Drug treatment of the patient with a borderline syndrome is at once a heretical and a pragmatic practice. The dynamic formulation of the borderline character disorder forms the paradigm of modern psychological theory just as that of hysteria once served Freud. It is such a critical fulcrum of thought that the descriptions of this syndrome approach an art as much as a science. The clinician treating the borderline is forced into an acrobatic dance of contorted communication just to maintain contact with the patient. A completed case is an object of admiration. Medication is commonly regarded as an impurity in this rarefied atmosphere.

Borderlines abuse drugs. If we give them more, we are abusing the patients, colluding with them in their psychopathology. The therapeutic relationship will be hopelessly damaged. The implication is that a competent treatment is a drug-free treatment.

There is no question that the objections are well founded from both a scientific and an artistic perspective. Yet drugs work. They clearly are not a complete treatment, just as lithium is not a complete treatment for manic-depressive syndrome, but they facilitate and sometimes are the sine qua non of treatment.

The state of the drug art is young. Treatment choices are frequently opportunistic. Understanding of the application of diagnostic techniques and medications to borderlines is incomplete, though related pathological processes such as the "atypical depressions" are coming into a fairly clear light. The physiology of affect, of course, is a burgeoning study. Response to
DEFINITIONS

There are three diagnostic categories we should consider: borderline syndrome without affective disorder; borderline syndrome with affective disorder; and affective disorder mimicking borderline syndrome. By "borderline syndrome without affective disorder" I mean a character disorder, probably with some degree of secondary depression in response to that character disorder, but not a primary or biologic affective disorder. Some people have more than one coexisting psychopathological process, borderlines with affective disorder are an example. Here I am referring to a primary affective disorder. It is reasonable to postulate that the affective disorder, if present in a subclinical or clinical form during early development, could be a significant factor in the formation of character. A dramatic example would be the child with subclinical manic-depressive syndrome, who, deprived of a constant internal psychological milieu, would relate to his parents in a clinging fashion. Although their initial response would be nourishing, the constancy of the clinging, coupled with the parents' perception of their child's inconstant affective identity, would produce a self-protective withdrawal by the parents, thus setting up the field for the growth of an abnormal character as well as the biologically determined affective disorder. A parent with an affective disorder can pass along a dual legacy: the genes for depression and the empty parenting of a depressive.

Affective disorder can mimic character pathology. Akiskal (1983) describes this phenomenon eloquently. There is the primary depression with residual chronicity, and the subaffective dysthymic disorder. The commonality is the fact that these are both biological affective disorders. The difference is that in one instance there is an initial, clear, major affective episode, while in the other the process is insidious. The latter is commonly mistaken for the borderline syndrome.

DIAGNOSIS

Kernberg (1975), Masterson (1976), and Kohut (1971), among others, present clear diagnostic schema based on personality dynamics, interpersonal relationships and behavior, and the transference occurring in psychotherapy. Grinker and Gunderson have compiled more phenomenological descriptions of the borderline (see Gunderson and Singer, 1975; Grinker and Werble, 1977). Given the potential for a concomitant affective disorder, or for an affective disorder to appear to be character pathology, one must also apply the diagnostic techniques used to define depression, anxiety, bipolar affective disorder, and the various associated syndromes. Affective disorders are psychophysiological events and as such can be characterized using the traditional medical model: diseases with a specific history, typical psychological and physical responses by the patient to the disease state, pedigree, and physiological peculiarities common to the disease state.

There are a variety of physiological tests that are abnormal in affective disorders. The best known is the dexamethasone suppression test. This is a measure of adrenal gland function, which is elevated in about one-half of the cases. The current hypothesis is that adrenal function is inhibited by changes in the hypothalamic-pituitary axis. There is an intimate biochemical relationship between the pituitary gland, which regulates all the other endocrine glands' function, and the hypothalamus, a part of the limbic system of the brain where affect is generated. The TRH (thyrotropin-releasing hormone) stimulation test is abnormal in 60% of cases, which overlaps that segment with adrenal abnormalities. This is another test of the hypothalamic-pituitary axis as it effects thyroid gland function and is somewhat more complex to administer than the dexamethasone test.

There are other, more exotic measures of the metabolites of neurotransmitters in the urine, blood, and spinal fluid that at present are impractical to consider from a clinical perspective, although they are valuable research tools. Of greater clinical value is the sleep electromyogram. This displays the patterns of electrical activity in the brain during sleep. In depression, the time between sleep onset and the first REM period (REM latency) is decreased significantly from normal. The methylphenidate (Ritalin) challenge is a clinical (versus laboratory) test. Here a patient is given one or more doses of the stimulant methylphenidate, which acutely mimics the effect of antidepressant drugs. If the target symptoms clear, it is likely that the person will respond to antidepressants. It is interesting to note that many borderlines abuse (use) stimulant drugs prior to successful professional treatment. If a borderline patient shows a positive history for affective disorder or is not progressing as expected in psychotherapy, physiological testing and treatment should be considered (Carroll et al., 1981). A positive response to medication is also a pragmatic diagnostic tool, but given the problems inherent in medical treatment, this "test" should not be used without a high index of suspicion.

