Spring Conference Showcase March 28, 2022

Cardiovascular Complications of COVID-19 and its True Mortality in a Large Metropolitan Health System

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ABSTRACT

Background

Patients with cardiovascular disease are reported to be more susceptible to severe forms of COVID-19. Previous studies also suggest that COVID-19 is a possible risk factor for developing cardiovascular complications. This study was designed to investigate the role of pre-existing and acquired cardiovascular disease in patients who died with a positive COVID-19 diagnosis in the largest health system in the Twin Cities (Allina Health). This study also aims to determine whether COVID-19 was the primary cause of mortality for patients with a positive COVID-19 diagnosis.

Methods

Retrospective chart review was used to analyze cardiovascular complications associated with COVID-19 mortality. Patients who were admitted from 3/1/2020 to 12/31/2020 and died in a metro Allina hospital with a positive COVID-19 diagnosis were included. Cause of death was adjudicated by at least 2 health professionals and was determined through hospital notes, discharge summaries, and labs.

Results

In patients who died primarily of COVID-19, 84% had a history of hypertension, 60% had a history of smoking, 51% had diabetes, 44% had a history of CAD, and 29% had a history of COPD. During hospitalization, 11% had an MI, 5% had a stroke, 41% had atrial fibrillation, and 44% had an elevated troponin level. Of the patients who died due to COVID-19, 56% were given antiviral agents, 72% were given dexamethasone, and 18% were given convalescent plasma. Of the COVID-19 deaths, 26 patients had a new MI and 10 had a new CVA/TIA.

Conclusions

Among COVID-19 positive patients who died in metro Allina hospitals, 82% died primarily of COVID-19 and 18% died primarily of other causes. Of the 18% who died of other causes, cardiovascular etiologies were the most common. Additionally, many pre-existing cardiovascular conditions and new in-hospital complications were found to be associated with deaths caused by COVID-19. When comparing the two groups, patients who died primarily of COVID-19 had a higher body mass index, as well as a higher incidence of COPD, obstructive sleep apnea, and history of smoking.

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BACKGROUND

- The first case of COVID-19 in Minnesota was reported in March 2020.
- Previous studies suggest that COVID-19 is a possible risk factor for cardiovascular complications such as myocarditis, stroke, acute myocardial infarction, and thromboembolic events.
- While cardiovascular complications have been observed in COVID-19 patients in aggregate, individual patient data is rarely reported.
- This study was designed to:
  - Examine the role of pre-existing cardiovascular conditions in patients who died from COVID-19 within the largest health system in the Twin Cities (Allina Health).
  - Measure and identify new cardiovascular complications that developed during the terminal hospitalization of patients who died with COVID-19.
  - Determine the true mortality from COVID-19 in a cohort of consecutive patients diagnosed with the COVID-19 infection.

METHODS

- Individual patient analysis was performed for all reported COVID-19 deaths from 3/1-12/31/2020 (n=455) within the three metro Allina hospitals.
- Medical charts were reviewed with all pre-existing cardiac co-morbidities and procedures recorded and the cause of death adjudicated by at least two health care providers.
- Patients were classified as dying from COVID-19 if they had a positive COVID-19 test and CXR consistent with COVID-19 and suffered from progressive respiratory failure/ARDS (n=371). Patients who had incidental finding of a positive COVID-19 test or whose respiratory status had recovered from COVID-19 and died from other causes were classified as non-COVID-19 deaths (n= 84).

PATIENT POPULATION

Table 1. Demographic characteristics of COVID-19 positive patients who died

	Total: (n = 455)	COVID-19 Deaths (n = 371)	Non-COVID-19 Deaths (n = 84)	*P*-value
Women (%)	181 (40)	143 (39)	38 (45)	0.3
BMI, median (kg/m²) (IQR)	29.6 (25.2, 34.8)	30.1 (25.5, 35.3)	27.8 (24.0, 32.4)	**0.03**
Obesity (%)	212 (47)	179 (48)	33 (39)	0.1
Age, median (yrs) (IQR)	75 (67, 83)	76 (68, 83)	74 (63, 82)	0.258
18-55 (%)	31 (7)	21 (6)	10 (12)	0.2
56-65 (%)	66 (15)	52 (14)	14 (16)	
66-75 (%)	135 (30)	111 (30)	24 (28)	
76-85 (%)	132 (29)	113 (31)	19 (22)	
>85 (%)	91 (20)	73 (20)	18 (21)	
Race				
White (%)	378 (84)	309 (84)	63 (84)	0.694
Black (%)	43 (10)	33 (9)	10 (12)	
Native American or Alaskan Native (%)	10 (2)	8 (2)	2 (2)	
Hawaiian or Pacific Islander (%)	2 (0.5)	2 (1)	0 (0)	
Asian (%)	15 (3)	14 (4)	1 (1)	
Ethnicity				
Non-Hispanic (%)	421 (93)	345 (93)	79 (94)	
Hispanic (%)	31 (7)	26 (7)	5 (6)	

RESULTS

Table 2. Clinical characteristics of COVID-19 positive patients who died

	Total (n = 455)	COVID-19 Deaths (n = 371)	Non-COVID-19 Deaths (n = 84)	*P*-value
Hypertension (%)	381 (85)	308 (84)	73 (89)	0.244
Hx or current smoker (%)	251 (57)	213 (60)	38 (48)	0.064
Dyslipidemia (%)	344 (77)	279 (76)	65 (79)	0.556
History of CAD (%)	196 (44)	160 (44)	36 (44)	0.92
Diabetes (%)	224 (50)	187 (51)	37 (44)	0.263
COPD (%)	122 (27)	106 (29)	16 (20)	0.082
Chronic kidney disease (%)	211 (47)	171 (47)	40 (49)	0.736
Hx of dialysis (%)	22 (5)	17 (5)	5 (6)	0.618
Pulmonary hypertension (%)	71 (16)	61 (17)	10 (12)	0.321
PAD/PVD (%)	121 (27)	96 (26)	25 (30)	0.424
Hx of MI (%)	105 (23)	89 (24)	16 (20)	0.347
Hx of CABG (%)	45 (10)	34 (9)	11 (13)	0.291
Hx of PCI (%)	99 (22)	81 (22)	18 (21)	0.907
Hx of CHF* (%)	141 (33)	118 (33)	23 (29)	0.5
HFpEF (%)	88 (19)	70 (19)	18 (23)	0.5
HFrEF (%)	57 (13)	49 (14)	8 (10)	0.4
Unspecified (%)	14 (3)	13 (4)	1 (1)	0.5
Hx of CVA (%)	102 (23)	81 (22)	21 (26)	0.5
Hx of valve disease (%)	61 (14)	49 (13)	12 (15)	0.8
Hx of valve replacement (%)	20 (4)	17 (5)	3 (4)	>0.99
Hx of atrial fibrillation/atrial flutter (%)	160 (36)	130 (35)	30 (37)	0.8
Hx of obstructive sleep apnea (%)	123 (28)	112 (30)	11 (14)	**0.004**
Prior PPM, ICD or CRT-D/P (%)	55 (12)	49 (13)	6 (7)	0.1

Table 3. In-hospital comparison between COVID-19 deaths vs. other primary causes of death

	Total (n = 455)	COVID-19 Deaths (n = 371)	Non-COVID-19 Deaths (n = 84)	*P*-value
Length of stay (days)	10 (5, 17)	10 (5, 17)	7 (2, 17)	**0.02**
AKI during hospital (%)	271 (60)	223 (60)	48 (57)	0.6
MI in hospital (%)	56 (12)	42 (11)	14 (16)	0.2
CVA in hospital (%)	32 (7)	18 (5)	14 (16)	<0.001
PCI in hospital (%)	8 (2)	2 (0.5)	6 (7)	0.001
DVT or PE (%)	46 (10)	38 (10)	8 (9)	0.8
In-hospital shock* (%)	128 (33)	117 (32)	31 (36)	0.389
Cardiogenic (%)	15 (3)	9 (2)	6 (7)	0.031
Septic (%)	84 (18)	66 (18)	18 (21)	0.474
Other/Unspecified (%)	54 (12)	45 (12)	9 (11)	0.686
ECCO2R (%)	9 (2)	9 (2)	0 (0)	0.2
ECMO (%)	12 (3)	9 (2)	3 (4)	0.476
CRRT or dialysis in hospital (%)	31 (7)	26 (7)	5 (6)	0.812
Atrial fibrillation/atrial flutter (%)	177 (39)	152 (41)	25 (29)	0.05

*Some patients had multiple types of CHF or in-hospital shock

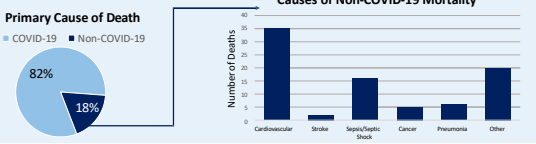
Table 4. Peak lab values for COVID-19 deaths vs. Non-COVID-19 deaths

Labs	Total (n = 455)	COVID-19 Deaths (n = 371)	Non-COVID-19 Deaths (n = 84)	*P*-value
Troponin, median (ng/ml) (IQR)	0.061 (0.020, 0.216)	0.053 (0.020, 0.202)	0.09 (0.019, 0.713)	0.2
BNP, median (pg/ml) (IQR)	199 (63, 528)	187 (59, 486)	253 (81, 1315)	0.1
Creatinine, median (mg/dl) (IQR)	1.68 (1.16, 2.74)	1.67 (1.18, 2.60)	1.80 (1.10, 3.57)	0.5

Table 5. COVID-19 therapies for COVID-19 positive patients who died

	Total (n = 455)	COVID-19 Deaths (n = 371)	Non-COVID-19 Deaths (n = 84)	P-value
Antiviral agents (%)	233 (51)	207 (56)	26 (31)	**<0.001**
Dexamethasone (%)	308 (68)	266 (72)	42 (49)	**<0.001**
Hydroxychloroquine (%)	14 (3)	14 (4)	0 (0)	0.083
Convalescent plasma (%)	75 (16)	65 (18)	10 (12)	0.194
Monoclonal antibodies (%)	44 (10)	39 (11)	5 (6)	0.19
BiPAP/CPAP (%)	301 (66)	265 (72)	36 (42)	<0.001
Supplemental oxygen (%)	419 (92)	349 (94)	70 (82)	<0.001
Mechanical ventilation (%)	208 (46)	173 (47)	35 (41)	
Time on ventilator (days)				
<5	55 (26)	41 (24)	14 (40)	0.19
5-10	50 (24)	45 (26)	5 (14)	
11-15	36 (17)	30 (17)	6 (17)	
>15	67 (32)	57 (33)	10 (29)	

Figure 1. Classification of total deaths



CONCLUSIONS

- In the largest health system in the metropolitan Minneapolis/St. Paul area, among COVID-19 patients who died in an Allina hospital, 82% had primarily COVID-19/ARDS-related deaths, and 18% had or acquired other diagnoses that were designated as their primary cause of death.
- Of the 18% that died of non-COVID-19 causes, cardiovascular-related death was the most common, including MI, stroke, pulmonary embolism, cardiogenic shock, and cardiac arrest.
- Pre-existing cardiovascular comorbidities and acquired cardiovascular complications are common among patients who died from COVID-19 and patients who had COVID-19 but died from other causes.
- Patients who died of COVID-19 had a higher body mass index, as well as a higher incidence of COPD, obstructive sleep apnea, and history of smoking compared to non-COVID-19 deaths.

