The Genesis of Gallop Sounds: Investigation by Quantitative Phono- and Apexcardiography

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SUMMARY The genesis of gallop sounds was investigated in 12 patients by simultaneous measurement of external apexcardiographic (ACG) and left ventricular (LV) pressure, dP/dt, and sound using infinite time constant piezo-resistive pressure transducers with identical sensitivity and frequency responses. Absolute intensity of internal and external sound was quantified. The external transducer was applied to the chest wall with a pressure of 200-400 mm Hg. Six patients had a third heart sound (S₃), eight had a fourth heart sound (S₄) and one patient had a summation gallop. Left atrial (LA) pressure, dP/dt, and sound were also recorded in one S₃ and four S₄ patients. The dP/dt of the rapid filling wave (RFW) and “a” wave of both apexcardiogram and left ventricle were measured. Similar data were obtained in 10 control patients without gallop sounds.

The intensity of gallop sounds was uniformly greater over the chest wall than inside the left ventricle or left atrium. In addition, the dP/dt of RFW and “a” wave tended to be higher in the apexcardiogram than the left ventricle of control patients. Also, the dP/dt of the LV RFW in S₃ patients and “a” wave in S₄ patients tended to be higher than those in control subjects, but there was overlap. The dP/dt of ACG filling waves in patients with gallop sounds was significantly greater (p < 0.01) than the respective filling wave of the left ventricle. The ACG dP/dt of the RFW in all S₃ patients and “a” wave in all S₄ patients was increased above the maximal values of the respective ACG filling waves in the control subjects.

The data suggest that the higher intensity of gallop sounds and the higher dP/dt of the filling waves over the chest cannot be caused by passive transmission of sound or pressure changes in the left ventricle. Therefore, we postulate that the greater vibratory energy of gallop sounds recorded over the precordium is caused by the impact of the heart on the chest wall. The strength of the impact is a function of several interacting mechanisms, including the momentum transfer and coupling between the heart and the chest wall.

SINCE POTAIN’S original description of gallop sounds in 1885, several mechanisms have been proposed to explain their genesis. From studies using phonocardiograms in association with the apexcardiograms with short-time constants, Benchimol et al. concluded that the third heart sound (S₃) occurs at the peak of the rapid ventricular filling wave (RFW), and that the fourth heart sound (S₄) appears at the peak of the “a” wave of the ACG.

On the basis of these findings and hemodynamic observations made by means of fluid-filled catheters, several investigators proposed that the S₃ is caused by the sudden deceleration of the RFW and that the S₄ was produced by an exaggerated “a” wave generated by the left atrium in an attempt to fill a noncompliant left ventricle. However, Arevalo et al. found in animals that the S₃ occurs when left ventricular (LV) pressure ceases to fall during relaxation. At this instant, the dP/dt of LV pressure is almost 0 mm Hg/sec, so their studies implied that the S₃ occurs at the end of active ventricular relaxation when the fall in ventricular pressure due to relaxation is equal to the rise in pressure due to filling. Prewitt et al. questioned this experimental finding and demonstrated that there is no feature of the LV pressure curve that is consistently related to the RFW of the ACG. They concluded that LV pressure cannot be solely responsible for the genesis of the S₃. Their studies also showed that the RFW of the ACG corresponds neither to the peak of rapid filling nor to the peak rate of filling. They also concluded that events other than rapid ventricular filling, or ventricular pressure rise, should be considered as the genesis of this sound. In 1940, Boyer et al. proposed that a shock of the ventricular apex against the chest wall caused the S₃, but this explanation was subsequently retracted. However, kymographic studies by Kuo et al. suggested that these workers that an impact or sudden stretch of the ventricular wall was the chief cause of production of diastolic gallop sounds.

The genesis of gallop sounds is a controversial issue, so we attempted to study them by applying several new techniques, including quantitative intracardiac and external phonocardiography in conjunction with high-fidelity, infinite-time-constant calibrated pressure apexcardiography.

Materials and Methods

External pressure, dP/dt, and sound were recorded with a high-fidelity infinite-time-constant, calibrated pressure apexcardiograph (fig. 1) developed in our laboratory. Our ACG transducer is a piezo-resistive strain gauge mounted on a thin, stainless-steel diaphragm approximately 2 mm in diameter.
dynamic range of the transducer is 0–1500 mm Hg, with a flat frequency response of 0–30 kHz ± 1% non-linearity and hysteresis.

