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Original Research Article

Variations of renal tissue oxygenation during abdominal compartment syndrome and sepsis

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ABSTRACT

Purpose: This experimental study was designed to evaluate the renal tissue oxygenation under the coexistence of abdominal compartment syndrome and sepsis.
Material and methods: Fourteen non-breed dogs were divided into two groups: the control group (8) and the study group (6). Sepsis was established with intravenous endotoxin infusion at 100 μg/kg for over 30 min. Insufflation of CO_2 in the peritoneal cavity was used for the increase in intra-abdominal pressure (IAP). A special catheter placed and fixed in the renal cortex at a depth of 3 mm from the renal capsule was used for the measurement of renal tissue oxygenation.
Results: Study parameters were recorded at the starting phase, at IAP of 15 mmHg and 30 mmHg and after decompression of the abdomen in the control group, and at the same intervals plus the induction of sepsis, prior to increasing abdominal pressure, in the study group. With the elevation of the IAP a reduction of renal tissue oxygenation presents itself, which is more pronounced in the presence of sepsis, especially for IAP over 15 mmHg. Like other parameters, after abdominal decompression the renal tissue oxygenation returns to the initial levels, independently of sepsis.
Conclusions: The afferent arterioles vasoconstriction, which takes place during sepsis, and the intra-renal shunt, which occurs and leads to blood diversion to the medulla from the renal cortex due to the combination of intra-abdominal hypertension (IAH) and sepsis, seem to explain this finding.

1. Introduction

The classic compartment syndrome is the outcome of the swelling of soft tissue after ischemia–reperfusion, bone fractures, or crush injuries. Treatment involves urgent recognition and decompression, and also treatment of problems caused in the systemic circulation by reperfusion [1]. The recognition of the abdomen as a compartment and the adverse effects of increased pressure in the abdominal cavity were firstly described at the end of the 19th century by Marey and Burt, who highlighted the effects of intra-abdominal hypertension (IAH) on the respiratory system [2]. The first specific reference to the effects of IAH on the renal system was recorded in the early 20th century by Wend, who first associated IAH with renal impairment [1,3].

In the last decades, an increased interest in IAH has been observed mainly due to the development of laparoscopy, which has contributed to the study and understanding of the pathophysiological mechanisms that are caused by the increased intra-abdominal pressure (IAP) in various systems [4–6]. The terms IAH and abdominal compartment syndrome (ACS) are not identical but different stages of the same pathological process. IAH is an early phenomenon, which if not corrected leads to the full development of ACS [7,8]. The exact level of IAP above which it is considered IAH has not yet been clarified. Malbrain refers to unfavorable effects of increased IAP on organ function for values over 10 mmHg [9]. Ivatury et al. indicate that during their practice they consider IAH as the constant pressure greater than 20–25 cm H_2O (14.7–18.4 mmHg) [7]. Burch et al. published in 1996 a classification system of IAH, which correlates the values of IAP with treatment [10] that is used today with slight modifications [11]. Almost everyone agrees that at the level of 15 mmHg (20.4 cm H_2O), the basic pathophysiological disorders of IAH have already been established, while at the level of 30 mmHg (40.8 cm H_2O) ACS is present.

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Very important is the relationship of ACS with sepsis. Sepsis may be the causal factor of ACS, as in the case of development of it after massive fluid therapy for the reduction of the septic shock in cases of fecal peritonitis and intra-abdominal abscess. Sepsis can also be a concomitant condition from the beginning or in the course of ACS, as in post-traumatic situations, after burns, obstructive ileus, and several postoperative complications [12].

This experimental study was designed to evaluate the renal tissue oxygenation under the coexistence of ACS and sepsis. Tissue oxygenation is a method that can provide significant information regarding tissue microcirculation, and its use in the near future in clinical practice can make the treatment of critically ill patient better and more effective.

2. Material and methods

All the experiments were performed at the Experimental Laboratory of the Intensive Care Unit of General Regional Hospital "George Papanikolaou" from March 2002 until December 2004 after the approval issued by the Veterinary Authority of Thessaloniki (Protocol Number: 13/6541/30.05.02). The experiment was executed according to the provisions set by the European Union and other applicable laws for the protection and proper treatment of vertebrates, which are used for research and experimental purposes, and was conducted under the supervision of a veterinarian.

Fourteen non-breed dogs from both genders who weighed 10–25 kg were studied. The animals were divided into two groups as follows: the control group A (8 animals) and the study group B (6 animals). The specific animals in the experiment were chosen due to their significant similarities with humans in terms of hemodynamic, respiratory, and renal functions.

