BEGINNER'S MIND: TRYING TO LEARN SOMETHING ABOUT OBESITY

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An annual meeting is an occasion in our busy lives for a moment of reflection—a time to consider what has been passed on to us from our predecessors and what we will pass on to those who follow us.[1] For me, it is a time to remember my teachers, and one in particular—Shunryu Suzuki.

Shunryu Suzuki was a slight, self-effacing Japanese man with a beautiful smile. He lived in San Francisco for 13 years, where he established a Zen Buddhist community. He did it without seeming to do anything—just by being a gently smiling person who made people feel good about themselves and want to do better than they had ever done.

Suzuki-roshi used to talk about "beginner's mind," an idea that expressed his teaching. What is "beginner's mind?" Suzuki rarely mentioned it in his only book, *Zen Mind, Beginner's Mind*, (1) and he did not define it.

Here is what he wrote about it:

> . . . beginner's mind, a mind free from possessing anything, a mind that knows that everything is flowing change. Nothing exists but momentarily in its present form and color. One thing flows into another and cannot be grasped . . . .
>
> In the beginner's mind there is no thought "I have attained something . . ." When we have no thought of achievement, no thought of self, we are true beginners. Then we can really learn something. . . .
>
> When we forget ourselves, we actually are the true activity . . . or reality itself. When we realize this fact, there is no problem whatsoever in the world and we can enjoy our life without any difficulties. The purpose of our practice is to be aware of this fact.

The "practice" that Suzuki-roshi was talking about was meditation, Zen meditation, and if he was an example of the fruits of that practice, it is a wonderful practice indeed. But there are other kinds of practice. Whatever we do with all our might, and a full heart, becomes our practice. So all of us have a practice in the sense that Suzuki-roshi meant it. Today I would like to talk to you about the practice of studying obesity, of times when it expressed "beginner's mind," moments of what might be called "grace."

Just before I began the study of obesity, there was such a moment. At the time I was fresh from years of psychoanalytic training, unsuccessful psychoanalytic training. As you know, psychoanalytic training involves the powerful influence of a personal analysis combined with intensive indoctrination in psychoanalytic theory. It is a redoubtable apparatus of thought control and it certainly controlled my thought. For some people the relief of symptoms and the enhanced morale that occur in the course of psychoanalysis are enough to reconcile them to the accompanying intellectual straitjacket. Unfortunately, or perhaps fortunately as things turned out, I was not one of those whose symptoms were relieved or whose morale was enhanced by psychoanalysis. I felt terrible—confused and terrible.

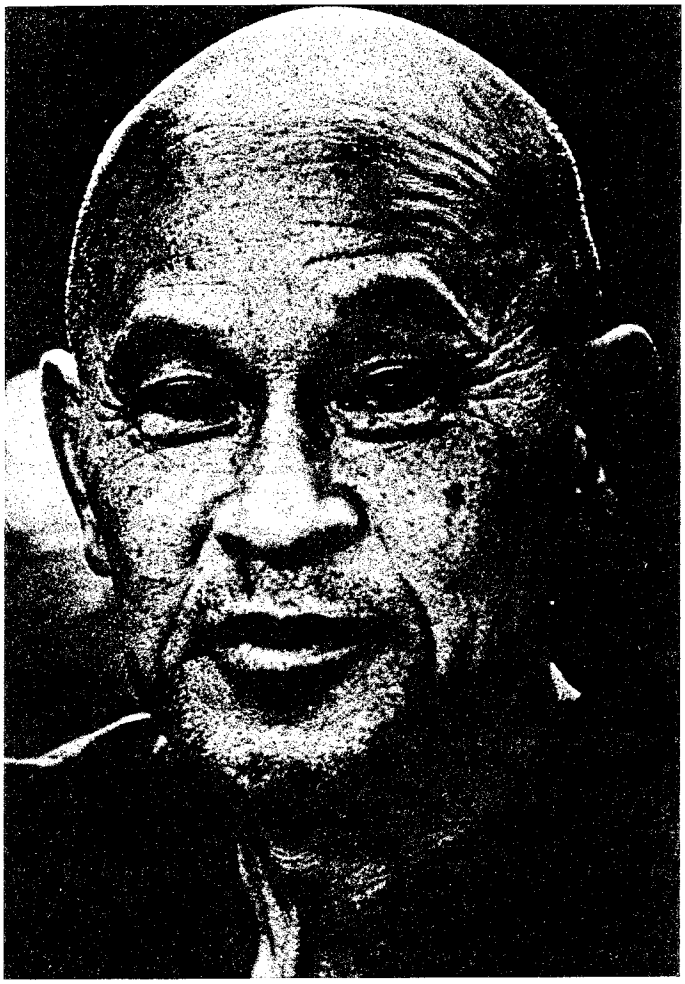

FIGURE 1: Shunryu Suzuki 1905-1971.

