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Editorial

# Fraudulent Science and Starling's Law for the Capillary-interstitial Fluid Transfer

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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. Persistent to defend such erroneous concepts is a futile attempt to defend fraudulent science. It is well known that Starling's law dictates the rules on fluid therapy for the management of shock which misleads physicians, and particularly anaesthetists and surgeons, into giving to much fluid during shock resuscitation, induction of anaesthesia and prolonged major surgery [3]. This practice induces the newly recognized volumetric overload shocks (VOS) that cause ARDS and hundreds of thousands of patients death per year [4-6] though remain unrecognized and underestimated.

Starling reported his hypothesis in 3 articles in the Lancet in 1886 and a fourth in J Physiology in 1896 [7,8]. He neither proposed a law nor equation. 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 strait uniform brass tubes. The oncotic pressure of plasma proteins becomes higher near the capillary exit and sucks fluid in over the distal part.

Starling's hypothesis became a law later after the report by Pappenheimer and SotoRivera in (1948) [9]. A serious experimental error by the authors occurred: The authors thought that elevating the capillary pressure may be achieved by elevating the venous pressure or arterial pressure alike, matching mmHg for mmHg. However, this has proved wrong, based on evidence from clinical practice: Elevating distal pressure (DP) akin to venous pressure augments capillary filtration as well known in 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).

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 [10] and wide porous wall of intercellular clefts [11] 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 [12]. So, both Starling's forces are wrong and the equations. It is time to say: Goodbye Starling's law, hello G tube [13].

## Conflict of Interest

None.

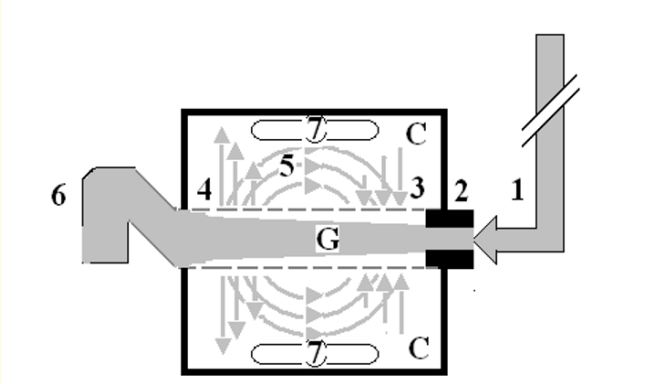


**Figure 1:** Shows a diagrammatic representation of the hydrodynamic of G tube based on G tubes and chamber C. This 37-years old diagrammatic representation of the hydrodynamic of G tube in chamber C is based on several photographs. The G tube is the plastic tube with narrow inlet and pores in its wall 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.

## Funding

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