Impulse Cardiogram in Early Diagnosis of Left Ventricular Dysfunction in Hypertension

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An abnormal apical impulse has long been recognized in hypertensive cardiac disease. Beilin and Mounsey (1962) described in great detail the changes in the apical impulse occurring in a series of 25 hypertensive patients who had been brought into hospital with a view to treatment with hypotensive drugs. They showed that with the development of hypertensive cardiac disease, as demonstrated by the electrocardiogram and x-ray, there was an abnormal prolongation of the apical impulse up to, or beyond, the second heart sound. They also stated that patients with borderline hypertrophy showed inconstant changes in the apical impulse while those without hypertrophy (as shown on the electrocardiogram and chest x-ray) had an entirely normal impulse.

The present paper is an extension of this work to determine the value of the apical impulse in determining changes in left ventricular function at an earlier stage than indicated either by the electrocardiogram or chest x-ray.

SUBJECTS AND METHODS

Studies were made on 47 patients (27 men and 20 women) with hypertension, whose ages ranged from 25 to 68 years, with an average of 51. Their height and weight were recorded and note was also made of their general physique. Four of them were acromegalic and another three had ischaemic heart disease as well. Their range of blood pressure was always measured at the time of recording the cardiac impulse. Careful clinical assessment of the position, amplitude, and form of the apical impulse was made. The apical impulse was then recorded with the impulse recorder, using a simultaneous phonocardiogram at the pulmonary area and an electrocardiogram as reference tracings (Beilin and Mounsey, 1962). Electrocardiogram and postero-anterior and lateral chest x-rays were made in all cases.

The criteria of a normal or abnormal impulse cardiogram were those of Beilin and Mounsey (1962) and Nagle et al. (1966). The analysis of each impulse cardiogram was done before examining the electrocardiogram and chest x-ray of the patient, and included the height of the atrial contraction wave or atrial beat and the height and duration of the ejection beat.

According to the duration of the ejection beat the patients were classified into 3 groups. In the first group hypertensive patients with a normal ejection beat lasting for up to two-thirds of systole were included (Fig. 1). The second group consisted of the patients with a sustained ejection beat, which extended through the last third of systole up to the second sound, and the third group those cases with very prolonged ejection beat which extended beyond the second sound.

Subsequently, the electrocardiograms of each of those groups were analysed to detect signs of left ventricular hypertrophy and left atrial hypertrophy. The electrocardiographic criteria of left ventricular hypertrophy used were those of Sokolow and Lyon (1949) and Goldberger (1953) summarized by Simpson (1960). On the chest x-ray the general form of the heart and the cardiothoracic ratio were examined. The following correlations were made:

(1) The relation of the duration of the ejection beat of the apical impulse with the electrocardiographic evidence of left ventricular hypertrophy. (2) The relation of the duration of the ejection beat on the impulse cardiogram with the left ventricular enlargement on the chest x-ray. (3) The relation of the height of the atrial beat on the impulse cardiogram with changes of the P wave on the electrocardiogram. (4) The height of the ejection beat with changes in the electrocardiogram and heart size on the chest x-ray.

RESULTS

Of the 47 patients with hypertension studied, 10 (21%) belonged to group 1 with a normal ejection beat on the impulse cardiogram, 8 (17%) to group 2

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with a sustained ejection beat which extended beyond the first two-thirds of systole up to the second sound, and 29 patients (62%) to group 3 with an ejection beat extending beyond the second sound (Table I).

The electrocardiogram was normal in 6 of the 10 patients of group 1. The remaining 4 patients had minimal non-specific T wave changes and only one of them presented with a tall R wave (+) as well. In group 2 the electrocardiogram was abnormal in all the 8 patients, though in 3 of them it showed only minimal non-specific T wave changes. In group 3 which included the 29 patients with a long sustained ejection beat, the electrocardiogram was normal in 5 patients and abnormal in 24 patients (Fig. 2). Of the last 24 cases, 5 showed only minimal non-specific T wave changes on an otherwise normal electrocardiogram, and 12 patients presented with the combined signs of left ventricular hypertrophy, namely high R wave voltage, depressed S–T segment, and T inversion in the left ventricular leads.

The following was the relation between the duration of the ejection beat and the radiological appearance of the left ventricle. In group 1 with a normal ejection beat, 7 patients had a normal heart size and
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Fig. 1.—Normal impulse cardiogram in a hypertensive man aged 26. The atrial beat is not present, whereas the ejection beat occupies less than the first two-thirds of systole. PRE-EB = Pre-ejection beat.

form, and 3 showed left ventricular enlargement. In group 2, 5 had a normal heart size and 3 had left ventricular enlargement, whereas of the 29 patients of group 3, 15 had a normal chest x-ray and 14 an enlarged left ventricle. In other words, whereas the ejection beat was abnormal in 79 per cent of the patients studied, the chest x-ray showed evidence of left ventricular enlargement in only 42 per cent.

