#ASEchoJC Twitter Chat
Tuesday, October 27, 8 PM ET

Accuracy of the Single Cycle Length Method for Calculation of Aortic Effective Orifice Area in Irregular Heart Rhythms

Introduction & Welcome: Ritu Thamman (@iamritu), Nadeen Faza (@NadeenFaza) & Enrique Garcia-Sayan (@EGarciaSayan) – *Moderators*

Welcome: @iamritu: Welcome to tonight's #ASEchoJC with @hahn_rt @OKhaliqueMD paper with moderators @NadeenFaza @EGarciaSayan @nishath_quader & me Suggest joining the new interventional #echofirst specialty interest @ASE360 group

Paper: https://bit.ly/35Inhya

Tweetorial Memo: https://bit.ly/3oxyNFs

Q1. @iamritu: @ASE360 #EACVI #ASEchoJC "Special care must be taken to select representative sequences of beats and to avoid post-extrasystolic beats."https://bit.ly/31Ozpww

How can we use post-extrasystolic beats in AS assessment clinically to differentiate pseudo versus true severe AS?

EACVI/ASE CLINICAL RECOMMENDATIONS

Recommendations on the Echocardiographic Assessment of Aortic Valve Stenosis: A Focused Update from the European Association of Cardiovascular Imaging and the American Society of Echocardiography

Helmut Baumgartner, MD, FESC, (Chair), Judy Hung, MD, FASE, (Co-Chair), Javier Bermejo, MD, PhD, John B. Chambers, MB BChir, FESC, Thor Edvardsen, MD, PhD, FESC, Steven Goldstein, MD, FASE, Patrizio Lancellotti, MD, PhD, FESC, Melissa LeFevre, RDCS, Fletcher Miller Jr., MD, FASE, and Catherine M. Otto, MD, FESC, *Muenster, Germany; Boston, Massachusetts; Madrid, Spain; London, United Kingdom; Oslo, Norway; Washington, District of Columbia; Liège, Belgium; Bari, Italy; Durham, North Carolina; Rochester, Minnesota; and Seattle, Washington*

Echocardiography is the key tool for the diagnosis and evaluation of aortic stenosis. Because clinical decision-making is based on the echocardiographic assessment of its severity, it is essential that standards are adopted to maintain accuracy and consistency across echocardiographic laboratories. Detailed recommendations for the echocardiographic assessment of valve stenosis were published by the European Association of Echocardiography and the American Society of Echocardiography in 2009. In the meantime, numerous new studies on aortic stenosis have been published with particular new insights into the difficult subgroup of low gradient aortic stenosis making an update of recommendations necessary. The document focuses in particular on the optimization of left ventricular outflow tract assessment, low flow, low gradient aortic stenosis with preserved ejection fraction, a new classification of aortic stenosis by gradient, flow and ejection fraction, and a grading algorithm for an integrated and stepwise approach of aortic stenosis assessment in clinical practice.
(J Am Soc Echocardiogr 2017;30:372-92.)

Three or more beats should be averaged for patients in sinus rhythm.

Averaging of more beats is mandatory with irregular rhythms (at least 5 consecutive beats). Special care must be taken to select representative sequences of beats and to avoid post-extrasystolic beats.

The shape of the CWD velocity curve is helpful in distinguishing

A1: Notable Responses:

@NadeenFaza: Postextrasystolic potentiation following a PAC or PVC leads to peak AV velocities & mean aortic gradients similar to DSE used in low flow low gradient AS. This helps differentiate severe from pseudosevere/moderate AS. #ASEchoJC

@AntonioBarros_Replying to @NadeenFaza Post-Extrasystolic Transaortic Valve Gradients Differentiate "Pseudo" and "True" Low-Flow, Low-Gradient Severe AS D…https://pubmed.ncbi.nlm.nih.gov/28109925/