A four-leg active bridge is supplied with a highly regulated DC voltage of ±3 V. The signal voltage is amplified by an instrumentation amplifier so that the output of 1 V is equal to a pressure of 100 mm Hg. The system is calibrated with air pressure using a sphygmomanometer.

The voltage generated by the total pressure (which is the sum of static pressure and dynamic pressure) is fed into the summing junction of an operational amplifier. A negative voltage is then applied to offset the output so that the 0 point of the ACG is close to the baseline. The loading pressure is determined from the applied negative voltage because the latter is calibrated so that 1 V corresponds to 100 mm Hg. The negative voltage opposing the loading pressure is read on a digital meter in steps of 1 mm Hg. The signal is then passed through a low-pass Butterworth filter with 40 db/decade roll-off above 25 Hz, and electronically differentiated to measure dP/dt of the ACG. The dP/dt was calibrated with a calibration switch so that a 1-V deflection was equal to 1000 mm Hg/sec. The undifferentiated signal was used to record the apexcardiographic pressure. The phono-processor amplifies (gain) the unfiltered pressure signal in multiples of 10, 20, 50, 100, 200, 500 and 1000. The processor also contains high-pass (Hz/roll-off db/decade: 30/60, 100/20; 250/20; 350/40; 450/60) and low-pass (Hz/roll-off db/decade: 50/40; 100/40; 250/40; 500/40; 1000/40; 2500/40) filters. All sounds were recorded using a high-pass filter with 60 db/decade roll-off below 30 Hz, and a low-pass filter with 40 db/decade roll-off above 500 Hz. A sound-calibration switch produced a voltage signal of 1 V, which was equal to 100 mm Hg divided by the gain factor; this permitted sound intensity to be quantified in mm Hg.

With the patient in the semi-left lateral recumbent position, the ACG transducer was held to the chest wall at the point of maximal impulse by means of a three-legged holder fastened to the patient with a three-way elastic strap. The loading pressure could be controlled by adjusting the advancement of the transducer. The transducer was then locked in position with a screw.

External pressure, dP/dt, and sound from the apex were recorded with loading pressures of 200–400 mm Hg. Loading pressure was increased beyond 200 mm Hg until the maximal amplitude of the systolic wave was obtained. Respiration was held when maximal amplitude was achieved. LV pressure, dP/dt, and internal sound were recorded with a Millar catheter. Transducers mounted at the tip of the Millar catheters have a flat frequency response from 0–20 kHz ± 0.5% nonlinearity and hysteresis. Micromanometric pressure was corrected for gravitational effects by superimposing its pressure over the fluid-filled pressure during slow rates of pressure changes in diastole. Millar pressure and dP/dt were attenuated above 25 Hz with 40 db/decade roll-off, similar to AGC pressure and dP/dt. Intracardiac sound was recorded using a phono-processor identical to that used for the ACG. Similar to external sound, the internal sound was quantified in mm Hg. External and internal pressure,
dP/dt and sound were recorded simultaneously with the same filtering and amplification so that both recordings were equivalent.

External (ACG) transducers and Millar catheters were equisensitive, with identical frequency responses when calibrated and tested in a liquid-filled chamber.

LV pressure, dP/dt, and sound were recorded simultaneously with external pressure, dP/dt, and sound from the apex in 12 patients with gallop sounds. Six had a ventricular diastolic gallop (S₃), eight an atrial diastolic gallop (S₂), and one a summation gallop. Table 1 shows their age, sex, rhythm and diagnosis. Left atrial pressure, dP/dt, and sound were also recorded in four patients with an atrial diastolic and one patient with a ventricular diastolic gallop.

Maximal peak-to-peak intensity of both internal and external gallop sounds, maximal dP/dt of the RFW of the ACG, and the maximal dP/dt of the “a” wave of the ACG were measured as the average value for five consecutive cycles in each patient. The dP/dt of the rapid filling wave and the dP/dt of the “a” wave of the left ventricle were measured at points corresponding to the peak dP/dt of the ACG RFW and peak dP/dt of the ACG “a” wave, respectively. The peak dP/dt of the ACG and/or LV RFW was taken as 0 mm Hg/sec when a measurable positive dP/dt was not present within 80 msec of the ACG 0 point (point at which ACG dP/dt first reaches 0 mm Hg/sec after completion of ventricular systole).

