A New Consensus Error Grid to Evaluate the Clinical Significance of Inaccuracies in the Measurement of Blood Glucose

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OBJECTIVE — The objectives of this study were *1*) to construct new error grids (EGs) for blood glucose (BG) self-monitoring by using the expertise of a large panel of clinicians and *2*) to use the new EGs to evaluate the accuracy of BG measurements made by patients.

RESEARCH DESIGN AND METHODS — To construct new EGs for type 1 and type 2 diabetic patients, a total of 100 experts of diabetes were asked to assign any error in BG measurement to 1 of 5 risk categories. We used these EGs to evaluate the accuracy of self-monitoring of blood glucose (SMBG) levels in 152 diabetic patients. The SMBG data were used to compare the new type 1 diabetes EG with a traditional EG.

RESULTS — Both the type 1 and type 2 diabetes EGs divide the risk plane into 8 concentric zones with no discontinuities. The new EGs are similar to each other, but they differ from the traditional EG in several significant ways. When used to evaluate a data set of measurements made by a sample of patients experienced in SMBG, the new type 1 diabetes EG rated 98.6% of their measurements as clinically acceptable, compared with 95% for the traditional EG.

CONCLUSIONS — The consensus EGs furnish a new tool for evaluating errors in the measurement of BG for patients with type 1 and type 2 diabetes.

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D iligent and timely control of blood glucose (BG) levels has recently emerged as a crucial element in diabetes therapy (1–4). This development places a renewed emphasis on the importance of accurate monitoring of BG levels and, particularly, on the accuracy of self-monitoring of blood glucose (SMBG) levels. Therefore, routine errors in SMBG may be an important determinant of the outcome of diabetes therapy, suggesting a need for the close scrutiny of this variable. However, the analysis of errors in SMBG presents a particularly troublesome problem, because the importance (i.e., the clinical consequence) of any particular error depends on the absolute value of

both the reference and measured values and not just on the percentage of deviation. Moreover, this dependence is not easily described by any simple mathematical relationship. Thus, standard simple statistical tools, such as linear regression analysis or correlation coefficients, are inadequate for the task of monitoring patients' SMBG errors or evaluating new SMBG methodologies. To address such concerns, a group of investigators from the University of Virginia (UVA) (5,6) introduced error grid (EG) analysis in the mid-1980s. EG analysis avoids dealing with statistical issues by assigning a specific level of clinical risk to any possible SMBG error. Each point on the grid (true BG, mea-

sured BG) is associated with 1 of 5 risk levels, as determined by the clinical judgment of the UVA group. The underlying assumptions made by Clarke and his UUA colleagues in order to establish the risk boundaries in their EG are the following: *1*) the target BG range is between 70 and 180 mg/dl, and patients will only attempt to correct values outside that range; *2*) corrective treatment by the patient is inappropriate if it results in BG levels outside the target range; and *3*) failure to treat BG <70 or >240 mg/dl is inappropriate (6).

These assumptions formed the basis of the 5 risk levels, which are labeled and defined as follows: *A*: SMBG <20% deviation from true BG or both SMBG and BG <70 mg/dl; *B*: deviation from true BG >20% but leads to no treatment or benign treatment; *C*: overcorrection of acceptable BG levels; *D*: dangerous failure to detect and treat BG errors; and *E*: erroneous treatment (i.e., treatment contradictory to that actually required).

EG analysis provides a clear representation of the clinical importance of SMBG errors and has gained widespread use (7–9). Nevertheless, the Clarke EG can be criticized on the basis of the placement of its risk boundaries (10,11), some of which skip risk categories. It was created 15 years ago by a small number of clinicians, and advances in the field since then justify a re-examination of the subject. In this article, we introduce a new EG based on a survey of 100 endocrinologists (the survey is available on the Becton Dickinson website at www.bdchc.com/). Our aim is to produce an updated EG based on the clinical judgment of a large number of experts. To this end, we have avoided, as much as possible, offering any of our own clinical opinions of the EG. The underlying assumptions are those of a consensus of the 100 experts. We retain the 5–risk level format of Clarke et al., but we have slightly altered the definitions of the risk levels so as to decouple them from the specific assumptions of the Clarke EG.

