THE MECHANISM AND SIGNIFICANCE OF THE AURICULAR SOUND

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The auricular sound (variously referred to by other writers as the fourth heart sound, presystolic gallop, or auricular gallop) is heard under certain pathological conditions. It is a dull, low-pitched sound, audible along a line joining the apex to the xiphoid, and is usually more obvious at the latter site. It may be heard either with a bell chestpiece or a thin diaphragm, and is appreciated best of all by direct auscultation with the ear on the patient’s xiphoid, as described by Potain, when the combination of thrust and sound is very obvious. It may indeed be better felt than heard, since its frequency is too low to be transmitted adequately by stethoscope tubing (Rappaport and Sprague, 1942).

The auricular sound must be differentiated from other additional sounds. Physiological splitting of the first heart sound is recognizable because its two components are high-pitched, whereas the auricular sound is of low frequency. The time interval between auricular and first sounds (0.08 sec.) is greater than that separating the two components of a split first sound (0.03 sec., Wolferth and Margolies, 1931); and splitting varies with respiration, becoming more obvious during expiration. An early systolic ejection sound which may occur with dilatation of the pulmonary artery or aorta (Leatham and Vogelpoel, 1954) is also of high frequency and is clearly audible over the base of the heart. Summation gallop (Wolferth and Margolies, 1933), due to superimposition of the third and auricular sounds, will disappear if the heart can be slowed by carotid sinus pressure, and can be differentiated from the auricular sound by this means.

Previous writers have expressed differing views on the production of the auricular sound and on its prognostic significance, especially in relation to cardiac failure. Since the introduction of multi-channel instruments has now made it possible to record heart sounds and intracardiac pressures simultaneously, it was decided to investigate the relationship of the auricular sound to other events in the cardiac cycle by this means, in order to obtain information about the mechanism of its production. In addition, clinical and phonocardiographic studies were made in a large number of patients to re-assess the significance of the sign.

Methods. Simultaneous sound and pressure recordings were available from 12 cases of congenital heart disease with either pulmonary hypertension or pure pulmonary stenosis in which an auricular sound was present: cardiac catheterization had been undertaken for diagnostic purposes. These tracings were used for direct timing of the auricular sound in relation to pressure changes. They were made with a Sanborn electromanometer and a four-channel recorder designed by New Electronic Products, Ltd.

A clinical study was made of 88 other patients in whom the auricular sound was audible, and a phonocardiogram taken in 62 of them. There were 46 patients with hypertension, 29 with cardiac infarction, 4 with aortic valve lesions, and 9 with heart block. In the hypertensive group there was one patient with coarctation of the aorta and another with chronic nephritis. The remaining 44 had essential hypertension and three of these were seen during a malignant phase. Six of the patients with infarction were studied within a few days of the acute attack, and the remainder at periods varying from three months to several years after; in all cases there was electrocardiographic confirmation.

The clinical, electrocardiographic, and radiological findings in these 88 patients were compared with those in 205 with similar diseases in whom the auricular sound was not detected. In this group there were
86 patients with hypertension (including five with coarctation of the aorta), 16 with recent and 62 with old cardiac infarction (with electrocardiographic confirmation), and 42 with pure aortic valve lesions, cases with co-existent mitral disease being excluded. In addition, over 100 adults with no evidence of cardiovascular disease were examined to find out if the auricular sound was ever audible in health. All patients were examined by the author.

The majority of phonocardiograms were obtained with amplifiers and string and mirror galvanometers made by the Cambridge Instrument Co. The medium frequency channel (Leatham, 1952) corresponds approximately to the findings on auscultation with the bell type of stethoscope or with a thin diaphragm, and was found suitable for recording the auricular sound. (The low frequency record shows it even more clearly, but also shows vibrations that may be inaudible.) The high-frequency record ("logarithmic" of Rappaport and Sprague) corresponds more closely to auscultation with the conventional diaphragm, but so attenuates the low-pitched sounds as to be of no value in this study. In all cases, phonocardiograms were recorded from the mitral area and the lower left sternal edge simultaneously with lead I or lead II of the electrocardiogram. In this investigation, vibrations starting after the onset of the P wave of the simultaneous electrocardiogram were considered to be of auricular origin only if their onset preceded that of the QRS complex, so as to avoid confusion with the muscular component of the first heart sound described by Rappaport and Sprague (1942).

**Mechanism of the Auricular Sound**

Recordings from patients with heart block demonstrate the features of auricular contraction uninfluenced by succeeding ventricular events. When separate auricular sounds were not heard, a group of small vibrations were seen on low-frequency traces, following the P wave of the cardiogram (Fig. 1). These were below the range of audibility (Rappaport and Sprague, 1942). In cases with audible auricular sounds the later vibrations of the group were greatly increased in amplitude so that the auricular vibrations appeared to be in two sets (Fig. 2). This "double" appearance of the auricular sound in recordings from cases of heart block has previously been noted by McLeod et al. (1932), Duchosal (1932), Braun-Menendez (1938), and Evans (1943). This intensification of the later auricular vibrations was a constant feature of phonocardiograms recorded from patients with audible auricular sounds, irrespective of the actual cardiac lesion present. Their relation to electrical and mechanical events in the right heart are shown in Table I.