(Boni RS: *S Suzuki.* In Suzuki S, *Zen Mind, Beginner's Mind.* Copyright 1988, John Weatherhill, Inc. Reprinted by permission.)

So, I stopped trying to become a psychoanalyst and went to work in the psychosomatic medicine program of Harold Wolff at the New York Hospital. Yet I hadn't abandoned some of the things that I had learned so well from my analyst and from my father before him, particularly fear of authority.

During the time that I was waiting to start work in Dr. Wolff's program, I reverted to some old bad habits, worrying what he would expect of me and whether I would be able to live up to his expectations. I started ruminating just as if I were back in psychoanalytic training: "Will I be able to measure up to his expectations?"

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A question posed by a good friend gave me back beginner's mind. After listening to my worries, he just asked, "Are you going over there to measure up to Harold Wolff's expectations or to try to learn something about obesity?"

This question had a surprising impact and it lifted a terrible burden. In a moment, the frustrations of the years of psychoanalytic training vanished. From then on, all that mattered was whether I could learn something about obesity. It might be a lot and it might be a little, but whatever happened was just between me and the work.

At the time I had no idea how long I would remember this glimpse of beginner's mind, but it has stayed fresh all these years.

Not long after I went to work with Dr. Wolff there was another such experience. Each week one of the postdoctoral fellows presented a case that was supposed to suggest a topic for further study. I had already seen a large number of obese patients and had learned some fascinating things about them, but for a while none seemed to suggest a topic for further study. Then suddenly one occurred to me, stimulated by the memory of an event that had occurred long before.

During the period when I was in psychoanalytic training, a few of us had begun to use wire recorders, which had just been developed, to record interviews with patients, and we met periodically to discuss them. One evening I presented the case of a 17-year-old obese girl whom I had been treating for more than a year and who had just broken off treatment under pressure from her father. I began by describing the patient's background and the course of treatment and then turned on the recording. We were sitting there listening quietly when abruptly an obese young woman psychiatrist stood up. Gasping for air, she staggered to the door. I followed her and, when she turned, I was startled to see the anguish in her face. She was in the midst of a panic attack.

She came back to the room reluctantly and only after reassurance that the wire recorder was turned off. Gradually she was able to describe what had happened: "It wasn't anything about the father. That was a problem but that wasn't what did it. That was nothing. It was how that girl talked about the way she eats. Nothing for breakfast. She is never hungry at all in the morning. Then what it's like at night, how she can't seem to stop eating. Supper doesn't satisfy her, and she just goes on and on. She even gets out of bed to eat. . . . That's how I eat and I never heard about it from anybody else in my whole life."

In the midst of my search for a topic for further study, this memory came back fresh and clear. The anguish on the face of the obese psychiatrist told a beginner's mind that this was what I had been waiting for. So I began to ask my obese patients about how they ate, and in a short time I had learned a good bit about this "night-eating syndrome."

Dr. Wolff and I wrote a paper (2) that ended:

> (a) A distinctive eating pattern of obese patients is described. This "night-eating syndrome" is characterized by nocturnal hyperphagia, insomnia, and morning anorexia.
>
> (b) Patients manifesting the syndrome had great difficulty losing weight and experienced a high incidence of complications in their attempts.
>
> (c) It is suggested that the syndrome represents a response to stress of a type peculiar to certain obese patients, and that it is intimately related to the pathogenesis of their obesity.

What happened after publication of this report was interesting. As I was trying to work out the key features of the night-eating syndrome, I began to wonder whether there were other distinctive eating patterns. This curiosity was heightened by a report on the eating patterns of laboratory rats. Following the production of lesions in the ventromedial nucleus of the hypothalamus, these rats lost their usual meal patterns, over-ate, and became obese. (3).

