

Left ventricular dysfunction — bedside Valsalva manoeuvre

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SUMMARY Thirty-seven patients were evaluated before cardiac catheterisation by bedside physical examination, including Valsalva manoeuvre, to assess the value of the sphygmomanometrically determined arterial pressure responses during the Valsalva manoeuvre and to compare its sensitivity, specificity, and predictive accuracy in the detection of left ventricular dysfunction with that of the commonly used diagnostic signs including the chest *x*-ray. Patients not on beta-blockade treatment could be separated into three distinct arterial pressure responses detectable at the bedside which corresponded well to three statistically different groups with regard to left ventricular ejection fraction (0.29 ± 0.11 , 0.48 ± 0.15 , 0.69 ± 0.11) and left ventricular end-diastolic pressure (38 ± 5 mmHg, 24 ± 10 mmHg, 14 ± 5 mmHg) at subsequent cardiac catheterisation. In patients not on beta-blockade it was shown for the first time that (1) the height of the systolic arterial pressure overshoot was directly related to left ventricular ejection fraction and inversely related to left ventricular end-diastolic pressure, and that (2) the bedside sphygmomanometrically determined arterial pressure response during Valsalva manoeuvre provided a semiquantitative estimate of left ventricular function and was unsurpassed in its ability to do so by any of the standard diagnostic signs including the chest *x*-ray film.

Although Weber and not Valsalva first described the circulatory effects of airway straining,¹ the clinical manoeuvre consisting of sustained forced expiratory effort against an obstructed airway (closed glottis or column of mercury) bears the name "Valsalva" after its description by this man almost two hundred years ago.² The normal arterial blood pressure response to the Valsalva manoeuvre is an initial rise associated with the onset of straining (phase 1), followed by a sharp fall to below baseline levels as the straining is maintained (phase 2). Release of strain (phase 3) is followed in the normal subject by a distinct overshoot of the arterial pressure (phase 4) creating a typical sinusoidal response (Fig. 1a). Hamilton first observed that during the straining phase of the Valsalva manoeuvre arterial pressure remained unchanged in patients with congestive heart failure instead of decreasing precipitously as in the

normal.³ Though the bedside application of this manoeuvre for the detection of congestive heart failure was advocated over 20 years ago⁴⁻⁶ haemodynamic studies demonstrating its relative sensitivity and specificity in this regard have been inadequate and perhaps are responsible for the conspicuous absence of this manoeuvre in most textbooks of physical diagnosis.⁷⁻⁹

Recent studies dealing with the usefulness of physical signs in detection of congestive heart failure depend upon a rise in left ventricular end-diastolic pressure obtained during left heart catheterisation to establish both the diagnosis and the degree of left ventricular dysfunction.^{10 11} The concomitant use of ejection indices of myocardial performance such as ejection fraction are considered by many to help substantially in establishing the diagnosis and degree of left ventricular failure.¹²⁻¹⁴

The present report assessed the clinical value of the application of the Valsalva manoeuvre in the

detection of left ventricular dysfunction by investigating (a) the diagnostic accuracy of bedside sphygmomanometric detection of the arterial pressure response when compared with central aortic pressure tracings obtained during cardiac catheterisation; (b) the previously unreported relation between the systemic arterial pressure response and left ventricular ejection fraction; (c) the previously unreported relation between systemic arterial pressure response and left ventricular end-diastolic pressure; and (d) the value of this manoeuvre when applied prospectively at the bedside for the clinical detection of left ventricular dysfunction compared with the routine physical diagnostic signs, including chest radiography.

Patients and methods

Thirty-seven consecutive patients undergoing left heart catheterisation for evaluation of chest pain and/or dyspnoea were chosen for study. Patients with mitral or aortic stenosis, constrictive pericarditis, coarctation of the aorta, and atrial septal defect were excluded by study design since previously reported data¹⁵⁻¹⁷ suggest an abnormal response during the Valsalva manoeuvre in such individuals, possibly related to haemodynamic indices other than congestive heart failure itself. Diagnoses in this group of patients included six normal individuals, 25 with coronary artery disease, one with mitral valve regurgitation, three with aortic valve regurgitation and two with primary myocardial disease with associated mitral regurgitation.

