Tablets and Talking—A Spurious Contrast in Psychiatry

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THE MODERN PSYCHIATRIST can resort to, roughly, two therapeutic strategies: "tablets" and "talking": to phrase it more professionally: psychotropic medication and psychotherapeutic techniques.

The two strategies are often regarded as representing a contrast. Psychotherapy is alleged to be unfavorably influenced by simultaneous administration of psychotropic drugs. Moreover, a difference in quality is claimed. Psychotropic drugs are qualified as symptomatic, constituting a therapy of the second order. They do not eliminate the cause, or so the argument holds; only psychotherapy can do that.

This antithetic view on types of psychiatric therapy has its roots in another antithesis, that concerning the causation of psychiatric disorders, which are viewed as determined either by organic brain lesions or psychosocial factors. A conceivable example is the unproductive discussion about the question of whether schizophrenia is a biochemical or a psychosocial disease.[1] In this vision, tablets are useful in particular in psychiatric disorders that are primarily of organic origin, while psychotherapy is indicated in the case of complaints and symptoms of predominantly psychogenic or psychosocial etiology. In the latter case, psychotropic medication is claimed to be a spurious therapy, a kind of anesthesia that effaces the true causes. Since psychogenic and psychosocial factors seem to play an etiologic role in the vast majority of psychiatric patients and in numerous patients with so-called functional somatic symptoms, the depreciation of psychotropic drugs is understandable from this point of view.

TABLETS VERSUS TALKING: BASIC POSTULATES

The contrast between tablets versus talking is based, as pointed out, on two postulates: (1) Psychosocial factors play a causative role in most cases of mental decompensation: (2) If psychosocial factors are predominant, then (A) organic (cerebral) factors are of little significance, and (B) psychotropic medication is of little therapeutic value.

This paper focuses on the plausibility of these two postulates.

PSYCHOSOCIAL STRESS AND MENTAL DECOMPENSATION

Psychosocial factors often play a role in the etiology of psychiatric disorders. This statement can be confirmed without much hesitation. In the past decade, a

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start has been made with systematic research into events preceding manifestation of psychiatric symptoms,[2] and methods were evolved to analyze these events and estimate their ability to upset the emotions.[3,4] This so-called life-event research has demonstrated the truth of the above statement. Life events occur significantly more often during the 6–12 months that precede the manifestation of a psychiatric disorder than in the average population.[5–7] However, this statement requires several qualifications.

To begin with, the specificity of life events proved to be low. A given syndrome, say vital (endogenous) depression, can be preceded by all sorts of more or less upsetting events; and, inversely, a given type of event can be followed by all sorts of psychiatric syndromes. In many cases, moreover, several "events" occurred during the period preceding the symptoms. The risk of mental decompensation seems to be determined not so much by a certain type of life event as by the total load of events.

Assuming that life events do play a role in the etiology of psychiatric disorders, a second question arises: what exactly is their etiologic weight? Do they play a decisive or a more subordinate role? There is some uncertainty about this question, which was studied most intensively for the depressions. The importance of life events in the etiology of depression should not be overrated.[8] While upsetting events often do precede depressions, the vast majority of individuals who experience such events do not become depressive. The factor "vulnerability" seems to be of decisive importance for the development of a depression.[9]

PSYCHOSOCIAL AND BIOLOGIC CAUSES OF PSYCHIATRIC DISORDERS

Let us accept the statement that psychosocial factors play a role in the etiology of many psychiatric disorders, and leave the question of their exact etiologic weight moot in this context.

Does this imply that somatic, or more specifically, cerebral factors are absent in these cases, or play a subordinate etiologic role? I can only answer this question in the negative. Nothing changes in human behavior unless it is made instrumentally possible by changes in the functional state of the brain. This applies to visible behavior but equally to emotional processes, which largely escape visual perception. In other words, psychogenic and psychosocial factors influence human behavior, via not a vacuum but changes in the state of the brain. Cerebral functions can, in principle, be described in biochemical and physiologic terms. The conclusion must therefore be that psychosocially determined psychiatric disorders also have a biochemical (neurochemical) substrate.

