

Left atrial appendage (LAA) flow profile of its different waves and its correlation with direct left atrial pressure measurement: Can LAA flow profile be a surrogate to estimate left atrial pressure

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ABSTRACT

Background: Left Atrial Appendage (LAA) is one of the most contractile structure of the heart. Elevated Left atrial pressure (LAP) can change the flow profile in and out of LAA. There is little data on the effect of LAP on LAA flow velocities for patients in sinus rhythm, and it's not properly known that by evaluation of LAA flow spectra and its velocities, the LAP can be predicted. We tried to find the relationship between LAA flow velocities and LAP, with the premise that LAA flow velocities can be used as a surrogate for measuring LAP, by obtaining a regression equation in this prospective observational study.

Methods: In forty patients with normal systolic and diastolic heart function undergoing elective off pump coronary artery bypass (OPCAB) under general anaesthesia, TEE based LAA flow velocities were measured and simultaneous direct measurements of LAP was done by the surgeon. We also studied the relation between the ratio of early mitral inflow velocity (E) and mitral lateral annular early diastolic velocity (E'), that is, (E/E') in all patients.

Results: We found significant correlation between E/E' and LAP ($r = 0.424$, $p = 0.024$) however there was no significant correlation between LAA flow velocities and LAP.

Conclusion: LAA flow profile can not be used under anaesthesia to evaluate LAP however E/E' shows a strong correlation with directly measured LAP.

Keywords: E/E', LAA flow velocities, LAP

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INTRODUCTION

LAA is a highly contractile part of the left atrium and likely plays an important role in cardiac hemodynamics. Transesophageal echocardiography (TEE) is currently the modality of choice for evaluation of LAA. It allows complete delineation of LAA anatomy, contraction and

quantitative assessment of LAA function by pulse wave Doppler (PWD) interrogation of LAA flow.^[1-3]

Several studies in patients with atrial fibrillation has shown that elevated LAP affects LAA flow velocities, and LAA

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function assessment provides information about risk of clot formation, embolic events and success of cardioversion.^[2-5] However there is little data on the effect of LAP on LAA flow velocities for patients in sinus rhythm. Therefore, it is also not properly known that by evaluation of LAA flow spectra and its velocities, the LAP can be predicted.

We studied the relationship between LAA flow velocities and simultaneous direct measurement of LAP in patients with IHD undergoing OPCABG with no arrhythmias, normal systolic and diastolic functions, and no regional wall motion abnormalities with the premise that LAA flow velocities can be used as a surrogate for measuring LAP by obtaining a regression equation.

Although E/E' is a frequently used and relatively load-independent method for estimation of LAP, it may not be feasible always due to technical considerations like improper alignment of the Doppler signal; it is unreliable in certain clinical conditions like significant mitral regurgitation, severe mitral annular calcification, and presence of left bundle branch block.^[6] In this study we also measured E/E' and examined its relation with simultaneous, direct measurement of LAP.

MATERIALS AND METHODS

50 patients who underwent OPCABG procedure were included in this prospective observational study. The exclusion criteria were hypertension, diabetes, diastolic or systolic dysfunction because of any reason, arrhythmias, and valvular dysfunction. All of them were having triple vessel IHD. The prior TTE was within normal limits. The study received clearance from institutional ethics committee (2017-250-IP-101, dated 06/03/2018).

In the operation room, the standard lines and monitoring along with minimally invasive CO monitoring was used (FloTrac transducer with Edwards Lifesciences HemoSphere Advanced Monitoring Platform, Irvine, USA). All patients were induced with etomidate 0.2–0.3 mcg/kg, fentanyl 5–10 mcg/kg, and midazolam 0.05–0.1 mg/kg. The maintenance of anesthesia was done with propofol infusion of 50–150 mcg/kg/min, isoflurane of 0.6–1.2%, incremental doses of fentanyl. and it was BIS-guided management. Endotracheal intubation was facilitated after muscle relaxation with pancuronium 0.1–0.2 mg/kg. TEE probe X7-2t/Adult (Philips iE33, Bothell WA, USA) was introduced and a quick and complete examination of the heart was done according to the Indian perioperative TEE guidelines.^[7]