The dP/dt of the RFW and “a” wave of the left ventricle and ACG were measured in 10 patients with neither audible nor recordable gallop sounds. Table 2 shows their age, sex, rhythm and diagnosis.

The effect of variable ACG loading pressure on the recorded intensity of external gallop sounds was evaluated in two patients with heart disease and two normal subjects. The loading pressure was varied from 200-700 mm Hg.

Differences between “a” wave of the left ventricle and ACG in the control and S₃ groups and between the left ventricle and ACG RFW dP/dt in S₃ patients were compared using paired data; the significance of differences was evaluated using the t test. Differences in the LV “a” wave dP/dt between control and S₃ patients were compared using group means.

Results

The maximal loading pressure applied to the chest wall for the measurement of external (ACG) dP/dt and sound in this study was 400 mm Hg. When the effect of varying loading pressure on the intensity of gallop sounds was evaluated in four subjects, increments of loading pressure beyond 400 mm Hg resulted in variable increases in sound intensity (fig. 2).

Third Heart Sound

Six patients had an externally recordable S₃, ranging in intensity from 0.3-3.75 mm Hg (table 1). The intensity was at least 1.3 mm Hg in five of the six, and 0.3 mm Hg in the remaining patient; this gallop was recordable but not audible. Figure 3 illustrates apexcardiographic and LV sound and pressure in a patient with mitral regurgitation and atrial fibrillation. The external phonocardiogram shows a mitral regurgitant murmur and an S₃ 2.4 mm Hg in intensity. The LV phonocardiogram shows a mitral regurgitant murmur of lesser intensity and no appreciable S₃. The developed pressure of the systolic and RFWs in the ACG are greater than the corresponding LV pressures. The RFW of the left ventricle also peaks much later than the RVW of the ACG. Figure 4 illustrates the left atrial phonocardiogram in the same patient and shows the mitral regurgitant murmur to be of greater intensity at the source than outside. An S₃

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Abbreviations: ACG = apexcardiogram; ADG = atrial diastolic gallop; AF = atrial fibrillation; AI = aortic insufficiency; AS = aortic stenosis; ASHD = atherosclerotic heart disease; LV = left ventricle; MI = myocardial infarction; MR = mitral regurgitation; RFW = rapid filling wave; SR = sinus rhythm; VDG = ventricular diastolic gallop.
Gallop Sounds and Quantitative Phonocardiography/Reddy et al.

Figure 2. Effect of increasing apexcardiographic application pressure on the intensity of externally recorded gallop sounds in four subjects. As application pressure is increased beyond 400 mm Hg, the sound uniformly increases in intensity, although to a variable degree in different patients.

could not be recorded from the left atrium. Figure 5 shows the mitral regurgitant murmur to be least intense in the central aorta.

An appreciable S₃ was not recordable in the left ventricle at the time of occurrence of an external S₃ in three of six patients. In the three other patients, an S₃ of variable intensity was recordable in the left ventricle (fig. 6). In two of these three patients, the S₃ occurred in the left ventricle synchronous with the external sound; the internal intensity was less than external sound (0.40 vs 1.3 and 0.3 vs 3.75 mm Hg, respectively). In the third patient, with an external S₃ of 1.8 mm Hg, an internal sound of 0.20 mm Hg was recorded in the left ventricle (fig. 7); however, the internal sound followed the external sound by about 20–30 msec. Some patients had a short flow murmur instead of a distinct sound in the left ventricle at the time of occurrence of an external S₃ (fig. 8).

The dP/dt of Rapid Filling Wave (fig. 9)

In 10 control patients without either atrial, ventricular or summation gallops, the dP/dt of the LV RFW was 0 mm Hg/sec in all but one, in whom it was 71 mm Hg/sec. The RFW dP/dt of the ACG was greater than 0 in four control patients, and in these four, it was at least 100 mm Hg/sec (table 2). The dP/dt of the ACG in S₃ patients was uniformly greater than those of control patients. The minimal value of 333 mm Hg/sec in S₃ patients was greater than the maximal value of 218 mm Hg/sec in the control patients. All patients with an S₃, except one, had an external dP/dt of at least 700 mm Hg/sec; one patient with a dP/dt of 333 mm Hg/sec had an externally recordable but not an audible gallop of 0.3 mm Hg. Four of six S₃ patients had an LV dP/dt greater than the max-

Figure 3. Simultaneous recordings of external (apexcardiographic [ACG]) and left ventricular (LV) pressure and sound in a patient with mitral regurgitation and atrial fibrillation. The S₃ (ventricular diastolic gallop [VDG]) intensity is 2.4 mm Hg in the external phonocardiogram. There is no appreciable vibration at that instant in the LV phonocardiogram. The rapid filling wave (RFW) is more prominent in the ACG than in the left ventricle and the latter peak is delayed compared with the ACG. The external phonocardiogram also shows a systolic murmur which has a greater amplitude than in the left ventricle. Systolic pressure of the ACG is also higher than that of the left ventricle.
EXT.

