Original Article

# Intra-operative administration of low-dose IV glucose attenuates post-operative insulin resistance

Hiroko Fujino DDS, PhD[1], Shoko Itoda DDS, PhD[2], Kanako Esaki DDS[2],
Masanori Tsukamoto DDS, PhD[2], Saori Sako DDS[2], Kazuki Matsuo DDS[2],
Eiji Sakamoto DDS, PhD[2], Kunio Suwa MD, PhD[3], Takeshi Yokoyama DDS, PhD[2]

[1]*Department of Dental Anesthesiology, Showa University Dental Hospital, Tokyo, Japan*
[2]*Department of Dental Anesthesiology, Faculty of Dental Science, Kyushu University, Fukuoka, Japan*
[3]*Department of Life Care, Teikyo Junior College, Tokyo, Japan*

**Background & Aims:** Insulin sensitivity often decreases after surgery in spite of normal insulin secretion, and may worsen the outcome. This post-operative insulin resistance increases according to the magnitude of surgical invasion. However, supplementation of carbohydrates before surgery attenuates the post-operative insulin resistance. This study aimed to investigate the effect of intra-operative administration of low-dose glucose on the post-operative insulin resistance. **Methods:** Patients undergoing maxillofacial surgery were randomly assigned to two groups throughout the surgical procedure: The glucose group receiving acetated Ringer solution with 1.5% glucose and the control group receiving acetated Ringer solution without glucose. Insulin resistance quantified by the mean glucose infusion rate (the glucose infusion rate) was evaluated by glucose clamp using the STG-22TM instrument on the previous day and on the next day of surgery. Blood glucose level was monitored continuously during surgery. In addition, serum insulin, ketone bodies and 3-methylhistidine were measured during perioperative period. **Results:** Patients in the glucose group (n=11) received 0.15±0.06 g/kg/h of glucose during surgery, while patients in the control group (n=11) received no glucose. In both groups, however, the mean blood glucose levels were maintained stable at less than 150 mg/dL during and after surgery. The serum ketone bodies significantly increased after surgery in the control group (*p*=0.0035), while it decreased significantly in the glucose group (*p*=0.043). The reduction rate in the glucose infusion rate was significantly lower in the glucose group, 43.3±20.7%, than that in the control group, 57.7±9.3% (*p*=0.041). **Conclusions:** Intra-operative small-dose of glucose administration may suppress ketogenesis and attenuate the post-operative insulin resistance without causing hyperglycemia.

**Key Words: insulin resistance, glucose clamp, artificial pancreas, ketogenesis, 3-methylhistidine**

## INTRODUCTION

There are three major problems about nutritional management during perioperative period. The first problem is hyperglycemia, possibly resulting in reperfusion injury in the tissues.[1-3] In addition, hypoglycemia causes suppression of the immune system.[4,5] It may, therefore, worsen the outcome of operations especially in neurosurgery.[1,3] Hyperglycemia often occurs during the administration of conventional fluid with high concentration of glucose of 2.5% to 10% during surgery.[6-9] Some reports have proposed that the administration of glucose might not always be necessary during surgery.[7,8,10,11] Furthermore, several studies suggest that glucose administration during surgery can be harmful.[12,13] The second problem is pre-operative fasting. Patients are not allowed to have a meal for several hours before surgery to avoid vomiting during induction of anesthesia. Glucose is necessary in the central nervous activity and for red blood cells even under general anesthesia. It is stored as glycogen in the body, and the total amount of glycogen is less than needed for basal metabolism for one day. Thus, shortage of glucose induces gluconeogenesis.[14] The third problem is decrease in

insulin sensitivity after surgery. Brandi reported that 8 times more insulin is needed post-operatively than before surgery to maintain normoglycemia.[15] This morbid state with decrease in insulin sensitivity is called insulin resistance. The post-operative insulin resistance increases according to the magnitude of surgical invasion.[16] Pre-operative supplementation of carbohydrates attenuates the post-operative insulin resistance.[17,18]

We previously reported that a small-dose of glucose administration, around one-third of basal metabolism, could reduce ketogenesis in patients undergoing elective surgery.[19] Therefore, its administration during surgery may affect the post-operative insulin sensitivity. In the

current study, we hypothesized that the administration of a small-dose of glucose may attenuate post-operative insulin resistance without causing hyperglycemia, and investigated the change of insulin resistance using the glucose clamp technique with the STG-22TM (Nikkiso, Tokyo, Japan) during perioperative period including blood glucose concentration, ketogenesis and muscle protein breakdown.

