Organic Mental Disorders as Hypothetical Pathogenetic Processes

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Abstract — A new diagnostic system for organic psychiatry is presented. We first define "organic psychiatry", and then give the theoretical basis for conceiving organic-psychiatric disorders in terms of hypothetical psychopathogenetic processes, HPP:s. Such hypothetical disorders are not strictly identical to the clusters of symptoms in which they typically manifest themselves, since the symptoms may be concealed or modified by intervening factors in non-typical circumstances and/or in the simultaneous presence of several disorders. The six basic disorders in our system are Astheno-Emotional Disorder (AED), Somnolence-Sopor-Coma Disorder (SSCD), Hallucination-Coenestopathy-Depersonalisation Disorder (HCDD), Confusional Disorder (CD), Emotional-Motivational Blunting Disorder (EMD) and Korsakoff's Amnestic Disorder (KAD). We describe their usual etiologies, their typical symptoms and course, and some forms of interaction between them.

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Introduction

In this work we want to present a new classificatory and diagnostic system for organic psychiatry. We believe that both theoretically and practically, this system has considerable advantages over the presently available alternatives[1].

We consistently use diagnoses referring not to directly observable symptoms as such, but to hypothetical pathogenetic processes (HPP:s). The classificatory categories in our system have mostly been derived from traditional (mainly central European) psychiatric diagnoses, but have been modified according to our experience and systematized according to logical and semantical considerations.

Organic psychiatry

We define organic psychiatry in two different ways depending on whether we use the concept (A) with reference to practical clinical work, or (B) in a nosological context. The concept of an organic cause (C) of a symptom is of course also central to our concerns.

A. Organic psychiatry as a practical, clinical field can be delimited as the psychiatric management of cases in which well-defined somatic diseases or injuries have brought about mental symptoms through a disturbance of cerebral function. However, the content of the term can also be captured by means of an enumeration of examples.

Among the somatic diseases and injuries which we meet in organic psychiatry are, then, such intracranially localized conditions as traumatic brain injuries, brain tumors, intracranial bleedings and thromboses, hydrocephalus, degenerative brain diseases and intracranial infections; but also states resulting from diseases which are primarily localized in other parts of the body but secondarily disturb brain function, as for example endocrinopathies, systemic infections, cardiac arrest, attempts at suicide by hanging and other causes of anoxia, and toxic states as for example eclampsia and uremia.

This means that a major share of the psychiatric consultations at different somatic wards — for example in endocrinological and neurosurgical departments — fall within organic psychiatry, but so do many cases which the psychiatrist primarily sees at the psychiatric ward or in a psychiatric outpatient setting.

One must not overemphasize the need for an exact delimitation of organic psychiatry as a practical field. In clinical practice, organic psychiatry should always be an integral part of general psychiatry. The practitioner of organic psychiatry must be prepared to encounter all kinds of psychiatric problems and so has to know the whole psychiatric field. But organic-psychiatric cases offer difficulties and possibilities

[1]For a comprehensive presentation of the system cf (1).

of their own, which motivate that they are given a separate treatment in the education and training of psychiatrists.

B. Organic psychiatry as a focus for nosological research embraces those mental disorders which particularly frequently occur in clinical organic-psychiatric contexts (as defined above). A well-known example of such a disorder is the confusional state or, as we say, Confusional Disorder. Organic psychiatry in this sense includes disorders which occur also outside the clinical organic-psychiatric situations; confusional disorder is again an example of this. Cf also (1), Fig 5, p 139.

The disorders which belong to organic psychiatry in this second sense we call "Organic Mental Disorders". Hence, no hypothesis about an obligatory organic etiology is built into our concept of an organic mental disorder (cf also below). Indeed, some of the major organic mental disorders which we describe below can also be psychogenic, namely, confusional disorder, mild forms of astheno-emotional disorder and hallucination-coenestopathy-depersonalization disorder.

Organic psychiatry and biological psychiatry are different concepts. "Biological psychiatry" so-called deals mainly with disorders due to hypothetical somatic causes which cannot yet be called "well-defined"; for example, the major affective disorders or classical schizophrenia do not particularly frequently occur in connection with well-defined somatic diseases or injuries. This means that our definition of organic psychiatry as a nosological research field excludes many disorders which are in the focus of interest of biological psychiatry (and vice versa). Although the specialist in organic psychiatry has to have considerable knowledge about disorders like schizophrenia, major affective disorders and Panic Disorder, he will have to concentrate his research efforts on the organic mental disorders as defined here.

(C) Being well aware that the term hides many problems, we will in the following designate as "organically (or somatically) caused" those mental symptoms which result from brain states or processes which are not psychologically characterizable; cf Jaspers (2). Somnolence, directly resulting from the raising intracranial pressure after an intracranial bleeding, is an example of such symptoms.

When describing such symptoms as "organically caused" (or "somatogenic") we are not implying a false dichotomy to the effect that the causes which we classify as "non-somatic", i e the psychologically characterizable states and processes, do not also have a somatic basis. The depressive reaction after the loss of a close relative is explainable in terms of a chain of intrapsychic events and processes, and we therefore name the reaction "psychogenic". But in such psychogenic cases, there is (we presume) always an underlying or parallel chain of brain events and processes.

In organic psychiatry, the focus of interest of course lies on those organic causes which are well-defined from a somatic point of view and ascertainable (in principle) with today's medical techniques. But an organic cause, i e one which is not characterizable in psychological terms, need not be accessible with today's medical techniques and hence need not yet be somatically well-defined either. An example might be the primary defect in transmittor mechanisms underlying Panic Disorder, if the biological psychiatrist's speculation is correct (and the psychoanalyst's is wrong). Theoretically, one must distinguish such purely hypothetical somatic causes from those well-defined causes which are the primary concern of the specialist in organic psychiatry. In the present text, it should be clear from the context which kind(s) of somatic causes we are referring to[1].

