SOME OBSERVATIONS ON THE ATRIAL SOUND*

JOHN B. BARLOW, M.B., B.CH., M.R.C.P., *Department of Medicine, University of the Witwatersrand, Johannesburg*

The atrial sound, also known as the fourth heart sound or presystolic gallop, has been defined[7] as an audible vibration occurring more than 0·07 seconds after the beginning of the P wave of the simultaneous electrocardiogram (ECG) but preceding the onset of the QRS complex. It has been shown[7] that, unrelated to any alteration in the P-R interval, the position of the atrial sound varies in relation to the first heart sound and to the simultaneous ECG.

It is the purpose of this paper to demonstrate this variation in the timing of the atrial sound and to discuss some of the clinical implications resulting from this variability.

METHODS AND OBSERVATIONS

Low-frequency phonocardiographic (PCG) tracings on an apparatus similar to that recommended by Leatham[9] were used throughout. Standard lead II was used for the simultaneous ECGs. In order to determine the position of the atrial sound, the distance from the beginning of the P wave to the commencement of the atrial sound was used. This measurement was suggested by Duchosal[3] and is referred to as the P-G interval (G=gallop).

Details of the variation in timing of the atrial sound are described more fully elsewhere.[7] For the purpose of this paper they are briefly referred to here as follows:

1. *Effect of clinical improvement and deterioration.* Serial PCGs on patients with ischaemic or hypertensive heart disease have shown that with clinical improvement the P-G interval increases so that the atrial sound approaches the first heart sound and may eventually fuse with it (Fig. 1). Likewise, clinical deterioration results in a decrease of the P-G interval, the atrial sound occurring earlier in the cardiac cycle and consequently further away from the first heart sound (Fig. 10). These changes may occur in a few minutes or may take many weeks or months, depending on the time and extent of the clinical improvement or deterioration. For example, the atrial sound took about 4 weeks to merge with the first heart sound in a man recovering from a severe myocardial infarction (Fig. 1), whereas in another patient an attack of ischaemic pain at rest caused the atrial sound to move in a few minutes from a position in which it was fused with the major components of the first sound to one considerably earlier in the cardiac cycle (Fig. 6). A similar movement of the atrial sound has been shown to occur in severe hypertensive patients when the blood pressure is rapidly reduced by the administration of intravenous hexamethonium. As the blood pressure is allowed to rise again the P-G interval shortens and the atrial sound returns to its original position (Fig. 2).

2. *Effect of rest and emotion.* Some movement of the atrial sound towards the first heart sound, usually not more than 0·02 or 0·03 seconds, has frequently been noted in patients with hypertensive or ischaemic heart disease after rest lasting from a few minutes to an hour (Fig. 3). Emotional factors probably play a large part in this; the P-G interval may quickly shorten again on the introduction of an intravenous needle or similar procedure.

3. *Effect of respiration.* Whereas respiration produces no effect on the P-G interval in patients with hypertension or ischaemia, the atrial sound of cor pulmonale both increases in intensity and occurs earlier in the cardiac cycle during inspiration as compared with expiration (Fig. 4).

4. *Effect of various procedures.* It has already been mentioned that the intravenous administration of hypotensive drugs will increase the P-G interval in hypertensive patients. The inhalation of amyl nitrite has a similar effect in hypertension (Fig. 5), though a change in the P-G interval is seldom seen in cases with ischaemic heart disease following inhalation of this drug. Venous cuffing will prolong the P-G interval in both ischaemia and hypertension, the atrial sound returning to its previous position a few seconds after the cuffs are released.[7] Carotid-sinus pressure will often diminish the intensity of an atrial sound.[14] If the P-R interval is prolonged by this manoeuvre the atrial sound is obviously more widely separated from the first heart sound. The P-G interval, however, remains virtually unchanged.