The patient was positioned supine and neutral, and before bilateral IMAs were harvested, the measurements were done. The LAA view was made by TEE, mid-esophageal (ME) two-chamber view (80–100 degrees) with slight lateral tilt and clockwise rotation, and aligned in such a way that Doppler cursor was parallel to the flow from LAA into LA and reverse flow as shown by colour flow Doppler (CFD). The PWD was used and the sample volume was placed inside LAA between one-third and two-thirds length of the LAA from its mouth. The Nyquist limit was 60 cm per second. Three simultaneous readings of maximum forward flow, reverse flow and duration of both the spectral velocities were taken and averaged [Figures 1 and 2]. At the same time, the surgeon used a saline-filled pressure measuring line, which was attached to a transducer to measure the LA chamber pressure directly by a 26 gauge needle inserted into the superior aspect of left atrium between SVC and ascending aorta. The zeroing of the transducer was meticulously done at the mid-axillary line as reference point for the LA measurement. Both the measurements were done simultaneously. The E/E', a non-invasive estimate of LA pressure measurement was done as per the ASE/SCA TEE guidelines.^[8] Mitral inflow early (E) and atrial (A) wave velocities with spectral doppler and mitral annular early diastolic velocity (E') on the lateral mitral annulus with tissue doppler were measured in ME four-chamber view as per ASE/SCA guidelines.

DATA ANALYSIS AND RESULTS

The data was collected and analyzed using SPSS Statistics Version 26. Continuous variables, which were normally distributed, are presented as mean \pm standard deviation. Categorical variables have been presented as percentages. Skewness of data was assessed using tests of normality. Pearson correlation coefficient was calculated using bivariate analysis. Scatter plots were also done for significant correlations.

Out of the 50 patients, we had complete data of 41 patients of which 37 were male and 4 female. Mean age of the study group was 59.2 ± 9.4 yrs., mean height 164.2 ± 6.7 cm, mean weight 67.4 ± 10.4 kg, and mean

Table 1: Demographic data and hemodynamic data

Variable (n=41)	Mean \pm SD
Age (yrs)	59.2 \pm 9.4
Height (cm)	164.2 \pm 6.7
Weight (kg)	67.4 \pm 10.4
BSA (m ²)	1.7 \pm 0.1
HR (beats/min)	76.3 \pm 10.8
SBP (mmHg)	129.2 \pm 20.8
DBP (mmHg)	70.6 \pm 13.3
MAP (mmHg)	88.5 \pm 15.2

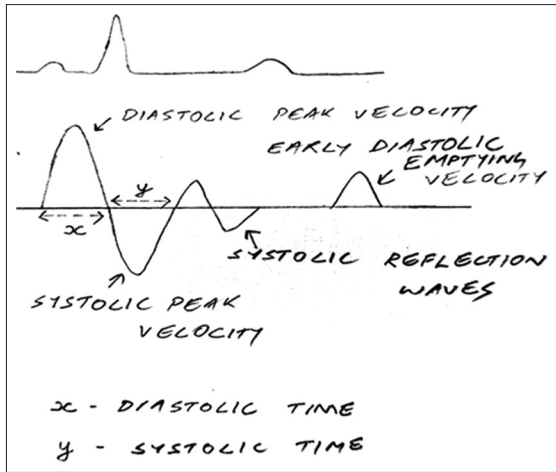


Figure 1: Schematic diagram of normal LAA flow profile

BSA 1.7 ± 0.1 m². At the time of Doppler evaluation, the mean HR was 76.3 ± 10.8 beats/min, mean SBP 129.2 ± 20.8 mmHg, mean DBP 70.6 ± 13.3 mmHg, and mean MAP 88.5 ± 15.2 mmHg. Demographic data and hemodynamic data at the time of Doppler evaluation are shown in Table 1.

The mean LAA peak diastolic velocity was 47.03 ± 15.05 cm/s and mean LAA systolic peak velocity was 40.7 ± 16.6 cm/s. The mean LAA diastolic time was 124.5 ± 25.7 msec, systolic time 207.4 ± 58.5 msec, ratio of diastolic peak velocity to systolic peak velocity 1.27 ± 0.52 , and ratio of diastolic time to systolic time was 0.63 ± 0.26 . The mean direct measurement of LAP was 12.7 ± 5.2 mmHg and mean E/E' lateral was 9.01 ± 3 . E' on the medial mitral annulus was not measured as the medial mitral annular movement was very less. The various Doppler parameters measured and simultaneous direct measurement of LAP are shown in Table 2.

