THE APEX CARDIOGRAM IN ISCHÆMIC HEART DISEASE*

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Techniques for recording low frequency movements of the chest wall caused by cardiac contraction have become the focus of increasing attention in the past few decades (Crehore, 1911; Weitz, 1917; Weber, 1920; Dressler, 1937; Wood *et al.*, 1951; Johnston and Overy, 1951; Luisada and Magri, 1952; Eddleman *et al.*, 1953; Groom and Boone, 1956; Hartman, 1956; Mounsey, 1957; Harrison *et al.*, 1958; Hollis and Vidrine, 1959; Rosa and Luisada, 1959; Benchimol *et al.*, 1961; Schneider and Klunhaar, 1961). With the use of these techniques, attempts have been made to establish abnormalities of the cardiac contraction in patients with ischæmic heart disease. Changes in the ballistocardiogram (Starr and Wood, 1943; Moss, 1961), röntgenkymogram (Dack *et al.*, 1940), kinetocardiogram (Eddleman *et al.*, 1953, 1957; Harrison and Hughes, 1958; Harrison, 1959; Suh and Eddleman, 1959; Skinner *et al.*, 1961), acceleration cardiograms (Mounsey, 1959; Rosa and Karsak, 1960) and others (Taquini, 1940; Wiggers, 1952; Harrison, 1954; Dressler, 1957; Hurst and Blackard, 1958) have been described: they were associated mainly with ventricular systole. Difficulties related to recording devices and interpretation of the tracings have been responsible for the lack of popularity of these methods.

In the present study, we intend to report the abnormalities of the apex cardiogram in patients with ischæmic heart disease with special emphasis on the "a" wave, which represents the active left ventricular filling due to atrial contraction.

As we described in previous reports (Benchimol *et al.*, 1960, 1961), the apex cardiogram represents the low frequency (0.1 to 50 cycles per second) displacement curves of the chest wall overlying the right or left ventricle (apex beat). The reliability and reproducibility of this method have been confirmed by Rosa and Luisada (1959) in a recent comparative study of different techniques for recording low frequency tracings of the chest wall caused by cardiac contraction.

SUBJECTS AND METHODS

Fifty-nine consecutive patients with well-documented ischæmic heart disease were studied. The criteria for selection were as follows. (1) Well-defined past history of myocardial infarction. (2) Electrocardiographic evidence of myocardial infarction. (3) Typical history of angina with or without myocardial infarction. (4) Atypical history of angina or no angina but with unquestionable positive exercise electrocardiogram with the double Master's two-step exercise test. One or more of the above was present in all subjects.

Patients were excluded from this study if any of the following findings were present, alone or in combination: (1) heart failure, (2) systemic hypertension, (3) valvular lesion, congenital or acquired, (4) pulmonary disease, (5) congenital heart disease.

In no case was the selection of the patient based on the "adequacy" of the apex cardiogram.

The patients were divided into the following groups.

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Group I. The control group comprised 52 normal subjects (27 female and 25 male) who were chosen on the basis of no history suggestive of angina, normal physical examination, and normal electrocardiogram. Ages ranged from 5 to 63 with an average of 36 years; 36 patients were above age 30 and 16 below 30 years. Thirty-two of them were exercised according to the Master's two-step exercise requirement and the apex cardiogram recorded at rest, immediately and every minute up to 15 minutes after exercise. The effect of nitroglycerine was studied in 10 of these patients.

Group II. This comprised 59 patients (40 men and 19 women) suffering from ischaemic heart disease (ASHD—arteriosclerotic heart disease), whose ages ranged from 34 to 82 years with a mean of 64 years. From this series of 59 patients, 25 had unequivocal evidence of myocardial infarction by clinical, laboratory, and electrocardiographic criteria. The remaining 34 had not had a myocardial infarction, but they had either angina or an abnormal electrocardiogram at rest and/or after exercise.

Twenty-two of these 59 patients had repeated episodes of angina, 32 did not have angina, and 5 presented atypical chest pain with an abnormal cardiographic response to exercise. Of these patients, 25 were exercised according to the double Master's two-step exercise requirement and the apex cardiogram was recorded. The effect of nitroglycerine was studied in 27 cases.

