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Intraoperative Hypotension and 30-D Mortality After Liver Transplantation

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Background. Intraoperative hypotension (IOH) is common and associated with mortality in major surgery. Although patients undergoing liver transplantation (LT) have low baseline blood pressure, the relation between blood pressure and mortality in LT is not well studied. We aimed to determine mean arterial pressure (MAP) that was associated with 30-d mortality in LT. **Methods.** We performed a retrospective cohort study. The data included patient demographics, pertinent preoperative and intraoperative variables, and MAP using various metrics and thresholds. The endpoint was 30-d mortality after LT. **Results.** One thousand one hundred seventy-eight patients from 2013 to 2020 were included. A majority of patients were exposed to IOH and many for a long period. Eighty-nine patients (7.6%) died within 30 d after LT. The unadjusted analysis showed that predicted mortality was associated with MAP <45 to 60 mm Hg but not MAP <65 mm Hg. The association between MAP and mortality was further tested using adjustment and various duration cutoffs. After adjustment, the shortest durations for MAPs <45, 50, and 55 mm Hg associated with 30-d mortality were 6, 10, and 25 min (odds ratio, 1.911, 1.812, and 1.772; 95% confidence interval, 1.100-3.320, 1.039-3.158, and 1.008-3.114; $P=0.002$, 0.036, and 0.047), respectively. Exposure to MAP <60 mm Hg up to 120 min was not associated with increased mortality. **Conclusion.** In this large retrospective study, we found IOH was common during LT. Intraoperative MAP <55 mm Hg was associated with increased 30-d mortality after LT, and the duration associated with postoperative mortality was shorter with lower MAP than with higher MAP.

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INTRODUCTION

Intraoperative hypotension (IOH) is a hemodynamic derangement frequently encountered by surgeons and anesthesia providers and is associated with postoperative

complications and mortality.¹⁻³ Studies in orthopedic, vascular, thoracic, abdominal, and other noncardiac surgeries have shown that exposure to mean arterial pressure (MAP) <60 to 70 mm Hg is associated with postoperative complications and mortality.^{1,3-8} Thus, many investigators and experts consider this MAP range a critical pressure and recommend maintaining MAP above 60 to 70 mm Hg during noncardiac surgery.^{1,6}

Patients with cirrhosis have unique hemodynamic pathophysiology characterized by low systemic vascular resistance (SVR) and low baseline blood pressure. Although hypotension can cause poor organ perfusion in all surgical patients, the relationship between intraoperative MAP and postoperative mortality in liver transplantation (LT) is not well studied. Previous investigations in LT are severely limited by small patient cohorts, low statistical power, and incomplete analysis.^{9,10} Findings from noncardiac surgery cannot be directly applied to LT because of distinctly different patient populations and surgeries. Developing intraoperative hemodynamic management strategies for LT patients relies on a better understanding of intraoperative blood pressure and its relationship with postoperative mortality.

The objective of this retrospective cohort study was to examine the prevalence and severity of IOH using various MAP indices and to determine if there was an association between certain MAPs and 30-d mortality in adult patients undergoing LT.

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MATERIALS AND METHODS

The study was approved by the institutional review board of the University of California, Los Angeles (UCLA), and written informed consent was waived. The perioperative data warehouse at UCLA has been established since 2013 to prospectively collect data on patient-related risk, operative procedures, and mortality.¹¹ Patients who underwent LT at Ronald Reagan UCLA Medical Center after the establishment of the data warehouse were included in the study.

We extracted data from the UCLA perioperative data warehouse and included patients who were identified as “adult liver transplant” in “procedure name” and had a primary Current Procedural Terminology code of 47135. The data were last accessed in November 2020. Exclusion criteria included patients who were 16 y old or younger. Extracted data included patient demographics, American Society of Anesthesiologists (ASA) class, etiology of liver disease, baseline laboratory values, and preoperative interventions including the use of vasoactive agents, hemodialysis, and mechanical ventilation. Pretransplant model for end-stage liver disease sodium (MELD-Na) score was calculated by using the highest values of the following laboratory tests: total bilirubin, international normalization ratio, serum creatinine (if not on hemodialysis) or status of hemodialysis, and serum sodium within 7 d before LT. Intraoperative data included the volume of transfused blood products, the use of vasopressors, and their concentrations.