5. *Effect of movement of atrial sound on the first heart sound.* The major components of the first sound, which are probably due to mitral and tricuspid valve closure,[10, 15] become louder as the P-G interval lengthens. This is best illustrated when the atrial sound approaches the first heart sound with rest (Fig. 3), for other factors, such as the specific effect of amyl nitrite, which invariably increases the intensity of the first sound,[1] or the possible increased intensity of the first sound due to a tachycardia associated with the hypotensive effect of hexamethonium, are then excluded. The change in P-G interval due to an alteration of the P-wave shape, but with the same heart rate and P-R interval, confirms this effect on the intensity of the major components of the first sound (Fig. 7).

DISCUSSION

In severe cases of ischaemic or hypertensive heart disease the P-G interval may be as short as 0·08 seconds. With improvement in the cardiac state, or lowering of the blood pressure in hypertensive patients, the P-G interval may increase to 0·20 seconds or more — depending to an extent on the length of the P-R interval. At this stage the atrial sound may have fused with the first heart sound and will then occur after the Q of the simultaneous ECG (Fig. 1). It is now indistinguishable, both clinically and by PCG, from a 'split' first sound in normal subjects in whom the earlier component is low pitched (Fig. 8). When an atrial sound has moved to this position, in which it occurs after the Q, it is, by definition, no longer an atrial sound and is better referred to as an atrial component of the first heart sound.[6]

The movement of the atrial sound can usually be appreciated on clinical auscultation. When the P-G interval is short and the atrial sound consequently widely separated from the first heart sound, the cadence is one of gallop rhythm, whereas, with improvement and thus lengthening

* Paper presented at the 42nd South African Medical Congress (M.A.S.A.), East London, C.P., September-October 1959.

of the P-G interval, the atrial sound approximates to the first sound and the cadence resembles that of splitting of the first heart sound.

It will be apparent, however, that the length of the P-R interval will also affect the position of the atrial sound in relation to the first heart sound and consequently may alter the cadence on clinical auscultation. In one study of the heart sounds in hypertension,[2] an atrial sound was present in at least 50% of cases and the average P-G interval for the series was 0·145 seconds. It is obvious that in any one patient in that group with a P-R interval of, say, 0·20 seconds, the atrial sound would occur earlier in relation to the first heart sound than in another patient with the same P-G interval but a P-R interval of, say, 0·15 seconds. In the first patient the cadence would be that of gallop rhythm, whereas in the second the atrial sound would be closer to the first heart sound and the cadence would be more like that of a 'split' first sound.

Weitzman[16] observed that the atrial sound, as shown on a PCG, often consists of an early 'inaudible' component and a second main, or 'audible', component. The inaudible component occurs 0·08 - 0·14 seconds after the beginning of the P wave[16, 7] and is unaffected by any change in clinical state or by the procedures mentioned above which alter the P-G interval. It may also be recorded on a PCG in normal patients. Weitzman[16] considered that this inaudible component was caused by the actual muscular contraction of the atrium. It certainly cannot be related to ventricular filling, which is probably the cause of the main audible component, since it can be recorded with atrial contraction during ventricular systole in patients with complete heart block (Fig. 9), when ventricular filling would clearly be impossible. It is to the second, and audible, component of the atrial sound to which all the above observations refer. This sound is never heard or recorded in a normal heart with a normal P-R interval. In, however, a heart otherwise normal but with a prolonged P-R interval, an atrial sound may be clinically audible and the P-G interval will then be anything from 0·21 to 0·28 seconds.[7] A similar long P-G interval is also seen when atrial contraction occurs in early diastole in patients with complete heart block. An atrial sound due to first-degree heart block in an otherwise normal heart is not moved by the inhalation of amyl nitrite or the application of venous tourniquets.[7]

