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**EPISODIC
BEHAVIORAL
DISORDERS**

An Unclassified Syndrome

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EPISODIC BEHAVIORAL DISORDERS: AN UNCLASSIFIED SYNDROME

Russell R. Monroe

Classification of syndromes, which has had heuristic value for the general physician, is in current disrepute among psychiatrists and behavioral scientists. Even in general medicine, classifications have been devalued; for example, there are those who consider diabetes as an abstraction, since the physician is confronted with a diabetic patient rather than the disease "diabetes." Nevertheless, this abstraction is useful both to the physician in treating his diabetic patient, and to the patient who is thereby allowed certain attentions, without being labeled lazy, a malingerer, or hypochondriacal.

In psychiatry, labels have been less useful, and sometimes merely for social or political reasons, they are assigned to establish the social role "sick," without benefit to the person so labeled. Among professionals, there are two reactions to this: either to do away with labels entirely, or to strive for a classification of behavior with precise etiologic, prognostic, and therapeutic implications. Such a pragmatic classification depends upon careful phenomenological analyses of symptoms and signs over a period of time, with a syndrome ultimately verifiable by precise laboratory methods. It is my contention that for most deviate behavior this disease model should not be

discarded but, instead, refined and developed.

Many of our past failures in establishing a clinically useful classification result from neglect of the course of illness. Identifying the syndrome here discussed, episodic behavioral disorders, depends less on the manifest symptoms, but rather on the illness course—the symptoms occurring episodically with equally precipitous appearance and remission. I suggest that this characteristic course often reflects a neurophysiologic mechanism which has specific etiologic, prognostic, and therapeutic implications, despite the otherwise diverse manifestations of the syndrome. Episodic behavioral disorders should be considered as generically equivalent to such currently utilized diagnostic terms as organic brain syndrome, neurosis, personality disorder, “functional” psychosis, etc.

The episodic behavioral disorders include subgroups: (1) those patients demonstrating disordered acts, referred to in the literature as “acting on impulse,” “impulse neurosis,” “irresistible impulse,” or “acting out,” which I designate *episodic dyscontrol*; and (2) episodic, psychotic, sociopathic, neurotic, and certain physiologic reactions, which I designate *episodic reactions*. The varied psychopathology covered by these previously poorly defined syndromes has been repeatedly described in the literature, although never officially labeled or included in the standard diagnostic manuals. However, the international classification (ICD-8) uses such labels as “reactive

excitation," "reactive confusion," "reactive psychosis unspecified," "acute schizophrenic episodes," "depersonalization neurosis," "explosive personality," and "episodic drinking"; these probably concord with what I designate *episodic behavioral disorders*.

The common features of such diverse psychopathology are precipitous onset of symptoms, equally abrupt remission, as well as the tendency for frequent recurrences. These episodes represent interruptions in the life style and life flow of the individual, and involve either a single act or short series of acts with a single intention (episodic dyscontrol), or more prolonged behavioral disturbances with complex psychopathology and multiple dyscontrol acts (episodic reactions). The value in identifying these syndromes is the possibility of providing more effective treatment.

A brief discussion of psychopathology in general may clarify this point. During the course of human development, the individual's interaction with both his external and internal environments elicits an increasingly complex repertoire of behavioral responses, some successful, others not. Through this trial and error, aided by parental guidance and societal attitudes, the maturing individual learns that certain behavioral patterns are more successful than others; that is, more likely to be rewarded or reinforced, or at least more effective in avoiding pain and punishment. These patterns tend to be used repeatedly in similar or related situations until characteristic

response patterns become established; these give the individual a unique “personality” or “character” and provide coherency and predictability to his “life style.” Thus, close associates can usually predict how an individual will respond in a given situation. These consistent established patterns of psychological adaptation may be adaptive (healthy or normal) or maladaptive (disordered behavior, disease, or illness). Maladaptive patterns, identified as personality disorders, neuroses, or psychoses, all have an insidious development, a monotonous repetition in life patterns, as well as a persistence which often defies even heroic therapeutic efforts towards change. Even when acute exacerbations occur under stress, the symptoms are congruent with the previous life style and life history of the individual. For example, the obsessive character develops obsessive symptoms, the cyclothymic individual develops a depression or hypomania.

In episodic behavioral disorders the symptoms abruptly interrupt both the life style and life flow of the individual, with the disturbed behavior appearing as a break between the past and the future. The behavior is both out of context for the situation and out of character for the individual. Occasionally, episodic acts are adaptive; for example, the spontaneous act that may represent the unique contribution of the genius or the man of action. Usually, however, the abrupt precipitous acts are based on primitive emotions of fear, rage, or sensuous feelings, without concern for the effect on the immediate environment or the long-term consequences to the actor or

society, and are either self- or socially destructive. These acts are often disinhibitions of behavior (in the motor sense), although there are also abrupt maladaptive responses that are inhibitions of action when action is necessary. Identifying and treating individuals with these disorders has particular social relevance, especially when episodic behaviors are sadistic or bizarre crimes, suicidal attempts, and aggressive or sexual acting out. Figure 11-1 summarizes these generalizations.

As mentioned previously, episodic disinhibition is further divided into two classes: episodic dyscontrol representing an abrupt single the pragmatic implication that anticonvulsant medication may aid in the treatment of such patients.

A careful phenomenological analysis, with a detailed history, is usually sufficient to distinguish the characteristic episodic behavioral disorders from chronic, insidiously developing behavioral deviations. However, at times it act or short series of acts with a common intention carried through to completion, with at least partial relief of tension or gratification of a specific need; and episodic reactions representing a more prolonged interruption in the life style and life flow of the individual, but also characterized by a precipitous onset and abrupt remission, as well as a tendency to recur. While episodic behavioral disorders may be superimposed on chronic, persisting psychopathology in other cases, behavior between episodes is relatively

normal. The abruptness of appearance of symptoms, particularly when of short duration and accompanied by confusion and other signs of clouding of sensorium, strongly suggests a basic epileptoid mechanism involving circumscribed areas of excessive neuronal discharge. Neurophysiologic data support this hypothesis with may be difficult to differentiate episodic behavioral disorders from the occasional acute “decompensation” to stress, or a persistent, impulsive life style. When differentiation is difficult, supplementary psychodynamic and neurophysiologic analyses may aid in making this discrimination. First, we will consider in detail the phenomenologic and psychodynamic aspects of episodic dyscontrol and reactions, and then we will consider the neurophysiologic differentiation.

