PSYCHODYNAMIC RELATIONSHIPS BETWEEN THE DEGREE OF ANXIETY AND OTHER CLINICAL SYMPTOMS[1]

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For a period of six years we have been studying anxiety under various experimental situations and also relationships between the quantitative degree of anxiety and the production and amelioration of other clinical psychiatric symptoms. These studies are a part of a broader investigation of the relationship between psychophysiological mechanisms and psychodynamics. In this particular phase of our work we were attempting to learn whether the pattern of symptom formation was similar under different types and degrees of stress.

METHODOLOGY

Several lines of research were used, material being gathered through the following techniques:

1. Intensive reconstructive psychoanalyses (12 patients)
2. Psychoanalytically oriented psychotherapy in combination with ataraxic drugs (175 patients)
3. Use of hallucinogenic drugs (mescaline and lysergic acid) (25 patients)
4. Use of intravenous chlorpromazine and promazine
   A. For the treatment of severely ill schizophrenic patients (35 patients)
   B. To block the effects of Mescaline or Lysergic Acid (25 patients)
5. Psychosurgical procedures performed on the conscious schizophrenic patient under local anesthesia
   A. During craniotomy proper (43 patients)
   B. During and immediately following prefrontal lobotomy (34 patients)

Most of the patients included under Group 5 were also exposed to techniques 3 and 4. A number of patients included under technique 2, initially, were treated with intravenous tranquillizing drugs (Group 4).

The diagnostic spectrum of psychiatric patients used in this study was very broad. All the patients treated by intensive reconstructive psychoanalyses could best be characterized as having psychoneuroses in combination with social and sexual maladaptation. Those treated with the combined brief psychoanalytically oriented psychotherapy-ataraxic drug technique included schizotypes in various degrees of compensation, patients with involutional depressions and patients with psychoneuroses. Only severely ill schizophrenic patients were exposed to techniques 3, 4 and 5.

DEFINITION OF ANXIETY AND TECHNIQUES OF EVALUATING DEGREE OF ANXIETY

In all phases of our studies, anxiety was defined for our purposes in the broadest possible aspect, namely, that it was a psychophysiological phenomenon which we considered synonymous with fear, in spite of whether the fear was generated by unconscious conflict or actual environmental threats whether these threats were biosocial, biochemical or exposure to intracranial operations under local anesthesia.

The definition was purposely very broad in its scope because as yet there are no clear-cut clinical biochemical or psychophysiological perameters available for the study of anxiety. Also in the acute experiment it is not feasible to attempt to differentiate between the fear of the experimental situation and the emergence of unconscious terrors, especially on a quantitative basis

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We found it helpful, indeed necessary, to divide and to analyze anxiety in terms of four components:

1. Motor component
2. Affectual component
3. Verbal component
4. Autonomic component

An attempt was made to quantitate the degree of intensity of each component as follows:

Motor Component

Palpation, passive and active movement used as a means of evaluation

Key:

(a) Calm—no restlessness, no tremor, no increase in muscle tonus

(b) Slight—slight increase in muscle tonus, slight restlessness, slight tremor

(c) Moderate—moderate increase in muscle tonus. Marked restlessness. Passive movement difficult

(d) Severe—thrashing about wildly, incessant movement

Affectual Component

Facial expressions; tone, quality and volume of voice used as means of evaluation

Key:

(a) Calm

(b) Slight

(c) Moderate—(patient obviously disturbed)

(d) Severe (patient in panic)

Autonomic Component

We could not accurately quantitate the degree of autonomic change. We measure vital signs and in some did psychogalvanometric recordings. Electroencephalographic studies were made in some of the psychosurgical cases and on some of the patients receiving hallucinogenic agents.

Verbal Component

Evaluated according to pressure of speech

(a) Normal conversational pattern

(b) Slight pressure of speech

(c) Moderately severe pressure of speech

(d) Extreme logorrhea

All of these components were then weighed together to give the final evaluation of the degree of anxiety present.

RELATIONSHIP BETWEEN MOUNTING ANXIETY AND SYMPTOM FORMATION

The results of the various research techniques mentioned above all appeared to indicate that there was a direct relationship between the quantitative degree of anxiety and the formation of other clinical psychiatric symptoms and signs (2, 3). The symptoms and signs such as phobias, obsessive-compulsive states, hypochondriasis, psychosomatic complaints, paranoid delusions, hallucinations, etc., appeared to be secondary defense mechanisms against the pressure of the mounting anxiety.

