Quebec Beer-Drinkers' Cardiomyopathy: Hemodynamic Alterations

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THE clinical, electrocardiographic, radiological and pathological features of the syndrome known as Quebec beer-drinkers' cardiomyopathy have been presented in separate communications.[1-4]

In 13 patients of this series, hemodynamic studies were performed to exclude other structural abnormalities of the cardiovascular system and to demonstrate to what extent the heart muscle was affected. These studies were performed one to four days after admission; every patient had already received digitalis or diuretics in variable amounts. None of the patients were severely ill at the time and all the patients studied eventually survived, compared to a 46.1% mortality for the group.

METHOD OF STUDY

The patients fasted and were each given 10 mg. of diazepam (Valium) intramuscularly one hour before the hemodynamic studies were started; the procedures were all carried out with the patients recumbent.

The studies were carried out by simultaneous retrograde transvenous right and left heart catheterizations. The reference level for central pressures was 10 cm. from the table top, pressures being recorded on an Electronics-For-Medicine Recorder* with Statham (P23G) strain gauges. Systolic and diastolic pressures were averaged over two respiratory cycles. Mean pressures were obtained by electrical integration. Base lines and standardization were checked before and after each recording.

Cardiac output was determined by indicator dilution according to a method we have described elsewhere.[5]

Isoproterenol was infused intravenously at a rate of 2 μg. per minute. Hemodynamic studies were made during the control period and 10 minutes after the infusion was started. Angiotensin infusions were done in the manner proposed by Ross and Braunwald.[6]

The patients exercised on a bicycle ergometer in a supine position at an energy level of 25 watts for 10 minutes.

TABLE I.—PULMONARY CIRCULATION: PRESSURE VALUES IN THE RIGHT ATRIUM, VENTRICLE AND PULMONARY ARTERY

	Right atrial pressure (mm.Hg)	*Right ventricular pressure (mm.Hg)*		*Pulmonary pressure (mm.Hg)*			*Pulmonary resistance (dynes/sec./cm.$^{-5}$)*
	Mean	*Early diastolic*	*Late diastolic*	*Systolic*	*Diastolic*	*Mean*	
A-8	7	0	8	52	7	22	533
A-2	16	0	20	50	18	30	
C-2	0	0	0	46	18	27	
A-14	7	0	20	45	17	25	666
A-5	—1	—5	0	35	15	20	576
A-3	5	5	9	25	5	18	197
A-6	0	—3	0	25	8	13	214
C-1	0	0	3	30	15	21	254

RESULTS

Table I shows pressure values in the right atrium, right ventricle and pulmonary artery. It may be seen that a few patients have normal pressures, but the majority show an elevation either of the systolic or of the diastolic ventricular pressures.

Table II illustrates pressures from the systemic circulation. In every case, the aortic pressures are low, while the end diastolic pressures in the left ventricle are abnormally elevated.

Fig. 1 shows the cardiac index of the patients before and after effort, the normal values for our laboratory being 2.9 ± 0.4. In three patients, isoproterenol infusion did not significantly increase cardiac output.

DISCUSSION

Goodwin[7] has divided cardiomyopathies into three categories: congestive, constrictive and

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TABLE II.—SYSTEMIC CIRCULATION: PRESSURE VALUES IN THE LEFT VENTRICLE AND AORTA.

	Left ventricular pressure (mm. Hg)			*Aortic pressure (mm.Hg)*		*Arterial resistance (dynes/sec./cm.$^{-5}$)*
	Early diastolic	*Late diastolic*	*Systolic*	*Diastolic*	*Mean*	
A-8	16	31	90	60	74	1793
A-14	5	30	90	60	68	1813
A-5	2	30	80	60	65	2080
A-3	0	20	105	65	80	876
A-1	3	16	105	80	100	1600

obstructive. We believe that this classification is unsatisfactory. "Congestion" has different meanings for different investigators, and muscular subaortic stenosis is now a well-known condition that has very little in common with the condition under discussion. We would therefore prefer a classification based on definite hemodynamic parameters and having some clinical import:

