Recurrent Complete Heart Block in a Healthy Patient during Laparoscopic Electrocauterization of the Fallopian Tube

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OVER the past three decades laparoscopy has become a routine procedure for tubal sterilization. Phillips¹ reviewed more than 100,000 pelvic laparoscopies and found a 0.65% incidence of hemorrhage and a 0.3% incidence of cardiac arrest. The most common anesthetic-related causes of death during laparoscopy were attributed to hypoventilation and cardiopulmonary arrest,² vague diagnoses that do not delineate the mechanism(s) involved. Excessive vagal activity is rarely mentioned as a cause of cardiac morbidity during laparoscopic sterilization.³

We report a third-degree atrioventricular block in a healthy woman during fallopian tube cauterization. The case clearly establishes the close temporal relationship between tubal cauterization and the onset of complete heart block.

Case Report

A 36-yr-old obese woman (height, 163 cm; weight, 92 kg) was scheduled for laparoscopic sterilization as an outpatient. Her medical history, the results of physical examination, and all laboratory data were within normal limits. She denied ever having any cardiac symptoms or disease. Her preoperative electrocardiogram (ECG) was entirely normal. She also denied ever having any fainting spells or vasovagal type reactions. She was not premedicated for the surgery. After a peripheral intravenous catheter was placed, ECG, pulse oximeter, and noninvasive blood pressure monitors were applied. The preinduction blood pressure (BP) was 125/67 mmHg, and the heart rate was 70 beats/min. After preoxygenation, 3 mg of curare and 150 μg of fentanyl were given intravenously, followed by 200 μg of propofol and 120 mg of succinylcholine to facilitate tracheal intubation. Anesthesia was maintained with a mixture of oxygen, nitrous oxide, and sevoflurane (1.3% end-tidal concentration), and muscle relaxation was accomplished with 5 mg of pancuronium. The patient’s BP and heart rate were stable during the first 15 min of the surgery. A Verres needle was placed through the infraumbilical area, and pneumoperitoneum was instituted with 4 l of CO₂ with a maximum pressure of 17 cm H₂O. The laparoscopic trocar was inserted into the abdominal cavity without any complications. Laparoscopic examination of the pelvic organs appeared normal. A second puncture was made for the coagulation instrument. The BP was 111/59 mmHg, and the heart rhythm was sinus with a rate of 55 beats/min, and the end-tidal CO₂ was 39 mmHg. Approximately 30 min after the beginning of the surgery, when the surgeon initiated bipolar electrocauterization of the fallopian tube, the ECG showed a 9-s period of complete heart block with six nonconducted P waves interrupted by one high-normal (or normal sinus) beat. This 9-s period was followed by another 4-s period of asystole. Several ECG complexes after this asystolic period were consistent with first-degree atrioventricular block; PR interval was 0.52 s, but it normalized over the next three beats to less than 0.2 s (fig. 1, upper and middle panels). The pulse oximetry tracing was absent during the period of ventricular asystole, confirming the absence of perfusion. We requested immediate cessation of all surgical activities and prompt decompression of the pneumoperitoneum. Before we were able to administer any drug and while unstimulated, the patient’s heart resumed normal sinus rhythm. Immediately after the event, the end-tidal CO₂ was 36 mmHg, and the oxyhemoglobin saturation measured with pulse oximetry was 99%. The patient was observed for several minutes, and because everything appeared normal, surgery was resumed without reintubating the abdomen with CO₂. However, the moment electrocauterity touched the fallopian tube a similar episode occurred (fig. 1, bottom tracing). We stopped the surgery and examined the equipment for malfunction (current leaks, short circuit, proper placement of electrocauterization dispersive plate), but everything appeared normal. A third electrocautery burst to the fallopian tube immediately reproduced complete heart block. Glycopyrrolate, 0.4 mg, was then administered, and this pharmacologic vagolyis increased the heart rate to 83 beats/min. Pneumoperitoneum was reinstalled, and the surgery was finished without any further problems. Postoperatively, a cardiologist with expertise in electrophysiology was consulted for a review of the intraoperative ECG tracings. He concluded that the complete heart block followed by a first-degree atrioventricular block suggested that the events were induced with parasympathetic-vagal stimulation, therefore, he did not deem it necessary to proceed with electrophysiologic studies.

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The patient's electrocardiogram was monitored overnight, and she was discharged home the following day.

