of blood pressure in these patients may also be a parallel recommendation.

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REFERENCES

1. Greenberg SM. Cerebral amyloid angiopathy: Prospects for clinical diagnosis and treatment. Neurology 1998;51:690–694.
2. Thal DR, Griffin WS, de Vos RA et al. Cerebral amyloid angiopathy and its relationship to Alzheimer's disease. Acta Neuropathol 2008;115:599–609.
3. Yamada M. Cerebral amyloid angiopathy: An overview. Neuropathology 2000; 20:8–22.
4. Rebeck GW, Kindy M, LaDu MJ. Apolipoprotein E and Alzheimer's disease: The protective effects of ApoE2 and E3. J Alzheimers Dis 2002;4:145–154.
5. Rensink AA, de Waal RM, Kremer B et al. Pathogenesis of cerebral amyloid angiopathy. Brain Res Brain Res Rev 2003;43:207–223.
6. Nicoll JA, Burnett C, Love S et al. High frequency of apolipoprotein E epsilon 2 allele in hemorrhage due to cerebral amyloid angiopathy. Ann Neurol 1997; 41:716–721.
7. Greenberg SM, Vonsattel JG, Segal AZ et al. Association of apolipoprotein E e2 and vasculopathy in cerebral amyloid angiopathy. Neurology 1998;50: 961–965.
8. McCarron MO, Nicoll JA, Ironside JW et al. Cerebral amyloid angiopathy-related hemorrhage. Interaction of APOE epsilon2 with putative clinical risk factors. Stroke 1999;30:1643–1646.
9. Rosand J, Hylek EM, O'Donnell HC et al. Warfarin-associated hemorrhage and cerebral amyloid angiopathy: A genetic and pathologic study. Neurology 2000; 55:947–951.

WEIGHT MAINTENANCE IN HOSPITALIZED GERIATRIC PATIENTS TREATED WITH BETA-BLOCKERS

To the Editor: Malnutrition is a significant problem in elderly people. Up to 7% of community-dwelling and 8% to 45% of hospitalized elderly people are malnourished.[1,2] The cause of malnutrition is usually multifactorial, including anorexia and increased energy expenditure during acute illness. Common comorbidities in elderly people, including hip fractures, heart failure, and obstructive pulmonary diseases, are associated with an increase in energy expenditure,[3,4] which may contribute to weight loss.

The sympathetic nervous system (SNS) is an important regulator of energy expenditure and substrate use. Sympathetic blockade using β-blockers in patients with head trauma and severe burns antagonizes catecholamine-induced tissue catabolism,[5,6] although the effect of β-blockade on weight loss in hospitalized elderly patients has not been examined. The aim of this study was to determine whether β-blocker use influences weight change during hospital admission in elderly patients.

A cross-sectional evaluation of anthropometric data was undertaken in 47 consecutive elderly patients admitted to the geriatrics ward at St. Vincent's Hospital, Sydney, Australia, between January and March 2008. Anthropometric measurements were repeated over 1 week of hospitalization on days 3 and 7. Patients with diabetes mellitus; organ transplants; severe cardiac, renal, or hepatic impairment; fever; or significant infection and patients taking corticosteroids or antipsychotic medications were excluded.

Body weight and height were measured in hospital gowns on the same electronic scale. Body mass index (BMI) was calculated as body weight in kilograms divided by the square of height in meters. The Human Research Ethics Committee, St. Vincent's Hospital, approved the study. Verbal consent was obtained from patients for anthropometric measurements.

Twenty-two (46.8%) of the 47 inpatients (mean age 83.2 ± 6.0) were treated with β-blockers. All patients had been taking selective β_1-blockers (atenolol 84.8%, metoprolol 15.2%) for at least 3 years. Most common causes for admission were falls (66.8%), delirium (55.2%), and progressive debility (48.0%). Frequency of diagnoses, blood pressure, and the number of medications taken by patients on admission were similar in β-blocker users and nonusers (data not shown). Admission body weight ($60.2 \pm 0.4\,kg/m^2$ vs $58.9 \pm 0.5\,kg/m^2$, $P = .4$) and BMI ($23.8 \pm 3.2\,kg/m^2$ vs $22.6 \pm 3.9\,kg/m^2$, $P = .09$) were not significantly different in β-blocker users and nonusers. BMI was less than $22\,kg/m^2$ in 43%. Pulse rate was significantly lower in β-blocker users (69 ± 10 vs 83 ± 12 beats/min, $P < .001$), suggesting adequate medication adherence.

During the 1-week follow-up, β-blocker nonusers lost weight, whereas users maintained their admission weight (Figure 1). The change in body weight was significantly greater in β-blocker nonusers than users by day 3 (-1.2 ± 0.7 vs $-0.1 \pm 0.9\,kg$, $P = .004$) and day 7 (-2.1 ± 1.2 vs $-0.2 \pm 1.1\,kg$, $P < .001$). Forward stepwise multiple regression was performed to determine the contribution of age, sex, blood pressure, pulse rate, and antihypertensive medication use to body weight reduction. In this model, β-blocker use was the only variable associated with weight change over the 1-week period (adjusted coefficient of variation = 0.43, $P < .001$).