This conclusion implies that we cannot speak of "the" cause of a psychiatric syndrome, nor of therapy that focuses on "the" cause. The term "cause" covers a multifactorial concept and comprises two components, the first of which is the cerebral substrate that made the disturbed behaviour instrumentally possible. Let me add that I use the term 'substrate' not in its anatomical sense but to designate a series of related cerebral functional disturbances. For this component of the disease-cause-concept, I use the term "pathogenesis."[10,28]

Secondly, there are the factors which are responsible for the development of the cerebral substrate of the behavioral disorders. They may be material as well as immaterial factors. I call them etiologic factors. The material factors can be hereditary or acquired during life. An example of a hereditary factor is a marginal primordium of an enzyme system. The result may be that the capacity for synthetizing a given neurotransmitter has little margin and quickly reaches its upper limit. Acquired influences include, for example, cranial injuries, various forms of encephalitis, and the cerebral consequences of malnutrition. These can leave traces, as a result of which certain cerebral systems can later be abnormally easily disrupted.

The immaterial etiologic factors include psychogenic and psychosocial influences. Few investigators doubt that these factors can influence the function of extracerebral organ systems—transiently, for a long time, or perhaps even permanently.

Perhaps superfluously, I present three examples:

1. Emotions can measurably influence a variety of somatic functions: blood pressure, heart rate, rate of blood flow, quality of sleep, appetite, and many others.
2. A placebo effect—that is an effect of medication based not on the medication but on the expectations raised by it—is a powerful (if underrated) weapon in medicine. The placebo effect is not confined to medication but can be produced by medical interventions in general. For example, in the 1950s, ligation of the internal thoracic artery was successfully used in the treatment of angina pectoris. Comparison of a group of patients thus treated with a group of patients treated by a sham operation (performance of the entire surgical procedure except ligation of the artery in question) failed to reveal any difference in therapeutic effect between the two groups. The favourable result of the operation appeared to be based on a placebo effect.[11]
3. The lethality of various somatic diseases in widows and widowers is significantly increased during the first year after the partner's death. There is every reason to assume that this phenomenon is based on psychologic factors.[12]

Two points are insufficiently taken into account:

1. There can be no question of a direct influence of psychogenic and psychosocial factors on somatic functions. The brain is the intermediary in such processes. It is in the brain that the so-called transduction takes place: the "conversion" of experience (a psychological process) to changes in the central regulatory mechanisms of peripheral organ systems. This transduction process has received insufficient attention as psychosomatics. Psychological processes have been and are still being studied, and so are the end situations at the periphery. But the cerebral intermediate links are hardly considered.
2. Psychogenic and psychosocial factors exert an influence not only on central regulatory mechanisms of peripheral organ systems but also on

central behavior-regulating mechanisms. Animal experiments have supplied convincing indications in this direction. Yet there is a certain hesitance to integrate this fact into day-to-day psychiatric thought and practice. Hence, there is an almost irradicable tendency to omit psychotropic medication in the treatment of psychiatric disorders with an unmistakable psychogenic "load" or even to consider it contraindicated; and, on the other hand, to consider psychotherapy superfluous whenever there are signs of what is called "organicity".

THE RECIPROCAL RELATION BETWEEN BRAIN FUNCTION AND BEHAVIOR

Changes of behavior develop by virtue of changes in the state of the brain; in other words, neurochemical changes of necessity precede changes of behaviour. Inversely, a given behavioral state can in turn induce neurochemical changes. These disappear as the behavior ceases, or they may persist (for some time). An example of the latter: certain anxiety/aggression-inducing stimuli alter the uptake of noradrenaline in noradrenergic nerve terminals in the brain. This can lead to changes in the sensitivity of the postsynaptic receptors and, therefore, in the functional "responsiveness" of the system involved. Such changes in receptor function persist after the aversive stimulus and the corresponding behavior have disappeared.[13]

In this way a state of behavior (a) which, as such, is transient can influence the likelihood that, in future, stimulus constellation (b) will induce behavior pattern (c). Behavior and brain function reciprocally influence each other incessantly.

THE TWO CHANNELS OF THERAPY IN PSYCHIATRY

Etiology and pathogenesis are both components of "the" cause of a psychiatric syndrome. This observation has therapeutic implications, namely that, in principle, the therapy of psychiatric disorders can take place via two channels. First, an attempt can be made to normalize the disturbed brain functions (the cerebral substrate of the psychiatric disorder). This is usually done with the aid of pills. The other possibility is to attenuate, and possibly eliminate, the pathogenic input from the individual's inner world and from the environment. Attempts to achieve this are usually made by talking.

Psychopharmacotherapy and psychotherapy therefore focus on different components of "the" cause of disease. Both are in principle cause-oriented. There is no reason to hold that pills are an inferior substitute for talking. There is every reason to maintain that both are full-fledged therapeutic methods which can be used together and simultaneously in the same patient, because they are complementary.