VDG

PHONO 1 M M H G

LA

-100 MMHG

DP/Dt

0 LV

FIGURE 4. External phonocardiogram and internal phonocardiogram, dP/dt, and pressure during the pullback of a retrograde Millar catheter from left atrium (LA) to left ventricle (LV) in the same patient as figure 3. The intensity of the mitral regurgitant murmur is greater in the LA than externally or in the LV. A ventricular diastolic gallop (VDG) cannot be appreciated in the LA. Calibration is not shown for dP/dt.

Fourth Heart Sound

Eight patients had an externally recordable S₄ (table 1). The intensity ranged from 0.25–3.5 mm Hg and was at least 0.60 mm Hg in all but one. The patient with an S₄ gallop of 0.25 mm Hg had an externally recordable but not an audible gallop (fig. 7).

The S₄ was indistinct in the left ventricle of all patients (fig. 11) except one. An internal S₄ of 0.2 mm Hg was recorded in one patient with an external S₄ of 0.25 mm Hg (fig. 8). Some patients had a short murmur instead of a distinct sound in the left ventricle with the occurrence of the external S₄ (fig. 8).

The dP/dt of the “a” Wave (fig. 12)

The dP/dt of the ACG “a” wave (115 ± 57 mm Hg/sec, mean ± sd; range 0–75 mm Hg/sec) was
FIGURE 7. External (apexcardiographic [ACG]) and left ventricular (LV) pressure, dP/dt and sound in a 43-year-old patient with cardiomyopathy. The intensity of an external ventricular diastolic gallop (VDG) is 1.8 mm Hg, while the dP/dt of the rapid filling wave is 813 mm Hg/sec. The dP/dt of the left ventricle at that instant is 100 mm Hg/sec with no appreciable vibration in the LV phono. However, the rapid filling wave of the left ventricle LV peaks later (20–30 msec) than in the ACG with a peak dP/dt of 156 mm Hg/sec; this peak is associated with a vibration of 0.20 mm Hg intensity in the internal phonocardiogram. This patient also had a palpable “a” wave but not an audible S₄. The external phonocardiogram shows an atrial diastolic gallop (ADG) of 0.25 mm Hg intensity, with an “a” wave dP/dt of 563 mm Hg/sec. The internal phonocardiogram shows an ADG of 0.2 mm Hg intensity, with an “a” wave dP/dt of 250 mm Hg/sec.

FIGURE 8. Simultaneous external (apexcardiographic [ACG]) and left ventricular (LV) pressure and sound in a patient with an audible S₄. The external phonocardiogram shows an ADG of 0.80 mm Hg and a VDG of 0.3 mm Hg intensity. The LV phonocardiogram shows a high-frequency flow murmur at the time of the external S₃ and S₄. ADG = atrial diastolic gallop; VDG = ventricular diastolic gallop.
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dP/dt.

Figure 10. External (apexcardiographic [ACG]) and left ventricular (LV) dP/dt and sound in the same patient as figure 3. Note the dP/dt of left ventricle is 0 mm Hg at the instant of peak dP/dt of the ACG rapid filling wave (RFW), which varies from 400–700 mm Hg/sec, depending on cycle length. Directional changes in intensity of external ventricular diastolic gallop (VDG) with varying cycle length are similar to changes in dP/dt.

Figure 9. Comparison of external (apexcardiographic [ACG]) and left ventricular (LV) dP/dt of the rapid filling wave (RFW) in control patients (closed circles) and patients with an S₃ (3). The ACG dP/dt of S₃ patients is higher than the corresponding LV value and uniformly higher than the ACG dP/dt of control patients. Note also that four control patients have higher values in the apexcardiogram than in the left ventricle. VDG = ventricular diastolic gallop.

