YES

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ON BEING SANE IN INSANE PLACES

If sanity and insanity exist, how shall we know them?

The question is neither capricious nor itself insane. However much we may be personally convinced that we can tell the normal from the abnormal, the evidence is simply not compelling. It is commonplace, for example, to read about murder trials wherein eminent psychiatrists for the defense are contradicted by equally eminent psychiatrists for the prosecution on the matter of the defendant's sanity. More generally, there are a great deal of conflicting data on the reliability, utility, and meaning of such terms as "sanity," "insanity," "mental illness," and "schizophrenia." Finally, as early as 1934, Benedict suggested that normality and abnormality are not universal. What is viewed as normal in one culture may be seen as quite aberrant in another. Thus, notions of normality and abnormality may not be quite as accurate as people believe they are.

To raise questions regarding normality and abnormality is in no way to question the fact that some behaviors are deviant or odd. Murder is deviant. So, too, are hallucinations. Nor does raising such questions deny the existence of the personal anguish that is often associated with "mental illness." Anxiety and depression exist. Psychological suffering exists. But normality and abnormality, sanity and insanity, and the diagnoses that flow from them may be less substantive than many believe them to be.

At its heart, the question of whether the sane can be distinguished from the insane (and whether degrees of insanity can be distinguished from each other) is a simple matter: do the salient characteristics that lead to diagnoses reside in the patients themselves or in the environments and contexts in which observers find them? From Bleuler, through Kretchmer, through the formulators of the recently revised *Diagnostic and Statistical Manual* of the American Psychiatric Association, the belief has been strong that patients present symptoms, that those symptoms can be categorized, and, implicitly, that the sane are distinguishable from the insane. More recently, however, this belief has been questioned. Based in part on theoretical and anthropological considerations, but also on philosophical, legal, and therapeutic ones, the view has grown that psychological categorization of mental illness is useless

at best and downright harmful, misleading, and pejorative at worst. Psychiatric diagnoses, in this view, are in the minds of the observers and are not valid summaries of characteristics displayed by the observed.

Gains can be made in deciding which of these is more nearly accurate by getting normal people (that is, people who do not have, and have never suffered, symptoms of serious psychiatric disorders) admitted to psychiatric hospitals and then determining whether they were discovered to be sane and, if so, how. If the sanity of such pseudopatients were always detected, there would be prima facie evidence that a sane individual can be distinguished from the insane context in which he is found. Normality (and presumably abnormality) is distinct enough that it can be recognized wherever it occurs, for it is carried within the person. If, on the other hand, the sanity of the pseudopatients were never discovered, serious difficulties would arise for those who support traditional modes of psychiatric diagnosis. Given that the hospital staff was not incompetent, that the pseudopatient had been behaving as sanely as he had been outside of the hospital, and that it had never been previously suggested that he belonged in a psychiatric hospital, such an unlikely outcome would support the view that psychiatric diagnosis betrays little about the patient but much about the environment in which an observer finds him.

This article describes such an experiment. Eight sane people gained secret admission to 12 different hospitals. Their diagnostic experiences constitute the data of the first part of this article; the remainder is devoted to a description of their experiences in psychiatric institutions. Too few psychiatrists and psychologists, even those who have worked in such hospitals, know what the experience is like. They rarely talk about it with former patients, perhaps because they distrust information coming from the previously insane. Those who have worked in psychiatric hospitals are likely to have adapted so thoroughly to the settings that they are insensitive to the impact of the experience. And while there have been occasional reports of researchers who submitted themselves to psychiatric hospitalization, these researchers have commonly remained in the hospitals for short periods of time, often with the knowledge of the hospital staff. It is difficult to know the extent to which they were treated like patients or like research colleagues. Nevertheless, their reports about the inside of the psychiatric hospital have been valuable. This article extends those efforts.

PSEUDOPATIENTS AND THEIR SETTINGS

The eight pseudopatients were a varied group. One was a psychology graduate student in his 20s. The remaining seven were older and "established." Among them were three psychologists, a pediatrician, a psychiatrist, a painter, and a housewife. Three pseudopatients were women, five were men. All of them employed pseudonyms, lest their alleged diagnoses embarrass them later. Those who were in mental health professions alleged another occupation in order to avoid the special attentions that might be accorded by staff, as a matter of courtesy or caution, to ailing colleagues. With the exception of myself (I was the first pseudopatient and my presence was known to the hospital administrator and chief psychologist and, so far as I can tell, to them alone), the presence of pseudopatients and the

nature of the research program was not known to the hospital staffs.

The settings were similarly varied. In order to generalize the findings, admission into a variety of hospitals was sought. The 12 hospitals in the sample are located in five different states on the East and West coasts. Some were old and shabby, some were quite new. Some were research-oriented, others not. Some had good staff-patient ratios, others were quite understaffed. Only one was a strictly private hospital. All the others were supported by state or federal funds or, in one instance, by university funds.

After calling the hospital for an appointment, the pseudopatient arrived at the admissions office complaining that he had been hearing voices. Asked what the voices said, he replied that they were often unclear, but as far as he could tell they said "empty," "hollow," and "thud." The voices were unfamiliar and were of the same sex as the pseudopatient. The choice of these symptoms was occasioned by their apparent similarity to existential symptoms. Such symptoms were alleged to arise from painful concerns about the perceived meaninglessness of one's life. It is as if the hallucinating person were saying, "My life is empty and hollow." The choice of these symptoms was also determined by the *absence* of a single report of existential psychoses in the literature.

Beyond alleging the symptoms and falsifying name, vocation, and employment, no further alterations of person, history, or circumstances were made. The significant events of the pseudopatient's life history were presented as they had actually occurred. Relationships with parents and siblings, with spouse and children, with people at work and in school, consistent with the aforementioned exceptions, were described as they were or had been. Frustrations and upsets were described along with joys and satisfactions. These facts are important to remember. If anything, they strongly biased the subsequent results in favor of detecting sanity, since none of their histories or current behaviors were seriously pathological in any way.

Immediately upon admission to the psychiatric ward, the pseudopatient ceased simulating *any* symptoms of abnormality. In some cases, there was a brief period of mild nervousness and anxiety, since none of the pseudopatients really believed that they would be admitted so easily. Indeed their shared fear was that they would be immediately exposed as frauds and greatly embarrassed. Moreover, many of them had never visited a psychiatric ward; even those who had, nevertheless had some genuine fears about what might happen to them. Their nervousness, then, was quite appropriate to the novelty of the hospital setting, and it abated rapidly.

Apart from that short-lived nervousness, the pseudopatient behaved on the ward as he "normally" behaved. The pseudopatient spoke to patients and staff as he might ordinarily. Because there is uncommonly little to do on a psychiatric ward, he attempted to engage others in conversation. When asked by staff how he was feeling, he indicated that he was fine, that he no longer experienced symptoms. He responded to instructions from attendants, to calls for medication (which was not swallowed), and to dining-hall instructions. Beyond such activities as were available to him on the admissions ward, he spent his time writing down his observations about the ward, its patients, and the staff. Initially these notes were written "secretly," but as it soon became clear that no one much cared, they were subsequently written on standard tablets of paper in such public places as the dayroom. No secret was made of these activities.

The pseudopatient, very much as a true psychiatric patient, entered a hospital with no foreknowledge of when he would be discharged. Each was told that he would have to get out by his own devices, essentially by convincing the staff that he was sane. The psychological stresses associated with hospitalization were considerable, and all but one of the pseudopatients desired to be discharged almost immediately after being admitted. They were, therefore, motivated not only to behave sanely, but to be paragons of cooperation. That their behavior was in no way disruptive is confirmed by nursing reports, which have been obtained on most of the patients. These reports uniformly indicate that the patients were "friendly," "cooperative," and "exhibited no abnormal indications."

THE NORMAL ARE NOT DETECTABLY SANE

Despite their public "show" of sanity, the pseudopatients were never detected. Admitted, except in one case, with a diagnosis of schizophrenia each was discharged with a diagnosis of schizophrenia "in remission." The label "in remission" should in no way be dismissed as a formality, for at no time during any hospitalization had any question been raised about any pseudopatient's simulation. Nor are there any indications in the hospital records that the pseudopatient's status was suspect. Rather, the evidence is strong that, once labeled schizophrenic, the pseudopatient was stuck with that label. If the pseudopatient was to be discharged, he must naturally be "in remission"; but he was not sane, nor, in the institution's vie had he ever been sane.

The uniform failure to recognize sani cannot be attributed to the quality of tl hospitals, for, although there were co siderable variations among them, sever are considered excellent. Nor can it be a leged that there was simply not enou time to observe the pseudo-patien Length of hospitalization ranged from to 52 days, with an average of 19 day The pseudopatients were not, in fa carefully observed, but this failure clear speaks more to traditions within psych atric hospitals than to lack of opportuni

Finally, it cannot be said that the failu to recognize the pseudopatients' sani was due to the fact that they we not behaving sanely. While there w clearly some tension present in all them, their daily visitors could dete no serious behavioral consequences- nor, indeed, could other patients. was quite common for the patien to "detect" the pseudopatients' sanit During the first three hospitalizatior when accurate counts were kept, 35 of total of 118 patients on the admissio ward voiced their suspicions, son vigorously. "You're not crazy. You' a journalist, or a professor [referrir to the continual note-taking]. You' checking up on the hospital." While mo of the patients were reassured by tl pseudopatient's insistence that he ha been sick before he came in but was fir now, some continued to believe that tl pseudopatient was sane throughout h hospitalization. The fact that the patien often recognized normality when sta did not raises important questions.

Failure to detect sanity during tl course of hospitalization may be du to the fact that physicians operate wit a strong bias toward what statisticia

call the type 2 error. This is to say that physicians are more inclined to call a healthy person sick (a false positive, type 2) than a sick person healthy (a false negative, type 1). The reasons for this are not hard to find: it is clearly more dangerous to mis-diagnose illness than health. Better to err on the side of caution, to suspect illness even among the healthy.

But what holds for medicine does not hold equally well for psychiatry. Medical illnesses, while unfortunate, are not commonly pejorative. Psychiatric diagnoses, on the contrary, carry with them personal, legal, and social stigmas. It was therefore important to see whether the tendency toward diagnosing the sane insane could be reversed. The following experiment was arranged at a research and teaching hospital whose staff had heard these findings but doubted that such an error could occur in their hospital. The staff was informed that at some time during the following 3 months, one or more pseudopatients would attempt to be admitted into the psychiatric hospital. Each staff member was asked to rate each patient who presented himself at admissions or on the ward according to the likelihood that the patient was a pseudopatient. A 10-point scale was used, with a 1 and 2 reflecting high confidence that the patient was a pseudopatient.

Judgments were obtained on 193 patients who were admitted for psychiatric treatment. All staff who had had sustained contact with or primary responsibility for the patient—attendants, nurses, psychiatrists, physicians, and psychologists—were asked to make judgments. Forty-one patients were alleged, with high confidence, to be pseudopatients by at least one member of the staff. Twenty-three were considered suspect by at least one psychiatrist. Nineteen were suspected by one psychiatrist *and* one other staff member. Actually, no genuine pseudopatient (at least from my group) presented himself during this period.