The combined study of the atrial beat in the impulse cardiogram and the P wave on the electrocardiogram showed that 21 out of 25 patients with a normal atrial beat (height less than 2 mm.) had a normal P wave and 4 had the pattern of left atrial hypertrophy. From the remaining 22 patients with an abnormal atrial beat, the P wave was normal in 16 patients and abnormal (compatible with left atrial hypertrophy) in only 6 patients. In other words, the atrial beat was found abnormal in 48 per cent of the patients studied, whereas the electrocardiographic evidence of left atrial hypertrophy was found in 21 per cent.

The relation of the amplitude of the ejection beat in the impulse cardiogram and electrocardiographic and radiological signs of left ventricular hypertrophy were studied (Table II). Of 25 patients showing an amplitude of the ejection beat ranging from 1 to 10 mm., 19 (76%) presented with electrocardiographic evidence of left ventricular hypertrophy and 12 (48%) with evidence of left ventricular enlargement on the chest x-ray. Of 15 patients

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Fig. 2.—Abnormal impulse cardiogram in young hypertensive woman aged 32 with chest x-ray and electrocardiogram reported normal (shown above). The ejection beat, high and grossly sustained, is extended beyond the second sound; the atrial beat is 4 mm. in height, suggesting left atrial hypertrophy.
with an ejection beat of 11 to 20 mm. high, 9 (69%) had signs of left ventricular hypertrophy in the electrocardiogram and 5 (33%) had signs of left ventricular enlargement in the chest x-ray. Finally, of 9 patients with a giant ejection beat (20 to 82 mm.), 7 (77%) had an abnormal electrocardiogram and only 3 (33%) had x-ray evidence of left ventricular enlargement. These results suggest that the so-called forceful or high amplitude apical impulse may not indicate left ventricular hypertrophy or enlargement.

The impulse cardiogram was examined in 23 hypertensive patients after treatment from 3 to 6 months. A considerable reduction of the blood pressure was recorded in 12 patients. Of the 23, 6 showed a shorter ejection beat on the impulse cardiogram, 4 had a more sustained and 13 an unchanged ejection beat, showing that the apical impulse does not closely follow the long-term decrease or increase of the arterial blood pressure. It tends to remain sustained even when the electrocardiogram and chest x-ray appear to be considerably improved after treatment. This is illustrated by the following case (Fig. 3).

A married woman aged 26 was admitted to the Hammersmith Hospital in December 1967 for investigation of hypertension. The blood pressure was 180/120 mm. Hg, the electrocardiogram showed only inverted T waves in the leads over the left ventricle, the chest x-ray showed a normal heart size but some deformity in the contour of the left ventricle, whereas the impulse cardiogram was abnormal with an ejection beat extending beyond the second sound and with a large atrial component (4 mm.). After a three-month period of hypotensive treatment, the blood pressure was measured and found to be 160/105 mm. Hg, the electrocardiogram was nearly normal, with the T wave upright, and the cardiac size smaller on x-ray. The impulse cardiogram remained grossly abnormal as far as the sustained ejection beat was concerned and only the atrial beat returned to normal levels (height of atrial beat less than 2 mm.).

**DISCUSSION**

This study has shown that the impulse cardiogram can disclose abnormalities of the heart consistent with left ventricular hypertrophy in hypertensive patients at a stage when the electrocardiogram and chest x-ray are normal. Of 37 hypertensive patients with sustained cardiac impulse suggestive of left ventricular disorder, 5 had normal electrocardiograms, and another 8 showed only minimal non-specific T wave changes. Of the same group of 37 patients with a prolonged ejection beat in the impulse cardiogram, 20 were found to have a normal heart size on x-ray examination and the remaining 17 patients an increased cardiothoracic ratio.

These findings underline the important place that the examination of the cardiac impulse may have in the early diagnosis of hypertensive heart disease. This is supported by the results which we had from 23 of our patients who were re-examined after a period of hypotensive treatment varying from 3 to 6 months. Though in these the electrocardiogram and x-ray size of the heart had returned to normal, the impulse cardiogram showed an unchanged abnormal sustained ejection beat. However, in 10 of our patients who presented with a normal impulse cardiogram, 3 had left ventricular enlargement on the chest x-ray, and 1 patient showed left ventricular hypertrophy on the electrocardiogram. All these patients had a barely palpable cardiac beat, and the failure of the impulse cardiogram to disclose signs of left ventricular hypertrophy could probably be explained by the difficulty in making an adequate record. This discrepancy between the impulse cardiogram, electrocardiogram, and chest x-ray was not recorded in the studies of Beilin and Mounsey who selected their patients to exclude those suffering from diseases of pleura and lung, obesity, etc., in order to obtain a reasonable amplitude of the apical impulse. On the other hand, all our patients with an easily palpable apex beat associated with hypertension or enlargement of the left ventricle on the electrocardiogram and chest x-ray were also found to have an abnormal ejection beat in the impulse cardiogram.