Left Atrial Pressure dP/dt and Sound

In four patients with an external atrial diastolic gallop in whom left atrial pressure, dP/dt and sound were recorded, no S₃ could be demonstrated in the left atrium (fig. 14). During spontaneous respiration, the intensity of the external S₃ usually decreased with inspiration. Although absolute pressure of the LV "a" wave tended to fall with inspiration, there was no significant change either in the developed pressure or the rate of pressure change associated with atrial contraction (fig. 15).
GALLOP SOUNDS AND QUANTITATIVE PHONOCARDIOGRAPHY/Reddy et al.

Figure 11. External (apexcardiographic [ACG]) and left ventricular (LV) dP/dt and sound in a patient with aortic stenosis. An external \( S_4 \) (atrial diastolic gallop) of 1.9 mm Hg corresponds to an ACG "a" wave peak dP/dt of 900 mm Hg/sec. When the latter occurs, the LV dP/dt is approximately 100 mm Hg/sec and is associated with no discrete vibration in the LV phonocardiogram.

Summation Gallop

One patient had a summation gallop of 1.3 mm Hg externally and 0.75 mm Hg in the left ventricle (fig. 16). The dP/dt of the summation wave was 800 mm Hg/sec, both internally and externally.

Discussion

Gallop sounds have been investigated by qualitative rather than quantitative phonocardiographic techniques. We developed special equipment that uses piezo-resistive pressure transducers and direct-coupled electronic circuitry with infinite time constants and calibrated amplification. Two identical systems with equal settings were used to record simultaneously internal and external pressure events, including sound. This permitted quantitative comparison of gallop sounds recorded internally in the left ventricle and externally on the chest wall.

Data obtained by these quantitative techniques indicate that the vibratory energy of the gallop sounds recorded over the precordium is greater than that recorded for the same events in the left ventricle or left atrium. These findings cannot be explained by a relative insensitivity of the intracardiac phonocardiogram, because murmurs recorded by the same technique were found to be loudest at their intracardiac source. For example, a mitral regurgitant murmur was louder in the left atrium than in either the left ventricle or central aorta or on the chest wall. Internally or externally recordable gallop sounds of

Figure 12. Comparison of external (apexcardiographic [ACG]) and left ventricular (LV) dP/dt of the "a" wave in control patients (closed circles) and patients with an \( S_4 \) (atrial diastolic gallop [ADG]) (4). There is overlap between LV dP/dt in control and \( S_4 \) patients. However, the ACG dP/dt of \( S_4 \) patients is uniformly higher than that in control subjects.
measurable amplitude were consistently associated with an increase in the dP/dt of their corresponding filling waves. The patient with the highest intensity of an internally recordable gallop sound also had the highest dP/dt of the corresponding LV filling wave. When the internal dP/dt was 0 at the time of the gallop sound externally, a measurable gallop sound was not internally recordable.

In some patients, the internal gallop sound in the left ventricle occurred 20–30 msec after the external sound; therefore, the origin of the external sound in such patients cannot be attributed to the later vibration in the left ventricle. Differences in the timing of external and internal gallop sounds were associated with similar differences in the timing of peak dP/dt of the respective ACG and LV RFWs. Therefore, an understanding of the genesis of the RFW of the ACG appears to be a key to learning the genesis of an external S₃.

We have shown that the external pressure developed during the RFW of the ACG can exceed that of the left ventricle. The present study demonstrated that the external rate of RFW pressure rise can exceed 100 mm Hg/sec when the LV dP/dt is 0 in patients without gallop sounds. In such instances, the higher pressure and higher rate of pressure rise of the external RFW cannot be explained by mere passive transmission of LV pressure. Furthermore, the dP/dt of the RFW of the ACG is markedly increased in patients with an S₃, while the LV dP/dt can be 0 at that instant. It is therefore unlikely that the LV pressure changes cause the exaggerated ACG RFW and associated S₃. Our observations are consistent with those of Prewitt et al., who found that no feature of LV pressure consistently corresponded to the RFW of the ACG. These investigators also showed that the RFW of the ACG corresponds neither to the peak of rapid filling nor to the peak rate of filling of the left ventricle.

**Figure 13.** External (apexcardiographic [ACG]) and left ventricular (LV) pressure and sound in the same patient as figure 11. The ACG "a" wave is more prominent than the LV "a" wave and its upstroke occurs during the downstroke of the LV "a" wave.