[1]It has been suggested (3, 4) that "organic" is an obsolete term, and it seems that the "organic mental disorders" section in DSM-III-R (5) will not remain in DSM-IV. The first main point of Spitzer et al (4) is that "organic" suggests a dichotomy between mental disorders which have a biological substrate in the brain and those which have not, a dichotomy which is not in accord with the generally accepted hypothesis that all mental disorders have a biological basis. Spitzer et al recognize the need for a word differentiating the mental effects of somatic diseases and substance abuse from those mental disorders which do not have such a readily recognizable somatic cause, but for the former they prefer the term "secondary" instead of "organic". Their second main point is that the principle of etiological neutrality of Axis I in DSM-IV is not compatible with having such "secondary mental disorders" as a main category.

Concerning the first point, we completely agree with the basic outlook of Spitzer et al, but we are not convinced that the change of term from "organic" to "secondary" will prevent fundamental philosophical confusions. Here we side with Lipowski (6) who prefers to keep the term "organic". The remedy is instead to define organic psychiatry and organic mental disorders carefully, as we have done above, so that no false dichotomy is entailed.

We also agree on the motive underlying the decision not to use "Secondary Mental Disorders" as a main heading in DSM-IV, namely, that the main diagnostic categories should be etiologically neutral. But note that one can define the concept of an organic mental disorder in an etiologically neutral way, namely as any mental disorder

In many a patient one or more symptoms turn out to be only partly somatogenic, and then of course partly psychogenic. In our analysis of organic-psychiatric cases, we lay much emphasis on trying to understand the relative contributions of psychic and organic causes to the patient's disorder.

The nature of diagnostic concepts in organic psychiatry

Our scientific work has focused on the task of developing an appropriate diagnostic system for the organic-psychiatric field.

Psychiatrists with little experience of organic psychiatry often believe that the diagnostic questions in this area are very simple, or at least not as problematic as in other psychiatric fields. For in organic psychiatry, the mental disorders very often have evident causes and it is often not difficult to delineate the clinical picture. It might seem a reasonable supposition that in these patients one could define disorders in terms of both a specific etiology and a specific symptomatology. Reading literature in which the authors rely on concepts like postoperative psychosis, puerperal psychosis, hydrocephalic dementia, frontal lobe dementia and post-concussional syndrome may well reinforce one's tendencies to think in terms of such a simplified etiological-symptomatological scheme.

However, already a little more experience of organic psychiatry usually reveals to the critical observer that it is only possible to a very limited extent to use diagnostic concepts tied to both specific etiology and specific symptomatology: patients with similar somatic causes of their mental disorders (for example, injury to the frontal lobe) may be symptomatologically very different; conversely, identical symptom clusters (e g, a typical, full-blown delirious picture) may result from widely different somatic causes.

This fact is now recognized by most authorities in organic psychiatry, and other ways of delimiting the organic mental disorders are being tried; a much-used method is to classify patients exclusively with regards to their symptoms. This way is the dominant one in DSM-III (7) and DSM-III-R (5). However, there is a third way beside using etiologically based psychiatric concepts and relying on purely symptomatological classifications. This way consists in defining the diagnostic categories in terms of psychopathogenesis, i e the processes linking somatic etiology and mental symptoms. Since these processes are not known in any detail, they have to be introduced as hypotheses; hence the designation Hypothetical Pathogenetic Processes (HPP:s).

HPP diagnoses are not tied to specific (distal)[1] etiologies such as frontal lobe injury or normal pressure hydrocephalus, but neither are they identical to specific symptom clusters. We want to emphasize, however, that our psychopathogenetic hypotheses are essentially founded on symptomatological data. Hence there is a strong symptomatological bias in our system. But we conceive of the relation between the symptoms and the diagnosis in a way which has to be explained further, since it is not the way of DSM-III and similar rigidly operationalistic systems.

The semantic status of operational diagnostic criteria

It is much discussed today whether the main diagnostic concepts of psychiatry should be introduced as lists of observable ("operational") diagnostic criteria, or as some kind of theoretical constructs. To understand this problem correctly one has to think just a little about the semantic status of operational criteria.

A certain mental disorder may be defined to be identical to an observable cluster of symptoms and signs. Having the disorder is then strictly the same as fulfilling a certain set of criteria. In other words, there is no possibility even in theory of the disorder occurring without the criteria being fulfilled, or vice versa. In philosophical terms, this means that the operational criteria for the disorder are taken as a strict definition of the diagnostic concept in question. This is the position of strict operationalism.

As shown in detail elsewhere (8), such a position results in awkward consequences if applied to real-life clinical situations. We will here only mention

which frequently, but not necessarily always, occurs in an organic-psychiatric context (cf above).

There may be other, more convincing arguments for excluding the organic mental disorders as a main category in DSM-IV, but even if so, it does not follow that one should treat them in a similar fashion in other contexts. Spitzer et al note that the needs of certain clinicians may motivate a special list in DSM-IV of those mental disorders which typically are secondary manifestations of a recognizable somatic disease. And, we would like to add, there will certainly still be a need for textbooks and reviews centering on those disorders, which of course also means treating them under one general heading.

[1]By "distal" etiology we refer to the causal factors which initiate the pathogenetical process, eventually leading to the symptoms.

the DSM-III criterion for Schizophrenic Syndrome, requiring at least 6 months duration for a positive diagnosis. If that is taken as a part of a strict definition, it is logically impossible that a patient should have a Schizophrenic Syndrome for only five months, and then die. This is clinically a very clumsy construct, to say the least; cf Strömgren (9). The same kind of argument can be applied to a great number of other diagnostic categories in DSM-III and DSM-III-R. Cf (8), especially Section 6 on Amnestic Syndrome and Dementia.

