KINETOCARDIOGRAPHY IN CORONARY ARTERY DISEASE

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The beating heart produces low frequency movements of the anterior chest wall. If these movements are recorded with an externally fixed pick-up device a kinetocardiogram (KCG) (Eddleman *et al.*, 1953) is obtained. The curve is a displacement record of the absolute movements and differs in this respect from tracings registered with pick-up devices resting directly upon the chest wall, e.g. apex cardiography (Marey, 1885; Luisada and Magri, 1952; Benchimol, Dimond, and Carson, 1961).

The normal KCG is well defined. In 30 of 51 normal men between 16 and 30 years of age KCGs similar in shape, relative size of amplitude, and time intervals were found. Only minor deviations were present in the remaining 21 men (von Bertrab, Reist, and Schweizer, 1963). The normal KCG has the following characteristics (Fig. 1): small outward movement during atrial

FIG. 1.—The normal kinetocardiogram (see text).
Arrow indicates start of the ejection period.

systole (A); minor and variable movements during isometric contraction (I); short outward movement (E) at the beginning of the ejection period, which is dominated by a retraction (R); outward movement in protodiastole (D) and a filling wave (F), beginning at the time of opening of the A-V valves. Small differences only exist between KCGs taken from different regions of the anterior chest wall. Similar curves have been published by authors previously working with this method

(Eddleman and Harrison, 1963). We did not find, however, a single case of sustained outward movement during the ejection period. The effect of age on the KCG has not yet been investigated. The impossibility of fully excluding coronary artery disease in clinically normal men above the age of 40 years is the major obstacle in investigations of this kind. We know, however, that normal KCGs can be found in normal men up to the age of 70 years (unpublished observations).

In heart disease the KCG is often abnormal. Abnormalities have been found in conditions with altered haemodynamics (Eddleman, 1959). Abnormal KCGs were obtained also in coronary artery disease (Harrison and Hughes, 1958; Eddleman and Langley, 1962). These latter results are of special interest as the diagnosis of coronary artery disease during the quiescent phase is often unsatisfactory.

This paper describes the result of a kinetocardiographic investigation in 75 patients with coronary artery disease.

SUBJECTS AND METHODS

The 75 patients fall into three groups.

(1) 31 patients with angina on effort (30 men and 1 woman aged 41–69 years) of variable duration (6 weeks to 7 years) without electrocardiographic evidence of cardiac infarction. The heart size was abnormal in 11 out of 30 cases; no radiography was available in one. All had sinus rhythm and none had heart failure. The electrocardiogram was abnormal in 24 cases; in 3 patients left bundle-branch block was present, whereas ST–T segment changes only were found in the others. The blood pressure was greater than 160/90 mm. Hg in 10 patients.

(2) 19 patients with recent cardiac infarction (16 men and 3 women aged 35–69 years) were investigated 10 to 59 days after the occurrence of the infarction. The electrocardiogram showed signs of anterior wall infarction in 8 and of infarction of the diaphragmatic wall in 7; posterior infarction was found in 1, and in 3 patients the localization of the infarction was not possible. The heart size was abnormal in 9 out of 11 patients; in 8 no radiography was carried out as complete bed-rest had been ordered by the physician in charge. All had sinus rhythm. Heart failure was present in 6 patients at the time of the investigation. Blood pressures higher than 160/90 mm. Hg were found in 5 patients.

(3) 25 patients with old cardiac infarction (23 men and 2 women aged 33–70 years) were studied 3 to 60 months after a cardiac infarction had occurred. The exact time of the infarction remained unknown in 6 cases. The electrocardiographic localization of the infarction was the anterior wall in 7, the diaphragmatic wall in 13, and the posterior wall in 2; the necrosis could not be localized in the remaining 3 patients. The heart size was abnormal in 16 and heart failure was found in 8. All had sinus rhythm. The blood pressure was above 160/90 mm. Hg in 7 patients.

