space was 25 per cent greater than that calculated to be necessary to ensure anesthesia of the ulnar nerve. This larger volume may explain the motor and sensory block of the left cervical and brachial plexus. However, a bilateral sensory block extending from C2 to T3 and bilateral diaphragmatic and intercostal paralysis cannot be explained on the basis of volume alone. Partial muscle paralysis, as evidenced by incomplete diaphragmatic paralysis and ability to lift the head, occurred in patient 1. The longer duration and completeness of muscle paralysis in patient 2 may be attributable to the higher concentration of lidocaine and the addition of tetracaine.

Subarachnoid injection of the anesthetic solution would explain the bilateral motor and sensory blockade in both cases. But the large volume of anesthetic solution and the high site of subarachnoid injection would then be expected to produce total spinal anesthesia, rather than the limited anesthesia which developed. Retained consciousness, integrity of vital centers, and intact cranial nerve activity were evident in patient 1, as no general anesthetic was used. In patient 2, consciousness returned at the end of the procedure although motor and sensory block persisted. These factors favor an epidural rather than a subarachnoid injection.

A caudad direction of the needle was maintained in performing these blocks. It is possible that the needle was inserted to a greater depth than intended and contacted the body rather than the U-shaped end of the transverse process of the sixth cervical vertebra. When the needle was "walked" caudally and paresthesias were elicited, it probably entered the epidural space. Thus, the anesthetic solution was deposited in the ipsilateral cervical epidural space (fig. 1) and spread to include the contralateral cervical and bilateral thoracic epidural spaces. The spread of anesthetic solution to the epidural spaces as a result of injection of a large volume, although possible, is unlikely. The resultant extensive epidural blockade, including bilateral phrenic nerve paralysis, could have been hazardous if unrecognized and untreated.

The danger of inadvertent epidural injection should be considered when interscalene block is used.

REFERENCE


Hyperosmolar Hyperglycemic Non-ketotic Coma
Following General Anesthesia:
Report of a Case

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Hyperosmolar hyperglycemic non-ketotic coma is rarely considered as a potential complication of anesthesia. The following is a case report of a young woman who died shortly after general anesthesia with a clinical syndrome of coma, hyperglycemia, and hyperosmolarity without ketosis.

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REPORT OF A CASE

A 32-year-old woman weighing 260 pounds was admitted to the hospital with a five-day history of epigastric and substernal discomfort, nausea and vomiting. Receiving ward laboratory data included: platelets 4,000/mm²; hemoglobin 8.7 gm/100 ml, leukocyte count 11,600/mm³. Blood pressure was 130/90 torr; temperature was 99.6 F. Physical findings were limited to a petechial rash over the neck, anterior chest and palate. Past medical history included elevated blood pressure during pregnancy at the age of 17 years, but the patient had been normotensive for the past five years. There was no history of recent viral infection or exposure to toxins, and her only medication was birth control pills.
RENAL DISEASE
DIURETICS
DEBILITATION

DEHYDRATION

OSMOTIC DIURESIS

HYPERALIMENTATION
MANNITOL
SORBITOL DIALYSIS

HYPEROSMOLARITY

INHIBITS INSULIN RELEASE

IMPARED FFA METABOLISM

CNS DYSFUNCTION

DEXTROSE
STEROIDS
BURNS
PANCREATITIS

NO KETOSIS

Fig. 1. Proposed pathogenesis of HHNK (modified from Gerich et al.), showing the numerous clinical situations which can result in the vicious circle of hyperglycemia, dehydration, and hyperosmolarity, eventually leading to CNS dysfunction and coma.

She was diagnosed as having thrombocytopenic purpura and received prednisone, 200 mg daily. During the next three days the platelet count rose to 18,000/mm$^3$, jaundice developed, the petechial rash spread, and splenectomy was deemed necessary. On the day prior to operation, urine became + for sugar but negative for acetone. No insulin was given. Results of preoperative laboratory studies were: hemoglobin 7.5 gm/100 ml; leukocyte count 22,000/mm$^3$; fasting blood sugar 350 mg/100 ml; BUN 21 mg/100 ml; Na$^+$ 139 mEq/l, K$^+$ 3.5 mEq/l, Cl$^-$ 99 mEq/l, total bicarbonate 27 mEq/l. Because of persistent anemia, 500 ml of packed cells and 500 ml of bank blood were given the evening before operation.