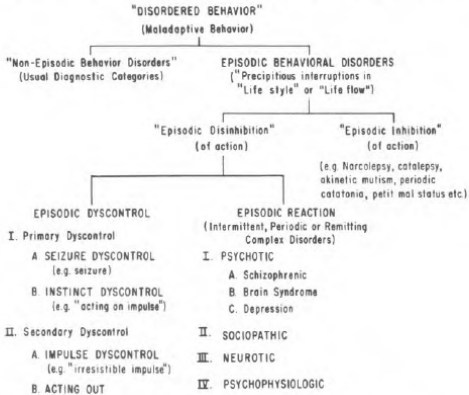


Figure 11-1.
“Disordered behavior.”

Episodic Dyscontrol

It is important in evaluating dyscontrol acts to establish the specificity of the drives or urges behind the act, the co-ordination and complexity of the motor behavior during the act, and the appropriateness of the motives or goals of the act. In doing so, one can identify more primitive dyscontrol acts (primary dyscontrol), and more sophisticated or complex dyscontrol acts (secondary dyscontrol). Primary dyscontrol is further divided into two levels, those of seizure dyscontrol and instinct dyscontrol, while secondary dyscontrol is divided into impulsive dyscontrol and acting-out dyscontrol.

Primary Dyscontrol—Seizure Dyscontrol

This is the most primitive dyscontrol act and is characterized by intense, indiscriminate affects, chaotic and uncoordinated motor patterns, and indiscriminate selection of the object acted upon, often the person closest at hand. Little specific need gratification is apparent, although there is usually tension reduction. Often the intention of the act is completely lost and the effectiveness of the act very limited; in this way, seizure dyscontrol can be used consciously or unconsciously to abort higher level dyscontrol acts, where the intentions are obvious and at the same time unacceptable to the actor. A typical example of seizure dyscontrol is the post ictal confusional state often manifested by a mixture of fright-fight responses, as well as demands for closeness.

A fourteen-year-old boy patient had grand mal epilepsy since the age of eight months. The attacks most often occurred at night. When they occurred during the day, they were associated with extremely aggressive outbursts. Rarely were there aggressive episodes without a prior seizure. Typically, the person attacked was the individual closest at hand, who in some way had physical contact with the patient. For instance, the father trying to loosen the patient's belt following a grand mal seizure was assaulted by the boy who the moment before appeared unconscious. The boy, despite his small size, picked up his much larger father, threw him against the wall and then beat him unmercifully. This was sometimes accompanied by the indiscriminate breaking of furniture. The boy's behavior was so frightening that it led to hospitalization, where the aggressive outbursts continued, terrorizing other patients and ward personnel. The boy himself would say, following such episodes, "I cannot understand why I become so destructive, I don't know what gets into me." When asked as a young child by a psychiatrist what three wishes he would like, the last wish was "to be a good boy and not lose control."

Primary Dyscontrol—Instinctual Dyscontrol

This is a higher level of dyscontrol act in which the effects are more clearly differentiated (e.g., fear, rage, sensuality), and more effectively gratified. The motor pattern, although lacking in subtlety, is nonetheless

efficient and coordinated. The object acted upon has at least simple associative links with past experiences; therefore, it is selected with some degree of appropriateness.

In both instinct and seizure dyscontrol, the behavior is characterized by an explosive, immediate response to an environmental stimulus that represents a “short circuit” between stimulus and action. The onlooker is startled at the individual’s behavior, and the individual may be equally surprised, saying, “I just did it, I don’t know why.” There is the experience of “having executed a significant action. . . without a clear and complete sense of motivation, decision or sustained wish, so that it does not feel completely deliberate or willfully intended.” The behavior is usually described by the actor as a whim, impulse, or sudden urge. Judgment during such acts is reckless or arbitrary, and the act more concerned with the actor’s need for gratification than the characteristics of the object acted upon. As an example, another patient would aggressively attack people. However, unlike the previous patient, she was not particularly confused nor amnesic for the attack itself, and the person involved was not necessarily the individual closest at hand, nor was the attack precipitated by physical contact. Instead, the object of her aggression was always male and, in retrospect, the person had mannerisms of speech reminiscent of her father. Further confirmation of the associative link with her father occurred during projective testing where, whenever the stimulus card elicited associations related to her father, she

would jump up, tear up the card that had provoked her, and then be at a loss to explain this precipitous act. There was no evidence for altered consciousness, except for the misidentification of the object attacked. Her response was stereotyped, as was her characteristic pattern for controlling urges; that is, obsessive-compulsive behavior.

EPISODIC DYSCONTROL

		PRIMARY DYSCONTROL		SECONDARY DYSCONTROL			
		SEIZURE	INSTINCT	IMPULSE	ACTING-OUT		
PHENOMENOLOGIC DIFFERENTIATION		NO DELAY BETWEEN STIMULUS AND RESPONSE		DELAY BETWEEN STIMULUS AND RESPONSE		PHENOMENOLOGIC DIFFERENTIATION	
		UNINHIBITED ACTION		---TRANSITION---	INHIBITED ACTION		
		UNCOORDINATED ACT	---TRANSITION---	SOPHISTICATED	COORDINATED ACT		
PSYCHODYNAMIC DIFFERENTIATION		TENSION RELIEF	DIRECT NEED GRATIFICATION	INDIRECT GRATIFICATION		PSYCHODYNAMIC DIFFERENTIATION	
		INHIBITED REFLECTION		EXCESSIVE REFLECTION			
		INHIBITED INTENTION	---TRANSITION---	CONSCIOUS INTENTION	UNCONSCIOUS INTENTION		

Figure 11-2.
Episodic dyscontrol.

Figure 11-2 summarizes the phenomenological characteristics of the primary dyscontrols where there is little or no delay between the impulse and act and undisguised, primitive motives. The two levels of primary dyscontrol can be differentiated by the uncoordinated motor behavior and the simple relief of tension apparent in seizure dyscontrol while instinctual dyscontrol is more coordinated with direct need gratification.