Now in hot pursuit of new eating patterns, I went out looking for a person with a similar history. Eventually I found one—a young man with a history of a major weight gain following a bout of encephalitis. He ate in a manner reminiscent of the rats with hypothalamic damage, and it looked as if he showed a second pattern—"eating without satiation" (4). Neither of these eating patterns had ever attracted much attention. But a third pattern, which seemed no more interesting at the time, has since aroused a great deal of interest. It is binge-eating.

Although I had undoubtedly encountered binge-eating early in my days in Dr. Wolff's clinic, I didn't pay much attention to it. The prevailing view was that "compulsive eating" caused obesity (how else would compulsive eaters eat?). It was not until I had thought about night-eating and "eating without satiation" that I began to consider alternatives. New concepts from neurophysiology played a large part in this search for alternatives, particularly the concept of a dual-control system of feeding behavior. Until then, satiety had been viewed as a purely incidental aspect of eating—the result of the drive to eat running out of steam. The discovery of specific brain areas that mediated satiety meant that satiety was a function in its own right. Very shortly thereafter, eating was viewed as a behavior under two types of control—with one mechanism (hunger) to start it and another mechanism (satiety) to turn it off. When I looked at it that way, it seemed as if both of the patterns I had studied resulted from a failure of satiety. Could there be a pattern of increased drive?

It required no more than the question to produce the answer. *Of course* there were people who over-ate as if they had an increased hunger drive. It was possible in a short time to describe the pattern of binge-eating, which has since been called bulimia (4,5). After a few years, this pattern began to arouse the intense interest that is now accorded it.

Another event some years later had some of this same quality of freshness and surprise. It occurred at a time when it was widely believed that obese people were emotionally disturbed and that their emotional disturbance caused their obesity. There were all sorts of ideas as to how emotional disturbances caused obesity; the one that sticks in my mind was "insatiable oral drives." This background made it hard to understand the results of a small study of obese men, for these men did not differ from a control group of non-obese men, either in the extent of emotional disturbance or in psychological traits—even in "insatiable oral drives" (6).

I think that this was the first study to use control groups in assessing psychological aspects of obese persons. It challenged the widespread belief about the connection between emotional disturbance and obesity, and set me to wondering about that belief. Was it true that obese people were more neurotic than non-obese people?

The opportunity to settle the question arose from a chance encounter in a bar during the medical meetings that used to fill Atlantic City in the spring. On this particular evening, I met Leo

Srole, a sociologist, who had just that day presented the first results of the Midtown Manhattan Project, at that time the most ambitious survey of mental illness yet undertaken (7). It was based on interviews with a community sample of 1,660 persons who had been carefully selected to represent a population of more than 100,000.

The interviews had collected information about the social class and ethnic background, as well as the physical and mental health of the subjects. The results were revolutionary for that time: 20% of the sample reported impairment in family relationships and work performance and significant symptoms of personal distress. These figures were so much higher than any previous estimate that Srole took pains to defend his findings: "With 1,660 subjects, you really can count on definitive results."

The mention of large numbers and definitive results intrigued me. I could hardly wait to ask,"Were your obese subjects more neurotic than your non-obese ones?"

He paused for a long time.

"You know," Srole began slowly, "We never did look at obesity."

"But you do have heights and weights, don't you?" I asked.

"Yes, we do" he replied. Well, why don't we look at obesity?

We did look at obesity and we got a definitive answer. But it wasn't very interesting. Obese people *were* more neurotic than non-obese people, or at least they scored higher on some scales, and the difference was statistically significant.

The problem was that they weren't much more neurotic. The statistical significance was a result of the large sample size; from a practical point of view, the findings were trivial.

So where did that leave us?

Leo Srole, Mary Moore and I dutifully wrote a paper with the dramatic message that obese people were a little more neurotic than non-obese people. We had no idea why nor any idea what to do with this tidbit, nor did the journals to which we submitted the paper. Both turned it down.

It seemed a shame that such fine data should come to such a sorry end, and the study kept bothering me. It wasn't clear what more could be done, but it seemed that something *should* be done.

The solution came while I was sitting at a counter in the Pennsylvania Station in New York City eating a dish of ice cream and waiting for a train to Philadelphia. In a kind of reverie, I found myself thinking about the relationship between social class and mental illness. The Midtown Study had been designed to find out if there was such a relationship and it had found it. So, since we had data on social class, we had entered it into our analyses along with a host of other variables without thinking very much about it. I seemed to remember that it had been useful and, if that were the case, it must mean that there was a relationship between social class and obesity. But I couldn't for the life of me remember whether we had found one or not. Then it occurred to me that I had never heard of a relationship between social class and obesity. Had we stumbled on one?

