Understanding the atrial sound

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Charcelay (1838) first described the presystolic sound which was later termed '*bruit de galop*' by Bouillard (Potain, 1894). Potain's description (1875) of this presystolic sound has not been bettered: 'This sound is much duller than normal sounds; it is a shock, a distinct pulsation, scarcely a sound. When the ear is applied to the chest it affects the sense of touch more perhaps than the sense of hearing.' Since the 1930's this sound has often been called the fourth sound to distinguish it from the protodiastolic third sound. Some clinicians prefer to call it the atrial sound because 'fourth' suggests that it may be the last rather than the initial sound of a complete cardiac cycle. Others use the terms atrial or presystolic gallop when it is associated with heart disease. We shall use the term 'atrial sound' to refer to the sound associated with atrial systole both in the healthy and the diseased heart; in bradycardia, tachycardia, and normal heart rate; in sinus rhythm and atrioventricular dissociation; and irrespective of whether the vibrations are audible through the stethoscope or require a phonocardiogram for their detection. We shall use the term 'atrial beat' to refer to the movement of the apex produced by atrial contraction (the 'a' wave of apex displacement). This discussion is only concerned with events arising from the left heart.

Characteristics of the atrial sound

The vibrations of the clinically detectable atrial sound are characteristically of low frequency, between 25 Hz and 100 Hz, and usually near the threshold of human hearing. The sound has a variable relation to the P wave of the electrocardiogram, starting not less than 0.07 sec and usually about 0.14 sec after the onset of the P wave. The peak vibrations of the sound coincide with the peak of the

atrial beat: the outward movement of the left ventricular apex caused by atrial contraction (Fig. 1). The relation of the sound to the rounded 'a' wave of the left atrial pressure pulse is variable.

The atrial sound is most easily detectable with the patient lying on his left side, semirecumbent. The palpating fingers, placed very lightly on the apex, can feel the presystolic thrust of an enlarged atrial beat. Listening at the same time as feeling enables the atrial beat to be distinguished from the double systolic impulse found in some patients with left ventricular disease. The atrial sound is most easily heard with the bell of the stethoscope placed very lightly over the cardiac apex, but is sometimes easier to hear at the left sternal edge or even at the base of the heart. It may be confused with a split sound or a first sound followed by an ejection click, but the atrial sound usually has vibrations of such small magnitude and low frequency that they can be filtered out by pressing firmly with the bell and so converting the skin into a diaphragm for the stethoscope.

Phonocardiograms recorded from microphones in the oesophagus (Orias and Braun-Menendez, 1939) or the left atrium (Crevasse *et al.*, 1962) show that the atrial sound may be preceded by low frequency vibrations, 0.04 sec to 0.06 sec after the onset of the P wave, which are not recorded at the chest wall. In the special case of heart block, instead of a single atrial sound, two components may be recorded at the cardiac apex. The significance of each component is not known.

The first of the two components has all the characteristics of the atrial sound of sinus rhythm. It occurs 0.07 to 0.14 sec after the onset of the P wave, its peak vibrations usually coincide with the peak of the atrial beat, and it disappears when the atrium contracts against the closed mitral valve during ventricular systole (Fig. 2). The second component occurs 0.20 to 0.28 sec after the onset of the P wave and also disappears when the atrium contracts during ventricular systole. These observa-

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FIG. 1 (A) The normal diastolic movements and sounds displayed by simultaneous recording of the apex cardiogram (ADC), the mitral area low frequency phonocardiogram (MALF), and lead II of the electrocardiogram. Diastolic filling of the ventricle occurs in three phases represented by the rapid filling wave (r fw), the slow filling wave (s fw), and the small 'a' wave caused by atrial systole. (B) The abnormal diastolic movements and sounds in a patient with ischaemic heart disease. The rapid filling wave is attenuated while the 'a' wave is exaggerated and is accompanied by a loud atrial sound (4).

tions do not support the widely held view (Kincaid-Smith and Barlow, 1959a) that the first component is an audible vibration which is retained when the atrium contracts against a closed mitral valve and that the second component corresponds with the atrial sound of sinus rhythm.