The method used has already been described (von Bertrab *et al.*, 1963). It can be summarized as follows: A capacitive pulse transducer* is fixed at one end of a metal bar. The bar is mounted on an arch above the thorax so that vertical setting of the transducer is possible at every point of the anterior chest wall. The points of recording are indicated in Fig. 2. They are defined by vertical lines used in electrocardiography (first number) and by intercostal spaces (second number). The KCG, standard lead II (ECG), and first and second heart sounds were recorded simultaneously. A carotid pulse tracing, standard lead II, and first and second heart sounds necessary for defining the beginning of the ejection period were taken afterwards. A three-channel direct recording Cardiopan III* was used.

The patients were in the supine position and the tracings were taken in apnoea at the end of a normal expiration.

RESULTS

The KCG was abnormal in 73 out of 75 cases. In only one case of angina pectoris and in one case of recent cardiac infarction was a normal KCG found. Six out of seven patients with angina on effort and normal ECG at rest had an abnormal KCG.

The following abnormalities were found:

(a) Large A-waves (more than 20% of the total amplitude) in about 80 per cent of cases (Fig. 3–5).

* Philips AG Zürich (Switzerland).

FIG. 2.—The points of recording the kinetocardiogram on the anterior chest wall.

FIG. 3.—The relative frequency of the kinetocardiographic abnormalities found in 75 patients with coronary artery disease. A+, large A waves; E-, small E waves and small retraction; "Bulges", outward movements during systole; and D+, large D waves.

(b) Outward movements during systole ("bulges"; Harrison and Hughes, 1958) in approximately 80 per cent of cases (Fig. 3). These "bulges" were seen early in systole beginning during isometric contraction (Fig. 6), in the middle of systole (Fig. 4), or late in systole (Fig. 7). In some cases the bulges were pansystolic (Fig. 8). Mid-systolic bulges were most frequent in angina on

FIG. 4.—KCG (K 4/4) from a man aged 59 years with angina on effort showing large A waves (A) and a mid-systolic "bulge" (B). Negativity of the T wave in lead aVF and standard leads II and III was the only abnormal sign. The blood pressure was 125/80 mm. Hg. Arrow, start of the ejection period.

FIG. 5.—KCG taken from a 59-year-old man with angina on effort showing large A waves (A) and large D waves (D). The heart size was within normal limits. A negative T wave in lead aVL, with isoelectric T in standard lead I was the only electrocardiographic anomaly. The blood pressure was 155/80 mm. Hg. Arrow, start of the ejection period.

TABLE
RELATIVE FREQUENCY OF VARIOUS ABNORMAL OUTWARD MOVEMENTS ("BULGES") IN 75 PATIENTS WITH CORONARY HEART DISEASE

	"Bulges" (%)			
	Early systolic	Mid-systolic	Late systolic	Pansystolic
Angina pectoris (31)	3	61	16	0
Cardiac infarction (44)	14	34	20	16
Recent (19)	10	26	32	16
Old (25)	16	40	12	16

effort whereas pansystolic bulges were found in cardiac infarction only (Table). Pansystolic bulges were confined to the positions K3/4, 3/5, 4/4, 4/5 whereas the other bulges were frequently present over a larger area.

(c) Small E waves and small retraction in approximately 40 per cent of cases (Fig. 3 and 7).

(d) Large D waves (more than 50% of the total amplitude) in 40 per cent of all cases (Fig. 3 and 5).

No definite relation could be established between the different kinetocardiographic abnormalities on the one hand and the heart size, presence of heart failure, electrocardiographic findings, and blood pressure on the other. Although all patients with heart failure and/or arterial hypertension had large A waves, this sign was frequently present in cases without clinically abnormal haemodynamics. No relation existed between the localization of the various bulges and the site of the infarction judged from the electrocardiogram.

FIG. 6.—KCG from a woman aged 69 with recent cardiac infarction showing an early systolic "bulge" (B). The heart size was abnormal and congestive heart failure was present. The blood pressure had been greater than 200/100 mm. Hg for years; at the time of the investigation it was 180/95 mm. Hg. Arrow, start of the ejection period.

FIG. 7.—KCG taken from a 67-year-old man with recent cardiac infarction, showing a late systolic "bulge" (B). The heart size was normal; a chest radiograph was not recorded. There was no heart failure and the blood pressure was 110/75 mm. Hg. Arrow, start of the ejection period.