I could hardly wait to get back to Philadelphia to look at the data. When I did, the results were stunning—there was a very strong relationship between social class and obesity.

Such a relationship had never even been suspected before and here was one of enormous power. Figure 2 shows that 30% of lower-class women were obese, compared with 18% of middle-class women and only 5% of upper-class women (8).

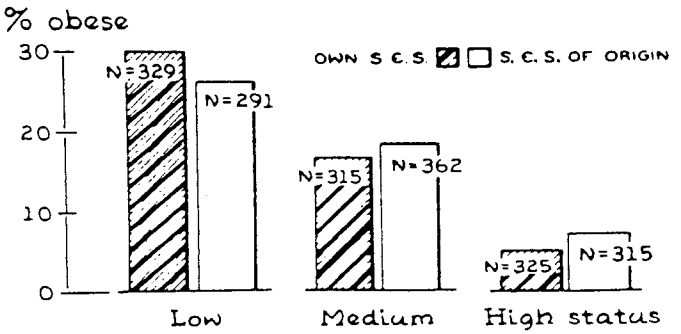

FIGURE 2: Prevalence of obesity in women as a function of social class. Obesity (at least 25% above ideal body weight) was present in 30% of women of lower socioeconomic status, 18% among middle-class and 5% among upper-class women. Socioeconomic status of origin (social class of the respondents' parents when respondents were 8 years of age) was almost as closely related to obesity as was their own social class, suggesting that the social class into which one is born is a determinant of obesity.

(Goldblatt PB, Moore ME, Stunkard AJ: Social factors in obesity. *Journal of the American Medical Association. 192:*1039–1044, 1965. Copyright 1965, American Medical Association. Reprinted by permission.)

Mary and I were pleased that we had made something more of our data. But it was more than pleasure; it was a feeling of awe. Even today when I see this figure, some of that feeling comes back. Clearly, we had stumbled onto a very powerful relationship; Mary Moore and I spent years learning more about it. Others have carried it even further; by last year 144 papers on social class and obesity had been published (9).

For a long time I did not study treatment, and when I began it was not with the treatment of obesity but with the treatment of anorexia nervosa. The work grew out of descriptive studies. They had been devised to answer a question that was simple enough: are anorectic patients more active than other people? Some psychiatrists, following Sir William Gull's original description of more than a century ago, maintained that they were. Others said no, that any activity on the part of these emaciated girls would seem like hyperactivity. We decided to answer the question by measuring the activity of the girls with pedometers.

We had found pedometers useful in the study of the physical activity of obese people. Obese women were clearly less active than non-obese ones: obese women walked an average of 1.8 miles a day, while non-obese controls walked 4.8 miles a day (10). So we asked anorectic patients to wear pedometers and we soon had the answer: anorectic girls were extremely active. Even those confined to the hospital logged more than 6 miles a day, a finding that seemed improbable until we found them walking up and down the ten flights of hospital stairs each night.

We were just completing this study in 1965 when I had my first experience with behavior therapy. John Paul Brady showed me a case report about an anorectic patient that had just been published (11). It described a 14-pound weight gain in eight weeks, which had been achieved by a simple operant paradigm—positively reinforcing weight gain by a variety of attentional measures. This report was notable because it described the sensitivity of an anorectic patient to immediate behavioral contingencies. I had always experienced anorectic patients as frozen, withdrawn, and

