

CASE REPORT · ELECTROPHYSIOLOGY & CARDIO-ONCOLOGY

The Seizure That Wasn't: A Case of Recurrent Unstable Bradycardia

Leadless pacemaker as a bridge to definitive therapy



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Division of Cardiovascular Medicine · University of Kentucky · Kentucky Chapter of the American College of Cardiology · 2026

A neck mass, a “seizure,” and a pulse in the 30s

History



55-year-old woman

COPD on 3 L home O₂ · 40+ pack-year smoker · CHF, HTN, obesity, chronic pain



Enlarging left neck mass × 3 months

Recurrent severe pain crises (several ER visits) · hoarseness, otalgia, dysphagia · ~10 lb weight loss · no prior oncologic workup



Witnessed event during a pain crisis

Described by her daughter as a seizure: lightheadedness, nausea, convulsive syncope, amnesia



Transferred for higher level of care

Expedited oncologic and cardiac workup; concurrently treated for UTI

30s_{bpm}

Sinus bradycardia

Symptomatic, recurrent

87/45_{mmHg}

Hypotension

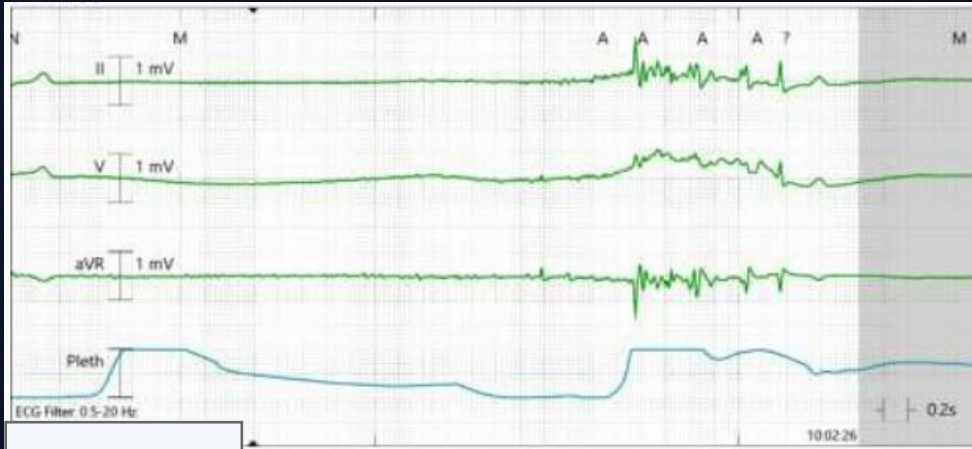
During the index episode

Atropine_{responsive}

Transient resolution

Both rate and pressure recovered

SA and AV nodal block, time-locked to neck pain



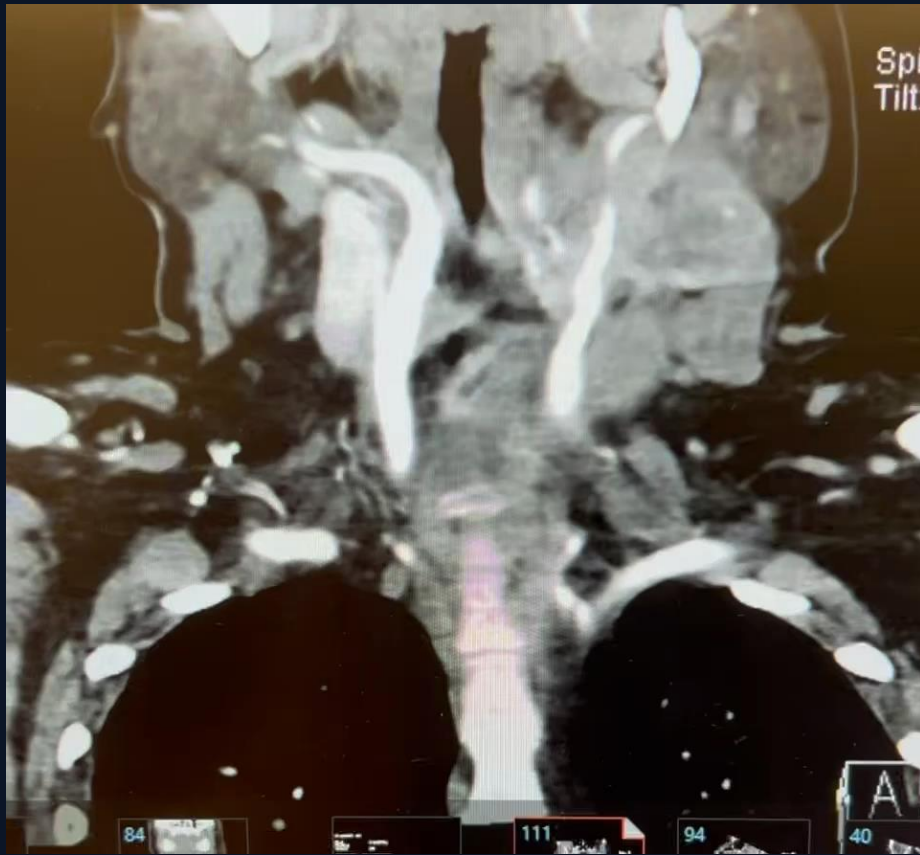
Pain → bradycardia

Recurrent SA and AV nodal block on telemetry, consistently coincident with jaw / neck pain crises; HR in the 30s

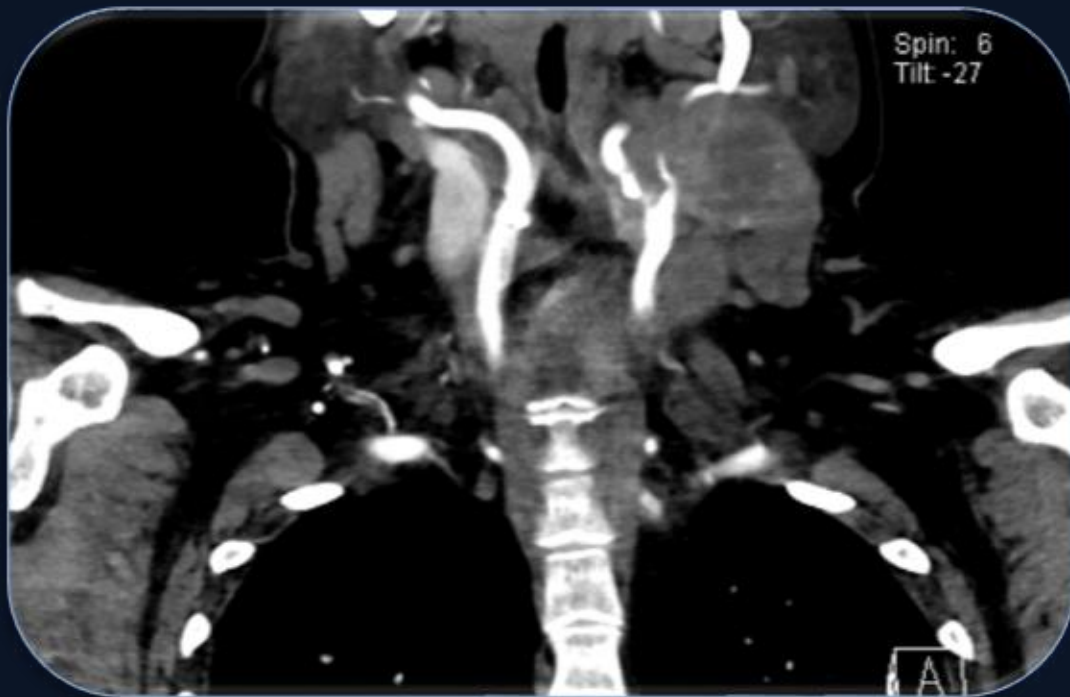


Code Blue, hospital day 3

Bradycardic hypotension; ROSC after ~20 s of compressions, atropine and an epinephrine infusion



A necrotic nodal mass encasing the carotid bifurcation



CT angiography · hospital day 1

~4 cm necrotic left level II nodal mass encasing the carotid bifurcation, ICA and ECA, with skull-base extension



Follow-up CT venogram

Mass $5.5 \times 4 \times 8$ cm; invades the IJ with tumor thrombus to the jugular bulb and sigmoid sinus

Locally advanced, unresectable supraglottic SCC



Laryngoscopy + biopsy · hospital day 3

p16-negative squamous cell
carcinoma of the epiglottis



Staging

Stage IVA (cT3, cN2b, cM0); no
distant metastasis.