The equipment used for recording the apex cardiogram of the left ventricle was the multi-channel Electronics for Medicine, DR-8, oscilloscopic photographic recorder. A crystal microphone (Sanborn No. 374) was attached to a microphone bell by a 5 in. long rubber tube. This crystal microphone reproduced an electrical signal proportional to changing pressure in the tubing. The crystal microphone was then connected to an AC amplifier with selective filters. The low limit filter was placed to 0.1, and the upper limit filter to 50, cycles per second. A low frequency (40–200 cycles per second) phonocardiogram at the apex and the electrocardiogram (præcordial lead at the point of the maximal impulse) were recorded simultaneously with the apex cardiogram. The tracings were recorded at a paper speed of 75 mm./sec.

Technique for Recording Apex Cardiogram. After completion of the routine phonocardiogram, the point of maximal cardiac impulse (apex beat) in the left lateral decubitus position was determined by palpation, and a left ventricular complex confirmed on the electrocardiogram (præcordial lead) from this point (R, Rs, qRs type of complex) (Fig. 1). The sound microphone was then positioned so that the pick-up bell was placed directly over the point of maximal impulse. The instrument was strapped to the chest by a rubber band or held in position by hand with slight pressure applied to the chest wall. The tracings were recorded at the end of expiration.

Analysis of the apex cardiogram was made in each case with special attention given to the diastolic filling wave which represents the reflected ventricular wave due to atrial contraction. Since no standardization was attempted, the amplitude of the "a" wave ("a" from atrial wave) was measured in millimetres of deflection from its base line and expressed as a percentage ratio of the total amplitude of the tracing. The total amplitude of the tracing was measured from the peak of the systolic wave (E-point, representing the opening of the aortic valve) to the base line (O-point, representing the opening of the mitral valve) (Fig. 2). The shape of the systolic wave, duration of the mechanical systole, isometric contraction, isometric relaxation, and duration and amplitude of the rapid filling wave in the apex cardiogram were studied carefully.

A time interval from the peak of the "a" wave to the E-point (peak of the systolic wave) was also measured and designated the a-E interval.

The results were analysed statistically, and the "t" test was used to determine the significance between the groups (Croxtton and Cowden, 1959).

All reported measurements and ratios represent the mean values of measurements made in three consecutive cardiac cycles. The following abbreviations have been employed throughout this study: 1, first sound; 2, second sound; 3, third sound; 4, fourth sound; ACG, apex cardiogram; "a", atrial wave; E(ejection) point, (opening of the aortic valve); O (opening) point, (opening of the mitral valve); RFW, rapid filling wave; and SW, systolic wave.

FIG. 1.—Chest radiograph showing the position of the apex beat (O) as determined by palpation. The apex cardiogram was recorded from that point. Note that the lead marker (O) coincides with the heart border which represents the point of maximal left ventricular impulse.

FIG. 2.—Apex cardiogram of the left ventricle (ACG) simultaneously recorded with the phonocardiogram at the apex (MA), carotid tracing (CT), jugular venous tracing (VT), and electrocardiogram (EKG), demonstrating the derivation of different measurements. RFW=rapid filling wave, IC=isometric contraction, IR=isometric relaxation.

RESULTS

Group I. 52 normal subjects.

Normal Apex Cardiogram. A normal apex cardiogram has been described in detail in our previous report (Benchimol *et al.*, 1961). In summary, it presents the following waves: "a" wave, due to atrial contraction; SW, systolic wave due to ventricular systole; RFW, rapid filling wave due to early passive rapid ventricular filling; SFW, slow filling wave due to late passive slow ventricular filling (diastasis); E-point, opening of the aortic valve, marks the beginning of ventricular ejection; O-point, opening of the mitral valve, marks the beginning of ventricular filling; IC, isometric contraction, from the end of the "a" wave to the E-point; and IR, isometric relaxation, from the second heart sound to the O-point (2-O interval). In a previous report, we called this the 2-FW interval (from the second sound and filling wave).

A normal apex cardiogram is illustrated in Fig. 3. It is characterized by the presence of a small

FIG. 3.—Apexcardiogram (ACG) of the left ventricle in six normal subjects. Note the small "a" waves in all tracings. All phonocardiograms recorded at apex. Compare with Fig. 6.