## METHODS

After obtaining approval from the Ethics Committee of Kyushu University Hospital (#21068), we got written informed consent from patients undergoing elective maxillofacial surgery, American Society of Anesthesiologists physical status I or II. Patients with diabetes and/or obesity [body mass index (BMI) >25 kg/m$^2$] were excluded from the study. Patients were randomly assigned to two groups: the glucose group receiving acetated Ringer solution with 1.5% glucose during surgery and the control group receiving acetated Ringer solution without glucose.[20,21]

### Glucose clamp technique using STG-22TM

Glucose clamp technique was achieved using the STG-22TM. It was developed as a type of artificial endocrine pancreas by the Nikkiso Company (Tokyo, Japan) in 1984. This device is incorporated with a closed-loop glycemic control system that provides continuous blood glucose monitoring and subsequent control of glucose levels by the automatic infusion of regular insulin and/or glucose into the blood, making it possible to maintain relatively stable glucose levels.[22] It provides continuous blood glucose monitoring through a dual lumen catheter blood sampling technique, high-quality roller pump (multichannel pump) and a glucose sensor electrode with glucose oxidase membrane (Yellow Springs, Dayton, OH). Before starting blood glucose monitoring, a two-point calibration was done using the standard solution for internal calibration (glucose concentration: 0 mg/dL) and a second standard glucose solution (200 mg/dL). During blood glucose monitoring, internal calibration using the standard solution for internal calibration was automatically done every 4 hours. After calibration of the equipment, blood was sampled continuously from the peripheral vein at a rate of 2 mL/h and continuously diluted with a heparinized isotonic solution. The diluted blood was further diluted with an isotonic buffer solution of phosphoric acid, pH 7.4, after which the glucose sensor electrode was exposed to the sampled blood. The multichannel pump and the glucose sensor electrode both had an accuracy of ±5%.

Insulin was infused intravenously at 1.25 mU/kg/min and glucose was simultaneously infused intravenously at a variable rate to maintain the blood glucose concentration at 90 mg/dL. In this method, insulin resistance was quantified by the mean glucose infusion rate (The glucose infusion rate) during a steady-state period.

### Evaluation of insulin resistance

The glucose clamp using the STG-22TM was performed before and after surgery. The procedure started at 9:00 in the morning after overnight fast on the previous day of surgery. A 20G IV catheter (InsyteTM, 20GA 1.16IN,

Becton Dickinson Infusion Therapy System, Sandy, UT) was inserted into the peripheral antebrachial vein and connected to the STG-22TM for continuous blood glucose monitoring, and another catheter was inserted into the peripheral antebrachial vein on the opposite arm for infusion of glucose and insulin. On the next day after surgery, this examination was repeated at 9:00 in the morning after 3 hours interruption of glucose administration on the next day of surgery.

### Anesthetic management

No patients were pre-medicated after overnight fasting with intravenous (IV) fluid administration. On arrival in the operating room, routine monitoring was applied, including electrocardiography, noninvasive blood pressure, pulse oximetry, and EtCO$_2$. General anesthesia was induced by 4 μg/kg of fentanyl and 0.1 mg/kg of midazolam. Intubation was facilitated with 0.1 mg/kg of vecuronium bromide. Anesthesia was maintained with 1~2% sevoflurane in 50% oxygen and 50% nitrogen to maintain bispectral index (BIS) levels <60. Continuous administration of remifentanil and intermittent administration of fentanyl were performed for analgesia to maintain the change in blood pressure and the heart rate within 25% of those before induction of anesthesia.

