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Review Article

Impact of percutaneous coronary intervention on Quality of Life in Patients with resistant hypertension

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Abstract

Background: Resistant hypertension frequently coexists with obstructive coronary artery disease, and whether percutaneous coronary intervention (PCI) meaningfully improves quality of life (QoL) in this overlap population remains incompletely defined.

Objective: This review synthesizes evidence on PCI's effect on quality of life and blood pressure trajectory in patients with resistant hypertension and coronary artery disease, situating revascularization within the resistant-hypertension management pathway.

Methods: We reviewed randomized ischemia trials with quality-of-life substudies, resistant-hypertension pharmacotherapy and device trials, and cohorts linking revascularization status to Seattle Angina Questionnaire and health-utility outcomes.

Findings: PCI produces early, durable improvements in angina-specific quality of life relative to medical therapy alone, most pronounced with frequent baseline angina, while its effect on blood pressure is modest; resistant hypertension amplifies cardiovascular risk independent of coronary anatomy, and spironolactone, renal denervation, and structured regimens lower blood pressure with variable quality-of-life benefit.

Conclusions: PCI relieves angina and improves disease-specific quality of life without substituting for resistant-hypertension therapy, supporting a combined strategy addressing ischemic burden and the hypertensive substrate together.

Keywords: resistant hypertension; percutaneous coronary intervention; quality of life; coronary artery disease; blood pressure

1. Introduction

Resistant hypertension, blood pressure above goal despite three optimized agents including a diuretic, or controlled only with four or more, affects a substantial minority of treated hypertensive patients and carries elevated cardiovascular risk [1,2]. Registry data place apparent treatment-resistant hypertension at roughly 12-15% of treated patients, with confirmed true resistance somewhat lower but still clinically substantial [3,4]. Patients meeting resistant-hypertension criteria are disproportionately older, more often diabetic, and more often carry obstructive coronary artery disease than patients whose pressure responds to standard regimens [5].

This overlap is not coincidental. Chronically elevated afterload drives left ventricular hypertrophy and subendocardial ischemia independent of epicardial stenosis, while the same endothelial, vascular, and neurohormonal processes sustaining resistant hypertension also promote atherosclerosis [6,7]. Pierdomenico and colleagues found that patients with confirmed true resistant hypertension had substantially higher cardiovascular event rates than normotensive responders or white-coat-resistant patients, establishing that the resistant phenotype itself drives excess coronary risk [8]. Cardiology clinics therefore regularly encounter patients with resistant hypertension found to have obstructive disease amenable to percutaneous coronary intervention (PCI), and clinicians face two intertwined questions: whether revascularization will improve quality of life, and how it fits alongside the pharmacologic and device-based therapies resistant hypertension itself requires.

This review synthesizes the quality-of-life evidence from major PCI and ischemia trials and situates it against what is known about spironolactone, renal denervation, and structured pharmacotherapy for the resistant-hypertension substrate revascularization does not treat, an intersection most existing trials study only in

isolation.

Table 1: Definitions used across the resistant-hypertension spectrum and their relevance to the coronary artery disease overlap population [1,2,4,22].			
Category	Definition	Key requirement	Relevance to overlap population
Apparent treatment-resistant hypertension	Office BP above goal on 3+ agents including a diuretic, or controlled on 4+	Office measurement only	Screening threshold that should trigger secondary-cause and adherence work-up
Confirmed resistant hypertension	Apparent resistance confirmed by ambulatory or home BP monitoring	Out-of-office confirmation	Excludes white-coat effect before escalating therapy or considering PCI referral bias
True refractory hypertension	Uncontrolled despite 5+ agents including a diuretic and MRA	Maximal guideline-directed therapy	Highest-risk subgroup; device therapy most often considered here
Secondary hypertension	Identifiable cause (aldosteronism, renal artery disease, OSA, CKD)	Directed screening	Correctable causes should be excluded before revascularization decisions are finalized

2. Quality of life as an outcome in ischemic heart disease

Quality-of-life measurement in coronary disease relies on disease-specific tools such as the Seattle Angina Questionnaire (SAQ), which quantifies angina frequency, physical limitation, and disease perception, and generic instruments such as the Short Form-36 (SF-36), which capture broader health domains [9,10]. The SAQ dominates contemporary revascularization trials partly because Spertus and colleagues validated that its scores independently predict mortality and hospitalization, establishing quality of life as a prognostically meaningful outcome rather than a patient-reported afterthought [11]. Rumsfeld and colleagues extended this principle to surgical revascularization, showing baseline health-related quality of life independently predicted mortality after bypass grafting [12].

Spertus and colleagues found that patients with more frequent baseline angina and worse baseline SAQ scores

derived the largest quality-of-life gains from revascularization, while mildly symptomatic patients had little room for measurable improvement, a ceiling effect later shaping how ischemia trials interpreted their quality-of-life substudies [13]. This baseline-dependency matters because a modest average treatment effect can conceal a large benefit concentrated among patients with substantial baseline angina, plausibly a sizeable subgroup within the resistant-hypertension overlap population.

