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Review Article

Impact of COVID-19 on Surgery and Anaesthesia: Fighting Another Unique War on ARDS Relevant to Covid-19 Pandemic

Ahmed N Ghanem*

Retired Consultant Urologist Surgeon and Independent Investigator, El-Mansoura University, Faculty of Medicine, Mansoura, Egypt

***Corresponding Author:** Ahmed N Ghanem, Retired Consultant Urologist Surgeon and Independent Investigator, El-Mansoura University, Faculty of Medicine, Mansoura, Egypt.

Received: November 26, 2020; **Published:** January 30, 2021

Abstract

This article reports the on the impact of Covid-19 pandemic on a retired Urologist Surgeon who was forced into home isolation while already busy fighting a parallel relevant war on the acute respiratory distress syndrome (ARDS) of another because that is important, unrecognized, and underestimated. The corona virus kills its victims by causing ARDS. I have been fighting my solo unique self-financed war against ARDS of a different cause that is no less common, important, or serious than ARDS of Covid-19 and sepsis. The home confinement self-isolation imposed by Covid-19 has been invested intensively and usefully in finalizing may unique solo self-financed war against another type ARDS. Sepsis and Covid-19 are well-recognized causes of ARDS. However, there is another important cause for ARDS that remains unrecognized and under-estimated. Once revealed and recognized it becomes obvious and easily provable. It has not been easy to recognize because it complicates other recognized shocks during its intravenous fluid therapy of shock and acutely ill patients.

Volumetric overload (VO) inducing shock reported as VO shock (VOS) or volume kinetic shocks is iatrogenic complication of fluid therapy that cause ARDS. This type of ARDS is usually attributed to sepsis, but recent evidence demonstrates that sepsis in most cases is most probably as innocent as the wolf in Josef's story. Although my war against ARDS induced by VOS started 40 years ago, it has been emphasized over the last 4 years and finalized, concluded, and triumphantly won in 2021. My war against ARDS of VOS has been won but this can only be triumphantly declared when the scientific and medical world acknowledge my related discoveries in physics, physiology, biochemistry, and medicine.

Keywords: *COVID-19; Volumetric Overload (VO); VO Shock (VOS); Acute Respiratory Distress Syndrome (ARDS)*

Introduction

The COVID-19 pandemic has plagued the whole world for nearly a year now. All governments, scientists and physicians of the world have been deeply engaged in fighting its war against humanity at an extremely high cost of human lives and finances.

The corona virus kills its victims by causing the acute respiratory distress syndrome (ARDS). I have been fighting my solo unique self-financed war against ARDS of a different cause that is no less common, important, or serious than ARDS of Covid-19 and sepsis. The home

confinement self-isolation imposed by Covid-19 has been invested intensively and usefully in finalizing may unique solo self-financed war against another type ARDS [1,2]. Sepsis and Covid-19 are well-recognised causes of ARDS. However, there is another important cause for ARDS that remains unrecognised and under-estimated. Once revealed and recognized it becomes obvious and easily provable. It has not been easy to recognize because it complicates other recognized shocks during its intravenous fluid therapy. Volumetric overload (VO) inducing shock reported as VO shock (VOS) [3-6] or volume kinetic shocks [7,8] is iatrogenic complication of fluid therapy in hospitals that cause ARDS. This type of ARDS is usually attributed to sepsis, but recent evidence demonstrates that sepsis in most cases is most probably as innocent as the wolf in Josef's story.

Although my war against ARDS induced by VOS had started back in 1981 it has been emphasised over the last 4 years and finalized and concluded in 2020 [1,2]. My war against ARDS of VOS has been won but this can only be triumphantly declared when the scientific and medical world acknowledges my related discoveries in physics, physiology, biochemistry, and medicine. These discoveries are listed and summarised here showing its physiological relevance and clinical importance:

Physics discoveries

1. The hydrodynamics of the porous orifice (G) tube [9-11].
2. The Tree Branching Law (TBL) [12,13].

Physiological discoveries

1. Proving Starling's law for the capillary-interstitial (ISF) fluid wrong and providing the correct replacement of the magnetic field like fluid hydrodynamics of the G tube [9-11].
2. The TBL Corrects two misconceptions on capillary physiology [12,13], namely:
   a. The cross-section areas of all the capillaries is larger than the aorta
   b. The red blood cells (RBCs) speed in a capillary is thought "very slow" to allow for the slow perfusion of the capillary-ISF transfer as based on Starling's forces.

