Section: Anaesthesia

Review Article

ISSN (O): 2347-3398; ISSN (P): 2277-7253

# Management of Occult Hypoperfusion Syndrome in Trauma Patients: A Narrative Review

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## Abstract

Occult hypoperfusion (OH) is connected with higher levels of morbidity and mortality after trauma. Occult hypoperfusion, defined as serum lactate concentration of more than 2.5 mmol / L persisting in the intensive care unit for more than 12 hours after admission. This refers to the reversible risk factor for negative results after traumatic injury. Occult hypoperfusion can be observed and patients at risk of complications should be classified other than frequently regulated metrics (blood pressure and heart rate), central venous oxygen saturation, and blood lactate levels. An elevated hospital duration of stay (LOS) and a greater incidence of postoperative difficulties are linked with OH. The focus of this review article is to assess the different approaches and methods involved in the management of OH syndrome in trauma patients. Identification of OH treatment methods can be helpful in reducing morbidity in patients with various injuries. The outcomes from this review article may prove beneficial to patients by rapid resuscitation and aggressive monitoring of OH. These management practices will severely reduce OH-associated morbidity and mortality. LOS, ICU readmission incidence and post-operative complications per patient was decreased by early treatment of OH.

**Keywords:** Management, Occult hypoperfusion syndrome, Trauma

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**Received: 11 August 2020** **Revised: 8 September 2020** **Accepted: 17 September 2020** **Published: 06 October 2020**

## Introduction:

The dictionary term 'occult' refers to any mystical, supernatural, or magical powers, practices, or phenomena. In clinical terminology, however, the occult case contains high serum lactate levels in unfavorably ill patients without symptoms of physiological shock. In the literature, several definitions of occult hypoperfusion (OH) are available, as enlisted below [Table 1].

By simple definition, hypoperfusion is the reduced amount of blood flow, which later develops into ischemia.[21] Any organ of the body can be afflicted by Ischemia. Occult hypoperfusion, also known as subclinical hypoperfusion (SCH) or occult shock, becomes apparent when the cellular hypoxia continues to be present with normal vital signs. The presence and length of duration of occult hypoperfusion are correlated with poor patient outcomes. Occult hypoperfusion instigates inadequate oxygen perfusion within the tissue for metabolic demands. This vital observation is missed during the standard clinical evaluation because of the presence of typical vigorous signs.[22] Occult hypoperfusion is related to increased hospital stay time and enlarged mortality. Low blood pressure, heart disease, or lack of blood flow are causes for hypoperfusion.[23]

The actual phenomenon of OH can be explained where traditionally, resuscitation of hypotensive trauma patients was dependent on traditional clinical markers like blood pressure and heart rate. In a limited number of patients, this technique was relatively successful, although some recent findings have indicated that normal heart rate and blood pressure were provided to a wide subset of patients while also providing significantly elevated lactate levels, which is suggestive of cellular hypoxia. The involvement is associated with poor health results, as well as the duration of supernatural hypoperfusion. The latest publications also indicated that patients who undergo surgery in this state with OH also reported poor outcomes.[24–27]

Studies also found that the leading cause of mortality is injuries during the first four decades of life.[28,29] When a survivor of injuries appears in the emergency room before the primary survey takes place, it is important to ask the ambulance team about the accident process. This offers useful data on the energy transfer, processes of deceleration, acceleration, and shear forces involved. Awareness of the damage process

