The Central Venous Pressure Causality Factors

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Received date: June 15, 2017; Accepted date: June 16, 2017; Published date: June 20, 2017

The pressure which is measured in the central veins located near to the heart is known as the central venous pressure (CVP). The CVP denotes mean right atrial pressure which is for the most part considered as a gauge of right ventricular preload. It doesn’t specifically compute blood volume, yet it is regularly used to assess for this reason. Generally, the CVP value is calculated by the right heart function and the venous blood (situated in the vena cava) pressure. Note that the CVP is not only influenced by venous return and intravascular volume, but also by intra-thoracic pressure and venous tone, along with myocardial compliance and right heart function. Generally, CVP is used for the hypotension patients who do not respond to primary clinical management. The present note examines the CVP causality factors of shock patients [1-3]. Interested hypotheses are: What are the causal components of CVP? How are the causal variables related with the CVP? What are their effects on CVP? Answers of these speculations are minimal known in the cardiology literature. These issues are addressed in the present note with a real data set which is displayed in [1].

The present data set is displayed in various sites [1]. The data set has been collected at the Shock Research Unit, The University of Southern California, Los Angeles, California. It includes 113 shock patients with 20 variables/ factors. On each patient, two measurements are taken. Initial measurement is taken at the entry time of hospital admission, and the final measurement is taken at the discharge time or just before death. The data collection method, patient population and shock types are given in [1]. Joint generalized linear gamma model analysis [4] is used to identify the causal elements of CVP. Based on the joint gamma fitted models, the causal factors of the CVP of shock patients are reported in the present note.

There are 20 attribute characters/ variables in the considered shock data set. The attribute characters/ variables are age, sex (male=0, female=1), height, systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial blood pressure (MAP), mean central venous pressure (MCVP), heart rate (HR), cardiac index (CI), mean circulation time (MCT), shock type (non-shock=1, hypovolemic=2, cardiogenic, or bacterial, or neurogenic or other=3), body surface index (BSI), urinary output (UO), plasma volume index (PVI), appearance time (AT), hematocrit (HCT), red cell index (RCI), hemoglobin (HG), survival stage (survived=1, death=2), order of card record (initial=1, final =2) (OCR).

The MCVP is considered as the dependent variable, and the rest others are dealt with as the illustrative factors in the joint gamma fitted models. The joint gamma fitted models interpret the following for all the above hypotheses.

- The mean CVP (MCVP) is directly associated with the sex (male=0, female=1) (P=0.0226), indicating that CVP is higher for female shock patients than male.
- The MCVP is positively associated with the mean arterial blood pressure (MAP) (P=0.0082), implying that CVP increases as MAP increases, and vice-versa.
- The MCVP is inversely related with the diastolic blood pressure (DBP) (P=0.0015), indicating that CVP increases as DBP decreases, and vice-versa.
- The MCVP is directly associated with the body surface index (BSI) (P<0.0001), indicating that CVP increases as the BSI increases.
- The MCVP is reciprocally related with the appearance time (AT) (P=0.0019), indicating that CVP increases as the AT decreases, and vice-versa.
- The MCVP is directly correlated with the mean circulation time (MCT) (P<0.0001), implying that CVP increases as MCT increases.
- The MCVP is negatively related with the urinary output (UO) (P=0.0105), indicating that CVP increases as the UO decreases, and vice-versa.
- The MCVP is directly related with the plasma volume index (PVI) (P=0.0156), indicating that CVP increases as the PVI increases, and vice-versa.
- The variance of CVP (VCVP) is directly associated with the appearance time (AT) (P=0.0338), indicating that VCVP increases as the AT increases, and vice-versa.
- The VCVP is inversely associated with the mean circulation time (MCT) (P=0.0057), implying that VCVP increases as MCT decreases.
- The VCVP is directly partially related with the plasma volume index (PVI) (P=0.1091), indicating that VCVP increases as the PVI increases, and vice-versa.
- The VCVP is inversely related with the red cell index (RCI) (P<0.0001), indicating that VCVP increases as the RCI decreases.
- The VCVP is directly related with the hematocrit (HCT) (P=0.0043), indicating that VCVP increases as the HCT increases.
- The determinants of the CVP, and their effects & associations with CVP are focused in the above interpretations. It is shown that mean CVP is positively associated with the mean arterial blood pressure, while it is negatively associated with the diastolic blood pressure. Note that it is independent of the systolic blood pressure. It is highly associated with the body surface index. Appearance time is negatively associated with the mean CVP, while it is positively associated with the variance of CVP. Mean circulation time is positively associated with the mean CVP, while it is negatively associated with the variance of CVP. Plasma volume index is directly associated with both the mean and variance of CVP. Urinary output is negatively associated with the mean CVP, and this knowledge may be used in the clinical...
management of CVP. Red cell index is negatively, while hematocrit is positively associated with the variance of CVP. So, it is clear that many factors are responsible for changing the central venous pressure. The above information may be very helpful for the cardiologists. Cardiology researchers are advised to consider basal & maximum blood pressure, basal, peak & maximum heart rate, ejection fraction, body mass index as explanatory variables of CVP in their future studies.

References