Indirect Measurement of Left Ventricular End-Diastolic Pressure in Congestive Cardiomyopathy and Constrictive Pericarditis

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SUMMARY

Pulmonary artery diastolic and pulmonary capillary wedge mean pressures were measured in 30 patients with congestive cardiomyopathy and in 30 patients with constrictive pericarditis. These measurements were compared with left ventricular end-diastolic pressure (LVEDP) to assess their value as indirect measurements of left ventricular function. There was close correlation between the indirect measurements and LVEDP in both disorders: a better statistical correlation was achieved using a power curve than with linear regression, since pulmonary artery diastolic pressure increased disproportionately when LVEDP was high.

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Left ventricular end-diastolic pressure (LVEDP) is a measurement of left ventricular function. It depends on ventricular pressure and volume at the beginning of diastole, the volume which fills the ventricle in diastole, and the diastolic pressure-volume curve (a function of ventricular compliance).[1-3] In ventricular dysfunction the LVEDP may be elevated: this causes an increase in left atrial and pulmonary venous pressure, interstitial oedema and shortness of breath.

If pulmonary vascular resistance is normal, there is close correlation between pulmonary artery diastolic, left atrial (or pulmonary capillary wedge) mean and LV end-diastolic pressure.[4-8] Left heart catheterisation may be a dangerous procedure in critically ill patients, but pulmonary artery diastolic and pulmonary capillary wedge pressures are simple to measure, using a flow-directed cardiac catheter.[9] Continuous monitoring of these pressures is useful to estimate LVEDP and to assess its response to therapeutic interventions in critically ill cardiac patients.[10,11] Pulmonary vascular resistance increases in patients with persistent elevation of left atrial and pulmonary venous pressure. Pulmonary arterial hypertension complicates mitral valve disease, ischaemic heart disease and congestive cardiomyopathy: it is less severe in patients with constrictive pericarditis.[12-14]

We have measured the pulmonary arterial diastolic (PADP), pulmonary capillary wedge (PCWP) mean, and left ventricular end-diastolic (LVEDP) pressures in patients with congestive cardiomyopathy (CMO) and constrictive pericarditis (CP), to assess the value of the indirect measurements PADP and PCWP as indices of LV dysfunction in these diseases, and to compare the response of the pulmonary vascular bed to prolonged elevation of left heart filling pressures in the two conditions.

PATIENTS

An unselected group of 30 patients with congestive cardiomyopathy (primary myocardial disease) was studied. The disease is common in South African Blacks, but the aetiology is uncertain.[15-19] The patients had severe cardiac failure without murmurs: 7 patients had additional functional mitral incompetence and another 2 had overt pulmonary thrombo-embolism.

Another consecutive group of 30 patients with constrictive pericarditis was studied before pericardiectomy. The constriction was of tuberculous origin in 27 patients, amoebic in one and post-traumatic in two.

All patients had rested in bed for 2 - 3 weeks before the study and were receiving digitalis and diuretics at the time of cardiac catheterisation. Their disability is summarised in Table I.[20]

METHODS

Cardiac catheterisation was performed in the fasting state. Premedication of 50 mg pethidine and 10 mg diazepam was given. Routine right and left heart studies were made using a percutaneous puncture of the femoral artery and vein, or a cutdown in the right antecubital fossa. The pressures were recorded with Statham P23 Db bonded strain gauges on an Electronics-for-Medicine DR-16 photographic recorder, which has an electronic analogue differentiating circuit with minimal phase lag and distortion. The mid-chest level was used as the zero reference for pressure measurements. Care was taken to debubble the catheter manometer system and the frequency response was flat to 20 Hz. In patients with constrictive pericarditis the pressures were measured over several respiratory

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TABLE I. DISABILITY AT TIME OF CARDIAC CATHETERISATION[20]

	No. of patients	
	Congestive cardiomyopathy	Constrictive pericarditis
Grade 1	2	1
2a	3	7
2b	3	6
3	14	9
4	8	7
Total	30	30

cycles and the mean values calculated. Cardiac output was measured by the direct Fick method.

The procedure was completed with left ventriculography in the right anterior oblique position using a slow injection of 50 ml 76% Urografin.

Critique of Methods

Right and left heart pressures were measured using different catheters, and these were not always of the same length: this may alter the results slightly. The right and left heart pressures were not always recorded simultaneously.

