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Case Report

A Case of Cardiac Arrest Caused by Vasovagal Reflex during Blepharoplasty under Local Anesthesia

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Abstract
Vasovagal reflex may cause benign self-limiting syncope triggered by factors such as pain and emotional stress. However, rarely, it may cause cardiac arrest requiring urgent resuscitation, which has heretofore not been reported during blepharoplasty. In this report, the case of a 28-year-old man who underwent blepharoplasty under local anesthesia is presented. He suddenly experienced bradycardia and subsequent cardiac arrest during resection of the orbicularis oculi. He was fearful after smelling the unpleasant odor of burned tissue and fainted. He recovered consciousness within 1 min following prompt chest compression. Based on intraoperative conditions and cardiological examinations, vasovagal reflex was considered to be the cause of the cardiac arrest. Blepharoplasty involves multiple factors that can lead to neurocardiogenic syncope, thus increasing the risk for intraoperative syncope and circulatory collapse. Hence, the procedure should be performed under close monitoring with the preparation of staff, intravenous access, drugs, and resuscitation cart to manage possible cardiac arrest.

Keywords
blepharoplasty, vasovagal reflex, cardiac arrest, local anesthesia, intraoperative monitoring

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Introduction

Minor plastic surgeries under local anesthesia are often conducted by two medical professionals (a surgeon and a scrub nurse) without monitoring the electrocardiogram (ECG) and oxygen saturation or securing intravenous access. This approach, together with patient masking and draping, poses difficulties in the identification of and response to syncope or circulatory collapse. Vasovagal reflex (VVR) is one of the intraoperative complications of plastic surgery performed under local anesthesia and is triggered by factors such as pain and emotional stress[1,2)]. VVR sometimes causes self-limiting syncope; on rare occasions, it can cause circulatory failure and cardiac arrest. Nevertheless, such a case has hitherto not been reported during blepharoplasty.

We present a case of cardiac arrest during blepharoplasty under local anesthesia. The patient recovered within 1 min following prompt chest compressions. VVR was considered to be the cause of the cardiac arrest based on intraoperative conditions and cardiological examinations, including ECG, echocardiography, and coronary computed-tomography (CT) angiography. This case highlights the need of considering and preparing for intraoperative syncope or circulatory collapse during blepharoplasty under local anesthesia. Additionally, we have reviewed the risk factors for neurocardiogenic syncope during blepharoplasty.

Case Presentation

A 28-year-old man visited our division and requested that we improve the appearance of his puffy upper eyelids and lower jaw. Based on the short margin-reflex distance 1 (MRD-1) of 2.0 mm in both eyes (**Figure 1**) and blepharoptosis-related symptoms, such as tension-type headache and shoulder stiffness, we diagnosed him with aponeurotic blepharoptosis. Three-dimensional CT of his face revealed a left-sided deviation of the maxilla and mandible, with the mentum protruding downward. We recommended

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facial contouring surgery, such as orthognathic surgery with two-jaw osteotomy. Nevertheless, he rejected our recommendation because of the long treatment course; thus, we decided on blepharoplasty with external levator aponeurotic advancement to improve the puffy and ptotic upper eyelids.

The patient had a history of uneventful surgical treatment under local anesthesia for a right finger injury. He had undergone cardiological examinations owing to arrhythmia (details unknown) but had no subjective symptoms (e.g., syncope); thus, he was untreated. Furthermore, he had no family history of cardiac or cerebrovascular diseases and was not on any medications. For patients undergoing blepharoplasty in our institution, the findings of general laboratory data, chest X-ray, and 12-lead ECG are routinely checked. The laboratory data showed 15.5 g/dL of hemoglobin, and no abnormalities in other blood count or general biochemical examination values, including electrolytes. The chest X-ray showed a normal cardiothoracic ratio of 45% and no abnormal findings. The 12-lead ECG revealed sinus arrhythmia with a heart rate of 79 bpm, complete right bundle branch block (RBBB), and left posterior fascicular block (LPFB) with a right-axis deviation of 130° (**Figure 2**). ST segment elevations in anterior precordial leads indicative of Brugada syndrome were not observed.

