THE ATRIAL SOUND IN HYPERTENSION AND ISCHÆMIC HEART DISEASE

WITH REFERENCE TO ITS TIMING AND MODE OF PRODUCTION

BY

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The clinical significance of an atrial sound or presystolic gallop in hypertension and other cardiovascular disorders is still a subject of varying interpretation. Until recently the presence of a gallop rhythm has been accepted as an evil prognostic sign (Holt, 1927; White, 1928; Mond and Oppenheimer, 1929; Duchosal, 1932, 1935; Bramwell, 1935; Lian, 1948). Miles (1951), Weitzman (1955), and Evans (1957), however, have all noted that an atrial sound may occur in hypertension in the absence of any evidence of left ventricular failure and with normal effort tolerance.

In a clinical and phonocardiographic study in hypertensive patients, we noted much variation in the timing of the atrial sound, unrelated to any alteration in the P-R interval. The interval between the first sound and the atrial sound may be very wide or no greater than that between the two components of a split first sound. As this variation in timing was noted in the same patient under different conditions, serial phonocardiograms were used to study the relationship between the clinical state and the timing of the atrial sound.

METHODS

Low frequency phonocardiograms were performed with an apparatus whose frequency response specifications were similar to those recommended by Leatham (1952) for clinical work. Standard lead II was used throughout for the simultaneous electrocardiograms because of possible variations in the commencement of the QRS in different leads.

We define an atrial sound as any audible vibration occurring more than 0.07 sec. after the P wave (Duchosal, 1935) and preceding the start of the ventricular electrocardiogram. Care was taken to avoid confusion with third sounds and summation sounds; each figure shows either the preceding second sound or a flat base line for 0.24 sec. before the atrial sound.

RESULTS

(i) *Clinical Improvement and Deterioration.* Serial phonocardiograms in patients with ischæmic and hypertensive heart disease showed that with clinical improvement the atrial sound approaches the first heart sound and may fuse with it (Fig. 1 and 2); subsequent deterioration was accompanied by migration of the atrial sound away from the first sound (Fig. 3). These changes are appreciable on auscultation: patients with the greatest impairment of cardiac function have an obvious gallop rhythm, whereas, with improvement, as the atrial sound approaches the first heart sound, the cadence resembles that due to splitting of the first sound. The intensity of the atrial sound may or may not diminish as it approaches the first sound.

The most useful phonocardiographic measurement in these cases is the distance from the beginning of the P wave to the atrial sound. Duchosal (1932, 1935) termed this the P-G (G=gallop) interval. With severe impairment of cardiac function the P-G interval may be as short as 0.08 sec. As the clinical state improves the P-G interval lengthens and the atrial sound may

FIG. 1.—Serial phonocardiograms (A, B, C, D, and E) taken at weekly intervals in a patient with malignant hypertension on hypotensive drugs. They show the effect of clinical improvement; the atrial sound is moving towards the first sound in tracings A, B, and C and becomes incorporated into the first sound in tracings D and E. The P-G interval lengthens from 0.10 in (A) to 0.13 in (B) and 0.15 sec. in (C). In (D) and (E) the P-G interval is greater than the P-R interval. The records were taken when the blood pressure was at its highest daily level and with the patient in a recumbent position in order to exclude the acute effects of lowering the blood pressure (see Fig. 5).

become incorporated into the first heart sound with a P-G interval of 0.20 sec. or more. When the atrial sound occurs after the commencement of the QRS complex and thus forms part of the first heart sound it is best termed an atrial component of the first heart sound (Kincaid-Smith and Barlow, 1959).

The length of the P-R interval influences the audibility of the atrial sound. With a short P-R the atrial sound may be close to or form part of the first heart sound, even where severe cardiac disability is accompanied by a short P-G interval. Conversely, a long P-R interval favours the appearance of an atrial sound, and with delayed A-V conduction time beyond 0.20 sec. an atrial sound may occur without any accompanying cardiac abnormality (Evans, 1943).