**Discussion**

Peterson *et al.* reviewed deaths during tubal sterilization in the United States between 1977 and 1981 and found that "complications of anesthesia," specifically cardiopulmonary arrest was the leading cause of mortality. The retrospective nature of this review and the lack of today's anesthesia monitoring standards make it difficult to precisely identify the intraoperative events that led to "cardiorespiratory arrest" in young and otherwise relatively healthy patients. In Peterson's review, one patient undergoing tubal sterilization died from refractory complete heart block that was attributed to CO₂ embolism. Carmichael was the first who postulated the possible role of vagal stimulation caused by CO₂-induced peritoneal stretch as a possible mechanism of collapse during laparoscopic sterilization. Brantley and Riley in 1988 described two healthy women who experienced severe bradycardia during electrocoagulation of the fallopian tubes. However, although their primary suspicion was a vagally induced bradycardia, they still considered the possibility of CO₂ embolization in their cases. Our case indicates that stimulation of the fallopian tube during bipolar electrocoagulation may increase vagal tone, which in turn may induce inhibition of cardiac conduction through the atrioventricular node. Although, theoretically the CO₂ embolism could have caused electrocardiographic changes similar to those we saw in our patient, we expect that they would not have been so transient in nature. Therefore, we promptly excluded CO₂ embolism from the differential diagnosis. In addition to complete heart block, our patient's ECG showed a transient, first-degree atrioventricular block that resolved quickly. This suggested the presence of acutely increased vagal tone, which dissipated immediately after cessation of surgical stimulation. Further, the temporal association of electrocardiographic changes with cauteryization, as well as their repetitive nature with the same electrical stimulus, suggests a directly elicited reflex rather than an indirect one caused by stretching of the peritoneum as was proposed by Carmichael, especially because the second and third episode occurred in the absence of pneumoperitoneum and were elicited solely by cauteryization.

The issue that remains to be reviewed is the extent of vagal innervation of the female reproductive organs. Traditional teaching is that the vagus nerve innervates all viscera from throat to transverse colon. However, recent anatomic studies in rats indicate that the vagus extends further than was originally thought (to innervate caudal colon, uterus, and ovary) and that the stimulation of reproductive organs in rats may produce BP changes. The animal studies have provided anatomic and electrophysiologic evidence to support the existence of a brainstem–uterus circuit that involves the vagus nerve. Changes in the discharge frequency...
of the nucleus of the tractus solitarius in one study were evoked by mechanical cervicovaginal distention in the rat. In a bovine study, Welento et al. showed that the central nervous system sends a small number of nerve fibers from the vagus nerve (medulla oblongata) directly to the uterus. Although little is known about vagal innervation of the reproductive organs in humans, Engel reviewed a group of rare reflexes that are mediated by the vagus nerve, and two of them — the auriculouterine reflex in women and the auriculogenital reflex in men and women — may be pertinent to our case because they suggest the existence of vagal innervation of the human uterus and sex organs. In these reflexes, the vagus nerve forms a bridge between a circum- scribed area of the skin, mostly the external auditory meatus, and the uterus and some male and female sex organs. Afferent impulses from female reproductive organs may be conducted via either the vagus nerve or spinal cord. Vagotomy completely eliminates the neuron response in the rat nucleus tractus solitarius to uterine horn stimulation but not to cervix or vaginal stimulation. However, spinal cord transection eliminates the response to stimulation of all three pelvic reproductive structures, suggesting that information from these parts reach the nucleus tractus solitarius along spinal routes. Responses to uterine horn stimulation may require spinal and vagal neural pathways.

Nothing is known how this vagal reflex operates in the male, such as during stimulation of organs of the same embryonic origin (e.g., spermatic cord, testicles). However, surgical literature occasionally described severe bradycardia during manipulation of spermatic cord, suggesting that this rare reflex may be elicited in both sexes with stimulation of embryologically equivalent structures.

We described a patient in whom electrocauterization of the fallopian tubes caused reflexive complete heart block. We believe that vagal overactivity could have been elicited either through an afferent impulse originating either from the fallopian tube itself or from the ovary or uterus after the electric current was propagated to these structures. Our treatment consisted of prompt interruption of surgical stimulation, release of abdominal insufflation, and administration of an anticholinergic drug, glycopyrrolate.

References