**Figure 14.** External (apexcardiographic [ACG]) and left atrial (LA) pressure and sound in the same patient as figure 11. Note the absence of an atrial diastolic gallop in the LA phonocardiogram. The ACG "a" wave occurs during the downstroke of the LA "a" wave.
ventricle. There is also no evidence that either the absolute ventricular inflow or the rate of ventricular filling is increased in patients with a ventricular diastolic gallop. In some patients, it may even be decreased because of considerably diminished stroke volume. Therefore, the RFW of the ACG does not appear to be caused by either passive transmission of the ventricular pressure or by instantaneous ventricular volume changes.

Similar conceptual difficulties had also been encountered in explaining the systolic wave of the ACG, particularly when it was greater than the LV pressure. The ACG systolic wave is not solely caused by ventricular volume change or passive transmission of ventricular pressure, but is the result of complex forces acting on the chest wall. These include ballistic, torsional and other forces related to cardiac motion whose energy is derived from systolic contraction. This cardiac motion may not only cause an impact of the heart on the chest wall, producing an exaggerated systolic wave of the ACG, but may also be a source of elastically stored potential energy that is released later during early diastole. Therefore, we hypothesize that in addition to the momentum transfer from the inrushing blood, the reversal of systolic cardiac motion in early diastole may also contribute to an impact of the heart on the chest wall, which produces the RFW of the ACG. The degree of impact produced by a heart on the chest wall in early diastole depends on the momentum with which it hits the chest wall. While momentum depends upon its mass and velocity, the impact produced by a given momentum also depends on the degree of contact the heart can make with the chest wall. In some young, healthy subjects, the early diastolic motion may be of sufficient magnitude to produce an S₃ if it can also make proper contact with
the chest wall. With advancing age, the impact of the heart on the chest wall may decrease because of changes in the chest wall configuration or decreased velocity of the heart motion. The $S_2$ may appear in such age groups if the systolic motion of the heart, and consequently the early diastolic motion, can be exaggerated as in hyperkinetic states. Enlarged hearts may often be associated with $S_2$ because of increased momentum resulting from increased mass and better contact with the chest wall despite decreased motion. Enlarged hearts are usually associated with elevated left atrial "v" wave and mean pressure. Injection of blood into the left ventricle from a heightened pressure may impart momentum to the blood and the heart with or without causing a rapid pressure rise in the left ventricle. If there is a rapid pressure rise in the left ventricle, as noted in some cases, there will be an associated sound inside the left ventricle. If there is no rapid pressure change inside the ventricle, there will be no associated sound. Since the internal sound is dependent upon the rate of pressure rise with filling inside the left ventricle, and the external sound is dependent upon the pressure rise produced by the impact of the heart on the chest wall, they may be temporally dissociated or coincide with each other. In the former case, the internal sound may precede or follow the external. Both are early diastolic events that usually occur in close proximity to each other.

Denef et al., using a calibrated displacement ACG, found a significant increase in the height of the "a" wave and peak da/dt in patients with a fourth heart sound. Just as the RFW of the ACG is related to the genesis of a ventricular diastolic gallop, the "a" wave of the ACG is related to the genesis of an atrial diastolic gallop. Atrial contraction was found to result in a greater rate of pressure rise in the ACG than in the left ventricle, even in control patients without gallop sounds. In patients with atrial diastolic gallops, the dp/dt of the ACG "a" wave was distinctly higher than in controls, but no such clear separation was found regarding the dp/dt of the LV "a" wave. The difference between the dp/dt of the LV "a" wave and the ACG "a" wave was thus further exaggerated in the atrial diastolic gallop group. It is therefore unlikely that the pressure rise in the left ventricle is solely responsible for the genesis of the "a" wave of the ACG, particularly in patients with atrial diastolic gallops.

We therefore postulate that atrial contraction not only causes a rise in ventricular pressure but also produces motion of the heart. The motion, in turn, causes an impact on the chest wall. Such motion may involve the total heart or merely stretching and elongation of the ventricle. The impact, measured externally as a pressure variation on the chest wall, may be either greater or less than the pressure change in the left ventricle, depending on the degree and direction of the momentum of the heart and its coupling with the chest wall.