After anesthetic induction, a 20G IV catheter (InsyteTM, 20GA 1.16IN, Becton Dickinson Infusion Therapy System, Sandy, UT) was inserted into the antebrachial vein and connected to the STG-22TM for continuous blood glucose monitoring. A catheter was inserted into the radial arterial for intermittent blood sampling. Patients in the control group were infused with acetated Ringer solution without glucose, while patients in the glucose group were infused with 1.5% glucose during anesthesia. These Ringer solutions were infused at the rate of 20 mL/kg/h after the start of anesthesia. Patients in the glucose group received 0.3 g/kg of glucose. One hour after the start of infusion, infusion rate was decreased to 5 mL/kg/h. In the glucose group patients received 0.075 g/kg/h of glucose approximately. Other fluids, such as plasma or colloids, were not given with the exception of 100 mL of saline for administration of antibiotics. The blood glucose levels of all patients were continuously monitored by the STG-22TM, and insulin therapy was permitted intra-operatively when a glucose level exceeded 180 mg/dL. A rectal probe was inserted for core temperature monitoring. Ambient temperature was maintained at approximately 23-24 ˚C. Patients were covered with a single cotton blanket during the study period. Rectal temperatures were recorded every 15 mins after the start of surgery. When rectal temperature decreased to 36 ˚C, warming was used upper body blanket (Bear Hugger Blanket; Augustine Medical, Eden Prairie, MN, USA) and the rectal temperature was maintained between 36 ˚C and 37 ˚C.

### Measurements of plasma glucose, insulin, ketone bodies and 3-methylhistidine

Blood samples were collected before anesthetic induction (T1), 1 h after the induction of anesthesia (T2), 3 h after the induction of anesthesia (T3), at the end of surgery (T4) and at the first day post-operatively before glucose clamp

(T5) to determine plasma glucose, insulin, ketone bodies and 3-methylhistidine (3-MH). The blood samples were centrifuged at 3,000 rpm for 10 mins and plasma obtained were frozen (-20℃) until analysis. Plasma glucose (ABL System 625; Radiometer, Copenhagen, Denmark) were measured at the same time. Ketone bodies were measured by enzymatic techniques. The plasma insulin (normal range: 3.06-16.9 μU/mL) level was determined by radio-immunoassay, and the plasma 3-MH level was measured by high-performance liquid chromatography.

### Statistics

Data values are expressed as mean ± standard deviation (SD). Nonparametric variables were compared between the groups using Mann-Whitney's $U$ test. Parametric data were compared between the groups using unpainsulin resistanceed Student's $t$ test. Each blood sample parameters and hemodynamic data were analyzed using two way analysis of variance with ANOVA repeated measures. When a significant difference was noted, the post-hoc Boneferroni-Dunn test was performed for multiple comparisons. For all determinations, $p$ value of <0.05 was considered as the level of statistical significance.

### RESULTS

Twenty-two patients, 11 in each group, participated in this study. All patients underwent osteotomy of the maxilla and/or the mandible due to the jaw deformity from June 2010 to March 2013. They have no systemic complications, and they were evaluated as American Society of Anesthesiologists physical status I. No patients received insulin, and no other fluids were administered during surgery with the exception of scheduled autologous blood transfusion. There were no significant differences in terms of demographic data (Table 1). There were no significant differences in hemodynamic status, BIS values and rectal temperature (Table 2).

There was no significant difference in the glucose level before anesthesia; 85±0.7 mg/dL in the glucose group and 86±0.2 mg/dL in the control group. Blood glucose level was monitored continuously for all patients from the start of operation to the next morning using the STG-22TM (Figure 1). The plasma glucose levels were also measured intermittently. Glucose concentration during operation was significantly higher in the glucose group than that in the control group at T2 ($p$=0.0007), T3 ($p$=0.0008) and T4 ($p$=0.0054). No hyperglycemia, however, was observed during surgery and the mean blood glucose levels were maintained stable at less than 150 mg/dL in both groups during and after surgery.