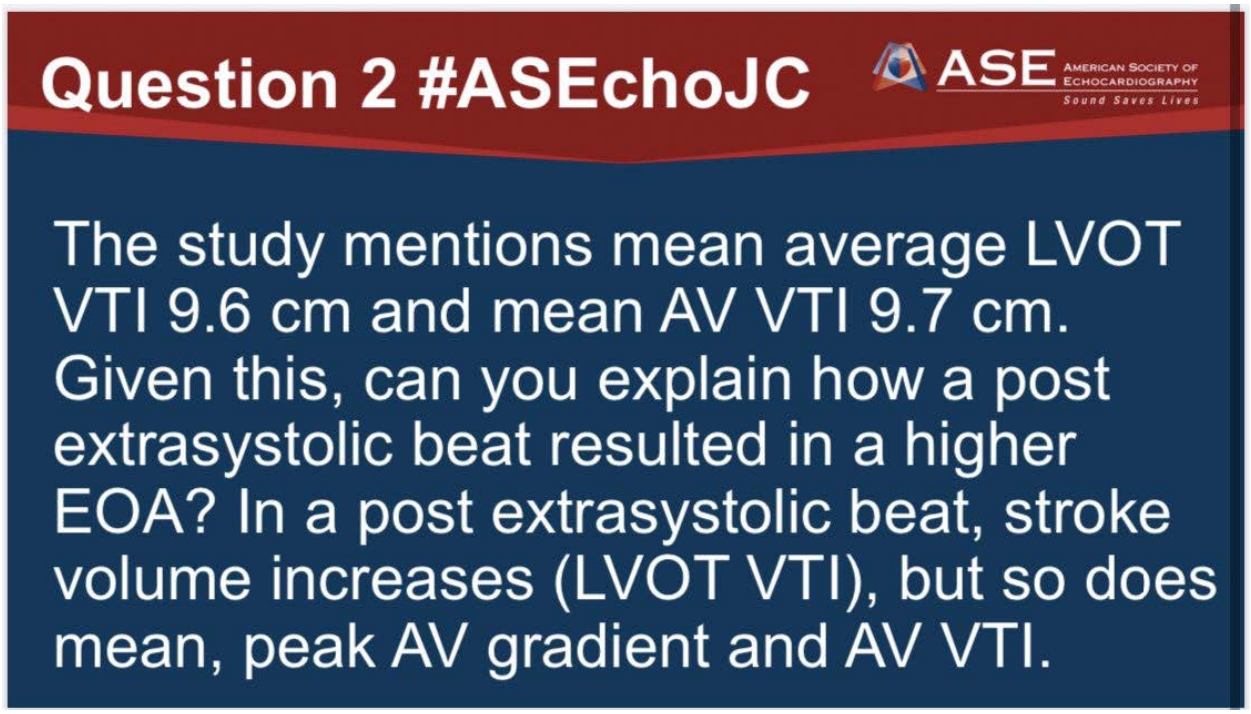
@PPibarot: Post-extrasystolic beats generate higher gradients because flow is higher on these beats. This is similar to the effect of dobutamine stress but one punctual beat. This approach is useful to differentiate true vs pseudo-severe AS.

@PPibarot: So if you get a mean gradient > 40 mmHg on a post-extra systolic beat, this is likely true severe AS and no need to do a DSE.

Q2: @iamritu: Study mentions mean average LVOT VTI 9.6 cm and mean AV VTI 9.7 cm.

Given this, can you explain how a post extrasystolic beat resulted in a higher EOA?

In a post extrasystolic beat, stroke volume increases (LVOT VTI), but so does mean, peak AV gradient and AV VTI



A2 Notable Responses:

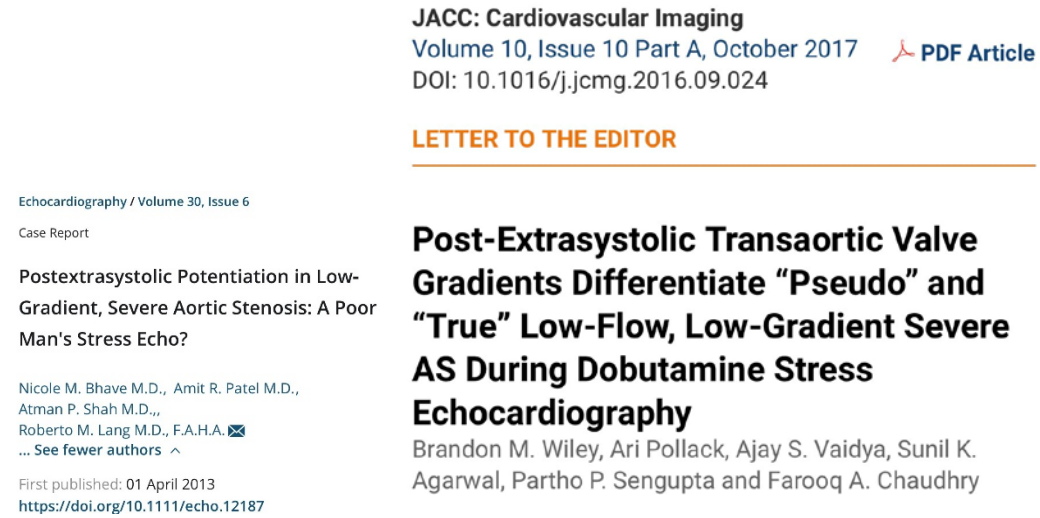
@hahn_rt: If the post-ectopic beat increases stroke volume to ≥35 ml/m2 you can: 1) identify true aortic stenosis if the resulting AVA remains <1.0cm2, 2) diagnose pseudo-severe or moderate aortic if resulting AVA increases to ≥ 1.0cm2.

We now know that low flow, low gradient severe AS may benefit from intervention and the TAVR UNLOAD trial (moderate AS with reduced LVEF) is enrolling. #ASEchoJC

@DavidWienerMD: #ASEchoJC Post extrasystolic potentiation = nature's dobutamine @asecho360

@NadeenFaza: Dr @hahn_rt, if you have a patient scheduled for DSE for low flow low gradient severe AS and the patient has PVCs at baseline, do you use the postextrasystolic beat to evaluate AS severity before proceeding with #DSE? Had a similar case recently. #ASEchoJC

@iamritu: @robertomlang @AmitRPatelMD called post extra-systolic potentiation a poorMan DSE #ASEchoJC postextrasystolic potentiation following PVCs leads to peak aortic velocities & mean aortic gradients similar to DSE for evaluation of low-flow, low-gradient AS



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LETTER TO THE EDITOR

Post-Extrasystolic Transaortic Valve Gradients Differentiate "Pseudo" and "True" Low-Flow, Low-Gradient Severe AS During Dobutamine Stress Echocardiography

Brandon M. Wiley, Ari Pollack, Ajay S. Vaidya, Sunil K. Agarwal, Partho P. Sengupta and Farooq A. Chaudhry

Echocardiography / Volume 30, Issue 6

Case Report

Postextrasystolic Potentiation in Low-Gradient, Severe Aortic Stenosis: A Poor Man's Stress Echo?

