Mini Review

Journal of Emergency Medicine, Trauma & Surgical Care

Volume Kinetic Shocks in Surgical Practice

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Abstract

Volume kinetic shocks are cardiovascular shocks induced by acute substantial volume changes of the cardiovascular system in either direction by decrease or increase. A decrease in cardiovascular volume induces the long established and well-known hypovolemic/hemorrhagic shocks. Cardiovascular shocks induced by volumetric overload have recently been reported first in 2016. Volumetric overload shocks (VOS) are of two types, depending on the type of fluid inducing it: Sodium-free fluid induce type 1 (VOS 1) and sodium-based fluid induce type 2 (VOS 2).

These VOS present with cardiovascular shock or cardiopulmonary arrest in theatre and the acute respiratory distress syndrome (ARDS) later. It is iatrogenic complication of fluid therapy that is under recognized and underestimated. VOS1 is induced by infusion of 3.5-5 liters of sodium-free fluid in one hour and is characterized with dilution hyponatraemia. VOS2 may complicate VOS 1 or may occur de novo complicating sodium-based fluid therapy during resuscitation of shock, acutely ill patients, and prolonged surgery. It has no obvious serological markers or none. Many errors and misconceptions mislead physicians into giving too much fluid for resuscitation of shock due to faulty rules on fluid therapy dictated by the wrong Starling's law. The correct replacement for this law is the hydrodynamic of the porous orifice G tube. Discovery of VOS has resolved the puzzles of the transurethral resection of the prostate (TURP) Syndrome, Hyponatraemia (HN) and the Acute Respiratory Distress Syndrome (ARDS) or the multiple organ dysfunction syndrome (MODS).

Keywords: Acute Respiratory Distress Syndrome (ARDS); Capillary physiology; Hyponatraemia; Shock; Starling's law; Transurethral Resection of the Prostate (TURP) syndrome; Volumetric overload Socks

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Received Date: July 16, 2020

Accepted Date: August 03, 2020

Published Date: August 10, 2020

Citation: Ghanem AN (2020) Volume Kinetic Shocks in Surgical Practice. J Emerg Med Trauma Surg Care 2: 010.

Introduction

Volume kinetic (VK) shocks are cardiovascular shocks induced by acute substantial volume changes of the cardiovascular system in either direction by decrease or increase. A decrease in cardiovascular volume induces the long established and well-known hypovolemic/hemorrhagic shocks. Cardiovascular shocks induced by volumetric Overload (VO) have been recently reported [1-4], first in 2016. Volumetric Overload Shocks (VOS) are of two types, depending on the type of fluid inducing it: Sodium-free fluid induce type one (VOS1) and sodium-based fluid induce type 2 (VOS 2). Both types of VOS complicate fluid therapy in clinical practice affecting mostly surgical patients during the resuscitation of shock, acutely ill patients and during prolonged major surgery.

There are many errors and misconceptions on fluid therapy [5,6] that mislead physicians [7] into giving too much fluid during shock resuscitation. These errors are dictated by faulty rules on fluid therapy induced by the wrong Starling's law [8-10] that dictates these faulty rules. This transfers the shock being treated such as hemorrhagic or septic shock into VOS, seamlessly and un-noticed when excessive crystalloids and/or colloids fluids are infused. Examples of VOS 1 are the condition known in urology as the transurethral resection of the prostate (TURP) syndrome [11] or hyponatremic shock [12].

The TURP syndrome is induced by massive fluid gain of 3.5-5 liters mostly of 1.5% Glycine and/or 5%Glucose infusion in one hour. Hyponatremic shock is induced by excessive 5%Glucose infusion during prolonged major surgery in women. The TURP syndrome occur when large volume of the irrigating 1.5% Glycine is absorbed during the one-hour TURP surgery. This VOS 1 occurs during or immediately after surgery presenting to anesthetists and surgeons and is usually mistaken for one of the known shocks of hemorrhagic or septic and gets wrongly treated by further volume expansion using crystalloids and/or colloids with disastrous or lethal outcome. By next morning after surgery it presents to physicians with hyponatremic encephalopathy manifesting with coma, convulsion. and paralysis [13]. Other manifestations of the multiple organ dysfunction syndrome (MODS) [14,15], also known as the acute respiratory distress syndrome (ARDS), do occur, but one system may predominate (Table 1).

Also occurs the excessive bleeding at the surgical site and Leucocytosis also occurred in the absence of sepsis and septic shock. Although sepsis and septic shock do certainly cause ARDS, it may perhaps be as innocent as the Wolf in Josef story in most situations.