An approximate estimation of the P-G interval can be made on clinical auscultation just as an experienced observer can assess whether the opening snap of mitral stenosis is occurring early or late. However, it will be apparent from the above that any clinical assessment of the P-G interval is liable to be hopelessly inaccurate unless the P-R interval is known. Clinical assessment of the P-G interval must depend on estimating, by the cadence of the sounds, the distance between the atrial sound and the first heart sound. This interval, however, could theoretically be the same in a patient with a normal heart except for first-degree heart block, a patient with a P-R interval of about 0·20 seconds with a moderate P-G interval, and a patient with a P-R interval of 0·14 seconds but with severe cardiac decompensation and consequently a short P-G interval (Fig. 11). An approximate clinical estimation of the P-R interval can sometimes be made, provided no valvular disease is present, by judging the intensity of the first heart sound. But it has already been mentioned that the position of the atrial sound in relation to the first heart sound has an effect on the intensity of the latter (Figs. 3 and 7) and thus this method must inevitably be very inaccurate. A recent ECG, however, is usually available; so when the P-R interval is known, some assessment of the P-G interval can be made on clinical auscultation. A subsequent change in P-G interval, following improvement or deterioration in cardiac function, can then usually be elicited without difficulty.

The decrease in the P-G interval during inspiration which is seen in patients with atrial sounds originating from the right side of the heart (Fig. 4), suggests that the increased venous return during this phase of respiration places an added strain on these chambers. A right atrial sound is best heard over the lower sternal area or in the epigastrium and will obviously be more easily audible during deep inspiration. In this phase of respiration it is both louder and more widely separated from the first heart sound. On some occasions a right atrial sound is audible only during deep inspiration, while with normal respiration it might be confused with a 'split' first sound or be completely inaudible. The movement of a right atrial sound with respiration, the P-G interval of a left atrial sound remaining unchanged, may sometimes be a help in determining from which ventricle a gallop is originating.

The exact mechanism of the atrial sound is still uncertain. It often occurs too late in relation to the P wave to result from the actual muscular contraction of the atrium and, in any event, the sound is too loud for this to be likely. However, the muscular contraction could well be the cause of the early 'inaudible' component which is sometimes recorded on a PCG.[16] Potain[13] postulated that the atrial sound resulted from sudden tension of the ventricular wall following the inflow of blood in atrial systole. Kuo *et al.*,[8] by recording simultaneous atrial and ventricular pressures, showed that the atrial pressure is higher than the ventricular at the time that the atrial sound occurs. It seems improbable, therefore, that the atrial sound can be attributed to closure of the atrioventricular valves — the theory originally suggested by Lewis[11] and still favoured by some.[4]

It seems likely that the atrial sound is associated with ventricular filling resulting from atrial systole, but that resistance to this ventricular filling is increased. The actual sound heard could well, as suggested by Potain,[13] be caused by a sudden rise in tension of the ventricular wall. In patients in heart failure, the early atrial sound could then be due to a raised end-diastolic pressure favouring a rapid rise, after the atrial contraction, to the tension necessary to produce the sound. Clinical improvement, the application of venous tourniquets, or the lowering of the blood pressure, could then be expected to delay the atrial sound by lowering the end-diastolic pressure in the ventricle.

The different effects of amyl nitrite on the P-G intervals in hypertensive and ischaemic heart disease can possibly also be explained on this mechanism. Amyl nitirite causes a temporary decrease in peripheral resistance and hence a fall in blood pressure. This may be sufficient reason to account for the prolongation of the P-G interval in hyper-

tensive patients (Fig. 5). In ischaemic heart disease, however, the P-G interval is usually unchanged.[7] Possibly in this condition the temporary drop in peripheral resistance does not result in the ventricular end-diastolic pressure falling, and hence prolongation of the P-G interval, because the ventricular muscle is itself inadequate and the decreased peripheral resistance makes little or no difference to the inefficient contraction of the ventricle.