Measurement of MAP was via the intra-arterial catheter and recorded every minute during LT surgery. MAP was extracted into the following categories: MAP before anesthesia induction, average MAP from hour 1 to hour 8 during LT surgery, the lowest MAP during the entire LT surgery, and durations (in minutes) of MAP at various thresholds (<45, 50, 55, 60, and 65 mm Hg). The endpoint of the study was 30-d all-cause mortality after LT.

Patient exposure to IOH measured by various MAP thresholds during LT was assessed. The relationships between 30-d mortality and various MAP categories were analyzed. The cumulative minutes on various MAP thresholds were further analyzed for the association with mortality using unadjusted and adjusted analyses. The cumulative minutes were categorized to 5, 10, and 20 min initially. To identify the shortest duration that was associated with mortality, minutes then increased beyond 20 min or decreased if needed. Baseline demographic, preoperative, and intraoperative variables that showed significant association with 30-d mortality in the multivariable logistic regression analysis were used for adjustment in the final analysis.

Data were presented as mean (\pm SD) or median (interquartile range) for continuous variables when appropriate. The percentage was used for categorical variables. Univariable analyses for patient demographics and preoperative and intraoperative variables were performed by using Student *t*, Mann-Whitney *U*, or chi-square tests when appropriate. Stepwise (forward and backward) multivariable logistic regression was used. Odds ratio and 95% of confidence interval were calculated in the logistic regression. All tests were performed using SPSS, version 26.0 (IBM, Armonk, NY), with $P < 0.05$ considered to be statistically significant.

RESULTS

Data extraction generated 1178 adult LT patients for the study. Mean age was 54.9 (± 12.1), male patients were 59.7%, ASA

TABLE 1.
Patient demographics

Variables	M (\pm SD) or %
Age (y)	54.9 \pm 12.1
Sex (male, %)	59.7
Weight (kg)	71.4 \pm 19.0
Height (cm)	168.9 \pm 10.7
Body mass index	24.9 \pm 5.8
ASA class 4 or higher	81.8
MELD-Na score	33.8 \pm 12.7
Preoperative sodium (mmol/L)	136.9 \pm 4.6
Preoperative total bilirubin (mg/dL)	17.0 \pm 15.5
Preoperative international normalization ratio	2.1 \pm 0.7
Preoperative creatinine (mg/dL)	1.4 \pm 1.1
Preoperative HGBA1C	5.2 \pm 1.0
Diabetic mellitus	12.1
Hepatitis B	3.1
Hepatitis C	10.0
Nonalcoholic steatohepatitis	20.2
Alcoholic cirrhosis	24.8
Hepatic neoplasm	21.1
Preoperative requirement of ventilation	15.6
Preoperative use of vasopressor	26.4
Preoperative requirement of renal replacement	30.5

ASA, American Society of Anesthesiologists; MELD-Na, model for end-stage liver disease sodium.

classes 4 or higher were 81.8%, mean MELD-Na score was 33.8 (± 12.7), and etiology of liver disease included alcoholic cirrhosis (24.8%) and nonalcoholic steatohepatitis (20.2%) (Table 1).

As shown in Figure 1, IOH defined by various MAP thresholds was common during LT surgery. A majority of the patients (71.2%–99.4%) were exposed to low MAPs (<45–65 mm Hg) for at least 1 min. The percentage of patients who were exposed to low MAPs (<45–65 mm Hg) for 5 and 10 min was 29.5% to 98.0% and 16.1% to 96.9%. Furthermore, a significant number of the studied patients were exposed to prolonged duration of IOH. For example, 64.5% to 88.9% and 41.8% to 71.1% of patients were exposed to at least 30 and 60 min of low MAPs <60 to 65 mm Hg; 42.7% of patients experienced MAP <65 mm Hg for 120 min.