Nicole M. Bhave M.D., Amit R. Patel M.D., Atman P. Shah M.D.,, Roberto M. Lang M.D., F.A.H.A.
... See fewer authors

First published: 01 April 2013
https://doi.org/10.1111/echo.12187

@EGarciaSayan: Post-extrasystolic beats increase aortic and LVOT DVI. Helpful to keep in mind in the presence of frequent ectopy. The "poor man's DSE"?

A2: RTHahnMD @hahn_rt

Average post-ectopic LVOT VTI = 23±6.4 cm and mean AV = VTI 101±22 cm. Associated with that is an increase in DVI. #ASEchoJC

A2: So both LVOT & AV VTIs increased post ectopy in the FE cohort #ASEchoJC

A2: @EGarciaSayan: #ASEchoJC, post-extrasystolic beats result in Upwards arrowaortic gradient, VTI & stroke vol. Increased EOA likely due to post-extrasystolic potentiation of contractility. May obviate the need for DSE on LFLG AS. See nice case report by @robertomlang @amitrpatelmd https://bit.ly/3e11S7g

A2: @AntonioBarros_ #ASEchoJC Probably because AF and irregular heart rhythms in general affects contractility.

A2: @KerryEsquitinMD: For clarification: 9.6 and 9.7 were the number of beats measured, not the value of the VTIs

A2: @iamritu: Thank you @KerryEsquitinMD for that clarification #ASEchoJC

Table 1 Doppler variables measured by different methods

Variable	Standard approach (a)	Single cycle long RR (b)	Single cycle short RR (c)	*P* value (a vs b)	*P* value (a vs c)	*P* value (b vs c)
VTI_{LVOT} (cm):						
AF	17 ± 4.9	20 ± 5.7	15 ± 5.0	<.0001	<.0001	<.0001
FE	19 ± 5.6	23 ± 6.4	N/A	<.0001	N/A	N/A
VTI_{AV} (cm):						
AF	81 ± 20	90 ± 22	68 ± 20	<.0001	<.0001	<.0001
FE	87 ± 21	101 ± 22	N/A	<.0001	N/A	N/A
Stroke volume (μg):						
AF	63 ± 18	73 ± 21	55 ± 18	<.0001	<.0001	<.0001
FE	78 ± 23	96 ± 28	N/A	<.0001	N/A	N/A
EOA (cm^2):						
AF	0.82 ± 0.27	0.84 ± 0.27	0.86 ± 0.28	.11	.01	.24
FE	0.94 ± 0.32	0.99 ± 0.32	N/A	.0006	N/A	N/A
DVI:						
AF	0.22 ± 0.07	0.23 ± 0.07	0.23 ± 0.07	.09	.01	.28
FE	0.23 ± 0.07	0.24 ± 0.07	N/A	.0002	N/A	N/A

Q3. @iamritu: How applicable is the data overall to a real-Earth globe americas severe AS population in FE group given that 40% had non severe AS? (18 patients w non severe AS & 27 w severe AS) hypotheses generating study or can we justify any definitive conclusions based on sample size? #ASEchoJC

A3 Notable Responses:

A3: @hahn_rt: Not all patients had severe AS. Thus this study is relevant to the general population of patients presenting typically with symptoms and evidence for some degree of AS

A3: @EGarciaSayan: It indeed makes sense that the same findings can be extended to patients with varying degrees of AS. Validation in larger cohorts and correlation with invasive hemodynamic data may lead to widespread application of this method.

A3: @iamritu: Makes sense that was a mix of severe and non severe AS patients #ASEchoJC

A3: @DavidWienerMD: If applicable across all AS grades in a larger study, could improve quality of reporting. I suspect busy labs don't always average 5-10 beats per @ASE360 AS Guideline

A3: @hahn_rt: The database of transthoracic echocardiograms (TTEs) performed on patients undergoing evaluation for #TAVR at Columbia University Medical Center in New York was retrospectively searched from October 2012 through September 2013. #ASEchoJC

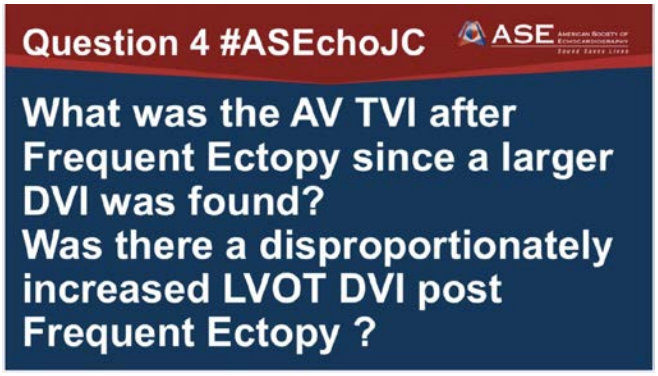
A3: @AtonioBarros_: #ASEchoJC I believe that it would be important to include patients with non-severe AS for a better assessment of reclassification of AS severity with this method. If the clinical difference is as important as the statistics.