VOS 2 may complicate the therapy of VOS 1 when excessive crystalloids and colloids are infused. It may complicate other types of known shocks when excessive crystalloids and/or colloids fluids are infused. It is hard to recognize in this setting and impossible to differentiate from the shock being treated as it has little or no serological marker like hyponatraemia of VOS 1. It presents later with ARDS or MODS thus VOS causing ARDS has been established beyond any doubt [14,15]. Although fluid retention in ARDS patients has recently been reported in prospective huge multi-center trials in surviving patients ranging from 3-10 liters [16,17], and in mortality cases ranging from 12-14 liters [18], it has never before been incriminated in the patho-aetiology of ARDS till recently [14,15].

Cerebral	Cardiovascular	Respiratory	Renal	Hepatic & GIT
Numbness Tingling SBB[1] COC[2] **Coma** Convulsions PMBCI[3]	**Hypotension** **Bradycardia** **Dysrhythmia** **CV Shock*** **Cardiac Arrest** **Sudden Death**	Cyanosis. FAM[4] APO)[5] RA[6] **Arrest** **CPA**[7] Shock lung ARDS8	Oliguria Annuria[8] Renal failure or AKI[9] Urea ↑ Creatinine ↑	Dysfunction: Bilirubin ↑ SGOT ↑ Alkaline Phosph. GIT symptoms. DGR[10] Paralytic ileus Nausea & Vomiting.

Table 1: Shows the manifestations of VOS 1 of the TURP syndrome which is the same as that of ARDS manifestations of the multiple organ dysfunction syndrome (MODS) induced by VOS2.

Abbreviations: SBB[1]: Sudden bilateral blindness; **PMBCI**[3]: Paralysis mimicking bizarre cerebral infarctions, but is recoverable on instant use of HST of 5% NaCl and/or NaCo3, and so is coma and AKI; **FAM**[4]**:** Frothing around the mouth; **APO**[5]**:** Acute pulmonary oedema; **RA**[6]**:** Respiratory arrest; **CPA**[7]**:** Cardiopulmonary arrest; **ARDS**[8]**:** Acute respiratory distress syndrome. Occurs later on ICU; **AKI**[9]**:** Acute kidney injury; **DGR**[10]**:** Delayed gut recovery; **CV Shock***: Cardiovascular shock of VOS reported here as VOS1 and VOS2; **Annuria**[8]**:** That is unresponsive to diuretics but responds to HST of 5% Nacl and/or 8.4%$NaCo_3$; **AKI**[8]**:** Acute kidney injury.

Professor Hahn from Sweden studied VK in healthy volunteers and patients [19,20]. He concluded that: "Guidelines for fluid therapy rarely take into account that adverse effects occur in a dose-dependent fashion. Adverse effects of crystalloid fluids are related to their preferential distribution to the interstitial of the subcutis, the gut, and the lungs. The gastrointestinal recovery time is prolonged by 2 days when more than 2 liters is administered. Infusion of 6-7 liters during open abdominal surgery results in poor wound healing, pulmonary oedema, and pneumonia. There is also a risk of fatal postoperative pulmonary oedema that might develop several days after the surgery. Even larger amounts cause organ dysfunction by breaking up the interstitial matrix and allowing the formation of lacunae of fluid in the skin and central organs, such as the heart. For both crystalloid and colloid fluids, coagulation becomes impaired when the induced hemodilution has reached 40%. Coagulopathy is aggravated by co-existing hypothermia. Although oedema can occur from both crystalloid and colloid fluids, these differ in pathophysiology."

Other authors also found a significant effect of crystalloids overload on mortality as they did the research during the first 24-48 hours from hospital admission. I have found only one prospective study on adults' trauma patients by Jones et al (2016) [21], and one prospective paediatrics study by Coons et al (2018) [22] and a remarkable review article by Schrier. Reported in 2010 [23], that incriminate saline overload and recommend judicious use of fluid infusion during resuscitation of shock and trauma. In patients of these adult and paediatrics trauma trials there is no sepsis involved and both were done over a period of 24 and 48 hours, respectively. Both articles detected a significant relationship of VO with morbidity and mortality of ARDS.

Jones et al. [21] reported in conclusion: "Large-volume crystalloid resuscitation is associated with increased mortality and longer time ventilated. Based on this data, we recommend judicious use of crystalloids in the resuscitation of trauma patients." The conclusion by Coons et al [22] was: "Early administration of high volumes of crystalloid fluid greater than 60 ml/kg/day significantly correlates with pulmonary complications, days NPO, and hospital length of stay. These results span the first 48 h of a patient's hospital stay and should encourage surgical care providers to exercise judicious use of crystalloid fluid administration in the trauma bay, ICU, and floor".

There are currently substantial physics [8,9] and physiological [10] evidence that Starling's law is wrong. It is responsible for the faulty rules on fluid therapy [5] that mislead physicians [7] into giving too much fluid during the resuscitation of shock that induce VOS causing ARDS [14,15].Other authors have found that Starling's forces do not hold in clinical practice [23-25]. The editor of The British Journal of Anaesthesia 2012 commented on this article [25]. "The classic Starling principle does not hold for fluid resuscitation in clinical setting." I have not only proved that Starling's law is wrong on both of its forces, but also have provided its correct replacement; the hydrodynamic of the porous orifice (G) tube [8-10] (Figure 1).

