

Metabolic response to maxillofacial trauma revisited: A retrospective study

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ABSTRACT

Purpose: Management of a trauma patient aims at stabilization or resuscitation and revival from critical condition resulting from various sequences of systemic pathophysiological responses in the body. Hematological changes are the first signs reflecting the homeostasis mechanisms starting in the body after injury. The aim of the current study is to evaluate the physiological changes following maxillofacial trauma and extrapolate the findings to understand the posttrauma responses. **Patients and Method:** This is a retrospective study involving 192 subjects divided into two groups, trauma group and control group. In both the groups, baseline vitals and complete blood picture were recorded for comparison. In trauma group, the recordings were made within 24 h after maxillofacial injury. **Results:** All the parameters were analyzed using SPSS version 18. Independent sample *t*-test was used to assess the nature of data distribution and statistical significance was considered only at *P* value < 0.05. On comparison of complete blood picture mean values of hemoglobin (13.63 vs 12.18), RBC count (4.51 vs 4.10), WBC count (8835.48 vs 8336.56) were seen to be higher in trauma patients compared to control subjects. The mean bleeding times are almost equal (2.35 vs 2.47) but the clotting times (5.42 vs 5.26), random blood glucose (94.78 vs 90.13), and blood urea (27.14 vs 26.30) were marginally higher in trauma group but were statistically insignificant. The mean value of serum creatinine (0.84 vs 0.80) was comparatively higher in trauma patients and was statistically significant. Study of vitals revealed that mean systolic blood pressures were almost equal (120.65 vs 121.08) in both the groups. The mean diastolic blood pressures (79.46 vs 88.49) and oxygen saturation (93.73 vs 98.86) in trauma patients are comparatively reduced. The mean values of temperature (99.30 vs 98.50) and pulse rate (102.38 vs 97.14) were on relatively higher side in trauma group compared with control group. **Summary and Conclusion:** Using basic blood parameters and vitals in the present study, the compensatory mechanisms happening in the body after maxillofacial trauma can be seen. These changes although significant on side by side comparison can still fall within the normal physiological range provided by various diagnostic setups. Hence, the need for maxillofacial surgeon to be sensitive to minor variations in these aspects to ensure safety of the patient cannot be overemphasized.

Keywords: Baseline vitals, complete blood picture, maxillofacial trauma, metabolic response

Introduction

In management of a trauma, there is patient need to focus on initial stabilization, resuscitation, and revival from critical

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Received: 20-09-2019

Revised: 22-09-2019

Accepted: 23-09-2019

Published: 15-11-2019

condition resulting from various sequences of systemic physiological or pathological responses to injury in the body. In 1942, Cuthbertson^[1,2] was the first to describe distinct phases of the metabolic changes which occur after major trauma and characterized the “ebb” and the “flow” of posttraumatic metabolic alterations. Since then many authors have tried to understand the complex mechanisms of the physiology after injury and tried to redefine the stress response.

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How to cite this article: Kuntamukkula VK, Sinha R, Tiwari PK, Bhogavaram B, Subramaniam H, Kumar BV, et al. Metabolic response to maxillofacial trauma revisited: A retrospective study. J Family Med Prim Care 2019;8:3713-7.

Access this article online

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DOI:
10.4103/jfmpc.jfmpc_798_19

Major trauma induces marked metabolic changes which contribute to the systemic immune suppression in severely injured patients and increase the risk of infection and posttraumatic organ failure. The hypercatabolic state of polytrauma patients includes nutritional compensations like protein and fat catabolism's, cardiac compensations aiming at tissue perfusions like tachycardia, renal compensations like fluid and electrolyte conservation, endocrinal compensations like hyperglycemic state, etc., The cause of the failure of these above-mentioned mechanisms is failure to quantify the extent of the injury, resuscitations needed to support various systems in maintaining homeostatic environment.

