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Case Report

Stroke Volume Variation Guided Fluid Management Intraoperatively Using Cardiac Output Monitor in a Case of Craniofacial Resection

J. Suresh[1], T. Rajaram Manoharan[2], A. L. Shanmuga Priya[3], T. Balaji[4]

[1]Junior Consultant, Department of Anesthesiology, Preethi Hospitals Private Limited, Tamil Nadu, India, [2]HOD and Professor, Department of Anesthesiology, Preethi Hospitals Private limited, Tamil Nadu, India, [3]Senior Resident, Department of Anesthesiology, Preethi Hospitals Private Limited, Tamil Nadu, India, [4]Senior Consultant, Department of Anesthesiology, Preethi Hospitals Private Limited, Tamil Nadu, India

Abstract

Early aggressive resuscitation of critically ill patients may limit or reverse tissue hypoxia, organ failure progression, and improve outcome. Similarly, a protocol to optimize preload and cardiac output in patients undergoing major surgery reduced post-operative complications and length of stay. However, overzealous fluid resuscitation has been associated with increased complications, increased length of intensive care unit and hospital stay, and increased mortality. Cardiac filling pressure, including central venous pressure and pulmonary artery occlusion pressure, have been traditionally used to guide fluid management. Studies conducted over the past decades demonstrate that cardiac filling pressures are unable to predict fluid responsiveness. At present, stroke volume variation that normally occurs during phases of the respiratory cycle is used to guide fluid responsiveness during major surgeries.

Key words: Cardiac output, Fluid responsiveness, Stroke volume variation

INTRODUCTION

Precise assessment of volume status is a prerequisite for adequate volume replacement which may achieve optimal organ perfusion and oxygen supply. Frequently used preload indexes such as central venous pressure (CVP), pulmonary artery occlusion pressure (PAOP), intra thoracic blood volume index (ITBI), and left ventricular end diastolic area index (LVEDAI), often fail to provide reliable information as an alternative to these static variables assessment of stroke volume variation has been used as an indicator for hemodynamic monitoring to predict fluid responsiveness in patients requiring major surgeries involving large fluid shifts.[1-4]

CASE SCENARIO

We report a 36-year-old male presenting with ulcers over the hard palate and bleeding from the ulcer site for 30 days and ear pain with discharge. No h/o epistaxis/giddiness. On examination, the patient is moderately built, weighing 60 kg. Another system examination was found to be normal. Echocardiography, spirometry, and other blood investigations are within normal limits. The patient had been diagnosed as recurrent carcinoma hard palate (well-differentiated squamous cell carcinoma) and subsequently underwent partial maxillectomy with supraomohyoid neck dissection in September 2011. Now, because of intracranial extension, middle craniofacial resection and reconstruction with temporalis flap and superficial temporal artery-based osteoplastic flap were planned [Figure 1].

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ANESTHETIC MANAGEMENT

After getting high-risk informed consent, the patient was scheduled for surgery. The patient was pre-medicated with inj. glycopyrrolate 0.2 mg iv, inj. ranitidine 50 mg iv, inj. ondansetron 8 mg iv, and inj. morphine 4 mg iv. In the operating room, peripheral line was established with 16 gauge Venflon. Other monitors such as NIBP, pulse oximeter, and E.C.G. were used. A continuous spinal catheter was placed at the L2-L3 level with 19G epidural catheter to reduce the intracranial pressure. After local infiltration and anesthetizing lower airway, tracheostomy was performed and 8 sizes

Corresponding Author: J. Suresh, Department of Anesthesiology, Preethi Hospitals Private Limited, Tamil Nadu, India.

