Alterations in left ventricular volumes induced by Valsalva manoeuvre[1,2]

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Five patients were studied with left ventriculography during different phases of the Valsalva manoeuvre. Small doses of contrast medium allowed adequate repetitive visualization of the left ventricle for volume calculation. During strain phase, the volume of the left ventricle decreased by nearly 50 per cent in each case, and stroke volume and cardiac output also dropped strikingly. Release of straining was attended by a sharp rebound of left ventricular volume to control levels, with a transient surge of increased cardiac output 42 per cent above that of the resting state.

The Valsalva manoeuvre, when forcefully performed, produces transient but dramatic alterations in venous return, stroke volume, and arterial pressure (Stone, Lyon, and Teirstein, 1965; Goldblatt et al., 1963). The various phases of the Valsalva manoeuvre as defined by Hamilton are associated with well-delineated haemodynamic alterations (Stone et al., 1965; Goldblatt et al., 1963; Hamilton, Woodbury, and Harper, 1936). Phase 1, corresponding to the initiation of strain, results in a transient rise in arterial pressure and a rapid decline in venous return. Phase 2, representing the entire duration of the strain phase, has been correlated with falling arterial pressure and increasing heart rate. During the latter portion of this phase, a gradual increase in blood pressure occurs. Phase 3, the abrupt release of the Valsalva manoeuvre, produces a sudden fall of arterial blood pressure and rapid rise of venous return, at a time when the heart rate remains raised. Phase 4 corresponds to the rising blood pressure and pulse pressure seen as the cardiac output increases due to the surge of venous return. In normal subjects, arterial blood pressure and pulse pressure exceed control values, and a reflex bradycardia ensues (Stone et al., 1965; Hamilton et al., 1936).

Although these classic phases for the circulatory adaptations to the Valsalva manoeuvre have been defined precisely in animals, several measurements have not been feasible in man. For example, previous studies have not reported direct measurement of the alteration of left ventricular volumes during the Valsalva manoeuvre. Techniques for measurement of stroke output and volume measurements during non-steady-state conditions have not generally been available for the study of transient cardiovascular alterations. Approximations have been reported, using the pressure gradient technique (Greenfield et al., 1967), or by evaluating changes in overall cardiac size using metallic epicardial markers (Goldblatt et al., 1963). At Stanford University Medical Center, the development of an angiographic technique for repeatedly measuring left ventricular volume during the course of an intervention in man permitted study of the Valsalva manoeuvre in detail. Thus, the present study was undertaken using this direct angiographic method to measure transient changes in left ventricular chamber size and stroke volume during phases of the Valsalva manoeuvre.

Patients and methods

Five patients with angina pectoris, including one (Case 1) with left ventricular dyskinesis and mild compensated congestive heart failure, and one (Case 2) with subsequent normal coronary arteriograms, were studied during diagnostic cardiac catheterization (Table 1). The one patient with congestive heart failure was receiving digoxin at the time of the study, and the remaining four

TABLE I   *Clinical profile of 5 patients with resting LVEDP before Valsalva manoeuvre*

Case No.	Age	Sex	Clinical diagnosis	Control LVEDP* (mmHg)
1	50	M	Coronary artery disease, angina, mild congestive heart failure	27
2	59	F	Coronary artery disease, angina	10
3	68	M	Coronary artery disease, angina	8
4	39	M	Coronary artery disease, angina	12
5	53	M	Coronary artery disease, angina	8

LVEDP = left ventricular end-diastolic pressure.
* Resting LVEDP before any Valsalva manoeuvre.

were receiving no medication. Informed consent was obtained from each patient. All patients were fasting and were premedicated with quinalbarbitone, 100 mg intramuscularly. A 125 cm No. 7 Cournand or Goodale-Lubin catheter was positioned in the pulmonary artery. The end-hole of a 90 cm Cook polyethylene 5·2 J catheter was positioned in the descending aorta. Pressures were recorded using a Statham Model P23Db transducer and a photographic multichannel recorder. A 110 cm Cook polyethylene 6·7 angio-catheter was positioned near the apex of the left ventricle. Serial injections of 76 per cent meglumine and sodium diatrizoate were made during successive phases of the Valsalva manoeuvre (Fig. 1). A model IU4A Ensco programmable injector introduced contrast under a pressure of 630 lb/in². Injections were generally initiated 0·2 sec after the patient's QRS and were terminated 0·45 sec later. Using this pressure and this catheter, approximately 9 to 12 ml

contrast were injected into the left ventricle during a single mechanical diastole. The time and duration of the injection were indicated on the strip-chart record by means of a syringe position marker.