Secondary Dyscontrol—Impulsive Dyscontrol

Secondary dyscontrol, that is, impulse dyscontrol and acting-out dyscontrol, are difficult to differentiate phenomenologically from instinct dyscontrol. But in secondary dyscontrol, if an immediate precipitating event exists, it seems spurious or unimportant, and the motivational aspects of the behavior are hidden or bizarrely elaborated. For this reason, impulse dyscontrol and acting-out dyscontrol can be better characterized at the psychodynamic level. To do this, we must first understand the short circuit between the impulse and the act characteristic of primary dyscontrol but not characteristic of secondary dyscontrol.

It is precisely the short circuit, or action without premeditation, that results in the maladaptive quality of the seizure and instinctual acts described above. Such precipitous behaviors usually mean that the action has not been corrected by reflection on past experiences or anticipation of future consequences. Because of this, the act is usually self-destructive or self-defeating for the individual, or appears antisocial to the onlooker. However, careful evaluation of some precipitous acts, which appear unpremeditated and otherwise indistinguishable from the instinctual acts, reveals considerable premeditation. This is obvious if the premeditation is conscious and willingly reported by the patient (impulse dyscontrol), but at other times the premeditation remains unconscious and can only be inferred on the basis

of other data (acting-out dyscontrol). When the premeditation is conscious, the patient usually reports long periods of wavering and doubt about whether he should act or not, with mounting tension, as well as conscious attempts, often bizarre, to resist the impulse to act. Hence, in legal jargon this has been labeled the “irresistible impulse.” As an example:

In one patient, impulse dyscontrol occurred following real or imagined rejection. She would become increasingly irritable and hyperactive, and report mounting tension with waves of hopelessness followed by conscious urges to be aggressive, and then attempts to control it. However, such periods almost always culminated in an explosive act, even though during the act itself she might deflect her behavior. For example, following rejection by her mother, she began ruminating about choking her infant sister. She attempted to resist this by locking herself in her room, but the tension became unbearable. She described this tension as a sensation of warmth or flushes traveling over her face and head with a “sense that everything was closing in on me.” She then suddenly broke out of the room, grabbed her sister around the neck and began furiously choking her. In the middle of the act, she dropped the child and ran to her mother to confess. Other times, the act was carried through to completion; for instance, she would drown, torture, or otherwise harm animals, destroy furniture, or occasionally indulge in self-mutilative acts. In each instance, however, there was considerable conscious premeditation as to whether she should succumb to the impulse, with

attempts to resist the impulse through bizarre control mechanisms.

Secondary Dyscontrol—Acting-Out Dyscontrol

In acting out, despite careful evaluation, the individual denies any awareness of premeditation, yet the objective observer can only assume that the eliciting event has been separated by considerable time from the dyscontrol act. The act itself is so patently inappropriate for the situation, so out of character and so inadequately explained by the actor, one can only conclude that the act is largely determined by unconscious motives to solve repressed conflicts; this I call acting-out dyscontrol. As an example, one patient was a docile adolescent girl very much attached to her father. Without any conscious goal to her behavior, she began to pawn her belongings and save money several months before acting out. Supposedly on impulse, she flew to Puerto Rico and upon her arrival at the airport met a policeman whom she married three days later. The precipitating event preceded this act by considerable time. Her father and she had become estranged. The father, obviously jealous of her adolescent boyfriends, had become harshly punitive and restrictive of her freedom. Disconcerted by angry confrontations with her father, she would retreat to her bedroom and lock herself in. Several weeks before she ran away, the father had broken down her door and refused to let her replace the lock. Therapeutic sessions revealed that she was running from a threatened incestuous encounter with her father. On her trip, she found her

own sexual partner, an older policeman who, as was revealed through her associations, was a father substitute.

In summary, then, the differentiation between primary and secondary dyscontrol is the lack of premeditation in the former, and presence of this premeditation, either conscious or unconscious, in the latter. The acts themselves may appear equally explosive or precipitous to the casual observer, but only in the primary dyscontrol is there a true short circuit between the impulse and the act.

Psychodynamics of Primary and Secondary Dyscontrol

It will elucidate other dynamic differences between primary and secondary dyscontrol if we examine the mechanisms of delay between a stimulus and the response. We can identify two facets to this delay. The first is designated “reflective delay” (often referred to in the psychoanalytic literature as “thought as trial action”), which is the time necessary for establishing the uniqueness or familiarity of the stimulus by associative connections with past experiences; the time necessary to contemplate alternative courses of action; the time necessary to project into the future and predict the outcome of alternative actions. The second facet is “choice delay,” which is only possible following the reflective delay. Choice delay is a decision to postpone immediate action or gratification for long-term rewards; that is,

“biding one’s time.” This choice delay is absent in the extractive sociopathic individual who gives in to his urges in seeking immediate gratification, consequences be damned, even though there may have been careful appraisal of the situation and realization of the possible long-term consequences. This sociopathic individual has often been labeled in the literature as an “actor outter,” but by the definition here proposed would not exemplify episodic dyscontrol. To fit our definition, the reflective delay either must be absent (with the associated absence of choice delay), as occurs in primary dyscontrol, or the reflective delay must be severely distorted, as in secondary dyscontrol. The reflective delay depends upon a complex interaction between affects, hindsight and foresight, reason, discrimination and appropriate generalizations, as well as conscience mechanisms. If any of these is significantly deficient or inappropriately dominant, the whole process may fail, resulting in a total absence of reflective delay, leading to primary dyscontrol and a concomitant absence of choice delay, resulting in action that seeks immediate need gratification, with no concept that there is even an alternative. This is a true short circuit between stimulus and response, characteristic of primary dyscontrol.

On the other hand, in secondary dyscontrol, the conscious or unconscious premeditation and the delay between the true stimulus and the act indicate the extent of reflection, as well as the ambivalent vacillating attitudes towards the choice of either succumbing to or restraining the

impulse. By and large, individuals showing secondary dyscontrol are, in their general life style, overly inhibited and at some level aware of the true, even though neurotic, intentions of their action. The act itself then, is more likely to represent a rebellion against overly strict conscience mechanisms or a devious substitute gratification of forbidden, unacceptable impulses, such as the explosive act in the overly controlled obsessive character or the hysterical patient's extramarital affair during her analyst's vacation.

