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Within the past 12 months, I or my spouse/partner have had a financial interest/arrangement or affiliation with the organization(s) listed below.

Affiliation/Financial Relationship

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<u>ILOs</u>

- Learn why non-compaction is not accurate and should be replaced with excessive trabeculation.

-Definition, Epidemiology and clinical picture.

-The prognosis of Excessive trabeculation in different populations.

-Suggested definition for Excessive Trabeculation.

-Algorithm for management of patients with Excessive trabeculations.















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	ronow-up of 54 Adults		N 1 (
With Isolated A Distinct C	l Left Ventricular Noncompaction: ardiomyopathy With Poor Prognosis		Number of Patients
Erwin N. Oechslin, M	D, Christine H. Attenhofer Jost, MD, Jerry R. Rojas, MD,	Heart failure requiring hospitalization	18 (53%)
Philipp A. Kaufmann,	MD, Rolf Jenni, MD, MSEE	Deaths	12 (35%)
Zurich, Switzerland		Heart failure	4 (33%)
OBJECTIVES	We sought to describe characteristics and outcome in adults with isolated ventricular	Sudden cardiac death	6 (50%)
PACKODOUND	noncompaction (IVNC).	Others*	2 (17%)
BACKGROUND	arrest of compaction of the loose interwoven meshwork. Knowledge regarding diagnosis,	Heart transplantation	4 (12%)
METHODS	morbidity and prognosis is limited.	Syncope	6 (18%)
METHOD3	segmental thickening of the left ventricular myocardial wall consisting of two layers: a thin,	NYHA class at last follow-up or before heart	
	compacted epicardial and an extremely thickened endocardial layer with prominent trabecu- lations and deep recesses. Thirty-four adults (age ≥ 16 years, 25 men) fulfilled the diagnostic	transplantation or death	
	criteria and were followed prospectively.	Class I/II	18 (53%)
RESULTS	At diagnosis, mean age was 42 ± 17 years, and 12 patients (35%) were in New York Heart Association class III/IV. Left ventricular end-diastolic diameter was 65 ± 12 mm and	Class III/IV	16 (47%)
	ejection fraction 33 \pm 13%. Apex and/or midventricular segments of both the inferior and	Ventricular tachycardia	14 (41%)
	complications were heart failure in 18 patients (53%), thromboembolic events in 8 patients	Thromboembolic events	8 (24%)
	(24%) and ventricular tachycardias in 14 patients (41%). There were 12 deaths: sudden in six,	Cerebrovascular accident	1 (3%)
	transplantation. Automated cardioverter/defibrillators were implanted in four patients.	Transient ischemic attack	6 (18%)
CONCLUSIONS	Diagnosis of IVNC by echocardiography using strict criteria is feasible. Its mortality and morbidity are high, including heart failure, thrombo-embolic events and ventricular arrhyth-	Mesenteric infarction	1 (3%)
	mias. Risk stratification includes heart failure therapy, oral anticoagulation, heart transplan-	Pulmonary embolism	3 (9%)
	tation and implantation of an automated deformator/cardioverter. As IVNC is a distinct entity, its classification as a specific cardiounyopathy seems to be more appropriate. (J Am Coll Cardiol 2000;36:493–500) © 2000 by the American College of Cardiology	*Two other deaths included pulmonary embolism and arrhyth NYHA = New York Heart Association.	nmic, nonsudden death



Magnetic Resonance Imaging

Prevalence and Prognostic Significance of Left Ventricular Noncompaction in Patients Referred for Cardiac Magnetic Resonance Imaging

Alexander Ivanov, MD; Devindra S. Dabiesingh, DO, MPH; Geetha P. Bhumireddy, MD; Ambreen Mohamed, MD; Ahmed Asfour, MD; William M. Briggs, PhD, MS; Jean Ho, BS; Saadat A. Khan, MD; Alexandra Grossman, BS; Igor Klem, MD; Terrence J. Sacchi, MD; John F. Heitner, MD

