Observations on the Mechanism of Atrial Gallop Rhythm

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An atrial gallop sound is a frequent finding in hypertensive cardiovascular disease, myocardial infarction, and in the presence of a prolonged atrioventricular conduction time. In view of evidence relating this sound to a ventricular pressure wave resulting from atrial contraction, modification of this hemodynamic event was studied by means of tourniquet pooling of blood in the extremities. In the 18 patients studied, this maneuver caused striking changes in the intensity and timing of the atrial gallop. Analysis of these changes helps explain the frequent appearance of this triple rhythm in severe hypertensive cardiovascular disease.

Gallop rhythm is said to be present when an audible diastolic sound is added to the 2 normal heart tones. The atrial gallop is a low-pitched sound occurring after atrial systole, but before the subsequent ventricular contraction. This sound is frequently heard in patients with hypertensive cardiovascular disease, myocardial infarction, and in the presence of atrioventricular conduction disturbances (heart block, whether complete or partial). It was thought that some information about the genesis of this sound might be obtained by altering intracardiac pressure relationships. This was accomplished by pooling of blood in the extremities through the use of occlusive venous cuffs.

Materials and Methods

Eighteen hospitalized patients were studied. Seventeen were patients with hypertensive cardiovascular disease, either “essential” or renal in origin, but all either severe or malignant in nature. The systolic pressure was over 200 mm. Hg and diastolic pressure over 110 mm. Hg in all. The patients were not in obvious cardiac failure at the time of study, although many had previously been decompensated. All had roentgenographic or electrocardiographic evidence of left ventricular hypertrophy. One patient thought to have an atrial gallop sound from the right side of the heart had pulmonary hypertension due to sarcoidosis, but a normal systemic arterial pressure. Only patients with gallop sounds that were readily audible and with phonocardiograms free from extraneous vibrations were utilized. Because the patients were studied while they were lying quietly on a flat comfortable surface, those who were dyspneic, restless, or orthopneic could not be satisfactorily investigated.

All sound tracings were taken on the logarithmic scale of the Sanborn “Twinbeam” photographic recording instrument at a paper speed of 75 mm. per second. The standard 50-mm. open bell chest piece was placed on the chest wherever the sounds to be investigated were best demonstrated. This was usually the apex or along a line drawn from the apex to the sternum at the level of the fifth interspace.

In many instances additional tracings were taken to record the linear or apex cardiogram with a piezo-electric crystal microphone. These vibrations were recorded simultaneously with and from the same chest piece as the logarithmic phonocardiogram.

In order to study the effect of peripheral blood pooling, pneumatic cuffs 6 inches wide were placed around all extremities. Care was taken to apply the cuffs as high as possible, yet not to cause discomfort. The cuffs were inflated simultaneously and rapidly to the level of 100 mm. Hg. The inflation was sustained for 5 minutes, and the cuffs then were released suddenly. Following control sound tracings recordings were made at minute intervals during the period of sustained inflation. The period during cuff release and 1 minute thereafter was also recorded.

Results

Control phonocardiograms demonstrated the atrial gallop deflections usually 2 to 3
cycles in duration, occurring late in diastole, 0.07 second or more before the onset of the first heart sound. Such deflections occurred between the inscription of the P wave and QRS of the electrocardiogram (fig. 1). In addition, apex cardiograms demonstrated prominent, sharply peaked presystolic waves, the peaks of which were simultaneous with the gallop sound deflections (fig. 2).

Upon inflation of the venous occlusive cuffs, the gallop sound moved progressively toward the first heart sound (fig. 1). The average migration in the 18 cases studied was 0.034 second, with a range of 0.016 to 0.060 second. As this movement progressed, the gallop sound completely disappeared in 6 cases, diminished to less than 50 per cent of its control magnitude in 6 cases, and remained essentially unchanged in amplitude in 6 cases. With cuff inflation, there was concurrent migration of the atrial gallop wave of the apex cardiogram toward the first sound, its peak remaining synchronous with the gallop sound (fig. 2). The amplitude of the wave diminished and its summit was no longer sharp, but became rounded and less conspicuous. With release of cuffs, sound and apex tracings returned to their control configuration and temporal relationships (figs. 1 and 2).

