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Dexmedetomidine ameliorates gut lactate production and impairment of exogenous lactate clearance in an endotoxic sheep model

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Introduction

The mechanisms of persistent hyperlactemia during endotoxic shock are probably multifactorial. Both hypoperfusion-related anaerobic production and adrenergic-driven aerobic generation have been implicated. More recently an early and severe impairment in exogenous lactate clearance has also been described [1]. Theoretically, an excessive adrenergic response could influence all these mechanisms and thus aggravate the problem.

Objectives

To assess the effects of dexmedetomidine (DEX) on lactate production and exogenous lactate clearance in an endotoxic shock model.

Methods

Twelve anesthetized sheep were subjected to a multimodal hemodynamic/perfusion assessment including hepatic and portal vein catheterizations, total hepatic blood flow, sublingual microcirculation, and muscle microdialysis. After the monitoring phase, all received a 5 mcg/kg LPS bolus (E coli O127:B8®) and then 4 mcg·kg^{1}·hr^{1} for the rest of the experiment. After 1hr they were volume resuscitated, and then randomized to placebo or DEX. Sampling and exogenous lactate clearances [2] were performed at 4 points (figure 1).

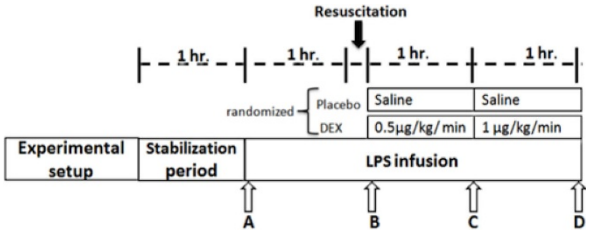

Figure 1

Results

DEX was not associated with any adverse hemodynamic effect in terms of cardiac output, heart rate, mixed venous oxygen saturation, central venous-arterial pCO_2 gradient and NE requirements as compared to placebo. DEX animals presented significant lower epinephrine levels (4.6 ± 1.3 vs 7.1 ± 1.4 ng/ml), arterial lactate levels (6.4 ± 3.1 vs 9.2 ± 1.8 mmol/l), portal vein lactate levels (6.2 ± 2.2 vs 8.1 ± 2.0 mmol/l); and higher portal vein O2 saturations (78 ± 16 vs 68 ± 11 %), and exogenous lactate clearance (7.2 ± 5.4 vs 2.9 ± 1.5 ml/kg/min) as compared to placebo at point D. No differences in muscle lactate production or sublingual microcirculatory parameters could be observed.

Conclusions

Dexmedetomidine ameliorates the increase in gut lactate production and impairment of exogenous lactate clearance in experimental endotoxic shock. This effect is associated with a significant reduction in systemic epinephrine levels.

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