Predictive Concepts: A Study in Gingival Inflammation and Carbohydrate Metabolism

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Introduction

For a long time, it has been conceded in clinical medicine and its subspecialities including dentistry, that seemingly similar people treated in a seemingly similar manner by seemingly similar practitioners often respond quite differently. The same headache pill eliminates the problem in one, exerts no effect in a second, and actually worsens the cephalalgia in the third person. Some people, for example, show less inflamed gingiva after the cleaning and polishing of their teeth. Others seem unchanged; and some few actually worsen.

The usual *explanation* is that these seemingly similar persons are, in reality, *different.* And this difference is usually ascribed to a lack of cooperation in taking the medicine or brushing the teeth (to pick but two of many so-called *explanations).* But, on occasion, we are willing to grant that the real difference may be one of "genes" or *constitution,* or *tissue tolerance,* or *predisposition,* or, as it is most commonly phrased, a difference in *resistance* and/or *susceptibility.*

If indeed seemingly similar subjects treated in seemingly similar ways by seemingly similar therapists and techniques respond differently because they are, in fact, *constitutionally* different, then it behooves us to take a hard look at this difference, its identification, and its quantification.

A propos, traditional medicine for a long time, by word more than by act, has contended that this so-called difference may, in some still-to-be-explained way, be related to the phenomenon termed *homeostasis,* a term coined by a nineteenth century French physician, Claude Bernard. He made the point, "illnesses hover constantly above us, their seeds blown by the wind, but they do not set in the terrain unless the terrain is ready to receive them."[1] And so, it was Bernard who invented the term *homeostasis* to describe the efforts of the organism to maintain a stable internal environment (a suitable terrain) meaning, for example, a steady internal atmosphere of precise and optimal temperature, circulation, movement of energy sources, elimination of toxic wastes, etc.

Hence, the logical notion prevails that so-called healthy people are *in* homeostasis; unhealthy people are not. And it therefore follows, for some students of the subject, that making people better is really a question of making them more homeostatic.

Is it possible to test this hypothesis by means of simple experimental methods which would allow models for verification with other demonstrations by other investigators?

Method of Investigation

For purposes of this discussion, three published reports will be utilized.[2"4] In all of the three demonstrations, gingival inflammation was graded by one of the commonly available scoring systems on a four-point scale (and a mean gingival score derived) at the beginning and end of the experimental period. Parenthetically, the same examiner performed all of the initial and final scoring with no knowledge of what transpired during the experimental period. Also, at both visits, nonfasting blood glucose (two-hour postprandial) was assessed by the Auto-Analyzer method.

In the first phase (Experiment "A"),[2] 45 presumably healthy male individuals were employed. At each of the two visits, separated by two weeks, the marginal gingivae and interdental papillae were graded in the upper and lower incisor areas. Thirty-two measures were obtained from each subject, and 45 subjects times 32 measurements yielded a total of 1440 values. At the initial visit, one-half of the teeth were scaled and polished. (The other side served as a control). The decision to treat the right or left side was made on a random basis. At this same first visit, the nonfasting blood glucose was also

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measured. Two weeks later, the clinical (gingival) and biochemical (blood glucose) values were again derived. *This process allowed a study of a clinical (gingival) change by means of altering (improving) the oral environment.*

The participants for the second phase of the program (Experiment "B") included children requiring orthodontic therapy[3]. Gingival inflammation surrounding the mandibular right and left lateral incisors were graded, as already described, on a four-point scale. Also, at the same visit, the nonfasting blood glucose was measured. On a random basis, the mandibular right or left lateral incisor was orthodontically banded. At the follow-up visit, one month later, the gingivae and blood glucose were recorded. *This experiment permitted a study of clinical (gingival) change by means of altering (potentially worsening) the oral environment.*