Methods of recording left atrial activity

A variety of techniques has been used to take graphic records of the chest wall vibrations caused by atrial and ventricular activity. The patterns obtained depend upon the aspects of motion to which the transducer is sensitive, its frequency response, time constant, and sensitivity, as well as any modifications created by its electronic circuitry. The techniques include apex cardiography (Marey, 1881), impulse cardiography (Beilin and Mounsey, 1962), kinetocardiography (Eddleman *et al.*, 1953), vibrocardiography (Agress *et al.*, 1961), and accelerocardiography (Rosa *et al.*, 1961). Apex cardiography is the oldest and most widely used of these techniques. By recording the displacement of the apex

beat, it most closely represents the physical signs felt by the fingers, and is well suited to the detection and measurement of the atrial impulse ('a' wave). A commonly used device is a cup connected to a piezoelectric device. The disadvantages of this system include the relatively large area covered by the cup, the unsuitability of most crystals to record very low frequency movements, a relatively short time constant, and dependence upon an air-filled conducting system. We have preferred an apparatus with a moving probe which is sensitive, specific for displacement, allows a single point to be selected for recording, and incorporates a microphone to pick up sound vibrations from an area around the point (Nixon, Hepburn, and Ikram, 1964). It is easy to use at the bedside and the records obtained are reproducible.

To assess whether a given 'a' wave is normal or abnormal, it is customary to compare its amplitude with the total apical movement, the upper limit of normal being taken as 15 to 20 per cent. There are obvious disadvantages to the comparison of one diastolic filling wave with the sum of three diastolic

FIG. 2 The apex cardiogram (ADC), mitral area low frequency phonocardiogram (MALF), and lead II of the electrocardiogram in a case of complete heart block. The P waves are labelled. The first P wave is followed by an apical 'a' wave and atrial sound (4) with two components. The first component starts 0.14 sec after the onset of the P wave and its peak vibrations coincide with the peak of the apical 'a' wave. The second component starts 0.23 sec after the onset of the P wave. The second P wave is followed by an 'a' wave which coincides with the rapid filling wave to produce a 'summation' sound (S). The third P wave occurs during ventricular systole and is not followed by either component of the atrial sound.

filling waves and a systolic movement. It is simpler and more logical to compare the 'a' wave amplitude with the total diastolic excursion since early and late diastolic filling are interdependent, one waning as the other waxes. Using this technique the upper limit of normal for the 'a' wave is 40 per cent. Pathologically enlarged 'a' waves are usually palpable.

It is less easy to assess the normality or abnormality of presystolic sound vibrations. The phonocardiographic appearance of atrial sound vibrations is not a reliable guide either to whether the sound is audible or whether it represents a physiological or pathological event. There is great need for standardization of methods for recording heart sounds and pulsations, so that comparable records can be produced from different centres and the problem of normal limits resolved, as has been done for the electrocardiogram.

Genesis of the atrial sound

The atrial sound coincides with the peak of outward movement of the left ventricular apex which is caused by atrial systole—it marks the end of a rapid filling phase when the apex and the mitral annulus are carried furthest apart. It is reasonable to suggest that sudden tension of the mitral cusps and chordae tendineae at this time causes vibrations heard as the atrial sound. This hypothesis is supported by the fact that the sound is amplified by manoeuvres which enhance atrial transport such as hypervolaemia and exercise; and myocardial diseases which reduce early diastolic filling of the ventricle.

The involvement of the mitral apparatus in the genesis of diastolic sound has been studied more fully in the case of the third heart sound (Dock, Grandell, and Taubman, 1955), and it seems likely that the third sound and atrial sound have a common origin. They have similar frequency character-

istics, both are related to rapid filling periods of the ventricle, and both have maximum vibrations at the peak of an outward movement of the ventricular apex. When they occur in disease they appear to represent exaggerations of normal physiological events. An illustration of the involvement of the mitral valve apparatus in the production of diastolic movement and sound is provided by mitral incompetence. In rheumatic cases, where the cusps and chordae are present, the rapid filling peak and third sound are prominent (Nixon, 1961), whereas in severe regurgitation round a prosthetic mitral valve, the filling peak and third sound are absent or rudimentary.

Factors influencing timing and audibility of the atrial sound

A variety of factors determines whether the atrial sound can be heard by the trained observer. Perhaps the most important of these is the time interval between the atrial sound and the first sound (Spodick *et al.*, 1971). This depends upon the PR interval, the interval between the P wave and the atrial sound, and the interval between the Q wave and the first sound.

