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Dear Library,

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American journal of psychiatry
1991 VOL 148 PP 388-390
Dr. Stone Replies
Stone A

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creasingly biological in orientation, and search committees for chairpersons of departments of psychiatry are interested in neuroscientists and, at times, have a negative attitude toward psychoanalysts.

How has the psychodynamic school responded to these changes? Unfortunately, by apology and argument ad hominem. It has not generated evidence for its claims of therapeutic efficacy. In this respect it is interesting that proponents of the newer psychiatric schools have increasingly used randomized, controlled trials to support claims for the efficacy of psychopharmacologic and behavioral therapies and as weapons in the rivalry among schools of psychiatry and, particularly, the interprofessional rivalry between psychologists and psychiatrists.

The title of Dr. Stone's paper (2) is indicative of this response of the psychodynamic school. The title announces his reply to my *indictment* of psychoanalysis. This is unfair to me. "Indictment" has a specific meaning in criminal law, implying a violation of criminal statute. While I am critical of psychoanalysis and skeptical of some of its claims, to invoke the term "indictment" is to go far beyond my argument. It is not an accurate representation of my views, and it serves to mobilize the believers in psychodynamic therapy by discrediting me and attributing to me a position that I have not taken.

Dr. Stone's argument seems to be that the professional and scientific difficulties that the psychodynamic school is experiencing result from the use of the courts by scientific/biological psychiatrists like myself to advocate our professional views. Of course, I have a point of view and have expressed it in various settings, including court cases such as the *Osheroff* case. Dr. Stone and his colleagues do not hesitate to express their point of view. There is nothing conspiratorial, undemocratic, or unprofessional in this advocacy. In fact, it was Dr. Stone who first drew attention to this case in the *New England Journal of Medicine* (5).

The current problems of psychoanalysis and the psychodynamic school are not the result of critics such as myself. Would that we were so powerful! Rather, the psychodynamic school has failed to respond adequately to new developments. For example, consider the status of the symptom neuroses: phobias, obsessions, compulsions, anxiety, depression, and dissociative states. In the past, psychoanalysis has contributed greatly to our diagnostic and therapeutic understanding. The situation has changed dramatically. If one looks at the programs of recent APA annual meetings, one finds that in the many lectures, discussions, and symposia on obsessive-compulsive disorder, phobia, depression, and panic disorder, the psychoanalytic school was hardly represented. What new contributions have psychoanalysts made to the understanding and treatment of phobias? Is there any evidence about new psychoanalytic treatment of obsessive-compulsive states?

Moreover, the new treatments—psychopharmacologic and behavioral—have supported their claims for efficacy by randomized, controlled trials. There are no reports on controlled trials of dynamic treatment of these clinical states. A few efforts are now underway, namely, the work of M. Horowitz on stress disorders, of L. Luborsky and his associates on the use of supportive-expressive therapy for heroin addicts maintained on methadone and, more recently, for patients with depression, and the efforts of a psychoanalytic study group on panic led by A. Cooper and K. Shear. However, these efforts are at a relatively early stage in their development and have not had an impact on research thinking or clinical practice.

Let me make my position on psychoanalysis and psychotherapy explicit. I am not a biological psychiatrist, nor am I hostile toward psychotherapy. I do not rely exclusively on psychopharmacologic agents, and I have been active in psychotherapy research for 20 years. I am not hostile toward psychoanalysis. My dominant feelings about psychoanalysis are frustration and disappointment.

There is resistance within the psychoanalytic community to confronting the issues of the scientific status of psychoanalysis and the need to generate evidence for the claims of efficacy of psychoanalysis and psychodynamic psychotherapy. Unless these issues are grappled with by the psychoanalytic leadership, I am pessimistic about the future of our profession, which has been greatly enriched by psychoanalytic ideas and psychodynamically oriented teachers and clinicians.

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GERALD L. KLERMAN, M.D.
New York, N.Y.

Dr. Stone Replies

SIR: This coda to the *Osheroff* dispute seems to be in the adversarial mode Dr. Klerman espoused when he chose to testify against Chestnut Lodge. Here Dr. Klerman labels me an "apologist" and implies that I have questionable motives.