Metabolic response after maxillofacial facial trauma patients is mostly conceptualized from poly-trauma patients but the involvement of the vital structures like brain can drastically alter the response. For example, in traumatic brain injury, there is a profound nutritional alteration and has a major impact on nitrogen metabolism and on intestinal trophicity.^[3]

The adaptive states after major maxillofacial injuries (soft tissue or bony injuries) are characterized by starvation, immobilization, and repair. The problematic areas include oronasal structures that impair nutrition and oxygenation, contaminated wounds and risk of infections due to mixed flora (oral/nasal) and also trying to achieve cosmetic and functional reconstruction that may need other surgical insults.

The hypothesis in this study is that metabolic responses after maxillofacial trauma should be reflected in parameters like complete blood picture (CBP), baseline vitals (blood pressure, pulse, temperature, oxygen saturation) although the values are within normal ranges. The impact of the subclinical signs like hemodilution, glomerular filtration rate, protein catabolism, glycogenolysis, and gluconeogenesis may have greater impact on the posttrauma recovery. During all these phases, various cellular elements and tissue level responses orchestrate a clinical presentation that is quite an interesting area of the study.

The purpose of this study is to compare the vitals and CBP of maxillofacial trauma patients obtained within 24 h after injury with control subjects to try and assess the reliability and importance of initial monitoring and resuscitation of maxillofacial trauma patients.

Patients and Methods

This is a retrospective study conducted at the Department of Oral and Maxillofacial Surgery between 2012 and 2016 at Sri Sai College of Dental Surgery, Vikarabad with IEC No. 545/SSCDS/SS/IRB/2017. The study sample consisted of 192 subjects in two groups of 96 each. Group A was the maxillofacial trauma Group while Group B was the control group including patients undergoing elective surgeries. All patients included in the study were between 20 and 40 years of age, included both the genders and also moderately nourished. The severities of

the maxillofacial injury are scored using FISS scoring systems given by Bagheri *et al.*^[4] All the patients did not have poly-trauma, head injuries, or fractures involving other regions of the body. Similarly in both the groups, patients with comorbid conditions that could affect the stress response or the normal values of blood investigations were excluded from the study. The study was approved by the institutional ethics committee and the patients were given detailed information about the study, following which written informed consent was obtained for the blood withdrawal as well as usage of patient data for publication.

Data regarding baseline vitals were obtained using CritikonDinamap Pro 1000 (Critikon Marketing Services, 4502, Woodland Corporate Boulevard, Tampa, FL 33614) monitor that included blood pressure (systolic/diastolic mm of Hg), pulse (beats per minute), oxygen saturation SpO₂ (percentage), and temperature (Fahrenheit). Complete blood picture included hemoglobin (g/dL), RBC count (million/mm³), WBC count (number/mm³), bleeding time, and clotting time (min/s), random blood glucose (mg/dL), blood urea (mg/dL), and serum creatinine (mg/dL) were obtained by standard diagnostic investigation methods used in the institutional diagnostic lab.

Statistical analysis was performed by using the SPSS 18 software version. Independent sample *t*-test was used to test the nature of data distribution and statistical significance was considered only at *P* value <0.05.

Results

The study involved 192 patients between the ages of 20 and 40 years (mean = 34 years). The sample consisted of 135 males (70%) against 57 females (30%). As show in Table 1, complete blood picture values are as follows: mean value of hemoglobin (13.63 vs 12.18), RBC count (4.51 vs 4.10), WBC count (8835.48 vs 8336.56) in trauma patients are comparatively higher than in normal subjects and also statistically significant (*P* < 0.001, *P* < 0.001, *P* = 0.002, respectively). The mean value of bleeding time are almost equal (2.35 vs 2.47) in both the groups and statistically insignificant (*P* = 0.236). The mean values of clotting times (5.42 vs 5.26), random blood glucose (94.78 vs 90.13), and blood urea (27.14 vs 26.30) although marginally higher in trauma patients than normal subjects but

Table 1: Statistical analysis showing CBP between trauma patients and normal subjects