ACKNOWLEDGMENTS

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DISCLOSURES

- The authors have no relevant financial or nonfinancial relationships to disclose.

Cardiac Resynchronization Therapy Optimization in Non-responders Using Electrical Dyssynchrony Mapping

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Disclosures

Honoraria/Speaking/Consulting Fee: Medtronic, Abbott Medical

Intellectual Property Rights: Medtronic

Research: Medtronic, Boston Scientific, EBR Systems

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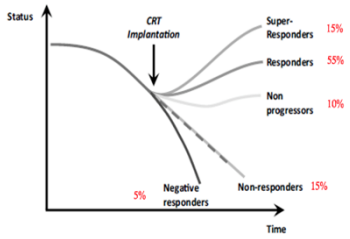
Background

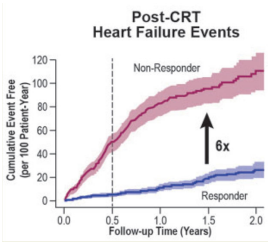
- ~ 160K CRT devices placed in US annually
- > 1M patients with CRT devices in place

≥30% of patients are NR

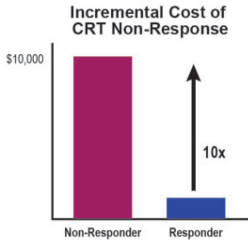


Steffel J, Ruschitzka F, Circ 2014;130:87-90

6-fold increase in HF events in NR



10-fold increase in cost in NR



Varma N, et al, Europace 2021;23:1586-95.

There is no well-accepted, proven approach to treating nonresponders (44% of NR receive no treatment)

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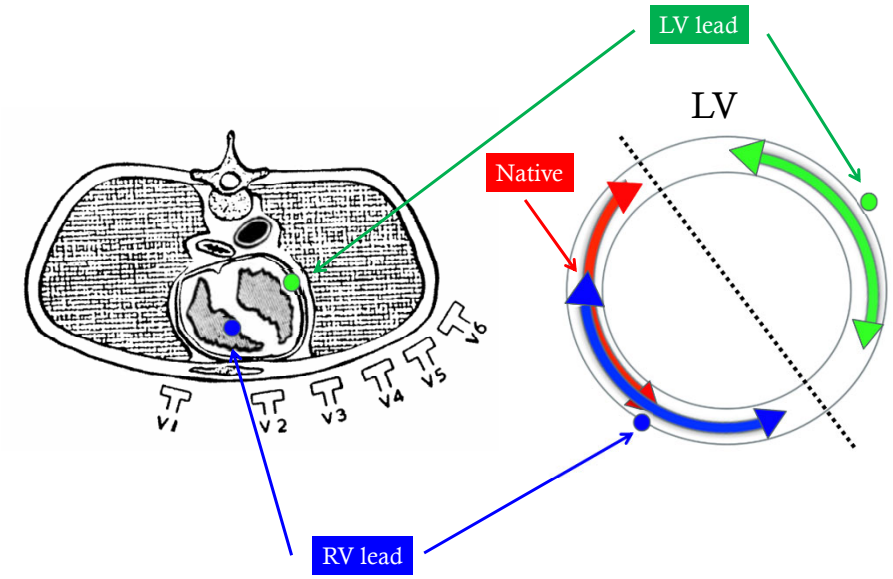
Wavefront Fusion in CRT



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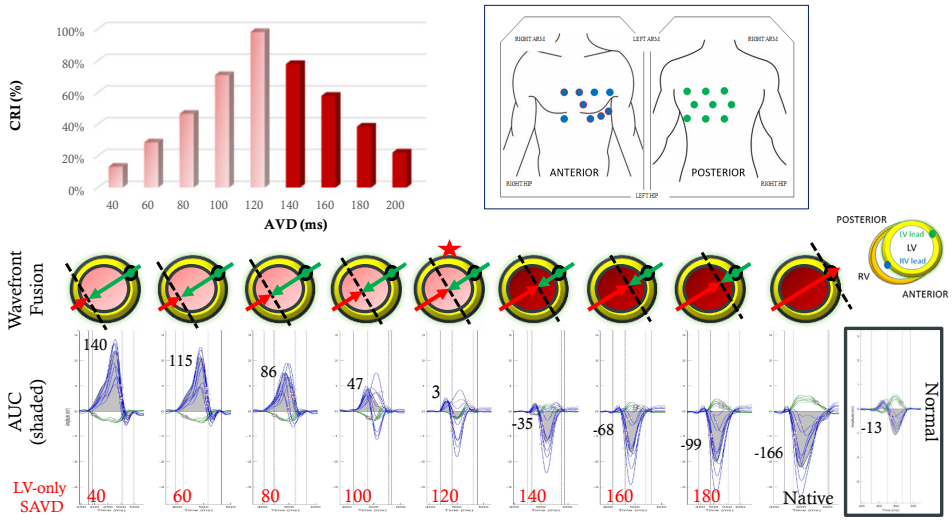
Cardiac Resynchronization Index: Multi-LEAD ECG



CRI: % change in AUC at any given setting compared to native

$$CRI = \frac{\left|AUC_{native}\right| - \left|AUC_{x}\right|}{\left|AUC_{native}\right|} \times 100\%$$

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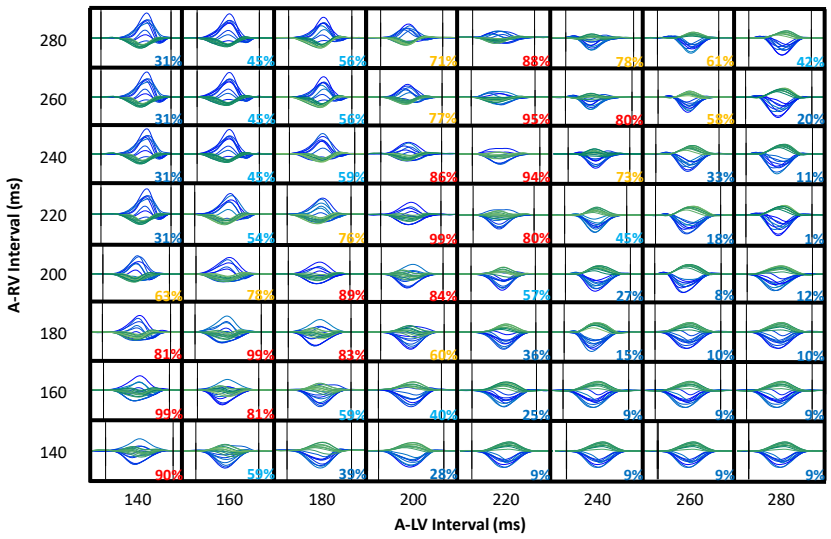

RAS

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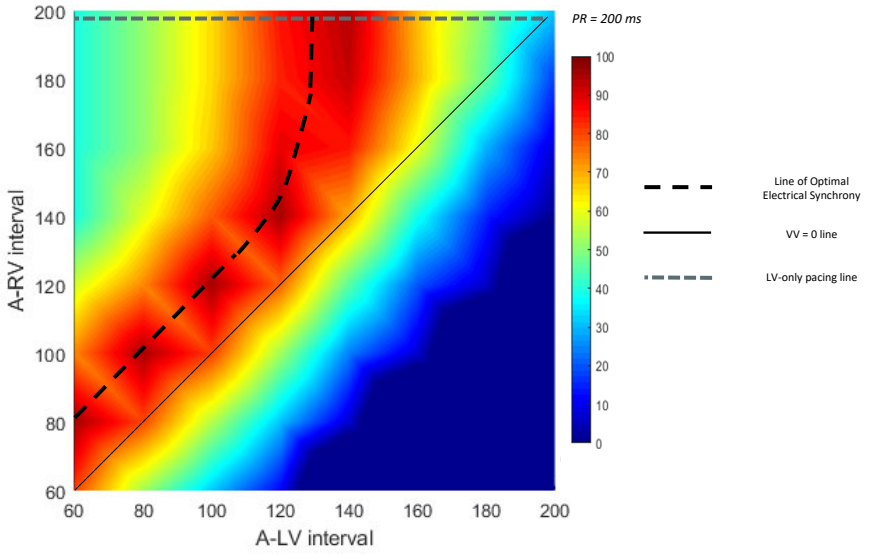
Electrical Dyssynchrony Map (EDM) in 62 y/o M with LBBB, QRSd 178 ms



HEO

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Study Design

Inclusion:
- Nonresponder (EF increase post-CRT ≤5%) OR Incomplete responder (EF increase >5% but final EF <40%)
- > 3 months post-CRT

Exclusion:
- Underlying RBBB
- Inadequate echo images for analysis
- CRT programming already optimal based on EDM (best setting ≤ 10 ms from current setting)
- CRI at best setting <80% (extremely poor lead position)
- < 90% CRT pacing (frequent PVC's or fast atrial fibrillation)

Protocol:
- Baseline echo → EDM → Program to CRI_{opt} → Repeat Echo ~6 months

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Baseline Characteristics Nonresponders to CRT (n=42)

Pre-optimization	Age	70.5 +/- 9.0
	Male n (%)	31 (74%)
	EF	31.7 +/- 4.5
	LVEDV (ml)	157 +/- 48
	LVESV (ml)	108 +/- 36
	AUC (absolute value)	51 +/- 31
	CRI (%)	50 +/- 24
	Time since implant (years)	4.1 +/- 3.9
Pre-CRT	EF	28.1 +/- 8.0
	QRS duration (ms)	159 +/- 24
	QRS morphology (LBBB/IVCD/RVp)	(23/12/7)
	AUC (native)	-110 +/- 65

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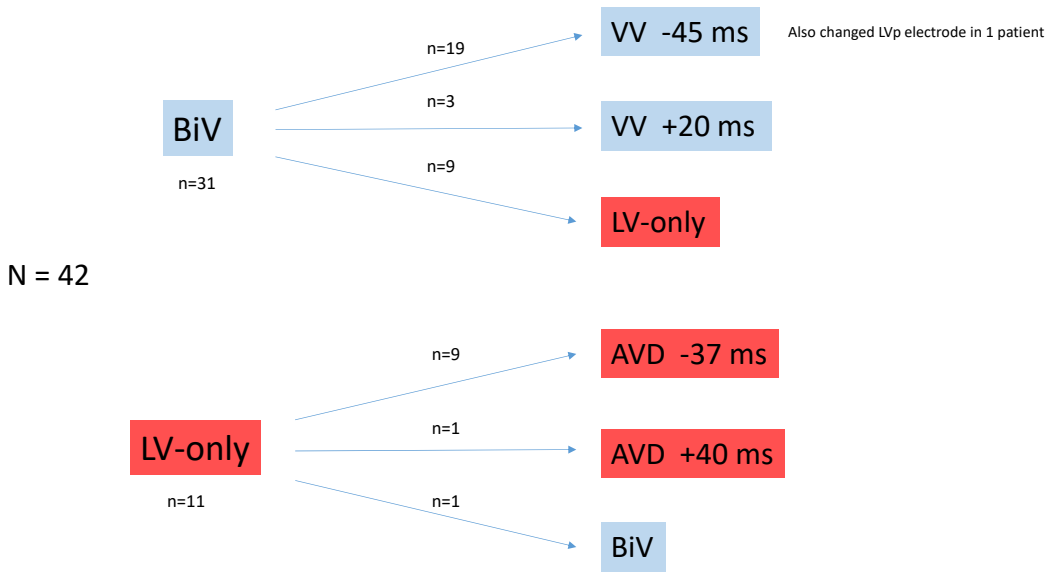
CRT Programming Pre- and Post-Optimization



