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Title: METHYLMETACRYLATE BLOOD LEVELS DURING HIP ARTHROPLASTY

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A direct effect of methylmetacrylate (MMA), one of the bone cement ingredients, has been involved in the severe hemodynamic complications occasionally observed during total hip arthroplasty (THP) at the time of femoral implantation. However, the results of intraoperative MMA plasma measurements remain confusing since both very low and high MMA plasma levels have been reported. Indeed, the methodological problems are numerous : site and delay for sampling, conditions of storage and measurements.

After approval by the IRB, 11 patients scheduled for THP, gave informed consent. Blood was withdrawn from a pulmonary and from a radial catheter at 0.5, 1, 1.5, 2, 3, 4, 5, 6 and 7 min. following implantation of cement in the acetabular(A) cavity and in the femoral(F) shaft.

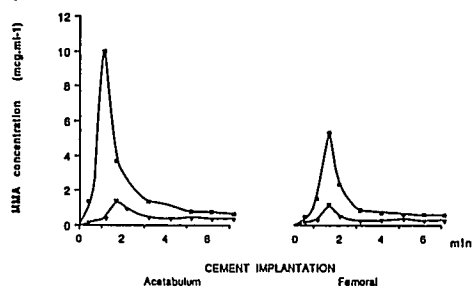
MMA plasma concentrations were assessed by a gas chromatography/mass spectrometry method. Results are given as mean \pm SEM. Maximal concentrations following A and F cement implantation were compared with a paired "t" test. When the peak value was higher than 1 mcg.ml⁻¹ in the pulmonary blood, 1) the decrease of MMA was fitted with a biexponential equation, 2) the area under the curve for pulmonary (AUCpa) and radial (AUCra) concentrations were computed, and the ratio Cpulm = (AUCpa-AUCra)/AUCpa was calculated.

A demonstrating individual evolution is shown in figure 1. The mean peak concentration of MMA in mixed venous blood was significantly higher after A than after F cement insertion (respectively 5.0 \pm 1.3 and 1.9 \pm 0.6 mcg.ml⁻¹) (p < 0.05). Significantly lower results were observed in arterial samples : respectively 1.9 \pm 0.8 mcg.ml⁻¹ (A) and 0.8 \pm 0.2 mcg.ml⁻¹ (F). In 13 occasions, the peak of MMA was above 1 mcg.ml⁻¹. The peak occurred at 1.4 \pm 0.02 min and the decrease fitted a biexponential equation (r = 0.91 \pm 0.02). The initial half-life was 0.3 \pm 0.1 min and the terminal 3.0 \pm 0.7 min. The ratio Cpulm was 55.1 \pm 7.8 %.

These data demonstrate that:

- the MMA peak in mixed venous blood is early (within 2 min.)
- MMA blood levels are higher after A than after F cement implantation, contrasting with previous studies, and are much lower than the experimental toxic levels.
- the decrease in MMA levels is very rapid.
- the clearance occurred mainly during transpulmonary passage.

FIGURE 1: Plasma concentration of MMA in mixed venous (upper curve) and arterial (lower curve) blood after cement implantation.



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TITLE: EFFECTS OF ACUTE CORONARY STENOSIS, ANESTHETIC AGENT, AND HYPOTENSION ON RENAL AND LIVER BLOOD FLOW

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Introduction: The effect of acute coronary stenosis on organs besides the heart has not been well studied. The purpose of this investigation was to examine the effects of acute coronary stenosis and hypotension on blood flow to the liver and kidney in pigs anesthetized with 4 different volatile anesthetics.

Methods: After approval by the Institutional Animal Care and Use Committee, 26 pigs weighing 21 to 37 kg (27.0 \pm 0.9kg) were randomized to receive 1 of 4 anesthetic agents: halothane, isoflurane, enflurane, and sevoflurane. Each pig underwent a randomly determined 10 min period of normal blood pressures (MBP>60) and low blood pressures (MBP <60). After 10 min, colored microspheres were injected, hemodynamic measurements taken, and arterial and venous blood gases drawn. A modification of the Gerwitz method of creating coronary stenosis was used.¹ Stenosis was confirmed by dye injection. Random order post-stenosis measurements were made at the same MBP's, after 15 min of stenosis. Blood flows to the perihilar and capsular areas of the liver, and to left and right renal cortex were calculated using the densities of the colored microspheres in each anatomical area post-mortem. Data were analyzed with repeated-measures ANOVA with significance at p<0.05.

Results: Four of the pigs died at placement of the acute stenosis. Technical problems with the flow measurements were encountered in 4 other pigs. Complete data were collected on 18 pigs. No differences in cardiac output were observed pre and post-stenosis at normal and low blood pressures. Results are tabulated in Table 1. Repeated-measures ANOVA revealed no agent effect or side (left vs right) effect for renal blood flow. However, a significant effect of stenosis (p=0.023) and blood pressure (p<0.001) was observed. No difference in capsular vs perihilar liver blood flow and liver blood flow with anesthetic agent were observed. In contrast to renal blood flow, liver blood flow was not affected by coronary stenosis. Only blood pressure affected liver blood flow (p<0.001).

Conclusions: Acute coronary stenosis differentially affects renal and liver blood flow. Renal blood flow decreases; liver blood flow does not. Different responses of these organs to catecholamine surges may provide an explanation for this effect.

TABLE 1. EFFECT OF CORONARY STENOSIS AND PERFUSION PRESSURE ON LIVER AND KIDNEY BLOOD FLOW

	PRE-STENOSIS		POST-STENOSIS	
	Normotensive (MBP=78 \pm 2 mmHg)	Hypotensive (MBP=33 \pm 2 mmHg)	Normotensive (MBP=77 \pm 2 mmHg)	Hypotensive (MBP=35 \pm 2 mmHg)
KIDNEY BLOOD FLOW (ml/min/g)				
Halothane (n=4)	2.18 \pm 0.20	1.19 \pm 0.12	1.42 \pm 0.13	0.86 \pm 0.06
Isoflurane (n=4)	2.55 \pm 0.67	1.65 \pm 0.38	2.07 \pm 0.67	1.42 \pm 0.25
Enflurane (n=5)	2.68 \pm 0.23	1.35 \pm 0.12	1.97 \pm 0.45	1.12 \pm 0.18
Sevoflurane (n=5)	2.00 \pm 0.29	1.09 \pm 0.21	1.87 \pm 0.54	0.87 \pm 0.17
TOTAL	2.35\pm0.18	1.31\pm0.11	1.84\pm0.23	1.06\pm0.10
LIVER BLOOD FLOW (ml/min/g)				
Halothane (n=4)	0.70 \pm 0.34	0.28 \pm 0.15	0.37 \pm 0.13	0.18 \pm 0.06
Isoflurane (n=4)	0.45 \pm 0.15	0.25 \pm 0.10	0.71 \pm 0.33	0.32 \pm 0.14
Enflurane (n=5)	0.63 \pm 0.17	0.26 \pm 0.06	0.69 \pm 0.29	0.25 \pm 0.08
Sevoflurane (n=5)	0.55 \pm 0.10	0.29 \pm 0.03	0.52 \pm 0.07	0.24 \pm 0.03
TOTAL	0.58\pm0.09	0.27\pm0.04	0.58\pm0.11	0.25\pm0.04

References: ¹Am J Cardiol 47:589-596 1981