Biochemical discoveries

1. Resolving the puzzle of acute dilutional hyponatraemia identifying its path-aetiology and finding a successful curative lifesaving therapy for it: The Hypertonic Sodium Therapy (HST) of 5%NaCl and/or 8.4%NaCo3 [14-18].
2. The transurethral resection of the prostate (TURP) syndrome is the most famous example of acute dilution hyponatraemia that represents VOS 1 induced by sodium-free fluid.
3. Revealing the effects of volume kinetics on the cardiovascular system pressure discovering VOS 1 and VOS 2 [6,7].

Medical discoveries

1. Discovering two new types of cardiovascular shocks: the volume kinetic shocks [7] or the volumetric overload shocks (VOS) [6] of type one (VOS 1) induced by sodium-free fluid and type two (VOS 2) induced by sodium-based fluid retention [3-7].
2. Resolving the puzzle of the acute respiratory distress syndrome (ARDS) by identifying its exact patho-aetiology being caused by VOS and a successful therapy of HST [1,2].
3. Resolving the puzzle of the TURP syndrome [19] also reported in urology as hyponatraemic shock [20] discovering its link with ARDS and finding the successful lifesaving therapy that also cures acute dilution hyponatraemia [20,21].
4. In discovering the above, the bridge connecting the science of physics, physiology, biochemistry, and medicine was constructed [2]

5. On a totally different subject, the patho aetiology of the loin pain haematuria syndrome (LPHS) was discovered revealing its link with symptomatic nephroptosis (SN), and 100% curative therapy surgery was devised [22].
6. A new surgical procedure for the therapy of cancer bladder with orthotopic bladder replacement was reported [23].

Summary of the new scientific discoveries

Despite multiple and powerful reporting in the literature on my multiple and important scientific discoveries the whole medical world is not responding. It seems to be in a deep coma. Even the top Medical, surgical, and scientific journals including *Nature, Nature Medicine, Science, Lancet, British Medical Journal, New England Journal of Medicine. Journal of The American Medical Association, The Surgeon- The Journal of the Royal College of Surgeons of Edinburgh, Physiology and Urology* journals have repeatedly done serious mistakes rejecting the many articles I sent to them. They persistently sustained obstinate refusal stand against my articles. They may ignore my person, but they cannot wrong any of my new discoveries. Here is a summary of my new discoveries to show you how wrong they all are.

My scientific discoveries are many and most important made over the last 32 years of my career life spent in investigating and reporting these articles. The articles recognize 2 new types of shocks and its treatment, proves that Starling's law for the capillary interstitial fluid transfer is wrong and provides an alternative mechanism: The hydrodynamics of a porous orifice (G) Tube. These discoveries resolve the puzzles of 3 syndromes discovering and precising its patho-aetiology and new successful treatments: namely the TURP syndrome and acute dilution hyponatraemia (HN), ARDS, and the loin pain haematuria syndrome (LPHS). Not only the exact patho-aetiologies of these syndromes were discovered but also successful treatments for it were found. The two new types of vascular shocks are volume kinetic shocks or VOS defined here.

Massive fluid infusions in a short time induce VOS of two types: Type one (VOS 1) and Type two (VOS 2). VOS 1 is induced by sodium-free fluid of 3.5-5 litres in one hour known as the TURP syndrome [19] or hyponatraemic shock [20]. Before we rejuvenated hypertonic sodium as effective curative therapy for hyponatraemia, it caused serious morbidity and was usually lethal in most cases [21]. Similarly, although ARDS was first reported in 1967 it has remained without any effective therapy till today [24]. VOS 2 may complicate VOS 1 or is induced by massive infusion of sodium-based fluids. VOS2 also complicates fluid therapy in critically ill and presents with ARDS. Volumetric gain of 12 - 14 litres of sodium-based fluids was reported in first ARDS article [24] but overlooked in all other article except mine and a few recently reported articles.

