Bispectral index may not reflect the depth of anaesthesia in a patient with glycogen storage disease type I

Editor—A 17-yr-old boy with glycogen storage disease (GSD) type I was undergoing resection of multiple hepatic adenomas under general anaesthesia. Before operation, the blood glucose was kept at 4–5 mmol litre$^{-1}$. After the induction of anaesthesia and tracheal intubation, continuous inhalation of sevoflurane and i.v. infusion of remifentanil were started. Rocuronium 0.2 mg kg$^{-1}$ was given i.v. every 30–40 min. Arterial and central venous cannulas were placed. Bispectral index (BIS) was used to measure the anaesthesia depth. Blood glucose level and arterial gas analysis were done every 30 min. Dextrose 10% and sodium bicarbonate 5% were given intraoperatively guided by the blood glucose level and the acid–base status, respectively. After induction, the inhaled concentration of sevoflurane and infusion speed of remifentanil were adjusted to keep the BIS score between 40 and 60. During the first 30 min, the heart rate and arterial pressure were normal. After skin incision, the heart rate and arterial pressure increased gradually, whereas the BIS score remained between 40 and 60. Blood glucose level remained below 5 mmol litre$^{-1}$ with a lowest reading of 3.5 mmol litre$^{-1}$. The rate of infusion of dextrose 10% was increased and the BIS score increased to 70 with the normalization of blood glucose level. To increase the anaesthesia depth, we increased the inhaled concentration of sevoflurane and the infusion rate of remifentanil. The heart rate and arterial pressure returned to normal and the BIS score decreased to 45. After operation, the patient was discharged home on day 10.

GSD type I is caused by deficiency of glucose-6-phosphatase activity which results in an inability to form glucose during periods of fasting which could cause intraoperative hypoglycaemia and lactic acidosis. The brain is dependent upon a continuous supply of glucose. Thus, keeping the blood glucose level normal is important for the brain function. Recording EEG during insulin-induced hypoglycaemia in 14 young adults, Glass and colleagues$^2$ reported a decrease in alpha-activity and an increase in delta- and theta-activities, which would be similar to the findings in patients given general anaesthesia. BIS is derived from pooled data of EEG changes in patients given different anaesthetics.$^3$ In our case, the patient’s BIS score fluctuated between 40 and 60 after skin incision. It appeared that the depth of anaesthesia was sufficient, but the heart rate and arterial pressure increased gradually. At the same time, hypoglycaemia developed. When the blood glucose level was returned to normal, BIS score increased followed by the changes in heart rate and arterial pressure. In view of the known EEG changes associated with hypoglycaemia state as described above, the decrease in BIS during anaesthesia may have been due to hypoglycaemia and not necessarily to deepening of anaesthesia. It is important to remember that BIS score might not reflect the depth of anaesthesia in patients with GSD type I who are prone to develop hypoglycaemia.

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