The significance of an atrial sound in any particular patient can be assessed only in relation to the nature of the underlying heart disease and the length of the P-G interval. An atrial sound in a patient with first-degree heart block and a P-G of not less than 0·21 seconds is of little significance. In such a case, of course, the cause of the heart block must, if possible, be ascertained and the prognosis assessed accordingly. In a series of hypertensive patients it was noted that only when the P-G interval was short (0·08 - 0·12 seconds) were moderate or severe cardiac symptoms present.[3] Hypertensive patients with mild cardiac symptoms or none, usually had a P-G interval of 0·15 - 0·20 seconds. Miles,[12] Weitzman[16] and Evans[5] have all agreed that atrial sounds occur in asymptomatic hypertensive patients, but it seems probable that the P-G interval is seldom less than 0·15 seconds in this group. Thus, if the blood pressure can be maintained at a level at which the P-G interval is long, then the prognosis as far as the heart is concerned is probably good. The presence of an atrial sound in normotensive patients with ischaemic heart disease is probably of more serious significance. In this condition it seems likely that the atrial sound implies inadequate ventricular function, and that, even with a relatively long P-G interval (0·15 - 0·20 seconds), symptoms of cardiac decompensation are likely to exist.

SUMMARY

The atrial sound is an audible vibration dependent on atrial contraction, which on a phonocardiogram (PCG) occurs more than 0·07 seconds after the beginning of the P wave of the simultaneous electrocardiogram (ECG) but precedes the onset of the QRS complex.

The position of the atrial sound is shown to vary in relation to the first heart sound and to the simultaneous ECG. This movement of the atrial sound, which is unrelated to any alteration in the P-R interval, has been studied in patients with hypertension, ischaemic heart disease, and cor pulmonale.

With improvement in cardiac function, the atrial sound approaches the first heart sound and may eventually fuse with it. When an atrial sound has moved towards the major components of the first sound to the extent in which it occurs after the Q wave of the simultaneous ECG, it is referred to as an atrial component of the first heart sound. In this position, it is clinically and by PCG indistinguishable from a 'split' first sound in normal patients in whom the first component of the 'split' is low pitched.

Some factors which cause movement of the atrial sound are described and discussed. These include clinical improvement and deterioration, rest and emotion, hypotensive drugs, the application of venous tourniquets, amyl nitrite inhalation, and respiration in patients with right atrial sounds in cor pulmonale.

The exact mechanism of the atrial sound is still uncertain. There is often an early 'inaudible' component, recorded on a PCG, which is possibly due to the actual muscular contraction of the atrium. The main 'audible' component probably results from increased resistance to ventricular filling at the time of atrial systole.

The significance of an atrial sound depends on both its position and the nature of the underlying heart disease. A clinical assessment of the position of the atrial sound can be made provided the P-R interval is known. It is thought that the presence of an atrial sound in ischaemic heart disease is of more serious significance than an atrial sound in a similar position in hypertensive heart disease.

I am grateful to the Editor of the *British Heart Journal* for permission to publish Figs. 1-10 inclusive.

REFERENCES

1. Barlow, J. B. and Shillingford, J. (1958): Brit. Heart J. **20**, 162.
2. Barlow, J. B. and Kincaid-Smith, P. S. (1960): *Ibid.*, In press.
3. Duchosal, P. (1935): Arch. Mal. Cœur, **28**, 345.
4. Editorial (1957): Circulation, **15**, 321.
5. Evans, W. (1957): Lancet, **2**, 53.
6. Kincaid-Smith, P. S. and Barlow, J. B. (1959): Brit. Heart J., **21**, 470.
7. *Idem* (1959): *Ibid.*, **21**, 479.
8. Kuo, P. T., Schnabel, T. G., Blakemore, W. S. and Whereat, A. F. (1957): J. Clin. Invest., **36**, 1035.
9. Leatham, A. (1952): Brit. Med. Bull., **8**, 333.
10. *Idem* (1954): Lancet, **2**, 607.
11. Lewis, T. (1915): *Lectures on the Heart*. London: Shaw and Sons.
12. Miles, B. E. (1951): Brit. Heart J., **13**, 327.
13. Potain, P. C. (1900): Sem. méd. (Paris), **20**, 175.
14. Read, J. L. and Porter, W. B. (1955): Amer. J. Med., **19**, 177.
15. Reinhold, J. and Rudhe, V. (1957): Brit. Heart J., **19**, 473.
16. Weitzman, D. (1955): *Ibid.*, **17**, 70.