In first-degree heart block the atrial sound is often audible and at certain heart rates it will summate with the protodiastolic third sound to create a 'summation gallop'.

The interval between the P wave and the atrial sound shortens with deteriorating heart function (Duchosal, 1931-1932). It lengthens with recovery from myocardial infarction (Hill *et al.*, 1969; Bennett, Smithen, and Sowton, 1972), with treatment of hypertension, and with manoeuvres which reduce cardiac filling, such as the application of venous tourniquets and the use of glyceryl trinitrate (Kincaid-Smith and Barlow, 1959b). These types of change tend to make the atrial sound merge with the first sound, to make it inaudible, and to make it appear as an 'atrial component' of the first sound on the phonocardiogram.

The interval between the Q wave and the first sound is lengthened by left ventricular hypertrophy, and so the atrial sound is often well separated from the first sound in hypertension. It is shortened by sympathetic stimulation of the heart as may be seen immediately after myocardial infarction (Jain and Lindahl, 1971).

Other factors which influence the audibility of the atrial sound include its amplitude and its frequency characteristics. Those manoeuvres and changes that shorten the interval between the P wave and the atrial sound also tend to amplify the atrial sound.

FIG. 3 The apex cardiogram (ADC), mitral area low frequency phonocardiogram (MALF), and lead II of the electrocardiogram in a patient with severe heart disease. The 'a' wave is prominent but rounded and an atrial sound is not discernible.

In some cases of long-standing left ventricular disease, the peak of the atrial beat tends to become sharp and the atrial sound may develop high frequency components, particularly in the physically active subject. In some cases with severe heart disease, the atrial beat is early and easily palpable, but rounded: vibrations of the corresponding atrial sound may be of low amplitude and frequency and be inaudible (Fig. 3).

Haemodynamic correlations of the atrial sound

In the normal heart, the pressure-volume relations of the left ventricle are such that relatively large increases in volume are accomplished with little rise in pressure. Most of the filling of the left ventricle is achieved in early diastole and the contribution from left atrial systole is small (Stott *et al.*, 1970). The apex cardiogram reflects this in a large rapid filling wave and a small 'a' wave. In myocardial disease the pressure-volume relation of the left ventricle alters: the ventricle fills less well in early diastole and increases in volume cause greater pres-

sure rises. The reduction in early diastolic filling leaves a larger volume of blood to be moved into the left ventricle by left atrial systole at a time when left ventricular pressure rises steeply with each increment of diastolic volume. The result is a rise in the left ventricular end-diastolic pressure, though the mean left atrial pressure and the left ventricular diastolic pressure before atrial systole remain normal (Cohn *et al.*, 1971). The apex cardiogram reflects this situation with a reduction in the size of the rapid filling wave and enlargement of the 'a' wave, which is usually accompanied by the atrial sound.

Deterioration in heart function tends to exaggerate these changes, but when the heart goes into clinical failure with abnormally high mean left atrial and left ventricular diastolic pressures, the pattern reverses. There is a return of early diastolic rapid filling, frequently accompanied by a pathological third sound ('ventricular gallop'), while the 'a' waves of the left ventricular pressure curve and of the apex cardiogram become less prominent. When the mean left atrial pressure exceeds 20 mmHg, the 'a' wave of the apex cardiogram usually becomes quite small and the atrial sound is lost (Taylor and Nixon, 1972). If the heart rate is fast enough to cause early diastolic rapid filling to coincide with atrial contraction, a summation gallop results, and this rate is often adopted tenaciously by the failing heart.

It is interesting to note that cardiac deterioration causes the interval between the onset of the P wave and the peak of the 'a' wave of left atrial and left ventricular pressure to lengthen, whereas it causes the time from the onset of the P wave to the atrial sound and the peak of the atrial beat to shorten (O'Rourke, 1970). This is because maximum movement of the apex away from the mitral annulus is achieved progressively earlier in left atrial systole as the heart function deteriorates.

Significance of the atrial sound

The significance given to a diastolic sound usually depends upon the diagnosis of the heart condition made by other methods. A third sound in a young athlete is called physiological while a similar sound in a patient with heart failure is called a gallop. In the past many clinicians have regarded any audible atrial sound as pathological. However, the earlier findings of Potain (1875) who believed that the diastolic sounds were exaggerations of normal events and that the atrial sound could be heard in health, are now being confirmed (Spodick *et al.*, 1971; Benchimol and Dessler, 1971). We have, therefore, to distinguish between the physiological and the pathological atrial sound.

