Transient Complete Heart Block Occurring during Nasal Irrigation

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Cardiac standstill and sudden death have been reported in operative procedures of the eyes, ears, nose, and throat and in intubation procedures. The exact mechanism and cause of these episodes are not entirely clear. The question of vagal effect with inhibition of the heart beat has been considered as a factor. The present case is reported because of the possibility of cardiac inhibition by vagal reflex. The stimulating factor in this patient was a high pressure spray of isotonic saline solution in the area of the sphenoid sinuses.

Case Report

The patient was a 54-year-old white woman who was admitted to St. Joseph's Infirmary in collapse on February 3, 1959. In her past history there was a rather marked psychoneurotic background after a surgical menopause in 1930. The chief symptom was headache, which had been related on occasion to sinusitis requiring frequent local treatments. Another symptom was substernal pain not typical of, but considered to be consistent with coronary artery insufficiency. This view was supported by minor electrocardiographic changes (fig. 1A) in January 1958.

On February 3, 1959, the patient collapsed in the chair of an otolaryngologist while receiving a forceful jet spray of isotonic saline solution into the area of the sphenoid sinus. She quickly became cyanotic and lost consciousness. She was placed in the recumbent position, and oxygen was administered by catheter orally. When examined 5 minutes later, she was unconscious, ashen, perspiring profusely, and had slow gasping respirations. The neck veins were distended but the lungs were clear. The blood pressure was unobtainable, and the pulse was faint and extremely slow. The heart beat was regular, with a rate of 32. During examination she had a brief major convulsion. Epinephrine 1:1000, 0.5 mL, and 1 mg. of atropine were given intramuscularly. An electrocardiogram showed an idioventricular rhythm and atrial fibrillary waves (fig. 1B). Over a period of 15 minutes her condition improved although the electrocardiogram remained unchanged. She was then transferred to the hospital by ambulance about 45 minutes after the onset of trouble. She was unconscious at this time, and oxygen was administered during the transfer by a Boothby mask.

On admission to the hospital she was placed in an oxygen tent and a dilute solution of norepinephrine (Levophed) was started intravenously, even though her pulse was about 70 on admission and her blood pressure was 112/84. She gradually regained consciousness, and 2 hours later the blood pressure was 125/85, and the pulse was 74. With this improvement Levophed was discontinued after the administration of only 20 mL. At this time weakness of the left hand, slurring of speech, and bilateral Babinski reflexes were observed. The situation seemed to be one of acute myocardial and cerebral anoxia, with the inciting episode uncertain. However, 12 hours later the electrocardiogram (fig. 1C) reverted to the same pattern as before the episode. The blood pressure and pulse were normal and the patient was comfortable except for minor precordial distress; but she still had weakness of the left hand, a Babinski reflex on the left, and her reactions were slowed. Evidence of myocardial damage was demonstrated by elevation of the serum transaminase (100 units on admission and 76 units on the seventh day), and by the development of T-wave changes (fig. 1D) on the fourth day, indicating inferior myocardial involvement. Blood electrolyte studies and routine blood examinations were normal, except for an admission white count of 12,600.

The patient was discharged after 2 weeks in the hospital and within 2 months had returned to normal activity. There has been no evidence of further cardiac difficulty and the electrocardiographic pattern (fig. 1E) returned to that taken prior to the episode. From the neurologic standpoint there remained only slight clumsiness of movements of the left hand.
Discussion

Clinically, this patient developed a sudden arrhythmia, probably a period of cardiac standstill followed by complete heart block, during a nasal procedure, and developed cerebral and myocardial anoxia and necrosis during the resultant period of decreased blood flow. The initiating factor in this arrhythmia is thought to be a vagovagal or trigemino-vagal reflex. A myocardial infarct occurring about the time of the nasal irrigation could have produced a sudden arrhythmia, but such a possibility seems remote.

