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#106

## **HEDONIC CAPACITY: Some Conjectures**

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One important historical consequence of the discoveries of Freud and his co-workers has been the widespread acceptance among psychotherapists (of various persuasions, many of whom would not describe themselves as “Freudian”) of what might be labeled the *impedance* view of almost all psychological aberration. In suggesting this locution I do not mean to pseudo-solve theoretical or therapeutic problems by a piece of novel semantics. Because I want to avoid discussing theoretical and technical differences among psychotherapists who would object to formulations in the “proper Freudian” terminology (e.g., *resistance*, *defense*, *superego anxiety*) while I focus attention upon a view that is ubiquitous and rarely questioned, I use the term *impedance* for it is descriptive and fairly neutral with respect to other theoretical and tactical controversies.

The essence of the impedance view of psychological malfunctioning and suffering can be simply stated: Most (all?) persons who seek professional help because of what they (or their friends, relatives, or internists) perceive as “psychological problems” are in psychological trouble because of the existence of certain mental forces or structures that *impede* effective behavior and resultant subjective impulse gratification. From this simple principle—that the trouble with neurotics is some psychological force impeding their functioning—it should be obvious that the theoretical posture and the therapeutic

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technique should be primarily focused on *removing* those impeding forces. Thus, the classical view of the essence of therapy<sup>\*</sup> rests basically upon this impedance conception of psychological distress and social or medical malfunction.

Of course, there are other broadly uncovering or insight-oriented psychotherapies (e.g., the Rogerian) that cannot be formulated in Freudian terms. These psychotherapies, despite their avoidance of such concepts as resistance, working through, or interpretation, share with the classical Freudian position the basic notion that people suffer psychologically, malfunction socially, or produce psychosomatically-mediated symptoms primarily because certain psychological counterforces do exist and operate as *impedances* to pleasure-seeking, gratification, loving, working, problem-solving, effective social conduct, and the like.

It is not my purpose in this paper to refute the impedance doctrine, which I happen to believe has a great deal of truth to it despite the difficulty in making a rigorous scientific case for it to our skeptical academic brethren.<sup>†</sup> This brief article is avowedly conjectural in nature. Since I am a neo-Popperian in my philosophy of science, I am unabashedly presenting a conjecture (in Popper's sense) without having any substantial so-called inductive "foundations" or "confirmation" in support of it (Popper 1959, 1962, 1972; Bunge 1964; Schilpp 1974). I think the conjecture is interesting and worth pursuing, and if the reader asks—despite my neo-Popperian philosophic orientation—where I "get such ideas from," I would say they come primarily from a mixture of casual observation with some thirty years experience as a psychotherapist. I do not, let me hasten to add, view the latter as very strongly evidentiary, since it appears obvious from the contemporary therapeutic scene that sane, intelligent, bedoctored persons of good will can spend thousands of hours listening to patients or clients and emerge with radically different views as to just "what is wrong with them." But being a good neo-Popperian, I do not believe that

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\* I use the phrase "essence of therapy" to refer to interpretations whose technical function is to enable the patient's ego to tolerate less distorted derivatives, characterized over the long run as a process of working through his resistances—equating resistance with defense as it occurs in the therapeutic situation.

† See Meehl 1970.

a conjecture requires positive support. All you need to justify in propounding a conjecture is the existence of a problem; and, so far as I know, the existence of psychological problems is not in dispute! So while I will mention some scattered bits and pieces of evidence, partly clinical, partly experimental, and partly psychometric that seem to me to bear upon this conjecture of mine, I want to emphasize the highly speculative nature of this contribution. Its aim is modest, being to call attention of clinicians and theoreticians to an interesting and important *possibility* which is researchable in principle and which, at the very least, we can stack up against our own clinical experience in trying to help people with their psychological difficulties.