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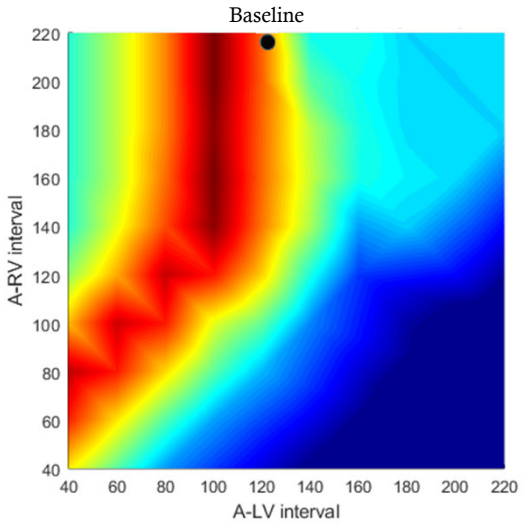
71 y/o F with NICM and non-response to CRT

- LBBB with PR 180, QRS 160
- EF 20-25% pre- and post-CRT



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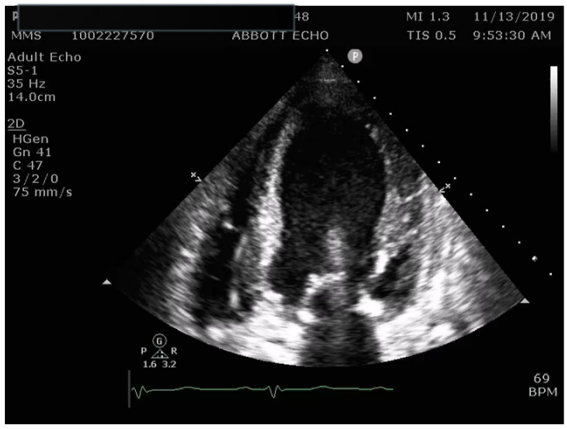
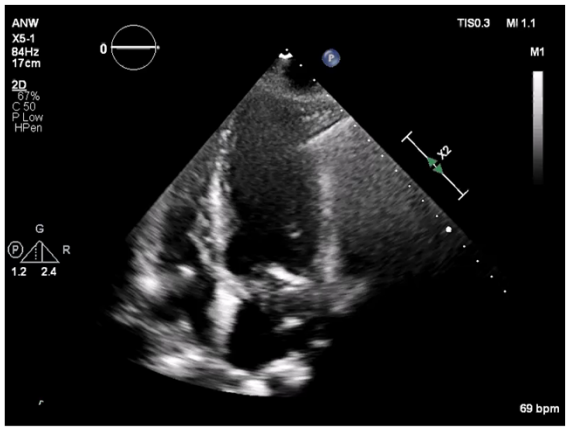


Pre-optimization
(LV-only, SAV 120 ms)



Post-optimization
(LV-only, SAV 100 ms)

AAR

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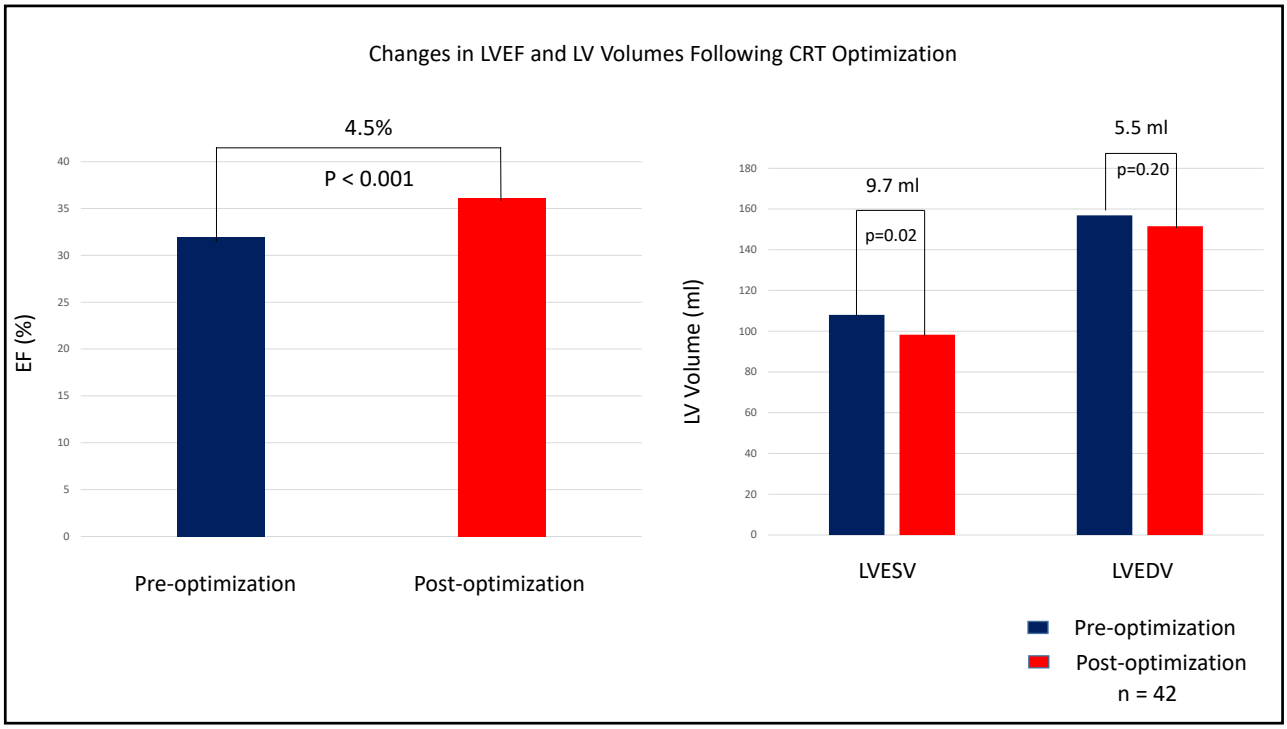
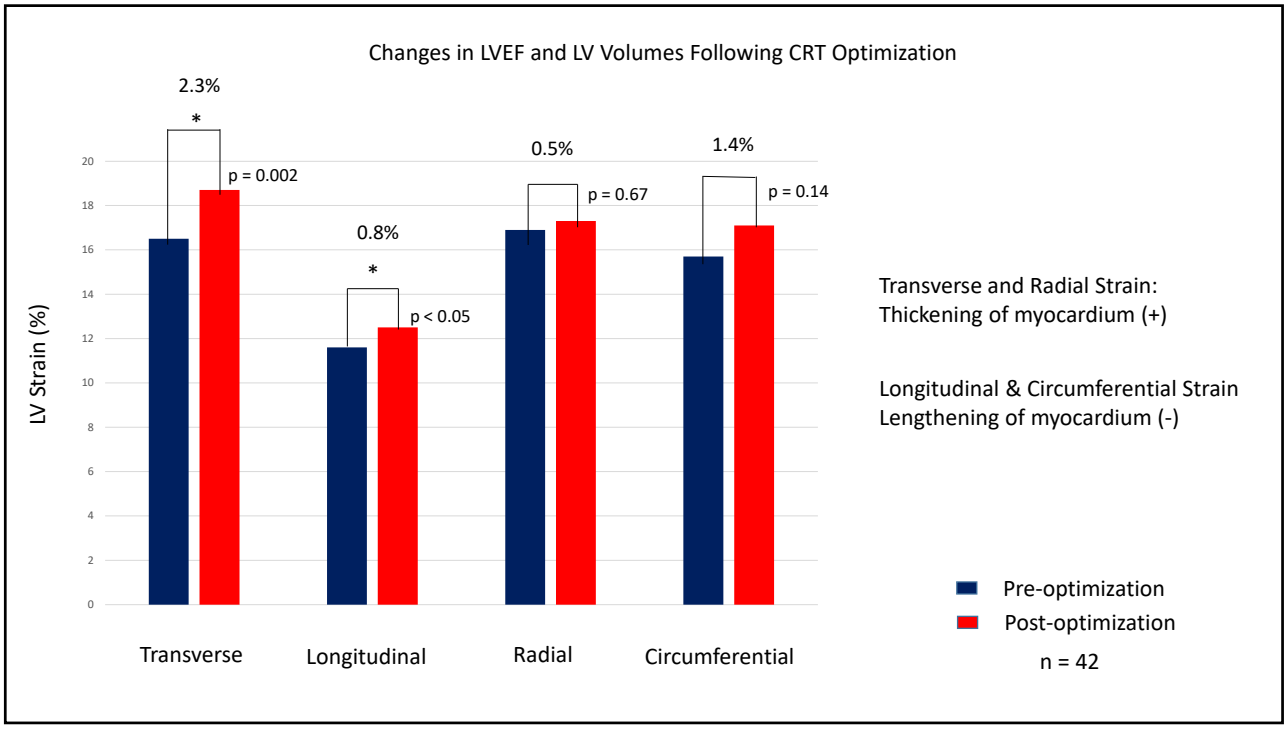

Changes in LVEF and LV Volumes Following CRT Optimization
4.5%
P < 0.001
EF (%)
Pre-optimization
Post-optimization
9.7 ml
p=0.02
5.5 ml
p=0.20
LV Volume (ml)
LVESV
LVEDV
Pre-optimization
Post-optimization
n = 42

13


Changes in LVEF and LV Volumes Following CRT Optimization
2.3%
*
p = 0.002
0.8%
*
p < 0.05
0.5%
p = 0.67
1.4%
p = 0.14
LV Strain (%)
Transverse
Longitudinal
Radial
Circumferential
Transverse and Radial Strain:
Thickening of myocardium (+)
Longitudinal & Circumferential Strain
Lengthening of myocardium (-)
Pre-optimization
Post-optimization
n = 42

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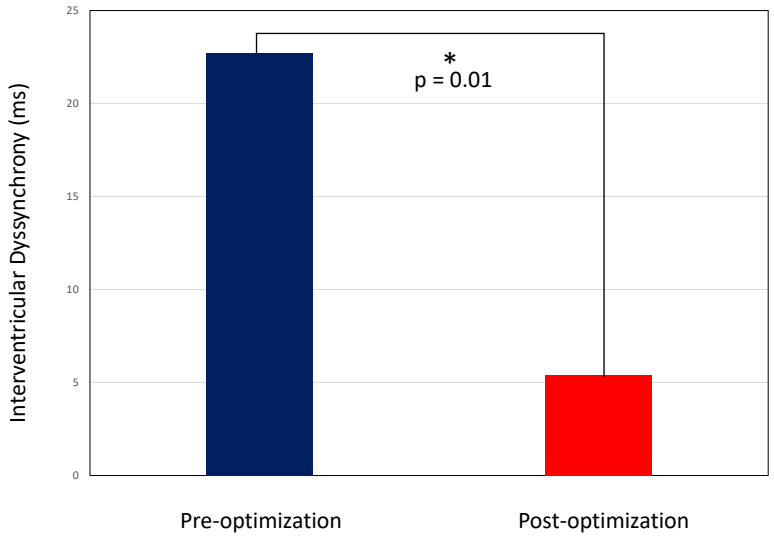
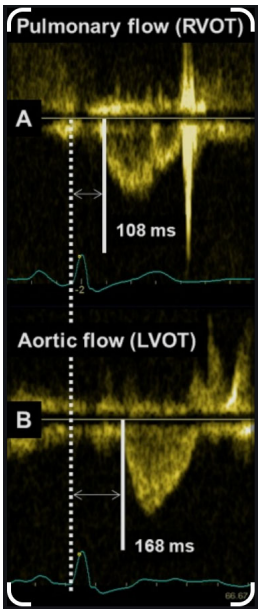
Interventricular Mechanical Dyssynchrony