Another dynamic consideration of episodic dyscontrol is that "urges overwhelm controls," but this has operational value only if we can determine where an individual falls between the extremes of excessively strong urges overwhelming normal control mechanisms on one hand, and normal urges uncontrolled by weak or deficient inhibitory mechanisms on the other. The traditional psychiatric view stresses weak control mechanisms. However, my view, supported by growing neurophysiologic evidence, is that sometimes intense dysphoric affects associated with excessive neuronal discharges in the limbic system overwhelm even normal control mechanisms. To identify this group where "urges overwhelm normal controls" is particularly important for planning an appropriate therapeutic regimen, because the goal becomes not so much to develop stronger inhibitory mechanisms, but in some way to blunt or neutralize the intense dysphoria. As discussed later, perhaps the most effective way to do this is with drugs.

Another very important dynamic consideration in planning treatment depends upon the patient's "retrospective self-evaluation." In episodic dyscontrol, during the act itself, the behavior is ego-syntonic, in that it is an abrupt, often explosively quick act, carried through to completion with relief of tension or need gratification. There is no procrastination or doubt about the action. However, in retrospect, often the actor sees the act as ego-alien. This is particularly true if the behavior represents an underlying epileptoid rather than a motivated mechanism. The individual's retrospective recognition of the act as ego-alien considerably facilitates any psychotherapeutic endeavor.

However, in other instances, responsibility for the dyscontrol act is defensively denied (e.g., amnesias), projected, or rationalized so that one can only assume the behavior is recognized as ego-alien at the unconscious level. This defensive behavior, of course, further complicates psychotherapy. Occasionally, one sees a patient who is completely unconcerned by the dyscontrol acts, no matter how much anguish such acts cause others. In these instances, the acts are truly ego-syntonic, both at the time the act is committed and also in retrospect. Even in this instance, if the acts were truly episodic and not just a way of life, this patient would represent an example of episodic dyscontrol. This group presents the most difficult psychotherapeutic problem, because their deviant behavior causes so little personal discomfort. Nevertheless, if the truly egosyntonic dyscontrol acts are also episodic, that is,

an interruption in the usual life style and life flow of the individual, and not just a “dyscontrol way of life,” appropriate therapeutic motivation can be developed.

The “dyscontrol way of life” has been described by Shapiro. These individuals truly demonstrate the “alloplastic readiness to act,” and may have the appearance of episodic dyscontrol, but closer scrutiny reveals such behavior as a waxing and waning of a persisting pattern, the intermittent quality representing need arousal alternating with satiation. This group represents a failure in choice rather than reflective delay, and is not truly an example of episodic dyscontrol.

Finally, one must consider the intentions of the act (Figure 11-2). In acting-out dyscontrol, the intention is thoroughly disguised, usually representing the symbolic fulfillment of a forbidden impulse. On the other hand, in impulse dyscontrol and instinct dyscontrol, the true intention is either directly expressed or only superficially disguised and rationalized. For example, the adolescent boy in the throes of an oedipal renunciation kills his mother’s lover.

At the most primitive level of episodic dyscontrol (seizure dyscontrol), the act is so diffuse and uncoordinated that it becomes totally ineffective, hence any unacceptable intentions are blocked. Some patients seem to use

this regressive seizural act as a substitute for more effective but unacceptable intentional acts, substituting tension reduction for a more specific (but unacceptable) need gratification. This is one explanation for the frequently reported clinical observation that with an increase in seizures there is a decrease in other dyscontrol acts. Likewise, patients manifesting the episodic disinhibitions discussed in this chapter frequently show episodic inhibitions of actions (Figure 11-1). These, too, protect them from the self-destructive consequences of the dyscontrol acts.

Episodic Reactions

Before turning to the possible neurophysiologic mechanisms behind the episodic behavioral disorders, we must consider the second large subgroup, the episodic reactions, which were defined as more prolonged interruptions of the life style and life flow of the individual, characterized by multiple dyscontrol acts, as well as other psychotic, neurotic, sociopathic, or physiologic symptoms. What the episodic reactions share in common with episodic dyscontrol is the precipitous onset and equally abrupt remission of symptoms, which in turn reflect a common etiologic (epileptoid) mechanism in many of the patients suffering from these disorders. Even though the symptoms in the episodic reactions may be prolonged for weeks or months, the possibility of epileptoid mechanisms in these more prolonged disorders should be considered. Data available on subcortical recordings in man have

demonstrated prolonged but circumscribed excessive neuronal discharges in the limbic system, associated with diverse psychiatric symptomatology. It is for this reason that both episodic dyscontrol and episodic reactions are grouped under episodic behavioral disorders.

Episodic Psychotic Reactions

Many patients showing episodic reactions manifest symptoms that are characteristic of schizophrenia. Most patients labeled as having remitting, intermittent, atypical, or reactive schizophrenia are examples of this episodic psychotic reaction. Some of these patients show clouding of sensorium, difficulties in orientation or identification, and a partial memory loss for the episode—symptoms suggestive of an acute brain syndrome rather than schizophrenia. However, their schizophrenic behavior so overshadows these sensorial defects that the possibility of a toxic reaction is either overlooked or disregarded, particularly in the absence of an obvious exogenous toxin. This disturbance in awareness undoubtedly reflects an underlying excessive neuronal discharge or epileptoid mechanism. It is this group of atypical psychotic patients with clouding of sensorium that Meduna designated as “oneirophrenia” and Mitsuda as “atypical” psychosis. Rodin discussed such a syndrome which, although phenomenologically related to schizophrenia, seemed etiologically closer to epilepsy, and proposed the term “symptomatic” schizophrenia. Vaillant emphasized the recurrent quality of the disturbance

and the frequent shift in diagnosis from schizophrenia to manic-depressive psychosis in an eighty-five-year retrospective study of remitting schizophrenias. Although this group of patients ultimately become chronic, for long periods they managed independent successful lives. Altschule and Williams observed that episodic schizophrenic symptoms with confusion occur with regular periodicity associated with the menstrual cycle in some women.

The epileptoid mechanism behind many of these episodic psychotic reactions is further emphasized by studies where extensive clinical EEG's were obtained on patients before, during, and after psychotic episodes. These studies suggested at least two types of epileptoid psychotic reactions—one correlated with centrencephalic epilepsy, either petit mal status in children or a prolonged post ictal response in adults, characterized by torpor, apathy, confusion with minimal hallucinations and delusions. A second type of psychotic reaction was correlated with focal, particularly temporal lobe, abnormalities, and characterized by florid psychotic behavior with mixed manic-depressive or schizophrenic symptoms and varying degrees of clouding of sensorium. It has also been observed that these atypical psychotic patients often have a history of epilepsy, a low convulsive threshold, and activated EEG abnormalities.' Because these patients show the clouding of sensorium and disorientation characteristic of the acute brain syndrome, I chose to designate them as manifesting an episodic brain syndrome rather

than episodic schizophrenia.

