

Background & Motivation

Aortic insufficiency (AI) or Aortic regurgitation is abnormal retrograde blood flow due to an incompetent aortic valve (AV) in the heart.

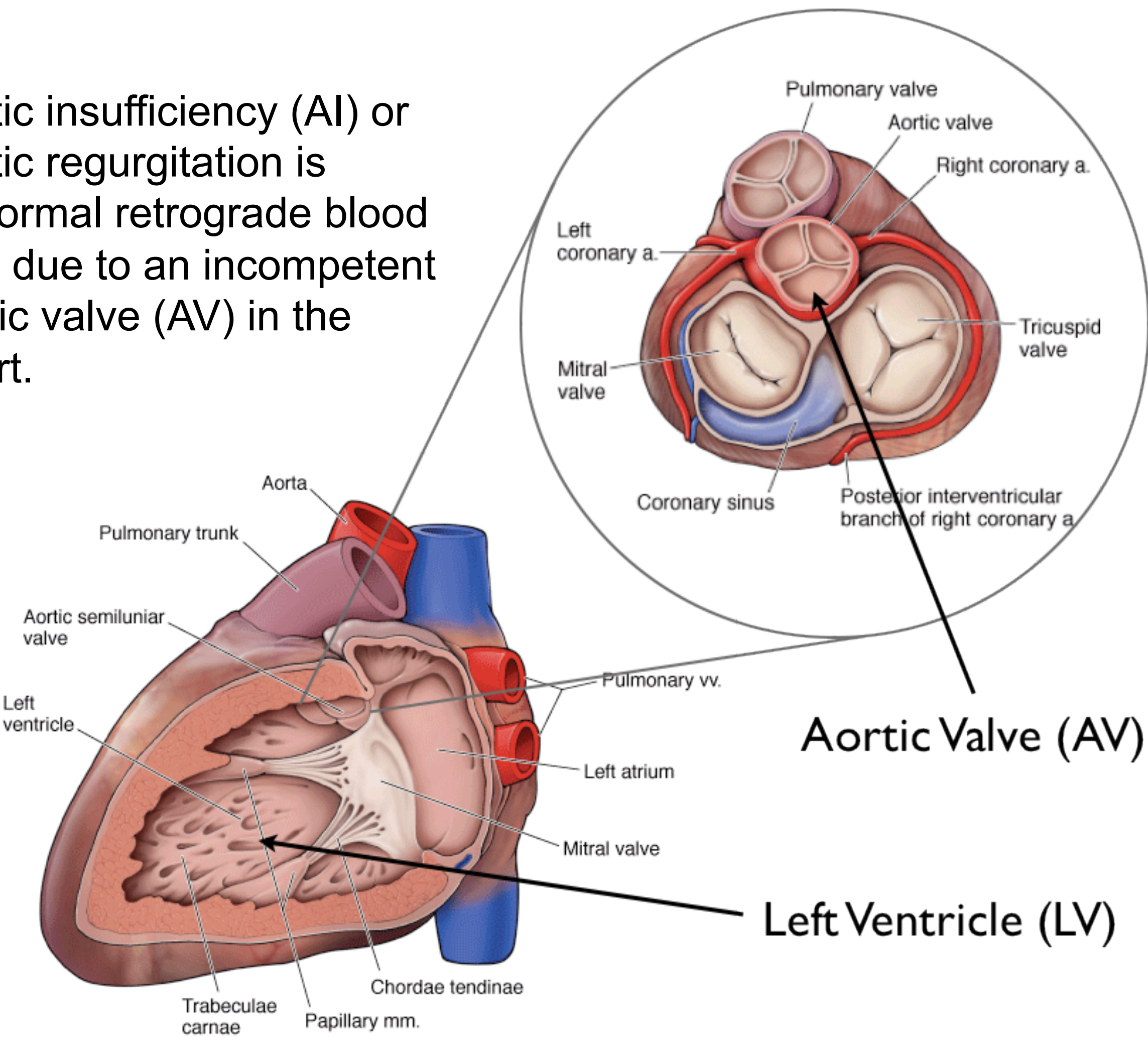


Figure 1 (above): Normal anatomy of the heart. Adapted from [5].

