

“Fluid Creep” in Critically Ill ARDS Surgical Patients: Time to Sop the Flood?!

Ahmed NM Ghanem^{1*} and Khaled Ghanem²

¹NHS The UK and Faculty of Medicine, Mansoura University, Egypt and Consultant Urologist Surgeon, Retired Independent Investigator and Scientist, Free Lance Author, Dreamer and White Revolutionary, London, UK

²Mansoura University, Egypt and Junior Clinical Fellow in Urology at Barking Havering and Redbridge University Hospitals NHS Trust, UK

***Corresponding Author:** Ahmed NM Ghanem, NHS The UK and Faculty of Medicine, Mansoura University, Egypt and Consultant Urologist Surgeon, Retired Independent Investigator and Scientist, Free Lance Author, Dreamer and White Revolutionary, London, UK.

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Fluid Creep (FC) is a new name for an old phenomenon of fluid retention in hospitalized critically ill Patients after resuscitation for major trauma, shock, and sepsis. The phenomenon is typically observed in patients suffering from the acute respiratory distress syndrome (ARDS). It is associated with high morbidity and mortality among the critically ill patients with ARDS. It has been reported since fluid therapy became the mainstay for shock and trauma resuscitation. Fluid creep is not really a new phenomenon, but the word creep may be relatively new use here. It has been consistently reported since the use of fluid therapy on a wide scale during World War 2. It was reported under the names of “fluid retention, fluid overload, positive fluid balance, fluid therapy complications and volumetric overload”. This phenomenon is both preventable and treatable.

This excellent article reviews the current knowledge and understanding of this phenomenon [1] which demonstrate that authors of the concerned authorities and physicians at large remain unaware of its true aetiology and best management. It also discusses Starling’s law as the main scientific foundation for fluid therapy in the shock resuscitation and acutely ill patients but falls short of incriminating it as the culprit for causing the phenomenon of fluid creep. My research that spans over the last 40 years and abundantly reported over the last decade has documented 13 new scientific discoveries in physics, physiology and medicine [2] that form the new scientific foundation for fluid therapy in shock management [3].

My research has demonstrated that Starling’s law is wrong on both of its forces and is the reason why good physicians are being misled into giving too much fluids during shock management. The discovery of the hydrodynamics of the porous orifice tube akin to capillary provide the correct replacement for the wrong Starling’s law [4,5]. The tree branching law corrects 2 widely received misconceptions on the capillary circulation [6].

Volumetric overload (VO) or fluid creep (FC) inflicts critically ill patients during fluid resuscitation for shock management. It is an iatrogenic condition. Most VO or FC starts by a flood in which large fluid volume is infused in a short space of time. It is of two types: Type 1 is induced by sodium-free fluids and is characterized by acute severe dilutional hyponatraemia of < 120 mmol/l for which the TUR syndrome is an example and Type 2 is induced by sodium-based fluids and presents as ARDS. VO presents initially with a cardiovascular shock that is mistaken for one of the well-known shocks and gets wrongly treated with further volume expansion [7,8]. The magnitude of VO or FC is shown in figure 1 and 2. Table 1 shows that VO or FC is most significant factor in the patho-etiology of the TUR syndrome and ARDS. Table 2 summarizes the clinical picture of the conditions. Table 3 shows the biological patients’ data and changes of serum solutes and therapy outcome. The first report on ARDS documented VO or FC of 12 - 14 litres in dead patients [9]. A recent huge, multi-center prospective study on ARDS document fluid retention or VO or FC of 7 - 10 liters in surviving ARDS patients [10].

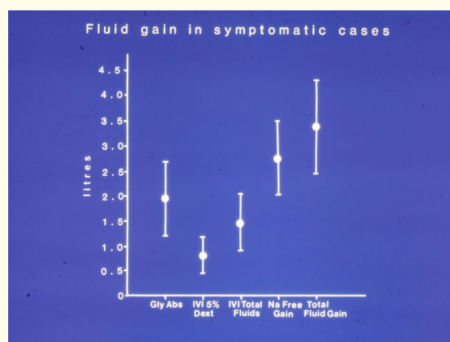


Figure 1: Shows the means and standard deviations of volumetric overload in 10 symptomatic patients presenting with shock and hyponatraemia among 100 consecutive patients during a prospective study on transurethral resection of the prostate. The fluids were of Glycine absorbed (Gly abs), intravenously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

