PAPER

Supranormal Trauma Resuscitation Causes More Cases of Abdominal Compartment Syndrome

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Hypothesis: Normal resuscitation (oxygen delivery index [DO_2I] ≥500 mL/min per square meter), compared with supranormal trauma resuscitation (DO_2I ≥600 mL/min per square meter), requires less crystalloid volume, thus decreasing the incidence of intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS).

Design: Retrospective analysis of a prospective database.

Setting: Twenty-bed intensive care unit (ICU) in a regional level I trauma center.

Patients: Patients with major trauma (injury severity score >15, initial base deficit ≥6 mEq/L, or need for ≥6 units of packed red blood cells in the first 12 hours) or age 65 years or older with any 2 of the previous criteria.

Interventions: Shock/trauma resuscitation protocol: pulmonary artery catheter, gastric tonometry, urinary bladder pressure measurements, lactated Ringer infusion, packed red blood cell transfusion, and moderate inotrope support, as needed, in that sequence, to attain and maintain a DO_2I greater than or equal to 600 mL/min per m^2 (16 months, ending January 1, 2001, n=85) or a DO_2I greater than or equal to 500 mL/min per square meter (16 months, starting January 1, 2001, n=71) for the first 24 hours in the ICU.

Main Outcome Measures: Lactated Ringer infusion volume (liters) at ICU admission, gastric partial carbon dioxide minus end-tidal carbon dioxide (GAP_{CO2}), IAH (urinary bladder pressure measurements >20 mm Hg), ACS (urinary bladder pressure measurements >25 mm Hg with organ dysfunction), multiple organ failure, and mortality.

Results: Demographics, injury severity, and shock severity parameters were similar in both groups. The supranormal resuscitation group required more lactated Ringer infusion volume in the first 24 hours in the ICU (mean ± SD, 13 ± 2 vs 7 ± 1 L; $P<.05$) and had higher GAP_{CO2} (16 ± 2 vs 7 ± 1 mm Hg; $P<.05$). In the supranormal group, IAH (42% vs 20%; $P<.05$) and ACS (16% vs 8%; $P<.05$) were more frequent. The conventional trauma outcomes, such as multiple organ failure (22% vs 9%; $P<.05$) and mortality (27% vs 11%; $P<.05$) were less favorable in the supranormal resuscitation group.

Conclusion: Supranormal resuscitation, compared with normal resuscitation, was associated with more lactated Ringer infusion, decreased intestinal perfusion (higher GAP_{CO2}), and an increased incidence of IAH, ACS, multiple organ failure, and death.

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POSTINJURY MULTIPLE organ failure (MOF) continues to be the major cause of prolonged intensive care unit (ICU) stay and mortality among trauma patients who survive the first 48 hours postinjury.[1-3] To prevent MOF, the population at risk was determined, and a prediction model was developed during the early 1990s.[4] The predictive model helps investigators to concentrate their preventive and therapeutic efforts on the population at highest risk for MOF. The independent predictors for MOF are age, injury severity measured by the injury severity score, and the severity of shock. Since age and injury severity score are not factors that we can control, resuscitation of hemorrhagic shock has become the focus in the prevention of MOF in many trauma centers. Shoemaker et al[5] proposed that a supranormal oxygen delivery index (DO_2I) (>600 mL/min per square meter) should be the optimal resuscitation goal, since they observed that DO_2I was increased to supranormal levels in survivors compared with nonsurvivors, whose DO_2I remained less than 600 mL/min per square meter. The concept that early supranormal DO_2I values improved the outcome was reported in a series of publications.[6-8] Later, several prospective randomized trials offered conflicting results.[9-11] Shoemaker et

al[12] recently showed, in a prospective randomized trial, that supranormal resuscitation does not have additional benefits on the outcome of critically injured patients.

We developed a computerized traumatic shock resuscitation protocol that is presumptively applied to trauma patients who are at high risk for MOF.[13,14] This standardized resuscitation process is a standard of care at our ICU and provides a unique opportunity for prospective resuscitation data collection. From the initiation of the resuscitation protocol in 1997, we used the supranormal DO_2I goal to attain and maintain during the first 24 hours after ICU admission. However, the ongoing concern regarding excessive volume loading prompted us to abandon the supranormal goal and, in January 2001, implement the "normal" DO_2I resuscitation goal (>500 mL/min per square meter). Our recent study,[15] comparing 18 patients who received supranormal resuscitation with 18 age-matched patients who received normal resuscitation, showed no difference in responses, but the supranormal group required more crystalloid loading. Clinical reports during the last 2 years have suggested the possible relationship between crystalloid loading and the development of intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS).[16-18] We described the epidemiologic factors and developed a prediction model for postinjury ACS to aid in the prevention of this syndrome, which is associated with an incidence of MOF and mortality rate greater than 50%.[19] Crystalloid volumes were identified by multiple logistic regression analysis as independent predictors for ACS. We hypothesized that normal resuscitation, compared with supranormal trauma resuscitation, would decrease the incidence of IAH and ACS because it requires less crystalloid volume.

