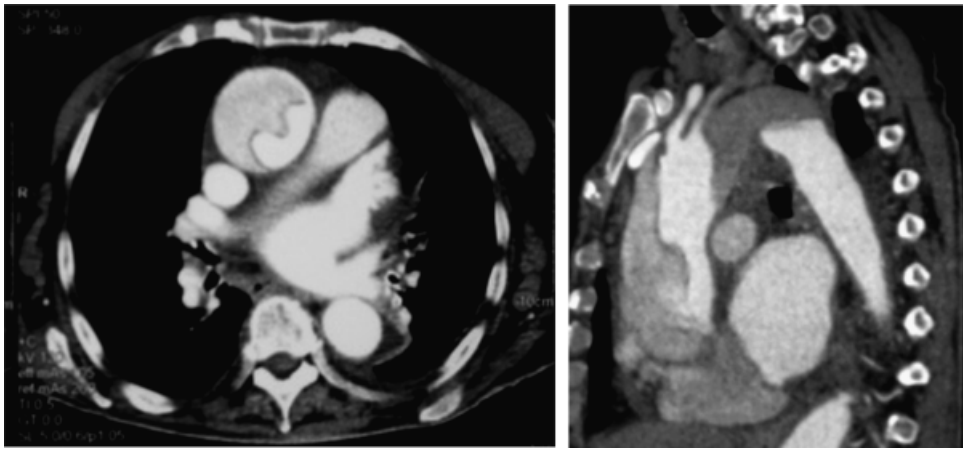

Figure 1. Computed tomography scan of the chest shows the aortic dissection of the aortic arch with extension to the great vessels of the arch.

insufficient blood supply to the masticatory muscles caused by inflammation of the branches of the external carotid.[1] Several diseases, including other vasculitis, atheromatous or embolic arterial occlusion, systemic amyloidosis, temporomandibular joint abnormalities as observed in rheumatoid arthritis, myasthenia gravis or myositis, and parotid or temporal tumors, may infrequently cause or mimic jaw claudication.[5–9]

Our observation suggests that acute aortic dissection should also be considered as a potential alternative diagnosis in elderly patients with acute-onset jaw claudication, especially when an inflammatory pattern is absent.

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ACKNOWLEDGMENTS

Conflict of Interest: The editor in chief has reviewed the conflict of interest checklist provided by the author and has determined that none of the authors have any financial or any other kind of personal conflicts with this letter.

Author Contributions: All the authors participated in the preparation of the letter.

Sponsor's Role: No sponsor.

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TACHYCARDIA-INDUCED CARDIOMYOPATHY IN A NONAGENARIAN

To the Editor: Tachycardia-induced cardiomyopathy (TIC) is the systolic or diastolic dysfunction caused by fast heart rates that resolves after normalization of the dysrhythmia.[1,2] TIC was first noted in the pediatric patient population.[3–5] Although TIC has now been well described in adults as well,[1,2] we believe the following case to be the first reported in a nonagenarian.

A 94-year-old female was hospitalized for progressive dyspnea on exertion, orthopnea, and paroxysmal nocturnal dyspnea. Physical examination revealed distended neck veins, bibasilar crackles, bilateral lower extremity edema, and an S3 gallop. Her brain natriuretic peptide level was 2,630 pg/mL.

Twelve-lead electrocardiograms and telemetry recordings demonstrated repetitive, incessant supraventricular tachycardia (SVT) with rates as high as 180/min. On the first hospital day, the patient was in SVT for 18 of 24 hours, although she was unaware of the extremely fast heart rate. The echocardiogram showed a slightly enlarged left ventricular (LV) chamber size, severe global hypokinesis, mild mitral and moderate tricuspid regurgitation, and an LV ejection fraction (LVEF) of 15%.