In 5 patients from each group, selected randomly, plasma insulin level was measured at 5 points. There were no significant differences between the groups in terms of plasma insulin concentration except at T2 ($p$=0.0034) (Figure 2). In the glucose group, the plasma insulin concentration did not change significantly, but it was maintained 7 μIU/mL after induction of anesthesia. In the control group, on the other hand, it decreased to below the normal limit from 2.7±2.0 μIU/mL (T1) to 2.6±1.3 μIU/mL (T4), and recovered to 10.0±5.9 μIU/mL after overnight infusion of glucose at 0.075 g/kg/hr (T5).

There was no significant difference in the serum ketone bodies before anesthesia, although baseline of the serum ketone bodies of the glucose group were higher due to increased ketone bodies in a few patients; 152±220 μmol/L in the glucose group and 61±38 μmol/L in the control group ($p$=0.27). The serum concentration of ketone bodies was significantly higher in the control group than that in the glucose group at T3 ($p$=0.023) and T4 ($p$=0.0007) (Figure 3). In the control group, the serum concentration of ketone bodies was significantly higher at T3 ($p$=0.0035) and at T4 ($p$=0.0035) than that at T1. In the glucose group, however, it decreased significantly from T1 to T5 ($p$=0.043). Acetone was not detected at any measurement in all patients.

The serum concentration of 3-MH was significantly decreased in both groups (data not shown). The decreasing rate was 9.6% in the glucose group and 13.0% in the control group, however, there were no significant differences in the serum concentrations of 3-MH between two groups.

The glucose infusion rate decreased after surgery in all cases, except for one case. In the control group, the pre-operative the glucose infusion rate of 9.4±0.8 mg/kg/min which decreased significantly to 3.8±0.2 mg/kg/min ($p$=0.0052). In the glucose group, the pre-operative glucose infusion rate of 8.0±2.0 mg/kg/min decreased significantly to 4.4±1.5 mg/kg/min ($p$=0.0045). Although the values of post-operative glucose infusion rate in both groups did not reach the significant level. The reduction rate in the glucose infusion rate was significantly lower in the glucose group, 43.3±20.7%, than that in the control group, 57.7±9.3% ($p$=0.041) (Figure 4).

### DISCUSSION

In the current study, we aimed to analyze the effect of the intra-operative administration of glucose on the devel-

**Table 1**. Patient characteristics and intra-operative variables

|  | The glucose group (n=11) | The control group (n=11) | $p$ value |
|---|---|---|---|
| Male/Female | 4/7 | 4/7 |  |
| Age (year) | 32.5±7.88 | 35.8±11.1 | 0.57 |
| Height (cm) | 163±8.54 | 164±7.79 | 1.20 |
| Weight (kg) | 58.2±9.41 | 57.1±8.93 | 1.36 |
| BMI (kg/m$^2$) | 21.7±2.23 | 20.9±2.17 | 0.32 |
| Fasting time (min) | 675 | 675 |  |
| Anesthesia time (min) | 367±68.2 | 323±75.7 | 0.09 |
| Operation time (min) | 273±62.4 | 244±48.4 | 0.14 |
| Fentanyl (μg/kg) | 5.41±0.76 | 5.90±1.11 | 0.52 |
| Remifentanil (μg/kg) | 80.8±29.8 | 69.2±24.8 | 0.57 |
| Infusion (mL/kg/h) | 9.04±1.01 | 9.67±3.04 | 0.71 |
| Urine output (mL/kg/h) | 1.60±0.74 | 1.48±0.92 | 0.66 |
| Blood loss (mL/kg/h) | 1.20±1.28 | 1.40±0.90 | 0.40 |
| Glucose dose (g/kg/h) | 0.15±0.06 | 0 |  |

There were no significant differences except glucose dose infused during anesthesia.

BMI: body mass index. Data are expressed as mean value ± standard deviation.

The data were statistically analyzed by Mann-Whitney's $U$ test or Student's $t$ test.

