Metoprolol-Induced Changes in Myocardial 
$^{123}$I-Metaiodobenzylguanidine Uptake in Parkinson’s Disease

Paul A.R. de Milliano, MD; Berthe L.F. van Eck-Smit, MD, PhD; Andre C. de Groot, MD; Kong I. Lie, MD, PhD

A 58-year-old man who had had Parkinson’s disease for many years was referred to the Department of Cardiology because of progressive and severe symptoms of orthostatic hypotension. Examination revealed a drop in systolic blood pressure on standing of 25 mm Hg. Heart rate at rest was 84 bpm, and it increased to 88 bpm on standing. Cardiac examination showed no abnormalities. 2D echocardiography was normal except for a slightly decreased ejection fraction (48%, Simpson’s rule). The patient was treated with metoprolol, with a gradual increase in dose to 200 mg/d. After 6 months of treatment, symptoms of orthostatic hypotension completely disappeared. On examination, no drop in blood pressure was observed. Before and after 6 months of treatment, single photon emission CT (SPECT) $^{123}$I-metaiodobenzylguanidine (MIBG) scintigraphy of the heart was performed. At baseline, almost no myocardial MIBG uptake was observed, as displayed in Figure 1, showing short-axis reconstructions of $^{123}$I-MIBG SPECT acquisitions. After treatment, a dramatic increase in MIBG uptake can be seen (Figure 2, showing the same reconstruction as Figure 1), indicating restoration of functional nerve endings in the myocardium with metoprolol.

Patients with Parkinson’s disease frequently exhibit symptoms of autonomic failure that suggest derangements of the sympathetic and/or parasympathetic nervous system.

Parkinson’s patients with sympathetic neurocirculatory failure have a loss of cardiac norepinephrine spillover and absence of myocardial 6-$^{18}$F]fluorodopamine–derived radioactivity. In early and late stages of Parkinson’s disease, a decreased uptake of $^{123}$I-MIBG has been described. MIBG is a structural analogue of norepinephrine and follows the same metabolic pathways as norepinephrine. A decreased myocardial uptake of $^{123}$I-MIBG indicates a loss of functional myocardial nerve endings.

References
Figure 1. Short-axis reconstructions of cardiac $^{123}$I-MIBG SPECT acquisitions before treatment with metoprolol in a patient with Parkinson’s disease.

Figure 2. Short-axis reconstructions of cardiac $^{123}$I-MIBG SPECT acquisitions after 6 months of treatment with metoprolol in the same patient as in Figure 1.
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