To return to our three diagnostic entities, the "borderline without affective disorder" has the characteristic clinical features and exhibits the dynamics outlined elsewhere in this book but is missing certain attributes: there is no recent or past history of a clear affective episode of significant proportion; although affective lability is typical in response to trauma, cyclothymia unrelated to trauma is absent; family history for affective disorder is unim-
pressive; the psychophysiological status is not retarded but agitated; and none of the aforementioned physiological tests is remarkable.

The "borderline with affective disorder" looks and acts like a borderline, but on careful investigation, exhibits other findings. I must emphasize careful investigation, because the defensive veneer is apt to be so dramatic that it can divert the most experienced clinician. These dramatic defenses are generally seen as responsive to the trauma of interpersonal situations, but can also result from the intrapersonal trauma of an affective disorder. One finds a depression minimally related in degree and form to identifiable trauma. There is usually some history of other, similar episodes, perhaps treated with prescription or street drugs. Family history will be positive—the reason for the maternal rejection when the patient was two was that mother was depressed, clinically depressed. Behavior is retarded during these periods, though not at other times—eating, sleeping, and defecating become consuming in a tone that suggests hibernation, not acting out. The DST or TRH test is abnormal; REM latency is reduced frequently; and the methylphenidate challenge shows a dramatic response.

With those "affective disorders that mimic borderline syndrome," one sees a picture that is frequently indistinguishable from the previous diagnostic group. This category should be divided in two: the chronic mild depression following a major affective episode, and the "subaffective dysthymic disorders." The former should be easy to discern from the history. There will have been a clear manic or depressive episode preceding the onset of the current symptoms. A lifelong problem is not apparent, and the present mental status should be clearly depressed while also exhibiting a variety of regressive traits which appear borderline. Physiological symptoms and testing will reflect those changes characteristic of a biological affective disorder. Subaffective dysthymia is a lifelong mild depression, which becomes inculcated in the personality. The individual is apt to be depressed and self-deprecatory. Involvement with friends or activities is truncated. Affect may follow a cyclothymic pattern (not labile) but is generally somewhat depressed, with only brief periods of euthymia. Mild vegetative symptoms are seen, as are a positive family history for affective disorder and frequent "abnormals" on testing. These two types of chronic affective disorders are often in therapy for an extended period of time before they are diagnosed as borderline, based on the refractory nature of their psychopathology—what appears to be reluctance to enter into a productive relationship is in fact simple biological inability.

TREATMENT

The drugs most commonly used for the borderline and associated syndromes are the antidepressants. These compounds effect the levels of the neurotransmitters noradrenalin, serotonin, and dopamine, among others, and therefore, hypothetically, the rate of nerve impulse transmission in the brain; hence they have ameliorative effects on mood. The difference between various drugs is the extent to which they alter each neurotransmitter: they are purely "adrenergic," or "serotonergic," or mixed. The other distinguishing factors are the type and quantity of side effects produced by the drugs. On the clinical side, certain drugs are more effective at relieving anxiety and panic; others are better for depression; and several are either less apt to exacerbate psychotic disorganization or even offer some therapeutic effect in psychosis. Two approaches may be taken to the administration of antidepressants. The conservative approach is to choose the drug on the basis of the side effects: it is likely to precipitate in a given individual, starting with a "reuptake inhibitor"—this category includes all the tricyclic antidepressants, plus most of the compounds introduced in the last two years. An overall drug sensitivity can be gauged by the person's sensitivity to other psychoactive medications or recreational drugs, including alcohol. It is also important to describe briefly the side effects typical of the chosen medication and reconsider the choice if the patient already suffers from similar symptoms. Then there are two reasons for changing the medication. If after any period of time debilitating side effects occur, a switch should be made to a drug with a different molecular structure. If after the appropriate trial period for a given drug (usually four weeks) the compound has no therapeutic effect, a drug that targets a different neurotransmitter should be used. If there is a suboptimal effect, first a blood level of the drug should be obtained. If the level is low, consider raising the dosage. If the dose is already quite high or the level is in the therapeutic range, consider adding a small dose of lithium or thyroid. Both compounds have been found to have a synergistic effect on antidepressants.

