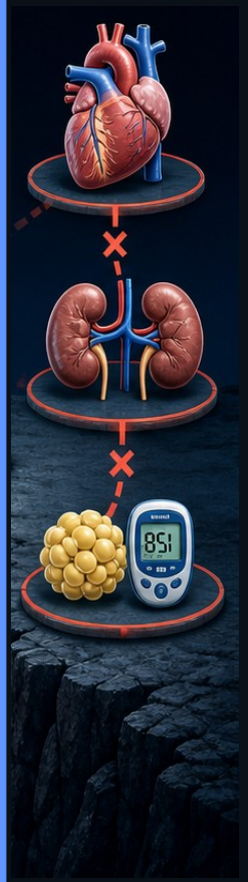


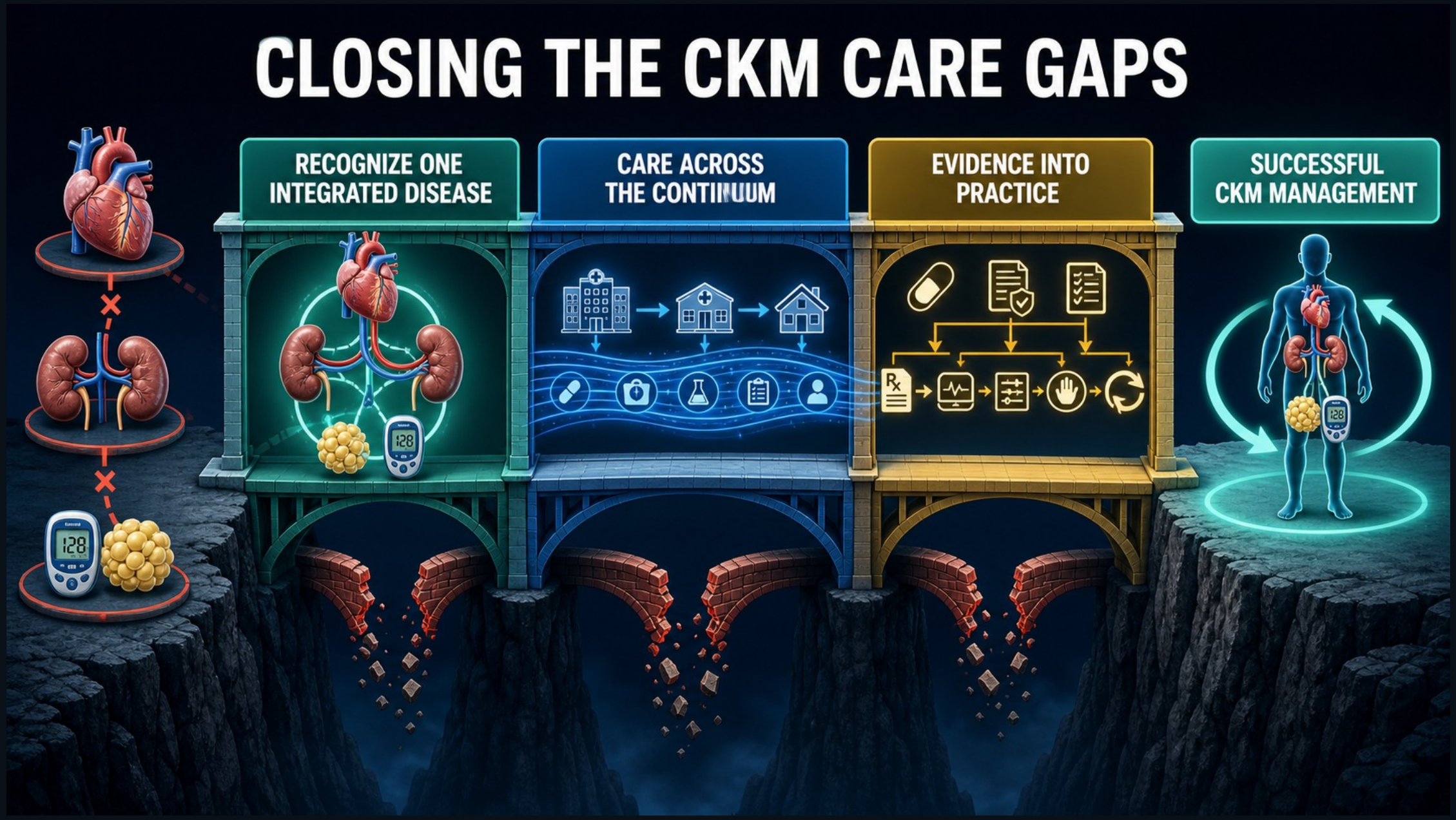
WCHQ CARDIOMETABOLIC ASSEMBLY

Cardiorenal continuity across the hospital–clinic boundary

Andrew C. Bland, MD, FACP, FAAP
Department Chair, Medical Associates Nephrology

HEART ↔ KIDNEY · ONE MANAGEMENT CONTINUUM

Sun Prairie, Wisconsin · August 14, 2026


CLOSING THE CKM CARE GAPS
RECOGNIZE ONE INTEGRATED DISEASE
CARE ACROSS THE CONTINUUM
EVIDENCE INTO PRACTICE
SUCCESSFUL CKM MANAGEMENT
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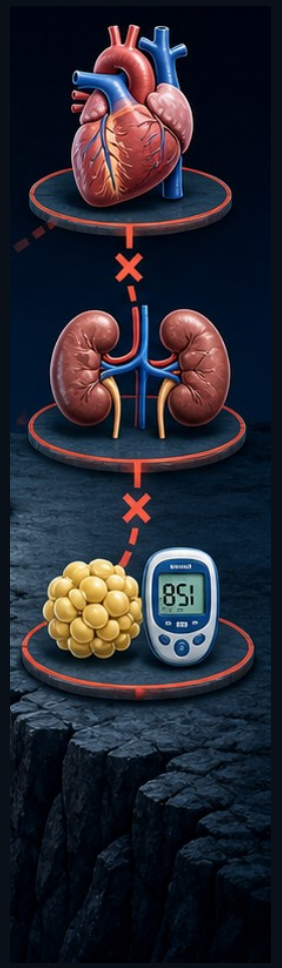
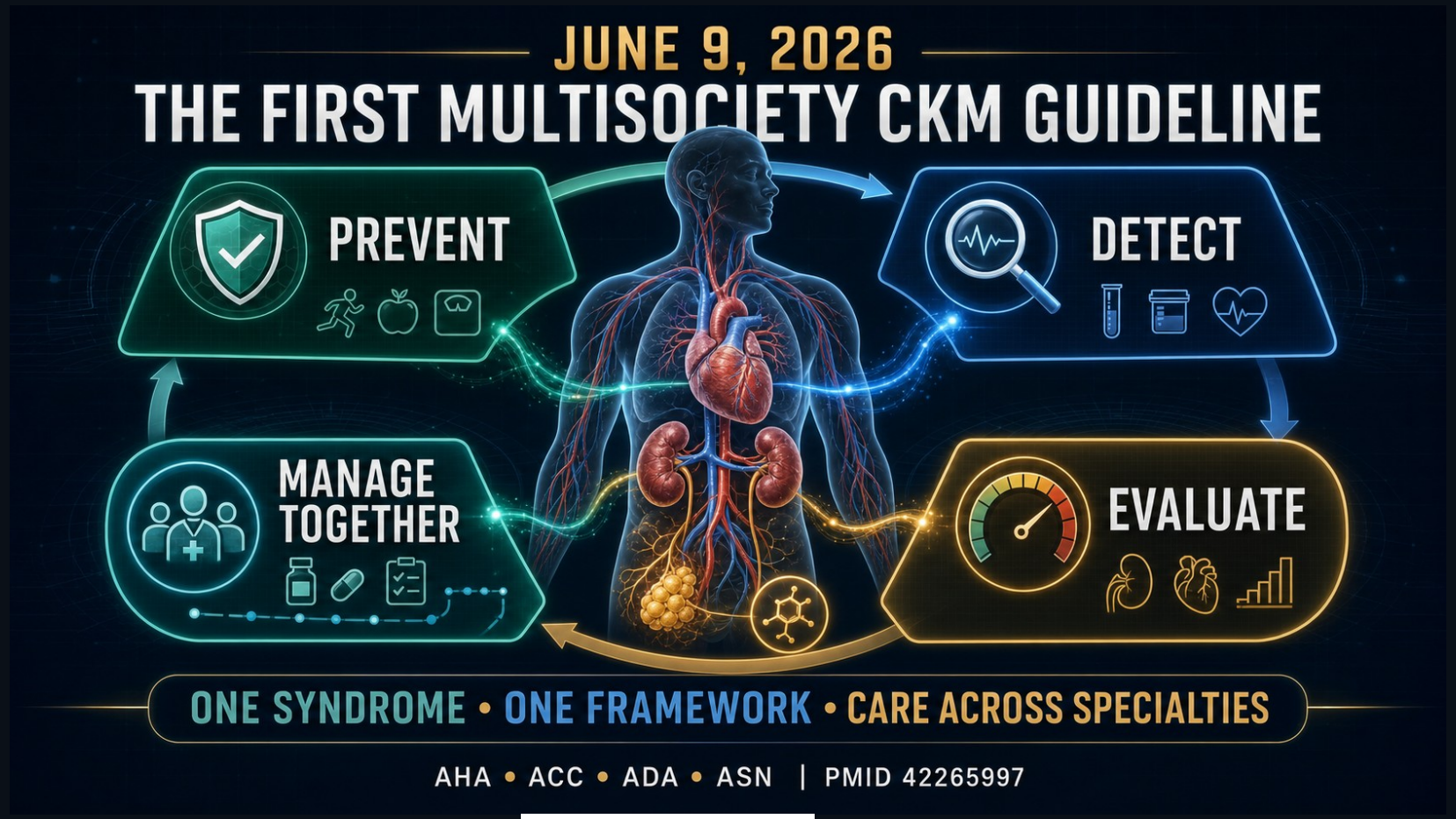
