Modification of the Atrial Sound by the Cold Pressor Test, Carotid Sinus Massage, and the Valsalva Maneuver

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The present study investigates changes produced in the atrial sound (fourth heart sound or presystolic gallop) by three procedures, the cold pressor test, carotid sinus massage, and the Valsalva maneuver, which among other effects are attended by changes in sympathetic or parasympathetic activity.

The atrial sound is defined as an audible vibration commencing more than 0.07 second after the start of the P wave, but before the onset of the QRS complex. It arises in the ventricle following atrial contraction. Rarely audible in normal hearts, it is frequently heard in systemic hypertension, myocardial disease, aortic stenosis, pulmonary hypertension, pulmonary stenosis, and atrioventricular block. Increases in ventricular end-diastolic pressure, venous return, and the force of atrial contraction appear to favor its production.

A change in these factors may be responsible for changes in amplitude of the atrial sound produced by the procedures used in this study.

Material and Methods

Cold pressor tests were performed on 41 subjects (table 1). Except for the two patients with normal hearts and one with thyrotoxicosis all had ventricular enlargement demonstrated by roentgenograms and by QRS voltage changes on electrocardiogram. All had sinus rhythm. In three cases the P-R interval was greater than 0.20 second (0.21, 0.26, 0.27 second). Five subjects were in congestive failure; mild in two and moderate in three.

Heart sounds and electrocardiograms were recorded simultaneously on magnetic tape and then converted to spectral phonocardiograms. Characteristics of the recording and spectral analyzing systems have been described. The microphone was kept on one area, usually at or medial to the cardiac apex. Amplification was kept unchanged throughout each patient’s recording and analysis.

A modified cold pressor test was performed. The subjects were supine throughout. The right hand was immersed in ice water for 5 minutes. During a control period immediately preceding immersion, and at approximately 1/2, 1, 1 1/2, 2, 3, 4, and 5 minutes after the onset of immersion, blood pressure was measured by auscultation in the left arm and heart sounds were recorded.

On a small group carotid sinus massage was subsequently performed, after the effects of the cold pressor test had subsided. An additional electrocardiogram (lead II) was recorded with a direct-writing electrocardiograph during massage. In several subjects the effects of the Valsalva maneuver were observed. Heart sounds and electrocardiograms were recorded continuously before, during, and after each procedure.

In spectral phonocardiograms frequency is displayed on the vertical axis and time on the horizontal axis, the intensity of sound being shown by the degree of blackness. An increase in the atrial sound was determined from inspection of the phonocardiographic record.

Results

Cold Pressor Test

Six hypertensive and five normotensive patients had no atrial sound; the sound appeared as a result of the test in one, a hypertensive (fig. 1). Twenty-eight patients had an atrial sound initially; in 15 (54 per cent) the sound increased in intensity during the test (figs. 2 and 3). In most such cases the increase was obvious clinically. Two additional patients had a presystolic murmur in association with mitral stenosis; in both the murmur increased strikingly during the test.

In positive responses the intensity of the atrial sound began to increase 20 to 45 seconds.
after the onset of immersion and reached its peak in one minute. In most, but not all, instances it began to decline after two to three minutes but remained above control levels throughout the test. It diminished rapidly in the first minute after the end of immersion and five to 10 minutes later it was back to control levels. The first and second heart sounds often became louder during the test. In cases in which the atrial sound responded its increase was often greater than that of the other sounds.

The interval between the onset of the P wave of the electrocardiogram and atrial gallop (P-G interval) averaged 0.16 second (range 0.12 to 0.20 second) in the control period. During the procedure it did not change in 22 subjects and decreased an average of 0.03 second (range 0.02 to 0.04 second) in four hypertensive and two normotensive subjects; in all six the atrial sound also became louder.

The heart rate sometimes increased shortly after immersion but usually returned to the control level or to slightly above it within one to two minutes. The mean values for changes in heart rate and blood pressure in the 23 hypertensive subjects are analyzed in table 2. The normotensive group was not analyzed in this way, owing to its small size and heterogeneous composition. The average increase in rate was similar in the hypertensive groups with or without a response of the atrial sound. The average rise in diastolic pressure was 20 mm Hg in both. The hypertensive group without atrial sounds had an average diastolic rise of 16 mm Hg.

The hypertensive groups with and without a response of the atrial sound did not appear clinically different, except for a slightly higher
Figure 1 (See legend on opposite page)
control rate in the former. Blood pressure and the atrial sound increased during the test in three patients on guanethidine. Sympathetic blockade was probably only partial as the blood pressure fell but little on standing. Five of nine patients taking reserpine responded with accentuated atrial sounds during the test.