The second part of this article uses both the new EG and the Clarke EG to evaluate the accuracy of SMBG as it is actually practiced by patients. We studied a sample of

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152 patients who routinely monitor their own BG. The study was performed in a diabetes clinic with patients using their own meters. The distribution of errors in our sample is considered in light of both EGs and the latest recommendations of the American Diabetes Association (ADA).

RESEARCH DESIGN AND METHODS

Consensus EG

We surveyed 100 physicians at the 1994 ADA Annual Meeting to construct an unbiased tool to analyze the clinical significance of SMBG measurement errors. All of the respondents were clinicians who treated diabetic patients. Pursuant to constructing an EG in the fashion of Clarke et al. (6), each doctor was asked to assign any plausible error in BG measurement to 1 of 5 risk categories. The risk categories, in order of increasing severity, were defined as follows: *A*: no effect on clinical action; *B*: altered clinical action or little or no effect on clinical outcome; *C*: altered clinical action—likely to effect clinical outcome; *D*: altered clinical action—could have significant medical risk; and *E*: altered clinical action—could have dangerous consequences.

The above definitions were intended to correspond to the definitions of the risk zones in the Clarke EG while allowing the respondents maximal freedom to set their own boundaries. For example, zone *A* of the Clarke EG is defined as <20% deviation or having both reference and measured BGs <70 mg/dl. In addition, the UVA authors (6) stated that "values falling within this range are clinically accurate in that they would lead to clinically correct treatment decisions." Our definition of zone *A* asks each respondent to define his or her own range of "clinically accurate measurements," which is clarified as having "no effect on clinical action." Our zone *B* definition does not overlap our definition of zone *A* because it specifies "altered clinical action," whereas the Clarke zone *B* is defined as a region of no treatment or benign treatment. Thus, a data point that indicates no altered action is always an *A* in our EG but could be a *B* in the Clarke EG if it does not meet the numerical requirements for the Clarke zone *A*. The Clarke zones *C*, *D*, and *E* are defined as overcorrection, failure to correct, and anticorrection (intervention to move BG in the wrong direction), respectively. Our definitions for these categories are less mechanistic, allowing the respondent to assign risk based on his or her own expertise. To allow for the possibility that our collection of experts would regard SMBG errors differently when they were made by patients with type 1 versus type 2 diabetes, we asked them to complete separate questionnaires for type 1 and type 2 diabetes. Because the gravity of an error in SMBG may depend on the clinical status of a particular patient, we provided separate clinical descriptions of hypothetical patients with type 1 and type 2 diabetes and asked the experts to complete 2 separate surveys. The respondents were asked to base their responses on those particular patients. From the survey results for each physician, we constructed 100 individual EGs by plotting measured BG values from 0 to 550 mg/dl versus the true BG value in increments of 10 mg/dl (a computer program that is capable of classifying values into error categories and plotting them on an EG after users enter values into the program is available at www.bdchc.com/). Each point on the grid had an assigned risk category: *A*, *B*, *C*, *D*, or *E*. For example, the point 90,240 (true BG, measured BG) was assigned risk category *C* by most respondents, whereas 300,310 was always assigned *A*.

To make each consensus EG from the 100 sets of responses, the 5 risk categories were assigned the values 0, 1, 2, 3, and 4. The consensus risk for each point on the EG was taken as the arithmetic average of the 100 responses. Risk fields were initially defined as the set of points on the grid with values from 0–0.5, 0.5–1.5, 1.5–2.5, etc. This linear mapping of the risk categories onto the EG has the effect of weighting the categories equally, which we believe is reasonable, but we acknowledge that it is not obligatory. The EG produced directly by this technique (data not shown) did not have smooth boundaries separating the risk fields. Because this small-scale unevenness—which is largely due to the finite size of the sample, the 10 mg/dl bin size chosen for the analysis, and the preference of physicians for familiar glucose boundaries (i.e., 80, 240, and 400 mg/dl)—was deemed to be of no clinical relevance, we chose to eliminate it by using an iterative averaging algorithm. The algorithm used (triangular filter) generates, at each iteration, a filtered set of points (Y_{ij}'') from the preceding set (Y_{ij}) according to the following equations:

$$Y'_{ij} = 0.25(Y_{[i-1]j}) + 0.5(Y_{ij}) + 0.25(Y_{[i+1]j})$$
$$Y''_{ij} = 0.25(Y'_{i[j-1]}) + 0.5(Y'_{ij}) + 0.25(Y'_{i[j+1]})$$

The penultimate EG thus produced was smoothed by hand to eliminate any remaining serrations. The final EG retains the essential features of the original without the obtrusive boundary irregularities.

SMBG clinical trial

We studied 152 subjects with type 1 and type 2 diabetes at the Princeton Diabetes Treatment and Education Center (Princeton, NJ) who regularly monitored their BG at home (Table 1). The study design was approved by an institutional review board, and all of the subjects gave informed consent. Most of the subjects ($n = 110$) used the One Touch II meter. The other meters represented in the sample were from Roche ($n = 17$), Abbott ($n = 11$), and Bayer ($n = 14$). Each of the 152 subjects participated on 2 separate occasions, thus generating 304 data points. For each visit to the clinic, the subject was instructed to abstain from eating for at least 2 h before arrival and to bring his or her own BG meter. The subject was then taken to a private room and observed (but not influenced) while personally obtaining a capillary sample of blood with his or her own lancet and used his or her own meter to take a BG measurement. The subject then went to another room, where a venous blood sample was collected into a #6510 serum separator tubes (SST) Vacutainer tube for glucose assay via the hexokinase method.

Within 30–60 min of collection, the blood was centrifuged at 1,000–1,300*g* for 10 min to separate plasma from cells to prevent the metabolism of BG. Samples were punctually sent to a licensed clinical laboratory to assure analysis within 5 h of collection. The glucose value obtained by the hexokinase assay was adjusted to account for the 11% increment of glucose concentration in plasma versus whole blood and thus compensate for the low aqueous content of the erythrocyte (4). Because the subjects had fasted for >2 h, the differences in the glucose level of capillary versus venous blood should have been small (<2–5 mg/dl) and were therefore ignored.

RESULTS — The EGs in Fig. 1, which are pertinent to patients with type 1 (Fig. 1*A*) or type 2 (Fig. 1*B*) diabetes, were based on the separate type 1 and type 2 diabetes surveys taken by the 100 endocrinologists. As shown in Fig. 1, the boundaries delineating the risk areas have been smoothed, as described in RESEARCH DESIGN AND METHODS. The smoothing process did not add or

Table 1—*Characteristics of study subjects*

n	152
Type 1 diabetic patients	60
Type 2 diabetic patients	92
Sex (M/F)	97/55
Diabetes duration (years)	16.4 ± 12.1
Age at time of study	37.8 ± 15.6
BMI (kg/m^2)	28.2 ± 5.2
Type of therapy	
Diet and exercise	7
Oral agent	35
Oral agent + insulin	20
Insulin alone	90
Observations of the condition of the subjects' BG meters*	
No obvious faults	148
Low battery	3
Broken meter	1
Observations of subjects' SMBG technique	
No obvious problem	151
Insufficient blood sample	1

Data are *n* or means ± SD. *All of the study subjects claimed to have a BG meter that was in good working order.