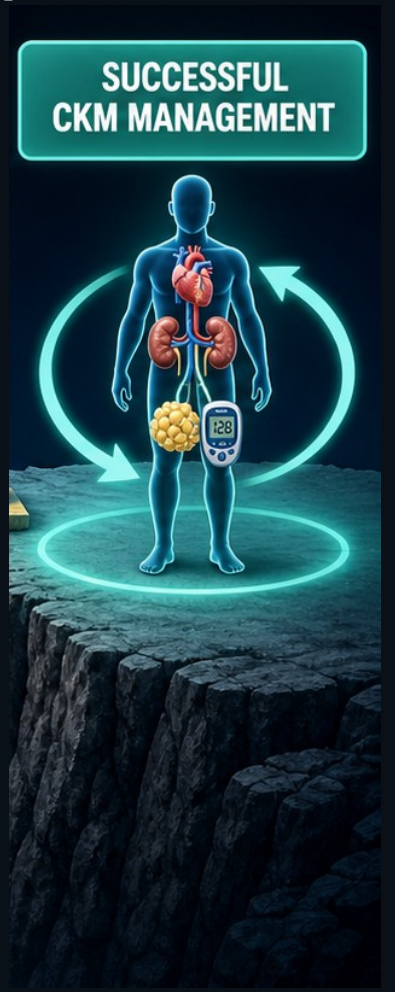
Rx

New Guideline for CKM Released







CKM is one syndrome—and now one care framework

RECOGNIZE + STAGE

One syndrome

- Metabolic risk, obesity/T2D, CKD and CVD are interconnected.
- Stage 0–4: preserved health → metabolic risk/CKD → subclinical → clinical CVD.
- Screen BP, adiposity, glucose, lipids, eGFR and UACR early.

DETECT + TREAT

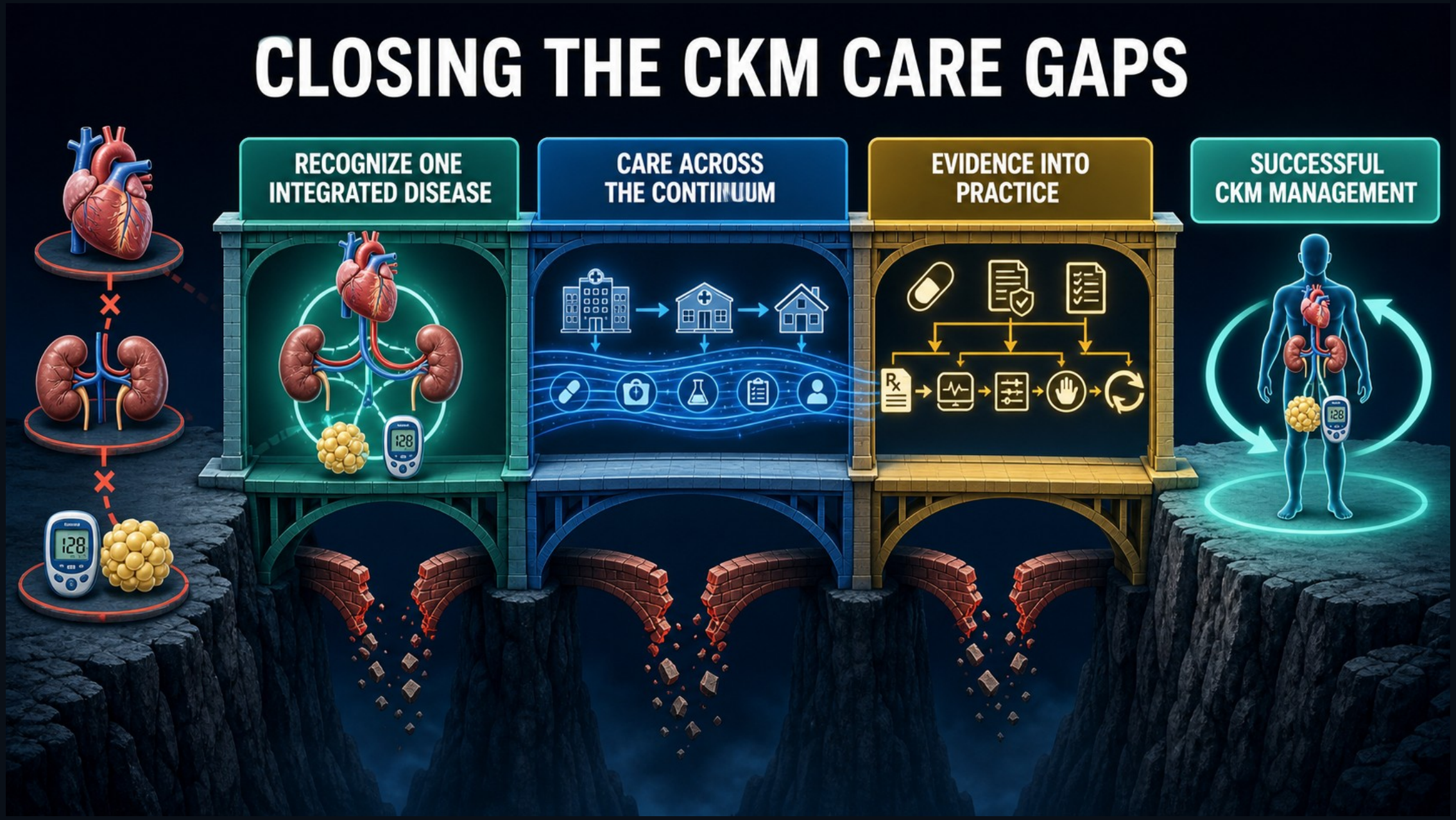
Risk drives care

- PREVENT, CKD severity and comorbidity—not EF or one diagnosis—guide intensity.
- Use multiorgan therapy: lifestyle/weight, RASi, SGLT2i, GLP-1 and nsMRA.
- Match treatment to stage and absolute risk.

