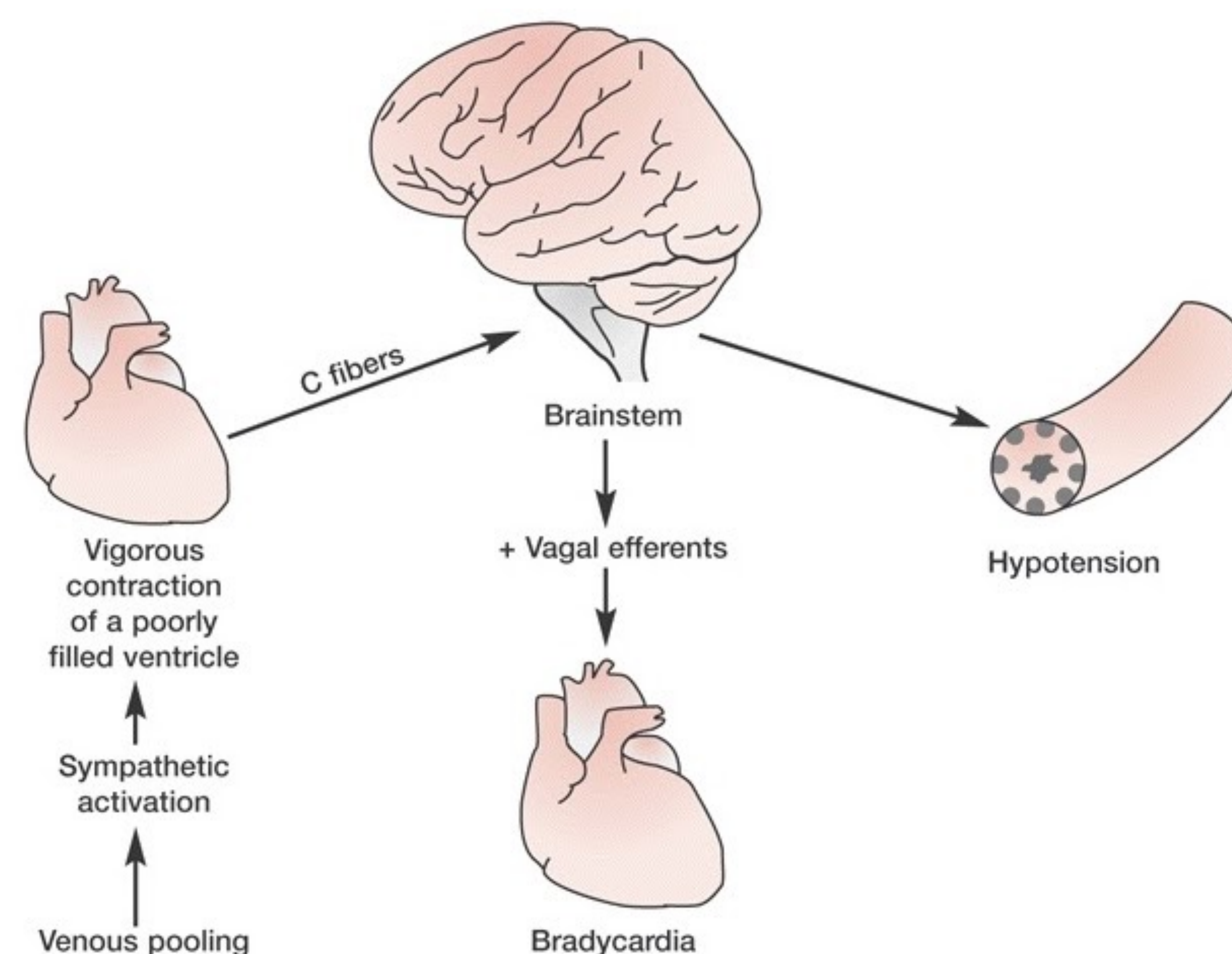


Introduction

The Bezold-Jarisch reflex is a cardioinhibitory reflex that often presents with a triad of bradycardia, hypotension, and peripheral vasodilation. The reflex originates from inhibitory mechanoreceptors located in the left ventricle. These receptors are stimulated especially during stretching and contraction of a poorly filled ventricle. Stimulation of the reflex then leads to an increase in parasympathetic activity via C-type nonmyelinated vagal afferent nerves. There is also simultaneously a decrease in sympathetic outflow, which combined with the increase in parasympathetic outflow leads to the triad seen with Bezold-Jarisch reflex.



Case Report

A 32-year-old female presented at 37w4d for a scheduled cesarean section due to newly diagnosed hypertrophic cardiomyopathy (HOCM) with severe left ventricular outflow obstruction. An interdisciplinary team meeting was had at 36w5d, in which the following steps were planned:

- Avoiding vaginal delivery in order to avoid Valsalva.
- For a cesarean delivery, spinal anesthesia was to be avoided due to concern for rapid hypotension and decrease in preload.
- An obstetric cardiologist was planned to be in the operating room for the duration of the case.
- The patient's disposition was to be the coronary intensive care unit (CCU) for closer post-operative hemodynamic management.
- Pre-operatively, the patient was preloaded 2-liters IV fluids.
- A slow-load epidural was to be used, to prevent any rapid hemodynamic changes.

During the case, while following each step of the plan, after a negative epidural test dose, the patient received 5cc of 2% lidocaine epidurally. Subsequently, while going from a sitting to supine position, the patient became profoundly bradycardic, hypotensive, vomited, then lost consciousness. As a pulse was being palpated, the patient spontaneously regained consciousness, with return to baseline vitals. However, a fetal deceleration was detected, and an emergency cesarean section needed.

In the CCU, immediate post-operative TTE showed hyperdynamic left ventricle and LVOT, consistent with patient's baseline. The patient's post-operative course was uncomplicated, and she was discharged home on POD3.

Discussion/Conclusion

In a patient with HOCM, rapid decreases in systemic tone, decreased venous return, and tachycardia are avoided. These can lead to less filling time, contraction of a poorly filled ventricle, and thus worsening LVOT. These were all important considerations to take into account leading to this parturient's delivery. Thus, a cesarean section with a slow-loaded epidural was planned. However, while lowering the patient to a supine position, a probable compression of the IVC, along with decreased preload to the left ventricle from the epidural, led to activation of the Bezold-Jarisch reflex. While the patient was bradycardic, the anesthesiology team was considering various pharmacologic agents such as glycopyrrolate and atropine, which could result in long-lasting tachycardia and worsen LVOT. However, it was important to balance that consideration with the patient's acute profound bradycardia and hypotension that needed immediate treatment. While our patient ultimately did not require any pharmacologic support and returned to her baseline vitals within minutes, this case demonstrated the complexity of trying to manage a patient in which tachycardia was being avoided, while the patient was also acutely bradycardic from the Bezold-Jarisch reflex.

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