I have twice used the qualification "in principle", thus indicating that the argument presented holds water so far as specific means (drugs) and (psychotherapeutic) methods are available for a given psychiatric syndrome. This applies only to a limited extent as yet. So far as psychotropic drugs are concerned, it applies only to vital depressions and psychoses. Very little has so

far been established concerning the specificity of various psychotherapeutic methods, because little pertinent research has been done. However, in my opinion, the actual state of affairs at this moment does not affect the essential truth of the argument presented.

PHARMACOTHERAPEUTIC AND PSYCHOTHERAPEUTIC DECONDITIONING

"Tablets" and "talking" bear a complementary relationship. Medication alone, without any effort to deal with the pathogenic input from the individual's inner world and from the environment, could at least have some transient success, but the relapse risk would be maximal.

Is the reverse also true? Is talking alone insufficient? Are tablets indispensable? I have postulated in the above that every disorder of behavior has its neurochemical substrate, which in turn can have been induced by (among others) psychogenic and psychosocial factors. May we not assume that the cerebral substrate and the corresponding psychiatric syndrome disappear when the pathogenic input is reduced, thus solely by talking? In some cases this certainly seems plausible, namely when mental decompensation acutely follows a psychotraumatic event in a personality of relatively firm structure. In such cases a limited series of "talks", in a clinical setting or otherwise, may be sufficient.

These cases, however, are relatively rare. Usually psychiatric syndromes result from a number of chronic elements, e.g., a disharmonious personality structure or a life situation which has long been disrupted. In such cases there is generally no longer any direct causal relation between psychotraumatic events and psychiatric syndrome. To phrase it more precisely, in such cases the psychotraumatic events only partly explain the development of the psychiatric symptoms. The morbid behavior has gradually become more and more deeply engraved over the years. It has become a reaction type, so to speak, that is ready for use and that is also easily triggered by situations and circumstances only remotely related to the original traumatizing situation(s). Finally, the symptoms can occur almost spontaneously. This applies to such conditions as symptom neuroses, psychosomatic disorders, and functional somatic symptoms.

It is an illusion to believe that harmonization of personality structure and/or life situation constitutes sufficient therapy in these cases. In addition, the learned dysfunctional behavior has to be unlearned. There are two ways to achieve this: behavior therapy and psychopharmacotherapy. In behavior therapy, the "unlearning" concept is taken literally. Psychopharmacotherapy can in principle be expected to reduce the facilitation (Bahnung), blocking the circuits responsible for the readiness with which dysfunctional behavior develops. This might be described as pharmacogenic deconditioning. Psychotropic drugs that can achieve this, are not available as yet. This is why the treatment of neuroses is still almost exclusively based on psychotherapy. This situation is determined by the present state of art; however, it is not based on principles. I venture to predict that in a not too remote future the treatment of neuroses too will benefit from developments in psychopharmacology.

Behavior therapy *is* available. In view of the above considerations, we may

expect that behavior therapy combined with psychotherapy should be superior to the use of either method separately. It is therefore surprising that the "either-or" principle is usually applied in determining indications.

ARRESTING A SELF-SUSTAINING DISEASE PROCESS

There are two other groups of psychiatric syndromes in which psychotherapy alone is probably not an adequate therapy: the group of the vital depressions and that of psychoses of the schizophrenic type. It is beyond doubt that these syndromes can be unleashed by all kinds of stress, including psychosocial. Once they are manifest, however, they often take an independent course, regardless of the factors that provoked them. It is true that these syndromes tend to improve spontaneously, but the duration of illness can be considerable. In these cases, agents capable of normalizing the cerebral substrate and thus abbreviating the duration of illness would be of great therapeutic value. Antidepressants and neuroleptics, we know, are agents able to effect this; and these agents have significantly improved the prognosis of recurrent vital depressions and recurrent psychoses.[9]

PHARMACOTHERAPEUTIC AND PSYCHOTHERAPEUTIC PREVENTION

There is a third field of mental-health care in which reliance should not be based exclusively on psychotherapeutic techniques: the field of prevention.