COORDINATE + DELIVER

One care team

- One framework spans primary care, cardiology, nephrology, endocrinology and pharmacy.
- High-risk diabetes + CKD + CVD needs interdisciplinary care.
- Use CKM coordination, virtual teams and navigation as complexity rises.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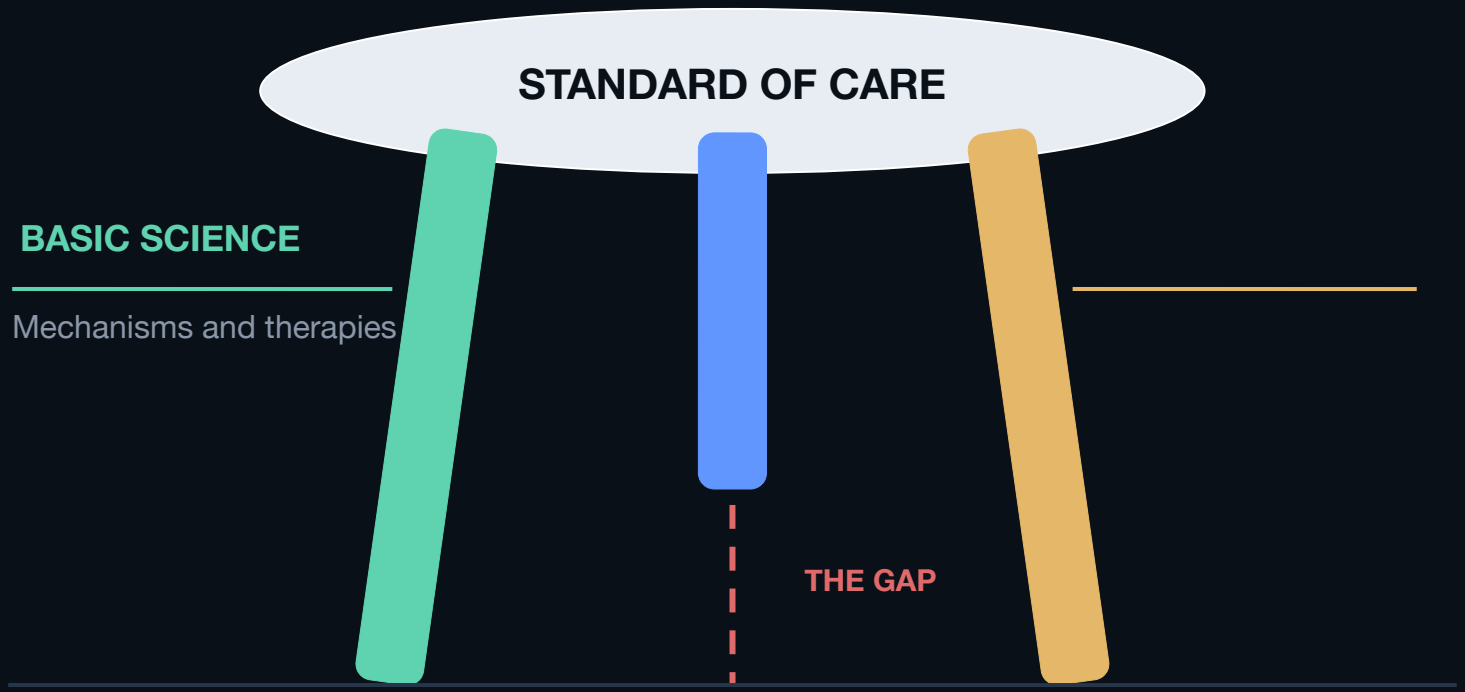
IMPLEMENTATION GAP

The standard of care rests on a short third leg



IMPLEMENTATION SCIENCE
Getting proven care to every patient, reliably

THE IMPLEMENTATION GAP

30,000 FT

DETAILED GUIDANCE

1,000 FT

LAND THE PLANE

FIND THE AIRPORT

ALIGN THE APPROACH

GEAR DOWN

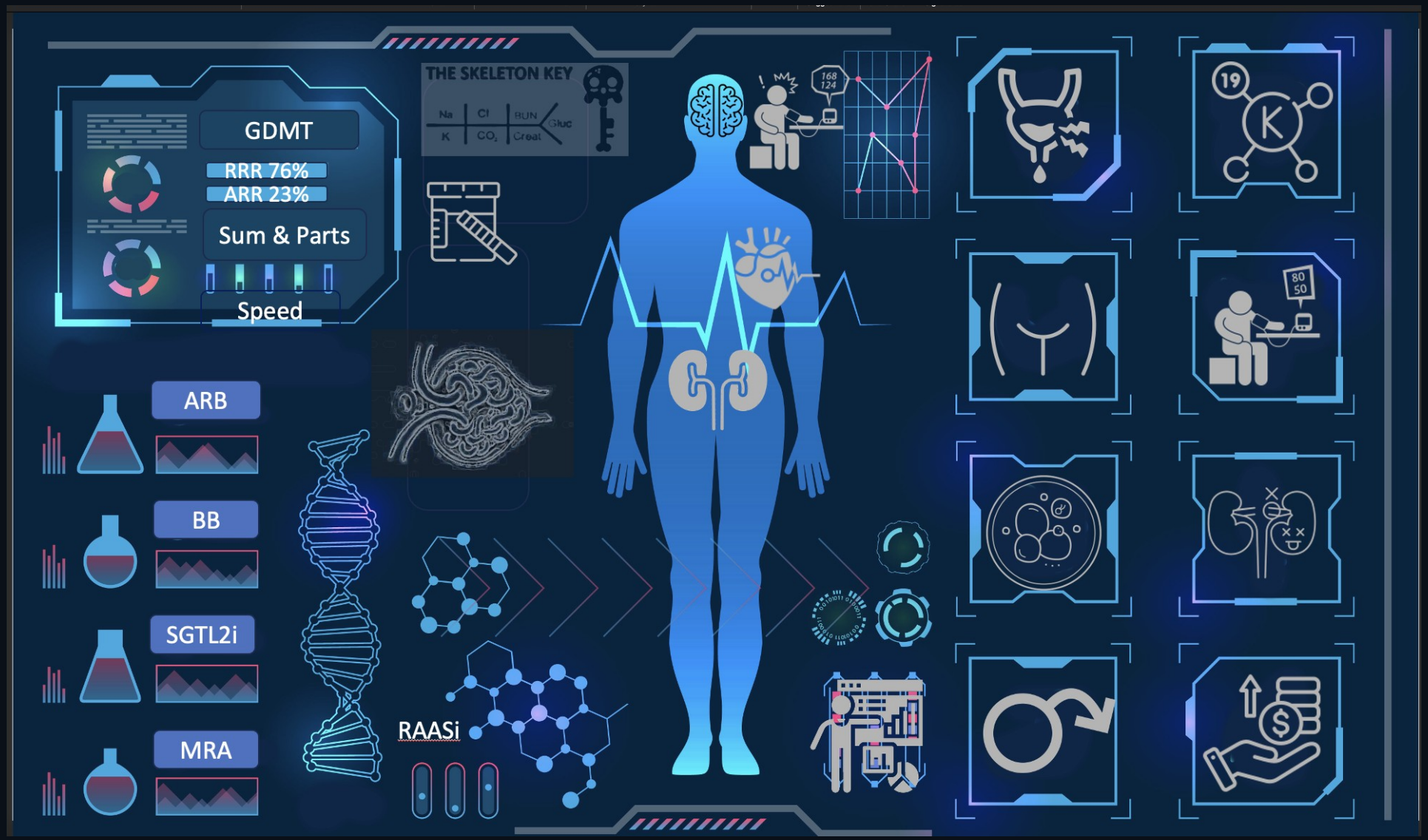
SET FLAPS

MANAGE AIRSPEED

TOUCHDOWN & FOLLOW THROUGH

GUIDELINES DEFINE WHAT TO DO • IMPLEMENTATION REQUIRES HOW

Translating GDMT from evidence to practice



APPENDIX

For HFrEF, a beta blocker is not automatically an evidence-based beta blocker

HFrEF OUTCOME EVIDENCE

Carvedilol · metoprolol succinate · bisoprolol

These agents—not any beta blocker—meet the HFrEF mortality/morbidity GDMT measure.