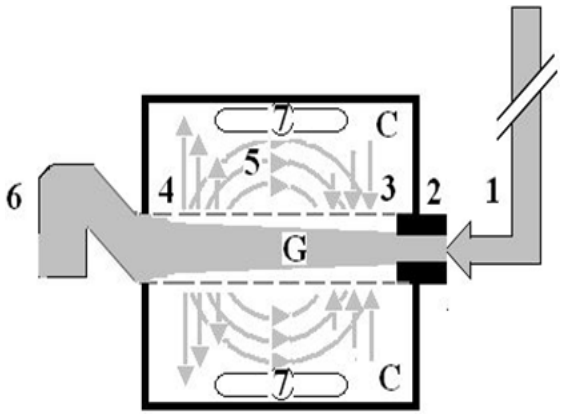

Figure 1: Shows a diagrammatic representation of the hydrodynamic of G tube based on G tubes and chamber C. This 38-years old diagrammatic representation of the hydrodynamic of G tube in chamber C is based on few photographs. The G tube is the plastic tube with narrow inlet and pores in its wall built on a scale to capillary ultra-structure of precapillary sphincter and wide inter cellular slit pores. The chamber C around it is another bigger plastic tube to form the G-C apparatus. The chamber C represents the ISF space. The diagram represents a capillary-ISF unit that should replace Starling's law in every future physiology, medical and surgical textbooks, and added to chapters on hydrodynamics in physics textbooks. The numbers should read as follows:

1. The inflow pressure pushes fluid through the orifice
2. Creating fluid jet in the lumen of the G tube**.
3. The fluid jet creates negative side pressure gradient causing suction maximal over the proximal part of the G tube near the inlet that sucks fluid into lumen.
4. The side pressure gradient turns positive pushing fluid out of lumen over the distal part maximally near the outlet.
5. Thus, the fluid around G tube inside C moves in magnetic field-like circulation (5) taking an opposite direction to lumen flow of G tube.
6. The inflow pressure 1 and orifice 2 induce the negative side pressure creating the dynamic G-C circulation phenomenon that is rapid, autonomous, and efficient in moving fluid and particles out from the G tube lumen at 4, irrigating C at 5, then sucking it back again at 3,
7. Maintaining net negative energy pressure inside chamber C.

****Note:** The shape of the fluid jet inside the G tube (Cone shaped), having a diameter of the inlet on right hand side and the diameter of the exit at left hand side (G tube diameter). I lost the photo on which the fluid jet was drawn, using tea leaves of fine and coarse sizes that runs in the center of G tube leaving the outer zone near the wall of G tube clear. This may explain the finding in real capillary of the protein-free (and erythrocyte-free) sub-endothelial zone in the Glycocalyx paradigm (Woodcock and Woodcock 2012) [25]. I also noted that fine tea leaves exit the distal pores in small amount maintaining a higher concentration in the circulatory system- akin to plasma proteins.

Discovery of VOS has resolved the puzzles of the TURP Syndrome, hyponatraemia and ARDS: Not only the exact patho-aetiology diagnosisof these conditions were precisely identified but also a lifesaving treatment of hypertonic sodium therapy (HST) of 5% NaCl and/or 8.4% $NaCo_3$was discovered and rejuvenated [14,15].

Conclusion

Acute, substantial volume kinetic in either direction of loss or gain causes cardiovascular shocks. Volumetric overload shocks (VOS) are newly recognized. It may present with shock or cardiopulmonary arrest in theatre and ARDS later. It is an iatrogenic complication of fluid therapy in hospitals that is under recognized and under-estimated. VOS is 2 types: VOS1 and VOS2. VOS1 is induced by 3.5-5 liters of sodium-free fluid and is characterized with dilution hyponatraemia (HN). VOS 2 may complicate VOS 1 or may occur de novo complicating sodium-based fluid therapy during the resuscitation of shock, acutely ill patients, and prolonged surgery. It has no obvious serological markers or none. Up to 10 liters of fluids are retained in surviving ARDS patients while those who die retain 12-14 liters. Many errors and misconceptions mislead physicians into giving too much fluid for resuscitation due to faulty rules on fluid therapy dictated by the wrong Starling's law. The correct replacement for this law is the hydrodynamic of G tube. Discovery of VOS has resolved the puzzles of TURP Syndrome, HN and ARDS.

Acknowledgements

I thank Dr. Khaled and Dr. Salma; son and daughter of the author, for constructing (Table 1) and for assistance with editing of this article.

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