

Frailty Assessment in the Cardiovascular Care of Older Adults



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Due to the aging and increasingly complex nature of our patients, frailty has become a high-priority theme in cardiovascular medicine. Despite the recognition of frailty as a pivotal element in the evaluation of older adults with cardiovascular disease (CVD), there has yet to be a road map to facilitate its adoption in routine clinical practice. Thus, we sought to synthesize the existing body of evidence and offer a perspective on how to integrate frailty into clinical practice. Frailty is a biological syndrome that reflects a state of decreased physiological reserve and vulnerability to stressors. Upward of 20 frailty assessment tools have been developed, with most tools revolving around the core phenotypic domains of frailty—slow walking speed, weakness, inactivity, exhaustion, and shrinking—as measured by physical performance tests and questionnaires. The prevalence of frailty ranges from 10% to 60%, depending on the CVD burden, as well as the tool and cutoff chosen to define frailty. Epidemiological studies have consistently demonstrated that frailty carries a relative risk of >2 for mortality and morbidity across a spectrum of stable CVD, acute coronary syndromes, heart failure, and surgical and transcatheter interventions. Frailty contributes valuable prognostic insights incremental to existing risk models and assists clinicians in defining optimal care pathways for their patients. Interventions designed to improve outcomes in frail elders with CVD such as multidisciplinary cardiac rehabilitation are being actively tested. Ultimately, frailty should not be viewed as a reason to withhold care but rather as a means of delivering it in a more patient-centered fashion.

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Abbreviations and Acronyms

- ADL** = activities of daily living
- AS** = aortic stenosis
- AVR** = aortic valve replacement
- CAD** = coronary artery disease
- CVD** = cardiovascular disease
- SPPB** = Short Physical Performance Battery
- TAVR** = transcatheter aortic valve replacement

Frailty, from the French *frêle* meaning of little resistance, is a biological syndrome that reflects a state of decreased physiological reserve and vulnerability to stressors (1). Stressors are broadly classified as acute or chronic illness (e.g., myocardial infarction) or iatrogenic (e.g., cardiac surgery). When exposed to such stressors, frail patients are at risk for marked and often disproportionate decompensation, adverse events, procedural complications, prolonged recovery, functional decline, disability, and mortality (2).

Frailty has become a high-priority theme in cardiovascular medicine due to the aging and increasingly complex nature of our patients (3). Evolving technical innovations have enabled clinicians to treat a wider array of patients with devices and procedures, many of whom were previously regarded as “ineligible” (4,5). Uncertainty regarding individual benefit from such treatments has been coupled with growing economic constraints on healthcare systems, such that the issue of appropriate patient selection has intensified. There is an unmet need to optimize resource allocation to prevent patients from receiving costly but futile interventions.

Assessment of frailty is instrumental to refine estimates of risk and guide patients toward personalized treatment plans that will maximize their likelihood of a positive outcome. For example, given 2 heart failure patients with similar chronological age and comorbidities, the presence of objectively-measured frailty alerts the clinician that 1 of the 2 patients has a substantially higher risk of mortality and major morbidity. Furthermore, the frail patient faces a higher risk from invasive procedures but also a potential benefit from interventions such as cardiac rehabilitation to counteract the physical weakness characteristic of frailty. A critical mass of clinicians, researchers, and policy makers have embraced the concept of frailty, yet the lack of a scientific road map to integrate frailty into practice has been a limiting factor.

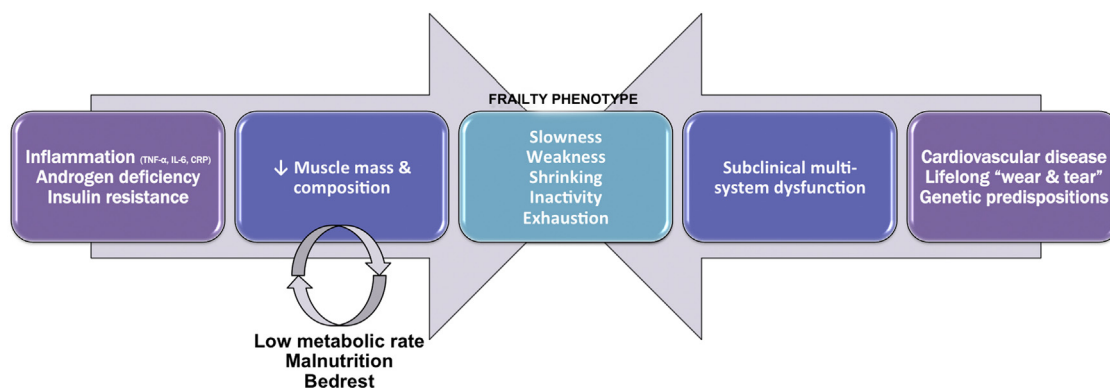
The objectives of this state-of-the-art paper are to: 1) summarize the existing body of evidence for frailty in patients with cardiovascular disease (CVD); 2) offer a perspective on integrating frailty into current clinical practice; and 3) point out the knowledge gaps for future research.

Pathobiology of Frailty

Frailty biology is a field of ongoing research and debate (6). Putative mechanisms revolve around dysregulation of the immune, hormonal, and endocrine systems (7)—notably, up-regulation of inflammatory cytokines (8–10), decreased

Figure 1

Two of the Pathways Leading Toward the Phenotype of Frailty



(Left) The age-associated activation of inflammatory cells and decline in androgen hormones upset the balance between catabolic and anabolic stimuli, respectively, leading to a decline in muscle mass and composition known as sarcopenia. This detrimental response is aggravated in patients with insulin resistance and metabolic syndrome. Addition of bed rest and malnutrition initiates a vicious cycle of further decline in muscle mass, limiting the necessary mobilization of amino acids in times of stress. **(Right)** The accumulation of subclinical impairments in multiple organ systems resulting from cardiovascular disease, lifelong “wear and tear,” and/or genetic predispositions lead to decreased homeostatic reserve and resiliency to stressors. Other pathophysiological pathways have been proposed. Biological pathways may manifest clinically as slow walking speed, weakness, weight loss, physical inactivity, and exhaustion—termed the phenotype of frailty. CRP = C-reactive protein; IL = interleukin; TNF = tumor necrosis factor.

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