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Title : DISTRIBUTION OF BLOOD FLOW WITH LIDOCAINE

Authors : M. L. Lescanic, M.D.; E. D. Miller, Jr., M.D.; C. A. DiFazio, M.D.

Affiliation: Department of Anesthesiology, University of Virginia Medical Center, Charlottesville, Virginia 22908

Introduction. The systemic use of lidocaine is common in clinical practice. We have examined the effects of two different plasma levels of lidocaine on the hemodynamics of the awake laboratory rat. Radioactive labelled microspheres were used to determine cardiac output and distribution of blood flow before and after lidocaine administration.

Methods. 22 fasted Wistar rats (230-357 gms) were anesthetized with diethyl ether and PE-50 tubing was inserted into the left ventricle, left femoral artery and vein. The catheters were exteriorized and the animals allowed to awaken in restraining cages. The protocol consisted of a one hour control period followed by a bolus injection and 40 minutes infusion of lidocaine or vehicle (0.9% saline). Two groups of animals received lidocaine (n=8 for both groups). The first group (low lidocaine dose) received a 250 µg bolus of lidocaine followed by an infusion of 75 µg/min. The second group (high lidocaine dose) received a 750 µg bolus followed by an infusion of 250 µg/min. A control group (n=6) was treated identically but received vehicle only. To determine cardiac output and distribution of blood flow 40,000-60,000 15 micron microspheres labelled with Strontium 85 were injected through the left ventricular catheter at the end of the control period. Blood was withdrawn from the femoral artery at a constant flow rate for 70 seconds to determine cardiac output. Cerium 141 labelled microspheres were similarly injected after the 40 minute infusion period. Arterial blood gases were obtained prior to and immediately after lidocaine or vehicle administration. Blood was sampled at the end of the infusion period for determination of plasma lidocaine levels by gas chromatography. The animals were then sacrificed and the organs of the body dissected out, weighed and counted in a gamma counter at the appropriate energy spectra for each isotope. All data are expressed as the mean ± the standard error of the mean. The data were analyzed using Student's t-test for unpaired data for comparisons within groups. A p value of less than .05 was taken as statistically significant.

Results. The control animals which received vehicle only showed no significant difference in cardiac output, blood pressure, heart rate or blood flow/min/gm tissue between the first and second microsphere injection. There were no significant changes in pH, PCO_2 or PO_2 in any of the three groups between the first arterial blood gas, prior to infusion of lidocaine or vehicle, and the second blood gas at the end of infusion. Animals with low lidocaine levels (1.98±.27 µg/ml) showed no significant differences in cardiac output, blood pressure, heart rate or blood flow/min/gm tissue. Significant differences in the group with high lidocaine levels (6.37±.29 µg/ml) are summarized in the following table:

	Control	Lidocaine (6.37±.29 µg/ml)	
C.O.	169±11	108±12	*
MAP	117±2	113±2	
H.R.	450±4	371±8	*
Blood Flow	(ml/min/gm tissue)		
Brain	1.05±.05	.62±.05	*
Heart	7.26±.72	4.89±.52	*
Lung	.93±.10	1.05±.18	
Skin	.19±.02	.14±.03	
Renal	14.29±.96	13.20±1.30	
Muscle	.20±.02	.10±.01	*
Liver	.46±.08	.36±.06	*
Spleen	1.70±.16	1.91±.31	
Stomach	1.35±.18	.64±11	*
S. Intestine	1.51±.18	1.36±.14	
L. Intestine	.70±.05	.77±.14	

*p<.05 paired analysis

Significant reductions in blood flow to brain, heart, muscle, liver and stomach occurred in the group given the high lidocaine dose. Despite a 35% decrease in cardiac output there were no significant changes in blood pressure, arterial blood gases or pH. Blood flow to the kidneys and intestines was preserved but brain blood flow declined 41%. Heart rate decreased 18% from 450 bpm to 370 bpm. Blood pressure was maintained by an increase in systemic vascular resistance. Muscle blood flow decreased 50%. Hepatic artery flow decreased 23%.

Discussion. No studies to date have examined the effect of intravenous lidocaine on the distribution of blood flow in the unanesthetized rat. The data from this study indicate that lidocaine levels of approximately 2 µg/ml, have no effect on cardiac output or organ blood flow. At higher concentrations of lidocaine (6 µg/ml) a reduction in cardiac output and heart rate is seen. This is most likely secondary to direct myocardial depression by lidocaine. The reduction in myocardial blood flow may be a result of the decreased metabolic demands of the heart associated with the reduction in heart rate. At this high concentration of lidocaine, blood pressure did not decrease in spite of the decreased cardiac output. Any tendency toward a decrease in blood pressure was offset by an increase in systemic vascular resistance. The majority of the increase in systemic vascular resistance was in muscle which comprises 45% of rat body mass. Two other vital organs did show decreases in blood flow. Hepatic blood flow decreased but total splanchnic flow was unchanged. Brain blood flow also decreased and may explain the attenuation of intracranial pressure rises seen when endotracheal tube suctioning is preceeded by intravenous lidocaine. The data from this model provide evidence that important changes in the distribution of blood flow occur when intravenous lidocaine is administered even though blood pressure is unaltered.