Descriptive Psychopathology

The Signs and Symptoms of Behavioral Disorders

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Preface

"Of all Persons who are Objects of our Charity, none move my Compassion, like those whom it has pleas'd God to leave in a full state of Health and Strength, but depriv'd of Reason to act for themselves. And it is, in my opinion, one of the greatest Scandals upon the Understanding of others, to mock at those who want it." (Daniel Defoe, 1697)[1]

As in seventeeth century England, today's society continues to subtly mock those of us "deprived of reason". Mental health insurance in the USA is inadequate and less than that for other conditions. The psychiatrically ill are left in large numbers on the streets and alleys of our cities, a situation the medical establishment would find intolerable if the distress were due to heart disease. Sufferers are ridiculed in mass entertainment, equated to clowns, fools, and criminals. While the necessity of care by specialists is widely recognized for patients with stroke, epilepsy, dementia, and other "neurologic" disease, it is acceptable for almost any interested party to "hang up a shingle" and offer almost any kind of treatment to those of us "deprived of reason".

Yet the loss of reason and other psychopathology are expressions of brain disease and dysfunction, and this recognition has diagnostic implications increasingly important as more exact treatments are introduced. The need for diagnostic accuracy, however, is subverted by the poor validity of present-day psychiatric classification. Better delineation of clinical populations will reduce heterogeneity and thus facilitate the application of more specific treatments. The recent call to separate melancholia from other depressions[2] and catatonia from the psychotic disorders,[3] for example, provide the framework for the more specific treatments for these conditions. Obsessive–compulsive spectrum disorders identified within the impulse control category also warrant their own treatment approach to avoid mismanagement. Recognizing psychoses associated with seizure disorder avoids sufferers being considered schizophrenic or hysterical and receiving inappropriate treatments.

To accurately delineate psychiatric disease, however, requires in-depth knowledge of the signs and symptoms of behavioral disorders, i.e. descriptive psychopathology,

and the examination skills to elicit clinically useful phenomena. Descriptive psychopathology, detailed here, considers the abnormal observable behavior and its subjective experience needed for this effort. Bedside assessment of cognitive functions complements the behavioral examination.

Hypothesized psychopathologic constructs (e.g. ego defense mechanisms, psychological reactions) represent a paradigm different from that of descriptive psychopathology. We do not discuss these ideas. For medical diagnosis, they are overly interpretive and lack objective definition. Their reliability is poor, and they are unhelpful in defining syndromes of the brain or in predicting treatment response and other clinical variables. In contrast, descriptive psychopathology can be reliably defined and its different patterns better predict pathophysiology and treatment response.[4]

Despite the detail we present, this book is not a dictionary of all psychiatric terminology. It is also not an encyclopedic compendium of the theories of the mind, or a wide-ranging dissertation on the psychology of behavior. We discuss theories and psychology only when helpful in clarifying the diagnostic or neurologic implications of psychopathology.

Thus, this book is not primarily written for the scientist or the theorist, although they should find it useful in defining their populations of interest. It is written to help clinicians in the care of their patients. Our approach is neuropsychiatric, derived from the understanding that all forms of descriptive psychopathology are observed in patients presently characterized as having neurologic disease (e.g. seizure disorder, stroke, and dementia), and that many classic neurologic signs and symptoms are in turn observed in patients recognized as having a psychiatric disorder. The separation of psychiatry and neurology is arbitrary. Both disciplines care for persons with brain dysfunction or brain disease. Their common ground is the clinical implications of the behavioral disturbances elicited by brain dysfunction. We delineate this common experience by detailing classic descriptive psychopathology and associated neurologic features. We show, often with clinical examples, how the presence of specific psychopathologic phenomena influences diagnosis. Within the limits of the present understanding of brain functioning, we also offer a neurologic understanding of classic clinical features as they affect diagnosis.

We divide the book into four sections.

In Section 1, we describe the problems and limitations of present classifications and through clinical examples show that they serve patients poorly. We illustrate that a command of the knowledge and skills of descriptive psychopathology provides more refined diagnosis and treatment.

As the study of descriptive psychopathology spans millennia, we review this history.[5] We detail the shifting tensions over the centuries between classification

"lumpers" and "splitters" that led to present classifications. We next show that the "mental status examination" is better considered the "behavioral examination of the brain". The limited neuroscience of psychopathology is presented.

In Section 2, we describe the principles of diagnosis, and detail the examination style, structure, and techniques.