N$2$O-O$2$—Ethane endotracheal anesthesia was used for splenectomy. Blood pressure ranged between 140/90 and 105/70 torr throughout the procedure. The patient received prednisone, 50 mg, preoperatively, and hydrocortisone, 200 mg, im, during the operation. Estimated blood loss was 2,500 ml; fluids replaced were 5 units of bank blood, 500 ml of 5 per cent dextrose solution, and 500 ml of Plasmalyte in water.

The patient remained in the recovery room for two hours. Blood pressure ranged between 120/70 and 140/90 torr, but respiratory rate increased from 20 to 28/min during this time. Plasmalyte, 1,500 ml in water, was infused. No Foley catheter or CVP monitor was placed due to the risk of precipitating hemorrhage. The patient was discharged from the recovery room awake and fairly well oriented, and when she returned to her room was noted to answer to her name and then doze. Temperature at that time was 101.2 F, and respiratory rate was 28/min.

During the next three hours respiratory rate increased to 46/min and heart rate increased to 134 beats/min, with blood pressure stable at 130/80 torr. The patient was restless and uncooperative. Laboratory tests revealed: hemoglobin 8.1 gm/100 ml; leukocyte count 31,300/mm$^3$; platelets 21,000/mm$^3$; arterial blood gases breathing room air, Po$_2$ 85 torr, Pco$_2$ 27 torr, pH 7.40, base excess $-$7 mEq/l. The patient received 3 units of bank blood, 89 mEq NaHCO$_3$, and 500 ml 5 per cent dextrose in water in the next two hours. A Foley catheter was placed and 1,700 ml of urine obtained which was + for sugar and negative for acetone. The patient was given 20 units of regular insulin subcutaneously and 200 mg hydrocortisone, iv. By this time the respiratory rate was 58/min, heart rate was 130 beats/min, and blood pressure was 117/70 torr. The patient was confused, could not respond to questions or commands, and thrashed about in bed, although there were no localizing neurologic signs. Laboratory
results at this time were: blood sugar 960 mg/100 ml; BUN 42 mg/100 ml; Na 134 mEq/l, K 5.5 mEq/l, Cl 86 mEq/l, total bicarbonate 13 mEq/l. Calculated serum osmolality was 374 mOsm/l.

At this point it was apparent that the patient was in hyperosmolar hyperglycemic non-ketotic coma. She received another 50 units of regular insulin, iv, but she became asystolic within the hour and could not be resuscitated. Permission for autopsy was not granted, but examination of microscopic sections of the spleen removed at surgery showed multiple small-vessel fibrous thrombi and microinfarctions characteristic of thrombotic thrombocytopenic purpura.

**DISCUSSION**

The pathogenesis of hyperosmolar hyperglycemic non-ketotic coma (HHNKC) is poorly understood, but seems to involve a vicious circle consisting of dehydration, hyperosmolarity, and hyperglycemia (fig. 1). This cycle can be initiated by several clinical situations. Among surgical patients the most common predisposing factors are large surface-area burns and massive steroid therapy. These both are capable of causing hyperglycemia, as are pancreatitis and excessive infusion of dextrose solutions. Hyperosmolarity leading to HHNKC can result from hyperalimentation infusions, mannitol therapy, peritoneal hemodialysis. Vigorous diuretic treatment, chronic renal disease, or debilitation from pneumonia or cerebrovascular accident can lead to dehydration and ultimately initiate HHNKC.

Absence of ketosis is thought to be caused by impaired release of free fatty acids from adipose tissue as a result of hyperosmolarity and decreased levels of lipolytic hormones. Central nervous system dysfunction in the presence of extracellular hyperosmolarity supervenes from intracellular dehydration, with brain shrinkage and damage to vessels, or perhaps from intracellular breakdown of larger molecules in a protective effort to generate new osmotic solute.