The vagal reflexes are mechanisms of great importance, and are often a factor in the production of cardiac arrest. Reid and associates' described “the reflex nature of the derangements in cardiac dynamics following mechanical irritation of the mucosa of the respiratory or gastro-intestinal tracts by a wide variety of contrivances, such as intratracheal tubes and catheters, bronchosopes, gastroscopes, esophagoscopes, inflation of cuffs, etc., or even spraying of the throat with water.” He described a child who developed cardiac arrest during adenoidectomy, and developed further episodes of arrest during removal of adenoidal packs the next day. Further manipulation was deferred until the administration of atropine, following which the packs were removed without incident. Weiss and Ferris6 described a patient with an esophageal diverticulum who experienced brief episodes of complete heart block with unconsciousness after swallowing. Similar attacks could be induced by inflating a balloon in the diverticulum, and they were abolished by atropine.

The vagovagal reflex is initiated by stimulation of the afferent endings of the vagus, which are widely scattered through the carotid sinus and the gastrointestinal and respiratory tracts, including the trachea and pharynx. In some instances the pathway of the reflex is to the vagal center in the medulla, and hence over the efferent fibers; in others the impulse travels by an axon reflex directly from afferent to efferent fibers of the vagus without reaching the brain. In what might be termed the “trigemino-vagal” reflex the impulse arises in the region of the eye, upper nasopharynx, or nasal sinuses and is trans-
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mitted through the trigeminal nerve to the brain, and hence to the vagal center and efferent pathway. This reflex is responsible for the effect of ocular pressure on paroxysmal atrial tachycardia, and occasionally for cardiac arrest during eye surgery. The exact point of stimulus in our case would be uncertain, but it was probably within the trigeminal sensory distribution.

The efferent vagal fibers are scattered throughout the atria, but are not found in the mammalian ventricle. Vagal stimulation reduces the rate of impulse formation in the sinoatrial node and may cause atrial arrest; also there may be delayed conduction through the atrioventricular node resulting in heart block of various degrees, from a slightly increased P-R interval to complete heart block. In a normal myocardium vagal atrial arrest is followed by the development of an idioventricular rhythm capable of maintaining circulation, but in a myocardium depressed by an anesthetic agent, anoxia, or preexisting disease, an adequate ventricular rhythm may not be initiated.

In the present case it is difficult to trace the sequence of events immediately after the vagal stimulation. Tracing (fig. 1B) taken 20 minutes later, during the period of unconsciousness, shows atrial fibrillatory waves, and ventricular complexes generally similar to those of the control tracing, with a rate of 46 to 54. At first appearance, the tracing suggests atrial fibrillation, with a high degree of atrioventricular block, such as might occur from digitalis or vagal stimulation from carotid sinus pressure. However, with the regular ventricular rhythm and extremely slow ventricular rate (32) when the patient was first seen, it would seem more likely that there is complete block with the ventricular impulse arising near the atrioventricular node. Although it seems likely that the vagal reflex precipitated the block, the preexisting coronary disease may have been a factor in this and in the subsequent myocardial damage.

Episodes of cardiac arrest and rhythm changes during operative procedures and intubation occur with such rapidity that observations as to the mechanism of the arrhythmia are difficult to obtain. Atropine abolishes the vagal reflex and is widely used preoperatively and the relation of the use of this drug to such episodes should be carefully determined.

**Summary**

Cardiac standstill associated with surgical work about the eyes, nose, and throat occurs but is not frequent.

A case is reported in which the patient collapsed while receiving a treatment with a jet spray in the sphenoid sinus area. There developed heart block and cerebral and myocardial damage. Vagal stimulation with inhibition of the heart beat is suggested as the probable cause.

**Summario in Interlingua**

Arresto cardiaco in association con manipulationes chirurgicas in le area del oculos, del naso, e del gurgite potre ocurrer sed non es frequente.

Es reportate un caso in que le patiente collabeva durante que le area de su sinus sphenoidale esseva tractate con un jetto irrigatori. Superveniva blocco cardiac e dannno cerebrale e myocardiale. Es opinate que le causa probable esseva stimulation vagal con inhibition del pulso cardiaco.

**References**