**Table 2.** Hemodynamic status and BIS values of patients

|  | T1 | T2 | T3 | T4 |
|---|---|---|---|---|
| HR (bpm) |  |  |  |  |
| The glucose group | 83.3±12.3 | 62.6±7.19 | 65.6±7.81 | 68.1±10.5 |
| The control group | 78.5±18.6 | 62.2±6.31 | 63.3±7.34 | 74.5±14.9 |
| SBP (mmHg) |  |  |  |  |
| The glucose group | 121±16.2 | 90.5±11.0 | 92.3±15.3 | 106±13.5 |
| The control group | 119±15.0 | 94.9±10.2 | 92.1±12.4 | 92.1±12.4 |
| DBP (mmHg) |  |  |  |  |
| The glucose group | 73.3±11.1 | 48.2±7.93 | 45.8±5.24 | 56.9±11.2 |
| The control group | 71.2±17.8 | 51.3±8.86 | 49.9±11.6 | 56.1±10.3 |
| BIS values |  |  |  |  |
| The glucose group | 91.9±12.5 | 53.0±6.15 | 49.1±8.89 | 61.3±6.58 |
| The control group | 97.6±1.28 | 45.2±7.86 | 45.5±5.66 | 64.1±15.6 |
| Rectal temperature (°C) |  |  |  |  |
| The glucose group |  | 36.5±0.27 | 36.4±0.80 | 37.2±0.46 |
| The control group |  | 36.4±0.42 | 36.5±0.50 | 37.2±0.66 |

T1: anesthetic induction; T2: 1 h after anesthetic induction; T3: 3 h after anesthetic induction; T4: at the end of surgery; HR: heart rate; SBP: systolic blood pressure; DBP: diastolic blood pressure; BIS: bispectral index. Data are expressed as mean value ± standard deviation. There were no significant differences between groups by ANOVA repeated measures.
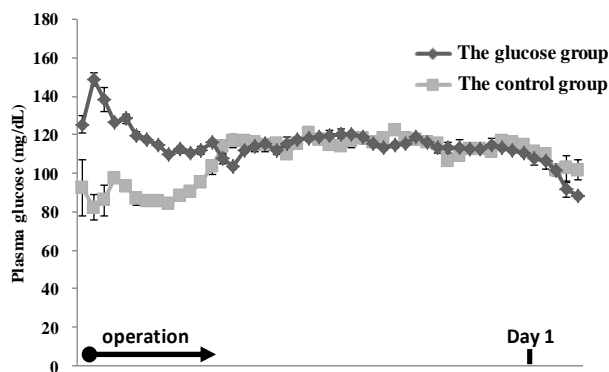


**Figure 1**. Continuous blood glucose monitoring. Alteration of mean blood glucose levels of the glucose group (n=11) and the control group (n=11).
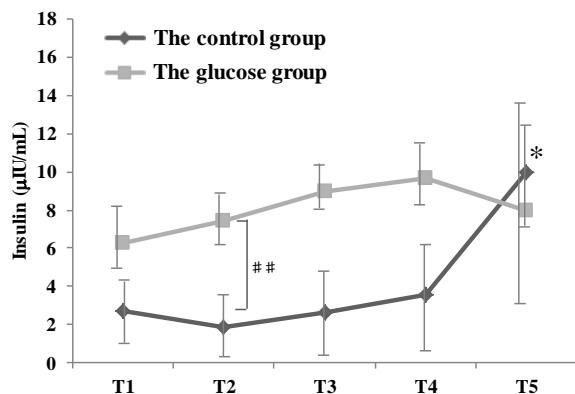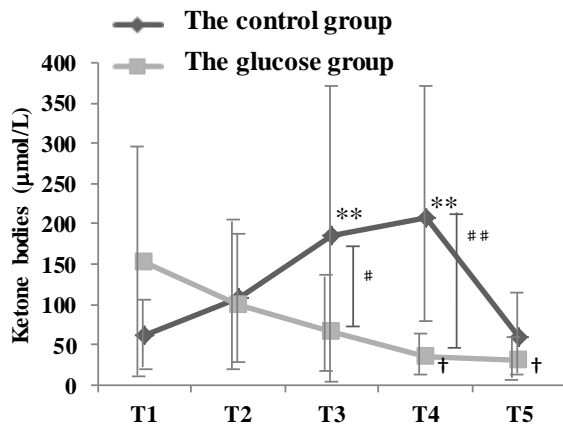


