some double binds of clinical psychopharmacology: a personal view

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The “double bind” is a situation in which an individual is exposed to conflicting messages and is punished, regardless of the response he makes. Other characteristics of the double bind are that it calls for the denial of some (often important) aspects of experience and that it is inescapable—“no matter what a person does, he ‘can’t win’” (3).

While the double bind originally formulated by Bateson’s group referred to a family interactional process, and was proposed as a theory for understanding the development of schizophrenic behavior, it was recognized that it also applied to many other kinds of situations (4). Clinical psychopharmacology, for example, is replete with double-bind situations—from selection criteria through data analysis. One double bind in the selection of patients is that of random versus matched samples. Insofar as a sample is random, the criticism goes, one does not know how it was selected and, as Huntsman has pointed out, “even in mathematical hands, probability, chance and random mean ignorance” (6). In the case of carefully matched pairs of subjects, on the other hand, one really cannot “generalize” on the basis of such a limited experimental sample. Similar criticisms apply to fixed dosage regimes (which are “artificial”) and to flexible dosage regimes (which are “uncontrolled”). Clearly, these are situations in which the investigator “just can’t win.”

In the course of my own clinical psychopharmacological work, I faced the double-bind situation first arose. Testing patients on their habitual medication, it was argued, would vitiate our findings, since the drugs themselves affect psychometric test performance. But by the same token, testing patients after the cessation of their habitual medication would contaminate our results by secondary withdrawal effects. In the face of this seemingly insoluble dilemma, we nevertheless pursued our work—after a considerable time lapse. At first, an attempt was made to test patients after drug withdrawal, but as had been suggested, confounding secondary withdrawal effects were noted in the test results. For this reason, we felt justified in testing patients without interfering with their treatment routine or habitual medication (provided these had been unsuccessful in abolishing psychopathological symptoms). Proceeding in this manner, we found that long-lasting pathology destroyed personal and nondiagnostic performance patterns and resulted in a “restructurization” into impersonal or group patterns. Or in other words, as patients became more chronic, the internal hierarchy of their factor scores tended to become restructured according to a pathology-specific stereotype (1). Thus, the discriminating power of the battery depended upon the differential features of manifest psychopathology, regardless of whether patients were on or off medication, provided only that their psychopathological symptoms could not be controlled by treatment.

With the adoption of the statistical method in our clinical psychopharmacological work, the double-bind situation soon arose for the second time. The power of a statistical test depends on, among other things, sample size; if sample size is inadequate, the chances are increased for Type II or beta error, i.e., failure to find a statistically significant difference when an actual difference exists. On the basis of empirical analyses, some authors suggest that approximately 40 to 50 patients are required for each treatment
group in controlled double-blind studies in which, for example, the Brief Psychiatric Rating Scale (7) is employed. On the other hand, I definitely knew that it was impossible for me to follow clinically, at any acceptable level of thoroughness, more than 30 patients in any single study. These were indeed incongruent messages—the dictates of methodological sophistication versus those of clinical experience. I could well understand the plight of the growing child who cannot escape the classic double-bind situation without denying some aspects of his experience. I, too, could have become progressively dependent on statisticians, ultimately denying my own clinical experience entirely. Like the growing child, I might eventually have lost my ability to discriminate the true meanings of my own and others' messages. But this did not happen: I rebelled!

Mine was a weak revolt indeed. I continued to accept all the basic rules of clinical investigation; e.g., that experiments should be designed to answer specific questions, that experimental design should give careful attention to proper control groups, criteria for selecting the experimental population, and nature of treatment. I even accepted as a basic rule that questions asked in a clinical investigation can best be answered on the basis of a controlled experiment. But then, in the course of clinical studies with nicotinic acid, I began to recognize—as had others before me (5)—that the requirements of a controlled experiment are far from being fulfilled in clinical psychopharmacological experimentation. Because of this and, probably even more importantly, because of the absence of criteria for selecting truly homogeneous populations, all psychopharmacological studies, even those conducted under the best possible conditions, fail to give a valid estimation of experimental error or to provide a genuine baseline for comparing the efficacy of the investigational substance with (1) no treatment, (2) a placebo, or (3) a standard comparison drug. Eventually, I came to recognize that rigid adherence to the scientific-statistical method, at this stage of development, could lead psychopharmacology into a deadlock (2).

There are those who claim that adherence to the "medical model," not to the "scientific-statistical method," is to be blamed for the impasse in clinical psychopharmacology. One may wonder whether this is another double-bind situation!

References


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