

The Hyperglycemic Response to Major Noncardiac Surgery and the Added Effect of Steroid Administration in Patients With and Without Diabetes

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BACKGROUND: The pattern and magnitude of the hyperglycemic response to surgical stress, the added effect of low-dose steroids, and whether these differ in diabetics and nondiabetics remain unclear. We therefore tested 2 hypotheses: (1) that diabetics show a greater increase from preoperative to intraoperative glucose concentrations than nondiabetics; and (2) that steroid administration increases intraoperative hyperglycemia more so in diabetics compared with nondiabetics.

METHODS: Patients scheduled for major noncardiac surgery under general anesthesia were enrolled and randomized to preoperative IV 8 mg dexamethasone or placebo, stratified by diagnosis of diabetes. Patients were part of a larger underlying trial (the Dexamethasone, Light Anesthesia and Tight Glucose Control [DeLiT] Trial). IV insulin was given when glucose concentration exceeded 215 mg/dL. The primary outcome measure was the change in glucose from the preoperative to maximal intraoperative glucose concentration. We also report the time-dependent pattern of intraoperative hyperglycemia.

RESULTS: Ninety patients (23% with diabetes) were randomized to dexamethasone, and 95 (29% with diabetes) were given placebo. The mean \pm SD change from preoperative to maximal intraoperative glucose concentration was 63 ± 69 mg/dL in diabetics and 72 ± 45 mg/dL in nondiabetics. The mean covariable-adjusted change (95% confidence interval) in nondiabetics was 29 (13, 46) mg/dL more than in diabetics ($P < 0.001$). For all patients combined, mean glucose increased slightly from preoperative to incision, substantially from incision to surgery midpoint, and then remained high and fairly stable through emergence, with nondiabetic patients showing a greater increase ($P < 0.001$). For nondiabetics, the mean increase in glucose concentration (97.5% CI) was 29 (9, 49) mg/dL more in patients given dexamethasone than placebo ($P = 0.0012$). However, there was no dexamethasone effect in diabetics ($P = 0.99$).

CONCLUSIONS: Treatment of intraoperative hyperglycemia should account for the hyperglycemic surgical stress response trend depending on the stage of surgery as well as the added effects of steroid administration. Denying steroid prophylaxis for postoperative nausea and vomiting for fear of hyperglycemic response should be reconsidered given the limited effect of steroids on intraoperative blood glucose concentrations. (Anesth Analg 2013;116:1116–22)

Nondiabetic surgical patients, especially those suffering acute illnesses, often become hyperglycemic.¹ Physiological stress associated with serious illness,

trauma, and surgery causes insulin resistance, glucose intolerance, and hyperglycemia, a syndrome sometimes referred to as the diabetes of injury.¹ Whether perioperative hyperglycemia causes serious complications remains controversial,^{2,3} but high plasma glucose concentrations are inflammatory⁴ and decrease immune competence.^{5,6} There are thus reasons to believe that at least some degrees of perioperative hyperglycemia may prove harmful. The hyperglycemic stress response to surgery remains poorly described, and the time dependence of intraoperative glucose concentrations during major noncardiac surgery remains unclear.

Perioperative use of steroids is common; for example, dexamethasone is often administered because the drug is an effective antiemetic.⁷ Some clinicians have used steroids to reduce postoperative edema and inflammation,⁸ and steroids may reduce postoperative fatigue.⁹ The most obvious potential complication of perioperative steroid administration is immune suppression and consequent impaired resistance to infection, but steroids also cause insulin resistance and hyperglycemia. Eberhart et al.¹⁰ have shown that in nondiabetic women, preoperative oral dexamethasone resulted

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Accepted for publication December 13, 2012.

This work was supported by internal funds and a grant from Aspect Medical Systems which was acquired by Covidien (Dublin, Ireland) in November 2009.

The authors declare no conflicts of interest.

This report was previously presented, in part, at the ASA Annual Meeting, October 9, 2009 and October 18, 2010.

Reprints will not be available from the authors.

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DOI: 10.1213/ANE.0b013e318288416d

in a short period of postoperative hyperglycemia. Others have evaluated the effects of small IV doses of dexamethasone in a relatively small number of patients with and without diabetes^{1,11-13} with varying results.

