CORRELATION OF INTRACARDIAC PRESSURE AND PRÆCORDIAL MOVEMENT IN ISCHÆMIC HEART DISEASE

BY

E. GREY DIMOND AND ALBERTO BENCHIMOL

From the Institute for Cardiopulmonary Diseases, Scripps Clinic and Research Foundation, La Jolla, California

Received December 8, 1962

A series of reports (Müller and Rørvik, 1958; Ross *et al.*, 1962; Malmborg, Pernow, and Eliasch, 1962) has indicated that angina pectoris may be associated with a rise in left atrial pressure and in end-diastolic left ventricular pressure. Separately, over a greater number of years, there have been reports to indicate that audible sounds due to atrial systole are present during angina pectoris (Duchosal, 1932; Kincaid-Smith and Barlow, 1959; Weitzman, 1955; Parry and Mounsey, 1961). Recently, an observation relative to the change in contour of the P wave in the electrocardiogram, involving the left atrial component of the P wave, in ischæmic heart disease has been described by Gross (1962).

Separate workers, using several instrumental approaches, have studied præcordial vibrations (Benchimol and Dimond, 1962; Agress and Wegner, 1962; Smith and Kountz, 1941; Rosa and Karsak, 1960; Skinner *et al.*, 1961), and have identified various alterations occurring in patients with angina pectoris. We have studied particularly the low frequency (0.1–20 cycles per second) displacement vibrations at the apex, the apex cardiogram. In ischæmic heart disease and especially after effort, we have uniformly (70 of 76 patients) noted the occurrence of a *large atrial thrust at the apex* (Benchimol and Dimond, 1962).

The purpose of this report is to document the observation that the large atrial component of the apex cardiogram is directly reflecting the end-diastolic rise in left ventricular pressure. We have recorded simultaneous apex cardiograms and left ventricular pressure curves at rest, after exercise, before and after nitroglycerin, before and after venous tourniquets; and have noted a direct relation between the amplitude of the atrial component of the apex cardiogram and the end-diastolic left ventricular pressure. We are referring particularly to angina pectoris, a manifestation of ischæmic heart disease, and not to left ventricular failure, to systemic hypertension, or to non-specific cardiomyopathies. We appreciate that abnormalities occur in these conditions also, but in this paper we are specifically discussing the single entity of effort pain, identified as angina pectoris and associated with ischæmic heart disease. We particularly stress that in this study and in a previous report (Benchimol and Dimond, 1962) we are not confusing angina pectoris with that condition that is usually described as left ventricular failure.

RESULTS

Fig. 1A illustrates the apex cardiogram recorded simultaneously with left intraventricular pressure and the phonocardiogram (mitral). The patient suffered from angina pectoris which was regularly provoked by exercise and relieved by nitroglycerin. The top left-hand tracing was recorded with the patient at rest, supine. Note the distinct fourth heart sound synchronous with the prominent atrial component of the apex cardiogram and the end-diastolic atrial wave form in the left

* Supported in part by grants from Timkin-Sturgis Cardiovascular Research Fund National Heart Institute Research Grants H-5575 and H-5730.

FIG. 1.—(A) Apex cardiogram recorded with intra-left-ventricular pressures. The patient had angina pectoris. The ratio of the “a” wave to the total amplitude of the apex cardiogram was 15 per cent at rest and increased to 32 per cent on exercise. Note that the end-diastolic pressure (at the point of the “a” wave) was 20 before exercise and at the end of exercise was 40 mm. Hg. Note also that the early diastolic pressure was 5 before exercise and increased to 12 after exercise. Note also that atrial systole is transmitted into the ventricle and is recordable. (B) The electrocardiogram, phonocardiogram, apex cardiogram, and intraventricular left ventricular pressure of patient with angina pectoris. Tracing recorded during angina and five minutes after nitroglycerin. Note the very prominent “a” wave (ratio 31%) in the left-hand tracing and the end-diastolic left ventricular pressure of 35 mm. Hg. Five minutes after nitroglycerin tablet notice the complete absence of an atrial component to the apex cardiogram and the diminution of the end-diastolic left ventricular pressure to approximately 7 mm. Hg.

