interatrial septum. We suggest, there-
fore, that “cross talk” between the two atria
might occur, so that increased mechanical
activity resulting directly from ventricular dis-
ease might not be confined to the atrium
involved. Cross talk is used in communica-
tion theory to imply unwanted spread of
information from the channel carrying it to
another, where it is irrelevant or even cor-
ruping. Here in the present paper, we mean
to suggest that the communication channel “overheard” is that between left ventricle and
left atrium: information arising from some
aspect of diastolic left ventricular disease is
transmitted to the left atrium causing its force
of contraction to increase. This information
flow spreads to the right atrium making its
behaviour inappropriate. If this explanation is
wrong, it might also apply in other diseases,
and to either side of the heart.

Our results also raise the question of how
the phenomenon should be named. There
was no evidence of the mechanical obstruc-
tion to right ventricular filling invoked by
Bernheim, neither is a modest “a” wave
superimposed on an otherwise normal venous
pressure in any way similar to the dilated
eck veins, fluid retention, and hepatic
engorgement that he described. We would
resist any change. Eponyms, even when inac-
curate or fanciful, have proved an effective
means of identifying clinical syndromes that
reflect unexpected associations, at a time
when their underlying mechanisms are
unclear. We suggest that rather than trying to
read into Bernheim’s original papers ideas
that he never clearly entertained, the claims
of tradition should be respected. More impor-
tant, however, the mechanisms underlying
these “a” waves should be investigated fur-
ther. In doing so, understanding of interatrial
and atrioventricular interactions will
undoubtedly be increased.

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