



Heart Failure with Improved Ejection Fraction: The Evidence Against Stopping Therapy That Appears to Have Worked

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Abstract

When left ventricular ejection fraction recovers from below 40% to 40% or higher on guideline-directed medical therapy, patients and clinicians alike are often tempted to see this as cure rather than remission and to ask whether treatment can be safely withdrawn. The randomized evidence says otherwise. In TRED-HF, a pilot trial that phased out heart failure medication in 25 patients with recovered dilated cardiomyopathy while 26 continued therapy, 11 (44%) of the withdrawal group relapsed within 6 months versus none of the continuation group ($p=0.0001$). Long-term follow-up at a median of 6 years found that 33 of the original 51 patients (65%) had relapsed overall, with a 5-year relapse rate of 61% measured from enrollment and 39% measured from the trial's end -- risk that keeps accruing well beyond the original 6-month endpoint. A Swedish national registry of 8,728 patients with improved ejection fraction confirmed the signal outside a trial setting: withdrawing renin-angiotensin-system inhibitors or neprilysin inhibitors was associated with a 38% higher hazard of cardiovascular death or heart failure hospitalization at 1 year (hazard ratio 1.38, 95% CI 1.08-1.77), and withdrawing a mineralocorticoid receptor antagonist with a 36% higher hazard (1.36, 95% CI 1.07-1.73), though beta-blocker withdrawal showed no such signal overall. A separate small trial, CATHEDRAL-HF, found that withdrawing every drug except a beta-blocker produced a far lower recurrence rate (14.8% at 52 weeks) than TRED-HF's full withdrawal, suggesting the withdrawal strategy itself, not just the underlying disease, shapes the risk. Candidate predictors of relapse -- older age, higher NT-proBNP, more baseline medications, no mineralocorticoid receptor antagonist use, and reduced peak radial strain -- remain hypotheses from a single small trial, not a validated way to select patients for safe withdrawal. This review sets the real trial and registry evidence against the understandable clinical impulse to de-prescribe once a patient looks recovered.

Keywords: Heart failure with improved ejection fraction; Guideline-directed medical therapy; Treatment withdrawal; Dilated cardiomyopathy; Relapse

Introduction

A meaningful share of patients treated for heart failure with reduced ejection fraction see their left ventricular ejection fraction climb from well below 40% into the normal range on guideline-directed medical therapy. This trajectory -- heart failure with improved ejection fraction, HFimpEF -- now

accounts for an estimated 20% to 30% of the treated heart failure population, though reported prevalence ranges from 9% to 52% depending on the definition and cohort used [8]. For the patient who feels well, takes four or more daily medications, and is told their heart function looks normal, the request to stop treatment is entirely understandable. For the

clinician, the same request raises a genuinely difficult question: is this recovery, or is it remission that depends on the drugs continuing to work?

This review reads the trial that answered that question directly -- TRED-HF -- alongside its long-term follow-up, a national registry study that tested the same question in unselected practice, a second small trial that tested a partial rather than complete withdrawal strategy, and the terminology and guidance that have followed, to set the honest evidence against stopping therapy that appears to have worked next to the equally honest desire, shared by patients and clinicians, to do exactly that.

The tension is not academic. Guideline-directed medical therapy for heart failure carries real burdens -- pill count, cost, hypotension, renal monitoring -- and a patient whose heart function has normalized has a reasonable claim to ask whether all of that remains necessary. The evidence reviewed here answers that question with real numbers rather than reassurance, and the numbers do not, on the whole, support the intuition that a recovered ejection fraction means recovered disease.

Material and Methods

This is a narrative review drawing on the primary publication of TRED-HF, its long-term follow-up report, a pre-specified secondary analysis of heart rate during withdrawal, a Swedish national heart failure registry study of guideline-directed medical therapy withdrawal, a second randomized trial testing partial withdrawal, an expert consensus document defining the improvement/remission/recovery spectrum, the 2022 AHA/ACC/HFSA heart failure guideline, and a contemporary review of HFimpEF epidemiology, identified through the sources cited here and citation tracking between them, without a pre-registered protocol or systematic search string. All percentages, hazard ratios, and p-values reported here are taken directly from the cited sources; none has been invented, estimated, or extrapolated.