**Figure 2**. Plasma insulin concentrations, mean±SD. Differences between groups were significant for plasma insulin concentration at $p<0.05$ by ANOVA repeated measures between groups. * indicate $p<0.05$ in comparison to T1 in the control group by Boneferroni-Dunn test, and ## indicate $p<0.01$ between groups by unpaired Student's t test.

opment of insulin resistance after surgery. In our results, administration of glucose with Ringer solution including 1.5% glucose effectively attenuated the increase in insulin resistance after surgery, while the control group without glucose showed severe post-operative insulin resistance.

We used STG-22TM in this study, since this device makes it possible to monitor blood glucose level continuously. The reliability of its glucose monitoring has already been verified.[23,24] In this study, blood glucose level was monitored during and after surgery, and no hyper/hypoglycemia was observed in any of the two groups (Figure 1). The normoglycemic hypeinsulinemic clamp technique is often used for evaluation of insulin resistance,[25] since this method is the global gold standard. The glucose clamp shows low coefficient of variation (0.10) and has good reproducibility.[26] In the hyperinsulin condition, glucose infusion rate correlates to insulin sensitivity. Therefore, glucose infusion rate is used as a good indicator of insulin resistance. Especially, the glucose clamp using the STG-22TM is more reliable, because glucose infusion rate could be evaluated during steady-state period under constant insulin infusion and constant glucose level by continuous glucose monitoring, which is necessary for avoiding hypoglycemia. Therefore, the glucose clamp using the STG-22TM has become the most standard method in Japan, although the procedure is very complicated. On the contrary, Homeostasis Model Assessment-Insulin resistance (HOMA-IR) is a simple, convenient method and adaptable for clinical evaluation for insulin sensitivity. In our recent study, however, HOMA-IR had poor correlation to the results by the glucose clamp using the STG-22 TM.[27] We, therefore, performed the glucose clamp using the STG-22TM twice before and after surgery in this study.

Surgical stress and body temperature affect glucose metabolism and blood glucose concentration. In the current study, there were no significant difference in dose of opioids and vital signs; blood pressure, heart rate and body temperature. We checked serum adrenaline, noradrenaline and dopamine as indicators of the degree of stress of surgery. There were no significant differences between groups either.

In this study, hyperglycemia was not observed. In the glucose group, the mean blood glucose level increased to almost 150 mg/dL in the first one hour after induction of anesthesia when the infusion rate was 20 mL/kg/hr.

**Figure 3**. Plasma ketone bodies concentrations. Changes in plasma ketone bodies concentrations at anesthetic induction (T1), 1 h (T2), 3 h after anesthetic induction (T3), at the end of surgery (T4), and at the first day post-operatively (T5). Differences between groups were significant for plasma ketone bodies concentrations at $p<0.05$ by ANOVA repeated measures between groups. ** indicate $p<0.01$ in comparison to T1 in the control group, † indicate $p<0.05$ in comparison to T1 in the glucose group by Boneferroni-Dunn test, and # and ## indicate $p<0.05$ and $0.01$ between groups by unpaired Student's $t$ test.
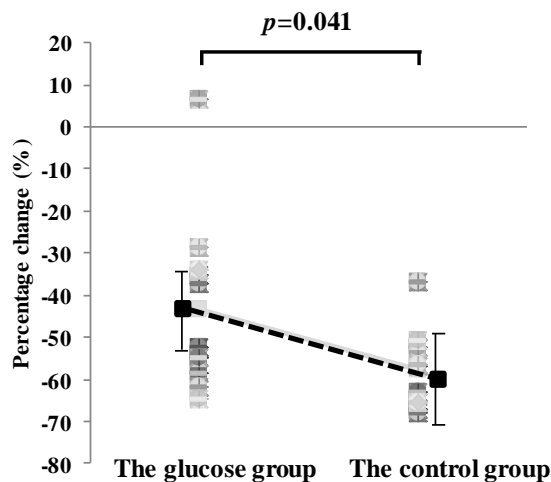