Causes of Aortic Insufficiency include:

- enlargement of the aortic root (aortic aneurysm, Marfan syndrome, aortic dissection etc.)
- abnormalities of the valve leaflets (bicuspid valve, degenerative, rheumatic) [1].

As a result of a chronic increase in regurgitant blood flow, the left ventricle (LV), which is responsible for pumping blood to the systemic circulation, undergoes compensatory remodeling. This adaptation includes:

- left ventricular hypertrophy
- enlargement of the chamber
- increased wall thickness (eccentric hypertrophy) [2].

Patients may be asymptomatic for some time owing to this chronic compensatory phase, however they are monitored with serial echocardiograms as ensuing LV dysfunction and dilatation can indicate poor surgical outcome [2, 3].

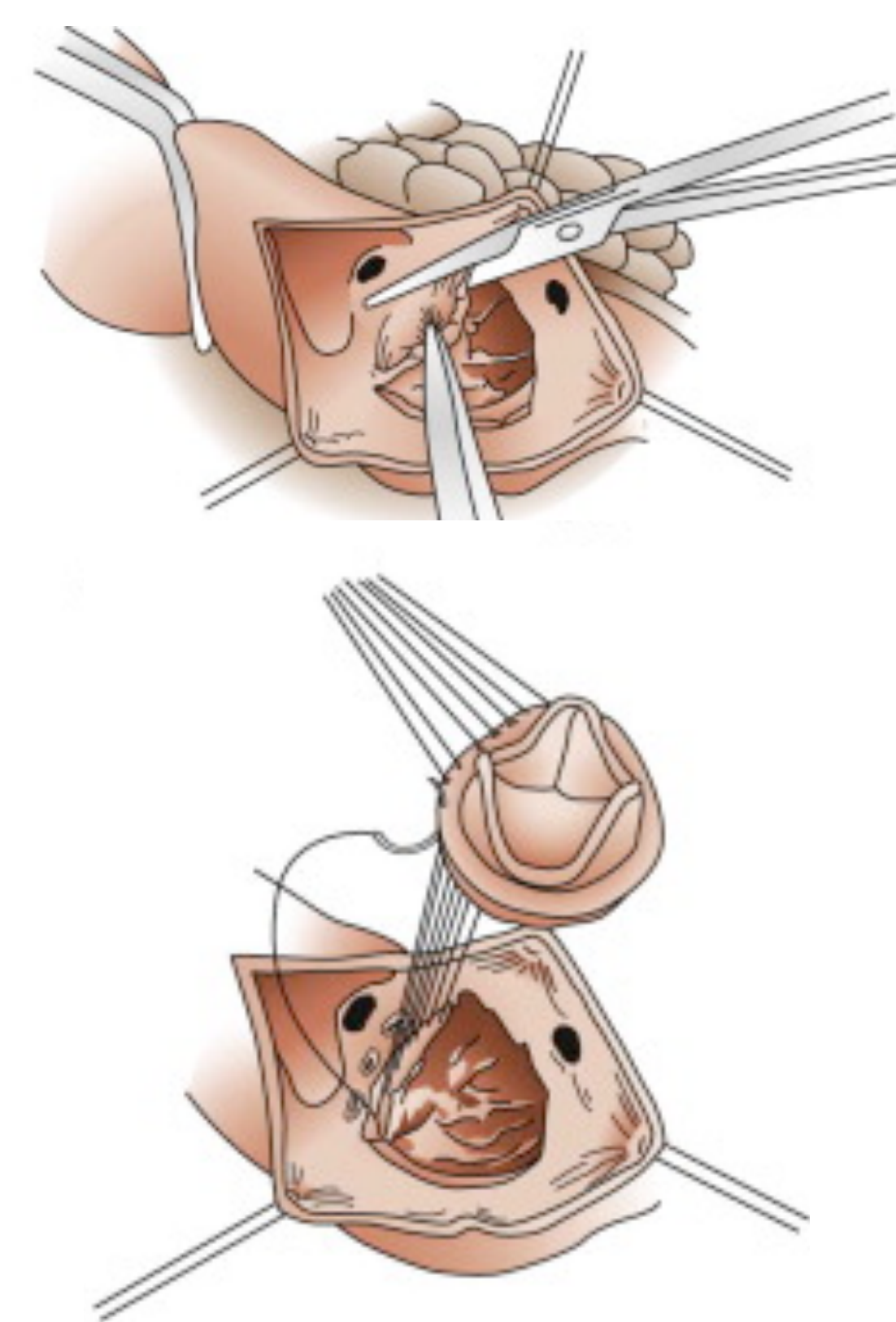
Surgical Correction:

