

Effect of Intraoperative Fluid on Blood Glucose Level in Neurosurgery

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Abstract

There is a considerable controversy regarding glucose administration during intracranial surgery. However, recent animal and human studies suggest that hyperglycemia exacerbates ischemic brain damage and intraoperative hypoglycemia may not be a concern if the surgical procedure is less than 4 hours.

We, therefore, studied the blood glucose in neurosurgery with craniotomy in 90 Thai patients, divided into 3 groups. 30 patients in each group received balanced salt solution (0.9% NSS), 5 per cent glucose rate 60-80 ml/h and 5 per cent glucose rate more than 120 ml/h. Blood for the determination of glucose concentration was obtained after induction and every 2 hours later until the end of the surgery.

There was one male patient in group I who received balanced salt solution (0.9% NSS) had blood glucose concentration lowered to 57 mg% at 4 hours after induction. The patients in group II who received 5 per cent glucose solution at maintenance rate did not have hyperglycemia (161.20 ± 38.30 mg%). In group III ; patients given 5 per cent glucose infusion at the rate of more than 120 ml/h had hyperglycemia (236.75 ± 63.57 mg%) at 6 hours. In conclusion, we suggest that in Thai patients undergoing neurosurgical procedures; blood glucose levels should be checked intraoperatively if glucose is withheld from the intraoperative fluid regimen. Otherwise 80 ml/h of 5 per cent dextrose intravenous infusion should be given to the patients to prevent hypoglycemia.

Intraoperative glucose administration in neurosurgical patients has been considered controversial. Normally, 50-150 g of glucose is given intraoperatively to prevent hypoglycemia, to provide energy, to allow free water administration, to conserve protein, and to prevent ketosis⁽¹⁾. These

effects may benefit patients undergoing general surgery. However, recent animal and human studies suggest that hyperglycemia exacerbates ischemic brain damage⁽¹⁻¹⁶⁾. Although some studies suggest the beneficial effect of hyperglycemia on cerebral ischemia⁽¹⁷⁻¹⁹⁾ and some show no sig-

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nificant deleterious neurologic outcome from giving glucose - containing fluid intraoperatively⁽²⁰⁻²²⁾. On the other hand, hypoglycemia is dangerous because the primary energy substrate of the brain is glucose. Thus, if one is concerned about intraoperative hypoglycemia, occasional glucose levels by capillary blood glucose analysis should be done.

The aim of this study was to compare the effects of different intraoperative fluid regimens on the blood glucose level in Thai patients undergoing intracranial surgery and receiving a standard anesthetic technique at Ramathibodi Hospital.

PATIENTS AND METHOD

90 non-diabetic Thai adults undergoing elective craniotomy were randomly assigned to 3 groups. Group I (N = 30) received no glucose infusion (NG) with 0.9 per cent NaCl at rate 60-80 ml/h, Group II (N = 30) controlled glucose infusion (CG) with 5 per cent N/2 rate 60-80 ml/hr and Group III comprised of 30 patients to whom no fluid regimen was assigned, but who inadvertently received hourly intraoperative 5 per cent glucose-containing fluid of more than 120 ml. (uncontrol glucose infusion, NG Group)

All patients fasted 6-15 h preoperatively. Total fluid infusion rates were adjusted to keep stable hemodynamics. Additional fluid such as blood or colloid was given as appropriate.

Monitoring included NIBP, pulse rate, direct arterial pressure, CVP (for patients who were prone to severe volume change overload), oxygen saturation and blood sugar. We routinely used capillary blood to determine blood sugar by glucometer. In our study, we measured blood sugar before surgery and every 2 hours till the end of surgery.

Anesthesia was induced intravenously with 3-5 mg/kg sodium thiopental and 1-2 mg/kg succinylcholine or nondepolarizing muscle relaxants

as appropriate and was maintained by using 50-70 per cent nitrous oxide in oxygen, with supplemental doses of pancuronium, atracurium or vecuronium. The patient received either isoflurane or halothane according to the individual anesthesiologist's preference.

Statistical Analysis

Statistical significance was tested by ANOVA. Bonferroni inequality was used to correct multiple comparisons.