**Figure 4**. Relative changes in insulin sensitivity. The relative reduction in insulin sensitivity (%) post-operatively for each patient was calculated from post-operative M-value/pre-operative M-value × 100 for all patients. Results are expressed as mean±SD.

According to this increase in blood glucose level, the insulin level also increased and was maintained to the end of surgery. The insulin level was 7.99±3.18 mIU/mL at T5. In the control group, the insulin level was kept lower during surgery than that before induction of anesthesia, but it was 9.99±5.95 mIU/mL at T5.

Shortage of glucose in the body induces gluconeogenesis and fat metabolism which leads to ketogenesis. Glucose is the most important nutrient especially for brain tissue, and storage of glucose in our body is usually less than basal energy expenditure for one day. Even in young patients, therefore, more than one-third of glucose production depends on gluconeogenesis after 22 hours of fasting.[14] However, glucose is not administered routinely during surgery as high concentrations of glucose might cause hyperglycemia,[6-9] which may lead to worse out-

come especially in neurosurgery.[1,3] It has been reported that carbohydrate-rich fluids given orally before anesthesia alters metabolism and reduces the catabolic response in the perioperative period, and that postoperative insulin resistance increased complications after cardiacsurgery.[28] The Enhanced Recovery after Surgery (ERAS) protocol has recently garnered attention as an evidence-based method for perioperative care used in hospitals worldwide to improve patient prognosis. This protocol recommends carbohydrate loading via oral administration before surgery.[29] But nothing about intra-operative glucose was indicated in the protocol of Enhanced Recovery after Surgery.

We earlier reported that overnight fast and fluid therapy with no glucose caused an increase in serum ketone bodies during surgery.[19] But intra-operative small-dose of glucose, corresponding to 43% of basal energy expenditure, effectively suppressed ketogenesis in orthopedic surgery. Mikura et al. reported that 0.1 g/kg/hr of glucose during surgery under general anesthesia effectively suppress protein-breakdown in the skeletal muscles in the rat model.[30] It has been reported that infusion of a small-dose of glucose (1%) during minor otorhinolaryngeal, head and neck surgeries might suppress ketogenesis and, suppress protein catabolism during surgery and prevent muscle protein -break down.[31] Serum 3-MH, which is an index of protein-breakdown.[31]

In this study, a small-dose of glucose (0.15 g/kg/hr) effectively suppressed serum ketone bodies (Figure 3). Temporary increase in ketone bodies was observed in patients in the control group who received no glucose during surgery, while serum ketone bodies level decreased in the glucose group. These results suggest that fat is used for energy production in the cells when glucose is not supplied during surgery. In the control group, gluconeogenesis using glucogenic amino acids might be accelerated as blood glucose level had been maintained.

The urine 3-MH level is a reliable indicator of skeletal muscle protein breakdown rate,[32] because 3-MH is not reused at all in the muscle tissue and is excreted from the kidneys. In our preliminary study, we tried to detect the change of urine 3-MH level, but it was difficult probably due to sampling timing. Muscle protein breakdown begins after the first one hour after surgery and the glucogenic amino acids during the early intra-operative period is supplied from smooth muscle, such as intestinal muscle, rather than skeletal muscle in recent reports.[33] When glycogenic amino acids are supplied from smooth muscle, 3-MH does not increase. Therefore, it may be difficult to detect muscle protein-breakdown from blood samples during surgery. However, it was reported that the low-dose glucose infusion caused a decreased in plasma 3-MH.[31] In our results, 3-MH decreased after surgery in both groups, but it did not reach significant level.