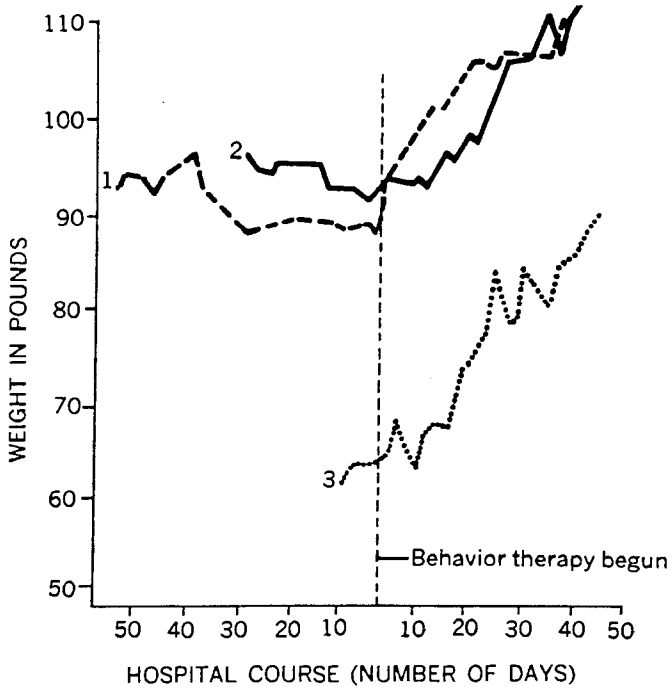

FIGURE 3: Weight gain by three anorectic patients following the application of a simple behavioral paradigm—access to physical activity contingent upon weight gain. Weight was stable during the period of usual hospital management and increased upon initiation of behavioral contingency.

(*American Journal of Psychiatry. 126:*1093–1098, 1970. Copyright 1970, the American Psychiatric Association. Reprinted by permission.)

stubbornly unresponsive, particularly to efforts to persuade them to eat.

When Barton Blinder, then a resident, and I discussed this report with Paul Brady and told him about the hyperactivity that we had found in these girls, he suggested that we incorporate it into a behavioral paradigm, making access to physical activity contingent on weight gain. Specifically, we gave the patient six hours outside the hospital on any day when her morning weight was at least half a pound above the previous morning's weight.

The idea seemed so reasonable that we were quite unprepared for the violent reaction of the first patient to whom we proposed it. This young landscape designer, up until then polite and well-mannered, exploded in a torrent of anger and abuse that made me think that we must have made a terrible mistake. Much to our surprise, however, she did not leave the hospital against medical advice as she had threatened, and within three days she settled into the program with a vengeance. During the next three weeks she gained more than 17 pounds and, what is more, her tense, frozen manner thawed. She began to be warm and friendly, and to seek out and talk with other patients and nurses. She said that she felt much better, and clearly she did.

We used this method on the next two patients who were hyperactive with comparable results. Then we encountered a patient who was so emaciated that she couldn't even get out of bed, let alone walk up and down the stairs. She complained bitterly about the large dose of chlorpromazine that we were giving her,

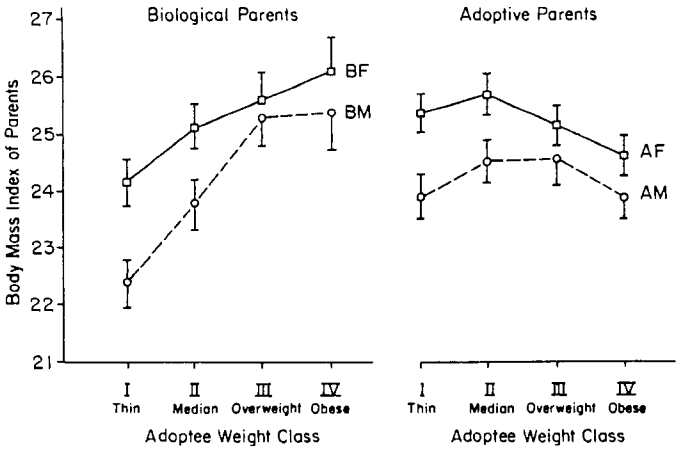

FIGURE 4: Body mass index of the parents of adoptees of differing weight classes. As the weight class of the adoptees increases, so does the body mass index of their biologic parents. There is no such increase among the adoptive parents. BF = biologic father; BM = biologic mother; AF = adoptive father; AM = adoptive mother.

(*The Pain of Obesity*. Copyright 1976, Bull Publishing Co., Palo Alto, CA. Reprinted by permission.)

and her complaints suggested another approach. When we introduced a schedule that reduced the dose of chlorpromazine contingent upon weight gain, we found a weight gain and a psychological improvement comparable to that seen in the first three patients.

After years of ineffectively trying to help these desperately ill little girls (12), the improvement in these patients was startling and immensely gratifying (see Figure 3). I remember being delighted by the comment of one of these patients when I visited her the evening before her discharge from the hospital. Looking at me intently, she demanded, "Why didn't you people do something as soon as I came into the hospital? I must have been out of my mind; I could have starved myself to death."