The migration of the atrial sound towards the first heart sound accompanying clinical improvement was followed in serial phonocardiograms in nine patients with malignant hypertension and six with benign hypertension. Moderate to severe effort dyspnoea or nocturnal dyspnoea had been present in all, together with electrocardiographic and radiological evidence of left ventricular enlargement. It was only within this dyspnoeic group that we encountered a short (0.08–0.12 sec.) P-G interval. An atrial sound may occur in benign hypertension in the absence of any cardiac symptoms

FIG. 2.—(A), (B), and (C) are serial phonocardiograms taken at 10-day intervals following an acute myocardial infarction in a patient with normal blood pressure. The atrial sound moves towards the first sound (B) and becomes incorporated as an atrial component of the first sound (C). The initial first sound component in (C) was shown to be an atrial component by its response to carotid sinus pressure (Kincaid-Smith and Barlow, 1959).

but in such patients we found a long (0.15–0.18 sec.) P–G interval and an atrial sound close to the first heart sound.

Where an atrial sound develops with an acute myocardial infarction it may disappear altogether with clinical improvement. In the six patients with normal blood pressure with serial records the mode of disappearance was the same as in hypertension (Fig. 2).

In three patients with ischæmic heart disease clinical deterioration was accompanied by a decrease in the P–G interval, and in one severely hypertensive patient who refused further treatment serial records showed a similar change (Fig. 3). Acute deterioration was observed in one patient with ischæmic heart disease who developed a further attack of ischæmic pain while a phonocardiogram was being taken. An immediate change occurred in the phonocardiogram: the atrial component of the first sound present in records taken before the pain developed became a presystolic atrial sound (Fig. 4). The proof that the initial first sound vibration in this patient was an atrial component is given elsewhere (Kincaid-Smith and Barlow, 1959, Fig. 9). The sound disappeared when the atrium was arrested by carotid sinus pressure. The ischæmic pain responded to amyl nitrite inhalation but this had no effect on the atrial sound.

(ii) *Hypotensive Drugs:* (a) *Long-term Treatment.* While an increase in the P–G interval accompanies clinical improvement in hypertensive patients when cardiac symptoms are treated by bed rest and digitalis, a more striking alteration in timing of the atrial sound is seen when hypotensive drugs are employed. Fig. 1 shows phonocardiograms at weekly intervals during hypotensive treatment in a man aged 30 with malignant hypertension. The atrial sound migrates towards the first heart sound and becomes incorporated as an atrial component of the latter. In the last two records (Fig. 1, D and E) the first sound is clinically and phonocardiographically indistinguishable from normal and has remained so over eight months further observation. The records were taken when the blood pressure was at its highest daily level to exclude acute hypotensive effect. It has been possible to maintain this patient's blood pressure at levels of 130/80 throughout the day whereas pre-treatment levels had been 260/140. In the majority of patients a less satisfactory therapeutic result is obtained and the atrial sound approaches the first sound in the initial stages of treatment but subsequently varies in timing from just before to just after the commencement of the QRS complex with a P–G interval of 0.15–0.18 sec.

(b) *Acute Effect of Hypotensive Drugs.* In hypertensive patients disappearance of the atrial sound was noted as an immediate sequence of intravenous administration of hypotensive drugs. Serial phonocardiograms were done in 20 cases during the fall in blood pressure following an

intravenous dose of pentolinium, hexamethonium, or pempidine. Fig. 5 (the same patient as Fig. 1) shows that the mode of disappearance of the atrial sound in this acute hypotensive response is the same as the more gradual change that occurs during maintenance treatment with hypotensive drugs.

FIG. 3.—Serial phonocardiograms (A), (B), and (C) at 10-day intervals during clinical deterioration in a severely hypertensive patient who refused further treatment. The atrial vibration forms part of the first sound in (A), just precedes Q in (B), and is 0.05 sec. ahead of Q in (C).

maximum change is seen about 30 seconds after a single large inhalation, when the atrial sound may appear as an atrial component of the first sound. Within one to three minutes the phonocardiogram has again become identical with the control tracing.

The P-G interval increases and the atrial sound approaches and becomes incorporated into the first sound, virtually disappearing when the blood pressure is at its lowest level (Fig. 5F). These changes may occur with small doses of hypotensive drugs and with relatively slight lowering of the blood pressure. The atrial sound migrates towards the first sound as the blood pressure falls (Fig. 5A-F) and moves away from the first sound as the blood pressure rises (Fig. 5G-J). The most convenient method for demonstrating this change over a short period is to produce postural hypotension with the patient in a semi-upright position and raise the pressure again by laying the patient flat. It can be shown, however, that the change is independent of posture as it accompanies blood pressure reduction in any position.

The effect of hypotensive drugs was not studied in patients with ischaemic heart disease or any other cardiac abnormality.