We tried to correlate LAA flow characteristics—LAA peak diastolic velocity, LAA peak systolic velocity, LAA diastolic time, LAA systolic time, ratio of diastolic to systolic velocity and ratio of diastolic to systolic time with simultaneous directly measured LAP—and generate an equation by which we can estimate LAP non-invasively from LAA flow characteristics. However, there was no correlation found. We also measured E/E' lateral and found significant correlation between E/E' lateral and simultaneous direct measurement of LAP (Pearson correlation coefficient 0.424 , $P = 0.024$) [Figure 3]. Correlations of LAP with the various Doppler parameters are shown in Table 3. Scatter plots showing the relation between LAP and LAA flow characteristics—LAA diastolic peak velocity, LAA systolic peak velocity, LAA diastolic time and LAA systolic time—where we found no significant correlation are shown in [Figure 4].

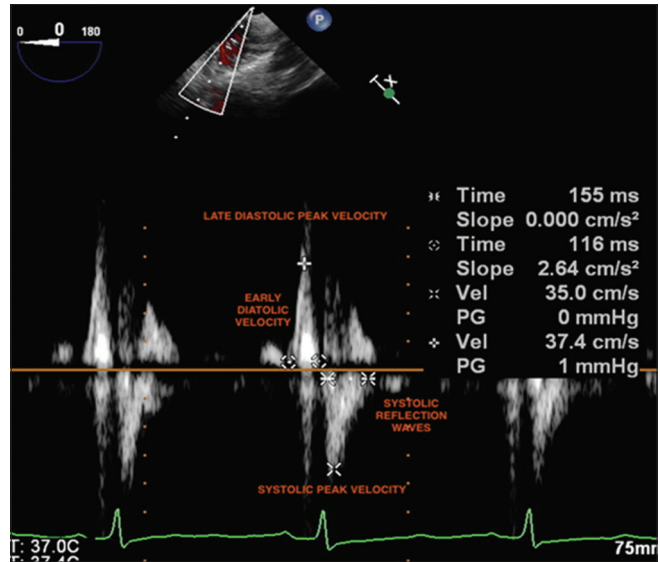


Figure 2: Real-time LAA flow profile by TEE in operation room

Table 2: LAA flow characteristics - velocities and duration, E/E' lateral and simultaneous direct LAP

Variable (n=41)	Mean±SD
LAA diastolic peak velocity (cm/s)	47.03±15.05
LAA diastolic time (msec)	124.5±25.7
LAA systolic peak velocity (cm/s)	40.7±16.6
LAA systolic time (msec)	207.4±58.5
LAA diastolic peak velocity/LAA systolic peak velocity (ratio)	1.27±0.52
LAA diastolic time/LAA systolic time (ratio)	0.63±0.26
LAP (direct measurement) (mmHg)	12.7±5.2
E/E' lateral (ratio)	9.01±3

Table 3: Correlation of LAP with the various Doppler parameters

	Correlation coefficient	P
LAA diastolic peak V (cm/s)	-0.211	0.185
LAA diastolic time (msec)	-0.127	0.436
LAA systolic peak V (cm/s)	-0.046	0.775
LAA systolic time (msec)	0.136	0.398
LAA diastolic peak V/LAA systolic peak V (ratio)	-0.181	0.257
LAA diastolic time/LAA systolic time (ratio)	-0.228	0.152
E/E' lateral (ratio)	0.424	0.024

DISCUSSION

LAA, a remnant of the embryonic left atrium, has a complex anatomical structure that is distinct from the rest of the left atrium and plays a more important functional role than thought previously. It acts as a reservoir during left ventricular systole, a conduit for blood transiting from the pulmonary veins to the left ventricle during early diastole, an active contractile chamber that augments left ventricular filling in late diastole, and a suction source that refills itself in early systole.^[9]

Although the blind cul-de-sac and multilobed anatomical structure can predispose to thrombus formation, it is

usually prevented by vigorous blood flow in the appendage cavity.^[1] However, it is the most common source of cardioembolic stroke in atrial fibrillation (AF) and is often described as the “most lethal human appendage.”^[10,11]