The extent to which low-dose steroids affect perioperative glucose concentrations thus remains controversial.^{1,11,12,14-16} Concerns over such potential hyperglycemic effects may have contributed to the lack of compliance with antiemetic prophylaxis protocols or to limiting steroid prophylaxis to high-risk patients.¹⁷ The differential effects of steroids on perioperative glucose concentrations in diabetics and nondiabetics also remain unknown, although it would be reasonable to hypothesize that the hyperglycemic effect of steroid administration would be increased in diabetics. Diabetics have long been known to either have absolute insulin deficiency (type 1 diabetes mellitus [T1DM]) or relative insulin deficiency with insulin resistance (type 2 diabetes mellitus [T2DM]).¹⁸

We therefore tested 2 hypotheses: (1) that intraoperative glucose concentrations increase more in diabetic than in nondiabetic patients; and (2) preoperative administration of dexamethasone (8 mg) results in a greater increase in intraoperative hyperglycemic surgical stress response in diabetic than in nondiabetic patients. We also report the time-dependent pattern of intraoperative hyperglycemia during major noncardiac surgery.

METHODS

With IRB approval and written informed consent (Cleveland Clinic IRB, Cleveland, OH, 216-444-2924, IRB#07-010), we studied patients ≥ 40 years of age, ASA physical status ≤ 4 , who were scheduled for major elective noncardiac surgery at the Cleveland Clinic. Exclusion criteria included: (1) recent IV or oral steroid therapy within 30 days, although inhaled steroids were permitted; (2) any contraindications to the proposed interventions; (3) ASA physical status > 4 ; and (4) procedures performed under regional anesthesia. The patients participated in an underlying trial called the Dexamethasone, Light Anesthesia and Tight Glucose Control (DeLiT) Trial which has been described in detail.¹⁹

The enrollment period extended from March 2007 through July 2010. All patients were given general anesthesia and endotracheal intubation with sevoflurane in air and oxygen, along with IV fentanyl infusion following a standardized protocol according to the randomization. Randomization codes were generated by the PLAN procedure in SAS statistical software (SAS Institute Inc, Cary, NC), and implemented using a Web-based system that was accessed by research physicians shortly before the planned surgery. Randomization was stratified according to the presence or absence of diabetes (history of either T1DM or T2DM, and/or receiving insulin or oral hypoglycemic medications) to ensure balance for each intervention comparison within diabetes status.

Patients were randomized to either tight glucose control with a target plasma concentration of 80 to 110 mg/dL or conventional glucose control of 180 to 200 mg/dL, regardless of diabetic status. The DeLiT Trial was stopped after 381 patients for futility. Because most patients in the tight glucose control group received insulin to achieve the desired

near-normal glucose concentration target, our study population is restricted to all patients in the conventional glucose group ($N = 185$ patients). In this conventional glucose control group, insulin was given when blood glucose concentrations exceeded 215 mg/dL and then adjusted to maintain it within the desired target of 180 to 200 mg/dL. As part of the underlying trial, patients were also randomized to immediate (1–2 hours preincision) preoperative administration of 8 mg IV dexamethasone or placebo.

We recorded baseline characteristics as well as preoperative and intraoperative glucose concentrations. Glucose concentration was measured at least hourly when stable (i.e., no intervention in terms of an insulin bolus or change of infusion rate was required, and the concentrations were similar for 2 consecutive readings) and every 30 minutes when insulin boluses were given or the insulin infusion rate was modified. The majority of our patients had an arterial catheter inserted as a part of their routine intraoperative care. In most patients, whole arterial blood was used for the blood glucose determination with the Accu-Chek Inform system (Roche Diagnostics, Indianapolis, IN) monitors. Whole blood samples had less chance of having an erroneous reading compared with the capillary blood samples (7% vs 15%, respectively).²⁰ Each Accu-Chek device was checked in 3 dimensions (linearity, inter-method [lab versus meter], and meter-meter) and used only when acceptable results on all 3 metrics were found. Additionally, calibrations with low and high controls were performed daily to ensure continued high performance.

The primary outcome, hyperglycemic surgical stress response, was defined as the glucose change from preoperative to maximal intraoperative glucose concentration. Patients and clinicians were fully blinded to dexamethasone versus placebo assignment.