AV replacement with a prosthetic valve is an established surgical option, however it has inherent limitations and complications:

- the requirement of lifelong anticoagulation
- durability of the valve
- risks of endocarditis [4]

A reconstructive surgical approach, namely AV repair has recently gained increasing interest as an alternative to AV replacement.

Figure 2 (right): Surgical aortic valve replacement. Adapted from [6].



Objectives

- elucidate the efficacy and safety of AV repair compared to AV replacement
- analyze characteristics of left ventricular recovery, particularly size and function following surgery (AV repair or replacement)
- identify trends in postoperative recurrence of AI following AV repair or replacement

Materials & Methods

Study Design:

Include (n = 57): All patients undergoing AV Repair/Replacement for primary indication of chronic severe AR with or without concomitant cardiac Sx, at UOHI Retrospective.

Patient factors:

- Age, Sex
- Comorbidities: HTN, CHF, prior MI, infective endocarditis, other cardiac

Preoperative Assessment:

- etiology of AR: annular dilation, bicuspid valve, cusp prolapse
- Preoperative Echo: LVEDD, LVESD, FS, LV Mass
- Degree of AI

AV Replacement

n = 28

AV Repair

n = 29

Postoperative Assessment:

- Echo: LVEDD, LVESD, FS, LV Mass
- Recurrence and degree of AI

Exclusion (n = 262):

- Patients without adequate echocardiographic followup
- acute AI
- Patients undergoing AVR for reasons other than primary moderate to severe AI

Echocardiography:

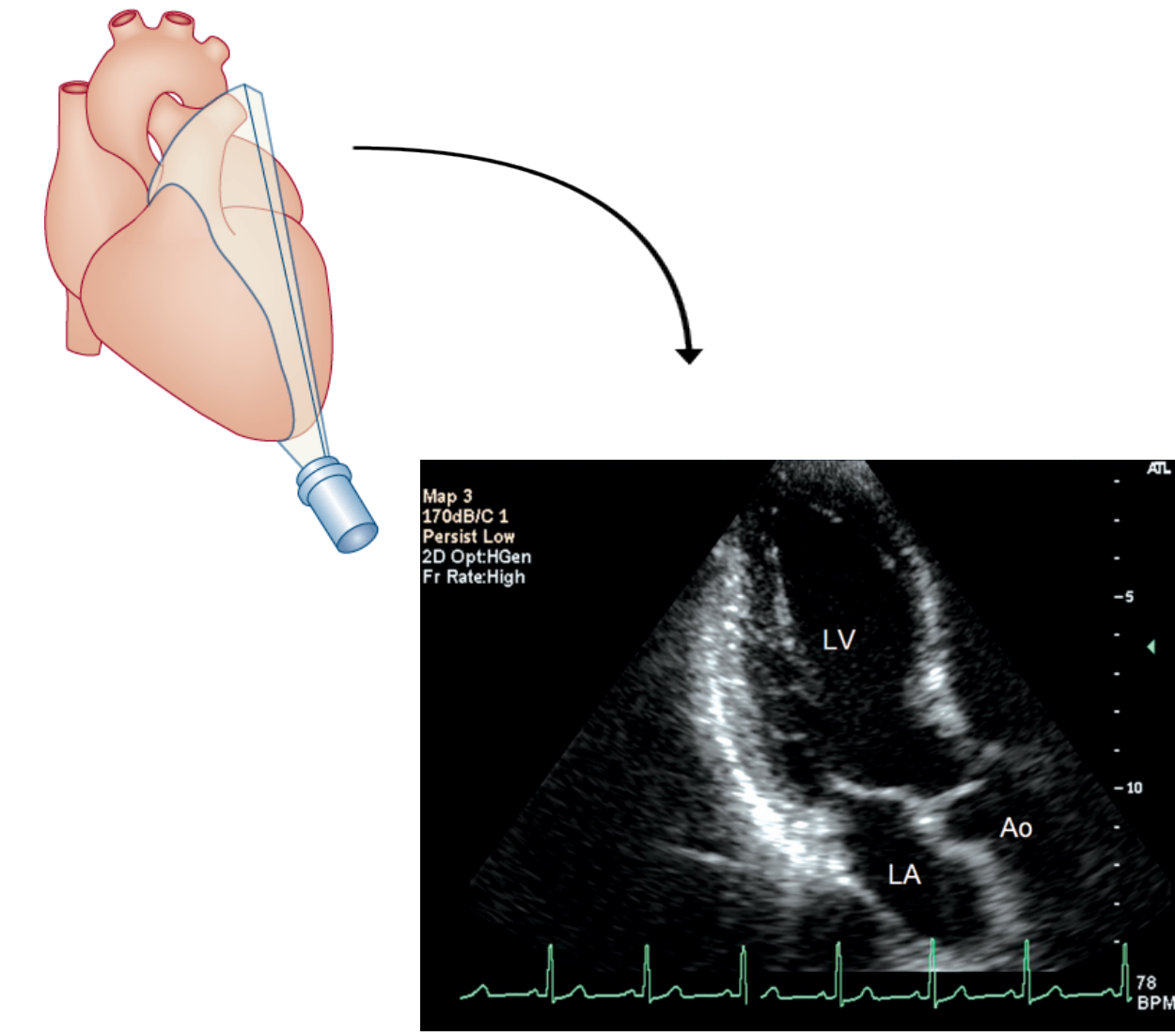
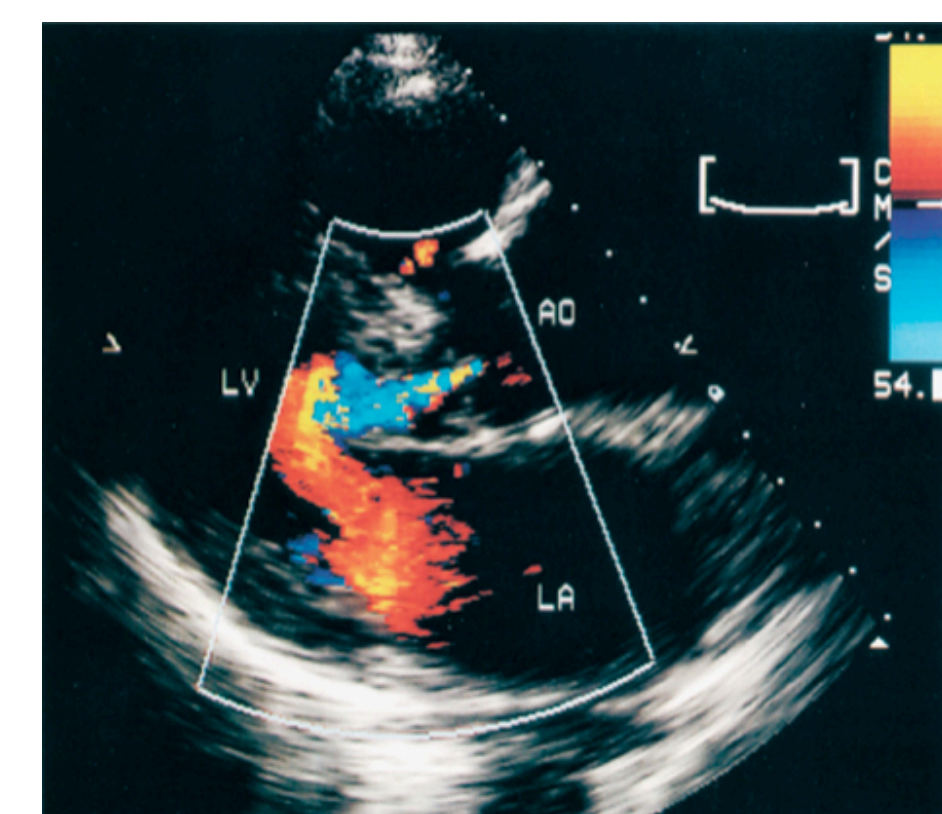