Unresectable: carotid encasement,
skull-base extension



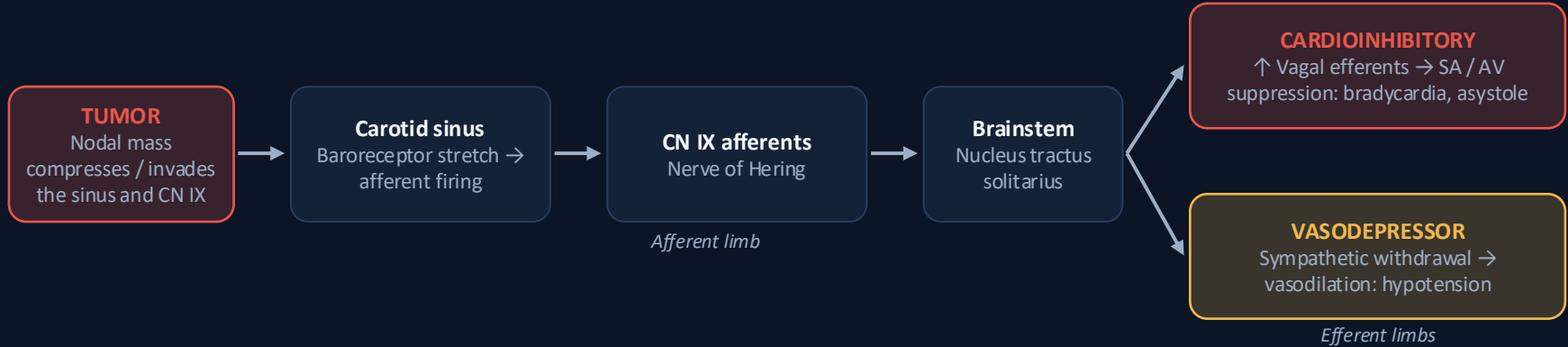
Echocardiogram

LVEF 65%, normal diastolic function,
no intracardiac mass or thrombus;
incidental small apical LV aneurysm

The pattern

Atropine-responsive, structurally normal heart, no intrinsic conduction disease identified → **a reflex mechanism**

The carotid sinus reflex, hijacked by a tumor



Intrinsic (classic) CSS

- Trigger: head turning, tight collar, carotid sinus massage [1]
- Older adults, atherosclerosis; ~1% of syncope [1]
- Predominantly cardioinhibitory → pacing is definitive (Class I / IIa) [1,2]
- Cardioinhibitory (70-75%) vs. Mixed (20-25%)

Extrinsic (tumor-induced) CSS

- Compression / invasion by nodal disease; pain-triggered (11 of 17); seizure activity in 8 of 17 [3,4]
- Vasodepressor / mixed physiology predominates: "most often vasodilatory"; pacing failed 3/3 as pure vasodepression emerged; pacing ± pressors succeeded in only 5 of 16 [3,5-8]
- Definitive therapy is oncologic: syncope resolved after radiotherapy or chemotherapy where pacing had failed [8-10]

Making the diagnosis without carotid sinus massage



The standard

- Carotid sinus massage reproducing symptoms with:
- Asystole ≥ 3 s or AV block $\rightarrow$ cardioinhibitory
- SBP fall ≥ 50 mmHg $\rightarrow$ vasodepressor
- Both $\rightarrow$ mixed [1,11]



Why not here

- Tumor encased the bifurcation and both carotid branches
- Massage is to be avoided with carotid bruit or significant carotid disease (embolic risk) [1]
- And often negative in tumor CSS anyway: positive in only 5 of 10 tested [3]



What we had instead

- Reproducible temporal correlation: pain $\rightarrow$ telemetry-documented SA / AV block
- Anatomic substrate on CTA at the carotid sinus
- No competing cause: EF 65%, normal structure, no metastases

Leadless pacing as a bridge, not a destination



1

Hospital day 5

Micra VR implanted, VVI 50 bpm



2

~2 weeks later · CCU

Still bradycardic and hypotensive; two further Code Blue events, atropine-responsive



3

Reprogrammed

Increase rate ↑ to 90 bpm & transfer to CVICU for vasopressor support → clinical improvement

Why leadless?



No pocket, no leads → minimal infection risk

Chemoradiation and immunosuppression imminent; ESC favors leadless when infection risk is high [2]



No hardware in or near the irradiated field

Bulky nodal disease and a planned 33-fraction neck field

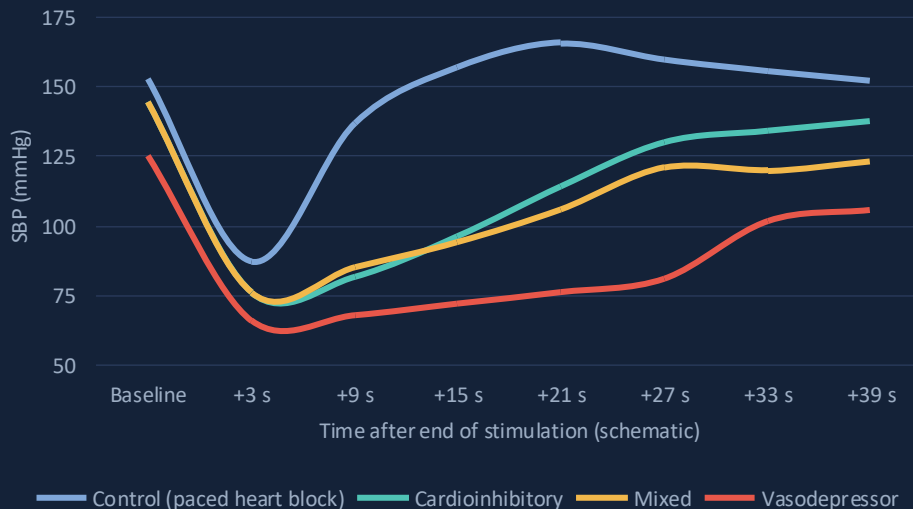


Evidence in exactly this patient profile

Micra CED subgroups: fewer complications than transvenous VVI in malignancy (HR 0.68) and COPD (HR 0.73); 3-year infection < 0.2% vs 0.7% [12–14]

Pacing fixes the rate, not the tone

Systolic BP recovery after carotid sinus stimulation, by subtype



Adapted (schematic, approximate values) from Wieling et al. 2013, Fig. 4 [15]. The vasodepressor curve stays low long after the rate is restored.