Figure 1—*EGs for type 1 diabetes* (A) *and type 2 diabetes* (B). *The EGs were constructed from a survey of diabetes specialists. The horizontal axis represents true BG values, and the vertical axis represents BG values measured by patients. The grid is divided into zones signifying the degree of risk posed by the incorrect measurement: zone* A *represents no effect on clinical action; zone* B *represents altered clinical action—little or no effect on clinical outcome; zone* C *represents altered clinical action—likely to affect clinical outcome; zone* D *represents altered clinical action—could have significant medical risk; and zone* E *represents altered clinical action—could have dangerous consequences.*

remove any region of risk. In both the final EGs (Fig. 1) and in the raw data (not shown), there are no discontinuities in risk at the boundaries (i.e., all boundaries separate regions that are adjacent in both proximity on the graph and severity of risk). For example, risk region *B* may border *A* and *C* but not *D* or *E*. This is a natural result of the survey, and it stands in contrast with the Clarke EG, which has several boundaries separating nonadjacent risk categories.

The new type 1 and type 2 diabetes EGs are similar but not identical to one another. The *A* zone of the type 1 diabetes EG is narrower than that of the type 2 diabetes EG in the middle BG ranges, but the difference is compensated by the *B* zone. Consequently, the *B/C* and *C/D* boundaries are almost identical in the 2 EGs, except at very low true BG values. At the true BG value of <30 mg/dl, where the upper zone boundaries are horizontal, the *C/D* boundary of the type 1 diabetes EG is 20 mg/dl higher than its type 2 diabetes EG equivalent. In contrast, the *D/E* boundary in the type 1 diabetes EG is 50 mg/dl lower, beginning at the measured BG value of 150 mg/dl, compared with 200 mg/dl in the type 2 diabetes EG.

In Fig. 2, the SMBG data from the clinical trial is plotted on both the Clarke EG (Fig. 2*A*) and the new EG for type 1 diabetes (Fig. 2*B*). We use the type 1 diabetes EG for comparison with that of Clarke EG, because, overall, the type 1 diabetes EG judges errors in SMBG somewhat more strictly than the type 2 diabetes EG, reflecting the potential for more serious consequences of BG measurement errors in type 1 versus type 2 diabetic individuals, because patients with type 1 diabetes generally have more severely impaired blood glucose regulation. Several differences
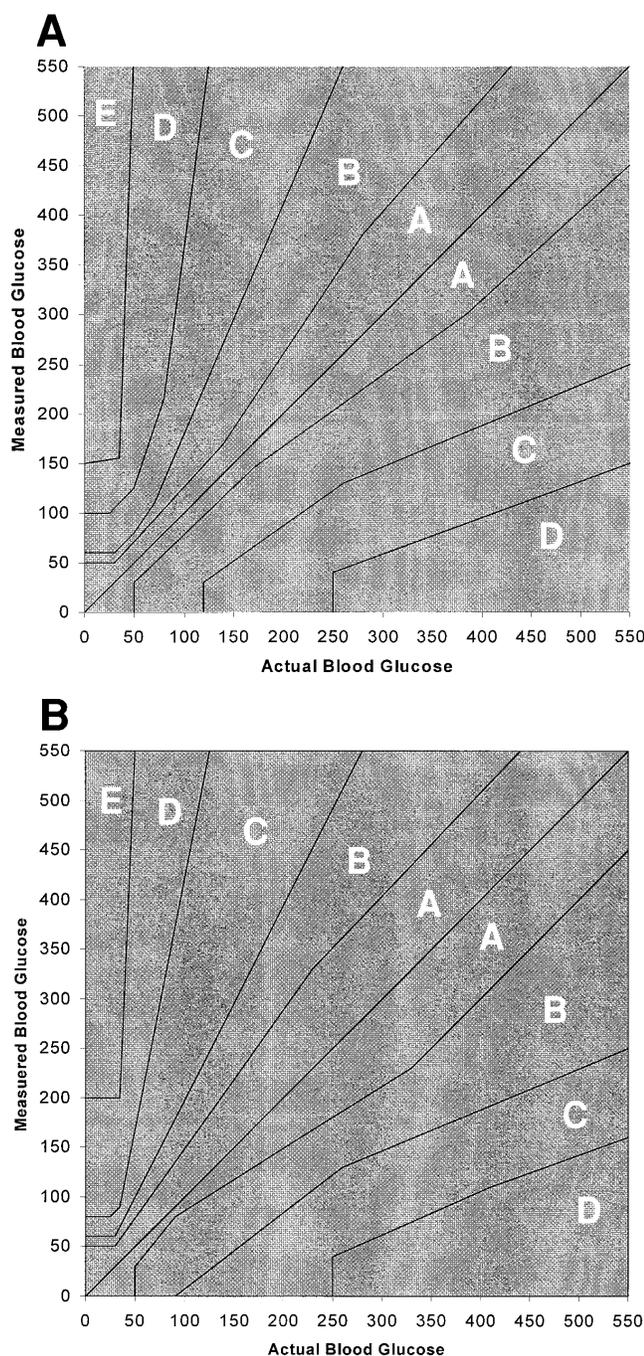
A

