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Cardiac arrest and volumetric overload shocks (VOS) complicating fluid therapy

Research Article

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Abstarct

Question: May cardiac arrest affect TURP patients?How may fluid resuscitation induce VOS and what is its link with the G tube?

Findings: Cardiac arrest occurs in TURP syndrome. Overzealous fluid infusion induces VOS1 and VOS2. VOS1 is induced by sodium-free fluids characterized with HN known as the TURP syndrome. VOS2 is induced with sodium-based fluids presenting as ARDS. The wrong starling's law caused errors misleading physicians into giving too much fluid. The correct replacement is the G tube hydrodynamic.

Meaning: Giving too much fluid during resuscitation of poly-trauma, shock and prolonged surgery induces the newly recognised VOS and causes ARDS. The faulty Starling's low underlies the errors and misconceptions on fluid therapy. 5%NaCl is lifesaving.

Introduction and objective: To report that volumetric overload shock (VOS) type 1 (VOS1) known as TURP syndrome and type2 (VOS2) may complicate fluid resuscitation of poly-trauma causing the acute respiratory distress syndrome (ARDS).

Materials and Methods: Evidence from analytical review of literature and results of my research demonstrates fluid therapy may induce VOS and cause ARDS. Errors on fluid therapy, underlying it all the wrong law of Starling, may mislead physicians into giving too much fluids. I conducted and reported 2 clinical, 1 physics and 1 physiological studies that investigated these issues.

Result: Overzealous fluid infusion induces 2 types of VOS, VOS1 and VOS2. VOS1 is induced by sodium-free fluids characterized with hyponatraemia (HN). VOS1 is known as the transurethral resection of the prostate (TURP) syndrome. Hypertonic sodium is lifesaving treatment. VOS2 is induced with sodium-based fluids and has no markers.VOS2 presents as ARDS. Both have features of the multiple vital organ dysfunction/failure (MVOD/F) syndromes. Coma, ARDS and acute kidney injuryoccur in any combination but one system may predominate. The wrong starling's law caused errors misleading physicians into giving too much fluid during resuscitation of shock and poly-trauma. The correct replacement is the hydrodynamic of the G tube.

Conclusion: Two new types of shocks are reported, VOS1 and VOS2. VOS1 is characterized with HN for which 5%NaCl is lifesaving treatment. Both present with MVOD/F syndromeor ARDS and induced by overzealous fluid infusion. The wrong Starling's law dictates the faulty rules on fluid therapy. The correct replacement is the hydrodynamic of the G tube.

Introduction

Cardiac arrest, dysrhythmia and cardiovascular shocks are the main presenting manifestations of the TURP syndrome. Coma and paralysis mimicking cerebro-vascular accidents are the main manifestations of the cerebral system. Respiratory arrest, acute respiratory distress syndrome (ARDS), and pulmonary oedema are respiratory manifestations. Acute kidney injury(AKI) is the manifestation of renal involvement. Hepatic dysfunction occurs. Coagulopathies affect the blood. These manifestations form the multiple vital organ dysfunction/ failure (MVOD/F) syndrome. These are the manifestations of a condition known in urology as the transurethral resection of the prostate (TURP) syndrome

In an editorial at BMJ on shock in poly-trauma Professor Paul Pepe [1] called for reconsideration and re-definition. He hinted at the role of fluid infusions in its patho-aetiology, mentioning that segregating confounders of hypotension, even taking a nihilistic approach, is a must to resolve this dilemma. My research has precisely achieved that. I have been investigating this condition with diverse clinical presentations reaching vital conclusions that hold the keys for resolving this dilemma but remained overlooked. These discoveries are summarised here.

Over 32 years of my career life I did many studies, some reported as communications [2-10], MD Thesis [11] and articles [12–13]. A summary of some overlooked issues, new concepts and discoveriesmade since my initial brain storming thoughts were reported at BMJ [2,3] is mentioned here. The shock that complicates overzealous fluid therapy during poly-trauma resuscitation [1], recognized as VOS2, may complicate various recognized shocks [14-17], diverse diseases requiring fluid therapy [18] or parentral 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.