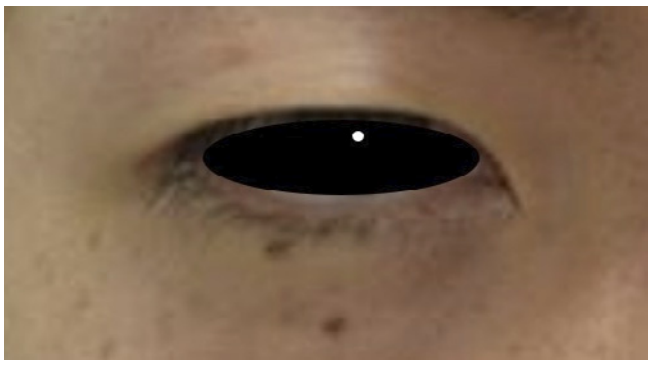


Figure 1. Preoperative appearance of the area around the patient's right eye
The entire areas of the palpebral fissures are blacked out, and the white points indicate the center of the pupillary light reflex. Preoperative MRD-1 is 2.0 mm.

An 18 mg lidocaine tape was placed on each upper eyelid for 2 h preoperatively. Upon entering the operating room, the usual monitors (i.e., ECG monitor, pulse oximeter, and noninvasive blood pressure monitor checked every 5 min) were set up. His initial vital signs included blood pressure of 119/65 mmHg, heart rate of 81 bpm, and oxygen saturation (room air) of 95%. An intravenous line was secured on his left forearm, and drip infusion of cefazoline (1 g) was started. Transverse spindle-shaped marks with a maximum width of 1 mm were drawn at a distance of 6.5 mm from the middle of the upper eyelid edge as guides for musculocutaneous resection. The patient was draped to expose the eyebrows, eyes, nose, and mouth, and 4 mL of local anesthesia comprising equal amounts of 0.5% lidocaine and 0.75% ropivacaine was injected into each upper eyelid. His heart rate rose to approximately 100 bpm and his blood pressure increased by approximately 5 mmHg during anesthesia injection, but he did not feel any uneasiness or pain.

The surgical procedure was started with a right upper eyelid incision 7 min after injection of local anesthesia. When musculocutaneous resection was completed, his vital signs were stable and he had no complaint of pain or discomfort. Nevertheless, when we proceeded to perform additional orbicularis oculi resection, he lost consciousness with no