RESULTS

Patient demographic data is shown in Table 1. Characteristics of the study population in terms of age and weight were comparable. The majority of the patients had ASA physical status either 1 or 2.

All patients had comparable blood glucose concentrations before surgery. (Table 2) At 2 hours after induction, the blood glucose in group II and III was significantly higher than in group I. The increase was greater in those receiving glucose-containing solution at the rate of more than 120 ml/h (Group III).

Between group I (0.9% NSS) and II (5% dextrose solution at the rate of 60 - 80 ml/h) there was no statistically significant difference in blood glucose concentrations at 4 hours after induction. Patients in group III had higher blood glucose levels (> 200 mg/dl) than in group I and II at 2 and 4 hours after induction. In addition, one male patient in group I had blood glucose of 57 mg/dl 4 hours after induction

DISCUSSION

At the present time, intraoperative glucose administration in neurosurgical patients has been considered controversial. Many recent animal and human studies suggest that hyperglycemia exacer-

Table 1. Patient demographic data (90 cases).

	Sex (F/M)	Age (yr)	Physical status				Weight (kg)	Steroid (# of patients) received
			1	2	3	4		
Group I	21 / 9	42.73±11.72	8	18	2	2	61.7±11.59	1
Group II	13 / 17	42.53±17.78	12	15	1	2	58.3±10.99	17
Group III	14 / 16	42.97±17.12	5	22	2	1	56.93±9.88	4

Table 2. Serial blood glucose throughout surgery : mg/dl

	N	NG Group Blood glucose	N	CG Group Blood glucose	N	UG Group Blood glucose
After induction	30	111.4±33.39	30	131.43±46.82	30	137.06±53.47
2 hours after induction	30	125.70±34.43	30	155.30±45.46	30	199.86±36.34 A
4 hours after induction	19	127.31±29.52 D	18	151.00±47.62	12	193.50±33.03 B
6 hours after induction	3	138.33±35.53	5	161.20±38.30	4	236.45±63.51 C

Data presented as mean±S.D.

A $p < 0.05$ in all groups, i.e., NG vs CG, CG vs UG, and NG vs UG

B $p < 0.05$ UG vs NG and UG vs CG

C No statistical analysis was done because of too small a sample size.

D Hypoglycemia developed in one patient (blood glucose 57 mg/dl).

bates ischemic brain damage^(4-6,23,24). Whereas, other studies suggest that it either decreases⁽¹⁷⁻¹⁹⁾ or has no significant effect on the neurologic outcome⁽²⁰⁾.

Hypoglycemia which is defined as a glucose concentration below the lower limit of normal fasting plasma glucose (e.g. less than 70 mg/dl) ; on the other hand, it is dangerous because the primary energy substrate of the brain is glucose. Until the controversy of intraoperative glucose administration comes to an end, it is wise to manage glucose conservatively that is to keep normo-glycemia. Normally, an intraoperative glucose concentration in neurosurgical patients of not more than 200 mg/dl is acceptable. Our data shows that a low rate of glucose infusion (60-80 ml/h) produces a glucose level less than 200 mg/dl at least for 6 hours after induction. We did not use data at 6 hours after induction for the statistical analysis because the number of population decreased to eleven. Nevertheless, the trend of blood glucose concentration increased in patients receiving glucose containing intravascular fluid compared to the group in which glucose was withheld.

In contrast to the previous study of Sieber⁽²⁵⁾ et al which found no hypoglycemia ; we found one patient in group I who received no glucose containing intraoperative fluid had hypoglycemia; (blood glucose concentration of 57 mg/dl) this patient after being given 50 per cent glucose 50 ml intravenously had blood glucose which increased to 128 mg%. This patient was 28 years old, physical status I, weight 50 kgs. He had fasted from

8 o'clock in the evening to 11 o'clock next morning, 15 hours before the surgery for a brain tumor (meningioma). The vital signs during the operation were stable. This result probably was due to the nutritional status of Thai patients that may effect the production of glucose by the liver or he probably fasted for a longer period than usual. Thus ; Thai patients may be more prone to hypoglycemia than Western people.

