grams serving the older homeless population, counseling on substance use, addressing risk factors for falls, and facilitating access to glasses or hearing aids may help avoid a substantial number of ED visits and hospitalizations.

The study has several limitations. We may not have captured all ED visits or hospitalizations, particularly if they occurred outside Boston. Moreover, because the study was conducted in Massachusetts, a state with universal health insurance, our results may not be generalizable to other states.

Providing primary care to older patients living in the street or a shelter is challenging. Focusing limited resources on targeting modifiable factors, including alcohol problems and common geriatric conditions, may lower rates of burdensome and costly acute care use in this vulnerable population.

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The Influence of Hyperglycemia on the Therapeutic Effect of Exercise on Glycemic Control in Patients With Type 2 Diabetes Mellitus

Randomized clinical trials show that aerobic exercise training improves glycemic control in patients with type 2 diabetes mellitus (T2DM).1 However, interindividual variability is large.2 This may be explained by genetic variability,3 but ambient hyperglycemia4 and pancreatic β-cell function5 may also contribute. We examined whether changes in glycemic control following a 12- to 16-week aerobic exercise training intervention were influenced by the pretrained glycemic state in 105 individuals with impaired glucose tolerance or T2DM.

Methods | This study was approved by our institutional review board, and subjects provided informed consent. Before and following a 12- to 16-week period of aerobic exercise training, body composition, aerobic fitness (maximal oxygen uptake [VO2max]), and glycemic control (hemoglobin A1c [HbA1c], fasting glucose, and oral glucose tolerance test [OGTT] levels) were determined in a total of 105 older (mean [SEM] age, 61 [1] years), overweight or obese (mean [SEM] body mass index, 33.1 [1] [calculated as weight in kilograms divided by height in meters squared]) individuals with impaired glucose tolerance (n = 56) or T2DM (n = 49; diagnosed a mean [SEM] 4.8 [0.9] years prior and not insulin treated). Relationships between preintervention variables and intervention-induced changes in variables were assessed by linear and nonlinear regression. See eMethods in the Supplement for full details of the study design.

Results | Mean (SEM) change in body weight (−4.6 [0.5] kg), whole-body adiposity (−1.9% [0.3%]), VO2max (±0.23 [0.03] L/min), fasting plasma glucose (−0.35 [0.08] mmol/L), and 2-hour OGTT (−0.8 [0.2] mmol/L) (to convert glucose to milligrams per deciliter, divide by 0.0555) were significantly improved following exercise training (full data are given in the eTable in the Supplement). Pretraining fasting plasma glucose level did not influence exercise-induced changes in glycemic control. However, there was a nonlinear quadratic relationship between pretraining 2-hour OGTT and exercise-induced changes in the 2-hour glucose response (r2 = 0.26; P = .06) (Figure, A). Subjects with a pretraining 2-hour OGTT
Individuals with impaired glucose tolerance or type 2 diabetes mellitus underwent 12 to 16 weeks of moderate-intensity exercise training, 5 days per week for 60 minutes per day. Individual subject data points are plotted on both panels; the x-axis represents the pretraining variable, and the y-axis indicates the exercise-induced change, such that the data points above the axis indicate an exercise-induced increase and vice versa. Open circles represent impaired glucose-tolerant subjects, and open triangles represent subjects with type 2 diabetes mellitus. The solid line represents the regression curve, and the dotted line represents the 95% confidence interval. A, There was a nonlinear quadratic relationship between pretraining 2-hour oral glucose tolerance test (OGTT) level and the training-induced change in 2-hour OGTT level (\( r = 0.06x^2 - 1.5x + 76 \) \( r^2 = 0.26; P = .06 \) [N = 105]). For every 1-mmol/L increase in pretraining 2-hour glucose level above 13.1 mmol/L (the inflection point of the curve), there was a 0.2–percentage-point loss of improvement in 2-hour glucose level following exercise. B, There was also a nonlinear quadratic relationship between pretraining hemoglobin A\(_1c\) (HbA\(_1c\)) level and the training-induced change in HbA\(_1c\) level (\( y = 0.33x^2 - 3.8x + 11.7 \) \( r^2 = 0.33; P = .02 \) [n = 52]). For every 1–percentage-point increase in pretraining HbA\(_1c\) level above 6.2% (the inflection point of the curve), there was a 0.2–percentage-point loss of improvement in HbA\(_1c\) level following exercise. C, An inverse linear relationship between pretraining HbA\(_1c\) level and the training-induced change in aerobic fitness was found (\( y = -0.11x + 0.91 \) \( r = -0.38; P = .006 \) [n = 52]). For every 1–percentage-point increase in pretraining HbA\(_1c\) level, there was 0.11-L/min loss of improvement in maximal oxygen uptake (\( \dot{V}O_2\max \)) following exercise training.