The experiment is instructive. It indicates that the tendency to designate sane people as insane can be reversed when the stakes (in this case, prestige and diagnostic acumen) are high. But what can be said of the 19 people who were suspected of being "sane" by one psychiatrist and another staff member? Were these people truly "sane," or was it rather the case that in the course of avoiding the type 2 error the staff tended to make more errors of the first sort—calling the crazy "sane"? There is no way of knowing. But one thing is certain: any diagnostic process that lends itself so readily to massive errors of this sort cannot be a very reliable one.

THE STICKINESS OF PSYCHODIAGNOSTIC LABELS

Beyond the tendency to call the healthy sick—a tendency that accounts better for diagnostic behavior on admission than it does for such behavior after a lengthy period of exposure—the data speak to the massive role of labeling in psychiatric assessment. Having once been labeled schizophrenic, there is nothing the pseudopatient can do to overcome this tag. The tag profoundly colors others' perceptions of him and his behavior.

From one viewpoint, these data are hardly surprising, for it has long been known that elements are given meaning by the context in which they occur. Gestalt psychology made this point vigorously, and Asch demonstrated that there are "central" personality traits

(such as "warm" versus "cold") which are so powerful that they markedly color the meaning of other information in forming an impression of a given personality.

"Insane," "schizophrenic," "manic-depressive," and "crazy" are probably among the most powerful of such central traits. Once a person is designated abnormal, all of his other behaviors and characteristics are colored by that label. Indeed, that label is so powerful that may of the pseudopatients' normal behaviors were overlooked entirely or profoundly misinterpreted. Some examples may clarify this issue.

Earlier I indicated that there were no changes in the pseudopatient's personal history and current status beyond those of name, employment, and, where necessary, vocation. Otherwise, a veridical description of personal history and circumstances was offered. Those circumstances were not psychotic. How were they made consonant with the diagnosis of psychosis? Or were those diagnoses modified in such a way as to bring them into accord with the circumstances of the pseudopatient's life, as described by him?

As far as I can determine, diagnoses were in no way affected by the relative health of the circumstances of a pseudo-patient's life. Rather, the reverse occurred: the perception of his circumstances was shaped entirely by the diagnosis. A clear example of such translation is found in the case of a pseudopatient who had had a close relationship with his mother but was rather remote from his father during his early childhood. During adolescence and beyond, however, his father became a close friend, while his relationship with his mother cooled. His present relationship with his wife was characteristically close and warm. Apart from occasional angry exchange friction was minimal. The children ha rarely been spanked. Surely there is not ing especially pathological about such history. Indeed, many readers may s a similar pattern in their own expe: ences, with no markedly deleterious co sequences. Observe, however, how su a history was translated in the psych pathological context, this from the ca summary prepared after the patient w. discharged:

> This white 39-year-old male . . . manifests a long history of considerable ambivalence in close relationships, which begins in early childhood. A warm relationship with his mother cools during his adolescence. A disfant relationship to his father is described as becoming very intense. Affective stability is absent. His attempts to control emotionality with his wife and children are punctuated by angry outbursts and, in the case of the children, spankings. And while he says that he has several friends, one senses considerable ambivalence embedded in these relationships also. . . .

The facts of the case were unintentior ally distorted by the staff to achieve cor sistency with a popular theory of th dynamics of a schizophrenic reactior Nothing of an ambivalent nature ha been described in relations with parent spouse, or friends. To the extent that an bivalence could be inferred, it was prob ably not greater than is found in a human relationships. It is true the pseu dopatient's relationships with his parent changed over time, but in the ordinar context that would hardly be remarkabl —indeed, it might very well be expectec Clearly, the meaning ascribed to his ver balizations (that is, ambivalence, affectiv instability) was determined by the diag nosis: schizophrenia. An entirely differ

ent meaning would have been ascribed if it were known that the man was normal.

All pseudopatients took extensive notes publicly. Under ordinary circumstances, such behavior would have raised questions in the minds of observers, as, in fact, it did among patients. Indeed, it seemed so certain that the notes would elicit suspicion that elaborate precautions were taken to remove them from the ward each day. But the precautions proved needless. The closest any staff member came to questioning these notes occurred when one pseudopatient asked his physician what kind of medication he was receiving and began to write down the response. "You needn't write it," he was told gently. "If you have trouble remembering, just ask me again."

If no questions were asked of the pseudopatients, how was their writing interpreted? Nursing records for three patients indicate that the writing was seen as an aspect of their pathological behavior. "Patient engages in writing behavior" was the daily nursing comment on one of the pseudopatients who was never questioned about his writing. Given that the patient is in the hospital, he must be psychologically disturbed. And given that he is disturbed, continuous writing must be a behavioral manifestation of that disturbance, perhaps a subset of the compulsive behaviors that are sometimes correlated with schizophrenia.

One tacit characteristic of psychiatric diagnosis is that it locates the sources of aberration within the individual and only rarely within the complex of stimuli that surrounds him. Consequently, behaviors that are stimulated by the environment are commonly misattributed to the patient's disorder. For example, one kindly nurse found a pseudopatient pacing the long hospital corridors. "Nervous, Mr. X?" she asked. "No, bored," he said.

The notes kept by pseudopatients are full of patient behaviors that were misinterpreted by well-intentioned staff. Often enough, a patient would go "berserk" because he had, wittingly or unwittingly, been mistreated by, say, an attendant. A nurse coming upon the scene would rarely inquire even cursorily into the environmental stimuli of the patient's behavior. Rather, she assumed that his upset derived from his pathology, not from his present interactions with other staff members. Occasionally, the staff might assume that the patient's family (especially when they had recently visited) or other patients had stimulated the outburst. But never were the staff found to assume that one of themselves or the structure of the hospital had anything to do with a patient's behavior. One psychiatrist pointed to a group of patients who were sitting outside the cafeteria entrance half an hour before lunchtime. To a group of young residents he indicated that such behavior was characteristic of the oral-acquisitive nature of the syndrome. It seemed not to occur to him that there were very few things to anticipate in a psychiatric hospital besides eating.

A psychiatric label has a life and an influence of its own. Once the impression has been formed that the patient is schizophrenic, the expectation is that he will continue to be schizophrenic. When a sufficient amount of time has passed, during which the patient has done nothing bizarre, he is considered to be in remission and available for discharge. But the label endures beyond discharge, with the unconfirmed expectation that he will behave as a schizophrenic again. Such labels, conferred by mental health professionals, are as influential on the patient as they are on his relatives and friends, and it should not surprise anyone that the diagnosis acts on all of them as a self-fulfilling prophecy. Eventually, the patient himself accepts the diagnosis, with all of its surplus meanings and expectations, and behaves accordingly.

The inferences to be made from these matters are quite simple. Much as Zigler and Phillips have demonstrated that there is enormous overlap in the symptoms presented by patients who have been variously diagnosed, so there is enormous overlap in the behaviors of the sane and the insane. The sane are not "sane" all of the time. We lose our tempers "for no good reason." We are occasionally depressed or anxious, again for no good reason. And we may find it difficult to get along with one or another person—again for no reason that we can specify. Similarly, the insane are not always insane. Indeed, it was the impression of the pseudopatients while living with them that they were sane for long periods of time—that the bizarre behaviors upon which their diagnoses were allegedly predicated constituted only a small fraction of their total behavior. If it makes no sense to label ourselves permanently depressed on the basis of an occasional depression, then it takes better evidence than is presently available to label all patients insane or schizophrenic on the basis of bizarre behaviors or cognitions. It seems more useful, as Mischel has pointed out, to limit our discussions to *behaviors*, the stimuli that provoke them, and their correlates.

It is not known why powerful impressions of personality traits, such as "crazy" or "insane," arise. Conceivably, when the origins of and stimuli that give rise to a behavior are remote or unknown, or when the behavior strikes us as immutable, trait labels regarding the *behaver* arise. When, on the other hand, the origins and stimuli are known and available, discourse is limited to the behavior itself. Thus, I may hallucinate because I am sleeping, or I may hallucinate because I have ingested a peculiar drug. These are termed sleep-induced hallucinations, or dreams, and drug-induced hallucinations, respectively. But when the stimuli to my hallucinations are unknown, that is called craziness, or schizophrenia—as if that inference were somehow as illuminating as the others.

THE EXPERIENCE OF PSYCHIATRIC HOSPITALIZATION

The term "mental illness" is of recent origin. It was coined by people who were humane in their inclinations and who wanted very much to raise the station of (and the public's sympathies toward) the psychologically disturbed from that of witches and "crazies" to one that was akin to the physically ill. And they were at least partially successful, for the treatment of the mental ill *has* improved considerably over the years. But while treatment has improved, it is doubtful that people really regard the mentally ill in the same way that they view the physically ill. A broken leg is something one recovers from, but mental illness allegedly endures forever. A broken leg does not threaten the observer, but a crazy schizophrenic? There is by now a host of evidence that attitudes toward the mentally ill are characterized by fear, hostility, aloofness, suspicion, and dread. The mentally ill are society's lepers.

That such attitudes infect the general population is perhaps not surprising,

only upsetting. But that they affect the professionals—attendants, nurses, physicians, psychologists, and social workers—who treat and deal with the mentally ill is more disconcerting, both because such attitudes are self-evidently pernicious and because they are unwitting. Most mental health professionals would insist that they are sympathetic toward the mentally ill, that they are neither avoidant nor hostile. But it is more likely that an exquisite ambivalence characterizes their relations with psychiatric patients, such that their avowed impulses are only part of their entire attitude. Negative attitudes are there too and can easily be detected. Such attitudes should not surprise us. They are the natural offspring of the labels patients wear and the places in which they are found.

Consider the structure of the typical psychiatric hospital. Staff and patients are strictly segregated. Staff have their own living space, including their dining facilities, bathrooms and assembly places. The glassed quarters that contain the professional staff, which the pseudopatients came to call "the cage," sit out on every dayroom. The staff emerge primarily for caretaking purposes—to give medication, to conduct a therapy or group meeting, to instruct or reprimand a patient. Otherwise, staff keep to themselves, almost as if the disorder that afflicts their charges is somehow catching.

So much is patient-staff segregation the rule that, for four public hospitals in which an attempt was made to measure the degree to which staff and patients mingle, it was necessary to use "time out of the staff cage" as the operational measure. While it was not the case that all time spent out of the cage was spent mingling with patients (attendants, for example, would occasionally emerge to watch television in the dayroom), it was the only way in which one could gather reliable data on time for measuring.

The average amount of time spent by attendants outside of the cage was 11.3 percent (range, 3 to 52 percent). This figure does not represent only time spent mingling with patients, but also includes time spent on such chores as folding laundry, supervising patients while they shave, directing ward clean-up, and sending patients to off-ward activities. It was the relatively rare attendant who spent time talking with patients or playing games with them. It proved impossible to obtain a "percent mingling time" for nurses, since the amount of time they spent out of the cage was too brief. Rather, we counted instances of emergence from the cage. On the average, daytime nurses emerged from the cage 11.5 times per shift, including instances when they left the ward entirely (range, 4 to 39 times). Late afternoon and night nurses were even less available, emerging on the average 9.4 times per shift (range, 4 to 41 times). Data on early morning nurses, who arrived usually after midnight and departed at 8 a.m., are not available because patients were asleep during most of this period.