ventricle. The end-diastolic pressure at the moment of atrial systole is 20 mm. Hg. Intraventricular systolic pressure is 160 mm. Hg. In the apex cardiogram the ratio of the "a" wave to the total amplitude of the apex cardiogram is 15 per cent. After mild exercise (raising from the supine 30 times) the following changes became apparent: increased loudness of the fourth heart sound synchronous with an increased atrial component of the apex cardiogram and a rise in the end-diastolic left ventricular pressure to 35 mm. Hg. The systolic intraventricular pressure remained at 160 mm. Hg. The over-all amplitude of the apex cardiogram at rest and after exercise cannot be compared because of adjustments of standard. Note, however, that the ratio of the atrial component to the total amplitude of the apex cardiogram has now increased to 32 per cent.

Fig. 1B demonstrates the effect of nitroglycerin in a patient experiencing angina pectoris. Four parameters were recorded, the præcordial electrocardiogram, the phonocardiogram from the mitral area, the apex cardiogram, and the left intraventricular pressure curve. In the left-hand tracing, the patient is supine and experiencing angina. Note the considerable amplitude of the fourth heart sound, the amplitude of the atrial component of the apex cardiogram ("a" wave to total amplitude ratio equals 31 per cent), and that the end-diastolic pressure in the left ventricle is 35 mm. Hg. The systolic intraventricular pressure is 150 mm. Hg. The right hand lower tracing was obtained five minutes after the sublingual administration of 1/200 grain nitroglycerin tablet. Note the disappearance of the fourth heart sound and the atrial component of the apex cardiogram, the very small atrial activity within the left ventricle, and the fall in the end-diastolic left ventricular pressure to normal. The ratio of the "a" wave to the total amplitude of the apex cardiogram is now 0, and the systolic left ventricular pressure has decreased to 135 mm. Hg.

Table I gives measurements obtained from the apex cardiogram and left ventricular pressure

TABLE I
ISCHÆMIC HEART DISEASE

	"a" wave ratio	LVED* pressure (mm. Hg)	LV early diastolic pressure (mm. Hg)	LV systolic pressure (mm. Hg)	A-E* interval (sec.)	Length of systole (sec.)	Heart rate
Control	24	30	5	133	0.18	0.29	78
After exercise							
30 sec.	28	40	9	142	0.17	0.39	83
1 min.	30	33	5	145	0.19	0.30	76
1½ min.	30	35	5	146	0.19	0.31	73
2 min.	29	36	6	146	0.20	0.30	71
4 min.	22	30	5	135	0.19	0.30	65
Control	29	30	5	141	0.20	0.31	68
After nitroglycerin							
30 sec.	29	28	5	136	0.19	0.31	63
1 min.	26	22	5	135	0.19	0.31	62
2 min.	12	20	5	140	0.17	0.29	63
3 min.	14	11	5	130	0.14	0.31	65
4 min.	—	10	5	120	—	0.30	65
10 min.	10	10	5	112	0.14	0.32	57
Tourniquets on 6 min.	11	10	5	117	0.17	0.28	61
Tourniquets released							
15 sec.	19	20	3	122	0.18	0.29	58
30 sec.	16	20	5	125	0.19	0.29	54
3 min.	14	20	5	122	0.19	0.30	57

*LVED=left ventricular end-diastolic; A-E interval=time interval from beginning of the "a" wave to the E-point of the apex cardiogram.

While the "a" wave ratio does not give absolute values of LVED pressure, it does faithfully reflect the relative value and the direction of change in the LVED pressures. Note also that the "a" wave ratio does not bear such a direct relation to any of the other parameters measured.