**Table 1: Changing definitions of occult hypoperfusion.**

| SN | Study | Year | Definition of hemodynamic stability |
|---|---|---|---|
| 1 | Blow et al, [1] | 1999 | SBP > 100 mmHg, Heart rate < 120 bpm, Urine output >1 ml/kg/h, LA >2.5 mmol/L |
| 2 | Claridge JA, [2] et al | 2000 | SBP > 100 mmHg, Heart rate < 120 bpm, Urine output > 1 ml/kg/h LA ≥ 2.5 mmol/L |
| 3 | Crowl AC et al, [3] | 2000 | SBP > 100 mmHg, Heart rate < 120 bpm, Urine output > 1 ml/kg/h LA ≥ 2.5 mmol/L |
| 4 | Meregalli et al, [4] | 2004 | SBP > 100 mmHg, Heart rate < 120 bpm, Urine output > 1 ml/kg/h Simplified acute physiology score II < 25±5 |
| 5 | Schulman Am et al, [5] | 2004 | SBP > 110 mmHg, Heart rate < 120 bpm, Urine output > 1 ml/kg/h LA ≥ 2.5 mmol/L |
| 6 | Howell MD et al, [6] | 2007 | MAP ≥ 70mmHg, Heart rate ≤ 110 bpm LA ≥4 mmol/L |
| 7 | Jansen T C et al, [7] | 2008 | SBP ≥ 100 mmHg, LA ≥ 3.5 mmol/L |
| 8 | Jansen T C et al, [8] | 2009 | SBP > 100 mmHg, Heart rate < 120 bpm, Urine output > 1 ml/kg/h LA > 2 mmol/L SOFA 0.62 SOFA-points/day |
| 9 | Martin JT et al, [9] | 2010 | SBP ≥ 90 mmHg, Heart rate < 120 bpm, Urine output > 1 ml/kg/h LA > 2.2 mmol/L |
| 10 | Thom, [10] | 2010 | SBP ≥ 100 mmHg, Heart rate ≤ 110 bpm, Urine output > 1 ml/kg/h Temperature > 35°C Cl < 2.6 L/min/$m^2$ |
| 11 | Corradi, [11] | 2011 | SBP ≥ 90 mmHg, Heart rate < 120 bpm, Urine output ≥ 30/h LA ≤ 2 mmol/L |
| 12 | Hu BY et al, [12] | 2012 | SBP > 100 mmHg, Heart rate < 120 bpm, Urine output > 1 ml/kg/h Postoperative ScvO(2) <70% LA ≥4 mmol/L |
| 13 | Grey B, [13] | 2013 | MAP ≥ 60 mmHg, Heart rate < 110 bpm, Urine output > 1 ml/kg/h LA ≥ 2.5 mmol/L |
| 14 | Salottolo et al, [14] | 2013 | SBP ≥ 90 mmHg, Heart rate < 120 bpm, Urine output > 1 ml/kg/h Venous LA ≥2.5 mmol/L |
| 15 | Zakrison et al, [15] | 2013 | SBP > 100 mmHg, Heart rate < 110 bpm, Urine output > 1 ml/kg/h BD 4 ≥ mmol/L |
| 16 | Lokhandwala S et al, [16] | 2015 | SBP > 100 mmHg, Heart rate < 120 bpm, Urine output > 1 ml/kg/h LA ≥ 4 mmol/L |
| 17 | Moore KM, [17] | 2016 | SBP > 110 mmHg, Heart rate < 110 bpm, Urine output > 1 ml/kg/h LA ≥ 2.5 mmol/L |
| 18 | Reinhart K et al, [18] | 2017 | SBP > 100 mmHg, Heart rate < 120 bpm, Urine output > 1 ml/kg/h Postoperative ScvO(2) <70% LA between 2 to 4 mmol/L MAP>65 mmHg |
| 19 | Das S et al, [19] | 2018 | SBP > 110 mmHg, Heart rate < 100 bpm, Urine output > 1 ml/kg/h |
| 20 | Wentling J et al, [20] | 2019 | SBP > 100 mmHg, Heart rate < 120 bpm, Urine output > 1 ml/kg/h |

allows foreseeing future lesions of the bony, spinal, soft tissue, and vascular. In blunt trauma with no obvious skin penetration, this is especially important where transmitted energy can cause tissue injury considerable distance from the original impact site. The entrance wound can be disproportionately less than the real internal injury in penetration wounds. Knowing the biomechanics of an accident will then direct the clinician's conclusion about whether to order urgent full-body CT or regular X-ray fracture collection, which may also be unremarkable despite substantial tissue harm.

Hemorrhagic shock is characterized as 'acute circulatory collapse, due to significant blood loss, with insufficient tissue perfusion causing cellular hypoxia'.[30] Initially, patients with hemorrhaging injuries can have normal hemodynamic criteria, since a large decrease in systolic blood pressure is

stopped by circulatory action (tachycardia, vasoconstriction). This compensatory function, however, is constrained and hypotension occurs as a result of hemorrhage afterward. The main driver of hypoperfusion and fatal triads (acidosis, coagulopathy and hypothermia) is the combination of tissue inflammation, hypovolaemia and vascular disturbance [Figure 1]. Coagulation observed in the injured patients is considered a response to massive bleeding, associated with acidosis, massive fluid resuscitation, and hypothermia. Among them, coagulopathy is regarded as an independent prognostic factor assisting in damage control resuscitation (DCR). It is hard to recover simple physiological and immunological roles after the lethal triad emerges, even with treatment in the intensive care unit. Prompt initial resuscitation of the pre-intensive care unit should also aim to minimize systemic inflammatory response syndrome to mitigate collateral loss from tissue hypoxia. Minimized physiological insult helps in an optimized treatment path for the trauma patient and stronger efficiency. Occult hypoperfusion can be additionally perpetuated by 'extreme triad' of hypothermia, acidosis, and coagulopathy [Figure 1] that can lead to an unavoidable progression and imminent death until blood loss is halted and natural physiology is preserved.

