ANNALS OF SURGERY
Vol. 227, No. 5, 726–734

Oxygen Delivery is an Important Predictor of Outcome in Patients With Ruptured Abdominal Aortic Aneurysms

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Objective

The purpose of this study was to evaluate the relation of oxygen delivery (DO_2) to the occurrence of multiple organ dysfunction (MOD) in patients with ruptured abdominal aortic aneurysms (AAA).

Summary Background Data

Patients with ruptured AAA are at high risk for the development of MOD and death. Previous reports of high-risk general surgical patients have shown improved survival when higher levels of DO_2 are achieved.

Methods

Hemodynamic data were collected at 4-hour intervals on 57 consecutive patients (mean age, 70.5 years) who survived 24 hours after repair of infrarenal ruptured AAA. Patients were resuscitated to standard parameters of perfusion (pulse, blood pressure, urine output, normal base deficit). MOD was determined based on six organ systems. Standard parametric (analysis of variance, t tests) and nonparametric (chi square, Wilcoxon) tests were used to compare hemodynamic data, red blood cell requirements, colon ischemia, and organ failure for patients with and without MOD.

Results

Patients who developed MOD had a significantly lower cardiac index and DO_2 for the first 12 hours; the difference was most significant at 8 hours. Logistic regression analysis demonstrated that the strongest predictors of MOD were DO_2, early onset of renal failure, and total number of red blood cells transfused.

Conclusions

DO_2 is an earlier and better predictor of MOD after ruptured AAA than previously identified risk factors. Failure to achieve a normal DO_2 in the first 8 hours after repair is strongly associated with the development of MOD and a high mortality. Strategies to restore normal DO_2 may be useful to improve outcome in these high-risk patients.

Patients with ruptured abdominal aortic aneurysms (AAA) constitute a high-risk surgical population.[1–16] For those who survive repair of their ruptured AAA, postoperative hemodynamic instability secondary to cardiac dysfunction and hypovolemia is common. Although stabilization can be achieved with vigorous resuscitation, patients remain at increased risk for the subsequent development of multiple organ dysfunction (MOD) and death.

Previous studies of patients with ruptured AAA have identified various risk factors for mortality, and most emphasize the relation of death to comorbid diseases of the patient or to early physiologic derangements that are often determined by the extent of aneurysmal rupture.[1–12] Although these preexisting and technical factors influence early mortality, late mortality has been linked to the development of organ dysfunction in the postoperative period.[2,4–7,9–11]

Previous studies of heterogeneous groups of high-risk general surgical patients have demonstrated that higher levels of oxygen delivery (DO_2) are associated with improved

Presented at the 109th Annual Meeting of the Southern Surgical Association, November 30 to December 3, 1997, The Homestead, Hot Springs, Virginia.
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Accepted for publication December 1997.

survival.[17–24] Although these studies have included patients with sepsis, acute respiratory distress syndrome, and trauma, patients who have ruptured AAA are unique in that they have blood loss without associated injury and they do not have the same metabolic derangements associated with infection. The purpose of this study was twofold: to evaluate the relation among postoperative hemodynamic function, oxygen transport, and the occurrence of MOD; and to compare the utility of DO_2 with previously identified predictors of outcome in patients with ruptured AAA.

MATERIALS AND METHODS

Patient Selection and Protocol

The study population consisted of consecutive patients who were referred to MetroHealth Medical Center with an infrarenal ruptured AAA from January 1989 through December 1996. Sixteen patients were retrospectively studied from 1989 until 1991, and the remainder were studied prospectively. All patients who survived 24 hours after surgical repair of the aneurysm were studied. Those with suprarenal aneurysms or those who required thoracic aortic cross-clamping were excluded. This study was approved by the Institutional Review Board of MetroHealth Medical Center.