Two clinical studies to understand the TURP syndrome and to recognize VOS were conducted in the eighties. A prospective study on 100 consecutive TURP patients of whom ten suffered TURP syndrome [19]. Volumetric overload was the only significant factor in causing the condition (Table 1 and figure 1). The second study was case series of 23 case cases s of the TURP syndrome manifesting as VOS1 [5]. Patient5s had Volumetric overload quantity and type is shown in figure 2. The clinical picture of all cases of ARDS are similar in all types though one system may predominate (Table 2). Of the case series, 3 patients died and remaining 20 patients were correctly diagnosed as VOS 1 and appropriately treated mostly with hypertonic sodium therapy (HST). Each patient passed 4 - 5 litres of urine followed by recovery from shock and coma. This treatment was successful in curing all patients bringing them back from dead.

A physics study of the hydrodynamics of the porous orifice (G) tube comparing it to that of Poiseuille's tube was done [9,10]. Measurements of pressures at various parts of a circulatory system incorporating the G tube in a chamber (c) to mimic the capillary interstitial (ISF) fluid compartment were done. The effect of changing the proximal (arterial), the distal (venous) pressures and the diameter of the inlet on side pressure of the G tube and chamber pressure as well as the dynamic magnetic field like fluid circulation around the G tube was evaluated. The dynamic magnetic field like fluid circulation around the G tube and surrounding it in C chamber (Figure 3) provides adequate replacement for Starling's law. The physiological equivalent of this physics study was done on the hind limbs of sheep [11]. It

Parameter	Value	Std. Err	Std. Value	T Value	P
Intercept			0.773		
Fluid Gain (l)	0.847	0.228	1.044	3.721	0.0007
Osmolality	0.033	00.014	-0.375	2.42	0.0212
Na+ (C_B)	0.095	0.049	0.616	1.95	0.0597
Alb (C_B)	0.062	0.087	0.239	0.713	0.4809
Hb (C_B)	-0.282	0.246	-0.368	1.149	0.2587
Glycine (C_B)	-4.973E-5	5.975E-5	-0.242	0.832	0.4112

Table 1: S*hows 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 hypo-osmolality are the only significant factors. The significance of volumetric overload is remarkable.*

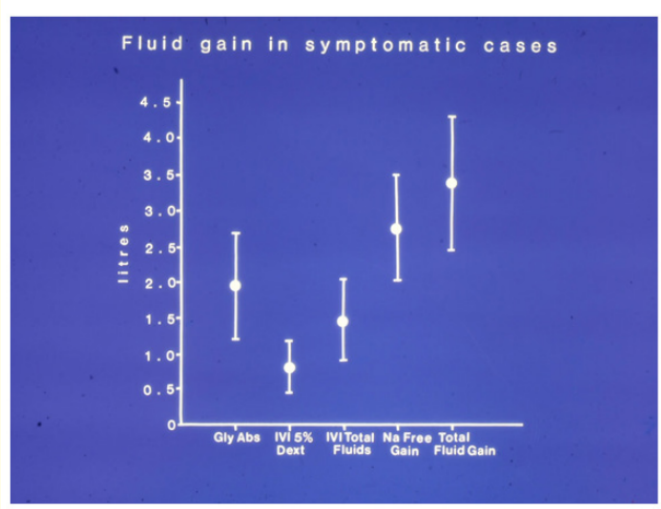

Figure 1: *Shows the means and standard deviations of volumetric overload in 10 symptomatic patients presenting with shock and hyponatraemia among 100 consecutive patients during a prospective study on transurethral resection of the prostate. The fluids were of Glycine absorbed (Gly abs), intravenously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

demonstrated that both saline and plasma induces oedema when run through the vein not the artery, and the arterial pressure causes suction not filtration due to effect of pre-capillary sphincter.

Starling's hypothesis was based on Poiseuille work on strait uniform brass tubes [25]. Eight decades latter evidence demonstrated that the capillary is a porous narrow orifice (G) tube as it has a pre-capillary sphincter [26] and pores that allow the passage of plasma proteins [27]. As the capillary pores allow the passage of plasma molecules, nullifying the osmotic pressure of plasma proteins, a call for reconsideration of Starling's hypothesis was previously made but there was no alternative then. The replacement came to light when the hydrodynamics of the G tube were discovered and reported preliminarily in 2001 at Medical hypothesis journal [9].