Volumetric overload shocks (VOS) that complicate iatrogenic overzealous fluid infusion may induce 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; sodium-free fluids (VO1) are characterized with hyponatraemia (HN) [12,15,21] with the first nadir proportional to severity [9,12]. The gain of VO1 such as 5%Dextruse, 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 TURP syndrome [12,15,16,21] or hyponatraemic shock [15]. It also affects women undergoing trans-cervical endometrial resection [22]. Other patients and children who are infused with excessive VO1 fluids such as 5% dextrose were reported [23,24].

Although this VO1 is characterized by the obvious marker of HN [12,14,15-19,21,22], it presents with cardiovascular shock or cardiac arrest to surgeons and anaesthetists in theatre [12,15,16] and with encephalopathic coma to physicians later [21-24]. Manifestations of respiratory, cardiac, renal and hepatic dysfunction or failures [17] are also evident and are recognized as the multiple vital organ dysfunction or failure (MVOD/F) syndrome [9,12] or the acute respiratory distress syndrome (ARDS). Acute renal failure currently known as acute kidney injury (AKI) prevents urinary excretion and sodium loss, so serum HNin VO1 shock is mainly dilution [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, colloid substitutes 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 [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].

Crystalloids, colloids, colloid substites and blood are sodium-based fluids (VO2)., though 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 3.5 L of fluid causes signs when intravenously infused over 2–3 h but can be a serious gain in <1 h. 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 andMVOD/F of the TURP syndrome are very variable in severity, up to cardiac 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 or arrest as signs of MVOD/F that may occur in any combinations [9,12]. Of the well documented presentation masks of VO1, one is shock apparent to surgeons and anaesthetists during or immediately 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-aetiology of the TURP syndrome and shock, highlighting its link with MVOD/Fsyndrome [7-13]. A prospective study on 100 TURP patients among whom 10 suffered the TURP syndrome demonstrated that VO is the most significant factor in its patho-aetiology (Figure 1 and Table 1) [12]. Also a case series of 23 patients affirmed VO as the correct patho-aetiology of the TURP syndrome(Figure 2) [24].

Table 1: shows the multiple regression analysis of total per-operative fluid gain, drop in measured serum osmolality (OsmM), sodium, albumin, Hb and increase in serum glycine occurring immediately post-operatively in relation to signs of the TURP syndrome. Volumetric gain and hypoosmolality are the only significant factors.Volumetric overload is the most highly significant factor .(Reproduced with the permission of author and editor of BJU Int. from reference 12)

P	T Value	Std. Value	Std. Err	Value	Parameter
		0.773			Intercept
0.0001	3.721	1.044	0.228	0.847	Fluid Gain [1]
0.0212	2.42	-0.375	00.014	0.033	**Osmolality**
0.0597	1.95	0.616	0.049	0.095	Na+ (C_B)
0.4809	0.713	0.239	0.087	0.062	Alb (C_B)
0.2587	1.149	-0.368	0.246	-0.282	Hb (C_B)
0.4112	0.832	-0.242	5.975E-5	-4.973E-5	Glycine (C_B)

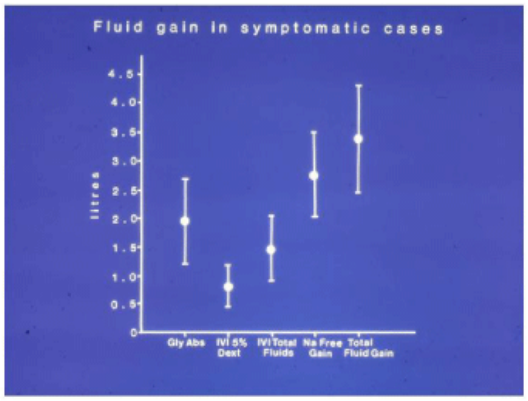

Figure 1: shows the means and standard deviations of volumetric overload in 10 symptomatic patients presenting with shock and hyponatraemia among 100 consecutive patients during a prospective study on transurethral resection of the prostate. The fluids were of Glycine absorbed (Gly abs), intravenously infused 5% Dextrose (IVI Dext) Total IVI fluids, Total Sodium-free fluid gained (Na Free Gain) and total fluid gain in litres..(Reproduced with the permission of author and editor of BJU Int. fromreference 12)