Most patients were taken directly to the operating room on arrival with a clinical diagnosis of ruptured AAA. Laparotomy was performed through a midline approach, and the aneurysm neck was rapidly controlled without entering the retroperitoneal hematoma. After infrarenal application of the proximal aortic clamp, distal control was achieved by clamping the common iliac arteries. Patients were not systemically anticoagulated, but iliac backflow was evaluated after graft repair of the aneurysm; if it was found to be absent, an embolectomy catheter was passed. Tube graft repair was preferred except in the presence of concurrent significant iliac artery aneurysms. The condition of the inferior mesenteric artery was assessed; if widely patent, it was implanted on the graft. The vascular supply of the colon was subsequently evaluated either by inspection or intraoperative Doppler studies. Unclamping of the graft was performed in stages, with close hemodynamic monitoring to avoid hypotension. The cell saver was used routinely. Efforts were made to avoid intraoperative hypothermia by increasing the ambient room temperature, applying regional warming devices, and warming all infused fluids.

Intraoperative anesthetic management of patients was left to the discretion of the anesthesiologist and mainly consisted of narcotic and volatile anesthetic agents. Hemodynamic monitoring was begun during surgery. Patients were admitted directly to the surgical intensive care unit (SICU) after operation. All patients were monitored with continuous electrocardiography and arterial blood pressure, transcutaneous pulse oximetry, and a pulmonary artery catheter. Intravenous crystalloid and colloid solutions, blood products, and inotropic agents were administered to maintain standard parameters of perfusion (pulse, blood pressure, urine output, normal acid–base balance) and a normal pulmonary artery occlusion pressure (PAOP). Vasoactive drugs were used when perfusion abnormalities did not respond to fluids. Packed red blood cells were transfused to maintain a minimum serum hemoglobin level of 10 g/dL. All patients were mechanically ventilated for at least 24 hours after operation. Analgesic medications and sedatives were provided as necessary, either by continuous or intermittent intravenous infusion.

Hemodynamic and oxygen transport data were collected for the first 24 hours after aneurysm repair. PAOP and cardiac index (CI) were measured at 4-hour intervals. Arterial oxygen saturation (SaO_2) was measured by pulse oximetry and was kept ≥95% when possible. DO_2 was calculated at each interval using the equation:

$$DO_2 = 1.34 \times \text{hemoglobin} \times SaO_2 \times (CI \times 10).$$

The following parameters were recorded for each patient: admission temperature; base deficit; prothrombin and partial thromboplastin times; average PAOP over the 24-hour study period; APACHE II scores on postoperative days 1, 3, and 7;[25] lowest PaO_2/FIO_2 ratio for each of the first 3 postoperative days; average hemoglobin over the first 24 hours; and transfusion of blood products (red blood cells, fresh frozen plasma, platelets) during surgery and over the first 24 hours after surgery. Additional risk factors noted for each patient were the occurrence of perioperative myocardial infarction, as determined by electrocardiogram or serial cardiac isoenzymes; early renal failure (oliguria with a serum creatinine level ≥3 mg/dL within 48 hours); colon ischemia and the need for colectomy; and return to the operating room within 48 hours of aneurysm repair.

Outcome Parameters

Organ dysfunction was determined according to criteria modified from Goris et al.[26] Each of six organ systems (pulmonary, hepatic, renal, cardiac, hematologic, and central nervous system) was assigned a grade of 0 (no failure), 1 (moderate failure), or 2 (severe failure).[26] Due to difficulty in its evaluation in this patient population, the gastrointestinal tract was omitted from scoring. The highest value of organ dysfunction for each system was noted beginning on the third postoperative day.

Patients were assigned to one of two groups based on a retrospective analysis of mortality. There was a high mortality observed in patients with an organ dysfunction score ≥6, and no patient with an organ dysfunction score <6 died from organ failure. Therefore, outcome was compared between patients with an organ dysfunction score ≥6 (MOD) and those with a score <6 (no MOD).

Table 1. DISTRIBUTION OF ORGAN FAILURE*

	Total	MOD	No MOD	p Value
Pulmonary	47	16	31	NS
Hepatic	44	16	28	NS
Renal	23	14	9	<0.001
Cardiac	16	14	2	<0.001
Hematologic	15	11	4	<0.001
Central nervous system	13	12	1	<0.001

MOD = multiple organ dysfunction.
* Values are number of patients.

