Intracardiac Pressure-Sound Correlates of Echographic Aortic Valve Closure

STEPHEN HIRSCHFIELD, M.D., JEROME LIEBMAN, M.D., GORDON BORKAT, M.D., AND CONNIE BORMUTH

SUMMARY Echographic aortic valve closure was compared to the diastolic notch of the aortic pressure and intracardiac A\textsubscript{2} to define the exact temporal relationship of the echographic, pressure, and sound parameters of aortic valve closure. Sixteen children, ages 3–20 years, were evaluated by simultaneous aortic valve echograms, manometric aortic root pressure tracings, and intracardiac phonocardiograms recorded at paper speeds of 200 mm/sec. Our observations demonstrated that echographic coaptation of the aortic valve leaflets coincides with the trough of the aortic pressure incisura and the onset of A\textsubscript{2}. The data suggest that A\textsubscript{2} is a result of valve closure.

ALTHOUGH considerable controversy exists regarding the genesis of the aortic component of the second heart sound (A\textsubscript{2}), two major views prevail. One view contends that A\textsubscript{2} is a direct result of aortic valve closure (AVC).

Another theory relates the genesis of A\textsubscript{2} to the vibrations that result from rapid acceleration and deceleration of blood within the cardiohemodynamic system. The latter theory de-emphasizes the role of semilunar valve closure.

Cineangiographic studies in man\textsuperscript{4} and studies employing electrical techniques in animals\textsuperscript{4} have demonstrated AVC to be coincident with the incisura of the aortic pressure curve but preceding A\textsubscript{2}. Data from these studies supported the view that AVC does not generate A\textsubscript{2}. Echocardiographic studies which allow clear, instantaneous delineation of aortic leaflet closure have been brought recently into the controversy.\textsuperscript{5} Some investigators\textsuperscript{5} appeared to have demonstrated that echographic AVC preceded the surface recording of A\textsubscript{2}, while data from Craig\textsuperscript{6} suggested that echographic AVC and the surface recorded A\textsubscript{2} were simultaneous.

Consequently, a study was undertaken of three simultaneously compared intracardiac events in order to delineate exact temporal relationships of echographic, pressure, and sound parameters of AVC. The three events were echo-

References

graphic aortic valve closure, aortic incisura recorded by micromanometer aortic root pressure tracings, and intracardiac A2. Semilunar valve closure as the immediate cause of A2 production may be inferred if the three events occur simultaneously.

**Methods**

Sixteen patients with a variety of congenital heart diseases were evaluated during diagnostic cardiac catheterization (table 1). In order to re-examine Kumar’s conclusion that A2 was markedly delayed from AVC in patients with valvular aortic stenosis, four children with valvular aortic stenosis were included in our study.

The echocardiogram was recorded with a Unirad 100 series ultrasonoscope employing an Aerotech 2.25 MHz transducer focused at 5 cm. The aortic valve echogram was recorded at the time of cardiac catheterization so that coaptation of the aortic cusps was always visualized (figs. 1 and 2).

The aortic pressure events were recorded utilizing a Millar catheter-tip micromanometer placed immediately above the aortic valve. The micromanometer serves as a variable inductance transducer from which low frequency vibrations are recorded as pressure and higher frequency vibrations are recorded as sound. These characteristics permit intracardiac sound and pressure events to be recorded simultaneously free of transmission delay.

Electrocardiographic, echographic, acoustic, and manometric events were recorded simultaneously on an Irex physiologic recorder at paper speeds of 200 mm/sec with

**TABLE 1. Patient Profile**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>Diagnosis</th>
<th>Onset to peak A2 (msec)</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>3</td>
<td>ALCA</td>
<td>2</td>
</tr>
<tr>
<td>2</td>
<td>5</td>
<td>VSD</td>
<td>4</td>
</tr>
<tr>
<td>3</td>
<td>8</td>
<td>MR</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>8</td>
<td>PDA</td>
<td>0</td>
</tr>
<tr>
<td>5</td>
<td>8</td>
<td>SAS</td>
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</tr>
<tr>
<td>6</td>
<td>9</td>
<td>PDA</td>
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</tr>
<tr>
<td>7</td>
<td>9</td>
<td>ASD</td>
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<tr>
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<tr>
<td>11</td>
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</tr>
<tr>
<td>12</td>
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<td>17</td>
<td>VAS</td>
<td>4</td>
</tr>
<tr>
<td>15</td>
<td>17</td>
<td>LBBB</td>
<td>3</td>
</tr>
<tr>
<td>16</td>
<td>20</td>
<td>VAS</td>
<td>0</td>
</tr>
</tbody>
</table>

