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The Bezold-Jarisch Reflex: Two Case Reports and a Literature Review:

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ABSTRACT

The Bezold-Jarisch reflex is a cardioinhibitory reflex characterized by triad of signs (bradycardia, hypotension, and apnea) and involved in some cases of vasovagal syncope. Its physiopathology is still largely unknown.We report two cases of young patients presenting with vasovagal syncope and whose autonomic system investigation showed a profile highly suggestive of Bezold-Jarisch reflex.

1. Introduction

Vasovagal syncope (VVS) is loss of consciousness as a result of reduced blood supply to the brain, mediated through neural mechanisms rather than cardiac dysfunction. Bradycardia, vasodilation, and systemic hypotension are hallmark signs of VVS [1]. The trigger may be central (pain, stress etc.), or peripheral by a reduction in venous return to the heart, especially when the patient is standing [2]. However, one may encounter these reactions during other situations such as regional anesthesia or hypovolemia.

The Bezold-Jarisch reflex (BJR) is part of the VVS spectrum. It is a triad of signs (bradycardia, hypotension, and apnea), initially described as a response to injection of alkaloid compounds, and later found to be mediated by cardiac chemoreceptors [3].

Our understanding of the BJR is incomplete. In this paper, we will illustrate this reflex through two clinical cases and provide a review of the current literature.

2. Case Reports

Case 1

A 36-year-old Caucasian man with no medical history was admitted in the Rhythmology Department for recurrent syncopal episodes beginning the year before. He complained of palpitations, vertigo, orthostatic intolerance, and abdominal pains. Physical examination, electrocardiogram, biology, and transthoracic echocardiogram were all normal.

Assessment of autonomic function concluded with vagal hyperactivity, preserved central alpha and beta sympathetic responses, and initially conserved peripheral alpha and beta sympathetic responses then decrease of the sympathetic tone causing syncope (see Table I). This profile was highly suggestive of BJR. The patient was treated with fludrocortisone, venotonics, vitamin therapy and magnesium. Lifestyle changes, such as abundant drinks and peripheral venous contention, were advised. The patient evolved favorably.

Table I. Autonomic function assessment in patient 1

Deep breathing test	• **Vagal response: 50% (normal: 30%)**
Hand grip test	• Vagal response: 25% (normal: 10%) • Peripheral alpha sympathetic response: 15%
Hyperventilation test	• HR increased from 60 to 75 BPM • BP decreased from 122/66 to 100/57 mmHg
Mental stress	• Central alpha sympathetic response: 15% • Central beta sympathetic response: 12%
Orthostatic test	• Initial response: HR increased from 60 to 90 then maintained at 85 BPM, and BP increased from 110/60 to 125/75 mmHg • 30th minute: HR decreased to 50 then 35 BPM, and BP decreased to 68/42 mmHg, causing syncope and interruption of the test • Supine position: HR and BP returned to normal

BP, blood pressure; BPM, beats per minute; HR, heart rate

Case 2

A 19-year-old Caucasian woman with no medical history was admitted in the Rhythmology Department for a single orthostatic syncope occurring a week prior. He complained of palpitations, vertigo, orthostatic intolerance and abdominal pains. Physical examination found no abnormalities. Electrocardiogram was in favor of sinus bradycardia at 50 beats per minute. Biology, transthoracic echocardiogram, and brain scan were all normal.

Autonomic function assessment proved severe vagal hyperactivity, mild central alpha and beta sympathetic hyperactivity, and weak orthostatic peripheral alpha sympathetic response (see Table II). This profile was highly suggestive of BJR. The patient was treated with phenylephrine, venotonics, vitamin therapy, magnesium, as well as lifestyle changes, with favorable evolution.