The third and last (Experiment "C") included 40 junior (third-year) dental students who, following the gingival grading and measurement of blood glucose were instructed to eliminate, as far as possible, refined carbohydrates from the diet.[4] The recommendation was also made that protein intake be increased. Four days later (Monday to Friday), gingival state and nonfasting blood glucose were remeasured. *This experiment allowed a study of clinical (gingival) change by ignoring the oral environment and possibly changing the homeostatic systems.*

It should be underscored that, by these experimental designs, it became possible to examine the gingival response to improving the oral environment by tooth cleansing alone (Experiment "A"), by worsening the oral environment alone (Experiment "B") by means of orthodontic banding, and by ignoring the oral milieu and simply changing the host system by dietary means (Experiment "C").

Results

In Experiment "A"[2] (the scaling and polishing study) the initial and final mean gingival scores in the unsealed side were unchanged with no statistically significant difference of the means ($P = 0.500$). This suggests, for one, the relative reliability of the measuring technique. In other words, when nothing was done, nothing resulted. However, in the scaled area, three points were noted. First, there was an overall 33% improvement in the gingival state. Secondly, the initial and final blood glucose values were statistically not significant ($P > 0.500$). Finally, while there was an overall reduction in gingival inflammation, not all subjects behaved the same. Specifically, 35 of the 45 (78%) improved; 6 (13%) remained unchanged; 4 (9%) actually worsened.

In Experiment "B", (the orthodontic study),[3] the initial and final gingival scores in the nonbanded side were not statistically significant. This underscores the validity of the scoring system. Clearly, when nothing was done, nothing resulted. However, in the banded area, three points warrant special mention. First, there was an overall 31% statistically significant ($P < 0.001$) worsening in the gingival state. Secondly, the initial and final blood glucose values were not statistically significantly different ($P > 0.100$). Finally, while there was an overall worsening in gingival state, not all of the subjects responded in the same manner. Actually, 27 (39%) worsened, 42 (61%) remained unchanged or improved.

Finally, in Experiment "C", with only dietary alteration, the 40 dental students demonstrated an overall statistically significant improvement ($P < 0.001$) in gingival inflammation of a magnitude of 35%.[4] Secondly, the blood glucuse values did not change significantly. Finally, not all subjects responded in the same way. Actually, 33 (83%) improved; 5 (12%) remained unchanged and 2 (5%) worsened.

Discussion

Three interdependent items emerge from these three experiments. First, it is possible to demonstrate effective clinical change following local therapy by improving the mouth with scaling and by worsening the oral cavity with orthodontic banding irrespective of any host considerations. Conversely, it is equally possible to improve gingival state by changing host conditions (with dietary means) and without altering any of the oral factors. Secondly, in all three instances, notwithstanding the overall direction of change, the individuals showed considerable variability; some worsened, some did not change at all, and some improved. Finally, within the limits of the study, the variability could not be explained by changes in nonfasting blood glucose as one measure of the homeostatic

profile.

However, a more detailed analysis of the postprandial blood glucose values in Experiment "A" shows that the scores ranged from a low of 55 mg% to a high of 120 mg%. If one grants (as most clinicians will) that approximately 60 to 100 mg% is the physiologic range, then only one subject is marginally hyperglycemic (120 mg%) at the initial visit, one is hypoglycemic (55 mg%) and three are hyperglycemic (102, 115, and 120 mg%) at the final visit.

Figure 1 (the outside square) pictorializes the initial blood glucose levels (on the abscissa) and the final scores (on the ordinate). It will be noted that only 4 of the 45 subjects are plotted outside the so-called normal range (60-100 mg%). On the assumption that within-the-square connotes homeostasis, then it can be hypothesized that the 41 subjects within the box are healthier (more homeostatic) than the 4 outside the square. It might be further theorized that (1) the gingival state of those inside the box would be better (lower) initially and finally. You will note (Table 1) that this is indeed the case (line 1 and 2). The initial gingival score inside is lower (0.6 versus 0.8) and the final is also lower (0.4 versus 0.5).

