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Valsalva maneuver: Insights into baroreflex modulation of human sympathetic activity

MICHAEL L. SMITH, DWAIN L. ECKBERG, JANICE M. FRITSCH,

LARRY A. BEIGHTOL, AND KENNETH A. ELLENBOGEN

Departments of Medicine and Physiology, Hunter Holmes McGuire
Department of Veterans Affairs Medical Center, and Medical College
of Virginia, Richmond, Virginia 23249

METHODS

We recorded R-R intervals, tidal volumes, arterial pressures, expiratory pressures, and muscle sympathetic nerve activity on electrostatic and FM recorders. Two protocols were followed. **Protocol #1:** Nine subjects performed five Valsalva maneuvers at each of three expiratory pressures (10, 20, and 30 mmHg) during which muscle sympathetic activity was recorded. The order of pressure levels was randomized. **Protocol #2:** Six subjects performed five Valsalva trials at 30 mmHg during which right atrial pressure was recorded from a saline-filled catheter connected to a pressure transducer. The same parameters listed above were measured except muscle sympathetic nerve activity. This protocol was used to determine the time course of return of right atrial pressure to baseline levels after a strain, and the relation of this time course to the return of arterial pressure.

RESULTS

A sample tracing of one Valsalva trial from one subject is depicted in Fig. 1. The typical phases of the Valsalva maneuver and their sympathetic responses are shown clearly. These include phase 1 elevation of arterial pressure and inhibition of sympathetic activity at the onset of straining; phase 2 reduction of pressure and increase of sympathetic activity as straining continues; phase 3 abrupt decrease of pressure and increase of sympathetic activity after release of straining; and phase 4 elevation of pressure and inhibition of sympathetic activity during resumption of controlled-rate breathing.

Relation between sympathetic nerve activity and arterial pressure. Average changes of peak muscle sympathetic nerve activity during straining and arterial pressure during phase 4 are listed in Table 1. Absolute sympathetic nerve activity correlated only modestly with absolute diastolic pressure during straining ($r = 0.55$, $p < 0.05$); whereas, beat-to-beat change of sympathetic nerve activity correlated well with beat-to-beat change of diastolic pressure ($r = 0.79$, $p < 0.01$). Peak sympathetic activity during straining correlated significantly with phase 4 increases of systolic and diastolic pressures ($r > 0.80$; $p < 0.0001$).

Post-strain sympathetic inhibition. Several measurements were made to determine what physiologic adjustments account for post-strain sympathetic inhibition. The time to return of baseline systolic pressure, sympathetic activity, and the occurrence of the first post-straining sympathetic burst correlated significantly with the intensity of straining. The time of return to baseline sympathetic nerve activity was consistently greater than the return to baseline arterial pressure (Fig 3).

Six subjects were studied to determine the time of return to baseline right atrial pressure after Valsalva straining. The time of return to baseline right atrial pressure (27.3 ± 3.0 s) was not different ($p = 0.40$) from time of return to baseline systolic arterial pressure (24.5 ± 2.2 s), and therefore was not predictive of the return to baseline sympathetic nerve activity.

SUMMARY

Valsalva's maneuver, voluntary forced expiration against a closed glottis, is a well-characterized research tool, used to assess the integrity of human autonomic cardiovascular control. Valsalva straining provokes a stereotyped succession of alternating positive and negative arterial pressure and heart rate changes mediated in part by arterial baroreceptors. Arterial pressure changes result primarily from fluctuating levels of venous return to the heart and changes of sympathetic nerve activity.

We measured muscle sympathetic activity directly in nine volunteers to explore quantitatively the relation between arterial pressure and human sympathetic outflow during pressure transients provoked by controlled graded Valsalva maneuvers. Our results underscore several properties of sympathetic regulation during Valsalva straining. First, muscle sympathetic nerve activity changes as a mirror image of changes in arterial pressure. Second, the magnitude of sympathetic augmentation during Valsalva straining predicts phase 4 arterial pressure elevations. Third, post-Valsalva sympathetic inhibition persists beyond the return of arterial and right atrial pressures to baseline levels which reflects an alteration of the normal relation between arterial pressure and muscle sympathetic activity. Therefore, Valsalva straining may have some utility for investigating changes of reflex control of sympathetic activity after spaceflight; however, measurement of beat-to-beat arterial pressure is essential for this use. The utility of this technique in microgravity can not be determined from these data. Further investigations are necessary to determine

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whether these relations are affected by the expansion of intrathoracic blood volume associated with microgravity.

