



# **Autonomic Nervous System: Chapter 22. Diabetic autonomic neuropathy (Handbook of Clinical Neurology)**

*Aaron I. Vinik, Tomris Erbas*

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Autonomic neuropathy, once considered to be the Cinderella of diabetes complications, has come of age. The autonomic nervous system innervates the entire human body, and is involved in the regulation of every single organ in the body. Thus, perturbations in autonomic function account for everything from abnormalities in pupillary function to gastroparesis, intestinal dysmotility, diabetic diarrhea, genitourinary dysfunction, amongst others. “Know autonomic function and one knows the whole of medicine!” It is now becoming apparent that before the advent of severe pathological damage to the autonomic nervous system there may be an imbalance between the two major arms, namely the sympathetic and parasympathetic nerve fibers that innervate the heart and blood vessels, resulting in abnormalities in heart rate control and vascular dynamics. Cardiac autonomic neuropathy (CAN) has been linked to resting tachycardia, postural hypotension, orthostatic bradycardia and orthostatic tachycardia (POTTS), exercise intolerance, decreased hypoxia-induced respiratory drive, loss of baroreceptor sensitivity, enhanced intraoperative or perioperative cardiovascular lability, increased incidence of asymptomatic ischemia, myocardial infarction, and decreased rate of survival after myocardial infarction and congestive heart failure. Autonomic dysfunction can affect daily activities of individuals with diabetes and may invoke potentially life-threatening outcomes. Intensification of glycemic control in the presence of autonomic dysfunction (more so if combined with peripheral neuropathy) increases the likelihood of sudden death and is a caveat for aggressive glycemic control. Advances in technology, built on decades of research and clinical testing, now make it possible to objectively identify early stages of CAN with the use of careful measurement of time and frequency domain analyses of autonomic function. Fifteen studies using different end points report prevalence rates of 1% to 90%. CAN may be present at diagnosis, and prevalence increases with age, duration of diabetes, obesity, smoking, and poor glycemic control. CAN also cosegregates with distal symmetric polyneuropathy, microangiopathy, and macroangiopathy. It now appears that autonomic imbalance may precede the development of the inflammatory cascade in type 2 diabetes and there is a role for central loss of dopaminergic restraint on sympathetic overactivity. Restoration of dopaminergic tone suppresses the sympathetic dominance and reduces cardiovascular events and mortality by close to 50%. Cinderella’s slipper can now be worn!

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