Background—Presence of prominent left ventricular trabeculation satisfying criteria for left ventricular noncompaction (L/NC) on routine cardiac magnetic resonance examination is frequently encountered; however, the clinical and prognostic significance of these findings remain elastyce. This registry and to assess L/NC prevalence by 4 current criteria and to prospectively caluate an association between disposis of L/NC by these criteria and adverse events. Methods and Results—There were 700 parients reference for cardiac magnetic resonance: 42% were wornen, median age was 70 years (mag. 45-71) years), intera fleft ventricular rejection fractions was 51% (±17%), and 32% had late gadoinium enhancement on cardiac magnetic resonance. The cohort underwent diagnostic assessment for L/NC by 4 separate imaging criteria—Teterence by their authors as Peteresen, Stacey, Jacquiet, and Captar, with L/NC prevalence of 39%, 25%, 25% and 3%, respectively. Primary clinical outcome was combined end point of time to death, ischemic stroke, ventricular techycardia/ventricular librillation, and heart failure hospitalization. Scondary clinical outcomes were (1) al-cause motality and (2) time to the first occurrace of any of the following events: cardiac death, ischemic stroke, ventricular techycardia/ventricular librillation, or heart failure hospitalization, During a median following of 7 years, there were no statistically significant differences in assessed outcomes noted between patients with and without L/NC inseptcive of the applied criteria.

