

Volume Kinetic Shocks in Clinical Practice

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ABSTRACT

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

These VOS present with cardiopulmonary shock or arrest in theatre and ARDS later. It is iatrogenic complication of fluid therapy in hospitals that is under recognized and underestimated. VOS is 2 types: VOS1 and VOS2. VOS1 is induced by 3.5-5 liters of sodium-free fluid and is characterized with dilution hyponatremia. 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 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 transurethral resection of the prostate (TURP) Syndrome, hyponatremia and the acute respiratory distress syndrome (ARDS).

KEYWORDS

Cardiovascular shocks; Hyponatraemia; Prostate (TURP) Syndrome; Acute respiratory distress syndrome; Volumetric overload shocks (VOS)

1. INTRODUCTION

Volume kinetic (VK) shocks are cardiovascular shocks induced by acute substantial changes of the cardiovascular system volume in either direction by decrease or increase. A decrease in cardiovascular

volume induces the long established and well-known hypovolemic or hemorrhagic shock. Cardiovascular shocks induced by volumetric overload (VO) have recently been reported [1-3]. Volumetric overload shocks (VOS) are of two types, depending on the type of fluid

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inducing it: Sodium-free fluid induces type one (VOS 1) and sodium-based fluid induce type 2 (VOS 2). Both types of VOS complicate fluid therapy in clinical practice during the resuscitation of shock, acutely ill patients and during prolonged major surgery. These VOS are under recognized and underestimated. It was first reported recently in 2016.

There are many errors and misconceptions on fluid therapy [4] that mislead physicians into giving too much fluid during shock resuscitation [5]. These errors are dictated by faulty rules on fluid therapy induced by the wrong Starling's law [6]. These errors transfer the shock being treated such as hemorrhagic or septic shock into VOS. It occurs seamlessly and un-noticed when excessive fluids are infused. Examples of VOS 1 are the condition known in urology as the transurethral resection of the prostate (TURP) syndrome [7] or hyponatremic shock [8]. The TURP syndrome is induced by massive absorption of 1.5% Glycine and/or 5% Glucose infusion. Hyponatremic shock is induced by excessive 5% Glucose infusion. 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 with further volume expansion using crystalloids and/or colloids.

By next morning after surgery VOS 1 presents to physicians with hyponatremic encephalopathy manifesting with coma, convulsion and paralysis [9]. Also, 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 fluids are infused. It has no clear serum marker like hyponatremia. Hence it is hard to recognize in this setting and impossible to differentiate from the shock being treated. It presents later with the acute respiratory distress syndrome (ARDS) or the multiple organ dysfunction syndrome (MODS) [9,10].

Professor Hahn studied VK of infused fluids in healthy volunteers and patients [11,12]. He reported 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 litres - 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. This is demonstrated in one study on adults' trauma patients by Jones et al. [13], and one paediatric study by Coons et al. [14] and a remarkable review article by Schrier [15], reported in 2010 that incriminate saline overload and recommend judicious use of fluid infusion during resuscitation. In patients of these adult and paediatric 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 [13] reported: "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 [14] 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 [16,17] and physiological [18] evidence that Starling's law is wrong. It is responsible for the faulty rules on fluid therapy that mislead physicians into giving too much fluid during the resuscitation of shock that induce VOS causing ARDS [6]. Other authors have found that Starling's forces do not hold in clinical practice [19]. I have not only proved that Starling's law is wrong on both of its forces, but also have provided its correct replacement of the hydrodynamic of the porous orifice (G) tube (Figure 1).

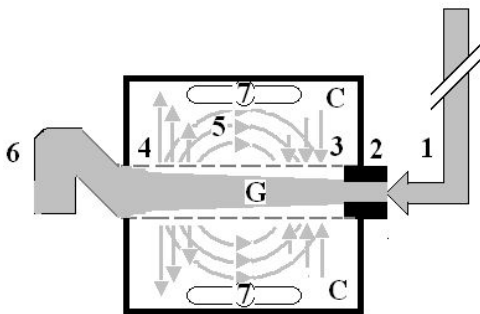


Figure 1: A diagrammatic representation of the hydrodynamic of G tube based on G tubes and surrounding chamber C. The G tube is the plastic tube with narrow inlet and pores in its wall built on a scale to capillary ultra-structure of precapillary sphincter and wide inter cellular slit pores, and the chamber C around it is another bigger plastic tube to form the G-C apparatus. The chamber C represents the interstitial fluid space.

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) [19].

The diagram represents a capillary-ISF unit, and 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.

Summary

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 cardiopulmonary shock or arrest in theatre and ARDS later. It is an iatrogenic complication of fluid therapy in hospitals that is under recognized and underestimated. VOS is 2 types: VOS1 and VOS2. VOS1 is induced by 3.5 litres - 5 litres of sodium-free fluid and is characterized with dilution hyponatremia. 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 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 G tube. Discovery of VOS has resolved the puzzles of TURP Syndrome, HN and ARDS.

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