These findings were in accordance with those of Beilin and Mounsey as far as the reliability of the impulse cardiogram is concerned for the detection of left ventricular hypertrophy or enlargement as demonstrated by the electrocardiogram and chest x-ray, in patients with a clearly palpable apex beat. Nevertheless, these results differ in that they show the impulse cardiogram to be the most sensitive method of detecting signs of left ventricular dysfunction in the early stages of hypertension. Possibly the greater number of our patients (47), compared with those of the above-mentioned authors (25), explains the difference between their results and ours.

The underlying pathological abnormality of ventricular systole responsible for the increased duration of the apical impulse in left ventricular hypertrophy has been illuminated by the work of Deliyannis et al. (1964). They studied the different muscle layers of the heart (external spiral, middle circular, and internal spiral), and found that in health, external and internal fibres retract the apex, while the basal portion is squeezed by middle circular fibres which do not extend to the apex. The anterior wall of the heart retracts from the thoracic cage as the heart empties in late systole. In left
FIG. 3.—Impulse cardiograms, electrocardiograms, and chest x-ray films in a 26-year-old woman with hypertension, taken before and after 3 months of hypotensive therapy. The pretreatment electrocardiogram shows inverted T waves in the left ventricular leads, which became upright after treatment. The pre- and post-
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Post-operative treatment x-ray films show normal heart size. However, in both of the examinations, the impulse cardiogram is distinctly abnormal, with a sustained ejection beat, suggesting that the left ventricular dysfunction persists after therapy.
ventricular hypertrophy, the middle circular fibres were found to extend to the apex and thus tend to oppose the retracting action of the external and internal spiral layers: therefore in systole the heart is squeezed and little retraction of the apex takes place. Other factors, however, probably also contribute to the genesis of the sustained impulse including general increase in heart size and sometimes dilatation as well as hypertrophy.

It is generally accepted that the electrocardiogram is not always an accurate method of establishing the diagnosis of left ventricular hypertrophy. Whereas Scott (1960) reports over-all accuracy to be 85 per cent, Selzer et al. (1958) report 70 to 80 per cent, and Rosenfeld et al. (1962) 60 to 70 per cent, Burch et al. (1958) thought that the electrocardiogram could not be relied upon to indicate chamber enlargement. In the particular context of hypertensive patients, the electrocardiogram as the main test of left ventricular hypertrophy has been found even less satisfactory. In 25 to 50 per cent (Friedberg, 1966) of the cases of moderate hypertension the electrocardiogram was normal. According to the study of Dawber et al. (1952), the electrocardiogram was abnormal in only 6 per cent of 465 patients with hypertension, and in only 33 per cent of 154 patients with hypertensive heart disease.

The reasons for the relative lack of correlation between the development of an abnormal electrocardiogram and the degree of cardiac hypertrophy or enlargement remain a matter of conjecture. A good explanation, suggested by Leishman (1951), may be that the electrocardiographic changes in hypertensive patients are possibly an expression of coronary insufficiency, and if in a hypertensive patient, whose coronary circulation is unimpaired, the heart can hypertrophy to a relatively large extent before muscle ischaemia will cause the appearance of an abnormal electrocardiogram.

X-ray examination of the heart is generally less reliable than is the electrocardiogram. Leishman (1951) in his study of 218 hypertensive patients found that a significantly greater proportion of patients with abnormal electrocardiograms had enlarged hearts, but even so this proportion was no higher than 50 per cent. This inaccuracy of the radiological examinations is well understood on the basis of the concentric type of left ventricular hypertrophy from which a great number of patients suffer during the early stages of hypertensive heart disease.

Evidence of left atrial hypertrophy in hypertensive heart disease was found more frequently in the impulse cardiogram than in the electrocardiogram. Twenty-two patients (48%) had an abnormal atrial beat in the impulse cardiogram, whereas only 10 patients (21%) showed P wave changes compatible with left atrial hypertrophy on the electrocardiogram.

Studies on the amplitude of the apical impulse did not correlate with signs of left ventricular hypertrophy in the electrocardiogram and chest x-ray.

SUMMARY

The impulse cardiogram has been compared with the electrocardiogram and chest x-ray in 47 patients with hypertension.

An abnormal prolongation of the apical impulse beyond the second sound was recorded in 5 patients with normal electrocardiogram and another 8 patients with minimal non-specific T wave changes. A sustained apical impulse was also found in 20 patients with normal heart size as seen radiologically. These findings suggest that by palpation of the apex beat it is possible to detect left ventricular dysfunction in hypertensive patients with a normal electrocardiogram and chest x-ray.

An abnormal atrial beat on the impulse cardiogram was present more often than an abnormal P wave in the electrocardiogram suggestive of left atrial hypertension.

The amplitude of the apical impulse did not have good correlation with the degree of left ventricular hypertrophy or enlargement as indicated by the electrocardiogram and chest x-ray film.

On long-term follow-up with hypotensive treatment which varied from 3 to 6 months, lowering of the blood pressure did not alter the length of the apical impulse, even when there was electrocardiographic and radiological improvement of the left ventricular state.

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