TABLE 1. Peak sympathetic nerve activity during straining and arterial pressure elevations after Valsalva straining

	Valsalva pressure, mmHg		
	10	20	30
Sympathetic activity, arbitrary units/10 s	1905±203	3086±374	4801±505
Change of systolic pressure, mmHg	10.4±1.2	17.9±1.4	25.5±1.8
Change of diastolic pressure, mmHg	3.6±0.7	7.8±1.0	13.1±1.1

All values are mean ± SEM. All changes were significantly different from zero ($P < 0.001$; n = 7). Pressure changes reflect the Phase 3 to 4 change; sympathetic increases reflect peak increase during Phase 2.
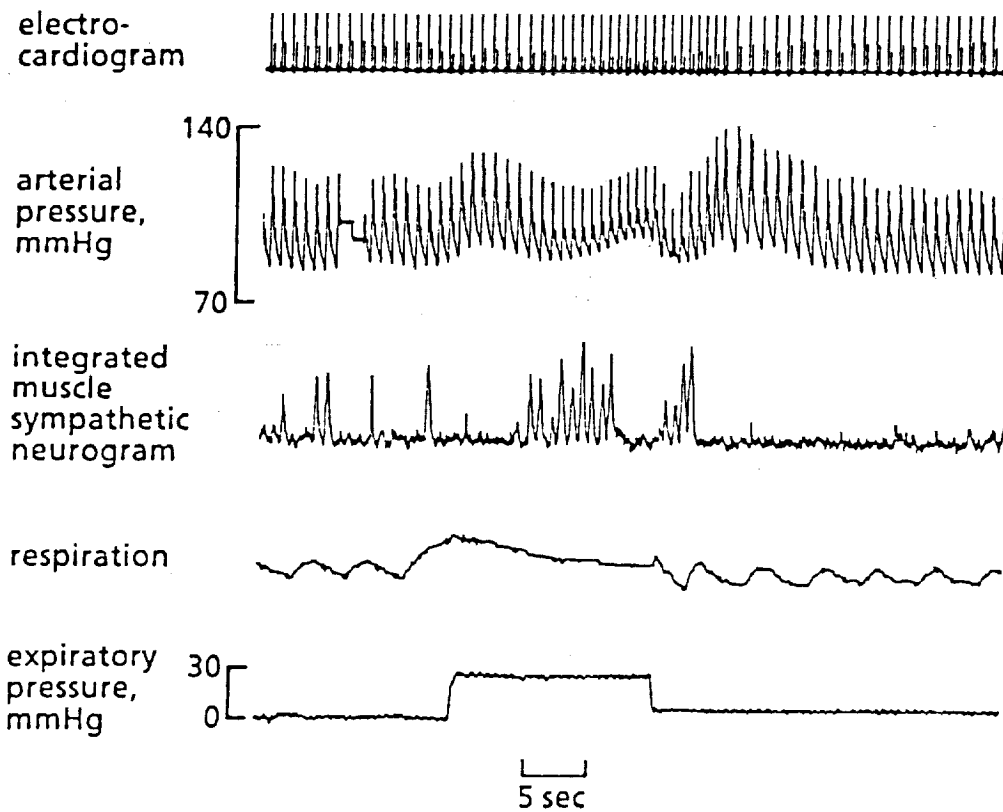
electro-
cardiogram

arterial
pressure,
mmHg

140

70

integrated
muscle
sympathetic
neurogram

respiration

expiratory
pressure,
mmHg

30

0

5 sec

Figure 1.   Original recording from a single trial for one subject.