Cardioinhibitory limb: corrected

Vagal SA / AV suppression. A rate floor restores output: 50 bpm, then 90 bpm to augment cardiac output against vasodilation.

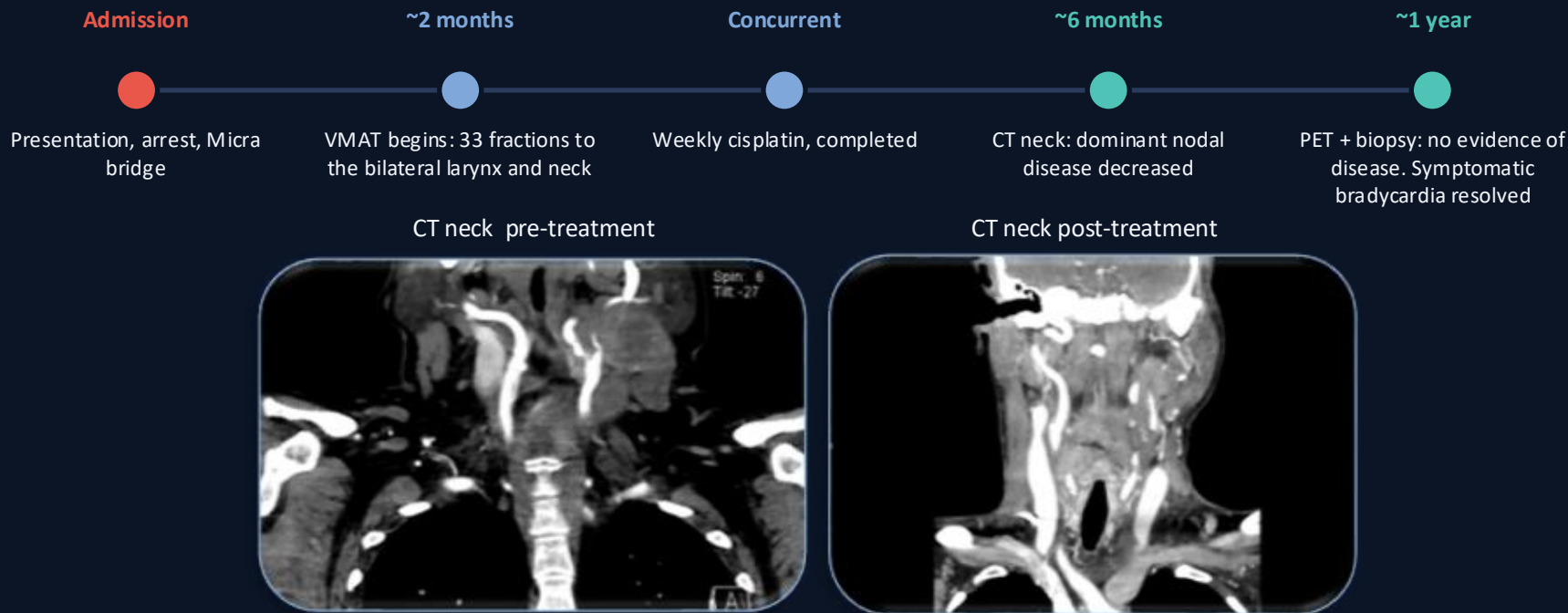


Vasodepressor limb: not corrected

Sympathetic withdrawal → vasodilation.
Vasopressors in the CVICU, then PRN midodrine [16]; PRN clonidine for hypertensive surges: true autonomic instability.

Tumor CSS is disproportionately vasodepressor or mixed [3,5–8]; isolated cardioinhibition without a pressure fall essentially does not occur [15].

Treatment Course



Two more cardiovascular decisions in the same neck



Tumor thrombus: anticoagulate?

- Follow-up CT venogram: IJ invasion with tumor thrombus filling the jugular bulb and extending into the sigmoid sinus; no cortical vein thrombosis
- **Rivaroxaban** started after a documented risk–benefit discussion
- Trade-off: bleeding from a necrotic, carotid-encasing mass vs propagation / embolism



Carotid encasement: stent prophylactically?

- Neurosurgery planned overlapping carotid stents to reduce radiation-related carotid injury; committing her to indefinite dual antiplatelet therapy
- Sequenced around PEG placement; later reconsidered by radiation oncology for post-RT edema risk
- Context: carotid blowout is rare with contemporary RT (~1%; pooled ~5%) but rapidly fatal; prior RT (89%) and nodal metastasis (69%) are the typical substrate [17,18]
- Stenting was ultimately **deferred**

What happened, and what we cannot claim



Outcome

- The device served as a bridge through the highest-risk window
- ~1 year: PET and biopsy with no evidence of disease
- ~14 months: CT: stable 17 × 14 mm left IIb node, post-radiation changes
- Died at home about one week later; cause undetermined



Limitations

- No carotid sinus massage: the diagnosis is inferred from correlation, imaging and exclusion
- A single case
- Cause of death unknown: aspiration, carotid blowout and thromboembolism are unconfirmed differentials, not conclusions
- Incidental apical LV aneurysm not implicated in the bradycardia

Four things to remember

1

Neck mass + unexplained bradycardia = think extrinsic CSS

Convulsive syncope and pain-triggered episodes are the clues; the first diagnosis is often 'seizure'. [3–5]

2

When massage is unsafe, correlation is the diagnosis

Telemetry-documented block time-locked to symptoms, an anatomic substrate on imaging, and exclusion of competing causes. [1,3]

3

Expect mixed physiology: pacing fixes rate, not tone

Plan for the vasodepressor limb: aggressive rate programming, vasopressors, midodrine. Persistent hypotension is physiology, not device failure. [3,5–8,15,16]

4

Pace to bridge, treat to cure

A leadless device suits infection risk and an irradiated field; chemoradiation, not the pacemaker, resolved the arrhythmia. [2,8–10,12–14]

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AngioVac Vegetation Debulking in Right-Sided Infective Endocarditis

A systematic review and meta-analysis

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Off-label use: AngioVac vegetation debulking is off-label