The earlier, inaudible component of the auricular vibrations corresponds in timing to the rise of pressure in the right auricle, while the audible component does not occur until 0.05–0.09 sec.

![Fig. 1.—Low amplitude auricular vibrations (A) in a case of complete heart block, in whom no auricular sounds were audible.](image-url)
later. These findings indicate that the audible component is a filling sound produced by blood entering the ventricle from auricular systole, while the preceding inaudible vibrations are due to the actual muscular contraction of the auricle. This view is in keeping with the experimental work of Wiggers (1949), who showed that the maximum increase in ventricular volume occurs later than the maximum rise in auricular pressure: and of Braun-Menendez and Solari (1938) who recorded two groups of auricular vibrations in dogs. They too considered these to be produced respectively by auricular contraction and ventricular filling.

While no direct comparison with pressure records from the left heart was available, the relationship between electrical and phonocardiographic events was found to be similar to that already described (see Table II).

The average interval between the onset of the P wave and the onset of the early auricular vibrations was 0.12 sec. A similar time interval was found between the onset of the P wave and the onset of left auricular contraction (as shown by the “a” wave in direct tracings from the left auricle) recorded by cardiac catheterization in eight cases of auricular septal defect. This supports the view that the early vibrations are produced by auricular contraction. The larger, audible vibrations

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**TABLE I**

**TIME INTERVALS IN 12 PATIENTS CATHETERIZED BECAUSE OF PULMONARY HYPERTENSION OR PURE PULMONARY STENOSIS**

<table>
<thead>
<tr>
<th>Description</th>
<th>Interval (sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset of P wave to onset of early auricular vibrations</td>
<td>0.09 (0.08-0.10)</td>
</tr>
<tr>
<td>Onset of P wave to onset of late (audible) auricular vibrations</td>
<td>0.12 (0.09-0.16)</td>
</tr>
<tr>
<td>Onset of P wave to onset of right auricular systole</td>
<td>0.06 (0.05-0.07)</td>
</tr>
<tr>
<td>Onset of right auricular systole to audible auricular vibrations</td>
<td>0.07 (0.05-0.09)</td>
</tr>
</tbody>
</table>

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**Fig. 2.—Inaudible (A) and audible (A) components of auricular sound.**
THE AURICULAR SOUND

occurred 0.05-0.11 sec. later, and so could be produced by filling of the left ventricle. The onset of left auricular vibrations occurred 0.03 sec. later than the right side and this is in keeping with the work of Groedel (1948), who showed that activation of the left auricle took place 0.03 sec. after the right. The duration of the whole group of auricular vibrations was found to range from 0.04 to 0.14, with an average of 0.095 sec.

TABLE II
RELATION OF AURICULAR VIBRATIONS TO ELECTROCARDIOGRAM IN LEFT-SIDED LESIONS

<table>
<thead>
<tr>
<th>Condition</th>
<th>Onset of P wave to onset of early auricular vibrations</th>
<th>Onset of P wave to onset of audible auricular vibrations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertensive heart disease (29 recordings)</td>
<td>0.125 sec. (0.08-0.16)</td>
<td>0.17 sec. (0.14-0.24)</td>
</tr>
<tr>
<td>Cardiac infarction (21 recordings)</td>
<td>0.10 sec. (0.07-0.15)</td>
<td>0.17 sec. (0.12-0.20)</td>
</tr>
<tr>
<td>Aortic valve disease (4 recordings)</td>
<td>.</td>
<td>0.18 sec. (0.16-0.23)</td>
</tr>
<tr>
<td>Heart-block (8 recordings)</td>
<td>. .</td>
<td>0.12 sec. (0.09-0.15)</td>
</tr>
</tbody>
</table>

Similar values for these time intervals and for the duration of the sound have been recorded by other observers (Wolferth and Margolies, 1933; Lewis, 1934; Duchosal, 1935; and Miles, 1951). These authors, however, did not make a clear distinction between audible and inaudible components.

There was no obvious respiratory variation in the amplitude of the audible component in most of the tracings in the present series. In a small number it became larger during inspiration (Fig. 3), but this was not appreciable by clinical auscultation.

![Figure 3](http://heart.bmj.com)
The findings in patients with hypertension are set out in Table III. Electrocardiographic or radiological evidence of left ventricular hypertrophy was present in 44 of the 46 cases with an auricular sound; in the remaining two the electrocardiogram showed ischaemic changes. The sound was never detected in any patient when both X-ray and electrocardiogram were normal.