B

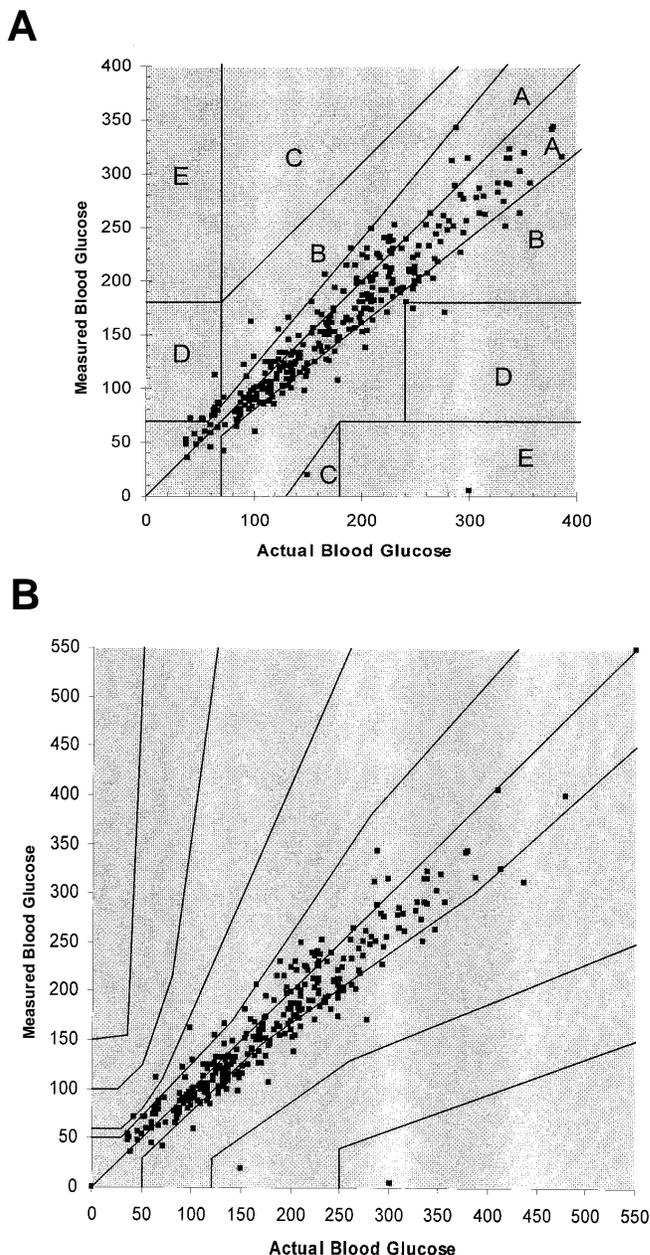**Figure 2**—*The SMBG data from the clinical trial superimposed on the EG of Clarke et al. (*A*) and on the type 1 diabetes EG (*B*).*

CONCLUSIONS — As long ago as 1987, the ADA stipulated that the accuracy of SMBG should be sufficient to exclude errors of >15% (12). Since then, it has become apparent that tight control of BG levels is a crucial parameter in the management of diabetes. It has been shown, for example, that an intense treatment regime, compared with conventional treatment, can reduce the risk of retinopathy, neuropathy, and nephropathy by 50–70% for type 1 diabetic patients (1). Similar benefits have been demonstrated for type 2 diabetes (2,3,13). These results suggest that normalization of BG levels may effectively eliminate or greatly reduce these dire complications of diabetes. Thus, the ADA now recommends at least 3 or 4 SMBGs per day for type 1 diabetic patients (2). Also, in 1987, the ADA proposed a goal for SMBG: total error (system plus user error) should be <10% for all SMBG systems for BG values between 30 and 400 mg/dl (12). Our data indicate that this seemingly modest goal remains elusive.