Normal < 40 ms



AVO – PVO = 168 ms – 108 ms = 60 ms

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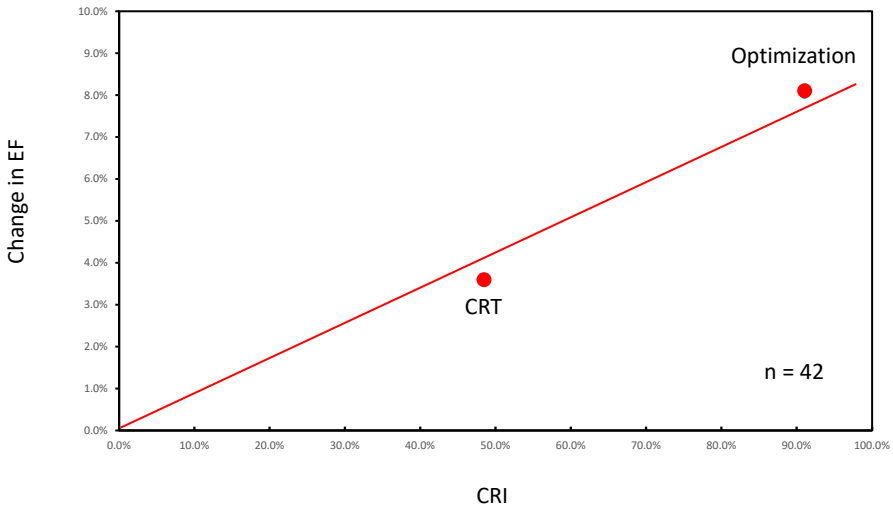
Effect of Implant and then Optimization on EF in Nonresponders and Incomplete Responders to CRT



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Potential Clinical Benefit

Reduction in HF event or death in CRT patients:

- 40% for every 5% increase in EF*
- 28% for every 10% reduction in LVESV*

130,000 CRT-D implants/yr in US x 30% nonresponder rate x 0.67 HF events per year** x 30% reduction in events

Annual reduction in HF events of about 8000

* Solomon SD et al. Effect of CRT on reverse remodeling and relation to outcome: MADIT-CRT. Circ 2010;122:985-992.
** Varma N et al. The cost of non-response to CRT: characterizing HF events following CRT. Europace 2021;23:1586-1595.

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Summary

1. CRT nonresponders and incomplete responders are 48% resynchronized at baseline settings and can be optimized using EDM (and changing AVD, VVD, pacing mode (BiV or LV-only) or LVp electrode) to 91% resynchronization

2. CRT optimization using EDM in nonresponders and incomplete responders results in significant improvements in:
   - LV systolic function (LVEF, transverse and longitudinal strain)
   - LV size (LVESV)
   - Mechanical interventricular dyssynchrony (AVO to PVO)

3. EDM is a non-invasive, practical, comprehensive (complete map of all combinations of AVD and VVD), methodology that can be used in the clinical setting to improve cardiac structure and function in nonresponders and incomplete responders to CRT

4. This approach to the treatment of nonresponders and incomplete responders to CRT could have major beneficial effects on clinical outcomes (HF hospitalization and death) and cost of care for these high-risk and high-cost patients

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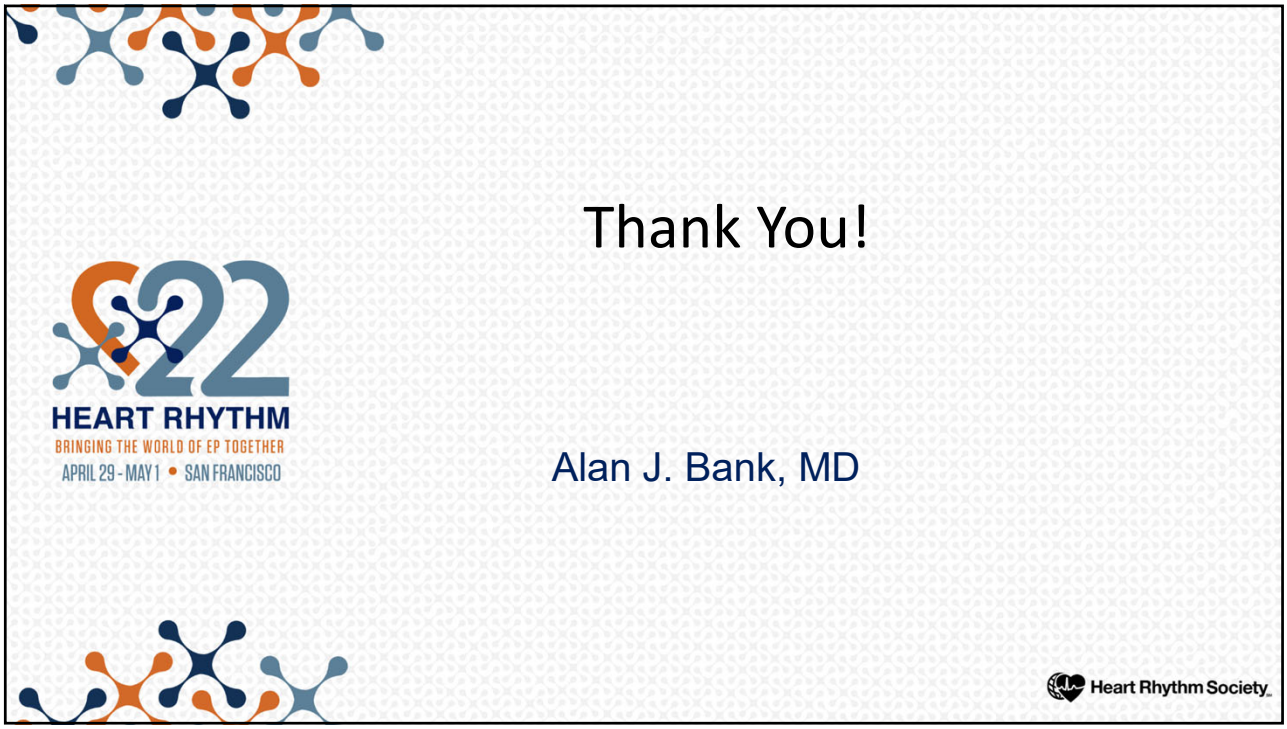
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A Systematic Review And Meta-analysis Of Clinical Outcomes Of Patients Undergoing Chronic Total Occlusion Percutaneous Coronary Intervention

March 28, 2022

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Disclosures

- I, Bahadir Simsek DO NOT have a financial interest/arrangement or affiliation with one or more organizations that could be perceived as a real or apparent conflict of interest in the context of the subject of this presentation.

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Background

- CTO PCI can improve patient symptoms; however, it remains controversial whether it impacts subsequent clinical outcomes.(1-6)
- 2021 ACC/AHA/SCAI guidelines 2b (level of evidence B).(7)

COR	LOE	RECOMMENDATION
2b	B-R	1. In patients with suitable anatomy who have refractory angina on medical therapy, after treatment of non-CTO lesions, the benefit of PCI of a CTO to improve symptoms is uncertain (1-4).

- 2018 ESC/EACTS guidelines 2a (level of evidence B)

Percutaneous revascularization of CTOs should be considered in patients with angina resistant to medical therapy or with a large area of documented ischaemia in the territory of the occluded vessel.[629,659–663]	IIa	B

4

Methods

- The study was performed according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines.
- The study was registered to The International Prospective Register of Systematic Reviews (PROSPERO).

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Methods

- A comprehensive search on: PubMed, Science Direct, Embase, Cochrane Library, and Web of Science with
- "chronic coronary occlusion" or "chronic total coronary occlusion" or "chronic total occlusion" or "total coronary artery occlusion"
- In addition, a manual search of the references of included studies was performed based on the 'snowball' method to identify any potentially relevant, but missed articles.

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Methods

We divided studies that reported clinical events* into 2 by design:

Observational studies

1. Successful versus failed CTO PCI
2. CTO PCI versus no CTO PCI (medical management only)

Randomized Controlled Trials

3. CTO PCI versus no CTO PCI

*All-cause mortality, CV mortality, MACE, MI, Stroke, HF hospitalization, Subsequent CABG, TVR

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Results

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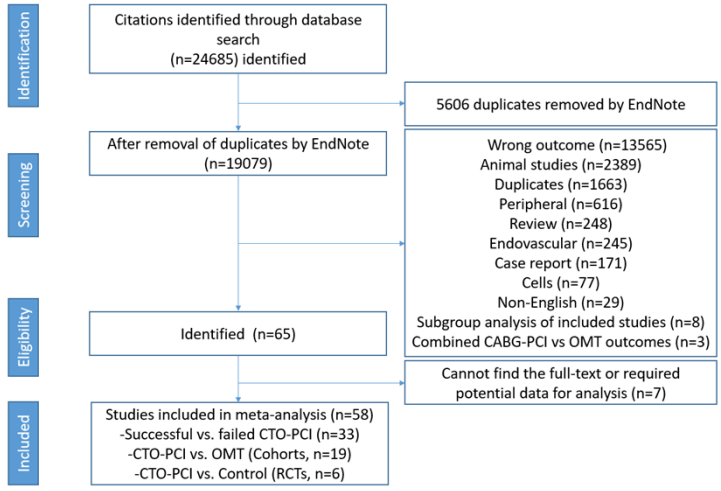
Results

- A total of 58 publications (54,540 patients) were included
- 33 were observational studies of successful vs. failed CTO PCI
- 19 were observational studies of CTO PCI vs. no CTO PCI
- 6 were RCTs



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Results

- Median f/u: 2.8 years (successful vs. failed CTO PCI)
- Median f/u: 2.6 years (CTO PCI vs. no CTO PCI)
- Median f/u: <1 year (RCTs)
- Baseline characteristics were similar between the groups