All patients were examined by three physicians (an internist and/or cardiologist, cardiology fellow, and medical resident) within 12 hours before the time of cardiac catheterisation. To maximise the sensitivity of the routine physical signs, their presence during the examination by any of the above physicians was considered diagnostic in the case of that individual patient. The bedside performance of the Valsalva manoeuvre with sphygmomanometric detection of the arterial pressure response was done by a single physician as originally described by Knowles and Gorlin.⁵ Briefly, the patient was instructed how to perform the Valsalva manoeuvre. The systolic blood pressure was routinely obtained while the patient was quietly breathing with normal tidal volumes in the supine position. The cuff pressure was then raised 15 mmHg above the systolic pressure and the patient was asked to perform the Valsalva manoeuvre at the end of a normal inspiratory effort. The effectiveness of the procedure was assessed by noting if the patient had developed a

florid face, distended neck veins, and increased abdominal muscle wall tone. After 10 seconds the patient was instructed to relax his abdomen and resume normal quiet breathing. During the strain phase of the Valsalva manoeuvre and for 15 seconds afterwards the cuff pressure was held inflated 15 mmHg above the previously determined systolic pressure while Korotkoff sounds were sought by auscultation over the brachial artery. Responses were characterised as either (a) sinusoidal, (b) absent overshoot, or (c) square wave (Fig. 1). Auscultation with cuff pressure held inflated 15 mmHg above the previously determined systolic pressure disclosed Korotkoff sounds at the initiation of the straining phase (phase 1) in all three types of pressure responses. In both the sinusoidal and absent overshoot responses these sounds were not perceived during continued later straining. In the square wave response, however, the Korotkoff sounds were heard at this level of systolic pressure for the entire duration of the straining phase. Only those patients with a sinusoidal response had return of Korotkoff sounds at this maintained cuff pressure approximately three to 10 seconds (phase 4 or overshoot) after relaxation (phase 3).

During cardiac catheterisation but before injection of any contrast material, the Valsalva manoeuvre was repeated with the patient sustaining a minimal effective intraoral pressure of 40 mmHg for 10 seconds. The arterial pressure response in the ascending aorta was recorded at a paper speed of 25 mm/s. These responses were analysed with special attention to the height of phases 1 and 4 as well as the presence or absence of a decrease in pulse pressure during phase 2.

End-diastolic pressure was identified as the pressure in the left ventricular chamber after atrial systole (post "a" wave) and before ventricular contraction. In patients in whom no "a" wave existed, end-diastolic pressure was measured 0.05 s after the onset of the QRS complex.¹⁸ Mean pulmonary capillary wedge pressure (\bar{P}_c) was substituted for left ventricular end-diastolic pressure in all three cases with chronic mitral regurgitation, since symptomatology and x-ray findings more often reflect this left ventricular filling pressure in these individuals. In all patients left ventricular end-diastolic pressure or \bar{P}_c was obtained from a series of representative cardiac cycles.

Left ventricular ejection fraction was determined by the single plane area length method¹⁹ as well as by Simpson's rule.²⁰ In all cases agreement between the two methods was excellent. The absolute values reported are those obtained by the area-length method.

Patients on and off beta-blockade treatment were

analysed separately to exclude the effects of propranolol on left ventricular haemodynamics and/or baroreceptor mediated neural reflex arc.

Significant differences between mean left ventricular end-diastolic pressures (\bar{P}_c) and ejection fractions of the various systemic arterial pressure groups were sought using the two-tailed Student's *t* test. Pearson "r" correlation coefficients between certain of the variables were calculated by the raw score method and linear regression equations derived by previously well-described methods.²¹ Differences between correlation coefficients were tested for significance by use of the Fisher transformation "z".²²

Where available, routine posteroanterior and lateral chest *x*-ray films taken within 48 hours of the time of cardiac catheterisation were read independently by two cardiovascular radiologists and graded for the presence of congestive heart failure without knowledge of the catheterisation findings. The grading system was as follows: 1, normal pulmonary blood flow with greater flow to the bases than the apices; 2, equal pulmonary blood flow distribution to the upper and lower lobes; 3, cephalisation of pulmonary blood flow with upper lobe greater than lower lobe flow; 4, alveolar air space oedema. Cardiomegaly was defined as a cardiothoracic ratio > 0.50.