A total of 89 patients (7.6%) died within 30 d after LT. Univariable analyses found that preinduction MAP and MAPs measured hourly during LT surgery were not significantly associated with postoperative mortality (Table 2). Median durations for the MAP thresholds of <45, 50, 55, and 60 mm Hg were significantly longer in the mortality group. Of note, there was no significant difference in durations for MAP <65 mm Hg between patients with and without mortality.

The predicted probabilities of mortality for patients who were exposed to various durations of MAP thresholds were assessed by univariable logistic regression (Figure 2). The predicted probabilities of mortality increased with both lower MAP thresholds and longer durations of exposure. The predicted probabilities of mortality were significantly associated with MAPs <45 to 60 mm Hg (odds ratio, 1.003–1.013; 95% confidence interval, 1.001–1.023; and $P = 0.002$ – 0.006). In contrast, exposure to MAP <65 mm Hg was not significantly associated with mortality (Figure 2).

Unadjusted associations between mortality and exposure to various MAP thresholds and durations were initially tested

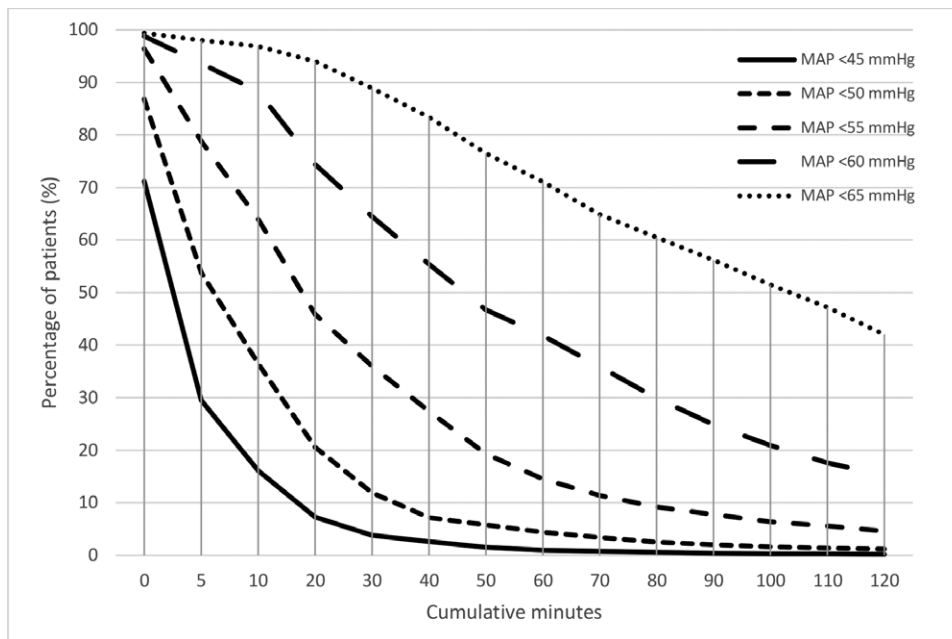


FIGURE 1. Percentage of patients in a relation with cumulative minutes spent on various MAP thresholds. MAP, mean arterial pressure.