In the absence of overt heart disease, the atrial sound may be heard in the following circumstances.

1) *In childhood* Here the circulation is brisk and the chest wall thin. All the detectable diastolic events appear amplified and the pattern of left ventricular filling movement is normal. The atrial sound, when present, is much less prominent than the third sound, and the apex cardiogram shows a normal relation between the rapid filling wave and the 'a' wave.

2) *In hyperdynamic and hypervolaemic states* The overactive circulation of certain emotional states, fever, anaemia, exercise, and pregnancy may so exaggerate the normal diastolic events as to make the atrial sound audible, but if the heart is normal the atrial sound and beat are overshadowed by the third sound and rapid filling wave. If a hyperdynamic state unmasks myocardial disease, an abnormal left ventricular filling pattern with a large 'a' wave and an atrial sound may be found.

3) *In middle age* As middle age advances, the incidence of the atrial sound in apparently normal subjects appears to increase. The increasing incidence may be due to increasing stiffness in the mitral valvar apparatus which causes it to become more productive of noise (Dock, 1959) or to changes in the left ventricular filling pattern. Unlike the sound, the 'a' wave of the apex cardiogram does not increase in amplitude with increasing age in normal subjects (Ginn *et al.*, 1967). The atrial sound, then, is encountered in two different clinical circumstances, either with or without enlargement of the 'a' wave. In some cases where the 'a' wave is normal in size and impalpable, the atrial sound may be a physiological event (Spodick *et al.*, 1971). In others, however, it may owe its presence to a healed myocardial infarction, to the cardiac effect of hypertension, or to covert heart disease. More investigation is needed to clarify the significance of the lone atrial sound, when it is the only questionable sign to be detected in a middle-aged heart.

We believe that an atrial sound associated with an enlarged 'a' wave and a palpable atrial beat is nearly always abnormal, and most frequently caused by hypertensive and/or ischaemic heart disease. Where this is found in subjects without other evidence of heart disease, it can be the consequence of a small healed myocardial infarction after which the electrocardiogram has returned to normal. In others it may be the first evidence of underlying ischaemic heart disease or cardiomyopathy. More study will be needed to determine the significance of the 'lone atrial gallop', and it would be useful to include apex recordings in long-term population studies.

Clinical applications of the atrial sound

Ischaemic heart disease

Acute myocardial infarction The atrial sound, usually accompanied by an enlarged atrial beat, is so consistently present after acute myocardial infarction (Hill *et al.*, 1969; Jain and Lindahl, 1971) that the diagnosis is unlikely in its absence. During the first day or two, when the first and second sounds may be quiet, the atrial sound can be very difficult to detect, presumably because the cardiac output is reduced and the atrium is transporting smaller volumes of blood. As the patient recovers, the first and second sounds become louder, and the atrial sound becomes easier to detect: it may be widely separated from the first sound, particularly when cardiac damage is severe (Krohn and Davis, 1964). As the patient continues to improve, the atrial beat wanes, the interval between the P wave and the atrial sound lengthens, and the atrial sound tends to become incorporated in the first sound (Hill *et al.*, 1969; Bennett *et al.*, 1972). Thus, serial observations of the state of the diastolic heart sounds and movements provide valuable information about heart function after infarction and can be a useful guide to the speed with which the patient should be mobilized. The persistence of an atrial sound after infarction carries a worse prognosis than its disappearance (Kincaid-Smith and Barlow, 1959b) and is more often associated with continuing symptoms (Benchimol and Dimond, 1963). The persistence of the atrial sound in association with a prominent third sound usually indicates a ventricular aneurysm (McGinn, Gould, and Lyon, 1968).

Diagnosis of early ischaemic heart disease

The atrial gallop is a common finding in angina pectoris (Banks and Shugoll, 1967). In cases without previous myocardial infarction it is commoner than an abnormal resting electrocardiogram (Nixon and Bethell, 1972). In some cases the atrial sound and enlarged atrial beat may become apparent only during attacks of angina, or may appear in response to exercise in patients with ischaemic heart disease (Benchimol and Dimond, 1963). The presence of an atrial sound and palpable atrial beat in subjects with 'atypical chest pain', or symptoms consistent with reducing left ventricular function, is a valuable indication of underlying heart disease and should lead to further investigation (Nixon and Bethell, 1971).