As those of you who work with anorectic patients know only too well, there is a great deal more to the treatment of anorexia nervosa than can be achieved by a brief stay on a psychiatric service. But for those of us who lived these events, it was inspiring, something that we will never forget. All of us experienced beginner's mind during those wonderful days, and these surprising results led me into years of research on the behavioral treatment of obesity.

These events occurred years ago, but the magic of discovery continues, and now I am a beginner in the field of behavior genetics. My first experience was a study of adoptees that I carried out with Thorkild Sorensen in Denmark. We studied 4,500 men and women who averaged about 40 years in age and who had been removed from their biological parents early in life. We gathered information about their heights and weights and then selected four samples—the 4% who were thinnest, the 4% who were fattest, the 4% who were next to the fattest, and the 4% who were at the median. Then we compared the weight category of the adoptees with the body mass index of their adoptive parents and of their biologic parents.

The most surprising finding of the study shown in Figure 4 was that there was no relationship between the weight category

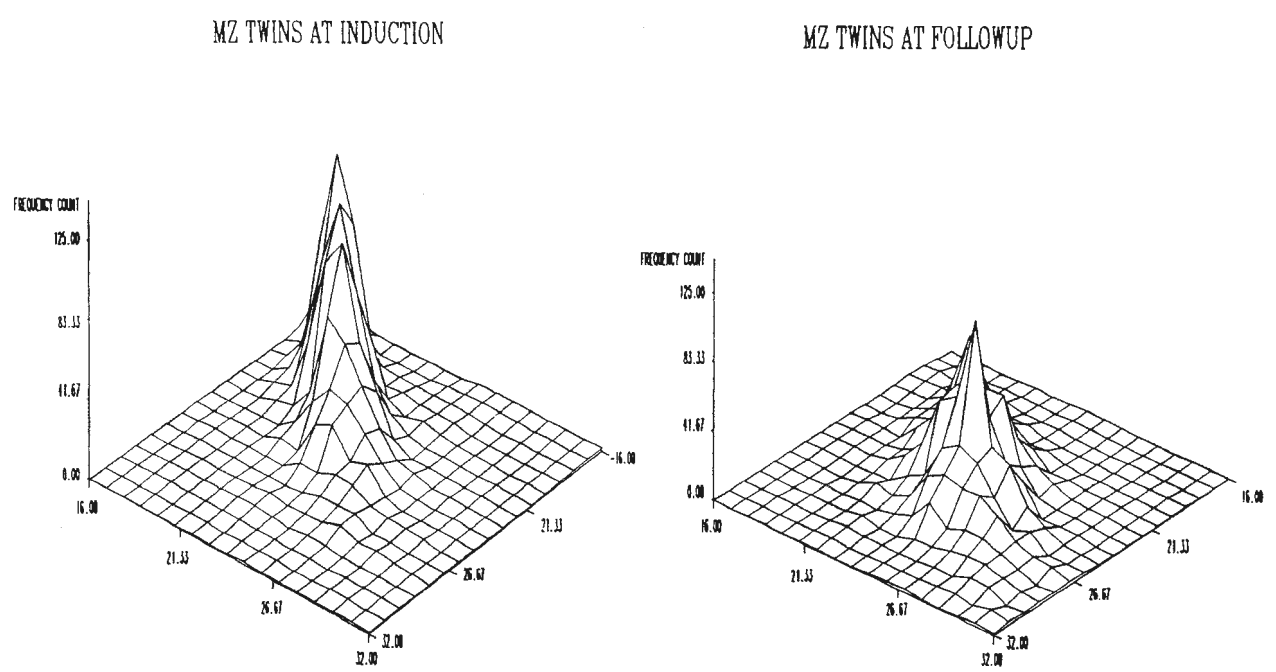

FIGURE 5: Relationship of the body mass index of members of identical twin pairs. The body mass index of one twin is plotted along one axis, that of his co-twin along the other.

(*Human Heredity. 39:*121–135, 1989. Copyright 1989, S. Karger Publishing, Basel, Switzerland. Reprinted by permission.)

of the adoptees and the body mass index of their adoptive parents (13). For someone who had been indoctrinated with the idea that the first five years of life were of paramount importance, these results were a real shocker.