How important are these shortcomings in the measurement of BG values to the treatment of diabetes? The ADA guideline offers one criterion, but it does not recognize that all BG measurement errors of a given percentage do not have equal clinical significance. A more nuanced evaluation of BG measurement errors was introduced by Clarke et al. (5,6), who proposed a 2-dimensional EG, which is essentially a lookup table in which any error in BG measurement is assigned to a risk category based on a 5-point scale. This is a more elegant approach, but the actual EG produced seemed flawed to us. The risk categories were assigned using the clinical judgment of a small number of experts, and even though we are not critical of their choices per se, it seems reasonable to expand and update the database to include a larger number of clinicians. More disturbingly, the EG of Clarke et al. has several strange discontinuities (i.e., boundaries where an infinitesimal change in BG causes the risk category to skip a level).

For example, a measurement of 200 mg/dl is judged to be a *C* (unnecessary correction of acceptable glucose) if the true BG value is 71 mg/dl, but it is considered an *E* (dangerous contrary treatment) if the true BG value is 69 mg/dl, skipping over the category *D* (dangerous failure to treat). These and other weaknesses have been discussed in the literature (10,11). To address such concerns, we have assembled a new EG based on a survey of diabetes specialists.

between the data analysis provided by the 2 EGs are evident by inspection. For example, the points falling into both the upper and lower *D* zones of the Clarke EG are all found in lower risk zones in the new EG. To assess whether such differences are a result of the nonequivalence of the 2 EGs, we performed a permutation test based on the contingency table describing the cross-classification of the data (Table 2). This test is a method for determining whether or not differences in classification are random errors or, alternatively, whether or not there is a greater proclivity for one grid to rate individuals into higher (more severe) categories. The test yielded a *P* value of <0.0001. Thus, we reject the null hypothesis that the likelihood of disagreement in classification is not dependent on direction (i.e., that a given data point is as likely to go up a risk level as down when analyzed by the other EG). Therefore, we conclude that the new EG and that of Clarke et al. are not equivalent.

The risk zones in the new EG fan out from the 45° line in a relatively simple pattern of increasing risk as one moves radially from the line of identity. This pattern contains no discontinuities in risk category, which is a gratifying result. It must be noted, however, that because the triangular filter used to smooth the risk boundaries could not eliminate all traces of small-scale jaggedness without compromising the data, it was necessary to perform the final boundary smoothing by hand. Furthermore, because there is some choice in how to implement the initial averaging process, there is, per force, some flexibility in how to place the risk boundaries. In particular, we arbitrarily assigned the values 0, 1, 2, 3, and 4 to the risk categories *A*, *B*, *C*, *D*, and *E*. This is a reasonable approach, but other weighings may also be reasonable and would of course produce different EGs. Finally, it should be noted that the surveys (type 1 and 2 diabetes) were based on specific descriptions of hypothetical patients, and the resulting EGs may not be insensitive to those descriptions. Thus, our EG, like that of Clarke et al., cannot be said to be completely free of arbitrariness and is not promoted here as the final word in evaluating BG measurement errors.