Statistical Methods

For univariable comparisons between diabetic and nondiabetic patients or between dexamethasone and placebo groups, we used the *t* test or Wilcoxon rank-sum test for continuous variables and χ^2 or Fisher exact tests for categorical variables.

We assessed the association between diabetic status (diabetic versus nondiabetic) and the primary outcome of glucose change from preoperative to maximal intraoperative glucose concentration (“maximal glucose change”) using multivariable linear regression. We forced into the model surgical type, dexamethasone use, and whether or not patients received intraoperative insulin (yes or no) as important confounding variables. We also adjusted for any unbalanced baseline factors in Table 1 (ASA physical status, age, and body mass index [BMI]) where $P < 0.05$. As a secondary analysis, we assessed the same association in the subset of patients who did not receive insulin during surgery.

In a subgroup analysis of the nondiabetic patients, we compared those with preoperative fasting plasma glucose (FPG) of < 110 to those who had FPG ≥ 110 mg/dL on the maximal glucose change using a *t* test. Meanwhile, for diabetic patients, we also performed comparisons between oral hypoglycemic-receiving diabetics and diet-controlled diabetics as well as between insulin-receiving diabetics and

Table 1. Baseline and Surgical Characteristics According to Diabetic Status or Dexamethasone Administration

Factor	Diabetics (N = 49)	Nondiabetics (N = 136)	P value	Dexamethasone (N = 90)	Placebo (N = 95)	P value
Age (y)	67 ± 10	63 ± 12	0.033	64 ± 11	64 ± 12	0.91
Body mass index (kg/m ²)	29 ± 7	27 ± 6	0.034	27 ± 7	28 ± 6	0.47
Female, no. (%)	12 (24)	44 (32)	0.30	26 (29)	30 (32)	0.69
ASA physical status, no. (%)			0.002			0.87
II	5 (10)	50 (37)		28 (31)	27 (28)	
III	36 (73)	75 (55)		52 (58)	59 (62)	
IV	8 (16)	11 (8)		10 (11)	9 (9)	
Surgery type, no. (%)			0.31			0.11
Abdominal aortic aneurysm	6 (12)	22 (16)		16 (18)	12 (13)	
Colectomy	11 (22)	47 (35)		30 (33)	28 (29)	
Cystectomy	9 (18)	27 (20)		19 (21)	17 (18)	
Peripheral revascularization	11 (22)	16 (12)		6 (7)	21 (22)	
Whipple/pancreas	11 (22)	21 (15)		17 (19)	15 (16)	
Other	1 (2)	3 (2)		2 (2)	2 (2)	
Dexamethasone, no. (%)	21 (43)	69 (51)	0.34	—	—	
Diabetics, no. (%)	—	—		21 (23)	28 (29)	0.34
Preoperative glucose (mg/dL)	143 ± 53	96 ± 19	<0.001	108 ± 43	108 ± 34	0.95
Received red cell transfusions, no. (%)	19 (38)	42 (31)	0.31	35 (39)	26(27)	0.10
Intraoperative insulin treatment, no. (%)	19 (39)	8 (6)	<0.001	16 (18)	11 (12)	0.33
Intraoperative insulin use in bolus units	0 [0, 4]	0 [0, 0]	<0.001	0 [0, 0]	0 [0, 0]	0.17
No. of glucose checks	8 ± 3	7 ± 3	0.56	8 ± 4	7 ± 3	0.024
Duration of glucose checks (h)	4.7 ± 1.9	4.5 ± 2.1	0.60	4.8 ± 1.9	4.3 ± 2.2	0.094
Duration of surgery (h)	4.9 ± 1.9	4.8 ± 2.4	0.88	5.1 ± 2.1	4.6 ± 2.4	0.092

Data are presented as means ± SD or median [interquartiles]. P values from the *t* test, Wilcoxon rank-sum test, χ^2 or Fisher exact test as appropriate; Wilcoxon sum-rank test for ASA physical status.

noninsulin-receiving diabetics using the *t* test. Covariable adjustment was not done here due to the limited sample size.