3. PCI and quality of life: trial evidence

COURAGE randomized patients with stable coronary disease and objective ischemia to PCI plus optimal medical therapy or medical therapy alone, finding no difference in death or myocardial infarction [14]. Its quality-of-life substudy told a different story: Weintraub and colleagues found PCI produced early, significant improvements in angina frequency and disease-specific quality of life, most pronounced in the first six to twenty-four months and among patients with more frequent baseline angina, though the gap narrowed by three years as medical therapy alone also improved symptoms [15].

ISCHEMIA extended this question to a larger, more contemporary population, again finding no difference in the primary ischemic composite between invasive and conservative strategies [16]. Its health-status analysis, led by Spertus and colleagues, found the invasive strategy produced significantly greater improvement in angina-related quality of life at three, twelve, and thirty-six months, concentrated among patients with daily or weekly baseline angina [17]. COURAGE and ISCHEMIA together established that PCI does not reduce hard events beyond optimal medical therapy in stable disease, but produces reproducible, clinically meaningful symptom-specific quality-of-life improvement.

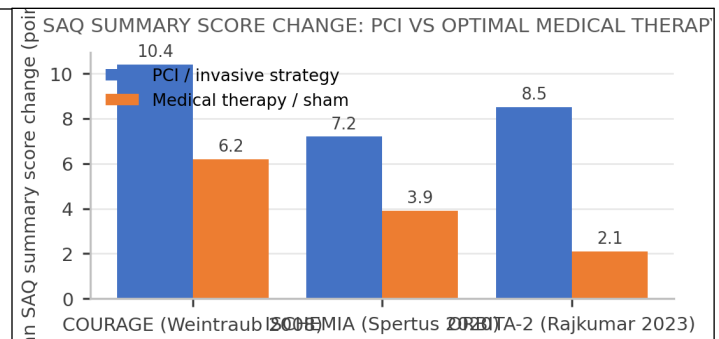


Figure 1: Angina-specific quality-of-life improvement (mean Seattle Angina Questionnaire summary score change from baseline) with an initial invasive/PCI strategy versus optimal medical therapy alone across major stable ischemic heart disease trials, showing a consistent early advantage for revascularization [15,17,19].

Two placebo-controlled trials sharpened this picture by removing the unblinded-comparison placebo effect. ORBITA randomized patients with single-vessel disease to PCI or a sham procedure, finding no significant difference in exercise time, its primary endpoint, though PCI showed numerically greater improvement in some angina domains [18]. ORBITA-2, which withdrew background antianginal medication before randomization, found PCI produced significantly greater improvement in angina frequency and SAQ summary score than sham, the clearest placebo-controlled confirmation that PCI has a genuine symptomatic benefit in selected patients [19]. Alongside the observation that even nonobstructive disease carries elevated ischemic risk, this sequence supports a symptom-driven rather than purely event-driven indication for PCI [20].

4. Resistant hypertension: burden, mechanisms, and the overlap population

Resistant hypertension is not homogeneous. Scientific statements distinguish apparent resistance, defined by office measurement alone, from confirmed resistance requiring ambulatory or home monitoring, and from true refractory hypertension uncontrolled despite five or more agents including a diuretic and mineralocorticoid receptor antagonist [2,21]. Secondary causes, primary aldosteronism, renal artery stenosis, obstructive sleep apnea, and chronic kidney disease, are disproportionately

represented and should be screened for before escalating empiric pharmacotherapy [22]. Calhoun's review of aldosteronism underscores that inappropriate aldosterone excess is common enough in resistant hypertension to justify mineralocorticoid antagonism as a near-default fourth-line step [23].

Sustained afterload elevation drives left ventricular hypertrophy, diastolic dysfunction, and microvascular rarefaction that impair coronary flow reserve and produce angina even without flow-limiting stenosis, relevant to why some resistant-hypertension patients with modest angiographic disease report substantial symptom burden [6]. Global epidemiology confirms uncontrolled blood pressure remains among the largest attributable contributors to cardiovascular mortality worldwide [24], and contemporary US surveillance shows blood-pressure control rates have plateaued after decades of improvement, a trend likely to enlarge rather than shrink this overlap population [25].

5. Pharmacologic and device-based therapy for the hypertensive substrate

Because PCI addresses epicardial ischemia rather than the neurohormonal and volume processes sustaining resistant hypertension, dedicated therapy remains necessary regardless of revascularization status. PATHWAY-2 provided the clearest randomized evidence for fourth-line drug selection, showing spironolactone produced significantly greater blood-pressure reduction than bisoprolol or doxazosin, establishing mineralocorticoid antagonism as the preferred fourth agent, a finding since incorporated into American and European guidelines [26,21,27].