COMMON MEASUREMENT ERROR

Counting atenolol as HFrEF GDMT

Atenolol may have another indication. It does not satisfy the evidence-based HFrEF beta-blocker measure.

CONTINUITY RULE

Document the indication

Medication reconciliation must preserve why the drug is used—not only the name and dose.

The measure is evidence-based HFrEF beta blocker when indicated—not generic beta-blocker exposure.

2022 AHA/ACC/HFSA guideline · PMID 35363499

FRAMEWORK

Decongest · Connect · Stabilize

DECONGEST

DECONGEST

Define the physiologic phase and treatment target.

CONNECT

CONNECT

Transfer therapeutic intent across every setting.

STABILIZE

STABILIZE

Close the monitoring, titration, hold and restart loops.

One framework. Three distinct failure modes.
Each requires a different reliability measure.

APPENDIX

Loop equivalence is a starting point—not the answer

FUROSEMIDE

40 mg PO ≈ 20 mg IV

Approximate conversion
Oral absorption can be delayed
Verify the patient's measured response

BUMETANIDE

1 mg PO ≈ 1 mg IV

Approximate conversion
Generally more predictable oral delivery
Still verify response

Gut edema · prior exposure · kidney function · route · measured diuretic response

Michigan Medicine loop-equivalence table · furosemide absorption: PMID 3970471

STABILIZE

MRA selection follows phenotype. Monitoring follows risk.

STEROIDAL MRA

Spironolactone / eplerenone

HFrEF and selected phenotypes

Spironolactone active metabolites persist ~14–17 h
K/Cr check within 1 week after start or titration

NONSTEROIDAL MRA

Finerenone

Phenotype-specific evidence and indication

Different PK; still requires labeled potassium and kidney monitoring

SHARED RULE

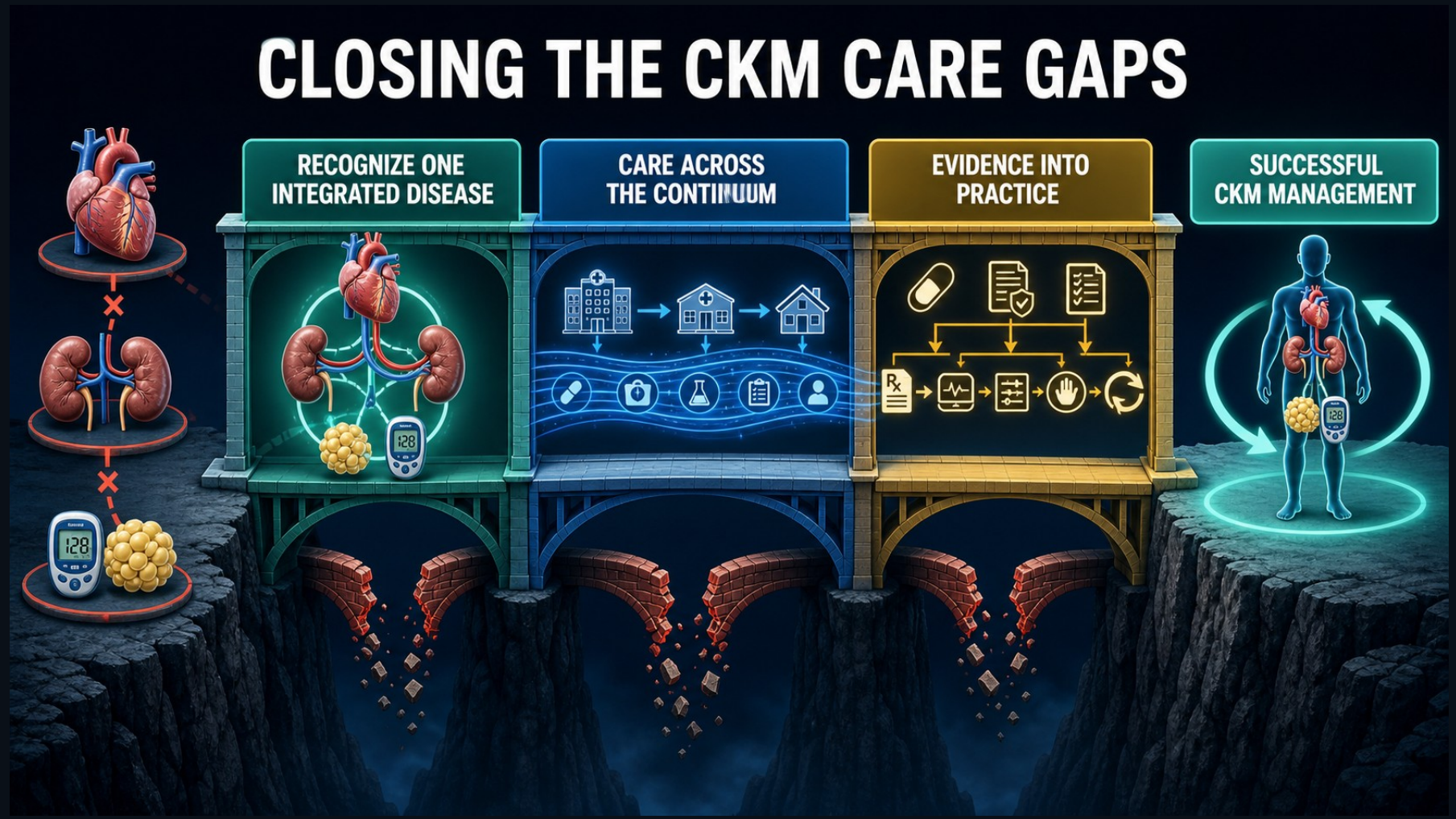
Choose. Monitor. Reassess.

No MRA is monitoring-free

Choose by indication, monitor on time, and document the result owner

The operational difference is not MRA versus no MRA—it is choosing by phenotype and reliably closing the right monitoring loop.

Spironolactone label · finerenone trials: PMIDs 33264825, 34449181 · use current FDA labeling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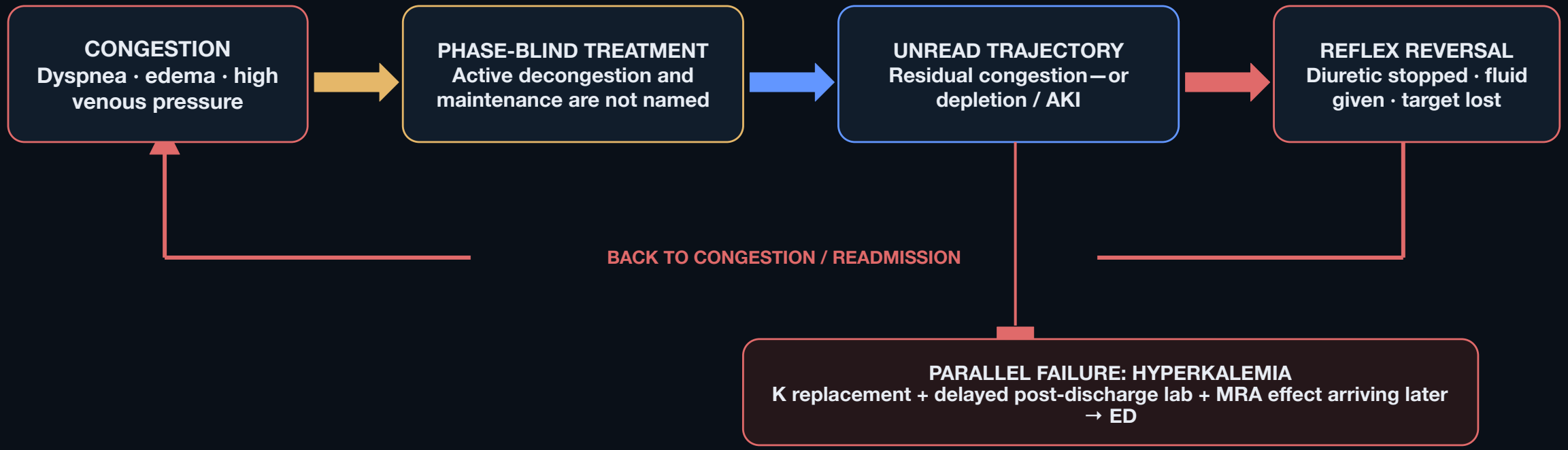
The Gap between hospital and office information is real and a challenge