Undoubtedly some episodic psychotic reactions are not determined by epileptoid mechanisms. Experience during World War II suggests that the “three-day schizophrenia” represents an individual’s adaptive potential to protect himself against extreme external stress. Such behavior is seen following natural catastrophes, such as earthquakes, floods, fires, etc., and under the official nomenclature would be designated as disassociative states. Although there is usually complete amnesia for these episodes and, in fact, during the episode the individual may be confused about his own personal identity, this disorientation can be readily differentiated from the clouded sensorium of the episodic brain syndrome. If the individual shows typical schizophrenic symptoms with a clear sensorium, I chose to designate the syndrome as an episodic schizophrenic reaction. The dual personality or split in orientation that occurs in hysterical disassociative states is distinguished from both the episodic brain syndrome and the episodic schizophrenic reaction.

One patient showed a true episodic schizophrenic reaction without any clouding of sensorium. She had a change in personality two weeks before admission to the hospital. Previously a devoted mother, she suddenly wanted to give up her children for adoption, and became so physically abusive towards them that she was hospitalized for the children’s safety. At the time

of hospitalization, she showed press of speech, incoherency, and poorly organized delusions that she was under some mysterious control from the outside and that unidentified enemies were going to destroy her family. She had auditory hallucinations of a sexually accusatory nature. The delusions and hallucinations disappeared within four days and within a week she had full insight regarding her bizarre behavior and illogical ideas, remembering clearly all that transpired during this episode.

Another patient on the other hand showed considerable clouding of sensorium during her acute psychotic episodes. Problems had been building up from the preceding year; the patient reported that at times “thoughts were racing through my mind and I couldn’t control them.” The family noted mild referential ideas. Two weeks before admission, her symptoms became florid. She was confused, disoriented, believed she was being influenced by mental telepathy, changed into a mulatto and that her husband was Eichmann. She identified the hospital personnel as persecutors, thought the hospital itself was a concentration camp, was disoriented as to date, time, place, and situation, and otherwise gave evidence of an acute brain syndrome. However, even at the height of the disorder, there were periods when she paused with considerable perplexity asking, “Why is everything so different?” At other times, she had a fleeting recognition that something was wrong with her. Two weeks after hospitalization, her sensorium was clear and she spontaneously said, “I haven’t imagined anything for the past four days.”

Episodic Psychotic Depressions

In this group of disorders, intense depression occurs and remits precipitously and unexplainedly, sometimes lasting for only a few hours, but often lasting for days or weeks. It is distinguishable from the usual psychotic depression, not only by the abruptness of onset, but also by less motor retardation, less conceptual elaboration of guilt and inferiority feelings, and more anxiety. The patient often refers to these episodes as “attacks,” and describes the depression as coming in waves, as if being engulfed in the ocean, or smothered in a cave, or surrounded by flames, sensations which occasionally occur as auras of typical epileptic seizures. In fact, some patients who develop epilepsy late in life have had such depressive episodes before the appearance of seizures, suggesting the likelihood that such depressions were prodromal symptoms of typical epilepsy, which preceded the appearance of convulsions by months or even years.

A patient would describe depressive episodes as if they were auras, saying that the sensation was like a wave overwhelming him. This would be so intense that he would have the visual sensation of being engulfed by flames or swallowed by the sea. At the same time, he would develop somatic preoccupations that he had a brain tumor, liver disease, heart trouble, or arthritis, all of which had some basis in reality. The sensations were so overwhelming that he contemplated suicide and made dramatic appeals for

hospitalization. Some months later, at the age of forty-four, he had his first grand mal seizure. After a second seizure and EEG evidence suggesting temporal lobe epilepsy, he was placed on a Dilantin-Mysoline combination, which controlled his seizures as well as the severe depressive episodes.

Episodic Physiologic Reactions

In view of the association of disturbing somatic sensations as an aura to typical epileptic seizures, it is not surprising that many of the episodic reactions are characterized by episodic hypochondriasis. In fact, Ging et al. report a significant relationship between paroxysmal EEG abnormalities and multiple physical complaints as scored on the MMPI; this supports Ervin et al.'s hypothesis that the frequency of hypochondriasis found in temporal lobe epilepsy patients might be a consequence of ictal visceral sensations these individuals experience. Often these episodic complaints are not hypochondriacal but a manifestation of true physiologic disturbances. Shimoda studied 2,500 patients with episodic somatic disturbances and found a high incidence of paroxysmal EEG abnormalities, which suggests the importance of an unrecognized group of disorders associated with an ictal phenomenon, the episodic physiologic reactions. Such disorders may involve almost any system and may lead, for example, to multiple laparotomies. From the clinical description of Shimoda's patients, many appear similar to what Reiman labeled "periodic disease." Also, the controversial "14 and 6 positive

spike” syndrome manifests both episodic physiologic reactions and occasional impulsive, aggressive acts. Deutsch’s fifty-year follow-up of Freud’s “Dora,” the patient to whom Freud first applied the concept of “acting out,” indicates that she apparently suffered from an episodic physiologic reaction, including migraine, coughing spells, hoarseness, palpitations, and multiple gynecological complaints, which kept “everyone in the environment in continual alarm.”

A thirty-three-year-old single female patient has had literally over one hundred hospitalizations starting at age five, and had undergone five medical hospitalizations during the three years prior to her present admission. Always, her complaints were severe, but the cause of the disability unclear. The first two times she was hospitalized for urinary retention, double vision, puffiness of the face, recurrent fever, and headaches. The third time she was admitted in an unresponsive state, supposedly having taken an overdose of barbiturates. However, the blood barbiturate levels were not significant and the patient denied any suicidal attempt. The coma was never adequately explained. Two months following this, she was again admitted, but this time for an actual suicide attempt wherein she had taken an overdose of barbiturates. Followed carefully on an outpatient basis, it was noted that she not only had transitory episodes of confusion, agitation, and depression, but also marked fluctuations in weight, diplopia, transitory slurring of speech, and unsteady gait. She improved considerably when on a carefully controlled

regimen of Dilantin.