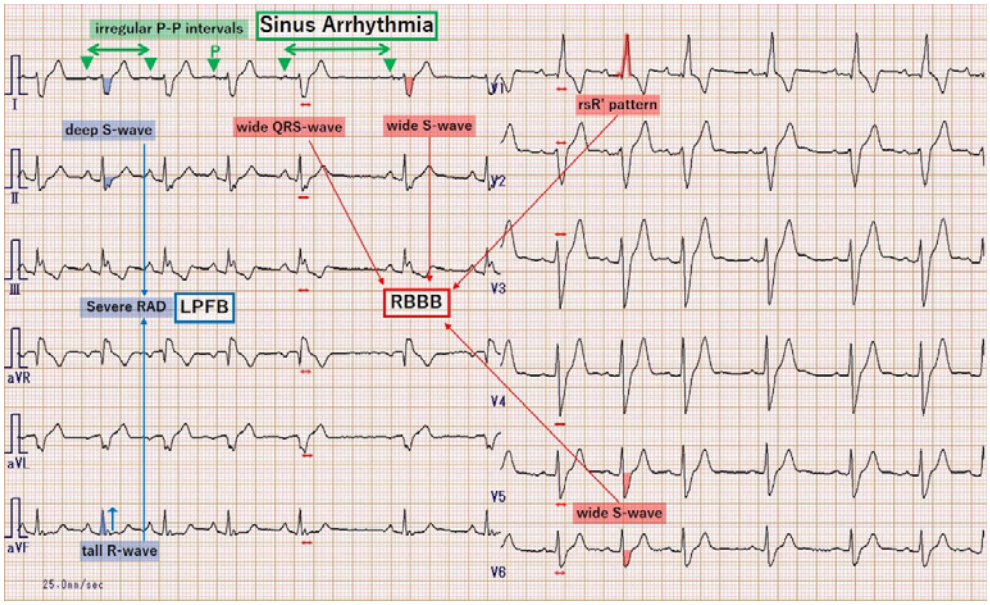


Figure 2. Preoperative 12-lead electrocardiogram
Sinus arrhythmia (related findings are annotated in green), complete RBBB (red), and LPFB (blue) are shown.
RBBB, right bundle branch block; LPFB, left posterior fascicular block

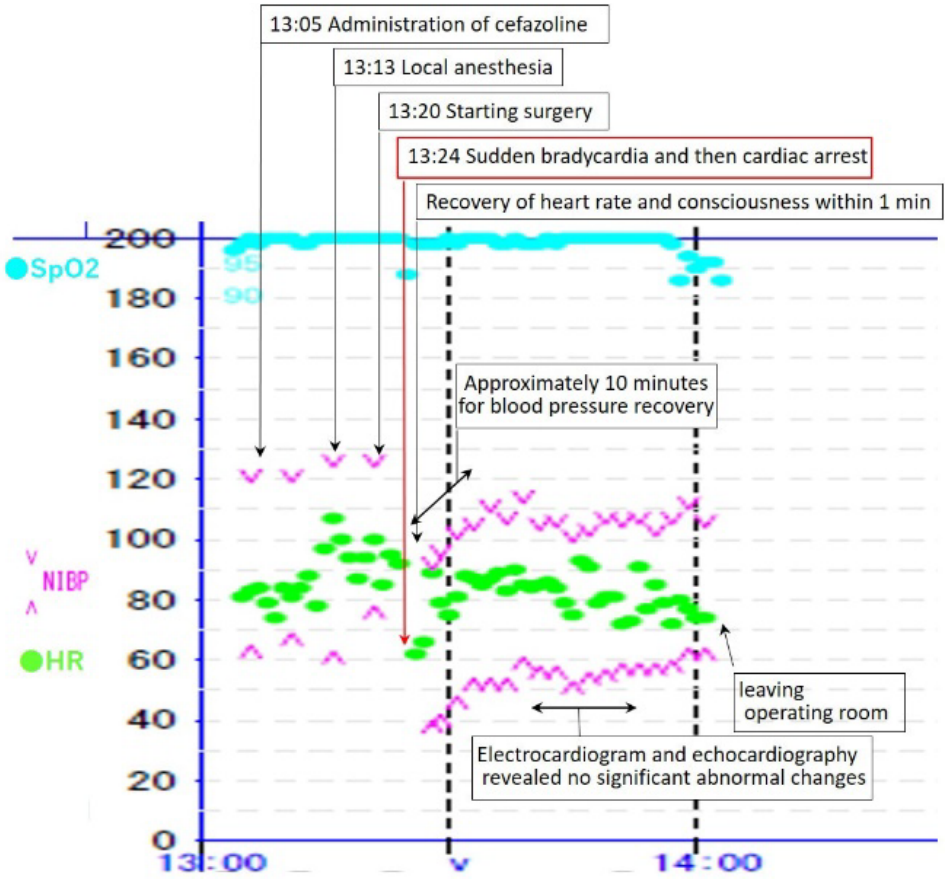
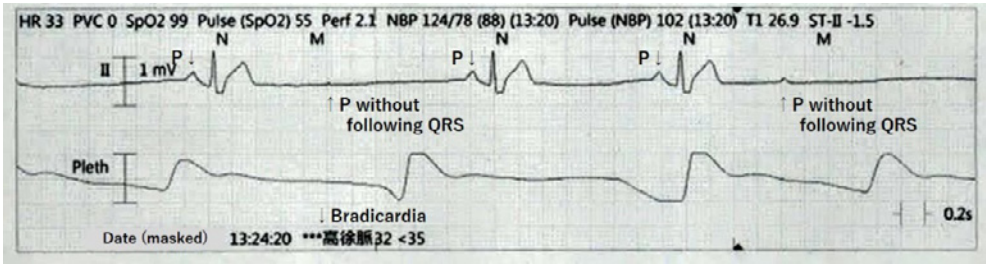
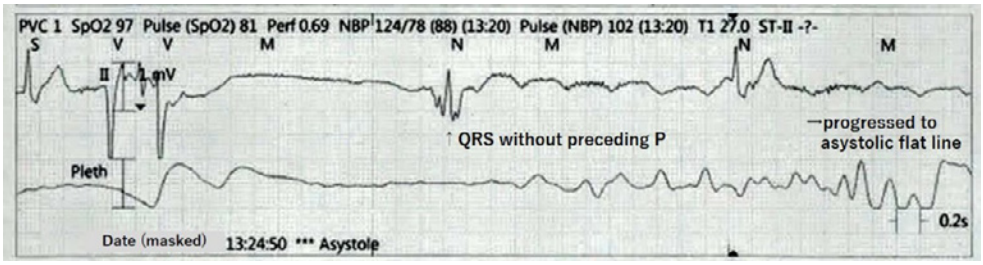