We assessed the effect of dexamethasone versus placebo and its interaction with diabetic status on mean maximal glucose change using linear regression. Because a significant interaction was observed between dexamethasone use and diabetic status ($P < 0.10$), the effect of dexamethasone on mean maximal glucose change was assessed within the diabetic (both T1DM and T2DM) and the nondiabetic patients, with a Bonferroni correction for 2 comparisons (significance criterion $P < 0.025$).

For the above 2 primary analyses of multivariable models, we used graphical diagnostic methods to check normality of the residuals (i.e., histogram plot of residuals) and the homogeneity of variance of the residuals (i.e., residuals plotted against fitted values).

We also assessed the linear relationship between preoperative glucose concentration and both the maximal glucose change and the percentage change from preoperative to maximal intraoperative glucose concentration using partial correlation analyses adjusting for baseline confounding variables.

We further assessed the pattern and magnitude of mean changes from baseline in glucose concentrations to specific surgical events (preoperative, incision, surgery midpoint, wound closure, and emergence) during surgery between diabetic and nondiabetic patients as well as between the dexamethasone and placebo groups using a linear mixed effects model with an unstructured within-patient correlation, adjusting for any potentially confounding baseline variables from Table 1 ($P < 0.05$) and forcing surgical type and whether or not patients received intraoperative insulin into the model.

With 49 and 136 patients in the diabetic and nondiabetic groups, respectively, we had 90% power at the 0.05

significance level with a 2-sided *t* test to detect a mean difference of ≥ 28 mg/dL between the 2 groups on the change from baseline to intraoperative maximal glucose concentration, using the observed overall SD of 52 mg/dL from this study.

SAS statistical software was used for all analyses. The significance level was 0.05 with 2-sided tests. The main results are reported as mean ± SD.

RESULTS

Baseline Characteristics

In the conventional glucose control group, 49 of 185 (26%) patients were diagnosed as having diabetes mellitus (Table 1). Nineteen of 49 (39%) diabetic and 8 of 136 (6%) nondiabetic patients were given insulin treatment intraoperatively ($P < 0.001$). Baseline characteristics were well balanced between patients randomized to dexamethasone and placebo but, as expected, unbalanced between diabetic and nondiabetic patients. Specifically, diabetic patients were more likely to be older, have higher ASA status and BMI, use insulin, and have larger baseline glucose concentrations. No severe hypoglycemic events were observed (i.e., all intraoperative glucose concentrations >40 mg/dL).

Intraoperative Maximal Glucose Change from Preoperative Concentrations

Univariably, mean maximal glucose change did not differ significantly between the diabetic and nondiabetic patients, with mean ± SD of 63 ± 69 mg/dL for diabetic and 72 ± 45 mg/dL for nondiabetic patients ($P = 0.39$). In a multivariable model adjusting for age, ASA status, BMI, surgical type, dexamethasone use, and insulin use, mean maximal glucose change was greater in nondiabetic than in diabetic patients, with a mean difference in maximal glucose change (95%

Table 2. Subgroup Analysis: Comparisons on Maximal Glucose Change Within Diabetic and Nondiabetic Patients

Group	N	Maximal glucose change (mean \pm SD mg/dL)	Univariable P value
Nondiabetics	136		
Baseline FPG			
<110 mg/dL	118	76 \pm 42	0.01
\geq 110 mg/dL	18	47 \pm 58	
Diabetics	49		
Insulin-receiving	14	62 \pm 92	0.94
Noninsulin-receiving	35	64 \pm 59	
Oral hypoglycemics only	21	66 \pm 66	0.79
Diet controlled	14	60 \pm 47	

FPG = fasting plasma glucose; maximal glucose change = intraoperative maximal glucose change from preoperative concentrations.

confidence interval [CI] of 29 (13, 46) mg/dL ($P < 0.001$). The same conclusion holds for the subset of patients who did not receive insulin during surgery ($P = 0.007$), with a mean difference in maximal glucose change (95% CI) of 22 (6, 38) mg/dL.

Subgroup comparisons on maximal glucose change within diabetic and nondiabetic patients are given in Table 2. In the nondiabetic group, the mean maximal glucose change in the patients with baseline FPG of <110 mg/dL was higher than in the patients with FPG \geq 110 mg/dL ($P = 0.01$). However, these particular results in Table 2 were not confounder adjusted due to sample size limitations and should be interpreted with caution.