In many of the patients the first sound was not so loud as the second heart sound at the apex. In 6 instances there was a definite increase in intensity of the first heart sound as the atrial gallop migrated into its initial deflections (fig. 3). In many, the deflections of the atrial gallop that had occurred before the onset of the QRS in the control tracings were now present as small slow vibrations, occurring just before the early rapid vibrations of the first sound during the inscription of the R wave (fig. 3). Upon sudden release of cuffs these low-frequency sounds migrated back into their former position in presystole (fig. 3).
OBSERVATIONS ON ATRIAL GALLOP RHYTHM

DISCUSSION

The audible atrial gallop sound does not occur synchronously with mechanical atrial systole.1 This sound occurs simultaneously with a pressure wave appearing in the ventricle as a result of atrial contraction. Such a presystolic ventricular pressure wave can be demonstrated by electrolymograms, which show an outward movement of the ventricular wall at this time,2 and apex cardiograms, which show a precordial thrust, the peak of which is simultaneous with the gallop sound. Ballistocardiograms under such circumstances reveal ventricular forces directed headward and to the right, the onset of which are synchronous with the gallop sound.3 4 It is true that at the time of mechanical atrial systole, vibrations occur that may be graphically recorded when the pickup is in the esophagus5 or in the atrial cavity.6 Recording instruments sensitive in the low frequencies (stethoscopic scale of Rappaport and Sprague;7 medium and low frequency of Leatham8) may register these sounds over the precordium. These vibrations do not represent the audible atrial gallop sound heard by the clinician. In the past failure to differentiate these subaudible vibrations from sounds audible to the human ear has resulted in confusion regarding the timing of the atrial gallop.

It is well recognized that atrial gallop sounds may be heard in patients with heart block when atroventricular conduction time is prolonged. Under these circumstances the vibrations related to atrial contraction precede the first heart sound by an interval long enough to render the atrial component audible as a separate sound (fig. 4c). The audibility is favored by the fact that the first heart sound itself is relatively faint when the P-R interval is prolonged.

The atrial sound may be frequently heard in additional patients with cardiovascular disease, even in the presence of a normal P-R interval1 (fig. 4d). In particular, patients with hypertensive cardiovascular disease are frequently found to have a readily audible atrial gallop sound. It is in this type of patient that tourniquet application has been observed to produce the changes reported here.

Numerous investigators have studied the
hemodynamic effects of pooling blood by venous occlusive cuffs. In normal subjects the cardiac output decreases slightly. A fall in central venous pressure occurs in association with a corresponding decrease in the pressures in the right atrium, right ventricle, pulmonary artery, and the left atrium. Thus, atrial systole results in somewhat less blood being ejected into the ventricle, which in turn is under less tension. Under these conditions the phenomena producing the atrial sound are altered so that it occurs later and often becomes faint or even inaudible. The peak on the apex cardiogram associated with the sound is also delayed and is usually decreased in magnitude. At this time the early vibrations of the first heart sound are apparently related to atrial activity, a point that has been debated in the literature.4

An additional factor that may enhance the audibility of the atrial sound in patients with hypertensive cardiovascular disease is the delay in the onset of the first heart sound, as demonstrated by slight, but significant, increase in the Q-1 interval* in the patients studied. The Q-1 time in the group was .066 ± .014 second, as opposed to a control group of 18 normal subjects in which the Q-1 time was .055 ± .01 second (p < .01). In the hypertensive group 7 patients had Q-1 times of .07 second or longer. This delay occurred in the absence of bundle-branch block, or a QRS complex longer than .10 second. The result of this increase in the Q-1 interval was to separate further the first sound from the atrial vibrations and thus aid in unmasking the atrial sound (fig. 4f).

