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Opinion

Shock in Polytrauma: Highlighting the Volumetric Overload Shocks and Hydrodynamic Phenomenon of the Porous Orifice (G) Tube

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Received: November 27, 2018; **Published:** December 28, 2018

Professor Paul Pepe's editorial [1] on shock in polytrauma is commendable for his call for reconsideration and re-definition. He hinted at the role of iatrogenic overzealous fluid infusions in its patho-aetiology, mentioning that segregating confounders of hypotension, even taking a nihilistic approach, is a must to resolve this dilemma. I have been investigating this condition with diverse clinical presentations reaching vital conclusions that hold the keys for resolving this dilemma but remained overlooked.

The following statements are based on 32 years of studies, some reported as communications [2-10], MD Thesis [11] and articles [12,13]. A summary of some overlooked issues, new concepts and discoveries made since my initial thoughts were reported at BMJ [2,3] is mentioned here. The shock that complicates overzealous fluid therapy during polytrauma resuscitation [1] may also complicate various recognized shocks [14-17], diverse diseases requiring fluid therapy [18] or parenteral nutrition [19] on intensive care units and during prolonged major surgery [20]. The prevalence of morbidity and mortality of this condition is staggering yet if it has attracted a fraction of the attention given to AIDS, it should have been resolved by now.

Iatrogenic overzealous fluid infusion may induce volumetric overload shocks (VOS) that have diverse clinical presentations depending on the type of fluid gained, volume and time [9]. The severity of the signs is directly proportional to the VO but inversely to time (VO/T) [12]. Fluid type determines the changes of serum solutes and presentation; pure water is most toxic causing haemolysis; sodium-free fluids (VO1) are characterized with dilution hyponatraemia (HN) [12,15,21] with a nadir proportional to severity [9,12]. The gain of VO1 such as 5% Dextrose, 3% Mannitol and 1.5% Glycine may occur either via excessive infusion [14,17,18] or inadvertent absorption of irrigating fluids used in endoscopic surgery- known as the transurethral resection of the prostate (TURP) syndrome [12,15,16,21]. It also affects women undergoing trans-cervical endometrial resection [22], other patients and children who are infused with excessive VO1 fluids [23,24].

Although this VO1 is characterized by the obvious marker of dilution HN [12,14,15-19,21,22], it presents with cardiovascular shock [12,15,16] and encephalopathic coma [21-24]. Manifestations of respiratory, cardiac, renal and hepatic failures and coagulopathy [17] are also evident and are recognized as the multiple vital organ dysfunction or failure (MVOD/F) syndrome [9,12]. Acute renal failure (ARF) prevents urinary excretion and sodium loss, so serum HN in VO1 shock is mainly dilutional [9-12]. The hypotension of VO1 is usually mistaken for known shocks of haemorrhage or septicaemia [17]. Hence, it is wrongly treated with further vascular expansion using crystalloids, colloids and blood.

Such VO1 shock and its hypertonic sodium therapy (HST) of 5% NaCl was reported 7 decades ago [14,15] and rejuvenated [3,12] as successful life-saving therapy but it was thought contra-indicated until it recently rectified by the authorities on HN [22,23]. Intravenously infused fluid of the same quantity, type and time induces identical systemic signs in both humans and animals [21], irrespective of the initial access route to the vascular system and later variable distribution into the body fluid spaces [9,12].

Sodium-based fluids (VO2) of crystalloids, colloids and blood, although better tolerated, induce signs of VOS without marked serological markers, affirming the concept of VOS [12] but it remains disbelieved. A normal daily intake of 3.5L of fluid causes signs when intravenously infused over 2 - 3h but can be a serious gain in < 1h. Why is it so difficult to recognize these facts on encountering serious cases even of VO1 that is characterized with HN? Although the systemic and bizarre signs of severe TURP syndrome are well documented in case reports, it is extremely difficult to relate to VO/T and fluid type, even on monitoring the gained volume, measuring blood loss and excluding septicaemia.

The complex signs of cardiovascular disturbance of shock and MVOD/F of the TURP syndrome are very variable in severity, up to arrest/death, with many presentation masks and differential diagnoses [9,12]. Hence, when seen in the complex surgical setting, they are wrongly attributed to known causes of shock, coma, respiratory distress, renal and heart failure as signs of MVOD/FF that may occur in any combinations [9,12]. Of the well documented presentation masks of VO1, one is shock apparent to surgeons and anaesthetists after the surgery [12,15-17] and another is coma [22,23], recognised later by physicians. More important, neither the concept nor mechanism of VOS by disturbing capillary dynamics has been recognized, despite explaining the patho-physiology of the TURP syndrome and shock, highlighting its link with MVOD/F [7-13].