Patients were studied in the supine position in the posteroanterior projection. End-diastolic and end-systolic volumes were calculated by either manual planimetry of tracings drawn from 35 mm cine film or by a Hewlett-Packard Model 2115A computer, using on-line data derived from a Tektronics Model 4551 light-pen tracing of the video image of the opacified left ventricular chamber. The angiogram was initially recorded on a video-disc recorder at 15 frames per second, and re-displayed on a video monitor for light-pen analysis (Alderman et al., 1973). End-diastolic frames were identified by noting the position of a QRS-triggered mechanical event marker superimposed on the angiographic field. Volumes were not calculated for any beats showing prematurity or aberrancy. Independent cardiac output measurements were obtained during the basal condition, using either standard Fick or indicator-dilution methods.

Patients performed the Valsalva manoeuvre by exhaling orally from a full inspiration against a resistance device which included a manometer for monitoring intra-oral pressure. Nose clips blocked nasal leakage of air. A 40 mmHg pressure was achieved almost instantly, maintained for 12 to 15 seconds, and then abruptly released.

There were no untoward complications from these angiographic procedures. Though multiple angiograms were obtained during one or two Valsalva manoeuvres, the necessity for high quality studies free of premature beats limits the data to those visualizations obtained during the mid-strain and release phases. These data were obtained in three patients during a single Valsalva manoeuvre, and in two patients from two separate, but
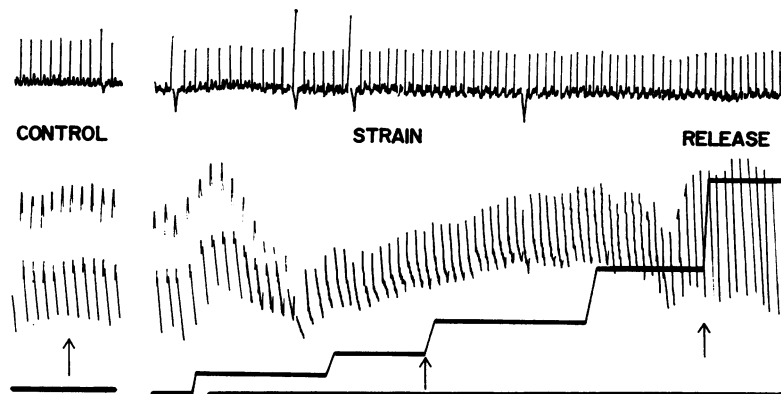

FIG. I   *Shown from above down are the electrocardiogram, aortic pressure trace, syringe injection marker, and zero pressure baseline during a typical Valsalva manoeuvre. This record was made during the study of Case 2. The upward steps of the syringe position indicator mark the time of separate left ventricular angiograms. Vertical arrows indicate the angiograms used for volume calculation.*

identically performed, manoeuvres. All statistical tests were performed using the paired t test, with significance defined at a P level of 0·05.

Results

Fig. 1 shows the record obtained from Case 2 during a single Valsalva manoeuvre of 30 seconds' duration. Five individual angiograms (indicated by the upward steps in the figure) were performed during this particular Valsalva manoeuvre, in addition to the control resting angiogram. The arrows on the figure indicate which angiograms were used for volume calculation in this study. Representative angiograms from which calculations were made are shown in Fig. 2. All five patients had comparable alterations of ventricular volumes during the strain and release phases of the Valsalva manoeuvre, as shown in Table 2.

Validation of the accuracy of the present technique of angiography in calculating left ventricular volumes was accomplished by comparison of stroke

volumes determined by Fick or indicator dilution methods during the control condition with those measured angiographically. Angiographic stroke volumes yielded a maximum variation of ±35 per cent, with mean variation of 5 per cent from those determined independently.

A summary of the means of the aortic pressure, pulse pressure, and heart rate response of all five patients during Valsalva manoeuvre is shown in Fig. 3, and the individual values are listed in Table 2. During the strain phase, the systolic blood pressure moved downwards, returning during the release phase towards normal in three of the five patients. Pulse rate rose steadily during strain and was still raised above control levels during relaxation.