Management of chronic ischaemic heart disease In any patient with established ischaemic heart disease, increasing amplitude of the atrial

sound and atrial beat is an indication of deteriorating heart function which often appears before the electrocardiogram worsens, and calls for appropriate treatment.

Hypertension

Potain first postulated that the atrial gallop was associated with arterial hypertension in some cases, though he was unable to measure the blood pressure. It has now been shown that in hypertensive disease, the atrial sound and beat correlate with the level of blood pressure (Kincaid-Smith and Barlow, 1959b) and electrocardiographic evidence of left ventricular hypertrophy (Beilin and Mounsey, 1962). Treatment of the hypertension tends to lengthen the interval between the P wave and the atrial sound which merges with the first sound and is lost (Kincaid-Smith and Barlow, 1959b), though an atrial sound persists in some patients, despite adequate blood pressure control (Grayzel, 1960). It is reasonable to use the presence of an atrial sound and palpable atrial beat as one indication for starting treatment of the hypertensive patient.

Aortic stenosis

In aortic stenosis, an enlarged apical 'a' wave and atrial sound usually signify a peak systolic gradient across the valve of at least 70 mmHg and a left ventricular end-diastolic pressure of more than 11 mmHg (Braunwald *et al.*, 1963; Tavel *et al.*, 1965). The development of these signs of augmented left atrial activity indicates that the left ventricle has become dependent upon atrial contraction for adequate filling. An arrhythmia which removes this atrial transport function may cause sudden death. Thus, the development of a palpable atrial beat and an atrial sound may be useful signs that the time has arrived for surgical treatment to be offered.

References

- Agress, C. M., Fields, L. G., Wegner, S., Wilburne, M., Shickman, M. D., and Muller, R. M. (1961). The normal vibrocardiogram. *American Journal of Cardiology*, **8**, 22.
- Banks, T., and Shugoll, G. I. (1967). Confirmatory physical findings in angina pectoris. *Journal of the American Medical Association*, **200**, 1031.
- Beilin, L., and Mounsey, P. (1962). The left ventricular impulse in hypertensive heart disease. *British Heart Journal*, **24**, 409.
- Benchimol, A., and Desser, K. B. (1971). The fourth heart sound in patients with proven normal hearts (abstract). *Circulation*, **44**, Suppl. 2, 140.
- Benchimol, A., and Dimond, E. G. (1963). The apexcardiogram in normal older subjects and in patients with arteriosclerotic heart disease. Effect of exercise on the 'a' wave. *American Heart Journal*, **65**, 789.