To this point we have not discussed what structure, when displaced by the pressure wave, actually produces the sound. The 2 most widely held theories are that the sound is caused by vibration of the ventricular musculature, or that a tensing or even reclosure of the valves takes place at this time. It is generally accepted that valve leaflets can produce a sound, but whether or not an audible sound can emanate from the ventricular musculature is debated.4 Henderson and Johnson,15 as well as Dean,16 using excited hearts and models showed to their satisfaction that valves may tense or reclose after atrial systole. Little17, 18 has shown in dogs that following atrial contraction, there is increased pressure in the ventricle. As atrial relaxation occurs and pressure in the atrium drops, that in the ventricle still elevated by the effects of atrial contraction may exceed it in late diastole before subsequent ventricular systole. Such a reversal of the diastolic atrioventricular pressure gradient could result in valve reclosure. The hemodynamic effects of tourniquet application may well alter these pressure relationships and lead to loss of the atrial sound.

The available information indicates, therefore, that an audible atrial gallop may occur

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*Time from the onset of the Q wave of the electrocardiogram to the onset of the first major vibrations of the first heart sound.
under several possible circumstances. When conduction from atrium to ventricle is delayed, as in heart block, atrial contraction with its augmentation of ventricular filling produces a sound in late diastole before the onset of ventricular contraction. Here temporal splitting off of the atrial sound occurs as the result of the separation of atrial and ventricular activity by the conduction defect. In other patients the sound may occur with normal conduction times when the pressure wave resulting from atrial activity appears to be transmitted more rapidly to the ventricle. As a result the sound occurs early and precedes the first sound by a time interval adequate to render it acoustically separate. Pooling of blood in the extremities results in modification of pressure volume relationships in such a way that the transmission of the wave is progressively slowed and the resulting sound migrates back toward the first heart sound. In some patients delay in onset of the first sound (prolonged Q-1 time) may enhance the tendency toward gallop production.

Summary

The effect of tourniquet pooling of blood with resultant lowering of intracardiac pressures has been observed in a group of patients with readily audible atrial gallop rhythm. A progressive migration of the atrial gallop sound toward the first heart sound appeared, accompanied by a disappearance or striking decrease in the amplitude of the sound in two thirds of the cases. Apex cardiograms showed concurrent migration and diminution in the precordial atrial gallop wave.

Available evidence indicates that atrial gallop sounds may occur (1) when atrioventricular conduction is prolonged or (2) when altered ventricular pressure volume relationships result in an earlier atrial sound. In some instances, delay in onset of the first heart sound (prolonged Q-1 time) may enhance the tendency toward gallop production.

Summario in Interlingua

Un gruppo de patientes con facilemente audibile rhythmos de galopo atrial eseva studiate con respecto al effetto del reduction del pression intracardiac resultante de stagnation de sanguine sub le application de tour-niquets. Appareva un migration progressive del sono de galopo atrial verso le prime sono cardiae, accompaniate per le disparition o un reduction frappante in le amplitude del sono in duo tertios del casos. Cardiogrammas apical monstrava simultaneitate de migration e diminution in le unda precordial de galopo atrial.

Le datos disponibile indica que sonos de galopo atrial pote occurrer (1) quando le conduction atrio-ventricular es prolongate o (2) quando alterationes del relation inter pression e volumine in le ventriculo resulta in un formation plus prompte del sono atrial. In certe casos, retardos in le declaration del prime sono cardiae (prolongation del tempore Q-1) promove possibilmente le tendentia al production del rhythmo de galopo.

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Ten dogs received hydralazine in varying dosages orally. The pattern and rate of toxic reactions varied directly with the dosage schedule. A progressive normocytic anemia, fever, anorexia, and cachexia appeared, often with terminal convulsions. Leukopenia was not apparent. LE cells were not found. Autopsy findings were compatible with a diagnosis of hemolytic anemia (i.e., congestion of the liver and spleen with hemosiderosis of both). No renal or other changes suggested systemic lupus erythematosus (SLE). From these experiments the authors concluded that hydralazine did not give rise to the SLE syndrome, and that in human beings there are 2 types of hydralazine reactions. The most common is a drug sensitivity in a patient with essential hypertension, the other type being a patient with latent SLE whose underlying disease is exacerbated by the use of hydralazine. In this latter type, the picture is typical of the underlying disease, and LE cells are found. The disappearance of symptoms and laboratory changes after withdrawal of a noxious agent in SLE is common.

MAXWELL
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