Sodium-based fluids VO2 induce VOS2 that has no marked serological markers but has all manifestations of MVOD/F. This is the type seen on treating recognized shock of polytrauma with overzealous infusions of crystalloids, colloids and blood [1]. The hypotension of VOS2 is unrecognizable from the shock being treated and the transition is undetectable. Advances in circulatory support and ventilation [25] have altered presentation but little improved outcome, adding more masks to the already confusing picture. So what was initially reported as the acute respiratory distress syndrome (ARDS) [20] became latter known as MVOD/F and is currently named systemic inflammatory response syndrome (SIRS) [25]. The volumetric overload is evident by oedema of the torso seen in VOS and MVOD/F or ARDS, detected by the increase in body weight. The VO gain may equal or double the total blood volume of 5 - 10% body weight. A researcher with interest in the mentioned conditions who keeps the above concepts in mind will not be confused by the variety of named syndromes and conditions documented in thousands or even millions of reported research articles in which VO is an obvious culprit but is rarely incriminated, why?

Ever since fluid therapy has proved life-saving therapy for polytrauma victims during the 2nd World War and in clinical practice later, it has become firmly implanted in the minds of generations of physicians that every hypotension is synonymous with hypovolaemia. Adding to the difficulties, VO/T concept as a cause of shock is paradoxical to the received concept of treating all shocks with vascular expansion, indiscriminately [7,25]. This is specifically correct in hypotension due to hypovolaemia or haemorrhage shock (where it should have a limit but is currently unknown), and otherwise flatly wrong. My research has traced this misconception to an erroneous underlying physiological law, namely

Starling's law on capillary-interstitial fluid exchange [12,13]. This along with the known direct proportional relationship of volume and pressure on filling solid reservoirs has deeply rooted the misconception. However, pressure-volume relation does not apply in most systems, and certainly not in the vascular system. Even in a solid reservoir it has a limit imposed by capacity of reservoir and maximal compression of the fluid/gas used.

Much research and arguments has been made on the type of resuscitation fluid used in shock but rarely the quantity versus capacitance of vascular system and time were considered [12]. The normal blood volume is 5L and maximum capacitance of the adult cardiovascular system is 7L, and although 3.5L is about the normal daily fluid requirements, infusing such volume in < 1h induces a typical VOS1 of the types mentioned above. The issues involved on discussing all shocks and in polytrauma are most complicated, generating endless

arguments on a faulty basis. Hence, it is vital to identify, re-evaluate and understand its correct basic physics and physiology. It should be realized that VO of such quantity gained in such time (VO/T) induces hypotension shock much like volume loss. This means that an acute change of circulatory volume in either direction induces shock. The capillary circulation is the place where all shocks act and induce its cellular and tissue harm, its re-evaluation should redefine shock, improve its management and resolve the dilemma.

Shock may be defined as disturbance of capillary circulation hindering oxygen/nutrient delivery and carbon dioxide/waste products removal causing damage of tissues and cells. Starling's law attributed capillary filtration mainly to arterial pressure and absorption to plasma proteins. This proposal was based on Poiseuille experiments on hydrodynamics of long uniform Bras tubes, and on vetro experiments on oncotic plasma pressure across membrane permeable only to water. The anatomy of the capillary tube, discovered 7 decades after Starling proposed his hypothesis, shows that it is essentially a "porous orifice tube" with pre-capillary sphincter and holes in its wall that allow the passage of molecules larger than plasma proteins. The latter fact nullifies the role attributed to plasma protein's oncotic pressure. The pre-capillary sphincter has a vital but overlooked role in the capillary-interstitial fluid circulation.

My research on the hydrodynamic of porous orifice tubes, akin to capillary, and incorporated in a circulatory system explored this issue [13]. It demonstrated that proximal pressure, akin to arterial pressure, induces an opposite effect to that proposed by Starling. The orifice created negative side pressure gradient maximal at proximal part near the inlet causing suction or absorption. The distal pressure, akin to venous pressure, is the force inducing filtration maximally at distal pores near exit [13,26].

The interacting effect of proximal and distal pressures on porous orifice tube revealed new hydrodynamic magnetic field-like circulation phenomenon between fluid in the tube lumen and that in a surrounding chamber, akin to the interstitial space. The circulation between fluid in the tube lumen and its surrounding chamber showed fluid to enter tube lumen through proximal and leaves through distal pores, creating a net negative pressure in the surrounding chamber akin to that in the interstitial space. Fluid in the surrounding chamber moves in the opposite direction to tube flow. The orifice, akin to pre-capillary sphincter, plays vital role in the dynamics and speed of this circulation [13,26]. A drop in proximal pressure, elevation of the distal pressure and too narrow or too wide orifice slows down the circulation.

Over expansion of the circulatory system markedly slows down the dynamic magnetic field-like circulation, reverting pressure in surrounding chamber from negative to positive as occurs when overzealous fluid infusion floods the interstitial space inducing a combination of shock and interstitial oedema of torso seen in VOS2 and MVOD/F. It is worth mentioning the clinical observation that although arterial hypertension is most common it never causes oedema of the interstitial space, while minor elevation of venous pressure does. Starling's law fails to explain this fact while the phenomenon of the porous orifice tubes demonstrates and explains it on inducing similar changes in proximal and distal pressures, and orifice diameter [26]. Understanding this phenomenon may help to redefine shock and identify the new VOS [27-29], rectify the physiological errors and misconceptions on intravenous fluid therapy [30] that resolve the enigma of MVOD/F. No clinical study will produce useful results before the mentioned issues are reconsidered and stratified.

Competing Interest

None declared by the author.

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Volume 3 Issue 1 January 2019