Patients were fully digitalised at the time of study and were receiving diuretic therapy. This had a different effect on haemodynamic measurements in different patients.

RESULTS

The haemodynamic data are summarised in Table II. Mean LVEDP was similar in the two groups of patients, but the range and standard deviation were large in patients with congestive cardiomyopathy. Mean PCWP and PADP were also similar in the two conditions: in 2 patients with cardiomyopathy PADP was greater than 40 mmHg.

TABLE II. HAEMODYNAMIC MEASUREMENTS

	Congestive cardiomyopathy	Constrictive pericarditis
LVEDP	24 ± 12	23 ± 7
PCWP	21 ± 12	20 ± 7
PADP	24 ± 11	21 ± 8

The mean values ± 1 SD are given.

There was significant linear relationship between LVEDP and the 'indirect' measurements PCWP or PADP in congestive cardiomyopathy and in constrictive pericarditis. PADP and PCWP increase disproportionately in patients in whom LVEDP is greater than 15 - 20 mmHg, and this confirms the findings of Kaltman *et al.*[4] (Figs 1 and 2, Table III). A power curve (least-squares fit) gave a higher correlation coefficient than linear regression analysis. There was a linear relationship between PCWP and PADP in congestive cardiomyopathy and in constrictive pericarditis (Fig. 3).

DISCUSSION

There was close correlation between 'indirect' measurements and LVEDP in congestive cardiomyopathy (CMO) and constrictive pericarditis (CP) although the relationship was less exact in CP. In CMO the range of LVEDP, PCWP and PADP was large: in 7 patients LVEDP was greater than 30 mmHg, while in 5 it was less than 10 mmHg, a consequence of bedrest, digitalis and vigorous diuretic therapy before cardiac catheterisation.

In constrictive pericarditis LV diastolic pressure was similar in many patients. The primary defect in CP is mechanical due to external compression of the cardiac chambers, so that the diastolic pressures cannot easily be altered and decreased to the normal range by digitalis and diuretic therapy.[14] Moreover, important pul-

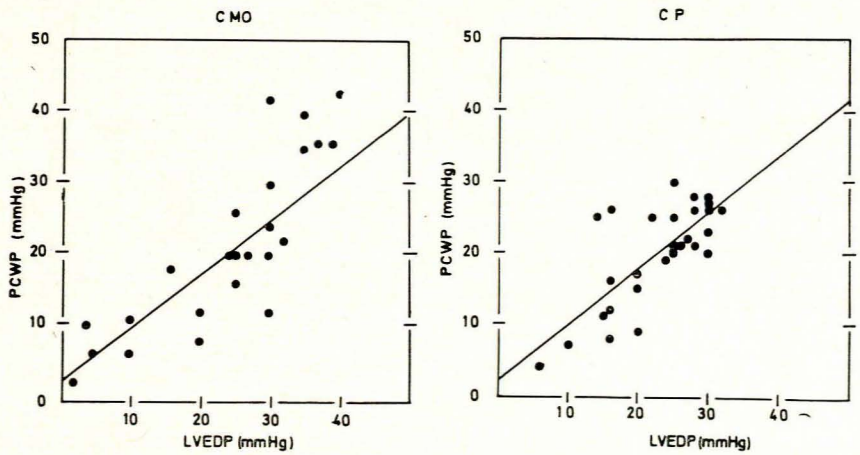

Fig. 1. Linear relationship between LV end-diastolic pressure (LVEDP) and mean pulmonary capillary wedge pressure (PCWP) in congestive cardiomyopathy (CMO) ($y = 1{,}95 + 0{,}77x \pm 7{,}25$; $r = 0{,}79$, $P < 0{,}001$) and in constrictive pericarditis (CP) ($y = 2{,}11 + 0{,}79x \pm 4{,}72$; $r = 0{,}75$ $P < 0{,}001$).