TABLE 2.
The univariable relationship between MAPs and 30-d mortality

	Mortality		P
	No (n=1089)	Yes (n=89)	
Preinduction MAP	83.9±16.1	82.5±15.1	0.554
MAP during first hour of surgery	72.4±9.6	70.7±9.6	0.109
MAP during second hour of surgery	73.5±9.5	71.9±9.5	0.133
MAP during third hour of surgery	76.0±10.4	74.1±11.7	0.165
MAP during fourth hour of surgery	77.4±10.0	76.1±13.1	0.397
MAP during fifth hour of surgery	76.6±9.9	75.0±11.5	0.263
MAP during sixth hour of surgery	74.6±9.1	74.6±11.7	0.986
MAP during seventh hour of surgery	74.4±8.6	73.4±10.8	0.456
MAP during eighth hour of surgery	75.3±8.5	73.1±11.2	0.152
Minutes on the MAP thresholds			
<45 mm Hg	2.0 (0–6.0)	7.0 (2.0–17.0)	<0.001
<50 mm Hg	6.0 (2.0–16.0)	19.0 (3.5–34.5)	<0.001
<55 mm Hg	17.0 (7.0–41.0)	43.0 (12.5–64.0)	<0.001
<60 mm Hg	45.0 (20.0–87.0)	77.0 (35.5–120.5)	0.005
<65 mm Hg	102.0 (52.0–164.0)	129.0 (70.0–186.5)	0.086

Data present in either mean ± SD or median (interquartile range). MAP, mean arterial pressure.

(Table 3). The shortest durations of exposure to MAP <45 and 50 mm Hg that were associated with mortality were both 5 min. Exposure to MAP <55 mm Hg required 10 min to reach statistical significance. Exposure to MAP <60 mm Hg ≥20 min was not associated with 30-d mortality. Extended exposure (≥30 min) to MAP <60 mm Hg was associated with postoperative mortality.

Selection of baseline characteristics and preoperative, and intraoperative variables except for blood pressure measurements for adjustment were first assessed by univariable and then multivariable analyses. Results of univariable

and multivariable analyses are shown in Table 4. Variables including body weight, body mass index, MELD-Na scores, ASA class scores, preoperative vasopressors, hemodialysis, mechanical ventilation, transfusion of red blood cells, fresh frozen plasma, platelet, and vasopressin were associated with postoperative mortality. Multivariable analysis showed that 3 non-MAP variables, pretransplant MELD-Na scores >25, pretransplant requirement of vasopressors, and intraoperative red blood cells transfusion, were significantly associated with 30-d mortality; these were used for adjustment in the final analyses. In addition, the shortest duration that was associated with mortality was sought by using 1-min increments.

Results of multivariable logistic regression using the 3 significant variables and 1-min increments are presented in Table 5. Exposure to MAP <45 mm Hg for ≥6 min, MAP <50 mm Hg for ≥10 min, or MAP <55 mm Hg to ≥25 min showed a significant association with postoperative mortality. One minute less than these durations failed to show the positive association. Exposure to MAP <60 mm Hg up to 120 min was not associated with mortality.

DISCUSSION

In this retrospective study of 1178 adult patients, we found that IOH during LT surgery was common and prolonged. This particular pattern of IOH encountered during LT is likely multifactorial in etiology. A low SVR state with decreased baseline blood pressure is common in LT patients and likely plays a major role. The complex pathophysiological hemodynamic changes often occur in patients with cirrhosis. Portal hypertension in these patients results in splanchnic and peripheral vasodilation, increased levels of circulating vasodilators, resistance to vasoconstrictors, extensive arteriovenous, and portal-caval shunting.¹² In addition to these preexisting factors, complex surgical procedure with manipulation of the major vascular structures, massive blood loss, severe coagulopathy, and allograft dysfunction may contribute to the development of prolonged IOH during LT.¹³