"a" wave, sharp E-point, rapid fall off of the systolic wave, and a small rapid filling wave. The original identification of all these components was based on a simultaneous recording of the apex cardiogram with intracardiac pressure curves during left and right heart catheterization in over 70 patients (Fig. 4). The percentage amplitude of the "a" wave in this group of 52 subjects averaged 7.8 per cent with a standard deviation of ± 1.4 per cent (Table I). The "a" wave in this group was quite small, with a duration not exceeding 0.06 sec., peaked, with a smooth contour and inscribed in the ascending limb of the systolic wave (Fig. 3). The a-E interval in this group averaged 0.09 sec. There were no significant abnormalities in the systolic wave nor in the rapid filling wave.

Effect of Exercise. Of these patients, 32 were exercised according to the Master's two-step criteria and the apex cardiogram recorded before, immediately after, and every minute after the exercise for 15 minutes. The response to exercise was considered negative in 30 (94%) cases in the sense that there was no increase in the amplitude of the "a" wave in the post-exercise period. In addition, there were no significant changes in the amplitude or configuration of the systolic and rapid filling waves (Fig. 5).

None of these patients complained of chest pain, dyspnœa or any other symptoms. Except for the physiological post-exercise tachycardia, there were no abnormalities in the præcordial electrocardiogram. Two cases out of 32 (6%) demonstrated a significant increase in the percentage amplitude of the "a" wave after exercise. These were considered false-positive responses. One was an asymptomatic 51-year-old man with a normal electrocardiogram at rest and after exercise, and the second patient was a 27-year-old asymptomatic woman. The amplitude of the "a" wave in these two patients increased from 9 per cent at rest to 18 and 21 per cent after exercise, respectively. Seven patients in this group of 32 had "a" waves at the upper limit of normal at rest with no change after exercise. The response to exercise, therefore, was considered negative.

The average percentage amplitude of the "a" wave for this group of 32 cases before exercise was found to be 8.0 ± 0.5 .

Group II. Ischæmic heart disease (ASHD—arteriosclerotic heart disease) 59 patients.

Rest. The most important abnormalities in this group were related to the amplitude of the "a" wave. It averaged 25.5 per cent with a standard deviation of ± 3.4 per cent (Table I and Fig. 6). The "a" wave, in addition to a significant increase in amplitude, was notched, had a longer duration, and was inscribed well before the E-point. Consequently, the a-E interval was somewhat increased in this group (mean 0.12 sec.) when compared with the control group (mean 0.09 sec.). The systolic wave was more rounded and the rapid filling wave less conspicuous.

FIG. 4.—Apex cardiogram (ACG) of the left ventricle recorded simultaneously with the left ventricular (LV) and right ventricular (RV) pressure curves during heart catheterization. Note the similarity of the systolic component (arrow) of the apex cardiogram as compared with left ventricular pressure curve. The time delay in the left ventricular curve relates to catheter length.

TABLE I

MEAN VALUES OF PERCENTAGE AMPLITUDE OF "a" WAVES OF APEX CARDIOGRAM IN NORMAL SUBJECTS AND IN PATIENTS WITH ISCHÆMIC HEART DISEASE AT REST, AFTER EXERCISE, AND AFTER NITROGLYCERINE

		Percentage amplitude of "a" wave (mean)				
		Resting	Immediately	2 min.	5 min.	10 min.
Group I	Exercise	8.0	8.3	8.2	8.1	7.0
Control group	32 cases					
Group II	Exercise	26.5	41.3	35.4	31.3	24.5
Ischæmic group	25 cases					
	Nitroglycerine	33.3	20.5	15.4	8.3	7.8
	27 cases

FIG. 5.—Apex cardiogram (ACG) of the left ventricle in four normal subjects before and after exercise. Note absence of any significant changes in the amplitude of the “a” wave after exercise. All phonocardiograms recorded at apex. Compare with Fig. 9.

FIG. 6.—Apex cardiogram (ACG) of the left ventricle in six patients with ischaemic heart disease recorded at rest. Note very large “a” waves in all tracings. All phonocardiograms recorded at apex. Compare with Fig. 3.

