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Research Article

Fraudulent Science and Starling's law for the capillary-Interstitial Fluid transfer

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Abstract

Objective

To demonstrate Starling's law is wrong by summarising the substantial evidence on the hydrodynamic of the porous orifice (G) tube.

Material and methods

Focussed open mind narrative review of the substantial evidence on the hydrodynamic of the G tube that proves Starling's law wrong is presented. It provides the replacement for explaining the capillary-interstitial fluid transfer.

Result

The G tube has negative side pressure (SP) 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 occurs through side holes near the inlet while filtration occurs 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.

Conclusion

Both physics and physiological evidence demonstrate that the capillary works as G tube in which the arterial pressure induce negative SP 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 VO induce VO shocks (VOS): Sodium-free fluid induce VOS 1 and sodium-based fluid induce VOS 2. Examples of VOS 1 is the TUR syndrome known in urology also as hyponatremic 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.

Keywords

Capillary physiology, Starling's Law, Hydrodynamic, Shock, Fluid therapy, ARDS, The TUR syndrome.

Declaration of Conflicting Interest

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Substantial evidence currently exists to demonstrate that Starling's law is wrong [1], the revised Starling Principle is a misnomer [2] and all the formulae that goes with it are also wrong. Commonly received but erroneous concepts and laws my represent fraud in modern science. Starling's law for the capillary-interstitial fluid (ISF) transfer is a famous example. Persistent to defend such erroneous concepts is a futile attempt to defend fraudulent science.

Starling reported his hypothesis in 3 articles in the Lancet in 1886 and a fourth in J Physiology in 1896 [3,4]. He proposed that fluid exchange across the capillary wall is dependent upon the balance of two main opposing forces. The hydrostatic pressure pushing fluid out and the oncotic pressure withdrawing fluid into the capillary lumen. The hydrostatic pressure is a function of the arterial pressure and is higher near the capillary inlet that pushes fluid out over the proximal part as based on Poiseuille's work on uniform brass tubes. The oncotic pressure of plasma proteins becomes higher near the capillary exit and sucks fluid in over the distal part.

In fairness to professor Starling, he nether proposed a law nor equation for his hypothesis. Starling's hypothesis became a law later with equation after the report by Pappenheimer and Soto-Rivera in (1948) [5]. A serious experimental error by the authors is identified and reported here. These authors thought that elevating the capillary pressure may be achieved by elevating the venous pressure or arterial pressure alike, matching mmHg for mmHg, and they reported this to be in support of Starling's hypothesis. However, this has proved wrong, based on evidence from clinical practice: Elevating distal pressure (DP) akin to venous pressure augments capillary filtration as known in clinical practice causing oedema formation while elevating proximal pressure (PP) akin to arterial pressure does not, it enhances suction or absorption via the negative side pressure (SP) maximum near the inlet as demonstrated in the porous orifice (G) tube (Figure 1 and 2), and chamber C around it (Figure 3 & 4).

In support of the above fact is: High venous pressure, or obstruction, is the main cause of the most common clinical oedema but arterial hypertension though quite common it never causes oedema. Off course neither Starling nor any of the authors who transferred his hypothesis into a law were aware of the brilliant discoveries of precapillary sphincter [6] and wide porous wall of intercellular clefts [7] of the capillary that allow the passage of plasma proteins thus nullifies oncotic pressure in vivo that were discovered later in 1967. The G tube discovery demonstrates that PP akin to arterial pressure induce negative SP gradient exerted on the tube's wall that is maximum near the inlet causing suction or absorption. In addition to this I have reported 21 reasons that prove starling's law wrong [8]. So, both Starling's forces are wrong and so is the equations.