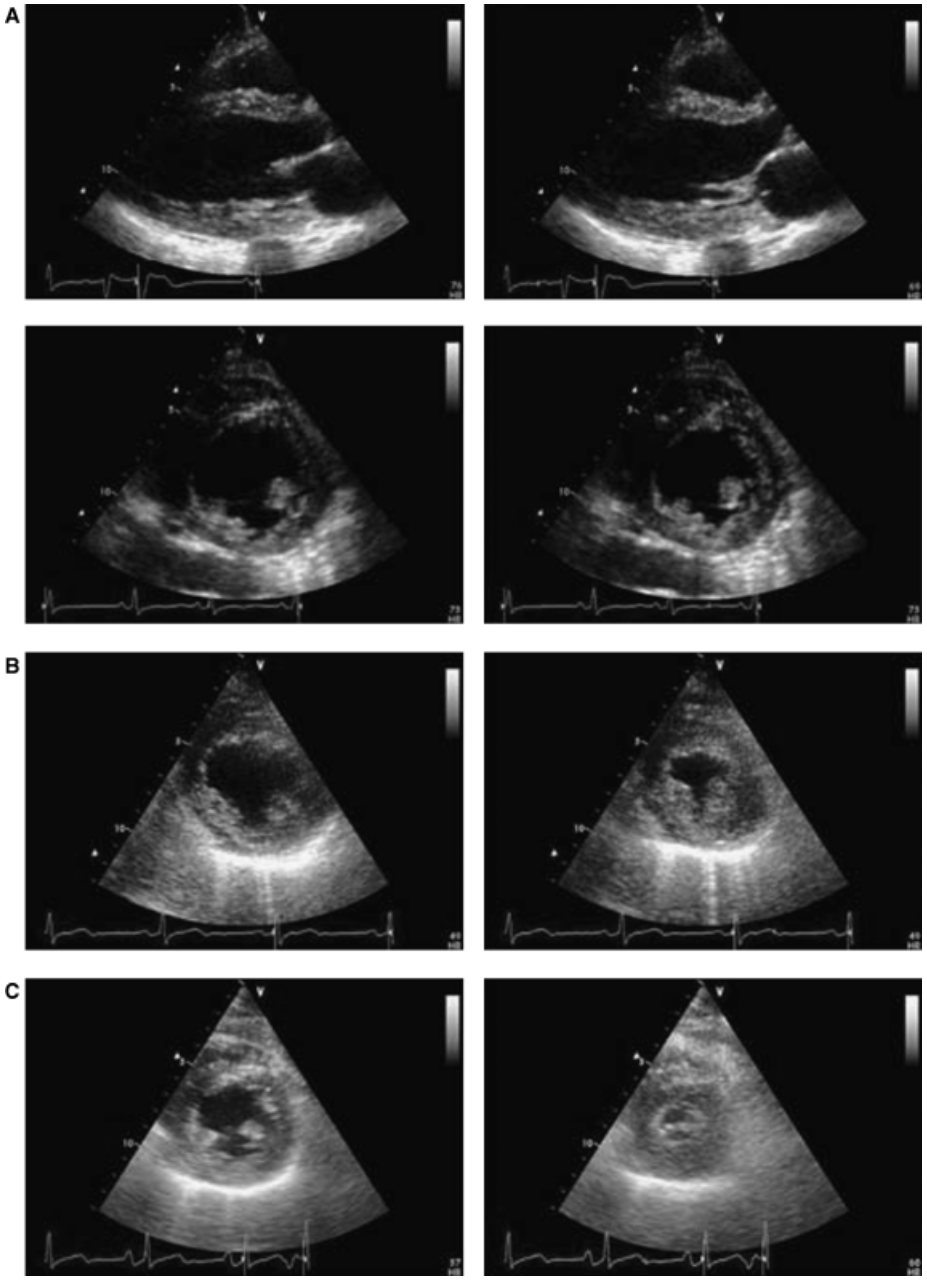

Figure 1. Representative end-diastolic (left column) and end-systolic (right column) frames of echocardiograms on presentation (A), 10 months later (B), and 2.5 years after the initial presentation (C). (A) Both the parasternal long axis views (top) and short axis views (bottom) are shown. B and C demonstrate the parasternal short axis views. Note the gradual decrease in left ventricular chamber size, as well as an increase in fractional shortening and systolic wall thickening over time.

The patient was treated with intravenous furosemide, and subsequently her heart failure symptoms improved, and the edema resolved. Because of her advanced age, electrophysiology studies were not performed. She was given amiodarone for arrhythmia control and was treated with a low-dose diuretic, an angiotensin-converting enzyme inhibitor, and a beta-blocker. She continued to be asymptomatic and physically active without further hospitalizations. Repeat 48-hour Holter monitoring demonstrated normal sinus rhythm without episodes of tachycardia. A transthoracic echocardiogram 10 months later revealed a near-normal LVEF of 50%. Two and a half years after the initial presentation, at age 97, she continues to be active and well. The echocardiogram now shows a small hypercontractile heart with concentric LV hypertrophy and an estimated LVEF of 60%. Representative end-diastolic and end-systolic frames of serial echocardiograms are shown in Figure 1.