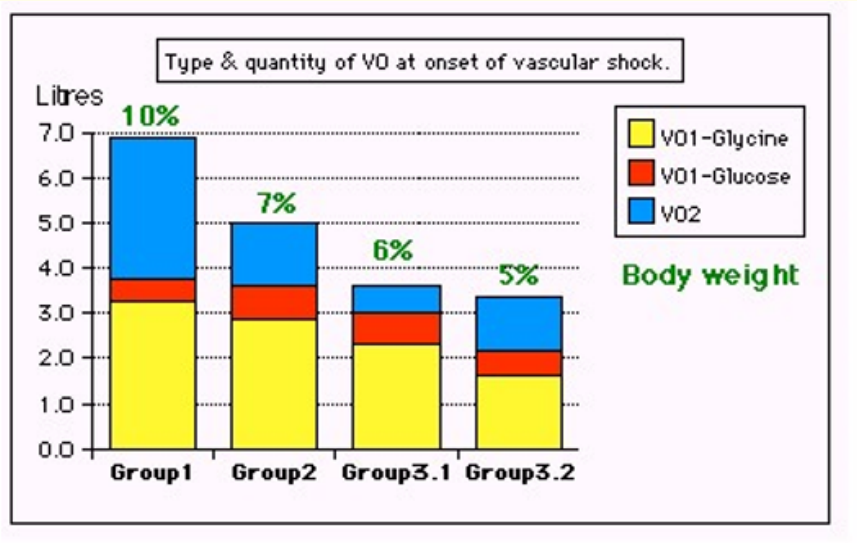

Figure 2: *Shows volumetric overload (VO) quantity (in litres and as percent of body weight) and types of fluids. Group 1 was the 3 patients who died in the case series as they were misdiagnosed as one of the previously known shocks and treated with further volume expansion. Group 2 were 10 patients 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.*

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

Table 2: *Shows the manifestations of VOS 1 of the TURP syndrome for comparison with ARDS manifestations induced by VOS2.*

Table abbreviations

SBB[1]: Sudden Bilateral Blindness; COC[2]: Clouding of Consciousness; PMBCI[3]: Paralysis Mimicking Bizarre Cerebral Infarctions, but is recoverable on instant use of HST of 5%NaCl and/or $NaCo_3$, 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[$]: Manifests Later, on ICU; AKI[9]: Acute Kidney Injury; DGR[10]: Delayed Gut Recovery; CV: Shock Cardiovascular shock of VOS reported here as VOS 1 and VOS2; Annuria[8]: That is unresponsive to diuretics but responds to HST of 5%Ncl and/or 8.4%$NaCo_3$; AKI[8]: Acute kidney injury; Also occurs the excessive bleeding at the surgical site and Leucocytosis occurred in the absence of sepsis and septic shock.*

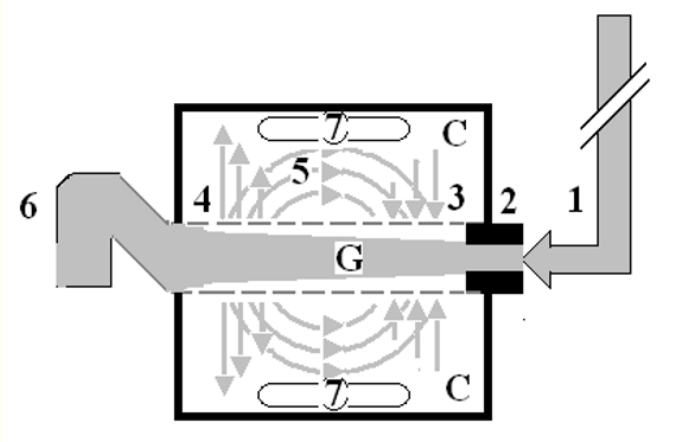

Figure 3: *Shows a diagrammatic representation of the hydrodynamic of G tube based on G tube and surrounding chamber C. This 37-years old diagrammatic representation of the hydrodynamic of G tube in chamber C is based on several photographs. The G tube is the plastic tube with narrow inlet and pores in its wall built on a scale to capillary ultra-structure of precapillary sphincter and wide inter cellular cleft pores, and 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 centre 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. It was also noted that fine tea leaves exit the distal pores in small amount maintaining a higher concentration in the circulatory system than that in the C chamber- akin to plasma proteins.*