The sources reviewed span three distinct study designs deliberately: a randomized controlled trial able to establish causation in a small, closely monitored population; a national registry able to test the same question at a scale and duration no trial could match, at the cost of the confounding inherent to observational data; and a second randomized trial testing whether the withdrawal strategy itself, not only the underlying disease, shapes outcomes. Reading these designs alongside one another, rather than any single one in isolation, is the central method of this review, because each addresses a limitation of the others and no single source in this literature currently answers the clinical question on its own.

Results

Defining heart failure with improved ejection fraction: improvement, remission, and recovery

A 2025 expert consensus document distinguishes three stages along the same trajectory rather than treating recovery as a single binary event [6]. Improvement (impHF) describes an LVEF rise of at least 10 absolute points that remains below 50%; remission (remHF) describes an LVEF that has reached 50% or higher alongside minimal symptoms and only mild or no biomarker elevation; recovery (recHF) adds normalized cardiac structure and function, no symptoms, normalized biomarkers, and stability sustained for at least 12 months [6]. Figure 1 sets these stages out alongside the consensus document's own graded recommendation: continuation of guideline-directed medical therapy is recommended at both the improvement and remission stages, and only at the recovery stage does the document allow that a shared-decision, gradual taper of therapy may be considered [6]. The 2022 AHA/ACC/HFSA heart failure guideline states this plainly for the broader HFimpEF population: "it is recommended that GDMT be continued in patients with HFimpEF, including those who are asymptomatic, to prevent relapse of HF and LV dysfunction" [3].

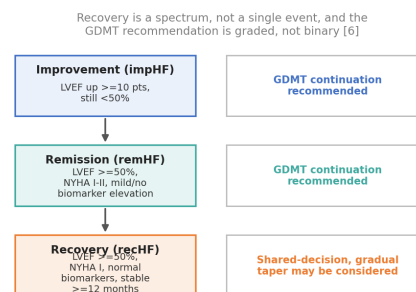


Figure 1: Improvement, remission, and recovery are distinct stages, and the recommended approach to GDMT differs across them [6].

It is also worth separating two different questions that this terminology can blur together: who recovers an improved ejection fraction in the first place, and what happens afterward if their therapy is withdrawn. A contemporary epidemiological review of the broader HFimpEF population identifies factors associated with a patient failing to sustain any ejection-fraction improvement at all -- male sex, non-White race, atrial fibrillation, a prolonged QRS interval or left bundle-branch block, ischaemic aetiology, diabetes mellitus, and older age combined with a lower resting heart rate have each been associated with a lower likelihood of durable improvement across observational cohorts [8]. These predictors of recovery itself are distinct from the predictors of relapse after withdrawal examined later in this review, and the distinction matters at the bedside: an echocardiogram report reading a normalized LVEF does not by itself say how durable that

number is, how it was reached, or whether the patient in front of a clinician resembles the population in which 'recovery' was defined [1,8].

TRED-HF: the trial that tested withdrawal directly

TRED-HF enrolled 51 patients whose LVEF had improved from below 40% to 50% or higher, with normalized left ventricular end-diastolic volume and an NT-proBNP below 250 ng/L, and randomized 25 to a phased withdrawal of all heart failure medication and 26 to continue therapy unchanged [1]. Relapse -- defined as a fall in LVEF of more than 10 points to below 50%, a rise in left ventricular end-diastolic volume of more than 10% above normal, a doubling of NT-proBNP to above 400 ng/L, or clinical evidence of heart failure -- occurred in 11 of 25 withdrawal patients (44%) within 6 months, compared with none of the 26 patients who continued therapy ($p=0.0001$) [1]. Table 1 sets out the trial's entry criteria and endpoint definition in full. Patient-reported health status moved in the same direction: the Kansas City Cardiomyopathy Questionnaire score fell by 5.0 points in the withdrawal group versus a rise of 1.0 point in the continuation group ($p=0.04$), though no patient in either group died or had a cardiovascular event during the initial 6-month comparison [1]. Figure 2 sets this primary result alongside a further data point from the trial's design: once the 6-month comparison closed, the continuation group was itself offered a supervised withdrawal, and 36% of that crossover group also relapsed (95% CI 20.6-57.8%) [1], a second independent estimate landing in the same range as the original randomized comparison.