**TABLE III**

**FINDINGS IN 131 HYPERTENSIVE PATIENTS**

<table>
<thead>
<tr>
<th>Condition of left ventricle</th>
<th>With auricular sound (46 patients)</th>
<th>With no auricular sound (85 patients)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertrophied</td>
<td>96%</td>
<td>75%</td>
</tr>
<tr>
<td>Normal size with ischaemic EC.</td>
<td>4%</td>
<td>5%</td>
</tr>
<tr>
<td>Normal</td>
<td>0</td>
<td>20%</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Functional capacity of patient</th>
<th>With auricular sound (46 patients)</th>
<th>With no auricular sound (85 patients)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Failure—Congestive</td>
<td>9%</td>
<td>6%</td>
</tr>
<tr>
<td>Failure—Left ventricular</td>
<td>14%</td>
<td>3%</td>
</tr>
<tr>
<td>Total</td>
<td>23%</td>
<td>9%</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Blood pressure</th>
<th>Average</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>210/125</td>
<td>290–160</td>
</tr>
<tr>
<td></td>
<td>200/120</td>
<td>100–110</td>
</tr>
<tr>
<td></td>
<td>290–150</td>
<td>150–110</td>
</tr>
</tbody>
</table>

It should, however, be noted that left ventricular hypertrophy was present in 75 per cent of patients without an auricular sound, and was not necessarily any less marked (see also Miles, 1951). The incidence of cardiac failure was higher (23%) in the group with the sound, than in those without (9%), but the majority of patients were ambulant and 20 per cent of the patients with the auricular sound were symptom-free. Blood-pressure readings in the two groups showed no significant difference and the age distributions were also similar.

The auricular sound was heard in 6 of 22 patients with recent cardiac infarction. No influence on immediate prognosis was noted in this small series. (In a larger, unpublished series of 176 cases of recent infarction admitted to the National Heart Hospital but not all examined by the author, data from the case-records showed a higher mortality and greater incidence of cardiac failure when the auricular sound was heard.) In the present series the sound was still present after recovery in 23 patients and persisted throughout the period of observation. Nine of these patients had hypertension and were excluded from the analysis because of this additional aetiological factor. Table IV sets out the findings in the 54 cases with normal blood pressures who were observed after recovery from the acute attack.

It will be seen that the incidence of cardiac failure was much higher among patients with the auricular sound (36%) than in those without (2.5%). Limitation of effort from this cause in the cases with an auricular sound accounts for the low incidence of effort pain in this group. Radiological enlargement of the left ventricle was present in a high proportion of this group, but was
absent in 20 per cent. Extensive changes in the electrocardiogram were found in 25 per cent of the patients with the sound, as against 16 per cent in those without. There was no significant difference in blood pressure readings or in the age distribution in the two groups.

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The auricular sound was heard in 4 of 46 patients with aortic valve disease. It was found in 3 of 23 cases with pure or dominant stenosis and in 1 of 23 with pure or dominant incompetence. In all four the electrocardiogram showed left ventricular hypertrophy: this was found in all but 3 of the 46 cases.

No detailed study was made of the incidence of the sound in congenital heart disease; the 12 cases in this series served principally to provide information about the timing of the auricular sound in relation to auricular systole; but it was noted that marked right ventricular hypertrophy was present in all twelve. A large "a" wave (Abrahams and Wood, 1951) was present in the right auricular pressure tracing in every case. Effort intolerance was present in all, but cardiac failure was not found.

The auricular sound could not be heard in any of over 100 healthy subjects, and it was concluded that it is not audible in the absence of cardiovascular disease.

DISCUSSION

The time relation of the auricular sound to electrical and mechanical events in the cardiac cycle suggests that it is produced in the ventricle by blood entering from auricular systole. Its configuration on the phonocardiogram resembles that of the third heart sound, which is also a filling sound produced in the ventricle. Unlike the third heart sound, however, the entry of blood from auricular systole does not give rise to audible vibrations in the normal ventricle: in every case with an auricular sound there was ventricular hypertrophy or ventricular ischemia. It may be that hypertrophied or infarcted ventricular muscle is less tolerant of distension. This theory was originally propounded by Potain in 1885, when he wrote "The gallop . . . is more pronounced if the heart is not distensible: the failure of distensibility may depend either on a sclerotic thickening . . . or to a decrease in muscular tonicity." Duchosal (1935) similarly attributed the sound to the
"shock of ventricular incompressibility" when the left ventricle was diseased. Increased force of auricular contraction may also play a part, since a large "a" wave was seen in the right auricular pressure tracings in all 12 cases catheterized. Under these circumstances, filling gives rise to vibrations whose amplitude may approach that of the first heart sound (Fig. 4 and 5).