Comparisons among the various physical diagnostic signs in the detection of left ventricular dysfunction were made according to the following definitions:

True positive: physical sign present, LVEDP ≥ 15 mmHg¹¹

True negative: physical sign absent, LVEDP < 15 mmHg

False positive: physical sign present, LVEDP < 15 mmHg

False negative: physical sign absent, LVEDP ≥ 15 mmHg

Sensitivity: true positives/total patients with LVEDP ≥ 15 mmHg

Specificity: true negatives/total patients with LVEDP < 15 mmHg

PV pos (predictive value of presence of a physical sign): true positives/total number in whom physical sign is present

PV neg (predictive value of absence of a physical sign): true negatives/total number in whom physical sign is absent

PE (predictive error): false negatives/total number in whom physical sign is absent

RR (relative risk): PV pos/PE.

Results

(A) CATHETERISATION FINDINGS

The three patterns of arterial pressure response during the Valsalva manoeuvre recorded at the time of cardiac catheterisation are shown in Fig. 1.

Of 16 patients not on propranolol therapy, the "sinusoidal" response (Fig. 1A) was observed in seven patients with a mean left ventricular ejection fraction of 0.69 ± 0.11 and a mean left ventricular end-diastolic pressure of 14 ± 5 mmHg. An "absent overshoot" of arterial systolic pressure < 15 mmHg above baseline,⁵ was seen in four patients with a mean left ventricular ejection fraction of 0.48 ± 0.15 and mean left ventricular end-diastolic pressure of 24 ± 10 mmHg. A "square wave" arterial pressure response (Fig. 1C) was recorded in five patients with a mean left ventricular ejection fraction of 0.29 ± 0.11 and mean left ventricular end-diastolic pressure of 38 ± 5 mmHg. Left ventricular ejection fraction means were significantly different for the square wave vs absent overshoot response ($p < 0.01$), square wave vs sinusoidal response ($p < 0.001$), and absent overshoot vs sinusoidal response ($p < 0.005$). Left ventricular end-diastolic pressure means were significantly different for the square wave vs

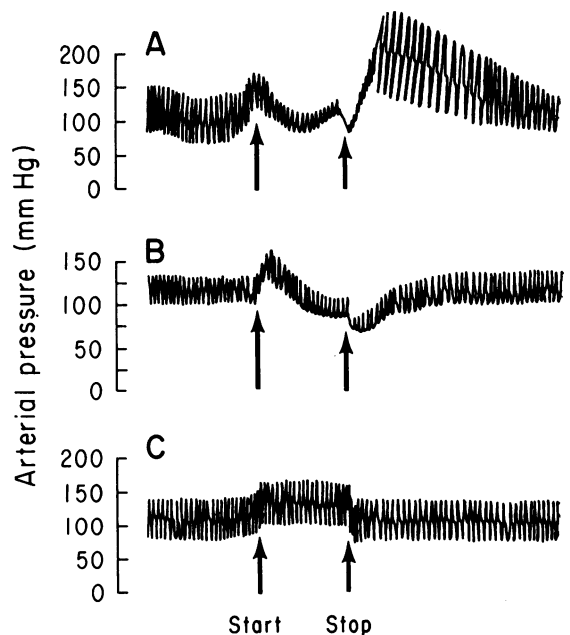


Fig. 1 Arterial pressure responses during Valsalva manoeuvre. (A) Sinusoidal arterial pressure response; (B) absent overshoot arterial pressure response; (C) square wave arterial pressure response.