Physicians, especially psychiatrists, were even less available. They were rarely seen on the wards. Quite commonly, they would be seen only when they arrived and departed, with the remaining time being spent in their offices or in the cage. On the average, physicians emerged on the ward 6.7 times per day (range 1 to 17 times). It proved difficult to make an accurate estimate in this regard, since physicians often maintained hours that allowed them to come and go at different times.

The hierarchical organization of the psychiatric hospital has been commented on before, but the latent meaning of that kind of organization is worth noting again. Those with the most power have least to do with patients, and those with the least power are most involved with them. Recall, however, that the acquisition of role-appropriate behaviors occurs mainly through the observation of others, with the most powerful having the most influence. Consequently, it is understandable that attendants not only spend more time with patients than do any other members of the staff—that is required by their station in the hierarchy—but also, insofar as they learn from their superiors' behavior, spend as little time with patients as they can. Attendants are seen mainly in the cage, which is where the models, the action, and the power are.

I turn now to a different set of studies, these dealing with staff response to patient-initiated contact. It has long been known that the amount of time a person spends with you can be an index of your significance to him. If he initiates and maintains eye contact, there is reason to believe that he is considering your requests and needs. If he pauses to chat or actually stops and talks, there is added reason to infer that he is individuating you. In four hospitals, the pseudopatient approached the staff member with a request which took the following form: "Pardon me, Mr. [or Dr. or Mrs.] X, could you tell me when I will be eligible for grounds privileges?" (or "... when I will be presented at the staff meeting?" or "... when I am likely to be discharged?"). While the content of the question varied according to the appropriateness of the target and the pseudopatient's (apparent) current needs, the form was always a courteous and relevant request for information. Care was taken never t approach a particular member of th staff more than once a day, lest the staf member become suspicious or irritated In examining these data, remember tha the behavior of the pseudopatients wa neither bizarre nor disruptive. One coul indeed engage in good conversation witl them.

The data for these experiments an shown in Table 1, separately for phy sicians (column 1) and for nurses anc attendants (column 2). Minor difference between these four institutions wer overwhelmed by the degree to whicl staff avoided continuing contacts tha patients had initiated. By far, their mos common response consisted of either a brief response to the question offerec while they were "on the move" and witl head averted, or no response at all.

The encounter frequently took th following bizarre form: (pseudopatient "Pardon me, Dr. X. Could you tel me when I am eligible for ground privileges?" (physician) "Good morning Dave. How are you today?" (moves of without waiting for a response).

It is instructive to compare thes data with data recently obtained a Stanford University. It has been allegec that large and eminent universities are characterized by faculty who are so busy that they have no time for students For this comparison, a young lady approached individual faculty member who seemed to be walking purposefully to some meeting or teaching engagemen and asked them the following questions.

1. "Pardon me, could you direct me to Encina Hall?" (at the medica school: "... to the Clinical Researcl Center?").

Table 1

Self-Initiated Contact by Pseudopatients With Psychiatrists and Nurses and Attendants, Compared With Other Groups

	Psychiatric hospitals		University campus (nonmedical)	University medical center Physicians		
Contact	(1) Psychiatrists	(2) Nurses and attendants	(3) Faculty	(4) "Looking for a psychiatrist"	(5) "Looking for an internist"	(6) No additional comment
Responses						
Moves on, head averted (%)	71	88	0	0	0	0
Makes eye contact (%)	23	10	0	11	0	0
Pauses and chats (%)	2	2	0	11	0	0
Stops and talks (%)	4	0.5	100	78	100	90
Mean number of questions answered (out of 6)	*	*	6	3.8	4.8	4.5
Respondents (No.)	13	47	14	18	15	10
Attempts (No.)	185	1283	14	18	15	10

*Not applicable

2. "Do you know where Fish Annex is?" (there is no Fish Annex at Stanford).
3. "Do you teach here?"
4. "How does one apply for admission to the college?" (at the medical school: "... to the medical school?").
5. "Is it difficult to get in?"
6. "Is there financial aid?"

Without exception, as can be seen in Table 1 (column 3), all of the questions were answered. No matter how rushed they were, all respondents not only maintained eye contact, but stopped to talk. Indeed, many of the respondents went out of their way to direct or take the questioner to the office she was seeking, to try to locate "Fish Annex," or to discuss with her the possibilities of being admitted to the university.

Similar data, also shown in Table 1 (columns 4, 5, and 6), were obtained in the hospital. Here too, the young lady came prepared with six questions. After the first question, however, she remarked to 18 of her respondents (column 4), "I'm looking for a psychiatrist," and to 15 others (column 5), "I'm looking for an internist." Ten other respondents received no inserted comment (column 6). The general degree of cooperative responses is considerably higher for these university groups than it was for pseudopatients in psychiatric hospitals. Even so, differences are apparent with the medical school setting. Once having indicated that she was looking for a psychiatrist, the degree of cooperation elicited was less than when she sought an internist.

POWERLESSNESS AND DEPERSONALIZATION

Eye contact and verbal contact reflect concern and individuation: their absence, avoidance and depersonalization. The data I have presented do not do justice to the rich daily encounters that grew up around matters of depersonalization and avoidance. I have records of patients who were beaten by staff for the sin of initiating verbal contact. During my own experience, for example, one patient was beaten in the presence of other patients for having approached an attendant and told him, "I like you." Occasionally, punishment meted out to patients for misdemeanors seemed so excessive that it could not be justified by the most radical interpretations of psychiatric canon. Nevertheless, they appeared to go unquestioned. Tempers were often short. A patient who had not heard a call for medication would be roundly excoriated, and the morning attendants would often wake patients with, "Come on, you m—— f——s, out of bed!"

Neither anecdotal nor "hard" data can convey the overwhelming sense of powerlessness which invades the individual as he is continually exposed to the depersonalization of the psychiatric hospital. It hardly matters *which* psychiatric hospital —the excellent public ones and the very plush private hospital were better than the rural and shabby ones in this regard, but again, the features that psychiatric hospitals had in common overwhelmed by far their apparent differences.

Powerlessness was evident everywhere. The patient is deprived of many of his legal rights by dint of his psychiatric commitment. He is shorn of credibility by virtue of his psychiatric label. His freedom of movement is restricted. He cannot initiate contact with the staff, but may only respond to such overtures as they make. Personal privacy is minimal. Patient quarters and possessions can be entered and examined by any staff member, for whatever reason. His personal history and anguish are available to any staff member (often including the "grey lady" and "candy striper" volunteer) who chooses to read his folder, regardless of their therapeutic relationship to him. His personal hygiene and waste evacuation are often monitored. The water closets may have no doors.

At times, the depersonalization reached such proportions that pseudopatients had the sense that they were invisible, or at least unworthy of account. Upon being admitted, I and other pseudopatients took the initial physical examination in a semipublic room, where staff members went about their own business as if we were not there.

On the ward, attendants delivered verbal and occasionally serious physical abuse to patients in the presence of other observing patients, some of whom (the pseudopatients) were writing it all down. Abusive behavior, on the other hand, terminated quite abruptly when other staff members were known to be coming. Staff are credible witnesses. Patients are not.

A nurse unbuttoned her uniform to adjust her brassiere in the presence of an entire ward of viewing men. One did not have the sense that she was being seductive. Rather, she didn't notice us. A group of staff persons might point to a

patient in the dayroom and discuss him animatedly, as if he were not there.

One illuminating instance of depersonalization and invisibility occurred with regard to medications. All told, the pseudopatients were administered nearly 2100 pills, including Elavil, Stelazine, Compazine, and Thorazine, to name but a few. (That such a variety of medications should have been administered to patients presenting identical symptoms is itself worthy of note.) Only two were swallowed. The rest were either pocketed or deposited in the toilet. The pseudopatients were not alone in this. Although I have no precise records on how many patients rejected their medications, the pseudopatients frequently found the medications of other patients in the toilet before they deposited their own. As long as they were cooperative, their behavior and the pseudopatients' own in this matter, as in other important matters, went unnoticed throughout.

Reactions to such depersonalization among pseudopatients were intense. Although they had come to the hospital as participant observers and were fully aware that they did not "belong," they nevertheless found themselves caught up in and fighting the process of depersonalization. Some examples: a graduate student in psychology asked his wife to bring his textbooks to the hospital so he could "catch up on his homework"—this despite the elaborate precautions taken to conceal his professional association. The same student, who had trained for quite some time to get into the hospital, and who had looked forward to the experience, "remembered" some drag races that he had wanted to see on the weekend and insisted that he be discharged by that time. Another pseudopatient attempted a romance with a nurse. Subsequently, he informed the staff that he was applying for admission to graduate school in psychology and was very likely to be admitted, since a graduate professor was one of his regular hospital visitors. The same person began to engage in psychotherapy with other patients—all of this as a way of becoming a person in an impersonal environment.

THE SOURCES OF DEPERSONALIZATION

What are the origins of depersonalization? I have already mentioned two. First, are attitudes held by all of us toward the mentally ill—including those who treat them—attitudes characterized by fear, distrust, and horrible expectations on the other. Our ambivalence leads us, in this instance as in others, to avoidance.

Second, and not entirely separate, the hierarchical structure of the psychiatric hospital facilitates depersonalization. Those who are at the top have least to do with patients, and their behavior inspires the rest of the staff. Average daily contact with psychiatrists, psychologists, residents, and physicians combined ranged from 3.9 to 25.1 minutes, with an overall mean of 6.8 (six pseudopatients over a total of 129 days of hospitalization). Included in this average are time spent in the admissions interview, ward meetings in the presence of a senior staff member, group and individual psychotherapy contacts, case presentation conferences, and discharge meetings. Clearly, patients do not spend much time in interpersonal contact with doctoral staff. And doctoral staff serve as models for nurses and attendants.

There are probably other sources. Psychiatric installations are presently in serious financial straits. Staff shortages are pervasive, staff time at a premium. Something has to give, and that something is patient contact. Yet, while financial stresses are realities, too much can be made of them. I have the impression that the psychological forces that result in depersonalization are much stronger than the fiscal ones and that the addition of more staff would not correspondingly improve patient care in this regard. The incidence of staff meetings and the enormous amount of record-keeping on patients, for example, have not been as substantially reduced as has patient contact. Priorities exist, even during hard times. Patient contact is not a significant priority in the traditional psychiatric hospital, and fiscal pressures do not account for this. Avoidance and depersonalization may.

Heavy reliance upon psychotropic medication tacitly contributes to depersonalization by convincing staff that treatment is indeed being conducted and that further patient contact may not be necessary. Even here, however, caution needs to be exercised in understanding the role of psychotropic drugs. If patients were powerful rather than powerless, if they were viewed as interesting individuals rather than diagnostic entities, if they were socially significant rather than social lepers, if their anguish truly and wholly compelled our sympathies and concerns, would we not *seek* contact with them, despite the availability of medications? Perhaps for the pleasure of it all?

