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. Yogev 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. Yogev 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 non-diabetic 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. Yogev said.

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 NK receptor represent the newest emetic pathway implicated in PONV.1

Key neurotransmitter receptors located in brainstem vomiting center

The brainstem vomiting center contains the essential neuromicrocircuity 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, and substance P. 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. 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.2 Because effector 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.3 For example, by targeting receptors in the vestibular apparatus, antihistamines are particularly useful for motion sickness and PONV associated with middle ear surgery.4 Conversely, 5-HT 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.5

Targeting NK receptors for an effective antiemetic

Evidencing efficacy in the treatment of 2,000 patients in a class IIb, randomized, double-blind, placebo-controlled study, it is well accepted that substance P/NK receptors are ideally suited as a site for antiemetic treatment of postoperative nausea and vomiting.6

Substance P/NK receptors

Serotonin/5-HT receptors

Figure. Proposed primary mechanism of 2 most recently identified emetic pathways.7,8

*Postoperative nausea and vomiting.