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All-cause mortality

OBSERVATIONAL

Success vs. Failed CTO PCI

A

Author, year	All-cause mortality	No	All-cause mortality	No	Odds ratios with 95% CI	Weight (%)
Byeong-Keuk Kim, 2014	48	1,997	17	506	0.72 [0.41, 1.25]	4.93
Axel de Labriolle, 2008	6	121	2	38	0.94 [0.18, 4.86]	1.33
Guoxiang Shi, 2014	11	89	12	36	0.37 [0.15, 0.92]	3.13
Zoran Olivari, 2003	3	286	3	84	0.29 [0.06, 1.48]	1.36
Renato Valenti, 2014	2	56	17	94	0.20 [0.04, 0.89]	1.53
Ji Young Park, 2018	15	306	11	90	0.40 [0.18, 0.90]	3.54
Francesco Borgia, 2012	19	218	9	56	0.54 [0.23, 1.26]	3.39
Chuan-Tsai Tsai, 2020	59	560	18	102	0.60 [0.34, 1.05]	4.89
Loes P Hoebers, 2013	75	1,151	49	516	0.69 [0.47, 1.00]	6.16
Yaling Han, 2010	280	922	45	85	0.57 [0.39, 0.84]	6.09
Erika Yamamoto, 2013	92	1,100	35	297	0.71 [0.47, 1.07]	5.93
Sudhakar George, 2014	492	9,707	259	3,981	0.78 [0.67, 0.91]	7.39
Leo Finci, 1990	5	95	3	97	1.70 [0.40, 7.32]	1.61
Jong-Pil Park, 2016	14	239	26	98	0.22 [0.11, 0.44]	4.16
Jah Yeon Choi, 2020	49	2,359	21	492	0.49 [0.29, 0.82]	5.20
Bahram Sohrabi, 2013	5	158	3	164	1.73 [0.41, 7.36]	1.63
Shahid Aziz, 2007	7	240	12	102	0.25 [0.09, 0.65]	2.91
Angela Hoye, 2005	30	537	29	275	0.53 [0.31, 0.90]	5.13
James A Suero, 2001	395	1,096	180	334	0.67 [0.54, 0.83]	7.12
Roxana Mehran, 2011	74	1,152	49	516	0.68 [0.46, 0.98]	6.15
Daniel A Jones, 2012	26	556	44	210	0.22 [0.13, 0.37]	5.26
Pil Hyung Lee, 2016	59	945	10	159	0.99 [0.50, 1.98]	4.16
Matjaž Bunc, 2016	4	121	29	59	0.07 [0.02, 0.20]	2.47
E Marc Jolicoeur, 2012	22	194	24	109	0.52 [0.28, 0.96]	4.55
Overall All-cause mortality	1792	24205	907	8500	0.52 [0.42, 0.64]	

Heterogeneity: τ^2 = 0.15, I^2 = 71.42%, H^2 = 3.50
Test of $\theta_i = \theta_j$: Q(23) = 66.23, p = 0.00
Test of θ = 0: z = -6.12, p = 0.00

Favors successful CTO-PCI | Favors failed CTO-PCI

1/32 1/8 1 2
0.52

Random-effects REML model

OBSERVATIONAL

CTO PCI vs. no CTO PCI

B

Author, year	All-cause mortality	No	All-cause mortality	No	Odds ratios with 95% CI	Weight (%)
Ugur Arslan, 2006	19	98	37	78	0.41 [0.22, 0.77]	6.67
Eun Ho Choo, 2019	45	379	83	391	0.56 [0.38, 0.83]	13.48
Eduardo J Flores-Umanzor, 2021	18	58	156	170	0.34 [0.19, 0.60]	7.76
Masashi Fujino, 2014	13	47	58	60	0.29 [0.14, 0.58]	5.40
Andrew Ladwiniec, 2015	43	362	170	497	0.35 [0.24, 0.50]	14.80
Taek Kyu Park, 2021	155	728	226	438	0.41 [0.33, 0.52]	22.56
Seung-Woon Rha, 2018	16	396	33	377	0.46 [0.25, 0.85]	6.93
Tamas Ungvari, 2011	8	36	33	58	0.39 [0.16, 0.94]	3.75
Xuhe Gong, 2021	7	293	17	246	0.35 [0.14, 0.85]	3.60
Victoria Martin Yuste, 2017	44	288	235	396	0.26 [0.18, 0.37]	15.05
Overall All-cause mortality	368	2685	1048	2711	0.38 [0.31, 0.45]	

Heterogeneity: τ^2 = 0.02, I^2 = 29.22%, H^2 = 1.41
Test of $\theta_i = \theta_j$: Q(9) = 10.34, p = 0.32
Test of θ = 0: z = -10.70, p = 0.00

Favors CTO-PCI | Favors Control

1/4 1/2 1
0.38

Random-effects REML model

RCTs

C

Author, year	All-cause mortality	No	All-cause mortality	No	Odds ratios with 95% CI	Weight (%)
Seung-Whan Lee, 2019	15	402	21	377	0.67 [0.34, 1.32]	82.18
Kambis Mashayekhi, 2018	1	100	2	102	0.51 [0.05, 5.71]	6.46
Gerald S Werner, 2018	2	257	0	137	2.67 [0.13, 56.01]	4.07
José P.S. Henriques, 2016	0	148	0	154	1.04 [0.02, 52.77]	2.45
Anton A Obedinskiy, 2018	0	39	0	33	0.85 [0.02, 43.91]	2.42
Stefan A Juricic	0	49	0	50	1.02 [0.02, 52.43]	2.43
Overall All-cause mortality	18	995	23	853	0.72 [0.39, 1.32]	

Heterogeneity: τ^2 = 0.00, I^2 = 0.00%, H^2 = 1.00
Test of $\theta_i = \theta_j$: Q(5) = 0.90, p = 0.97
Test of θ = 0: z = -1.07, p = 0.28

Favors CTO-PCI | Favors Control

1/32 1/4 1 2 16
0.72

Random-effects REML model

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Major adverse cardiovascular events

OBSERVATIONAL

Success vs. Failed CTO PCI

A

Author, year	MACE	No MACE	MACE	No MACE	Odds ratios with 95% CI	Weight (%)
Byeong-Keuk Kim, 2014	201	1,844	49	474	1.05 [0.76, 1.46]	5.05
Shao-liang Chen, 2009	19	113	7	13	0.31 [0.11, 0.88]	2.50
James Sapontis, 2017	34	558	9	79	0.53 [0.25, 1.16]	3.34
Guoxiang Shi, 2014	28	72	24	24	0.39 [0.19, 0.79]	3.55
Zoran Olivari, 2003	35	254	21	66	0.43 [0.24, 0.79]	3.98
Renato Valenti, 2008	24	320	30	112	0.28 [0.16, 0.50]	4.08
Ji Young Park, 2018	40	281	18	83	0.66 [0.36, 1.21]	3.96
Zhen Kun Yang, 2013	19	68	19	30	0.44 [0.20, 0.95]	3.36
Francesco Borgia, 2012	35	202	28	37	0.23 [0.12, 0.42]	3.96
Giampaolo Niccoli, 2012	17	179	32	89	0.26 [0.14, 0.50]	3.83
Chuan-Tsai Tsai, 2020	80	539	20	100	0.74 [0.43, 1.27]	4.26
Loes P Hoebers, 2013	157	1,069	135	430	0.47 [0.36, 0.60]	5.29
Francesca Sanguineti, 2013	167	833	76	267	0.70 [0.52, 0.95]	5.14
Yaling Han, 2010	625	577	87	43	0.54 [0.37, 0.78]	4.86
Erika Yamamoto, 2013	583	609	296	36	0.12 [0.08, 0.17]	4.93
Leo Finci, 1990	33	67	41	59	0.71 [0.40, 1.26]	4.09
Jong-Pil Park, 2016	20	233	21	103	0.42 [0.22, 0.81]	3.78
Jah Yeon Choi, 2020	227	2,181	50	463	0.96 [0.70, 1.33]	5.08
Bahram Sohrabi, 2013	49	114	73	94	0.55 [0.35, 0.87]	4.58
Angela Hoye, 2005	206	361	177	127	0.41 [0.31, 0.54]	5.20
J Aaron Grantham, 2010	3	66	8	48	0.27 [0.07, 1.08]	1.75
Dariusz Ciećwierz, 2013	21	117	41	97	0.42 [0.24, 0.77]	4.03
Pil Hyung Lee, 2016	103	901	43	126	0.33 [0.22, 0.50]	4.78
E Marc Jolicoeur, 2012	76	137	56	77	0.76 [0.49, 1.19]	4.62
Overall MACE	2802	11695	1361	3077	0.46 [0.37, 0.58]	

Heterogeneity: τ^2 = 0.22, I^2 = 80.44%, H^2 = 5.11
Test of $\theta_i = \theta_j$: Q(23) = 133.06, p = 0.00
Test of θ = 0: z = -6.89, p = 0.00

Favors successful CTO-PCI | Favors failed CTO-PCI

1/8 1/4 1
0.46

Random-effects REML model

OBSERVATIONAL

CTO PCI vs. no CTO PCI

B

Author, year	MACE	No MACE	MACE	No MACE	Odds ratios with 95% CI	Weight (%)
Ugur Arslan, 2006	74	43	96	19	0.34 [0.18, 0.63]	9.72
Eun Ho Choo, 2019	120	304	141	333	0.93 [0.70, 1.24]	14.00
Masashi Fujino, 2014	44	16	103	15	0.40 [0.18, 0.88]	7.83
Lei Guo, 2021	185	978	307	906	0.56 [0.46, 0.68]	14.92
Ji-won Hwang, 2016	16	131	41	247	0.74 [0.40, 1.36]	9.77
Seung-Woon Rha, 2018	88	324	82	328	1.09 [0.78, 1.52]	13.40
Hsin-I Teng, 2018	18	49	16	23	0.53 [0.23, 1.22]	7.36
Salvatore D Tomasello, 2015	20	756	68	758	0.29 [0.18, 0.49]	11.14
Xuhe Gong, 2021	36	264	58	205	0.48 [0.31, 0.76]	11.86
Overall MACE	601	2865	912	2834	0.57 [0.42, 0.78]	

Heterogeneity: τ^2 = 0.16, I^2 = 78.19%, H^2 = 4.59
Test of $\theta_i = \theta_j$: Q(8) = 33.79, p = 0.00
Test of θ = 0: z = -3.56, p = 0.00

Favors CTO-PCI | Favors No CTO-PCI

1/4 1/2 1
0.57

Random-effects REML model

RCTs

C

Author, year	MACE	No MACE	MACE	No MACE	Odds ratios with 95% CI	Weight (%)
Seung-Whan Lee, 2019	86	331	82	316	1.00 [0.71, 1.41]	46.65
Kambis Mashayekhi, 2018	6	95	17	87	0.32 [0.12, 0.86]	22.07
Gerald S Werner, 2018	13	246	9	128	0.75 [0.31, 1.81]	24.95
José P.S. Henriques, 2016	0	148	0	154	1.04 [0.02, 52.77]	2.18
Anton A Obedinskiy, 2018	2	37	0	0	0.07 [0.00, 4.12]	1.98
Stefan A Juricic, 2021	0	49	0	50	1.02 [0.02, 52.43]	2.17
Overall MACE	107	906	108	735	0.69 [0.38, 1.25]	

Heterogeneity: τ^2 = 0.17, I^2 = 35.10%, H^2 = 1.54
Test of $\theta_i = \theta_j$: Q(5) = 6.22, p = 0.29
Test of θ = 0: z = -1.23, p = 0.22