**Carotid Sinus Massage**

In 10 subjects carotid sinus massage slowed the heart rate without producing sinus arrest or changing the P-R interval. In five the atrial sound decreased in intensity (fig. 4) and occasionally disappeared. In four there was no change. One patient consistently developed marked increase in the atrial sound during slowing induced by carotid sinus massage (fig. 5). His test was repeated 20 times with the same result in each. We can find no other report of such an occurrence in the literature.

**Valsalva Maneuver**

The atrial sound disappeared during the strain period in all cases tested, usually reappearing three to five beats following release of the strain (fig. 6). Phonocardiograms taken in seven patients showed that during the five to six beats after its return the atrial sound increased progressively in intensity and was much louder than before the strain period. At the same time, in four patients, the P-G interval shortened progressively, sometimes with a concomitant decrease in the intensity of the first sound.

**Cold Pressor Test**

Sakamota, Kaito, and Ueda\(^{18}\) performed cold pressor tests on 18 hypertensive subjects and reported that the arterial diastolic pressure and the atrial sound increased in each. They concluded that the diastolic pressure rise caused the increase of the sound. Our results, however, demonstrate that these two effects are at least partly independent of one another. In our hypertensive subjects the average rise of diastolic pressure was the same whether or not the atrial sound responded. In the normotensive subject H.J., striking accentuation of the atrial sound occurred although the diastolic pressure failed to rise significantly (fig. 3). These observations suggest that the increase of the atrial sound may be due to factors other than, or in addition to, the rise in arterial pressure.

Recent investigations\(^{19-23}\) have shown that the cold pressor test can raise peripheral resistance, peripheral venous tone, superior vena cava pressure, and, sometimes, cardiac output, presumably by means of sympathetic nervous impulses to peripheral arteries, peripheral veins, and perhaps the heart. Vasconstriction, by opposing left ventricular ejection, may increase the left ventricular diastolic pressure during atrial systole. Venous constriction, by augmenting venous return,\(^{24}\) may increase left atrial volume; the force and volume of atrial ejection, increased in accordance with Starling's law,\(^{25}\) may be further enhanced by the positive inotropic influence which sympathetic activity has upon the left atrium.\(^{26}\) The more vigorous atrial contraction, and possibly the increased ventricular diastolic pressure during atrial systole, may be responsible for the louder atrial sound; the former mechanism, by increasing flow across the mitral valve, may explain the increased atrioventricular diastolic murmur in mitral stenosis.

The effect on the atrial sound appears to be independent of changes in heart rate also.

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**Figure 1**

*E. B., hypertensive woman, age 67. Cold pressor test. Simultaneous electrocardiogram appears at 540 cps. a = atrial sound; 1,2 = first and second heart sounds; SM = systolic murmur. a. Control. Atrial sound is absent. Blood pressure 200/120 mm. Hg. Heart rate 86/min. b. 20 seconds after right hand placed in iced water. Atrial sound has appeared and is quite loud. B.P. 220/150 mm. Hg. Heart rate 97/min. c. 75 seconds after hand removed from iced water. Atrial sound is disappearing although heart rate has increased slightly. B.P. 240/135 mm. Hg. Heart rate 100/min.*

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Figure 2

N. D., hypertensive woman, age 47. Cold pressor test. a. Control. A faint atrial sound occurs intermittently. B.P. 200/150 mm. Hg. Heart rate 97/min. b. 40 seconds after onset of cold immersion. A loud atrial sound is present in each beat. B.P. 230/165 mm. Hg. Heart rate 100/min.
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Figure 3

H. J., normotensive man, age 38, mild congestive failure due to myocardial infarction two months previously. Cold pressor test. a. Control. A faint atrial sound is present. B.P. 110/90 mm. Hg. Heart rate 100/min. b. 120 seconds after onset of cold immersion. Atrial sound has increased in intensity and is now as loud as, or louder than, the third heart sound. First heart sound has also increased. B.P. 120/90 mm. Hg. Heart rate 107/min.

It cannot be inferred from the minor degree or absence of cardiac acceleration during the test that cardiac sympathetic activity is not increased. The former may, in part, result from reflex bradycardia secondary to the acute rise of blood pressure. It should also be noted that efferent sympathetic stimulation may sometimes increase cardiac contractility with little or no effect on heart rate.27 28

Accentuation of the first heart sound during the test is perhaps due to increased sympathetic activity in the ventricles. A similar finding during epinephrine administration has been attributed to a steeper rise of ventricular pressure, which produces more rapid atrioventricular valve closure.8 29 30

The increase in the second heart sound is probably due to the higher aortic pressure, which produces more forceful aortic valve closure.8

Carotid Sinus Massage

The finding that carotid sinus massage frequently decreases the amplitude of the atrial sound confirms previous reports.6 31 33
cept for Read and Porter, who attributed it to cardiac slowing, writers on the subject have offered no explanation of this phenomenon.