The same wrong conception that elevating CVP to levels of 20- 22 cm H20 may elevate the arterial pressure in shock management by infusing too much fluid was prevailing in clinical practice till recently. Fortunately, such practice has stopped now since it was realized that it induces volumetric overload shocks [9] that cause the interstitial oedema of vital organs and subcutaneously causing ARDS [10,11] It is worth mentioning the relation of G tube orifice diameter to SP of the G tube and the surrounding chamber C pressure (CP) shown in (Figure 1,2). This is relevant to the negative ISF pressure measured by Guyton and Coleman subcutaneously to be of -7 cm water [12]. This negative pressure of the ISF space can only be explained by hydrodynamics of the capillary working as G tube. Starling's forces cannot account for this negative pressure of ISF space and lymph vessels at all.

It was in 1981 based on clinical observations that Ghanem first incriminated Starling's law for the wrong rules dictating fluid therapy in shock management that mislead physicians into giving too much fluid during shock resuscitation [13]. He concluded his physics study on the porous orifice (G) tube by 1983 that was preliminary reported in 2001 [14] and concluded 2021 [1]. The G tube was built on a scale to the ultrastructure anatomy of the capillary of having precapillary sphincter (Orifice) and intercellular clefts as pores that allow the passage of plasma proteins- hence nullify oncotic pressure. The hydrodynamic of the G tube is totally different from the Poiseuille's tube on which basis Starling proposed his hypothesis. The high proximal (arterial) pressure induces a fluid jet through the orifice (precapillary sphincter) that creates negative SP gradient inside the wider lumen tube (capillary) that causes suction maximum over the proximal part of the tube near the inlet. It turns gradually positive to cause filtration maximum over the distal part near the exit. This creates a net negative pressure in a surrounding chamber (Figure 4) and induces a magnetic field like fluid circulation between fluid in the tube lumen and the surrounding chamber (Figure 1 and 2). This discovery has most important, serious, and relevant physiological and clinical significance.

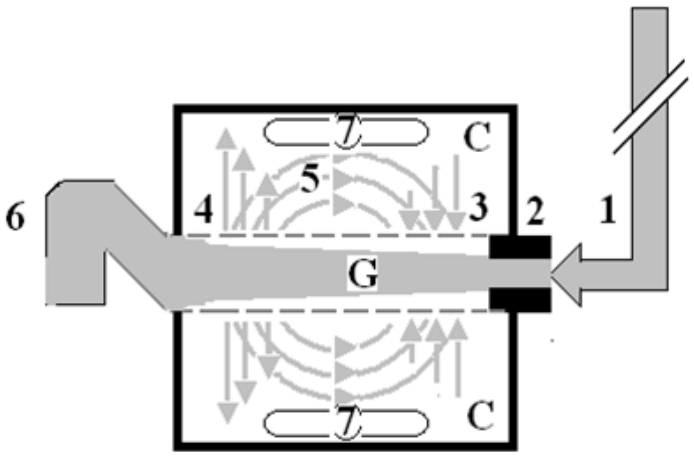

Figure 1 *shows a diagrammatic representation of the hydrodynamic of G tube based on G tubes and chamber C. This 37-years old diagrammatic representation of the hydrodynamic of G tube in chamber C is based on several photographs. The G tube is the plastic tube with narrow inlet and pores in its wall built on a scale to capillary ultra-structure of pre-capillary 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 run 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.*