TEE with 2D assessment and, in particular, with 3D reconstruction is one of the most accurate non-invasive imaging modalities to define LAA anatomy. LAA, a

posterolateral structure, is visualized with a very good resolution on TEE. LAA is maximally visualized in ME two-chamber view (80–100 degrees) and ME aortic valve short axis view (30–60 degrees).^[8] We used ME two-chamber view with slight lateral tilt and slight clockwise rotation so that the Doppler cursor was parallel to the flow from LAA to LA and reverse flow. Assessment of LAA function is done by PWD interrogation of LAA flow. Normal LAA flow pattern includes early diastolic emptying, late diastolic emptying or LAA contraction (diastolic peak velocity), LAA filling (systolic peak velocity) and systolic reflection waves [Figures 1 and 2].

Accurate measurement of LA pressure still remains a big challenge in cardiac ailments. Various surrogates like echo assessments of LA size, volume and indexed volume with BSA, estimations from mitral regurgitation velocities by Bernoulli’s equation, pulmonary venous flow profile, and pulmonary artery catheter-based pulmonary capillary wedge pressure estimation are some of the parameters that can give some rough estimate of LA pressure. E/E’ is one of the nearest simple non-invasive way of corroborating LA pressure but Doppler alignment in both the mitral inflow and the lateral mitral annulus may be challenging. Although the correlation of E/E’ with LAP is best in the setting of impaired LV systolic function, it also holds true with preserved systolic function.^[12]