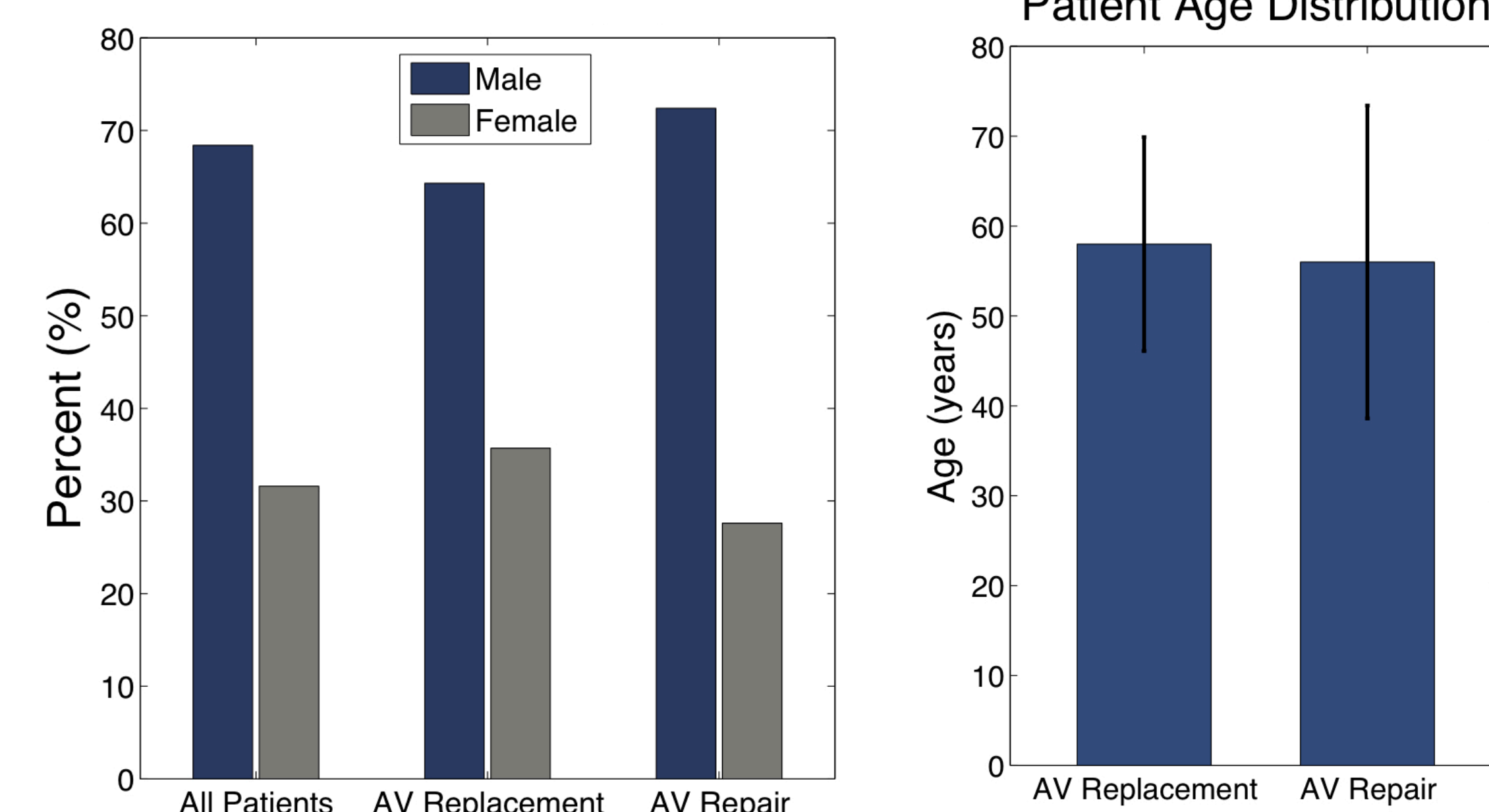
Figure 3 (above): Depicts basic 2D echocardiogram three chamber view. LV: Left Ventricle, LA: Left Atrium, Ao: Aorta.