Favors CTO-PCI | Favors Control

0.10 1 8
0.69

Random-effects REML model

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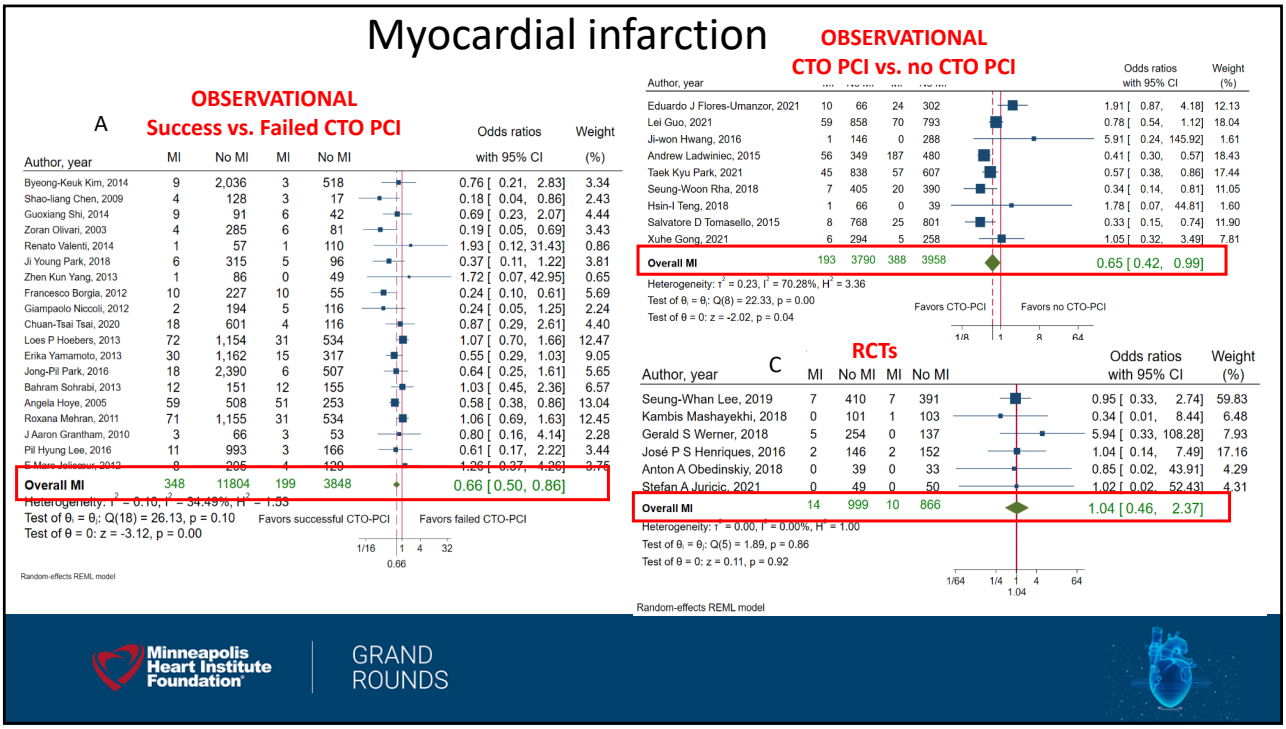
Myocardial infarction



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Results

- Similarly, observational studies showed significantly better outcomes favoring successful CTO PCI or CTO PCI over failed CTO PCI or no CTO PCI for
- CV mortality
- Subsequent CABG
- Heart failure hospitalizations
- Target vessel revascularization

- However, RCTs did not demonstrate any benefit favoring CTO PCI over no CTO PCI.

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Discussion/Limitations

Observational studies

- Selection bias/confounding

RCTs

- The two largest (EuroCTO and DECISION-CTO) stopped enrollment prematurely
- Low event rates
- Mild or no symptoms in patients (14%, DECISION-CTO)
- Crossover (20%, DECISION-CTO)

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Conclusion

- Current observational studies show an association of CTO PCI with better subsequent clinical outcomes whereas RCTs do not.
- Additional data from RCTs are needed to determine the impact of CTO PCI on 'hard' clinical events (ISCHEMIA-CTO, NOBLE-CTO).

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Evaluation of the OPEN-CLEAN Chronic Total Occlusion Percutaneous Coronary Intervention Perforation Score in the PROGRESS-CTO Registry

March 28, 2022

Spyridon Kostantinis, MD
(on the behalf of the PROGRESS-CTO investigators)

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Disclosure Statement of Financial Interest

I, Spyridon Kostantinis DO NOT have a financial interest/arrangement or affiliation with one or more organizations that could be perceived as a real or apparent conflict of interest in the context of the subject of this presentation.

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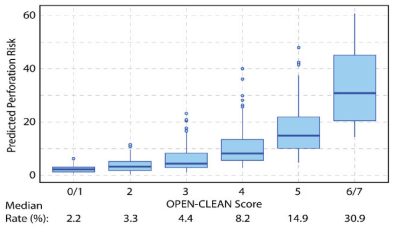
Background

- Coronary artery perforation is a feared complication of chronic total occlusion (CTO) percutaneous coronary intervention (PCI).
- The **OPEN-CLEAN CTO** (**O**utcomes, **P**atient health status, and **E**fficiency i**N** **CTO** hybrid procedures-**C**ABG, **L**ength of occlusion, **E**F<50%, **A**ge, severe calcificatio**N**) perforation score[1] is computed from 5 parameters:

Variables	Points
CABG	1
Occlusion length	
20 to <60 mm	1
≥60 mm	2
Ejection fraction <50%	1
Age	
50 to <70	1
≥70	2
CalcificatioN	1



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3

Goal

- We sought to evaluate the performance of the OPEN-CLEAN score in the **PROGRESS-CTO** (**PRO**spective **G**lobal **RE**gi**S**try for the **S**tudy of **C**hronic **T**otal **O**cclusion Intervention) registry.

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4

Methods

- The performance of the OPEN-CLEAN score in CTO PCI was assessed using complete data from the PROGRESS-CTO registry of 6,422 patients who underwent the procedure between 2012 and 2021 at 38 US and international centers.
- The OPEN-CLEAN score was calculated for each patient.

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Statistical analysis

- Categorical variables are presented as percentages and compared using Pearson's chi-square test or Fisher's exact test. Continuous variables are presented as mean ± SD or as median (interquartile range [IQR]) and compared using the Student's t-test and the Wilcoxon rank sum test. A 2-sided p value of 0.05 was considered to indicate statistical significance.
- The risk of perforation as a function of the OPEN-CLEAN score was estimated from a robust Poisson regression with a canonical log-link and sandwich variance estimator to allow for overdispersion in the data. The model fit was assessed using the c-statistic and its nonparametric bootstrap confidence interval.

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Results

- Mean age of patients in the study was 64±10 years, most patients were men (81%), and 60% had a prior PCI.
- The right coronary artery was the most common target vessel (52%).
- Technical and procedural success was 87% and 85%, respectively.
- The incidence of major adverse cardiac events was 2.1% (n=135) whereas that of perforation was 5.1% (n=326).

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Table 1. Baseline clinical characteristics

Variable	Overall	Perforation	No perforation	*P* value
	n=6,422	n=326, 5.1%	n=6,096, 94.9%	
Clinical characteristics				
Age (years)	64±10	68±9	64±10	0.0001
Men	81%	79%	81%	0.488
BMI (kg/m2)	31±8	29±5	31±8	0.001
Hypertension	89%	94%	89%	0.003
Diabetes	42%	36%	42%	0.032
Dyslipidemia	85%	84%	85%	0.685
Prior MI	45%	52%	45%	0.031
Prior CABG	28%	37%	27%	0.0002
Prior PCI	60%	69%	59%	0.0004
LVEF (%)	51±12	50±13	51±12	0.505

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BMI = Body Mass Index; MI = Myocardial Infarction; CABG = Coronary Artery Bypass Grafting; PCI = Percutaneous Coronary Intervention; LVEF = Left Ventricular Ejection Fraction.

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Table 2. Angiographic characteristics

Variable	Overall n=6,422	Perforation n=326	No perforation n=6,096	*P* value
Angiographic characteristics				
CTO Target Vessel				0.076
▪ RCA	52%	59%	52%	
▪ LAD	27%	22%	27%	
▪ LCX	20%	17%	20%	
Proximal cap ambiguity	35%	53%	33%	<0.0001
Occlusion length (mm)	29±18	32±19	29±18	0.003
Severe calcification	18%	29%	17%	<0.0001
In-stent restenosis	16%	16%	16%	0.751

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CTO = Chronic Total Occlusion; RCA = right coronary artery; LAD = left anterior descending; LCX = left circumflex.

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Table 3. Procedural characteristics

Variable	Overall n=6,422	Perforation n=326	No perforation n=6,096	*P* value
Procedural characteristics				
Successful Crossing Strategy				<0.0001
▪ AWE	56%	24%	58%	
▪ Retrograde	18%	29%	18%	
▪ ADR	12%	13%	12%	
▪ None	13%	34%	12%	
IVUS used	43%	48%	43%	0.104
Procedure time (min)	115 (75, 170)	177 (125, 237)	112 (73, 165)	<0.0001
Fluoroscopy time (min)	42 (26, 68)	71 (50, 101)	41 (25, 66)	<0.0001
Radiation dose (air kerma, mGy)	2.2 (1.3, 3.7)	3.0 (1.9, 4.8)	2.1 (1.2, 3.6)	<0.0001
Contrast volume (ml)	205 (150, 300)	250 (175, 350)	200 (150, 290)	<0.0001

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AWE = antegrade wire escalation; ADR = antegrade dissection and re-entry; IVUS = Intravascular ultrasound; Gy = Gray

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Table 4. Procedural outcomes

Variable	Overall	Perforation	No perforation	*P* value
	n=6,422	n=326	n=6,096	
Procedural outcomes				
Technical Success	87%	66%	88%	<0.0001
Procedural Success	85%	55%	87%	<0.0001
MACE	2.1%	17.8%	1.3%	<0.0001
Death	0.5%	4%	0.3%	<0.0001
MI	0.6%	1.8%	0.6%	0.005
Emergency CABG	0.1%	0.9%	0.03%	0.0001
Re-PCI	0.2%	1.2%	0.2%	0.0001
Stroke	0.2%	0.6%	0.2%	0.051
Pericardiocentesis	1%	13%	0.3%	<0.0001

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MACE = Major Adverse Cardiovascular Events; MI = Myocardial Infarction; CABG = Coronary Artery Bypass Grafting; PCI = Percutaneous Coronary Intervention.

