Ocular pressure waveform reflects ventricular bigeminy and aortic insufficiency

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Ocular pulse amplitude (OPA) is defined as the difference between maximum and minimum intraocular pressure (IOP) during a cardiac cycle. Average values of OPA range from 1 to 4 mmHg. The purpose of this investigation is to determine the source of an irregular IOP waveform with elevated OPA in a 48-year-old male. Ocular pressure waveforms had an unusual shape consistent with early ventricular contraction. With a normal IOP, OPA was 9 mmHg, which is extraordinarily high. The subject was examined by a cardiologist and was determined to be in ventricular bigeminy. In addition, he had bounding carotid pulses and echocardiogram confirmed aortic insufficiency. After replacement of the aortic valve, the bigeminy resolved and the ocular pulse waveform became regular in appearance with an OPA of 1.6–2.0 mmHg. The ocular pressure waveform is a direct reflection of hemodynamics. Evaluating this waveform may provide an additional opportunity for screening subjects for cardiovascular anomalies and arrhythmias.

Key words: Aortic insufficiency, arrhythmia, ocular pulse amplitude, ventricular bigeminy

Intraocular pressure (IOP) is pulsatile in nature due to the filling of the intraocular blood vessels in systole. Ocular pulse amplitude (OPA) is defined as the difference between maximum IOP (systolic) and the minimum IOP (diastolic). Average values of OPA in healthy subjects range from 1 to 4 mmHg.[1] The PASCAL dynamic contour tonometer (DCT) (Ziemer, Port, Switzerland) is capable of measuring IOP continuously and plotting the data as a waveform. Normal appearance of the waveform includes regular cycles corresponding to the heartbeat [Fig. 1]. The purpose of the current investigation was to determine the source of an irregular waveform of extreme values.

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Case Report

Ocular pressure waveforms from the right eye of a 49-year-old white male subject were digitally recorded during training to use the PASCAL DCT. Custom software was provided by the manufacturer for continuous recording. It was noted that the waveform had an unusual shape consistent with an early ventricular contraction every other beat [Fig. 2a]. It was also noted that the OPA was > 9 mmHg. Blood pressure (BP) was 138/64 and heart rate was 64 beats/min. A digital recording of the patent's OPA during Valsalva was then performed [Fig. 2b]. It was noted that the OPA became significantly lower during Valsalva, and the waveform became more regular in appearance.

The subject then presented for a thorough examination by a cardiologist. He was known to have congenital absence of the pericardium, which was diagnosed 12 years prior when he was evaluated for nonexertional chest pain.[2] Past medical history was significant for repair of a right inguinal hernia at age 27. He took omeprazole 20 mg p.o. daily for gastroesophageal reflux disease. On the review of systems, he reported generalized fatigue for 6 months, paroxysmal nocturnal dyspnea and orthopnea for 2 months, and a sensation of a bounding pulse in the neck for several weeks. He denied chest pain or lower extremity edema. Family history was significant for acute ascending aortic dissection in his younger brother at age 43.

Physical examination revealed an early descending diastolic murmur. The lungs were clear to auscultation. Holter monitor testing for 24 h revealed a baseline rhythm of normal sinus with first degree atrioventricular block and intermittent episodes of sinus tachycardia, sinus bradycardia and sinus arrhythmia, including isolated premature atrial complexes, premature ventricular complexes and ventricular bigeminy [Fig. 3a]. Echocardiography estimated left ventricular (LV) ejection fraction to be 50–55%. There was normal right ventricular systolic function and at least moderate aortic regurgitation [Fig. 4a]. Mild dilatation of the aortic root was noted on the echocardiogram. Cardiac magnetic resonance imaging demonstrated severe aortic insufficiency and a dilated left ventricle with a calculated LV ejection fraction of 42%. The ascending aorta was dilated with a maximum dimension of 4.3 cm at the level of the sinuses and 4.1 cm in the mid ascending aorta. Absence of the pericardium and posterior rotation of the heart were again noted.

Preoperative diagnoses included congenital absence of the pericardium, annulo-aortic ectasia with aortic root aneurysm and symptomatic, severe aortic insufficiency with LV dysfunction. He underwent surgery to replace the ascending aorta and the aortic valve with a pericardial tissue graft. A Bentall procedure was performed with a composite graft composed of a 32 mm Valsalva graft and a 29 mm Carpentier-Edwards PERIMOUNT Magna valve. Re-implantation of the coronary artery ostium was also performed.

The subject's postoperative course was uneventful. His symptoms of dyspnea and orthopnea resolved. Postoperative electrocardiogram readings revealed a progressive decrease in the frequency of arrhythmias, with the patient predominantly in normal sinus rhythm [Fig. 3b]. OPA was measured at 1 month

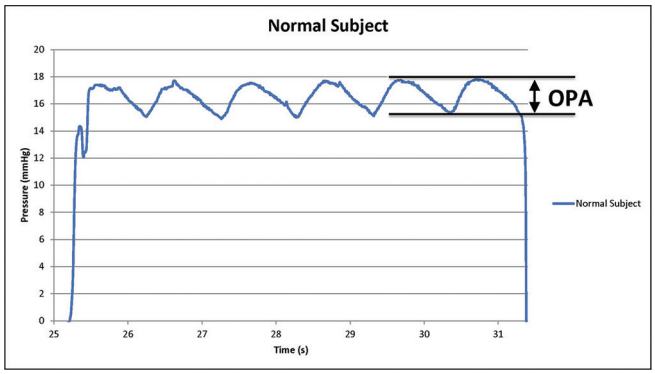

Figure 1: Ocular pulse amplitude waveform tracing in a normal, healthy subject

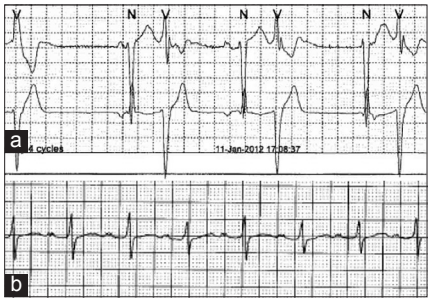

Figure 3: (a) The upper panel shows a section of the preoperative Holter monitor showing bigeminy. (b) The lower panel is an electrocardiographic recording after replacement of the ascending aorta and aortic valve showing normal sinus rhythm