Some of his comments might suggest to readers that, like Dr. Klerman, I had direct personal or professional involvement in this matter. I must therefore reemphasize the fact that I was not retained as an expert witness, nor did I have any clinical connection with Chestnut Lodge. My only involvement has been my academic commentaries on the legal significance of the litigation. In that connection, my response to Dr. Klerman's paper emphasized that he had mischaracterized the precedential value of a case settled out of court and that there was no legal authority for what he called the "right to effective treatment." Therefore, any standards based on his misunderstanding of the law were not valid. Furthermore, the standards he proposed had ominous implications for the psychodynamic school, and in the long run, his approach would be counterproductive even for the "scientific" psychiatry he favored. Dr. Klerman does not respond to these basic criticisms set out in my response. Instead, he replays two themes: the diagnosis and transfer of the patient and the scientific status of psychodynamic psychiatry.

As to the theme of the diagnosis and transfer, it should be recalled that Dr. Klerman asserted that a working diagnosis of narcissistic personality disorder was negligent, even though an affective disorder was also diagnosed. My response, based on published information, set out details of the clinical history omitted by Dr. Klerman which demonstrated

that such a diagnosis was entirely plausible. I also emphasized the frequency of the narcissistic personality diagnosis in that time period as further evidence against Dr. Klerman's uncompromising judgment that his colleagues behaved negligently.

My suggestion that the diagnosis might have been reasonable at the time is apparently reprehensible to Dr. Klerman. He is prepared to borrow a familiar page from the antipsychiatry polemicists who refuse to recognize that a psychiatric diagnosis can be both clinically correct and personally offensive. Dr. Klerman, like many "expert witnesses," has simply overstated his case. To make his point, he is now willing to repudiate the *DSM-III* personality disorders. Ironically, in his original paper, adherence to *DSM-III* was the very standard of care he wanted to impose on his psychodynamic colleagues.

I continue to believe that the diagnosis of narcissistic personality disorder was not negligently made, nor was it intended to be pejorative or morally condemnatory. And I did not suggest that such a diagnosis would justify the 7-month course of exclusively psychoanalytic treatment at Chestnut Lodge. As Dr. Klerman recognizes, I clearly stated that when Dr. Osheroff's condition deteriorated and when there were obvious symptoms of a major depressive disorder, consultation and alternative treatment were indicated. Therefore, Dr. Klerman's statement that I "would deny justice to a patient" through psychoanalytic characterology is, at the least, ill considered.

Dr. Klerman finds my opinion about Dr. Osheroff's sudden and dramatic improvement after his transfer "fanciful" and illustrative of the "strange clinical logic" common to psychoanalytically oriented colleagues. He apparently prefers to believe that Dr. Osheroff's dramatic improvement was entirely due to the drugs he was given. Perhaps it will help readers to sort out this disagreement if I restate my understanding of the issues. First, before his hospitalization, Dr. Osheroff had several times been medicated for depression in exactly the efficacious fashion Dr. Klerman would require, without dramatic improvement. Second, Dr. Osheroff was at a therapeutic impasse at Chestnut Lodge, with an intense negative transference, when an experienced consultant suggested transfer to a different hospital. Third, Dr. Osheroff's own account of the effect of the transfer indicates that he experienced a profound psychological improvement before the time at which one would expect the medication to take effect. Also, the quality of his psychological improvement was unlike that usually seen in patients on a combined regimen of phenothiazines and tricyclics. Fourth, the psychiatrist who was directly responsible for Dr. Osheroff's overall treatment at the second hospital emphasized the importance of the different psychiatric milieu in the patient's dramatic improvement. Fifth, it has been my clinical experience that patients frequently benefit from a change when they are at a negative therapeutic impasse. The same (presumably efficacious) treatment works better when given by a different doctor in a different setting. If Dr. Klerman believes that this account demonstrates a "strange clinical logic" which he would eschew, then I am at a loss to know how he understands the role of transference in the clinical practice of psychiatry.