Figure 4 (left): Doppler echo shows turbulent flow of aortic insufficiency. Preoperative and postoperative assessments were based on echocardiographic parameters such as Left ventricular end-diastolic dimension (LVEDD), Left ventricular end-systolic dimension, fractional shortening (FS), and left ventricular mass (LV Mass). Both figures adapted from [4].

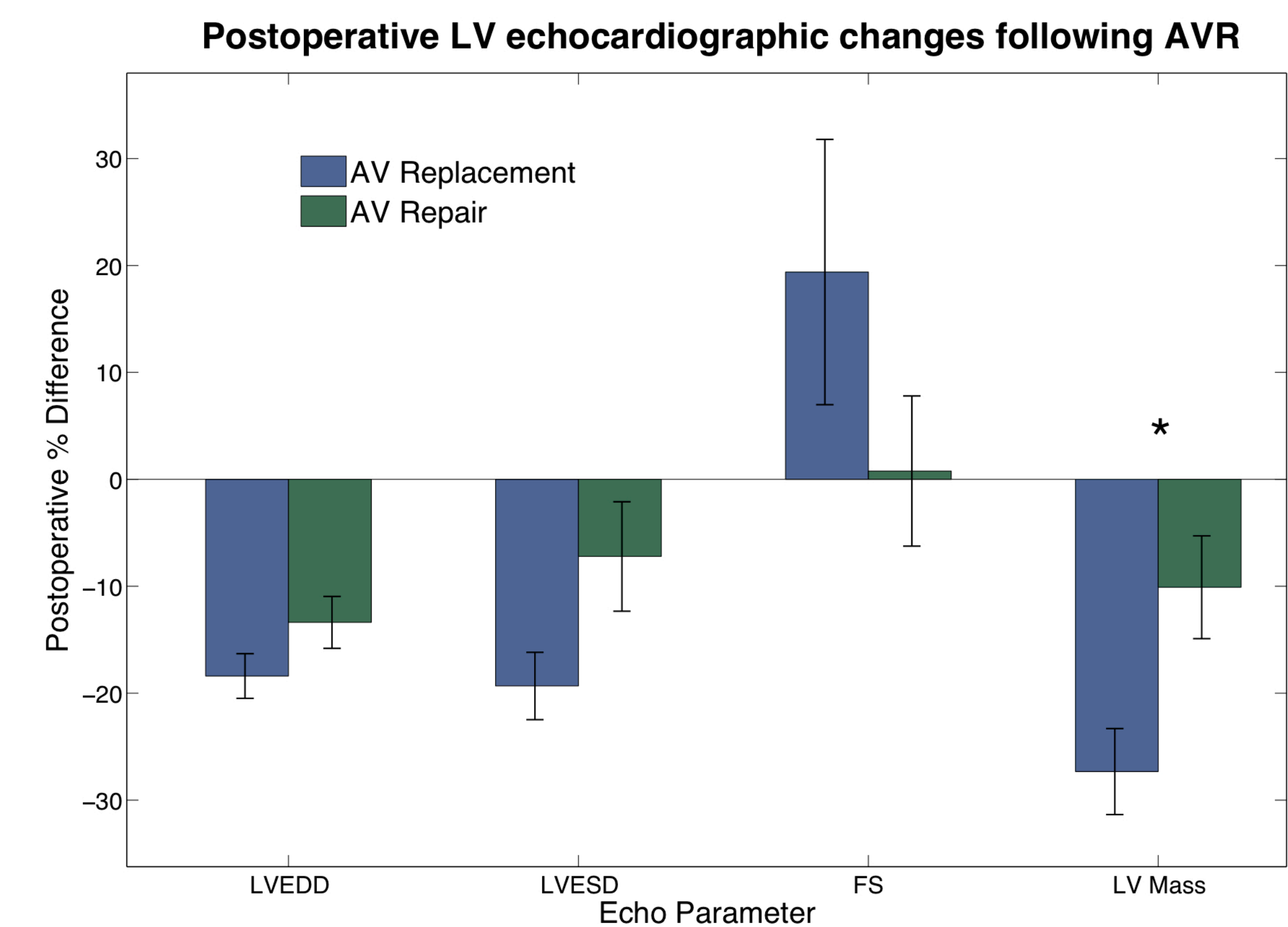
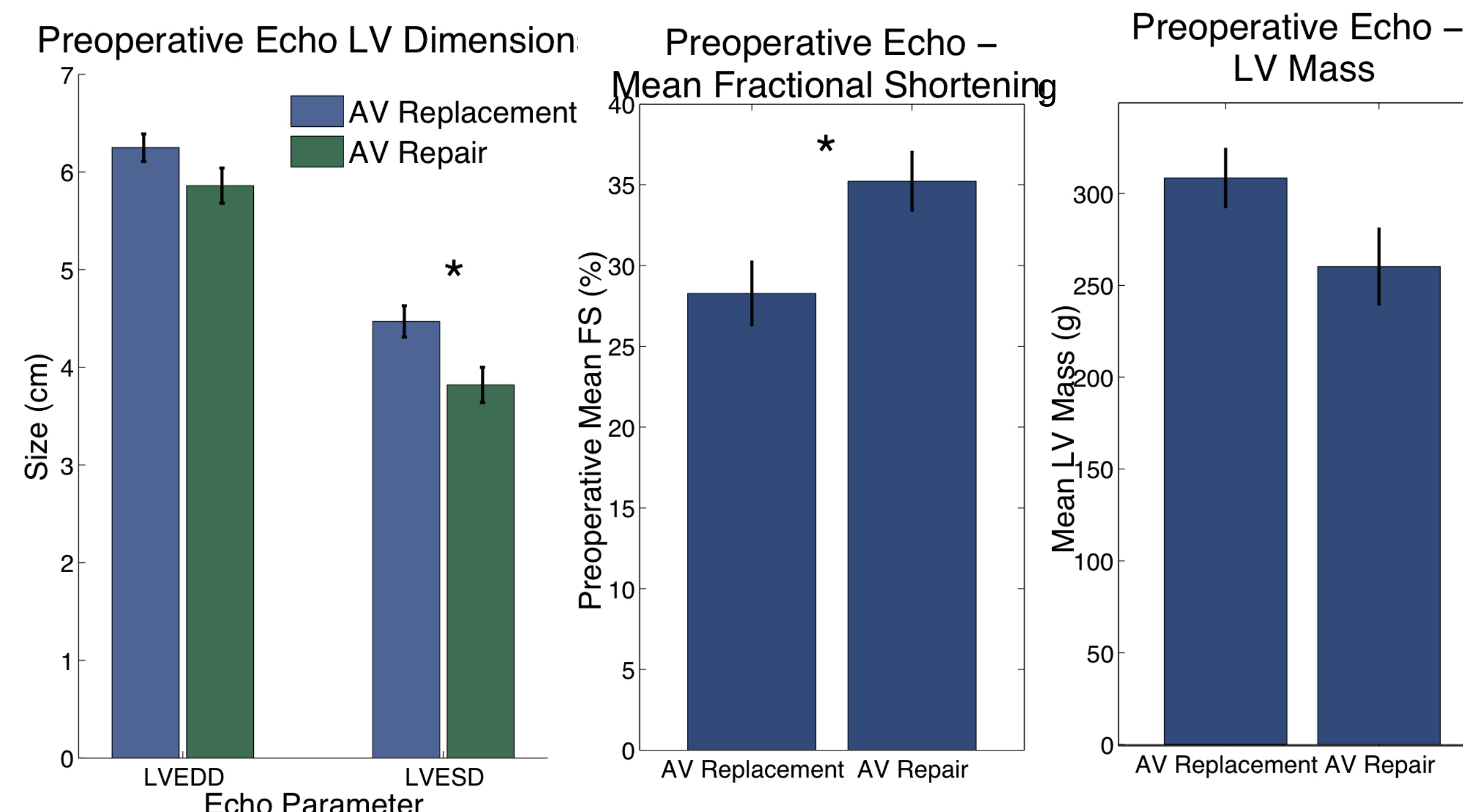