(iii) *Application of Tourniquets to the Limbs.* The same movement of the atrial sound towards the first heart sound can be produced by the application of sphygmomanometer cuffs to occlude venous return from the four limbs (Fig. 6). The maximum alteration in the timing of the sound occurs within three minutes of the application of the cuffs and fifteen seconds after the pressure is released the atrial sound returns to its original position.

Venous "cuffing" produced this change in ischaemic heart disease (3) and hypertension (8) but no similar alteration in timing of the atrial sound occurred in heart block (2) or prolonged P-R interval (1). In the latter three cases there was no cardiac abnormality other than the conduction disorder and the P-G interval was 0.21-0.24 sec.

(iv) *Amyl Nitrite.* In most hypertensive patients (20/22) the inhalation of amyl nitrite produces a similar change in the timing of the atrial sound to that following ganglion-blocking drugs and the application of venous tourniquets. Serial phonocardiograms taken at 15-second intervals for 3 minutes after amyl nitrite inhalation show that the atrial sound migrates towards the first heart sound with lengthening of the P-G interval (Fig. 7). The

FIG. 4.—Records taken before (A) and during (B) an acute attack of ischæmic pain in a patient with normal blood pressure and severe ischæmic heart disease. An atrial component of the first sound present in the first tracing has become a presystolic atrial sound in the second one. The atrial component of the first sound disappeared when carotid sinus pressure produced atrial arrest, thus establishing its identity (Fig. 9, Kincaid-Smith and Barlow, 1959).

In ten of eleven patients with ischæmic heart disease no change in the atrial sound followed amyl nitrite inhalation. In five patients with ischæmic heart disease, four with hypertension and one with normal blood pressure, the atrial sound altered as in the pure hypertensive group.

In patients with an atrial sound due to prolongation of the P-R interval beyond 0.20 sec. or complete heart block, but with no other detectable cardiac abnormality, the timing of the atrial sound remained unchanged after amyl nitrite inhalation. The P-G intervals in these patients were 0.21, 0.22, and 0.24 sec. However, in three patients with hypertension and prolongation of the P-R interval, the P-G interval lengthened from 0.15 to 0.19, from 0.16 to 0.19, and from 0.18 to 0.22 sec. respectively, following amyl nitrite inhalation.

(v) *Respiration.* In cor pulmonale the right atrial sound is best audible in the epigastrium or at the left sternal edge. Phonocardiograms in five such patients showed that the timing of the atrial

FIG. 5.—(A–J) are serial phonocardiograms taken during acute reduction of the blood pressure in a patient with malignant hypertension (the same patient as in Fig. 1). An injection of 5 mg. of ansolysen was given intravenously after the control tracing (A) had been taken. Records (B–F) were taken as the blood pressure fell, with the patient sitting in a semi-upright position in a cardiac bed. Records (G), (H), (I), and (J) were taken as the patient was gradually lowered into a recumbent position and as the blood pressure rose. The atrial sound approaches and disappears into the first sound as the pressure falls and moves out into a presystolic position as the pressure rises.

sound varies with respiration. During inspiration it is most widely separated from the first sound with a short P–G interval, and in expiration the P–G interval lengthens (Fig. 8). This change, together with the increase in amplitude of the atrial sound often seen in inspiration in such patients, makes it more easily audible at this stage of the respiratory cycle.

No constant change in the timing of the atrial sound accompanies the phases of respiration in hypertension or ischaemic heart disease, though the intensity of the sound is often decreased on inspiration, perhaps due to increasing depth of overlying lung.

(vi) *Effect of Rest.* The P–G interval may lengthen by 0.02–0.03 sec. as a result of ten minutes' to an hour's rest in hypertension and ischaemic heart disease. Where the atrial sound just precedes the first sound this change in timing will convert it into an atrial component of the first sound (Fig. 9). Rest does not constantly produce this change and emotional factors may explain this variability of response, as several patients showed shortening of the P–G interval on introduction of an intravenous needle or when warned of any such procedure.