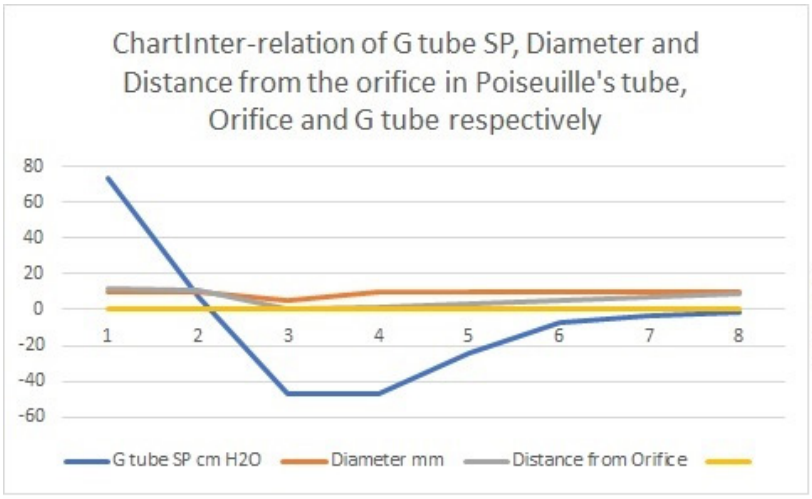

Figure 2 *shows the relationship between SP to the Diameter and length of the G tube which demonstrate a negative SP starting at the orifice (Point 2) (akin to precapillary sphincter) and extends as high negative pressure gradient over the proximal part of the G tube (Point 2-6) to cross 0 line at point 8 and then turn positive of 7 cm water at Point 9. This SP gradient from orifice at Point 2 to G tube lumen {Points 2-6) is negative to become positive DP at point 9 of 7 cm H20 water along the G tube. The wide section diameter of G tube is 7 mm all along the entire tube. The orifice is 5 mm while the distance from orifice to exit represent the tube' length in which the Fluid jet diameter change with increasing gradient (Figure 1a). Neither Poiseuille's law nor Bernoulli's equation can predict.*

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 [3,4], 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 [6]. 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 [7]- 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 proposed a law nor wrote any equations. Here we demonstrate that Starling's law is wrong on both of its forces [1,2], and the equations must be also wrong. This affirms the principle of what is built on wrongdoing must also be wrong. Both physics [1] and physiological [15] evidence demonstrate that the capillary works as G tube in which the arterial pressure induce negative SP gradient that causes absorption by suction not filtration that is maximum near the inlet. This is based on the hydrodynamic of the G tube summarized here. It has also been demonstrated that the oncotic pressure does not exist in vivo as the capillary has wide intercellular slit pores that allow molecules larger than plasma proteins to pass through it [7]. 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" [16].

THE PATHOLOGICAL CLINICAL SIGNIFICANCE OF HYDRODYNAMIC OF THE G TUBE WHEN THE CAPILLARY ACTS AS POISEUILLE'S TUBE INDUCING VOS AND ISF EDEMA OF ARDS

Starling's law being wrong has resulted in many errors and misconceptions on fluid therapy [17]. These errors mislead physicians [13] 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) [9,18] which cause the acute respiratory distress syndrome (ARDS) [19,20] or MODS. 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 are the transurethral resection of the prostate (TUR) syndrome [21] known in urology also as hyponatremic shock [22]. These are induced by massive absorption of 3.5-5 l 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 hemorrhagic or septicemic shock and is wrongly

treated with further volume expansion using crystalloids and/or colloids that transfer it into VOS 2. 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 [18, 19] or 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 [18,19].

What is the TUR Syndrome? And what is its relevance to VOS and ARDS?

The TURP syndrome is a condition induced by gaining large volume of sodium-free fluid (Figure 3 and 4) overloading the cardiovascular system and spelling into the interstitial fluid space of vital organs and subcutaneous. The fluid of 1.5%Glycine used as irrigating fluid gets absorbed during the TURP surgery as well as all endoscopic surgeries performed under sodium-free fluid irrigation of any type, BUT intravenous infusion of 5% Glucose considerably and significantly contributes to it- as well as saline. What is more saline or any sodium-based fluid such as Saline, Hartman, Ringer, plasma, and plasma substitutes, and blood worsens it transferring the shock being treated of VOS 1 into VOS 2 [24,37] and causing ARDS 1 and 2 [6,7] with its high morbidity and mortality later. The TUR syndrome has a characteristic severe drop of serum sodium level causing acute dilution hyponatraemia (HN) (Table 1, Figure 4) with severe clinical symptoms affecting all vital organs causing the multiple organ dysfunction syndromes (MODS) [42], or ARDS with recognizable clinical picture but one system may predominate such as AKI (Table 2). The HN of <120 mmol/l has 2 paradoxes and 2 nadirs that have eluded authorities and physicians on HN, and that has made the TUR syndrome most elusive and invisible with 2 paradoxes making it though obvious it has remained invisible even to authorities on HN. Professors and consultant urologists who are swift good resection experts have testified that the TUR syndrome does not exist with a negative prospective study of 100 patients [23]. Off course no such hyponatraemia occurs when the irrigating fluid is saline whatever the volume absorbed and infused. Another reason that prevents massive 1.5% glycine absorption and the TUR syndrome is not to open the prostate capsule and venous sinuses. There was also a urologist who did >1000 consecutive TURP surgeries without seeing the TUR syndrome.