	Trauma		Normal		<i>P</i>
	Mean	SD	Mean	SD	
Hb (g/dL)	13.63	1.81	12.18	2.02	<0.001
RBC count (million/mm ³)	4.51	0.62	4.10	0.71	<0.001
WBC count (number/mm ³)	8835.48	1,137.18	8,336.56	1,065.14	0.002
BT (min. sec)	2.35	0.58	2.47	0.74	0.236
CT (min. sec)	5.42	0.63	5.26	0.95	0.182
RBS (mg/dL)	94.78	21.92	90.13	17.54	0.111
BU (mg/dL)	27.14	5.27	26.30	6.17	0.318
SC (mg/dL)	0.84	0.14	0.80	0.15	0.029

statistically not significant ($P = 0.182$, $P = 0.111$, $P = 0.318$, respectively). The mean value of serum creatinine (0.84 vs 0.80) was comparatively higher in trauma patients and was statistically significant ($P = 0.029$).

As show in Table 2, mean systolic blood pressure values are almost equal (120.65 vs 121.08) in both the groups and also statistically not significant ($P = 0.819$). The mean values of diastolic blood pressure (79.46 vs 88.49), oxygen saturation (93.73 vs 98.86) in trauma patients are comparatively reduced when compared with normal subjects and also statistically significant ($P < 0.001$, $P < 0.001$, respectively). The mean values of temperature (99.30 vs 98.50), pulse rate (102.38 vs 97.14) in trauma patients are comparatively on higher side when compared to normal subjects and also statistically significant ($P < 0.001$, $P < 0.001$, respectively).

Discussion

What defines maxillofacial injuries? The answer dates back to 1947 when Lt Col Daniel Klein^[5] explained the severity of the resulting cosmetic defects and also miraculous rapidity with which they heal. Maxillofacial region confines the hard and soft tissue components of middle and lower thirds of the face. There is trend of increased maxillofacial injuries due to motor vehicle accidents, interpersonal violence, or sports-related activities, etc.

According to the famous statement by John Hunter,^[6] the body's responses to injury were defensive and had survival value. These survival tactics include complex events involving local and systemic effects trying to preserve homeostasis at tissue and organ levels. These responses are modulated by autonomic nervous system, hormones and inflammatory mediators based on severity of injury, comorbid conditions, and nutritional status. Clinicians can assess the physiological state of the patient and the prognosis based on various scientific parameters. Some parameters of the blood counts, for example, WBC count and hemoglobin/hematocrit can be used as predictors of mortality or rehospitalizations.^[7] In this study, it was hypothesized that metabolic responses after maxillofacial trauma can be reflected in CBP which might still be within normal limits for maxillofacial trauma patient.

According to the present study, there was a transient increase in hemoglobin and RBC count within 24 h after injury. This can be explained by the reduction in blood volume leading to

hemo-concentration, leading to sequestration of fluid from venous system. Another compensatory mechanism undertaken by renal system is release of erythropoietin which causes release of premature erythrocytes (reticulocytes) into circulation to maintain tissue oxygenation. These results are in agreement with the guidelines given by Donat R Spahn *et al.*^[7] The author stated that the above mechanism allows for the maintenance of tissue oxygenation in the immediate posttraumatic phase despite the blood loss.

Bauer AR^[8] in their study observed marked reduction in WBC counts by injury day 1 in mild to moderate injuries but returned to normal within approximately 5 days. Similarly, Claudia A. Santucci *et al.*^[9] also identified elevation in WBC counts in blunt trauma patients even with minimal initial signs of severe injury. In the present study in agreement also identified significant leukocytosis within 24 h after maxillofacial trauma. This can be explained by activation of body defense in repair and also the mild extent of the maxillofacial injury that may not show significant initial lymphocytopenia. On the contrary, the injuries in the maxillofacial region mostly are contaminated wounds with mixed aerobic and anaerobic flora which can trigger the spontaneous immune response. A watchful eye needed to be kept on any signs of septic shock like states during evaluation of severity of injury.