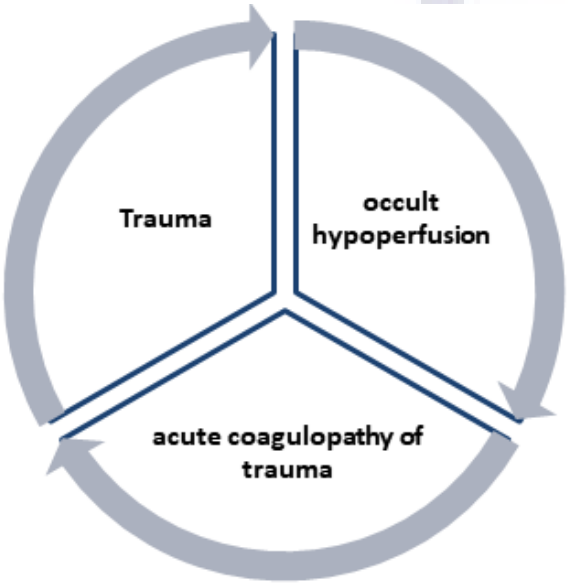


**Figure 1: Lethal triad of Trauma, occult hypoperfusion and acute coagulopathy of trauma**

Studies [Table 2&3] have shown that chronic OH is allied with elevated morbidity as well as mortality, and early correction tends to improve the final result.[3,4] Many studies have very well described the concept of OH in patients undergoing trauma.[5–7] It is shocking to understand that both the elder and young trauma patients are severely affected by occult

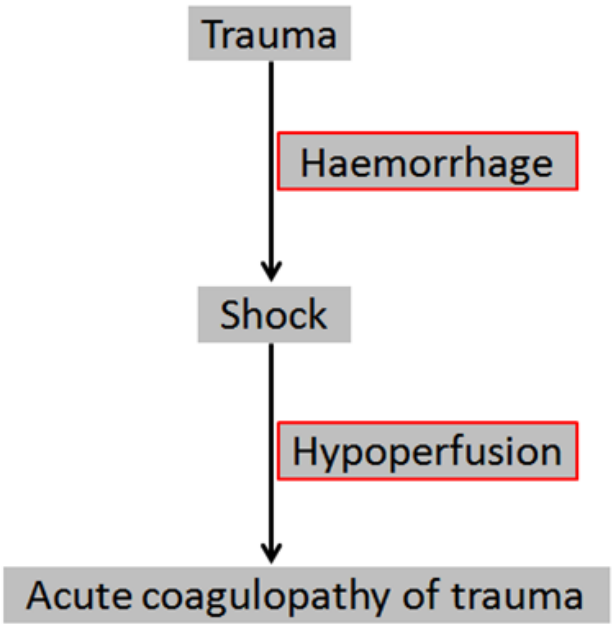


**Figure 2: Relations between trauma, shock and acute coagulopathy of trauma**

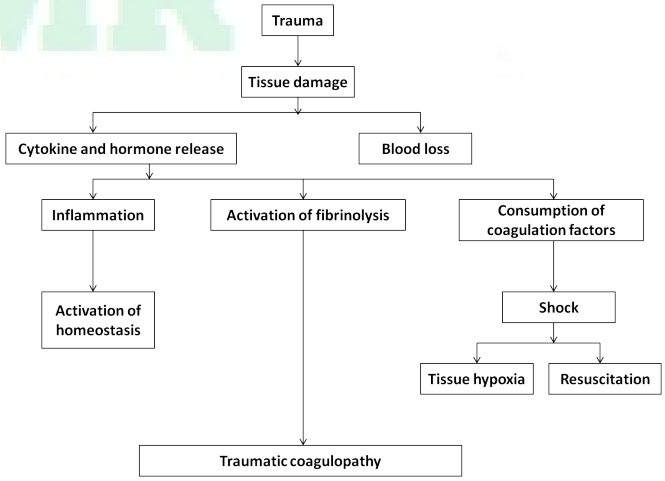


**Figure 3: Diagrammatic view of the factors that contribute to traumatic coagulopathy**

hypoperfusion.[31] Among both these categories of trauma patients, even when the vital signs achieved normalcy, there was a profound presence of oxygen debt and persistent cellular hypoperfusion. The existence of co-existing medical circumstances and the continuous use of drugs such as $\beta$-blockers can be one possible cause of this condition. In these groups of trauma patients, these variables can mask typical signs of shock.[32]

Occult hypoperfusion is correctly termed as the dynamic pathophysiologic state. Identification of this disease is important as an inability to detect and correct OS is related to a rise in both infectious and non-infectious difficulties and mortality. The occurrence of OH among patients with badly injured but hemodynamically stable trauma has been stated to range from 16 to 70%.[14] According to some studies, the traditional clinical endpoints of resuscitation (urine output, blood pressure, heart rate) are not foolproof to reveal the disparity between cellular oxygen delivery and cellular oxygen demand between critically ill patients.[33–35] In view of this, the objective of this review article is to summarize different approaches and methods elaborated in the management of OH syndrome in trauma patients.

