Introduction and case presentation:

A sixty seven old year man scheduled for left parotid tumor resection surgery. He didn't have previous surgery, didn't take any medication and didn't have any intraoperative accident in his first degree family. Also he didn’t mention any disease in medical history.

In physical examination the parotid tumor didn’t have significant effect on patient’s airways. Lungs were clear, heart sounds were normal and heart rate was 56 beat per minute.

In Para clinic: hematocrit, Na, K and FBS respectively were 49%, 137 meq/l, 3.9 meq/l and 84 mg/dl. EKG was within normal limits in all measures except heart rate which shows bradycardia but it increased by exercise.

After 8 hours fasting while he took 700 ml Dextrose/Saline solution, he lied on the operating table, and after beginning of standard monitoring (EKG, SpO2, NIBP) basal vital signs were measured as follow:

Heart rate (HR) = 64/min, Blood pressure (BP) = 138/87 mmHg, SpO2 = 96%

Anesthesia induction was done by intravenous injection of 100 microgram fentanyl, 350 milligram Na thiopental and 50 milligram atracurium after 4 minutes pre oxygenation and then tracheal intubation easily was done after 90 seconds manual ventilation.

Isoflorane 1-1.2 % in 50% mixture of O2 and N2O were used for maintenance of anesthesia. Tracheal tube secured, eyes covered and surgeons began their work. According to surgeon desire and for reducing surgical site bleeding operating table position changed to 15 degree head up.
A few minutes after surgical incision (left submandibular incision) while surgeon assistant was manipulating surgical site to localize tumor, heart rate dropped from 62 to 35 beat per minute.

Immediately 0.5 mg atropine prescribed intravenously, but heart rate dropped more and more, anesthesia ventilator turned off and controlled ventilation continued manually with more rate and tidal volume, but bradycardia worsened and finally asystole occurred.

At the moment anesthesia drugs discontinued, surgery stopped and CPR begun. After 30 minutes of CPR and prescription of 1 mg atropine, 7 mg epinephrine in 7 incremental doses and 3 doses of cardio version (200, 300 and 360 joule) normal heart beat returned. Forty five minutes later patient transferred to intensive care unit with BP= 130/80 mmHg, HR= 88 beat/min while lungs were ventilated manually with 100% oxygen.

Two days after admission in ICU he had a myocardial infarction.

Finally the patient couldn’t gain his consciousness and died 2 weeks later but in these days his family told that he had suffered from dizziness and orthostatic hypotension. In autopsy severe atherosclerosis in large vessels and coronary arteries discovered.

**Discussion:**

The patient was an old man which in appear did not suffer from any disease except a tumor in the neck but his heart rate was in the range of bradycardia definition, additionally patient went to cardiac arrest by the scene of bradycardia and he had a surgery on the neck and manipulation was in the field of an important organ: carotid sinus.

**Carotid sinus hypersensitivity:**

Carotid sinus hypersensitivity (CSH) is an exaggerated response to stimulation of carotid sinus baroreceptor.
Although baroreceptor function usually decreased with age, some people show hypersensitive carotid baroreflexes. In these patients, even mild stimulation to the neck results in marked bradycardia and hypotension.

CSH predominantly appears in older males and can occur secondary to the presence of an underlying head and neck cancer.\[^1,2^\] It is a potent contributory factor and a potentially treatable cause of unexplained falls and syncope in elderly people,\[^3,4^\] even those with no history of syncope, dizziness, or falls.\[^5^\]

In CSH, mechanical deformation of the carotid sinus (located at the carotid artery bifurcation) leads to an exaggerated response with bradycardia or vasodilatation, resulting in hypotension, syncope or cardiac arrest.\[^6^\]

CSH may be a part of a generalized autonomic disorder associated with autonomic dysregulation.\[^7^\] Data have been reported on neuronal degeneration with accumulation of hyperphosphorylated tau or alpha-synuclein in neurones in medulla, leading to impairment of central regulation of baroreflex responses and predispose elderly patients to CSH.\[^8^\]

In the cardioinhibitory type (the most common type which comprises 70-75% of cases), The predominant manifestation is a decreased heart rate, which results in sinus bradycardia, atroventricular block, or asystole due to vagal action on sinus and atroventricular nodes. This response can be abolished with atropine.\[^9^\]

CSH is observed in up to 14% of elderly nursing home patients and 30% of elderly patients with unexplained syncope and drop attacks. It is more common in males than in females and predominantly is a disease of elderly people.

In a review by Krediet et al, the scientific basis of the current diagnostic criteria for carotid sinus hypersensitivity (ie, blood pressure drop of $\geq 50$ mm Hg and/or asystole of $\geq 3$ s with carotid sinus massage) are called into question. Because the existing criteria is too sensitive to detect carotid sinus hypersensitivity, authors propose a new set of criteria (ie, blood pressure drop of $\geq 60$ mm Hg, lasting for $>6$ s and/or asystole of $\geq 6$ s with carotid sinus massage). This new set of criteria has yet to be validated by prospective studies.\[^10^\]

Permanent pacemaker implantation is generally considered an effective treatment for cardioinhibitory CSH and mixed forms of CSH.\[^11,12,13,14^\]
Surgical denervation and radiological denervation of the carotid sinus nerve were techniques used previously, but they have been largely abandoned because of high complication rates. Surgery remains an option for a patient with a neck tumor that is compressing the carotid sinus.

In this case manipulating of surgical site by the surgeon’s assistant in aim to find tumor and distinguish it from adjacent tissues made an action like carotid sinus massage so bradycardia, vasodilation and hypotension occurred (hypotension didn’t discovered in this patient because of rapidity of this events and time interval of NIBP which was adjusted for every 5 minutes). Hypotension delayed circulatory time and atropine couldn’t reach to site of action (heart) before complete asystole. Additionally hypotension reduced preload and it worsened by head up position of the patient during surgery finally manual ventilation with higher tidal volume and rate had increased intra thoracic pressure and shot last bullet to the heart and asystole happened.

**Conclusion:**

Carotid sinus hypersensitivity is an exaggerated response to stimulation of carotid sinus baroreceptor which can lead to severe bradycardia, hypotension and even asystole, It is more common in old males, and patients who suffer from head and neck tumors or inflammations. Pretreatment with vagolytic drugs and infiltration of local anesthetics is recommended but reliably can’t abolish it. A heightened awareness of this syndrome, close monitoring, and preparedness for timely diagnosis and management are essential for a successful outcome.

**References:**


Writing Committee to Revise the ACC/AHA/NASPE 2002 Guideline Update for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices

