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TITLE: CATECHOLAMINE CAUSE PULMONARY HYPERTENSION BUT NOT EDEMA IN THE ISOLATED PIG LUNG.

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Introduction: The primary mechanism of neurogenic pulmonary edema (NPE) is believed to be massive sympathetic discharge that results in a redistribution of blood from systemic to pulmonary circulation. Marked increased in circulating catecholamines occurs immediately following mechanical head injury. Infusion of catecholamines to animals pulmonary edema similar to NPE develops. We studied the direct pulmonary effects of catecholamines without systemic influence in an isolated porcine lung preparation.

Methods: Twenty-three in situ, isolated blood perfused lungs were studied. Group 1: time control, n-4; Group 2: Epinephrine (EP) infusion (200 μg/kg bolus + 30 μg/kg/m infusion), n=6. Group 3: Norepinephrine (NE) infusion, (200 μg/kg bolus + 30 μg/kg/m infusion), n=6. Group 4, EP + Phenoxybenzamine (0.15 mg/kg) posttreatment, n=4. Lungs were perfused at a fixed flow rate and at zero left atrial pressure (Pla) (venous reservoir placed at heart level). Lung microvascular permeability was measured prior to and 4 hrs after administration of catecholamines. Lung weight and pulmonary arterial pressure (Ppa) were followed for 4 hours after catecholamine infusion. Lung wet/dry (W/D) weight ratio was used as an index of edema formation.

Results: Infusion of EP or NE caused an immediate and sustained elevation of Ppa (Fig. 1) and marked decrease in lung weight, mean lung weight gain and Ppa at each Pla before and after EP or NE are unchanged. Lung W/D weight ratio were also unchanged from the control (Fig. 2). With phenoxybenzamine posttreatment Ppa decreased precipitously to baseline value.

Discussion: The present study showed that administration of catecholamine into isolated lung preparation at zone II condition caused a marked increase in Ppa but do not damage the pulmonary vessels directly to induce pulmonary edema. We conclude that catecholamines does not appear to be the primary mediators that cause pulmonary edema. Pulmonary edema seen in intact animals after infusion of catecholamines probably is caused by the subsequent systemic effects.

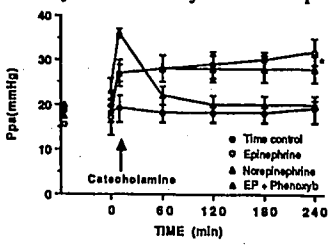

Fig. 1. Ppa changes during 240 min of catecholamines infusion. *P<0.05 from time control.

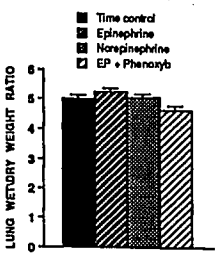

Fig. 2. Lung W/D weight ratio following 240 min of catecholamine infusion.

A1022

Title: ATRIO-VENTRICULAR INTERDEPENDENCE WITH TAMPONADE

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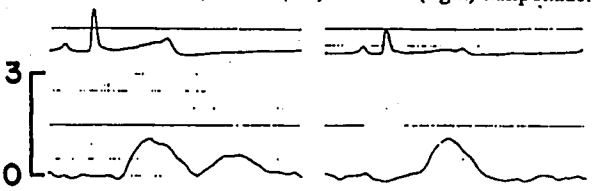
Introduction: The disappearance of the diastolic y descent in the right atrial pressure (Pra) trace is a classic hemodynamic sign of cardiac tamponade. It has been hypothesized to reflect an impediment of venous flow in diastole. However the exact mechanisms responsible for these changes have not been clearly established. We hypothesized that a homogeneous pericardial constraint of tamponade couples the volume changes of the atria and ventricles, inducing a situation of atrio-ventricular interdependence, i.e. reciprocal changes in volume.

Methods: Pra and superior (SVC) and inferior (IVC) vena caval flows were measured with ultrasound transit time probes in 6 anesthetized open chest dogs. Systolic and diastolic inflow volumes per beat through the SVC and IVC were obtained by integration and a ratio calculated. Measurements were performed at baseline and following production of 2 degrees of tamponade by injection of warm saline into the pericardial space to obtain pericardial pressures (Ppe) of 10 and 15 mmHg. Ratios were compared by ANOVA with Scheffe's tests.

Results: Diastolic/systolic inflow volumes (x ± SE; * P<.05):

	Control	Ppe > 10 mmHg	Ppe > 15 mmHg
SVC	0.47 ± 0.13	0.05 ± 0.05 *	- 0.09 ± 0.03 *
IVC	0.77 ± 0.16	0.33 ± 0.05 *	0.09 ± 0.05 *

ECG and Qsvc (L/min) before (left) and after (right) tamponade:



The normal systolic-diastolic pattern of venous flow was replaced with tamponade by a predominant systolic flow, the diastolic/systolic inflow volume ratio decreasing. This corresponded to a disappearance in Pra of the diastolic y descent as the mean Pra approached Ppe.

Discussion: Hoffman and Ritman's concept of "an invariant total heart volume" (1) implies that atrial inflow is limited to ventricular systole, a condition approximated only with tamponade since substantial diastolic atrial filling occurs in the control state. With tamponade, the homogeneous pericardial constraint induced by the pericardial liquid pressure couples atrial filling and ventricular emptying. During systole, ventricular emptying decreases the total intrapericardial volume and thus Ppe, permitting atrial filling. During diastole, reciprocal (interdependent) atrial emptying and ventricular filling will not change intrapericardial volume or Ppe. Thus diastolic Pra remains constant (disappearance of y descent), and diastolic venous flow will be absent.

Conclusion: Under normal conditions, the influence of pericardial constraint on atrio-ventricular coupling is small, allowing the atria and the ventricles to be relatively independent. During cardiac tamponade, a high degree of atrio-ventricular interdependence is present due to the increased pericardial liquid pressure that couples atrial and ventricular volume changes.

Reference: 1. Am J Physiol 249: H883, 1985