**Management of Occult Hypoperfusion Syndrome in Trauma Patients by Following Methods:**

The detection of OH is exceptionally challenging since the suffering patient's central hemodynamic status cannot be resolute by analysis. Additionally, the basic definition of typical and major signs in the trauma patient is in consideration.[45] The list of several methods to manage the OH syndrome in trauma patients is as follows:

**Immediate control of bleeding post-trauma:**

Extreme trauma is a significant public health concern worldwide and is a major challenge for trauma survivors in terms of death and morbidity. It is worth mentioning that 10% of cases of mortalities around the world are due to traumatic injuries. It is due to this estimate that close to 6 million people die globally every year and this number is going to drastically increase to more than 8 million by the year 2020. The scope of traumatic injuries includes any minor isolated wounds to complex injuries. Such injuries may also include multiple organ systems. A systematic approach is essential for all trauma patients. This will help to manage not only maximum outcomes but also reduce the risk of undiscovered injuries. In the initial studies, the "golden hour" concept was given tremendous emphasis. These studies highlighted the elevated risk of death and therefore necessitated prompt intervention during the first hour of care post-major trauma.[46]

**Algorithm for management of massive blood loss due to trauma**

**Step 1:** Activate the hospital trauma team before the patient coming. This hospital trauma team must have a dedicated trauma team leader, along with a general surgeon and anesthesiologist.

↓

**Step 2:** Receive the patient in the emergency room. Establish IV access. Send blood for grouping studies. This step is important to guarantee that the specimens are accurately labeled. After labeling, they are to be hand-delivered to the blood bank

↓

**Step 3:** Start fluid resuscitation previous to more transport (Failure to respond to crystalloid and blood dictates the requirement for direct definitive intervention)

↓

**Step 4:** Assess injuries and prioritize treatment (aortic, head injury)

↓

**Step 5:** Ensure availability of specialists based on injuries (neurosurgeon, thoracic surgeon, obstetrician) and alert clinical lab, blood bank and hematologist.

↓

**Step 6:** If Bleeding is uncontrolled, then initial surgical intervention mandatory to stop bleeding. In most cases, bleeding does stop. Transfer to ICU to monitor for shock and continued bleeding, if any.

↓

**Step 7:** In a few cases, bleeding does not stop. Such cases warrant three immediate interventions.

**I. First Intervention** involves the maintenance of tissue perfusion and oxygenation. The objective of this intervention is restoring circulatory volume.

**II. Second Intervention** involves maintenance of Hb > 8gm/dl. The objective of this intervention is to assess the urgency of transfusion.

**III. Third Intervention** involves Coagulopathy. This intervention helps to keep patients warm (>35°C) and maintain Platelets > $75X10^9$/L and fibrinogen > 1.0 g/L.

2. **Tissue markers for establishing occult hypoperfusion :**

In critical illness, organ damage is because of insufficient oxygen delivery which eventually fails to fulfill metabolic requirements. Occult hypoperfusion plays a vital role to cause the resultant risk of multiple system organ failures. Of the many markers, the maximum usually used markers in the intensive care unit department are lactate and base deficit. These two markers serve as probable markers for the establishment and management of OH.