FIGURES 1 - 11

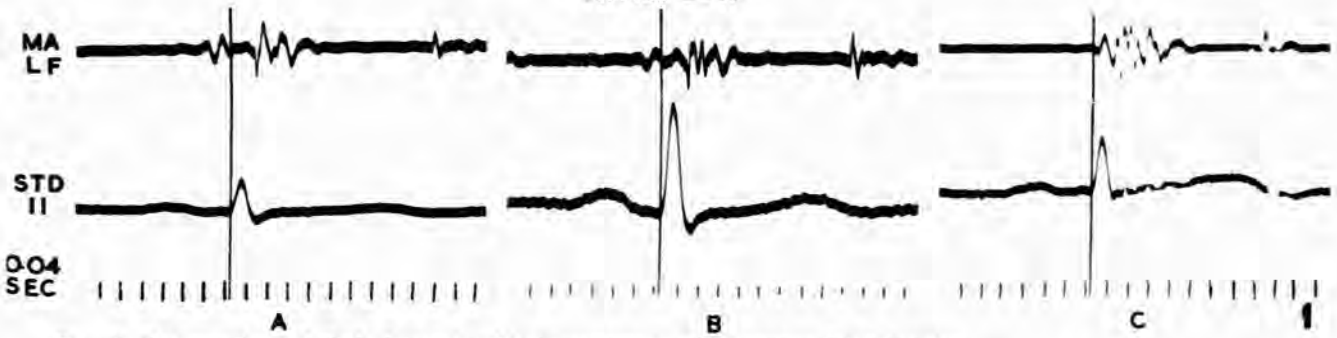

Fig. 1. Prolongation of P-G interval with improvement after myocardial infarction.
A. Phonocardiogram performed within 1 week of myocardial infarction. P-R interval 0·17 sec. P-G interval 0·10 sec.
B. Phonocardiogram about 2 weeks later. P-R interval 0·17 sec. P-G interval 0·13 sec.
C. Phonocardiogram about 5 weeks after myocardial infarction. P-R interval 0·17 sec. P-G interval 0·17 sec. Atrial sound no longer precedes Q wave of ECG and is now an 'atrial component of first heart sound'—see text. In this position it is very similar to a normal heart—compare Fig. 9.
In this and other figures, the line drawn through the Q wave serves to illustrate the movement of the atrial sound.