The determination of the p values for the amplitude of the "a" wave between Groups I and II showed $p < 0.001$ ($t = 7.45$). There was no significant difference in the amplitude of the "a" wave between the group with and that without an infarct. Furthermore, patients without angina but with unquestionable positive exercise cardiograms had abnormal "a" waves in the apex cardiogram. Two patients developed spontaneous angina while the apex cardiogram was being recorded, and both showed significant increase in the amplitude of the "a" wave during angina (Fig. 7).

FIG. 7.—Apex cardiogram (ACG) of the left ventricle in two patients with ischæmic heart disease. These two patients developed spontaneous angina when the tracings were being recorded. Note increase in the amplitude of the "a" wave during angina: this decreased five minutes after administration of nitroglycerine. There was marked increase in the amplitude of the rapid filling wave (RFW) which was inconspicuous in the control tracing. The phonocardiograms were recorded at the apex.

Phonocardiographic findings in these patients revealed a loud fourth sound in the majority of the cases. The third sound was seldom present. These findings were in keeping with other observations of the mechanism of atrial contraction (Duchosal, 1932, 1935; Jochim, 1938; Weitzman, 1955; Kuo *et al.*, 1957; Kincaid-Smith and Barlow, 1959a and b; Parry and Mounsey, 1961).

Effect of Exercise. The effect of exercise was studied in 25 of the patients. The exercise had to be discontinued in nine who developed angina during exercise. In these patients, tracings were obtained before administration of nitroglycerine which relieved angina in all of them. There were no complications in this series, as far as the exercise was concerned.

The average amplitude of the "a" wave for the pre-exercise tracing was 26.5 per cent (Table I, Fig. 8 and 9). The maximal increase in the amplitude of the "a" wave occurred immediately after exercise with an average for the group of 41 per cent (Fig. 8), which was found to be statistically significant ($p < 0.01$). There was a statistically significant difference for the maximal post-exercise amplitude of the "a" wave between the normal subjects and the patients with ischaemic heart disease ($p < 0.01$, $t = 2.70$).

FIG. 8.—Mean values for the percentage amplitude of the "a" wave of the apex cardiogram at rest and after exercise in 22 normal subjects and in 25 patients with ischaemic heart disease (ASHD). Note significant increase in the amplitude of the "a" wave in the ASHD group as compared with the control group.

The presence or absence of myocardial infarction did not interfere with the abnormal response in this group of patients since all of them, regardless of the presence of infarct, presented an abnormal post-exercise "a" wave. Six patients in this group of 25 had normal "a" waves before exercise that became very abnormal after exercise. Four of them have had a myocardial infarction in the past. The majority of patients in this group developed an abnormal *systolic* wave after exercise (Fig. 10), and these changes will be reported in a following communication. A negative response to exercise was not seen in patients with ischaemic heart disease. The average increase in the post-exercise tracing for the "a" wave, in comparison with the resting tracing, was 36 per cent, and, as can be seen, an abnormally large "a" wave at rest uniformly became "more abnormal" after exercise.

Effect of Nitroglycerine. The effect of nitroglycerine was studied in 10 normal subjects and in 27 patients with ischaemic heart disease. The effect of this drug in normal subjects could not be fully appreciated because of the small "a" wave and short a-E interval. Nevertheless, there was a tendency toward a decrease in the amplitude of the "a" wave with shortening of the a-E interval in normal subjects.

In this group of 27 patients with ischaemic heart disease, marked changes occurred in the apex cardiogram after administration of nitroglycerine (1/150 grains) (Fig. 11 and 12).

FIG. 9.—Apex cardiogram (ACG) of the left ventricle in three patients with ischæmic heart disease showing significant increase in the amplitude of the “a” wave after exercise. A fourth sound is recorded in all tracings coincident with the peak of the “a” wave. These changes returned to the control values six minutes after exercise. Note also increase in the amplitude of the rapid filling wave (RFW) with a third sound after two minutes of exercise. Phonocardiograms recorded at apex. Compare with Fig. 5.

The “a” wave disappeared completely after nitroglycerine in 11 patients, decreased in amplitude in 12, and did not change in 4. The a-E interval was shortened, with the “a” wave being recorded on the ascending limb of the systolic wave and during or shortly after the QRS complex in the electrocardiogram. This was followed by a disappearance or reduction of the fourth sound in the phonocardiogram (Fig. 11).