My conjecture, like the tradition of the impedance doctrine, can be stated (as a first approximation) quite simply: In the impedance doctrine, we assume that all neurotics are suffering an impairment of both their positive gratifications and effective instrumental behaviors. Such impairment is a result of the impeding influence of negative affects (notably the anxiety signal) and the defensive systems acquired to handle them. This formula constitutes a theoretically-biased orientation on our part. What we know of the mental machinery (whether from the laboratory or from common observation) should lead us to suspect that there is, in many people, an equally important quantitative influence in the *opposite* direction, namely, a quantitative feebleness or weakness of the positive reinforcers that normally function as “softeners” of aversive states (such as rage and fear). Such persons—who might be characterized as being at the low end of the continuum for *basic hedonic capacity*—are not, therefore, suffering primarily from impedances; and a psychotherapy oriented fundamentally toward clearing away impedances may be inappropriate for them.

Before the discovery of specific positive and negative reinforcement centers in the central nervous system, such a theoretical conjecture had not only considerable armchair plausibility but also corroboration by anecdotal and clinical observation. Anybody who observes children is struck by the remarkable range of individual differences in what appears to be their capability for experiencing pleasure, even at an early age.\* The impedance theory

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\* See Escalona 1968.

implies that the enjoyment potential of the apparent low hedonic type is impaired by interference from defensive characterological rigidities, excesses of the anxiety signal, and the like. I am, of course, aware that this answer would be the standard one to my conjecture. However, my aim is to call that standard answer into question by raising the possibility that just as there are some organisms whose pleasures are impeded by fear, so are there other organisms whose fears are insufficiently softened, attenuated, or, I may even say, impeded by adequate pleasure. Still, at the anecdotal level, people totally unlearned in technical psychodynamics are well aware that if you want, for instance, to discuss an emotionally-charged topic with a potential enemy, among the things you are well advised to do is feed him and sex him and otherwise make him feel good, with the purpose of generating an overall psychological state that will be more or less incompatible with anxiety and rage. But, there is also the old “Wild West” maxim: “Some men are just born three drinks behind.”

In the course of intensive psychotherapy, many patients—by no means always depressed or schizoid in makeup—spontaneously report (and more could do so if the therapist were alert to the idea of basic hedonic deficiency) that even as children they noticed they were different from the others because they had the distinct impression that everything seemed to be more fun for other children than for themselves. Here again one who is strongly committed to the impedance interpretation of neurosis can usually succeed in eliciting information supporting the view that the child was being impeded from adequate pleasure by some aversive state, such as social fear. But I think it takes some forcing of the facts to make all of the cases fit this pattern, even phenomenologically. My clinical impression is that some persons, whether reporting childhood or current adult situations, do *not* provide us with adequate support for the postulation of an impeding anxiety signal or characterological rigidity of a defensive nature; they seem simply not to get the usual quantitative “kick” out of life’s purported positive experiences.

Speaking now as an academic psychologist, taking off my clinician’s hat for the moment, I suggest this conclusion could have been theoretically expected. In practically any physical or psychological trait we have bothered to investigate systematically there

are tremendous individual differences. Given our direct experimental knowledge of the existence of Olds (+) centers in the brain, I think most psychologists would be willing to guess, with relatively high armchair confidence, that considerable differences among persons exist—and I include here “normal” persons not seeking psychotherapeutic help—in the distribution, number, density, or reactivity of these positive reinforcement (“pleasurable”) brain centers. Secondly, the growing mass of data in behavioral genetics suggests that almost any temperamental disposition in which there exists important individual differences—especially one known from direct laboratory research to reside in specific neuro-anatomical loci—is, at least to some degree, inheritable.\* Since we take for granted today that not everybody is born with an equal innate disposition to the anxiety signal, we should also take for granted that one important source of the differences between those who fall ill of a neurosis and those who, despite life’s stresses and frustrations, manage to stay reasonably healthy lies in differential susceptibility to anxiety. Therefore, I conjecture, clinicians and theoreticians ought to consider seriously the possibility that not only are some persons born with more cerebral “joy-juice” than others but also that this variable is fraught with clinical consequences.