Wenjun Zhou Martini^[10] identified reduced fibrinogen levels due to increased fibrinogen breakdown during hemorrhage and also because of dilatational coagulopathy (vascular stasis) due to administration of crystalloids or colloids for maintaining systolic blood pressure for tissue perfusion. On the contrary, bleeding time and clotting time in trauma patients is almost equal with the control group in our study. This may be explained by the minor extent of maxillofacial injury, moderate severity of the injury, and also relatively lesser hemorrhage. These factors might not cause such marked changes in the above-mentioned standard tests (B.T, C.T) but definite evaluation methods like activated thromboplastin time, prothrombin time may give us the clearer picture.

Duane *et al.*^[11] stated that blood glucose levels at the time of admission and also at 24 h after trauma did not correlate with outcome, particularly if the patient is adequately resuscitated with a normal lactate. Similar study conducted by Asimos AW *et al.*^[12] stated that blood values of Na⁺, Cl⁻, K⁺, and blood urea nitrogen levels in emergency department did not influence the initial management of major trauma patients. In agreement with the present study, mild elevation of the blood glucose levels and blood urea levels although was observed in trauma group, it did not influence the decision making for surgery during preanesthetic evaluation as well as the postoperative recovery because of adequate fluid resuscitation.

In the present study, significant elevation of serum creatinine levels in trauma groups was observed when compared to normal subjects. The body has limited carbohydrate reserves in the

Table 2: Statistical analysis showing baseline vitals between trauma patients & normal subjects

	Trauma		Normal		P
	Mean	SD	Mean	SD	
Systole (mm/Hg)	120.65	15.24	121.08	9.83	0.819
Diastole (mm/Hg)	79.46	8.52	88.49	8.72	<0.001
SPO ₂ (%)	93.73	2.20	98.86	1.08	<0.001
Temperature (°F)	99.30	0.82	98.50	0.10	<0.001
Pulse (beats/min)	102.38	4.01	97.14	1.25	<0.001

body^[13] and no storage deposits of protein. This lines up with the fact that any starvation after injury leads to loss of skeletal muscle mass (proteolysis) that eventually leads to negative nitrogen balance and a sepsis like state. Similar findings are observed in the study by Minville V *et al.*^[14] who suggested that poly-trauma patients required constant monitoring of the creatinine clearance for adjusting the medication dose regimes, especially for drugs with renal elimination.

Perry M *et al.*^[15] in his study debated about the optimal blood pressure, fluid administration, and role of surgical intervention in the actively bleeding patient. Maintaining the systolic blood pressure above 100 mmHg maintains optimal tissue perfusion and also emphasized that following ATLS management protocols prevents the lethal triad (acidosis, coagulopathy, and hypothermia) stepping in during resuscitation phase. In agreement with the present study, systolic blood pressure is maintained without much variation between the two groups. The sequestration of blood from venous system, action of catecholamines, and also marginal bleeding occurring in the test group explains the minimal effect on systemic circulation.

The changes in the diastolic pressure are statistically significant and also reduced in the test group. This reduction in diastolic pressures may also be compensation by fluid diversion from central circulation. This supports the fact that during posttrauma phase and also surgery phase, coronary perfusion to myocardium is altered but adequate levels are maintained at the extent of reduction in circulating blood to less critical organs and diverting them to more vital organs (brain, myocardium, etc.) circulation except in critically ill patients. There is a need for advanced diagnostic like echocardiogram to check the ejection fraction to evaluate the cardiac fill. The fluid resuscitation regimes and also choice of the after injury as well as during surgery is critical to maintain optimal fluid resuscitation with isotonic solutions.

