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Mini Review

# Does Raising the Central Venous Pressure (CVP) in Treating Shock with Fluids Induce Volumetric Overload Shocks (VOS)?

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## Introduction

The answer to the heading question is an affirmative yes! Not only persistence to elevate the central venous pressure (CVP) but also pulmonary capillary wedge pressure (PCWP) is among the fundamental commonly practiced but most misleading reasons for overzealous fluid infusion in managing haemodynamic shock of poly-trauma victims and inducing volumetric overload shock (VOS). The generally received practice on raising CVP and PCWP to level of 18-22 cm water in the management of shock as well as in acutely ill patients is a misconception [1]. All physiology textbooks testify that the normal CVP is 0 and ranges between -7 to +7 cm water measured at middle auxilary line- the level of right atrium on supine posture [2]. The best advice here is that we should imitate nature exactly if we can't understand how it truly works. Raising CVP to such high level vastly increases filtration force at capillaries inducing VOS and causing oedema of the multiple vital organ dysfunction/ failure (MVOD/F) as detailed in the articles [3,4].

Deep at the bottom of all such physiological errors and clinical misconceptions [5] concerning elevating venous and arterial pressures in managing shock are the erroneous Starling's law and the overlooked newly discovered hydrodynamic magnetic phenomenon of porous orifice (G) tube [3]. The latter makes sense of circulatory haemodynamic in health and in shock. All errors are rectifiable when the new phenomenon redefines shock précising the correct role played by the arterial and venous pressures, and the unique role played by the pre-capillary sphincter in both the capillary-interstitial circulation and systemic circulation [5]. The orifice diameter induces maximum efficient flow of the hydrodynamic capillary-interstitial circulation when its ratio to tube diameter is 0.7 that equals half of the tube lumen area. The relation of orifice size to the negative pressure exerted on the tube's wall is an inverted Bell shape. At orifice diameter of 0.7 the interstitial space is well circulated, hydrated and ventilated while staying "dry" under optimum negative pressure not "wet" with oedema of accumulated stagnant fluid that drown cells inducing VOS and MVOD/F that occurs when CVP is elevated. The orifice thus fundamentally affects both the hydrodynamic of G tube and the capillary haemo-dynamics of systemic circulations. Too narrow or too wide orifice induces hampering effect. This suggests that the pre-capillary sphincter, rather than the replaceable heart, is indeed the master of circulation.

Another thought-provoking point that may be considered in managing shock in polytrama and may limit tendency for overzealous infusions, is to wonder what is the maximum volume of blood loss that a patient can bear should he reaches the hospital alive? Most available evidence on animal and human studies indicates that rapid loss of about half the circulatory volume is incompatible with life. Thus, nothing will be gained if more than such volume is given in resuscitating shocked patient. Should this be considered as limiting factor for infusion quantity in shock management? In most practices double or triple the circulatory volume is rapidly infused into such patient and much larger volume is required to raise CVP above 10 cm water. Even in the most difficult circumstances of polytrauama in which bleeding into body cavities or around broken large bones may continue, an attempt should be made to stop that bleeding rather than persist on resuscitation by elevating CVP and arterial pressures before wheeling the patient for surgery. In current practice the opposite is employed. Many patients die during this futile attempt before taken to theatres to stop the bleeding, at which time the original insult causing shock of polytrauma is blamed without a question. Is it, perhaps, a method

of medico-legal defence because doctors who may try but fail to rescue such patient's life will risk their own if such patient died on the operating table? The last 32 years of my career life were spent in investigating and reporting these articles [5-9].

The articles recognise 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 2 clinical syndromes discovering its patho-etiology and its new successful treatments; namely the transurethral resection of the prostate (TURP) syndrome and the acute respiratory distress syndrome (ARDS). It also corrects many errors and misconceptions on fluid therapy [5]. Volumetric Overload Shock (VOS) is a condition induced by large fluid infusions in a short time 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 in one hour such as Glycine, Glucose, Mannitol and Sorbitol. It is seen in the TURP syndrome [4] or hyponatraemic shock. VOS2 is induced by massive infusion of sodium-based fluids such as normal saline, Ringer, Hartmann, plasma, plasma substitutes and blood transfusions 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 septic shocks and presents with ARDS. VOS2 is induced by the gain of 12-14 litres of sodium-based fluids when reported in ARDS. The presence of massive interstitial tissue oedema with engorgement of vital organs, pleural and peritoneal effusions, in the presence of hypotension shock, casted doubt on Starling's law! These issues were investigated at the clinical and physiological/physical fronts [3-9].

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