First category—patients presenting with an increased cardiac output; the increase may or may not respond to thiamine.[8, 9]

Second category—patients presenting with a normal or decreased cardiac output, which may or may not respond to exercise or isoproterenol.[10] A better evaluation of left ventricular function in these patients may be determined using the method proposed by Ross and Braunwald,[6] i.e. by progressively elevating aortic pressure with infusions of angiotensin and observing the relation of ventricular-filling pressure to stroke-work.

Third category—patients presenting with abnormal right or left ventricular compliance. Right ventricular compliance may be considered abnormal if the end diastolic pressure to systolic pressure ratio is equal to or greater than 1:3. Left ventricular compliance is abnormal if the same ratio is equal to or greater than 1:5.[10]

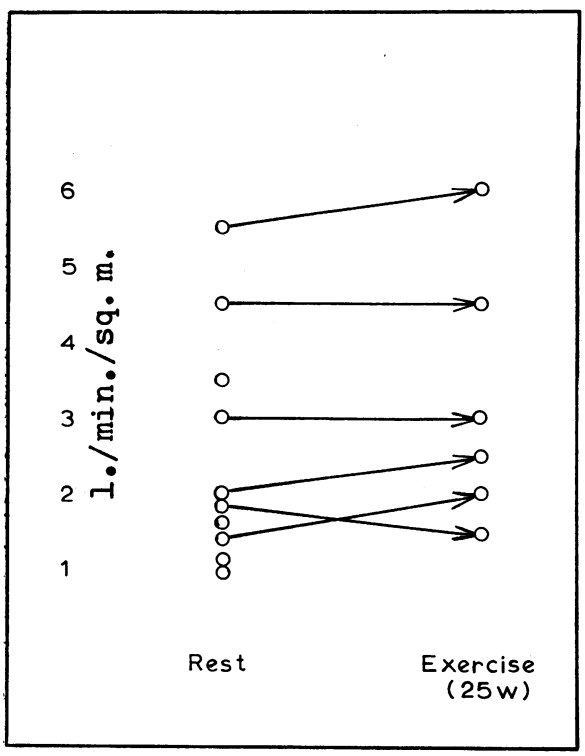

Fig. 1.—Cardiac index of patients before and after exercise (25 watts for 10 minutes) in litres per minute per square metre of body surface.

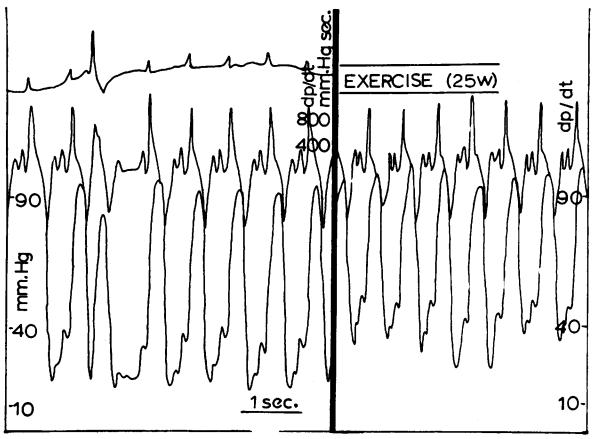

Fig. 2.—Left ventricular pressure pulse and dp/dt before and after exercise at an energy level of 25 watts for 10 minutes. During exercise there is a fall in the stroke-work index.

In our series (Fig. 2), the cardiac index was initially normal or low and did not rise with an exercise which usually doubles this value. Fig. 2 shows that during exercise there is actually a fall in the stroke-work index and there is no rise of the first derivative of the left ventricular systolic pressure (dp/dt) (the first derivative of the left ventricular pressure means the initial rate of increase in pressure within the left ventricle just before the aortic valve opens and after the mitral valve is closed; the measurement is in mm. Hg [pressure dp] over seconds of time [dt].) In spite of this marked decrease in myocardial reserve, a post-extrasystolic improvement in ventricular function is observed.