The second theme in Dr. Klerman's coda laments, as did his paper, the unscientific nature of psychoanalysis and its current decline. He adds only that the word "indictment" as used in the title of my paper discredits him. I must confess that my earlier word choices were harsher. It seemed to me that Dr. Klerman had done more than indict; he had, in fact, judged and sentenced. He had declared psychoanalysis and psychodynamic psychiatry obsolete by his scientific standards and had proposed legal barriers to their practice. Whether he did this out of frustration or hostility seems irrelevant. Certainly, his proposal would undermine the professional credibility of psychodynamic practitioners.

Dr. Klerman made two kinds of arguments against the psychodynamic school. First, he attacked the scientific status of psychoanalytic theory, citing the philosopher of science Grunbaum. Second, he criticized the lack of clinical efficacy studies, such as those required by the Food and Drug Administration for drugs. Neither of these arguments is new or original with Dr. Klerman. They have both been made by various critics for decades. What was novel was Dr. Klerman's insistence that they were now relevant to legal determinations of what constitutes negligence in the practice of psychiatry. Therefore, my response focused on the legal considerations.

Here I would briefly add that whether or not psychoanalysis is a "science" is a matter of competing definitions of science (1). Psychoanalysis is not a science as that concept is defined by logical positivists like Grunbaum or by the paradigm of biological materialism that now dominates much of American psychiatry. Biological materialism may eventually explain memory, but it will never explain memories. As long as psychiatry deals with human memories and the relation between symptoms and the human experience, it will need more than the limited understanding supplied by these narrowly defined concepts of science.

As to the efficacy arguments, Dr. Klerman's strict standard of scientific scrutiny has not been equally applied to the available clinical efficacy technology on which he relies. The recent disputes about the interpretation of the results of the NIMH collaborative research on treatment of depression (2) amply demonstrate the lack of scientific unanimity produced by current methods of studying clinical efficacy. One does not have to be a Luddite or an enemy of scientific psychiatry to suggest that until we know much more about the neurosciences, we are still crawling on our hands and knees. We are at the early stage of a long journey in search of definitively efficacious treatments for our patients.

Dr. Klerman, for the first time in this coda, explicitly acknowledges that he used the *Osheroff* litigation as an occasion to advocate his own professional views in court. He therefore confirms a basic thesis of my criticism. During the past two decades, psychiatry has been turning and twisting in the winds of litigation. At every turn and twist we were told, as Dr. Klerman tells us, that it was a matter of justice for patients. But in fact the impetus for legal change in this field has come from lawyers and self-interested professional experts who have advocated their own views. Sometimes the professionals, ignoring or discounting efficacy, were hostile to ECT or to antipsychotic medications. Typically, they sought to reject the medical model of mental illness and treatment. The one consistent result of this litigation has been to limit the discretionary judgment of the psychiatrist.

Dr. Klerman's attack is from the other side, so to speak, but it would have the same dispiriting result. It would narrow and limit our profession to a cookbook of recipes sent down from on high. The threat of malpractice liability invoked by Dr. Klerman and the fiscal discipline of managed care join forces in this cookbook mentality that now erodes professional autonomy in the name of science and efficacy. Our patients who need more than recipe treatments will be the victims.

I agree with Dr. Klerman that psychiatry "has been greatly

enriched by psychoanalytic ideas and psychodynamically oriented teachers and clinicians." This enrichment took place even though the scientific criteria Dr. Klerman now mandates were never met. That may be a paradox for Dr. Klerman, but for me it demonstrates that the "learning" of psychiatry comes from a family of humanistic and scientific disciplines. Indeed, that is the vital attraction of the profession. The art of clinical practice is in weaving these disciplines together; science may be our guide but it is not our master.

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ALAN A. STONE, M.D.
Cambridge, Mass.