By contrast, there was a strong relationship between the weight class of the adoptees and the body mass index of their biologic parents (13). These findings were so clear that when I first saw them I was reminded of the growl of my old chief, Harold Wolff, "Dr. Stunkard, if there is something there, you don't need statistics to show it."

The adoption study did not provide a quantitative estimate of the strength of the genetic effect on obesity, but a twin study, done with Terry Foch and Zdenek Hrubec, did (14). This study involved 4,000 male twin pairs whose heights and weights had been measured at age 20 and again at age 45. We calculated an estimate of heritability and compared it with the heritability of other disorders that had been studied by the twin method (see Table 1). The heritability of the body mass index was .77 at age 20 and .84 at age 45, considerably higher than that of several other disorders.

When this strong genetic influence on human obesity became apparent, there was an unfortunate reaction both in the lay press and among our colleagues. They proclaimed that since the genetic influence on obesity is so strong, there wasn't really very much that could be done about it. I knew that this wasn't true; I had seen too many obese people beat the odds, lose weight, and stay reduced. But for a time it wasn't clear how to counter this counsel of despair. My colleague Arlen Price showed how to do it.

His solution involved making use of the unique characteristics of identical twins. Because identical twins are genetically identical, any difference between members of a pair must be due to environmental influences. Arlen Price proposed that we explore these differences among the identical twins whom Foch, Hrubec, and I had studied earlier. When we arrayed the twins according to the extent of their body mass index, a striking relationship emerged (15).

In the normal weight range, the differences in body mass index within twin pairs was very small; as the subjects became more obese, there was a marked increase in the difference between the pairs. This finding is represented graphically in Figure 5, which shows a peak in the normal weight range, representing the high frequency of twin pairs with a very similar body mass index. As the twins become more obese, this peak shrinks to a small hill,

TABLE 1

Heritability Estimates of Twin Studies of Obesity and Other Medical Conditions (14)

Condition	Heritability
Obese adults	.64
Obese adults	.77
Obese adults	.84
Schizophrenia	.68
Hypertension	.57
Alcoholism	.57
Cirrhosis of the liver	.53
Epilepsy	.50
Coronary artery disease	.49
Breast cancer	.45

(*Journal of the American Medical Association. 256:* 51–54, 1986. Copyright 1986, American Medical Association. Reprinted by permission.)

indicating that fewer twin pairs have a very similar body mass index and a larger number show differences in their body mass index. The finding is even more striking among the 45-year-old twins than it is among the 20-year-olds.

This analysis goes a long way in helping to explain the nature of the genetic influence. Although genes are a necessary condition for obesity, they are not a sufficient condition. Even with a genetic predisposition, whether people become obese and how obese they become depends upon their environment.

Clearly, in the case of body weight, genes are not destiny: genetic influences do not doom people to a life of obesity. Indeed, it provides them with a reasonable explanation of their obesity, one more respectful of them than the traditional views that obesity represents a personal failure or is the result of defective impulse control or insatiable oral drives. Since environmental influences are so effective in determining the extent of obesity, it gives hope.

I have been talking about discovery and the joy of discovery. It is something that we have all known—the release from striving and the experience of grace, of some gift quite beyond our control. In a way it *is* beyond our control; but in another sense we can do something about the conditions for discovery, for they arise from beginner's mind—taking each moment as it arises as a moment of discovery and giving ourselves wholly to that moment.

The wonderful freedom of our society makes it possible for us to give ourselves wholly to the moment. For me, in the words of my friend so long ago, it is to try to learn something about obesity, to follow whatever leads seem most promising, whether or not I know anything about the subject, even though each time I am just a beginner.

Being a beginner brings problems, too. Granting agencies don't look kindly on beginners; they prefer experts. Compared to the experts you are always the new boy on the block. It seems as if everybody knows more than you do. I have this feeling most of the time, especially now, trying to learn something about genetics.

But enough of this. Suzuki-roshi had a caution about old men telling war stories: "Repeating his recollections in this way, his personality will be twisted more and more, until he becomes quite a disagreeable, stubborn fellow. This is an example of leaving a trace of one's thinking. We should not forget what we did, but it should be without an extra trace."

How do you do that? "In order not to leave any traces, when you do something, you should do it with your whole body and mind . . . you should do it completely, like a good bonfire. You should not be a smoky fire. You should burn yourself completely" (1).

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