2.1. Anesthesia

All animals were treated with pre-anesthetic intramuscular injection of xylazine hydrochloride (ROMPUN® 2%/BAYER) at a dose of 1 ml/kg body weight, and atropine sulphate (Atropine Sulfate®/DEMO) at a dose of 0.02 mg/kg body weight. Percutaneous puncture of the right brachial vein and placement of an angiocatheter 18G (Abocath®-T) was performed after 15–30 min. From this catheter, sodium thiopental (Pentothal®/ABBOTT) 1.25% was bolus injected at a dose of 5 mg/kg body weight to induce general anesthesia. Following this, tracheal intubation was performed with a Shiley tube with an air chamber of appropriate size that was connected with the Siemens SERVO 900C ventilator. The breathing pattern used was controlled ventilation throughout the experiment in which the fraction of inspired oxygen ensured $PaO_2 > 100$ mmHg and the per minute ventilation ensured $PaCO_2$ 30–50 mmHg and pH 7.2–7.6.

Anesthesia was preserved by continuous drop wise administration of thiopental sodium solution with an infusion rate of 2 mg/kg weight/hour through the brachial vein. In order to provide analgesia, fentanyl (Fentanyl®/JANSSEN) was given at a dose of 15 mg/kg/h. As a muscle relaxant, atracurium was selected at a dose of 1 mg/kg/h. The choice of this particular muscle relaxant was made due to its specific metabolism mode (self-decomposed) which does not affect renal function. Additionally, adequate liquids were administrated through the brachial vein (solution Ringers/Lactate and Normal Saline) in order to maintain the central venous pressure, the pulmonary capillary wedge pressure, and the cardiac output at the starting levels.

2.2. Measurements – catheterization

The pulmonary compliance was continuously monitored by the breathing system Lung Mechanics Calculator 940 Siemens, and the electrophysiology of the cardiac muscle was monitored through the recording of Lead II by attached electrodes on the chest wall. For this reason a Datex Engstrom CS/3 monitor was used.

Thereafter, the right femoral vessels were surgically exposed for the installation of the following:

A. Angiocatheter 16G (Abocath®-T) was placed in the femoral artery not only for the continuous monitoring and recording of blood pressure, but also for the arterial blood sampling to determine blood gases. The blood gas analysis was made by the Radiometer Copenhagen ABL 330 gas analyzer and the determination of hemoglobin in each sample was performed with Hemoximeter OSM 3 by Radiometer Copenhagen.
B. The Swan-Ganz catheter, 8F (Opti-Q®, Abbott) was placed in the pulmonary artery via the femoral vein, the inferior vena, and the right atrium and ventricle. The catheterization was necessary for the measurement and recording of the pressure in the right atrium, the pulmonary artery wedge pressure in pulmonary capillaries, and also for the continuous monitoring of cardiac output.

The display and recording of these parameters were made with the monitor.

2.3. Surgical interference

With a lateral thoracolumbar incision approximately 10 cm long, the left kidney was surgically exposed. Thereafter, the special probe for the measurement of the tissue oxygenation was placed in the cortical part of the kidney, 3 mm under the kidney's capsule. The stabilization of the probe was followed by the airtight closure of the incision. The tissue oxygenation and the local temperature were recorded by the monitor Tissutrak (Pfizer Biomedical Sensors Ltd., England).

With a small subumbilical incision on the midline (open technique), a 5 mm trocar (VERSAPORT® 5 mm, Autosuture) was placed in the peritoneal cavity for the administration of carbon dioxide, the creation of pneumoperitoneum, and the achievement of the required IAP. The gas (carbon dioxide) was administered through an appropriate insufflator (SOLOS ENDOSCOPY, PAP III).

At the end of the experiment, the animals were euthanized with a bolus injection of sodium thiopental at a dose of 15 mg/kg along with the simultaneous disconnection of the mechanical ventilation.

2.4. Statistical analysis

During the statistical analysis of the experimental data, the following method was observed: Initially, the One-sample Kolmogorov–Smirnov test was performed for each parameter and for each stage of the experiment individually with the aid of which it was found that the data followed a normal distribution. Then, the Paired samples t-test and the Independent samples t-test were used for paired and independent observations respectively to determine whether there are significant statistical differences in the parameters during the different phases of the experiment. For the parameter $PtiO_2$, the Wilcoxon test and the Sign test were also used. Statistical difference was considered significant for $p < 0.05$.

2.5. Study protocol

After the end of the catheterization and the surgical incision ($t = 0$) in both the groups, no measurement or intervention was made for 30 min so as to achieve the required balance.

After 30 min, the values of the parameters were measured at rest for the control group (A) (PHASE A1) and for the study group (B) (PHASE B1) ($t = 30$).