Criterion	TRED-HF requirement
Prior LVEF	<40%, historically documented
Current LVEF	$\geq 50\%$ at screening
LV end-diastolic volume	Normalized
NT-proBNP	<250 ng/L
Relapse (primary endpoint)	>10-pt LVEF fall to <50%, OR >10% LVEDV rise above normal, OR NT-proBNP doubling to >400 ng/L, OR clinical heart failure

Table 1: TRED-HF entry criteria and the composite definition of relapse used as its primary endpoint [1].

These criteria were deliberately conservative: requiring a normalized end-diastolic volume and a low NT-proBNP, in addition to a normalized LVEF, selected patients who looked recovered by every available non-invasive measure, not simply patients whose ejection fraction alone had crossed a threshold [1]. That matters for reading the relapse rate that follows: it was observed in a population chosen to look about as convincingly recovered as this literature can currently define, which makes the finding harder to dismiss as an artefact of an

unusually lenient definition of recovery [1].

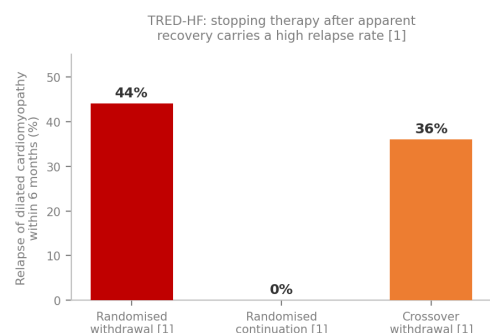


Figure 2: TRED-HF's primary result: 44% of patients randomized to withdrawal relapsed within 6 months versus none who continued therapy, and 36% of the crossover group relapsed when later withdrawn [1].

A pre-specified secondary analysis asked whether a simple bedside sign could flag relapse as it happened rather than only after the fact. Resting heart rate, checked every 4 weeks during withdrawal, tracked relapse closely: each 10 beats-per-minute higher attained heart rate carried a hazard ratio of 1.92 (95% CI 1.34-2.73) for relapse, and each 10 bpm rise from baseline carried a hazard ratio of 1.99 (95% CI 1.37-2.89), with patients who relapsed showing a 10.4 bpm greater rise in heart rate than those who did not (95% CI 4.0-16.8), independent of baseline heart rate, beta-blocker dose, age, NT-proBNP, and baseline LVEF [2]. A rising heart rate during a supervised drug taper is, on this evidence, a genuine early-warning sign rather than an incidental finding, though it identifies relapse already under way rather than predicting which patients will relapse before withdrawal begins.

How long does relapse risk last? Long-term follow-up of TRED-HF

A question the original 6-month trial could not answer is whether relapse risk is a one-time hurdle cleared by patients who make it past the initial withdrawal period, or a risk that persists. Long-term follow-up of the same 51 patients, at a median of 6 years (interquartile range 6-7) after enrollment, found that 33 of the 51 (65%) had relapsed by some point during that follow-up, whether or not they had relapsed during the original trial [4]. Measured specifically from the point 5 years after enrollment, the cumulative relapse rate was 61% (95% CI 45-73); measured instead from 5 years after the trial itself ended, it was 39% (95% CI 19-54) [4]. Figure 3 places these figures on a single timeline. Of the 20 patients who had relapsed during the original trial, 9 relapsed again during the extended follow-up and a further 13 patients relapsed for the first time only after the trial had concluded [4], meaning a clean 6-month trial window materially understates the true burden of relapse over a patient's remaining years, and that patients who tolerate an initial withdrawal are not thereby proven safe from relapse in the future.

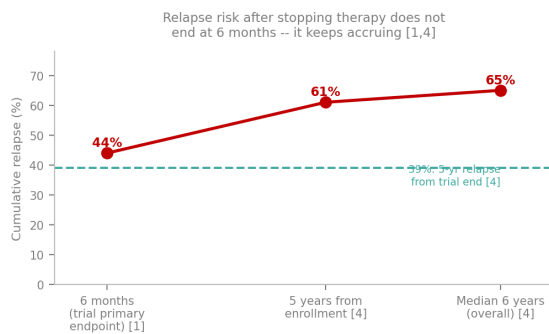


Figure 3: Relapse in TRED-HF's original cohort continued to accrue for years after the trial's 6-month primary endpoint [1,4].