A3: @iamritu: Would be interesting to do such a comparison using this single Cycle method versus standard in AFib/ ectopy patients and see the rate of reclassification

A3: @hahn_rt: The reclassification in the AFib group was only 2/52 (3.8%). #ASEchoJC

Q4. @iamritu: What was the AV TVI after Frequent Ectopy since a larger DVI was found?

Was there a disproportionately increased LVOT DVI post Frequent Ectopy? #ASEchoJC



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AF	0.22 ± 0.07	0.23 ± 0.07	0.23 ± 0.07	.09	.01	.28
FE	0.23 ± 0.07	0.24 ± 0.07	N/A	.0002	N/A	N/A

A4 Notable Responses:

A4: @iamritu: Yes, that is how the AVA increased post-ectopic beat. See the DVI in the prior table image. #ASEchoJC

@iamritu: in AFib cohort the DVI only increased in the long RR interval . Did the Aortic valve & LVOT VTI increased similarly? If you don't know the CW & PW are from the same R-R interval,best to measure two R-R intervals that are the same duration,(short,long) & average those?

@hahn_rt: Thus the stroke volume increased and the valve area became larger (thus DVI also increased).

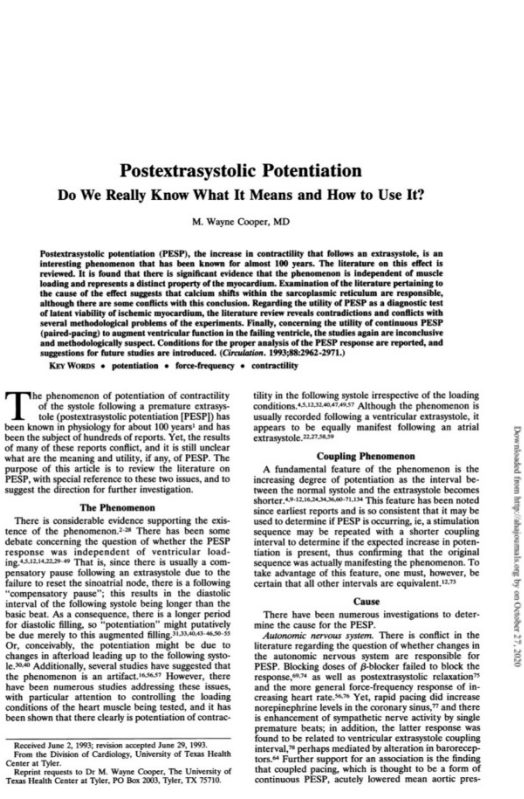
@NadeenFaza: Thank you Dr @hahn_rt! How about patients with frequent #PACs?

@EGarciaSayan: Good question! Are post-extrasystolic beats due to PACs vs PVCs equivalent in terms of post-extrasystolic potentiation and an increase in stroke volume + DVI?

@VlSorrellImages: Since you don't have a compensatory pause after a PAC (the sinus simply resets), I don't think the same physiology will be seen.

@hahn_rt: Post-extrasystolic potentiation has been seen following both APCs and VPCs. See: Cooper MW. Postextrasystolic potentiation. Do we really know what it means and how to use it? Circulation. 1993;88(6):2962-71.

@iamritu: This is a great paper 223 references!



Postextrasystolic Potentiation

Do We Really Know What It Means and How to Use It?

M. Wayne Cooper, MD

Postextrasystolic potentiation (PESP), the increase in contractility that follows an extrasystole, is an interesting phenomenon that has been known for almost 100 years. The literature on this effect is reviewed. It is found that there is significant evidence that the phenomenon is independent of muscle loading and represents a distinct property of the myocardium. Examination of the literature pertaining to the cause of the effect suggests that calcium shifts within the sarcoplasmic reticulum are responsible, although there are some conflicts with this conclusion. Regarding the utility of PESP as a diagnostic test of latent viability of ischemic myocardium, the literature review reveals contradictions and conflicts with several methodological problems of the experiments. Finally, concerning the utility of continuous PESP (paired-pacing) to augment ventricular function in the failing ventricle, the studies again are inconclusive and methodologically suspect. Conditions for the proper analysis of the PESP response are reported, and suggestions for future studies are introduced. (*Circulation*. 1993;88:2962-2971.)

KEY WORDS • potentiation • force-frequency • contractility

The phenomenon of potentiation of contractility of the systole following a premature extrasystole (postextrasystolic potentiation [PESP]) has been known in physiology for about 100 years[1] and has been the subject of hundreds of reports. Yet, the results of many of these reports conflict, and it is still unclear what are the meaning and utility, if any, of PESP. The purpose of this article is to review the literature on PESP, with special reference to these two issues, and to suggest the direction for further investigation.