In Section 3, we define and describe psychopathology that goes beyond that found in present classification manuals, and show how the identification of these phenomena is of diagnostic importance. We present the behavioral domains of the examination in the order commonly addressed clinically. We start with chapters on general appearance, motor behavior, and emotion, areas of the assessment that rely heavily on inspection rather than extensive conversation.

In the chapter on motor disturbances we also delineate catatonia, and distinguish the motor disturbances of basal ganglia, cerebellar and frontal circuitry disease. We describe the differences in the speech and language problems encountered in patients with aphasia, mania, catatonia, and the "formal thought disorder" associated with psychosis. In the discussion of perceptual disturbances we detail the phenomena associated with temporal–limbic disease. We discuss delusions and aspects of abnormal thought content. The spectrum of obsessive–compulsive behaviors is presented as a more coherent picture than the present scattering of related conditions throughout classification. We detail the behaviors and cognitive impairment patterns of patients with delirium and different forms of dementia. We describe the dimensional structure of personality and personality disorder and how this approach is more productive than the present categorical system in predicting co-morbidities and in shaping behavioral treatments.

Lastly, in Section 4, we propose a re-structuring of present-day classification based on the psychopathology literature and its validating data. Our goal is to re-establish the best of the past within the framework of modern insights into brain function and psychopathology.

Nevertheless, present-day psychiatry retains much ambiguity. There are no laboratory tests that define psychiatric illness to the precision achieved in identifying specific strains of a virus or the number of trinucleotide repeats in a genetically based illness. Sustained pleasure for the psychiatric clinician must come from examining and making sense of diverse psychopathological expressions of illness and the satisfaction from using that understanding to shape treatments and resolve distress. "Figuring it out" and "getting all better" patients with complex patterns of psychopathology are experiences that sustain clinical practice. Telling the distraught mother and sister of an 18-year-old man who had been hospitalized for "encephalitis" and was considered "a hopeless case", but who in fact had a mood disorder and malignant catatonia that "We're going to get him all better, not just a little better" and then doing it, finally watching the previously mute

and immobile patient walk out of the hospital with his family is an experience that cannot easily be achieved without a full understanding of descriptive psychopathology.

Defining psychopathology to delineate behavioral syndromes and to choose specific treatments is a practical effort for the trainee and the experienced clinician alike. All who accept the responsibility for the care of patients with behavioral syndromes should find useful information in this book. But our effort is aimed at those new to that responsibility – trainees in psychiatry, neurology, and neuropsychology. For them, our book offers a crossroad in their career journey, the path now less taken, but we think more rewarding than the cookbook psychiatry that has been created to complement present classification. Karl Jaspers expressed the same challenge more than 90 years ago in the prefaces to the 2nd and 3rd editions of his classic textbook *General Psychopathology*:

> The opinion has been expressed in medical quarters that this book is too hard for students, because it attempts to tackle extremely difficult and ultimate problems. As far as that is concerned, I am convinced that either one grasps a science entirely, that means in its central problems, or not at all. I consider it fatal simply to adjust at a low level. One should be guided by the better students who are interested in the subject for its own sake, even though they may be in the minority. Those who teach should compel their students to rise to a scientific level. But this is made impossible if "compendia" are used, which give students fragmentary, superficial pseudo knowledge "for practical purposes", and which sometimes is more subversive for practice than total ignorance.[6]

NOTES

1 Defoe (1697), cited by Hunter and Macalpine (1963), page 265.
2 Taylor and Fink (2006).
3 Fink and Taylor (2003).
4 Present classification does not predict treatment response. See the discussion in Chapter 4 and in Taylor and Fink (2006) chapter 1.
5 The Western interest in psychopathology dates from classical Greece, evolved in central Europe and France, captured the interest of physicians in Great Britain and then crossed the Atlantic to the USA and Canada. It is now worldwide. Chapter 2 provides a discussion of the history of Western classification of psychiatric illness. Medical traditions from Asia are not discussed because they have not influenced modern medical psychiatry.
6 Jaspers (1963), page x xi.