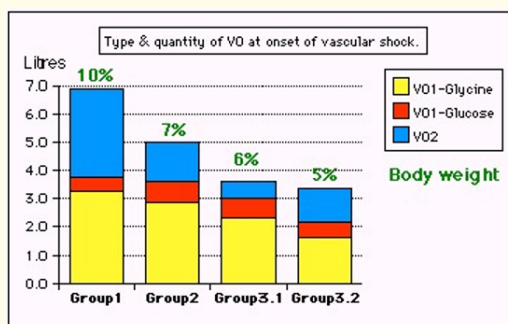


Figure 2: Shows volumetric overload (VO) quantity (in litres and as percent of body weight) and types of fluids. Group 1 was the 3 patients who died in the case series as they were misdiagnosed as one of the previously known shocks and treated with further volume expansion. Group 2 were 10 patients from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

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: 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.

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

Table 2: 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; ARDS⁵: Occurs on ICU later; Annuria⁸: That is unresponsive to diuretics but responds to HST of 5%Ncl and/or 8.4%NaCO3; AKI⁹: Acute Kidney Injury. Also occurs the excessive bleeding; AKI⁹: Acute Kidney Injury; DGR¹⁰: Delayed Gut Recovery; CV Shock*: Excessive bleeding may occur at the surgical site and leucocytosis occurred in the absence of sepsis and septic shock.

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	Mmol/1	
9	K ⁺ (P<.05)	5.6	4.8	5.0	4.9	5.0	4.46	Mmol/1	
10	Co ₂ (P=.002)	23.0	23.0	25.5	24.0	26.4	27.30	Mmol/1	
11	Glucose	13.2	17.3	16.4	15.9	16.9	6.20	Mmol/1	
12	Urea (P=.0726)	26.5	9.0	6.6	6.8	6.4	6.7	Mmol/1	
13	Bilirubin (P<.05)	19	16	8	6	9	7	Mmol/1	
14	AST	124	32	20	18	21	20	Mmol/1	
15	Protein	43	52	48	44	52	62	g/l	
16	Albumin	23	30	30	28	32	39	g/l	
17	Hb (P=.0018)	119.3	127.9	114.5	105.2	123.8	123.8	g/l	
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 on biochemical abnormalities, therapy and outcome comparing the 3 groups of 23 case series patients whose VO is shown in figure 2. Group-1 was the 3 patients with ARDS and fluid creep. who died and had post-mortem examination, Group-2 were a series of severe TURP syndrome cases successfully 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 bold 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 Anaesthesia) and representation of VOS 1 (General Anaesthesia). 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.

Also, my discoveries have demonstrated that both types have an effective lifesaving therapy using hypertonic sodium therapy (HST) of 5% NaCl and 8.4% NaCO₃ [11]. This HST should be given instantly, rapidly and adequately as early as possible in the course of the illness immediately after suspecting the diagnosis. By the end of this HST that lasts one hour giving less than one liter, the patient sheds urine of 4.5 - 5 liters of the fluid that was retained in his body. This massive urinary output should not be replaced by further fluid creep. The response to HST on correcting serum osmolality and sodium with matching remarkable clinical improvement is shown in figure 3. It is now possible to implement a useful policy based on the new sound scientific foundation for fluid therapy for resuscitation of shock that prevents and cures fluid creep in ARDS patients who are being killed in hundreds of thousands each year.

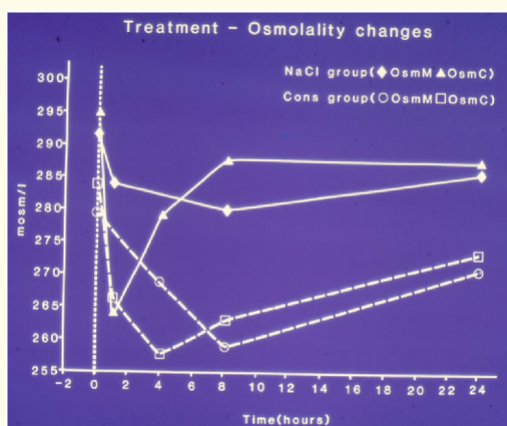


Figure 3: Shows mean changes in measured serum osmolality (OsmM) and calculated osmolality (OsmC) in patients with the TUR syndrome comparing those infused with 5% hypertonic sodium (solid lines) and those treated conservatively (slashed lines). OsmC was calculated from the formula $2 \times \text{Na} + \text{urea} + \text{glucose}$ in mmol/l of serum concentration thus reflecting changes in serum sodium concentration. The vertical dotted line represents the start of operation (Time B) followed by C, C1, C2 (end of one hour treatment) and D (next morning), respectively.

Conflict of Interest

None declared by the author.

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