It should be apparent from what I have said that I do not always think of what Rado called “anhedonia” as a pathological entity. My conjecture involves the usual academic psychologist’s emphasis upon a *normal individual differences variable*; however, this emphasis considerably alters the likelihood that social or medical pathology will arise at the statistical extremes of the distribution. Since I suppose very few people come to a psychotherapist for help because life is too much fun, our clinical sampling of the two ends of this distribution is biased. But I am convinced that “high-joy” people do exist; and I should be surprised if my readers, in contemplating their range of acquaintances, disagree with me. There are persons who seem able to take considerable pleasure from almost any circumstance not distinctly loaded with aversive components and for whom the most ordinary experiences appear to be a source of considerable gratification. I conjecture that these people

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\* See Meehl 1974-75 for references to genetic factors.

are the lucky ones at the high end of the hedonic capacity continuum, i.e., they were “born three drinks” *ahead*.

Because activation of the Olds (+) centers has a tendency to inhibit, soften, or attenuate the negative (aversive) experiences with which everyday life, alas, so often abounds, this normal range of individual differences has the potential for becoming extremely important clinically. I suggest that a person who has the misfortune of being on the low end of the hereditary hedonic capacity continuum—a *fact not, in itself, clinically pathological in my view*—may experience life as unduly burdensome and stressful for the simple reason that although he has to put up with the same kinds of annoyances, anxieties, and threats the rest of us do, his difficulties are not being softened or statistically counterbalanced by adequate hedonic “kicks.”\* In fact, some patients have spontaneously reported something very close to this assessment. Most psychotherapists I know have had patients (again, I emphasize, patients who are not severely depressed or convincingly schizotypal) who say such things as, “Well, you know, I have to get up in the morning when I hear the alarm clock ring and go out and shovel the walk and all that kind of junk, and what do I really get out of it? I mean, it strikes me that life is often pretty much a big pain in the neck—it just isn’t worth it; it’s more trouble than it’s worth. And I really don’t understand why most of my friends seem to feel differently about it.” Sometimes such a statement is a typical impedance neurosis complaint. Sometimes it is a cry for help. Sometimes it is part of what my colleague Schofield (1964) would describe as the “philosophical neurosis.” But *sometimes*—and, I suggest, more commonly than most clinicians are ready to recognize—it is a substantially accurate account of the patient’s reinforcement schedule (Ferster & Skinner 1957), mainly attributable to his inheriting unlucky polygenes that put him at the low end of the hedonic capacity continuum. So, in effect, he is complaining to the therapist that he is like a white rat in a Skinner box who spends most of his time pressing the lever to keep the shock turned off but does not receive very many pellets—and, for him, unfortunately, this simile represents the *literal truth*.

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\* This notion runs counter to the impedance theory but has an analogous quantitative meaning.

I should like to digress slightly to discuss my views on depression since they relate to the present conjecture about hedonic capacity. I believe (partly on the basis of the published genetic data but admittedly more on the basis of clinical impressions) that there are at least five, and possibly as many as seven, distinguishable kinds or types of depression (not associated with coarse brain disease or other organic illness). Since it is not appropriate to document this claim here, I will simply list the types: (1) endogenous bipolar (manic-depressive), (2) endogenous unipolar, (3) retroflex rage, (4) object loss, (5) extinction depression (the only kind Skinner recognizes and the kind greatly underestimated by psychotherapists in the psychodynamic tradition), (6) depression secondary to schizotypy, and (7) *depression secondary to low hedonic capacity*. Skinner's extinction depression applies to a person who becomes depressed not because of any complex intrapsychic dynamics and not because an inherited psychotic depression gene is activated, but because he has recently been put on a quantitatively unsatisfactory reinforcement schedule, perhaps quite by happenstance. For instance, if I hit a period during which I do not sell much insurance and my wife does not have orgasms and my friends do not laugh at my jokes and my new car is full of "bugs," I will tend to become depressed.\* The layman often understands this kind of depression more readily than the psychotherapist. "No wonder the poor guy is depressed. Everything bad has happened to him all at once lately."