The next step is to try monoamine oxidase inhibitors (MAOIs). These drugs act by blocking the breakdown of all of the neurotransmitters, the distinction between MAOIs being the level of side effects. One of these side effects is the reason why these drugs, although effective, are generally a second choice. Since their mode of action is to sensitize the body to the neurotransmitters by decreasing their breakdown, exogenously introduced chemicals similar to those neurotransmitters can be overstimulating, producing anxiety, elevated blood pressure, and a variety of other related symptoms. In the extreme this could result in cerebral-vascular accidents. This awful problem has a simple solution: the easily accomplished avoidance of the offending chemicals. In any population but borderlines, this is easily said and done, but only the former is true with the uncooperative patient. Ingesting a "forbidden" chemical can be anything from a challenge to a communication. On the positive side, the MAOIs are generally regarded as the most effective drug for severe anxiety, atypical depressions, and anxiety "equivalents."
behavior is, among other things, a way of mitigating anxiety, and if the MAOIs treat that anxiety, it will be used as a tool and not a weapon by the patient.

A variety of other drugs are sometimes used for borderlines instead of antidepressants, or if antidepressants have not been effective. Sedative-hypnotics, such as secobarbital and methaqualone, and minor tranquilizers, such as diazepam (Valium) are useful for the anxiety-related symptoms in rather large doses, but are almost always abused. A new drug in this category—alprazolam, or Xanax—may be an exception in that it ablates much major anxiety in relatively modest doses. Although it is chemically similar to diazepam, because of its greater efficacy of action, it may obviate acting out as a self-treatment for anxiety and thus have a lower abuse potential. Even though they are only rarely effective, major tranquilizers such as chlorpromazine have been used in the treatment of borderline symptoms of anxiety and depression—because at least these drugs are not liable to be abused. This kind of treatment is really indefensible, especially given the potential for permanent and serious side effects from major tranquilizers. The rare exception is the borderline with a psychotic diathesis who requires that kind of psychic organization these drugs provide.

Lithium is similar to the major tranquilizers in its indications. Although it provides a mild antianxiety effect and occasionally is an effective antidepressant, the complexity of its administration and the potential for serious side effects make this a drug of "last choice," or one to be reserved for that borderline with a clear cyclical mood disorder—not just severe lability of mood.

Amphetamines represent an interesting choice of medication for borderlines. Here is a drug that is frequently self-administered. Clinically it seems to enhance both healthy and pathological defenses and if overused can lead to frank psychosis. Effective use of stimulants has been reported in several special affectively disordered populations and in "adult minimal brain dysfunction" (AMBD) (see Wender, Wood, and Reimherr, 1984). The latter is frequently associated with massive anxiety. This would be a ripe area for controlled research.

A second approach to administering medication to these kinds of patients uses the safest and easiest drug first. Evidence is mounting to suggest that certain syndromes tend to respond to certain drugs, and that some of the physiological tests may predict specific drug response as well as diagnose depression.

There is an increasing literature of case reports that the secondary dysthymia of the borderline responds very well to the MAOIs (Klein, 1977). It is not unusual to hear several weeks after the onset of such treatment, "You know, I told you I never had anxiety attacks, but since I've been on this drug and started to get some relief, I realize I was having anxiety attacks con-

stantly but I didn't know what they were—I just hurt." The overall effect is to reduce the "background psychic noise," allowing the individual to focus better on a variety of extra- and intrapsychic events.

The "reuptake inhibiting" antidepressants and lithium are the drugs of choice for the primary affective disorders, which include all but the "pure borderline" groups dealt with here. One can differentiate the "pure affective disorder" from the borderline with affective disorder by drug response. The former show what appear to be changes in their character pattern following drug treatment: regressive, dependent qualities, along with their dynamic concomitants, melt away, to be replaced by maturation so quick that it reminds one of time-lapse photography. Sometimes, of course, this process marches right into hypomania, or mania, in which case appropriate responses must be made. More often depression is replaced by euphoria, with behavioral changes lagging behind mood changes—therapy at this point is critical to enhancing affect-congruent growth in behavior. The depressed borderline also converts to euphoria, but the character style remains borderline. No lesson seems to have been learned from the affective episode, its very memory an anathema to the patient. The pace picks up, but the individual does not grow up. No miracle cure here!

Although, in my opinion, it is best to prescribe following the foregoing diagnostic guidelines, two of the physiological tests offer some additional input. A positive response to methylphenidate has been reported to auger well for response to adrenergic reuptake inhibiting drugs (Sabelli et al., 1983). A positive DST has been a positive response to either adrenergic or serotonergic drugs, depending on the author (Fraser, 1983). Based on nonobjective clinical experience, I prefer the serotonergic compounds in this situation.