There was a negative correlation between the preoperative glucose and maximal glucose change (adjusted $r = -0.65$; 95% CI, $-0.73, -0.56$; $P < 0.001$; Fig. 1A) after adjusting for age, ASA status, BMI, dexamethasone use, diabetic status, surgical type, and insulin use. The preoperative glucose was also negatively correlated with the percent change from preoperative to maximal glucose (adjusted $r = -0.69$; 95% CI, $-0.76, -0.61$; $P = 0.001$; Fig. 1B).

The effect of dexamethasone on the primary outcome of maximal glucose change varied by diabetic status (dexamethasone-by-diabetes interaction $P = 0.094$, Table 3). As shown in Table 3, dexamethasone increased the mean maximal glucose change (versus placebo) for nondiabetic patients (86 ± 41 vs 58 ± 45 mg/dL), with a

covariable-adjusted difference in the mean change (97.5% CI) of 29 (9, 49) mg/dL ($P = 0.0012$); however, there was no dexamethasone-induced hyperglycemic effect for diabetic patients (63 ± 66 vs 63 ± 72 mg/dL), with difference in mean maximal glucose change (97.5% CI) of 0 ($-33, 33$) mg/dL ($P = 0.99$). We did not find any severe violations of the assumptions of normality of residuals or the homogeneity of variance of the residuals in the 2 primary multivariable models.

Intraoperative Hyperglycemic Response Pattern over Time

For all patients combined, mean glucose increased slightly from preoperative to incision, substantially from incision to surgery midpoint, and then remained high and fairly stable through emergence. However, as shown in Figure 2, the change in glucose from baseline over time depended on diabetic status ($P < 0.001$). Most of the glucose increase occurred at midpoint, with nondiabetic patients showing a greater increase ($P < 0.001$). Meanwhile, we observed a dexamethasone-by-time interaction ($P = 0.018$) on the mean change in glucose from baseline. Again, most of the glucose increase occurred at midpoint, with the greatest increase evident in the dexamethasone group ($P = 0.01$).

DISCUSSION

The causes of intraoperative hyperglycemic surgical stress response include release of stress hormones such as growth hormone, glucagon, catecholamines, and glucocorticoids,²¹ all of which contribute to insulin resistance in the liver and in skeletal muscle by modifying signaling properties of insulin receptor substrates.²² We evaluated surgery-induced hyperglycemia in relation to dexamethasone administration and patients' diabetic status. Although dexamethasone administration could be randomly assigned, diabetic status obviously could not. As might be expected, diabetic patients had slightly higher ASA physical status, higher preoperative glucose concentrations (143 vs 96 mg/dL), and were more likely to be given intraoperative insulin treatment (39% vs 6%).

Blood glucose concentrations increased substantially from baseline to the intraoperative period, but contrary to our first hypothesis, the increase was greater in nondiabetic patients than in diabetic patients. In patients with no history of diabetes, the increase in blood glucose concentration was greater among those with normal or near-normal

Figure 1. The univariable negative relationship between the preoperative glucose and (A) change and (B) percent change from preoperative (PreOP) to intraoperative maximum glucose concentration for all patients.

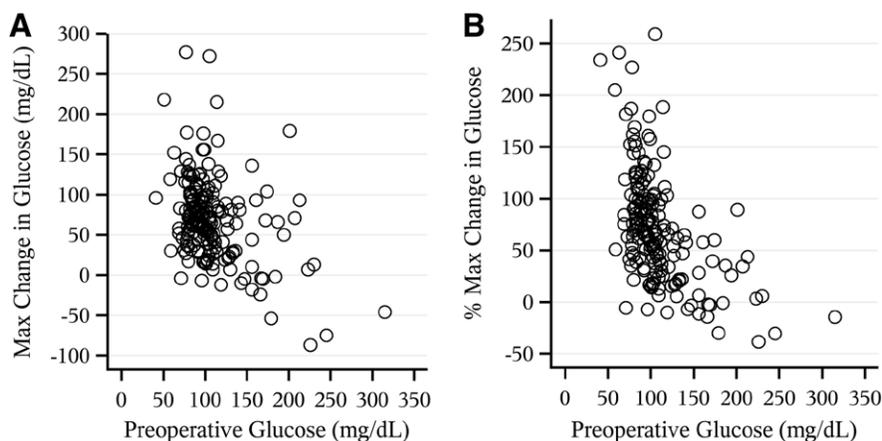


Table 3. The Effect of the Dexamethasone Intervention on Glucose Increase from Preoperative to Intraoperative Maximal Glucose (mg/dL) for Diabetic and Nondiabetic Patients