Twenty-one of the 25 patients, who received either mescaline intravenously or lysergic acid orally, demonstrated definite evidences of mounting anxiety before there were any other symptomatic expressions (Table 1). In seventeen of the 21 patients, motor restlessness and increased muscle tonus was the heralding indication of mounting anxiety. The various psychiatric symptoms began to manifest themselves usually only after there were definite alterations in the affectual component. Autonomic changes, where our measurements indicated a significant alteration, usually occurred simultaneously with a definite in-

TABLE 1

Mounting Anxiety and Symptom Formation

Pattern of Investigation	Number of Patients	Patients Manifesting Increased Anxiety Prior to Other Symptoms	
		Number	Percentage
Mescaline or Lysergic acid	25	21	84
Craniotomy under local anesthesia	43	40	93
Combined psychotherapy-drugs	112	91	81

crease in the affectual component. There were 3 patients in catatonic stuperous states who demonstrated autonomic changes before motor changes occurred.

A similar general pattern existed in the studies made on patients exposed to the stress of a large craniotomy performed under local anesthesia. Forty of the 43 patients studied during craniotomy manifested a definite increase in various components of anxiety before there were manifestations of other psychiatric symptoms. In this series of observations, as in the studies of the hallucinogenic agents, the motor component of anxiety was altered first in the vast majority of cases. Thirty-five of the 40 patients (88 per cent) who showed a definite increase in anxiety, prior to the appearance of secondary symptoms, demonstrated restlessness or increased resistance to passive movement before affectual or autonomic changes were elicited. In 31 of these patients the secondary symptoms appeared only after the affectual component was definitely increased.

In 112 of the patients, who were eventually treated by the combined psychotherapy-ataraxic drug technique, we were able to obtain very detailed histories, which, when analyzed from the standpoint of the relationship of anxiety and symptom-formation, convincingly indicated, in 91 cases (81 per cent), a definite mounting anxiety level prior to the onset of other clinical manifestations.

Another striking observation was that in patients exposed to, what may be called "maximum stress", (*i.e.* mescaline or lysergic acid, craniotomy under local anesthesia and patients seen in acute panic reaction from overwhelming social pressures) showed, in almost all instances, a diffuse pan-anxiety state by the time the affectual component became very obvious. All patients, at this point were reacting not only to the stress of the immediate situation, but to the emergence of unconscious conflicts which were released under the pressure of the mounting anxiety. The combination of the immediate and more remote traumas caused the crescendo into the pan-anxiety state. It was in this psychophysiological setting that other clinical symptoms made their debuts.

From these studies, the psychophysiological relationship between the pressure of anxiety and appearance of other psychiatric symptoms and signs, which we consider as being secondary defense mechanisms, seemed to be as follows: Initially a state of mounting anxiety was produced by an environmental stress (*i.e.* an internal or external biophysical stress or an external biosocial problem) with which the patient could not cope. This in turn stimulated, or perhaps permitted the emergence, of unconscious conflicts, originating from past traumas, this combination of stresses precipitating a diffuse pan-anxiety state. It was then that psychiatric symptoms, such as phobias, hypochondriasis, obsessive thinking, etc., appeared individually or in a succession, the exact symptoms and pattern of appearance being unique to each patient. As the anxiety mounted, more and more of these secondary defense mechanisms were brought into play, in an attempt to diminish, or perhaps serving as a buffer against the mounting anxiety. This pattern brought to our minds the mental image of an engineer attempting to stop the forward rush of a runaway locomotive by applying ever increasing pressure on the brakes. Eventually if these "mental breaking mechanisms" did not quantitatively decrease the level of anxiety, the patient's ego defenses were overwhelmed. In patients with a schizoid matrix, this overwhelming of the ego defenses, lead to impairment of reality contact with the patient being dominated by feelings of dissociation or dissolution or other bizarre aberrations. In a number of patients who had been studied previously with the hallucinogenic agents or who otherwise had been observed during one or more panic states, we were able to abort the

mounting anxiety by the use of intravenous chlorpromazine or promazine, thereby preventing the emergence of the symptom pattern that otherwise would have appeared.