CONNECT

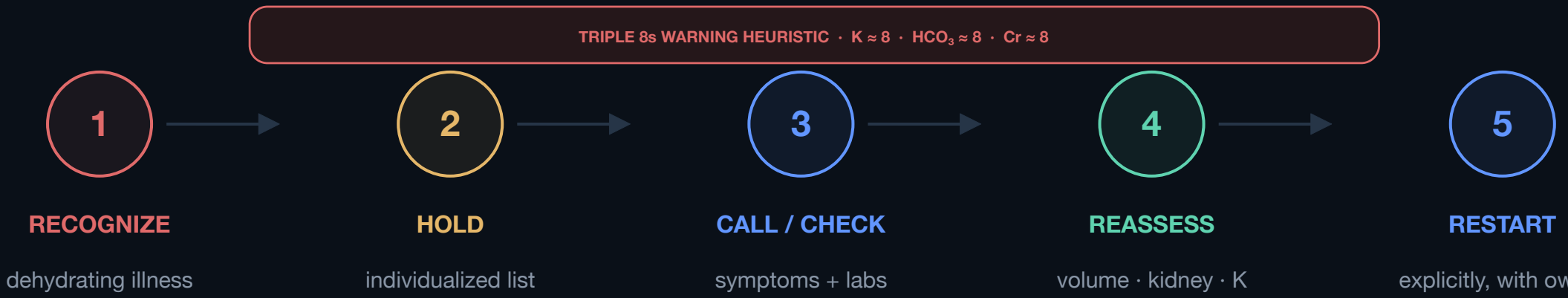
The merry-go-round is a systems failure—not a bad actor



BREAK BOTH LOOPS: named phase · target weight · diuretic intent · lab date · result owner · hold/restart path

STABILIZE

Every temporary hold needs an explicit restart path



Sick-day management is a temporary safety pathway

Individualize temporary holds to illness and volume state. Every hold needs a trigger to reassess and an explicit owner to restart—preventing avoidable ED visits and permanent therapeutic loss.

4 Blinded Clinicians and the Elephant

CONNECT

One patient. Four partial views.

MISSING CONTEXT
‘That medicine nearly killed you’ → durable GDMT refusal

SHARED CONTEXT
Indication + phase + trajectory + restart owner → safe hold → intentional restart

CONNECT

A medication list says what. The continuity contract says why.

The contract travels with the patient. The visit is one checkpoint—not the intervention itself.

The same creatinine direction can mean different things

TRAJECTORY 1

Congestion + kidney improve

ACTIVE DECONGESTION
Congestion falls; kidney function improves

Continue toward the target.

TRAJECTORY 2

Congestion improves; Cr rises

ACTIVE DECONGESTION
Congestion falls; creatinine may rise

Interpret the whole response.

TRAJECTORY 3

Persistent congestion + kidney injury

REASSESS PHASE + DELIVERY
Congestion persists; kidney function worsens

Escalate evaluation.

Creatinine is a response signal. Congestion, perfusion, flow, symptoms and treatment phase establish the phenotype.

PMID 29352071

STABILIZE

A monitoring instruction is not a monitoring plan

AMBIGUOUS

“Labs before follow-up”

No order
No date
No named result owner
No planned response

OPERATIONAL

Order · date · owner

K/Cr ordered before discharge
First-week collection window
Named result owner
Predefined escalation path

Reliability begins before the patient leaves the hospital.

FDA Aldactone label · potassium within 1 week after initiation or titration

APPENDIX

Cohort and outcome definitions

FIELD	DEFINITION	RESULT
POOLED WINDOW	2025 through 2026-H1	810 discharges / 577 patients
CARDIORENAL	cardiac + kidney diagnosis history	65 discharges
FOLLOW-UP	≥1 nephrology office visit ≤28 days	2 / 32 readmitted
NO FOLLOW-UP	no nephrology office visit ≤28 days	11 / 33 readmitted
EFFECT MODIFICATION	full-cohort interaction term	p=0.0046

OPENING

The appointment is visible. The work around it is not.

The analytic exposure is simple:

≥1 NEPHROLOGY OFFICE VISIT WITHIN 28 DAYS

The clinical intervention is larger

- interpret congestion and perfusion
- order and own first-week labs
- preserve medication intent
- make holds temporary
- document the next decision

The visit is a marker. Continuity is the mechanism we need to build and test.

STABILIZE

The first 28 days are a sequence—not a visit

The visit becomes valuable because earlier observations and labs arrive with a decision owner.

Attendance alone cannot decongest, titrate, monitor potassium, or restart therapy.

Author exposure definition: office visit ≤28 days

BUILD

Minimum viable continuity bundle

DECONGEST

DECONGEST

Define the physiologic phase and treatment target.

CONNECT

CONNECT

Transfer therapeutic intent across every setting.

STABILIZE

STABILIZE

Close the monitoring, titration, hold and restart loops.

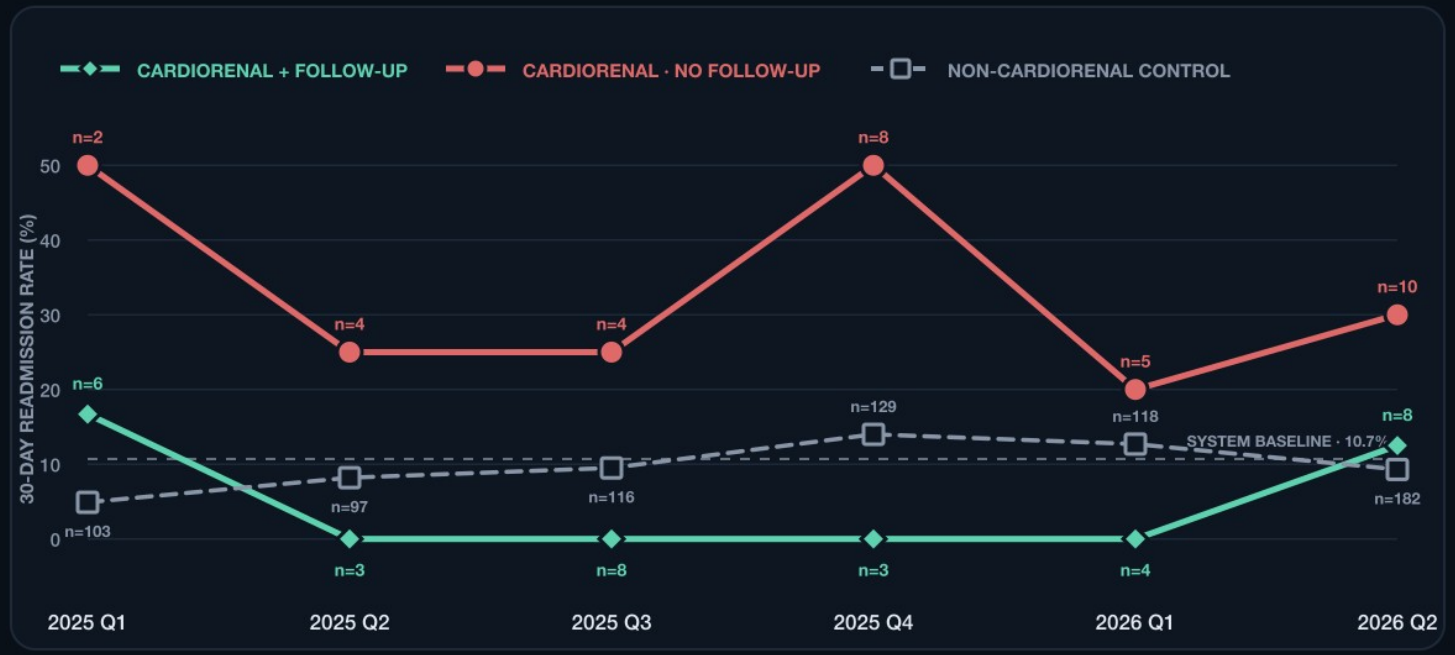
BEFORE DISCHARGE	phase + perfusion/flow state + target weight + diuretic plan + K/Cr order/date
AT RESULT	named owner interprets weight, congestion, kidney trajectory and potassium; closes the result
AT FOLLOW-UP	adjust / hold / restart + next checkpoint documented for patient, clinic and ED