**2a:Blood lactate levels:**

**Table 2: List of published literature with the management of occult hypoperfusion syndrome in trauma patients**

| SN | Year | Author | Title | Inference |
|---|---|---|---|---|
| 1 | 2015 | Fordham A and Hudson A, [36] | Exploring the problem of occult hypoperfusion in the context of prehospital trauma | The result of this study reported that OH was recognized in 7% of patients who did not receive packed red blood cells in the pre-hospital phase. Systolic blood pressure seemed to have a significant effect on the doctors' option of whether or not to transfuse red blood cells packed in the pre-hospital process. This report proposed a prospective review to determine whether to use portable instruments capable of calculating serum BD and/or lactate to identify the tissue perfusion state of trauma patients in the pre-hospital process. This step might help to diagnose and treat all patients with hemorrhaging trauma more accurately, including patients with OH. |
| 2 | 2012 | Shere-Wolfe RF, Galvagno SM and Grissom TE, [37] | Critical care considerations in the management of trauma patient following primary resuscitation | This study reported that patients suffering from multiple trauma along with severe shock from considerable tissue injury and hemorrhage exhibit significant challenges towards management and resuscitation in the ICU. |
| 3 | 2006 | Dutton RP, [38] | Fluid management for trauma; where are we now? | This research showed that future attempts to improve hemorrhagic shock outcomes would need to focus on faster diagnosis and bleeding control (as with recombinant factor VIIa or different topical hemostatic agents) and improved management of the condition of shock. |
| 4 | 2007 | Ganapathy N, [35] | End Points in Trauma Management | This research stated that fluid resuscitation is an essential, essential part of traumatic hemorrhage shock patient care. It also indicated that the function of the anesthesiologist and intensivist is to recognize the effect of shock during traumatic hemorrhage and to resuscitate the patient with the necessary fluid, in the correct quantity, at the right moment. |
| 5 | 2013 | Bridges and Beilman, Gregory, [39] | OH in seriously injured combat casualties Pre/Post Medevac transport | This research concludes that there was a high frequency of hypoperfusion in critically wounded battle victims at POI, post-transport from the field and prior to evacuation from Position II. Oxygen saturation (StO2) of skeletal tissue (muscle) < % observed OH in these patients, and StO2 > % also indicated hypoperfusion. Hypoperfusion did not rule out a standard StO2 (75%-90%). A single irregular StO2 (< 75% or > 90%) suggests the need for patients to be reassessed and can be useful as the patient progresses down the spectrum of treatment. |

Whenever the distribution of oxygen is inadequate to sustain normal oxygenation in the skin, blood lactate levels continue to increase under both laboratory and clinical conditions. In chronically ill patients, the levels of blood lactate are closely linked to the results. As the outcome of anaerobic digestion, in hypoperfusion states, lactate levels are observed to be elevated. This is how it is converted to lactate as the pyruvic acid is unable to join the Krebs cycle due to inadequate oxygen supply. In OH, the condition is not unusual where the development lactate reaches its metabolism limit, which eventually allows the blood lactate levels to increase. Similarly, the cells are deprived of oxygen in the state of

**Table 3: List of published literature with the management of occult hypoperfusion syndrome in trauma patients**

| SN | Year | Author | Title | Inference |
|---|---|---|---|---|
| 6 | 2003 | Victorino GP, Chong TJ and Pal JD, [40] | Trauma in an elderly patient | This particular article stated that the treatment of trauma-related accidents would involve an increasingly growing number of elderly patients. It also indicated that the age positions a trauma patient in a higher-risk group, and it is recognized that elderly patients with major trauma have higher injury and death rates than their younger complements. In trauma victims, aged age is a potentially dangerous factor for adverse results. Aggressive treatment, comprehensive supervision and full care delivered by an advanced health-care trauma staff will yield better results in elderly trauma patients. |
| 7 | 2016 | Mitchell C, [41] | Tissue oxygenation monitoring as a guide for trauma resuscitation | Hypoperfusion is the most common occurrence reported during cardiac resuscitation prior to the onset of multiple organ dysfunction syndromes. To ensure sufficient tissue oxygenation and perfusion, identifying small variations in perfusion is important. Blood pressure scales, urine intake, heart rate, lactate serum levels, mixed venous oxygen saturation, and central venous oxygen saturation are standard ways of monitoring physiological changes. Continuous non-invasive testing of muscle tissue oxygen saturation has the ability to signal shock intensity, diagnose OH, direct resuscitation, and anticipate the need for multiple organ failure syndrome prevention therapies. In emergency centers, ambulance centers, operating rooms, and emergency medical facilities, tissue oxygen saturation is required. Technology for tissue oxygen saturation is almost as effective as it of mixed venous oxygen, core venous oxygen saturation, and serum lactate. |
| 8 | 2017 | Yeslawath ND, [42] | Primary recognition with renal Doppler resistive index measurements of hemorrhagic shock in patients with polytrauma | This study proved that Renal Doppler RI measurement may represent a clinically useful non-invasive technique for early detection of occult hemorrhagic shock. |
| 9 | 2007 | Antonelli M et al, [43] | Hemodynamic monitoring in shock and implications for management | This consensus paper from the $8^{th}$ International consensus conference on intensive care medicine recommended that the concept of shock should not require hypotension and, as a consequence, the occurrence of insufficient tissue perfusion on physical inspection is of significance. The only biomarker proposed for diagnosis or staging shock as blood lactate, considering the existing data. This paper also planned that (1) the pulmonary artery catheter in shock and (2) static preload tests used alone to assess fluid awareness are not regularly used. |
| 10 | 2013 | Kaczynski J, [44] | Prevention of tissue hypoperfusion in the trauma patient: primary administration | This article summarized the challenges involved in f identification and management of tissue hypoperfusion due to hemorrhagic shock in the civilian polytrauma cases. |