Mental disorders: Hypothetical Pathogenetic Processes

Recent developments in the philosophy of science have made clear that a scientific construct which has been introduced in terms of an observable cluster need not be strictly identical to that cluster, but is instead often properly conceived as a hypothetical entity or process, underlying (causing) the observed phenomena (10).

Both somaticists and psychiatrists often work with constructs of such a nature. A good illustration is offered by the discovery of European sleeping sickness (von Economo's disease)[1]. Although the symptoms of that disease were so striking in the typical cases that they were regarded as indicating the presence of a new disease, relatives of patients with typical von Economo's disease often showed much less conspicuous symptoms. In these cases only a probable diagnosis could be made since the symptoms were uncharacteristic. Hence, the disease is certainly not identical with any cluster of symptoms[2]. Also, our knowledge about the pathology of von Economo's disease is, on the whole, limited to severe cases, and is most probably not universally valid. Neither do we know its specific distal etiology.

Hence, although we can be quite certain that European sleeping sickness did exist, we are not in the possession of necessary and sufficient symptomatological or pathologico-anatomical criteria for its occurrence. A fortiori, we cannot formulate an operational, strict definition of the disease. Since we do not know which agent caused the disease, we cannot strictly define it in specific, etiological terms either — it can only be defined as "that contagious encephalitis which, in the epidemics in the beginning of the century, typically caused such and such symptoms and signs, and such and such cerebral pathology".

The really interesting element in this is not that von Economo's disease is not strictly defined in terms of its usual symptoms. This property also belongs to a concept such as myocardial infarction, which alongside its symptomatic criteria has a strict definition in terms of pathology; cf (8) p 36. In contrast, von Economo's disease does not have, and never had, any such strict definition; all that we have ever had for a definition of it is the specification in terms of its usual manifestations. Still, the workers in the field had no great difficulties in identifying it (at least when they saw typical cases). And although we still cannot strictly define the disease, we do talk about it (and do understand the question whether this disease could atypically manifest itself as a psychosis).

We think that the situation is similar with regard to mental disorders. Several of them can be identified as underlying, unitary factors, although we still know only to a limited extent how they manifest themselves. Of course we do not believe that all — or even most — such disorders are manifestations of a unitary etiology in the sense in which European sleeping sickness probably was. But there is a level between observable symptoms and distal etiology, namely, the level of mediating pathogenetic processes (cf (13), Ch. 6). These mediating (or "intervening") processes may each be the result of different distal etiologies and may each, in turn, cause observable signs and symptoms. We know them only hypothetically, as the probable causes of such and such symptoms, and as probably interacting in such and such ways with each other and with other factors.

The line of reasoning which we have followed in this section should be familiar to the psychiatrist who has been trained in traditional nosology. Diagnoses such as "Depression without depression" or "Hysteric conversion" are difficult to understand if one does not accept hypothetical pathogenetic processes, but they are easy to understand in terms of HPP:s. Depression without depression is simply the same psychopathogenetic process as that which typically causes depressive mood (together with a number of other symptoms) — but now it is, for some reason, not causing a depressive mood.

[1]See (11) for a detailed analysis of this and another example, Legionnaires' Disease.

[2]Note, for example, that it is still an open question whether there were - or even still are - atypical cases of von Economo's disease presenting themselves exclusively as psychoses. Cf (12), p 294.

Indeed, we think that the unity of most diagnostic entities in psychiatry is to be sought on such a hypothetical, pathogenetic level, intermediate between distal etiology and manifest symptoms. In this respect (but not in others), the somatic diagnosis of respiratory insufficiency offers a better parallel to psychiatry than does von Economo's disease. Respiratory insufficiency is a pathophysiological disease entity which can have a number of different, specific etiologies. It typically manifests itself in certain symptoms and signs (e g, dyspnoea and cyanosis). It is, however, not strictly identical with any set of symptoms and signs, since these may be concealed (e g, by oxygen treatment), or they may be due to other diseases (heart insufficiency).

Of course, a main difference between psychiatry and internal medicine is that much more is known about pathophysiological mechanisms in the latter field than in the former. In other words, the diagnostic categories in psychiatry must rely on less well-founded theories, and they will inherit the preliminary and tentative character of these (psychological, psychopathological and psychophysiological) theories. This is actually one main reason why they still have to be identified via their typical manifestations. In the case of defects of known somatic functions, these can now be strictly defined in theoretical terms referring to these functions. In psychiatry, where only very general principles about mental functioning can be established, the delimitation of disease processes must still be firmly anchored in symptoms and signs.

However, as explained above, this does not entail that these processes should be strictly defined in operational terms. We think that it is simply impossible to get a clear, consistent and clinically useful view of the psychiatric disorders, if we do not use concepts which essentially refer to a deeper level than the purely observational one. By referring to an intermediate level in the pathogenesis of symptoms — i e by using HPP diagnoses — one may hope to bring some order into the extremely complex psychiatric field. Our system therefore consistently uses such concepts.

Elements of a psychophysiological theory

In order to explain why HPP diagnoses in organic psychiatry are clinically more informative and relevant than other kinds of diagnoses, we have to briefly discuss the probable intrinsic nature of the disorders themselves, conceived as hypothetical brain processes.

We have elsewhere offered the outlines of a psychophysiological theory (1, pp 106ff). The fundamental postulate of this theory is that the brain is a complex adaptive system which tends to behave convergently, i e to react in a limited number of ways to many different noxae.

The existence of such adaptive[1] convergent processes is, in our view, the basic explanation why very similar symptoms can be seen in widely differing distal-etiological conditions and why, after all, organic-psychiatric symptoms tend to group themselves into a limited number of clusters. We conceive of the different disorders in our systems as more or less stable phases of convergent processes. As such, these processes are unobservable, but they manifest themselves in more or less typical symptoms.