Another phase of our studies concerned itself with the problem of whether individual patients have essentially the same sequence of symptom appearance in response to repeated bouts of marked anxiety developing secondary to severe stress (Table 2). In a study of 88 patients who were followed for more than one year, and who, during this period, had two or more periods where they manifested acute severe psychiatric flare-ups, 74 (84 per cent) had essentially the same sequence of symptom-formation during the repeated periods of stress. Thirty-eight of the 43 schizophrenic patients, who were studied while craniotomies were being performed under local anesthesia, had two or more episodes during which time there was a marked increase in the level of anxiety in response to the stress of the operations. Thirty-three (87 per cent) of these 38 patients showed essentially the same sequence of symptom appearance with each bout of anxiety. The number of symptoms manifested roughly seemed to depend upon the quantitative degree of anxiety on each occasion.

TABLE 2

Similarity of Symptom Appearance in Response to Repeated Bouts of Anxiety

Pattern of Investigation	Number of Patients	Patients Demonstrating Similar Response to Repeated Stress	
		Number	Percentage
Craniotomy under Local Anesthesia	38	33	87
Combined Psychotherapy-Drugs	88	74	84

RELATIONSHIP BETWEEN DECOMPRESSION OF ANXIETY AND SYMPTOM AMELIORATION

We observed that the degree of anxiety must be first decreased *before* a symptom or succession of symptoms disappears. It was noted, among patients successfully treated by means of the combined tranquillizer-psychoanalytically oriented psychotherapy technique, that although symptoms might remain for a period of days, after the level of anxiety was first decreased, there was less emotional impact associated with these symptoms. A given symptom or group of symptoms did not seem to completely disappear until the degree of anxiety decreased below a certain threshold which was unique for the individual patient. This transition period, in which a symptom was relatively denuded of its original emotional impact prior to the complete clearing of the symptom, was usually very striking. It is not usually so dramatically seen in patients treated with psychotherapy alone, because the process of improvement is much more gradual in this instance.

Our observations during the past few years have pointed up to us that the last symptom to appear, during the period when tension is mounting, is usually to first symptom to disappear when the level of anxiety is reduced (4, 5). Of the 175 patients treated with the combined psychotherapy-drug therapy technique, we found that the general pattern of disappearance of symptoms was the reverse of the sequence of symptom appearance in 144 patients (82 per cent). Similarly, in the study of 88 patients, followed for more than 1 year and who, during this time had 2 or more episodes in which they manifested severe psychiatric flare-ups, 66 (75 per cent) had essentially the same pattern of symptom amelioration following the repeated periods of anxiety decompression.

Of the 25 patients given intravenous chlorpromazine or promazine to block the effects of mescaline or lysergic acid 17 (68 per cent) showed a general reversal of the pattern of symptom appearance during the phase of anxiety amelioration.

TABLE 3

Decrease in Components of Anxiety in Relation to Fractional Pre-Frontal Lobotomy

	Component of Anxiety	Number of Cases
Quadrant No. 1	Motor	16
	Affectual	2
	Verbal	0
Quadrant No. 2	Motor	11
	Affectual	11
	Verbal	1
Quadrant No. 3	Motor	0
	Affectual	11
	Verbal	8
Quadrant No. 4	Motor	2
	Affectual	1
	Verbal	11

We also found that the various components of anxiety were not ameliorated simultaneously. While this often was noted following the intravenous administration of chlorpromazine or promazine to patients under severe stress secondary to mescaline or lysergic acid, it was demonstrated most dramatically during or immediately following the 34 pre-frontal lobotomies performed under local anesthesia (2, 6). For descriptive reasons, we had divided each frontal lobe into two halves, medial and lateral, making four quadrants in all that had to be cut. Twenty-nine of the 34 patients demonstrated a definite decrease in anxiety during or just minutes following the lobotomy. All 29 showed a definite lessening of the motor component, while in 25 the affectual component was ameliorated and in 20 the verbal component was changed. Table 3 shows that a greater amount of frontal lobe white matter had to be sectioned to cause a change in affect than was required to produce a change in muscle tension or motor activity. Similarly it appeared that an even larger leukotomy was necessary to produce a change in verbal content. We also found that the various clinical psychiatric symptoms (*i.e.* phobias, hypochondriacal reactions, obsessive thinking, etc.) did not begin to fade until the affectual component of anxiety was first significantly ameliorated.