Episodic Neurotic Reactions

In discussing episodic dyscontrol, we have mentioned the dysphoric affects that accompany the dyscontrol acts. At times, this dysphoria, although appearing precipitously and remitting in the same way, persists for a significant period of time, either without dyscontrol acts or with multiple dyscontrol acts which do not relieve the tension. We have already described such depressive episodes, but there also may be typical attacks of anxiety, acute rages, and even sexual excitement with neurotic defense mechanisms. The pragmatic value of making these observations is the possibility that these, too, may be epileptoid phenomena that will respond to a specific therapeutic regimen, including the use of anticonvulsants.

An adolescent chronic schizophrenic boy patient would be suddenly overwhelmed by phobias, particularly fear of the dark, being alone, being on the street or among crowds, without any particular change in his schizophrenic symptoms. At such time, he would come to the hospital begging for admission. These experiences would be transitory, sometimes lasting only a few hours or at most a few days, clearing up again with no particular change in the underlying chronic schizophrenia.

Another patient was admitted to the neurological service in status

epilepticus. After this terminated, she was transferred to the psychiatric service, because she developed blindness, hemiparesis, hemianesthesia, and aphonia. These symptoms cleared rapidly under minimal reassurance. However, she was considered to be representative of an episodic conversion syndrome, because these symptoms occurred repeatedly when she was under stress, with the varied conversion symptoms either occurring simultaneously or in rapid sequence.

A third patient, a very conscientious, prudish mother and respected member of the community, would periodically experience fugue states during which time she adopted a new name and the identity of a wild, promiscuous single girl. These changes in personality were sometimes transitory, occurring several times a day, and at other times persisted for two to three days. They occurred in clusters, sometimes following obvious external stress, and at other times seemingly unexplained. She also manifested episodic physiologic symptoms.

Episodic Sociopathic Reactions

Social values, to have meaning, depend on a stability and continuity in the world, so that antisocial behavior, in the broadest sense, is typical of most episodic behavioral disorders, because frequent breaks in life style and life flow leave the patient's world discontinuous and inconstant. For this reason,

the diagnosis of the episodic sociopathic reaction must be made carefully, and should be limited to the occasional patient who shows “bursts” of sociopathic behavior which completely overshadows other episodic behavior, such behavior being totally out of character for the individual, except for brief and infrequent intervals, yet sustained enough so that we cannot consider it as merely a dyscontrol act.

One patient, a twenty-seven-year-old married housewife, demonstrated momentary blackouts, periods of slow, hesitant speech, and episodes where she would impulsively break up furniture or destroy her belongings. The sociopathic episodes involved a period of mounting tension followed by sustained promiscuity, lasting for weeks or months, where she actively solicited favors from young men, often having sexual contacts with many men during the same evening. Such periods resulted in several pregnancies and at least two illegitimate children. However, these promiscuous periods alternated with long intervals of marital fidelity and even periods of complete sexual abstinence.

Episodic Behavior Associated with Chronic Psychopathology

Although behavioral characteristics of episodic dyscontrol or episodic reactions may be the presenting difficulty, perhaps serious enough to precipitate hospitalization, this may not be the primary problem. Careful

scrutiny may reveal that the episodic disorder is but a minor aspect of the total psychopathology, exploited for its secondary gains. For example, a dyscontrol act may be a cry for help (such as the impulsive suicidal attempt), rebellion against phobic or obsessive inhibitions, or an attempt to differentiate one's self from others in chronic schizophrenic patients. It is important to distinguish between the dynamic and etiologic mechanisms of the underlying persistent non-episodic psychopathology and the superimposed episodic dyscontrol or reactions.

A comparative evaluation between the level of regression of dyscontrol behavior and the regression of the basic chronic psychopathology gives significant diagnostic and prognostic clues. Usually, the more regressive the basic psychopathology, the more regressive will be the episodic behavioral disorder. If there is a marked disparity between the level of regression of the chronic psychopathology and the episodic behavior, the more complicated will be the dynamic and the etiologic interpretation. For instance, if the patient has a well-organized defense neurosis, yet manifests episodic dyscontrol at the level of seizure or instinctual acts, one would have to investigate several alternatives to explain this disparity:

Does the current reality have an overwhelming traumatic implication?
(2) Was the structured neurosis really covering a more regressive psychotic potential? (3) Was the seizure or instinctual act epileptoid rather than

motivated?

Where the superimposed episodic behavior is used in the service of the basic psychopathology, or has attained value because of the secondary gains received by the patient, symptomatic improvement of the episodic behavior will lead to an exacerbation of the basic psychopathology. This may in part explain the frequent observation that successful treatment of typical epileptic seizures leads to overt psychosis. Of course, the presence of severe chronic psychopathology alters considerably the prognosis and therapeutic regimen of these patients.

Neurophysiologic Mechanisms

Man's capacity to respond appropriately to environmental demands depends on two factors: the individual's endowment or the functional integrity of the equipment he possesses, and the appropriateness and extent of his learned behavior. It is difficult to evaluate how much of a maladaptive response is due to "faulty equipment," and how much is due to "faulty learning." In clinical practice, a patient seldom falls at one or the other of these extremes. Rather, the maladaptive behavior results from some mixture of faulty equipment and faulty learning. Nevertheless, it becomes important for the clinician to evaluate the extent each deficit contributes to the maladaptive behavior, in order to make an accurate prognosis and to

establish appropriate therapeutic goals. The possible epileptoid nature of many episodic behavioral disorders is suggested by the frequent concurrence of more typical epileptic phenomena (e.g., grand mal, petit mal, or simple automatisms).

Unfortunately, there is no reliable laboratory procedure to measure neurophysiologic deficits, not even the clinical EEG, which does not reveal the excessive subcortical neuronal discharges that are most likely correlated with dyscontrol behavior. Statistically, clinical EEG's with sleep recordings may indicate a higher number of abnormalities among these patients than would be expected in the normal population, or for that matter in a population of chronic psychotic patients. However, this is of little help in diagnosing a given patient who has had one routine electroencephalogram which is read as within normal range. In fact, typical epileptic behavior can occur in proven epileptics with known pathophysiologic dysfunction without EEG abnormalities, in the scalp recordings during the aura, the seizure itself, or the post-ictal period. For this reason, the routine scalp EEG is no absolute measure of whether epileptoid phenomena play a significant role in episodic dyscontrol or episodic reactions.