The TURP syndrome starts by presenting with cardiovascular shock to anaesthetists and surgeons in theatre [9,18] and at times by cardiac or cardiopulmonary arrest [24] and sudden death. By next morning the surviving patients present with coma, convulsion and bizarre paralysis to physicians, neurologists and ICU specialists and the characteristic serum hypo-osmolality BUT other solute contents dilutions seem to be apparently improving due to water shift into cells (Table 3, Figures 5 and 6). The HN of <120 mmol/l causes cardiovascular shock. Volumetric overload (VO) is the most highly significant factor causing its patho-aetiology with a (p=0.0007). Osmolality was also significant (p=0.02) while all other serum solute changes including sodium and glycine did not reach statistical significance in the multiple regression analysis, yet it did alone when pre- and post-operative levels are compared!? (Table 3). This cardiovascular shock is easily confused with haemorrhagic or septicaemia shock and gets wrongly treated with further massive volume expansion!?

background.

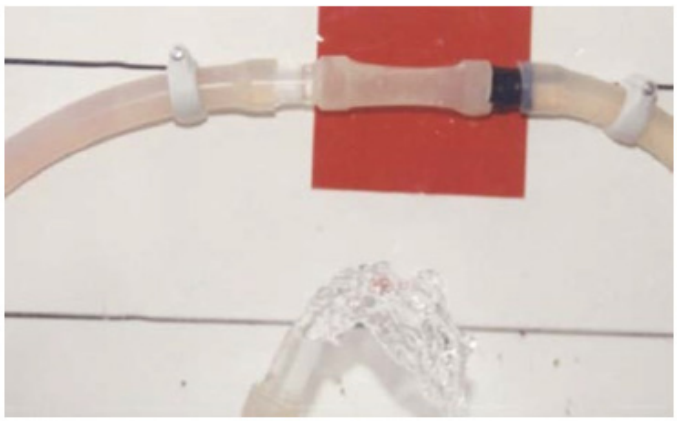

Figure 3. *The G-Tube enclosed in a rubber chamber (C) which is sucked in as water passes through the G tube indicating net negative pressure in C.*

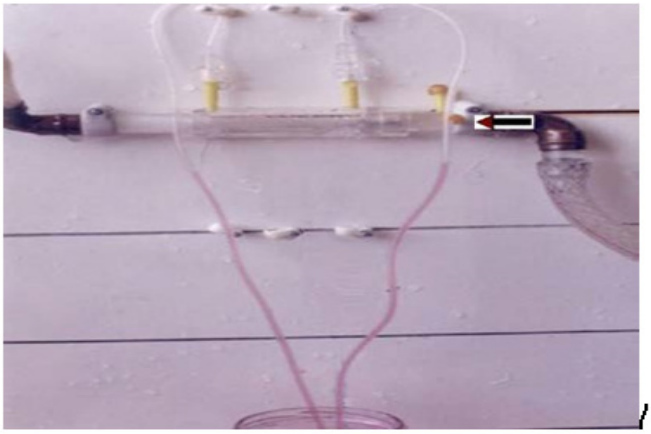

Figure 4. *The G-C model measuring the negative pressure in C with manometers sucking water from a jar 30 cm below.*