METHODS

PATIENTS

Severely injured patients who meet specific criteria are resuscitated using a standardized shock resuscitation protocol in the shock trauma ICU of Memorial Hermann Hospital (Houston, Tex), a level I regional trauma center and teaching affiliate of the University of Texas–Houston Medical School, which serves the greater Houston area (southeast Texas; population, 4 million). During the 32-month period ending in April 2002, there were 13 588 trauma admissions and 1983 shock trauma ICU admissions in Memorial Hermann Hospital. We compared patients' responses and outcomes between the supranormal resuscitation goal (DO_2I >600 mL/min per square meter), used during the 16-month period before January 2001, and the normal resuscitation goal (DO_2I >500 mL/min per square meter), used starting in January 2001. These goals were to be attained and maintained during the first 24 hours in the ICU.

The patients included in this study were those who required shock resuscitation. The criteria identifying the need for resuscitation included (1) major injury (≥2 abdominal organs, ≥2 long bone fractures, complex pelvic fracture, flail chest and/or major vascular injury), (2) blood loss (anticipated need for ≥6 units of packed red blood cells for transfusion during the first 12 hours after hospital admission), and (3) shock (arterial base deficit ≥6 mEq/L during the first 12 hours after hospital admission), or a trauma victim 65 years or older with any 2 of the previous criteria. Patients with these criteria who also had incurred severe brain injury (defined as Glasgow Coma Scale score ≤8 in the shock trauma ICU and abnormal brain computed tomographic scan results) were not resuscitated by the protocol unless the patient's brain injury was assessed by the attending neurosurgeon to be at low risk of worsening cerebral edema with volume loading. There were no additional criteria for the patients described in this study. Injury severity score was determined by trauma registry coders. Consecutive patients during the 32-month period were received as 2 cohorts, supranormal (before January 2001) and normal (from January 2001).

RESUSCITATION PROTOCOL

The resuscitation protocol is a goal-directed, logical, rule-based process that emphasizes hemoglobin and volume loading to attain and maintain an oxygen delivery goal for the first 24 hours of ICU admission. Described previously,[13,14] a hierarchical sequence of therapy interventions and thresholds for intervention is uniformly applied to all patients. Resuscitation is data driven to tailor the standardized process to the needs and responses of the individual patients. To achieve a DO_2I greater than or equal to 600 mL/min per square meter (before January 2001) or a DO_2I greater than or equal to 500 mL/min per square meter (from January 2001) for 24 hours, the protocol comprises a hierarchy of 5 sequentially applied therapies (with intervention thresholds): (1) packed red blood cell transfusion (hemoglobin <10 g/dL [<12 g/dL if age ≥65 years]); (2) lactated Ringer solution (pulmonary capillary wedge pressure [PCWP] <15 mm Hg [<12 mm Hg if age ≥65 years]); (3) Starling curve to optimize the relationship between cardiac index and PCWP (hemoglobin ≥10 g/dL, PCWP ≥15 mm Hg, and DO_2I <600 mL/min per square meter or DO_2I ≥500 mL/min per square meter); (4) inotrope (cardiac index–PCWP optimized and DO_2I <600 mL/min per square meter or DO_2I ≥500 mL/min per square meter); and (5) vasopressor (mean arterial pressure <65 mm Hg).

As a part of the resuscitation protocol, intra-abdominal pressure was determined indirectly by measuring urinary bladder pressure (UBP) every 4 hours (or more frequently if it was ordered). Intra-abdominal hypertension was defined as a UBP greater than 20 mm Hg. Abdominal compartment syndrome was defined as a UBP greater than 25 mm Hg with acute organ (cardiac, pulmonary, or renal) dysfunction, reversible on abdominal decompression. A pulmonary artery catheter with continuous cardiac output and mixed venous hemoglobin oxygen saturation monitoring and a gastric tonometer-sump catheter with gastric mucosal interstitial partial pressure of carbon dioxide monitoring were placed. Current, essential measurements (hemoglobin concentration, cardiac index, arterial oxygen saturation, and PCWP) are used in decision making for therapy intervention in real time. The hierarchy sequence is based on these measurements and a comparison with established intervention thresholds.