The hydrodynamics of the G tube [9,10] (Figure 3) demonstrated that the proximal (arterial) pressure induces a negative side pressure gradient on the wall of the G tube causing suction most prominent over the proximal half and turns into positive pressure over the distal half. Incorporating the G tube in a chamber (C), representing the ISF space surrounding a capillary, demonstrated a rapid dynamic magnetic field-like fluid circulation between C and G tube lumen. Incorporating the G tube and C in a circulatory model driven by electric pump induced proximal pressure akin to arterial pressure causing suction from C into the lumen of G tube. This proves that the arterial pressure causes suction not filtration at the capillary interstitial fluid circulation, and hence Starling's law is wrong on both forces and

equations. The hydrodynamics of the G tube provide adequate correct replacement for Starling's law [9-11]. This illustrates how 2 new types of vascular shocks [6,7] and a replacement of Starling's law were discovered that have resolved the puzzles of 3 clinical syndromes of TURP, HN and ARDS.

The physiological relevance of the hydrodynamic of the G tube to normal capillary physiology

The provided evidence demonstrates that the hydrodynamic of the G tube is totally different from Poiseuille's tube. This is relevant to the physiological function of capillary regarding the capillary-ISF transfer. When Starling proposed his hypothesis on the formation of oedema in 1886 and 1896 [25], he assumed that the capillary works as Poiseuille's tube of uniform diameter and its hydrostatic pressure induced by the high arterial pressure is responsible for filtration of fluid higher over the proximal part of the capillary near the inlet. He wrongly thought the capillary works as Poiseuille's tube. It was discovered > 80 years later in 1967 that the capillary has a narrow orifice; the precapillary sphincter [26]. He also wrongly assumed that absorption of fluid is induced by the oncotic pressure of plasma proteins as he thought that the capillary wall is impermeable to albumin. It was also discovered in 1967 that the capillary has wide pores of intercellular clefts that allow molecules larger than plasma proteins to pass through [27]- hence nullify oncotic pressure *in vivo*. Starling's hypothesis was made into a law later. In fairness to Professor Starling, who was a great physiologist, he never wrote any equations nor proposed a law. Here we demonstrate that Starling's law is wrong on both of its forces, and the equations must be also wrong. This affirms the principle of what is built on wrongdoing must also be wrong.

Both physics [9,10] and physiological [11] evidence demonstrate that the capillary works as G tube in which the arterial pressure induce negative side pressure gradient that causes absorption by suction not filtration that is maximum near the inlet. This is based on the hydrodynamic of the G tube summarised here. It has also been demonstrated that the oncotic pressure does not exist *in vivo* as the capillary has wide intercellular cleft pores that allow molecules larger than plasma proteins to pass through it [27]. Starling's law is thus wrong on both of its forces and the equations must also be wrong. It is time to say farewell: "goodbye Starling's law, hello G tube" [28]. The revised Starling's principle [29] has also proved misnomer and wrong [30].

The pathological importance of hydrodynamic of the G tube when the capillary acts as Poiseuille's tube inducing VOS and oedema of ARDS

Starling's law being wrong has resulted in many errors and misconceptions on fluid therapy [31]. These errors mislead physicians [32] into giving too much fluid during the resuscitation of shock, the acutely ill patient and prolonged major surgery. The resulting volumetric overload (VO) induce VO shocks (VOS) [3-7] which cause ARDS [1,2]. VOS are two types depending on the type of fluid inducing it: Sodium-free fluid induces VOS 1 and sodium-based fluid induces VOS 2. Examples of VOS 1 is the transurethral resection of the prostate (TURP) syndrome [19] known in urology also as hyponatraemic shock [20]. These are induced by massive absorption of 3.5 - 5 litres in one hour of 1.5% Glycine used as irrigating fluid for the TURP surgery and/or excessive infusion of 5% Glucose. This VOS 1 is always mistaken for the known haemorrhagic or septicaemic shock and is wrongly treated with further volume expansion using crystalloids and/or colloids that transfer it into VOS 2. Before we rejuvenated hypertonic sodium therapy as successful curative lifesaving therapy for acute dilution HN, it caused coma, convulsion, paralysis, shock, and respiratory distress and caused death in most cases [21]. The later VOS 2 may also complicate fluid therapy of recognized shocks during therapy using crystalloids and/or colloids and blood. This is in turn cause ARDS [1,2] or the multiple organ dysfunction syndromes (MODS). Both VO and sepsis adversely affect the hydrodynamic of the capillary working as G tube transferring it into Poiseuille's tube causing both shock (VOS) and oedema of ISF space particularly in the subcutaneous tissue and of vital organs that characterize and cause ARDS or MODS [1,2].