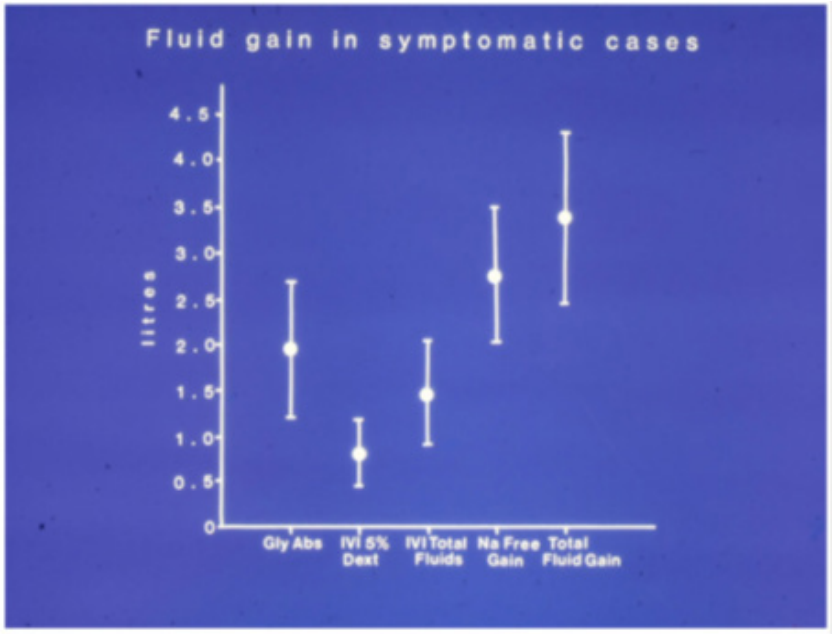

Figure 5 *shows the means and standard deviations of volumetric overload in 10 symptomatic patients presenting with shock and hyponatraemia among 100 consecutive patients during a prospective study on transurethral resection of the prostate. The fluids were of Glycine absorbed (Gly abs), intravenously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

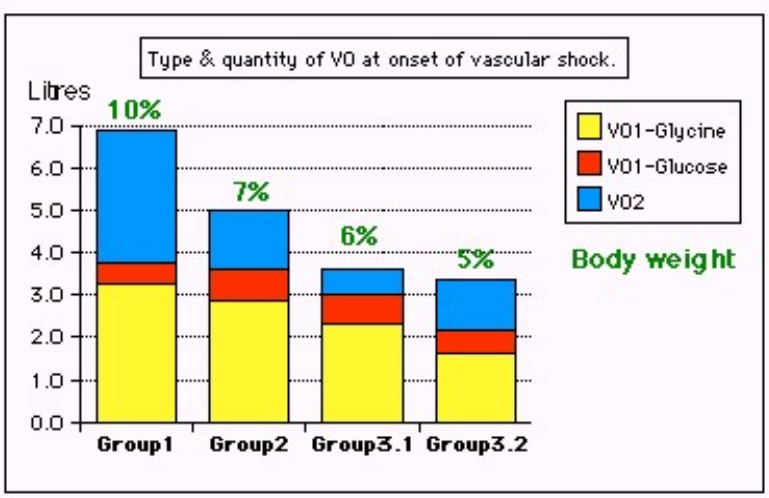

Figure 6 *shows volumetric overload (VO) quantity (in liters and as percent of body weight) and types of fluids. Group 1 was the 3 patients who died in the case series as they were misdiagnosed as one of the previously known shocks and treated with further volume expansion. Group 2 were 10 patients from 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.*

Cerebral	Cardiovascular	Respiratory	Renal	Hepatic & GIT
Numbness	Hypotension	Cyanosis.	Oliguria	Dysfunction:
Tingling	Bradycardia	FAM[4]	Annuria[8]	Bilirubin ↑
SBB[1]	Dysrhythmia	APO)[5]	Renal failure or	SGOT ↑
COC[2]	CV Shock*	RA[6]	AKI[9]	Alkaline Phosph.
Convulsions	Cardiac Arrest	Arrest	Urea ↑	GIT symptoms.
Coma	Sudden Death	CPA[7]	Creatinine ↑	DGR10
PMBCI [3]		Shock lung		Paralytic ileus
		ARDS$		Nausea & Vomiting.

