TITLE: EFFECTS OF VASOACTIVE AGENTS, HYPOXIA AND VOLUME ON ESTIMATION OF LEFT ATRIAL PRESSURE IN PIGS

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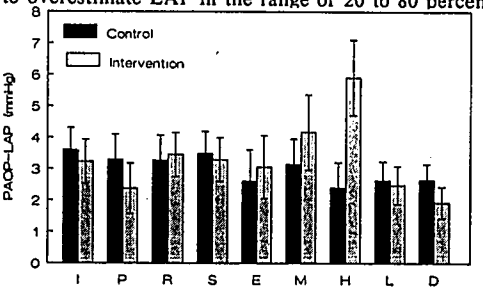
Introduction. The use of the pulmonary artery catheter and pulmonary artery occlusion pressure (PAOP) to estimate left atrial pressure (LAP) is a common clinical practice. However, in the face of altered pulmonary hemodynamics PAOP may not be an accurate representation of LAP. The purpose of this study was to investigate the relationship between PAOP and LAP under conditions of pharmacologic therapy, hypoxia and blood volume changes.

Methods. Following approval of the Animal Care Committee, six immature pigs (18 to 29 kg) were studied. Anesthesia was maintained with 1.5% isoflurane in 100% oxygen. Controlled mechanical ventilation with 5 cm PEEP was used to achieve an end-tidal CO_2 of 35 ± 5 mmHg. Femoral artery and vein, and flow-directed pulmonary artery catheters were inserted, along with 20 ga. PE tubing placed in the right and left atria for pressure monitoring. Cardiac output was measured continuously using an electromagnetic flowmeter placed on the ascending aorta. Hemodynamic parameters measured or derived included MAP, HR, CO, PAM, SVR, PVR, CVP, LAP, and intermittent PAOP. Responses to the following randomized drug infusions were evaluated: isoproterenol (I), phentolamine (R), nitro-prusside (S), PGE_1 (P), esmolol (E), and methoxamine (M). Other non-pharmacologic interventions included hypoxia (H), rapid fluid volume loading (L), and hypovolemia (D). Drug infusions were titrated to elicit a hemodynamic response. Hypoxia was induced with 100% N_2 maintaining tidal volume constant. Volume loading was achieved by rapid administration of 1L RL and 500 ml hetastarch. Hypovolemia was produced by withdrawal of 1,500 ml of blood over five minutes. PAOP-LAP gradients were determined prior to and at steady state following interventions, The results were compared using t-tests.

Results. Mean baseline gradients (mean±sem) from the Figure ranged from 2.4 to 3.6 mmHg (19.5% to 49.3% error in the PAOP estimate of LAP). A significant mean increase in gradient to 5.7 mmHg (80.3% error in PAOP (12.8mmHg) estimate of LAP (7.1mmHg)) occurred following hypoxic challenge ($p<0.025$). Statistically significant changes in the gradient did not occur following pharmacologic or volume interventions.

Discussion. These data demonstrate a relatively consistent gradient between PAOP and LAP under a variety of pharmacologic and volume challenges with PAOP over estimating LAP in the range from 20 to 50 percent. Hypoxia resulted in a marked increase in the gradient as the result of severe pulmonary vasoconstriction. Although not statistically significant, vasodilators (I,P,R,S,L) tended to decrease the gradient, while constrictors (E, M, H) increased the difference. Overall, PAOP tended to overestimate LAP in the range of 20 to 80 percent.



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TITLE VALIDITY OF MICROSPHERE DETERMINATIONS OF CEREBRAL BLOOD FLOW DURING ACUTE HEMODILUTION IN CATS

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Introduction: Determination of cerebral blood flow (CBF) using radiolabelled microspheres (MS) depends upon collection of accurate arterial reference samples (ARS). Hemodilution, used experimentally and clinically to improve cerebral perfusion in stroke, may erroneously decrease ARS concentrations of MS depending on hematocrit, reference sampling rate and the size of the artery from which the ARS are withdrawn.[1] Decreasing MS concentrations in ARS due to hemodilution would lead to falsely high estimates of CBF. We studied the effects of vessel size, hematocrit and withdrawal rate on ARS MS concentrations in cats.

Methods: In a protocol approved by the IACUC, 8 adult cats (3 ± 0.1 kg) were anesthetized with ketamine hydrochloride (25mg/kg), tracheotomized, paralyzed with pancuronium bromide and ventilated with 1.6% isoflurane in N_2O:O_2 (70:30). Both brachial and the left femoral arteries were cannulated with polyethylene tubing (i.d.= 0.8mm). The abdominal aorta (AA) was cannulated with a larger diameter tubing (i.d.=1.2mm) via the right femoral artery. A left atrial cannula was placed for microsphere injection. Isoflurane concentration was then decreased to 0.8% in N_2O:O_2 (70:30) and arterial pH, pCO_2 and pO_2 were adjusted to normal limits and 2 sequential MS injections were made using each of 5 isotopes. Since MS are rapidly cleared from the systemic circulation, repeated injections of the same isotope could be performed. During the first injection, ARS were withdrawn from one brachial artery at 1.03 ml/min and from the other brachial artery and the AA at 2.06 ml/min. Subsequently, ARS were withdrawn from one brachial artery at 2.06 ml/min and from the other brachial artery and the AA at 1.03 ml/min. ARS were gamma-counted and the number of MS in each was calculated. This procedure was repeated as the cats were hemodiluted with 10% hetastarch to hemoglobin levels of 85, 70, 55 and 40% of the baseline hemoglobin concentration. This protocol permitted the comparison of the number of MS in ARS collected at slow and fast withdrawal rates from both small and large arteries at each hematocrit. Results were analyzed using ANOVA.

Results: ARS MS concentrations are displayed as mean ratio±SD. Ratios of MS concentrations did not differ significantly between 100% and 40% HgB at any withdraw rate or site. There were no significant interactions among any withdrawal sites (i.e. vessel size) or HgB concentrations.

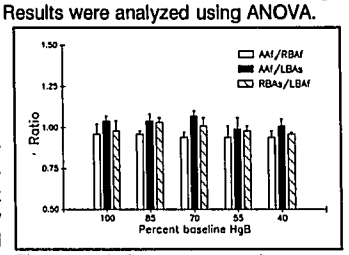

Figure: f=fast rate; s=slow rate; R/LBA=right/left brachial arteries

Discussion: The numbers of MS in ARS were independent of vessel size, withdrawal rate or degree of hemodilution. The differences between these and previous results[1] may reflect the fact that, in the present study, the cannulae completely occluded the small vessels and minimized the effects of axial streaming. Axial streaming may have contributed to the observations that MS numbers in ARS can be erroneously low in small vessels at low flow rates and low hematocrits.[1] While we observed no effects of hemodilution on ARS MS concentrations, we agree that careful validation of the MS method is important to insure accuracy in new pathological models.[1,2]

References:
1. Rosenberg, et al, Am J Physiol 244:H308-311, 1983
2. Heymann, et al, Prog Cardiovasc Dis 20:55-79, 1977