DATA ANALYSIS

Data were obtained prospectively during the protocol resuscitation process. A computer with touch screen and keyboard interfaces was used at bedside to enter necessary data, compare data with threshold rules, execute the logical process, and present instructions for intervention and/or monitoring. With the computer, both diagnostic data and interventions were recorded in real time for review. Retrospectively, the resuscitation response, interventions, and outcomes of supranormal and normal resuscitation patients were compared. The hemodynamic variables, cardiac index, PCWP, gastric regional mucosal carbon dioxide minus end tidal carbon dioxide (GAP_{CO2}), UBP, and blood and fluid volumes during the time course of the resuscitation process were compared. Cohort demographics, the incidence of IAH and ACS, MOF, and survival outcomes were also compared.

Data are presented as mean ± SEM. Data were analyzed using an analysis of variance, χ^2, and *t* tests. The analysis of variance was used to analyze and compare time sequence data between the 2 cohorts. The χ^2 test was used to compare the injury severity score between the male and female cohorts, a nonparametric scale assessment. *t* tests (unpaired, 2-tailed) were used to compare parametric quantities (eg, clinical laboratory measurements, fluid volumes, and stay times). A difference between or within the supranormal and normal cohorts with $P<.05$ was considered significant.

RESULTS

During the 32-month study period, 156 patients were resuscitated in the shock trauma ICU using our standardized shock resuscitation protocol. Eighty-five of these patients received supranormal DO_2I resuscitation and 71 received normal DO_2I resuscitation. Demographic, injury, and shock severity data are presented in **Table 1**. Supranormal and normal cohorts were not different in age, sex, injury mechanism, injury severity score, and initial severity of shock. In response to the initial shock, both cohorts were given the same amount of blood and crystalloids during the pre-ICU (nonprotocol-driven) phase of the resuscitation. As we described earlier, the normal DO_2I resuscitation goal resulted in the same response in terms of cardiac index and mixed venous oxygen saturation (**Figure 1**). Supranormal and normal resuscitation optimized the arterial base deficit and serum lactate during a similar time frame (**Figure 2**). To achieve the supranormal goal, significantly more crystalloid volume infusion was required, and there was a tendency toward more packed red blood cell transfusion ($P=.07$) (**Figure 3**). The UBP of the supranormal cohort was significantly higher at 4 hours after ICU admission and remained higher throughout the first 24 hours (**Figure 4**A). The gastric regional perfusion described by the GAP_{CO2} was higher in the supranormal group (Figure 4B). The supranormal cohort experienced worse outcomes, with higher frequency of IAH, ACS, MOF, and mortality (**Table 2**).

COMMENT

Our shock resuscitation results support that supranormal trauma resuscitation is not superior to normal traumatic shock resuscitation. The supranormal and normal resuscitation cohorts had the same resuscitation response to our standardized shock resuscitation protocol. From the 2 demographically similar cohorts with identical shock and injury severity, the supranormal resuscitation patients required more LR loading to achieve the goal of a DO_2I greater than 600 mL/min per square meter. The normal resuscitation cohort (DO_2I goal >500 mL/min per square meter), with less crystalloid requirement, had better intestinal perfusion, lower UBP, and better outcome than those of the supranormal cohort.

Our observation is consistent with recent clinical reports that have suggested that supranormal resuscitation does not have an additional benefit over normal resuscitation.[9-12] Furthermore, our study advocates that supranormal resuscitation can be harmful in a population of severely injured patients with predominantly blunt injuries.

Table 1. Demographics, Injury, and Shock Severity of Supranormal and Normal Resuscitation Cohorts*

	Supranormal Resuscitation (n = 85)	Normal Resuscitation (n = 71)
Age, y	37 ± 3	33 ± 2
Sex, % male	76	74
Injury mechanism, % blunt	84	80
ISS	28 ± 3	27 ± 2
Initial base deficit, mEq/L	9 ± 1	9 ± 1
Serum lactate, 12-24 h, mmol/L	4.2 ± 1	3.9 ± 1
Pre-ICU PRBC transfusions, U	5 ± 1	5 ± 1
Pre-ICU crystalloid infusions, L	6 ± 1	5 ± 1

Abbreviations: ICU, intensive care unit; ISS, Injury Severity Score; PRBC, packed red blood cell.
*Data are given as mean ± SD unless otherwise indicated.