Frequently, these patients are not known to be diabetic prior to the onset of coma, and rarely are they insulin-dependent after treatment. Our patient was receiving massive doses of steroids in an attempt to elevate a severely depressed platelet count, and as a result became hyperglycemic and glycosuric. Without insulin coverage before or during operation, she rapidly developed HHNKC in the postoperative period.

The anesthesiologist confronted with a patient at risk of developing HHNKC should maintain a high index of suspicion. Blood samples for sugar and ketones are indicated during anesthesia, and in long procedures urinary catheterization is warranted. Judicious infusion of dextrose-containing solutions, particularly combined with isotonic saline solution, is mandatory. Appropriate insulin coverage should be given when necessary.

Postoperatively, should signs of restlessness, disorientation or prolonged somnolence occur concomitant with non-ketotic hyperglycemia (600–1,000 mg/100 ml), treatment should be directed toward cautiously lowering blood sugar with insulin. Regular insulin, 50 units iv and another 50 units subcutaneously, is a maximum recommended starting dose, with subsequent doses depending on the rapidity of the decrease in blood sugar concentration. Too-strict reduction in plasma glucose not only may cause loss of intravascular volume and hypovolemic shock, but also may result in severe intracellular edema and increased intracranial pressure as plasma hypertonicity decreases. Clements, Procek, and Winegrad showed that hyperglycemia raises brain sorbitol content via the polyol pathway, and that a rapid decrease in plasma glucose results in walter’s entering brain cells, causing cerebral edema.

If cardiovascular stability is maintained, 0.45 per cent saline solution is advocated as the replacement solution of choice to correct dehydration; usually, 2–3 liters are needed in the first hour or two. Should dehydration be severe enough to cause hypotension, physiologic saline solution should be infused rapidly until blood pressure is satisfactory. Potassium replacement must also be supplied, since tissue K+ stores are depleted even though serum [K+] may be elevated as a result of acidosis. Because serum [K+] falls rapidly in patients with sudden intracellular movement of glucose and infusions of fluids, 20 mEq of KCl in the second or third bottle of 0.45 per cent saline solution is recommended.

HYNKC is alleged to have 40 per cent mortality. Our case demonstrates that the syndrome can develop rapidly just prior to, dur-
Thiopental Anaphylaxis: A Case and a Method for Diagnosis

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Severe allergic reactions to sodium thiopental are extremely rare, especially in light of the vast number of injections given throughout the world. Seven cases 1-7 of anaphylactic shock due to thiopental have been reported to date. This seems to indicate either that very few such episodes occur, or that this event may go undiagnosed or unsuspected. The following is a case report of thiopental anaphylaxis confirmed by the basophil degranulation test.8,9

REPORT OF A CASE

The patient, a healthy 40-year-old caucasian woman, was admitted to the hospital early in 1971 for repair of bilateral hallux valgus. Thiopental had been given for uterine dilatation and curettage in 1937, 1938, and 1967. There was a history of a rash following treatment of a sore throat with penicillin, and the patient had been instructed to avoid penicillin. She had also experienced coryza from perfumed cosmetics, but did not have seasonal allergies or eczema. Physical examination revealed no abnormalities other than the orthopedic problem. Preoperative hemoglobin, urinalysis, serum electrolytes, chest x-ray, and electrocardiogram were normal.

The patient had an uneventful one-hour anesthetization for correction of the hallux valgus. Anesthesia was induced with 450 mg sodium thiopental and maintained with nitrous oxide, oxygen, and halothane.

Fourteen days later the patient returned unpregnated to the operating room for application of below-knee casts. Induction of anesthesia with 275 mg of thiopental, 2.5 per cent, was followed by atropine sulfate, 0.2 mg, iv. Immediately following induction, N2O:O2 (6:3 liters) was administered by mask via a Magill circuit. After a few breaths, salivation and cyanosis were noted, together with some difficulty in manual ventila-