Also, excessive neuronal discharges in sub-cortical structures may be sustained for considerable periods of time, that is, weeks or months, without spread to the cortex and without typical epileptic seizures. Often, such limited

excessive neuronal discharges are accompanied by marked behavioral changes not usually identified as epileptic. These include dysphoria, depression, mounting irritability, altered levels of awareness, impulsivity, depersonalization, and even hallucinations or delusions. The only common characteristic of these varied symptoms with that of typical centrencephalic or temporal lobe epilepsy is a precipitous onset and equally precipitous remission.

An EEG technique that would significantly reduce the number of false negatives without an excessive increase in false positives is needed. If one uses a combination of activation techniques, such as sleep, hyperventilation, and drugs, such as Alpha-chloralose, false negatives are virtually nil in classic epileptic patients and rare in the group of episodic behavioral disorders that on other criteria seem to be epileptoid. However, there is an increase in the number of false positives (up to 20 percent in nonpatient groups). I have preferred to use Alpha-chloralose rather than Metrazol, or other drugs, because of its effectiveness in activating latent abnormalities, as well as the patient's acceptance of the subjective concomitants of this drug. Alpha-chloralose gives one a feeling of pleasant, mild intoxication, in contrast, for instance, to Metrazol which induces unpleasant anxiety. There are two drawbacks to Alpha-chloralose; namely, while it has been used in Europe for many years as a sedative, it has never been marketed in this country, therefore, it is considered an experimental drug requiring a Federal Drug

Administration IND number. The other practical disadvantage is that Alpha-chloralose is not readily soluble and must be given orally. This means an unpredictable response appearing from fifteen to sixty minutes after ingestion, with the duration of pharmacological action from two to five hours, during which time the patient should be under medical observation. Thus, this activation procedure requires from three to five hours, an excessive amount of time for most busy clinical EEG laboratories. However, for the psychiatric hospital with a clinical EEG laboratory, the effort and time would be rewarded by affording more accurate diagnoses and more effective therapeutic planning.

“Positive activation” induced by Alpha-chloralose or other drugs can be seen as representing two types of EEG responses. The first pattern is “specific,” characterized by the focal appearance of hypersynchrony and/or slow waves or typical generalized patterns of centrencephalic epilepsy. If this pattern occurs in the resting baseline, it is augmented by the activation procedure. The second type of EEG activation pattern is “aspecific” and characterized by high amplitude, paroxysmal slow waves (three-seven per second) that are generalized and bilaterally synchronous, sometimes with intermixed hypersynchronous wave forms in the same distribution. According to the literature, the prevalence of such “aspecific” patterns is high in uncomplicated epilepsy, and my findings indicate it is almost equally high in patients showing episodic behavioral disorders.

Most patients who show activated “aspecific” EEG patterns, as well as episodic dyscontrol or episodic reactions, also have a clear-cut history of a psychologically traumatic past, underlining the fact that the excessive neuronal discharges probably are not sufficient causes of episodic behavior. Studies have consistently reported that dyscontrol patients were usually subjected to intense overstimulation during the first several years of life; that is, exposure to extreme aggression in parents, siblings, or other significant adults in the environment, or exposed to severe panic reactions, or to persistent sensual and often overtly sexual stimulation. Thus, it may be conservatively generalized that if a person, for whatever reason, is destined to become neurotic, psychotic, or sociopathic, he will likely manifest this as an episodic disorder if there is an associated epileptoid mechanism (epileptoid meaning periods of circumscribed excessive neuronal discharges within the central nervous system). This underlying epileptoid mechanism can usually be demonstrated by special EEG activating techniques, which, if positive, suggest that anticonvulsant medication will significantly facilitate an effective therapeutic regimen.

Certain phenomenological characteristics give us further clues regarding whether the prominent mechanism is epileptoid or learned. Epileptoid mechanisms are probable if the dyscontrol acts are primitive and diffuse (primary dyscontrol), the eliciting situation neutral or ambiguous, and the secondary gains slight or absent. Although these statements have

common sense obviousness, the complexities of coming to such conclusions on the basis of clinical data can be surprisingly difficult. Another distinguishing characteristic between epileptoid or motivated episodic dyscontrol is the disparity between the hierarchical levels of dyscontrol behavior and the behavior between episodes. This is greater in the epileptoid patient. For instance, in a sophisticated, intelligent man whose episodic dyscontrol is primary dyscontrol, that is, a seizural or instinctual act, one must assume that there is a likelihood of an epileptoid element, particularly if there is a stereotyped repetitive quality to the dyscontrol acts. On the other hand, this would not necessarily be true if the patient were a mental defective showing organic perseveration and obsessive-compulsive traits.

At the phenomenological level, there are several other criteria for differentiating between epileptoid or motivated episodic dyscontrol. If there is clouding of sensorium during the episodic act, it is more likely epileptoid. Because it is usually impossible to evaluate the clouding of sensorium during the dyscontrol act itself, we are forced to rely on a history of amnesia to determine the likelihood of such clouding. Contrary to what is usually thought, it is the epileptoid patient who is more likely to have partial recall for the episode (except during a grand mal seizure or simple automatism). Also, he is more willing to accept the responsibility for this behavior. He feels his behavior is “driven,” is perplexed by both the quality and intensity of the act, and is willing to be confronted with his behavior in the hope of excising the

“foreign body.” He sees his act as truly ego-alien. On the other hand, the motivated or so-called hysterical patient often has complete amnesia for the episodic behavior, because he recognizes unconsciously the unacceptable intentions of his act and, therefore, denies responsibility for his behavior.

Treatment

The concept of episodic behavioral disorders aids in planning an effective therapeutic regimen for a group of patients who are otherwise difficult therapeutic problems. Some of the difficulties are enumerated below.

1. Although these patients episodically may be seriously dangerous to both themselves and society, they have relatively long periods of quiescence or normality, making it difficult to insist upon long-term hospitalization for the protection of the patient or society. The symptoms often abate within a few days after hospitalization; on the other hand, they may recur a few days after discharge, following months of intensive milieu therapy within the hospital.
2. Outpatient therapy is complicated by the tendency of these patients to precipitously terminate therapy as a dyscontrol act, or to otherwise manipulate the therapist with their dyscontrol behavior.
3. Dyscontrol patients with episodic physiologic reactions or abnormal EEG's are likely to have multiple physicians, hence multiple, often conflicting, therapeutic regimens. The

corollary is that an adequate therapeutic program often requires combined pharmacologic and re-educational techniques; this complicates the delegation of responsibility for “change” to the various physicians on the one hand, and to the physician and the patient on the other.

