Title: CARDIOVASCULAR, PULMONARY, AND RENAL EFFECTS OF MASSIVELY INCREASED INTRA-ABDOMINAL PRESSURE IN CRITICALLY ILL PATIENTS

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Introduction: Massive elevation of intra-abdominal pressure (IAP) causes cardiovascular, respiratory and renal dysfunction which progressively becomes difficult or impossible to manage unless IAP is reduced. This report describes the cardio-respiratory-renal profile which enables a better understanding of the pathophysiologic consequences of high IAP.

Methods: Six critically ill patients with massive intra-abdominal hemorrhage causing elevated IAP, measured by water manometry via a nasogastric or gastrostomy tube, had cardiovascular measurements determined immediately before and 15 minutes after a volume challenge of 10 ml/kg over 10 minutes. Six patients underwent surgical decompression of the abdomen, and data immediately before and 15 minutes after decompression are reported on the four patients in whom complete data were obtained.

Ejection fraction was calculated from portable mTC 99 gated pool scans or bedside portable echocardiograms. Cardiovascular measurements were obtained from indwelling arterial and pulmonary artery catheters. All patients were ventilated with volume ventilators to maintain $PaCO_2$ as normal as possible [43 ± 3 (SE) mm Hg].

Data were statistically analyzed by paired t tests, comparing each patient before and after volume challenge or surgical decompression. P values <0.05 were considered significant.

Results: Baseline Data. Mean IAP averaged 51 ± 7 cm h_20. (Normal=0 or less). Left ventricular (LV) ejection fraction averaged 55 ± 6% and LV end diastolic volume averaged 64 ± 14 ml. LV contractile pattern by gated pool scan typically revealed a small to normal hyperdynamic ventricle. Peak inspiratory pressures were very high (65 ± 5 cm H_20--dynamic compliance was extremely low (17 ± 2 ml/cm H_20)--PaO_2 and $PaCO_2$ were adequate on mechanical ventilatory support and urine output averaged 10 ml/hr.

Volume Challenge of 10 ml/kg improved cardiovascular function within 10 minutes-Fig. 1. Blood gases and respiratory measurements were unchanged. Laparotomy performed on four patients immediately and significantly improved cardiorespiratory function and urine output-Fig. 2. Three of six patients who underwent decompressive laparotomy survived to hospital discharge. The other three patients survived laparotomy, but died before hospital discharge. The two patients who did not undergo surgical decompression died within a few days of their volume challenge.

Discussion: Patients with elevated IAPs due to massive intra-abdominal hemorrhage manifested a cardiovascular profile of high filling pressures and low cardiac output despite apparent hypovolemia. The presence of tachycardia, oliguria and respiratory variation in the arterial pressure tracing suggest hypovolemia, but high left and right atrial pressures may confuse the clinical picture. Pericardial tamponade was ruled out as the reason for high filling pressures and low cardiac output. Furthermore, an ejection fraction of 55% and small to normal LV volumes do not support a cardiogenic basis for the low-output state. Either measured filling pressures are not accurately transmural in this condition or LV and RV compliance is dramatically decreased given the end diastolic volumes observed. Similar but less dramatic responses to high IAP have been reported in patients with ascites, in women undergoing laparoscopy and in dogs whose IAP is elevated by fluid installation or gas insufflation. Elevated IAP probably interferes with ventricular filling by compressing the diaphragm upward, transmitting pressure to the heart and great vessels, leading to a hemodynamic profile of low ventricular compliance, high filling pressures and low output. Therapy must be directed towards decompressing the abdomen since medical management of the low output state by raising filling pressures further, increasing heart rate or increasing inotropy to an already vigorously contracting ventricle becomes counterproductive. Increased dead space and high compression volume resulting from the high peak airway pressures were probably why we were unable to lower $PaCO_2$ below 43 mm Hg despite V_E 13 ± 1.5 l/min.

Conclusion: Once the reasons for the low output state and the required maximal respiratory support are understood, patients with massively elevated IAP who are beyond help with medical therapy should proceed to surgical decompression to maintain their viability.

Fig. 1

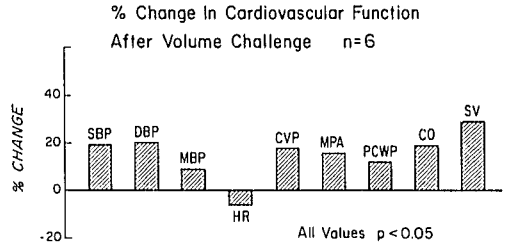

Fig. 2