In their recent prospective randomized study of 35 control and 40 supranormal resuscitation patients, Velmahos et al[12] concluded that early optimization does not improve the outcome in severely injured patients. Their study population had the same mean age (33 years) but their Injury Severity Score was lower (21 points) than in our present study population. Seventy-three percent of their population sustained penetrating trauma, while in our population, more than 80% of the patients had blunt trauma. The difference in injury mechanism can explain why Velmahos et al did not find any difference in outcome between supranormal and normal patients and why we found that supranormal resuscitation resulted in worse outcome. Velmahos et al also concluded that penetrating trauma is a predictor of optimal resuscitation response because it is associated with specific injuries that can be addressed surgically, unlike blunt trauma, which is usually caused by diffuse impact with multisystem effects that cannot always be controlled surgically.

A meta-analysis of 21 randomized controlled trials on hemodynamic optimization revealed a statistically significant reduction in mortality if patients with acute severe illness or injury were treated early to achieve optimal (supranormal) goals before the development of organ failure.[20] The hemodynamic optimization after the development of organ failure did not improve the outcome compared with control groups.

While the concept that maximizing oxygen delivery to eliminate flow-dependent oxygen consumption decreases MOF and the optimal DO_2I value remains controversial, we believe that a shock resuscitation protocol facilitates appropriate prioritization of resuscitation in the initial evaluation and management of major trauma. Moreover, a protocol facilitates early recognition of complications that would not be evident with standard monitoring. Examples include myocardial dysfunction, ongoing hemorrhage, and the development of ACS. The end point in resuscitation is difficult to define. Our approach is to aim for a DO_2I greater than 500 mL/min per square meter during the first 24 hours of resuscitation and to continue resuscitation beyond that time only if evidence of persistent perfusion deficits exists. Efforts to maintain a DO_2I greater than 500 mL/min per square meter beyond 24 hours is rarely beneficial, and to persist may be harmful.

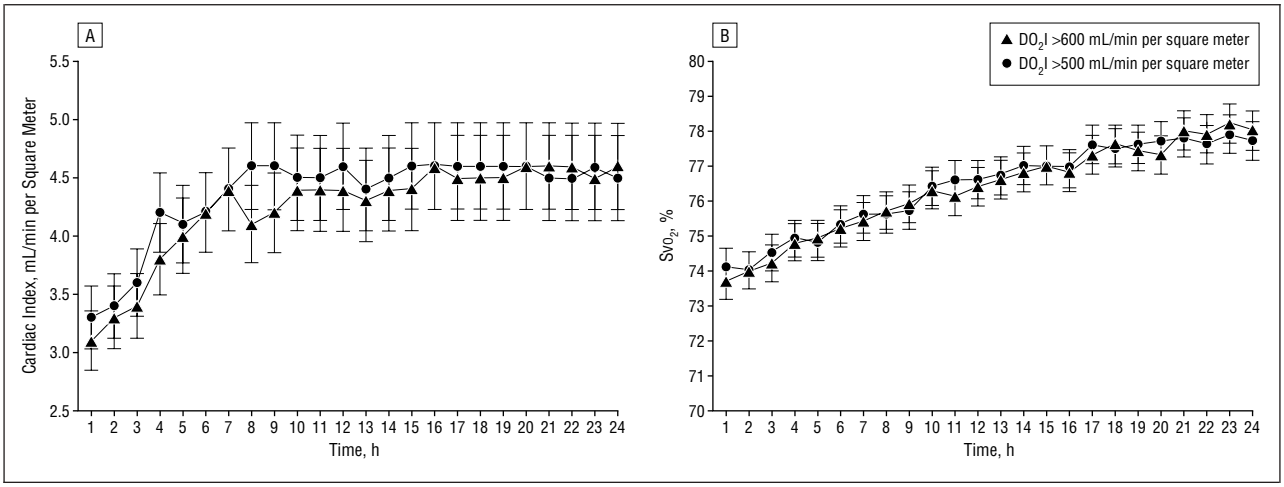

Figure 1. A, Cardiac index response of patients undergoing supranormal and normal resuscitation. B, Mixed venous oxygen saturation of patients undergoing supranormal and normal resuscitation. DO_2I indicates oxygen delivery index; SvO_2, mixed venous oxygen saturation.