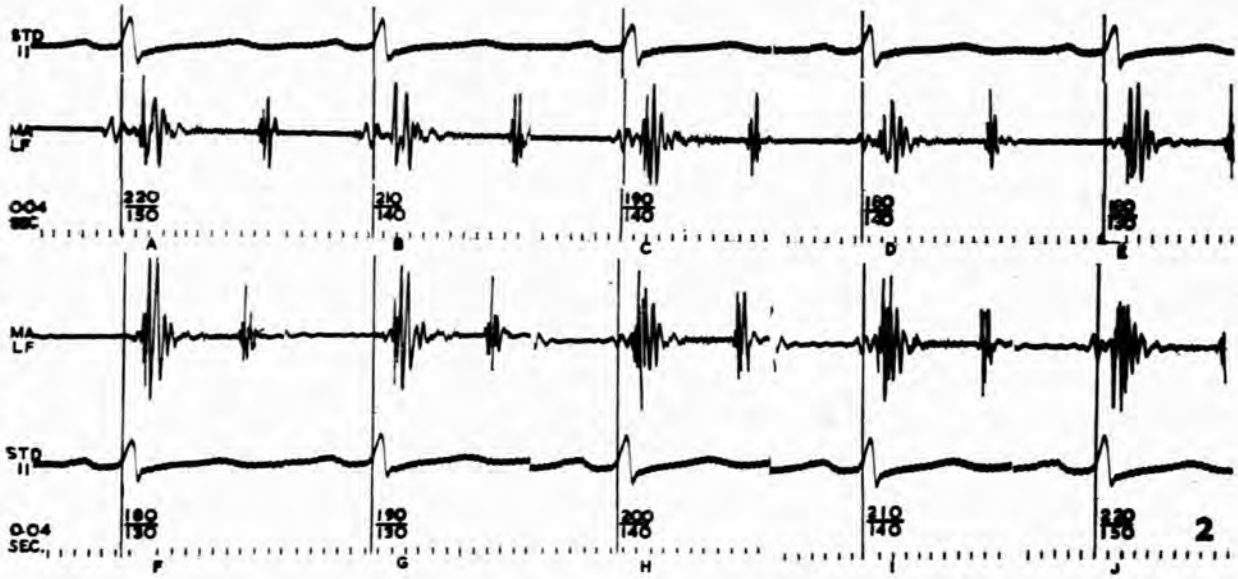

Fig. 2. Prolongation of P-G interval following intravenous hexamethonium in hypertensive patient. P-G shortens again when blood pressure is allowed to rise. Note that in tracings E, F and G, the atrial sound has become an atrial component of the first heart sound.

Fig. 3. Prolongation of P-G interval during rest for a period of about 1 hour. The increased intensity of the first heart sound as the P-G lengthens is clearly shown. The P-R interval is unchanged throughout.

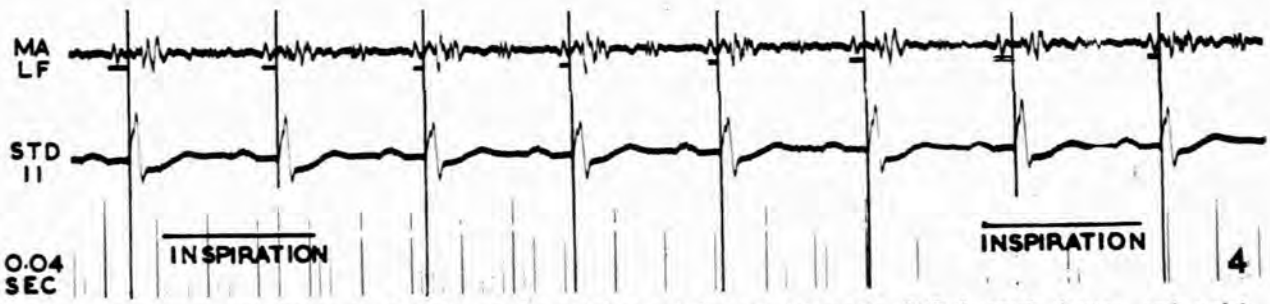

Fig. 4. Movement and change in intensity of the atrial sound in cor pulmonale. P-G interval shorter, and atrial sound louder, during inspiration, compared with expiration. The position of the atrial sound is readily seen by comparing its relationship to the line drawn through the Q of the ECG.

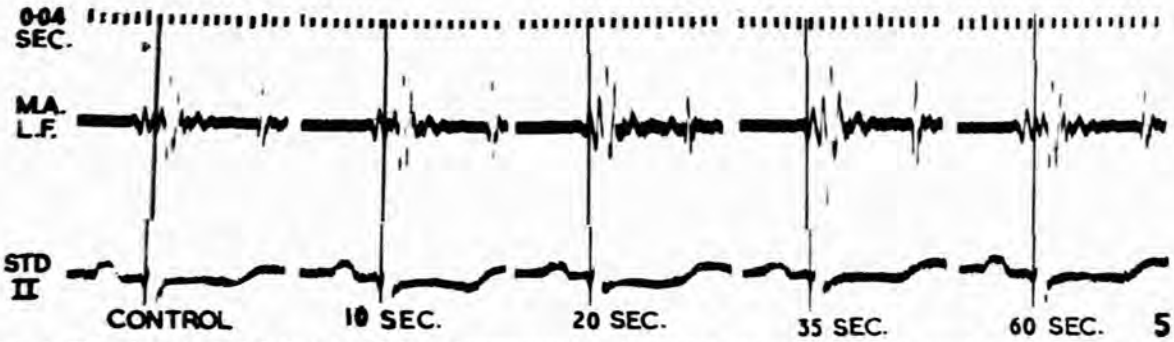

Fig. 5. The alteration of P-G interval in a hypertensive patient after the inhalation of amyl nitrite. Note also the increased intensity of the first heart sound in tracings taken 20 and 35 seconds after inhalation of the drug.