During strain, mean end-diastolic volume declined nearly 50 per cent and returned by release to control levels (Fig. 4). In similar fashion, endsystolic volume declined during strain phase, but unlike end-diastolic volume did not return to pre-

TABLE 2 *Measured and calculated haemodynamic and volumetric data of each of 5 patients*

	Case No.	Heart rate	Stroke volume (ml)	Cardiac output (l./min)
Independent cardiac output				
	1	83	51	4·3
	2	81	74	6·0
	3	63	60	3·8
	4	75	72	5·4
	5	68	66	4·5
	Mean	64	65	4·8

| Case No.	Heart rate	End-diastolic	End-systolic	Stroke	Systolic	Diastolic	Pulse	Cardiac output (l./min)	Ejection fraction
		Left ventricular volumes (ml)			*Aortic pressures (mmHg)*				
Angiographic control									
1	80	228	165	63	98	66	32	5·0	0·28
2	90	87	20	67	166	88	78	6·0	0·77
3	70	113	64	49	158	76	82	3·4	0·43
4	75	160	86	74	106	68	38	5·6	0·46
5	77	83	34	49	122	66	56	3·8	0·59
Mean	78	134	74	60	130	73	57	4·8	0·51
Strain or phase 2 of Valsalva									
1	94	165	146	19	93	74	19	1·8	0·12
2	105	45	13	32	124	87	37	3·4	0·71
3	77	49	37	12	100	80	20	0·9	0·24
4	76	84	43	41	118	84	34	3·1	0·49
5	86	47	15	32	97	74	23	2·8	0·68
Mean	88	78	51	27	106	80	27	2·4	0·45
Release or phase 4 of Valsalva									
1	102	233	139	94	70	48	22	9·6	0·40
2	128	82	13	69	174	96	78	8·8	0·84
3	72	100	48	52	124	84	40	3·7	0·52
4	82	164	77	87	110	70	40	7·1	0·53
5	103	75	26	49	106	60	46	5·0	0·65
Mean	97	131	61	70	117	72	45	6·8	0·59
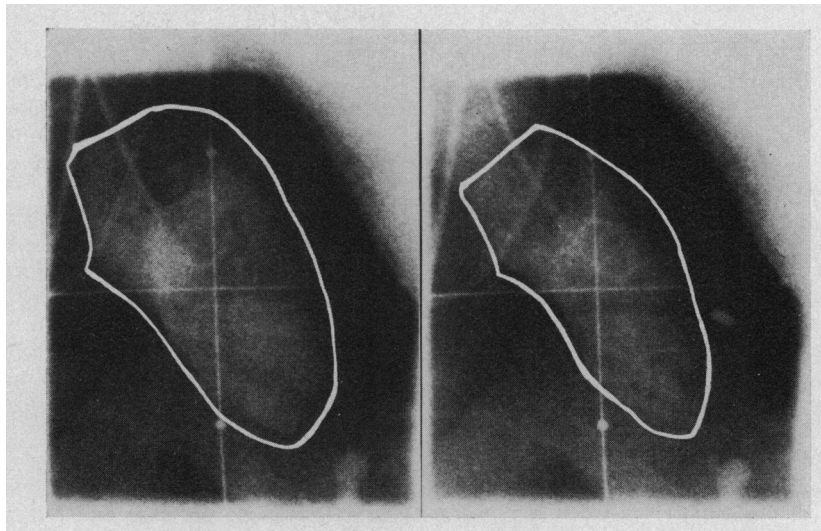
FIG. 2   *Representative angiograms from which volume calculations were made. The angiogram on the left is at end-diastole, that on the right at end-systole.*
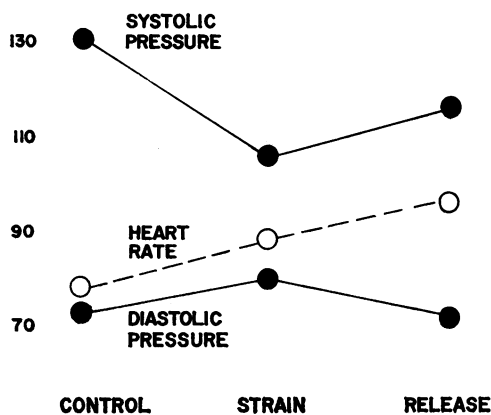

FIG. 3   *Depicted above are the means of the five patients' aortic systolic and diastolic pressures and heart rates during control, strain (phase 2), and release (phase 4) of the Valsalva manoeuvre. There is a pronounced decrease in systolic pressure with an increase in heart rate during strain phase. During release, mean systolic pressure returns towards control levels, and the heart rate continues to rise.*

FIG. 4   *This figure shows the striking reduction in left ventricular end-diastolic volume during strain phase ($P < 0.01$) with rebound to control levels upon release ($P > 0.05$).*

Valsalva levels during release (Fig. 5). As can be anticipated, then, from these measured volumes, stroke volume during squeeze decreased 50 per cent, but rebounded to just above control levels upon release of straining (Fig. 6, Table 2).