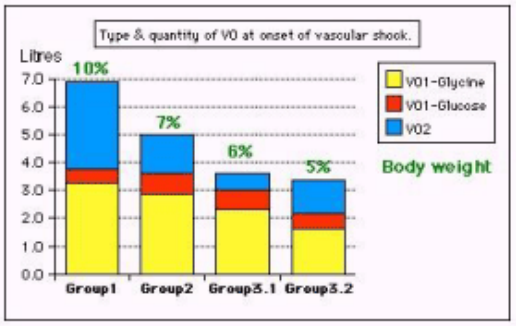

Figure 2: Volumetric overload (VO) quantity (in litres and as percent of body weight) and types of fluids. Group 1 was the 3 patients who died in the case series as they were misdiagnosed as one of the previously known shocks and treated with further volume expansion. Group 2 were 10 patients from the series who were correctly diagnosed as volumetric overload shock and treated with hypertonic sodium therapy (HST). Group 3 were 10 patients who were seen in the prospective study and subdivided into 2 groups; Group 3.1 of 5 patients treated with HST and Group 3.2 of 5 patients who were treated with guarded volume expansion using isotonic saline.(Reproduced with the author permission from open access journal reference 25)

VOS2has no markedserological markers but has all manifestations of MVOD/Fsyndrome recognized as ARDS. This is the type seen on treating recognized shock of poly-trauma with overzealous infusions of crystalloids, colloids, colloid substitutes 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 ARDS [20] became latter known as MVOD/F and is currently named systemic inflammatory response syndrome (SIRS) [26]. The volumetric overload is evident by oedema of the torso seen in VOS2 and MVOD/F or ARDS, detected by the increase in body weight. The VO or fluid 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 notincriminated, why?

Fluid therapy has proved life-saving therapy for poly-trauma victims during the 2nd World War and in clinical practice later. This has firmly implanted in the minds of generations of physicians that every hypotension is synonymous with hypovolaemia. Addingto the difficulties, VO/T concept as a cause of shock is paradoxical tothe received concept of treating all shocks with vascular expansion, indiscriminately [7,26]. 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 [12,13,27,28]. 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 5 litres (L) and maximum capacitance of the adult cardiovascular system is 7L. Thus although 3.5L is about the normal daily fluid requirements, infusing such volume in <1 h induces a typical VOS1 of the types mentioned above. The issues involved on discussing all shocks and in poly-trauma 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 by loss or gain 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 andcarbon 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 8decades 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 [27]. 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 (G) tubes, akin to capillary, and incorporated in a circulatory system explored this issue [13,27]. 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 pressure gradient on the wall of the G tube turns positive causing filtration over the distal part of the tube, maximum near the exit' The distal pressure, akin to venous pressure, is enhances filtration through the distal pores near exit [13,27]. The hydrodynamic of the porous orifice (G) tube is summarised in (Figure 3).