Figure 3. Anesthesia chart of the first surgery.



(a)



(b)

Figure 4. Monitor electrocardiogram just before the asystolic cardiac arrest
(a) Severe sinus bradycardia and atrioventricular block
(b) Sinus pause and QRS-wave without preceding P-wave
When there are no effective pulse waves in the pulse oximeter, it means peripheral circulatory failure. This electrocardiogram progressed to an asystolic flat line.

physical discomfort. Furthermore, he experienced bradycardia (heart rate < 30 bpm), followed by asystolic cardiac arrest within 1 min (**Figure 3 and 4**). We immediately called for assistance and began cardiopulmonary resuscitation. Si-

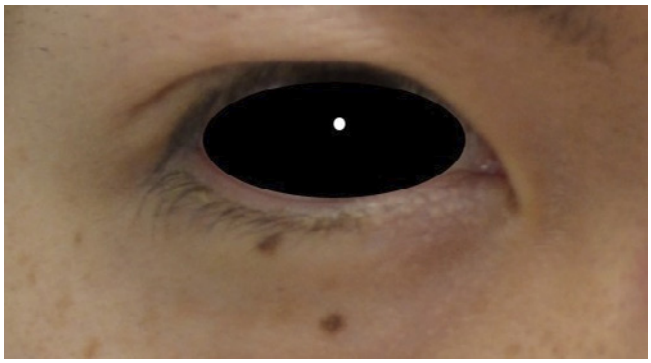


Figure 5. Postoperative appearance of the area around the patient's right eye
Image at seven postoperative months indicates increased MRD-1 of 8.0 mm.

nus rhythm was restored within 1 min following chest compressions, and he soon regained clear consciousness. We prepared an intravenous infusion of atropine but did not administer it because of the patient's prompt recovery. Although his blood pressure recovered in approximately 10 min and his vital signs were stable afterward, we decided to discontinue the procedure because we could not definitively identify the cause of the cardiac arrest (**Figure 3**). We sutured the right upper eyelid incision and terminated the surgical procedure without further events. Echocardiography in the operating room after recovery showed no abnormal cardiac activity, and 12-lead ECG revealed no new changes compared with the preoperative findings of sinus arrhythmia, RBBB, and LPFB. After his recovery, the patient stated that he had felt strong anxiety and fear because of the proximity of the surgical manipulation to his eyes and because he smelled something burning. He also expressed that he had felt uncomfortable for a moment before fainting. Based on the situation before the cardiac arrest and the quick recovery, we considered severe VVR, rather than local anesthetic toxicity or a newly developed cardiovascular event, to be the cause of the cardiac arrest. The patient was hospitalized for follow-up and discharged the following day without incident. The stitches on the right upper eyelid were removed on the seventh postoperative day, and the wound was clear.