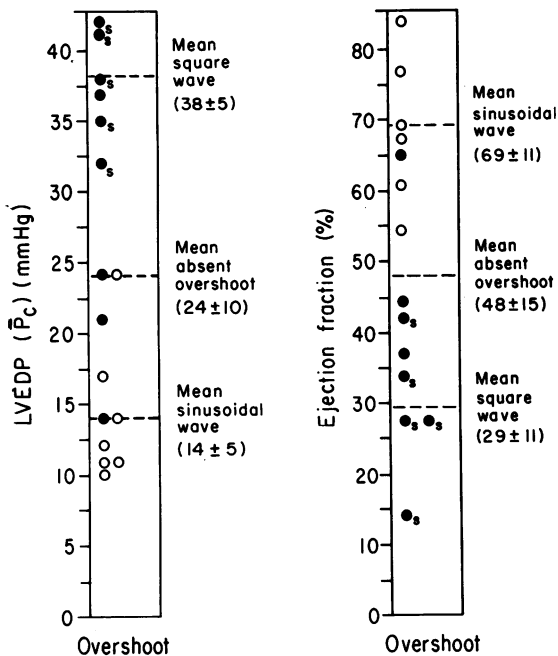


Fig. 2 Left ventricular ejection fraction and left ventricular end-diastolic pressure (LVEF and LVEDP) vs arterial pressure response during the Valsalva manoeuvre. ○, sinusoidal arterial pressure response; ●, absent overshoot arterial pressure response; ●_s, square wave arterial pressure response.

sinusoidal response ($p < 0.001$) and absent overshoot vs sinusoidal response ($p < 0.05$) (Fig. 2).

In a similar group of patients it has been previously shown that airway pressure during straining approximates closely to intraoesophageal pressure¹⁶ and that the latter during initial straining approximates to the magnitude of the early systolic arterial pressure rise (phase 1).²³ In our study there was no significant correlation between the degree of systolic arterial pressure initially generated during the Valsalva manoeuvre (magnitude of phase 1) and the height of the overshoot (magnitude of phase 4), provided a sufficient increase in intrathoracic pressure had taken place (intraoral pressure > 40 mmHg for 10 seconds). The height of the pressure overshoot was, however, related directly to the magnitude of the left ventricular ejection fraction ($r = 0.72$) (Fig. 3) and inversely to the magnitude of the left ventricular end-diastolic pressure ($r = -0.79$) (Fig. 4).

In 21 patients receiving propranolol no significant correlation between left ventricular ejection fraction or left ventricular end-diastolic pressure and the height of the systolic arterial pressure overshoot was found (Fig. 5). Though propranolol did appear to decrease the level of overshoot, an overshoot > 15 mmHg was always associated with a left ventricular ejection fraction > 0.50 and left ventricular end-diastolic pressure < 24 mmHg in all patients studied regardless of the aetiology of

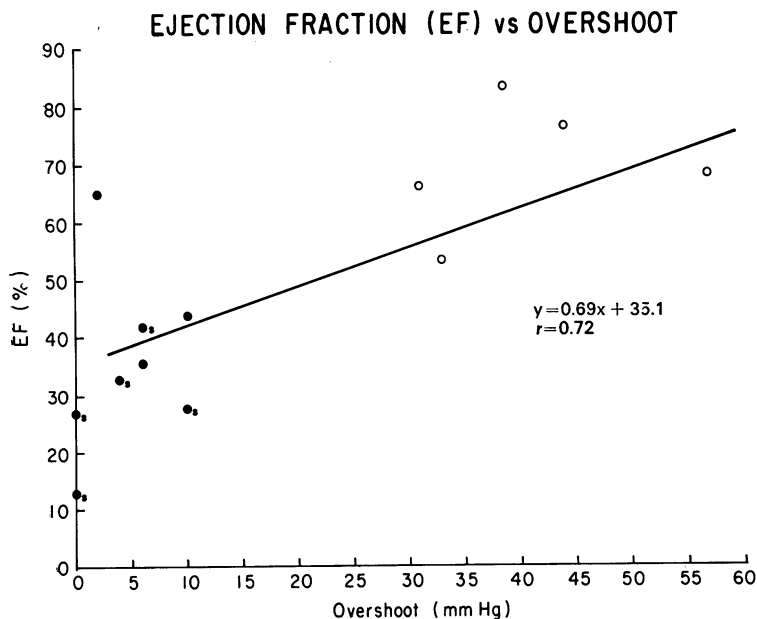


Fig. 3 LVEF vs height of systolic arterial pressure overshoot. ○, sinusoidal arterial response; ●, absent overshoot arterial pressure response; ●_s, square wave arterial pressure response.

their heart disease and the presence or absence of beta-blockade treatment.

No patient in this series suffered angina pectoris, syncope, or ST-T segment alterations during the Valsalva manoeuvre.