One may think steroid use can effect plasma glucose. However, we did not take this into account nor were the amount of intraoperative blood loss and standardization of anesthetic agents. The fluid adjusted to keep stable hemodynamics was 0.9 per cent NSS, colloid, blood or blood component which had no effect on blood glucose. We designed the study this way so that we could use the conclusion of the study in our actual clinical practice for the whole neurosurgical patient population.

SUMMARY

Our study results suggest that blood glucose levels should be checked intraoperatively if glucose is withheld from the intraoperative fluid regimen, in neurosurgical patients otherwise 60-80 ml/h of 5 per cent dextrose intravenous infusion should be given to the patients to prevent hypoglycemia if surgery lasts more than 4 hours.

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REFERENCES

1. Biebuyck JF. Glucose : A reevaluation of its intraoperative use. *Anesthesiology* 1987; 67: 72-81.
 2. Newberg LA. Use of intravenous glucose solutions in surgical patients. *Anesth Analg* 1985; 64: 557-9.
 3. Pulsinelli WA, Levy DE, Sigsbee B, et al. Increased damage after ischemic stroke in patients with hyperglycemia with or without established diabetes mellitus. *Am J Med* 1983; 74: 540-4.
 4. Drummond JC, Moore SS. The influence of dextrose administration on neurologic outcome after temporary spinal cord ischemia in the rabbit. *Anesthesiology* 1989; 70: 64-70.
 5. Lam AM, Winn HR, Cullen BF, et al. Hyperglycemia and neurological outcome in patients with head injury. *J Neurosurg* 1991; 75: 545-51.
 6. Pulsinelli WA, Waldman S, Rawlinson D, et al. Moderate hyperglycemia augments ischemic brain damage : A neuropathologic study in the rat. *Neurology* 1982; 32: 1239-46.
 7. Nakakimura K, Fleischer JE, Drummond JC, et al. Glucose administration before cardiac arrest worsens neurologic outcome in cats. *Anesthesiology* 1990; 72: 1005-11.
 8. Berger L, Hakim AM. The association of hyperglycemia with cerebral edema in stroke. *Stroke* 1986; 17: 865-71.
 9. Longstreth WT, Inui TS. High blood glucose level on hospital admission and poor neurological recovery after cardiac arrest. *Ann Neurol* 1984; 15: 59-63.
 10. Duckrow RB, Beard DC, Brennan RW. Regional cerebral blood flow decreases during hyperglycemia. *Ann Neurol* 1985; 17: 267-72.
 11. Ashwal S, Schneider S, Tomasi L, et al. Prognostic implications of hyperglycemia and reduced cerebral blood flow in childhood near-drowning. *Neurology* 1990; 40: 820-3.
 12. Warner DS, Smith M-L, Siesjo BK. Ischemia in normo- and hyperglycemic rats : effects on brain water and electrolytes. *Stroke* 1987; 18: 464-71.
 13. De Salles AA F, Muizelaar JP, Young HF. Hyperglycemia, cerebrospinal fluid lactic acidosis, and cerebral blood flow in severely head-injured patients. *J Neurosurg* 1987; 21: 45-50.
 14. Lanier WL, Stangland KJ, Scheithauer BW, et al. The effects of dextrose infusion and head position on neurologic outcome after complete cerebral ischemia in primates : examination of a model. *Anesthesiology* 1987; 66: 39-48.
 15. Myers RE, Yamaguchi S. Nervous system effects of cardiac arrest in monkeys. *Arch Neurol* 1977; 34: 65-74.
 16. Gardiner M, Smith ML, Kagstrom E, et al. Influence of blood glucose concentration on brain lactate accumulation during severe hypoxia and subsequent recovery of brain energy metabolism. *J Cereb Blood Flow Metab* 1982; 2: 429-38.
 17. Zasslow MA, Pearl RG, Shuer LM, et al. Hyperglycemia decreases acute neuronal ischemic changes after middle cerebral artery occlusion in cats. *Stroke* 1989; 20: 519-23.
 18. Ginsberg MD, Prado R, Dietrich WD, et al. Hyperglycemia reduces the extent of cerebral infarction in rats. *Stroke* 1987; 18: 570-4.
 19. Jernigan J, Evans OB, Kirshner HS. Hyperglycemia and diabetes improve outcome in a rat model of anoxia / ischemia. *Neurology* 1984; 34 (suppl) : 262.
 20. Shapira Y, Artru AA, Cortev S, et al. Brain edema and neurologic status following head trauma in rat. *Anesthesiology* 1992; 77: 79-85.
 21. Parish BA, Webb KS. Hyperglycemia is not a poor prognostic sign in head-injured children. *J Trauma* 1988; 28: 517-9.
 22. Woo J, Lam C W K, Kay R, et al. The influence of hyperglycemia and diabetes mellitus on immediate and 3-month morbidity and mortality after acute stroke. *Arch Neurol* 1990; 47: 1174-7.
 23. Warner DS, Gionet TX, Todd MM, et al. Insulin - induced normoglycemia improves ischemic outcome in hyperglycemic rats. *Stroke* 1992; 23: 1775-81.
 24. De courten-myers GM, Kleinholz M, Wagner KR, et al. Fatal strokes in hyperglycemic cats. *Stroke* 1989; 20: 1707-15.
 25. Sieber F, Smith DS, Kufferberg J, et al. Effects of intraoperative glucose on protein catabolism and plasma glucose levels in patients with supratentorial tumors. *Anesthesiology* 1986; 64: 453-9.
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ผลของการให้สารน้ำต่อระดับกลูโคสในเลือดในผู้ป่วยที่ได้รับการวางยาสลบผ่าตัดสมอง†

