In Reply: There is no evidence that guidelines or training improve EMR note quality. In the absence of evidence, only expert opinion remains. It is my opinion as a medical educator, experienced if not expert, that training has minimal lasting impact.

Mark Twain wrote, “I didn’t have time to write a short letter, so I wrote a long one instead.”1 With EMR features such as copy-and-paste and one-click data import, it takes less time and effort to write long random notes than it does to write short structured ones. Until EMR systems are modified to reverse this time incentive, long notes will remain the norm.

A similar situation existed with regard to handwashing by health care workers. Decades of training and cajoling did little to improve handwashing compliance rates. Compliance improved only after it was made easier and faster to comply—by installation of conveniently located hand sanitizer dispensers.2 If short, cogent EMR notes are the goal, EMR system redesign is necessary.

I am impressed that Cornell’s Department of Medicine has committed faculty time and money to these efforts. However, while I sincerely hope that Dr Siegler proves me wrong, I remain skeptical regarding the efficacy of training in improving EMR notes.

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PANCREAS ORGAN WEIGHT IN INDIVIDUALS WITH DISEASE-ASSOCIATED AUTOANTIBODIES AT RISK FOR TYPE 1 DIABETES

To the Editor: Autopsy and imaging studies suggest that human pancreata from adults with type 1 diabetes (T1D) are smaller and weigh less than those without the disease.1,2 However, it is unknown when pancreatic atrophy begins in T1D.

Therefore, we examined pancreas weight early in the natural history of T1D from at-risk individuals without diabetes but with disease-associated autoantibodies, obtained through the Network for Pancreatic Organ Donors with Diabetes program.3 All donors were identified by organ procurement organizations that coordinate organ and tissue donations for clinical transplantation or research.

Methods. Between October 16, 2006, and April 27, 2012, 193 pancreata were recovered from cadaveric multiorgan donors in the United States and considered for the study following written informed research consent from the next of kin. Study procedures were in accordance with the Declaration of Helsinki and the University of Florida institutional review board, and were detailed previously.3

Information on demographics and medical history were obtained from chart review or through a data-sharing agreemen

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Table. Organ Donor Variables Considered for Inclusion in a General Linear Model of Factors Influencing Pancreas Weight\(^a\) (continued)

<table>
<thead>
<tr>
<th>Characteristic(^b)</th>
<th>No Diabetes ((n = 23))</th>
<th>Positive for Autoantibody Only ((n = 8))</th>
<th>Type 1 Diabetes ((n = 20))</th>
<th>Overall (P) Value(^c)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Autoantibody screening results, No. (%)</td>
<td>0</td>
<td>8 (100)</td>
<td>17 (85)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>C-peptide, median (range), ng/mL(^d)</td>
<td>6.7 (2.4-20.6)</td>
<td>3.4 (0.05-13.6)</td>
<td>0.05 (0.05-0.2)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Duration of diabetes, mean (SD), y(^e)</td>
<td>0</td>
<td>0</td>
<td>18.0 (10.0)</td>
<td>NA</td>
</tr>
<tr>
<td>Negative donor history of diabetes, No. (%)</td>
<td>22 (96)</td>
<td>8 (100)</td>
<td>0 (100)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Insulin dependence, No. (%)</td>
<td>0</td>
<td>0</td>
<td>19 (95)</td>
<td>NA</td>
</tr>
<tr>
<td>Non-heart beating donors, No. (%)</td>
<td>3 (13)</td>
<td>1 (12)</td>
<td>1 (5)</td>
<td>.57</td>
</tr>
<tr>
<td>Organ recovery time, mean (SD), h(^m)</td>
<td>14.5 (5.9)</td>
<td>18.7 (4.7)</td>
<td>17.0 (4.6)</td>
<td>.13</td>
</tr>
</tbody>
</table>

Abbreviation: NA, not applicable.

\(^a\)Collinear terms assessed using pairwise correlations for all continuous variables defined as any 2 factors with a \(P\) value of less than .05 and \(r\) value of 0.80 or greater. Multicollinearity was defined when the variance inflation factor was greater than 1 as previously described.\(^f\) Multicategorical variables were independently screened for collinearity using a \(\chi^2\) test. Interaction terms included both age and body mass index by disease status group. For all remaining candidate covariates considered in the final model, the assumptions of analysis of covariance were evaluated as described elsewhere.\(^g\)

\(^b\)Use of mean or median for continuous variables was based on the evaluation of normal distribution.

\(^c\)Determined for variables using analysis of variance if continuous and parametric, the Pearson \(r^2\) test if nominal, categorical, and values in all cells greater than 5, or by Freeman-Halton extension of the Fisher exact test if nominal, categorical, and values in 1 or more cells less than 5. \(P\) values were not calculated if values from 2 or more cells were equal to 0.

\(^d\)Donors aged 18 years or younger were excluded to minimize the possibility that changes in the outcome variable were due to normal organ growth during the early postnatal stages of life.

\(^e\)No histopathological or clinical evidence of pancreatitis.

\(^f\)Meditations with broad action placed into this category.

\(^g\)Calculated as weight in kilograms divided by height in meters squared.

\(^h\)All data from confirmatory testing were converted to National Institute of Diabetes and Digestive and Kidney Disease units and defined as positive if 1 or more of the following applied: glucagonic acid decarboxylase 65 level of 20 or greater, insulinoma-associated 2c level of 5 or greater, zinc transporter 8 antigen level of 0.020 or greater, or insulin level of 0.016 \(\mu\text{U/mL}\) or greater.