The Phenomenon

There is considerable evidence supporting the existence of the phenomenon.[2-28] There has been some debate concerning the question of whether the PESP response was independent of ventricular loading.[4,5,12,14,22,29-49] That is, since there is usually a compensatory pause following an extrasystole due to the failure to reset the sinoatrial node, there is a following "compensatory pause"; this results in the diastolic interval of the following systole being longer than the basic beat. As a consequence, there is a longer period for diastolic filling, so "potentiation" might putatively be due merely to this augmented filling.[31,33,40,43-46,50-55] Or, conceivably, the potentiation might be due to changes in afterload leading up to the following systole.[30,40] Additionally, several studies have suggested that the phenomenon is an artifact.[16,56,57] However, there have been numerous studies addressing these issues, with particular attention to controlling the loading conditions of the heart muscle being tested, and it has been shown that there clearly is potentiation of contractility in the following systole irrespective of the loading conditions.[4,5,12,32,40,47,49,57] Although the phenomenon is usually recorded following a ventricular extrasystole, it appears to be equally manifest following an atrial extrasystole.[22,27,58,59]

Coupling Phenomenon

A fundamental feature of the phenomenon is the increasing degree of potentiation as the interval between the normal systole and the extrasystole becomes shorter.[4,9-12,16,28,34,36,60-71,134] This feature has been noted since earliest reports and is so consistent that it may be used to determine if PESP is occurring, ie, a stimulation sequence may be repeated with a shorter coupling interval to determine if the expected increase in potentiation is present, thus confirming that the original sequence was actually manifesting the phenomenon. To take advantage of this feature, one must, however, be certain that all other intervals are equivalent.[12,72]

Cause

There have been numerous investigations to determine the cause for the PESP.

Autonomic nervous system. There is conflict in the literature regarding the question of whether changes in the autonomic nervous system are responsible for PESP. Blocking doses of β-blocker failed to block the response,[69,74] as well as postextrasystolic relaxation[75] and the more general force-frequency response of increasing heart rate.[56,76] Yet, rapid pacing did increase norepinephrine levels in the coronary sinus,[77] and there is enhancement of sympathetic nerve activity by single premature beats; in addition, the latter response was found to be related to ventricular extrasystole coupling interval,[78] perhaps mediated by alteration in baroreceptors.[64] Further support for an association is the finding that coupled pacing, which is thought to be a form of continuous PESP, acutely lowered mean aortic pres-

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Reprint requests to Dr M. Wayne Cooper, The University of Texas Health Center at Tyler, PO Box 2003, Tyler, TX 75710.

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Question 5: @iamritu: Paper mentions that the DVI was larger in Frequent ectopy with post ectopic beat: DVI 0.24 +/- 0.07 vs 0.23 +/- .0.07.

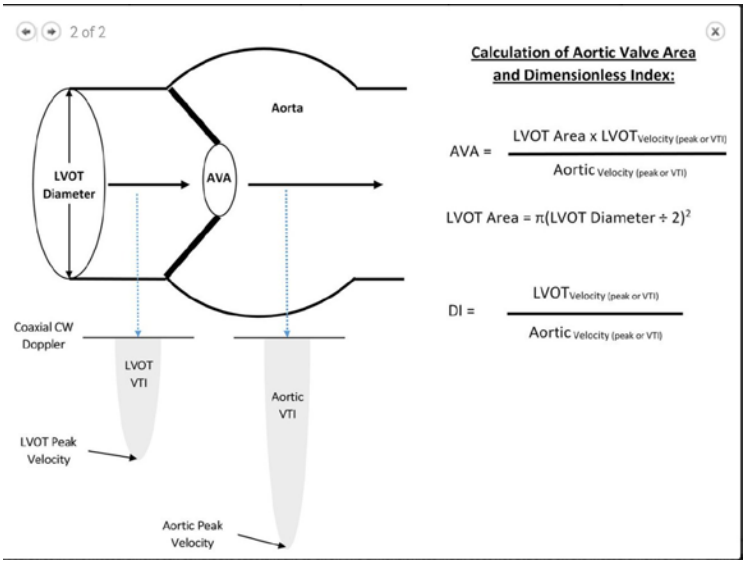
A5 Notable Responses:

@hahn_rt: The significance of an increase would depend on whether that DVI for any single individual actually increased to >0.25. DVI is a powerful parameter for assessing AS severity.

@iamritu: 100% there is prognostic importance of dimensionless index in patients with low flow low gradient severe aortic stenosis & preserved ejection fraction

@hahn_rt: "A measure incorporating peak velocity, mean gradient and dimensionless index (either by velocity time integral or peak velocity ratio) achieved a balance of sensitivity (92%) and specificity (99%)". https://pubmed.ncbi.nlm.nih.gov/29925543/

@NadeenFaza: "A measure that used DVI in place of AVA addressed discrepancies between quantitative echo data and the documented interpretation of severe aortic stenosis" pubmed.ncbi.nlm.nih.gov Q6. How did APCs vs VPCs differ in terms of LVOT VTI, AV VTI, EOA in the study? #ASEchoJC



@hahn_rt: We will be submitting our manuscript on DVI and outcomes from the PARTNER trials soon!

@ash71us: How about the acceleration time/ejection time ratio?

@iamritu: AVA can be underestimated at low transvalvular flow rate. Yet, the impact of flow rate on prognostic value of AVA ≤1.0 cm2 is unknown and is not incorporated into AS assessment.

