# TRANSIENT ANALYSIS OF RESPIRATION USING ECG-TRIGGERED PHRENIC NERVE STIMULATION

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Introduction The etiology of the fall in left ventricular stroke volume (LVSV) and arterial pressure with negative intrathoracic pressure (NITP) during normal and obstructed inspiration is controversial with changes in both LV preload and afterload proposed. Unfortunately, with inspiration in the intact animal, both mechanisms would occur over the course of several cardiac cycles making evaluation of any single mechanism difficult. We have shown previously that NITP applied during systole only diminishes LVSV while increasing systolic intrathoracic aortic diameter ($D_{AO}$), consistent with NITP imposing an effective afterload. This suggested that NITP may decrease arterial blood flow from the intrathoracic to the extrathoracic arterial compartment independent of its effect on LVSV. Accordingly, this study evaluated the effects of NITP confined to diastole on arterial blood flow out of the thorax and the ensuing LVSV.

Methods Using a left thoracotomy and femoral cutdowns, 12 anesthetized dogs were acutely instrumented. In series I (n=6) $D_{AO}$ was measured by sonomicrometry using 5 MHz piezoelectric crystals sewn to the aortic adventitia in both the anterior - posterior (AP) and right - to - left (RL) dimension. Ascending aortic blood flow ($Q_{AA}$) and LVSV (integrated $Q_{AA}$), arterial, left ventricular ($P_{LV}$) and esophageal pressures ($P_{ESO}$, air-filled balloon) were recorded. In series II (n=6) phasic extrathoracic carotid ($Q_{CAR}$) and/or intrathoracic descending aortic blood flow ($Q_{DA}$) were measured along with $D_{AO}$. In both series the phrenic nerves were dissected, cut and the peripheral ends used for phrenic nerve stimulation (PNS, duration: 100-200 ms) triggered from the R-wave of the ECG to produce NITP during early (ED), mid (MD), or late diastole (LD) after the thorax was resealed. PNS was carried out with the endotracheal tube obstructed and unobstructed. Data were collected after discontinuing mechanical ventilation for a brief period of apnea until a steady hemodynamic state had been established. 1-3 PNS were then performed, each seperated by 6-10 s of apnea. The stimulation in which the R-R-interval was closest to that of the immediately preceding beat was used for analysis. All parameters were compared quantitatively (series I, ANOVA followed by Scheffe's test, with $p<.05$) or qualitatively (series II) to the immediately preceding beat.

Results Series I: With the airway obstructed, NITP (-21.6mmHg, duration: 310ms±40 SE) caused a fall in the subsequent LVSV after PNS during ED (-7.7%, n.s.), MD (-11.7%, $p<.05$) and LD (-14.6%, $p<.001$) despite $P_{ESO}$ having returned to baseline before this systole. LV end diastolic transmural pressure ($P_{LV}$-$P_{ESO}$) increased from ED (+1.0mmHg, n.s.), to MD (+2.7mmHg, $p<.05$) and LD (+5.9mmHg, $p<.01$). End diastolic $P_{LV}$ relative to atmosphere also increased significantly during MD and LD. The R-R-interval remained unchanged. $D_{AO}$(AP) (14.7mm±2.1 to 15.4mm±2.08, $p<.0001$), $D_{AO}$(RL) (14.9mm±2.07 to 15.4mm±2.12, $p<.0001$) and calculated aortic cross sectional area (+8.3%, $p<.0001$) increased. Despite shorter (270ms±20) and smaller (-13.5mmHg±2.1) changes in NITP with the airway unobstructed during PNS, LVSV decreased progressively and to a similar extent during ED, MD, and LD with significant increases in LV end diastolic pressure in MD and LD both relative to atmospheric pressure and $P_{ESO}$. Series II: NITP decreased instantaneous $Q_{CAR}$ and $Q_{DA}$ and increased $D_{AO}$ both with and without airway obstruction in all dogs with retrograde $Q_{CAR}$ or $Q_{DA}$ seen in 4 dogs. In both series diastolic $D_{AO}$ showed a less rapid decrease, a plateau or even an increase with NITP, followed by a rapid decrease when PNS ceased. Increased $D_{AO}$ were still seen with PNS when an increase in abdominal pressure with diaphragmatic descend was minimized (abdomen open, n=3), but not with the chest open (n=2).

Discussion NITP during diastole alone was sufficient to decrease the subsequent LVSV. Since end diastolic $P_{LV}$ increased, a primary decrease in pulmonary venous return is ruled out. When lung volume was maintained constant by airway obstruction, pulmonary compression of the heart was excluded and the rapidity of the events makes changes in left ventricular contractility unlikely. This suggests that ventricular interdependence may occur with NITP within 300ms decreasing LV preload and the subsequent LVSV. With the airway unobstructed, NITP may further decrease LV preload and LVSV by direct compression of the heart by the expanding lungs. NITP reduces aortic flow run-off and can cause transient diastolic retrograde aortic flow. Both factors summate to distend the intrathoracic aorta which can be viewed as an elastic container driven by changes in ITP modulating arterial flow out of the thorax. The analogous mechanism is accepted for the venous circulation where NITP promotes flow into the thorax. Thus, transient analysis of respiration improves our understanding of the hemodynamic effects of respiration by demonstrating that the fall in peripheral arterial flow and pressure (pulsus paradoxus) with NITP during normal and obstructed inspiration is associated with both a diminished LVSV and a retention of blood within the intrathoracic arterial compartment.

References

1. Peters J, Kindred MK, Robotham JL. Negative intrathoracic pressure during systole: evidence for a left ventricular afterload. Anesthesiology 65: A44, 1986