hypovolaemic shock, resulting in anaerobic respiration. This mechanism contributes to the production of lactic acid. In lieu of normal vital signs, elevated blood lactate levels (OH) often serve as stronger indicators of morbidity and mortality in patients with surgery and trauma. In addition, serum lactate levels often help determine lethality in the initial period following high-risk surgery.[4] In addition, elevated blood lactate levels in many forms of shock were closely interrelated with mortality. This argument has been backed by many studies documenting that lactate levels have been the most common marker that helps detect OH. And, to be responsible for OH, experiments have established the minimum value of 2.5 mmol / L. This discovery from various studies helps to define the concept of OH as the disorder of serum lactate levels greater than 2.5 mmol / L in presence of usual major signs. Few studies have recommended the existence of supernatural hypoperfusion in patients with clinically intact trauma. These findings suggest that OH was observed to be linked with a non-significant improvement in the duration of stay in the hospital, but caused a fourfold rise in the risk of mortality. In spite of this, the screening of wound patients with serum lactate for OH is of utmost importance. Few reports have indicated that trauma patients who have experienced surgical fracture fixation in the occurrence of OH need further ventilation in the state of surgical fracture fixation. And, they were at an elevated risk of risks of inflammation and respiration. Therefore, some physicians recommend that fracture fixation should be postponed wherever possible before OH is fixed. Some reports suggest that lactate output values are higher than most clinical indicators of shock or organ failure.[47–52] Calculation of diuretics, heart rate, and mean arterial pressure or metabolic acidosis indexes, is superior. Data from some research indicates that lactic acid removal is closely associated with the increased recovery and better action of patients with wounds within the first 24 hours following injury. Similarly, measuring lactic acid helps to guide the resuscitation process. Despite its clear association with OH, it is often important to view the fluctuation of lactic acid levels within the therapeutic sense for many reasons. The elevated levels of lactic acid can be triggered, first and foremost, by previous injury or increased physical activity (exercise). Whereas, on the other hand, in a patient with a highly traumatized ischaemic leg, decreased or normal lactic acid levels can be found where the lack of tissue perfusion can give a falsely low outcome. Finally, owing to continuing resuscitation, lactate levels will also increase, leading to the return of occult perfusion.

**2b. Base deficit:**

The values of the base deficit reveal an indirect assessment of total tissue acidosis because of occult impaired perfusion. In the early studies, many researchers used the initial base deficit as a single independent predictor of post-traumatic mortality within the first 24 hours. In fact, in patients suffering from hemorrhagic shock, the base deficit is a superior predictor of mortality over pH for the measurement of arterial blood gas. Nevertheless, as in the case of lactate, the efficacy of the base deficit in hemodynamically stable patients is not yet completely recognized. Some contradictory studies have also reported that the elevated initial levels of lactate and base deficit do not correlate with tissue hypoxia. And, in fact, both the levels of blood lactate and base deficit are influenced by several non-hypoxic reasons for metabolic acidosis. Traditionally, the resuscitation of surgical patients has been conducted by normalization of major signs. These vital signs include urine output, heart rate, and blood pressure. Along with hemodynamic instability and with this in the background, invasive monitoring becomes essential.

3. **Scoring systems:**

Some studies recognize that the identification of hemorrhagic shock may not be an easy task. This is because, without drastic improvements in hemodynamic parameters, young adults are capable of tolerating substantial blood loss. Approximately 44 percent of trauma patients may have relative bradycardia (heart rate < 90 / min after trauma-related hypotension (SBP < 90 mmHg). The research was conducted around a decade ago by Ley et al stated.[53] Because of this, the situation demanded different scoring systems, and hence, several scoring systems have been developed. These scoring systems can combine different parameters, which eventually assist with definite identification and appropriate management of hemorrhaging trauma patients. Out of many scores, shock index (SI), rate overpressure evaluation and assessment of blood consumption score have found profound significance. The Shock index which was first described in 1967 by Allgower is widely acknowledged as a viable option to both the systolic blood pressure and blood pulse. In the literature, there have been several studies that investigated the importance of shock index as a hemodynamic marker to predict outcomes of trauma. In fact, the shock index is the most suitable index to predict trauma mortality. The shock index is a mathematical quotient resulting from the heart rate divided by the SBP. Therefore, it is considered an indicator of hemodynamic instability; therefore it also helps as an indicator of systemic oxygenation, increased likelihood of death, and cardiac function. The normal range of shock index falls within the range of 0.5 to 0.7. Aftershock index score, the rate overpressure evaluation (ROPE) scores another score to manage OH. Dividing heart rate by pulse pressure (heart rate / systolic blood pressure-diastolic blood pressure) determines this ROPE ranking. An extensive paper has the most appropriately described ROPE evaluation. In this paper, around 200 trauma patients with a level of 3.0 or greater ROPE score have been identified with predictive for developing decompensated shock. This study helped to establish that the ROPE score of less than 3.0 can be being predictive of the patient remaining