Factor	Dexamethasone (N = 90)	Placebo (N = 95)	Mean difference (97.5% CI) ^a	P value ^a
Diabetics (N = 49)	63 ± 66	63 ± 72	0 (-33, 33)	0.99
Nondiabetics (N = 136)	86 ± 41	58 ± 45	29 (9, 49)	0.0012
Overall ^b	81 ± 49	59 ± 54	22 (7, 37)	0.004

Data are presented as mean ± SD.

CI = confidence interval.

^aLinear regression model including factors for dexamethasone versus placebo and diabetic versus nondiabetic; the interaction between dexamethasone and diabetic status was significant ($P = 0.094$, less than criterion of $P < 0.10$); we used a Bonferroni correction for 2 comparisons ($\alpha = 0.05/2 = 0.025$).

^bAnalysis ignoring the interaction.

blood glucose concentrations versus those who had a fasting glucose concentration of ≥ 110 mg/dL (indicating either impaired fasting glucose or an undiagnosed diabetes mellitus). We previously reported that 21% of nondiabetic general surgical patients at Cleveland Clinic present with glucose concentrations ≥ 110 mg/dL.²³ Our current and previous results thus suggest that a large proportion of noncardiac surgery patients are subject to a significant hyperglycemic surgical stress response. Therefore, the increased attention which diabetic patients have traditionally experienced should be extended to nondiabetics as well.

In nondiabetic patients, glucose concentrations increased substantially from incision to midpoint of surgery, but then changed little during the remaining hours of surgery. This pattern of intraoperative glucose concentrations resembles to some extent that described by Hans et al.¹³ who identified a peak concentration at 2 hours after induction. Although Hans et al.¹³ attributed hyperglycemia to administration of 10 mg of dexamethasone at induction, they did not exclude the possibility that a hyperglycemic stress response was the cause. Cumulatively, these results suggest that optimal algorithms for intraoperative glucose control might best be based not only on absolute glucose concentration, but also on a context-sensitive component that considers the time elapsed since incision, such as the one we used.²⁴

As might be expected for a randomized factor, patients given dexamethasone and placebo were comparable on baseline factors. In our study, a small dose of dexamethasone, 8 mg

given IV preoperatively, moderately augmented the hyperglycemic response to surgery (86 vs 58 mg/dL) only in patients without diabetes. This is an intriguing finding because diabetic patients have either absolute insulin deficiency (T1DM) or relative insulin deficiency with insulin resistance (T2DM)¹⁸ and would thus be thought to have limited ability to adjust for the dexamethasone-induced hyperglycemic effect.

Our results contrast with those of Hans et al.¹³ who, in a nonrandomized study of nondiabetic and T2DM patients undergoing noncardiac surgery, found a comparable dexamethasone-induced hyperglycemic response in the 2 groups. Others, however, have reported similar (to our results) dexamethasone-induced hyperglycemia in nondiabetic patients. Lukins and Manninen,¹² for example, in their nonrandomized trial of nondiabetic patients ($n = 34$) for craniotomy reported a 50 mg/dL increase after 10 mg of dexamethasone. A difference of 37 mg/dL was reported by Pasternak et al.¹¹ when they randomized nondiabetic neurosurgery patients ($n = 20$) for craniotomy to 10 mg of dexamethasone or placebo. Similarly, Nazar et al.¹ in their randomized trial of obese patients ($n = 30$) with impaired glucose tolerance, and scheduled for gastric bypass surgery, observed a significantly different blood glucose increase of 29 mg/dL in the dexamethasone group.