By prolonging diastole, bradycardia increases the time available before onset of atrial systole for the ventricles to relax, which may increase their distensibility, and for the atria to empty passively, which may decrease their volume. In addition, carotid sinus stim-

Figure 4
M. M., hypertensive woman, age 48. Carotid sinus massage (during last four beats on the record). The atrial sound becomes faint and almost disappears. Another electrocardiogram (not shown), which was taken simultaneously on a direct-writing electrocardiograph, demonstrated that the P-R interval did not change.

Figure 5
G. R., hypertensive man, age 47. Carotid sinus massage. Atrial sound becomes louder, an unusual finding which occurred consistently in 20 repetitions of the procedure.
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The increased venous return results mainly from restoration of normal intrathoracic pressure, but peripheral venoconstriction caused by sympathetic activity may also contribute to it.

Other Comments

Atrial sounds may become louder during exercise and in emotional states, such as apprehension and anger, in which sympathetic activity is prominent. Their frequent occurrence in thyrotoxicosis may be due not only to increased venous return, but also to potentiation of catecholamines in the atrium by excess thyroid hormone.

Although the autonomic nervous system is invoked to a greater or lesser extent by the three procedures used in this study, the hemodynamic changes involved are so complex that only with more direct methods such as cardiac catheterization and intracardiac phonocardiography can the relative importance of the various factors involved be elucidated.

Summary

Three procedures were investigated for their effect on the atrial sound. The cold

ulation has a negative inotropic effect on the left atrium, and by reducing peripheral venous and arteriolar tone, decreases venous return and peripheral resistance, respectively. Decreased force of atrial contraction, together perhaps with altered ventricular diastolic pressure and distensibility, may be responsible for diminution and disappearance of the atrial sound. (Reduced venous return may partly explain the disappearance of protodiastolic gallops during carotid sinus massage.)

Valsalva Maneuver

A reasonable explanation for disappearance of the atrial sound during the strain period of the Valsalva maneuver is the marked reduction of cardiac filling. During the poststrain period the fact that venous return, peripheral resistance, and cardiovascular sympathetic tone all exceed control levels may be responsible for the striking increase in the atrial sound. Its migration from the first sound, which diminishes, appears to be mainly the result of increasing venous return; the converse occurs when venous return is reduced by applying tourniquets.

Figure 6
M. M., same patient as in figure 5. Valsalva maneuver. During the strain period (first four beats) the atrial sound, previously present, disappeared. The bush-like figure is due to a forceful resumption of respiration. The atrial sound reappears by the third poststrain beat and during the next four beats increases progressively, becoming much louder than before the strain period. It also migrates from the first sound, which becomes fainter.
pressor test caused the atrial sound to appear in one subject and to become louder in 15 of 28 subjects; it increased the atriysystolic murmur in two subjects with mitral stenosis. These effects were at least partly independent of changes in arterial diastolic pressure and heart rate. Carotid sinus massage decreased the atrial sound in five of 10 subjects and produced an unexplained increase in one. Following release of the strain of the Valsalva maneuver the atrial sound became louder than before the strain; in seven subjects its amplitude increased progressively and the sound also migrated from the first heart sound.

Increase of the atrial sound by the cold pressor test is attributed to sympathetic action upon peripheral veins, arteries, and the left atrium; its decrease by carotid sinus massage, to parasympathetic action upon these structures and the cardiac pacemaker; and its increase following release of the strain of the Valsalva maneuver, to combined increases of venous return and cardiovascular sympathetic activity. The proposed explanations require investigation by more direct methods.

Sympathetic activity may contribute to the increased amplitude of atrial sounds during states of exercise and heightened emotion and, in part, for their frequent occurrence in thyrotoxicosis.

References
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Diseases of the Pericardium

Raymond Vieussens in 1706 recorded the morbid appearances of pericardial adhesions and diagnostic signs of effusion into the pericardium. Joseph Exupère Bertin is quoted in 1824 by his son as having observed a case of acute pericarditis in 1739, but the first published account of the disease was by de Senne in 1749. Laennec described some of the physical signs of acute pericarditis, but stated that "of all the severe lesions of the thoracic organs three alone remain without pathognomonic signs to a practitioner expert in auscultation and percussion—namely aortic aneurysm, pericarditis, and polyph in the heart previous to death." Collin in 1824 was the first to give an adequate account of pericardial friction, which he compared to the creaking of new leather. Andral in 1829 recorded pericarditis in acute rheumatism, and in 1839 Bright said that he had long been aware of the association of pericarditis with chorea.—Sir Humphry Davy Rolleston. The Harveian Oration. Great Britain, Cambridge University Press, 1928, p. 14.
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