Additional cardiological examinations were later performed. Coronary CT angiography revealed no significant coronary stenosis, and echocardiography showed a normal ejection fraction of 61% without valvular disease or pulmonary hypertension. These results led us to consider severe VVR as the cause of the cardiac arrest.

Two months later, we completed the suspended blepharoplasty under general anesthesia without incident, and his MRD-1 and blepharoptosis-related symptoms improved (**Figure 5**). Seven months later, mandibular contouring surgery was performed under general anesthesia without incident.

Discussion

VVR can be triggered by various afferent pathways and causes parasympathetic activation of the efferent cardiac vagus nerve and inhibition of the sympathetic medullospinal pathway. These changes result in bradycardia and vasodilation, which lead to hypotension[1,2]. The afferent pathways are mainly classified into two types: central type, which involves the direct stimulation of the cortico-hypothalamic center by strong pain or emotional stress, and peripheral type, which involves the stimulation of the mechanoreceptors in the left ventricular wall or aortic baroreceptors to afferent vagus nerves by reduced venous return[1]. VVR sometimes causes self-limiting syncope because of transient brain hypoxia; in rare cases, it can cause severe bradycardia, hypotension, and cardiac arrest. Cardiac arrest has been reported with epidural anesthesia or spinal anesthesia, especially in obstetric cases before and after administering local anesthetics[3-6]. Cardiac arrest has also been reported following the administration of brachial plexus block or peripheral intravenous access[7,8]. Common risk factors for onset and exacerbation of VVR include young age, history of syncope, and strong anxiety or fear during procedures. Other reported risk factors include sympathetic blockade and reduced venous return due to compression of the inferior vena cava or excessive hemorrhaging in pregnant women undergoing a cesarean section.

Although there are no previous clinical reports on cardiac arrest during blepharoplasty, three features of this surgery serve as risk factors for neurocardiogenic syncope, a type of which is VVR. First, surgical maneuvers performed near the eyes elicit strong feelings of anxiety and fear in patients. Second, olfactory irritation affects autonomic nerve activities, and patients undergoing blepharoplasty smell the unpleasant odor of tissue being burned using the electrical scalpel. Olfactory irritation exerts emotional effects and directly modulates the sympathetic and parasympathetic activities via adrenergic and muscarinic receptors on the olfactory epithelium[9]. Unpleasant odor is known to cause parasympathetic activation[10] and can, therefore, be considered to be a risk factor for VVR. Based on the intraoperative conditions and the patient's words, we consider the above two factors to be the main causes of VVR in this patient. By contrast, pain and reduced venous return are unlikely triggers of VVR, because the patient had no complaints of pain, got administered intraoperative fluid infusion without preoperative water restriction, and was consistently in a supine position.

Third, the oculocardiac reflex (OCR) can be considered a risk factor. OCR has the same efferent pathway as VVR, but its afferent pathway is the trigeminal nerve, which is stimulated by pressure on the eyeballs or traction of extraocular muscles, ocular fat pad, and orbicularis oculi[11,12]. Unlike VVR, OCR occurs more easily under general anesthesia than under local anesthesia[11]. Studies have shown that OCR is the cause of bradycardia during blepharoplasty under local anesthesia[12] and cardiac arrest during surgery for orbital floor fracture[13]. It has been reported that patients have different sensitivities to OCR[14]. Sensitive patients experience bradycardia and hypotension, which are repeatedly triggered by even gentle eyelid traction, and require atropine to pre-