Some of the impact may be dampened, depending on the thickness and character of the chest wall, leading to a decrease rather than an increase in recorded amplitude of gallop sounds. The application pressure of the ACG transducer compresses the wall tissue and reduces losses in transmission of the force. The intensity of the external sound increased to a variable extent with increasing loading pressures beyond 400 mm Hg. Although we only used a maximum loading pressure of 400 mm Hg, the external sound was uniformly louder than that recorded inside the left ventricle or left atrium. If the losses were further minimized by applying a higher loading pressure, the intensity of the external sound could only have increased.

The impact of the heart on the chest wall due to atrial contraction may be greater in the hypertrophied ventricle when the left atrium contracts more vigorously in an attempt to fill a less compliant left ventricle. Better coupling between the hypertrophied heart and chest wall may also contribute to the genesis of the exaggerated "a" wave of the ACG and associated atrial dialetolic gallop. The ultimate source of energy for both the LV and ACG "a" wave lies in atrial contraction, so the heights of these two waves may be related; however, there may be a finite delay between the occurrence of atrial contraction, observed as a pressure change in the left ventricle or left atrium, and the "a" wave of the ACG, which is due to impact of the heart. Therefore, it is not surprising that, in some patients, the upstroke of the ACG "a" wave occurred during the downstroke of the "a" wave of LV and left atrial pressure. The magnitude of the impact of the heart on the chest is dependent upon the coupling between these two structures, so it may be maximal at only one localized spot on the chest wall and may require the patient to lie in a particular position with the breath held in a particular phase of respiration. In this regard, we observed that the "a" wave of the ACG and the associated atrial diastolic gallop may greatly vary with the position of the patient and respiratory phase without a change in developed pressure or rate of pressure change inside the left atrium. This is also true regarding the ventricular gallop. The external recordings of atrial flutter sounds during ventricular systole also support the "impact theory" of gallop sound genesis.

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References


6. Arevalo F, Meyer EC, MacCanon DM, Luisada AA: Hemo-
Choice of Electrocardiographic Leads for Recording the Earliest QRS Onset in Noninvasive Measurements

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SUMMARY A significant error may be introduced in intervals measured from the onset of the QRS if an electrocardiographic lead that does not record the earliest deflection is used. To ascertain to what extent the commonly used leads can be relied on to show the earliest QRS onset, 100 normal subjects and 219 patients with heart disease were studied by means of simultaneous recording of three leads: a right precordial lead chosen to show an R S configuration, lead II, and another limb lead chosen to show a q R configuration.

Lead II most frequently showed a delayed QRS onset — in 34% of normal subjects and 36% of the patients. In the other limb lead the initial QRS deflection was delayed in 24% of the normal subjects and 23% of the patients. The QRS onset in the right precordial lead was never delayed in the normal subjects; however, it was delayed in this lead in 6% of the patients. The delays in each of the leads ranged from 5–20 msec.

We conclude that while a right precordial lead is by far the most reliable single lead that can be used for interval measurements, simultaneous recording of a right precordial lead and a limb lead assures the recording of the earliest QRS onset in all cases.

AN ELECTROCARDIOGRAPHIC (ECG) recording is routinely performed simultaneously with recordings made of various noninvasive testing procedures. One of the important uses of this ECG recording is the precise measurement of intervals from the onset of ventricular depolarization (the QRS complex). Such measurements are essential in the assessment of ventricular performance by means of systolic time intervals,1 in the timing of heart sounds on the phonocardiogram and in the timing of the motion of various structures on the echocardiogram such as opening or closure points of the valves2 and systolic wall motion.3,4 The reliability of all these measurements depends on the accuracy and precision of the determination of the earliest onset of the QRS complex.

The onset of the initial QRS deflection is frequently not simultaneous in all ECG leads. Nevertheless, the error in measurement that may be introduced by the use of a lead that does not demonstrate the earliest onset of the QRS is often disregarded. Many laboratories still routinely use lead II. However, Danzig et al.5 showed by comparing simultaneously recorded ECG leads in a large series of subjects that the mean onset of the QRS is significantly earlier in right precordial leads (V1 and V2) than in lead II. They used these results to justify use of a right precordial lead in measurements of the onset of the QRS. Their study, however, was performed on normal subjects only, and it does not necessarily follow that in pathologic states a right precordial lead will always show the earliest onset of the QRS. An orthogonal lead system has been suggested for time interval measurements.6,7 Such leads have advantages on theoretical grounds, but their superiority over the

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P S Reddy, F Meno, E I Curtiss and J D O'Toole

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