Referring again to Figure 1, there is a slightly smaller square which characterizes a physiologic range of 65 to 95 mg%. If indeed our hypothesis prevails, then all that was mentioned about the larger (60-100 mg%) rectangle should obtain. In fact, it does. The initial and final gingival (lines 3 and 4) scores in the more homeostatic zone are lower (0.8 versus 0.5 and 0.5 versus 0.3).

Figure 1. A pictorial analysis of the blood glucose levels before and after the cleaning and polishing of the teeth. Progressively restricted limits of normality for nonfasting glucose are shown by the progressively smaller rectangles. (Experiment "A")

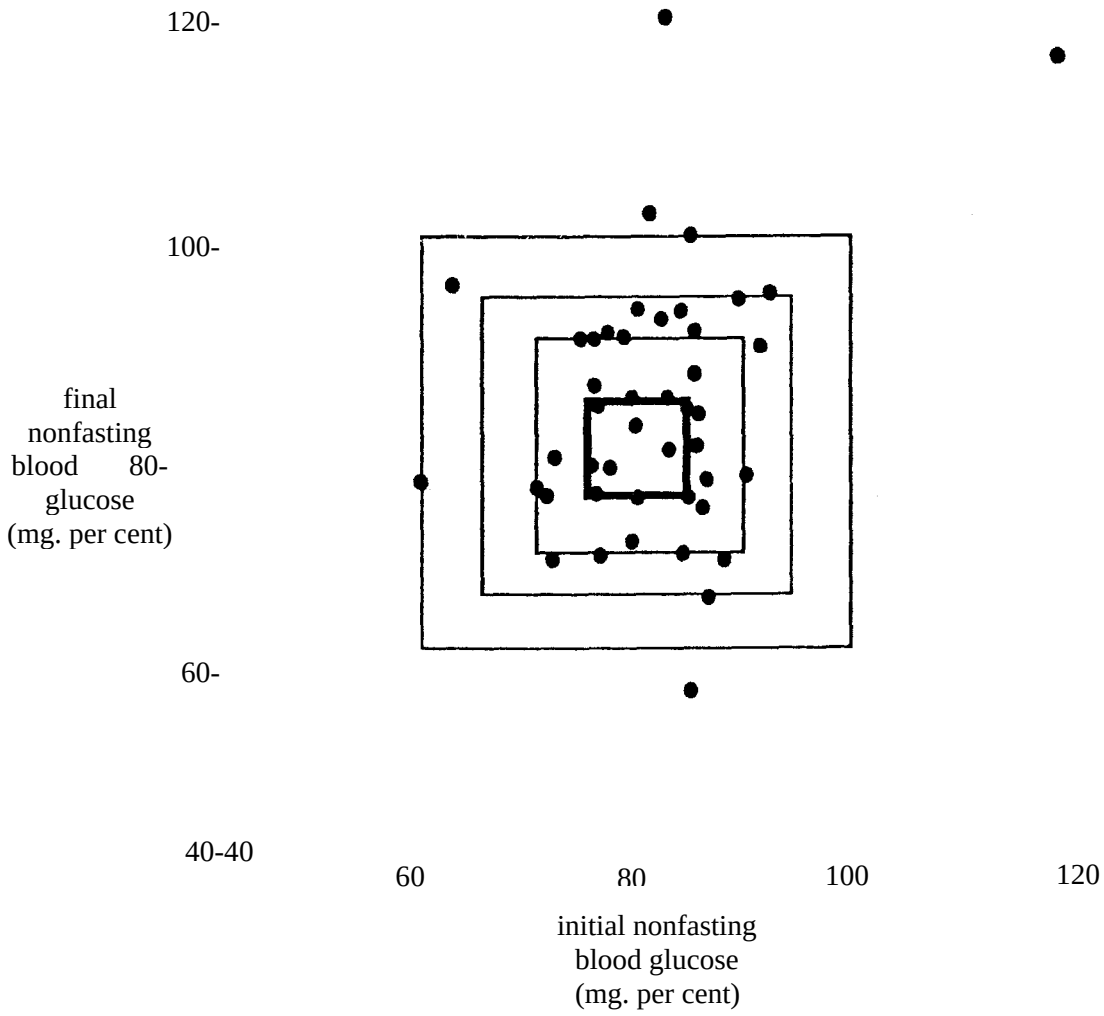


Table 1
pre-and postscalling scores (Experiment "A")