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Beyond the DSM and ICD: a rationale for understanding and using descriptive psychopathology

The straight jacket imposed on psychiatry by the introduction of [DSM III] and its successors, reverberating in Europe with the [IDC 10] has had a profound effect on the practice of psychiatry. An earlier generation's more elegant constructions of a hierarchal basis for diagnos tic classifications has disappeared, so that a patient now may end up with 3, 4, or even more DSM IV diagnoses, and patients failing to fulfill one of the criteria for entry for a condition may be deemed not to be suffering from that condition, although logical clinical evaluation would suggest otherwise. Furthermore, many of the diagnostic inclusions are broad, ambigu ous, and open to misuse in inappropriate settings . . . it seems the DSM IV and its forerunners were created by committees which appear not to have been appreciative of the broad spectrum of conditions met with in psychiatric practice, and particularly in neuropsychiatry.[1]

Present-day psychiatry is dependent upon the *Diagnostic and Statistical Manual for Mental Disorders* (DSM) and *International Classification of Diseases* (ICD) classifications. The DSM has become the main reference source of recognized psychopathology and is the standard system for research subject selection world-wide. The ICD, rarely used in the USA, is favored in many parts of the world as a clinically useful document.[2] The ICD offers separate research and clinical versions in several languages.[3] Training programs in many parts of the world also rely on the DSM and ICD. Endorsed treatment algorithms are linked to classification labels. Manual category numbering is required for clinical documentation, and insurance reimbursement, and is referenced in legislative and legal proceedings. This dependence is accepted under the assumption that the manuals maximize reliability and contain validated conditions and groupings that encourage the best diagnostic decisions and treatment choices.

The dependence on the classification manuals has permitted a paradigm shift in psychiatry, particularly in the USA. The more leisurely psychological approach to patient care has been largely replaced by a primary care treatment model. Rapid diagnosis, followed by reflexive pharmacotherapy is encouraged

Developed from an article, Vaidya and Taylor (2006).

to accommodate high patient turnover. "I don't have enough time to see my patients . . . they only give me a half hour for intakes and follow-up visits" has become the mantra of psychiatry house officers.[4]

Applying the primary care model is facilitated by the ICD short descriptive prose or the DSM telegraphic lists. While the two systems differ in some categories (e.g. psychotic disorders, dementia, disorders in children and adolescents, and generalized anxiety disorder) and terminology (e.g. the ICD "organic" versus the DSM "secondary to" for syndromes with established etiology), both offer a skeletal view of psychopathology designed to be applied quickly.[5] The time-consuming detailed investigation of the unfolding of the patient's illness, the nuances of the sequence of symptom emergence, patterns of features, and the importance of some features over others is deemed superfluous and has been abandoned. Once diagnostic criteria are met, a treatment algorithm based on the DSM or ICD diagnosis is chosen. Treatment algorithms, often endorsed by an "expert" panel,[6] can be applied as if cooking recipes.[7]

Paralleling the format changes has been an expansion in diagnostic choices from a handful of syndromes in DSM-II (APA, 1968) and the ICD-6 mental disorders section to presently over 280 options. The expansion is meant to assure recognition of any psychiatric affliction,[8] implicitly promising that the classification contains all the known psychiatric conditions, that these conditions are sufficiently validated, and that the diagnostic criteria for each are reliable and sufficient to identify each condition. There should be no practical need to know more psychopathology than what is in the manuals. The promise, however, is unfulfilled, as validity is poor for many classification groupings (e.g. personality disorders, impulse control disorders) and the reliability of the systems is marginal.

The weakness in present classifications is illustrated in the startling and clearly implausible announcement that a study supported by the National Institutes of Mental Health in the USA determined that 55% of persons in the USA are at lifetime risk for psychiatric illness. In response, Paul McHugh, professor of psychiatry at Johns Hopkins and retired department chairman, blamed inexperienced interviewers relying on the DSM. He wrote:

> In addition to relying solely on respondents' yes or no answers to a checklist, the investiga tors are committed to employing the official Diagnostic and Statistical Manual of Mental Disorders Fourth Edition (abbreviated DSM IV), which bases all psychiatric diagnoses on symptoms and their course, not on any fuller knowledge of the person. It is as if public health investigators studying the prevalence of pneumonia over time in the American popula tion were satisfied to call every instance of a cough with a fever and a mucoid sputum a case of pneumonia.[9]