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Figure 1. Incidence of perforations for each value of the OPEN-CLEAN score

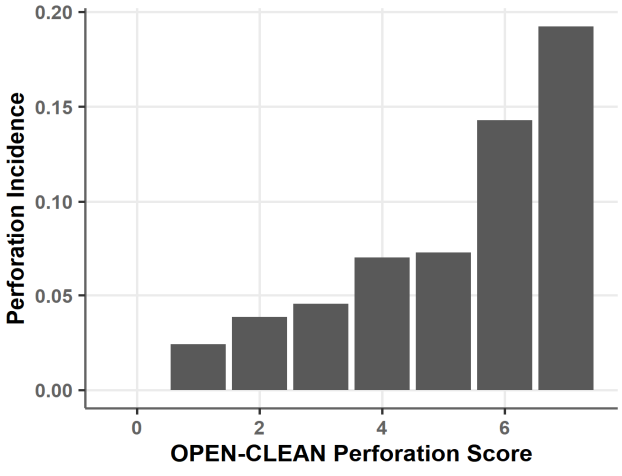

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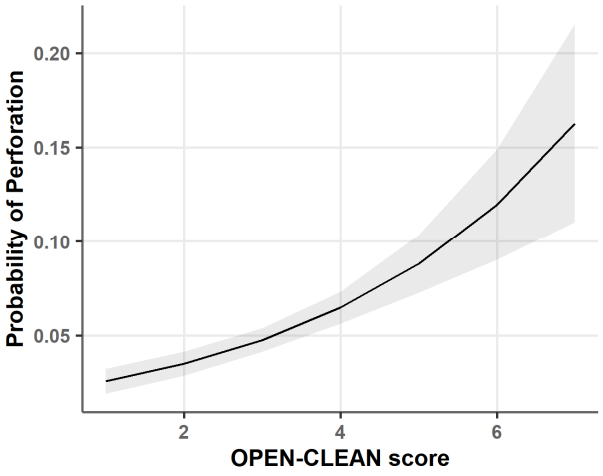
Figure 2. Probability of perforation stratified by OPEN-CLEAN score



- The estimated increase in the risk of perforation with one score unit increase was 36% (95% confidence interval [CI], 25%-48%; p<0.001).
- The c-statistic was 0.62 (95% CI, 0.59-0.65).

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Main findings

- In an independent cohort of patients, the OPEN-CLEAN perforation score was positively associated with the risk of perforation during CTO PCI.
- However, the estimated c-statistic of 0.62 was lower than 0.75 reported in Hirai et al.
- Given the association with the risk of perforation observed in this study and ease of calculation, the OPEN-CLEAN perforation score may be useful for quantifying the risk/benefit ratio for each individual patient.

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Limitations

- Observational study without adjudication of clinical events by an independent committee
- Quantitative coronary angiographic analyses were not performed.
- CTO PCIs in the PROGRESS-CTO registry are performed at dedicated, high-volume CTO centers with experienced operators, limiting the generalizability of the findings to centers with limited CTO PCI experience.

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Conclusion

- The OPEN-CLEAN CTO perforation score is a valuable tool for estimating the risk of perforation during CTO PCI and can be useful for quantifying the risk/benefit ratio for each individual patient.

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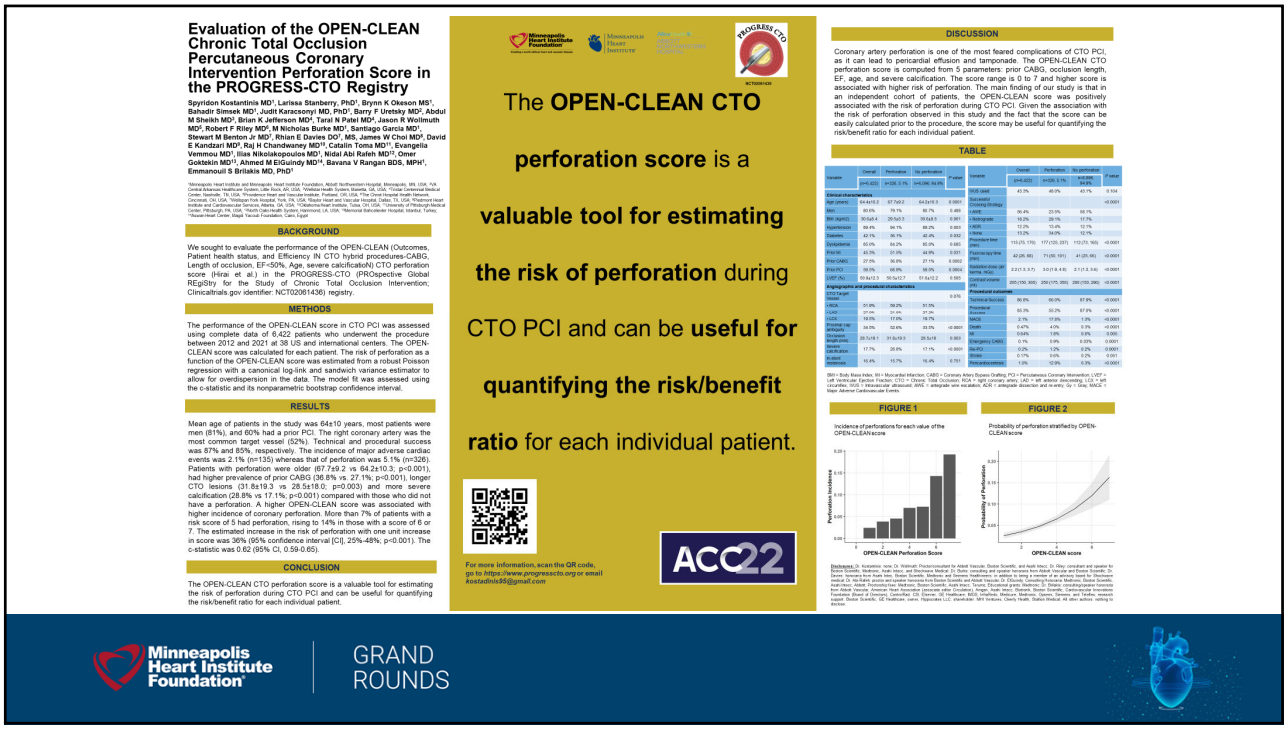

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Thank you!

Spyridon Kostantinis, MD
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MHIF
WOMEN'S
Penny Anderson Women's Cardiovascular Center

STRESS MYOCARDIAL BLOOD FLOW IS ABNORMAL DURING ACUTE TAKOTSUBO SYNDROME

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1

BACKGROUND

- Coronary microvascular dysfunction (CMD) is a proposed mechanism for takotsubo syndrome (TS).
- To date, it is unknown whether changes in stress myocardial blood flow (MBF) exist during acute TS presentation, their associated mechanisms and the natural course of such abnormalities.
- In a proof-of-concept mechanistic study, cardiac magnetic resonance imaging (CMR) with T2 mapping and quantitative stress perfusion were used to comprehensively evaluate during the acute TS presentation and at 6-months later.

METHODS

- Between March and September 2021, patients who met the criteria for acute TS with mid-apical regional wall motion abnormalities (WMA) and no significant coronary artery obstruction were approached to participate.
- After informed consent, patients underwent comprehensive Adenosine stress CMR study with cine imaging, T2-mapping, quantitative perfusion with automated in-line quantification of global and regional MBF, in addition to late gadolinium enhancement (LGE) imaging. Same protocol was repeated at 6-months from initial CMR exam to assess for interval changes.

RESULTS

- A total of 4 patients were so far recruited. All women, age range 49-77 years, with emotional trigger and mild troponin elevation (**Table 1**).
- CMR was performed within 3±SD (or median, IQR) days from presentation. Left ventricular ejection fraction was 30-45% with regional myocardial edema (**Figure 1**) and hypokinesis or akinesis in the mid-apical segments (**Figure 2**). LGE was absent in all patients.
- In the acute TS setting, although global rest (>1.0 ml/min/g) and global stress MBF (> 2.0 ml/min/g) were normal, apical stress MBF was abnormal with a base/apex ratio ≥ 1.5 (normal=1.0) (**Table 1**) (**Figure 3**)
- Repeat CMR at 6 months from acute presentation in 3 out of 4 patients, has so far demonstrated resolution of myocardial edema, normalization of LV contractility and of stress MBF (**Figures 1, 2, 3**).

Table 1 – Baseline Clinical and Imaging Characteristics

	Patient 1	Patient 2	Patient 3	Patient 4
Sex	Female	Female	Female	Female
Age	68	49	77	67
Peak troponin (pg/mL)	1.998	11.097	4.257	0.228
Ballooning Type	Apical	Apical	Apical	Apical
Left Ventricular EF % (transthoracic echo)	44	45	34	30
Type of TS stressor	Emotional	Emotional	Emotional	Emotional
Acute Stress Global MBF (>2.0)	**2.34**	**2.23**	**2.23**	**2.25**
Acute Rest Global MBF (0.8-1.0)	**1.30**	**0.80**	**1.20**	**0.98**
Acute Global Myocardial perfusion Reserve (MPR) (>2.4)	**1.80**	**2.79**	**1.86**	**2.30**
Acute Basal Stress MBF	3.00	2.35	2.10	2.82
Acute Apical Stress MBF	2.00	1.48	1.40	1.70
Acute Stress Basal/Apex Ratio	**1.50**	**1.59**	**1.50**	**1.66**
Acute Rest Basal MBF	1.50	0.97	1.30	0.95
Acute Rest Apical MBF	1.30	0.80	0.90	0.81
Acute Rest Basal/Apical Ratio	**1.15**	**1.21**	**1.44**	**1.17**
Recovery Stress Global MBF (>2.0)	**3.52**	**2.85**	**3.62**	
Recovery Rest Global MBF (0.8-1.0)	**1.04**	**0.70**	**1.30**	
Recovery Global MPR (>2.4)	**3.38**	**4.07**	**2.78**	
Recovery Basal Stress MBF	3.50	2.50	4.15	
Recovery Apical Stress MBF	3.20	2.50	3.23	
Recovery Stress Basal/Apex Ratio	**1.09**	**1.00**	**1.28**	
Recovery Rest Basal MBF	1.02	0.70	1.20	
Recovery Rest Apical MBF	1.05	0.70	1.20	
Recovery Rest Basal/Apical Ratio	**0.97**	**1.00**	**1.00**	

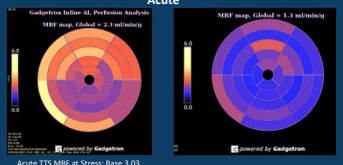
STRESS MYOCARDIAL BLOOD FLOW IS ABNORMAL DURING ACUTE TAKOTSUBO SYNDROME

Retu Saxena[1,2], João L. Cavalcante[1,2], Dawn R. Witt[2], Stephanie Zinken Ebnet[2], Gretchen Benson[2], Ross Garberich[2], Opema Lohese[2], Derek Vang[2], Peter Kellman[3], Hue Xu[3], Scott Sharkey[2]

(1) Minneapolis Heart Institute at Abbott Northwestern Hospital, Minneapolis, MN
(2) Minneapolis Heart Institute Foundation, Minneapolis, MN
(3) NIH/NHLBI, Bethesda, MD

Figure 3

Acute



Acute TTS MBF at Stress: Base 3.03, Apex 2.1, Ratio 1.5

Normal Stress MBF > 2.0 ml/min/g

Acute TTS MBF at Rest =1.3 ml/min/g

Normal Rest MBF 0.8-1.0

Recovery

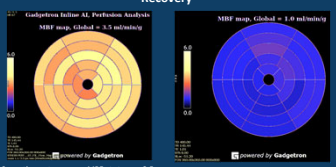


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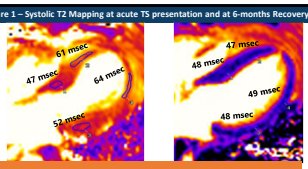
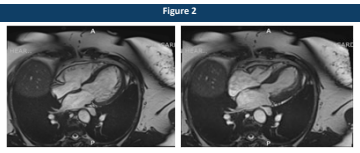
Figure 1 – Systolic T2 Mapping at acute TS presentation and at 6-months Recovery



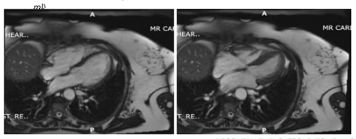
Normal myocardial T2 mapping < 55 msec

Figure 2



ACUTE END DIASTOLE (EDV 194 ml)

ACUTE END SYSTOLE (ESV 87 ml)



RECOVERY END DIASTOLE (EDV 180 ml)

RECOVERY END SYSTOLE (ESV 74 ml)

CONCLUSIONS

During acute TS, rest MBF is normal in all segments despite abnormal mid-apical contraction, regional MBF response to adenosine stress is decreased in abnormally contracting LV segments. Acute global MPR is reduced.