THE CONSEQUENCES OF LABELING AND DEPERSONALIZATION

Whenever the ratio of what is known to what needs to be known approaches zero, we tend to invent "knowledge" and assume that we understand mor than we actually do. We seem unabl to acknowledge that we simply don' know. The needs for diagnosis and re mediation of behavioral and emotiona problems are enormous. But rather thar acknowledge that we are just embark ing on understanding, we continue t label patients "schizophrenic," "manic depressive," and "insane," as if in thos words we had captured the essence of un derstanding. The facts of the matter ar that we have known for a long time tha diagnoses are often not useful or reliable but we have nevertheless continued t use them. We now know that we cannot distinguish insanity from sanity. It is depressing to consider how that informa tion will be used.

Not merely depressing, but frighten ing. How many people, one wonders, ar sane but not recognized as such in our psychiatric institutions? How many have been needlessly stripped of their privileges of citizenship, from the right t vote and drive to that of handling thei own accounts? How many have feigned insanity in order to avoid the criminal consequences of their behavior, and, conversely, how many would rather stand trial than live interminably in a psychiatric hospital—but are wrongly thought to be mentally ill? How many have been stigmatized by well-intentioned, but nevertheless erroneous, diagnoses? On the last point, recall again that a "type 2 error" in psychiatric diagnosis does not have the same consequences it does in medical diagnosis. A diagnosis of cancer that has been found to be in error is cause for celebration. But psychiatric diagnoses are rarely found to be in error. The label sticks, a mark of inadequacy forever.

Finally, how many patients might be "sane" outside the psychiatric hospital but seem insane in it—not because craziness resides in them, as it were, but because they are responding to a bizarre setting, one that may be unique to institutions which harbor nether people? Goffman calls the process of socialization to such institutions "mortification"—an apt metaphor that includes the processes of depersonalization that have been described here. And while it is impossible to know whether the pseudopatients' responses to these processes are characteristic of all inmates—they were after all, not real patients—it is difficult to believe that these processes of socialization to a psychiatric hospital provide useful attitudes or habits of response for living in the "real world."

SUMMARY AND CONCLUSIONS

It is clear that we cannot distinguish the sane from the insane in psychiatric hospitals. The hospital itself imposes a special environment in which the meanings of behavior can easily be misunderstood. The consequences to patients hospitalized in such an environment—the powerlessness, depersonalization, segregation, mortification, and self-labeling—seem undoubtedly countertherapeutic.

I do not, even now, understand this problem well enough to perceive solutions. But two matters seem to have some promise. The first concerns the proliferation of community mental health facilities, of crisis intervention centers, of the human potential movement, and of behavior therapies that, for all of their own problems, tend to avoid psychiatric labels, to focus on specific problems and behaviors, and to retain the individual in a relatively nonpejorative environment. Clearly, to the extent that we refrain from sending the distressed to insane places, our impressions of them are less likely to be distorted. (The risk of distorted perceptions, it seems to me, is always present, since we are much more sensitive to an individual's behaviors and verbalizations than we are to the subtle contextual stimuli that often promote them. At issue here is a matter of magnitude. And, as I have shown, the magnitude of distortion is exceedingly high in the extreme context that is a psychiatric hospital).

The second matter that might prove promising speaks to the need to increase the sensitivity of mental health workers and researchers to the *Catch*-22 position of psychiatric patients. Simply reading materials in this area will be of help to some such workers and researchers. For others, directly experiencing the impact of psychiatric hospitalization will be of enormous use. Clearly, further research into the social psychology of such total institutions will both facilitate treatment and deepen understanding.

I and the other pseudopatients in the psychiatric setting had distinctly negative reactions. We do not pretend to describe the subjective experiences of true patients. Theirs may be different from ours, particularly with the passage of time and the necessary process of adaptation to one's environment. But we can and do speak to the relatively more objective indices of treatment within the hospital. It could be a mistake, and a very unfortunate one, to consider that what happened to us derived from malice or stupidity on the part of the staff. Quite the contrary, our overwhelming impression of them was of people who really cared, who were committed and who were uncommonly intelligent. Where they failed, as they sometimes did painfully, it would be more accurate to attribute those failures to the environment in which they too, found themselves than to personal callousness. Their perceptions and behavior were controlled by the situation, rather than being motivated by a malicious disposition. In a more benign environment, one that was less attached to global diagnosis, their behaviors and judgments might have been more benign and effective.

NO

James Mauro

AND PROZAC FOR ALL . . .

The year 1993 proved a big one for Eli Lilly & Co., makers of Prozac. *Listening to Prozac,* a testimonial to the drug's healing powers, make the best-seller list, while Peter Kramer, its author, touted his tiny benefactor on various talk shows. Again and again the pill popped up in endless *New Yorker* cartoons, computer-network discussions, even David Letterman jokes. In February, the pill itself graced a cover of *Newsweek.*

Slowly, stealthily, Prozac is slithering into more and more of our lives and finding a warm place to settle.

Even the most casually aware citizen can feel the shift in thinking brought about by the drug's ability to "transform" its users: We speak of personality change, we argue over the drug's benefits over psychotherapy (all those expensive hours of parent-bashing as compared to a monthly dash to the pharmacy); and we let ourselves imagine a world in which our pain is nullified, erased as easily and fully as dirty words on a school blackboard.

Most of all, we envision a race of people both frighteningly bland and joyously healed as the ultimate double-edged sword. While Prozac may indeed be our gift horse of the decade, at least we're staring it straight in the jagged molars.

Of all the fears and concerns, the one barely spoken of but no less valid apparently has more to do with the good news than the bad: It seems the drug is *more* effective, and works to relieve *more* symptoms, than previously imagined.

Without a doubt, Prozac is exiting the realm of clinical depression and entering the murkier world of subclinical, subsyndromal, sub-"sick" disorders. Clinicians in particular are worried that the definition of "subsyndromal" disorders (psychological complaints that fail to meet the criteria for a specific illness) is expanding to include more of what were once thought of as ordinary life stresses. (The unofficial term for this is "bracket creep.")

And as this illness invitation list grows, so, of course, do the numbers of patients who now fall into this category—people somewhere short of being honest-to-God sick but who are nevertheless in some sort of pain.

Robert Trestman, M.D., director of the outpatient program at Bronx VA Medical Center, sums up the dilemma: "There are many situations where people do not meet the minimum criteria for a disorder. Where a specific diagnosis may require five criteria, for instance, some people will have only two, perhaps even one. And yet they're suffering."

And receiving psychiatric medication when once they were shipped off to a therapist's couch. Trestman neatly breaks down the dividing lines between the sick and the uncomfortable:

- Traditional patients, who say, "Doc, can you fix me? I'm hurting."
- Nontraditional patients, who say, "I'm not broken, but make me better. I want to be more assertive, I want to feel better, I want to accomplish more."

In the past, both groups would be recommended for therapy. Now, more and more are being tried on Prozac. Because of its fewer side effects and lower toxicity, the risk-to-benefit ratio is a lot lower.

"It's lower," agrees Trestman, "but it's not zero. There are side effects, risks that raise concern in the medical community."

GOOD NEWS OR BAD?

Historically, the use of drugs as fixers of the world's private ills has run into serious, if unanticipated, snags. At the turn of the century, the medical community thought that cocaine was a completely appropriate, nonaddictive drug, and widely prescribed it. In the 1950s and '60s, first barbiturates and then amphetamines were doled out for various psychological maladies. We now know that each of these drugs came with significant risks. So what yet-to-be imparted knowledge may cause scienc once again, to admit sheepishly that th exuberance over Prozac was somewh premature, if not wholly overblown?

While much remains to be learne about Prozac, so far the bad news ma be that there's no bad news. If, aft all, it does turn out to have no seriot drawbacks, what are the implications a drug that is a shortcut to healing?

It is a concern that potentially affects a psychologists, who may find themselv short of angst-laden clients in the comir years; that places dubious power i the hands of primary care physician who may prescribe the drug without fully articulated understanding of the patients' distress; and that strikes a chor of defensive fervor in the hearts an minds of everyone raised with the Judec Christian ethics—that nothing in life ca be worthwhile, or effective, unless yo *work* for it.

There's more to the story. Questior abound regarding the drug and its chem ical cousins, Zoloft and Paxil: What othe types of disorders, aside from clinical di pression, are they being prescribed for Do the medications work? What other op tions exist? What are the potential risks t individuals and to society?

I'M DYSTHYMIC, YOU'RE DYSTHYMIC

Of all the distresses, ailments, and infir mities patients complain of nowaday perhaps none is so broad or so muddl in definition as "dysthymia"—a chroni discontent involving either depressio (but not *clinical* depression) or irritabi ity. Its symptoms—not eating or eatin too much, not sleeping or oversleepin poor concentration or difficulty makin decisions—reveals the unexclusivity c

its rank and file. In terms of requirements for diagnosis, dysthymia may be the only club that would have Groucho Marx for a member.

According to a recent survey, approximately 48 percent of Americans—almost half the population—has experienced some form of dysthymic disorder. And all of them may qualify for Prozac. Robert Millman, M.D., professor of psychiatry and public health at Cornell, sees the irony of it: "There's *nobody* nonsyndromal. You can give Prozac to anyone you want."

Which is anathema to what medical science is supposed to be about. "We try to convince people there's some specificity to what we do," says Millman. "But this is embarrassing."

And the list doesn't stop there. Simon Sobo, M.D., director of psychiatry at New Milford Hospital, reports that "Prozac has been successfully used for obsessive hair-pulling, panic disorder, eating disorders, and social and other phobias. It has proven useful to people to free themselves from addictive relationships; to dispel doubts about performance; to overcome obstacles that once seemed impossible. I have even added it to my watering can and found geraniums grow better on it."

He's joking, of course, but only about the geraniums. Add obesity, gambling addiction, and PMS to the spectrum of complaints now being helped by Prozac.

BETTER RECEPTION?

If little is known yet of just how effective these drugs are for psychological distress, even less clear is the actual impact they have on those who benefit from them. Are they simply mood brighteners or are they re-regulating systems that are out of balance? Do they actually *change* personality, making you feel better than normal, or merely fine-tune it? Do people say, "Gee, I feel more myself on this drug" or "Gee, I'm a different person now"?

Some clinicians, such as Larry Siever, M.D., director of the Outpatient Psychiatry Division at Mt. Sinai School of Medicine in New York, offer an opinion between the two: "If you have a staticky, bland picture on your TV set, you can fix the reception by adjusting the tuning and contrast. Or simply change the channel. My understanding of the medications personally is more the former than the latter."

Of course the big fear surrounding the "channel-changing" aspect of the drugs is that society will evolve into a battalion of "happy soldiers." Exhumed by Kramer himself, the specter of Aldous Huxley's soma—*Brave New World's* fictional drug that anesthetized citizens into a content unawareness—continues to haunt us and cloud the argument surrounding Prozac. Yet to many, the analogy seems false.

"The drugs, if properly used," says Siever, "shouldn't dampen normal signals of anxiety, not even normal depression. It should not snow under in the way that a hypnotic does a person's normal level of arousal or awareness, but should allow all of these signals to emerge more clearly."

And, he continues, extending the argument, "If depression or other symptoms emerge, whether from psychological or social stresses, aren't people entitled to treatment for these conditions, just as they would get if they had an ulcer in relation to the stresses in their lives?"