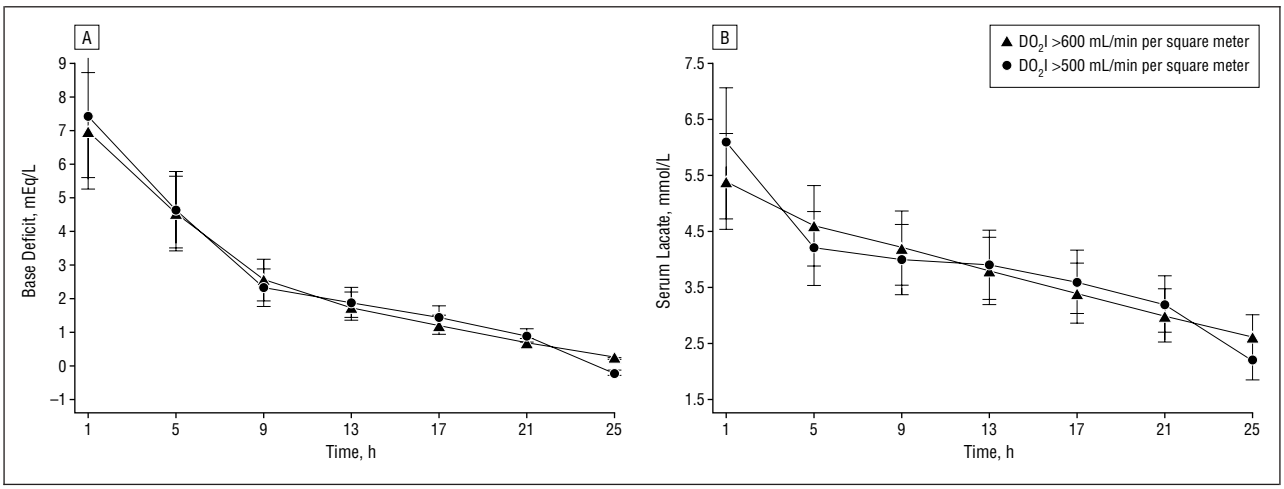

Figure 2. A, Arterial base deficit of patients undergoing supranormal and normal resuscitation. B, Serum lactate concentration of patients undergoing supranormal and normal resuscitation. DO_2I indicates oxygen delivery index.

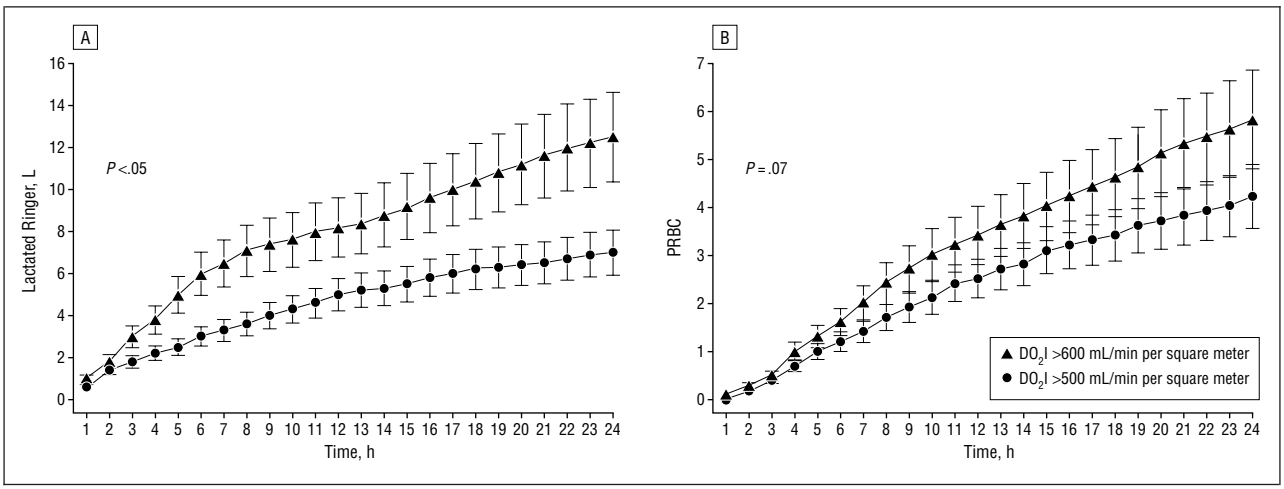

Figure 3. A, Crystalloid infusions of patients undergoing supranormal and normal resuscitation. B, Packed red blood cell (PRBC) transfusions of patients undergoing supranormal and normal resuscitation. DO_2I indicates oxygen delivery index.