The current study revealed a clinically significant weight-maintaining effect of β-blockers in geriatric patients, evident within 1 week of hospitalization. This study has potential clinical importance, given the high rates of malnutrition in elderly hospitalized patients.[1,2] In one study, more than 50% of elderly hospitalized patients had a BMI less than $22\,kg/m^2$.[7] In another, approximately 1 kg of weight loss accompanied every week of hospitalization.[2] Malnutrition is associated with adverse hospital and

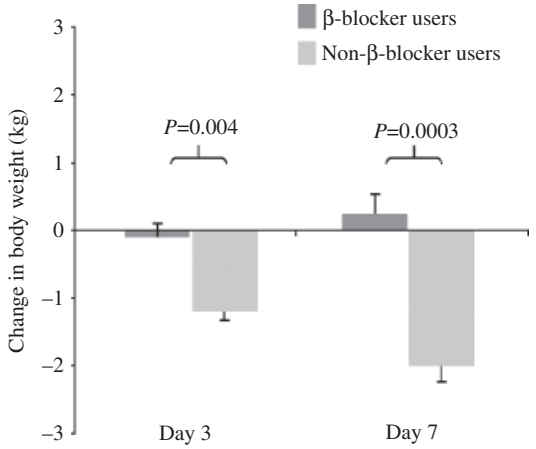

Figure 1. Change in body weight at days 3 and 7 (β-blocker user = 22, nonuser = 25) (data are means ± standard errors).

postdischarge clinical outcomes, including longer length of stay, higher rates of minor and major complications and mortality, and higher costs.[1–4,7]

The mechanisms that contribute to a possible effect of β-blockers on weight and body fat remain unknown. SNS activation has been shown to be an important inhibitor of food intake in rodents.[8] Inhibition of the SNS using β-blockers would then be expected to stimulate food intake. In addition, SNS inhibition by β-blockers may reduce weight loss in hospitalized patients through a concomitant reduction in energy expenditure and stress-induced lipolysis.[9] Although these metabolic changes would be detrimental in obese patients using β-blockers in the treatment of hypertension, they may be beneficial in elderly patients for the maintenance of body weight.

The current study may have significant clinical implications. Therapeutic interventions for weight loss are limited in the elderly population. Adherence to nutritional supplements is generally poor.[10] Although the cross-sectional design of the current study cannot establish causality, the observation of a rapid divergence in weight within 1 week of hospitalization suggests that short-term β-blocker therapy may have a beneficial role in weight maintenance in elderly hospitalized patients. Further research is required to investigate the role of short-term β-blockade on energy balance in hospitalized elderly patients.

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REFERENCES

1. Brantervik AM, Jacobsson IE, Grimby A et al. Older hospitalised patients at risk of malnutrition: Correlation with quality of life, aid from the social welfare system and length of stay? Age Ageing 2005;34:444–449.
2. de Luis D, Lopez Guzman A. Nutrition Group of Society of Castilla-Leon (Endocrinology, Diabetes and Nutrition). Nutritional status of adult patients admitted to internal medicine departments in public hospitals in Castilla y Leon, Spain—A multi-center study. Eur J Intern Med 2006;17:556–560.
3. Gaillard C, Alix E, Sallé A et al. Energy requirements in frail elderly people: A review of the literature. Clin Nutr 2007;26:16–24.
4. Gariballa S, Forster S. Energy expenditure of acutely ill hospitalised patients. Nutr J 2006;29:9.
5. Herndon DN, Hart DW, Wolf SE et al. Reversal of catabolism by beta-blockade after severe burns. N Engl J Med 2001;345:1223–1229.
6. Cotton BA, Snodgrass KB, Fleming SB et al. Beta-blocker exposure is associated with improved survival after severe traumatic brain injury. J Trauma 2007;62:26–33.
7. Brantervik AM, Jacobsson IE, Grimby A et al. Older hospitalised patients at risk of malnutrition: Correlation with quality of life, aid from the social welfare system and length of stay? Age Ageing 2005;34:444–449.
8. Tsujii S, Bray GA. A beta-3 adrenergic agonist (BRL-37,344) decreases food intake. Physiol Behav 1998;63:723–728.
9. Lamont LS, Brown T, Riebe D et al. The major components of human energy balance during chronic beta-adrenergic blockade. J Cardiopulm Rehabil 2000; 20:247–250.
10. Gariballa S, Forster S, Walters S et al. A randomized, double-blind, placebo-controlled trial of nutritional supplementation during acute illness. Am J Med 2006;119:693–699.

SURVIVAL OF NONAGENARIAN PATIENTS ADMITTED TO THE INTENSIVE CARE UNIT FOR SYMPTOMATIC CARDIAC RHYTHM DISTURBANCES IN NEED OF A PACEMAKER

To the Editor: A progressive increase in life expectancy has led health care to specialize in elderly patients. Various studies have shown that admission of these patients to intensive care units is justified,[1,2] but nonagenarians could be