Ejection fraction showed no consistent change during strain. In relaxation, however, there was a uniform and statistically significant rise over control measurements (Fig. 7).

Discussion

These angiographic studies permitted a quantitative assessment of volumetric alterations occurring during the Valsalva manoeuvre in man and confirm conclusions that were heretofore based on animal studies or extrapolations of pressure data for volume

END SYSTOLIC VOLUME

FIG. 5 *Left ventricular end-systolic volume diminishes during phase 2 (P < 0·05). In phase 4, however, control volumes are not reached (P < 0·05).*

estimation. Previous indirect data in humans are consistent with the conclusion that the fall in systemic arterial blood pressure during phase 2 results from diminished venous return, and thus, diminished cardiac output. The results of this study

STROKE VOLUME

FIG. 6 *Stroke volume changes in the five patients reflect the changes seen in the left ventricular end-diastolic volume and left ventricular end-systolic volume.*
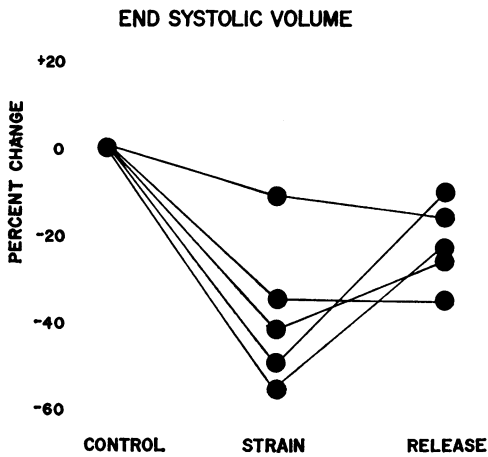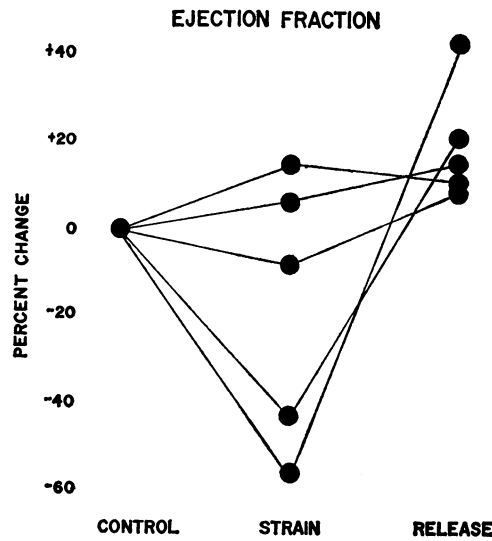
EJECTION FRACTION

FIG. 7 *Ejection fraction changes variably during the strain phase of Valsalva, but is uniformly augmented in phase 4 after release of straining (P < 0·01).*

support this concept by demonstrating a pronounced reduction in left ventricular end-diastolic volume and end-systolic volume which is greater than could be accounted for by heart rate change alone. Thus, the cardiac output falls, and despite the 'support' (Hamilton *et al.*, 1936) from the raised intrathoracic pressure and raised calculated peripheral vascular resistance, arterial blood pressure falls. The fact that the systolic pressure falls more than the diastolic pressure can be explained in two ways. First, a smaller stroke volume for a given aortic pressure-volume relation would produce a smaller systolic peak pressure. Second, as the cardiac output decreases, the aorta 'empties', and thus, less pressure change is required to accommodate a given stroke volume.

In the present study, a definite overshoot in the systolic pressure was not observed after release of straining, though the pressure did tend to return towards normal. This lack of overshoot of blood pressure may be explained in two ways. The first is that since four of the patients studied did have coronary artery disease proven by previous infarction in some and significantly abnormal coronary arteriograms in all, the abnormal response was an indicator of their heart disease. The other factor which probably contributed is the cumulative effect of contrast material *per se*. The changes in systolic and diastolic blood pressure and cardiac output are consistent with the added effect of phase 4 Valsalva, plus the previously described effects of angiographic contrast medium on circulatory dynamics (Kloster
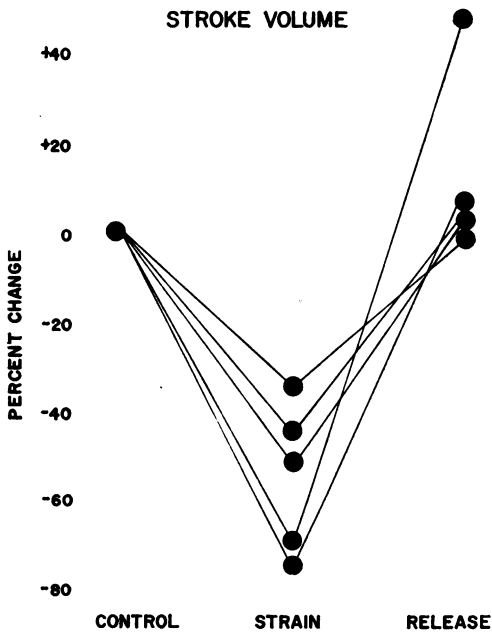
et al., 1966). By the time angiograms were performed during phase 4, a cumulative mean dose of 0·7 mg/kg of contrast medium had been administered over the preceding 7 to 22 sec.

