



# Hyponatraemia: Nadirs and Paradoxes of the Missing Volumetric Overload



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## Short Communication

Hyponatraemia (HN) affects adults and children [1]. The authors concluded that: "acute hospital induced hyponatraemia is caused by electrolyte-free fluids, or 'volumetric overload type1 (VO1)', in the presence of elevated vasopressin. The condition affects men, women and children, particularly postoperative patients. It is responsible for far greater morbidity and mortality than generally received." It concerns not only physicians and intensive care therapists among all clinicians who prescribe fluid therapy in the most compelling of clinical emergencies, but also primarily surgeons.

The authors' call for a change of the policies on fluid therapy is fully supported but requires scientific justification. It is interesting that such revolutionary conclusion is based on a retrospective study and observations. Had I been gifted with the clarity and authority with which the conclusion was given, not only HN but also the puzzle of the multiple vital organ dysfunction/ failure (MVOD/F) syndrome or acute respiratory distress syndrome (ARDS) should have been resolved long ago. I commend the Editor and reviewers of BMJ for considering this study an evidence-based medicine.

I have long recognized these conditions as killers of surgical patients and have spent most of my career life investigating and understanding. My initial observations were communicated over 32 years ago [2]. For resolving giant clinical puzzles such as HN and MVOD/F, one needed to fly high for a bird's eye view and dive deep for a close-up examination of a piece of evidence that lies at the bottom of the ocean of knowledge on the subject. In doing so, artificial boundaries of science and super specialties of medicine and surgery needed crossing for gathering the scattered pieces of evidence. Missing pieces were either excavated and rejuvenated or newly discovered. Facts needed segregation from fallacies, and reorganization, in order to reconstruct the real picture.

Although acute HN is 'iatrogenic complication' of fluid therapy, it should not cause any 'guilt feeling'. It is a complex problem with wide range of severity and many masks of presentations. It underlies many variously named but identical clinical syndromes such as the transurethral resection of the prostate (TURP) syndrome and others affecting medical and surgical patients [2].

It may be confused with many recognized medical conditions as well as features of MVOD/F [3]. Its primary nadir and paradox remain overlooked. The key for resolving the puzzles relates to a century old erroneous physiological law that dictated the 'faulty rules' on fluid therapy [4]. The authors alluded to the 'missing' VO1 at the second nadir of HN on tertiary referral. This article's main objective is to identify, locate and quantify the 'missing' VO1 and uncover its primary nadir and paradox. Major surgery in general and TURP are examples where and when VO1 may induce the condition and its first nadir [5]. Not only the faulty rules on fluid therapy dictate the infusion of large VO during surgery but also, Invariably and incorrectly, every hypotension episode is considered synonymous with hypovolaemia [3].

The observed paradox was this: VO1 of about 5% body weight (BW) gained in one hour may cause hypotension shock. This misleadingly calls for further vascular expansion using electrolyte-based fluids (VO2), inducing shock, coma and acute renal failure (ARF) among other features of the MVOD/F syndrome.

Dilutional hyponatraemic shock was reported 55 years ago [6] and later in patients suffering from the TURP syndrome [7]. It represents VO1 vascular shock. Our prospective study [5] precisely quantified VO1 by adding the per-operative infused fluids to the measured volume of absorbed 1.5% Glycine irrigant. After providing anecdotal evidence [8], our prospective study also re-juvenated the life-saving therapy of instant rapid infusion of hypertonic sodium therapy (HST) such as 5% NaCl or 8.4 NaCO<sub>3</sub> [7].

Further experience showed that 8.4% sodium bicarbonate is a good alternative therapy but lower sodium concentrations of HST are less effective. All VO2 fluids such as isotonic saline is harmful, as it clouds the serum markers while aggravating VO and establishing the MVOD/F syndrome [3-5]. VO1 dilutes all serum contents and induces the first nadir of HN [5], manifesting at the immediate post-operative period. The second nadir of HN may appear less seriously later, reflecting much less VO1 than was gained. This is due to the intracellular osmotic shift of fluid. Cellular oedema manifests with encephalopathic coma and ARF, among other features of MVOD/F [3,4].

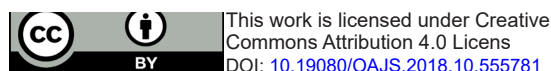
Further delay, before and after involving the physicians, may make HN chronic, establishing the permanent brain damage. The latter was incorrectly attributed to HST. The second nadir of HN may also follow another common scenario: any urinary output that represents a recovery from VO1, when chased by fluid input may keep the retained VO1 constant or increased. No pitting oedema occurs because most of the 'missing' VO1 resides inside the cells. Time plays a vital hidden role on the nadirs of HN of VO1. This was the hardest to understand and unravel. "VO versus time" (of fluid infusion) is a helpful concept to consider in clinical settings requiring massive fluid therapy or vascular expansion.

Contrasting the physiological effects of a normal daily fluid intake (about 5% BW or 3.5 l of 70 Kg) to its pathological effects when infused in one hour, should be considered very seriously. Mathematical calculation reveals that such VO1 lowers the serum sodium from 140 to 112 mmol/l (the first HN nadir). Assuming no further input or output occurred, the time (of osmotic equilibrium) might spontaneously elevate the second nadir to 129 mmol/l. This is the result of extracellular fluid space dilution by only 1.2 l. The 'missing' 2.3 l of gained VO1 resides in cells.

Perhaps, a better approach for resolving such complex clinical problem was to go 'back to basics and to the future': to recognize the error of the physiological law that dictated the faulty rules on fluid therapy. Physical studies, done in 1984 and reported in 2001 [4], discovered a hydrodynamic phenomenon with physiological and clinical relevance to the mentioned conditions. This may provide a tenable alternative hypothesis for the capillary-interstitial fluid transfer, and the scientific basis for the call to change the policies on fluid therapy. Recently I have reported all the evidence to consider volumetric overload shock (VOS) a clinical reality [9-11] and to prove that Starling's law is wrong providing the alternative of hydrodynamics of a porous orifice (G) tube [12].

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