well and ROPE of above 3.0 being predictive of patient evolving decompensated shock.[54] This score also predicts the development of hypovolaemic shock. Finally, the blood intake (ABC) level measurement is the greatest indicator of a massive prerequisite for transfusion. The following parameters are used for this score: penetrating pathways, positive Quick scan, systolic blood pressure, 120/minute.

4. **Other methods to detect hypoperfusion :**

It has been examined, such as the Lenox polarographic tissue oxygen monitor. The Lenox microcatheters have been approved by USFDA towards their use in measuring muscle tissue and brain tissue oxygenation. The case in the trauma patient is a bit different because monitoring the subcutaneous tissue is cumbersome due to the changes in perfusion induced by pain, hypothermia, and peripheral edema. All these three are hallmarks of a patient undergoing trauma. Furthermore, Licox polarographic tissue oxygen monitor actions partial pressure of oxygen within the deltoid muscle in addition to renal and splenic doppler resistive indices. Both these catalogs are advised to be explored further as futuristic indicators for the management of OH.[55]

5. **Focused assessments with sonography for trauma (FAST) scans:**

FAST scans used pre-hospital phase have evolved into the non-invasive, rapid, and repeatable examination for the management of OH. These scans have the definite advantage that these can be applied to any main body section, counting chest, pericardium, pelvis and abdomen. In spite of several advantages offered by these FAST scans, they suffer from some limitations. FAST scans lack sensitivity for the site of blood loss. Further, it fails to provide any information on retroperitoneal structures, it is specific for the presence of free fluid, and it is highly dependent upon operator skills. More significantly, when used in conjunction with standard trauma series X-rays, FAST scans mostly fail to recognize underlying injuries. This failure can principal to delay in the distribution of definitive treatment.

6. **Multi-detector computed tomography :**

In the initial management of trauma patients, identification of OH plays a very important role. Analysis of the inferior vena cava (IVC) ratio on computed tomography (CT) scan has revealed tremendous potential in predicting the intravascular volume. In the major trauma victims, a flat inferior vena cava ratio on a CT scan has proved to be an accurate marker for OH. This marker also helps to identify patients who need more belligerent resuscitation, checking, and provision.

**Advanced trauma life support (ALTS grading system):**

Advanced Trauma Life Support (ATLS) is a protocol-based system useful for standardizing early evaluation and administration of injured patients. ALTS also helps to evade the omission of potentially life-saving interventions. Several studies have reported the influence of advanced trauma life provision on lowering cases of mortality and morbidity after. One of the several big challenges towards the management of the injured patients is the recognization of shock. This shock is identified within the period of early management of the injured patient by evidence of end-organ hypoperfusion.

**Continuous glucose monitoring (CGM):**

Originally, a continuous glucose monitoring (CGM) method was established specifically for diabetic patients. Additionally, this method is also a beneficial implement for monitoring glucose changes in a pediatric intensive care unit (PICU). Many studies have worked earlier to identify any correlation between OH and accuracy of continuous glucose monitoring (CGM) with laboratory markers. It is important to consider that the correctness of continuous glucose monitoring (CGM) measurements is not dependent on laboratory parameters that are applicable to tissue hypoperfusion.

**Central venous oxygen saturation ($ScvO_2$ and central venous pressure (CVP):**

Around two decades ago, a study was undertaken by Rady et al.[57] confirmed the presence of OH in their analysis. By showing the drop of central venous oxygen saturation ($ScvO_2$), this analysis demonstrated the presence of OH. It is significant to recognize central venous pressure so as to understand the central venous saturation of oxygen. A calculation of the pressure in the vena cava is central venous pressure. As an assessment of right atrial and preload strain, this pressure control may be used. This central venous pressure is also used, especially in the intensive care unit, as an indicator of hemodynamic status. In order to determine central venous pressure, the standard procedure requires the aid of a central venous catheter brought forward via the inner jugular vein and then positioned near the right atrium in superior vena cava. Between 8 mmHg and 12 mmHg, a standard central venous pressure reading is assessed. The elevated levels of central venous pressure reflect the possibility of fluid retention and/or myocardial contractile dysfunction. On the contrary, the dropped levels of central venous pressure are reflected in decreased venous tone or volume depletion. The range of values of central venous oxygen saturation ($ScvO_2$)may suggest a lower rate of oxygen extraction which is secondary to mitochondrial impairment. Overall, this is indicative of deprived outcomes and effectively bounds the lower levels of central venous oxygen saturation ($ScvO_2$) sensibility. Studies have reported a positive correlation between postoperative complications (anastomotic shock) and hypoperfusion.[58] For instance, in the severe condition of abdominal trauma, an anastomotic leak is considered as the best morbid complications. And, hypoperfusion is considered a significant factor for its development. In the case of identifying the risk of anastomotic