FIG. 8.—KCG K 3/4 from a man aged 47 with recent cardiac infarction showing a pansystolic “bulge” (B). The heart size was abnormal and a third heart sound was audible. There was no pulmonary venous congestion or venous hypertension. The blood pressure was 110/80 mm. Hg. Arrow, start of the ejection period.

failure irrespective of the underlying heart disease (Skinner, 1961; Skinner, Nickerson, and Ingram, 1961b). Systolic bulges on the other hand are frequently found in cases with systolic overload of either ventricle. It is highly probable, however, that bulges in the absence of abnormal systolic ventricular pressure and bulges in the K3 position are signs of coronary artery disease. Furthermore the combination of large A waves, mid-systolic bulges, and large D waves seems to be a typical pattern in angina on effort. Little can be said, therefore, about the diagnostic value of kinetocardiography in coronary artery disease.

The pathophysiology of the kinetocardiographic abnormalities found in coronary artery disease is only partially known. Large A waves, as mentioned before, are frequently found in congestive heart failure. They diminish in height or disappear altogether during successful treatment of failure. It is probable therefore that large A waves are caused by atrial contraction in the presence of an abnormal end-diastolic ventricular pressure. If this is correct one has to assume that in coronary artery disease heart failure is frequent. Müller and Rørvik (1958) have been able to demonstrate a rise of the pulmonary capillary pressure during anginal pain. It is further known that patients with angina on effort are frequently stopped not only by pain but also by breathlessness. An increased resistance to distensibility with abnormal end-diastolic pressure is not excluded however. Tennant and Wiggers (1935) and Prinzmetal *et al.* (1949) described paradoxical outward movements of the ischaemic myocardial area after the ligation of a large coronary artery branch in dogs (“functional aneurysm”). Kymographic studies, especially electrokymographic investigations have further shown abnormal outward movements of the border of the heart in a considerable proportion of patients with coronary artery disease. (Dack, Sussman, and Master, 1940; Dack, Paley, and Sussman, 1950; Sussman, Dack, and Master, 1940; Luisada and Fleischner, 1948; Samet, Schwedel, and Mednick, 1950.) Finally it is well known that at necropsy anatomical aneurysms are frequently found in these cases (Scherf and Boyd, 1942). It is not known if the bulges are direct evidence of functional or anatomical aneurysm.

It is well known that in patients with coronary artery disease careful palpation of the anterior chest wall sometimes reveals abnormal outward movements. It is said that the frequency of these paradoxical motions is higher in cases of cardiac infarction than in those with angina on effort only, where the abnormality is more often only found during pain (Dressler and Pfeiffer, 1940; Harrison, 1955, 1959; Vakil, 1955; Hurst and Blackard, 1958). The KCG represents the displace-

DISCUSSION

In coronary artery disease two main kinetocardiographic abnormalities are found in the majority of cases: large A waves and outward movements during systole or “bulges”. In this respect we have confirmed the observations of the groups of workers who introduced the method of recording (Harrison and Hughes, 1958; Suh and Eddleman, 1959; Skinner *et al.*, 1961a; Davie *et al.*, 1962; Eddleman and Langley, 1962; Eddleman and Harrison, 1963). In our experience, however, the frequency of these abnormalities is even higher than in the investigations mentioned. So we cannot agree with the statement that patients with isolated angina on effort usually have normal KCGs. We found abnormal KCGs in the majority of cases with a normal ECG at rest.

The specificity of these alterations is not yet fully established. It has been shown that large A waves are usually present in congestive heart

ment of the anterior chest wall relative to a fixed external point. As far as outward movements are concerned the KCG shows therefore what can be felt on careful palpation. This close relation between the KCG and the palpatory findings enhances the value of kinetocardiography. The KCG is an objective record: it gives an accurate relation between a given palpatory finding and the cardiac cycle. Finally it reveals more details than palpation, especially the inward movements which are not easily felt.

SUMMARY

The results of a kinetocardiographic investigation in 75 patients with coronary artery disease are described. The kinetocardiogram was abnormal in 73 cases. An increase of the outward movement during atrial systole and outward movements during ventricular systole were the main abnormalities found. It is probable that systolic outward movements in the absence of abnormal ventricular pressure are specific signs of coronary artery disease. The diagnostic value of the method is not yet known; its establishment is desirable.

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