4. Often, these patients misuse drugs, either frantically trying to control their dysphoria by indiscriminate drug use, with habituation to drugs or alcohol becoming an additional problem, or they may not remember to take the prescribed medication because of the clouded sensorium. Furthermore, toxic symptoms of drug overdose and the episodic symptoms themselves are often similar, so that the differential between too much or too little medication cannot be easily determined.

On the positive side, patients with episodic behavioral disorders often have long periods of relative rationality, during which they can examine the episodic disturbances with realistic concern. Thus, there is the “split in the ego” which is so necessary for any re-educational insight psychotherapy.

The first prerequisite in the therapy of episodic behavioral disorders is that the therapist must be willing to take chances. If he becomes overly concerned about his patients’ behavior, they will unmercifully manipulate and punish him. The second prerequisite for effective therapy is to anticipate the type of dyscontrol acts and make preparatory plans (discussed with the patient) for handling such behavior. This may require the aid of responsible

family, peers, or co-operating professionals. A third requirement is that as soon as there is a quiescent period after any episodic behavior, the therapist should relentlessly confront the patient with his behavior, particularly those patients most reluctant to reconsider it. Fourth, the therapist must be willing to combine drug and re-educational (psychotherapeutic) techniques, but on the other hand, he must be prepared to withhold all medication, even though it might otherwise be indicated, if the patient will not follow carefully the prescribed drug regimen. Fifth, the therapist must be flexible within the therapeutic setting itself, so that dyscontrol acts will occur in the office and can be microscopically scrutinized by the patient and therapist together. Conjoint sessions with patient and significant peers are sometimes beneficial, particularly in confronting the patient with dyscontrol behavior which he is defensively rationalizing. This flexibility, however, does not apply to the frequency, time, or setting of the therapeutic sessions. A schedule must be rigidly adhered to if the therapist is to resist manipulation. Sixth, it is desirable to have one clinician responsible for the total medical management; he may utilize consultants, but he should interpret the consultants' findings and dispense recommended medications himself. These aspects of therapy are discussed in greater detail elsewhere.

It is particularly important to develop a therapeutic motivation in those dyscontrol patients who lack it, by twenty-four-hour control of environment with immediate, inescapable punishment for failures and equally immediate

and appropriate reward for successes. Hopefully, the frustration and anxieties caused by such an environment will instill an appropriate motivation for change. This disciplined environment rarely can be provided by the same individual who is responsible for the re-educational or insight therapy.

Procedures must be established for preventing acting out in the form of termination or avoidance of effective therapeutic sessions. Also, one must insist that the sole responsibility for the patient's behavior lies with the patient himself, no matter what neurophysiologic deficits there may be, or how capricious the environment.

Insight can be developed in these patients by making the dyscontrol acts ego-alien and then examining the full implications of the act itself. Resistance can be minimized by utilizing first the patient's narcissism rather than confronting it head on, avoiding disapproval of the patient's behavior by emphasizing alternative and more effective ways for meeting his needs. Understanding the dyscontrol acts is more rapidly accomplished by focusing on why the patient committed himself to action at a given time and place. Later, one examines the behavioral pattern itself; that is, the associational connections with and consequences of the act. This is particularly true for secondary dyscontrol, which occurs in patients whose lives are generally characterized by a neurotic inhibition of action rather than an "alloplastic readiness to act." In fact, dyscontrol acts may be the first sign of improvement

in neurotically inhibited patients, an indication that the patient is ready to overcome his inhibitory fears of action.

Medication, as an adjunct to, and sometimes as a primary form of, therapy, can be recommended in the following manner: If epileptoid etiologic mechanisms are presumed, or have been demonstrated by EEG activation techniques, anticonvulsants should be used, keeping in mind several limitations. Anticonvulsants are not universally effective, even in typical epilepsy; and sometimes one will work when another fails. Also, there is little range between effective therapeutic dose and toxic level. The therapeutic index can often be increased by combining several synergistically acting drugs. In episodic dyscontrol, this is best obtained by using the benzodiazepines, either alone or in combination with the usual anticonvulsants.

If control of the dyscontrol acts results in other disordered behavior, close scrutiny usually reveals that inadequate attention has been given to the re-educational psychotherapeutic program; or the importance of secondary gains of the dyscontrol behavior has been overlooked; or no opportunity has been provided for the realistic expression of affects and the gratification of needs. Phenothiazines, if used to control episodic psychotic reactions and not to treat a chronic, persistent psychosis underlying the episodic disorder, need not be maintained beyond clinical improvement. In fact, maintenance

medication may be contraindicated. If at all possible, phenothiazines should be avoided, as they may aggravate dyscontrol acts. If they are needed, they should be combined with anticonvulsants and/or benzodiazepines. Patients with episodic depressions may respond well to benzodiazepines, or even to anticonvulsants, whereas they may be untouched or perhaps aggravated by the usual antidepressants. The fact that such a complicated pharmacologic combination may be necessary, indicates that the ideal pharmacologic agent has not been found.

Long-term follow-up suggests that contrary to the usually expressed pessimism regarding therapy with these patients, they respond well to an appropriate regimen and may contribute significantly to society. Despite the turmoil of the treatment process itself, the most gratifying therapeutic results, both for the patient and the therapist, occur with this group. A top executive who had been incapacitated for five years, drank heavily, completely neglected his family, and whimpered childishly for help, is now abstinent, authoritatively assuming his role as head of the household, and functioning well in his executive position. A mother whose fugue states were so prolonged and irresponsible that she lost her husband and control of her children, becoming utterly dependent on elderly parents, has finished her professional training, is gainfully employed, and has resumed caring for her children. Another woman whose life was so totally chaotic that she, too, gave up caring for her children and divorced her husband, is now working in a

highly competent professional position and at the same time caring for her children and household. A man who could function only at a menial clerical level because of his impulsiveness and frequent habituation to drugs now successfully manages his own large business. These results are typical of many patients with episodic behavioral disorders who have been treated intensively, both pharmacologically and psychotherapeutically, over a sustained period.

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