high-sensitivity cardiac troponin T) and with coronary heart disease (CHD), stroke, or death (1). They concluded that particularly among subjects with systolic blood pressure ≥120 mm Hg, and thus elevated pulse pressure, low DBP was associated with subclinical myocardial damage and coronary events.

In this insightful analysis, a very low DBP coupled with a high pulse pressure seemed to be very deleterious and these results put new perspective on studies that support lower targets for blood pressure lowering therapy (2).

It has been shown that overaggressive antihypertensive treatment, that leads to low DBP and thus hypoperfusion of the coronary arteries, results in cardiac ischemic events (3). McEvoy et al. (1) focused their attention on DBP as the predominant determinant of coronary perfusion. However, it should be remarked that several factors in addition to DBP, such as perfusion time, vessel wall diameter, and vasomotor tone, may importantly affect coronary blood flow. It has been reported that any increase in heart rate (HR) impinges on diastolic time more than on systolic time, reduces the perfusion time, and leads to subendocardial ischemia, especially in patients with coronary artery disease (4). Furthermore, a large body of evidence indicates that high HR can be considered a strong predictor of cardiovascular morbidity and mortality in different clinical settings (4). In their report, the authors, unfortunately, did not address the association of HR with subclinical myocardial damage and events across the various DBP categories. Considering that HR is a simple parameter that strongly correlates with diastolic coronary flow it should be routinely taken into account for refining risk stratification of CHD in treated hypertensive patients.

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http://dx.doi.org/10.1016/j.jacc.2016.11.086

Please note: Both authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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Diastolic Hypotension and Myocardial Ischemia

A Reason to Remember Cuff Artifact in Blood Pressure Measurement

In their important paper showing that diastolic hypotension increased the risk of myocardial ischemia, McEvoy et al. (1) nicely explained the mechanism: similar to cerebral perfusion, most of myocardial perfusion occurs during diastole. They found that the risk was largely driven by a wide pulse pressure (>60 mm Hg), meaning that the persons at risk had stiff arteries. This supports the hypothesis (2,3) that the J curve may be due to diastolic pressures that are actually much lower than measured by a cuff.

In 1978, colleagues and I reported (4) that among patients >60 years of age with diastolic pressures >100 mm Hg but no end-organ disease, one-half had a cuff diastolic pressure that was 30 mm Hg higher than the intra-arterial pressure. At the time I called this “pseudohypertension” because I was focusing on the cutoff of 90 mm Hg then used to define the need for antihypertensive therapy. A better name would be cuff artifact. We found that mean arterial pressure calculated from cuff pressures more closely approximated intra-arterial pressures (5). I have estimated that this problem occurs in ~4% of patients attending hypertension clinics, but it is likely that lesser degrees of cuff artifact are much more common.

In patients with stiff arteries, bradycardia widens pulse pressure because a larger stroke volume is being pushed into a stiff aorta. Thus beta-blockers or diltiazem may aggravate the problem of unrecognized diastolic hypotension. In elderly patients who complain of hypotensive symptoms, but whose cuff pressures do not seem to be hypotensive, it is important to measure intra-arterial pressure. The findings of McEvoy et al. (1) serve as a reminder to consider that the blood pressure measured by a cuff may actually be much higher than the true blood pressure.

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http://dx.doi.org/10.1016/j.jacc.2016.11.085

Please note: Dr. Spence has reported that he has no relationships relevant to the contents of this paper to disclose.

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The Role of Heart Rate in Diastolic Coronary Perfusion and Subclinical Myocardial Ischemia

We read with interest the paper by McEvoy et al. (1) describing the association between low diastolic blood pressure and adverse clinical and subclinical (elevated troponin level) cardiovascular outcomes. The authors postulate that low diastolic blood pressure impairs coronary perfusion and thus causes adverse cardiac events. This mechanism is certainly plausible, but we wonder whether heart rate could have had an influence on the observed results. Heart rate affects diastolic pressure-time index, which is more important than diastolic pressure alone in determining coronary perfusion (2). Diastolic perfusion index is strongly influenced by heart rate and cardiac ejection duration, as demonstrated in large cohorts of both cardiology outpatients (3) and healthy volunteers (4). Additionally, the role of systolic pressure-time loading and its relationship to diastolic pressure-time index and overall myocardial oxygenation (i.e., myocardial demand-supply ratio) (2–4) is important to consider, and is itself strongly affected by heart rate and cardiac ejection duration. The findings of McEvoy et al. (1) highlight an important clinical issue—maintenance of adequate diastolic blood pressure to facilitate coronary perfusion. We would be interested to know to what extent heart rate interacted with their findings.

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http://dx.doi.org/10.1016/j.jacc.2016.11.087

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In the Treatment of Hypertension, Lowering of Diastolic Pressure to <70 mm Hg Is Often Unavoidable

In the treatment of hypertension, lowering of diastolic pressure to <70 mm Hg is often unavoidable. McEvoy et al. (1) examined the association of diastolic blood pressure (DBP) with coronary heart disease (CHD), stroke and death using the ARIC (Atherosclerosis Risk In Communities) study cohort. They noted increased CHD risk of 1.5, 1.2, and 1.2 for DBP <60, 70, and 80 mm Hg, respectively. A similar relationship was seen in subjects treated for hypertension at baseline. The authors concluded that in the treatment of hypertension it may be prudent to ensure that DBP levels do not fall to <70 mm Hg. In our opinion, the linkage between