Table 2. NUMBER OF PATIENTS WITH ORGAN DYSFUNCTION

Organ Systems Failed	MOD	No MOD
0	0	6
1	0	9
2	0	13
3	2	8
4	5	2
5	6	0
6	6	0

MOD = multiple organ dysfunction.

Statistical Analysis

The time patterns of CI and DO_2 between patients with and without MOD were compared by repeated measures analysis of variance. Standard parametric (analysis of variance, t tests) and nonparametric (chi square, Wilcoxon) tests were used to compare age, hemodynamic and oxygen transport data, and risk factors for MOD between groups. Comparisons of predictive variables and MOD were made using logistic regression analysis.[27] Mean values are expressed along with the standard deviation of the mean. A level of $p < 0.05$ was considered significant.

RESULTS

Eighty-six patients were referred for ruptured AAA over the 8-year study period. Fourteen patients died during surgery, and 13 additional patients died within 24 hours after operation (31.4% mortality). Of the 59 patients who survived >24 hours, 57 had a pulmonary artery catheter placed; these 57 patients make up the study population.

The average patient age was 70.5 ± 8.3 years (range, 51 to 91 years). Forty-seven patients were transported via aeromedical transport from referral hospitals. Seventeen patients had free rupture of the aneurysm into the peritoneal space; the majority of the remaining patients had a large retroperitoneal hematoma. Fifty-eight percent of the patients with MOD had free rupture, compared to 42% of those without MOD (not significant).

Nineteen patients had an organ dysfunction score ≥6 (MOD) and 38 had a score <6 (no MOD). The pulmonary system was the most common organ failure. Of the 23 patients with renal failure, 9 had early renal failure and 15 needed hemodialysis (Table 1). Seven patients who developed MOD had early onset of renal failure, compared with two patients without MOD ($p = 0.004$). Patients with MOD had an average of 4.8 ± 1 failed organs, compared to 1.8 ± 1.1 organ failures for patients without MOD (Table 2, $p < 0.001$). Twenty patients died, 16 in the group with MOD (84% mortality) and 4 without MOD (11% mortality, $p < 0.001$). Three patients without MOD had chronic respiratory or renal failure and were allowed to die according to a living will or family wishes. The fourth patient, who had an organ dysfunction score of 1, had an unexplained death secondary to acute bronchospasm and cardiac arrest 13 days after operation.

The average length of stay was 73 ± 38 days for patients with MOD who survived, compared to 27 ± 19 days for survivors without MOD ($p = 0.02$). The average SICU length of stay was 38 ± 23 days for survivors with MOD and 13 ± 10 days for patients without MOD ($p = 0.04$).

All patients had an increase in both CI and DO_2 during the 24-hour study period. CI ($p < 0.05$) and DO_2 ($p < 0.0005$) for the first 16 hours were significantly lower in patients who developed MOD; the greatest difference between groups was noted at 8 hours ($p = 0.0001$, Figs. 1 and 2). There was no difference in CI or DO_2 between the two groups after 16 hours. A DO_2 <400 mL/min/m^2 at 8 hours was more strongly predictive of the development of MOD ($p = 0.003$; odds ratio, 6.07; 95% confidence interval, 1.8, 20.7) than DO_2 at any other time period. The distribution of DO_2 at 8 hours is shown in Figure 3.

There was no difference in age between patients with MOD (72.9 ± 8.4 years) and those without MOD (69.4 ±

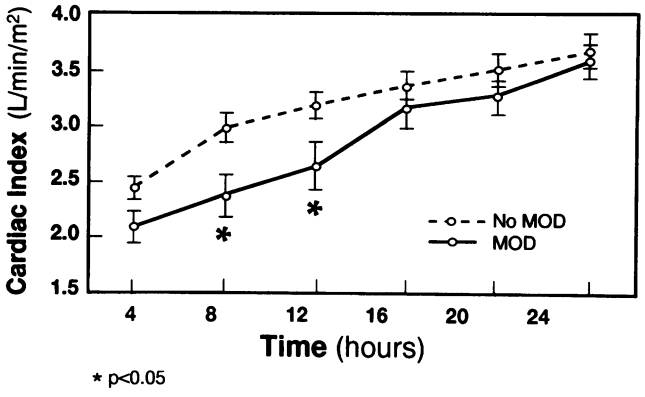

Figure 1. Cardiac Index and multiple organ dysfunction (MOD) (mean values ± SEM).