In the present study, tissue oxygen saturation levels are low in test group when compared to control group. Oxygen delivery is dependent on oxygen availability, the ability of arterial blood to transport oxygen, and tissue perfusion. The standard values of partial pressures of oxygen in arterial system is >79 torr which is corresponding to >94% oxygen saturations on pulse-oxymeter and similarly venous partial pressure of oxygen is 30--40 torr that corresponds to 75% oxygen saturation.^[16] This difference in the pressures regulates gaseous flow and tissue oxygenation. Tissue hypoperfusion occurring in shock results in failure to meet the metabolic demands of various systems and development of multiorgan dysfunction syndrome. The findings are similar to the study conducted by Mitchell C^[17] which emphasized that the continuous noninvasive monitoring of tissue oxygen saturation has the potential to indicate severity of shock, detect occult hypoperfusion, and also act as guide for trauma resuscitation.

In a study conducted by Hsieh TM *et al.*^[18] revealed that low body temperature is associated with the mortality outcome and

also hypothermia being the third of the lethal triad in trauma. Fortunately in the present study, the study group had mild but significant elevation in body temperature (99.30°F) when compared to control group (98.5°F). This can be explained by pyrogen-induced release of prostaglandin E2 (PGE2) and their effect on hypothalamus. Hypovolemia although mild to moderate in the present study may have initially lead to hypothermia but because of the compensatory mechanisms in restoring the blood volume lead to mild elevated temperatures and also the primary care undertaken may also influence this outcomes.

The modulatory effect of elevated body temperature on adaptive immunity was studied by Appenheimer MM.^[19] The author hypothesized that elevated body temperatures caused enhanced rate of activation, function, and differentiation of WBCs. In agreement with the above-mentioned study, mild elevation of WBC counts was observed with elevated body temperature in trauma patients when compared to control group.

In the present study, tachycardia (102 Bpm) was identified in trauma group when compared to control group which can be explained by the post-hemorrhagic reduction in blood volume. The main compensations being the adrenalin mediated and also baro receptor stimulation of myocardium to maintain the circulatory mechanisms. Similar findings were identified in a study conducted by Reisner AT,^[20] who observed that trauma patients with hemorrhage are continuously tachycardic while others have a normal heart rate.

Primary medical care and primary healthcare are very important in trauma. A primary healthcare individual provides all care delivered at the point of first contact to a trauma patient. Understanding the metabolic response of traumatized patient is a crucial phenomenon due to blood loss and other causative etiologic factors which alters the metabolic response. It is very important to deliver essential and comprehensive healthcare in an integrated manner to the patient at the point of first contact, which help the access to integrated curative, preventive and health. Regardless of the intervention chosen, the family and patient can be reassured by a family healthcare professional that the medical service can be an ongoing source of support for them as they heal from the traumatic event(s) both physically and psychologically which will increase the quality of life of patient as well as his family.

The authors recognize some limitations of the study in its current form. Chief among those is that we cannot rule out any pre-existing variations in the baseline values of the parameters. The influence of socioeconomic status, gender differences that directly influence nutritional status (although all the patients are moderately nourished), the stress response could show some amount of interpersonal variations in the blood parameters although all are obtained from single diagnostic setup. Another point to ponder is that the study was conducted including basic blood parameters and baseline vitals based on the rural setup of the hospital and affordability of the population. We may

understand complex physiology better, if advanced diagnostic tests like enzyme assay and blood markers are used.

Summary and Conclusion

Using basic blood parameters and vitals in the present study, the compensatory mechanisms happening in the body after maxillofacial trauma can be seen. These changes although significant on side by side comparison can still fall within the normal physiological range provided by various diagnostic setups. Hence the need for maxillofacial surgeon to be sensitive to minor variations in these aspects to ensure safety of the patient cannot be overemphasized.

There is a need for longer studies with advanced diagnostic methods to understand the homeostatic environment disturbances after maxillofacial trauma alone or in combination with other poly-trauma subjects.

Patient Consent

Informed and written consent taken. Institutional ethical board committee clearance obtained.

Ethical approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

This article does not contain any studies with animals performed by any of the authors.