Readmission Prevention

DATA

The separation persists quarter after quarter

30-day all-cause readmission · pooled 2025–2026 H1 · denominators shown at each point



POOLED RESULT

6.2%
WITH FOLLOW-UP

VS

33.3%
NO FOLLOW-UP

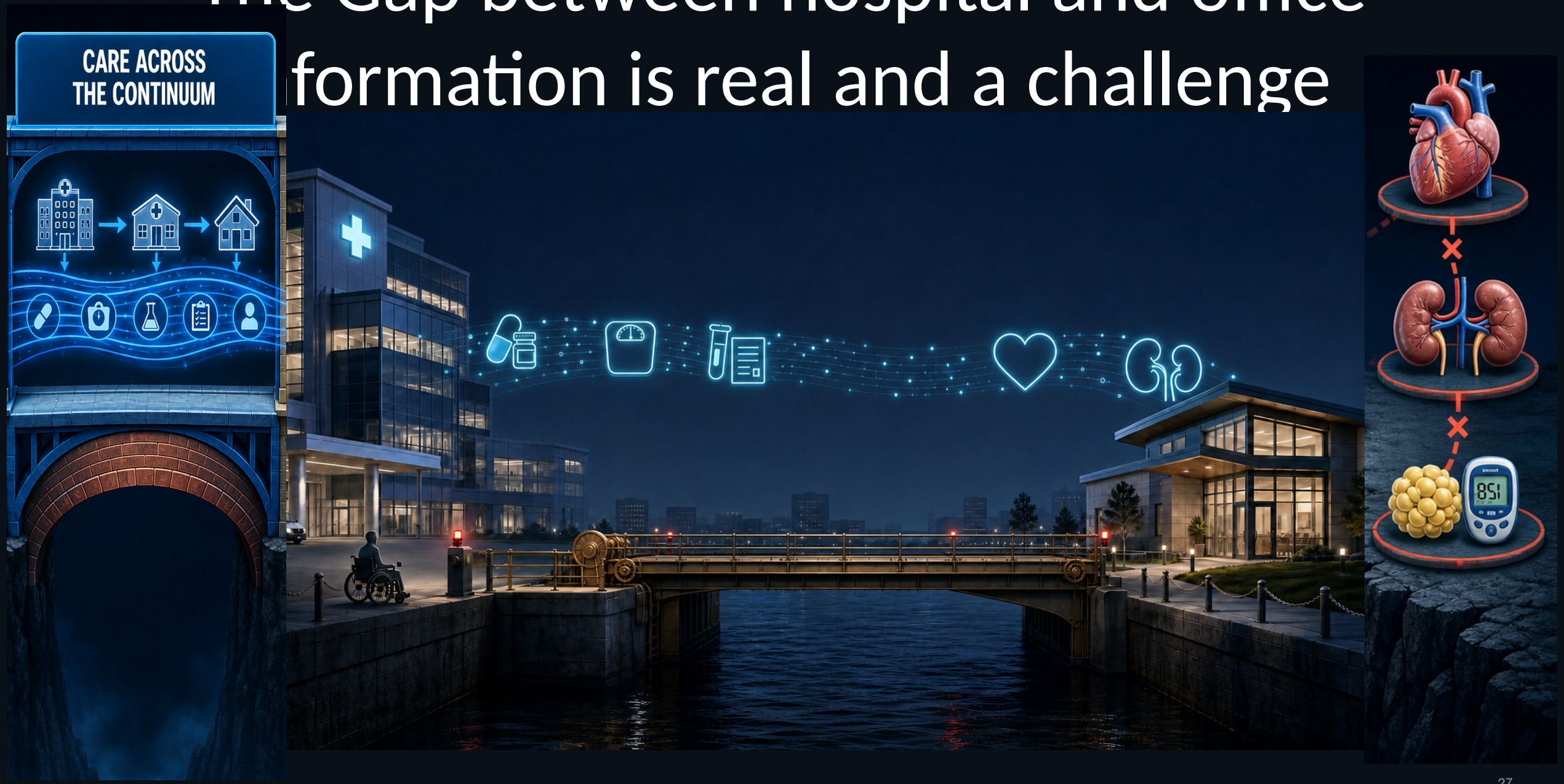
ARR 27.1 points
Fisher p=0.011

Follow-up = ≥1 nephrology office visit within 28 days. Quarterly values are descriptive; small cardiorenal denominators limit point-by-point inference.

The pooled comparison and full-cohort interaction are the primary statistical evidence.

Author data · 810 discharges · 577 patients · observational

The Gap between hospital and office formation is real and a challenge

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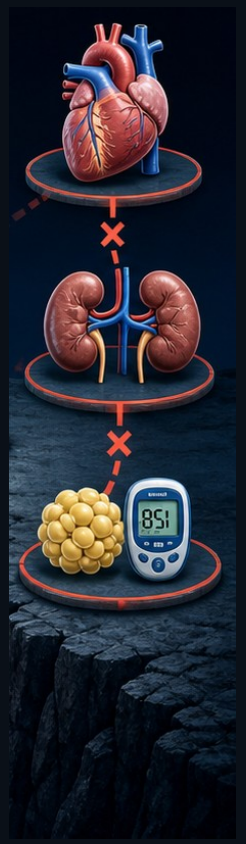
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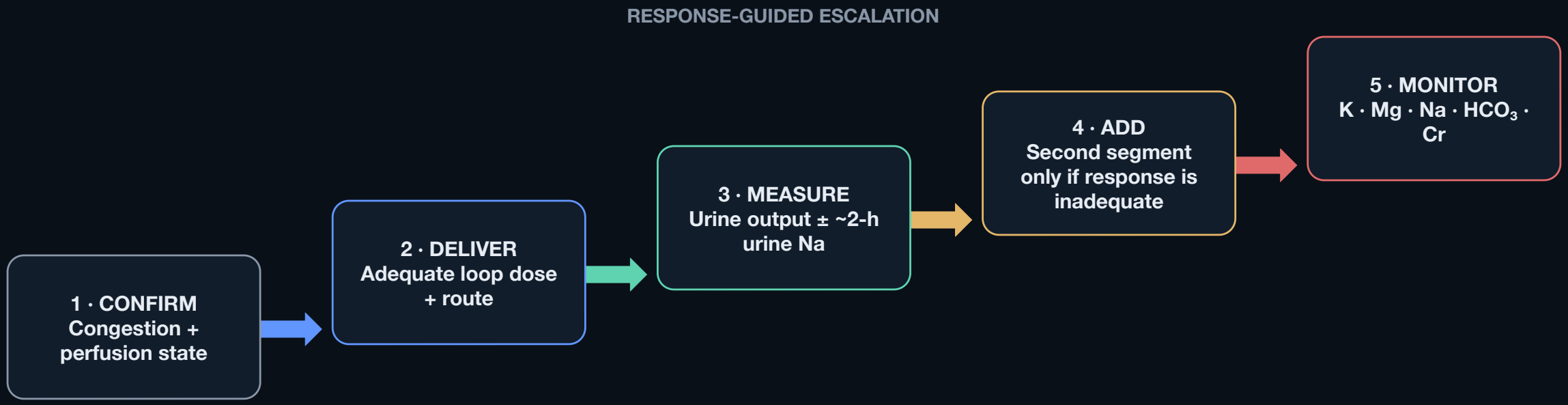
APPENDIX