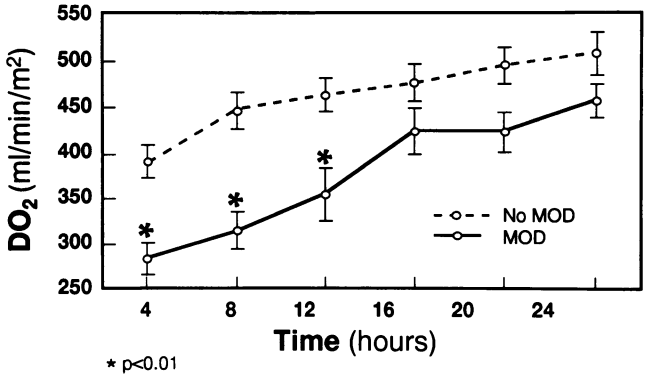

Figure 2. DO_2 and multiple organ dysfunction (MOD) (mean values ± SEM).

8.2 years). Patients in both groups had similar levels of hypothermia and base deficit on admission to the SICU (Table 3). APACHE II scores were higher on all days in patients who developed MOD. PaO_2/FIO_2 ratios were not significantly different between groups in the first 24 hours after surgery; however, patients who developed MOD showed a persistent impairment in the PaO_2/FIO_2 ratio, while patients without MOD had significant improvement in oxygenation on SICU days 2 ($p < 0.005$) and 3 ($p < 0.002$). Patients who developed MOD had a greater mean postoperative prothrombin time (17.7 ± 3.3 *vs.* 15.0 ± 2.2 seconds; $p = 0.0012$) and partial thromboplastin time (84.7 ± 57 *vs.* 49.0 ± 39 seconds; $p < 0.001$) and lower hemoglobin levels and received more units of red blood cells, fresh frozen plasma, and platelets than patients who did not develop MOD (Table 4). Average PAOP values were not different between groups; however, more patients who developed MOD received vasoactive drugs than those without MOD ($p = 0.01$). No patient with MOD sustained a perioperative myocardial infarction, but three patients in the group without MOD had this complication.

Sixteen patients returned to the operating room within 48 hours. The indications for reoperation were bleeding in five patients, colectomy in four, laparotomy for colon ischemia without resection in three, arterial thrombectomy in three, and acalculous cholecystitis in one. There was a similar incidence of reoperation in both groups of patients.

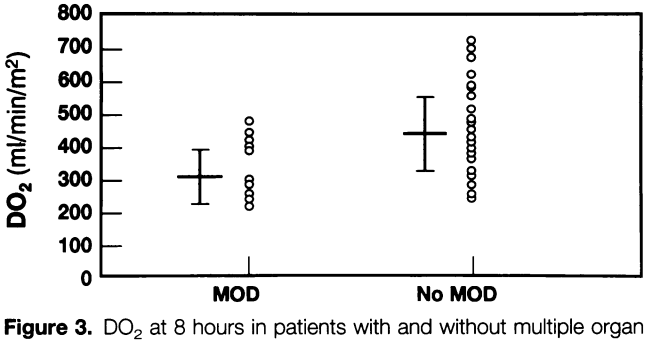

Figure 3. DO_2 at 8 hours in patients with and without multiple organ dysfunction (MOD).

Table 3. POSTOPERATIVE PHYSIOLOGIC PARAMETERS*

	MOD	No MOD	p Value
Temperature (°C)	34.1 ± 1.6	34.9 ± 1.3	NS
Base deficit	9.1 ± 3.5	7.3 ± 4.4	NS
APACHE II score			
Day 1	20.5 ± 4.6	16.3 ± 4.6	0.0036
Day 3	17.6 ± 4.3	12.0 ± 3.3	0.0001
Day 7	15.6 ± 4.5	11.3 ± 3.8	0.0016
PaO_2FIO_2			
Day 1	134.2 ± 56.1	165.4 ± 57.5	NS
Day 2	134.0 ± 45.7	178.7 ± 54.2	0.0046
Day 3	131.8 ± 47.8	187.8 ± 61.4	0.0014

MOD = multiple organ dysfunction; NS = not significant.
* Values are mean ± standard deviation.

