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Effects of weight loss pharmacotherapy on vascular function in humans with obesity-hypertension

Obesity is associated with sympathoactivation, hypertension, and atherosclerosis. It is not known whether increased sympathetic neural outflow translates into exaggerated sympathetically mediated vasoconstriction in obesity, nor whether weight loss can reduce the impact of obesity and hypertension on sympathetic tone and resistance vessel function.

We assessed sympathetic tone in humans by measuring forearm vasodilatation to intra-arterial administration of the alpha-adrenergic receptor antagonist phentolamine, and by recording microneurographic sympathetic nerve activity to muscle (mSNA). Obese and hypertensive subjects demonstrated increased mSNA. However, vasodilatation to phentolamine was not augmented in obese normotensive and hypertensive subjects versus lean. Intriguingly, lean hypertensives had markedly augmented sympathetic vasoconstrictor tone. Obese subjects then underwent a 3-month weight loss program using the intestinal lipase inhibitor, orlistat (but with no change in physical activity). Weight loss of approximately 10% reduced mSNA and BP, but did not alter vasodilatation to phentolamine. We tested forearm resistance vessel function using intra-arterial nitroprusside (nitric oxide donor) and isoproterenol (beta-adrenergic receptor agonist). Dilatation to both was blunted only in obese hypertensive subjects. Orlistat-induced weight loss normalized the response to nitroprusside but not to isoproterenol in obese hypertensive subjects.

Thus, resistance vessel sympathetic tone is not increased in obesity despite higher mSNA. This dissociation could be due to a different target of sympathoactivation. In contrast, hypertensive subjects without obesity have exaggerated sympathetically-mediated vascular tone. Obesity without hypertension does not impair resistance vessel function in humans. However, obesity plus hypertension causes vascular smooth muscle dysfunction, which is improved by pharmacologically-induced weight loss.

Biography

William G. Haynes is a Professor of Internal Medicine at the University of Iowa (Divisions of Endocrinology and Cardiology). Dr. Haynes's NIH-funded research focuses on the mechanisms of hypertension and vascular damage in obesity. He provided the first description of the sympathoexcitatory effects of leptin. He has published over 150 papers, reviews and book chapters. Dr. Haynes is a member of the American Society for Clinical Investigation, has won awards from the International Society of Hypertension, and the American Heart Association, and was Associate Editor of Arteriosclerosis, Thrombosis and Vascular Biology.

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