Alternating atrial electromechanical dissociation as contributing factor for pulsus alternans

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SUMMARY Ten patients with mechanical pulsus alternans were studied by echocardiography and mechanocardiography. All had been or were in congestive heart failure. An atrial mechanism for pulsus alternans could be identified in two patients: one with primary congestive cardiomyopathy and one after aortic valve replacement for calcific aortic stenosis. Each strong systole was preceded by an “a” wave, while each weak systole was not. This was documented on both the apexcardiogram and the M-mode echocardiogram. Since both patients were in normal sinus rhythm with regular PP intervals, it was concluded that alternating atrial electromechanical dissociation was either the underlying mechanism or contributed to the pulsus alternans.

Thus, alternating atrial electromechanical dissociation exists and may cause pulsus alternans. Pulsus alternans is not necessarily the result of left ventricular myocardial dysfunction alone.

Pulsus alternans, originally described by Traube in 1872, consists of an alternation of strong and weak ventricular contractions at regular intervals. In most cases, it is clinically recognised by palpation of peripheral arteries. It is never seen in subjects without heart disease and is usually associated with moderate to severe cardiac failure.

Several theories have been postulated to explain pulsus alternans including alternating contractile failure of some myocardial segments, incomplete metabolic recovery, and alternating high and low filling pressures of the left ventricle.

We noted an atrial mechanism for pulsus alternans in one patient, and subsequently retrospectively studied a series of patients with pulsus alternans using external pulse recordings to see if this mechanism occurred in others as well.

Patients and methods

Ten patients with pulsus alternans were identified in the Thoraxcentre between 1974 and 1981, using external pulse recordings. All patients underwent M-mode echocardiography and phonomechanocardiography. The clinical data of the patients are listed in the Table.

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sex</th>
<th>Age (y)</th>
<th>Clinical state</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>24</td>
<td>Congestive cardiomyopathy</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>56</td>
<td>Two months after aortic valve replacment</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>60</td>
<td>Six years after aortic valve replacment</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>62</td>
<td>One month after aortic valve replacment</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>34</td>
<td>Coronary artery disease, congestive heart failure</td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>26</td>
<td>Ventricular septal defect, Ensennenger’s syndrome</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>63</td>
<td>One month after aortic valve replacment</td>
</tr>
<tr>
<td>8</td>
<td>M</td>
<td>42</td>
<td>Congestive cardiomyopathy</td>
</tr>
<tr>
<td>9</td>
<td>M</td>
<td>33</td>
<td>One month after aortic valve replacment</td>
</tr>
<tr>
<td>10</td>
<td>F</td>
<td>66</td>
<td>Coronary artery disease, congestive heart failure</td>
</tr>
</tbody>
</table>

Results

In the presenting patient as well as in a second patient, alternating absence of the “a” wave in both the apexcardiogram and M-mode echocardiogram was observed. Before each strong systole an “a” wave was present on both recordings and before each weak systole the “a” wave was absent. In the other patients this abnormality was not seen.

Fig. 1 shows the simultaneous recording of the electrocardiogram, apexcardiogram, and phonocardiogram of case 1. Note the alternating presence of atrial activity in relation to pulsus alternans visible in the apexcardiogram. In Fig. 2 the M-mode echocardiogram shows the same alternating absence of the “a” wave on the anterior mitral valve leaflet motion.

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Fig. 1  Electrocardiogram (ECG), apexcardiogram (ACG), and phonocardiogram (Phono) of case 1. Note that each strong systole is preceded by an "a" wave (arrows) and the weak systoles are not.

Fig. 2  M-mode echocardiogram of case 1. Note the alternating presence of the "a" wave (arrows) in the anterior mitral valve leaflet (aML) motion. The left ventricular posterior wall (LVPW) amplitude and velocity of motion are higher after an "a" wave. ECG, electrocardiogram; IVS, interventricular septum.

Fig. 3  Electrocardiogram (ECG), apexcardiogram (ACG), and phonocardiogram (Phono) of case 2. Note that each strong systole is preceded by an "a" wave (arrows) and the weak systoles are not. The electrocardiogram shows complete left bundle-branch block.

pattern. Alternating strong and weak left ventricular posterior wall contractions are present: both the amplitude of motion and endocardial velocity of anterior motion are alternating.

Fig. 3 and 4 show the recordings of case 2. In Fig. 3 the alternation of atrial mechanical activity is seen on the apexcardiogram and this was associated with pulsus alternans, as seen in Fig. 4 on the carotid pulse recording. Note that the "a" wave on alternate beats as seen in the anterior mitral valve leaflet tracing always precedes a strong beat on the carotid pulse recording.

Since both patients were in sinus rhythm with

Fig. 4  M-mode echocardiogram of case 2 with simultaneous carotid pulse recording (CR). The alternating presence of the "a" wave on the anterior mitral valve leaflet (aML) motion pattern correlates well with the alternation in the carotid pulse. The motion of the interventricular septum (IVS) is paradoxical (probably the result of operation). The electrocardiogram shows complete left bundle-branch block. ECG, electrocardiogram; LVPW, left ventricular posterior wall.
Alternating atrial electromechanical dissociation

regular PP intervals, the alternating absence of atrial
contraction is caused by a 2:1 electromechanical
dissociation and appears responsible for the pulsus
alternans in these patients.

Discussion

Alternating atrial electromechanical dissociation is a
previously undescribed mechanism. Presumably it
causes alternating strong and weak filling of the
ventricles and therefore pulsus alternans.

Continuous atrial electromechanical dissociation has
been reported after conversion of atrial flutter or fibrila-
tion into sinus rhythm. Often this is only temporary,
but sometimes it can be permanent when atrial fibril-
lation is longstanding. The mechanical failure of the
atrium, in most cases the left atrium, is attributed to
severe atrial muscle damage during the arrhythmia.
Alternating atrial electromechanical dissociation may
also be the result of atrial muscle damage. Left atrial
enlargement seems to be a less important factor, since it
occurred in both the patients with alternating atrial
electromechanical dissociation and those without.

Pulsus alternans is usually associated with severe
left-sided heart disease, such as severe coronary artery
disease, aortic valve disease, and cardiomyopathy. Its
exact cause is not understood, but it is perhaps caused by
(1) alternating contractile failure of some myocardial
segments, or (2) alternating filling of the
ventricles. The most persuasive demonstration of
the latter mechanism was by Veenendaal and Nanda, who
studied a patient with a mitral valve prosthesis and
pulsus alternans by echocardiography. They found
that the echocardiogram showed alternating failure of
prosthetic valve opening, with alternating filling of the
left ventricle. The mechanism of pulsus alternans in the
two patients illustrated here is probably related to that
of Veenendaal's patient, whereas this mechanism
played a minor role in the other eight patients in our
series. The regular presence of the fourth heart sound
in the patients showing alternating atrial electro-
mechanical dissociation should be noted. In spite of
pulsus alternans, its intensity did not vary on the
phonographic recordings. Since the fourth heart sound
is believed to represent rapid changes in late ventricular
filling, the contribution of atrial contraction to the
generation of this sound must be reconsidered, at least
in patients with alternating atrial electromechanical
dissociation. The constant fourth heart sound favours
the theory of alternating contractile failure of some left
ventricular myocardial segments as a cause of pulsus
alternans.

Thus, it is likely that pulsus alternans can be caused
by a variety of mechanisms. Unfortunately, our series
of patients with pulsus alternans is too small for definite
conclusions to be made on the mechanism described in
this report. Further studies may indicate more
precisely its incidence and may provide further insight
into its pathophysiology.

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