Fourteen patients developed colon ischemia, six of whom required colectomy. Four patients underwent colectomy on the first postoperative day, the other two patients on postoperative days 3 and 4. Four of the patients who required colectomy subsequently developed MOD. There was no relation between the occurrence of colon ischemia and the development of MOD. Although there was no difference in DO_2 between patients with and without colon ischemia, DO_2 at 8 hours was significantly lower in the six patients who had colon ischemia treated with colectomy ($p = 0.015$).

Parameters that were significantly different between groups on univariate analysis were used as independent variables in a logistic regression analysis to determine the probability that a patient would develop MOD. DO_2 ($p = 0.0064$), early onset of renal failure ($p = 0.0064$), and the total number of red blood cells transfused ($p = 0.008$) were associated with the occurrence of MOD by this analysis.

Table 4. PARAMETERS OF RESUSCITATION*

	MOD	No MOD	p Value
Hemoglobin (g/dL)	10.6 ± 1.4	11.8 ± 1.6	0.014
Blood products (units)			
Red blood cells	16.5 ± 8.0	8.9 ± 5.0	0.0003
Fresh frozen plasma	9.3 ± 5.0	5.2 ± 3.7	0.0016
Platelets	12.9 ± 8.3	7.5 ± 9.3	0.01
PAOP (mmHg)	14.6 ± 4.6	14.7 ± 3.7	NS
Patients who received vasoactive agents (percentage of patients)	12 (63%)	11 (29%)	0.013

MOD = multiple organ dysfunction; PAOP = pulmonary artery occlusion pressure; NS = not significant.

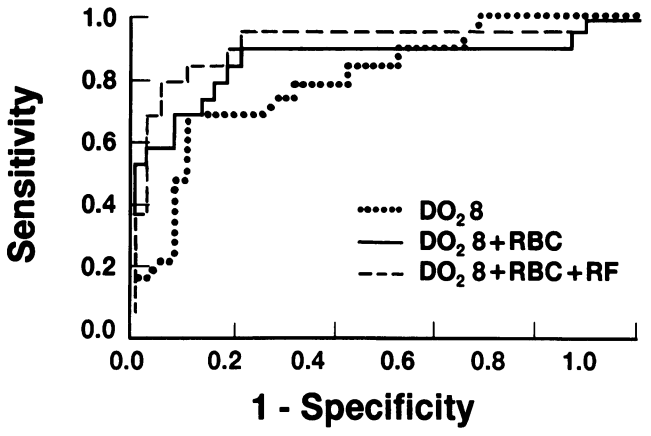

Figure 4. Receiver operating characteristic curves based on three logistic regression models: probability of multiple organ dysfunction (MOD) based on DO_2 at 8 hours alone; DO_2 and total red blood cells transfused; and DO_2, total red blood cells transfused; and the development of early renal failure (RF).

Receiver operating characteristic curves for the parameters of the logistic regression model are shown in Figure 4.

DISCUSSION

Patients with ruptured AAA are at high risk for perioperative death and postoperative complications.[1–16] Most previous studies have focused on preoperative risk factors such as age,[3,5,8,10] hypotension,[5–7,9,11] cardiac arrest,[4,5,9–11] and coma,[4,10] which greatly influence intraoperative or early mortality. Intraoperative risk factors, including the presence of free rupture of the aneurysm into the peritoneum,[3] blood loss,[5,6,9,11] and site and duration of aortic cross-clamping,[7] also have been demonstrated to affect outcome. Least studied are postoperative factors such as hemodynamic instability,[10] colon ischemia,[13–15] and the occurrence of organ failure.[2,4–7,9–11]

Patients with ruptured AAA share common risk factors such as underlying cardiovascular disease, perioperative hemodynamic perturbations (blood loss, hypovolemia, shock), and therapeutic interventions (fluids, blood products, vasoactive agents). The postoperative evaluation of oxygen transport of these patients can be performed without the influence of other factors that can affect oxygen consumption and DO_2, as may occur in other populations of critically ill patients (*e.g.*, sepsis, fever, nutrition). In the present study, all patients were monitored immediately after aneurysm repair and oxygen transport patterns were measured at uniform time intervals. All patients were resuscitated to standard clinical parameters of perfusion, including PAOP, and all received ventilatory support and sedation.

