

Levels at Night Near Hypoglycemic

Glucose from page 1

The investigators fit the women with continuous glucose monitoring devices that measured their blood glucose levels every 5 minutes for 72 hours.

Patients were asked not to modify their lifestyles or nutritional habits during the study.

The women's overall mean blood glucose (79.3 mg/dL) and mean fasting blood glucose levels (75 mg/dL) were "much, much lower than was previously reported by others."

Mean nighttime blood glucose levels (66 mg/dL) "almost represented hypoglycemia," but such values may actually represent "normal physiology during the first trimester in nondiabetic patients," Dr. Yogeve said.

The postprandial glycemic profile of the women was the same after each meal. Mean blood glucose values started at 79 mg/dL just before a meal and rose to 106 mg/dL 60 minutes after the meal; it reached a high of 112 mg/dL 74 minutes

after the meal. The values reached 99 mg/dL at 2 hours and 82 mg/dL at 3 hours.

The fasting and overall mean blood glucose levels were similar in 18 obese (defined as a body mass index greater than 27.3 kg/m²) and 44 nonobese women. Compared with nonobese women, however, those who were obese had significantly higher mean preprandial blood glucose levels (73 mg/dL vs. 88 mg/dL) and significantly lower mean nighttime blood glucose concentrations (69 mg/dL vs. 60 mg/dL).

The obese patients were characterized by a higher postprandial peak value, a

longer time interval to reach the postprandial peak value, and higher mean blood glucose levels during the 3 hours after each meal, Dr. Yogeve said.

In different measures of blood glucose levels, the women as a whole were at least one standard deviation below the recommended threshold for the treatment of diabetes during pregnancy, he said.

Because of the difficulty of performing continuous glucose monitoring in nondiabetic women with a normal pregnancy, the study did not involve a specific cohort of patients but instead mostly included doctors' wives, midwives, and nurses, Dr. Yogeve said. ■

Epidurals Can Aid Parturients With Aortic Stenosis

BY SHARON WORCESTER
Southeast Bureau

HOLLYWOOD, FLA. — Maintaining hemodynamic stability is particularly important in the anesthetic management of parturients with aortic stenosis, and the use of a slowly titrated epidural or combined spinal-epidural with a reduced spinal anesthesia dose appears to provide this stability in most patients, findings from a case series suggest.

The cases, including six patients with moderate aortic stenosis and six with severe

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aortic stenosis, also suggest that invasive monitoring facilitates anesthetic management in some patients, and that special attention to postoperative analgesia, monitoring, and volume status can prevent hemodynamic instability and complications.

Dr. Alexander Ioscovich reported in a poster at the annual meeting of the Society for Obstetric Anesthesia and Perinatology.

The 12 patients were treated at two university hospitals, and compose all the cases of aortic stenosis in parturients seen at those hospitals from 1990 to 2005. Five of six patients with moderate aortic stenosis, and three of six with severe aortic stenosis had regional anesthesia; two with moderate aortic stenosis, and four with severe aortic stenosis had invasive monitoring; and one with critical symptomatic aortic stenosis had intraoperative transesophageal echocardiography under general anesthesia. There were no cases of hemodynamic instability or anesthetic complications, although one patient had a failed epidural, wrote Dr. Ioscovich of Sunnybrook Women's College Hospital, Toronto.

Although neuraxial anesthesia has traditionally been considered contraindicated in aortic stenosis patients, the findings suggest this approach is useful in all but the most severe cases, he concluded. ■



PONV* & Your Practice: A Novel Target in PONV, Article 3 of 3

The Emerging Role of Substance P in PONV

A new emetic pathway to target for PONV

More than 50 years after its discovery, substance P is now recognized as an important neurotransmitter in the central and peripheral nervous systems. Substance P, which belongs to the neurokinin (NK) family of neurotransmitters, plays an integral role in relaying noxious and aversive sensory information to the brain. Originally studied in disease models associated with pain transmission, substance P and its NK₁ receptor represent the newest emetic pathway implicated in PONV.¹⁻³

Key neurotransmitter receptors located in brainstem vomiting center

The brainstem vomiting center contains the essential neurocircuitry required for producing the emetic response (see Figure). This anatomical region contains high concentrations of several key neurotransmitters involved with emesis, including receptors for choline, histamine, dopamine, opioids, serotonin,⁴ and substance P.² By serving as sensors that can be stimulated by drugs, electrolytes, and metabolic chemicals, these receptors relay impulses to the vomiting center and initiate the vomiting reflex.⁵ Blockade, or antagonism, of these receptor sites is the mechanism of action of many pharmacologic antiemetic agents commonly used for PONV. Various antiemetics exhibit different affinities for emetic neuroreceptors and, therefore, target different emetic neuroreceptors.

Different types of stimuli trigger different emetic pathways

The vomiting center can receive stimuli from several areas, including afferents from both the periphery and the central nervous system.⁵ Because afferent systems trigger the release of various neurotransmitters, receptor antagonists may be particularly effective against one type of vomiting and less effective against emesis induced by other stimuli.⁶ For example, by targeting receptors in the vestibular apparatus, antihistamines are particularly useful for motion sickness and PONV associated with middle ear surgery.^{4,5} Conversely, 5-HT₃ receptor antagonists, which act primarily on abdominal vagal afferents, do not exhibit an antiemetic response to motion, yet exhibit potent antiemetic response activity against acute chemotherapy-induced nausea and vomiting.⁶

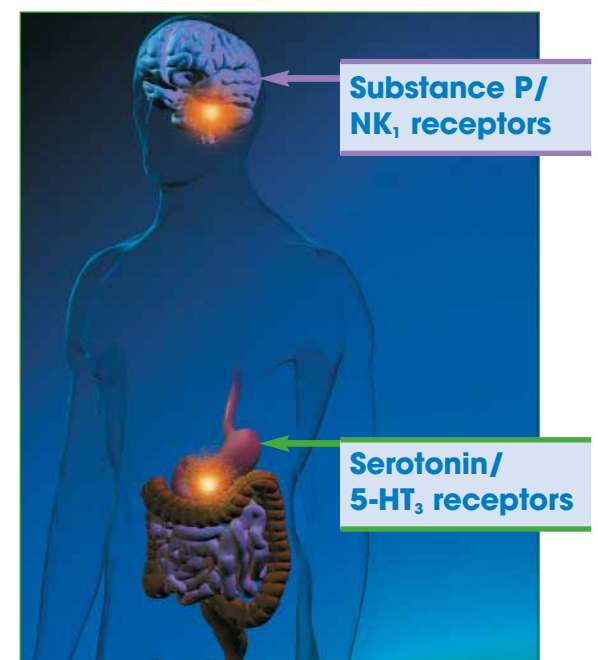


Figure. Proposed primary mechanism of 2 most recently identified emetic pathways.⁶⁻⁹

*Postoperative nausea and vomiting.