episodes (34.6%) was digestive septic focus, following 13 cases of post-surgical abdominal intervention (25%). Main reason of PCD was 11 cases of pulmonary empiema (21.1%), following 9 cases of infected intraabdominal collections (17.3%) and pneumoefrosis in 8 cases (15.4%). In 46 cases (88%) the PCD was performed under ultrasound guidance. PCD was performed 1 day mean after ICU admission (0–5.75), it was maintained 4 days mean (2–13.75).

No complications related to PCD were observed in most episodes (44 cases, 85%). The most frequent complications were drainage obstruction in 5 cases (9.4%), and local bleeding in 2 episodes (3.4%), none of them related with decease.

Subsequent surgical intervention after PCD was required only in 8 cases (15.4% of patients). ICU mortality was 13.5% (7 cases) and in-hospital 19.2% (10 cases).

CONCLUSIONS. PCD can be a useful and safe tool for evacuation of collections in ICU patients. Prospective studies are needed to quantify surgical intervention reduction in these high-risk patients.

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INTRODUCTION. A metabolic shift towards lipid use occurs during critical illness. This may represent an evolutionary conserved response to utilize body fat stores as a energy substrate. Ketone bodies, often regarded as a by-product of fatty acid beta-oxidation, are emerging as important metabolic regulators. Ketogenic diets are being used therapeutically in various diseases.

OBJECTIVES. To assess the impact of exogenous administration of the ketone, beta-hydroxybutyrate (BHB), on metabolism and cardio-respiratory function in a long-term rat model of fluid-resuscitated faecal peritonitis.

METHODS. Instrumented male Wistar rats had sepsis induced by intraperitoneal injection of faecal slurry. Sham-operated animals received no injection. Animals were then placed in metabolic cages for indirect calorimetric measurement of O₂ consumption and CO₂ production. They had free access to food and water. Fluid resuscitation with a balanced solution was started at 2h. At 6h animals were randomized to receive iv BHB infusion (180mg/kg/h) or an equivalent isocaloric volume of glucose solution. At 24h, blood and tissue samples were collected and animals euthanized.

RESULTS. At 24h BHB-treated animals had a metabolic alkalosis (7.53 ± 0.04 vs 7.49 ± 0.03 untreated; p = 0.001) with increased bicarbonate (33.4 ± 2.5 vs 28.2 ± 3.2 mEq/L; p < 0.001) and base excess (10.8 ± 3.2 vs 4.5 ± 3.9 mmol/L; p < 0.001), yet PaCO₂ levels were unchanged. BHB had no effect on respiratory exchange ratio (RER = CO₂ production/O₂ consumption) in sham-operated animals (0.85 ± 0.05 vs 0.89 ± 0.03 in placebo; p = 0.169). However, the septic animals, regardless of BHB treatment, had similarly reduced RER values at 24h (0.80 ± 0.03 vs 0.78 ± 0.01 untreated, p = ns; p < 0.001 compared to sham). O₂ consumption was unchanged at 24h, whereas CO₂ production was reduced in the septic groups (1245 ± 146 mL/kg/h vs 1441 ± 171 mL/kg/h; p = 0.003). Compared to placebo, BHB infusion in septic animals produced no significant changes in clinical severity score, temperature, blood pressure or cardiac function.

CONCLUSIONS. Sepsis produced a shift towards fat oxidation signified by a fall in RER. BHB infused over 18 hours resulted in a metabolic alkalosis but no effect on illness severity, haemodynamics or RER. No positive impact was seen with a ketogenic diet in this animal model of sepsis.