Intracardiac Pressure-Sound Correlates of Echographic Aortic Valve Closure

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SUMMARY Echographic aortic valve closure was compared to the diastolic notch of the aortic pressure and intracardiac A₂ 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, micromanometric 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 A₂. The data suggest that A₂ is a result of valve closure.

ALTHOUGH considerable controversy exists regarding the genesis of the aortic component of the second heart sound (A₂), two major views prevail. One view contends that A₂ is a direct result of aortic valve closure (AVC). 1, 2 The other theory relates the genesis of A₂ to the vibrations that result from rapid acceleration and deceleration of blood within the cardiohemic system. The latter theory de-emphasizes the role of semilunar valve closure. 3

Cineangiographic studies in man 4 and studies employing electrical techniques in animals 5 have demonstrated AVC to be coincident with the incisura of the aortic pressure curve but preceding A₂. Data from these studies supported the view that AVC does not generate A₂. Echocardiographic studies which allow clear, instantaneous delineation of aortic leaflet closure have been brought recently into the controversy. 6, 7 Some investigators 8 have demonstrated that echographic AVC preceded the surface recording of A₂, while data from Craigen 9 have suggested that echographic AVC and the surface recorded A₂ 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-
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

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**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>
<td>0</td>
</tr>
<tr>
<td>6</td>
<td>9</td>
<td>PDA</td>
<td>2</td>
</tr>
<tr>
<td>7</td>
<td>9</td>
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<td>13</td>
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<td>VAS</td>
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<td>14</td>
<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 of the aortic root pressure. 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.¹, ²

In recent years the genesis of heart sounds has been carefully evaluated by techniques employing micromanometers, intracardiac phonocardiograms, and analysis of angiocardiograms performed in conjunction with sound tracings.³ Data accumulated from these studies suggested that aortic valve closure occurred prior to the onset of A₂. MacCanon et al.⁴ employed an electrical conducting device to time the relationship between AVC 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. These 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 S₂.³

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. Chandraratna⁶ and Anastassiades⁵ 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 A₂.⁸ 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 Chandraratna⁶ and Anastassiades⁵ 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 Chandraratna⁶ and Anastassiades.⁵

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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S Hirschfeld, J Liebman, G Borkat and C Bormuth

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