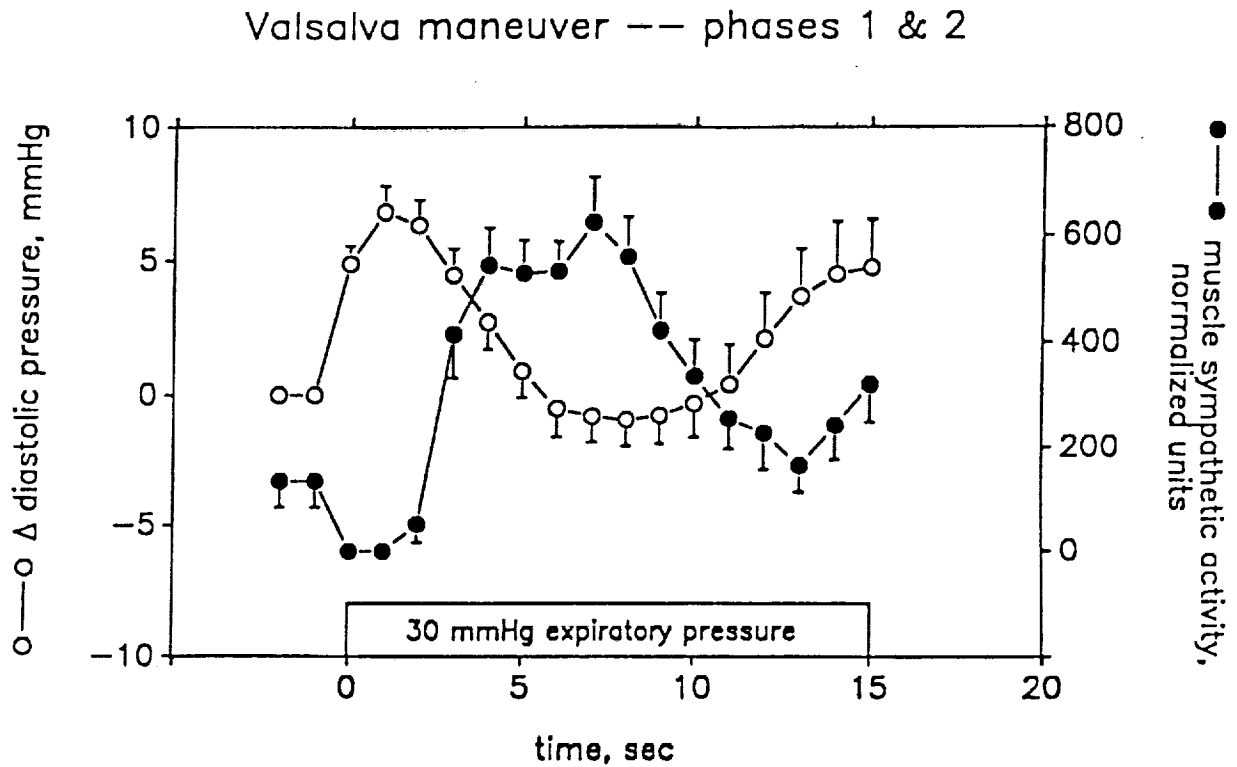**Valsalva maneuver — — phases 1 & 2**

Figure 2.   Average changes of muscle sympathetic nerve activity and diastolic pressure during 1-s time bins during 30 mmHg Valsalva maneuvers in 7 subjects. Brackets encompass one SEM. Muscle sympathetic activity was offset 1.3 s to account for nerve conduction latency.
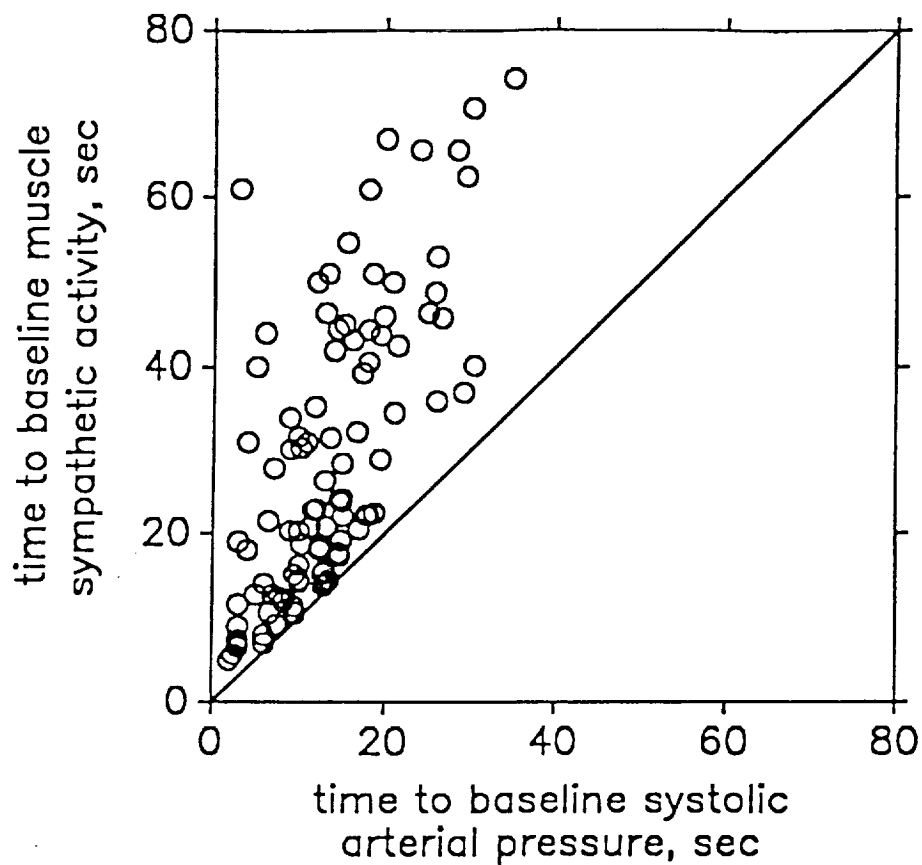
Figure 3   Relation between post-strain latency of return to baseline muscle sympathetic activity (ordinate) and post-strain latency of return to baseline systolic pressure (abscissa). These data indicate clearly that post-strain sympathetic inhibition extends beyond the return of arterial pressure to to baseline levels.