Title: **Lidocaine alters flow-mediated changes in large arteries in humans**

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Since the effects of lidocaine (L) on large and small peripheral arteries remain unclear to date, we investigate the influence of graded doses of L on the flow-diameter relationships of the brachial artery (BA) in humans.

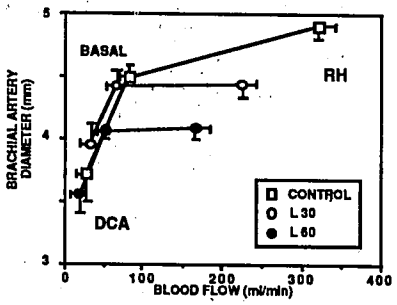
Methods. After approval by the IRB and obtening informed consent, 9 healthy volunteers (23±2yrs) were studied before (control) and during an iv infusion of L at two dosages [30 (L30) and 60 (L60) µg/kg/min following an iv bolus of 1.5 mg/kg]. A dual-crystal pulsed Doppler system was used to measure BA diameter (D) and blood flow (BF) proximal to the antecubital fossa[1]. Mean arterial pressure (MAP, Finapress®) and heart rate (HR) were continuously recorded. Forearm vascular resistances (FVR) were calculated as FVR=MAP:60BF. Blood samples were drawn during control, L30 and L60 to assay plasma norepinephrine (NE) levels (HPLC method) and serum L levels (gas chromatography). Following basal measurements during control, L30 and L60, BF was altered by an occluding cuff placed on the forearm distal to the site of BAD and BF measurements (distal circulatory arrest, DCA). By deflating the cuff after 10 min, a typical reactive hyperemia (RH) was observed. Data are expressed as mean±SEM and compared using ANOVA and Fisher PLSD test.

Results. Table shows that L60 increased MAP and NE. Simultaneously, velocity, BAD and BF decreased whereas FVR increased significantly. As depicted on figure, during RH, L reduced the increase in BF (P<0.05 : L30 and L60 vs control, L60 vs L30) and suppressed the BF-induced increase in BAD.

Comments. These data show that L dose-dependently reduces the RH-induced increase in BF and alters the subsequent flow-related increase in BAD. These effects are unlikely related to a major change in distending pressure since they are observed at L30 when MAP remains unchanged or due to a direct action of L on large artery smooth muscle since L does not change BAD during DCA. These effects of L might rather be due to (1) an indirect increase in sympathetic tone, or (2) an alteration of the flow-diameter relation through an action of L on the vascular endothelium[2].

	control	L30	L60
HR (bpm)	55±2	57±3	59±2
MAP (mmHg)	85±4	93±5	98±5*
Brachial artery			
diameter (mm)	4.49±0.05	4.42±0.09	4.07±0.07* **
velocity (cm/s)	8.42±0.59	6.91±0.60	6.20±0.95*
blood flow (ml/min)	80±7	64±7	50±7*
FVR (mmHg/ml/s)	67±7	93±10	143±19* **
Lidocaine (µg/ml)	0	1.72±0.09	2.39 ±0.14*
NE (ng/ml)	227±37	285±47	306±54*

* = P<0.05 vs control; ** = P<0.05 vs L30



References

1. Anderson EA, Mark AL. Circulation 79: 93-100, 1989.
2. Johns RA. Anesthesiology 70: 805-811, 1989.

TITLE: **FACTORS INFLUENCING CEREBRAL BLOOD FLOW VELOCITY DURING CARDIOPULMONARY BYPASS**

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The influence of nasopharyngeal temperature (NPT), $paCO_2$, hemoglobin (Hb), systemic blood flow (Q), mean arterial pressure (MAP), systemic vascular resistance (SVR) and age on cerebral blood flow (CBF) during cardiopulmonary bypass (CPB) have been characterized (1). However, the influence of those variables on cerebral blood flow velocity (CBFV) still has to be defined. Aim of the current study was to establish the factors most highly associated with changes in CBFV during CPB.

With informed consent and institutional approval CBFV of the middle cerebral artery was determined by transcranial Doppler technique (TC2-64B, EME) in 70 patients undergoing coronary artery bypass grafting. Simultaneously to CBFV recording NPT, $paCO_2$, Hb, Q, MAP and central venous pressure were measured after establishment of hypothermic resp. normothermic (rewarming) CPB. Stepwise linear regression technique was used to establish the factors significantly influencing CBFV during CPB.

The table shows the range of investigated variables. By stepwise linear regression, the effect of $paCO_2$, NPT and Hb on CBFV reached statistical significance.

As already demonstrated for CBF (1), NPT and $paCO_2$ are likewise the most powerful determinants ($p < 0.001$) of CBFV during nonpulsatile CPB. In contrast to the findings of Govier et al (1) but in correspondence with the results of Roy et al (2) for CBF, we observed an inverse relationship ($p < 0.05$) between Hb and CBFV during hypothermic CPB. Our results furthermore indicate that under the below mentioned CPB conditions (Table) CBFV autoregulation is retained (3) and CBFV is independent of age (4).

References:

1. An. Thorac. Surg. 38: 592, 1984
2. Anesthesiology 71: A34, 1989
3. Thorac. Cardiovasc. Surgeon 38: 106 (A75), 1990
4. Anesthesiology 71: A37, 1989

Variable	Range	r	p
$paCO_2$* (mmHg)	33 - 77	0.689	< 0.001
NPT** (°C)	27.0 - 37.7	0.540	< 0.001
Hb (g/dl)	5.1 - 9.3	- 0.227	< 0.05
SVR (dyn.sec.cm^{-5})	466 - 1639	0.145	NS
Q (L/m^2/min)	1.4 - 2.4	0.098	NS
MAP (mmHg)	33 - 87	0.097	NS
Age (yrs)	38 - 75	- 0.048	NS

Table: Influence of different variables on CBFV during hypothermic CPB.
*Temperature uncorrected
**Hypothermic and normothermic (rewarming) CPB