Training of descriptive psychopathology relies on classification manuals

The primary care model has elicited a metamorphosis in psychiatric instruction, notably in a reduction in the teaching of the *mental status examination* and descriptive psychopathology. Once the lynchpin of training, interest in psychopathology now focuses on how to recognize the clinical features needed to apply DSM or ICD labels. For example, in a 2002 mailed survey to all accredited psychiatry residency training programs in the USA ($N=149$),[10] of which 68 (45.6%) responded, while nearly 80% stated that they offered a course in descriptive psychopathology (often only one semester),[11] and another in the mental status examination (typically less than 5h), less than 30% of respondents taught the classic features of psychopathology (e.g. catatonia, first rank symptoms), and less than 20% used any of the well-known psychopathology texts.[12] Twenty percent of programs offered no formal lecture series in descriptive psychopathology or mental status examination. Psychopathology was seen in many teaching programs as the signs and symptoms described in the present DSM, but nearly half did not provide classroom instruction or discussion of the features in the criteria, and those that did typically devoted less than 5h to it. A 1991 survey of all psychiatric clinical tutors in the UK also found substantial reliance on the DSM for the teaching of basic psychopathology.[13] Surveys of the teaching of psychopathology in other parts of the world are lacking.

Problems in present classifications

Table 1.1 displays the problems in present classification. These are discussed in detail below.[14]

DSM and ICD reliability is weak

Reliability in diagnosis is its degree of precision, i.e. agreement among clinicians. If reliability is poor, validity of diagnoses (accuracy) is unclear.[15] Systematized "field trials" of the interrater reliability of the recent DSM and ICD iterations describe mixed results. Diagnostic agreement was also inflated by defining agreement as two clinicians placing the patient in the same diagnostic class rather than explicitly agreeing.[16] If the clinicians differed in the specific disorder within the class (e.g. if one diagnosed "schizophrenia", while the other said the patient had "delusional disorder"), agreement was accepted.[17] Such agreement is equivalent to clinicians agreeing that a patient has a respiratory problem, but not whether it is bronchitis or pneumonia, bacterial or viral or allergy-related.

Table 1.1. Problems in present classification

Problem	Effect
Reliability is weak	The "claim to fame" of recent DSM iterations is high reliability. Weak reliability insures idiosyncratic diagnosis
Encourages false positives and false negatives with over inclusive diagnostic criteria	Some conditions are over diagnosed (e.g. depression) resulting in false positives, research sample heterogeneity, and unneeded or dangerous treatment for patients. Some conditions are not recognized (e.g. catatonia), resulting in false negatives and inappropriate treatment
Offers false choices	False choices lead to the prescription of inappropriate treatments. Conditions such as schizophreniform have no validity. Dissociation, a symptom, is treated as a disease. Abnormal bereavement and puerperal depressions are given separate status
Omits or marginalizes established syndromes	Catatonia is incorrectly linked to psychotic disorders, melancholia is reduced to a modifying term, the different frontal lobe syndromes are not included
Diagnostic criteria are poorly defined	Terms such as "disorganized speech" encourage misdiagnosis (e.g. misidentifying a fluent aphasia as flight of ideas or formal thought disorder)
Checklist format limits meaningful examination	Items are incorrectly given equal weight. Type of illness onset, sequence of symptom emergence, and patterns of features are mostly ignored, resulting in misdiagnosis
Omits important discriminating psychopathology	Psychopathology associated with neurologic syndromes (e.g. psychosensory features and seizure disorder) are not mentioned, resulting in illnesses going unrecognized
Claiming theory neutrality, it avoids neuroscience and laboratory criteria	Patterns of features that indicate the involvement of a specific brain region or system are not included (e.g. features indicating right hemisphere disease). Laboratory assessments are not included as helpful criteria (e.g. hypothalamic pituitary functioning in depressive illness, CPK levels and response to lorazepam in catatonia)
Longitudinal criteria are not used	The pre psychosis findings in schizophrenia are ignored, resulting in the over diagnosis of the condition and research sample heterogeneity. The dimensional traits of personality are ignored, resulting in poor reliability and validity for the personality disorders

In the two DSM-III trials, the overall agreement for Axis I for adults was marginally acceptable (kappas of 0.68 and 0.72, with 0.70 the minimal agreement coefficient). For affective disorders they were 0.69 and 0.83. For schizophrenia, both trials obtained kappas of 0.81. The range among diagnostic classes, however, was broad and many had kappas near 0.50 (i.e. closer to chance agreement).[18] Kappas for children and adolescents were poor for most conditions, as were kappas for Axis II. Often, only a few patient vignettes per category were used, lessening the likelihood of meaningful agreement. DSM-III-R field trials have similar shortcomings (APA, 1987).