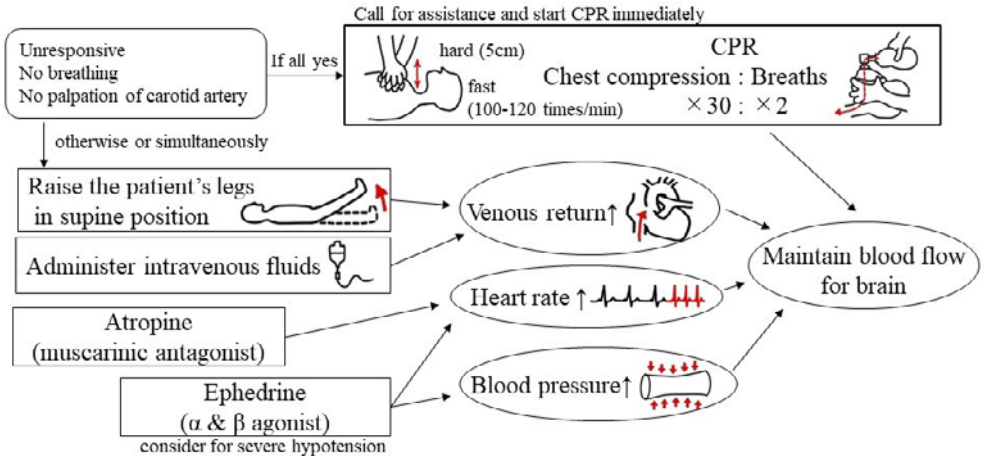


Figure 6. Recommended treatments for severe vasovagal reflex
The most important point is maintaining blood flow for the brain. In case of cardiopulmonary arrest, immediately start cardiopulmonary resuscitation (CPR) and consider other treatments without interrupting CPR.

vent recurrent reflexes[11]. In our patient, we could not eliminate slight pressure on the eyeball or direct stimulation of the orbicularis oculi during resection. Nevertheless, the patient is not considered hypersensitive to OCR because he later uneventfully underwent two surgeries under general anesthesia; therefore, we do not regard OCR to be the cause of the cardiac arrest.

Preoperative ECG abnormalities in this case may be related to the exacerbation of VVR to cardiac arrest. The patient had abnormalities in the sinus node and intraventricular conduction. Because this condition is not likely involved in initiating the afferent pathway of VVR and because most patients with cardiac arrest do not exhibit preprocedural ECG abnormalities, preoperative ECG can be considered to play only a small role in the prediction of VVR onset. However, sinus arrhythmia and intraventricular conduction block in this case might have exacerbated the inhibition of the sinus node and intraventricular conduction caused by VVR, and the risk for circulatory collapse increased once VVR occurred. This finding shows that preoperative ECG evaluation is useful for the risk prediction of VVR exacerbation despite its limited role in the risk prediction of VVR onset. Preoperative evaluation of 12-lead ECG is necessary for high-risk patients with a history of VVR or syncope and is recommended for all patients undergoing blepharoplasty.

Appropriate treatment of VVR includes maintenance of venous return by raising the legs and administration of intravenous fluids, atropine, or ephedrine. Severe hypotension is sometimes resistant to atropine and requires ephedrine[4,8]. We should have considered administering ephedrine because it took 10 min for the blood pressure of the patient to revert to normal. In case of cardiopulmonary arrest, calling for assistance and cardiopulmonary resuscitation must be begun immediately (**Figure 6**)[15]. Prophylactic strategies include addressing patient anxiety, fear, and pain with adequate communication, appropriate anesthesia, and sedation (in some cases). General anesthesia is a viable option to prevent VVR[5], and the patient underwent two surgeries under general anesthesia uneventfully.

In the absence of close patient monitoring, intraoperative circulatory collapse due to VVR can be overlooked, leading to irreversible organ injuries, as reported in cases of prolonged cardiac arrest requiring 1-15 min of cardiopulmonary resuscitation[3,6,7]. Even for minor surgical procedures, such as blepharoplasty under local anesthesia, appropriate treatment options for severe VVR should be made available.

Conclusion

During blepharoplasty under local anesthesia, we encountered a case of cardiac arrest caused by VVR. A patient with no history of syncope can experience cardiac arrest in the background of asymptomatic ECG abnormalities. Blepharoplasty should be viewed as a procedure associated with a high risk for syncope and should be performed under close monitoring with adequate preparation for the possibility of cardiac arrest.

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Consent to Participate: The authors have not obtained consent to participate because a case report does not require it.

Consent for Publication: The authors have not obtained written consent because the person is not identifiable.

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