Why this question matters



Right-sided IE is 5–10% of endocarditis

Native tricuspid valve or infected device leads



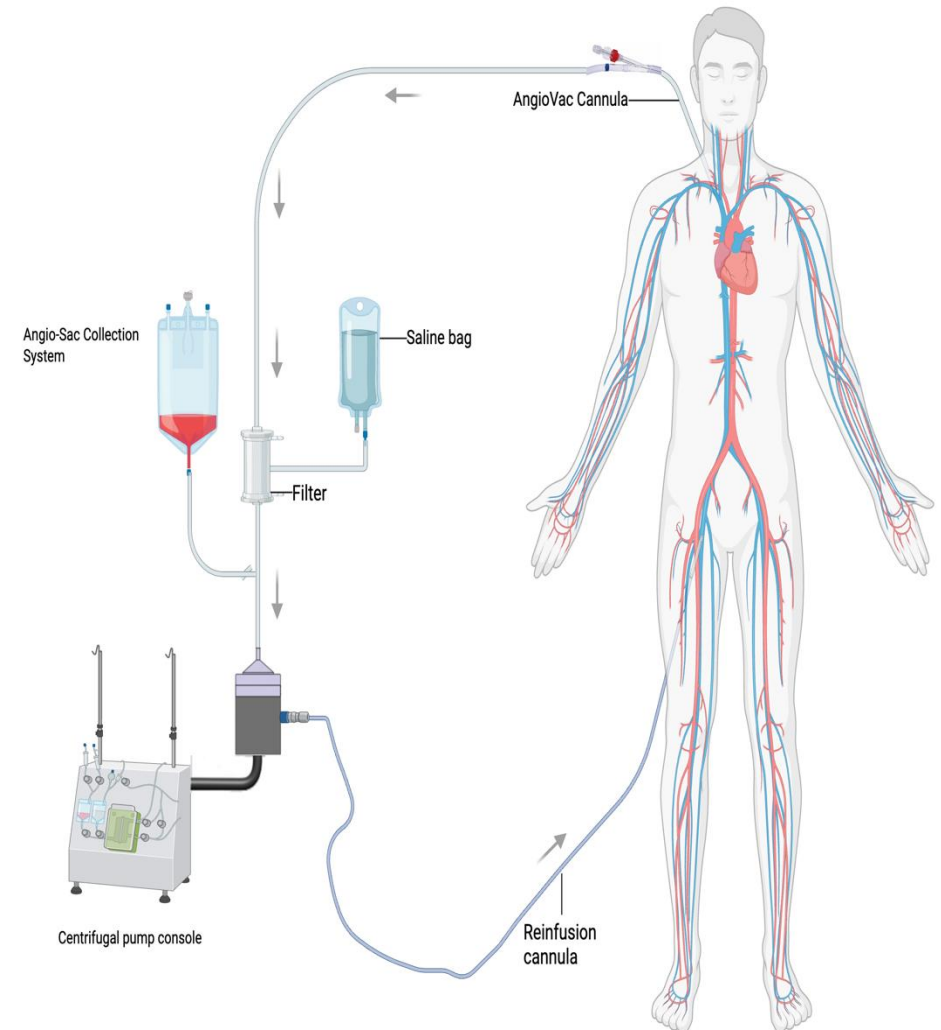
AngioVac use is growing

Off-label debulking before lead extraction or surgery



Is it effective and safe?

Existing evidence: small, mixed, overlapping series



Methods



- 3 databases through July 2026 · PRISMA 2020
- ≥ 5 AngioVac-treated right-sided IE patients
- Random-effects GLMM of proportions
- Two independent reviewers · JBI

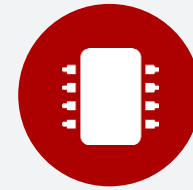
Two clinically different populations



Native tricuspid valve

5 reports

- Younger (mean age $\approx$ 29–42 y)
- Largely people who inject drugs
- Next step: valve surgery if needed



Cardiac device (CIED)

8 reports

- Older (mean age $\approx$ 52–76 y)
- Debulking before lead extraction
- Next step: full system removal

6 further reports included mixed native, prosthetic and device infection.

Efficacy:

93%

Study-defined success

95% CI 89–96

10 cohorts · 223 patients

93%

Vegetation reduced >50%

95% CI 81–99

11 cohorts · 223 patients

91%

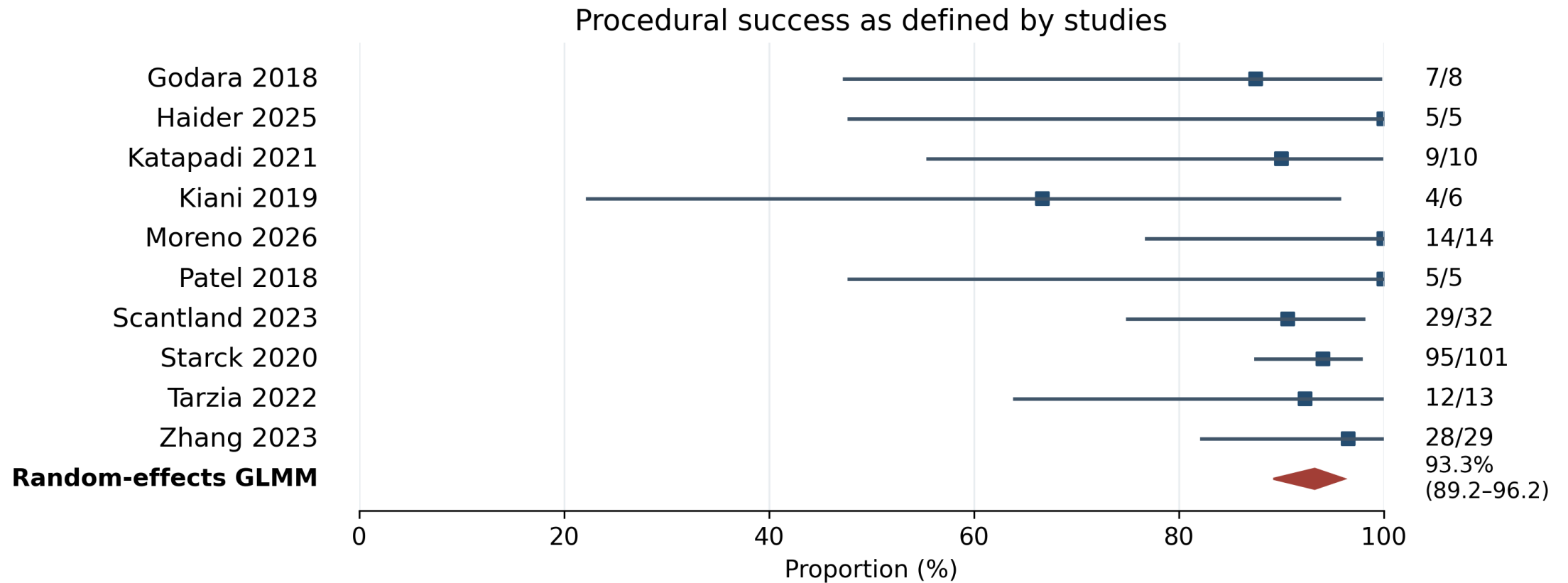
Bacteremia cleared

95% CI 83–97

5 cohorts · 132 patients

Clearance also reflects antibiotics, extraction and surgery — not aspiration alone.

Pooled study-defined procedural success: 93.3%



k = 10 independent cohort contributions. Study CIs: exact binomial. Pooled CI: profile likelihood.

Safety and subsequent clinical outcomes

Immediate and early outcomes

99.6%

Survived the procedure

95% CI 98.7–100 · 16 cohorts · N=492

3.5%

Early mortality

95% CI 1.0–7.7 · 14 cohorts · N=489

2.3%

Procedural complications

95% CI 0.3–7.1 · 11 cohorts · N=438

Needs follow-up — less certain

35%

Needed transfusion

95% CI 21–52 · 2 cohorts · N=49

27%

New/worse TR

95% CI 3–72 · 5 cohorts · N=98

7%

Later tricuspid surgery

95% CI 3–17 · 6 cohorts · N=301

Limitations



Single-arm, mostly retrospective

No comparison with antibiotics or surgery



Different definitions of success

Outcomes measured differently across studies



Patient selection often unclear

Diagnosis unclear in 16 of 20 reports

Take-home messages



Reported debulking and study-defined success exceeded 90%



Transfusion and tricuspid valve dysfunction warrant follow-up



Best used as an adjunct or bridge, decided by an endocarditis team

Uncontrolled data do not establish comparative benefit.

Thank you

Questions?