My conjecture about depression secondary to low hedonic capacity is a kind of *physiological equivalent* to Skinner's extinction depression. However, instead of the external social Skinner box administering an inadequate number of positive reinforcers, those "objective" pellets that are being delivered are not psychophysiologicaly *received* as reinforcers because of the aberrated hedonic parameter. Of course, the important practical difference is that extinction depression will either go away by itself (if it is merely a random "bad run" of extinction trials) or can be altered by helping the patient into a different social Skinner box in which the pellet deliveries will happen more frequently, e.g., change jobs, wives, cars, or whatever. Although with this kind of patient a

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\* See Meehl 1962b.

therapist may be operating mistakenly upon a pure impedance view of neurosis, he nevertheless may do the patient a great deal of good. In the course of interpreting the alleged impedances there can occur a cognitive restructuring and a freeing up of risk-taking trial and error; and, as a result, the patient moves into a positive reinforcement schedule and undergoes significant quantitative improvement. However, for the patient whose depression is secondary to his being genetically located at the low end of the hedonic capacity continuum, no such effective behavior is available. Although Rado (1956) has talked about teaching patients (mainly schizotypes) to learn to live with a "scarcity economy of pleasure," I must confess I have not found his writings on this problem very practical.

I must emphasize that, given the conjectural nature of this brief contribution, I make no pretense whatever to a systematic literature search of either experimental or differential psychology studies. Instead, I shall mention certain well-known, exemplifying studies that are, at least, consistent with the proffered conjectures. The classic undergraduate textbook example of interfering with a conditioned anxiety signal by associating the anxiety elicitor with a positive (reinforcing) stimulus is Jones' (1924) report on how a child's conditioned fear of a rabbit was overcome by contiguous feeding. Then we have Farber's (1948) finding that the well-known rigidity or "response fixation" under anxiety in the T-maze (using rats as subjects) could be reduced by feeding in the experimental situation, although no reinforcement contingencies were involved. "Counterconditioning" or "reciprocal inhibition" experiments provide the theoretical basis underlying Wolpe's therapeutic method (Wolpe & Lazarus 1966). Brady and Conrad's (1960) study revealed that direct intracranial limbic system self-stimulation of positive centers could markedly attenuate a conditioned aversive emotional response in rats and monkeys, although not in cats.\* I would also consider relevant the analgesic effect of masturbation reported by Marchand (1961) and, along similar lines, the regrettable and (according to my colleagues in psychopharmacology) rather consistent correlation between the potency and dependability of antipain

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\* See also Olds 1958; Hernandez-Péon 1964, especially the mutual antagonism diagram 17 on p. 199; Wilson & Davison 1971, replied to by Reid 1973.

drugs and their addictive and euphoria-inducing properties. In the search for better pain relievers, repeated claims have been made that some new drug (e.g., Demerol) is just as effective as morphine but less addictive. Such claims have regularly proven incorrect.\* The psychological element in subjective physical pain is universally recognized today and hardly in need of further documentation. How much something hurts, even though it has a clear-cut peripheral origin in organic disease, depends considerably (some pain experts would argue for the word *mainly*) upon what might be broadly called the anxiety component involved.

At present it would not be possible to marshal a comparable body of clinical or research evidence from either differential psychology or behavior genetics. As for differential psychology, I will mention, as a plausible candidate for identification with hedonic capacity, Cattell's (1935) *surgency*, an individual differences factor which he says shows up consistently not only in his work but also in the work of most investigators but usually under other names (Cattell 1957). For example, English and English (1958) define it as "a trait inferred to account for behaviors that are cheerful, lively, responsive, social, trustful." In a statistical study of personality ratings on patients by psychotherapists (Meehl *et al.* 1971), my colleagues and I were surprised (since neither we nor the rating clinicians were routine users of Cattell's factor scheme) to detect a large individual differences factor which we tentatively called *surgency* and which Cattell<sup>†</sup> unreservedly agreed with on reading the item content. Readers of that paper (Meehl *et al.* 1971) might reasonably argue that our *surgency* factor is too much permeated with social or interpersonal item content to qualify as a hedonic potential variable, and I would want to give that argument due weight. However, while I cannot present a rebuttal here, I suggest that in a variety of clinical and research contexts—not only those involving hedonic capacity—one might expect an overrepresentation of social content in differential psychology studies (1) because of the relatively higher clinical visibility of the social indicators of psychological factors to raters, and, in my view more significantly, (2) because parametric differences in basic temperamental variables

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\* See, for example, Modell 1961.