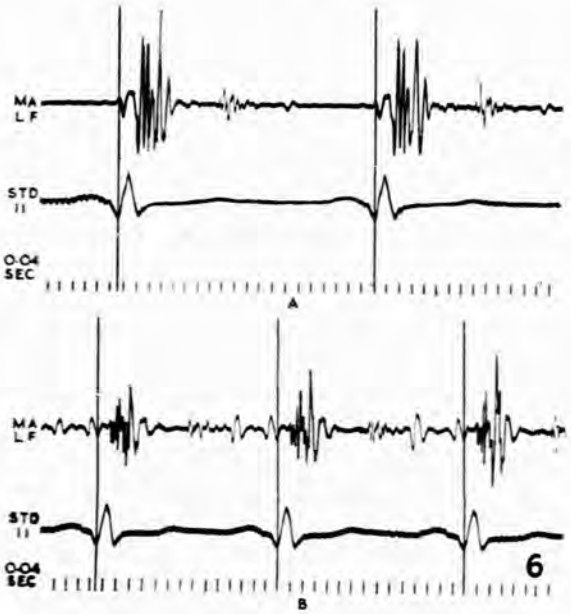

Fig. 6. Patient with ischaemic heart disease; showing shortening of P-G interval during an attack of angina at rest. (A) Before pain. (B) During pain. In both tracings a third heart sound is also present.

Fig. 7. Alteration in P-wave shape, but with same P-R interval. The first heart sound is louder in the complex on the right, where the atrial sound is delayed (i.e. prolonged P-G interval).

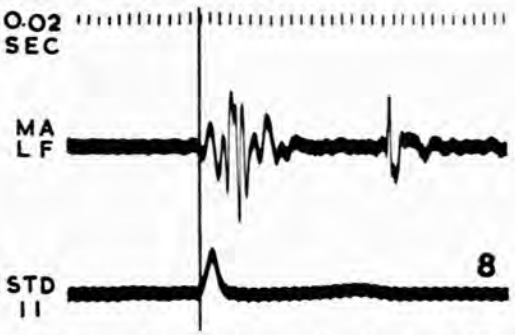

Fig. 8. Phonocardiogram of normal patient aged 30 years, with 'split' first heart sound in which the earlier component is low pitched. The beginning of this low pitched component is seen to be coincidental with the Q of the ECG. (Compare with Fig. 1 C). The P wave is poorly demonstrated but P-R interval was about 0·18 sec.

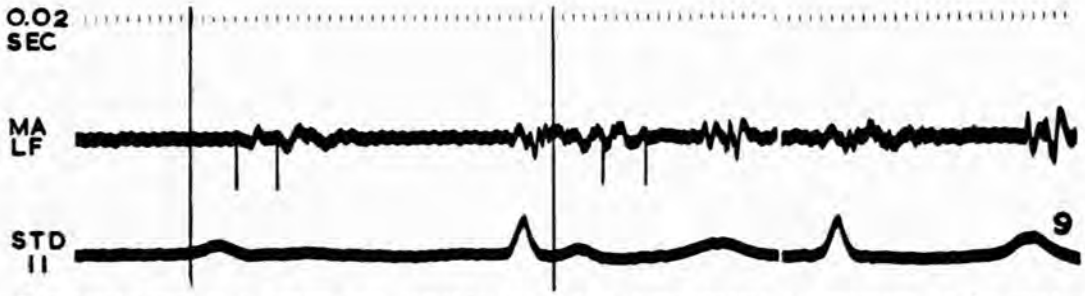

Fig. 9. Phonocardiogram in a patient with complete heart block demonstrating 2 components of the atrial sound. A long vertical line is drawn through the beginning of the P wave and the short vertical lines show the commencement of the two components of the atrial sound. It can be seen that the second component disappears when atrial contraction occurs during ventricular systole. The tracing on the right shows a flat base line between the first and second heart sounds when no P wave is present.