Siever's example inadvertently reveals yet another controversy surrounding the use of drugs—any drugs—in fighting these disorders: the contention that pharmacology focuses on the individual rather than examining the larger societal problems that lie behind depression and other ills. Epidemiological studies have shown that more people are suffering from major depression that ever before—at ever-younger ages. Prozac, some argue, puts a Band-Aid on individual symptoms rather than addressing why people are seeking help in ever-increasing numbers.

The response of clinicians is to answer the question with a question: Why must one solution preclude any other?

The fundamental error, they argue, is to assume that the use of Prozac as a therapeutic tool equals an interest only in the biological causes of depression (or sub-depression, or just plain old feeling lousy). Those who can prescribe medication are, by profession, at least partly invested in biological *solutions*. And since Prozac is usually recommended along with some form of psychotherapy, the conclusion that interpersonal relationships are somehow ignored—or that individual brain chemistry is the one and only root being addressed—seems erroneous.

"We're not saying this is the *only* way to help," insists Trestman. "We're saying, 'This is one way, but of course there are others.' Many more people can be helped by changing the structure of society than through medicine. But we also have the ability apparently to help many more people with medication than before. Now we have to figure out should we? And for whom? And where does it stop?"

OUT OF THE WOODWORK

Other concerns stem from the staggering numbers of people for whom Prozac would prove beneficial. In 1991, this

advertisement appeared in New York *Times* and *Village Voice*.

"ARE YOU DEPRESSED? DO YOU SU FFER FROM FATIGUE? INABILITY TO CONCE TRATE? HAVE TROUBLE SLEEPING OR EA ING? IF SO, CONTACT..."

The ad was placed to gather subjec for a study of the effectiveness of Proz in treating dysthymia. The respons according to researcher Jesse Rosenth M.D., Director of psychopharmacology Beth Israel Medical Center in New Yor was "literally thousands of phone calls. was amazing—all these bright, educate hardworking people just came out of th woodwork. We found a mother lode nice people who were able to functio but who were quite literally the walkin wounded of New York."

After selecting a core group who m the criteria for dysthymia, Rosenthal an his team divided them up and gave on half Prozac, the other half a placeb Results? An astonishing 62 percent the Prozac group showed significar improvement after only eight weeks opposed to 18 percent given placebos Other studies conducted by Rosenth have shown a more than 70 percer success rate.

The number of people who responde to the advertisement is evidence widespread, if low-level, depression– and in greater numbers than were pre viously imagined. But what struck Ros enthal was that, while their averag age was 36, almost 80 percent of them were single, and another 9 percent wer divorced. Nearly 90 percent of them ha been in therapy on and off over the years

"They had a lot of insight," report Rosenthal. "But they still had symp toms."

Which begs the question: Were thes people dysthymic (read "unhappy") be

cause they couldn't get themselves involved romantically, or were their persistent blues preventing them from successfully interacting with others?

The distinction is an important one, and crucial in the argument of a "drugs vs. societal change" approach to combating low-level depression. Romantic courtship may be more difficult now than ever before—which may lead many to remain single and unhappy. If so, working toward easier social interaction would benefit. If, however, the reverse were true, and the subjects' dysthymia was what prevented them from dating, then focusing on the individual—*in order to correct the social*—seems justified. "And that focus is not to be dismissed," stresses Trestman.

DOES PROZAC = LEARNING?

Whatever the root, one can see them, sipping Cranzac (Prozac and cranberry juice—a popular cocktail for those unable to tolerate full doses of the drug), nuzzling up to potential mates at the local singles' bar, smiling, their psychological wounds successfully sutured. Given time, wouldn't a more positive outlook lead to better interactions, and the potential relationships that developed continue to promote good cheer once Prozac is tapered off?

"Of course," agrees Trestman. "If people start responding differently to you, and you start feeling different about yourself, you set up new habit patterns that reinforce your changed state of affairs. It may be that Prozac resets the adjustment in the brain after a number of months, and that afterward people would be at this new point and could taper off without relapse."

In other words, first the drugs make you better, happier, more in control—then you do the rest of the work on your own. Cornell's Robert Millman concurs: "The drugs change a person's emotional reward system. Your sense of acceptance increases. Your feeling state is changed. Then hopefully you take this new ammunition and go out and use it on your own."

Wait a minute. What are we saying here? That "real learning" occurs on Prozac? That the drug does not simply solve your problems medically, but requires you to do half the legwork yourself? Yes, believes Millman, "So that even when you take away the medication, the same situation in life may create different responses in an individual. Where once the thought of initiating romance seemed too stressful, it now seems possible. Where once life seemed sad, lonely, and defeating, it now appears worthwhile and conquerable."

SYNDROME VS. CHARACTER

Still, there are fears. Is Prozac bringing to light the frightening number of people who suffer from some sort of distress? Or is it that what were once called "character traits" are now being reclassified as "syndromes"—because they can be smoothed out by medication? And, if such a trend continues, will there be anyone left who *isn't* "disordered"? Who *doesn't* need drugs?

Some doctors bristle at the distinction between syndrome and character. "It's a false and meaningless boundary," insists Steven Roose, M.D., of Columbia University. "People implicitly cross the border from, well, it's a syndrome, that means there's something wrong with the brain, to, well, that's just their character, their personality, so that's psychology."

Such dualism is destructive, believes Roose: "If somebody has a bad temper and works to control it, we don't say they're altering who they are. But there's a paranoia that somehow with medication, we're trying to control the essence of individuality, that we're manipulating someone."

No doubt the moral arguments about character altering are being applied more severely when treatment involves medication as opposed to psychotherapy. Consider one recent *New Yorker* cartoon: "If they had Prozac in the 19th century." One panel features Karl Marx saying, "Sure, capitalism could work out its kinks!" In another, Edgar Allen Poe is on friendly terms with the raven. A third shows Nietzsche outside a church with his mother, saying, "Gee, Mom, I like what the priest said about the little people."

The implicit message is that, without suffering, without the character quirks that made Poe poetic, for example, we would be deprived of his brooding masterworks. True, perhaps, but if suffering is so enlightening, if it is part of what makes us "us" and we should try our best to preserve it rather than medicate it away—isn't that also an argument against *any* kind of treatment? Shouldn't we then avoid seeking *any* kind of relief, for fear that we may be damaging, even destroying, the human spirit, the creative urge, that which defines all of us, the brilliant and the dullard?

"The notion that suffering is good is paternalistic and, at worst, sadistic," says Roose. But even if we take that moralistic, almost religious view, why point our swords only at the dragons marked "take as directed"? Why not apply the same questions and concerns to p chotherapy? "The use of psychother in this country has been grandfathe in," points out Bob Trestman. "It's b accepted already for many years, firs terms of counseling from religious le ers, and more recently in the prac of formal therapy. So that we no lor question either its intrusiveness on v we are or its relative safety."

Does psychotherapy have side effe Is it intrusive? Does it change essence of who we are? The answe yes to all. "If psychotherapy coul manipulate or effect change, ther wouldn't work," states Roose. "The i that therapy isn't intrusive, that don't alter behavior or control peop thoughts is fundamentally untrue."

What about side effects? "By def tion, if a treatment is powerful enou to work, it's powerful enough to h adverse effects. Every journal on p chotherapy will talk about people v regress in treatment, people who h psychotic reactions, people for wh therapy has caused deterioration rat than progress.

"Still," Roose continues, "beca these so-called nonsyndromal disord are considered to be in the realm of p chology, we don't think there's anyth wrong in treating them with psychoth apy. We believe that *isn't* manipulat while medication is—regardless of c come."

MANIPULATION VS. CHANGE

Yet what if the brain reacted, readjus itself in the same way, whether response to a pill or a therapeu directive?

Last year, in the *Archives of Gen Psychiatry*, a research team headed

UCLA's Lewis Baxter, M.D., reported a study of two groups suffering from obsessive-compulsive disorder (recurrent, unwanted thoughts accompanied by ritualized acts, such as excessive handwashing). In treatment, one group was given Prozac with no formal therapy, the other behavior therapy, in the form of exercises designed to prevent their compulsiveness, with no drugs. After 10 weeks, scans of their brains were compared with those taken at the beginning of treatment.

Approximately two-thirds of each group improved. More important, for those who did improve, rates of glucose metabolism (an indicator of brain activity) decreased in *exactly* the same areas of the brain, in statistically similar amounts, regardless of treatment. The behavioral techniques actually altered brain function —and did so no differently, no less intrusively, than Prozac.

"Some may wonder," writes Baxter et al, "how behavior therapy could produce brain-function changes similar to drugs. [But] the possibility of both having the same neural effects is not as farfetched as it might seem."

The brain is the organ of the mind, and its function affects personality. So how far do we go in treating its disorders and distresses, its syndromes and its character flaws? By all accounts, the resounding answer seems to be: as far as it is safe to go. The unanimous opinion among professionals is that more information is needed.

Yet what about the concern that we are entering an age when even the slightest wrinkle in character can be defined as a "disorder." Will we become a Prozac nation? Hardly, thinks Robert Millman, who does not believe the whole of society is going to became dependent upon these drugs. The reason? Evolution, which, over the course of time, has created in us the brain functions that dictate the way we deal with thoughts and emotions. That intricate interplay, he offers, is way beyond the primitive effects of any of these drugs.

"The system is so refined," believes Millman, "and drugs are so primitive, that one can never really replace the other. With drugs, you're always giving away more than you're getting—if you're not really debilitated. You're giving away sensitivity, receptivity, some capacity for pleasure. But it's a reasonable trade-off if you're in pain."

The only question, then, is for what degree of pain do we seek medical treatment. And, as Bob Trestman puts it, where will it end?

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Words

A proposal to classify happiness as a psychiatric disorder

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Author's abstract

It is proposed that happiness be classified as a psychiatric disorder and be included in future editions of the major diagnostic manuals under the new name: major affective disorder, pleasant type. *In a review of the relevant literature it is shown that happiness is statistically abnormal, consists of a discrete cluster of symptoms, is associated with a range of cognitive abnormalities, and probably reflects the abnormal functioning of the central nervous system. One possible objection to this proposal remains – that happiness is nor negatively valued. However, this objection is dismissed as scientifically irrelevant.*

Introduction

Happiness is a phenomenon that has received very little attention from psychopathologists, perhaps because it is not normally regarded as a cause for therapeutic concern. For this reason, research on the topic of happiness has been rather limited and any statement of existing knowledge about the phenomenon must therefore be supplemented by uncontrolled clinical observation. Nonetheless, I will argue that there is a *prima facie* case for classifying happiness as a psychiatric disorder, suitable for inclusion in future revisions of diagnostic manuals such as the American Psychiatric Association's Diagnostic and Statistical Manual or the World Health Organisation's International Classification of Diseases. I am aware that this proposal is counter-intuitive and likely to be resisted by the psychological and psychiatric community. However, such resistance will have to explain the relative security of happiness as a psychiatric disorder as compared with less secure, though established conditions such as schizophrenia. In anticipation of the likely resistance to my proposal I will therefore prefacemy arguments with a brief review of the existing scientific literature on happiness. Much of the following account is based on the work of Argyle (1).