Audibility is further enhanced when, as the result of a P–R interval of 0.15–0.18 sec., the first heart sound is soft and the auricular and first sounds are widely separated. In one patient, the

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**Fig. 4.**—Large auricular sound in a patient with hypertensive heart disease.

**Fig. 5.**—Large auricular sound in a patient, following cardiac infarction (X= artefact).
first component of a split first heart sound, although close to the auricular sound, was so small as to be inaudible at the apex: the auricular sound was therefore easily separated by the ear from the later component of the first sound (Fig. 6).

Clinical Significance. The particular association of the auricular sound with hypertension was first noted by Potain in 1875. Subsequently many observers have regarded the sign as having a bad prognostic significance and have stressed a relationship with cardiac failure (Lewis and Dock, 1934; Bramwell, 1935; Duchosal, 1935; Evans, 1943; and Levine and Harvey, 1949). In Miles's (1951) series, failure was not always present, but effort intolerance was invariably found.

In the present series only 23 per cent of hypertensive patients with an auricular sound showed evidence of failure. The great majority were ambulant and able to follow their ordinary occupations. Death occurred in only 2 of 24 patients observed over periods of from three months to six years after the sound was first noted; and in four others in whom failure occurred, response to treatment was satisfactory. In five patients (including three with malignant hypertension who responded to medical treatment) effort tolerance has remained normal for 2 to 6 years. The auricular sound in hypertension indicates left ventricular hypertrophy rather than cardiac insufficiency; but either may occur without this sign being present (Table III).

Following myocardial infarction, the long-term prognosis was worse in patients in whom the auricular sound persisted after the acute attack. In 56 per cent there was cardiac failure or effort dyspnea compared with an incidence of 22 per cent in those without the added sound. The lower incidence of angina in the patients with an auricular sound, shown in Table IV, is misleading, since so many were too dyspneic to undertake much effort. Correlation with left ventricular enlargement was again high (80%), but the left ventricle was of normal size in 20 per cent. It should be noted that the sound may persist for months or years after the acute episode even in symptom-free patients.

The incidence in aortic valve disease was low. This was surprising in view of the considerable degree of left ventricular hypertrophy that was usually seen on the electrocardiogram. In cases
of aortic incompetence, it may be that the reflux from the aorta during diastole results in a pressure gradient which is unfavourable to atrioventricular bloodflow at the time of auricular systole. The rarity of the sound in aortic stenosis is more puzzling.

The incidence of failure among the 100 patients with an auricular sound was not high (21%). In fact, the auricular sound was essentially a sign of ventricular hypertrophy (or ischaemia). The usual cause of triple rhythm in heart failure is an abnormal third sound, which may appear to be mid-diastolic because of tachycardia. The latter may result in summation of the third sound with auricular vibrations thus giving rise to the loud triple rhythm commonly heard in heart failure.

**Summary and Conclusions**

One hundred patients with an audible auricular sound were studied in order to determine the mechanism and significance of this finding. The series comprised 46 cases with hypertension, 29 with myocardial infarction, and 25 with other conditions. Phonocardiograms were recorded in 74: in the 12 congenital cases they were taken synchronously with intracardiac pressure pulses. The clinical findings in these patients were compared with those in 205 patients with similar diseases, in whom the auricular sound was not detected, and in over 100 healthy subjects.

The sound occurs 0·12-0·17 sec. after the onset of the P wave and 0·05-0·09 sec. after the onset of auricular systole. These findings suggest that it is a filling sound produced in the ventricle by blood entering during auricular systole. The vibrations caused by this are normally inaudible, but are accentuated when the ventricle is hypertrophied or has undergone infarction.

The auricular sound was not heard in healthy subjects, nor in any case of heart disease in which both electrocardiogram and X-ray were normal.

In hypertension the sound was always associated with hypertrophy of the left ventricle. Although the patients with the added sound showed a higher incidence of cardiac failure three-quarters (77%) were free of heart failure and about one-quarter (25%) were free of all symptoms.

In cardiac infarction persistence of the sound was associated with left ventricular enlargement in 80 per cent of cases, and with cardiac failure or effort dyspnoea in 56 per cent, but 16 per cent remained symptom-free. The sound was seldom heard in aortic valve disease.

It is concluded that the auricular sound is a filling sound produced in the ventricle. It always indicates abnormality of the ventricle and is not audible in health, but is not direct evidence of cardiac failure.

I would like to express my thanks to Dr. Aubrey Leatham for the great interest he has shown in this study, and for advice and assistance in the preparation of this paper given by him and also by Sir John Patkisson, Dr. Paul Wood, Dr. William Evans, and Dr. Maurice Sokolow. My gratitude is due also to the other Physicians at the National Heart Hospital for permission to study the patients under their care: and to the technical staff of the Institute of Cardiology for their share in the recordings.

**References**