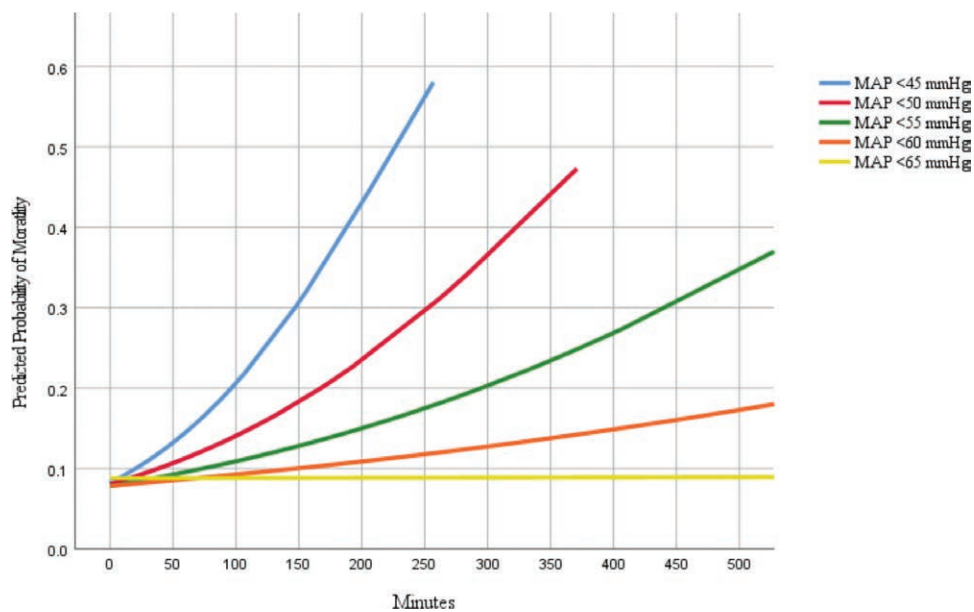


FIGURE 2. Predicted probability of 30-d mortality in a relationship with minutes spent on various MAP thresholds. Graphs were constructed by univariate logistic regression. Odds ratios, 95% CI, and *P* values for MAP <45, 50, 55, 60, and 65 mm Hg were 1.013, 1.004-1.023, and 0.004; 1.008, 1.003-1.014, and 0.002; 1.005, 1.002-1.008, and 0.002; 1.003, 1.001-1.005, and 0.006; and 1.002, 1.000-1.003, and 0.087, respectively. CI, confidence interval; MAP, mean arterial pressure.

TABLE 3.

Univariable analysis on the relationship between the minutes spent on various MAP thresholds and 30-d mortality

MAP thresholds (mm Hg)	Minutes	Univariable analysis		
		OR	95% CI	<i>P</i>
MAP <45	≥5	3.048	1.962-4.735	<0.001
	≥10	3.905	2.490-6.126	<0.001
	≥20	3.964	2.285-6.877	<0.001
MAP <50	≥5	2.014	1.250-3.247	0.004
	≥10	3.202	2.035-5.037	<0.001
	≥20	3.684	2.367-5.734	<0.001
MAP <55	≥5	1.493	0.798-2.795	0.210
	≥10	2.030	1.192-3.457	0.009
	≥20	2.801	1.755-4.471	<0.001
MAP <60	≥5	1.243	0.441-3.502	0.681
	≥10	1.281	0.605-2.714	0.517
	≥20	1.600	0.903-2.835	0.107
	≥30	2.064	1.225-3.479	0.006

CI, confidence interval; MAP, mean arterial pressure; OR, odds ratio.

Another major finding of our study is that, despite frequent and prolonged episodes of IOH, LT patients in our study are relatively resilient to early postoperative mortality. First, MAP that is associated with postoperative mortality in our LT patients is lower than that reported in noncardiac patients.^{1,3} Exposure to MAP <60 to 70 mm Hg, which is widely considered an intraoperative risk for mortality and complications in noncardiac surgery, is not associated with mortality in this study. The risk of 30-d mortality can be only observed after exposure to lower MAPs (<55 mm Hg and less) during LT. Furthermore, the duration of various MAP thresholds that are associated with mortality appear to be longer in our study than in the previous studies. For example, exposure to MAP <55 mm Hg requires 25 min or longer to reach the statistical significance for postoperative 30-d mortality in our study.

This duration of IOH is longer than previously reported.^{5,9} The underlying chronic low SVR state with decreased baseline blood pressure in conjunction with the compensatory increases in cardiac output may preserve tissue perfusion despite relatively low blood pressure and contribute to the relative resilience to IOH that we observed in our LT patients.