It is perhaps premature to attempt an exact definition of happiness. However, despite the fact that formal diagnostic criteria have yet to be agreed, it seems likely that happiness has affective, cognitive and behavioural components. Thus, happiness is usually characterised by a positive mood, sometimes described as 'elation' or 'joy', aithough this may be relatively absent in the milder happy states, sometimes termed 'contentment'. Argyle, in his review of the relevant empirical literature, focuses more on the cognitive components of happiness, which he describes in terms of a general satisfaction with specific areas of life such as relationships and work, and also in terms of the happy person's belief in his or her own competence and self-efficacy. The behavioural components of happiness are less easily characterised but particular facial expressions such as 'smiling' have been noted; interestingly there is evidence that these expressions are common across cultures, which suggests that they may be biological in origin (2). Uncontrolled observations, such as those found in plays and novels, suggest that happy people are often carefree, impulsive and unpredictable in their actions. Certain kinds of social behaviour have also been reported to accompany happiness, including a high frequency of recreational interpersonal contacts, and prosocial actions towards others identified as less happy (3). This latter observation may help to explain the persistence of happiness despite its debilitating consequences (to be described below): happy people seem to wish to force their condition on their unhappy companions and relatives. In the absence of well-established physiological markers of happiness, it seems likely that the subjective mood state will continue to be the most widely recognised indicator of the condition. Indeed, Argyle has remarked that 'If people say they are happy then they *are* happy' (4). In this regard, the rules for identifying happiness are remarkably similar to those used by psychiatrists to identify many other disorders, for example depression.

The epidemiology of happiness has hardly been researched. Although it seems likely that happiness is a relatively rare phenomenon, exact incidence rates must depend on the criteria for happiness employed in any particular survey. (In this respect happiness is also not unique: similar problems have been encountered when attempts have been made to investigate the

Key words

Happiness; major affective disorders; psychiatry.

epidemiology of other psychiatric disorders such as schizophrenia (5)). Thus, although Warr and Payne (6) found that as many as 25 per cent of a British sample said that they were 'very pleased with things yesterday', Andrews and Withey (7), studying a large US sample, found that only 5.5 per cent of their subjects rated themselves as scoring maximum on a nine-point scale of life-satisfaction. One problem with these kinds of data is that they have been generated in the absence of good operational criteria for happiness and have focused on the cognitive components of the condition (perhaps because these are comparatively easy to measure) rather than the affective and behavioural components. It is therefore quite possible that informal observation is a better guide to the prevalence of happiness in community samples. Certainly, if television soap operas in any way reflect real life, happiness is a very rare phenomenon indeed in places as far apart as Manchester, the East End of London and Australia. Interestingly, despite all the uncertainty about the epidemiology of happiness, there is some evidence that it is unevenly distributed amongst the social classes: individuals in the higher socio-economic groupings generally report greater positive affect (8) which may reflect the fact that they are more frequently exposed to environmental risk-factors for happiness.

Further light might be shed on the nature of happiness by considering its aetiology. Although the cause or causes of happiness have yet to be identified aetiological theories have implicated both environmental and biological factors. With respect to the environment, there seems little doubt that discrete episodes of happiness typically follow positive life-events (9). However, the observation that some people are generally happier than others suggests that less transient factors may also play an important role. While it has been suggested that a general disposition towards happiness is related to self-esteem (10) and social skills (1), two variables which presumably reflect early learning experiences, the finding that extroversion is a good predictor of happiness even years in the future (11) suggests that biological factors may be implicated.

Evidence that happiness is related to cognitive abnormalities will be outlined below when I discuss the proposition that happiness is irrational. Genetic studies of happiness are a neglected avenue of research but neurophysiological evidence points to the involvement of certain brain centres and biochemical systems. Thus, stimulation of various brain regions has been found to elicit the affective and behavioural components of happiness in animals (12) as has the administration of drugs which affect the central nervous system such as amphetamine and alcohol (13). Taking the environmental and biological evidence together it may be necessary to discriminate between various different types of happiness. Thus, it may be useful to distinguish between *reactive* happiness, usually manifesting itself as an acute episode followed by a rapid remission of symptoms, and endogenous happiness which may have a relatively chronic onset and which may be less often followed by symptomatic improvement. The differential diagnosis of these two types of happiness is an obvious project for future studies. Given the apparent similarities between happiness and depression, it seems possible that endogenous happiness will be characterised by positive mood first thing in the morning, a heavy appetite, and persistent erotomania.

Happiness as a psychiatric disease

Since the emergence of the profession of psychiatry in the nineteenth century it has commonly been assumed that psychiatric disorders are forms of disease. Whilst this assumption has not gone unchallenged in recent years (14) it remains so pervasive within the mental health professions that the demonstration that happiness qualifies as a disease would be a powerful argument for including it within future nosologies of psychiatric disorder.

Historically, there have been two approaches towards the definition of disease (15). The first, which is best exemplified by the work of the doctor Thomas Sydenham in the eighteenth century, involves the identification of syndromes consisting of clusters of symptoms thar occur together. The second, which is best exemplified by the later work of Virchow, involves the identification of a pathological process that is causally implicated in a disturbance of body or behaviour. In practice, medical scientists usually hope thar the two types of classification will converge to enable the generation of causal models of disease. However, for most psychiatric disorders this prospect lies somewhere in the future (16). For this reason, when considering the evidence that happiness is a disease, it will be useful to bear in mind for comparison the evidence pertaining to the disease status of recognised psychiatric disorders such as schizophrenia.

The question of whether or not it is possible to identify a meaningful syndrome of happiness has been the subject of only very limited research. According to Argyle (1), most investigators agree that happiness is best thought of as a dimension of affect, rather than as a discrete category of emotional disequilibrium: in this respect at least happiness appears to be similar to both schizophrenia and perhaps the majority of psychiatric disorders (17). However, the relationship between the dimension of happiness and other affective dimensions remains unclear. Thus, in a factor-analytic investigation (8) it was observed that reports of happiness and reports of negatively valued affective states loaded on separate factors, suggesting that they are independent of each other. Interestingly, people who report high-intensities of happiness also report high intensities of other emotions (18), which might be regarded as evidence for the hypothesis (to be discussed below) that happiness is related to a neurophysiological state of disinhibirion. Nonetheless,

the *frequencies* with which people report happiness and the negatively valued affective states appear to be negatively correlated (19). Some confusion also exists about the relationship between happiness and the psychiatric disorder of mania; although it might be expected that these are related conditions Argyle (1) has noted that mania, in contrast to happiness, is mainly characterised by excitement. Nonetheless, the diagnostic criteria for hypomanic episodes employed by the American Psychiatric Association (20) seem to allow happiness to be regarded as a subtype of hypomania. Taking all this evidence together, it might be argued that there is only modest empirical support for the notion of a discrete happiness syndrome. On the other hand, the evidence is really quire favourable when compared with the evidence supporting other widely accepted psychiatric syndromes such as schizophrenia (21).

Some evidence that happiness is related to a disturbance of the central nervous system has already been noted. Just as it is possible to elicit schizophrenic symptoms in some individuals by stimulating the parietal lobes, so too it is possible to produce happiness by brain stimulation, though of subcortical centres (12). Cortical centres also seem to be implicated however, as both left hemisphere seizures and right hemispherrctomy have been associated with prolonged euphoric states; indeed it has been suggested that emotional states in general are regulated by a complex balance of excitatory and inhibitory centres in both hemispheres, and that abnormal affective states of any kind reflect a disturbance of this balance (22). Clearly, further biological research is needed to specify in any detail the role of neuropsychological abnormalities in happiness but a promising start has been made, and quite a clear picture is apparent in comparison to the mixed results of nearly one hundred years of research into schizophrenia (21).

Indeed, it is the lack of progress in identifying a biological pathology for schizophrenia and other psychiatric disorders that has led someauthors to reject the notion that schizophrenia is a disease (14) and others to argue that the criteria for disease should not require the identification of an underlying biological pathology (23). Clearly, if, as I have argued, happiness meets the narrower criteria for disease employed in physical medicine it is also likely to meet any such broader criteria advocated for psvchiatry. For example, it has been suggested that, for the purposes of psychiatric research, a disease be simply regarded as any deviation from the norm by way of excess or deficit which confers upon the sufferer some form of biological disadvantage (24). Evidence that happiness is statistically abnormal has already been discussed and, despite the lack of clear data, there is at least some reason to suppose that happiness confers a biological disadvantage, at least in the short term. Consistent clinical evidence of an association between happiness, obesity and indulgence in alcoholic beverages has existed from before the time of scientific medicine (Julius Caesar, for example, is reputed to have asked for the company of fat men on these grounds (24)). Given the well-established link between both alcohol and obesity and life-threatening illnesses it seems reasonable to assume that happiness poses a moderate risk to life. The common observation that happiness leads to impulsive behaviour is a further cause for concern.

More clear evidence that happiness confers a biological disadvantage can be discerned from the literature relating various cognitive measures to mood state, but before discussing this evidence it will first be useful to consider the proposition, advocated by some philosophers, that *irrationality* rather than disease be considered the criterion for psychiatric disorder.

Happiness, irrationality and cognition

Mainly because of persistingdoubts about the value of applying the concept of disease to psychiatric disorders, a number of philosophers have suggested that the quality of *rationality* is the most appropriate criterion for distinguishing between such disorders and types of behaviour and experience not worthy of psychiatric attention. According to Radden (26), behaviour may be described as irrational if it is bizarre and socially unacceptable, reduces the individual's expected utilities, or is not grounded on good (ie logically consistent and acceptable) reasons; in the latter case, in particular, Radden believes that the behaviour should be the subject of psychiatric scrutiny. A similar view has been taken by Edwards (27) who claims that *bona fide* cases of psychiatric disorder are characterised by actions that fail to realise manifest goals, thinking that is illogical and replete with contradictions, beliefs that should be falsified by experience, the inability to give reasons for actions, unintelligible or nonsensical thinking, and a lack of impartiality and fairrnindedness.

Some definitions of irrationality clearly make more sense than others. Bizarreness and social disapproval are weak criteria for irrationality because they are culturally constrained and difficult to apply with any consistency: the Lancastrian's predilection for dried pig's blood may seem bizarre to the Hotentot, who prefers to eat slugs. Against this, some authors have argued that delusional beliefs should be tested against their cultural background, although this has the disadvantage of allowing totalitarian regimes to diagnose political dissidents as insane (28).

In testing whether or not happiness is irrational it may therefore be safer to fall back on the other approaches to defining irrationality outlined by Radden and Edwards. Thus, although there is a lack of relevant data, it seems reasonable to assume that happiness often results in actions which fail to realise manifest goals, and which therefore decrease the happy person's expected utilities. The potentially life-threatening consequences of happiness have already been discussed. In addition, happy people may experience great difficulties when faced with mundane

but essential tasks.