ET can be a source of error though https://www.onlinejacc.org/content/75/15/1758

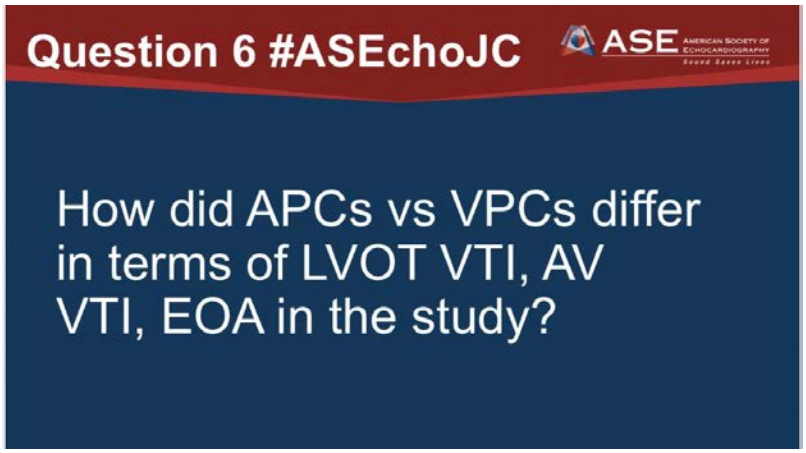
@davidwienerMD: The @ASE360 AS guidelines note the same

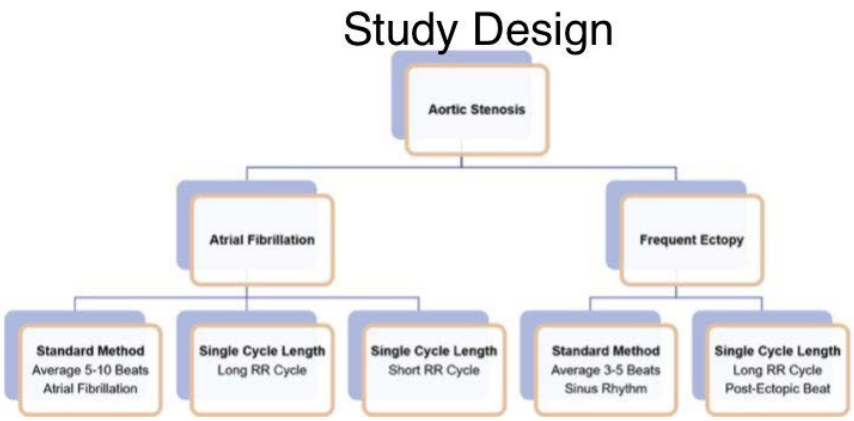
Velocity Ratio and VTI Ratio (Dimensionless Index). Another approach to reducing error related to LVOT area measurements is removing LVOT CSA from the continuity equation. This dimensionless velocity or VTI ratio expresses the size of the valve effective area as a proportion of the CSA of the LVOT:

$$\text{Velocity ratio} = \frac{V_{LVOT}}{V_{AV}}$$

$$\text{VTI ratio} = \frac{VTI_{LVOT}}{VTI_{AV}}.$$

Q6: @iamritu: How did APCs vs VPCs differ in terms of LVOT VTI, AV VTI, EOA in the study?

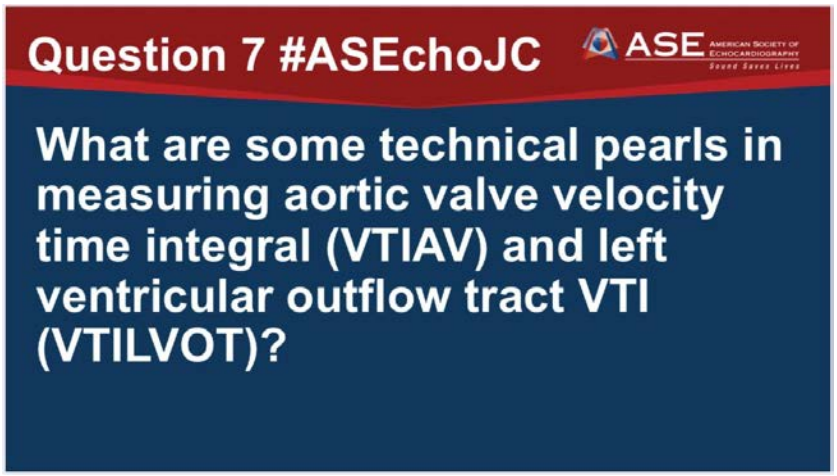




A6 Notable Responses:

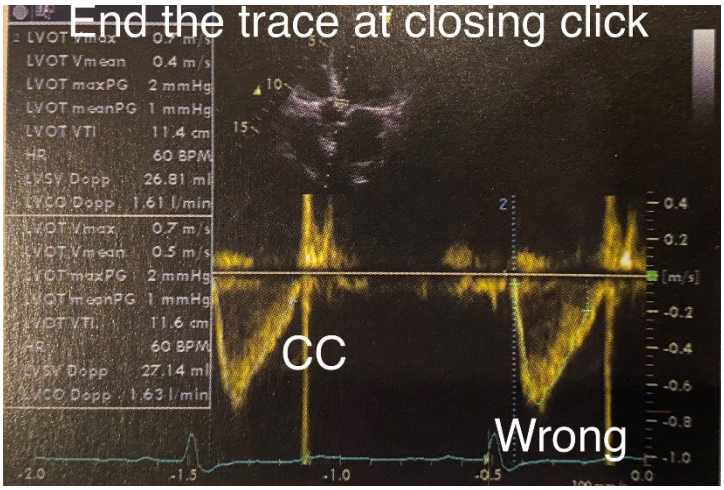
@EGarciaSayan: #ASEchoJC both APCs and VPCs were included in the FE cohort, with increases in aortic gradients, stroke volume and DVI. @hahn_rtpointed out earlier in the discussion a 1993 article describing complex mechanism that goes beyond loading conditions: https://bit.ly/35GOy4k

Q7. @iamritu: What are some technical pearls in measuring aortic valve velocity time integral (VTIAV) & left ventricular outflow tract VTI (VTILVOT)?