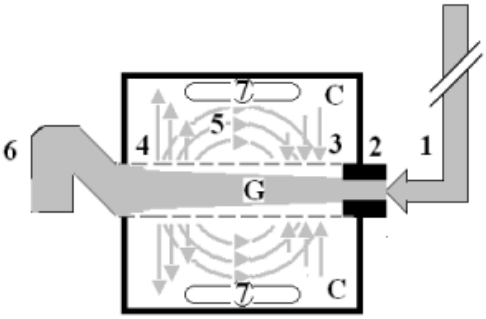

Figure 3: Diagram of the porous orifice (G) tube enclosed in chamber (C) based on several photographs demonstrating the magnetic field-like G-C circulation phenomenon. The proximal inflow (arterial) pressure (1) pushes fluid through the orifice (2) creating fluid jet in the lumen of the G tube. The fluid jet creates negative side pressure gradient causing suction maximal over the proximal half of the G tube near the inlet (3) that sucks fluid into lumen. The side pressure gradient turns positive pushing fluid out of lumen over the distal half maximally near the outlet (4). Thus the fluid around G tube inside C moves in magnetic field-like fluid circulation (5) taking an opposite direction to lumen flow of G. tube. The inflow (arterial) pressure (1) and orifice (2) induce the negative side pressure energy creating the dynamic G-C circulation phenomenon that is rapid, autonomous and efficient in moving fluid out from the G tube lumen at (4), irrigating C at (5), then sucking it back again at (3), maintaining net negative energy pressure (7) inside C. The distal outflow (venous) pressure (6) enhances outflow at (4) and its elevation may turn the negative energy pressure (7) inside C into positive, increasing volume and pressure inside C chamber.(Reproduced with the author permission fromBHC open access journal reference 27)

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 C, 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 C moves in the opposite direction to G tube flow. The orifice, akin to pre-capillary sphincter, plays vital role in the dynamic and speed of this circulation [13,27]. A drop in proximal pressure, elevation of the distal pressure and too narrow or too wide orifice slows down the G tube to chamber C circulation.

Over expansion of the circulatory system markedly slows down the dynamic magnetic field-like G-C 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 VOS2and 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 G tubes demonstrates and explains it on inducing similar changes in proximal and distal pressures, and orifice diameter. Understanding this phenomenon may help to redefine shock and identify the new VOS [28,29] by rectifying the physiological errors and misconceptions on intravenous fluid therapy [30] that resolve the enigma of MVOD/F or ARDS. No clinical study will produce useful results before the mentioned issues are reconsidered and stratified.