line	blood glucose range	sample size	mean gingival scores initial	final
1	**<60>** 100	**4**	**0.6**	**0.4**
2	60-100	**41**	**0.8**	**0.5**
3	**<65> 95**	**8**	**0.5**	**0.3**
4	**65— 95**	**37**	**0.7**	**0.5**
5	**<70> 90**	**15**	**0.5**	**0.3**
6	**70— 90**	30	0.6	**0.4**
7	**<75> 85**	**32**	**0.5**	**0.2**
8	**75- 85**	**13**		
0.8	**0.5**			

Table 2
pre-and post dietary scores (Experiment "C")

line	**blood glucose range**	**sample size**	**mean gingival initial**	**scores final**
1	< 60 >100 60-100	**8**	0.69	0.44 0.36
2		**32**	0.56	
3 4	< 65 > 95 65— 95	**14 26**	0.63 0.56	0.36 0.39
5 6	< 70 > 90 70—90	**25 15**	0.63 0.51	0.40 0.35
7	<75> 85 75— 85	**36 4**	0.60	0.39 0.31
8			0.47	_
— ... —				

As a matter of fact, as one shrinks the so-called normal or physiologic range, the clinical pattern just described sharpens. Hence, the healthiest gingiva (the lowest score) is represented after scaling (line 8) in the very smallest square (0.2).

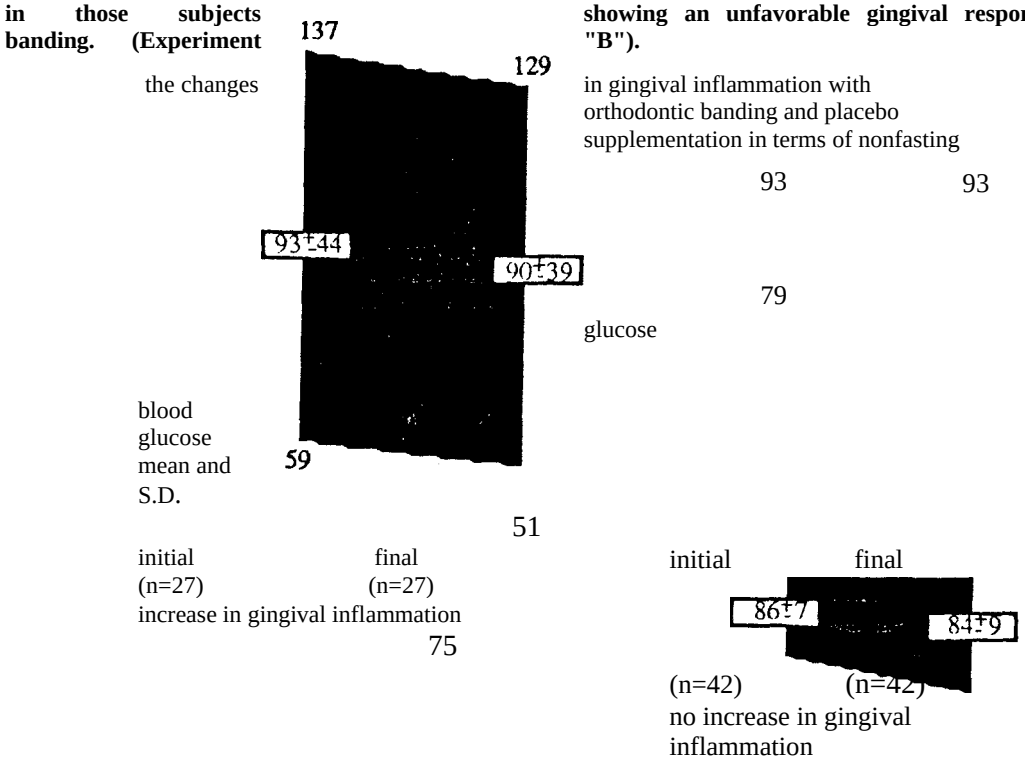
It would, therefore, seem that carbohydrate metabolism may well indeed be one reflector of host resistance/susceptibility. Also, it would appear that carbohydrate