Following Hughlings Jackson (17), we also see the formation of symptoms in light of a hierarchical model of the nervous system, and accordingly classify many symptoms (e g most hallucinations) as "release phenomena". The disorder and its basic manifestations (functional deficits and release symptoms) interact with spared psychical functions and with external stimuli, finally resulting in observable constellations of symptoms and signs (1, Fig 4, p 120). Because the factors which interact with the disorder may be very different from case to case and from time to time, the symptoms and signs are also sometimes considerably modified although the underlying disorder remains the same. Cf also (18).

Hence, in our system the organic-psychiatric disorders can be characterized as convergent, intermediate psychopathogenetic pathways for the brain's reactions to noxious influences. If our psychophysiological hypothesis is true, these convergent processes are fairly stable over time, and concepts referring to them should have greater predictive validity for prognosis, therapy and care than categories based either on specific, distal etiology or on observable symptoms and signs. In large series of patients which we have followed for long periods of time, we have observed that our concepts do have a considerable value for predicting the development of symptoms and the final prognosis. This has convinced us of the existence of the pathogenetic processes to which these concepts refer.

[1]Cf also Bleuler (14), Conrad (15) and Ey (16). That the processes are "adaptive" does not, of course, entail that the attempts at adaptation always succeed.

Structural analysis in organic psychiatry

We believe that it is important to use all available knowledge about the patient in the causal analysis of symptoms and signs in organic psychiatry. The psychiatric symptoms constitute the primary data, and their occurrence and course should be carefully documented. We also have to register a number of somatic, intrapsychical and social factors which might possibly contribute to the formation of the symptoms. Further, we must consider the possibility that more or less idiosyncratic vulnerability factors — hereditary dispositions, habitual reaction patterns — may contribute to the clinical course. Finally, one must take the possibility of other, intercurrent psychiatric disorders into regard.

Psychological causes abound in organic psychiatry, and psychological explanations (partial or complete) should therefore be sought for. It seems to be not at all rare for psychological stress or trauma to release the same kind of attempted adaptive reaction as does a physical injury to the brain. An example in point is psychogenic confusional disorder; another is psychogenic mild astheno-emotional disorder (see below; a common label is "neurasthenic syndrome"). Incomplete but contributing psychological causes occur even more often.

It often turns out that similar clinical symptoms must be given radically different explanations already at the level of psychological functions. For example, a failure on a memory test may of course be a manifestation of a malfunctioning of specific memory mechanisms (as in Korsakoff's Amnestic Disorder, cf below), but is often instead basically the result of lack of motivation or of a defective capacity for concentration. Lack of concentration may, in turn, be the result of the patient's disturbing thoughts about her serious illness (normal psychology), or of some yet-to-be-explained lowering of the patient's basic capacity to concentrate (probably pathological).

On the other hand, absence of a certain mental symptom, say fatigue, need not imply that there is no corresponding disturbance at a somewhat deeper level — the patient may have a pathological fatiguability, although this does not show up since no demands are put on him in the hospital. In other words, we try to avoid overly simplistic inferences from data about the patient's achievements (or failures to achieve) to hypotheses about his underlying mental functions, capacities and dispositions.

We consider this psychological phase of the "structural analysis" of a case a necessary prerequisite for the judgment whether, and to what degree, the patient's various symptoms indicate the presence of a particular organic-psychiatric disorder. Each such disorder usually manifests itself in disturbances of several specific mental functions. This in turn means that the structural analysis of any given case also involves an inference from the pattern of disturbances of mental functions (and of release symptoms) to the underlying disorder. In uncomplicated cases this inference can be easy, since the clinical picture is typical. It is more difficult in other cases, for example if there are reasons to suspect an intercurrent mental disorder.

It should be pointed out that in the structural analysis, specific (distal) etiologies may be used as criteria for disorders in the same (probabilistic) way as symptoms, signs and course. This is because some organic mental disorders are particularly common with certain somatic etiologies. For example, the presence of a Monroic cyst supports the hypothesis that a certain failure on a memory test is really due to Korsakoff's Amnestic Disorder, although such an etiology is of course neither necessary nor sufficient for that diagnosis. Considerations like this are not seldom helpful when the symptom pattern is ambiguous.

A special difficulty is presented by the co-occurrence of several organic mental disorders. In a patient with a disturbance of brain function, the etiological conditions for two or more such disorders may very well be fulfilled simultaneously. Then, the particular disorders can manifest themselves in varying degrees in the total symptomatology. In some cases one disorder dominates the picture completely and the other pathogenetic process(es) is (are) only sparsely represented in the symptomatology, or even not at all. In other cases the constellation of symptoms is not characteristic of any of the disorders present.

It is often difficult or impossible in complex cases to identify all current disorders on the basis of the patient's mental symptoms on one single occasion. However, if we follow such patients over a longer period of time, the continuous changes in the symptoms usually enable us to make a complete retrospective diagnosis. To be able to "retropolate" reliably in this way, one must of course be familiar with the natural courses of uncomplicated cases of the individual organic disorders, and also with the usual development of their underlying etiological conditions.

The six basic disorders of organic psychiatry

Most of the basic categories in our system have been derived from traditional concepts, especially from those originally created in the classical European school by E. Bleuler, K. Jaspers, M. Bleuler, K. Conrad and others, but we have modified them in several respects. These modifications rest (i) on our own observations, in consecutive patients with various somatic illnesses, concerning the distribution of symptoms and symptom pictures, (ii) on observations concerning the longitudinal courses of these symptom pictures, (iii) on the existing knowledge about common etiological mechanisms, and (iv) on theoretical, psychological and psychophysiological considerations concerning the interaction between various mental mechanisms.