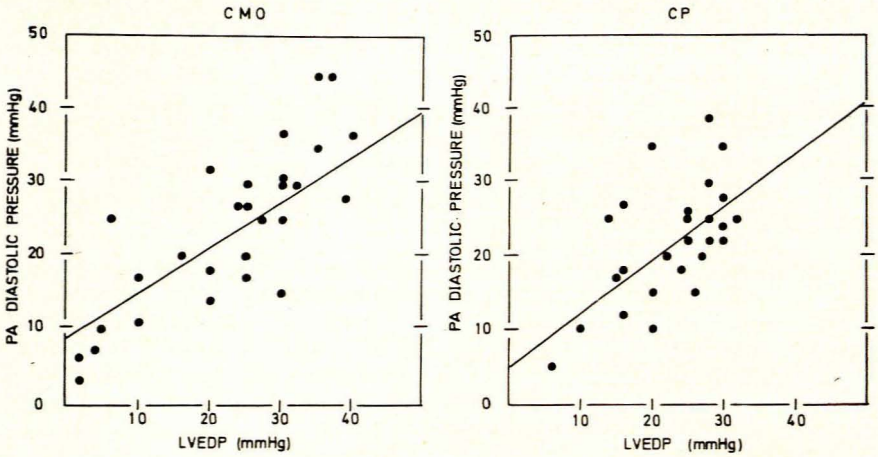

Fig. 2. Linear relationship between LV end-diastolic pressure (LVEDP) and pulmonary artery (PA) diastolic pressure in congestive cardiomyopathy (CMO) (y = 8,74 + 0,64x ± 7,29; r = 0,73, $P < 0,001$) and in constrictive pericarditis (CP) (y = 4,94 + 0,72x ± 6,25; r = 0,62, $P < 0,001$). PA diastolic pressure increases disproportionately in some patients with a high LVEDP.

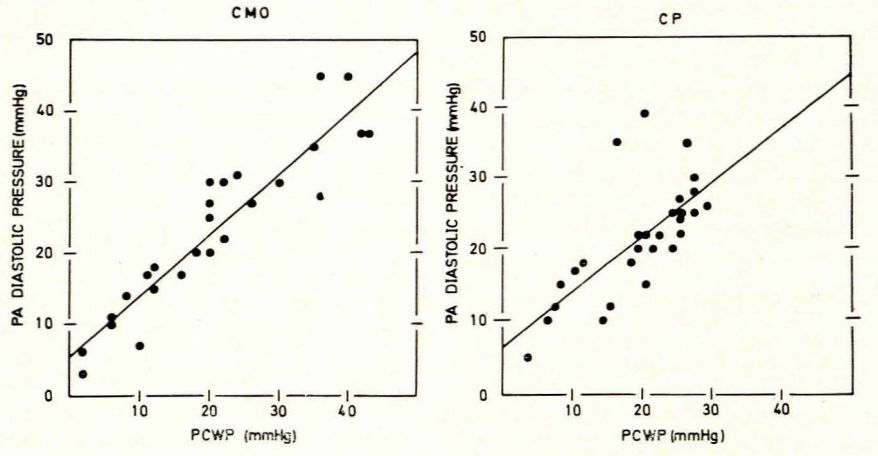

Fig. 3. Linear relationship between mean pulmonary capillary wedge pressure (PCWP) and pulmonary artery (PA) diastolic pressure in cardiomyopathy (CMO) (y = 5,56 + 0,86x ± 4,14; r = 0,93, $P < 0,001$) and in constrictive pericarditis (CP) (y = 5,98 + 0,77x ±5,52; r = 0,70, $P < 0,001$). Three patients with CP had an unusually high PA diastolic pressure and disturbed the statistical analysis.

TABLE III. CORRELATION COEFFICIENTS

		Linear regression $y = ax + b$		Power curve $y = ax^b$	
x	y	CMO	CP	CMO	CP
LVEDP	**PCWP**	**0,79**	**0,75**	**0,90**	**0,82**
LVEDP	**PA diastolic pressure**	**0,73**	**0,62**	**0,85**	**0,74**
PCWP	**PA diastolic pressure**	**0,93**	**0,70**	**—**	**—**

monary arterial hypertension is uncommon in constrictive pericarditis. Right ventricular filling and stroke output are limited, and exercise does not cause a significant increase in pulmonary artery pressure as in mitral valve disease:[21,22] this may protect the pulmonary veins and left atrium so that florid pulmonary oedema is uncommon in CP. LVEDP and left atrial pressure vary with respiration and may decrease by 5 - 10 mmHg on inspiration: this may be

another explanation for the lack of reactive pulmonary arterial hypertension.

Pulmonary capillary wedge mean (PCWP) or pulmonary artery diastolic pressure (PADP) is a useful guide to LV end-diastolic pressure in patients with congestive cardiomyopathy and constrictive pericarditis: serial measurement of right heart pressures may be of value in the management of critically ill patients with these disorders.

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