Other factors may also explain why our patients tolerated prolonged IOH without experiencing increased mortality. Our patients have high MELD-Na scores, which indicate more severe liver disease with lower baseline SVR, and preserved tissue perfusion.¹⁴ Before listing for transplant, LT candidates in our institution usually undergo a comprehensive preoperative cardiac evaluation. Patients with advanced cardiac disease undergo pretransplant interventions for conditions such as obstructive coronary artery disease or aortic stenosis before transplant. Additionally, patients with advanced cardiac disease not amenable to intervention and those with multiple comorbidities may be excluded from candidacy if they are deemed at excessive perioperative risk. This rigorous screening may have played a role in our study's results because patients with robust cardiac function are likely to demonstrate better resilience to prolonged IOH. Perioperative beta-blockade may also have played a role in our findings. Beta-blockade is widely used in patients with end-stage liver disease for the prevention of variceal bleeding. Preoperatively administered beta-blockade may contribute to the development of IOH during LT surgery; however, it has not been associated with postoperative mortality in LT patients.¹⁵

A good understanding of the degree and duration of hypotension that are associated with mortality has important clinical implications. When IOH reaches a dangerously low level, prompt treatment is necessary. Blood transfusion and vasopressor therapy are typically used to resuscitate LT patients. However, over treatment of IOH has been associated with risks as well, as we showed in this study that both blood transfusion and vasopressor were ironically independent risk factors for postoperative mortality. Blood transfusion

TABLE 4.**Preoperative and intraoperative variables by 30-d mortality****Univariable analysis**

Variables	Mortality		P
	No (n = 1089)	Yes (n = 89)	
Age (y)	54.8 ± 12.2	55.6 ± 10.6	0.492
Sex (male, %)	60.3	52.8	0.163
Weight (kg)	70.7 ± 18.2	79.0 ± 25.3	0.003
Height (in)	66.5 ± 4.2	66.0 ± 4.6	0.363
Body mass index	24.7 ± 5.6	27.9 ± 7.6	0.001
ASA class 4 or higher	80.5	95.5	0.001
MELD-Na score	33.4 ± 12.9	39.5 ± 7.7	<0.001
MELD-Na score ≥25	75.0	97.0	<0.001
Hepatitis B	2.9	4.5	0.689
Hepatitis C	9.9	11.2	0.695
Nonalcoholic steatohepatitis	20.4	16.9	0.661
Alcoholic cirrhosis	25.3	18.0	0.124
Hepatic neoplasm	22.4	13.5	0.051
Preoperative requirement of ventilation	14.7	27.0	0.002
Preoperative requirement of renal replacement	29.8	40.4	0.036
Preoperative use of vasopressor	24.9	46.1	<0.001
Preoperative sodium (mmol/L)	136.9 ± 4.6	137.2 ± 4.8	0.686
Preoperative total bilirubin (mg/dL)	16.5 ± 15.2	23.4 ± 17.0	0.004
Preoperative international normalization ratio	2.1 ± 0.7	2.2 ± 0.6	0.216
Preoperative creatinine (mg/dL)	1.4 ± 1.1	1.2 ± 0.9	0.055
Preoperative HGBA1C	5.2 ± 1.0	5.1 ± 0.9	0.636
Phenylephrine bolus (µg)	690.9 ± 715.0	677.8 ± 711.3	0.906
Vasopressin bolus (unit)	9.3 ± 9.1	15.7 ± 17.2	0.002
Maximum vasopressin infusion rate (units/h)	3.6 ± 1.4	4.0 ± 1.5	0.026
Maximum norepinephrine rate (µg/kg/min)	0.5 ± 2.4	0.5 ± 0.3	0.817
Red blood cells (unit)	20.3 ± 18.8	32.5 ± 30.1	<0.001
Fresh frozen plasma (unit)	21.9 ± 19.7	32.3 ± 29.8	0.002
Platelet (ml)	363.0 ± 355.8	504.6 ± 479.6	0.008
Cryoprecipitate (ml)	233.6 ± 230.9	270.4 ± 258.3	0.196
Surgery time (min)	611.7 ± 140.6	551.1 ± 248.8	0.025