The recent supporting evidence on VOS causing ARDS

Professor Hahn studied VK in healthy volunteers and patients [33,34]. He reported in conclusion 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 litres is administered. Infusion of 6 - 7 litres 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 haemodilution 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 study on adults' trauma patients by Jones., *et al*. (2016) [35], and one paediatrics study by Coons., *et al*. (2018) [36] and a remarkable review article by Schrier reported in 2010 [37] 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*. [35] 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*. [36] 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 48h 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".

The huge prospective multicentre trials [38,39] also documented massive volumetric overload (VO) in surviving ARDS patients of 3 - 10 litres of retained fluid volume but have neither recognized VOS nor incriminated VO in the patho-aetiology of ARDS. They also did not recognize the high association of VO with the mortality which was estimated at 60 or 90 days not at the immediate period of 24 - 48 hours after admission as demonstrated by the above reports [38,39]. Excellent example of these huge multicentre trials is that study reported by Rowan., *et al*. in 2017 [38].

In the results section, Rowan., *et al*. reported: "Each study day the liberal-strategy group received more fluid than the conservative-strategy group and on days 1 through 4 had a lower urinary output, resulting in a higher cumulative fluid balance (Table 2). During the study, the seven-day cumulative fluid balance was -136 ± 491 ml in the conservative-strategy group, as compared with 6992 ± 502 ml in the liberal-strategy group ($P < 0.001$) (Figure 1 of the supplementary material). For patients who were in shock at baseline, the cumulative seven-day fluid balance was 2904 ± 1008 ml in the conservative-strategy group and 10,138 ± 922 ml in the liberal-strategy group ($P < 0.001$). For patients who were not in shock at baseline, the cumulative fluid balance was -1576 ± 519 ml in the conservative-strategy group and 5287 ± 576 ml in the liberal-strategy group ($P < 0.001$)".

Therapy of VOS and ARDS

Prevention

Being iatrogenic complications of fluid therapy, both VOS and ARDS are preventable.

To prevent VOS and ARDS a limit to the maximum amount of fluid used during shock resuscitation or major surgery must be agreed upon (New guidelines are required).

Surgical care providers must exercise judicious use of crystalloid fluid administration in the trauma bay, ICU, and floor.

Replace the loss in haemorrhagic hypovolemic shocks but do not overdo it.

If hypotension develops despite volume replacement later during ICU stay, inotropic drugs, hydrocortisone 200 mg and hypertonic sodium therapy (HST) should be used-please, see later. The latter restores the pre-capillary sphincter tone (peripheral resistance) so that the capillary works as normal G tube again, but No isotonic crystalloids or colloids over-infusions is required.

To learn the new correct science, one must unlearn the old incorrect habits.

The following practices should be abandoned:

A. Bolus fluid therapy in surgical patients
B. Abandon the aggressive current liberal regimen of Early Goal-Directed Therapy (EGDT) in treating shocked and septic patients [40]. Multiple huge multicentre trials have proved it to be the wrong practice [38,39].
C. Please refrain from persisting to elevate CVP to levels above 12 and up to 18-22 cm saline in shock management. This is a major cause for inducing VOS and ARDS during shock resuscitation, particularly septic shock [41].

Therapeutic

Hypertonic sodium therapy (HST) of 5%NaCl and/or 8.4%$NaCo_3$ has truly proved lifesaving therapy for the TURP syndrome and acute dilution HN as well as secondary VOS 2 that complicates fluid therapy of VOS 1 causing ARDS. It works by inducing massive diuresis being a potent suppressor of antidiuretic hormone. It may also work on the pre-sphincter capillary restoring its tone.