Abbreviations: A2 = aortic component of second heart sound; ALCA = anomalous left coronary artery; ASD = atrial septal defect; CoA = coarctation of the aorta; LBBB = left bundle branch block; MR = mitral regurgitation; PDA = patent ductus arteriosus; PS = pulmonic stenosis; SAS = subvalvular aortic stenosis; VAS = valvular aortic stenosis; VSD = ventricular septal defect.
time markers of 10 msec (figs. 1 and 2). Five to ten cardiac cycles were measured to determine temporal relationships of echographic aortic valve closure to intracardiac A2 and the incisura aortic valve. The time from onset of A2 to the initial high frequency component of A2 was measured also from each sound complex.

Results

In all patients echographic coaptation of aortic valve leaflets coincided with the incisural trough and the onset of A2 (figs. 1 and 2).

Peak intensity of A2 was delayed 0–8 msec (mean 3 msec) from echographic aortic valve closure (table 1). In 13 of 16 children, peak A2 was less than 5 msec from onset of A2 and in 5 of 16 high frequency A2 was coincident with onset of A2.

No difference in results was obtained from children with or without aortic valve stenosis.

Discussion

Two major theories have evolved regarding the genesis of heart sounds in general and A2 in particular. Early investigators believed that A2 was produced by apposition of the aortic valve cusps and until recently this theory generally has been accepted.

In recent years the genesis of heart sounds has been carefully evaluated by techniques employing microphones, intracardiac phonocardiograms, and analysis of angiograms performed in conjunction with sound tracings. Data accumulated from these studies suggest that aortic valve closure occurred prior to the onset of A2. MacCanon et al. employed an electrical conducting device to record the relationship between A2 and the aortic incisura in closed chest anesthetized dogs. Their data showed that the onset of A2 was coincident with the aortic pressure incisura, but followed valve closure by a mean of 10 msec. Subsequent observations were consistent with the hypothesis that deceleration of a blood column in the aortic root at the termination of systole, and not closure of aortic leaflets, leads to vibrations audible as S2.

These studies require accurate delineation of excursions of valve leaflets, which echocardiography provides with clarity and in a manner that permits correlation with those intracardiac events related to production of sound. Chandraratna and Anastassiades have reported that echographic aortic valve closure preceded the onset of A2 by 5–25 msec (mean 13). Their data support the contention that A2 is not caused by coaptation of aortic leaflets, but by events which occur after valve closure.

In contrast to these studies, Craig demonstrated unvarying simultaneity of AVC and initial high frequency vibrations of A2. Evaluation of 30 patients in our laboratory, employing external phonocardiography and aortic valve closure reflected by ultrasound, likewise demonstrated simultaneity of echographic AVC and the onset of A2. Peak intensity of high frequency components of A2 followed coaptation by less than 3–5 msec in our patients.

Persistence of this disagreement regarding the genesis of A2 and our own inability to demonstrate convincing differences between echographic AVC and surface recorded A2 prompted our study comparing echographic AVC with two intracardiac events: incisura of the aortic root pressure and intracardiac A2. In all of our patients the onset of intracardiac A2 coincided exactly with the pressure incisura and echographic AVC. This occurred even in patients with valvular aortic stenosis. The higher frequency vibrations of A2 occurred less than 8 msec after echographic AVC in all patients and were delayed by less than 5 msec in 13/16 children.

The average age of our subjects was 11 years, whereas subjects studied by Chandraratna and Anastassiades ranged in age from 10–80 years. The younger patient would have more compliant systemic arterial vessels, but we do not believe this would account for differences in our data.

Intracardiac phonocardiography is free of distortion and dampening that may accompany surface sound recording; initial phononic evidence of intracardiac A2 as well as high frequency A2 could be clearly recorded. The clarity of intracardiac sound recordings may account, in part, for the discordance of our data from that of Chandraratna and Anastassiades.

The onset of intracardiac A2 correlated with the incisural trough of the aortic pressure tracing, which is generally considered as a hemodynamic index of AVC. The onset of A2 was therefore chosen as the sound event that should correlate with echographic AVC. Our results demonstrated simultaneity of echographic aortic closure, aortic pressure incisura and onset of intracardiac A2, and indicate that closure of the aortic valve initiated A2. They also suggest that the higher frequency components of A2 were the results of tensing of the closed semilunar valve.

References

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