Multivariable logistic regression

Variables	Odds ratio	95% CI	P
MELD-Na score >25	6.413	1.513-27.175	0.012
Pretransplant use of vasopressors	2.282	1.359-3.831	0.002
Red blood cells (unit)	1.018	1.009-1.028	<0.001

ASA, American Society of Anesthesiologists; CI, confidence interval; MELD-Na, model for end-stage liver disease sodium.

can be associated with acute lung injury, renal dysfunction, and transfusion-related reactions. Transfusion-induced acute hypocalcemia and hyperkalemia may further worsen IOH.¹⁶ Massive transfusion may lead to volume overload, increased central venous pressure, and reduced perfusion to the newly transplanted liver.¹⁷ Vasopressor therapy induces vasoconstriction, which may compromise tissue perfusion at a microvascular level, even when blood pressure is within a normal range. Vasopressor has been associated with cardiac dysfunction such as stress-induced (Takotsubo) cardiomyopathy in LT patients.¹³ Vasopressors also have numerous pleiotropic effects that are not easy to predict or measure, and some of these may be deleterious.¹⁸ Therefore, a delicate balance of therapies is required for the hemodynamic management of LT patients.

There are several important limitations in our study. It is a retrospective study with inherent shortcomings. Our study is based on single-center data that may not be generalizable to other centers with different patient populations and

practices. The UCLA perioperative data warehouse stores large amounts of perioperative data; however, some data points that may have impacted mortality may have not been obtained with our method. Cumulative minutes of IOH are the primary MAP variable used for analysis in our study, which is consistent with other previous studies. However, it is not known if this method is the best to characterize IOH during LT. Nevertheless, the durations of MAPs associated with mortality may be useful for clinicians managing hypotension during LT. Finally, we only analyzed the relationship between intraoperative MAP and 30-d mortality. Therefore, it cannot be assumed that the MAP thresholds we found for mortality can be extrapolated to postoperative complications such as acute kidney injury or myocardial injury.

In summary, in this large retrospective study, IOH is found to be common and prolonged during LT surgery. It appears that LT patients are relatively resilient to IOH. Intraoperative MAP <55 mm Hg seems to be a critical threshold to associate with increased 30-d mortality in LT. Our findings provide

TABLE 5.**Multivariable analysis on the relationship between minutes of MAP thresholds and 30-d mortality**

MAP thresholds (mm Hg)	Duration (min)	Multivariable analysis		
		OR	95% CI	P
MAP <45	≥5	1.634	0.943-2.833	0.080
	≥6	1.911	1.100-3.320	0.022
	≥10	1.964	1.100-3.505	0.022
MAP <50	≥5	1.156	0.645-2.069	0.626
	≥9	1.670	1.005-1.025	0.074
	≥10	1.812	1.039-3.158	0.036
MAP <55	≥5	0.676	0.322-1.420	0.301
	≥10	1.105	0.576-2.117	0.764
	≥20	1.508	0.855-2.660	0.156
	≥24	1.672	0.952-2.936	0.074
	≥25	1.772	1.008-3.114	0.047
MAP <60	≥5	0.389	0.127-1.187	0.097
	≥10	0.582	0.247-1.374	0.217
	≥20	0.778	0.391-1.552	0.477
	≥60	1.154	0.667-1.996	0.609
	≥120	0.966	0.498-1.873	0.918

Multivariable analyses were performed by including 1 MAP threshold/duration combo and 3 independent non-MAP risk factors (pretransplant MELD-Na scores ≥25, pretransplant requirement of vasopressors and intraoperative RBC transfusion) for 30-d mortality. CI, confidence interval; MAP, mean arterial pressure; MELD-Na, model for end-stage liver disease sodium; OR, odds ratio; RBC, red blood cells.

important insights into intraoperative hemodynamics and may contribute to the further development of management strategies in LT patients.

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