Sequential blockade should follow measured response



Evidence boundary: guidelines support escalation after inadequate loop response; exact 2-hour sodium thresholds and specific 2-, 3-, or 4-agent combinations require a local protocol and patient-level judgment.

Use RHC when uncertainty is blocking the next decision

1 Persistent dyspnea / congestion

2 Bedside state still uncertain

3 Measure RA · PCWP · CI

4 Name the physiology

5 Treat physiology—not assumptions

WHAT THE NUMBERS MAY REVEAL

CONGESTION-DOMINANT
High RA/PCWP · adequate CI
Continue decongestion if perfusion is intact

RESTRICTIVE / LOW OUTPUT
High filling pressure · low CI
Evaluate restrictive or infiltrative physiology

CONSTRICTIVE CLUES
Respiratory ventricular interaction
Focus the constriction evaluation

RHC defines physiology. Etiologic diagnosis—including amyloidosis—requires separate evaluation.

APPENDIX

RHC can separate three management phenotypes when decongestion is failing

CONGESTION-DOMINANT

High filling pressure · adequate CI

Persistent venous congestion supports continued or intensified decongestion when perfusion remains adequate.

RESTRICTIVE / LOW OUTPUT

High filling pressure · low CI

A normal EF can coexist with low cardiac index. Consider restrictive or infiltrative evaluation in the right context.

CONSTRICTIVE CLUES

Ventricular interaction

Discordant respiratory ventricular changes and pressure patterns can support focused constriction evaluation.

RHC defines the physiology and the next treatment decision; it does not by itself diagnose amyloidosis.

Pressure, perfusion, and flow answer different questions

VENOUS PRESSURE

RA / CVP

Back-pressure can impair renal function.

Question: is congestion still driving the kidney signal?

PERFUSION

MAP – RAP

Renal perfusion pressure ≈ MAP – RAP
A contextual approximation—not a universal target.

Question: is end-organ perfusion intact?

FORWARD FLOW

Cardiac index

EF does not equal flow.

Question: is low output limiting the plan?

Lower blood pressure may be tolerated only while forward flow and end-organ perfusion remain intact.

PMIDs 19215833, 35611889

A normal EF can conceal critically low forward flow

50 × .60 = 30 mL · 30 × 70 = 2.1 L/min · 2.1 ÷ 1.8 = 1.2 L/min/m²

EF is a fraction. Cardiac index is delivery.

DATA

A 27-point absolute difference in 30-day readmission

NEPHROLOGY FOLLOW-UP

6.2%

2 readmissions / 32 discharges

NO NEPHROLOGY FOLLOW-UP

33.3%

11 readmissions / 33 discharges

ARR 27.1 percentage points · Fisher p=0.0112 · descriptive NNT-equivalent 3.7

Author data · pooled 2025–2026 H1 · observational

THE SIGNAL

6.2% vs 33.3%

An association worth testing—not a causal conclusion.

DECONGEST

DECONGEST

Define the physiologic phase and treatment target.

CONNECT

CONNECT

Transfer therapeutic intent across every setting.

STABILIZE

STABILIZE

Close the monitoring, titration, hold and restart loops.

The appointment opens the door. Continuity is the intervention.

DATA

Strong signal. Limited causal certainty.

POOLED COMPARISON

6.2% vs 33.3%

Cardiorenal follow-up vs no follow-up
Fisher exact p=0.0112

SUPPORTS ACTION

Direction repeats

Quarterly separation is descriptive
Small denominators; pooled signal leads

DOES NOT PROVE

Causation—or one mechanism

Selection, severity, access, survivorship and treatment continuity may contribute

Use the signal to justify a prospective continuity bundle—not to declare a treatment effect.

Author data · pooled Fisher p=0.0112 · full-cohort interaction p=0.0046

EXPERIENCE

Three health systems exposed the same boundary failure

HSHS DECATUR

≈1 in 3 → 4%

Enhanced navigation + home monitoring
Earlier five-month pilot

UNITYPOINT

Workflow + GDMT

Identification, order-set use and discharge measurement
No readmission outcome claimed

CURRENT COHORT

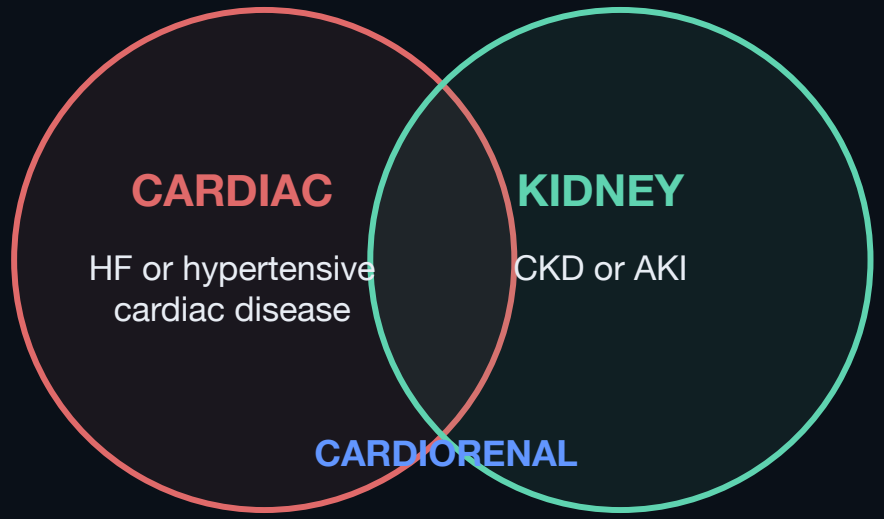
6.2% vs 33.3%

Pooled observational cardiorenal analysis
Interaction p=0.0046

Different programs. The same missing transfer of therapeutic intent.

Internal program records · different eras and designs

Cardiorenal is a bidirectional phenotype—not heart failure alone



What the definition does

- identifies coexisting cardiac and kidney disease
- supports a reproducible cohort
- does not assume identical hemodynamics
- does not substitute diagnosis codes for bedside state

BUILD

Measure reliability before claiming outcome improvement

01	CONTRACT	% eligible discharges with phase + target + owner
02	LAB ORDER	% with K/Cr order placed before discharge
03	RESULT CLOSED	% results acknowledged within 24 hours
04	FOLLOW-UP	% completing nephrology visit ≤28 days
05	RESTART	% holds with documented restart decision · balancing: severe K/AKI ED events

THEN TEST READMISSION + SAFETY PROSPECTIVELY

APPENDIX

Infusion and ototoxicity: avoid universal thresholds

BOLUS VS INFUSION

No universal winner

Primary symptom and creatinine outcomes do not support a single default strategy.