Assessments of DSM-IV also detail mixed results. The DSM-based clinical interview is reported to reliably identify patients with eating disorders,[19] symptoms and diagnoses in relatives of psychiatric patients,[20] and diagnoses from information obtained from personal interview or from an informant.[21] Other reports are less positive. For example, an assessment of 362 outpatients using the DSM-IV interview instrument for anxiety and mood disorder obtained good test–retest reliability for the two categories, but there was substantial overlap and "a common source of unreliability was disagreements on whether constituent symptoms were sufficient in number, severity, or duration to meet DSM-IV diagnostic criteria."[22] Participants also had difficulty categorizing clinical features presented randomly as representing an Axis I or II criterion, and in one study they misclassified 31% of Axis I criteria as representing an Axis II disorder and 25% of Axis II criteria as representing an Axis I condition.[23] They could not identify whether a feature was a symptom of disease or trait behavior. This is equivalent to not knowing whether the patient's cough represents a nervous tic or respiratory disease.

The DSM-IV field trials revealed diagnostic uncertainty. The mood disorder field trials of 524 patients from inpatient, outpatient, and community settings from 5 sites used structured interviews and reported good intra-site but only fair inter-site reliability with deteriorating reliability in a six-month retest.[24] Test–retest reliability is reported below for statistical reliability standards for psychosis, somatization, eating disorder, dysthymia, mania, generalized anxiety disorder, attention deficit hyperactivity disorder, and hypochondriasis.[25] Studies of the multiaxial systems of both classifications find poor agreement (i.e. which axis to place clinical features) and poor agreement on axes assessing environmental stressful events.[26]

The reliability of "bizarre" delusions, the hallmark of the criteria for the psychotic disorders, has also been found unsatisfactory.[27] An assessment for substance-induced psychiatric syndromes in 1951 acute psychiatric inpatients found a dimensional approach to have better reliability and predictive validity than the dichotomous DSM-IV strategy.[28]

ICD-10 field trials worldwide assessed over 15000 patients at 112 clinical centers in 39 countries. Good reliability was achieved except for the personality disorders.[29] Independent examinations of 150 patients assessed with a European diagnostic instrument also found good reliability for schizophrenia,[30] mania, and major depression, but unsatisfactory reliability for schizoaffective disorder.[31] The validity of the psychotic disorders category, however, was questioned.[32] Systematic application of ICD descriptions to clinical samples also finds instability over time for the diagnosis of bipolar and recurrent depressive disorder.[33] The low interrater reliability for a depressive episode[34] and difficulties with the ICD depression subscales for endogenous and psychogenic depression[35] partially account for the diagnostic instability of the ICD mood disorder category. ICD reliability was found enhanced by the addition of clinical descriptions to the operational criteria, a strategy not used in the DSM.[36]

The mixed reliability results are particularly alarming because the field trial participants were intensively trained in the use of the system and examination instruments. Also, about 40% of the patient evaluations were done conjointly. These procedures are rarely used in clinical practice where diagnostic agreement among clinicians remains low.[37]

Further, while the assessment of patients by structured examination can obtain fair to good reliability,[38] this method has poor agreement with the more likely clinical circumstance of a clinician doing a semi-structured evaluation[39] or a standard clinical assessment.[40] Even when using semi-structured assessments, reliability is marginal for some diagnostic options.

Bertelsen (1999) cautions against the exclusive reliance on simplified list-based criteria. He points out that the best clinical approach is an initial comprehensive traditional clinical examination to first identify the syndrome followed by the matching of the findings to criteria for nosologic labeling, rather than reliance solely on the manuals.[41]

Classification validity is uncertain

Accuracy in diagnosis defines validity, i.e. the patient has the illness that is diagnosed. Poor validity leads to false positive and false negative classifications. A false negative occurs when the patient's illness is unrecognized. A false positive occurs when a patient is given a diagnosis he does not have. Present classification methods encourage both types of errors.