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Table II. Autonomic function assessment in patient 2

Deep breathing test	• Vagal response: 105% (normal: 30%) • BP decreased from 100/59 to 93/58 mmHg
Hand grip test	• Vagal response: 27% (normal: 10%) • Peripheral alpha sympathetic response: 14% • BP increased from 98/51 to 112/61 mmHg
Hyperventilation test	• HR decreased from 63 to 59 BPM • BP decreased from 102/56 to 97/50 mmHg
Mental stress	• Central alpha sympathetic response: 14% • Central beta sympathetic response: 19% • HR increased from 59 to 70 BPM • BP decreased from 101/52 to 115/62 mmHg
Orthostatic test	• Vagal response: 37% (normal: 10%) • Initial response: HR increased from 62 to 71 then reached 89 BPM, and BP increased from 100/53 to 103/53 mmHg • 30th minute: HR decreased to 43 BPM, and BP decreased to 82/51 mmHg, causing dizziness and interruption of the test • Supine position: HR and BP returned to normal
BP, blood pressure; BPM, beats per minute; HR, heart rate	

3. Discussion

The BJR is a cardioinhibitory reflex characterized by a triad of responses: bradycardia, hypotension, and apnea. It was first observed following intravenous injection of Veratrum plant alkaloids in animals by von Bezold and Hirt in 1867 and Cramer in 1915. In the late 1930s, Jarisch and Richter demonstrated that the receptor area was ventricular, the afferent pathway was in the vagus nerve, and the efferent pathway involved inhibition of sympathetic outflow to peripheral vessels and increased activity in the vagus nerve to the heart [4].

Although Veratrum alkaloids are not normally present in animals, physiologic factors such as mechanical stimulation can trigger the BJR and cause VVS [5, 6]. For example, it can cause "paradoxical" bradycardia during severe hemorrhage in humans. Studies in a rabbit model demonstrated that this response is mediated by the ventricular receptors and by the ability of the BJR reflex to override the arterial baroreceptor response. During severe hemorrhage, the ventricular receptors can be excited by abnormal squeezing of the myocardium due vigorous contraction around a nearly empty chamber (see figure 1) [7].

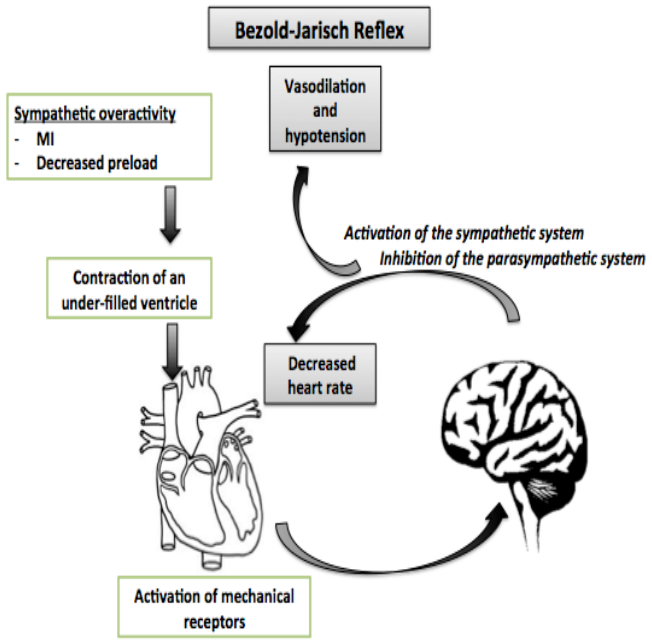

Figure 1. Bezold-Jarisch reflex mechanisms and pathways

The BJR can be activated through a large variety of mechanisms. Other classical causes include orthostasis, compression of inferior vena cava during late pregnancy and pain and anxiety during general, epidural, and spinal anesthesia [1]. The BJR has also been suspected to be the main mechanism of VVS occurring in patients with myocardial ischemia, heart failure, and severe aortic stenosis [6].

Our series is an example of orthostasis-induced BJR. When a supine individual stands up, up to a liter of blood is redistributed from the intrathoracic capacitance vessels to the veins in the lower body, therefore compensative mechanisms are needed to maintain adequate venous return to the heart and systemic arterial pressure [8]. Turning a subject into the head-up position on a tilt table induces the change in blood distribution, but without muscle contraction in the legs which assists venous return during standing. VVS is more likely to occur in subjects with a smaller blood volume [9]. If a standing subject faints, immediate collapse into the recumbent position will tend to increase venous return towards normal and thus limit the event.

4. Conclusion

The BJR is a rare phenomenon with major clinical implications. Our case series illustrates the profile of orthostasis-induced BJR. Autonomic system assessment is highly recommenced in young patients presenting with VVS and no other obvious cause.

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