Ivanov et al. LVNC in Patients Referred for CMR



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Long-Term Outcome of		Children (n = 52)					Adults n	= 275		
Noncompaction Cardiomyopathy		3	No Mutation (n = 29) (55%)				No Mutation (n = 194) (70%)			
Jaap I. van Waning, MD, ⁵ Kadir Caliskan, MD, PnD, ⁵ Yvonne M. Hoedemaekers, MD, PnD, ⁶ Karin Y. van Snaendonck-Zwarts. MD. PnD. ⁶ Annette F. Baas. MD. PnD. ⁶ S. Matthiis Boekholdt. MD. PnD. ⁶		Mutation (n = 23) (45%) (n Genetic Prot	Familial (n = 8) (15%)	Not Familial (n = 21) (40%)	Not Familial (n = 21) (40%) Sporadic Total	Mutation (n = 81) (30%) Genetic	Familial (n = 45) (16%) Probably Genetic	Not Familial n = 149 (54%) Sporadic	Total	p Value
Joost P. van Melle, MD, ^g Arco J. Teske, MD, PHD, ^h Folkert W. Asselbergs, MD, PHD, ^h Ad P.C.M. Backx, MD, ⁱ			Probably Genetic	Sporadic						
Gideon J. du Marchie Sarvaas, MD, ¹ Michiel Dalinghaus, MD, PHD, ^k Johannes M.P.J. Breur, MD, PHD, ¹	Median follow-up, months	81 (13-114)	24 (1-102)	40 (19-120)	60 (18-113)	26 (6-69)	29 (10-71)	22 (4-49)	25 (4-58)
Marijke P.M. Linschoten, MsC, Laura A. Venooij, MD, Isabella Kardys, MD, PHD, Dennis Dooijes, PHD, Ronald H. Lekanne Denrez, PuD ^d Arne S. Homa, PuD ^a Maarten P. van den Berg, MD, PuD ^a	Heart failure									
Robert M.W. Hofstra, PhD, ^a Marjon A. van Slegtenhorst, PhD, ^a Jan D.H. Jongbloed, PhD, ^c Danielle Maioor-Krakauer, MD, PhD ^a	Heart failure requiring hospitalization	9 (39)	3 (38)	1 (5)	13 (25)	20 (25)	9 (20)	30 (20)	59 (21)	
	Thromboembolic events									
ABSTRACT	TIA					2 (2)	1 (2)	5 (3)	8 (3)	
	Stroke	1 (4)			1 (2)	1 (2)	4 (9)	12 (8)	17 (6)	
BACKGROUND The clinical outcomes of noncompaction cardiomyopathy (NCCM) range from asymptomatic to heart failure arrhythmiss and sudden cardiac death. Genetics play an important role in NCCM	Peripheral thromboembolism	1 (4)			1 (2)	4 (5)	1 (2)	6 (4)	11 (4)	
neur latar, annyannas, and sadden cardie death. denetics pay an important role in receiv.	Arrhythmias									
OBJECTIVES This study investigated the correlations among genetics, clinical features, and outcomes in adults and abilities dispared with NCCM	Atrial fibrillation	3 (13)	1 (13)	1 (5)	5 (10)	15 (19)	8 (18)	21 (14)	44 (16)	
children diagnosed with NCCM.	Sustained VT/VF	1 (4)	2 (25)	1 (5)	3 (6)	3 (4)	2 (4)	11 (7)	16 (6)	
METHODS A retrospective multicenter study from 4 cardiogenetic centers in the Netherlands classified 327 unrelated	ICD	5 (22)	2 (25)	2 (10)	9 (17)*	34 (42)	15 (33)	57 (38)	106 (39)	0.004
NCCM patients into 3 categories: 1) genetic, with a mutation in 32% (81 adults; 23 children) of patients; 2) probably genetic, familial cardiomyopathy without a mutation in 16% (45 adults; 8 children) of patients; or 3) sporadic, no family	Secondary prevention (% of ICD)		1 (50)		1 (11)	3 (9)	2 (13)	5 (9)	12 (4)	
history, without mutation in 52% (149 adults; 21 children) of patients. Clinical features and major adverse cardiac events (MACE) during follow-up were compared across the children and adults.	Appropriate shock (% of ICD)					1 (3)	3 (20)	6 (11)	10 (9)	
RESULTS MYH7, MYBPC3, and TTN mutations were the most common mutations (71%) found in genetic NCCM. The risk	LVAD	1 (4)			1 (2)	2 (2)		1 (1)	3 (1)	
of having reduced left ventricular (LV) systolic dysfunction was higher for genetic patients compared with the probably	Heart transplantation	4 (17)			4 (8)	2 (2)	2 (4)	2 (1)	6 (2)	
genetic and sporadic cases (p = 0.024), with the highest risk in patients with multiple mutations and TTN mutations.	Death	4 (17)	3 (38)	1 (5)	8 (16)	3 (4)	5 (11)	8 (5)	16 (6)	
Mutations were more frequent in children ($p = 0.04$) and were associated with MACE ($p = 0.025$). Adults were more likely to have sporadic NCCM. High risk for cardiac events in children and adults was related to LV systolic dysfunction in	MACE in patients with LV systolic dysfunction	8 (35)	3 (38)	0 (0)	11 (21)	12 (15)	6 (13)	18 (12)	36 (13)	
mutation carriers, but not in sporadic cases. Patients with MYH7 mutations had low risk for MACE (p = 0.03).	MACE in patients with	1 (4)	1 (13)	1 (5)	3 (6)	1 (1)	5 (11)	16 (11)	22 (8)	0.027
CONCLUSIONS NCCM is a heterogeneous condition, and genetic stratification has a role in clinical care. Distinguishing	normal LV function									
genetic from nongenetic NCCM complements prediction of outcome and may lead to management and follow-up										

Van Waning et al. Noncompaction Cardiomyopathy Features and Genetics

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1.Baseline Characteristics of patients with Excessive trabeculation, preserved	d EF and ICD implanted. (N=20)
Gender Female.	10 (50)
Age at diagnosis (yrs).	39 (27,54)
LVEF at presentation (%)	*57.5 (7.5)
Follow up time (yrs)	13 (9,15)
2.Risk factors for SCD and VAs	
Family history of SCD/Genetic cardiomyopathy.	7(35)
History of Syncope Prior to ICD implant.	8(40)
History of NSVT Prior to ICD implant.	7(35)
LV morphological or Functional abnormality on CMR.	8(40)
3.Device Characteristics	
Age at ICD implantation (yrs).	41.1 (29,56)
Time from device Implantation to appropriate therapy	2.1 (2.4,6)
Type of device implanted	Single chamber 10 (50%)
	Dual chamber 8 (40%)
	Biventricular 2 (10%)
4.Adverse Outcomes	
Composite outcome	9 (45)
Appropriate ICD Therapy	5 (25)
Stroke	2 (10)
Development of Cardiomyopathy	2 (10)
Death	0 (0)

ICD, Implantable Cardiac Defibrillator; LVEF, Left Ventricular Ejection Fraction; SCD, Sudden Cardiac Death; VAs, Ventricular Arrythmias; CMR, Cardiac Magnetic Resonance. Numerical variables Median (IQR). Categorical variables; N (%). *LVEF, Mean (SD).