Ramtin Khanipour, DO · University of Louisville, Division of Cardiovascular Medicine



Frontal QRS-T Angle Distinguishes Cardiac Amyloidosis from Non-Amyloid Heart Failure but Does Not Correlate with Cardiac Magnetic Resonance Findings

Carrigan Wasilchenko MD, Jeffrey Spindel DO



Outline



- Disclosures
- Background
- Objectives
- Methods
- Results
- Limitations
- Conclusions
- References



Disclosures



- Funding source: UK Myocardial Recovery Alliance (MYRA) and UK Center for Clinical Translational Science (CCTS) grant #UL1TR001998
- Biopsy Collection and Repository Database
 - Clinicaltrials.gov #NCT05521503



Background



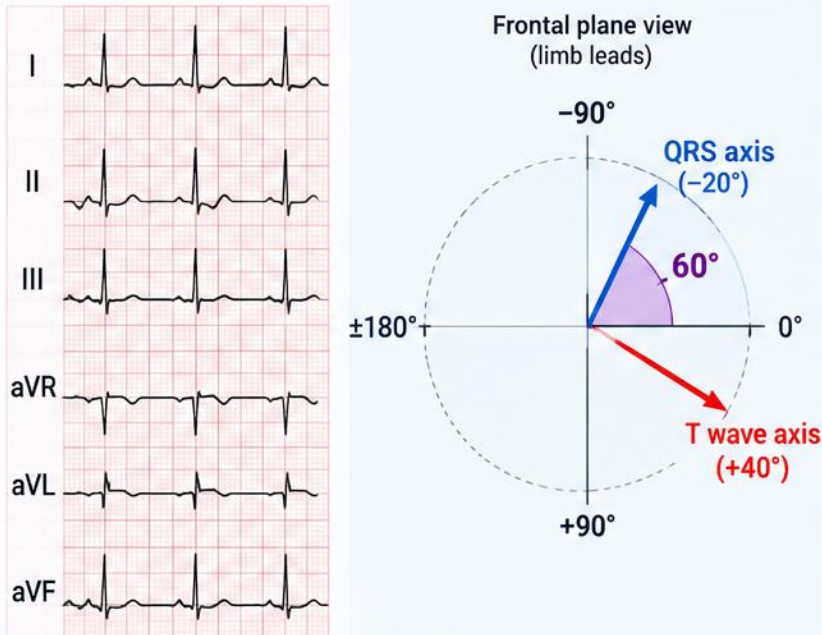
- Cardiac amyloidosis (CA) was historically rare and difficult to diagnose and treat; now, advances in effective therapies have made early and accurate diagnosis critical
- Frontal QRS-T angle has been associated with structural heart disease and adverse cardiovascular outcomes
- A prior study (Medvedosky et al) demonstrated a wide QRS-T angle in AL amyloidosis is associated with advanced amyloid disease and is an independent predictor of survival, but its role across amyloid subtypes and relationship to CMR findings remains unclear.



What is the Frontal QRS-T Angle?

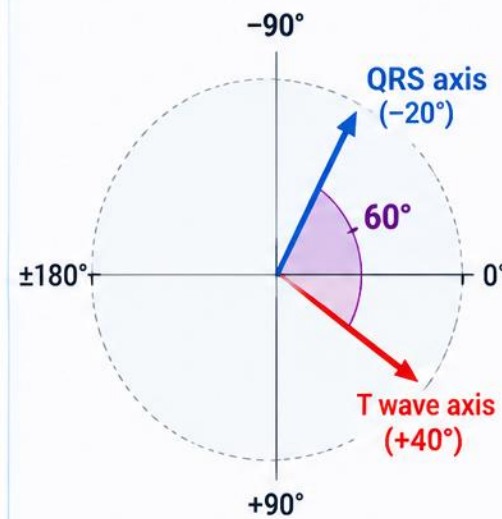


A. 12-lead ECG with Frontal Plane Axes



Frontal QRS-T angle = |QRS axis – T wave axis|
(acute angle used if >180°)

B. Example with Vector Angles



Frontal QRS-T angle
= 60°
(absolute difference)



QRS axis

Direction of ventricular depolarization (QRS complex)



T wave axis

Direction of ventricular repolarization (T wave)



QRS-T angle

Absolute angular difference between the QRS and T wave axes (0°–180°)

A wider QRS-T angle may reflect greater ventricular electrical heterogeneity.



Objectives



- **Primary Question:** Is the frontal QRS-T angle wider in patients with cardiac amyloidosis (CA) compared with patients with non-amyloid heart failure?
- **Secondary Question:** Does the QRS-T angle correlate with CMR markers of myocardial involvement?
- **Hypothesis:** We hypothesized that patients with CA would have a wider QRS-T angle and that greater electrical abnormality would correlate with CMR degree of late gadolinium enhancement (LGE) and prolonged T1 times.



Methods



1 Study Population

Consecutive patients with symptomatic NYHA II–III diastolic heart failure referred to the University of Kentucky Cardiac Amyloidosis Clinic for investigation of underlying cardiomyopathy were prospectively enrolled prior to final diagnosis.



Symptomatic NYHA II–III diastolic heart failure
Referred to UK Cardiac Amyloidosis Clinic



Pre-diagnosis evaluation
(ECG, clinical data)



Excluded
Ventricularly paced
(n = 8)

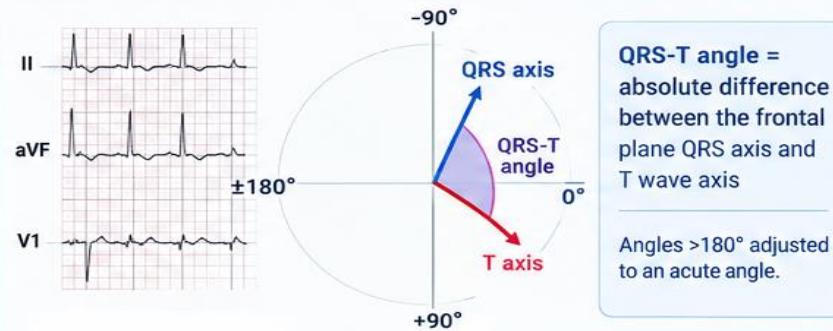
Final Cohort
n = 98

Cardiac amyloidosis
n = 48

- ATTRwt: 23
- ATTRv: 15
- AL: 10

Non-amyloid HF
n = 50

2 QRS-T Angle Definition



Wider angle → greater ventricular electrical heterogeneity
(potential marker of abnormal electrical function)

3 Statistical Analysis



Performed using
STATA



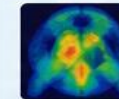
Image generation
using Python

- Circular variables (QRS and T wave axes) compared using the Mardia–Watson–Wheeler test of circular distributions.
- Frontal QRS–T angle compared using Welch's two-sample t-test (unequal variances between groups).