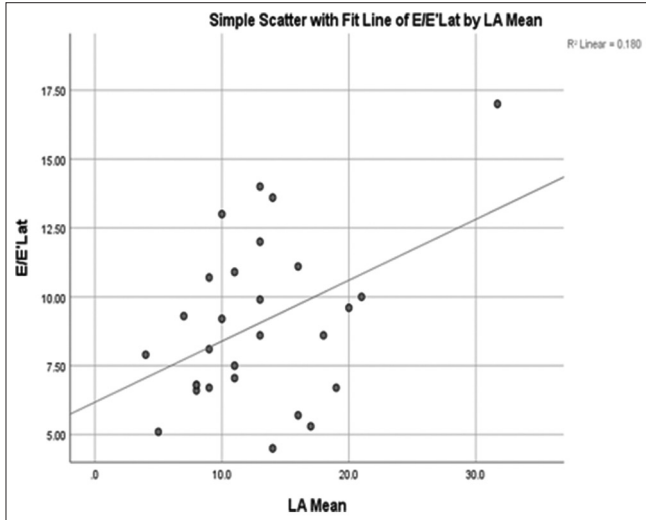


Figure 3: Scatter plots showing correlation between LAP and E/E’ lateral

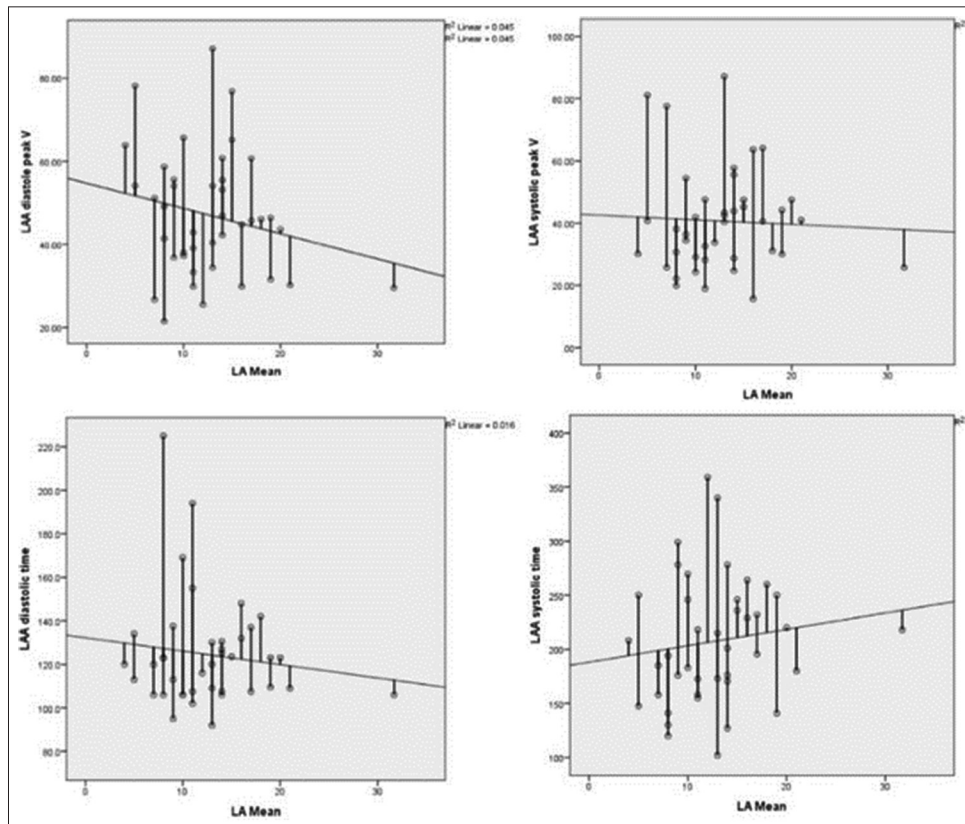


Figure 4: Scatter plots showing no correlation between LAA flow characteristics and LAP

Changes in LAP can affect LAA flow spectra. A study by Tabata *et al.*^[2] to examine changes in LAA flow pattern in relation to LAP measured by right heart catheterization in patients with myocardial disease suggests that marked elevation in LAP reduces LAA peak contraction velocity even in patients in sinus rhythm, when compared to control group without cardiovascular disease. In our study, we included patients with normal LV function in sinus rhythm undergoing elective OPCABG and tried to find a correlation between LAA flow velocities and simultaneous direct measurement of LAP, so that we could produce an equation to estimate LA pressure non-invasively by measuring LAA flow velocities.

The hemodynamic changes caused by anesthetic agents may reduce the measured velocities; however we measured the LAA flow velocities when the hemodynamic parameters were in the pre-induction range and at the same time the surgeon measured the LA pressure directly using a 26-gauge needle inserted into the left atrium and attached to a pressure measuring line. We couldn't find any previous study in which LAA flow velocities were measured intraoperatively. In our study, the mean LAA peak contraction velocity was 47.03 ± 15.05 cm/s and mean LAA filling velocity was 40.7 ± 16.6 cm/s. These measurements were within the previously defined average LAA contraction (50–60 cm/s) and filling (40–50 cm/s) velocities measured by TEE under topical anesthesia and moderate intravenous sedation.^[13,14]

The mean direct measurement of LAP was 12.7 ± 5.2 mmHg and mean E/E' lateral was 9.01 ± 3 . On analyzing the correlations of directly measured LAP with the LAA flow profile, we found no correlation between LAA peak diastolic velocity, systolic velocity, and the durations of both the spectra with simultaneous direct measurement of LAP. But we found positive correlation between E/E' and simultaneous direct measurement of LAP (correlation coefficient 0.424, $P = 0.024$), and it is already known that measuring E/E' is a simple non-invasive method of predicting LAP.

There are very few studies examining the relation between E/E' and direct measurement of LAP. A study by Ritzema *et al.*^[15] to determine the accuracy of Doppler echocardiography and tissue Doppler imaging (TDI) measurements in detecting elevated left atrial pressure (LAP) in 15 ambulant subjects with chronic heart failure and a permanently implanted direct LAP monitoring device found a positive correlation between E/E' and direct measurement of LAP. In our study, we found positive correlation between E/E' and simultaneous direct measurement of LAP in patients with preserved LV function [Table 3].

As we found positive correlation between E/E' and LAP in the intraoperative setting, under anesthesia, and there was no correlation between LAP and LAA flow velocities, LAA flow velocities cannot predict LAP in patients with normal LV function in sinus rhythm.

CONCLUSION

In this study, LAA flow velocities and duration were correlated with direct measurement of LAP in order to generate an equation by which we could do an estimation of LAP non-invasively. However, we found no correlation and therefore LAA flow velocities cannot help in that assessment. Whereas E/E' showed a definite correlation and this parameter is already used in Nagueh formula to calculate LAP.^[16]

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Conflicts of interest

There are no conflicts of interest.

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