metabolism as a barometer of host state gains importance when the physiologic parameters are progressively restricted (more homeostatic). Finally, it would appear that herein lay part of the explanation for the different responses previously described in gingival condition following the seemingly similar cleaning and polishing of the teeth in seemingly similar subjects by seemingly similar therapists.

One can demonstrate confirmation of this hypothesis by utilizing other methodology. Figure 2 (Experiment "B") depicts the initial and final blood glucose values for the group of 27 subjects in whom the gingivae worsened versus the 42 who responded with no change or improvement. Following orthodontic banding, the most cursory inspection (the area of shading) suggests that these two groups are distinctly different. First, the initial and final scores in the group that worsened are similar (93 ± 44 versus 90 ± 39 mg%) and this also obtains in those who did not (86 ± 7 versus 84 ± 9) suggesting that blood glucose did not vary significantly (P> 0.100) during the the one month experiment. However secondly, while the initial values did not differ significantly on a mean basis (93 versus 86 mg%), the spread of the values, the variance was significant (44 versus 7 mg%). This was essentially the pattern at the end of the experiment (39 versus 9 mg%). This suggests that the blood glucose scores varied (44/7) significantly (P< 0.0005) about sixfold more in those who fared more poorly.

Hence, we have here an opportunity to observe that the homeostatic profile of those children who responded unfavorably to orthodontic bands is markedly different than those who were able to tolerate these unnatural foreign bodies. And so, once again, utilizing different methodology, these data underscore the point that variability of therapeutic response may be related to blood glucose levels. The subjects who showed no worsening following orthodontic bands, demonstrating good tissue tolerance (better homeostasis) showed blood glucose values closely grouped about the mean. On the other hand, those who worsened following band placement showed blood glucose values widely dispersed around the mean both

Figure 2. The changes in gingival state with orthodontic banding and placebo supplementation as it relates to nonfasting blood glucose. Note that the initial and final values for each group are very similar. There is a statistically significant variance between the group which worsened versus the group which did not suggesting greater dysglycemia in those subjects showing an unfavorable gingival response to orthodontic banding. (Experiment "B").



