New Contribution to Medicine, Physiology and Urology

Ahmed N Ghanem*
Retired Consultant Urologist, Egypt
*Corresponding author: Ahmed N Ghanem MD, FRCS, Retired Consultant Urologist, No 1, Jasmine Tower, President Mubarak Street, Mansoura 35511, Egypt, Tel: 002050367028; Email: amyghanem@gmail.com
Submission: November 07, 2017; Published: November 15, 2017

Introduction

These newly reported articles present new discoveries in Medicine, Physiology and Urology [1-4]. The articles recognizes 2 new types of socks 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. Volumetric Overload Shock (VOS) is a condition caused by massive fluid infusions and is of two types; Type one (VOS1) and Type two (VOS2). VOS1 is induced by sodium-free fluid gain of 3.5-5 litres such as Glycine, Glucose, Mannitol and Sorbitol. It is known as the TURP syndrome or hyponatraemic shock [5]. VOS2 is induced by massive infusion of sodium-based fluids such as normal saline, Ringer, Hartmann, plasma and plasma substitutes that may complicate the therapy of VOS1. VOS2 also complicates fluid therapy in critically ill patients suffering from other known shocks such as hypovolaemic, haemorrhagic and septicemia and presents with the adult acute respiratory distress syndrome (ARDS) [6]. VOS2 is induced by the gain of 12-14 litres of sodium-based fluids when reported in ARDS. The occurrence of massive interstitial tissue oedema with congestion of vital organs, pleural and peritoneal effusion, in the presence of hypotension shock, casted doubt on Starling’s law!

The last 32 years have been spent in investigating these issues at both clinical and physical fronts. Two clinical studies aiming to understand the TURP syndrome and recognizing VOS were done. A prospective clinical study on 100 consecutive TURPP patients of whom the condition affected 10 patients with severe hypotension and bradycardia and severe acute hyponatraemia of <120mmol/l was conducted. Volumetric overload was the only significant factor in causing the condition. The second clinical study involved a case series of 23 cases of the TURP syndrome manifesting as VOS1. The first 3 cases died as they were treated erroneously as one of the recognised shocks and treated with further volume expansion. The remaining 20 patients were treated with hypertonic 5% sodium chloride. Each patient passed 4-5 litres of urine followed by recovery from shock and coma.

The physical investigation involved studies of the hydrodynamics of the porous orifice (G) tube comparing it to that of Poiseulle’s tube. Thousands of experimental measurements of pressures at various parts of a circulatory system incorporating the G tube in a chamber to mimic the capillary-interstitial fluid compartment. 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. It is quite remarkable how this circulatory model mimic the circulatory system in heath and disease.

Shock is a disturbance at the capillary cellular level impairing the capillary-interstitial fluid transfer; delivery of oxygen and removal of waste products. The process is governed by Starling’s law [7]. In this law the arterial pressure is considered the force causing capillary filtration! If this is true, how come that arterial hypertension though common never causes oedema? Starling based his hypothesis on Poiseuille work on strait uniform brass tubes. Latter evidence however demonstrated that the capillary is a porous narrow orifice (G) tube as it has a pre-capillary sphincter and pores that allow the passage of plasma proteins [8,9]. As the capillary pores allow the passage of plasma molecules, nullifying the osmotyic pressure of plasma proteins, a call for reconsideration of Starling’s hypothesis was previously made but there was no alternative at that time [10].

The hydrodynamics of the G tube 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 [11,12]. Incorporating the G tube in a chamber (C), representing the interstitial space surrounding a capillary, demonstrated a rapid dynamic magnetic field-like fluid circulation between the C and G tube lumen. Incorporating the G tube and C in a circulatory model driven by electric pump inducing proximal pressure similar to arterial pressure; causing suction from C into the lumen of G tube. The distal (venous) pressure is responsible for filtration. This proves that the arterial pressure causes suction not filtration at the capillary interstitial fluid circulation, and hence Starling’s law is wrong. The reported hydrodynamics of the G tube provide an adequate mechanism for the capillary interstitial fluid circulation.
References