(B) BEDSIDE VALSALVA FINDINGS

The sphygmomanometrically observed arterial

pressure response during the Valsalva manoeuvre accurately predicted the response recorded at catheterisation in (7/7), (3/4), and (4/5) patients with the sinusoidal, absent overshoot and square wave responses, respectively. A single case of absent overshoot response at catheterisation was misclassified clinically as sinusoidal. Similarly a case with square wave arterial pressure pattern

Fig. 4 LVEDP vs height of systolic arterial pressure overshoot. \circ , sinusoidal arterial pressure response; \bullet , absent overshoot arterial pressure response; \bullet_s , square wave arterial pressure response.

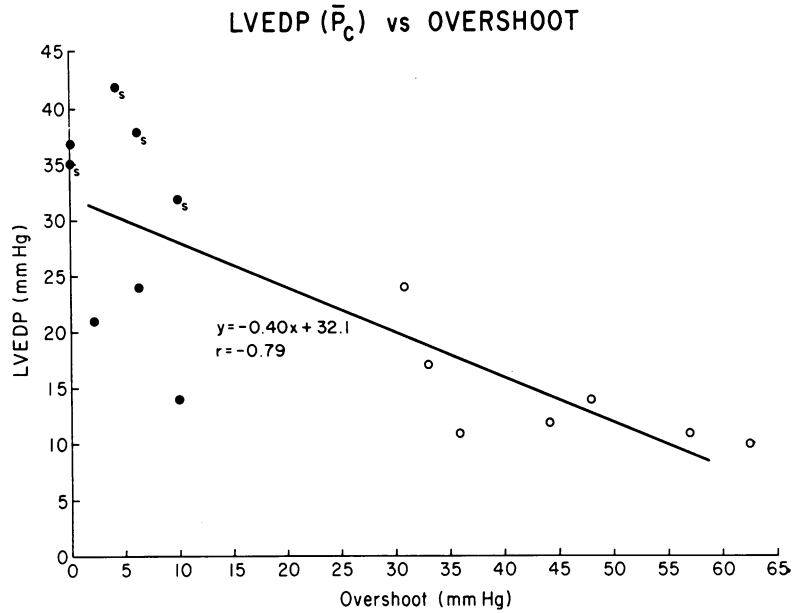
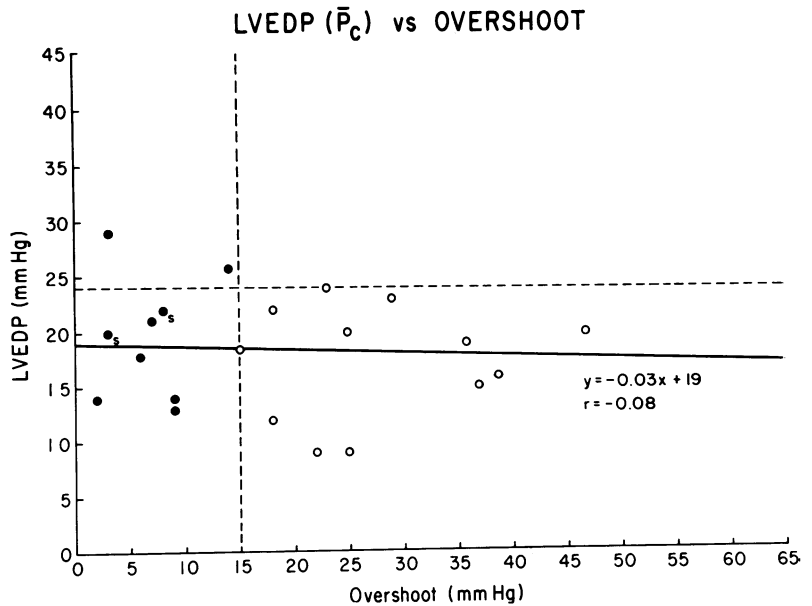


Fig. 5 LVEDP vs height of systolic arterial pressure overshoot (patients on propranolol). \circ , sinusoidal arterial pressure response; \bullet , absent overshoot arterial pressure response; \bullet_s , square wave arterial pressure response.



was clinically diagnosed as absent overshoot response.

Râles (20%, 5/25), ventricular gallop sounds (S_3G) (16%, 4/25), hepatojugular reflux (20%, 5/25), jugular venous distension (20%, 5/25), and peripheral oedema (8%, 2/25) were extremely insensitive indicators of raised left ventricular end-diastolic pressure (\bar{P}_c) (Fig. 6). An atrial gallop sound (S_4G), while sensitive (68%, 17/25), suffered from relative lack of specificity (50%, 6/12) in this regard (Table).