The following is a brief presentation of six particularly important organic mental disorders[1]. A special emphasis is put on the astheno-emotional disorder (AED), which in our opinion is the most important of them all. This mental reaction is common throughout all parts of organic psychiatry, but especially the mild and moderately severe forms of it are not given adequate attention in modern psychiatry. Also, the hallucination-coenestopathy-depersonalisation disorder (HCDD) is described in some detail, since it is not generally recognized as a diagnostic entity.

Astheno-Emotional Disorder, AED

AED is a diagnosis which includes a large spectrum of cases of varying severity, ranging from disturbances which are hardly distinguishable from everyday psychopathology, to very severe cases, as for example in many cases of severe multi-infarct dementia. Our conviction that, after all, these very different clinical pictures belong together is mainly founded on a great number of longitudinal patient studies, where we have been able to follow in detail the gradual process of improvement or deterioration over large parts of the whole scale of severity.

Primary symptoms

In its mild ("neurasthenic") and moderately severe forms, the disorder typically produces an impaired concentration ability, memory difficulties, increased mental fatiguability, irritability and emotional instability. In the severe forms there is also a general impoverishment of and sluggishness in associational processes, a lack of overview and a reduced capacity for abstraction.

None of these terms has a uniform meaning in common psychiatric usage, and we will therefore give a short description of these various types of astheno-emotional symptoms.

Primary symptoms in mild to moderate AED

The concentration difficulties in a mild or moderate AED consist in a diminished capacity to uphold a good, uninterrupted attention; cf (1), Fig 7, p 144. In very mild cases the symptoms may mimic those common in everyday fatigue states, but even in most of the mild cases the disturbance is significant enough to considerably reduce the patient's level of achievement. In patients with a moderately severe AED the power of concentration may be so disturbed that they cannot, for example, read the daily paper but only the headlines. They may become more or less disoriented. The psychiatrist can judge the severity of the concentration difficulties from anamnestic data about the patient's performance in reading, looking at TV, talking with other people etc. Psychometric tests which require a steady mental concentration can be very helpful.

The memory difficulties which are caused by mild and moderately severe AED seem to be largely secondary to the patient's attentional deficits. Here, too, detailed anamnestic data and psychometric tests are informative when judging the degree of severity of these disturbances. Most memory tests are very sensitive to memory difficulties of the astheno-emotional type, which is important to keep in mind when determining the clinical significance of an inferior performance on a memory test. The AED diagnosis should always be considered when memory disturbances are observed, since the great majority of organic memory disturbances are astheno-emotional in nature.

Patients with AED are sensitive to disturbances from their environment. For example, they might have difficulties participating in a conversation if several persons are present or if there is much background noise, and they often prefer to go aside when several members of the family come together. In a moderately severe AED, the experience of strong lights or sounds may be unpleasant and even pain-like. The patients are often irritable and may react with outbursts of anger to various disturbing stimuli, and they may even show minor forms of violent behavior.

The emotional lability in a mild to moderate AED manifests itself as a hypersensibility to emotional stress. Tearfulness when exposed to sentimental topics is typical; there may also be short attacks of sobbing. In moderately severe cases of AED there is

[1]In spite of their undoubted importance in organic psychiatry, the aphasias are not on our list. This is mainly because as a field of research, the aphasias traditionally belong to neurology. For the same reason, various forms of agnosia are not discussed here.

sometimes a considerable "affective incontinence" with frequent such attacks.

Primary symptoms of severe AED

When a patient with a moderately severe AED deteriorates, the old symptoms become more accentuated, but new kinds of symptoms also appear. The concentration difficulties come to be very incapacitating, and they are now not even temporarily amendable by relaxation. The flow of associations becomes impoverished and sluggish; the capacity for overview and judgment is impaired. Already the patient with a moderately severe AED is often incapable of changing the topic of a discussion in a natural, flexible manner, and an ordinary conversation usually means an obvious strain for him. In a severe case of AED these difficulties can be so marked that it is impossible to sustain a meaningful dialogue with the patient. Perseveration may occur. Memory fails severely, and it becomes evident that the memory problems are not any longer only secondary to the concentration difficulties — memories are recalled with only few details, and in advanced cases fragmentarily. Large parts of the memory stores (including old memories) seem to have been irreversibly lost. The tendencies towards outbursts of anger may be aggravated, and occasionally lead to severe acts of violence. In some patients the emotional lability is more marked than in moderate cases, but in other patients one instead sees an emotional flattening. We cannot tell whether the latter symptom is a manifestation of the patient's AED or if the flattening has to be explained by an intercurrent EMD (cf below).

In the borderland between moderately severe and severe cases of AED it is sometimes impossible either to confirm or to exclude the presence of the symptoms specific to the severe forms. Such cases we classify as "fairly severe".

Etiology and course

Mild astheno-emotional disorder can have either an organic or a psychogenic etiology. Moderately severe cases are most often of an organic origin, but, for example, in some patients with really severe chronic pain and a grave disturbance of sleep the mental stress may result in an AED of barely moderate severity. Severe forms of AED as far as we know always have an organic etiology.

Symptoms of AED is a most common finding in all parts of organic psychiatry. All kinds of diffuse or localized organic brain diseases or injuries may give rise to this disorder: traumatic injuries, tumors, infections, degenerative diseases, vascular diseases, etc.

In psychogenic cases of AED, even if they are moderately severe, the symptoms of the disorder usually disappear quickly if the releasing etiological factor is eliminated. In organic cases the prognosis of course depends on the underlying somatic disease, but also on the severity of the disorder and, importantly, on the patient's age. Mild organic cases usually heal completely if the underlying somatic disease is eliminated; this holds also for most young patients with moderately severe AED. In severe AED the prognosis, according to our opinion, is mostly unfavorable. Although in some cases an improvement can be seen, it does not reach even the level of moderate severity.

