Motorized Scooters: Boon or Bane?

We read with interest the report by Zagol and Krasuski1 on motorized scooters. The investigators suggested that scooters can have detrimental long-term effects on cardiovascular risk and that their findings pertinent to risk should influence physician practice. We are concerned that the study’s methods do not support such a conclusion. Specific limitations include the following: (1) the use of a retrospective cohort study design with no attempt to account for other factors potentially affecting outcomes (e.g., by using statistical, case-matched, or historical control), (2) incomplete data on other treatment pertinent to the outcomes being studied, and (3) very low survey response rates. These limitations must be considered in interpreting the study findings.

A retrospective cohort study design does not permit attributions of causality. It is impossible to know whether observed changes were caused by the natural histories of patients’ diseases. One recent study of the natural histories of diabetes and hyperlipidemia in a tertiary care outpatient program showed changes very similar to those in this population over the course of a year or so of follow-up: 25% of patients had diabetes at baseline, and on average, diabetic control decreased substantially over time (e.g., rates of diabetes doubled and mean glucose levels increased), while 60% had hyperlipidemia and average lipid levels improved substantially over time.2 The lack of a comparator group undoubtedly magnifies the attributed risks.

The problems with using a retrospective cohort study design are compounded by differential reporting on the medical management of diabetes, hypertension, and hyperlipidemia and differential attribution of causality. Zagol and Krasuski1 attributed unfavorable changes in blood pressure and diabetes control to the scooters and attributed favorable changes in the control of hyperlipidemia to medical management. Various alternative explanations exist that were not considered (e.g., variable efficacy and/or adherence with treatment for the various conditions).

The survey response rate was only 28% (far too low to attribute results to the population as a whole). Some journals, such as JAMA, will publish reports on survey results only when response rates are ≥60%.3 Despite this low response rate, Zagol and Krasuski1 reported metabolic parameters for the entire group, and they were not reported for the survey respondents. Thus, the survey nonrespondents are disproportionately represented in the metabolic outcomes. A more definitive conclusion could have been drawn by analytically coupling metabolic parameters with survey results. For all we know, the survey respondents may have been the group benefiting most from scooters, using them to get out and become more active, while the nonrespondents were recalcitrant to medical and lifestyle interventions, not using the scooters, and having more disease progression.

The data on scooter use were self-reported, and the actual survey instrument was not provided. Scooter use 4 hours/day is a surprising result. Typically, scooters are used for mobility outside the home, and patients walk inside. Because of scooters’ wide turning radii, their use within most homes is difficult; furthermore, scooter seats are uncomfortable for prolonged use. Patients who require power mobility inside and outside usually use power wheelchairs with specialized seating. Even then, the amount of use is highly variable,4 for example, averaging only 2 hours/day in patients with severe neurologic disease.5 In our randomized trial of motorized scooters for ambulatory patients, the scooters were used almost daily, commonly to transition between locations, and steps per day did not decrease in scooter users.6

We agree with Drs. Zagol and Krasuski1 that further investigation is needed to understand health and functional impacts of scooters and identify the types of patients who benefit from scooters. It would be unfortunate if physicians and third-party payers were biased by this very limited study against providing equipment that may substantially improve the quality of life.


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Reply: Dr. Hoenig et al highlight some of the intrinsic limitations of our retrospective cohort study. Although no control group was used, the annual incidence and prevalence of diabetes in our study group over the 1 year of follow-up was dramatically higher than expected in the general population. According to the Centers for Disease Control and Prevention, the average age-adjusted annual incidence rate of diabetes in adults from 2005 to 2007 was 11.1 per 1,000 individuals in Texas and 9.1 per 1,000 individuals for the entire United States population.1 The observed incidence rate for diabetes (301.6 per 1,000 subjects) in our cohort was markedly higher. Furthermore, the prevalence of diabetes in adults
aged 65 to 74 years from 2005 to 2007 was 21.2% for Texas and 18.5% for the United States population. Although our patient population had a somewhat higher prevalence of diabetes at baseline (38%), at the end of 1 year of follow-up, the prevalence of diabetes (57%) was >2 times their Texas age-matched counterparts and >3 times that of similar United States individuals, a magnitude suggesting a true finding.