† Personal communication.

reflected in acquired molar behavior traits should theoretically be activated by an intermittent reinforcement schedule as is the case for social dispositions. My hunch is that this factor explains why the aversive drift invariably seen in schizotypes tends—although there are striking individual exceptions—to be largely reflected in the interpersonal area (Meehl 1962a, 1964, 1972b). The reinforcement schedule the external world imposes upon most of us with regard to *most* domains of instrumental behavior (such as the act of reaching for a pencil or, as we see in many compensated schizotypes, seeking aesthetic and intellectual pleasures) *tends*, at least in the statistical sense, to be more “consistent”<sup>\*</sup> than is true of most interpersonal responses.

Finally, as to the inheritability of hedonic capacity, I must repeat that I rely here (probably much more than most of my readers would consider legitimate) on an armchair formula: Any basic temperamental variable that shows pronounced individual differences, especially one for which specific neuro-anatomic loci are known, will to some considerable extent be variable because of genetic differences.<sup>†</sup> As it happens, Lieblich and Olds (1971) have reported successful genetic selection for readiness to respond to intracranial stimulation as a positive reinforcer.

Admittedly these miscellaneous clinical, psychometric, and experimental findings provide only weak corroboration for my composite conjecture. Strong tests are imaginable, but probably must await better assessment technologies than we presently have. The inheritable hypohedonia could be checked by monozygotic-dizygotic twin studies and foster child studies, given a psychometric or psychophysiological “pleasure measure” of high validity. But the conjectured causal chain would presumably require follow-up studies of infants whose early developmental stages revealed normal emergency affectivity but a hedonic defect nevertheless—a tough measurement problem indeed!

I conclude by admitting ruefully that the clinical implications of these theoretical conjectures are, at present, almost wholly negative

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<sup>\*</sup> In Skinnerian language, one might describe them as having a smaller VR environmental parameter.

<sup>†</sup> See, for example, Williams 1956; Meehl 1972a and the research literature cited therein.

One justification for this communication is to set some good clinical brains working on constructive suggestions for the therapeutic management of patients whose complaints arise largely from their having inherited a defective hedonic capacity. The negative clinical implication is, of course, that interview tactics predicated upon the impedance view of all maladjustment are in such cases contraindicated for the simple reason that they take time and money, yet are unlikely to work.

My positive suggestions are, I am afraid, largely cognitive as well as tentative. I think people with low hedonic capacity should pay greater attention to the “hedonic bookkeeping” of their activities than would be necessary for people located midway or high on the hedonic capacity continuum. That is, it matters more to someone cursed with an inborn hedonic defect whether he is efficient and sagacious in selecting friends, jobs, cities, tasks, hobbies, and activities in general. Secondly, I have a strong clinical impression that, at least in the American culture, many people develop a kind of “secondary guilt” or shame about hedonic defects, particularly in the interpersonal domain, because the dominant ethos prescribes that everybody must be an extravert and must receive tremendous gratification from social interaction. I am convinced that active therapeutic maneuvers of the rational-emotive type (Ellis 1962, 1971; Ellis & Harper 1961) can be highly effective in reducing this secondary guilt and shame by helping the patient accept the fact that not everybody gets a big “kick” out of social interaction—there is no compelling reason why everybody has to be the same in this respect, any more than we all have to be the same in our needs for exercise, food, play, sleep, sexual outlet, music, poetry, art, beer, or whatever. But I must confess, again speaking conjecturally, that I have more faith in the ultimate efficacy of a psychopharmacological approach to this problem than I do in psychological intervention. Judging from what Freud had to say about the ultimate biochemical approach to constitutional issues, I believe he would probably go along with this suggestion.

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