The Effect of Multiple Sclerosis on Carotid Baroreflex Control of Heart Rate and Blood Pressure

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ABSTRACT

Multiple sclerosis (MS) is marked by conduction abnormalities within the central nervous system that can lead to impaired blood pressure regulation. However, the impact of this disease on dynamic neural control—responsiveness and timing (i.e., latency)—of blood pressure has not been examined. Utilizing a variable neck chamber system, we tested the hypothesis that patients with MS (MS: n=4) exhibit an altered response following baroreflex perturbation compared to sex and age matched healthy controls (CON: n=4). At rest, 5-sec pulses of neck suction (NS; -60 Torr) and neck pressure (NP; +40 Torr) were applied to simulate carotid hypertension and hypotension, respectively. Mean arterial pressure (MAP; Finometer) and heart rate (HR) were continuously measured in response to the perturbations. Carotid baroreflex (CBR) latencies (i.e., time-to-peak responses) were examined using carotid-cardiac (peak HR responses), carotid-vasomotor (peak MAP responses), and change in MAP at the peak HR response of the corresponding stimuli (MAP@HR_{peak}), all of which were not significant for both NP and NS. Following NS, responses in MAP (MS: -12±5, CON: -10±3 mmHg; p=0.43) and HR (MS: -9±3, CON: -8±4 BPM; p=0.58) were similar between groups. Following administration of NP, HR responses (MS: 4±2, CON: 5±4 BPM; p=0.47) were no different. However, the differences found in MAP were significant (MS: 5±2, CON: 8±2 mmHg; p=0.05), providing some evidence that baroreceptor responsiveness may be compromised when faced with a hypotensive challenge.