We have learned from a small group of age-matched shock trauma patients that normal and supranormal resuscitation can yield the same physiologic response to the standardized shock resuscitation protocol.[15] It was clear from this study that with supranormal resuscitation, significantly more LR was infused. There was

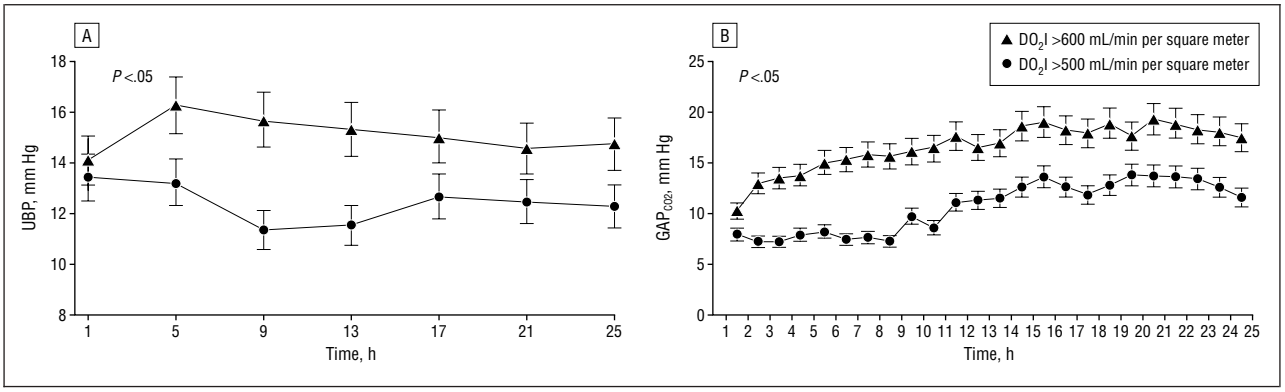

Figure 4. A, Urinary bladder pressure of patients on supranormal and normal resuscitation. B, Gastric regional mucosal carbon dioxide minus end-tidal carbon dioxide (GAP_{CO2}) of patients on supranormal and normal resuscitation. DO_2I indicates oxygen delivery index; UBP, urinary bladder pressure.

Table 2. Outcomes of the Supranormal and Normal Resuscitation Cohorts*

	Supranormal Resuscitation (n = 85)	Normal Resuscitation (n = 71)
Intra-abdominal hypertension	42†	20
Abdominal compartment syndrome	16†	8
Multiple organ failure	22†	9
Death	27†	11

*Data are given as percentage of patients.
†$P<.05$.

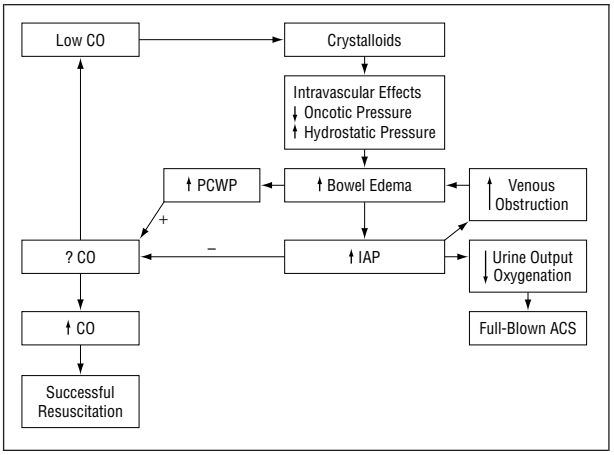

Figure 5. CO indicates cardiac output; IAP, intra-abdominal pressure; PCWP, pulmonary capillary wedge pressure; +, positive effect; –, negative effect; and ACS, abdominal compartment syndrome.

no difference between the supranormal and normal groups in terms of mortality.