Persistent supraventricular or ventricular tachyarrhythmias cause TIC. In the late 1980s, several case series were reported in the pediatric cardiology literature that demonstrated partial or complete resolution of "idiopathic" dilated cardiomyopathy after successful suppression of an incessant tachycardia.[3–5] Subsequently, TIC has also been found to be present in adults, in whom the most frequent cause is long-standing atrial fibrillation with rapid ventric-

ular response.[1,2] Patients who are especially at risk are those who have no awareness of their tachycardia. Children can typically have very fast heart rates for long periods of time without symptoms,[3–5] and our case suggests that the very elderly may also be vulnerable because of decreased perception of abnormally fast heart rates.

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ACKNOWLEDGMENTS

Conflict of Interest: The editor in chief has reviewed the conflict of interest checklist provided by the author and has determined that none of the authors have any financial or any other kind of personal conflicts with this letter.

Author Contributions: All authors contributed to the collection, analysis and interpretation of data, and preparation of the letter.

Sponsor's Role: No sponsor.

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ARE THE ASSESSING CARE OF VULNERABLE ELDERS QUALITY INDICATORS FOR HYPERTENSION WRONG?

To the Editor: With regards to the article "Quality Indicators for the Care of Hypertension in Vulnerable Elders,"[1] we feel that there were two clinical issues of great importance that were not given due diligence. First, is the effect of low diastolic blood pressure (DBP) within the elderly population and, second, is the lack of good data on which the goals for the treatment of systolic hypertension in patients aged 80 and older are based. Although not all patients who are vulnerable elders are aged 80 and older, all patients aged 80 and older are vulnerable elders. This letter is an attempt to discuss these issues.

Under quality indicator 9, *Intervening for Hypertension: Above SBP Goal*, the recommendation is to provide pharmacological intervention for any elderly patient with persistent systolic blood pressure (SBP) of 140 or greater without comorbidities, 135 or greater on home ambulatory monitoring, or 130 or greater with diabetes mellitus or chronic kidney disease. Although many studies have shown some benefits of antihypertensive treatment in all patients with SBP above 160, two meta-analyses of these studies in patients aged 80 and older found a nonsignificant relative excess of death from all causes in the treated group.[2,3] The results of the pilot study for Hypertension in the Very Elderly Trial, with a mean entry blood pressure of 181/99, also showed a trend toward excess deaths with treatment.[4] Treatment of elderly patients with blood pressures ranging from 140 to 160 may be more hazardous. Neither meta-analysis included elderly patients whose baseline SBP was less than 160; the mean baseline blood pressure of the elderly groups in the two meta-analyses were 180/84[2] and 176/78.[3] Under the recommendations published, a patient with a blood pressure of 143/68 should be placed on antihypertensive therapy, but clinicians who subscribe blindly to this do so at their own peril. A growing body of literature is suggesting that low DBP may also contribute to greater mortality in elderly people. One such article that is cited as evidence for treating isolated systolic hypertension[5] also notes that DBP is inversely correlated with total mortality. A post hoc analysis of the Systolic Hypertension in the Elderly Program study[6] revealed that patients whose treatment with antihypertensives resulted in a 5-mmHg decrease in DBP suffered more strokes, coronary heart disease, and cardiovascular disease. It was also noted that a DBP less than 70 mmHg was associated with greater risk of cardiovascular disease.

Further complicating this issue is the subject of dementia in elderly patients specifically with regard to the management of blood pressure. One study noted that, in patients aged 85 and older,[7] lower DBP, especially in patients taking antihypertensives, conferred a greater risk of developing all-cause dementia. This finding was also noted in a second study that compared baseline DBP in an elderly population followed for 6 years.[8] Patients with DBP less than 66 mmHg were much more likely to suffer from dementia at the conclusion of the study. Nevertheless, not all studies have confirmed this association.[9]

In patients with SBP ranging from 131 to 159 who are aged 80 and older, it is hard to arrive at an evidence-based statement regarding treatment. By establishing quality indicators with little data, it will be difficult to initiate the studies that are required. Perhaps the decision to initiate antihypertensives should be based upon DBP. In patients whose baseline SBP is less than 160, if a decision is made to treat, therapy should be initiated only if DBP can remain above 70 mmHg. In so doing, clinicians may prevent morbidity and mortality not only in patients with high SBP, but also in those with a low DBP.

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ACKNOWLEDGMENTS

Conflict of Interest: The editor in chief has reviewed the conflict of interest checklist provided by the author and has determined that none of the authors have any financial or any other kind of personal conflicts with this letters.