Real-world confirmation: the Swedish Heart Failure Registry

Because TRED-HF enrolled only 51 patients with non-ischemic dilated cardiomyopathy under close trial supervision, its findings needed testing in a larger and more representative population. A Swedish Heart Failure Registry study drew on data collected between June 2000 and December 2023, spanning nearly a quarter-century of ordinary Swedish clinical practice across multiple hospital systems, and identified 8,728 patients with an initial LVEF below 40% that later improved to 40% or higher, of whom 366 (4.4%) had renin-angiotensin system inhibitor or angiotensin receptor-neprilysin inhibitor therapy withdrawn, 696 (17.2%) had a mineralocorticoid receptor antagonist withdrawn, and 270 (3.3%) had a beta-blocker withdrawn, against a composite outcome of cardiovascular death or heart failure hospitalization occurring in 9% of the cohort overall (9.9 per 100 patient-years) [5]. After adjustment, withdrawal of renin-angiotensin system inhibition or neprilysin inhibition was associated with a 38% higher hazard of that composite outcome at 1 year (hazard ratio 1.38, 95% CI 1.08-1.77; number needed to harm 17; absolute risk difference 6.0%), and mineralocorticoid receptor antagonist withdrawal with a 36% higher hazard (1.36, 95% CI 1.07-1.73; number needed to harm 25; absolute risk difference 3.9%) [5]. Beta-blocker withdrawal showed no significant association overall, though a signal emerged in the subgroup with only moderately improved LVEF, 40% to 49% (interaction $p=0.03$) [5]. Figure 4 sets these three hazard ratios out together, and the authors concluded that their results "support continuing RASi/ARNi and MRA in patients with HFimpEF" while calling for a dedicated randomized trial of beta-blocker withdrawal specifically [5]. A registry of this size and duration converts TRED-HF's small-trial signal into a population-level finding that cannot be dismissed as an artefact of a highly selected 51-patient cohort followed under unusually close trial supervision.

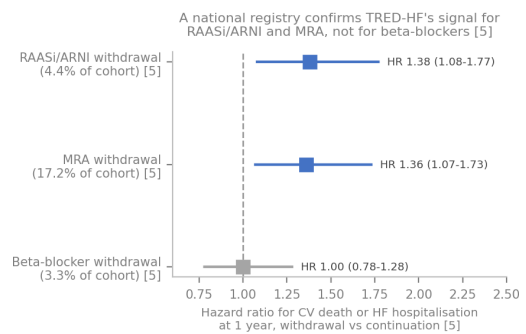


Figure 4: In 8,728 registry patients with improved ejection fraction, withdrawing RAASi/ARNi or an MRA was associated with a significantly higher hazard of death or hospitalization; beta-blocker withdrawal was not [5].

Does the withdrawal strategy matter? CATHEDRAL-HF and selective de-escalation

TRED-HF and the Swedish registry both examined withdrawal of one drug class at a time or withdrawal of everything at once, but a further trial asked a different, practically important question: is a partial withdrawal that deliberately keeps one drug in place safer than stopping treatment altogether? CATHEDRAL-HF randomized 60 patients with HFimpEF 1:1 to a stepwise withdrawal of all heart failure medication except carvedilol, or to continued usual HFimpEF-pattern therapy [7]. Recurrence of ventricular dysfunction occurred in 3 of 30 intervention patients (10%) versus 1 of 30 controls (3.3%) at 24 weeks, and in 4 of 27 intervention patients (14.8%) versus 4 of 26 controls (15.4%) at 52 weeks, with no hospitalizations, deaths, or sustained ventricular arrhythmias in either arm [7]. Figure 5 sets out the trial's design and these outcome counts. By 52 weeks, the two arms had essentially converged, and the recurrence rate in the arm that stopped everything except a single beta-blocker (14.8%) was substantially lower than the 44% relapse TRED-HF observed after stopping all therapy including beta-blockade [1,7]. The trial's own authors describe this as the first demonstration that discontinuing heart failure therapy while maintaining carvedilol is feasible in HFimpEF, while explicitly calling for larger confirmatory studies before the approach is adopted in practice [7].

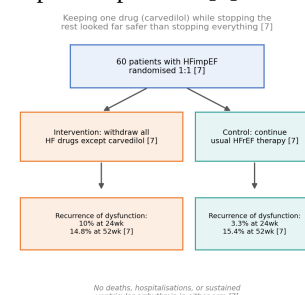


Figure 5: CATHEDRAL-HF: withdrawing every drug except a beta-blocker produced a lower recurrence rate than TRED-HF's full withdrawal, hinting that strategy, not just disease biology, shapes relapse risk [1,7].