Several features of the new EG differ qualitatively from the EG of Clarke et al. The 9 geometric risk zones of Clarke et al.'s EG are replaced with 8 irregular risk zones in the new one. The rectangular *E* zone occupying the lower right-hand corner of Clarke et al.'s EG (high true glucose, low measured glucose) is absent in the new EG. This difference may be a result of our definition of zone *E* as containing measurements that "could have dangerous consequences" rather than the UVA group's definition of contrary treatment. Evidently, our respondents did not feel that the clinical actions implied here rose to the level of "dangerous," although Cox et al. (5) refer to their zones *C*, *D*, and *E* as "potentially dangerous." Elsewhere on the new EG, the intermediate risk categories of Clarke et al. (*C* and *D*) are transformed to form concentric contours radiating from the 45° line. The upper *B* and *C* zones in the new EG extend into the low BG range and separate the upper *D* zone from the *A* zone. The lower *C* zone in the new EG is vastly enlarged compared with Clarke et al.'s EG, so that it separates the displaced lower *D* zone from the *B* zone. The low tolerance for error centered around 70 mg/dl is retained, but the details of the 2 EGs differ substantially in this region. For example, the

Table 2—*Degree to which the type 1 diabetes EG and the Clarke EG agree and disagree about classifying measurements*

	Risk zones of type 1 diabetes EG					The Clarke EG totals
	A	*B*	*C*	*D*	*E*	
Risk zones of Clarke et al.'s EG						
A	226*	23	0	0	0	24
B	4	36	0	0	0	40
C	0	0	1	0	0	1
D	9	2	2	0	0	13
E	0	0	0	1	0	1
Type 1 diabetes EG totals	239	61	3	1	—	

Data are *n*. *For example, there were 226 measurements classified as *A* by both EGs. Yet, there were 23 measurements classified as *A* by the EG of Clarke et al. but as *B* by the type 1 diabetes. Moreover, there were only 4 measurements classified as *B* by the Clarke EG but as *A* by the type 1 diabetes.

square of the *A* zone in the Clarke et al. EG, which extends out to 70 mg/dl, extends only to 50 mg/dl in the new EG. This indicates that the 100 clinicians surveyed felt that at an actual blood glucose level of 60 mg/dl, there were therapeutic differences between a reading of 10 and 60 mg/dl.

Our study of 152 patients who were experienced in SMBG served as a basis to compare the different methods of error analysis discussed above. Only 45% of the patients' measurements were within 10% of the hexokinase value, and only 63% were within 15%; these figures fall far short of the ADA benchmarks. In addition, these results are in accord with numerous studies that report the failure of patients to achieve the ADA goal for SMBG accuracy (14–17). Nevertheless, in both the new EG and that of Clarke et al., most of the measurements fall into zones *A* and *B*, that is, they are errors that would have no effect on clinical outcomes. Overall, the new EG is significantly more forgiving toward this set of errors than the EG of Clarke et al.: 98.6% of the measurements are *A*'s or *B*'s on the new EG versus 95% on the Clarke et al. EG. Most of the difference can be traced to the modification of the abrupt *A*/*B* and *B*/*D* boundaries on the Clarke et al. EG. The new EG is more exacting than that of Clarke et al. when both true and measured BG levels are <70 mg/dl; however, very little of this data set was affected by this fact. Some of the more errant data points serve to illustrate the difference between the 2 EGs. For example, the point Hex 65, SMBG 112 is regarded as a serious *D* by the Clarke EG, whereas in the new EG, it falls into the part of the *C* zone absent from the Clarke EG that is interposed between *D* and *B* near 70 mg/dl. The egregious Hex 305,

SMBG 5 is rated an *E* by the Clarke EG but only a (still serious) *D* by our panel, because the new EG eliminates the entire high glucose *E* zone. The point Hex 241, SMBG 181 is very near one of the Clarke *D*/*B* borders, but is an unambiguous *B* in the present EG. (We make no judgment favoring any one of these interpretations over any other. They are noted only to illustrate situations in which the EGs differ.)

The new EG was created to reflect the opinion of a large number of clinical diabetes experts. The consensus EG lacks the discontinuities of Clarke et al.'s EG but, otherwise, it gives similar results. Overall, the new EG was more tolerant of SMBG errors made by our test group than the EG of Clarke et al. These consensus EGs provide a new tool to aid in evaluating the quickly evolving technology of BG measurement.

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