My experience in using it for treating established ARDS with sepsis and primary VOS 2 that causes ARDS is not tested. However, evidence on HST suggests it will prove successful if given early, promptly and adequately to ARDS patients while refraining from any further isotonic crystalloid or colloid fluid infusions using saline, HES and/or plasma therapy- just give the normal daily fluid requirement and no more. After giving HST over one hour using the CVP catheter already inserted, the patient recovers from AKI and produces through a urinary catheter massive amount of urine of 4 - 5 litres as you watch. This urine output should not be replaced. Just observe the patient recovering from his AKI, coma and ARDS and asks for a drink. This is done in addition to the cardiovascular, respiratory, and renal support on ICU. Patients with AKI on dialysis, the treating nephrologist should aim at and set the machine for inducing negative fluid balance.

The HST of 5%NaCl and/or 8.4%NaCo3 is given in 200 ml doses over 10 minutes and repeated. I did not have to use more than 1000 ml during the successful treatment of 16 ARDS patients. Any other hypertonic sodium concentration is not recommended. A dose of intravenous diuretic may be given but it does not work in a double or triple the normal dose. A dose of 200 mg of hydrocortisone is most useful. Antibiotic prophylactic therapy is given in appropriate and adequate doses to prevent sepsis and septic shock. No further fluid infusions of any kind crystalloids, colloids and blood is given. The urinary loss should not be replaced as this defeats the objective of treatment.

A suggested recommended future trial on ARDS of Covid-19 and sepsis

I would recommend a small pilot prospective controlled randomized comparative cohort study on 100 patients as a start to try HST in established ARDS cases of both sepsis and Covid-19 that would be something to look forward to reading a report on it, hopefully soon. No multicenter trial or high expenses is needed for that. Not much time is required either. Any centre or hospital receiving Covid-19 cases can do this study. If you cannot do it on a hundred patients, you probably cannot (as Mr. JP Ward put it to me before the start of our prospective study [19]).

The 100 Covid-19 patients are divided randomly into two groups of 50 patients each at the time of presentation of ARDS. The 1st group receive the standard supportive treatment for ARDS. The 2nd group receive the same standard treatment plus hypertonic sodium therapy (HST) of 5%NaCl and/or 8.4%NaCo3 of a maximum of one litre. The Study starts on hospital admission and ends on the day of hospital discharge or death. It should record precise daily volumetric balance in hospital and the patient's body weight from hospital admission to discharge or death. The results and statistics compare the two groups with regards to morbidity and mortality. All patients should receive the standard clinical monitoring of the systems shown in (Table 2) and the standard laboratory monitoring.

I can assure the investigators that no harm will come to patients. It is a guaranteed win bit; you may win but you do not lose anything. In the worst-case scenario, the patient may not respond because of chronicity of ARDS or after sepsis complicates ARDS and gets the capillary damage established. As the author of all self-referenced articles here, published in open access journals, and as copyright holder I give open permission to any interested investigator to use any of my articles as template, particularly recommended article [19]- the appropriate permission from the editors of BJUI and authors are given. The scientific basis of this study is detailed in this book [42]. If it proved successful in a trial on ARDS of Covid-19, it shall demonstrate that I have been sharing in the war against Covid-19 right at the front line in my own unique, remarkable, and exclusive way.

Conclusion

I have been sharing in the war against Covid-19 right at the front line in my own unique, remarkable, and exclusive way. I have fought my war against ARDS of another unrecognized and underestimated cause of volumetric overload occurring during fluid therapy of shock inducing volumetric overload shocks (VOS) and causing ARDS. During this war I have made many discoveries in physics, physiology, biochemistry and medicine. The G Tube has totally different hydrodynamic from Poiseuille's tube. The G tube has negative side pressure gradient that is maximum negative near the inlet and turns gradually positive to become maximum near the exit. Thus, in the G tube suction or absorption of fluid occur through side holes near the inlet while filtration occur through holes near the exit. This creates autonomous rapid dynamic magnetic field-like fluid circulation in a surrounding chamber (C) between fluid around the G tube and fluid inside its lumen. The negative SP of G tube creates net negative pressure in chamber (C) with a direction of flow opposite to that in the G tube.