Table 1 shows the manifestations of VOS 1 of the TURP syndrome for comparison with ARDS manifestations induced by VOS2. The manifestations are the same but one vital organ-system may predominate.

Table Abbreviation
SBB1: Sudden Bilateral Blindness
COC2: Clouding of Consciousness
MBCI3: 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
FAM4: Frothing Around the Mouth
APO5: Acute Pulmonary Oedema.
RA6: Respiratory Arrest.
CPA7: Cardiopulmonary Arrest; ARDS$: Occurs on ICU later.
Annuria8: That is unresponsive to diuretics but responds to HST of 5%Ncl and/or 8.4%NaCO3; AKI8: Acute Kidney Injury. Also occurs the excessive bleeding at
AKI9: Acute Kidney Injury
DGR10: Delayed Gut Recovery; CV Shock*:
Excessive bleeding may occur at the surgical site and leukocytosis occurred in the absence of sepsis and septic shock.

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 2 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 hypo-osmolality are the only significant factors.

1		Gr1	Gr2	Gr3	Gr3.1	Gr3.2	Normal	Units
2	Number of patients	3	10	10	5	5	mean	
3	Age	71	70	75	72	78	72	Years
4	Body weight (BW)	69	70	68	71	65	69	Kg
5	Postoperative serum solute concentration						Preoperative	
6	Osmolality	271	234	276	282	271	292	Mosm/1
7	Na+	110	108	120	119	121	139	Mmol/1
8	Ca++	1.69	1.79	1.85	1.84	1.86	2.22	''
9	K+ (P<.05)	5.6	4.8	5.0	4.9	5.0	4.46	''
10	Co2 (P=.002)	23.0	23.0	25.5	24.0	26.4	27.30	''
11	Glucose	13.2	17.3	16.4	15.9	16.9	6.20	''
12	Urea (P=.0726)	26.5	9.0	6.6	6.8	6.4	6.7	''
13	Bilirubin (P<.05)	19	16	8	6	9	7	''
14	AST	124	32	20	18	21	20	''
15	Protein	43	52	48	44	52	62	g/l
16	Albumin	23	30	30	28	32	39	''
17	Hb (P=.0018)	119.3	127.9	114.5	105.2	123.8	123.8	''
18	WCC (P<.005)	18.9	16.2	7.5	7.8	7.2	8.0	per HPF
19	Glycine			10499			293	µmol/1
20	Therapy	CT	HST	Randomized:	HST	CT©		
21	Outcome	Death	Full Recovery		Full Recovery	Morbidity		

Table 3 Shows the mean summary of data, therapy and outcome comparing the 3 groups of 23 case series patients who's (whose) VO is shown in (Figure 2). Groip-1 was the 3 patients who died and had post-mortem examination, Group-2 were a series of severe TURP syndrome cases successful ly treated with hypertonic sodium therapy (HST), and Group-3 were 10 patients encountered in the prospective study who were randomized between HST (3.1) and conservative treatment (CT) (3.2). The significant changes of serum solute contents are shown in bald font with the corresponding p- value. Most of the patients showed manifestation of ARDS of which the cerebral manifestation predominated, being on initial presentation (Regional Anesthesia) and representation of VOS 1 (General Anesthesia). However, most patients were given large volume of saline that elevated serum sodium to near normal while clinical picture became worse. They suffered VOS2 that caused ARDS. The VO of patients to whom these data belong is shown in Figure 4.

Conclusion

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 occurs through side holes near the inlet while filtration occurs 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 SP 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 VO induce VO shocks (VOS): Sodium-free fluid induce VOS 1 and sodium-based fluid induce VOS 2. Examples of VOS 1 is the TUR syndrome known in urology also as hyponatremic 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 edema of ISF space and of vital organs that cause ARDS.

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