Fig. 6 also shows the data from posteroanterior and lateral chest radiography in this group of

patients. Five of six patients with either cephalisation of pulmonary blood flow or alveolar air space oedema also had cardiomegaly. Four of six of these patients possessed square wave arterial pressure responses during the Valsalva manoeuvre.

No patient in this series had angina pectoris or syncope during the bedside performance of the Valsalva manoeuvre. Subsequent to analysis of these data, 25 additional patients with documented coronary artery disease and stable angina pectoris, who underwent the bedside Valsalva manoeuvre as previously described, had no development of chest pain during its execution.

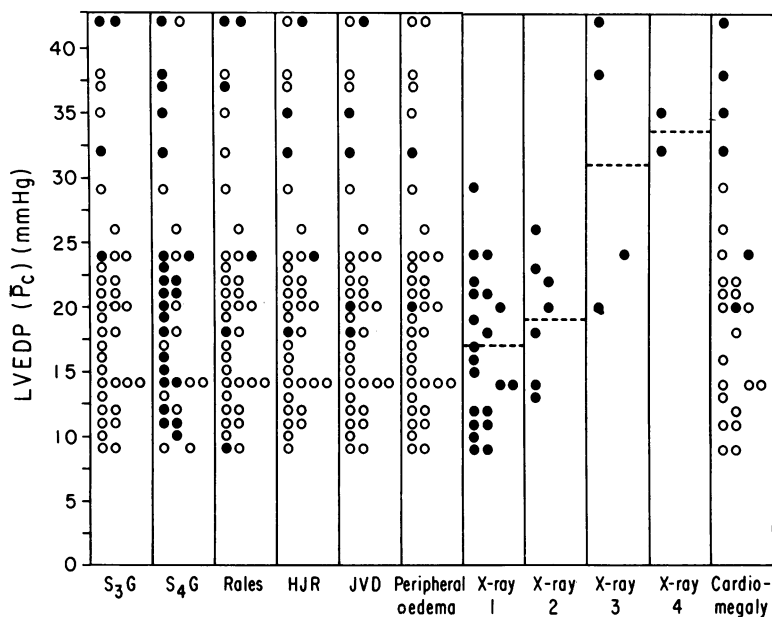


Fig. 6 Physical examination and x-ray findings in patients with various LVEDPs; symbols in corresponding positions in various columns relate to same individual. ●, presence of physical sign or x-ray finding; ○, absence of physical sign or x-ray finding.

Table Physical and radiological findings in left ventricular dysfunction

Sign	True +	True -	False +	False -	Sens	Spec	PVpos	PVneg	RR
S_3G	4	12	0	21	16 (31)	100 (95)	100	36	1.6
S_4G	17	6	6	8	68	50	74	43	1.3
Râles	5	11	1	20	20 (13)	92 (91)	83	35	1.3
HJR	5	12	0	20	20 (17)	100 (91)	100	38	1.6
JVD	5	12	0	20	20 (10)	100 (97)	100	38	1.6
Peripheral oedema	2	12	0	23	8 (10)	100 (93)	100	34	1.5
Non-sinusoidal sphygmomanometer, no propranolol	8	6	0	2	80	100	100	75	4.0
Chest x-ray Class 2, 3, 4 pulm flow	12	9	2	11	50	82	85	45	1.5
Chest x-ray cardiomegaly	6	9	0	11	35 (62)	100 (67)	100	45	1.8

() data reproduced from Harlan *et al.*¹¹ See Methods section of text for correlation definitions.