In all the patients, despite the increased heart rate during phase 4, the left ventricular end-diastolic volume returned to control values, stroke volume exceeded control slightly, and thus, cardiac output was significantly, though transiently, increased 42 per cent above resting levels. The return to control left ventricular end-diastolic volume, despite the increased heart rate, is consistent with increased return of blood from the venous reservoir which had accumulated during the strain phase.

The increases in heart rate and ejection fraction during phase 4 produced a significantly increased cardiac output and are consistent with a continuing adrenergic influence on the heart. As has been noted previously, though the peripheral vascular resistance may seem to fall acutely when calculated from data measured in this changing haemodynamic situation, this classic quotient may not reflect the actual state of constriction of the resistance vessels at which adrenergic influence would be mediated. It may only represent the fact that the aorta, having previously emptied, is now lower on its pressure-volume curve (Greenfield *et al.*, 1967). Therefore, this fall of calculated peripheral vascular resistance should not necessarily be construed as evidence countering a continuous adrenergic influence.

The use of contrast material for direct beat-by-beat determination of cardiac output and ejection fraction has in the past been limited to the resting steady-state condition. Use of this method to evaluate transient haemodynamic changes has been limited previously by the large volumes of contrast usually employed (1–2 ml/kg) which produce significant haemodynamic changes (Kloster *et al.*, 1966). In the present study, by choosing patients with relatively normal-sized hearts and critically timing the injection from the patient's electrocardiogram, a much smaller dose of contrast could be employed (10 ml per angiogram) for adequate left ventricular visualization. Viamonte (1971) has indicated that for adequate visualization during angiography a ratio of 1 ml contrast of 76 per cent concentration to 8 to 10 ml

blood is required. This ratio was attained in the four patients with normal-sized hearts. In the one exception (Case 1), the dose of contrast was similar to that used in the other patients, and adequate visualization was achieved.

Conclusion

In conclusion, the above study showed a significant reduction in left ventricular volume during the strain phase of the Valsalva manoeuvre (phase 2). The data are consistent with the abrupt blockade of venous return from the periphery as the basis for this fall of left ventricular volume. By similar logic, the demonstration of the return of left ventricular volume to control values, despite the increase of heart rate above control levels in that period just after release of straining (phase 4), seems best explained by transiently augmented venous return.

References

Alderman, E. L., Sandler, H., Brooker, J. Z., Sanders, W. J., Simpson, C., and Harrison, D. C. (1973). Light-pen computer processing of video image for the determination of left ventricular volume. *Circulation*, **47**, 309.

Goldblatt, A., Harrison, D. C., Glick, G., and Braunwald, E. (1963). Studies on cardiac dimensions in intact, unanesthetized man. II. Effects of respiration. *Circulation Research*, **13**, 455.

Greenfield, J. C., Jr., Cox, R. L., Hernandez, R. R., Thomas, C., and Schoonmaker, F. W. (1967). Pressure-flow studies in man during the Valsalva maneuver with observations on the mechanical properties of the ascending aorta. *Circulation*, **35**, 653.

Hamilton, W. F., Woodbury, R. A., and Harper, H. T. (1936). Physiologic relationships between intrathoracic, intraspinal and arterial pressures. *Journal of the American Medical Association*, **107**, 853.

Kloster, F. E., Bristow, J. D., Jacobs, W. R., Porter, G. A., and Griswold, H. E. (1966). Hemodynamic effects of angiocardiography. *Investigative Radiology*, **1**, 398.

Stone, D. J., Lyon, A. F., and Teirstein, A. S. (1965). A reappraisal of the circulatory effects of the Valsalva maneuver. *American Journal of Medicine*, **39**, 923.

Viamonte, M., Jr. (1971). Innovations in angiography. *Radiologic Clinics of North America*, **9**, 361.