Psychogenic, secondary symptoms

Even a mild AED can reduce the patient's mental capacity enough to cause him trouble in his daily life. Eventually, this often results in a considerable load which leads to psychogenic, secondary reactions. There may appear a lowered self-esteem, feelings of uncertainty, anxiety, depressive reactions (even severe depressions), and psychosomatic complications including headache. Even paranoid reactions may occur, especially when insurance controversies are involved. Not seldom, these secondary symptoms are so severe that they dominate the clinical picture, and it is not unusual that the less dramatic primary AED symptoms — which in fact make up the core of the patient's symptomatology — are not registered at all.

Psychophysiology

Psychophysiologically, mild to moderate AED may correspond to a functional disturbance of a number of cognitive and emotional control processes which unconsciously regulate the attentional level, evaluate the emotional significance of stimuli and filter out irrelevant information. This disturbance may be an effect either of too high external demands on the system (the typical psychogenic case), or of incoming pathological information stemming from a primary defect elsewhere in the central nervous system (internally generated "noise"). In severe forms of AED, a direct effect of the etiological factor on the control mechanisms may be of greater importance than the overload postulated in the mild and moderate cases.

Comparison with DSM-III and DSM-III-R

When patients with symptoms of severe, pure AED are diagnosed according to DSM-III and DSM-III-R, they should as a rule be classified as Dementia (concerning mixed cases, cf p 15 below).

Moderately severe forms, especially if the emotional symptoms are prominent, would often be classified as Organic Personality Disorder. Although very common, mild AED (if of a proven or presumed organic etiology) can only be classified as Organic Mental Syndrome NOS in DSM-III-R (5, p 119); in DSM-III the corresponding label is Atypical or Mixed Organic Brain Syndrome (7, pp 123f).

Somnolence-Sopor-Coma Disorder, SSCD

In its severe forms, this well-known disorder manifests itself as a coma of varying depth. In moderately severe cases, the disorder typically causes psycho-motoric dampening with a general impairment of most cognitive, emotional and motivational performances. Moderately severe SSCD also causes an increased tendency to fall asleep. A mild SSCD produces slight and rather unspecific symptoms which may be difficult to notice. The symptoms can be limited to a low attentional intensity, slow comprehension or a somewhat reduced simultaneous cognitive capacity with slightly lessened powers of judgment.[1]

When, as is often the case, slight symptoms of a somnolence-sopor-coma disorder arise in a mixed organic condition, it is particularly difficult and sometimes impossible in practice to establish their correct nature.

SSCD symptoms usually arise as a result either of chemical, toxic influences on the brain or of raised intracranial pressure; however, it may also occur in some other clinical situations, for example pituitary adenomas with suprasellar extension (20, p 160). The course varies, mainly depending on the varying nature of the distal etiology.

SSCD is psychopathologically distinct from psychogenic sleepiness and from the unspecific fatigue reaction which usually follows episodes of great somatic stress. Psychophysiologically, SSCD can probably be described as a malfunctioning of the brain's arousal systems (or of their target neurons).

In DSM-III or DSM-III-R, somnolence-sopor-coma disorder is not described as a separate organic mental syndrome, but is only represented in the form of an associated symptom ("reduced level of consciousness") in Delirium. However, Delirium often occur without an SSCD component, and vice versa. In a reasonably severe, mixed condition the SSCD would often contribute to fulfilling the criteria for a DSM-III diagnosis of Dementia. This is particularly relevant in cases of normal pressure hydrocephalus (19).

[1]For a fuller description of the symptoms cf (19).

Confusional Disorder, CD

Decisive for a diagnosis of this well-known disorder is a certain kind of incoherence in thought and speech (21). In addition, the disorder often gives rise to such symptoms as disorientation, anxiety, hallucinations and illusions in varying extent, but these symptoms are not pathognomonic for the disorder to the same extent as is incoherence. Disorientation, for example, also occurs in most patients with Korsakoff's Amnestic Disorder, and partial disorientation may occur in patients with concentration difficulties due to an AED which is only moderately severe.

Confusional disorder is a common sequel of diffuse or localized mechanical or chemical injury to the brain, but often the disorder is psychogenic. The course is usually acute. Cases of mixed etiology are very common; cf confusional disorder in people with senile dementia of Alzheimer's type, when they are exposed to a completely new environment.

In DSM-III, confusional disorder of organic etiology would normally be classified as Delirium. Psychogenic cases would often receive the label "Brief Reactive Psychosis".

Hallucination-Coenestopathy-Depersonalisation Disorder, HCDD

In full-blown cases of this disorder, the patient experiences visual hallucinations or other visuo-perceptual disturbances, coenestopathies and feelings of depersonalisation and/or derealisation. As a rule, auditory hallucinations do not belong to the picture.

The visuo-perceptual disturbances are mostly visual pseudo-hallucinations (i e, the patient is aware of their hallucinatory nature) but there may also be micropsia, metamorphopsia etc.

Coenestopathies are changes of body image (for example, the feeling of enlargement of a bodily part) which are experienced with hallucinatory clearness. The HCDD kind of coenesthopathies are distinguished from similar schizophrenic symptoms by the fact that the HCDD patients (except in the most severe cases) do not believe that their body has really changed. Hence, most HCDD coenestopathies may also be classified as bodily pseudohallucinations.

Depersonalisation and derealisation phenomena (which, following an old European tradition, we treat under the common heading "depersonalisation") occur in several psychiatric contexts, but those which are caused by an HCD disorder are more distinct and obtrusive than those which one typically finds in, for example, depressive and anancastic states.