Both physics and physiological evidence demonstrate that the capillary works as G tube in which the arterial pressure induce negative side pressure gradient that causes absorption by suction not filtration. Starling's law is thus proved wrong on both forces and equation.

Starling's law being wrong has resulted in many errors and misconceptions on fluid therapy that mislead physicians into giving too much fluid during the resuscitation of shock. The resulting volumetric overload (VO) induce VO shocks (VOS): Sodium-free fluid induce VOS 1 and sodium-based fluid induce VOS 2. Examples of VOS 1 is the TURP syndrome known in urology also as hyponatraemic shock. This VOS 1 is always mistaken for a known shock and is wrongly treated with further volume expansion that transfer VOS 1 into VOS 2. The later VOS 2 may also complicate fluid therapy of recognized shocks. This is in turn cause ARDS or MODS. Both VO and sepsis adversely affect the hydrodynamic of the capillary working as G tube transferring it into Poiseuille's tube inducing both shock (VOS) and oedema of ISF space and of vital organs that cause ARDS or MODS.

The TBL is a fundamental law of nature that govern the ramifications of all trees of green and red, particularly the Aorta-arterial tree. It corrects two important misconceptions on the capillary physiology: The cross-section area of the capillaries is greater than that of the aortas, and the RBCs speed in a capillary is "very slow" to allow for the slow perfusion based on the wrong Starling's forces. This evidence sums up to demonstrate that the capillary-ISF transfer occurs according to a precise fast circulation of the magnetic field like fluid not the slow perfusion. That provides adequately for the demands of cells at rest and increased demand during strenuous physical activity.

Final notes on Covid-19 and ARDS

Being retired for 10 years now and having no access to clinical practice and research facilities, I could not directly contribute to fighting the war and being at the frontline on Covid-19 pandemic. The home confinement and complete isolation from the outside world imposed on me by Covid-19 has been most usefully invested in intensifying my war on ARDS of volumetric overload and finalizing my reports on VOS causing ARDS [1,2].

Covid-19 pandemic is a temporary transient disaster that will eventually goes into oblivion. It will be placed dormant and harmless in the history of similarly serious infectious diseases that have been eradicated by active immunization. Sepsis as a cause of ARDS can be easily eradicated by the effective powerful and adequate antibiotics we have today. Then we are left with ARDS induced by volumetric overload. This requires eliminating the culprit wrong Starling's law. Being wrong has dictated many errors and misconceptions on fluid therapy that mislead physicians into giving too much fluid during the resuscitation of shock, acutely ill patients, and prolonged major surgery. This induced VOS that cause ARDS. This require devising new guidelines on fluid therapy, making every practicing physicians aware of the scientific discoveries mentioned above and letting it shine and prevail. This article has been referenced in 4 relevant books that provide further evidence on the claims and theory reported here that are available from amazon.com and the publishers [42-45].

"A suggested Protocol for the above desperately needed study

Objectives:

1. To validate the role of Volumetric Overload (VO) in the patho aetiology of VO Shocks (VOS) and ARDS.
2. To evaluate and validate the effectiveness of hypertonic sodium therapy (HST) for treating VOS, ARDS and Covid-19

Patients and Methods:

1. Only 100 patients are studied (Initially for quick results) during their hospital stay from admission to discharge
2. Baseline evaluation of all patients on hospital admission that include clinical, laboratory and body weight (gravimetric measurement).
3. Further evaluations are done on daily basis and when indicated during the hospital and ICU stay.
4. All patients should receive the standard current therapy for shocks of Trauma, haemorrhage, Sepsis, ARDS and Covid-19.
5. All patients have precise documentation of fluid therapy (Input): type, and volume and time of infusion (Volumetric measurement). Fluid output is recorded too.
6. Only half of the patients are randomized to receive HST of 5%NaCl and/or 8.4%NaCo3, and NO other concentration such as 1.8% and 3% are used.
7. The morbidity and mortality of both groups are recorded and statistically analyzed and compared on discharge or death.

Conflict of Interest

None declared by the author.

Funding Support

None declared by the author.

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