10 MG/HOUR RCT

More urine ≠ better relief

More urine output
No better symptom relief
More hypokalemia
PMID 23793945

OTOTOXICITY

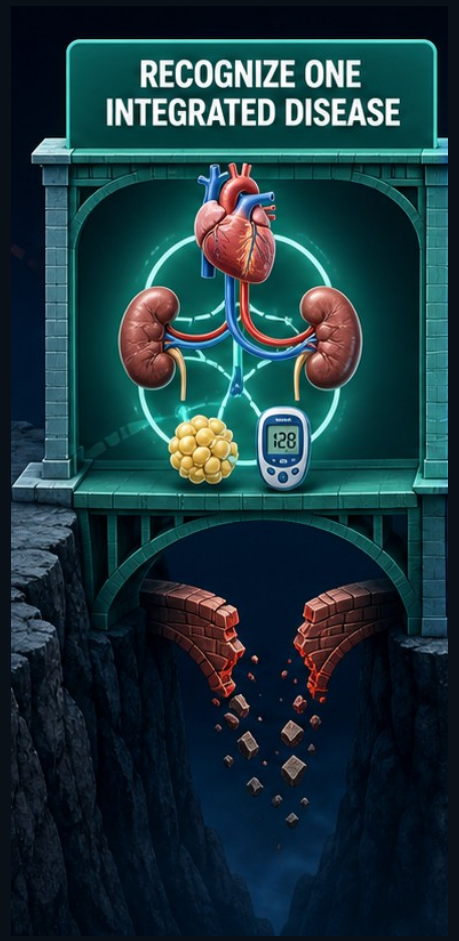
Risk is contextual

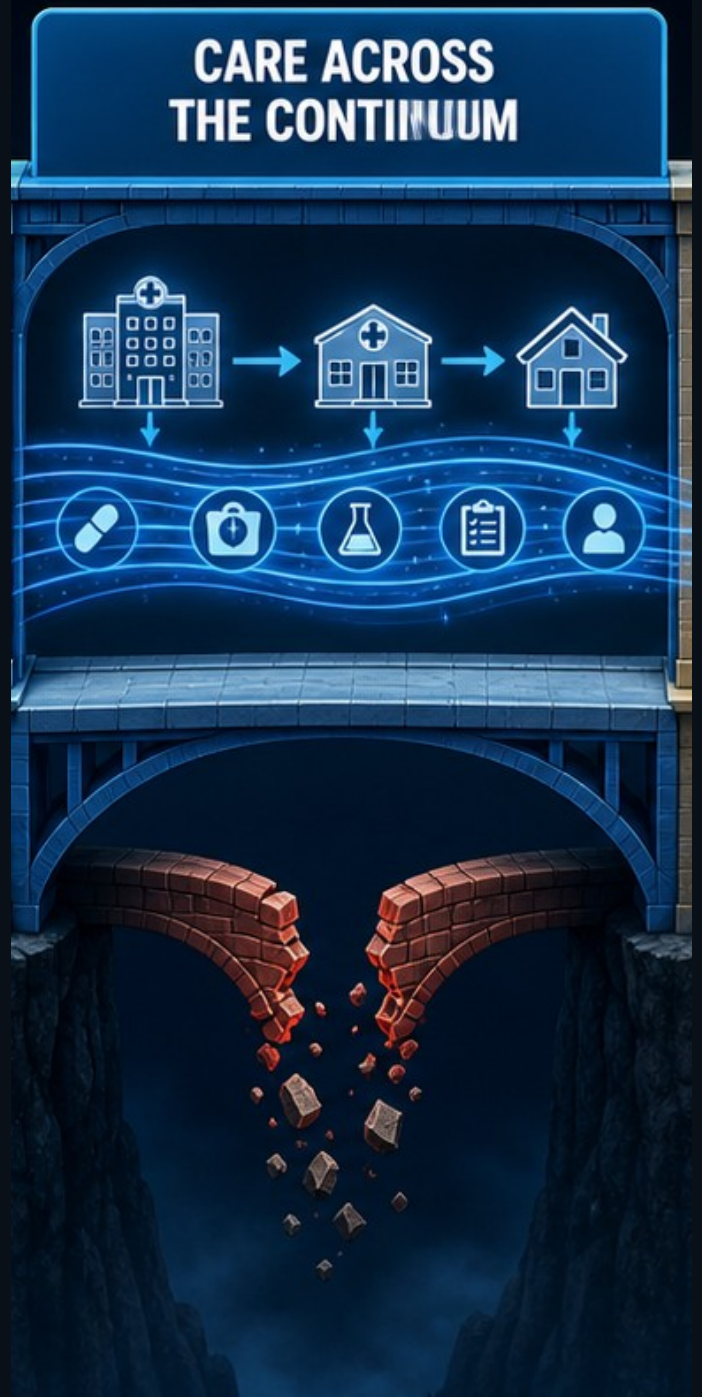
Rate · dose · renal impairment · hypoproteinemia · other ototoxins

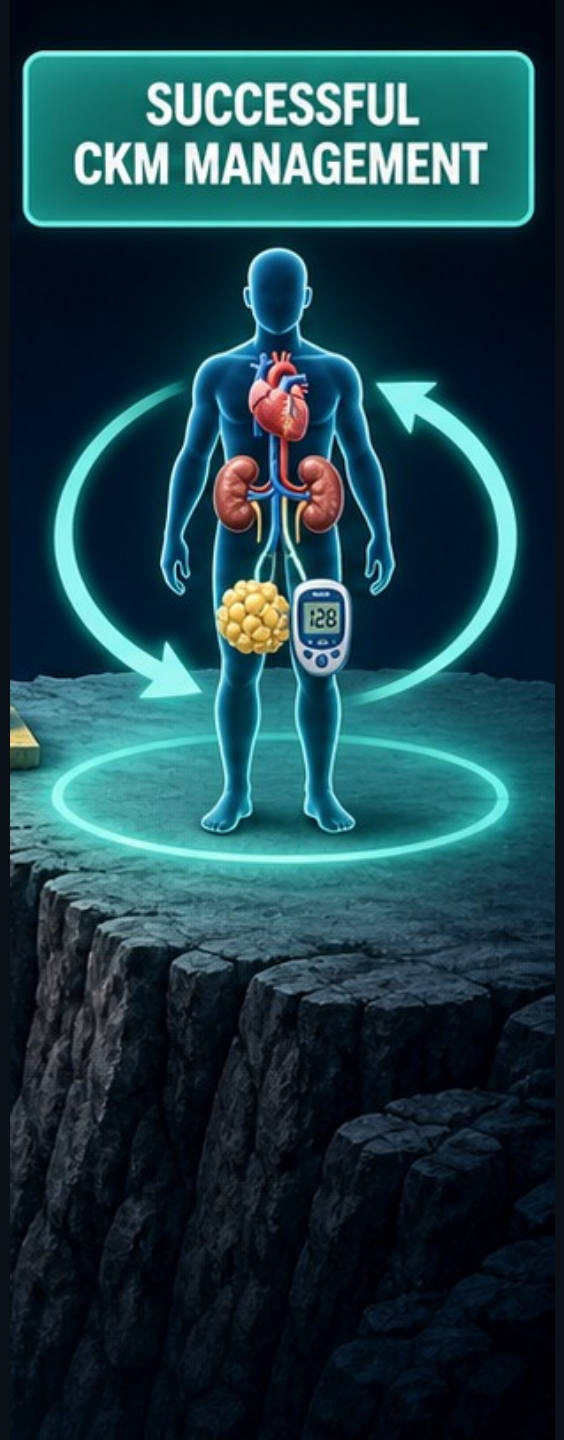
Use the label + patient context

Evidence guardrails—not a dosing recommendation.

DOSE: PMID 21366472 · 10 mg/hour RCT: PMID 23793945 · FDA furosemide label


RECOGNIZE ONE
INTEGRATED DISEASE


CARE ACROSS
THE CONTINUUM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