Full-blown cases of HCDD are not very frequent, and in most cases symptoms from only one or two of the three main groups are present. Nevertheless, there are cogent reasons to consider these three kinds of symptoms to be manifestations of a unitary pathogenetic process. Firstly, there is an obvious phenomenological similarity between the symptoms. They all have a similar obtrusive character and a hallucinatory clearness. Secondly, they strongly tend to occur in the same clinical settings. These settings are mainly various forms of intoxication (especially with hallucinogens), migraine, endocrinological abnormalities, and traumatic injuries and other causes of dysfunction of the hypothalamus and/or the temporal lobes (e g, in partial epilepsy of temporal origin)[1].

HCDD may also be psychogenic (e g, in sensory deprivation experiments), and mixed-etiology cases of HCDD are probably not uncommon. In a large series of patients (20), symptoms of this disorder were significantly more frequent in the patients who had a hysteroid personality (DSM-III: Histrionic Personality Disorder); cf Lindqvist, Carlsson and Malmgren (forthcoming).

In severe cases, the HCD disorder sometimes progresses to a confusional disorder, but usually it soon heals spontaneously even if the releasing etiological factor is unchanged.

When, nowadays, symptoms of HCDD are reported from departments of internal medicine or infectious disorders, they are often classified as an "Alice in Wonderland Syndrome". In DSM-III and DSM-III-R, the natural syndrome diagnosis of a somatogenic, reasonably pure HCDD would be Organic Hallucinosis.

Emotional-Motivational Blunting Disorder, EMD

This disorder gives rise to disturbances of emotional, motivational and cognitive functions; of these, the emotional and motivational changes are easier to observe than the cognitive ones. The motivational blunting can be more general — "Antriebsschwäche" — or may seem to affect particular ambitions, for example, personal hygienic goals or professional ambitions. The emotional shallowness of EMD patients often takes the form of lack of feelings for and consideration of other people, including a noteworthy lack of concern for their relatives. A shallow euphoria is often present. Beside these rather easily noted emotional and motivational changes, EMD also produces serious cognitive changes which, however, may be more difficult to identify. These are changes which belong to the core of personality, namely, the capacities for abstraction, foresight, planning and self-criticism.

Overt behaviour can vary a great deal: some patients' behaviour is hardly affected; some become extremely inactive, unspontaneous, while in others the disorder leads to thoughtless, unrestrained, economically rash, promiscuous or even criminal behaviour.

Most doctors are probably aquainted with the emotional-motivational blunting disorder as appearing in connection with damage to the frontal lobes, the so-called "frontal lobe syndrome". However, the disorder also very often occurs with injuries to limbic structures (22), thalamus and hypothalamus. It can also be caused by severe endocrine disturbances[2].

In EMD patients with progressive diseases such as cerebral gliomas, a progressive worsening of the disorder is seen. In a few cases of very serious brain injury, an EMD may manifest itself which is severe from the beginning and then does not improve at all. In the great majority of cases however, EMD shows some tendency towards healing. Even a moderately severe case of EMD may heal completely or almost so, for example when occurring after a severe head trauma or in connection with an endocrine disorder.

In the 1940's, the course of EMD of different severity could be studied in large series of patients having undergone extensive frontal lobotomies. When, today, an EMD occurs after stereotactic psychosurgery it should be mild, healing within a few weeks or a couple of months.

Psychophysiologically, the manifestations of EMD may tentatively be explained as resulting from a disruption or disturbance (at any level) of bidirectional functional connections between primary motivational regions and cerebral systems responsible for abstract thought and foresight, which results in a reduced overall motivational level and/or lessened higher control over basic drives.

According to DSM-III and DSM-III-R mild and moderately severe — as well as a number of more severe — forms of a pure EMD would be classified as Organic Personality Disorder. This category however also includes patients with other disorders

[1]Shared distal etiology is, after all, an indicator that two pathological processes in the brain are similar to some degree.

For the same reasons, we also believe that the great majority of isolated visual hallucinations of organic origin are manifestations of a mild HCDD.

[2]It should also be noted that the most common organic-psychiatric disorder in cases of not too extensive frontal lobe damage is not EMD but astheno-emotional disorder, AED.

than EMD, for instance cases with astheno-emotional disorder and marked lability of affect. Many of the really severe cases of reasonably pure EMD would, on the other hand, be classified as Dementia.

Korsakoff's Amnestic Disorder, KAD

Fully developed, KAD manifests itself as the well-known combination of retrograde amnesia, short-term memory impairment, disorientation and confabulation. The memory disturbance is not secondary to a reduction of attentional or motivational capacities.

This disorder arises only with bilateral damage to, or dysfunction of, limbic structures, the hypothalamus or certain parts of the thalamus. The course can be chronic and stationary (as in most cases of alcoholic dementia), subchronic and gradually healing (as in moderately severe post-traumatic amnesia) or transient (as in transient global amnesia). Before the microneurosurgical era, KAD (with a course varying from transient to stationary) was a frequent complication of operations for a ruptured aneurysm on the anterior communicating artery (23, 24).

Patients with Korsakoff's amnestic disorder, KAD, almost always have more or less evident symptoms of an emotional-motivational blunting disorder, EMD; this is understandable in view of the etiology of the two disorders. In many psychiatric classifications, the emotional and motivational changes are included in the diagnosis "Korsakoff's syndrome". We have however decided to keep the two categories distinct in order to emphasize the identity between the emotional and motivational changes in patients with KAD and in those with a pure EMD.

According to DSM-III and DSM-III-R, pure cases of Korsakoff's amnestic disorder would generally be classified as Amnestic Syndrome. However, this diagnosis excludes all patients who at the same time have serious general deficits in intellectual functions (for example, patients with simultaneous symptoms of severe AED). These would receive a DSM-III diagnosis of Dementia.