4 Cardiac Amyloidosis Diagnosis^[1]



Endomyocardial biopsy



PYP scan
(grade 3 uptake)



Abdominal fat pad biopsy
(rare)



Genetic testing + CMR
(for ATTR amyloid)

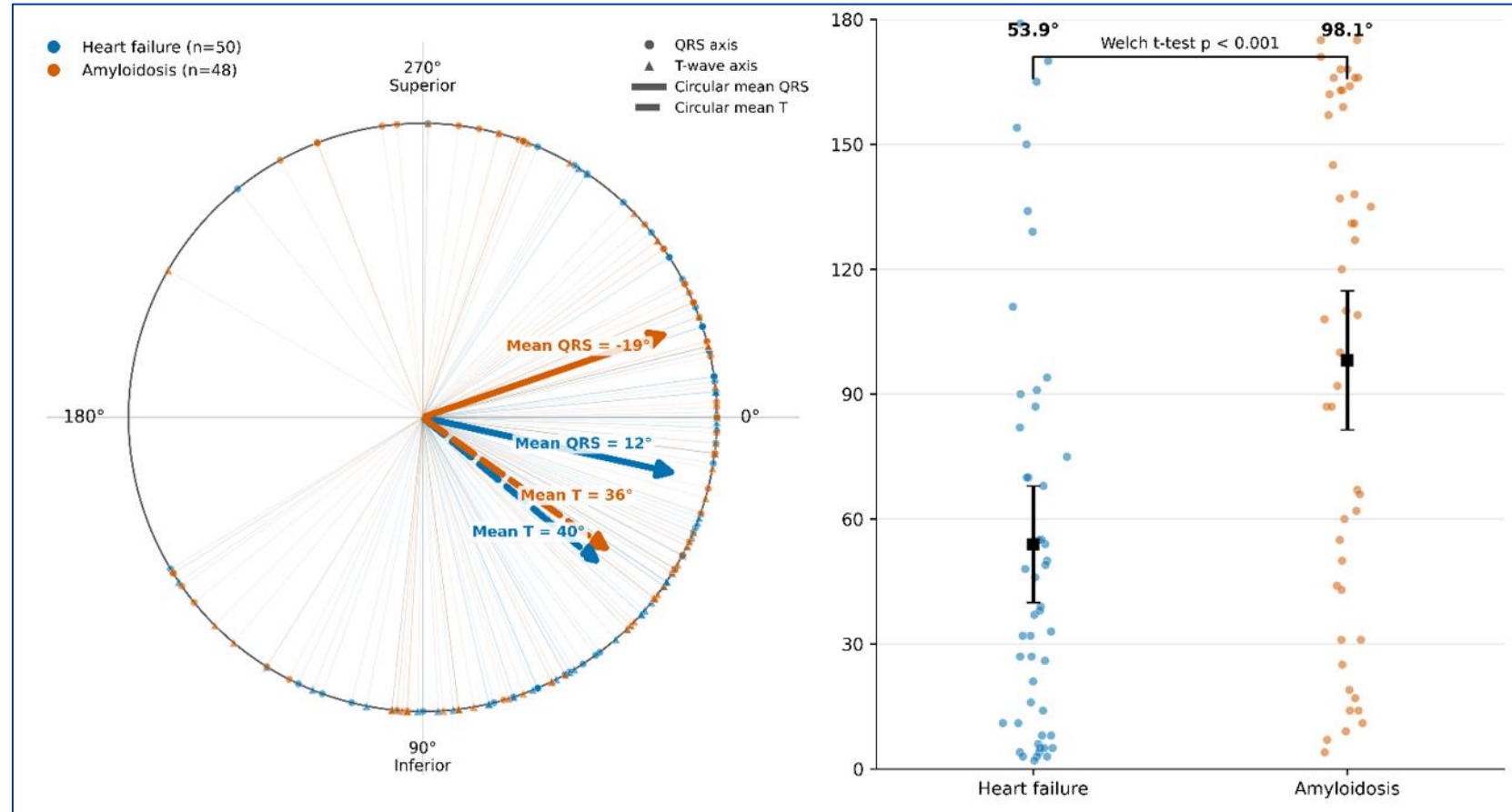


Bloodwork + CMR
(for AL amyloidosis)

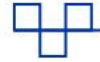
Results – Primary Question



- No significant difference in the QRS axis ($p = 0.074$) or T axis ($p = 0.32$) nor significant difference in QRS duration
- **Frontal QRS-T angle is significantly wider among patients with cardiac amyloidosis**
- Frontal QRS-T angle >92 identified cardiac amyloidosis with AUC 0.714, sensitivity 56.3%, specificity 82.0%, PPV 75%, and NPV 66%



Results – Secondary Question



- QRS-T angle was not associated with degree of late gadolinium enhancement on a categorical scale (n = 27, p=0.24)
 - LGE semi-quantitative scale based on extent of myocardial involvement
 - Grade 0: None
 - Grade 1: < 15%
 - Grade 2: >25%
 - Grade 3: Diffuse/atrial involvement
- Native T1 times were slightly prolonged in the CA group compared to non-amyloid (1140.4 ± 52.5 vs 1058.5 ± 42.8 , p = 0.004)



Limitations



- Sample size and single-center design
- Limited CMR availability
- Heterogeneity in CMR assessment
- Amyloid subtype heterogeneity
- Potential confounding
- Diagnostic performance and validation



Conclusions



- Frontal QRS-T angle was significantly wider in patients with CA than patients with non-amyloid HF
- A QRS-T angle of >92 demonstrated high specificity but limited sensitivity for CA
- Frontal QRS-T angle was not correlated with LGE or T1 times, suggesting that QRS-T angle may capture electrical abnormalities not reflected by conventional CMR tissue characterization
- Frontal QRS-T angle may serve as a simple, inexpensive adjunctive ECG marker to identify patients who warrant further evaluation for cardiac amyloidosis



Table 1:

	Heart Failure (n=50)	Cardiac Amyloidosis (n=48)	p-value
Mean QRS Axis, circular	12.4°	-19°	0.074
QRS axis, Mean Resultant Length	0.63	0.4	
Mean T wave Axis, circular	39.5°	35.5°	0.32
T Wave Axis, Mean Resultant Length	0.76	0.61	
Frontal QRS-T Angle	53.9° ± 50.5°	98.1° ± 59.2°	0.0001
QRS Duration (ms)	99.7 ± 20.0	101.5 ± 23.5	
T1 Times (ms)	1058.5 ± 42.8	1140.4 ± 52.5	0.004
LGE Quantification	1	1.6	0.24

References



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CT-Based Quantitative Assessment of Visceral and Superficial Abdominal Fat and Obesity Measurement Parameters and Their Associations with Macrovascular and Microvascular Complications in Diabetes: A Systematic Review and Meta-Analysis

Presenter: Matthew Dyer, MBA

**Fatemeh Dehghani Firouzabadi, Zara Ali, Ali Sheikhy, Heinrich Dreyer,
Matthew Frank, Nathaniel Zamaite, Meisam Hoseinyazdi, Aida Fallahzade,
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Disclosure