However, the larger cohorts and greater statistical power in this study reveal a difference in various outcome measures. The larger amount of crystalloids used during supranormal resuscitation surprisingly led to worse intestinal perfusion, most likely because of intestinal edema. Although the exact mechanism of the postinjury intestinal edema is unknown, the postreperfusion capillary leak, the decreased colloid oncotic pressure, and the increased hydrostatic pressure in the capillary vascular bed are possible contributors. All of these factors can be related to excessive crystalloid loading.[18] Bowel edema, a major component of postinjury IAH and ACS, can occur after hemorrhagic shock and resuscitation in the absence of abdominal injuries, and is known as secondary ACS.[16-18]

We recently developed a prediction model for postinjury ACS. In this model, greater than 7.5 L of infused crystalloids and a GAP_{CO2} greater than 16 mm Hg are independent predictors of ACS.[19] As shown in **Figure 5**, patients who are admitted to the ICU and have a low cardiac index are volume-loaded with crystalloid infusions, which decrease the intravascular colloid oncotic pressure and increase the hydrostatic pressure. This intervention can have a positive effect on cardiac output by increasing the preload (PCWP), but can have a negative effect through the increased edema of the reperfused gut. The bowel edema is worsened by the impaired venous return caused by the elevated intra-abdominal pressure. The result of the negative (IAH) and positive (increased PCWP) effects determines the cardiac output response. If the response is not optimal and the intra-abdominal pressure is not recognized, resuscitation can enter the futile crystalloid preloading cycle, where further crystalloid infusions worsen the bowel edema and increase the intra-abdominal pressure until the patients develop further organ dysfunctions (renal, pulmonary) and full-blown ACS.

According to our present data, the incidence of IAH and ACS was higher in the supranormal resuscitation group, in whom more LR infusions were administered. The worse outcome of the supranormal group was due to the suboptimal intestinal perfusion and the increased incidence of ACS. The impaired perfusion (indicated by higher GAP_{CO2}) of the gut and subsequent reperfusion is a known instigator of MOF.[21] Numerous investigators have shown that gastric tonometry can be a reliable clinical tool to monitor early intestinal circulatory compromise caused by IAH or ACS.[22-24] Clinical and laboratory observations during the last decade have reported a poor outcome in ACS and a frequent association of ACS with MOF.[25-28] In the prospective ACS database of Memorial Hermann Hospital, shock resuscitation is associated with an incidence of MOF and mortality rate of greater than 50%. Based on logistic regression analysis, ACS is a predictor for both MOF and death.[19]

In summary, our standardized shock resuscitation protocol provided a unique opportunity to compare se-

verely injured patients in shock who were resuscitated with supranormal or normal DO_2I goals. The review of the prospective resuscitation data demonstrates that supranormal resuscitation, compared with normal resuscitation, was associated with more LR infusion, decreased intestinal perfusion (higher GAP_{CO2}), and an increased incidence of IAH, ACS, MOF, and death.

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DISCUSSION

Donald E. Fry, MD, Albuquerque, NM: The authors have presented to us a very provocative study which shows that resuscitation to a supernormal level of oxygen delivery in severe trauma patients resulted in increased requirements for crystalloid resuscitation, demonstrated an increased gastric P_{CO_2} accumulation with their gastric telemetry studies, which indicates that there really was reduced gastric mucosal perfusion, which, if you followed the data closely, was evident at 2 hours into the patient's management—well before increased intra-abdominal pressure would be an issue. They showed increased intra-abdominal pressure and increased frequency of the abdominal compartment syndrome, reflecting increased extracellular water accumulation, in my view, within the intestine and its contents, and increased multiple organ failure and deaths in the patients.

This is particularly interesting since the SVO_2 in their studies were the same between the 2 groups; thus, the patients had increased oxygen consumption in the groups that had increased mortality rates and increased organ failure complications. So let's dissect apart this a little bit. If we have oxygen delivery as a function of cardiac output of oxygen saturation of hemoglobin and of PO_2, we have the 2 groups being equal. The difference can only be explained in terms of oxygen delivery by differences of hemoglobin in the 2 groups, which the authors have not presented. It means that increased hemoglobin is in fact potentially the variable that makes this a study of genuine significance. The issue may in fact not be how much oxygen was being delivered, but this may be a landmark study in provoking the idea that higher hematocrits may end up in the acute resuscitative area of time of being adverse to patient outcome. Increased hematocrit, increased blood viscosity results in microcirculatory increased resistance to flow, increased extravasation of our resuscitative extracellular water, and the consequences of increased intra-abdominal pressure, increased probability of abdominal compartment syndrome. So I believe that the data here are incredibly interesting, as we all wrestle with the issue of what is the appropriate hemoglobin that patients should be maintained at in their postresuscita-

tive period and, for that matter, during the entire period of patient management in the critical care unit after major injury.

So I would like to offer 3 questions to the authors. What in fact were the postresuscitative hemoglobin differences in the 2 groups of patients? I would suspect that at 24 hours, those differences were significant. Can the authors tell us now that they have abandoned hyperoxygenation as a strategy? What did they shoot for as the appropriate hematocrit in the postresuscitative patient? This inevitably draws enormous debate in our institution every time this subject is discussed.