Diagnostic false positives

Diagnostic criteria in the manuals are mostly imprecise and overly broad, encouraging the identification of illness when none exists, or misidentifying one illness for another. The identification of over half the population in the USA as meeting

such criteria for illness dramatically demonstrates the degree of false positive diagnosis inherent in using the DSM. Half of persons in normal bereavement also meet criteria for major depression, but neither the researchers nor the bereaved in the studies considered the state to be illness.[42] The major depression criteria of apathy and motor slowing are seen in frontal circuitry disease, while low energy, shyness, and anxiety are found in some persons with personality deviations. These patients may be misdiagnosed as depressed and needlessly prescribed antidepressant agents.[43]

The DSM diagnosis of major depression requires five or more items in any combination.[44] Depressed mood need *not* be present for the diagnosis of depression. A loss of interest or the inability to experience pleasure are acceptable alternatives. "Fatigue or loss of energy" and "diminished ability to think or concentrate" are choices. The criteria are not operationally defined (e.g. what degree of diminished concentration is needed to be a symptom and how concentration is to be measured, are not detailed). In the quest for diagnostic reliability, criteria are over-simplified, thereby lowering the bar for admission into the category of depression. Taken literally (which is a necessity to obtain expected reliability) the following patient meets DSM-IV criteria for major depression.

Patient 1.1

A 51-year-old man experienced substantial loss of interest and anhedonia for almost a year. He slept much of the day (hypersomnia is a criterion choice), and his movements and thinking were slowed (psychomotor retardation is a criterion choice). He had trouble concentrating his thoughts, and had no energy. He was pessimistic about the future. He did not want to kill himself, but he did not want to live in his present state. His symptoms caused "clinically significant distress and impairment in social functioning." His condition could not be explained as the "direct physiological effects of a substance . . . or a general medical condition." His general neurologic examination was normal, except for slowness of movement and thought. His symptoms began after his trailer home burned, destroying it and all his possessions. He was not burned and did not suffer significant smoke inhalation. Posttraumatic stress disorder was ruled out, because he did not have nightmares and was neither anxious nor ruminating about the event. Major depression was diagnosed by several clinicians and antidepressant medications were prescribed without improvement.[45]

On examination, the man's mood was reactive, and although subdued, he showed mildly diminished emotional expression rather than sadness or apprehension. A frontal lobe avolitional syndrome[46] **was diagnosed and carbon monoxide poisoning hypothesized as the cause of his behavioral change.**

CT scan showed bilateral basal ganglia calcifications, a finding consistent with the diagnosis of carbon monoxide exposure. Methylphenidate treatment improved his condition.

Patient 1.1 also meets criteria for "treatment-resistant depression", because he did not respond to two drug trials with different classes of antidepressants. However, about 10–15% of depressed patients labeled "treatment-resistant" are incorrectly considered depressed, and therefore do not benefit from antidepressant treatments.[47]

The DSM criterion A common to all the psychotic disorders is also problematic. Two of five features are needed, but sustained auditory hallucinations and "bizarre" delusions may stand alone. This provision is a vestige from the ideas of Kurt Schneider, who considered some psychotic features to be pathognomonic of schizophrenia if a neurological disease could not be recognized.[48] The identification in the 1970s of Schneider's "first rank symptoms" in patients with mood disorder and other conditions, however, demonstrated definitively that these features are not pathognomonic, but the error persists in DSM-IV. Consider Patient 1.2.

Patient 1.2

A 32-year-old woman was hospitalized because she barricaded her home and rearranged the furniture so that her two young children would not have to walk on the floor. She said she had overheard neighbors constantly plotting to electrify the floor and that she could feel static electricity. She was irritable and walked constantly throughout the inpatient unit on tiptoe and had several other catatonic features.

She responded to questions such as "What do you think is the reason for your neighbors doing those things to your house?" with:

"They're jealous, mean spirited, I'm the spirit of 1776, they see the spirit in me, I have an aura, an aura borealis, a whore (eyes filled with tears for a moment), a four by four."

Patient 1.2 meets the DSM criterion A for a psychotic disorder. She experienced sustained auditory hallucination (tactile also) and many would accept her delusional ideas as "bizarre". She also exhibited "disorganized speech", another criterion A choice. However, other psychopathology can be recognized. Her tiptoe gait is consistent with catatonia, and she exhibited other catatonic features, phenomena not detailed in the manuals.[49] Criterion A includes catatonia as a choice in the diagnosis of schizophrenia. Nevertheless, irritability and constant walking about the inpatient unit suggests hyperactivity or agitation and along with catatonia are consistent with a manic episode. She had grandiose delusions. Characterizing speech with such vague terms as "disorganized" is also poor practice.

Flight-of-ideas with clang associations describes her language better, and are features of mania. The patient was treated with lithium monotherapy and fully recovered.