Of eight patients, three showed decreased right ventricular compliance according to the

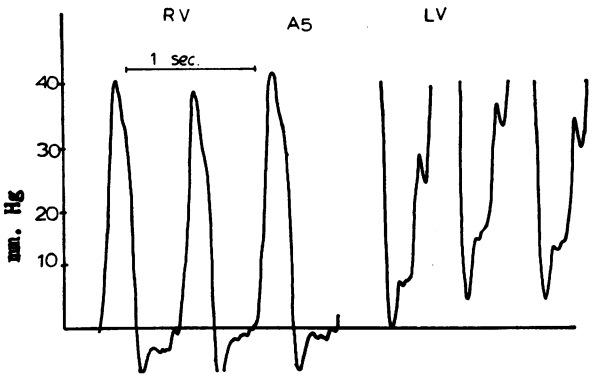

Fig. 3.—Right and left ventricular pressure curves. There is a different degree of compliance in the right and in the left ventricle.

criteria mentioned above. All but one showed decreased left ventricular compliance.

Fig. 3 illustrates, in the same subject, a different degree of compliance in the right and in the left ventricle.

Another frequent hemodynamic feature of this condition was increased atrial activity as shown

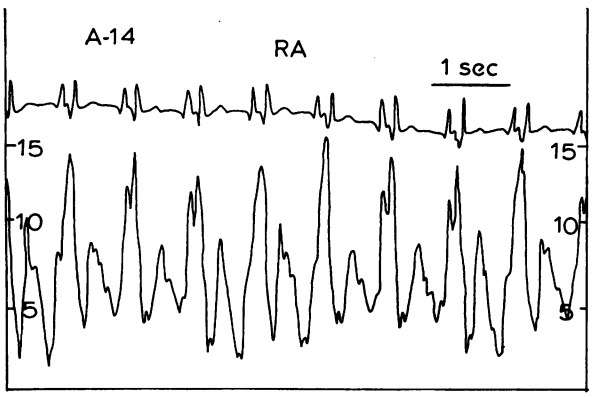

Fig. 4.—Right atrial pressure curve showing giant "a" wave.

by giant "a" waves (Fig. 4), loud fourth heart sounds, and ventricular "atrial kicks", which could at times even be seen in the aortic pressure curves (Fig. 5). This phenomenon probably represents another aspect of decreased myocardial compliance, since, microscopically, the atrial wall was as involved as the rest of the myocardium.

FOLLOW-UP STUDIES

Three patients were re-examined a year after their initial hemodynamic evaluation. They were symptom-free, and the usual cardiovascular investigation showed no abnormalities. These three patients were heavy beer drinkers and had their usual alcoholic intake up to the day preceding

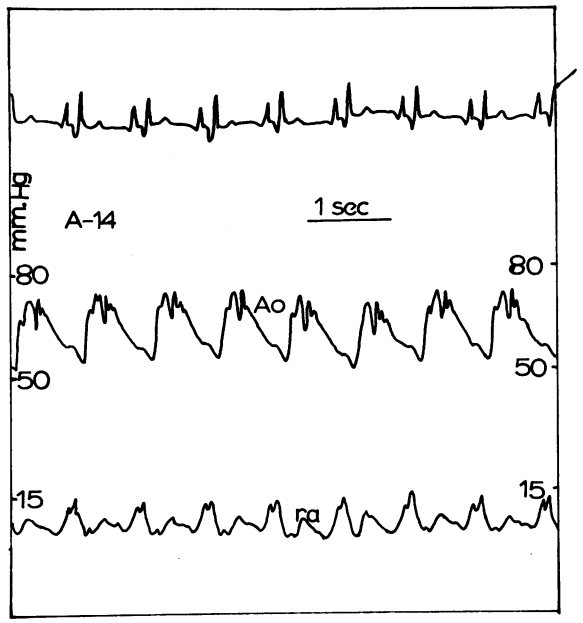

Fig. 5.—Right atrial and aortic pressure curves. The giant "a" wave is seen in the aortic pressure curve.