Large doses of dexamethasone (1 mg/kg) or methylprednisolone (15–30 mg/kg) produced substantial hyperglycemia in patients having cardiac surgery.^{25,26} In contrast, a low dose of hydrocortisone (equivalent to 8 mg

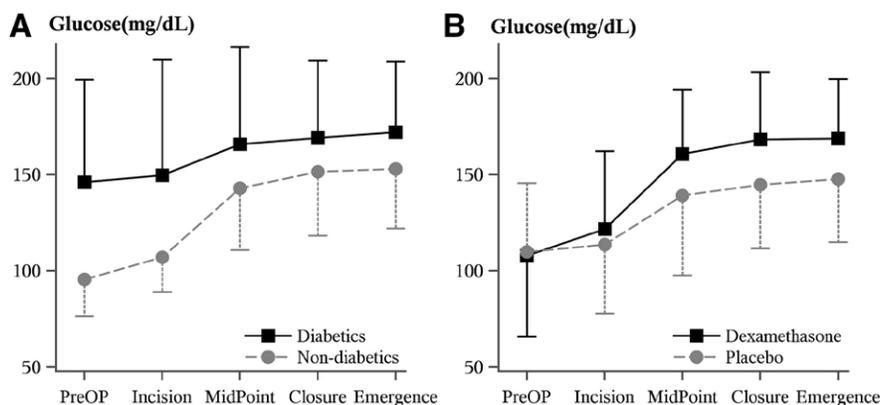


Figure 2. Pattern of intraoperative hyperglycemic stress response over time. The time points were not equidistant, and surgery midpoint was defined as halfway between induction and closure. Half error bar presents the unadjusted mean ± SD of intraoperative glucose over time for each group. Results are from a linear mixed effects model with unstructured within-patient correlation, adjusting for the potentially confounding baseline variables (ASA physical status, age, body mass index, surgical type, and intraoperative use of insulin). (A) Mean glucose increased for both diabetic and nondiabetic groups during surgery, with a largest increase at the midpoint. There was a larger and longer increase in nondiabetic patients than in diabetic patients ($P < 0.001$). (B) Glucose values increased for both dexamethasone and placebo groups during surgery, with a largest increase at the midpoint, and the greatest increase evident in the dexamethasone group ($P = 0.01$).

of dexamethasone) did not.¹⁵ Available evidence thus suggests that doses of dexamethasone administered prophylactically for postoperative nausea and vomiting (typically 4–8 mg) are unlikely to considerably increase intraoperative glucose concentrations.

Our study is limited by the fact that glucose concentrations exceeding 215 mg/dL were treated. Consequently, some patients from each group were given insulin. However, we statistically adjusted for insulin treatment, which is a better approach than simply excluding these patients. In addition, a sensitivity analysis was performed to assess the same association for the subset of patients who did not receive insulin. The same conclusion was reached. We also note that the results presented here were not the primary outcome, but instead a subgroup analysis of a larger underlying DeLiT Trial. Moreover, glucose concentrations were evaluated both by our central laboratory and with the criticized albeit more practical point-of-care testing. However, point-of-care testing is generally considered reliable and is widely used both clinically and in research.

In summary, diabetic patients had significantly greater preoperative blood glucose concentrations than nondiabetic patients. Both groups showed a sizeable intraoperative hyperglycemic response that was inversely related to preoperative glucose concentration. Glucose increased mainly from incision to the midpoint of the surgery, and subsequently remained nearly stable. Preoperative administration of 8 mg of dexamethasone increased the intraoperative hyperglycemic response compared with placebo, but only in nondiabetic patients.

In conclusion, intraoperative glucose control strategies should consider the stage of surgery and whether patients were given steroids. Denying steroid prophylaxis for postoperative nausea and vomiting for fear of a hyperglycemic response should be reconsidered given the limited effect of steroids on intraoperative blood glucose concentrations. ■

DISCLOSURES

Name: Basem B. Abdelmalak, MD.

Contribution: This author helped design the study, conduct the study, and write the manuscript.

Attestation: Basem B. Abdelmalak has designed the study, seen the original study data, reviewed the analysis of the data, written the manuscript, approved the final version, and is the author responsible for archiving the study files.

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Attestation: Angela M. Bonilla has seen the original study data and approved the final manuscript.

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Contribution: This author helped design the study, conduct the study, and write the manuscript.

Attestation: Daniel I. Sessler has seen the original study data, reviewed the analysis of the data, and approved the final manuscript.

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