This abnormal response may reflect intrinsic CMD or the consequence of regional myocardial edema.

These acute findings are rather transient which may reflect circumstantial CMD as these findings resolved at 6 months, along with normalization of myocardial edema and myocardial contractility

Stress CMR may offer insights into the mechanism for TS

2

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4

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6

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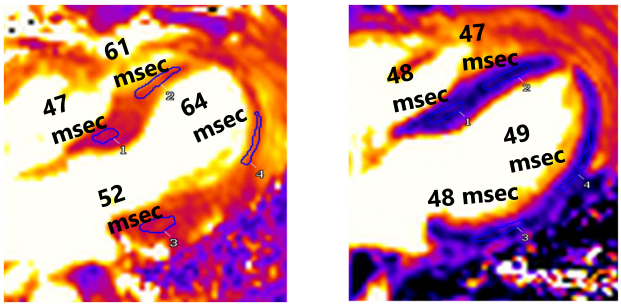

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Figure 2

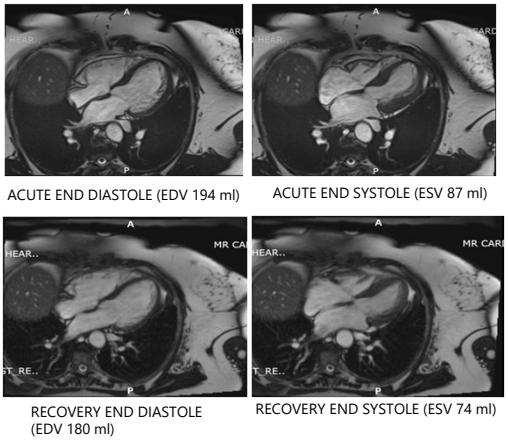

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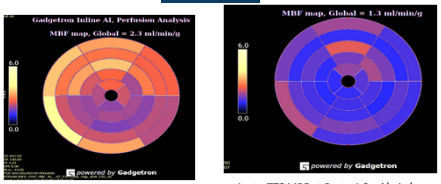
Figure 3

Acute



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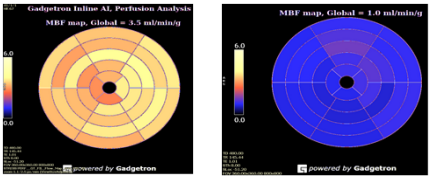
Recovery



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Conclusions

During acute TS, rest MBF is normal in all segments despite abnormal mid-apical contraction, regional MBF response to adenosine stress is decreased in abnormally contracting LV segments. Acute global MPR is reduced.

This abnormal response may reflect intrinsic CMD or the consequence of regional myocardial edema.

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Stress CMR may offer insights into the mechanism for TS

8

Deformation of Self-expanding Transcatheter Aortic Valve Prostheses for Hypo-Attenuating Leaflet Thickening

Miho Fukui, Vinayak N. Bapat, Santiago Garcia, Hirotomo Sato, Maurice Enriquez-Sarano, John R. Lesser, João L. Cavalcante, and Paul Sorajja
Minneapolis Heart Institute Foundation, Minneapolis, Minnesota, USA

BACKGROUND

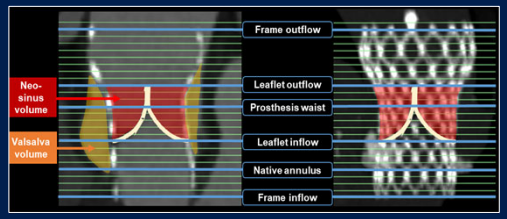
- Mechanistic insight into causative factors for HALT in TAVR prostheses remains limited

STUDY AIM

- To determine relation between prostheses deformity in self-expanding TAVR valves and occurrence of HALT

METHODS

- 213 native AS patients prospectively examined with cardiac CTA screening for HALT 30 days after TAVR
- Study exclusions were valve-in-valve procedure, inadequate image quality for transcatheter heart valve (THV) or CT unavailable due to death or risk of CIN .



- ***Eccentricity*** = $\sqrt{1-\frac{(minor\ diameter)2}{(major\ diameter)2}}$
- ***Asymmetric leaflet expansion*** = sum of the difference between 120º and each angle formed by each prosthetic leaflet
- ***Neo-sinus volume*** = the volume above the THV leaflets within the THV frame.

RESULTS

Baseline Characteristics

Clinical Characteristics	HALT (n=35)	No HALT(n=178)	*p*
Age (years)	84 (80-87)	82 (77-87)	0.25
Male – no. (%)	17 (49)	90 (51)	0.83
Diabetes mellitus – no. (%)	4 (11)	55 (31)	0.02
Hypertension – no. (%)	29 (83)	154 (87)	0.58
Atrial fibrillation/flutter – no. (%)	8 (23)	57 (32)	0.28
Coronary artery disease – no. (%)	20 (57)	88 (49)	0.41
eGFR (mL/min/1.73 m²)	59 (48-72)	67 (54-75)	0.11
LVEF (%)	60 (55-65)	60 (55-65)	0.90
Aortic valve area index (cm²/m²)	0.42 (0.36-0.48)	0.39 (0.33-0.46)	0.19
STS-PROM score (%)	3.9 (2.8-5.5)	3.2 (1.9-5.3)	0.13
TAVR procedure			
Transfemoral Access – no. (%)	33 (94)	167 (94)	0.92
Pre-dilatation – no. (%)	11 (31)	43 (24)	0.37
Post-dilatation – no. (%)	1 (3)	6 (3)	0.88
Medications at discharge			
Antiplatelet therapy – no. (%)	34 (97)	176 (99)	0.43
Anticoagulant therapy – no. (%)	**6 (17)**	**64 (36)**	**0.03**

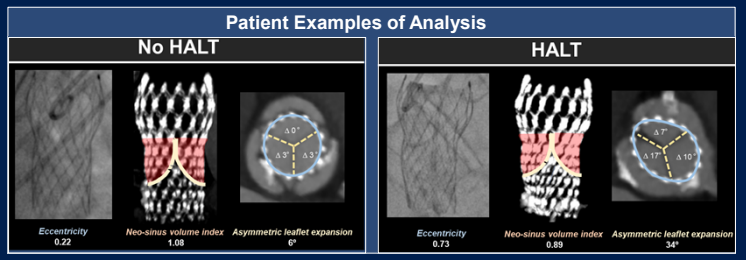
Cardiac CTA Characteristics

	HALT (n=35)	No HALT (n=178)	*p*
Baseline variables			
Bicuspid aortic valve – no. (%)	2 (6)	3 (2)	0.15
Aortic valve calcium score (AU)	1828 (1462-3181)	2324 (1557-3258)	0.44
Post-procedural variables			
Eccentricity index at			
Frame outflow	0.31 (0.18-0.42)	0.30 (0.22-0.37)	0.52
Leaflet outflow	0.29 (0.20-0.38)	0.26 (0.19-0.31)	0.05
Prosthesis waist	**0.38 (0.32-0.44)**	**0.31 (0.23-0.43)**	**0.008**
Leaflet inflow	**0.54 (0.49-0.61)**	**0.42 (0.35-0.55)**	**<0.001**
Native annulus	**0.55 (0.47-0.64)**	**0.49 (0.37-0.59)**	**0.03**
Frame inflow	0.56 (0.47-0.65)	0.51 (0.38-0.60)	0.06
Asymmetric leaflet expansion	**18 (10-24)**	**10 (6-14)**	**<0.001**
Neo-sinus volume index	**0.98 (0.94-1.00)**	**1.01 (0.98-1.04)**	**<0.001**
Implant depth (mm)	5.0 (4.0-6.6)	5.3 (3.6-7.1)	0.84
Canting (mm)	2.3 (1.5-4.0)	2.3 (1.3-3.7)	0.93
Commissure malalignment			
Right coronary – no. (%)	6 (17)	49 (28)	0.20
Left coronary – no. (%)	8 (23)	45 (25)	0.76
LVEF by CT (%)	63 (42-67)	58 (50-65)	0.13
LVSVI by CT (ml/m²)	45 (34-50)	47 (39-52)	0.43

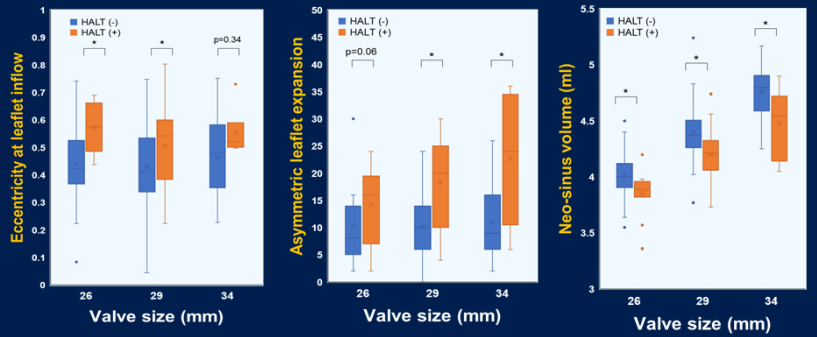
Multivariable Regression Analysis for HALT

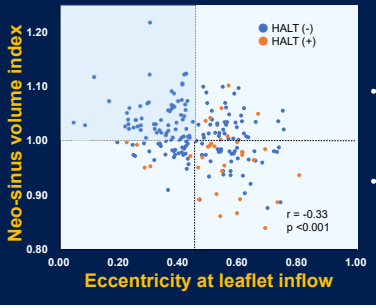
	Model 1		Model 2	
	OR (95% CI)	*p*	OR (95% CI)	*p*
Anticoagulant therapy at discharge	0.18 (0.05-0.65)	0.009		
Valve size (26-, 29-, 34 -mm)			0.46 (0.24-0.85)	0.01
Eccentricity at leaflet inflow (>0.44)	**3.74 (1.25-11.1)**	**0.02**	**4.23 (1.44-12.5)**	**0.009**
Asymmetric leaflet expansion (per 1-degree)	**1.15 (1.07-1.23)**	**<0.001**	**1.13 (1.06-1.20)**	**<0.001**
Neo-sinus volume index (per 0.1)	**0.31 (0.13-0.75)**	**0.01**	**0.32 (0.13-0.76)**	**0.01**

Patient Examples of Analysis



TAVR Prosthesis Deformation and HALT





CONCLUSIONS

- TAVR prosthesis deformation (i.e. eccentricity, asymmetric leaflet expansion, neo-sinus volume) might explain HALT occurrence following self-expanding TAVR prosthesis.
- These data may have implications for both design and deployment techniques to improve clinical outcomes with TAVR.

<Disclosures> The authors have no disclosures related to this study to report.