**Table 4:** Signs and symptoms of hemorrhage by grade.[56]

| Parameter | Class I | Class II (Mild) | Class III (Moderate) | Class IV (Severe) |
|---|---|---|---|---|
| Approximate blood loss | <15% | 15 to 30% | 31 to 40% | > 40% |
| Heart rate | <15% | 15 to 30% | 31 to 40% | > 40% |
| Blood pressure | <15% | 15 to 30% | 31 to 40% | < 40% |
| Pulse pressure | <15% | 15 to 30% | 31 to 40% | < 40% |
| Respiratory rate | <15% | 15 to 30% | 31 to 40% | > 40% |
| Urine output | <15% | 15 to 30% | 31 to 40% | < 40% |
| Glassgow Coma scale score | <15% | 15 to 30% | 31 to 40% | < 40% |
| Base deficit | 0 to 2 mEq/L | 2 to 6 mEq/L | 6 to 10 mEq/L | >10 mEq/L |

leak development, a parameter named as the central venous oxygen saturation ($ScvO_2$) serves as a vital hypoperfusion marker. It has been reported that the phenomenon of OH is the consequence of circulatory disturbances during hemorrhagic and hypoxic periods. Further, some evidence suggests that central venous oxygen saturation exposes the initial and early stages of hypoperfusion. Additionally, the central venous oxygen saturation ($ScvO_2$) acts as a timely hypoperfusion marker. This attribute benefits in early support and serves the objective during early decision-making of the surgical procedure. However, in some patients in whom the indices of hypoperfusion may not be generated, this method is not optional since this approach could lead to excessive fluid management.[59]

## Summary

The occurrence of OH is considerably high after cardiac surgery. These patients have a lengthy ICU course in the hospital, and they require more services in the hospital. The quest for the perfect marker for early OH diagnosis continues. Several clinicians have agreed that conventional markers like blood pressure and urine output fail to assist in identifying OH. In this situation, it becomes imperative to identify a dependable indicator of OH. The explanation for this study is that the bulk of deaths are due to multiple organ failure within an ICU, which is an end result of chronic hypoperfusion. The most favorable marker to identify OH shall include several sought after attributes. This includes reproducibility in a wide variety of OH states, rapidity of acquisition accuracy, simple, uniform and reliable results among the sample specimens. This current article reported that calculating blood lactate concentrations in the early phase of high-risk surgery is accurate in predicting lethality. In conjunction with other hemodynamic factors, blood lactate levels are quickly collected and measured efficiently and accurately. This is feasible even before the introduction of any invasive monitoring in the concerned patient. However, the early measurements of blood (serum) lactate concentrations face some inherent disadvantages. These disadvantages become more profound more precisely in the septic conditions when the metabolic circumstances can be complicated. Washing of stored lactate in tissues, extracorporeal renal assistance with hemofiltration, enhanced aerobic development and altered clearance in the liver and other organs are included in the list of complicated metabolic disorders. The biochemical markers (lactate and base deficit), which measure the OH are considered to be more specific and trustworthy as compared to the traditional pre-hospital methods. Therefore, we suggest that biochemical testing stands out as the most viable method to manage OH.

## Conclusion

In recent times, it has become of paramount importance to identify patients with OH. This identification is necessary to minimize morbidity and mortality. While laboratory tests such as measuring lactic acid help to define OH and revival recommendations, previous prognostic indicators of OH are necessary in order to introduce earlier and additional effective surveillance and resuscitation in patients at risk. Future perspectives: In order to determine if the detection and alteration of hyperlactatemia can effective in falling morbidity and mortality in a healthy surgical population, further studies are needed. Larger, multicenter trials would also aid comprehensively assess the treatment of ritual hypoperfusion in patients with trauma.

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**How to cite this article:** Prasad G V K. Management of Occult Hypoperfusion Syndrome in Trauma Patients: A Narrative Review. Asian J. Med. Res. 2020;9(3):1-12.

DOI: dx.doi.org/10.47009/ajmr.2020.9.3.AN1

**Source of Support:** Nil, **Conflict of Interest:** None declared.