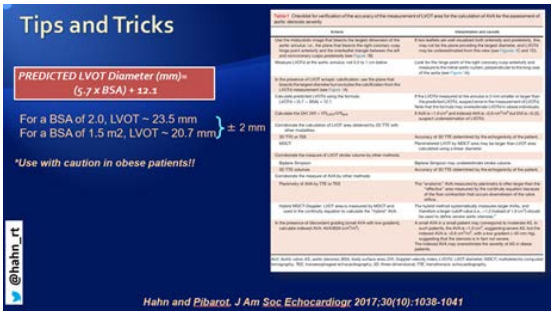


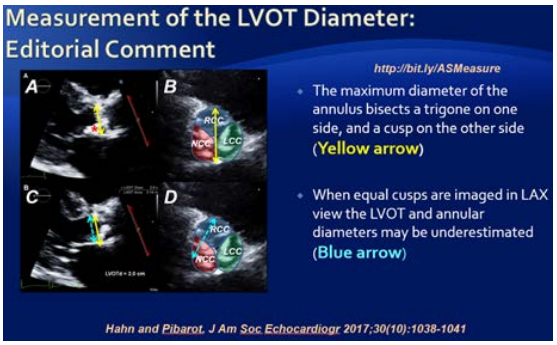
A7 Notable Responses:

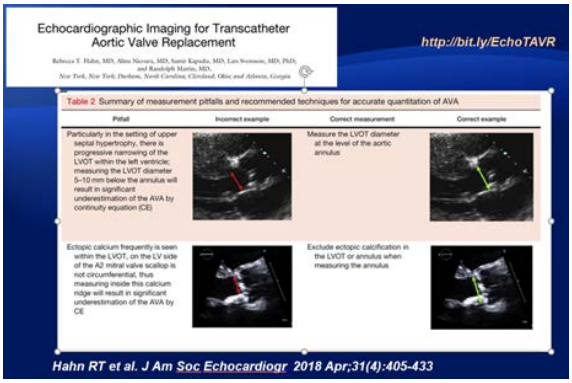
A7: @iamritu: Remember to trace LVOT VTI to the closing click #ASEchoJC



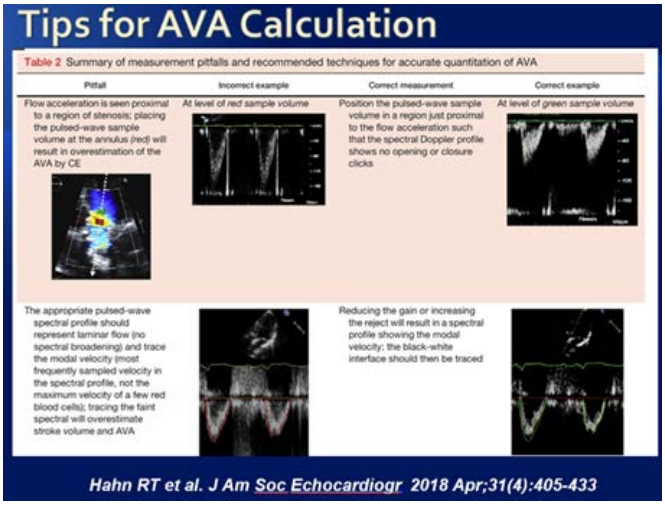
A7: @hahn_rt: Important tips: http://bit.ly/ASMeasure http://bit.ly/EchoTAVR. First: Measure the LVOT diameter close to the annulus, and exclude the ectopic calcification on the anterior mitral valve.