Psychiatric syndromes (depressions, psychoses, neuroses, addictions and many others) tend to be recurrent. There are few exceptions to this rule. This is not merely a question of recurrent or chronic psychosocial stress, but also of increased vulnerability to the decompensating effect of this stress. An effort at prevention should therefore be made from two sides: to reduce the psychotoxic load of the life environment and to reduce the individual's vulnerability. The latter goal one may try to achieve by psychotherapy, in an effort to stabilize the personality structure. However, the concept of (psycho)vulnerability can be viewed from a biologic as well as from a psychological angle. What imperfection of the brain causes certain behaviour-regulating systems to be so easily upset by various stressors? If biologic vulnerability could be reduced, a preventive goal of the first order would be achieved.

In this respect, important progress has been made in the groups of depressions and psychotic syndromes. The risk of relapse of psychoses of the schizophrenic type can be substantially reduced with the aid of long-acting neuroleptics.[14] Lithium has produced similar effects in recurrent vital depressions, both in the unipolar types (vital depressions only) and in the bipolar types, which combine vital depressions with (hypo)manic periods.[9] In the field of biochemical prevention, new developments are in progress, in particular, in the group of vital depressions. It has been demonstrated in a subcategory of patients with unipolar and bipolar depressions that a chronic deficiency of the neurotransmitter 5-hydroxytryptamine (5-HT; serotonin) in the brain is likely to play a role in increased vulnerability to depression in these patients.[15] These patients were consequently treated by oral administration of 5-hydroxytryptophan (5-HTP), a precursor of 5-HT, which in the brain is converted to 5-HT. This physiologic substance proved to have both a therapeu-

tic effect[9,16-18] and a prophylactic effect.[29,30] 5-HTP therefore is the first "aimed" prophylactic to be used in psychiatry. By "aimed" I mean aimed at a suspected pathogenetic component of a given psychiatric syndrome.

Perhaps superfluously, chemoprophylaxis is not a substitute for psychotherapeutic prophylaxis but supplements this adequately.

TABLETS AND TALKING

Psychotropic medication and psychotherapy form a sensible combination. This is primarily a theoretical conception, but it is supported by the facts. The facts derive from research done in patients with vital depressions and psychoses of the schizophrenic type, the only syndromes for which relatively specific psychotropic drugs are now available. By way of example, I mention the work of Hogarty and associates[19,20] and Golstein et al.[21] They studied the relative values of long-acting neuroleptics and individual and family psychotherapy in reducing the risk of relapse of schizophrenic psychoses. This risk was found to be lowest in patients receiving a combination of these treatments. Leff[22,23] demonstrated that the prophylactic effect of long-acting neuroleptics increases as the life environment of a patient is more stressful. This supplies an indirect argument in favor of combined treatment.

Comparable research results have been reported in the group of recurrent vital depressions. The prognosis for both duration of depression and risk of relapse was most favorable in those patients given combined treatment.[24] Lithium greatly reduces the risk of relapse of unipolar and bipolar depressions. When lithium prophylaxis is provided casually in a busy outpatient clinic, its results are less favorable than those obtained in a special outpatient setting known as a lithium clinic,[25] where pharmacotherapy is combined with supportive psychotherapy and social guidance.[26] Moreover, negative interactions between pharmacotherapy and psychotherapy have not been demonstrated.[24,27]

Tablets and talking form a sensible combination, at least in the diagnostic categories discussed here. To go even further, they form a necessary combination. Omission to use this combination is a serious disservice to the patient.

SUMMARY

Pharmacotherapy and psychotherapy of psychiatric patients are not given equal appreciation. Particularly in syndromes with marked psychogenic or psychosocial overtones, psychotherapy is regarded as the causal therapy par excellence. In these cases (the vast majority), psychotropic drugs are believed to have at best a symptomatic importance and to entail the risk of effacing the true causes. I consider this view to be as unfelicitous as it is wrong, because: (1) Psychosocially determined behavior disorders, too, have a neurochemical substrate; (2) It is by all means sensible to make an attempt to normalize behavior by correction of this substrate; (3) Normalization of the cerebral substrate with the aid of pharmacotherapy is no less a causal type of therapy than is reduction of the pathogenic input with the aid of psychotherapy; (4) This means that pharmacotherapy and psychotherapy are complementary, and that each separately is an incomplete therapy.

This argument is valid where psychotropic drugs with a relatively specific

effect are available, as they are for the vital depressions and psychoses of the schizophrenic type. In these groups of patients, empirical findings confirm the theoretical expectation formulated (4) above. It is postulated that, for the treatment of neuroses which has so far been based entirely on psychotherapeutic intervention, much is yet to be expected of future developments in psychopharmacology.

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