Finally, I would actually question the value of the routine placement of the pulmonary artery catheter of the Swan-Ganz catheter as was apparent that they did through this study. If you are not going to calculate oxygen delivery and if the majority of patients in fact can be appropriately resuscitated with clinical criteria that they used, do they continue to recommend that postresuscitative trauma patients, that trauma patients going into the intensive care unit, really require the pulmonary artery catheter? There continues to be some debate whether the pulmonary artery catheter itself may in fact be a source of morbidity, and I would be interested in their reflections on that.

Steven Stain, MD, Nashville, Tenn: My question relates to Dr Fry's comments. The end point of resuscitation in both groups was oxygen delivery, and that requires a Swan Ganz catheter for calculation. If you have a relatively young trauma patient with an ISS (injury severity score) greater than 15 who requires blood transfusion for a splenectomy, does that patient also require a Swan or can that patient be managed just with urine output and clinical criteria?

Gregory Jurkovich, MD, Seattle, Wash: Ten years ago, we were encouraged to use supranormal oxygen delivery to drive our care during the initial resuscitation of critically injured patients. In this short time period, not only has this been questioned, but this paper refutes it as not only being of no benefit but also of being harmful.

Dr Fry has emphasized the importance of and questions the relative hemoglobin or hematocrit that should be idealized in the circulation during resuscitation. I want to add to that question my concern for what now should be the appropriate measure of adequacy of resuscitation. You have argued that a delivery of oxygen of greater than 600 mL/min would be inappropriate and that 500 mL/min is the same. But is there another number that is better? Is 400 okay; is 300 okay? What is and what should be our end point of resuscitations? Particularly bothersome to me is that the SVO_2, my preferred measure of adequacy of resuscitation, was the same in both of these groups. So I am perplexed now as to what I should use as a resuscitation end point or resuscitation goal. I would appreciate your comments on what you now use to determine effective resuscitation.

Dr Cocanour: We are using a computerized resuscitation protocol. One of its strengths is that it allows us to do these kinds of studies and decrease the variability between groups even though this study was done over 2 consecutive periods of time. Other than the drop in DO_2I, the protocol did not change.

What was the difference in the postresuscitation hemoglobin between the 2 groups? We did not include it in the manuscript or presentation but it was similar to the transfusion data, which, if you remember, the *P* value was only.07. There was a slightly higher hemoglobin at postresuscitation in those who had a DO_2I of 600 as a goal. We will need to go back and review the actual numbers.

What is the appropriate hemoglobin goal for resuscitation? We still have arguments within our own group. I am not sure that anybody knows for sure. I am satisfied with a hemoglobin above 7 or 8, although if someone is bleeding, you need to keep the hemoglobin higher to avoid having the next hemoglobin come back at 4. It also must be individualized for each patient.

What is the appropriate hemoglobin to aim for in the ICU? Again, there is still a great deal of controversy about this. It will take many more studies before we can definitely say what is an appropriate hemoglobin.

Yes, we still are using PA [pulmonary artery] catheters. The senior author feels that this is the gold standard for being able to monitor oxygen delivery, and oxygen delivery is still one of the best resuscitation end points. We have looked at using other devices, such as the Lidco, which measures cardiac output using lithium dilution. We have also looked at a couple of other devices as well. We have not come up with anything that we really like better, so we are still using the PA catheter.

What about the young, healthy patient who has had a splenectomy? These patients usually don't get put on the resuscitation protocol. We didn't go over the criteria for placement into the resuscitation protocol in depth, but if someone is not going to require an additional 6 units of blood over the next 24 hours, and their base deficit is corrected, I see no need to put those patients on the resuscitation protocol. But for patients with pelvic fractures from blunt trauma who are going to continue to lose blood, patients with continued ooze from raw surfaces in the abdomen or chest, or those in whom the base deficit is not correcting, these are the patients most likely to benefit from being placed on the resuscitation protocol.

What's the appropriate end point of resuscitation? If anybody ever comes up with an easily measured and appropriate end point that works for every single different patient, they will probably be able to make a lot of money. I don't know what the appropriate end point is. We have gone from a DO_2I of 600 because we noticed that a noticeable number of patients developed pulmonary edema during the resuscitation process, so as a consequence, our group dropped it to 500, and whether or not we should drop it further, I don't know. I really am not sure what the appropriate end point is if you are using DO_2I. I also do not know of anything better at this point.