In normal circumstances, the delivery of oxygen greatly surpasses the usual levels of oxygen consumption ($\dot{V}O_2$). When $\dot{V}O_2$ is elevated, as in exercise, compensatory increases in DO_2 and tissue oxygen extraction will balance the increase in $\dot{V}O_2$. When $\dot{V}O_2$ exceeds these normal compensatory mechanisms, oxygen debt and anaerobic metabolism result.[28] Similarly, when DO_2 is subnormal, oxygen extraction increases to maintain $\dot{V}O_2$. A decrease in DO_2 below a critical level required to maintain $\dot{V}O_2$ results in anaerobic metabolism.[29]

Observational studies in heterogeneous populations of critically ill surgical patients have demonstrated improved outcomes in those who attain higher values of CI and DO_2.[17–19] The rapidity with which critically ill patients augment oxygen transport also has been associated with improved survival. In two studies of high-risk surgical patients, reversal of oxygen debt within 8 to 12 hours after operation was associated with a higher likelihood of survival without the development of organ failure, in contrast to persistent oxygen debt, which often resulted in organ failure or death.[20,21] Similar observations have been made in other studies of medical and surgical patients, severely injured patients, and those who have undergone orthotopic liver transplantation.[22–24]

The present study demonstrates that a population of patients with ruptured AAA also had worse outcome when there was a delay in achieving an adequate CI and DO_2. All patients demonstrated an improvement in oxygen transport over the course of the initial 24 hours after operation. However, CI and DO_2 in the first 16 hours were significantly lower in patients who developed MOD, with the most significant difference occurring at 8 hours after surgery. Patients without MOD attained higher CI and DO_2 within 4 to 8 hours after admission to the SICU; patients who developed MOD did not reach comparable levels of CI and DO_2 for an additional 8 to 12 hours.

One study of high-risk surgical patients has identified a CI of 4.5 L/min/m^2 and a DO_2 of 650 mL/min/m^2 as predictive of survival.[19] The achievement of these "supranormal" levels of CI and DO_2 during resuscitation has resulted in an inconsistent improvement in the outcome of critically ill patients.[30–40] Patients in the present study did not spontaneously achieve supranormal levels of CI or DO_2. In fact, a DO_2 level of only 450 mL/min/m^2 at 8 hours after operation was predictive of a good outcome without the occurrence of MOD.

Efforts to augment DO_2 with optimization of cardiac function may be limited in patients with ruptured AAA due to patient age and underlying cardiac disease. Patients in the present study were older (mean age, 70.5 years) than in most of the previous studies (mean age, 41 to 60 years).[17–24,31,32,34–36] CI has been shown to decrease progressively with increasing age,[19] although the ability of elderly surgical patients to achieve a higher CI correlates with survival. Patients with sepsis, acute respiratory distress syndrome, and hypovolemic shock who are >50 years old are also less likely to generate a DO_2 of >600 mL/min/m.[2,41] Besides being an inherently older population, patients with AAA have a high incidence of coronary artery disease, which can impair their ability to compensate for the hemodynamic instability and intravascular volume changes that occur in the perioperative period.[16]

Patients who developed MOD had a lower CI despite the minimal difference in the postoperative physiologic parameters between groups. There was no difference in base deficit between patients with and without MOD. The level of hypothermia, a previously identified risk factor for organ dysfunction in patients undergoing elective AAA repair,[42] was similar among groups. Cardiac dysfunction of the patients who developed MOD did not appear to be due to differences in resuscitation. PAOP was similar between groups, suggesting that intravascular volumes were equal. Patients who had a lower CI (and subsequently developed MOD) received more inotropic and vasoactive support, which may reflect the severity of perioperative cardiac dysfunction in this group. Although the occurrence of myocardial infarction has been correlated with poor outcome in previous reports,[7,10] perioperative myocardial infarction was not associated with the development of MOD in the present group of patients.