(vii) *The Effect of Migration of the Atrial Sound on the Intensity of the First Heart Sound.* As the atrial sound approaches the first heart sound the intensity of the latter increases (Fig. 9). This change was noted in many records, including those showing the effect of clinical improvement, hypotensive drugs, amyl nitrite, venous cuffing, and rest. Other factors such as the specific effect of amyl nitrite on the intensity of the first sound, or the tachycardia accompanying lowering of the blood pressure, may account for the change in some records but these can be excluded in the group showing the effect of rest. Rest usually slows the pulse and should, if anything, decrease the intensity of the

FIG. 6.—Serial phonocardiograms in a hypertensive patient, showing the effect of venous pooling in the four limbs resulting from the application of sphygmomanometer cuffs inflated to 100 mm.Hg. (A) was taken before the cuffs were inflated, (B) 30 seconds after, and (C) 3 minutes after inflation of the cuffs. (D) was taken 15 seconds after the pressure was released. The atrial sound moves towards and into the first sound, while the cuffs are inflated, and becomes presystolic again when the cuffs are released. Note that the major components of the first sound increase in intensity as the atrial sound migrates towards the first sound.

first sound, but whenever the atrial sound moves towards the first sound during rest the intensity of the latter increases (Fig. 9). Records showing a change in P-wave shape without a change in P-R interval but with alteration of the timing of the atrial sound (Kincaid-Smith and Barlow, 1959, Fig. 6 and 7) substantiate this observation.

(viii) *Studies in Complete Heart Block and Prolonged P-R Interval.* The atrial vibrations of heart block and prolonged P-R interval are usually clearly separated into two components (Fig. 10). We agree with Weitzman (1955) that the first component is inaudible and represents atrial contraction, and that the second component is the audible atrial sound. The first component occurs 0.07–0.14 sec. after the P wave and the second component in the cases with no other cardiac abnormality that we studied occurred 0.21–0.28 sec. after the P wave.

FIG. 7.—Serial phonocardiograms, showing the effect of a single inhalation of amyl nitrite. The atrial sound moves towards the first sound and at 35 seconds it forms an atrial component of the first sound: at 60 seconds the tracing is the same as the control.

FIG. 8.—Phonocardiogram, showing the effect of respiration on the timing of the right atrial sound in cor pulmonale. The atrial sound is more widely separated from the first sound (i.e. the P-G interval is shorter) in inspiration than in expiration. The maximum effect is seen in early inspiration. The time marking refers to the smaller subdivisions only just visible at the left of the figure. The larger clear markings are at 0.2 sec. intervals.

In three patients with severe hypertension and prolongation of the P-R interval the timing of the first component was the same as that where the conduction defect was the only abnormality, but the second component occurred earlier; 0.15–0.18 sec. after the P wave. This timing of the second component would allow it to precede the first sound even in the absence of any conduction defect.

In cases with only conduction defects we were unable to alter the timing of the second component with amyl nitrite or venous tourniquets whereas the hypertensive cases with prolonged P-R interval behaved similarly to other hypertensive cases, the P-G interval increasing in response to amyl nitrite and venous cuffing.

In complete heart block the P-G interval (measured to the second component) varies with the stage of diastole at which atrial contraction occurs. Fig. 11 shows that in early diastole the P-G interval is long (average 0.26 sec.) whereas in late diastole it is shorter (average 0.23 sec.). When atrial contraction occurs during ventricular systole the second component does not occur (Kincaid-Smith and Barlow, 1959).

FIG. 9.—Phonocardiograms, showing the effect of rest on the atrial sound. Tracing (A) was taken as soon as the patient reached the phonocardiographic room. Tracing (B) was taken after 10 minutes' rest, and tracing (C) after an hour's rest. The atrial vibration moves from a presystolic position (A) to become an atrial component of the first sound (C). The intensity of the first major component of the first sound increases greatly as the atrial sound approaches it.

FIG. 10.—Showing that the atrial phonocardiogram in heart block appears as two well-defined components.

The audible atrial sound in heart block may consist of several vibrations on a phonocardiogram, and clinically it may be heard as a single sound, an apparent double sound, or as a short rumble resembling a mid-diastolic murmur.

DISCUSSION

Our observations on the atrial sound support Duchosal's (1932) thesis that with improvement in the clinical condition the P-G interval lengthens and the atrial sound approaches, and may fuse with, the first heart sound. Wolferth and Margolies (1933) pointed out that several of Duchosal's examples were invalid because he had not considered the third heart sound or the phenomenon of summation. In 1935 Duchosal published further observations on unequivocal atrial sounds confirming his previous findings. Evans (1943) raised a valid objection to Duchosal's work when he pointed out that the start of the atrial sound in a phonocardiogram from a healthy subject may be the same as that in patients with cardiac disease (namely P-G 0.08 sec.). We think that this

