TITLE: DOES SEVERE NORMOVOLEMIC HEMODILUTION ADVERSELY AFFECT SPLANCHNIC OXYGENATION?

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Introduction. Perioperative hemodilution (HD) has become an accepted means of reducing transfusion requirements. This study evaluates the effects of moderate and severe normovolemic HD on splanchnic oxygenation.
Methods. In 9 pigs following laparotomy for instrumentation (catheterization of hepatic and portal [P] veins, placement of EMF probes around hepatic artery [HA], portal vein [PV] and superior mesenteric artery [SMA], and of PO_2 surface electrodes [1] onto liver [HEP] and small intestine [SI]), hematocrit (Hct) was lowered to 20% (HD1) and 14% (HD2) by replacing blood with equal amounts of 6% hydroxyethyl-starch.
Results (Table). Absolute flows increased continuously. Relatively (%), HABF increased most. Therefore, despite similar reductions in all O_2 contents, only DO_2HA was preserved. Splanchnic VO_2 was maintained due to increases in O_2 extractions. During HD2, surface PO_2 decreased with broadening of the summary liver PO_2 histogram (one third of PO_2 values below 30 mmHg).

Discussion. It appears that during severe normovolemic HD splanchnic VO_2 is preserved due to activation of compensatory mechanisms (ie, increases in flows and O_2 extractions). However, decreases in surface PO_2 and abnormal distribution in the liver PO2 histogram would indicate that during severe HD compensatory mechanisms begin to become exhausted.
Reference. 1. Anesthesiology 44:184,1976

VARIABLE	CONTROL	HD 1	HD 2
MAP(mmHg)	92±3	93±3	82±4*
CVP(mmHg)	2.5±0.4	2.5±0.4	2.6±0.4
CO(l/min)	3.6±0.2	4.1±0.2*	4.5±0.2*
THBF(ml/min)	653±20	794±29*	895±33*
HABF(ml/min)	87±8	136±9*	166±13*
PBF(ml/min)	577±28	675±30*	750±43*
SMABF(ml/min)	393±34	447±52*	498±52*
DO2TH(ml/min)	65±5	49±5*	39±3*
DO2HA(ml/min)	11±1	11±1	9±1
DO2PV(ml/min)	57±4	40±4*	29±3*
DO2SMA(ml/min)	48±4	36±5*	28±3*
VO2TH(ml/min)	17±2	16±3	15±4
VO2SI(ml/min)	10±1	10±2	9±2
PO2HEP(mmHg)	55±3	54±1	41±5*
PO2SI(mmHg)	56±2	54±1	45±3*

Means ± SEM. * = $p<0.05$ compared to preceding value. BF = blood flow. TH = total hepatic. DO2 = O2 delivery. VO2 = O2 uptake. (See text for further abbreviations.)

BUPIVACAINE AND MYOCARDIAL ISCHEMIA. STUDY IN THE PIG IN SITU HEART.

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The impairment of intraventricular conduction is the most serious cardiac disorder caused by bupivacaine (Bup), since it possibly results in ventricular fibrillation[1]. As the impulse propagation in the ventricular contractile fibers is also adversely affected by circulatory deficiency, when severe[2], the use of Bup for regional anesthesia might be dangerous under such circumstances. The hazards would be even real peroperatively, since numerous factors are then likely to stimulate myocardial activity and metabolism and, thus, to reveal an ischemia which was not apparent. Consequently, it was of interest to achieve an electrophysiological study of the Bup effects in an ischemic area of the myocardium.

This study was carried out in 20 anesthetized, open-chest pigs, weighing 18-24 kg, which were artificially ventilated and heated to maintain constant their core temperature. Ischemia was produced by completely occluding temporarily (4-8 min) the left anterior descending coronary artery near its origin and checked by the expected changes of ST segments and waves in D_2 and V_2-V_3 ECG leads. Monophasic action potential (MAP) was recorded by an appropriate electrode introduced into the ventricular wall thickness of the area subjected to ischemia. Ventricular beats remained governed by sinus node, except during short 10 s periods every minute : the ventricles were then paced at a constant rate, relatively high (180 bpm), using an electrode fixed near the apex, therefore also in the ischemia area, to determine depolarization velocity, conduction time (CT) and effective refractory period (ERP) in the ventricular contractile fibers.

From the comparison of the data obtained in 10 animals under ischemia alone and 10 under ischemia plus Bup (2 mg/kg, i.v.) it results that : 1) CT is lengthened by ischemia only slowly (> 2 min) and moderately (< 75%). With adding Bup, its lengthening is faster and far more considerable (150%). Similarly, the width of the QRS complex, little affected by ischemia alone, is increased to a large extent by the association of Bup. 2) On the contrary, Bup tends to prolong MAP duration which is shortened by ischemia (33%). The decrease in ERP due to ischemia (20%) is however better counteracted by Bup. 3) The ischemia-induced fall in MAP amplitude, probably related to reduction in resting membrane potential, is also lessened by Bup in some measure, but Bup does not delay or even hastens ventricular fibrillation following the major impairment of MAP.

The possible detrimental action of ischemia on intraventricular conduction is liable to be aggravated by Bup, with occurrence of fibrillation despite antagonistic effects on repolarization.

References
1. Anesthesiology 70 : 799-804, 1989
2. Physiol. Rev. 69 : 1049-1169, 1989