- Bennett, E. D., Smithen, C. R., and Sowton, E. (1972). Significance of atrial sound in acute myocardial infarction (abstract). *British Heart Journal*, **34**, 202.
- Braunwald, E., Goldblatt, A., Aygen, M. M., Rockoff, S. D., and Morrow, A. G. (1963). Congenital aortic stenosis: clinical and haemodynamic findings in 100 patients. *Circulation*, **27**, 426.
- Charcelay, A. (1838). Memoire sur plusieurs cas remarquables de défaut de synchronisme des battements et des bruits des ventricules du coeur. *Archives Générales de Médecine*, 3rd new series, Vol. 3, 393.
- Cohn, P. F., Vokonas, P. S., Williams, R. A., Herman, M. V., and Gorlin, R. (1971). Diastolic heart sounds and filling waves in coronary artery disease. *Circulation*, **44**, 196.
- Crevasse, L., Wheat, M. W., Wilson, J. R., Leeds, R. F., and Taylor, W. J., (1962). The mechanism of the generation of the third and fourth heart sounds. *Circulation*, **25**, 635.
- Dock, W. (1959). The forces needed to evoke sounds from cardiac tissues, and the attenuation of heart sounds. *Circulation*, **19**, 376.
- Dock, W., Grandell, F., and Taubman, F. (1955). The physiologic third heart sound: its mechanism and relation to protodiastolic gallop. *American Heart Journal*, **50**, 449.
- Duchosal, P. (1931-1932). A study of gallop rhythm by a combination of phonocardiographic and electrocardiographic methods. *American Heart Journal*, **7**, 613.
- Eddleman, E. E., Willis, K., Reeves, T. J., and Harrison, T. R. (1953). The kinetocardiogram. I. Method of recording precordial movements. *Circulation*, **8**, 269.
- Ginn, W. M., Sherwin, R. W., Harrison, W. K., and Baker, B. M. (1967). Apex-cardiography: use in coronary heart disease and reproducibility. *American Heart Journal*, **73**, 168.
- Grayzel, J. (1960). Gallop rhythm of the heart. *American Journal of Medicine*, **28**, 578.
- Hill, J. C., O'Rourke, R. A., Lewis, R. P., and McGranahan, G. M. (1969). The diagnostic value of the atrial gallop in acute myocardial infarction. *American Heart Journal*, **78**, 194.
- Jain, S. R., and Lindahl, J. (1971). Apex cardiogram and systolic time intervals in acute myocardial infarction. *British Heart Journal*, **33**, 578.
- Kincaid-Smith, P., and Barlow, J. (1959a). The atrial sound and the atrial component of the first heart sound. *British Heart Journal*, **21**, 470.
- Kincaid-Smith, P., and Barlow, J. (1959b). Atrial sound in hypertension and ischaemic heart disease. *British Heart Journal*, **21**, 479.
- Krohn, B. G., and Davis, W. H. (1964). The apex cardiogram as a prognostic tool in acute ischaemic heart disease. *4th European Congress of Cardiology, Abstracts*, p. 188.
- McGinn, F. X., Gould, L., and Lyon, A. F. (1968). The phonocardiogram and apexcardiogram in patients with ventricular aneurysm. *American Journal of Cardiology*, **21**, 467.
- Marey, E. J. (1881). *La Circulation du Sang a l'Etat Physiologique et dans les Maladies*. Masson, Paris.
- Nixon, P. G. F. (1961). The third heart sound in mitral regurgitation. *British Heart Journal*, **23**, 677.
- Nixon, P. G. F., and Bethell, H. J. N. (1971). Coronary deaths - how unexpected? *British Medical Journal*, **4**, 486.
- Nixon, P. G. F., and Bethell, H. J. N. (1972). Atrial gallop in diagnosis of early coronary heart disease (abstract). *British Heart Journal*, **34**, 202.
- Nixon, P. G. F., Hepburn, F., and Ikram, H. (1964). Simultaneous recording of heart pulses and sounds. *British Medical Journal*, **1**, 1169.
- Orias, O., and Braun-Menendez, E. (1939). *The Heart Sounds in Normal and Pathological Conditions*. Oxford University Press, London.
- O'Rourke, R. A. (1970). The atrial sound. Factors regulating its occurrence and timing. *American Heart Journal*, **80**, 715.
- Potain, P. C. E. (1875). Du rythme cardiaque appelé bruit de galop, de son mécanisme et de sa valeur séméiologique. *Bulletin et Mémoires de la Société Médicale des Hôpitaux de Paris*, **12**, 137. (Quoted by V. A. McKusick in *Cardiovascular Sound in Health and Disease* (1958). Williams and Wilkins, Baltimore.)
- Potain, P. C. E. (1894). *Clinique Médicale de la Charité*. Masson, Paris.
- Rosa, L. M., Constantino, J. P., Reich, R., Karsak, N., and Zezmer, B. (1961). The precordial accelerogram in normal subjects and noncardiac patients. *Experimental Medicine and Surgery*, **19**, 207.
- Spodick, D. H., Rectra, E., Khan, A., and Pigott, V. (1971). Audibility of the fourth heart sound: a prospective, blinded investigation (abstract). *Circulation*, **44**, Suppl. 2, 33.
- Stott, D. K., Marpole, D. G. F., Bristow, J. D., Kloster, F. E., and Griswold, H. E. (1970). The role of left atrial transport in aortic and mitral stenosis. *Circulation*, **41**, 1031.
- Tavel, M. E., Campbell, R. W., Feigenbaum, H., and Steinmetz, E. F. (1965). The apex cardiogram and its relationship to haemodynamic events within the left heart. *British Heart Journal*, **27**, 829.
- Taylor, D. J. E., and Nixon, P. G. F. (1972). Assessment of left ventricular function after myocardial infarction. *British Heart Journal*, **34**, 905.

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