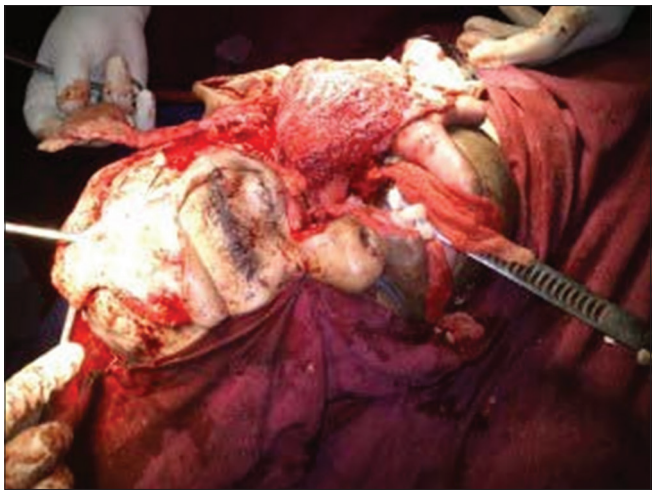

Figure 1: Craniofacial resection being done

Figure 2: Cardiac output monitor showing stroke volume variation and stroke volume index

flexometallic tube inserted. After checking tube position and air, entry patient was induced with inj. propofol 130 mg iv and inj. atracurium 35 mg. Anesthesia was maintained with isoflurane oxygen-nitrous oxide volume-controlled ventilation with a tidal volume of 8 ml/kg. The right subclavian vein was cannulated with 7 Fr 20 cm triple lumen catheter for central venous pressure (CVP) measurement and the left radial artery was cannulated with 18 gauge Venflon and connected to Vigileo cardiac output monitor. After induction, the following variables are measured: CVP, S.V., CO, and stroke volume variation (S.V.V.). Then, S.V.V. was measured at 15 min intervals whenever S.V.V. is more than 13% fluid was given with crystalloids and hetastarch in increments of 50–100 ml to maintain S.V.V. around 10 and CVP of 6–10 mmHg [Figure 2].

DISCUSSION

Several dynamic tests of volume responsiveness are reported. These tests dynamically monitor change in stroke volume after a maneuver that increases or decreases venous return. Tests such as pulse pressure variation (PPV) derived from analysis of arterial waveform, S.V.V. derived from pulse contour analysis, and variation of the amplitude of pulse oximeter plethysmographic waveform are highly predictive of fluid responsiveness.[5] Systolic arterial pressure variation (SPV) and PPV are also influenced by the vasomotor tone which is supposed to be less the case with S.V.V. We used S.V.V. for assessing fluid responsiveness in this patient. S.V.V. is the change in stroke volume during respiratory cycle. It occurs due to cyclic changes of intrathoracic pressure induced by mechanical ventilation and can be assessed continuously by any beat-to-beat cardiac output monitor.[6] The following equation assesses S.V.V. as a percentage of S.V. during the ventilator cycle: SVV=SV max-SV min/S.V. mean where maximum and minimum S.V. are mean values of four extreme values of S.V. during a period of 30 s and mean S.V. is the average value for this period.[7] The major determinant of this variable is the reduced venous return during mechanical inspiration. Both arrhythmias and spontaneous breathing will lead to misinterpretation of PPV and S.V.V. For any specific preload, PPV/SVV will vary according to tidal volume and airway pressure. Many studies show that a rise in S.V.V. predicts the subsequent appearance of hypotension and necessity for additional fluid, particularly when S.V.V. exceeds 15%. Intermittent positive pressure ventilation induces cyclic changes in loading conditions of the left and right ventricles. Mechanical ventilation decreases preload and increases afterload of the left ventricle. The right ventricle preload reduction is due to a decrease in venous return pressure gradient. The inspiratory reduction in the right ventricular ejection leads to decreased LV filling after a phase lag of 2–3 heartbeats. Thus, the left ventricular preload reduction may lead to a decrease in the left ventricular stroke volume.

In case of ARDS low tidal volume which is used reduces the amplitude of change in intra thoracic pressure that causes the SVV. If the tidal volume challenge results in increase in absolute value of SVV more than 2.5 % fluid responsiveness is likely.[8] A variation of >12–13% is highly predictive of volume responsiveness. S.V.V. was advocated to be more accurate than SPV and PPV. The patient during sepsis state and undergoing neurosurgery demonstrated the ability of S.V.V. to predict fluid responsiveness.

CONCLUSION

Thus, S.V.V. can serve as a useful indicator of fluid responsiveness in mechanically ventilated major surgical patients. However, further studies are required to demonstrate the usefulness of both automatically

calculated variables to guide volume optimization in critically ill patients to improve outcomes.

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