Table 1: Mean OPA, IOP, and BP values of test subject before and after aortic root and valve replacement

	Mean OPA right eye (mmHg)	Mean IOP right eye (mmHg)	BP (mmHg)
Preoperative	5.8	12.9	138/64
1-month postoperative	2.0	13.3	110/64
6 months postoperative	4.0	12.3	122/68

OPA: Ocular pulse amplitude, IOP: Intraocular pressure, BP: Blood pressure

and 6 months postoperatively [Table 1]. The average values for OPA were 2.0 mmHg at 1 month and 3.5 mmHg at 6 months. The mean IOP at the 1 and 6 month postoperative visits was 13.3 mmHg and 12.2 mmHg, respectively. The waveform revealed occasional ectopic beats at both postoperative visits; however, the frequency of these and the amount of time in bigeminy was drastically reduced [Fig. 2c and d]. Postoperative two-dimensional echocardiography demonstrated resolution of the aortic regurgitation [Fig. 4b].

Discussion

The pulsatile nature of IOP was first described as early as 1967.[3] Later, Perkins reported the dependence of OPA on both ocular blood flow and ocular rigidity.[4] More recently, a model was presented demonstrating the interdependence of the static aqueous circulation and the pulsatile ocular blood flow, resulting in the ocular pulse waveform.[5] The influence

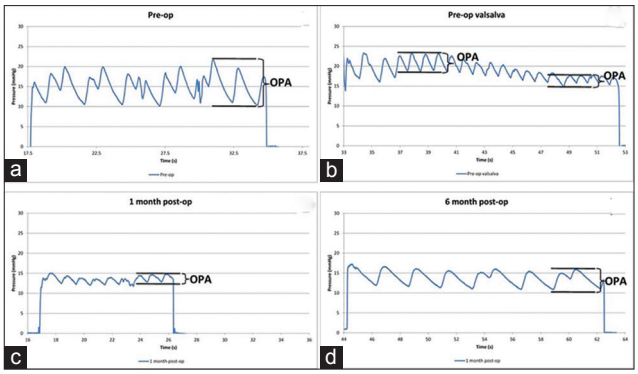

Figure 2: Ocular pulse amplitude (OPA) waveform tracings of a 49 year old test subject. (a) The upper left panel shows an incidental finding of an unusual shape consistent with an early ventricular contraction. Note that the max OPA is also significantly elevated at >9 mmHg. (b) The upper right panel shows an OPA waveform tracing of the same subject during Valsalva, demonstrating a reduction in the OPA. (c) The lower left panel shows an OPA tracing 1 month postoperatively demonstrating an occasional ectopic beat and reduced OPA amplitude in the normal range. (d) The lower right panel shows an OPA waveform tracing at 6 months postoperatively

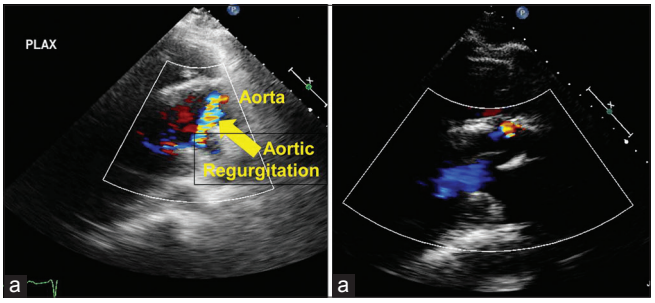

Figure 4: (a) The left panel shows the preoperative two-dimensional (2D) echocardiography demonstrating aortic regurgitation. (b) The right panel shows the postoperative 2D echocardiography demonstrating resolution of aortic regurgitation

of systemic pulse pressure (dependent upon both systolic and diastolic BP) on OPA has been shown both by a decrease in OPA in response to nifedipine in a series of patients[6] and after surgical correction of aortic regurgitation in a single patient,[7] similar to the current case. Reduction of systemic pulse pressure consistently led to a reduction of OPA in both of these reports. However, both trials relied on Goldmann applanation tonometry to estimate OPA. Neither had electronically recorded waveforms as were produced in the current case.

Analysis of the recorded waveforms in the current case demonstrates that the ocular pulse waveform is a direct reflection of real-time hemodynamics. This is clearly evident as reduction of cardiac output in response to the Valsalva maneuver results in immediate lowering of OPA as the preload on the heart is reduced, and the regurgitation is subsequently ameliorated. In patients without regurgitation, the OPA is not affected by a Valsalva maneuver.[8] Of notable clinical interest, the OPA waveform is sufficiently sensitive to demonstrate the hemodynamic consequence of arrhythmias such as ventricular bigeminy. Previous investigators have reported the tachycardia found incidentally on OPA performed at routine follow-up for glaucoma evaluations.[9] In addition, OPA has been shown to be

diminished unilaterally in patients with carotid artery stenosis.[10]

The advantage of the ocular pulse waveform is that it can be recorded from the cornea using a tonometer probe much like those already in widespread use by ophthalmologists and optometrists. The testing is noninvasive, takes minutes to complete, and could easily be incorporated into routine eye examinations. This could become an important tool in the detection of arrhythmias and hemodynamic aberrations in asymptomatic patients, leading to earlier diagnosis and treatment.

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