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*H*eart rate (HR) is the most important determinant of myocardial oxygen demand, playing a fundamental role in cardiac metabolic requirements.[1] There are several reasons for its well-recognized detrimental effect on myocardial ischemia. Increasing HR increases myocardial oxygen demand, creating an imbalance in the demand/supply ratio. It also shortens diastole, which diminishes coronary filling, hence myocardial perfusion, while also decreasing collateral flow to the ischemic area. Paced increases in HR in patients with coronary artery disease (CAD) induce vasoconstriction, thereby compounding the hemodynamic impact of coronary artery stenosis.[2] Several studies have shown that an increase in HR precedes most episodes of ischemia. An increase from 60 to 80 beats per minute (bpm) doubles the incidence of ischemic episodes,[3] while reversal of the β-blocker-induced reduction in HR attenuates or even reverses its benefits in left ventricular dysfunction.[4] HR is thus a key determinant of myocardial ischemia and cardiac function. Recent large-scale studies indicate that an increased resting HR is associated with a continuous increase in all-cause and cardiovascular mortality.[5,6] It has also been related to an increased predisposition to plaque rupture, leading to acute coronary syndromes.[7] HR reduction after acute myocardial infarction has a positive impact on prognosis, with several studies showing significant reductions in cardiac death, sudden death, and reinfarction.[8,9] HR reduction has also proved a good predictor of survival in heart failure studies.[10,11] These findings have had a direct impact on recent guidelines regarding the use of HR as a therapeutic target. The American College of Cardiology/American Heart Association guidelines recommend maintaining HR at 55 to 60 bpm in chronic stable angina.[12] The European Society of Cardiology guidelines recommend selective I_f current inhibitors (ivabradine), as alternatives to β-blockers for HR reduction (Class IIa; Level of evidence B).[13] Given the adverse impact of an elevated HR on myocardial ischemia, its association with poor prognosis in CAD, and the prognostic benefit of HR lowering, the question "When do you start taking resting HR into account when choosing a treatment in patients with stable CAD?" should not be too difficult to answer. It is, however, remarkable that there should be no clear recommendations in this regard. However, in accordance with current guidelines and consensus opinion, a resting HR of 55 to 60 bpm should be targeted in patients with stable CAD. In conclusion, I think it is becoming clearer than ever that HR should be considered a therapeutic target in CAD, although this still requires backing by further clinical data. Valuable information in this regard should soon be forthcoming from large-scale trials, in particular the morBidity-mortality EvAl-UaTion of the I_f inhibitor ivabradine in patients with coronary disease and left ventricULar dysfunction (BEAUTI_fUL). ❏

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