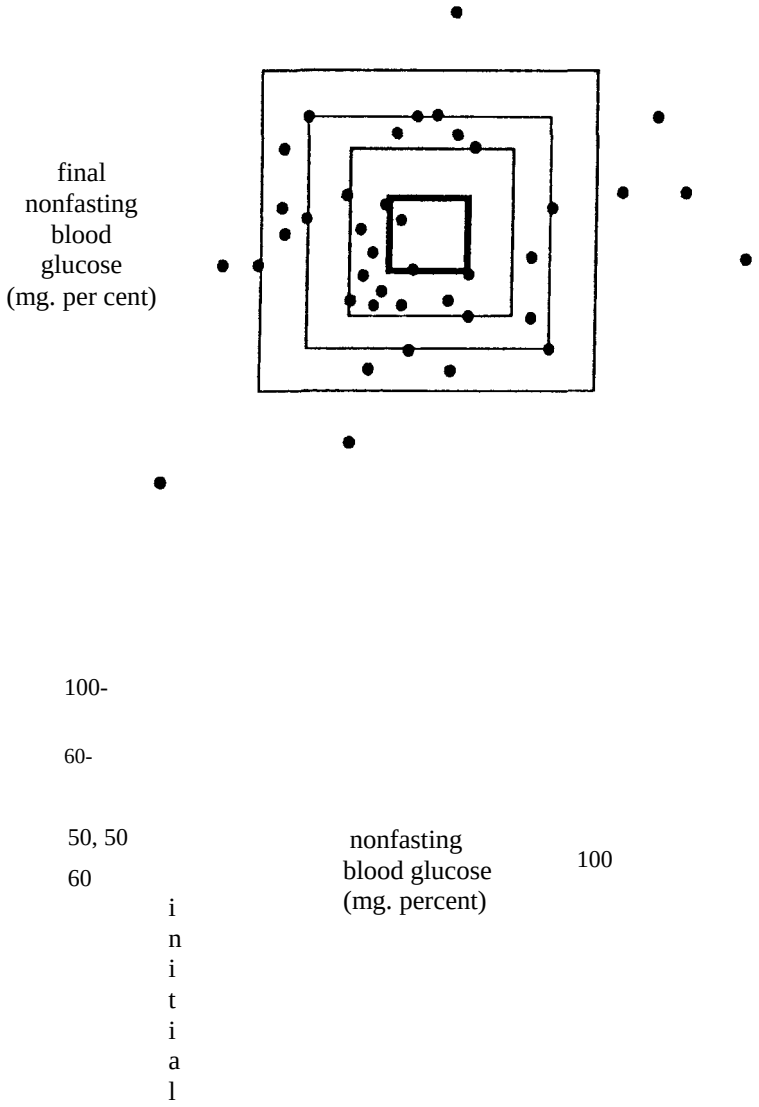


Figure 3. A pictorial analysis of the blood glucose levels before and after a reduction in refined carbohydrates food-stuffs. Progressively restricted limits of normality for nonfasting blood glucose are shown by the progressively smaller rectangles. (Experiment "C")

before and after the experimental period. Here is a different demonstration (Experiment "B") underlining the possibility of discriminating why seemingly similar patients, treated seemingly similar by seemingly similar therapists, often do not respond the same.

Finally, Experiment "C" exemplifies the same principle by altering only the host state. Returning to the earlier model, Figure 3 pictorially portrays the blood glucose scores for the 40 dental students initially on Monday (on the horizontal axis) and on the ordinate on Friday of the same week. Eight of the subjects (Table 2) were outside (presumably not in homeostasis); 32 inside. It is abundantly clear that in all instances, the scores within are lower than outside initially and finally. And, most importantly, the gingival score is lowest and best (0.31) at the end of the week in those individuals with the most homeostatic blood glucose range (line 8).

It would appear, within the limits of this study, that those subjects who demonstrated gingival improvement (regardless of the nature of the therapy) seemed to be those with the most optimal homeostasis (as measured by nonfasting blood glucose); those who worsened or remained unchanged were characterized by less homeostasis.

Because of the newness of this approach, it would be highly desirable to check out other clinical parameters (e.g. periodontal pocket depth, blood pressure) and other biochemical measures (e.g. serum cholesterol, serum

creatinine). Accordingly, reports to follow will examine periodontal pockets (in contrast to gingival inflammation) versus nonfasting blood glucose[5] and both gingival inflammation and periodontal pocket depth in the light of serum cholesterol[6-7] (instead of blood glucose as in this report).

Summary and Conclusions

The scientific community has long been struggling with a definition of *wellness.* Even the renowned World Health Organizations (WHO) admits to difficulty with an adequate description. Within the limits of this preliminary study, it would appear that wellness connotes and may well be synonymous with homeostasis (in this particular case a blood glucose steady state). Conversely, sickness is represented by a lack of homeostasis (as judged by blood glucose).

Reports to follow will continue to examine this hypothesis by analyzing other and different clinical and biochemical indices.

References

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