Discussion

The Valsalva manoeuvre is generally divided by most cardiac physiologists into four more or less well-defined phases: phase 1—onset of straining with its associated arterial pressure rise; phase 2—straining; phase 3—release of strain; and phase 4—arterial pressure overshoot (Fig. 1A). Studies in man using the pressure-gradient technique have shown that in normal subjects the arterial pressure response during the four phases of Valsalva is the result of an acute increase in intrathoracic pressure (phase 1); decreased stroke volume secondary to decreased venous return and compensatory rise in peripheral vascular resistance with narrowing of pulse pressure (phase 2); an acute decrease in the level of intrathoracic pressure (phase 3); and a distinct increase in stroke volume over control level with concomitant decrease in peripheral vascular resistance (phase 4).²⁴ M-mode echocardiographic studies have shown reduced left atrial size,²⁵ and reduced left ventricular internal dimension in systole and diastole with a resultant fall in stroke volume during phase 2.^{25 26} Angiographic methods have also shown decreased left ventricular end-diastolic volume and stroke volume during phase 2.²⁷ Echocardiography has failed, however, to document an increase in stroke volume during phase 4 above that of control levels.²⁵ Phases 2 and 4 are associated with a relative tachycardia and bradycardia, respectively.

Experimental work in the dog^{28 29} has yielded results consistent with the hypothesis that the normally present overshoot (phase 4) is the result of reflex sympathetic activity engendered by the hypotension present in several circulatory baroreceptors, including the carotid sinuses during the period that venous return is impaired.³⁰ Arterial baroreceptor hypotension during phase 2 causes decreased carotid sinus nerve stimulation with enhanced alpha and beta sympathetic efferent traffic via the cardiac sympathetic nerves^{31 32} resulting in tachycardia (beta), enhanced contractility (beta), and increased peripheral vascular resistance (alpha).^{33 34}

Physiological events are different in patients with left ventricular dysfunction and the square wave arterial pressure response during the Valsalva manoeuvre. Stroke volume increases initially (phase 1) and then continues to remain raised over control levels during the remainder of the strain period (phase 2). Immediately before phase 3 there is a fall in stroke volume and a rise in peripheral vascular resistance.³⁵ The relative tachycardia and bradycardia of phases 2 and 4, respectively,

are also conspicuously absent in this group of patients.²⁶

Previous studies in people dealing with the value of the Valsalva manoeuvre in the detection of congestive heart failure have been haemodynamically limited and have not dealt with the question of relative sensitivity and specificity when compared with the other physical diagnostic signs. Though Gorlin presented right heart catheterisation data in 78 patients, there were only seven presumed normal controls and six patients with presumed coronary artery disease in his series.¹⁶ In addition perhaps 42 of 78 possessed cardiac lesions which, independent of associated congestive heart failure, might have been responsible for the abnormal circulatory response noted. As detailed previously, data from these patient groups were not included in our analysis. Moreover, of the remaining 36 patients in Gorlin's series, 13 had recordings of only the right atrial pressure as an estimate of the degree of "heart failure". In two other reports of haemodynamic studies using right heart catheterisation techniques, the arterial response to the Valsalva manoeuvre could be attributed to the presence or absence of congestive heart failure in only 17 patients³⁶ and 10 patients.²³

Results of this study suggest for the first time that (1) the height of the arterial overshoot may be directly related to the magnitude of the left ventricular ejection fraction and inversely related to (\bar{P}_c) left ventricular end-diastolic pressure and (2) the clinical value of the bedside sphygmomanometrically determined arterial pressure response in assessing the degree of left ventricular dysfunction in patients not on propranolol is unsurpassed by any of the standard diagnostic signs including the chest x-ray film. The Table summarises the sensitivity, specificity, positive and negative predictive accuracies, and relative risk for left ventricular dysfunction of the various diagnostic signs based upon a left ventricular end-diastolic pressure (\bar{P}_c) ≥ 15 mmHg¹¹ at rest. Excellent agreement was noted between our series and that of Harlan and associates¹¹ regarding the ability of the standard physical signs to indicate left ventricular failure. Thus the superiority of the bedside Valsalva manoeuvre for detecting left ventricular failure in our study was not merely secondary to poor clinical acumen by our physicians in the recognition of the conventional signs on physical examination.

The ability to separate patients not on beta-blockade treatment into three systolic arterial pressure response groups during Valsalva at the bedside was confirmed, and it was determined for the first time that such clinically detectable re-

sponses corresponded to three statistically different groups of individuals with regard to left ventricular ejection fraction (0.29 ± 0.11 , 0.48 ± 0.15 , 0.69 ± 0.11) and left ventricular end-diastolic pressure (38 ± 5 mmHg, 24 ± 10 mmHg, 14 ± 5 mmHg).