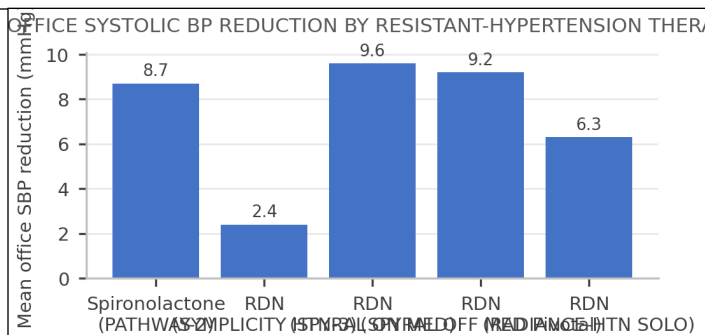


Figure 2: Mean office systolic blood pressure reduction with fourth-line spironolactone versus catheter-based renal denervation trials in patients with resistant hypertension, illustrating the substrate-directed blood-pressure benefit that percutaneous coronary intervention does not provide [26,28,29,31,32].

Catheter-based renal denervation followed a more turbulent path. The initial sham-controlled SYMPPLICITY HTN-3 trial failed to show significant benefit over sham, prompting redesign with more rigorous technique and adherence confirmation [28]. The SPYRAL HTN program subsequently demonstrated significant blood-pressure reductions both with background medication and, in medication-naïve patients, against sham, restoring confidence in a genuine antihypertensive effect [29,30,31]. Ultrasound-based denervation produced similar sham-controlled reductions in RADIANCE-HTN SOLO, and a contemporary review concluded the device class now has a consistent, modest antihypertensive effect across platforms [32,33]. SYMPPLICITY HTN-Japan extended this evidence to a population comparable to the patients discussed here, consistent with Japanese hypertension society guidance favoring individualized, stepwise escalation [34,35].

Where blood pressure itself is the target, revascularization has at best a modest, inconsistent effect. INVEST, comparing calcium-antagonist and non-calcium-antagonist strategies in hypertensive coronary patients, found comparable blood-pressure control between pharmacologic strategies regardless of revascularization status [36]. Stable ischemic heart disease guidelines accordingly frame revascularization as symptom- and prognosis-directed therapy,

complementary to rather than a substitute for blood-pressure management [37], and older heart-failure evidence for spironolactone in severe left ventricular dysfunction further supports mineralocorticoid antagonism's cardioprotective rationale independent of its pressure-lowering effect [38].

6. Integrating revascularization within resistant-hypertension management

PCI and resistant-hypertension therapy address different, complementary targets, and neither substitutes for the other. In a patient with resistant hypertension, obstructive disease, and significant angina, trial evidence supports proceeding with PCI, extrapolating from COURAGE, ISCHEMIA, and ORBITA-2 a meaningful, durable improvement in angina-specific quality of life, particularly with substantial baseline symptom burden [15,17,19]. That patient's hypertension nonetheless requires independent escalation, confirming true resistance with out-of-office monitoring, excluding secondary causes, and adding spironolactone before device therapy, since revascularization does not reliably lower blood pressure [2,26].

This also shapes how post-PCI quality of life should be interpreted: because uncontrolled resistant hypertension independently degrades physical functioning and general health perception through mechanisms the SF-36 captures but the angina-specific SAQ does not, a patient may report residual impairment after a technically successful PCI if the hypertensive substrate remains uncontrolled [10,6]. Clinicians should reassess blood-pressure control before attributing persistent symptoms solely to residual ischemia, since the two conditions contribute to quality of life through distinct mechanisms responsive to different interventions. Coordinated cardiology- and hypertension-specialist-led management represents the most evidence-consistent approach, though no randomized trial has yet tested this combined strategy as a single intervention.

Table 2: Complementary treatment targets of percutaneous coronary intervention and resistant-hypertension therapy in the overlap patient [15,17,26,33].			
Domain	PCI	Resistant-hyperten sion therapy	Primary evidence base
Primary target	Epicardial ischemic burden	Neurohormonal / volume-mediated blood pressure elevation	COURAGE, ISCHEMIA, ORBITA-2
Expected qu ality-of-life effect	Reduced angina frequency, improved SAQ summary score	Improved general physical and mental health (SF-36 domains)	Weintraub 2008; Spertus 2020
Effect on office blood pressure	Modest, inconsistent	Substantial with spironolactone or renal denervation	PATHWAY-2; SPYRAL HTN program
Role in overlap patient	Symptom- and i schemia-directe d	Substrate-directed, non-substitutable	Guideline synthesis (Carey 2018; Whelton 2018)

7. Conclusion

PCI produces a reproducible improvement in angina-specific quality of life in stable obstructive coronary disease, now confirmed under placebo-controlled conditions and concentrated among patients with substantial baseline symptom burden [18,19]. In patients with resistant hypertension and comorbid coronary disease, a population of elevated cardiovascular risk with a mechanistically plausible link between afterload elevation and ischemic symptom burden, this benefit is expected to generalize, but addresses only the ischemic component of the overall risk profile [5,8]. Effective management therefore requires two parallel, non-substitutable tracks: revascularization guided by angina burden as in the general stable coronary disease population, and dedicated resistant-hypertension therapy, confirmed with out-of-office monitoring and escalated through spironolactone and, where appropriate, renal denervation, treating the hypertensive substrate PCI does not touch [26,33]. Trials that prospectively stratify by resistant-hypertension status within revascularization cohorts, rather than treating hypertension merely as a covariate, would meaningfully strengthen this evidence base.

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