Results

Patient Demographic Data:



Preoperative echocardiographic parameter results:



• **Preoperatively**, there was no statistically significant difference in LVEDD or LV Mass between patient groups ($p > 0.05$). There was however a statistically significant difference in preoperative LVESD and FS for patients undergoing AV replacement vs. AV repair (paired t-test, $p = 0.0129$ and $p = 0.0126$ respectively).

• **Postoperative** left ventricular echocardiographic parameters were compared to their respective preoperative value for each patient. Mean % difference is depicted for each parameter. Error bars represent standard error of the mean (SEM). There was a statistically significant difference in % change in Left Ventricular Mass (LV Mass) for patients who underwent AV Replacement compared to AV Repair (t-test, $p < 0.05$).

Discussion

- There was a statistically significant reduction in LV Mass in patients who underwent AV replacement compared to AV repair (t-test, $p < 0.05$)
- This data should be analyzed in light of loss of echocardiographic follow-up in patients and should be correlated with follow-up in months, in future studies
- There was a greater degree of improvement in all echocardiographic parameters following AV replacement when compared to AV repair
- However it is not clear whether this degree of improvement reflects a poorer preoperative status in AV replacement patients

Conclusion

- This preliminary study has identified characteristics of LV recovery in patients who have undergone AV repair or replacement at the University of Ottawa Heart Institute
- There are however some important limitations. In particular, this project has identified potential future avenues of research that may include expanding the patient sample, clinical correlation, identification of preoperative predictors of outcome, and analysis of left ventricular recovery over a longer follow-up time course

References & Acknowledgements

Works Cited: [1] Lilly LS. Pathophysiology of Heart Disease. Baltimore, MD: Lippincott Williams & Wilkins; 2011. [2] Bashore TM, Granger CB, Hranitzky P, Patel MR. Chapter 10. Heart Disease. In: McPhee SJ, Papadakis MA, Rabow MW, eds. CURRENT Medical Diagnosis & Treatment 2012. New York: McGraw-Hill; 2012. [3] Bonow RO, Carabello B a, Kanu C, et al. ACC/AHA 2006 guidelines for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Circulation. 2006;114(5):e84-231. [4] Fuster V, Walsh RA, Harrington RA, eds. Hurst's The Heart. 13th ed. New York: McGraw-Hill; 2011. **Figure Citations:** [5] Morton DA, Foreman KB, Albertine KH. Chapter 4. Heart. In: Morton DA, Foreman KB, Albertine KH, eds. The Big Picture: Gross Anatomy. New York: McGraw-Hill; 2011. [6] Surgical approach in aortic valve replacement: Fom Albertucci M, Karp RB: Prosthetic valve replacement. In Al Zaibag M, Duran CMG [eds]: Valvular heart disease. New York, 1994, Marcel Dekker, p 615.) and [4]. **Acknowledgements:** Financial support was provided through the University of Ottawa, Undergraduate Research Opportunity Program (UROP).