Bahbah et al. ICD implantation in patients with Excessive Trabeculation and preserved Ejection fraction.

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Definition

Excessive trabeculation is a deviation from the conventional developmental process of the human structural myocardium, which in the presence of certain genetic, familial and clinical factors can give rise to a Cardiomyopathic disorder with potentially fatal outcomes.







































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Conclusions

- Women practicing IC are less likely to be married or have children
- Women and non-white individuals encounter obstacles in career advancement
- Women, non-native English speakers, and non-white individuals have a higher likelihood of experiencing discrimination from patients and families, peers, supervisors, support staff, and nursing staff
- A portion of the participants (41%) expressed concerns that DEI initiatives might result in unintended consequences



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Thank you!

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What is burnout?	
A syndrome conceptualized as resulting from chronic workplace stre managed. 3 dimensions:	ss that has not been successfully
feelings of energy depletion or exha	ustion
↑ mental distance from one's job, or feelings of negativism of	or cynicism related to one's job
↓ professional efficacy	
	WHO definition
Minneapolis Heart Institute Foundation CENTER FOR CORONARY ARTERY DISEASE	



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	Author (publication year)	Country	Participants (n)	How was burnout assessed	Main Results
	Mehta, L.S. et al. (2019)	USA	Cardiologists (2,274)	Mini-Z survey	26.8% reported burnout symptoms
	Lobo, A.S. et al. (2019)	USA	Cardiovascular workers (481) [70% interventional cardiologists]	"Do you feel burnout?"	56.5% reported burnout
	Shah, S. et al. (2020)	USA	IC, structural, CHIP, and peripheral vascular fellows (135)	Not applicable	65% reported significant stress at work and at home because of the COVID-19 pandemic
	Cullen, M.W. et al. (2021)	USA	Cardiovascular Diseases Fellowship Program Directors (141)	Mini-Z survey	21% reported symptoms of burnout
	Joshi, S.S. et al. (2022)	International	Cardiac imaging specialists (125)	Not reported	58% reported burnout (64% reported worsening during the COVID-19 pandemic)
	Sharma, G. et al. (2023)	International	Cardiologists (5,890)	Not applicable	28% reported any self-reported MHC, of which 76.1% reported psychological distress
7	Simsek, B. et al. (2023)	International	Attending IC (1,159) and IC fellows (192)	"Rate the impact of your burnout on your life"	69% of the interventional cardiologists are affected by burnout
	Koval, M.L. (2023)	USA	Cardiologists (367)	"Do you feel burnout/and or depressed?"	43% felt burned out (29%) or both burned out and depressed (14%)
2	Simsek, B. et al. (2023)	USA/Canada	First-year IC fellows (111)	Not applicable	84% considered the interventional cardiology fellowship somewhat (62%) or very stressful (22%)
	Bogerd, R. et al (2023)	Netherlands	Cardiologists (382)	Not applicable	3.85/5 on professional fulfillment, 2.25/ 5 on work exhaustion and 2.04/5 interpersonal disengagement
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	Minneapol Heart Inst Foundation	is itute CENT	ER FOR CORONARY ARTERY DISEASE		Allina Health Winneapolis Heart institute















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Causes of Long-Term Mortality in Patients with Microvascular Obstruction Following ST-segment Elevation Myocardial Infarction

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Historical Observations of MVO (no-reflow)

- First observed in the Brain !
- First described in the heart by Kloner et al (JCI, 1974) "The no-reflow phenomenon after temporary coronary occlusion in the dog".