There was a significant reduction in the total amplitude of the *Systolic wave* after

FIG. 10.—Apex cardiogram (ACG) of the left ventricle in two patients with ischaemic heart disease, showing change in the systolic wave (SW) after exercise. Observe that these changes occurred immediately after the E-point; thus representing abnormality of the cardiac contraction during the period of rapid ejection. There was also significant increase in the amplitude of the "a" wave. Note increase in the amplitude of the rapid filling wave (RFW) at two minutes after exercise. Phonocardiograms recorded at apex.

nitroglycerine. In many instances, the apex beat, which was forceful before administration of the drug, became very weak or impalpable after nitroglycerine. In these circumstances, an apex cardiogram could not be recorded adequately. These changes in the systolic component were preceded, temporarily, by changes in the "a" wave as described above and occurred two to three minutes after the drug had been given. It was noted that these changes occurred mainly in the component of the systolic wave that immediately followed the E-point, thus representing changes during the initial rapid left ventricular ejection.

There was a significant decrease in the amplitude of the *rapid filling wave* which became inconspicuous in the majority of patients.

Nitroglycerine and Exercise. Nitroglycerine was administered one minute before exercise in 14 patients with ischaemic heart disease. These 14 had previously been studied and had abnormal "a" waves before exercise which became "more abnormal" after exercise. The exercise was then repeated, preceded by the administration of nitroglycerine. Nitroglycerine prevented the appearance of the abnormal post-exercise "a" wave. There was also an increase in the tolerance to exercise for this group of patients.

FIG. 11.—Apex cardiogram of the left ventricle showing the effect of nitroglycerine in six patients with ischæmic heart disease. Note the very large “a” waves in the control tracings which almost disappeared after nitroglycerine. Observe, also, the decrease or disappearance of the fourth sound after nitroglycerine. The abnormal systolic wave (SW) marked with an arrow disappeared after nitroglycerine. Phonocardiograms recorded at apex.

Effect of Smoking. The effect of cigarette smoking was studied in 12 patients with ischæmic heart disease with abnormal resting “a” waves in the apex cardiogram. In 8, there was a significant increase in the amplitude of the “a” wave during smoking which returned to the control values ten minutes later. In 4 patients, no significant change occurred in the “a” wave.

Effect of Digitalis. The effect of acute digitalization was studied in two cases of ischæmic heart disease with large “a” waves in the resting apex cardiogram. No significant changes occurred in the amplitude of the “a” waves in these two patients after digitalization. The effect of this drug on the apex cardiogram as well as the effect of smoking and exercise preceded by nitroglycerine is being studied at the present time and the results will be reported later.

DISCUSSION

This study indicates that the apex cardiogram of the left ventricle adds information about the abnormal cardiovascular dynamics in patients with ischæmic heart disease.

FIG. 12.—Effect of nitroglycerine on the amplitude of the “a” wave of the apex cardiogram in patients with ischaemic heart disease (ASHD). The curve represents the mean values of measurements in 27 patients for each time interval after nitroglycerine.

Genesis of the “a” Wave. The apex cardiogram “a” wave reflects the ventricular filling wave associated with the impact of blood upon the ventricular wall during atrial systole. This conclusion is based on a simultaneous recording of the apex cardiogram with intracardiac pressure curves and on information obtained from patients with mitral valve lesions (Benchimol *et al.*, 1960).

(a) The “a” wave in the apex cardiogram occurs almost simultaneously with the “a” wave in the atrial pressure curves. (b) The apex cardiogram “a” wave disappears during atrial fibrillation or flutter. (c) In the presence of atrial prematurities or atrioventricular block, the “a” wave is inscribed at different time intervals in relation to the main systolic wave and follows the P wave in the electrocardiogram. This suggests a correlation between the mechanical and electrical atrial systole. (d) In cases of mitral stenosis, in which there is obstruction to blood flow across the mitral valve, the apex cardiogram “a” wave is inconspicuous or absent. (e) The apex cardiogram “a” wave is always coincident with the fourth sound (Fig. 2).