References

1. Pepe PE. Editorial: Shock in polytrauma. Br Med J. 2003;327:1119-1120
2. Ghanem AN. Hypoalbuminaemic hyponatraemia: a new syndrome. Br Med. Jour. 1985; 29:1502
3. Ghanem AN. Wojtulewski JA, Penney MD. Dangers in treating hyponatraemia. Br Med J. 1987; 294: 837.
4. Ghanem AN, Ward JP. Fluid absorption during urological surgery. Br J Uro, 1988; 61: 168-9.
5. Ghanem AN. Hyponatraemia and hypo-osmolality. Lancet 1988; ii:572.
6. Ghanem AN. Frequency and causes of fluid absorption: a comparisonof the three techniques for resection of the prostate under continuous pressure monitoring. BJU Int 1999; 84: 891-2.
7. Ghanem AN. Monitoring the critically ill patient. J R Coll Surg Edinb 2000; 45 (2): 138-9.
8. Ghanem AN. Preventing renal failure in the critically ill: identifying the problem and finding solution. bmj.com/cgi/eletters/322/7300/1437#15409, 2 Jul 2001 [Full text]
9. Ghanem AN. Hyponatraemia during and after TUR prostatectomy. Saudi Med J 2002; 23 (4): 477-9
10. Ghanem AN. Dysnatraemia or hyponatreamia: highlighting the volumetric overload/time (VO/T) concept. BMJ 2003: Rapid response Feb 17, 2003
11. Ghanem AN. The Transurethral Prostatectomy (TURP) Syndrome: An Investigation of the Osmotic and Metabolic sequelae of Volumetric Overload (VO). MD Thesis. Institute of Urology and Nephrology, Mansoura University, Mansoura, Egypt. November 6, 1988.
12. Ghanem AN, Ward JP. Osmotic and metabolic sequelae of volumetric overload in relation to the TURP syndrome. Br J Uro 1990; 66: 71-78
13. Ghanem AN. Magnetic field-like fluid circulation of a porous orifice tube and relevance to the capillary-interstitial fluid circulation: Preliminary report. Medical Hypotheses 2001 Mar; 56 (3): 325-334.
14. Danowski TS, Winkler AW, Elkington JR. The treatment of shock due to salt depression; comparison of isotonic, of hypertonic saline and of isotonic glucose solutions. J. Clin. Invest. 1946; 25: 130.
15. Harrison III RH, Boren JS, Robinson JR. Dilutional hyponatraemic shock: another concept of the transurethral prostatic reaction. J Uro. 1956; 75 (1): 95-110.
16. Spencer Hoyt H, Goebel J L, Lee HI, Schoenbrod J. Types of shock reaction during transurethral resection and relation to acute renal failure. J Uro 1958; 79: 500-7.
17. Bird D, Slade N, Feneley RCL. Intravascular complication of transurethral prostatectomy. Br J Uro 1982; 54: 564-5.
18. Dandonna P, Fonseca V and Baron. Hypoalbuminaemic hyponatraemia: a new syndrome? Br Med Jour 1985; 291: 1253-5.
19. Watters DAK, Chamroonkul MA, Eastwood MA, et al. Changes in liver function associated with parentral nutrition. J Roy Coll Surg Edin 1984; 29: 339-44
20. Ashbaugh DG, Bigelow DB, Petty TL, Levine BE. Acute respiratory distress in adults. Lancet 1967; ii: 319-23.
21. Hahn RG. Irrigating fluids in endoscopic surgery. Br J Uro; 1997: 79: 669-80
22. Arieff AI. Ayus JC. Endometrial ablation complicated by fatal hyponatraemic encephalopathy. JAMA 1993; 270: 1230-2
23. Arieff AI. Hyponatraemia, convulsion, respiratory arrest and permanent brain damage after elective surgery in healthy women. N Engl J Med 1986; 314 (24): 1529-34.
24. Arieff AI. Management of hyponatraemia. Br Med Jour 1993; 307: 305
25. Ghanem KA. and Ghanem AN. Volumetric overload shocks in the patho-etiology of the transurethral resection prostatectomy syndrome and acute dilution hyponatraemia: The clinical evidence based on 23 case series. Basic Research Journal of Medicine and Clinical Sciences ISSN 2315-6864 Vol. 6(4) pp. xx-xx April 2017
26. Webster NR. Monitoring the critically ill patient. J R Coll Surg Edin 1999; 44 (6): 386-93
27. Ghanem KA. and Ghanem AN. 2017. The proof and reasons that Starling's law for the capillary-interstitial fluid transfer is wrong,

advancing the hydrodynamics of a porous orifice (G) tube as the real mechanism. Blood, Heart and *Circ,* Volume 1(1): 1-7 doi:10.15761/BHC.1000102 Available online

28. Ghanem, A.N. and Ghanem, S.A. Volumetric Overload Shocks: Why Is Starling's Law for Capillary Interstitial Fluid Transfer Wrong? The Hydrodynamics of a Porous Orifice Tube as Alternative. Surgical Science, 2016; 7: 245-249.
29. Pindoria N, Ghanem SA, Ghanem KA and Ghanem AN, (2017) Volumetric overload shocks in the patho-etiology of the transurethral resection prostatectomy syndrome and acute dilution hyponatraemia. *Integr Mol Med,* 2017 doi: 10.15761/IMM.1000279.
30. Ghanem AN. The Adult Respiratory Distress Syndrome: Volumetric Overload Shocks in Patho-Etiology, Correcting Errors and Misconceptions on Fluid Therapy, Vascular and Capillary Physiology. Surg Med Open Acc J. 2(2). SMOAJ.000534.2018. DOI: 10.31031/SMOAJ.2018.02.000534