**Discussion**

These findings emphasize that exercise-induced improvements in glycemic control are dependent on the pretraining glycemic level. We demonstrate that although moderate-intensity aerobic exercise can improve glycemic control, individuals with ambient hyperglycemia are the most likely to be nonresponders. Our key observation is that pretraining hyperglycemia predicts exercise-induced improvements in glycemic control: for every 1-mmol/L rise in pretraining 2-hour OGTT glucose level above 13.1 mmol/L (the curve inflection point in Figure, A) we predict a 0.2-mmol/L loss of improvement in 2-hour OGTT glucose following exercise. Accordingly, for every 1–percentage-point increase in pretraining HbA\(_1c\) level above 6.2% (the curve inflection point in Figure, B), we predict a 0.2 percentage point loss of improvement in HbA\(_1c\) level following exercise. Pretraining hyperglycemia also predicted the exercise-induced increment in aerobic fitness: for a 1–percentage-point increase in pretraining HbA\(_1c\) level, we predict a 0.11 L/min loss of improvement in \( \dot{V}O_2\max \) following exercise.

Prior work shows that diabetes remission following exercise and diet intervention is more likely in individuals with a shorter disease history and lower HbA\(_1c\) level. We show that aerobic exercise-induced improvements in glycemic control are blunted by ambient hyperglycemia, particularly in subjects with T2DM. Mechanistic studies are required to help us understand this phenomenon, but underlying impairments in β-cell function are likely to be very important. That hyperglycemia blunted the cardiovascular adaptations to exercise (\( \dot{V}O_2\max \)) is in agreement with some prior reports and may be explained by the causal association between chronic hyperglycemia and microvascular and macrovascular dysfunction.

The clinical relevance of these new findings is paramount and highlights the need to understand the metabolic “nonresponder.” Because chronic hyperglycemia (>6.2% HbA\(_1c\) level; >13.1 mmol/L glucose level) potentially predicts a poor therapeutic effect of aerobic exercise on glycemic control and fitness, using exercise to treat patients with poorly controlled T2DM may have limited chances of a successful outcome.

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**Propofol for Screening Colonoscopy in Low-Risk Patients: Are We Paying Too Much?**

Use of propofol for sedation for screening colonoscopy in low-risk patients has increased markedly recently. In the United States, propofol is administered only by an anesthesiology provider, which can substantially increase cost (typically $600-$2000). Given the number of screening colonoscopies performed every year, additional health care costs associated with this practice are substantial. The advantages of propofol over standard moderate sedation using a narcotic and/or benzodiazepine include quicker onset of action, a shorter half-life, and deeper level of sedation. However, evidence supporting the potential benefits of propofol is limited. For screening colonoscopy in low-risk patients, the question thus becomes, what is propofol worth? For a patient who has never had a colonoscopy, this is a very difficult question to answer, since it is challenging to associate a dollar amount to comfort without knowledge of the likely degree of discomfort. Hence, we approached this question by asking those who are most familiar with colonoscopy and sedation medications—namely gastroenterologists and endoscopy nurses.

**Methods** | We validated a 3-question questionnaire by interviewing and asking 20 endoscopy physicians and nurses from 3 different endoscopy units to answer questions in an opened-ended manner; responses were used to construct the final questionnaire, which was then reviewed by additional gastroenterologists. The questions were as follows:

1. If you were to have screening colonoscopy, what sedation would you prefer?
   - Unsedated.
   - Midazolam-fentanyl (moderate sedation).
   - Propofol (deep sedation).

2. If you prefer propofol, how much extra would you be willing to pay out of pocket?
   - $0
   - $1-$100
   - $101-$200
   - $201-$300
   - $301-$500
   - More than $500
   - Doesn’t matter

3. I prefer propofol because (check all that apply):
   - I do not want to feel anything.
   - My recovery time will be faster.
   - I want to be taken care of by an anesthesiologist/certified registered nurse anesthetist.

The questionnaire was placed on SurveyMonkey.com and the link emailed to specific gastroenterology division directors asking them to distribute it to their faculty. Responses were also obtained by direct distribution and/or interview at the Digestive Diseases Week (the major national gastroenterology meeting). To obtain nurse responses, we attended 6 Society of Gastroenterology Nurses Association meetings and distributed paper questionnaires at the beginning of random oral sessions; responses were collected immediately after the session.

**Results** | Responses were received from 451 gastroenterologists and 460 nurses. The response rate for nurses, calculated as number of questionnaires with responses divided by number of questionnaires distributed, was 84%. For the gastroenterologists, the response rate was 87% for directly distributed questionnaires and 23% by web (SurveyMonkey.com).