As Dr. Hoenig et al correctly point out, the mean response rate among mail surveys published in medical journals is 60%. The general response rates for postal questionnaires, however, is 31.5%, a number similar to our response rate. Interestingly, a much larger study surveying Medicare-aged patients in the same catchment area received only a 12% response rate to a very short postal survey (4,653 respondents of 39,222 questionnaires). To assess whether our survey respondents differed from nonrespondents, we compared glycemic effects and found that blood glucose increased similarly in the 2 groups over the year of follow-up and that respondents actually trended toward more new diagnoses of diabetes (32% vs 11%, p = 0.056) compared with nonrespondents. Because the survey results were only a small aspect of our study, we do not believe that the low response rate should diminish the overall study findings.

Although we agree that our study design limitations preclude the attribution of causality, we believe that the findings do support our conclusions that further study in this area is warranted and that discussion with patients to ensure maintenance of tolerated physical activity after scooter prescription is important. We also believe very strongly that physicians and third-party payers should not be biased by our small, limited study against providing equipment that may substantially improve overall quality of life.

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Limiting Adverse Cardiac Remodeling After Acute Myocardial Infarction by Blocking Interleukin-1

Abbate et al reported success in limiting adverse cardiac remodeling after acute myocardial infarction by blockade of interleukin-1. This promising new approach used anakinra, a natural substance.

Their report also has significance for another noncardiac disorder, sudden infant death syndrome (SIDS). In 1989, I suggested that interleukin-1 was the intermediary causing prolonged sleep apnea and SIDS during respiratory infections. Interleukin-1 and infections cause fever, activation of the immune system, and increased slow-wave sleep. As any parent knows, mild respiratory infections are common in infants and children, usually only a nuisance, but they have a statistical correlation with SIDS, and SIDS almost always occurs during sleep. The combination of respiratory infection and deep sleep may cause prolonged apnea and, if not interrupted by arousal, lead to hypoxic apnea and death.

This possibility of involvement of interleukin-1 was supported by a Norwegian group in 1995, who found that at autopsy, SIDS cases had increased interleukin-6 in the cerebrospinal fluid. The prevention of SIDS theoretically might be possible if anakinra were given to infants with respiratory infections at bedtime, but that is not justified, unlike the “back-to-sleep” positioning of all sleeping infants, which lowered the rate of SIDS by 50% and has no side effects.

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Valsartan Inefficacy or Ill-Effects of Concomitant Medications!

In the provocative review by the Nateginlind and Valsartan in Impaired Glucose Tolerance Outcomes Research (NAVIGATOR) investigators, a single daily dose of valsartan reduced the risk for diabetes by 14% compared with placebo, a relative risk reduction lower than that seen even with lifestyle modification in some trials. However, approximately 40% of patients were taking β blockers and 40% diuretics, agents that have been consistently shown to have adverse metabolic effects. In a meta-analysis of randomized trials of β blockers in patients with hypertension, treatment of 1,000 patients for 4.4 years resulted in 14 excess cases of diabetes. Similarly, thiazide diuretics increased the risk for diabetes by 32% compared with placebo or non-β-blocker agents.

Conceivably, angiotensin-converting enzyme inhibitors or angiotensin receptor blockers, by mitigating hypokalemia associated with thiazide diuretics and by other effects, may abolish some of these adverse glycemic effects if given concomitantly with β blockers or diuretics. However, in the Study of Trandolapril/Verapamil SR and Insulin Resistance (STAR), in which patients with the metabolic syndrome were randomized to either verapamil and trandolapril or to losartan and hydrochlorothiazide, at the end of 1 year, losartan and hydrochlorothiazide increased plasma glucose significantly more than verapamil and trandolapril after all oral glucose tolerance testing. Similar results were documented in the International Verapamil-Trandolapril (INVEST) study and the Effects in Diabetes Mellitus: Carvedilol-Metoprolol Comparison in Hypertensives (GEMINI) study.