Thiazide Diuretics and Polydipsia in Schizophrenic Patients

SIR: The literature linking polydipsia, hyponatremia, water intoxication, and hyponatremic encephalopathy in chronic schizophrenic patients is voluminous (1). Several cases of polydipsia in which a thiazide diuretic is thought to have triggered water intoxication and its sequelae have also been reported (2, 3). We report here a serious case of hyponatremic encephalopathy that was very likely precipitated by hydrochlorothiazide and add the warning that polydipsia should be ruled out before thiazide diuretics are prescribed for patients with schizophrenia.

Mr. A, a 32-year-old schizophrenic patient whose condition had been stable with a regimen of fluphenazine decanoate, 1 cc every 2 weeks, for more than a year, was hospitalized after his mother noted that he was more withdrawn than usual, not sleeping, and drinking excess fluids. The patient, hospitalized for 24 days, continued to receive fluphenazine decanoate injections and was also started on hydrochlorothiazide, 50 mg/day, for hypertension (his blood pressure was 150/90 mm Hg).

Three weeks after discharge, Mr. A's mother found him in bed, diaphoretic and mute. He was admitted to the intensive care unit in status epilepticus, which was terminated with intravenous phenytoin sodium and diazepam. On admission, his serum sodium level was 108 meq/liter. A drug toxicity screen was negative. When he was given a short course of 3% normal saline while a large free water diuresis developed, his serum sodium level rose to 135 meq/liter in about 8 hours, suggesting that pituitary function was normal. He had no further seizures.

Mr. A's mother reported that during the week before this hospitalization, he had been drinking 3–4 quarts of beer and up to 6 liters of soft drinks each day. He was also smoking 3–4 packs of cigarettes daily. Because of the large amount of liquid consumed, and because his serum sodium level was quickly corrected, a diagnosis of psychogenic polydipsia and diuretic-induced hyponatremia was made. The diuretic was discontinued, and the patient was discharged on a regimen of oral captopril, 25 mg t.i.d., to control hypertension. The suspicion that hydrochlorothiazide had precipitated this episode was supported when, several weeks later, Mr. A again consumed large quantities of liquids while continuing to smoke heavily, was rehospitalized, but was asymptomatic this time and had a normal serum sodium level.

It is not known why some patients with chronic schizophrenia drink excessive amounts of water, particularly during relapse, or why about 3% of those who do so develop hyponatremia (4). It is suggested repeatedly in the literature on polydipsia that the increased dopamine activity associated with schizophrenia perturbs the mechanism controlling thirst and/or stimulates excess secretion of antidiuretic hormone, leading to greater retention of water and dilution of sodium in serum (3). Neuroleptics and nicotine from heavy cigarette smoking (4) are also mentioned as possible contributing causes of hyponatremia.

Thiazide diuretics inhibit reabsorption of sodium in the kidney, causing loss of both sodium and water from serum. Impaired urinary dilution and inappropriate antidiuretic hormone secretion can also contribute to thiazide-induced hyponatremia (5). By drinking excess water, schizophrenic persons who take these diuretics can then further dilute serum sodium to life-threatening or lethal levels.

Excess water consumed by polydipsic schizophrenic patients should be considered a "drug" in a potential drug-drug interaction with any substance that alters water balance. The polydipsia diagnosis should be conspicuously noted in the charts of such patients, and a warning about prescribing thiazide diuretics should appear.

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RENÉ J. MULLER, PH.D.
HELEN D. LANN, M.D.
Baltimore, Md.

Unexpected Intracerebral Pathology in Older Schizophrenic Patients

SIR: We wish to report a high frequency of unexpected intracerebral pathology in a group of older schizophrenic patients, most of whom were men. We obtained CT scans of 29 patients (27 men and two women) over age 55 during a longitudinal study (1). Patients with histories of degenerative brain disease, stroke, substantial alcoholism, or brain injury were excluded. Several of the scans exhibited lucencies suggestive of past cerebrovascular accidents as well as other pathology. The scans were examined by a neuroradiologist (M.T.B.) who was blind to information about the patients. If there was an obvious, well-defined cortical or subcortical lucency consistent with a past infarct, the patient was considered to have had a cerebrovascular accident. Ten of the patients (34%) had evidence of a probable past cerebrovas-