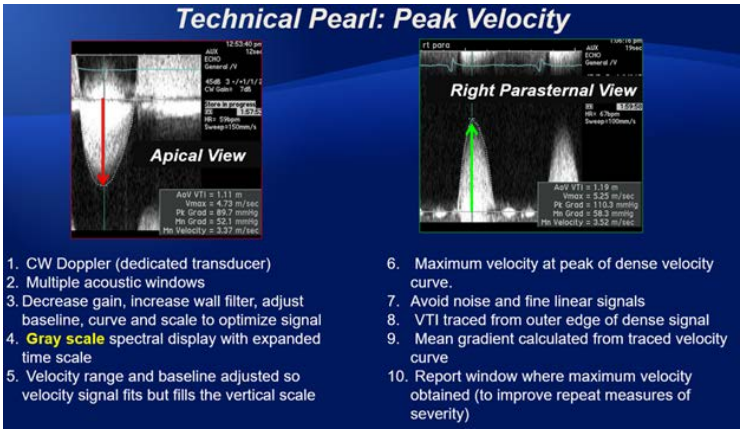


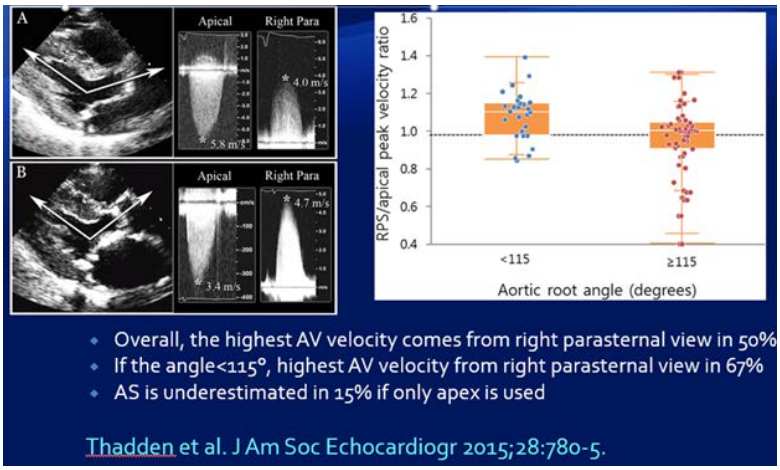


@hahn_rt: Second: use the modal velocity (reduce gain) and position sample volume just proximal (apical) to flow acceleration if possible. NOTE: Sample volume is place on the annulus in the setting of a non-stenotic AV.



@hahn_rt: Third: always check the right parasternal view since in >50% of cases, this will yield the highest transaortic velocity.https://bit.ly/3jB8M4r





@MaheshAnandCh: How close is close? I make sure it is an inner edge to inner edge measurement within 0.5 cm from the annulus on a zoomed PLAX https://t.co/1524D5Bpof?amp=1

@E_Guzzetti: Annulus or very close (i.e., 2 mm) yield better estimations than 5-10 mm below

@DavidWienerMD: Interrogation from multiple windows is a marker of a quality study and is an @IACaccred Echo requirement

vii. For aortic stenosis, the systolic velocity must be evaluated from multiple transducer positions (e.g., apical, suprasternal and right parasternal). This must include interrogation from multiple views with a dedicated non-imaging continuous wave Doppler transducer (at least one clear envelope must be obtained); and

Q9: @iamritu: In this study protocol, the LVOT diameter was measured in mid-systole, within 2-4 mm apical to the annulus. Is this the recommended approach to measuring LVOT diameter?

A9 Notable Responses:

@hahn_rt: Here is the figure from our most recent guidelines: we use mid-systole, however we ONLY measure close to (within 2 mm) or at the annulus. : http://bit.ly/ASMeasure

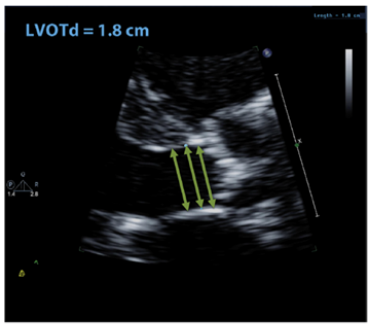


Figure 5 Left ventricular outflow tract diameter (LVOTd) is measured in a zoomed parasternal long-axis view in mid-systole from the white-black interface (inner-to-inner) of the septal endocardium to the anterior mitral leaflet, parallel to the aortic valve plane. Some experts prefer to measure within 0.3–1.0 cm of the valve orifice whereas others prefer the measurement at the annulus level (see text). Note that in many patients, as in this case, the LV outflow tract is relatively rectangular within 1 cm of the aortic annulus. Green double headed arrows show the LVOTd measurement at the annulus and with different distances from it yielding identical measurements in this patient.

Other notable Questions:

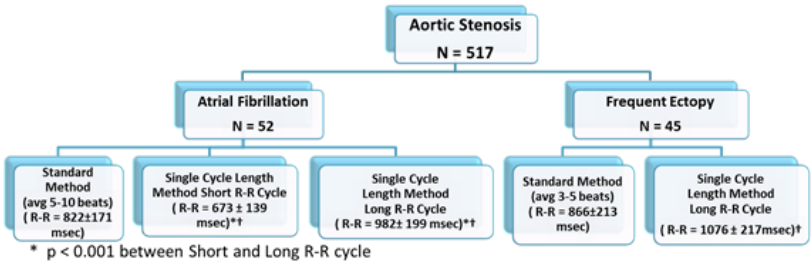
@DavidRubenson: How much CL variability do you think would allow the method to remain accurate? I think you tried to match within 10%

Notable Responses:

@hahn_rt: Similar cycle length was defined as preceding R-R intervals for VTIAV and VTILVOT are within 10% of each other. Short and long R-R cycles were defined relative to the average heart rate. EOA and DVI were then calculated for a short R-R cycle and for a long R-R cycle. #ASEchoJC

@hahn_rt: From @KerryEsquitinMD presentation (see below). #ASEchoJC The p value was statistically significant but given the numbers (0.23 vs 0.24), would you consider this a significant difference?

Table 1: Study Design



* p < 0.001 between Short and Long R-R cycle

† p < 0.001 between Standard and Short R-R cycle or between Standard and Long R-R cycle

@DavidRubenson: I am also curious why you did not assess the correlation between EROA during stable NSR in the FE group, and the EROA values in the other groups. Perhaps I missed that. It would be like a control group