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ได้ทำการศึกษาการให้สารน้ำที่มีกลูโคสและไม่มีกลูโคสทางหลอดเลือดดำในผู้ป่วยที่ได้รับการวางยาสลบความรู้สึกแบบทั่วไปเพื่อผ่าตัดสมอง ทั้งหมด 90 ราย โดยผู้ป่วย กลุ่มที่ 1 จำนวน 30 ราย ได้รับ 0.9% NSS ตามความต้องการสารน้ำของผู้ป่วย ผู้ป่วยกลุ่มที่ 2 จำนวน 30 ราย ได้รับสารน้ำ 5% D/N/2 ปริมาณ 60-80 มิลลิลิตรต่อชั่วโมง และผู้ป่วยกลุ่มที่ 3 จำนวน 30 รายได้รับสารน้ำ 5% D/N/2 ปริมาณมากกว่า 120 มิลลิลิตรต่อชั่วโมง ได้ทำการตรวจวัดระดับกลูโคสในเลือดโดยใช้ Glucometer ในระยะเวลาก่อนเริ่มให้ยาระงับความรู้สึก และหลังเริ่มให้ยาระงับความรู้สึกทุก 2 ชั่วโมง จนเสร็จสิ้นการผ่าตัด

ผลของการศึกษาพบว่าในผู้ป่วยกลุ่มที่ 1 มีผู้ป่วยชาย 1 รายมีภาวะกลูโคสในเลือดต่ำ (กลูโคสในเลือดเท่ากับ 57 มก/ดล.) ที่เวลา 4 ชั่วโมง หลังเริ่มการผ่าตัด ส่วนในกลุ่มที่ 2 พบว่าระดับกลูโคสในเลือด มีค่าต่ำกว่า 200 มก/ดล เป็นเวลาอย่างน้อย 6 ชั่วโมงของการผ่าตัด โดยในกลุ่มที่ 3 พบว่ามีระดับกลูโคสสูงเกินกว่า 200 มก/ดล ใน 6 ชั่วโมงสรุปได้ว่าการให้สารน้ำที่มีกลูโคส เช่น 5% D/N/2 ในผู้ป่วยที่ได้รับการวางยาสลบความรู้สึกผ่าตัดสมองในปริมาณ 60-80 มล/ชม ในผู้ใหญ่ เป็นวิธีการที่ปลอดภัย โดยไม่พบว่ามีภาวะกลูโคสในเลือดต่ำหรือสูงเกินไป ในกรณีที่ไม่สามารถตรวจวัดระดับกลูโคสในเลือดได้.

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