In view of these facts, we are convinced that the apex cardiogram “a” wave does represent the reflected ventricular wave during atrial systole. These explanations are supported by Hawthorne’s findings (1961) on the determinations of the instantaneous change in the cross-sectional areas of the left ventricle during the period of atrial contraction. This atrial component area tracing indicated a contribution to the volume of the ventricle by atrial contraction amounting to 30 to 40 per cent of its end-diastolic size. Rushmer (1954) also demonstrated a significant increase in the left ventricular diameter at the time of atrial contraction. In his records, the atrial systole was represented by a small upward deflection.

Mechanism of Abnormal “a” Wave in Ischaemic Heart Disease. Our findings seem to indicate an abnormal ventricular filling in patients with arteriosclerotic heart disease. The exact mechanism of this process is unknown. It appears that the abnormal mechanisms of the ventricular filling could be the result of three different factors acting alone or in combination: (a) abnormal atrial contraction as a consequence of increased left atrial pressure; (b) change in the distensibility of the ischaemic left ventricular wall; or (c) increase in the left ventricular residual volume due to incomplete emptying of the ventricle during systole.

Buckley *et al.* (1956) demonstrated that the intraventricular pressure inflow ratio is the result of the balance between a stress on the ventricular wall and its opposition to the stress. This ratio was

expressed as the mechanical impedance of the ventricular wall. The increased resistance to ventricular distensibility, according to their findings, tends to be much higher at the end of diastole, and this probably corresponds with changes in the elasticity and viscosity of the components of the ventricular wall before ventricular contraction. Therefore, a decrease in the ventricular distensibility should require, theoretically, a more effective filling pressure and consequently a more vigorous atrial contraction.

In 1958, Müller and Rørvik suggested that the anginal state was associated with some degree of left ventricular failure on the basis of a measured significant increase in the pulmonary capillary pressure and symptoms of left heart failure, whether the anginal pain was precipitated spontaneously or by exercise.

There is experimental evidence to support the fact that by constricting the left coronary artery (Case *et al.*, 1954), a decrease in the left ventricular stroke work occurs despite an increase in the left atrial pressure. This unilateral depression of the left ventricular wall function was quantitatively related to the degree of coronary insufficiency. Therefore, it appears feasible to believe that a decrease in the coronary blood flow in patients with ischæmic heart disease, resulting in an ischæmic ventricular wall, would produce a decrease in ventricular distensibility. During the episode of angina, the increase in the left ventricular end-diastolic pressure would be an additional factor opposing atrial systole.

Thus, in our experience, we are finding the apex cardiogram, simple as it is, a useful extra test to supplement and confirm the clinical and hæmodynamic findings in assaying patients with ischæmic heart disease. Even more important, this easily obtained measurement lends itself readily to physiological and pharmacological studies and may serve as a tool for elucidating that elusive condition, angina. Like most tests in medicine, it must be used in combination with other findings and its limits of error should be recognized.

SUMMARY AND CONCLUSION

Changes in the apex cardiogram were studied in 59 patients with ischæmic heart disease, of whom 25 had had a myocardial infarction and 34 either angina or an abnormal electrocardiographic response to exercise. Fifty-two normal subjects were used as a control group.

The apex cardiogram "a" wave was thought to represent the pulse wave originating in the ventricle from the impact of blood against the ventricular wall as a result of atrial systole. An abnormal "a" wave was not present in normal subjects but was found in nearly all patients with ischæmic heart disease, thus indicating an abnormal ventricular filling during atrial contraction. These changes were attributed to abnormally high ventricular resistance to atrial systole. The percentage amplitude of the "a" wave in relation to the total amplitude of the tracing in the normal subject was found to be 7.8 ± 1.4 per cent, and in the group with ischæmic heart disease, 25.5 ± 3.4 per cent.

The effect of exercise was studied in 25 patients with ischæmic heart disease and in 32 normal subjects. There was significant increase in the amplitude of the "a" wave after exercise in all patients with arteriosclerotic heart disease. There were two "false-positive" responses to exercise in the control group.

Nitroglycerine given to 27 patients with ischæmic heart disease produced complete disappearance of the "a" wave in 11, significant reduction in amplitude in 12, and no change in 4 patients. In 10 normal subjects, there was also reduction in the amplitude of the "a" wave. In 14 patients, the administration of nitroglycerine before exercise prevented the appearance of the previously abnormal post-exercise "a" wave.

It is concluded that this simple and easy method is useful to detect the abnormal mechanism of the ventricular filling in patients with ischæmic heart disease.

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