In addition to direct enhancement of cardiac function, DO_2 can be augmented by increasing the amount of circulating hemoglobin and by ensuring a high oxygen saturation of hemoglobin. The perioperative sequelae of ruptured AAA, however, may compromise the ability to augment these elements of DO_2. In the present study, patients who developed MOD had greater derangements of coagulation on admission to the SICU. Transfusion requirements were higher for these patients and average hemoglobin levels were lower during the first 24 hours after operation. The PaO_2/FIO_2 ratio was low on the first postoperative day in all patients. These disturbances in oxygenation may impair the optimal saturation of hemoglobin with oxygen. Hence, it is likely that the intrinsic cardiac dysfunction of this population of patients, in addition to continued blood loss and respiratory dysfunction, resulted in an inadequate DO_2, which may have contributed to the development of MOD.

The purpose of the present study was to evaluate outcome in terms of organ dysfunction. Intensive care unit mortality has been correlated with the number of failed organs in various situations.[43] Although definitions of MOD have varied,[26,43–45] the intent of such classifications is to correlate the number of failed organs and the severity of organ dysfunction with outcome. Accordingly, "MOD syndrome" has been used to describe a continuum of organ failure that is related directly to an insult, such as sepsis or injury, or that occurs as a consequence of the host response.[43] Isolated organ failure, such as renal failure,[4,7,10,11] respiratory failure,[2,4,7] and cardiac failure,[10] has been demonstrated to correlate with mortality in patients with ruptured AAA. Patients in this study who developed MOD had a mortality of 84%, which is similar to previous reports that identify failure of two or more organ systems as a predictor of high mortality in this patient population.[9,11]

Low DO_2 was a risk factor for transmural colon ischemia. The etiology of colon ischemia may be related to microvascular hypoperfusion. Some reports have implicated hypotension in the development of this condition.[13,15] Ischemia is not necessarily prevented with reimplantation of the inferior mesenteric artery.[15] The development of colon ischemia after ruptured AAA has been correlated with decreased cardiac output during the early postoperative period.[14] In the present study, there was no difference in DO_2 among all patients who developed colon ischemia; however, patients who developed transmural necrosis had DO_2 values that were significantly lower at 8 hours. Although anatomic or hemodynamic factors in the perioperative period may contribute to the development of mucosal ischemia, inadequate DO_2 in the early postoperative period is associated with the development of transmural ischemia of the colon.

The presence of low DO_2, early renal failure, and a high transfusion requirement of red blood cells identifies patients at greater risk for the development of MOD. Low DO_2 in the early postoperative period should prompt interventions to increase DO_2, which may result in a lower incidence of MOD. The administration of fluids to achieve a higher PAOP (range of 18 to 20 mmHg), the use of inotropic support, transfusion to hemoglobin levels of 12 to 15 g/dL, and optimization of oxygenation should improve DO_2.

CONCLUSIONS

This study describes the postoperative hemodynamic patterns of patients with ruptured AAA and identifies DO_2 in the early postoperative period as an important predictor for MOD. These findings concur with other studies of heterogeneous populations of critically ill and injured patients that have shown improved outcome with early augmentation of DO_2. These patients should not be expected to achieve supranormal goals, but those at risk for MOD may benefit from more aggressive support of cardiac function, higher levels of hemoglobin, and maintenance of 100% oxygen saturation of hemoglobin. Modification of these parameters beyond current conventional goals could potentially affect the outcome in these patients.

Acknowledgments

The authors thank Ruth Lange, RN, for her assistance with data collection and Joan Hagen for her assistance with statistical analysis.

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Discussion

DR. MARTIN ALEXANDER CROCE (Memphis, Tennessee): Thank you, Dr. Nunn, Dr. Copeland, Members, and Guests. Dr. Malangoni and his colleagues should be commended for doing this study. When an investigator chooses any of the variables of cardiopulmonary physiology as a focus for studying a large number of critically ill patients, the difficulty in obtaining interpretable data is immense, because there are so many variables.