\(^i\)When levels were reported below the lower limit of detection (ie, <0.05), a fill value of 0.05 was used; data missing from 1 donor in the group positive for a single autoantibody only.

\(^j\)Data are missing for 1 donor in the type 1 diabetes group.

\(^k\)For the 3 persons in the no diabetes group, the warm ischemia times were 8, 23, and 25 minutes; for the 1 person in the group positive for a single autoantibody only, the warm ischemia time was 11 minutes; and for the 1 person in the type 1 diabetes group, the warm ischemia time was 16 minutes. The remaining 46 were brain-dead donors with no elapsed time prior to organ retrieval.

\(^m\)Indicates the period from aortic cross-clamp to laboratory receipt. Data are missing for 2 persons in the group positive for a single autoantibody only and for 1 person in the type 1 diabetes group.

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**Figure.** Pancreas Weight of Organ Donors by Disease Status Using an Analysis of Covariance Model

The boxes represent the mid 50% of the data, the line within box represents the group mean value adjusted for age and body mass index. The high and low whiskers represent the 95th and 9th percentiles, respectively. The filled black circles represent outliers. Using the \(t\) test, the comparison between donors without diabetes and those positive for a single autoantibody only yielded a \(P\) value of .02; and for the comparison between donors without diabetes and those with type 1 diabetes a \(P\) value of less than .001. A comparison between the donors positive for a single autoantibody only and those with type 1 diabetes was not performed. Statistical significance was indicated at a Bonferroni-corrected nominal \(\alpha\) level of .025. Of note, although age and body mass index were poorly correlated with pancreas weight and failed to meet one of the assumptions of analysis of covariance, both were included into the final model because linear models that included age or body mass index by disease status group interaction terms showed that the interaction was not statistically significant.

ment with the United Network for Organ Sharing. Organ recovery, processing, autoantibody testing methods, histopathological review, and disease classification procedures have been described.\(^3\) Prior to organ recovery, initial screening for autoantibodies to glutamic acid decarboxylase 65 and insulinoma-associated 2c was performed. Following receipt of the pancreas, confirmatory testing was completed and also included autoantibodies to insulin and zinc transporter 8 antigens. T1D was confirmed by laboratory markers and medical chart review.

We included 23 controls without T1D, 8 T1D-free single islet autoantibody-positive donors, and 20 T1D cases. Cases were excluded with damaged pancreata (\(n = 17\)) missing organ weight (\(n = 26\)) or confirmatory autoantibody testing results (\(n = 10\)); other diseases or unresolved medical history (\(n = 34\)); unconfirmed (\(n = 1\)) or with multiple (\(n = 3\)) autoantibodies; pancreatitis (\(n = 20\)) or pancreatic endocrine tumor (\(n = 1\)); and donors aged 18 years or younger (\(n = 30\)).

The primary outcome variable was pancreas weight of donors in the 3 groups, examined using a general linear model to account for the influence of covariates on the end point (TABLE). Only age and body mass index (BMI) were retained in the final model. Other variables were not included due to multicollinearity, nonsignificance, or lack of previous evidence for an association.

All \(P\) values are unadjusted, 2-tailed, and statistically significant if less than .05, unless otherwise noted. Statistical analysis was performed using SAS software version 9.2 (SAS Institute Inc).
Results. Of the 51 donors, 46 were classified as brain dead and 5 as nonheart beating. Baseline characteristics were similar across groups, with the exception of all diabetes-related variables, reflecting criteria used to categorize the donors (Table). Most donors received cardiovascular drugs prior to death, but there was variation between the 3 disease status groups (Table).

Differences existed in the mean pancreas weight of individuals stratified by disease status and adjusted for age and BMI (Fig 1E). The mean weight of pancreata from those without diabetes (controls) was 81.4 g (95% CI, 73.0–89.8 g) compared with 61.3 g (95% CI, 46.8 g–75.8 g; P = .02) from the group positive for a single autoantibody only and 44.9 g (95% CI, 36.0 g–53.9 g; P < .001) from the T1D group.

Comment. In this study, the weight of pancreata in individuals without T1D, but with serum markers that potentially precede the clinical manifestations, as well as in individuals with T1D, was less than in controls. This suggests that early atrophy of the organ may be an important subclinical feature of T1D pathogenesis.

Limitations exist due to the small sample size and expected low incidence of subsequent T1D in single autoantibody-positive individuals. Additionally, functional assessment of pancreata was not possible, precluding evaluation of both the exocrine and endocrine compartments of the gland. In this data set, there is no evidence that cause of death or organ retrieval procedures affected organ weight.

Future studies should include validation and analysis of the potential mechanisms underlying this observation to understand whether early pancreatic atrophy contributes to the pathogenesis of T1D.

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Author Contributions: Dr Kaddis had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Study concept and design: Campbell-Thompson, Atkinson, Kaddis.

Acquisition of data: Campbell-Thompson, Wasserfall, Montgomery, Kaddis.

Analysis and interpretation of data: Campbell-Thompson, Atkinson, Kaddis.

Drafting of the manuscript: Atkinson, Kaddis.

Critical revision of the manuscript for important intellectual content: Campbell-Thompson, Wasserfall, Montgomery, Atkinson, Kaddis.

Statistical analysis: Kaddis.

Obtained funding: Campbell-Thompson, Atkinson.

Administrative, technical, or material support: Campbell-Thompson, Wasserfall, Montgomery, Atkinson.

Study supervision: Campbell-Thompson, Wasserfall, Atkinson.

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Disclaimer: The interpretation and reporting of organ donor data are the responsibilities of the authors and in no way should be seen as an official policy of or interpretation by the Organ Procurements and Transplantation Network or the US government.

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