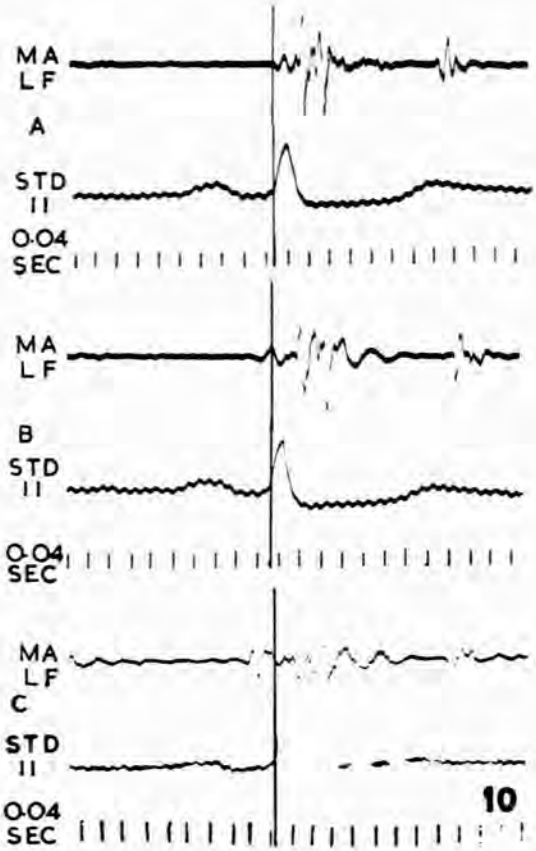

Fig. 10. Shortening of P-G interval over a period of several months in a hypertensive patient who refused therapy. (A) P-R=0·17 sec.; P-G=0·17 sec. (B) P-R=0·17 sec.; P-G=0·15 sec. (C) P-R=0·17 sec.; P-G=0·11 sec.

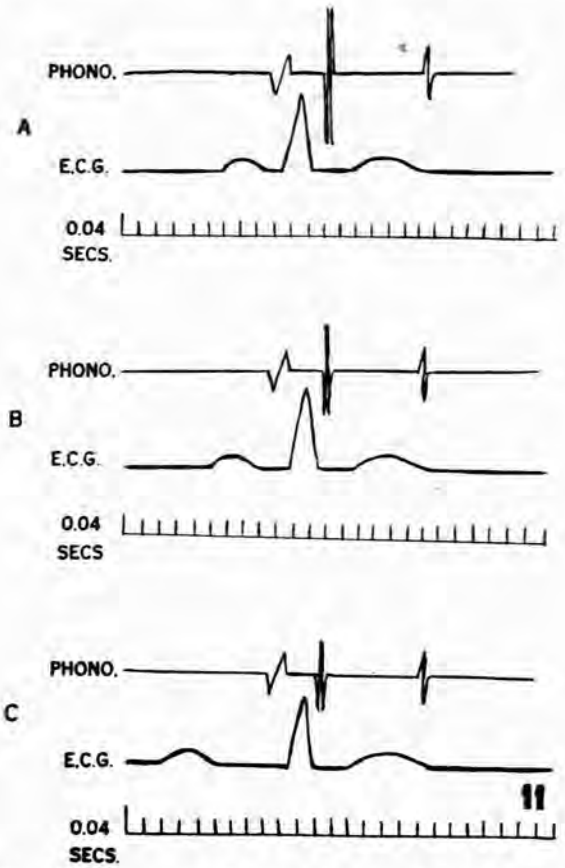

Fig. 11. Diagram to illustrate the necessity of knowing the P-R interval before the P-G interval can be clinically assessed. In the three examples shown, the atrial sound always precedes the first heart sound by 0·14 seconds. (A) P-R 0·14 sec.; P-G 0·10 sec. (B) P-R 0·20 sec.; P-G 0·16 sec. (C) P-R 0·30 sec.; P-G 0·26 sec. The intensity of the first heart sound has been decreased in (B) and (C) on account of the prolongation of the P-R interval.