# Damage Control Resuscitation for Catastrophic Bleeding

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**KEYWORDS**

• Hemorrhage • Shock • Transfusion • Resuscitation • Coagulopathy • Thrombelastography

**KEY POINTS**

- The timely recognition of shock secondary to hemorrhage from severe facial trauma or as a complication of complex oral and maxillofacial surgery presents formidable challenges clinically.
- Specific hemostatic disorders are induced by hemorrhage and several extreme homeostatic imbalances may appear during, or in the aftermath of, resuscitation of patients with catastrophic bleeding that dominate the pathophysiology of an acutely bleeding patient.
- Damage control resuscitation (DCR) has evolved from a definition of massive transfusion (MT) to a more complex therapeutic paradigm that includes hemodynamic resuscitation, hemostatic resuscitation, and homeostatic resuscitation.
- A detailed DCR protocol must be and should be an essential element of a well-defined response to catastrophic blood loss that may complicate several surgical and medical specialties.
- In virtually every clinical setting, however, definitive control of bleeding is the principal objective of any comprehensive resuscitation scheme for hemorrhagic shock (HS), and DCR should not overshadow emergent intervention to that end.

## INTRODUCTION

In the past decade, MT for exsanguinating hemorrhage has evolved into a more inclusive paradigm referred to as DCR. DCR centers on 3 distinct therapeutic objectives: hemodynamic resuscitation, hemostatic resuscitation, and homeostatic resuscitation. The principles of DCR are based on extensive laboratory investigation and comprehensive clinical study of HS pathophysiology, hemorrhage-associated coagulopathy, and hemorrhage-induced homeostatic imbalance, which appear as a consequence of massive blood loss or in the aftermath of resuscitation.[1–7] Implementation of team-based DCR programs is associated with an increase in survival of the severely injured in concert with a reduction in costly waste of blood bank products.[8,9] Blood component transfusion best practices were developed by the AABB to enhance outcome measures of hospital blood management programs,[10] and, currently, hospitals in the United States are required by accreditation and regulatory agencies[a] to institute specific resuscitation policies and guidelines. Moreover, trauma center verification by the American College of Surgeons Committee on Trauma

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[a]The National Quality Measures Clearinghouse, sponsored by the Agency for Healthcare Research and Quality, and the US Department of Health and Human Services, have included The Joint Commission measures in its public database for evidence-based quality measures and measure sets.

Oral Maxillofacial Surg Clin N Am 28 (2016) 553–568
http://dx.doi.org/10.1016/j.coms.2016.06.010

requires hospital to demonstrate both organized capability to carry out DCR and a commitment to the process.[11] DCR, however, is demanding with respect to resource utilization prompting considerable discourse and ethical debate on, for example, blood resource allocation in MT scenarios that create significant blood product shortages. Also at issue are the inherent exigencies of an emergency setting that compromise determination of futility, obtaining informed consent, and awareness of advanced directives.[12]

This article reviews the essential elements of DCR and describes application of this complex therapy to catastrophic bleeding in severely injured patients. Furthermore, the authors believe that the basic tenets of DCR elucidated in this review pertain to management of significant blood loss as a complication of complex oral and maxillofacial surgery as well as other conditions, for example, massive gastrointestinal bleeding, gynecologic hemorrhagic crises, and intraoperative and postoperative hemorrhage complicating vascular or cardiovascular procedures. Thus, clinicians of varying specialties must maintain familiarity with rapid and significant developments in this aspect of resuscitation. However, DCR is not the definitive management of life-threatening hemorrhage in most cases. The primary treatment of ongoing hemorrhage remains source control by surgical, angiographic, or endoscopic intervention.

## HEMORRHAGIC SHOCK

### *Clinical Aspects*

The early recognition of HS and timely initiation of resuscitation remain formidable challenges,[13] in part because of a complex mosaic of compensatory mechanisms activated in response to an acute loss of blood. Compensatory physiology tends to obscure signs of tissue hypoperfusion and impending hemodynamic collapse. Consequently, a substantial loss of blood sustained by a trauma patient prior to hospital arrival may pass unnoticed, or occult hemorrhage that continues in the trauma bay may not be recognized in a timely manner. In a previously healthy patient, these early compensatory processes are highly effective and create a deceptive clinical state that may suggest stability when rather a considerable oxygen debt has accumulated and significant cellular dysfunction has developed.[14]

Compensatory mechanisms are mediated by an increase in sympathetic autonomic activity in conjunction with secretion of vasoactive hormones (predominantly catecholamines, vasopressin, and angiotensin[15]) that mitigate the reduction in perfusion of vital organs despite a fall in circulating blood volume. Decompensation generally means that the limits of these particular responses have been exceeded. Thus, under intense neurohormonal stimulation, an initial clinical state of compensated shock develops. Ongoing hemorrhage eventually progresses, however, to decompensated shock, characterized by hemodynamic instability and accelerating functional deterioration in vital physiologic systems,[16] and finally to refractory shock (also referred to as irreversible shock), associated with hemodynamic collapse and death.[17,18] Resuscitation initiated promptly and guided by established standards of care generally is effective for compensated HS. Resuscitation becomes progressively less effective during the decompensated phase of shock as physiologic reserves are exhausted and is rendered completely ineffective during the refractory phase due to accumulated cell death preventing recovery of organ function.

Because HS is fundamentally a problem of blood flow rather than blood pressure, systemic arterial hypotension (systolic blood pressure [SBP] $\leq$90 mm Hg) is widely regarded as an inaccurate clinical indicator of shock.[19–23] A change in heart rate (HR) assessed out of context with other parameters is also considered an insensitive as well as nonspecific sign of hemorrhage.[24] The ratio of HR to SBP, or shock index (SI), however, has been examined as a clinical measure that may identify hypoperfusion before a patient decompensates.[25–30] Furthermore, age diminishes physiologic reserve, and the product of SI and age is suggested as a more sensitive indicator compared with HR, SBP, or SI alone.[28] A yet more sensitive indication of occult hemorrhage may be obtained by dividing HR by the pulse pressure (SBP $-$ diastolic blood pressure), referred to as the pulse rate over pressure evaluation (ROPE) index.[31] The ROPE index may be somewhat more sensitive to blood loss than SI.[32] A reduction in pulse pressure (also referred to as narrowing) reflects a downward trend in SBP due to diminished stroke volume in parallel with an upward trend in DBP due to compensatory vasoconstriction and an increase in vascular resistance.

Detection of a lactic acidosis corroborates the diagnosis of HS. A normal hydrogen ion ($H^+$) concentration ([$H^+$]) in extracellular fluid is 40 nmol/L, approximately one-millionth the extracellular concentration of $HCO_3^-$ (normal, 24 mmol/L). Thus, the rise in [$H^+$] secondary to a hemorrhage-induced acidemia is very small. For example, a substantial decrease in pH from 7.4 to 7.1 represents merely a 40-nmol/L increase in [$H^+$]. In addition to pH, this extraordinarily small number can be