The glucose infusion rate is a good indicator of insulin resistance, and it decreased in both groups compared to that before surgery. The decreasing rate, however, was significantly larger in the control group than that in the glucose group ($p=0.041$) (Figure 4). Insulin resistance means decrease in sensitivity of insulin in the peripheral tissues, which is a transitional phenomenon appears usually for the first several days after surgery. In the current

study, the infusion rate was not constant and changed from 20 mL/kg/hr to 5 mL/kg/hr at one hour after the start of infusion. The average administration rate of glucose was about 0.15 g/kg/hr during surgery, which covers about 56.1±23.1% of basal energy expenditure calculated by Harries- Benedict equation. After surgery 0.08 g/kg/hr of glucose was given continuously until the next morning 3 hours before glucose clamp using STG-22TM. This small-dose of glucose did not cause hyperglycemia during and after surgery, but it effectively suppressed increase in serum ketone bodies and attenuated post-operative insulin resistance, which may contribute enhanced recovery after surgery.

The study is small sample size and the surgical procedure was limited by maxillofacial surgery. Further study with large sample size, and other surgical procedure may be required.

In conclusion, infusion of acetated Ringer solution with 1.5% glucose during surgery did not cause either hyperglycemia or hypoglycemia, and suppressed ketogenesis. In addition, the infusion of a small dose of glucose during surgery attenuate post-operative insulin resistance, a similar result to that found for pre-operative carbohydrate.

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Original Article

# Intra-operative administration of low-dose IV glucose attenuates post-operative insulin resistance

Hiroko Fujino DDS, PhD[1], Shoko Itoda DDS, PhD[2], Kanako Esaki DDS[2],
Masanori Tsukamoto DDS, PhD[2], Saori Sako DDS[2], Kazuki Matsuo DDS[2],
Eiji Sakamoto DDS, PhD[2], Kunio Suwa MD, PhD[3], Takeshi Yokoyama DDS, PhD[2]

[1]*Department of Dental Anesthesiology, Showa University Dental Hospital, Tokyo, Japan*
[2]*Department of Dental Anesthesiology, Faculty of Dental Science, Kyushu University, Fukuoka, Japan*
[3]*Department of Life Care, Teikyo Junior College, Tokyo, Japan*

## 术中静脉小剂量给予葡萄糖减弱术后胰岛素抵抗

背景与目的：手术后尽管胰岛素分泌正常，但胰岛素的敏感性往往会降低，这可能会使预后不良。手术后胰岛素抵抗的增加跟手术侵害的程度有关。然而，手术前碳水化合物的补充减弱了手术后胰岛素的抵抗。本研究旨在探讨术中给予低剂量葡萄糖对术后胰岛素抵抗的影响。方法：接受颌面手术的患者根据手术程序被随机分为两个组。葡萄糖组接受含 1.5% 葡萄糖的醋酸格林溶液，对照组接受不含葡萄糖的醋酸格林溶液。用平均的葡萄糖输注率来量化胰岛素抵抗，分别在手术前一天和手术后第二天用 STG-22TM 仪器根据葡萄糖钳夹来估计葡萄糖输注率。手术中连续监测血糖水平。另外，围手术期测量血清胰岛素、酮体和 3-甲基组氨酸的变化。结果：葡萄糖组患者（11 人）在手术中输入葡萄糖 0.15±0.06 g/kg/h，对照组患者（11 人）没有输入葡萄糖。然而，两组在手术中和手术后血糖平均水平均稳定在低于 150 mg/dL。手术后，对照组的血清酮体显著增加（$p=0.0035$），而葡萄糖组血清酮体显著降低（$p=0.043$）。葡萄糖组的葡萄糖输注率的降低率（43.3±20.7%）显著低于对照组（57.7±9.3%）（$p=0.041$）。结论：术中小剂量给予葡萄糖能够抑制生酮作用和减轻手术后胰岛素抵抗，而不会导致高血糖症。

**关键词：胰岛素抵抗、葡萄糖钳夹、人工胰腺、生酮作用、3-甲基组氨酸**