Who might relapse, and who might not

TRED-HF's own investigators looked for predictors that could identify, before withdrawal begins, which patients are more likely to relapse. In multivariable analysis, older age ($p=0.03$), the absence of mineralocorticoid receptor antagonist use before withdrawal ($p=0.004$), taking more than two heart failure medications at baseline ($p=0.004$), higher baseline NT-proBNP ($p=0.02$), and reduced peak radial strain on imaging ($p=0.02$) were each associated with relapse [1]. Figure 6 sets these associations out directly. None of these factors has been validated in an independent cohort as a prospective tool for deciding which patients can safely stop therapy; they describe who happened to relapse in a single 51-patient trial, and the trial's own authors state plainly that reliable predictors of persistent recovery remain undefined [1].

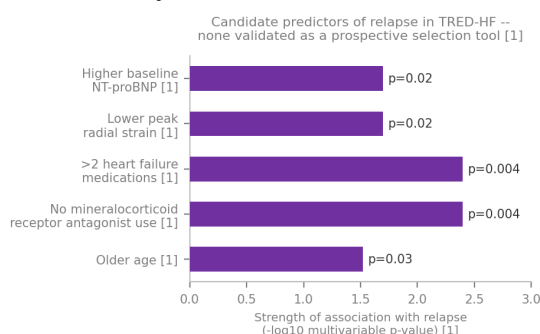


Figure 6: Candidate predictors of relapse identified in TRED-HF, ranked by the strength of their multivariable association -- hypotheses for future study, not a validated selection rule [1].

It is worth keeping these two predictor lists conceptually separate: the broader epidemiological predictors of failing to sustain any ejection-fraction improvement discussed earlier -- male sex, non-White race, atrial fibrillation, ischaemic aetiology, and related factors -- describe who recovers in the first place, drawn from large observational cohorts [8], while TRED-HF's own predictors of relapse after withdrawal describe a narrower and different question, drawn from only 51 patients within a single trial [1]. A clinician cannot yet combine the two into a single risk score, and no study reviewed here has attempted to.

Study	N	Design	Headline finding
TRED-HF [1]	51	RCT, full withdrawal vs continuation	44% vs 0% relapse at 6 months ($p=0.0001$)
Swedish registry [5]	8,728	Observational, class-specific withdrawal	RAASi/ARNi HR 1.38, MRA HR 1.36 for CV death/HF admission
CATHEDRAL-HF [7]	60	RCT, withdrawal except beta-blocker vs continuation	14.8% vs 15.4% recurrence at 52 weeks

Table 2: Design and headline result of the three withdrawal studies reviewed here [1,5,7].

Discussion

Taken together, this evidence points in one direction more consistently than most debates in heart failure management: stopping guideline-directed medical therapy after ejection fraction has improved carries a real and, for renin-angiotensin-system inhibition and mineralocorticoid receptor antagonism specifically, a statistically robust risk of relapse, confirmed in both a small controlled trial and a large national registry [1,5]. The honest complication for practice is that this collides with a reasonable patient request and a reasonable clinical instinct: a person who feels well and has been told their heart failure is 'in remission' has grounds to ask why medication should continue indefinitely. The data answer that with a number rather than a reassurance -- a 44% relapse rate within 6 months of full withdrawal, and a persistently elevated hazard of death or hospitalization when renin-angiotensin-system inhibition or mineralocorticoid receptor antagonism is withdrawn in unselected practice [1,5].

CATHEDRAL-HF's finding that a partial withdrawal strategy, retaining a single beta-blocker, produced a substantially lower recurrence rate than TRED-HF's complete withdrawal is the most clinically actionable nuance in this literature, but it is a nuance, not a change in the overall conclusion [1,7]. A 60-patient trial cannot establish that beta-blocker-sparing withdrawal is safe in the way the much larger Swedish registry has established that renin-angiotensin-system inhibitor and mineralocorticoid receptor antagonist withdrawal is risky, and the registry's own beta-blocker subgroup finding -- confined to patients with only moderately improved, rather than fully normalized, ejection fraction -- suggests the safety of beta-blocker withdrawal may itself depend on how completely the disease has resolved [5]. Table 2 draws the three studies' headline findings together for direct comparison. The rising heart rate identified during withdrawal in TRED-HF's own sub-analysis offers one practical safety net for a patient and clinician who do proceed with a supervised taper: a checked pulse every few weeks is a simple, low-cost way to catch relapse as it begins [2].