Our clinical data derived from patients on propranolol treatment suggest that beta-adrenergic antagonists may alter the relation between left ventricular end-diastolic pressure (or left ventricular ejection fraction) and the height of the phase 4 overshoot (Fig. 5). Though a significant percentage of patients in this study was receiving propranolol, an overshoot greater than 15 mmHg, regardless of treatment was always associated with a left ventricular ejection fraction >0.50 and left ventricular end-diastolic pressure <25 mmHg. Thus the absence of a clinically detectable overshoot (<15 mmHg) is non-diagnostic in patients on propranolol, but the presence of such an arterial overshoot excludes the possibility of moderate to severe left ventricular dysfunction.

Experimentally, norepinephrine (alpha-agonist) infusion will diminish the extent of the arterial overshoot in normal subjects.²⁹ Recent clinical studies disclose increased plasma concentrations of norepinephrine in patients with congestive heart failure.³⁷ Thus the absent overshoot response during Valsalva manoeuvre in patients with raised left ventricular end-diastolic pressure and diminished left ventricular ejection fraction may be related to plasma norepinephrine levels. Moreover, the direct and inverse correlations shown between the height of the systolic arterial pressure overshoot and left ventricular ejection fraction and left ventricular end-diastolic pressure respectively, may be a function of plasma norepinephrine concentrations which are proportional to the degree of congestive heart failure.³⁷ The almost immediate altered response of certain patients during the Valsalva manoeuvre to acute postural changes¹⁶ suggests, however, that the absent overshoot is the result of peripheral vasoconstriction leading to a relative expansion of pulmonary blood volume²⁹ and pulmonary extravascular water volume,³⁸ the latter being related to hydrostatic pulmonary capillary wedge pressure by the Starling hypothesis for capillary filtration.

Syncope while in the supine position, at least in normal subjects at rest, is rare during the performance of the Valsalva manoeuvre. In the presence of respiratory alkalosis, however, with its associated cerebral vasoconstriction, the decreased cardiac output and mean aortic blood pressure which occurs during late phase 2 and phase 3³⁴ may be sufficient to produce syncope even while supine in

approximately 50 per cent of normal subjects.³⁹ In our experience with over 60 patients at rest, we have not encountered a single syncopal episode. This may be attributable to the purposeful time limitation of the straining phase in our patients by the accompanying physician. In addition the greatest risk for respiratory alkalosis during basal conditions would be present in those with the most severe left ventricular dysfunction. Such patients showed a square wave arterial pressure response during Valsalva, raising and not lowering both cardiac output and mean aortic blood pressure during the period of strain (phase 2).³⁵

Although some have questioned the safety of the Valsalva manoeuvre in patients with coronary artery disease,⁴⁰ others have reported no adverse clinical effects and even occasional relief of angina pectoris when performing this manoeuvre.⁴¹ The effects of this manoeuvre upon myocardial oxygen consumption as well as coronary arterial blood flow will vary with the type of arterial pressure response considered. In this regard, at least three of Levine *et al.*'s⁴¹ six patients who experienced symptomatic relief from their anginal attacks during the Valsalva manoeuvre possessed normal sinusoidal arterial pressure responses.

Although conditions other than left ventricular dysfunction such as prerenal azotaemia or haemorrhage (decreased intravascular volume),²⁸ and idiopathic orthostatic hypotension⁴² may also cause an absent overshoot arterial pressure response, these conditions can usually be differentiated from failure on the basis of the clinical history.

Until further data are available, we must conclude that this procedure may not be completely innocuous in patients with acute coronary heart disease both because of haemodynamic considerations as regards coronary artery blood flow and possibly because of the associated increases in vagal tone which may be arrhythmogenic under such circumstances.^{43 44} In patients with coronary artery disease, stable angina pectoris, and a wide range of left ventricular ejection fractions (0.21 to 0.71) and end-diastolic pressures (10 to 42 mmHg), however, it has been our experience that performance of this manoeuvre by the patient in the presence of a physician is simple, safe, and can be readily accomplished by almost all individuals with the proper instruction. Moreover, bedside sphygmomanometric assessment of the arterial pressure response during Valsalva far surpasses all other routinely available bedside diagnostic signs in the detection of left ventricular dysfunction and provides in certain subjects a reliable estimation of its severity.

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