FIG. 11.—Showing that in complete heart block the timing of the atrial sound (second component of the atrial phonocardiogram) varies with the stage of diastole at which it occurs. In early diastole (0.30 sec after the second sound) it occurs 0.26 (average) sec. after the P wave whereas in late diastole it occurs 0.23 (average) sec. after the P wave. Each small square in the figure represents one observation.

apparent weakness in Duchosal's thesis is explained by a study of the components of the atrial phonocardiogram. This consists of two groups of vibrations (Lewis, 1915; Weitzman, 1955) and we agree with the latter author that the first component (0.08–0.14 sec. after the P wave) is inaudible. This inaudible component occurs in the normal phonocardiogram as well as in patients with cardiac disorders and we think that Evans' (1943) observations referred to this component. The audible component of the atrial sound is usually of higher pitch and higher amplitude and in our experience is only found in cardiac disorders or with prolongation of the P–R interval. The components of the atrial sound are best seen in heart block (Fig. 10) where atrial vibrations may last 0.14 sec. and the initial low-pitched vibrations are often well-defined from those of higher frequency and amplitude which constitute the audible atrial sound. It is to this latter component that Duchosal's (1932, 1935) and our own observations on variations in the timing of the atrial sound apply. Where both can be shown, the initial atrial vibrations remain constant in timing, during acute and long-term observations on the movement of the audible atrial sound.

The shortest P–G interval which we have observed was 0.08 sec. at which stage the audible atrial sound seems to coincide with or follow very shortly upon the inaudible atrial vibrations. With improvement, the atrial sound may move by as much as 0.15 sec. thus prolonging the P–G interval to 0.23 sec. (Fig. 1). At this stage the atrial vibration occurs after the beginning of the QRS complex and thus by definition forms part of the first heart sound. A more detailed analysis of this atrial component of the first sound is given elsewhere (Kincaid-Smith and Barlow, 1959). It is only in patients with moderate or severe cardiac symptoms in whom we have observed a short (0.08–0.12 sec.) P–G interval, although an atrial sound is a common manifestation in hypertension even in the absence of any symptoms. We found an audible atrial sound in 50 per cent of patients with average casual blood pressure readings above 180/100 (Barlow and Kincaid-Smith,

1959). This is a higher percentage than that quoted previously (Miles, 1951; 23%). Miles (1951), Weitzman (1955), and Evans (1957) agree that atrial sounds occur in asymptomatic hypertensive patients. In hypertensive patients with mild or absent cardiac symptoms the P-G interval was usually 0.15–0.20 sec. and the atrial sound just preceded the QRS complex. Most patients on hypotensive treatment retain an audible atrial sound although this may disappear temporarily when the blood pressure is low. It is only where it is possible to maintain the blood pressure at normal levels throughout the day that we have been able to demonstrate permanent disappearance of the atrial sound (Fig. 1).

In ischæmic heart disease an atrial sound that develops after an acute myocardial infarction may disappear altogether, remain as an atrial component of the first sound, or persist. Where it persists but the patient has no symptoms, the timing corresponds to that in the asymptomatic hypertensive group (P-G 0.15–0.20 sec.). We agree with Weitzman (1955) that cardiac symptoms are more common when the atrial sound persists; he noted a poor prognosis in this group.

The P-G interval lengthens with clinical improvement and shortens with clinical deterioration, suggesting that the early occurrence of an atrial sound is a manifestation of less efficient myocardial function. Two of the acute procedures (hypotensive drugs, and venous pooling) whereby we can produce an immediate lengthening of the P-G interval are also used as therapeutic measures in left ventricular failure. This may suggest that the change in the timing of the atrial sound produced is due to relief of overloading of the left ventricle. As, however, an atrial sound occurs in asymptomatic hypertensive subjects it is unlikely to signify any gross rise in diastolic pressure in the ventricle.

The observation that inspiration shortens the P-G interval of the right atrial sound in cor pulmonale whereas it does not alter the left atrial sound of hypertension or ischæmic heart disease suggests that the increased right ventricular filling pressure places an added strain on the right ventricle, thus potentiating the factors that produce the sound.

The fact that venesection does not prolong the P-G interval in cases with complete or first-degree heart block supports the view that in such cases the audible atrial sound is due merely to a temporal separation of atrial and ventricular events and does not signify functional defect. The P-G interval in such cases is considerably longer than in the hypertensive and ischæmic group, in whom, with improvement, the P-G interval approaches that found with normal function but delayed A-V conduction time.