By themselves, or combined with each other, the six disorders which we have now described give rise to the large majority of symptoms seen in organic psychiatry. In (1) we also describe other organic mental disorders which are less important to organic psychiatry as a whole, but sometimes of course of dominant interest, e g the auditory hallucination disorder often met with in chronic alcoholism. We also discuss, from etiological and psychopathogenetical viewpoints, a number of symptoms and symptom constellations like tiredness, depressive symptoms, anxiety, aggressive behaviour and changes in sexual behaviour.

Interactions between two or more mental organic disorders

Interactions between organic disorders can produce symptom patterns which are not typical of the disorders in question. In some such cases it may be fairly easy to read the correct multiple diagnoses from the present symptoms, but mostly a longitudinal study of the patient is needed. This is especially so if one wants to reliably judge the severity of the component disorders, which is often important for prognosis.

In the following, a few important kinds of interactions between disorders are briefly described.

— A simple and common example is offered by the patient who suffers a traumatic brain injury, which results in a combination of an SSCD and an AED. Initially the patient is comatous; it is then impossible to know which disorders are at hand except SSCD. When the patient later awakens and the SSCD symptoms gradually diminish over a couple of weeks, it is often possible to observe the occurrence of AED symptoms, but as a rule not possible to determine the severity of the AED with any degree of certainty. This can not be done until the SSCD symptoms have regressed completely, and not until then is one in the position to judge the prognosis in a reliable way.

— When a patient awakens from a coma it may instead be the case that the clinical picture is dominated by symptoms of EMD and/or KAD. In such cases one should presuppose the existence of a non-negligible AED, even if it does not give any identifiable contribution to the complex symptom picture. The glaring primary defects of memory function arising through the KA disorder usually overshadow the secondary memory disturbances of an astheno-emotional type, resulting from concentration difficulties, and the motivational shallowness determined by the EMD often completely masks the mental fatiguability arising from the AED. In these patients, EMD and KAD usually heal faster than AED. The AED symptoms therefore become gradually more apparent. In cases

where the KAD and EMD symptoms regress completely, there is usually a protracted final phase with a gradually diminishing, purely astheno-emotional symptomatology (1, Fig 6, p 132).

— With patients who have recently awakened from a coma, it is of course natural that one searches for mild SSCD symptoms. The risk of overlooking such symptoms is much higher when they are mild from the beginning. But even in cases of the latter kind, a correct diagnosis may be important. For example, in patients with the combination of a not particularly severe AED and a mild SSCD (i e, ceteris paribus a prognostically favourable condition) the two disorders mutually result in a clinical picture which is very similar to a pure AED of considerable severity. This constellation is found in many patients with normal pressure hydrocephalus (19). They regularly receive the diagnosis "hydrocephalic dementia". When such a patient's symptoms are dramatically relieved by a shunt operation which immediately eliminates the SSCD, and only mild or moderately severe AED symptoms remain, the case is classified as one of "treatable dementia".

— Confusional Disorder also gives rise to diagnostic problems concerning other possibly co-existing organic mental disorders. The confusional symptoms often dominate so completely that other disorders are wholly concealed. It is, for example, usually impossible to say whether a confusional patient also has astheno-emotional concentration difficulties or a retrograde amnesia characteristic of KAD. The prognosis of a brain-injured patient with a confusional disorder therefore cannot be determined until the confusional symptoms have regressed.

— In patients with dysphasia it can be difficult to evaluate the severity of other, coexisting disorders, and sometimes even to identify them. For example, the presence of an EMD in a patient with a left fronto-temporal lesion may easily be overlooked because of the accompanying dysphasia.

A common feature of these examples is that a prognostically relevant diagnosis often cannot be made if one relies only on the momentary symptom picture. By means of a structural and longitudinal analysis of the symptom picture and a separate consideration of each underlying disorder one can, however, usually arrive at a much greater prognostic precision.

Note on the concept of Dementia

As the reader may have already inferred, in our opinion Dementia is a "social", pragmatically motivated diagnosis, which theoretically and clinically is to be regarded as a heterogeneous mix of different organic disorders. The most common components are the AE, EM and KA disorders, dysphasia and various agnosias. In different cases of Dementia, different disorders dominate the symptoms picture. The most "pure" occurrences of severe AED are probably seen in multi-infarct dementia. In some rare cases of "dementia" there is an appreciable SSCD component.

We have elsewhere (19) reported a clinical study of organic mental syndromes in normal pressure hydrocephalus, trying to separate and analyze the different components of the "dementia" of these patients. It would seem to be of interest for prognosis, treatment and care to try to describe other cases of Dementia in a similar manner.

Some advantages of our diagnostic system

Compared with narrowly operationalistic systems like DSM-III and DSM-III-R, our conceptual frame of reference has a number of advantages.

— The system is founded on an explicit logico-semantical theory which makes it possible to use the diagnostic concepts in a clear and unambiguous way in clinical as well as in research settings.

— The system makes it possible to analyze not only the momentary symptom picture, but also the whole course of disease, and it offers a conceptual framework for describing the interaction between psychological and somatic etiological factors. In the proper sense of the word, it therefore represents a dynamical point of view in organic psychiatry.

— The same diagnostic concepts can be used in all parts of organic psychiatry and in all kinds of clinical situations: (i) when a well-defined cerebral lesion is at hand, (ii) when a somatic distal etiology is probable but not certain, and (iii) when similar symptom pictures are at hand but there is probably no organic distal etiology at all.

— The same diagnostic concepts are used to describe simple and mild cases as for complex and severe cases. This is possible because the system allows for multiple simultaneous diagnoses and for quantification of the severity of the disorders. The

importance of this emerges clearly in longitudinal studies of patients with changing symptom pictures and in studies of consecutive cases of etiological categories which are associated with highly varying psychiatric manifestations.

— The system allows for probability diagnoses of disorders in incompletely investigated cases, and for retrospective revision of these diagnoses.

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