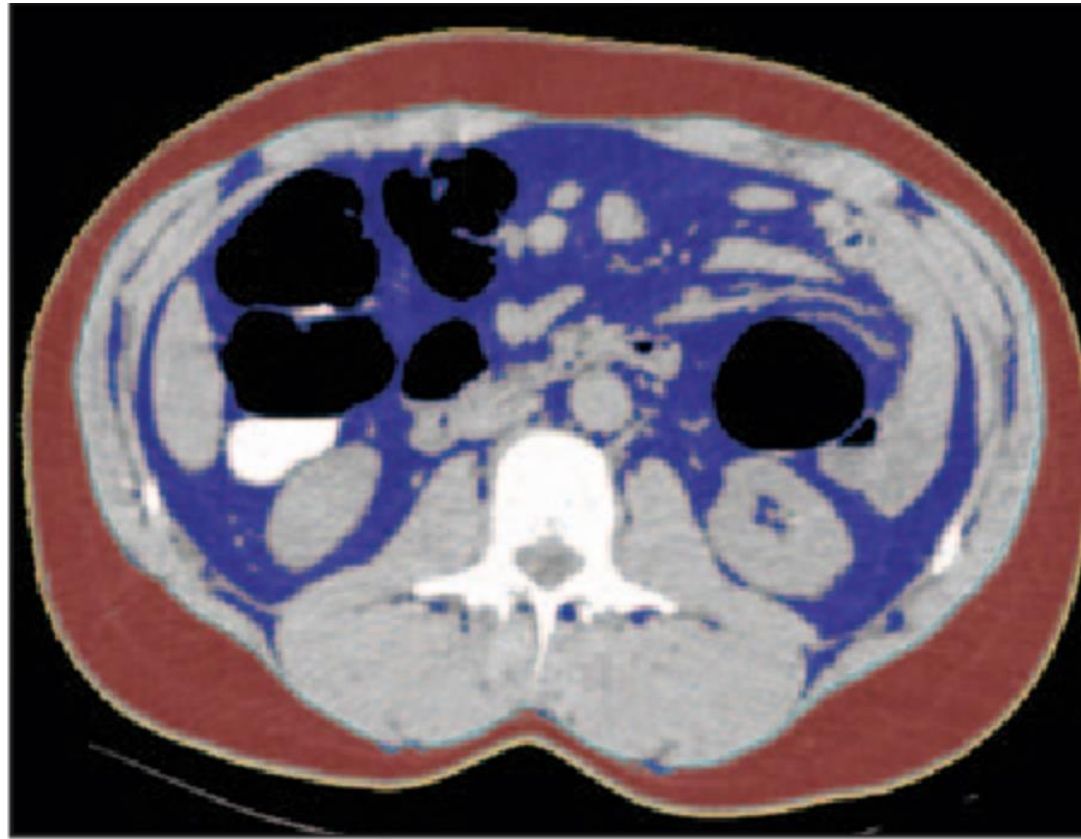
- Noting to disclose.

Background

- BMI and waist circumference do not distinguish between different abdominal fat compartments.
- CT can separately measure **visceral fat, subcutaneous fat, and ectopic fat.**
- These compartments may have different associations with diabetic vascular complications.

Objective

- Summarize the association between CT-measured adiposity and macrovascular complications and microvascular complications.



Study Approach

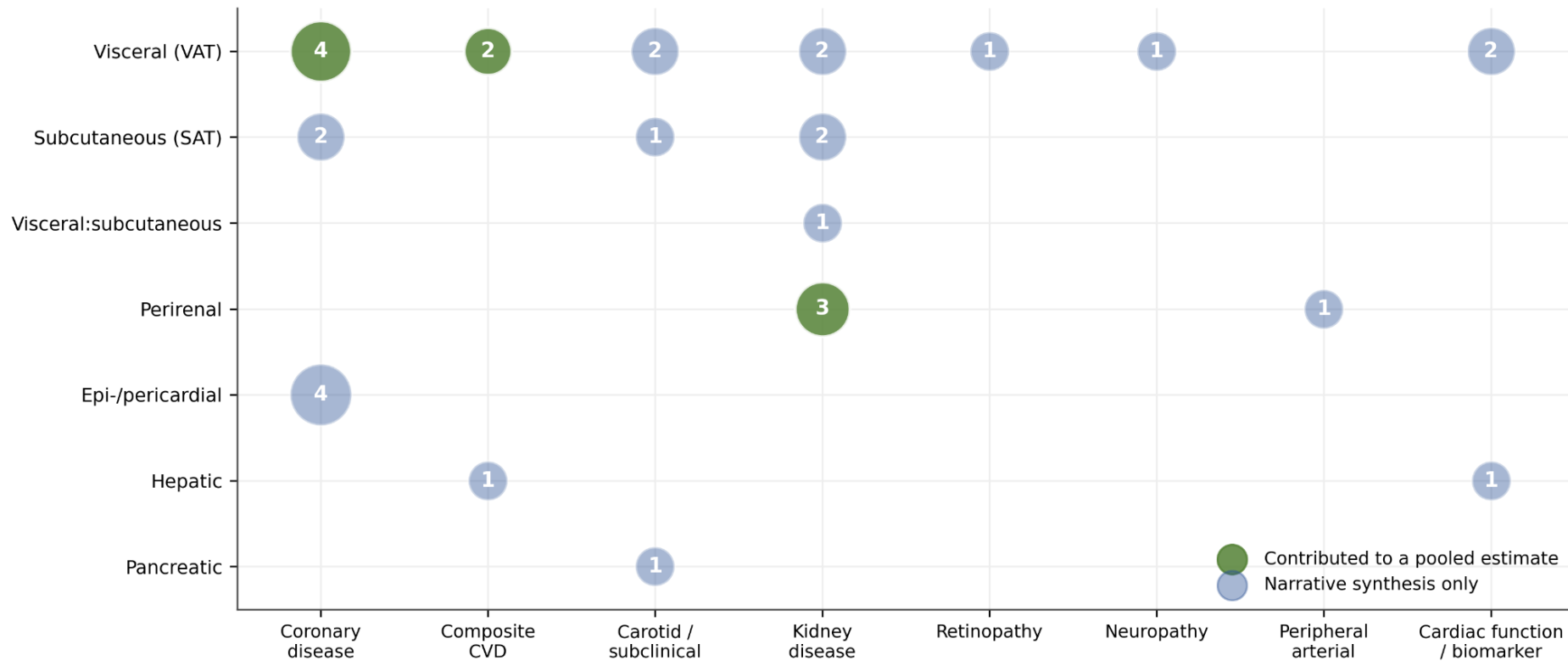
- PRISMA-guided systematic review of MEDLINE, Embase, Scopus, and Web of Science.
 - 960 records identified → 64 full texts assessed → 55 studies included.
- Most studies were cross-sectional, with fewer longitudinal and interventional studies.

Study Approach

- Two exposure–outcome groups supported quantitative synthesis:
 - **Visceral fat and coronary/cardiovascular outcomes**
 - **Perirenal fat and kidney outcomes**

Estimates were standardized per one standard deviation increase in fat measurement, with sensitivity analyses to assess stability.

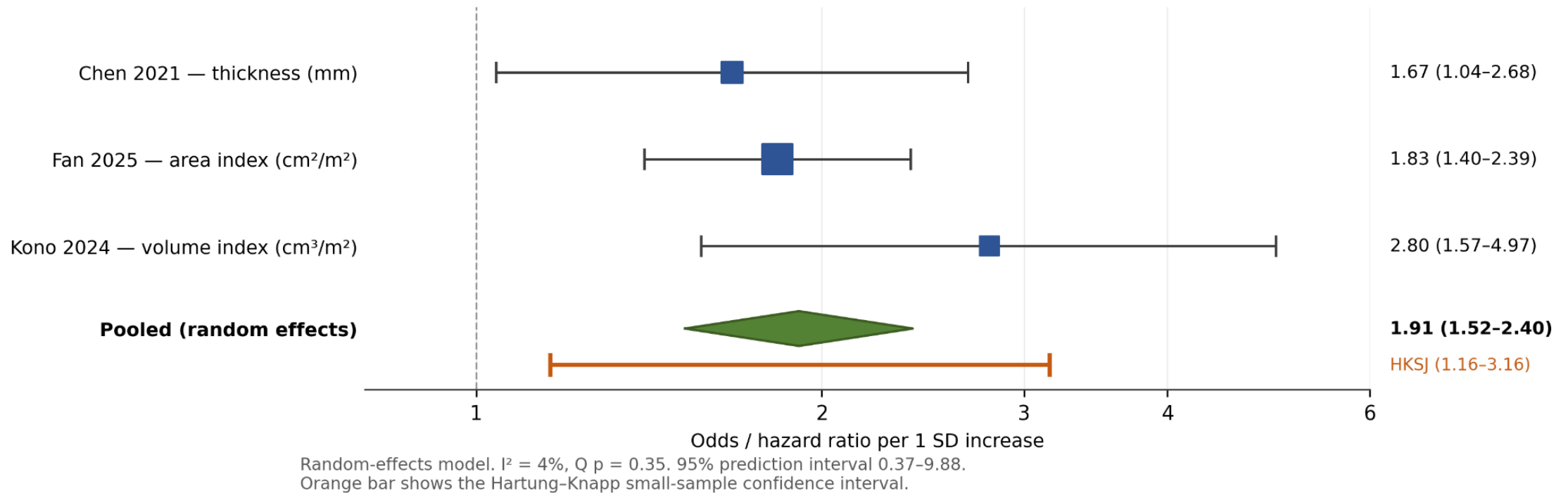
Overall Findings Across Fat Compartments