The results obtained with amyl nitrite are difficult to interpret. This drug lowers the blood pressure and its temporary effect in prolonging the P-G interval in hypertensive patients in whom increased blood pressure is the primary disorder may be due to this. It is of interest that although amyl nitrite relieves ischæmic pain it does not as a rule alter the P-G interval in ischæmic heart disease, and did not do so in spite of pain relief in the patient (Fig. 4) who developed an atrial sound with an attack of ischæmic pain.

MECHANISM OF THE ATRIAL SOUND

The mechanism of the atrial sound is still uncertain and some of our observations may help to elucidate this problem.

Charcelay (1837), who first described the atrial sound, thought it was due to atrial contraction. Potain (1900) thought that the sound resulted from sudden tension of the ventricular wall following the inflow of blood in atrial systole. Lewis (1915) described the two components of the atrial sound in heart block and attributed the first component to atrial contraction and the second to closure of the atrio-ventricular valves after the cessation of blood flow from the atrium. Weitzman (1955) demonstrated that the first (inaudible) component of the atrial sound coincided with atrial contraction and agreed with Potain that the second component was a ventricular filling sound. Potain's (1900) theory assumes that the atrial sound is produced by a sudden rise in tension of the ventricular

wall. The early atrial sound present in patients with heart failure could then be due to a raised end diastolic pressure and hence a quick rise to the tension required to produce the sound. The migration of the sound accompanying blood pressure reduction or venous pooling by tourniquets could be due to a decrease in the end diastolic pressure as could the changes accompanying clinical improvement. The diastolic state of the left ventricular myocardium in asymptomatic cases is uncertain, but it is difficult to imagine it is entirely free from "strain".

A recent study (Kuo *et al.*, 1957) in which atrial and ventricular pressures were recorded in patients with gallop rhythm throws doubt on the valvular theory of atrial sound production. These authors showed that atrial pressure was higher than ventricular pressure at the time of the atrial sound and hence thought it very unlikely that it could be due to atrio-ventricular valve closure.

One of our observations that provides further evidence against the valvular theory of atrial sound production is the increase in intensity of the first sound that occurs as the atrial sound migrates towards the first sound with the patient lying quietly at rest.

If the major components of the first sound are due to mitral or tricuspid valve closure their intensity should diminish when atrio-ventricular valve closure due to an atrial sound immediately precedes the first sound. The increase in intensity of the first sound that occurs as the atrial vibration approaches the major components of the first sound may support the "ventricular filling sound" theory of atrial sound production. If a ventricular filling sound immediately precedes systolic mitral and tricuspid closure it implies that flow from atrium to ventricle shortly precedes valve closure. The cusps would thus be widely separated as in mitral stenosis and the intensity of the first sound would be greater. The increase in intensity of the first sound accompanying migration of the atrial sound during the application of tourniquets to the limbs has been confirmed by a recent publication (Leonard *et al.*, 1958).

Observations on the timing of the atrial sound in complete heart block support the explanation that the initial state of ventricular filling determines the length of the P-G interval. Isolated atrial sounds occurring in the early diastolic period have a P-G interval of 0.24-0.28 sec. (average 0.26) while those in late diastole when the ventricle would be more nearly filled have a P-G interval of 0.21-0.25 sec. (average 0.235). This suggests that the critical degree of ventricular distension necessary for the production of the sound follows more closely on atrial contraction when there is a high initial pressure in the ventricle.

Thus we support Duchosal's (1932, 1935) thesis that the P-G interval gives an indication of the degree of cardiac involvement in hypertension and ischaemic heart disease. This has the clinical application that a classical presystolic gallop with wide separation of the atrial sound and first sound indicates greater impairment of function than the close atrial sound or "Galop presystolique retardé" (Lian, 1948). The latter may be present for several years in asymptomatic hypertensive patients.

SUMMARY

In hypertension and ischaemic heart disease the atrial sound approaches and may fuse with the first heart sound during clinical improvement. With deterioration the atrial sound moves in the reverse direction.

In hypertension the same movement of the atrial sound towards the first sound can be produced by the intravenous administration of hypotensive drugs, venous pooling by means of sphygmomanometer cuffs, and amyl nitrite inhalation. The interpretation of these, and of other changes in the timing of the atrial sound, is discussed.

The mechanism and production of the atrial sound is discussed, and evidence is advanced supporting the theory that it is a ventricular filling sound.

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