Both Radden and Edwards imply that irrationality may be demonstrated by the detection of cognitive deficits and distortions of one sort or another. There is excellent experimental evidence that happy people are irrational in this sense. It has been shown that happy people, in comparison with people who are miserable or depressed, are impaired when retrieving negative events from long-term memory (29). Happy people have also been shown to exhibit various biases of judgement that prevent them from acquiring a realistic understanding of their physical and social environment. Thus, there is consistent evidence that happy people overestimate their control over environmental events (often to the point of perceiving completely random events as subject to their will), give unrealistically positive evaluations of their own achievements, believe that others share their unrealistic opinions about themselves, and show a general lack of' evenhandedness when comparing themselves to others (30). Although the lack of these biases in depressed people has led many psychiatric researchers to focus their attention on what has come to be known as *depressive realism* it is the unrealism of happy people that is more noteworthy, and surely clear evidence that such people should be regarded as psychiatrically disordered.

Possible objections

I have argued that happiness meets all reasonable criteria for a psychiatric disorder. It is statistically abnormal, consists of a discrete cluster of symptoms, there is at least some evidence that it reflects the abnormal functioning of the central nervous system, and it is associated with various cognitive abnormalities – in particular, a lack of contact with reality. Acceptance of these arguments leads to the obvious conclusion that happiness should be included in future taxonomies of mental illness, probably as a form of affective disorder. This would place it on Axis I of the American Psychiatric Association's Diagnostic and Statistical Manual (20). With this prospect in mind, I humbly suggest that the ordinary language term 'happiness' be replaced by the more formal description *major affective disorder, pleasant type*, in the interests of scientific precision and in the hope of reducing any possible diagnostic ambiguities.

There are two possible objections to the proposed inclusion of major affective disorder, pleasant type, as a psychiatric disorder. First, it might be argued that happiness is nor normally a cause for therapeutic concern. Therapeutic concern has in fact been proposed as a criterion for disease by Kraupl-Taylor (31) because of the difficulties of formulating a less arbitrary criterion. However, Kendell (15) has criticised this definition as worse than no definition at all because of its obvious circularity and because of the inevitable implication that diseases are culturally and historically relative phenomena. On this account, sickle-cell anaemia, anorexia nervosa and psychopathy (to name but three unequivocal examples of disease described only in recent times) were not diseases before their discovery. In any event, once the debilitating consequences of happiness become widely recognised it is likely that psychiatrists will begin to devise treatments for the condition and we can expect the emergence of happiness clinics and anti-happiness medications in the not too distant future.

The second, related objection to the proposal that happiness be regarded as a psychiatric disorder points to the fact that happiness is not normally negatively valued. Indeed, it is testimony to the insidious effects of happiness on some of the greatest minds in history that some philosophers have argued that the pursuit of happiness is the ultimate aim of all human endeavours. However, it is notable that even some of those who have been rash enough to advocate the greatest happiness for the greatest number have been explicit in rejecting those extreme forms of happiness associated with gluttony of the senses (32). More importantly, the argument that happiness be excluded from future classifications of mental disorder merely on the grounds that it is not negatively valued carries the implication that value judgements should determine our approach to psychiatric classification. Such a suggestion is clearly inimical to the spirit of psychopathology considered as a natural science. Indeed, only a psychopathology that openly declares the relevance of values to classification could persist in excluding happiness from the psychiatric disorders.

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The verbal information pathway to fear and heart rate changes in children

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Background: Although many studies have now demonstrated that threat information is sufficient to change children's beliefs and behaviours towards novel animals, there is no evidence to suggest that it influences the physiological component of the fear emotion. **Methods:** An experiment is reported in which children ($N = 26$) aged between 6 and 9 were given threat, positive or no information about three novel animals and then asked to place their hands into boxes that they believed to contain each of these animals. Their average heart rate during each approach task was measured. **Results:** One-way analysis of variance revealed significant differences in the average heart rate when approaching the three boxes: heart rates were significantly higher when approaching the box containing the animal associated with threat information compared to when approaching the control animal. **Conclusions:** These findings suggest that fear information acts not only upon cognitive and behavioural aspects of the fear emotion, but also on the physiological component. **Keywords:** Anxiety, conditioning, fears, information processing.

Despite the fact that anxiety disorders typically originate in childhood (Öst & Treffers, 2001), research on theories of anxiety are still very adult-focused and treatments aimed at children are virtually non-existent (Cartwright-Hatton, Roberts, Chitsabesan, Fothergill, & Harrington, 2004). Children are likely to acquire fears in a variety of ways and the variance in childhood anxiety appears to be attributable to shared (11%) and non-shared (37%) environments, with genetic factors explaining the remaining variance (Eley et al., 2003). One of the challenges facing us is to identify these environmental factors such that theories of fear acquisition and preventions and treatments based on these theories can be better informed.

Rachman proposed three possible pathways through which environmental factors might lead to fear (Rachman, 1977): (1) direct experience (and association) with a traumatic event; (2) vicarious learning, in which a stimulus comes to evoke fear through observing another's fear of that stimulus; and (3) the transmission of information, in which a stimulus comes to evoke fear through verbal communication about the possible threat arising from the stimulus. These pathways are not mutually exclusive. For example, verbal information is likely to have an impact on the strength of an association between a stimulus and an aversive outcome in future direct conditioning experiences (Davey, 1997; Mineka & Zinbarg, 2006).

Reviews of the literature have concluded that there is good evidence for all three of Rachman's pathways (King, Gullone, & Ollendick, 1998; Merckelbach, deJong, Muris, & van den Hout, 1996); and some have concluded that verbal information is the most important of these pathways (Muris, Merckelbach, Gadet, & Moulaert, 2000; Ollendick & King, 1991) and is the main pathway through which anxious parents transmit fears to their children (Hadwin, Garner, & Perez-Olivas, 2006). However, most of this evidence has been based on retrospective reports from adult phobics and so is limited in what it can tell us about the causal influences on the development of fear (see Field et al., 2001; King et al., 1998 for more detailed critiques).

Recent attempts to explore the causal influence of verbal information on fear have manipulated the information given to children about novel animals (Field et al., 2001). Field and Lawson (2003) gave threat, positive or no information to children about three Australian marsupials (the quoll, quokka and cuscus), which were unfamiliar to children in the UK. They found that threat information significantly increased children's fear beliefs as indexed by both self-report and indirect measures (the implicit association task). In addition, children showed increased reluctance to approach a box inside which the animal was believed to be. The same paradigm has been used to demonstrate the causal influence of threat information on several aspects of children's fear cognitions. Field, Lawson, and Banerjee (submitted) have shown that both directly and indirectly measured fear beliefs can last beyond the immediate experimental manipulation (Muris, Bodden, Merckelbach, Ollendick, & King, 2003) and up to 6 months. Threat information is also sufficient to induce an attentional bias towards an animal (Field, 2006c), similar to the kinds of bias seen in adult phobics (Mogg & Bradley, 2002). This paradigm has also been used to look at the interactional effects of threat information and other variables: behavioural inhibition system sensitivity or trait anxiety

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facilitates behavioural avoidance and attentional bias following threat information (Field, 2006a) and the effects of threat information were more potent when coming from an adult compared to a peer (Field, Argyris, & Knowles, 2001).

The evidence has shown changes in cognitions and behaviours associated with the fear emotion. However, to date, there is no evidence that verbal information will affect physiological responses to fear. This is not a trivial lacuna in our knowledge given the frequent observation that fear cognitions, behavioural avoidance and physiological responses do not correlate (Hodgson & Rachman, 1974; Lang, Melamed, & Hart, 1970; Rachman & Hodgson, 1974; Zinbarg, 1998). Based on this desynchrony in measures of anxiety, Lang (1968) proposed a model of emotion in which an emotion consists of three response systems: (1) subjective states and cognitions associated with those states (verbal-cognitive responses); (2) behavioural changes; and (3) physiological states. More recently, Zinbarg (1998) has proposed a hierarchical model of anxiety in which he suggests that anxiety is a higher-order unitary construct but is multidimensional at lower levels of the hierarchy. As such, cognition, behavioural and physiological responses can be thought of as indicator variables for a latent variable 'anxiety'. This model usefully explains the desynchrony between different measures of fear by suggesting that factors other than the higher-order construct of anxiety will influence these response systems differentially, and that in a given situation the influence of these external variables on different response systems will vary. As such, it cannot be assumed that because verbal information affects fear cognitions and avoidance behaviour, it will also affect physiological responses: desychrony should be high when emotional responses are relatively mild, as you might expect after verbal information, and synchrony can be expected only when emotional arousal is strong (Hodgson & Rachman, 1974). Regardless of whether you adopt a multidimensional or hierarchical conceptualisation of the fear emotion, it would be a powerful addition to our understanding of the causal role that verbal information plays in fear acquisition to show that it influences physiological responses.

Heart rate is a physiological response that shows a good correspondence to self-reported fear (Lang, Melamed, & Hart, 1970; Sartory, Rachman, & Grey, 1977): for example, heart rate increases show an on–off correspondence with the presence or absence of a phobic stimulus (Lang, 1971), and animal phobics' heart rates increase linearly as their proximity to their fear-eliciting animal increases (Sartory, Rachman, & Grey, 1977) and when confronted with imagery of their phobic stimuli compared to control imagery (McNeil, Vrana, Melamed, Cuthbert, & Lang, 1993). Although barely any research has looked at children's heart rate responses to phobic stimuli, children aged 6–17 did show increased heart rates to a mildly phobic stimulus: a videotape of a large dog (Weems, Zakem, Costa, Cannon, & Watts, 2005). As such, if verbal information is having an effect on the physiological component of the fear emotion, then there should be corresponding changes in children's heart rate when confronted with animals about which they have heard threat information. The aim of this experiment was, therefore, to look at the effect of the verbal information pathway on the physiological response system of fear. It was predicted that threat information would lead to increased heart rates during what children believed to be an encounter with a novel animal.

Method

Participants

The participants were 26 primary school children (11 male, 15 female) aged 6–9 years ($M = 8.0$ years, $SD = 1.56$). This age range was selected because normative fears are focused on animals during this developmental period (Field & Davey, 2001). Opt-in parental consent was obtained before the study began. The children were given the information alone but completed parts of the behavioural task in pairs.

Stimulus materials

Animals. Pictures of three Australian marsupials, the Quoll, the Cuscus and the Quokka, were used. These were animals about which the children had no prior experience and so they would have no prior fear expectations.

Information. The two sets of information (one threat, one positive), matched for length and word frequency, used by (Field, 2006a, 2006c; Field & Lawson, 2003) were used.

Touch boxes. Avoidance was assessed with a behavioural task used by Field and Lawson (2003) and Field et al. (submitted). For each animal, a touch box was created consisting of a large wooden box, with a round hole at one end and a plaque showing the name of its animal inhabitant. A Hessian curtain covered the hole, with a slit in the middle such that the child could put their hand into the box but could not see what the box contained. Each box contained a furry cuddly toy at the back.

Heart rate. It was not pragmatically possibly to take bulky laboratory equipment for measuring heart rate into the school and instead a portable device was used: a 2003 610i POLAR heart rate monitor. This device consisted of an elastic belt with two plastic sensors that went around the child's chest and measured their heart rate. A wristwatch connected to the elastic belt measured data. The watch was set up to record the average heart rate over a 15 second period.