Six patients had leukotomies performed while they were actively hallucinating. In five the hallucinations either improved or disappeared. It was clearly discernible that the improvements occurred only after there was first a marked decrease in anxiety. For example, three patients were hallucinating in all spheres and demonstrated very primitive structuring of their aberrations. When the motor and affectual components of anxiety had been greatly reduced, the tactile and olfactory aspects disappeared and the manifest content of the auditory and visual hallucinations was better organized. In two others, the hallucinations disappeared entirely even before the last quadrant had been cut.

Dreams behave in a similar manner. The combined psychotherapy-drug therapy technique offers an excellent opportunity to observe just what happens psychodynamically to dreams when anxiety is rapidly decompressed. The emotional impact of dreams was affected first. This was most clearly seen with nightmares and rage dreams. In the early phase of phenothiazine therapy, the patients continued to have the same dreams that were accompanied by marked terror or rage prior to the onset of treatment, but now the emotional impact was blunted. The manifest content was the same, but the dreams were denuded of rage or terror. After a few more days the manifest content of the dreams changed too. It appeared that it was not until the level of anxiety was decreased below a certain threshold, which was unique for a given patient, that the manifest content of the dream was changed. The same phenomenon can be seen during the course of a successful psychoanalytic therapy in the absence of drug therapy, in that patients who have shown significant improvement may often relate dreams with similar content to some that had nightmare quality early in

treatment but which in the advanced stage of treatment had lost their terror.

DISCUSSIONS AND CONCLUSIONS

In these studies patients were exposed to marked stress of one or more types; namely, biosocial, biochemical, neurophysiological and the stress of an intracranial operation performed under local anesthesia. In spite of the technique used to produce the trauma, the response to the threatening situation followed patterns that were similar in many ways. We believe that our results indicate an intimate and direct relationship between the quantitative degree of anxiety and the emergence of other clinical psychiatric symptoms and signs. The greater the stress, the greater the degree of anxiety; the greater the degree of anxiety the more symptoms and signs that are called into play.

We believe that our studies also show that the quantitative degree of anxiety must first be decompressed before there can be any amelioration of the various clinical symptoms. The different phases, through which the patients passed before there was psychic integration on a high level appeared to be the same whatever the therapeutic technique. Only the rate or reintegration and improvement appeared to vary with the different therapies. It was also striking to us that as the degree of anxiety repeatedly mounted, in response to repeated dangers, whether real or apparent, the pattern of symptom appearance tended to be more or less stereotyped for a given patient. Similarly, the repeated patterns of clinical improvement, in the individual patient, seemed to follow a trend.

The question then arises as to just how the various quantitative degrees and components of anxiety are related to other clinical psychiatric symptoms and signs. We believe that an explanation is available, if we consider the degree and components of anxiety and the other symptoms as the expressions of a hierarchical order of higher central nervous system functioning. We postulate that the manifestations of anxiety are expressions of a state of disequilibrium within the more primitive phylogenetically older physiological mechanisms of the brain. The various components of anxiety that present themselves clinically as the stress is increased might be considered as manifestations of disequilibrium as phylogenetically newer and higher subcortical mechanisms are called into play in an attempt to govern the failing more primitive neural defense mechanisms. It seems that it is not until these subcortical processes of neural integration are overwhelmed that the higher cortical regulatory mechanisms are challenged. Following the same frame of reference, the different psychiatric symptoms such as phobias, obsessive-compulsive states, and the like, might then be considered as reflections of disfunction at the highest level of neural activity.

The hierarchical concept of neural organization is certainly not new. Hughlings Jackson first proposed it is 1898 (1) in his attempt to explain the mechanisms of epileptic seizures. A large body of neurological and neurophysiological research, during the past half century, has given substance to the concept of a hierarchically ordered system of neural integration and to the dynamic interrelationship between all of the levels within the system.

The various psychotherapeutic techniques have contributed greatly to our understanding of normal and abnormal expressions of the highest level of neural activity. If one can conceive of dreams as being a manifestation of subcortical activity temporarily released from cortical control, then current psychiatry has also contributed to our knowledge of this neural phenomenon. However, a very broad hiatus still exists in our understanding of the psychiatric manifestations of the phylogenetically older neural mechanisms. We believe that further multi-disciplinary research directed toward such an understanding would

greatly enhance our appreciation of the dynamics of psychiatric illness.

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