Circle size and the number inside indicate how many studies addressed each combination. Studies reporting several depots or outcomes appear more than once.

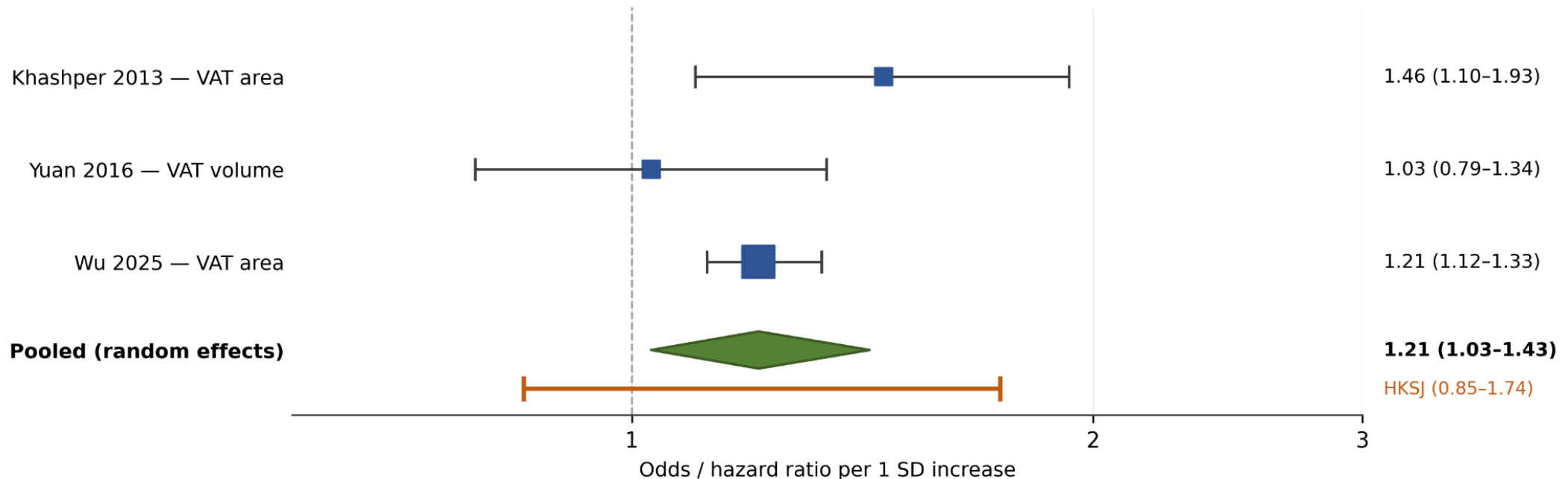
Evidence spans several fat compartments and outcomes. Only two exposure–outcome groups supported quantitative pooling.

Perirenal Fat and Kidney Outcomes



Pooled association: 1.91 per SD (95% CI 1.52–2.40). The association remained significant with the small-sample correction.

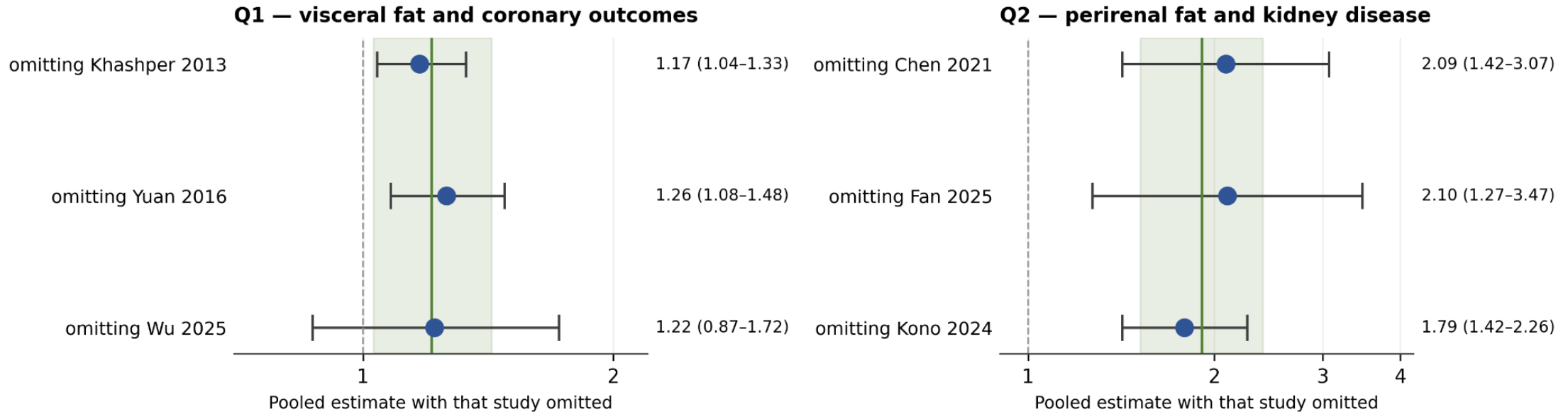
Visceral Fat and Coronary/Cardiovascular Outcomes



Random-effects model. $I^2 = 36\%$, $Q p = 0.21$. 95% prediction interval 0.22-6.70.
The Hartung-Knapp interval crosses the null and the pooled estimate is not robust to omission of any single study.

Pooled association: 1.21 per SD. The small-sample interval crossed the null (0.85–1.74), limiting confidence in the pooled finding.

Sensitivity Analyses



Perirenal–kidney findings remained significant in every omission analysis. Coronary findings lost significance when Wu 2025 was removed.

Discussion and Clinical Implications

- The relationship depends on both fat location and the vascular outcome examined.
- The **perirenal–CKD** association was more stable than the visceral–coronary association in the available analyses.
- Different outcome definitions, population characteristics, and adjustment strategies may explain some of the variability in coronary findings.

Discussion and Clinical Implications

- Predominantly cross-sectional designs limit causal inference, and only three studies contributed to each quantitative analysis.
- Measuring fat on existing CT scans may offer additional risk information, but improvement beyond established clinical risk models remains unproven.

Conclusions

- **Perirenal fat** showed a consistent association with **adverse kidney outcomes** in a small evidence base.
- **Visceral fat** associations with **coronary/cardiovascular outcomes** were less robust.
- Prospective studies with standardized CT measurements are needed to determine whether these markers improve risk prediction and clinical decisions.

Thank You!