Informed consent

Informed consent was obtained from all individual participants included in the study.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Stahel PF, Flierl MA, Moore EE. "Metabolic staging" after major trauma-A guide for clinical decision making? *Scand J Trauma Resusc Emerg Med* 2010;18:34.
2. D'alessandro A, Nemkov T, Moore HB, Moore EE, Wither M, Nydam T, *et al.* Metabolomics of trauma-associated death: Shared and fluid-specific features of human plasma vs lymph. *Blood Transfus* 2016;14:185-94.
3. Charrueau C, Belabed L, Besson V, Chaumeil JC, Cynober L, Moinard C. Metabolic response and nutritional support in traumatic brain injury: Evidence for resistance to renutrition. *J Neurotrauma* 2009;26:1911-20.
4. Yadollahi M, Behzadi Seyf Abad M, Pazhuheian F. Pattern of maxillofacial injuries and determinants of outcome in a large series of patients admitted to a Level-I Trauma Center. *Bull Emerg Trauma* 2019;7:176-82.
5. Klein D. Classification of maxillofacial injuries. *Plast Reconstr Surg* 1947;2:133-8.
6. Singer M, Jones AM. Bench-to-bedside review: The role of C1-esterase inhibitor in sepsis and other critical illnesses. *Crit Care* 2011;15:203.
7. Spahn DR, Bouillon B, Cerny V, Coats TJ, Duranteau J, Fernández-Mondéjar E, *et al.* Management of bleeding and coagulopathy following major trauma: An updated European guideline. *Crit Care* 2013;17:R76.
8. Bauer AR, Mc Neil C, Trentelman E, Swift SA, Mason JD. The depression of T lymphocytes after trauma. *Am J Surg* 1978;136:674-80.
9. Namas RA, Vodovotz Y, Almahmoud K, Abdul-Malak O, Zaaqoq A, Namas R, *et al.* Temporal patterns of circulating inflammation biomarker networks differentiate susceptibility to nosocomial infection following blunt trauma in humans. *Ann Surg* 2016;263:191-8.
10. Martini WZ. Coagulation complications following trauma. *Mil Med Res* 2016;3:35.
11. Olariu E, Pooley N, Danel A, Miret M, Preiser JC. A systematic scoping review on the consequences of stress-related hyperglycaemia. *PLoS One* 2018;13:e0194952.
12. Kapoor D, Srivastava M, Singh P. Point of care blood gases with electrolytes and lactates in adult emergencies. *Int J Crit Illn Inj Sci* 2014;4:216-22.
13. Hastings AB. The electrolytes of tissue and body fluids. *Harvey Lect* 1940-1941;36:91.
14. Udy AA, Morton FJ, Nguyen-Pham S, Jarrett P, Lassig-Smith M, Stuart J, *et al.* A comparison of CKD-EPI estimated glomerular filtration rate and measured creatinine clearance in recently admitted critically ill patients with normal plasma creatinine concentrations. *BMC Nephrol* 2013;14:250.
15. Tucketta JW, Lynham A, Lee GA, Perry M, Harringtone Ú. Maxillofacial trauma in the emergency department: A review. *Surgeon* 2017;12:106-14.
16. Ganong WF. *Review of Medical Physiology*. 23rd ed. USA: McGraw Hill; 2013.
17. Mitchell C. Tissue oxygenation monitoring as a guide for trauma resuscitation. *Crit Care Nurse* 2016;36:12-70.
18. Hsieh TM, Kuo PJ, Hsu SY, Chien PC, Hsieh HY, Hsieh CH. Effect of hypothermia in the emergency department on the outcome of trauma patients: A cross-sectional analysis. *Int J Environ Res Public Health* 2018;15:E1769. doi: 10.3390/ijerph 15081769.
19. Appenheimer MM, Evans SS. Temperature and adaptive immunity. *Handb Clin Neurol* 2018;156:397-415.
20. Reisner AT, Edla S, Liu J, Liu J, Khitrov MY, Reifman J. Tachycardic and non-tachycardic responses in trauma patients with haemorrhagic injuries. *Injury* 2018;49:1654-60.