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-40 min LAD coronary occlusion = normal reperfusion following release of occlusion.-90 min occlusion = impaired blood flow following release of occlusion.
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Histopathology (electron microscopy):

- Sig capillary damage in subendocardium with swollen endothelium and intraluminal endothelial protrusion.
- Intraluminal platelets and fibrin thrombi.
- Interstitial and intramyocardial edema (extrinsic compression)

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Historical Observations of MVO

- Increases with Ischemic duration.
- No-Reflow area increases over time (process, not an event).
- Cell contracture may contribute to microvascular compression
- Platelet and leukocyte depletion may reduce extent of no-reflow.
- Confined to region of myocardial infarction

Kloner R, et al. JCI 1974







The Presence of MVO as Powerful as Infarct Size or LV function in Predicting Event-Free Survival Event-Free Survival Event-Free Survival 30 LVEF ≤479 IS ≥19%L\ 20 10 LVEF >47% IS <19%LV Multicenter Study from c 4 6 ż 10 12 10 Time (Months After Infarction) Time (Months After Infarction) Germany Of 738 ne0 001 p<0.00 **STEMI** patients Event-Free Survival зс Event-Free Survival 30 20 MO presen 20 10 MSI >3 No MC 6 8 10 12 6 å 10 Time (Months After Infarction) Time (Months After Infarction) FIGURE 4 Event-Free Survival According to IS, Myocardial Salvage Index LVEF, and MO Kaplan-Meier curves showing the risk of cardiac events, stratified by the size of the infarct (IS), the myocardial salvage index (MSI), left ventricular ejection fraction (LVEF), and the presence of microvascular obstruction (MO). LV = left ventricle.

The NHLBI TIME Trial: Role of Microvascular Obstruction in 2-Year Clinical and MRI Follow-up

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2016 Scientific Sessions of the AHA

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Long-Term Causes of Mortality in Patients with MVO Following STEMI – Rationale and Methodology

Rationale: The long-term natural history and mortality is not known in patients with MVO following STEMI beyond 2 years.

- We analyzed the long-term follow-up and cardiac MRIs of 475 patients admitted through the Level 1 Program (Mean age = 60 years, 76% male) for the presence (n=337) or absence (n=138) of MVO following STEMI and successful reperfusion with PCI between 2007 -2017.
- Causes of death were determined from the patient's electronic medical record or death certificate.

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Long-Term Causes of Mortality in Patients with MVO Following STEMI – RESULTS

- Patients with MVO had greater ischemic times (159 vs. 141 mins; p = 0.012) and were more likely to have TIMI 0 flow on presentation (71 vs 54%; p <0.002).
- Patients with MVO had greater infarct size by cardiac enzymes resulting in greater LV end-diastolic volume index (LVEDVI) (80 vs.75 ml/m2) and LV end-systolic volume index (LVESVI) (39 vs 30 ml/m2) LV mass (146 vs 134 g) and reduced LVEF (48 vs 58 %; all p < 0.01) by cardiac MRI performed 1-3 days following STEMI and PCI.
- During long-term follow-up a total of 56 patients with MVO died compared to 18 patients without MVO.
- Patients with MVO died sooner after STEMI (5.9 vs 7.8 years) and were more likely to die from cardiovascular causes such as progressive heart failure or sudden cardiac death (Table). LVEF before death was lower in the MVO group (46 vs 52%).

Long-Term Mortality of STEMI Patients who have MVO on cardiac MRI							
Cause of Death	Cardiovascular - CHF - Cardiac arrest - MI	Neurological - Dementia - Stroke - ICH	Sepsis	Pulmonary -pneumonia -respiratory failure - COVID- ARDS	Cancer	Natural Causes	Time from STEMI to death (years)
MVO + (n=56)	22*	8	6	7	9	4	5.9 ± 4
MVO – (n=18)	1	1	0	3	7	7	7.8 ± 4
* P < 0.0	004 Fishers Exact Test						



CONCLUSIONS

 There is significant long-term mortality associated with MVO following STEMI with the majority of patients dying from cardiovascular causes. In contrast, patients with STEMI without MVO rarely died from a cardiac etiology but from cancer and natural causes.

These findings may suggest that MVO acts as a surrogate marker for greater underlying atherosclerosis, microvascular dysfunction or other unknown co-morbidities that enhances Long-term cardiovascular mortality.