curves before and after exercise, before and after nitroglycerin, and during and after venous tourniquets in a patient with classical angina pectoris: (a) control or rest period; (b) 30 seconds, 1 minute, 1½ minutes, 2 minutes, and 4 minutes after exercise; (c) a second period of control observation; (d) 30 seconds, 1 minute, 2 minutes, 3 minutes, 4 minutes, and 10 minutes after 1/200 grain of sublingual nitroglycerin; (e) after 6 minutes with venous tourniquets on all four limbs, and (f) 15 seconds, 30 seconds, and 3 minutes after the sudden release of the four limb venous tourniquets. The ratio of the "a" wave to the total amplitude of the apex cardiogram is expressed in the second column, the left ventricular end-diastolic pressure as recorded by transaortic catheter is listed in the third column; the left ventricular early diastolic pressure obtained in the same manner is listed in the fourth column; the left ventricular peak systolic pressure is listed in the fifth column; the interval of time, expressed in seconds, from the onset of the atrial component of the apex cardiogram to the ejection peak (e-point) is listed in the sixth column; the total length of systole (from the moment of the onset of ejection until the closure of the aortic valve) is listed in the seventh column; and the heart rate is identified in the eighth column. Note the relation between the "a" wave ratio and the end-diastolic left ventricular pressure. For example, during the control period the "a" wave ratio is 24 per cent and the left ventricular end-diastolic pressure is 30 mm. Hg. Ten minutes after a nitroglycerin tablet the "a" wave ratio is 10 per cent and the left ventricular end-diastolic pressure is 10 mm. Hg. Note that a similar effect is obtained from the placement of four limb venous tourniquets, namely an "a" wave ratio of 11 per cent and a left ventricular end-diastolic pressure of 10 mm. Hg. When the venous tourniquets are suddenly released the "a" wave ratio increases to 19 per cent and the left end-diastolic pressure to 20 mm. Hg. Note that left ventricular systolic pressure and heart rate cannot be directly correlated. Note also that the a-E interval and the length of systole have a rough variation but do not correlate as well as the "a" wave ratio and the left ventricular end-diastolic pressure.

CONCLUSION

The relation between the amplitude of the atrial component of the apex cardiogram and the left ventricular end-diastolic pressure suggests that use of this indirect, præcordial procedure may offer a safe, rapid, reproducible, inexpensive method of documenting the presence of end-diastolic left ventricular pressure rise, and may decrease the dependence upon the complicated intraventricular pressure measurements, thus making it easier to study, to recognize, and to treat ischæmic heart disease.

REFERENCES

- Agress, C. M., and Wegner, S. (1962). The vibrocardiographic exercise test for coronary insufficiency. *Amer. J. Cardiol.*, **9**, 541.
- Benchimol, A., and Dimond, E. G. (1962). The apex cardiogram in ischæmic heart disease. *Brit. Heart J.*, **24**, 581.
- Duchosal, P. (1932). A study of gallop rhythm by a combination of phonocardiographic and electrocardiographic methods. *Amer. Heart J.*, **7**, 613.
- Gross, D. (1962). The clinical significance of P waves with delayed ascent. *Amer. Heart J.*, **63**, 497.
- Kincaid-Smith, P., and Barlow, J. (1959). The atrial sound in hypertension and ischæmic heart disease; with reference to its timing and mode of production. *Brit. Heart J.*, **21**, 479.
- Malmberg, R. O., Pernow, B., and Eliasch, H. (1962). Effects of exercise on the cardiopulmonary circulation in patients with coronary heart disease. Abstract-IV World Congress of Cardiology, Mexico City, p. 225.
- Müller, O., and Rørvik, K. (1958). Hæmodynamic consequences of coronary heart disease; with observations during anginal pain and on the effect of nitroglycerin. *Brit. Heart J.*, **20**, 302.
- Parry, E., and Mounsey, P. (1961). Gallop sounds in hypertension and myocardial ischæmia modified by respiration and other manœuvres. *Brit. Heart J.*, **23**, 393.
- Rosa, L. M., and Karsak, N. (1960). Precordial pulsatory mechanism in coronary heart disease. *Circulation*, **22**, 801.
- Ross, R. S., Schwartz, N. Gaertner, R. A., and Friesinger, G. C. (1962). Electrocardiographic and hemodynamic observations during selective coronary cine-angiocardiology. Abstract of 54th Annual Meeting of the American Society for Clinical Investigation, p. 60.
- Skinner, N. S., Leibeskind, R. S., Phillips, H. L., and Harrison, T. R. (1961). Angina pectoris. Effect of exertion and of nitrites on precordial movements. *Amer. Heart J.*, **61**, 250.
- Smith, J. R., and Kountz, W. B. (1941). Total cardiac vibrations in aged hearts and in coronary disease. *Proc. Soc. exp. Biol. (N.Y.)*, **47**, 353.
- Weitzman, D. (1955). The mechanism and significance of the auricular sound. *Brit. Heart J.*, **17**, 70.