For the practicing cardiologist, the clearest current position mirrors the graded recommendation in the 2025 improvement/remission/recovery consensus and the 2022 AHA/ACC/HFSA guideline alike: guideline-directed medical therapy should be continued, not withdrawn, in essentially all patients whose ejection fraction has improved, reserving any discussion of gradual taper for the minority who have reached full, sustained recovery and who have a specific reason -- most often a genuine side effect -- to consider it, and then only as a shared decision with close monitoring rather than a routine step once a follow-up echocardiogram looks normal [3,6]. Table 3 sets this graded approach out against the specific criteria defining each stage.

Stage	Defining feature	Recommended approach to GDMT
Improvement (impHF) [6]	LVEF up ≥ 10 pts, still $< 50\%$	Continue
Remission (remHF) [6]	LVEF $\geq 50\%$, NYHA I-II, mild/no biomarker rise	Continue
Recovery (recHF) [6]	LVEF $\geq 50\%$, NYHA I, normal biomarkers, stable ≥ 12 months	Shared-decision taper may be considered

Table 3: The graded relationship between recovery stage and the recommended approach to guideline-directed medical therapy [3,6].

Study limitations

This is a narrative review, not a systematic review with a pre-registered protocol, and its conclusions rest on one small randomized trial, its long-term follow-up, one registry study, and one small partial-withdrawal trial rather than an exhaustive search of the HFimpEF withdrawal literature. TRED-HF enrolled only patients with non-ischemic dilated cardiomyopathy, a population that may recover through different mechanisms than patients whose ejection fraction improves after an ischemic insult, so its 44% relapse figure should not be assumed to transfer unchanged to every cause of HFimpEF [1]. The Swedish registry, despite its size, is observational: patients whose medication was withdrawn may have differed systematically from those whose was not, in ways adjustment cannot fully remove [5]. CATHEDRAL-HF randomized only 60 patients and was not powered to detect a difference in hard clinical outcomes, so its reassuring recurrence-rate comparison with TRED-HF should be read as hypothesis-generating, not as proof that a beta-blocker-sparing strategy is safe [7]. Finally, none of TRED-HF's candidate predictors of relapse -- age, medication count, NT-proBNP, mineralocorticoid receptor antagonist use, or peak radial strain -- has been tested prospectively in an independent cohort [1].

A further point worth stating plainly for practice: the terminology used across this literature is not fully standardized, and a clinician reading TRED-HF, the Swedish registry, and CATHEDRAL-HF side by side will notice that each defines its own version of 'improved' or 'recovered' ejection fraction, with different thresholds for LVEF, symptom status, and biomarker normalization [1,5,6,7]. The 2025 expert consensus document's three-stage terminology postdates all three primary studies reviewed here, so none was designed around it [6]. The heart-rate warning sign in TRED-HF's sub-analysis was derived from only 49 patients with available data and 20 relapse events, a small enough sample that its hazard ratios, while statistically significant, carry wide confidence intervals and have not been replicated independently [2]. This review's own search was not systematic, and a formal systematic review with meta-analysis of GDMT withdrawal in HFimpEF, better suited to pooling

smaller studies, was identified during the search but was not itself analyzed here.

Conclusion

Ejection fraction that improves on guideline-directed medical therapy is genuinely good news, but the trial and registry evidence reviewed here does not support treating a normalized ejection fraction as a cure that permits stopping treatment. TRED-HF found a 44% relapse rate within 6 months of full withdrawal, a risk that a long-term follow-up shows continuing to accrue for years rather than resolving once withdrawal is survived, and a national registry of nearly 9,000 patients found a significantly higher hazard of cardiovascular death or heart failure hospitalization specifically after withdrawal of renin-angiotensin-system inhibition or mineralocorticoid receptor antagonism. A partial withdrawal strategy that keeps a beta-blocker in place looked considerably safer in one small trial, a genuinely useful lead for future research, but not yet a validated alternative to continuing therapy. For now, the honest answer to the patient who asks whether their pills can stop now that their heart looks normal is that the evidence, examined directly rather than assumed, still says no.

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