TABLE III.—RESPONSE OF LEFT VENTRICLE TO EXERCISE DURING THE ACUTE STAGE OF THE DISEASE AND A YEAR AFTER RECOVERY

	1966		1967	
A-8	*Rest*	*Exercise (25w)*	*Rest*	*Exercise (25w)*
Cardiac output (l./min.)	3.0	3.5	4.5	8.4
Stroke output (ml.)	39	33	66	78
Stroke-work index (g./sq.m.)	20.2	13.2	39.3	72.2
Left ventricle dp/dt (mm. Hg/sec.)	720	792	875	1125

the investigation. Table III shows the response of the left ventricle to exercise during the acute stage of the disease and a year after recovery. Two other subjects similarly reinvestigated showed normal left ventricular function after

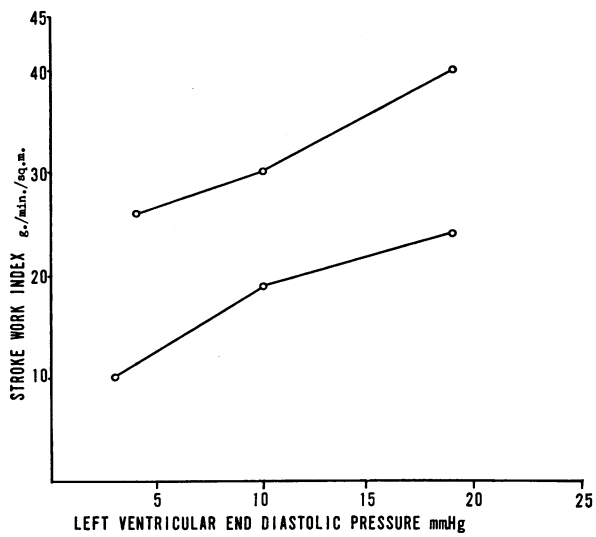

Fig. 6.—Left ventricular function curves during infusion of angiotensin.

exercise by doubling at least their cardiac output and their left ventricular stroke-work. At the follow-up, in two of these patients, stroke-work index and left ventricular end-diastolic pressures were measured during infusion of angiotensin: the left ventricular function curves thus obtained (Fig. 6) show some impairment of myocardial performance and fit into group 2 of Ross and Braunwald.[6] However, Regan *et al.*,[11] applying this technique to chronic alcoholics who clinically had normal cardiovascular findings, obtained identical curves, which may be considered "normal" for the group under study.

Summary Hemodynamic studies performed in 13 patients suffering from Quebec beer-drinkers' cardiomyopathy showed a normal or low cardiac output which failed to rise on exercise and in the majority of cases diminished myocardial compliance.

At follow-up a year later, three patients showed normal left ventricular function at rest and during exercise. During angiotensin infusion, the left ventricular function curves were identical to those found in chronic alcoholics with normal cardiovascular findings.

Résumé Des études hémodynamiques ont été effectuées chez 13 malades souffrant de la cardiomyopathie des buveurs de bière québecois. Ces études ont démontré un débit cardiaque normal ou abaissé, qui n'augmentait pas à l'exercise. On a également pu noter, dans la majorité des cas, une diminution de la compliance myocardique.

Un an après l'épisode initial, trois malades ont démontré, à l'étude hémodynamique, une fonction ventriculaire gauche normale au repos et à l'effort. Au cours de l'infusion à l'angiotensine, la fonction ventriculaire gauche était identique à celle trouvée chez des alcooliques chroniques dont le cœur était cliniquement normal.

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