MANAGEMENT

The management of the medical treatment of the borderline is complex. The clinician, having chosen a drug, must now administer it. Given the difficulty of even entering into a dialogue with the patient, this is no easy task. Administration of the drug starts with the initiation of the subject, which, it is hoped, is couched in a data-based interpretation. It traverses the analysis of the patient's response to the topic of drugs. After the drugs are actually given, one must consider an even wider variety of issues, remembering that there will be not just a drug effect, but a psychological response to that effect, which may completely overshadow the effect. Critical to an understanding of all these complexities is an acceptance of the fact that medication is something that the therapist brings to the therapy, not the patient. Although it has some similarities to other psychotherapeutic and psychoanalytic modes that
are generally responsive to the psychopathology, to a much greater degree it is a novel presentation of the therapist.

Ostow’s (1979) treatise covered this area in considerable detail. It is pointed out that, with the proper presentation, drugs are seen as a gift, or as an act of affectionate concern on the part of the therapist. In this spirit, it is important to forewarn the patient of the potential side effects of the medication. This helps to obviate a variety of psychological responses to those symptoms that may be countertherapeutic: side effects may produce resistance to all drug effects; they can be perceived as punishment for feeling better; or they could be a proof of the failure of either the patient or the doctor. Drugs treat not only the patient, but also the therapist: countertransference anxiety resonates with that of the patient, changing the character of the work and therefore exacerbating the psychopathology. Ostow and his contributors emphasize the importance of delaying drug treatment until analysis is well under way, thus making the differentiation of reality from fantasy somewhat easier. I think this course also facilitates the formation of the therapeutic relationship, which is strained considerably by the prescription of drugs by either the treating therapist or by a consultant.

A fascinating and frequently observed paradox is the *traumatizing* effect of a therapeutic drug effect: many drugs, especially antidepressants, can ablate certain phenomena, such as anxiety, depression, and psychotic thought patterns. What appear to be symptoms to the pharmacologist are also defenses for the psyche, and drug treatment can render a person defenseless in the face of stress. This situation must be addressed. The stress should be reduced in the environment or in the person by the use of tranquilizers. Even better would be the enhancement of coping skills, perhaps by focusing on these skills specifically in the therapy or by increasing the contact with the therapist, thus increasing the mechanical support for the patient. It is not uncommon for a person who has been “well medicated” to discontinue drugs to escape just such a problem.

There are other pitfalls not related to actual drug effects, but to the fact that drugs have been prescribed at all. Prescription seems to be proof that the patient is damaged physically and thus permanently. The drug is a method of psychological euthanasia, and the doctor is an assaulant. Or perhaps the therapist is damaged and has to use a medicine-prosthesis. Maybe the therapist is separating, leaving the drug as a transitional object, or delegating a medical consultant as the surrogate parent.

There is some debate about whether or not to separate the therapist from the medical management. This is frequently a necessity when the therapist is not a physician or is unfamiliar with the present “state of the art.” This therapeutic split allows for the talking therapy to be conducted uncontaminated by the mechanical issues of management and facilitates analysis of that management situation by the therapist at an objective distance. It decreases the extent to which the issue of the medicine may be used defensively in the therapy. In the acting-out patient, it removes a very dangerous self-destructive tool from the electric arena of the therapy. The major problem of the therapeutic split is that it may turn into a splitting defense. This necessitates some level of very clear communication, plus a significant amount of trust and confidence between the two professionals. Even with a single doctor, splitting may occur between the prescriber and the therapist. The data base will be more complete with one doctor, though it is messier and more difficult to clean it up. No extra communication is required, and there is no threat to the confidentiality of the treatment. One person is more likely to be able to follow the clinical course and make accurate responses to changes in the condition of the patient.

If a consultant is to be used, I would concur with Ostow that he not be called in early in the case. If it is an emergency situation, the support of the hospital is far safer than a precipitous trial on chemicals. From the start, communication must be open between therapist, patient, and medical consultant; otherwise, valuable information will be missed and the whole treatment will be undermined. It is important for both treaters to have a good grasp of the other’s camp, but not to do the other’s job. If some problem becomes apparent in the course of the consultation, it should not be ignored but referred to the appropriate dyad. As soon as an effective medication is found, the consultant’s role should attenuate, until there are problems or it is time to consider cessation of drug treatment.

**CONCLUSION**

Drugs rarely produce a cure in the borderline disorders or, for that matter, in any severe psychopathology. What they do is ameliorate symptoms, immediately reducing psychic pain; facilitate psychotherapy; and provide a psychological milieu more amenable to growth and development. If the wrong drug or the wrong dose is used, it will obfuscate the therapeutic issues past the point of recognition. Even the right medication places a tremendous strain on the therapy, challenging the most talented practitioner, yet that same drug will produce or allow changes not possible in years of work by both patient and therapist.

**REFERENCES**


