The value of praecordial pulsations in the diagnosis of heart disease

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PALPATION of the praecordium is as indispensable as auscultation of the heart in clinical examination of the cardiac patient. Indeed, the two should be practised simultaneously, checking the timing of heart sounds and praecordial movements in relation to one another. To some extent the introduction of the modern binaural stethoscope with its flexible rubber tubes has been a retrograde step, since this instrument is less suited to simultaneous analysis of praecordial pulsations and heart sounds than was the original solid tubular stethoscope invented by Laennec. In the Traité de l’Auscultation Mediate, Laennec (1819) emphasized that his instrument could be used not only to auscultate the heart but also to appreciate accurately the cardiac impulse. He includes in his book a chapter on the cardiac impulse and states that a forcible impulse should be regarded as the principle sign of cardiac hypertrophy.

Improved methods of recording the cardiac impulse have been slower to develop than has phonocardiography, although apex cardiography was one of the earliest methods introduced for recording the heart beat (Marey, 1878). Apex cardiography suffers from the disadvantage that it is only a record of the relative displacement of a point in an intercostal space in relation to the immediately surrounding area of chest wall. In cardiac diagnosis it is often the displacement of a large area of the thoracic cage by the movements of the underlying heart that is important diagnostically, (as in the left parasternal lift of right ventricular hypertrophy), and not the relative movement of a small area in an intercostal space. To record this, an instrument is required that measures absolute displacement, that is the total movement of the chest wall in relation to a fixed point in space. Various methods have been used for making this measurement, by Dressler in 1937, Eddleman et al. in 1953 and by Beilin & Mounsey in 1962. We have called our instrument the impulse cardiogram and it aims at being a graphic record of what the physician’s hand and fingers feel.

The technique of palpating the praecordium

It is often a good practice when examining the praecordium to place the whole palm of the hand over the area of the chest that underlies the heart. In this way, one can imagine that one is holding the anterior surface of the heart in the cup of one’s hand, thus appreciating both right and left heart events simultaneously. Care should be taken, however, not to assume that pulsations at the apex are always due to the left ventricle, while those in the left parasternal area are due to the right. Although this statement is true probably in about 90% of patients, in the other 10% it is misleading. With great enlargement of either left or right ventricle the relationship of underlying chambers of the heart to the praecordium are altered (Fig. 1). Thus, with marked left ventricular hypertrophy this chamber may underlie the whole area of praecordium from the apex beat to the left sternal edge, the right ventricle being pushed over to the right. Similarly, with great right ventricular enlargement this chamber forms the apex of the heart, the left ventricle being rotated posteriorly (Deliyannis et al., 1964). It is more accurate, therefore, when describing praecordial pulsations to relate these to the areas on the chest wall where they are felt, rather than to term them ‘right or left ventricular types’ of impulse.
The genesis of the cardiac impulse in health and disease

Angiocardiographic and cinefluoroscopic studies have shown close correlation between the movements of the heart and the form of the cardiac impulse.

1. The apical impulse in ventricular systole

The first part of the apical impulse, both in health and disease, is probably caused by similar mechanisms. In William Harvey's words (1628), "the heart erects and raises itself into a point, so that at this moment it strikes the chest wall."
and externally a pulsation can be felt. It is during the latter part of systole that the heart behaves abnormally in the presence of hypertrophy. Whereas in health the anterior wall of the heart retracts from the thoracic cage as the heart empties in late systole (Fig. 2), in left ventricular hypertrophy the antero-apical portion of the heart fails to retract in late systole (Fig. 3), thus giving rise to the sustained cardiac impulse felt over the praecordium. Studies of the different muscle layers of the heart have suggested that the cause of this phenomenon is extension of the middle circular layer of muscle fibres toward the apex of the heart in the presence of hypertrophy, thus tending to inhibit the apical retracting action of the spiral fibres (Fig. 4) (Deliyannis et al., 1964). 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.

The cause of the sustained impulse in ventricular aneurysm is not far to seek. The aneurysmal wall which is composed entirely of fibrous tissue without any living myocardium, is unable to take part in concentric contraction of the heart and herniates outwards during ventricular systole (Fig. 5).

The marked systolic retraction and diastolic expansion seen in some cases of constrictive pericarditis is probably related to tethering of the inflow and outflow tracts of the ventricles, in annular constrictive pericarditis (Fig. 6). In this condition, the anterior wall of the ventricles is often relatively free from constriction and hence shows exaggerated contraction during ventricular systole and expansion during diastole. Ventricular filling is abnormally abrupt and forceful due to the high venous filling pressure. The marked systolic retraction and large diastolic rapid inflow beat seen in the impulse cardiogram closely reflect the underlying heart wall movements (Mounsey, 1959).

Apical retraction seen in tricuspid incompetence is due to a different mechanism, reflecting the increased stroke output of the dilated right ventricle which is both ejecting blood into the pulmonary artery and regurgitating it through...
the incompetent tricuspid valve into the right atrium. Cineradiological studies show a marked seesaw motion of the heart, with inward movement of the left cardiac border during systole and simultaneous outward movement of the right atrial border. This seesaw cardiac motion is clearly reflected in the movements imparted to the whole praecordium (Boicourt, Nagle & Mounsey, 1965).

2. The atrial beat

Increased atrial transport function with decreased ventricular compliance is probably the basic abnormality responsible for an augmented atrial beat associated with ventricular hypertrophy. In hypertrophic obstructive cardiomyopathy, where great ventricular hypertrophy is seen, the major portion of ventricular filling has been shown to take place during atrial systole.

**Fig. 5.** Impulse cardiogram, chest X-ray, cineradiogram left cardiac border, and cross-section of heart at autopsy in patient with cardiac aneurysm. Paradoxical systolic pulsation shown in cineradiogram tracings, accounting for overlying sustained cardiac impulse. Bulging of left cardiac border in chest X-ray. Extensive infarct involving whole of lateral wall of left ventricle at autopsy. ECG showing ST elevation and tall T waves in anterior chest leads.
FIG. 6. (a) Annular constrictive pericarditis as seen at operation. Pericardial constriction, (1) in A-V groove, and (2) around base of aorta and pulmonary artery. (b) Praecordial impulse cardiogram (MID P) showing systolic retraction in constrictive pericarditis. The outward diastolic pulsation is the diastolic rapid inflow beat (DRI beat). The early diastolic sound (EDS) coincides with the steep portion of the upstroke of the DRI beat. Phonocardiogram: PA, MF: pulmonary area, medium frequency. 2' and 2": split second heart sound. LSE, MF: left sternal edge, medium frequency. Electrocardiogram lead II.

FIG. 7. Superimposed tracings of cineangiocardio-
grams of right ventricle in left lateral view, at three points during cardiac cycle, indicated in simultaneous electrocardiogram. Note major increase of ventricular cavity area during downstroke of P wave (--- --- line) and beginning of QRS (--- · --- line), i.e. in atrial systole and end-diastole.

in cineangiocardio-
graphic studies. This is associated with a large outward movement of the anterior ventricular wall which in turn causes the giant atrial beat in the cardiac impulse (Fig. 7) (Nagle et al., 1966). In ventricular aneurysm, however, another factor is probably contributing to the large amplitude of the atrial beat, namely the thin tambour-like quality of the aneurysmal wall.

Acknowledgments

References