Procedure

The children were randomly assigned to one of three counterbalancing orders that differed with respect to which animal was associated with which type of information. (Order 1: quoll (threat), cuscus (positive), quokka (none); Order 2: quokka (threat), quoll (positive), cuscus (none); Order 3: cuscus (threat), quokka (positive), quoll (none)).

First, the children were shown the three pictures of the Australian animals. They were told the animals' names, and then the pictures were placed where the child could see them all clearly. The child was then told the information about the animals by the female experimenter. The animal associated with a particular type of information depended on the counterbalancing group to which the child was assigned, and within each of these groups half of the children heard the threat information before the positive and vice versa for the remaining children.

After the information, a behavioural task was administered to assess the children's heart rate as an index of their fear. To ensure parents were happy with the procedure, the experimenter did not fit the chest belt to the children. Instead, children were put in same-sex pairs and the experimenter explained to both children how to put the belt on. One child then fitted the belt on the other and stood behind a screen. (Likewise, when the first child had completed the touch-box task, they fitted the belt onto the second child and stood behind the screen.)

The touch-box task itself began with the experimenter checking that the belt was fitted properly and that data were being collected. The three touch boxes were placed side by side on a table, with one metre of space between them, as Cuscus, Quokka, Quoll, respectively. A line was marked out with tape 1 metre in front of the boxes, which was the starting point for each approach task. Each child stood at the line for 15 seconds, after which they were told to approach the first box. The children were asked to stroke the first animal and were given 15 s to place their hand into the box. When 15 s had elapsed the children were asked to return to the starting line. The mean heart rate during this 15 s period was recorded. The child remained at the starting line for 15 s before being asked to stroke the second animal. Again, they were given 15 s to complete the task and the average heart rate during this period was recorded. Children returned to the starting line for 15 s before approaching the final box.

All children placed their hands into all boxes within the 15 s limit and all children were fully debriefed at the end of the experiment using fact sheets and puzzles about the animals.

Results

Figure 1 shows the mean heart rate taken during the 15 s approach to each of the boxes containing the animals associated with threat, positive or no information. The children's heart rate was, on average, highest when approaching the box containing the animal associated with the threat information, and lowest when placing their hand in the box containing the animal associated with positive information. A 3 (type of information: threat, positive, none), one-way repeated measures ANOVA was conducted on the data. There was a significant main effect of the type of information, $F(2, 50) = 6.05$, $p < .05$. Contrasts showed that heart rates were significantly higher when approaching the box containing the animal associated with threat information compared to the box containing the control animal (no information), $F(1, 25) = 5.51$, $p < .05$, and this was a medium to large effect by Cohen's (1992) criteria, $r = .42$. However, the average heart rate when approaching the box containing the animal associated with positive information was not significantly different to the heart rate when approaching the box containing the control animal (no information), $F(1, 25) = 1.07$, $p > .05$, and this was a small to medium size effect, $r = .20$.

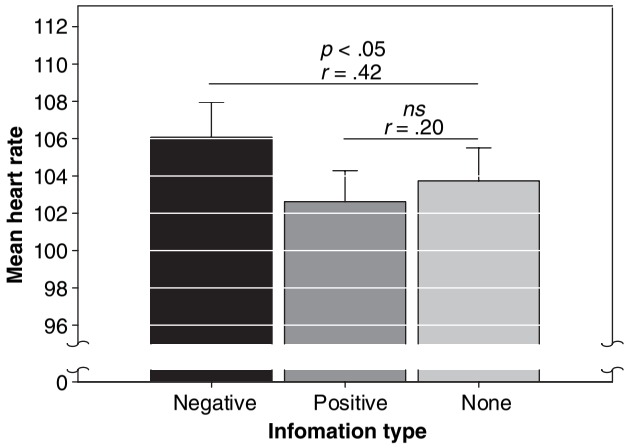

Figure 1 Graph showing the mean heart rate (and standard error) during the 15 s during which the child placed their hand into each of the three touch boxes. Effect sizes are reported as *r*

Discussion

The main finding from this experiment was that threat information had a direct effect on the physiological system of the fear response. This finding adds to the existing body of research showing that the threat information pathway is a viable causal mechanism through which the subjective and behavioural response systems can be changed. Taken together, this evidence shows that threat information can affect all three of Lang's systems of the fear response (Lang, 1968). This finding is important given the desynchrony in measures of fear (Zinbarg, 1998), and, in conjunction with previous work (Field, 2006a; Field, Argyris, & Knowles, 2001; Field & Lawson, 2003), shows convergence in the effect of verbal information across different response systems of anxiety. Also, given the proposition that convergence is expected when emotional arousal is strong (Hodgson & Rachman, 1974), the current

results might suggest that verbal information does not merely create weak levels of emotional arousal. However, to fully conclude this, future research must measure cognitive, behavioural and psychological indices and anxiety concurrently to see the correlation between these measures within children.

Implications for theories of fear acquisition in children

This experiment clearly supports Rachman's (1977) theory that fears develop directly through verbal information. Although a variety of past work has shown that verbal information can lead to cognitive and behavioural phenomena associated with clinical anxiety (i.e., attentional biases and avoidance), this is the first empirical demonstration that children's physiological responses change as a direct result of threat information. This finding, therefore, offers a causal link through which verbal information creates childhood anxiety. For example, if children's heart rate responses to novel animals are altered by information from adults, it supports the suggestion that verbal information (and vicarious learning) is the likely pathway through which anxious parents transmit anxiety to their children (Hadwin, Garner, & Perez-Olivas, 2006). However, the finding could suggest that some theories of fear acquisition may have underplayed the direct role of verbal information in fear acquisition. For example, in Davey's model, fear information is seen as important in influencing future conditioning, and in revaluing situations, but this experiment suggests that verbal information has a direct effect too: it is not simply a variable that interacts with future learning (Davey, 1997). There are similar implications for Mineka and Zinbarg's (2006) model, in which verbal information is also viewed as a vulnerability factor influencing future learning. However, there are two important points to consider. First, the data here do not rule out a conditioning account of fear acquisition (see below) and, at most, the results would imply only a minor modification of these theories to include verbal information as a potentially direct learning experience rather than a vulnerability factor. Second, although significant increases in heart rate were observed in this study, these increases were relatively minor. It is certainly not the case that phobic levels of fear were induced, but merely subtle differences in heart rate. This gels with the suggestion that verbal information is likely to give rise to milder fears than direct conditioning (Rachman, 1977), but also implies, as Davey (1997) and Mineka and Zinbarg (2006) suggest, that for clinical anxiety to develop more directly traumatic experiences may be needed, or that the fear created by information may have to undergo some subsequent process such as incubation before it reaches clinical levels.

Implications for theories of how the threat information pathway works

Although Rachman (1977) did not propose a formal mechanism through which the threat information pathway operated, Field (2006b) suggests that fear information can be conceptualised in terms of associative learning in which a stimulus (e.g., a quoll) acts as a conditioned stimulus (CS) and the information acts to elicit a mental representation of threat, which acts as an unconditioned stimulus (US). The evoked concept of threat need not, in itself, be aversive: Field points out that modern conceptualisations of associative learning/conditioning no longer assume that an outcome or unconditioned stimulus is biologically significant. For example, humans can readily learn that pictures of butterflies (CS) will mutate (US) when exposed to radiation (Collins & Shanks, 2002; Lober & Shanks, 2000) or that certain foods (CS) predict an allergic reaction (US) (Aitken, Larkin, & Dickinson, 2000; Le Pelley & McLaren, 2003), and the learnt associations show many phenomena characteristic of autonomic conditioning (see Dickinson (2001) for a review). Past work using the Implicit Association Task (Field & Lawson, 2003; Field et al., submitted) could be interpreted as suggesting that fear information acts by creating an association between the novel animal and a concept of 'nasty/bad/threatening', or even a memory of the information itself, and that it is this representation that is evoked during the behavioural task, and the behavioural output of this cognitive representation is an increase in heart rate. Consistent with this idea, cognition in a threat situation has been shown to be a precursor of cardiovascular change (Tomaka, Blascovich, Kibler, & Ernst, 1997).

The data from the current experiment support this view: the design of this experiment can be seen as a CS+/CS− discriminative conditioning paradigm in which certain CSs (animals) are paired contiguously with information (US) and act as CS+s, whereas other animals have no CS (no information) and act as a CS−. The term 'conditioning' can applied to a process, a procedure or a mechanism (Field, 2006b) and at least procedurally the current experiment can be conceptualised as conditioning. The conditioned response was a physiological response (increases in heart rate) to the threat CS+ compared to the CS−; this comparison tells us that the pairing of the animal and the information has had the effect. However, what it does not tell us is whether the differences in heart rate are being governed by the information itself, or by a representation of 'threat' that the information evokes. As such, future work needs to use techniques to re-value (Rescorla, 1974) the information to see whether the cognitive, behavioural and physiological CRs demonstrated in these experiments are driven by a direct association between the animal and the information or an

association between the animal and a representation of threat evoked by the information.

Developmental implications

The current experiment was based on an age range during which animal fears are typically common (6–9 years old) and at which animal phobias develop. The developmental implications of the experiment are hard to gauge because only one age group was tested. As such, it is unclear whether the observed differences in heart rates were specific to this age group and, therefore, represent a particular vulnerability to fear information that is specific to this developmental period. Cognitive biases, selective attention and memory biases are associated with anxiety symptoms in children but do not appear to differ across age groups (Watts & Weems, 2006); however, there is no evidence on whether such anxiety-related cognitions differ across ages when anxiety symptoms are partialled out. Furthermore, although Field et al. (submitted) have shown that fear cognitions and behavioural avoidance to novel animals are statistically comparable in 6–8 and 12–13-year-olds, future work needs to be done on whether information-induced physiological reactions to novel stimuli are age dependent, and, in general, more research is need on the developmental pattern of information processing of threat material.

Clinical implications

If children are showing increased physiological responses to novel stimuli after hearing information from adults, then this has obvious clinical implications. First, in terms of prevention, the suggestion is that parents and other significant adults could reduce the likelihood of fears developing by reducing the amount of threat information they provide to their children (at least about specific phobic stimuli): there is clear evidence that giving children threat information increases their physiological responses when interacting with these animals. There is also the possibility that by providing positive information, adults could immunise against future negative learning episodes (although positive information had only a small, and nonsignificant, positive effect on heart rate responses during interactions with the animals).

If children's heart rates react to information given by adults, then this could explain the increased reluctance to interact with these animals (Field, 2006a; Field & Lawson, 2003). Avoidance of the animals is potentially problematic because it prevents disconfirmation of threat beliefs (in clinical terms it acts as a safety behaviour) and, therefore, makes any fear cognition, or physiological responses, more likely to persist.

In terms of interventions for child animal anxiety, the implications are less clear. Positive information did not reduce heart rate responses significantly (and gave rise to a relatively small effect size), suggesting that positive information is not a useful strategy for reducing fear. However, the implication from any learning model is that if fear can be learnt through information then there should be ways to unlearn that fear, or to break the association between the animal and the representation of threat, or the memory of the information. In this context, future work could again usefully explore exactly what associations are formed (if any) when verbal information is given and, therefore, inform interventions to break or reduce the strength of those associations.

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