False negative diagnosis and "not otherwise specified" (NOS)

The high proportion of patients receiving the DSM *Not Otherwise Specified* (NOS) choice further attests to the limits of the system.[50] To support treatment choices, the "catch-all" option permits clinicians to assign patients to a likely diagnostic category despite being unable to fit them to a specific illness descriptor (e.g. the diagnosis "psychosis, NOS" justifies prescribing an antipsychotic agent).

Use of the NOS choice occurs in several circumstances. Most commonly, the patient meets some but not all necessary criteria. When a patient has an established syndrome not recognized in the DSM, but has a clinical feature that suggests a diagnostic category, NOS is also applied. The frontal lobe avolitional and disinhibited syndromes, several seizure-related syndromes and the paraphrenias are not included in the DSM. Patients with these conditions go unrecognized and are typically labeled "psychotic disorder" or "mood disorder, NOS".[51]

Hirschfeld (2001) reviews the behaviors consistent with a *manic-depressive spectrum*, a construct not implicitly incorporated in the DSM. Such syndromes elicit the NOS suffix. The manic-depressive spectrum concept, however, leads to more effective treatment (e.g. mood stabilizers and antidepressants rather than psychotherapy alone) for many patients now considered as having personality disorders. *Cyclothymia* represents part of that spectrum.

The *Oneiroid Syndrome*, a dream-like state, known to European psychiatrists, but all but forgotten in the USA, is another example.[52] Recent reviews of the diagnostic usefulness of psychopathology associated with traumatic brain injury[53] and epilepsy[54] further highlight the omission of important syndromes.

The failure to define the catatonia syndrome illustrates another ICD and DSM shortcoming. Catatonia has strong linkage to mood disorder, more so than to schizophrenia. Yet, the DSM primarily places catatonia as a subtype of schizophrenia, while all patients with catatonia not clearly the result of a neurologic or general medical condition must be diagnosed as suffering from a psychotic disorder by the ICD. There are over 40 classic catatonic features and associated behaviors, but the DSM briefly mentions only 12 and the ICD fewer. Neither manual offers instructions on how to identify or elicit the features. A patient could easily have many catatonic features not elicited or recognized by the clinician trained to the DSM or ICD standard. It is not surprising that most DSM-trained clinicians think catatonia is rare despite the consistent finding that when systematically assessed, 10% of acutely hospitalized psychiatric patients, 40% of hospitalized manic patients, and many patients with developmental

disorders meet criteria for catatonia.[55] Some catatonic and stuporous patients are mostly mute and cannot communicate the information needed to assess criteria. Patient 1.3 illustrates.

Patient 1.3

A 28-year-old woman became withdrawn and then mute over a period of several days. She sat staring for long periods, and when she did move, her efforts were slow. Without evidence of a general medical or structural neurologic cause for her condition, she was admitted to a psychiatric inpatient unit. Laboratory tests results revealed no explanation for her state other than dehydration. Her drug screen was negative. She was diagnosed "psychotic" "NOS". The brief psychotic disorder/schizophreniform/schizophrenia option was considered.

A consultant elicited several catatonic features not identified in the DSM (Gegenhalten, automatic obedience, ambitendency) consistent with catalepsy, bradykinesia and mutism. IV midazolam in preparation for an MRI temporarily disinhibited the patient at which time she looked about the drab hallway to the MRI suite and said "Good, you're taking me to the basement incinerator. I deserve to die. I am a bad person." She described her depressed mood, hopelessness and desire to die. The MRI revealed no structural disease, and she was diagnosed as having melancholia with catatonic features. A course of lorazepam resolved her catatonia.

Some patients marginally match a large DSM or ICD category. Patient 1.4 initially diagnosed as having psychotic disorder NOS, with consideration of "late-onset schizophrenia", illustrates the need to recognize other forms of psychopathology.

Patient 1.4

A 63-year-old woman previously in good health stopped answering her phone. Concerned, her daughter went to the woman's house, but at first the mother would not open the door, saying that rays were being beamed into the house to gain control of her mind. She said her neighbors and their homes had been replaced by aliens from another planet and that the aliens were now probing into her mind and had started to gain control of her body. When she finally opened the door she screamed at her daughter, calling her an imposter and an alien. Once in the hospital she was unable to return to her room from the dining area, and angrily accused the nurses of hiding her room so that she could not find it.[56]

The attending psychiatrist considered late-onset schizophrenia or psychosis, NOS as likely diagnoses. A consultant, however, recognized the woman to

have Capgras syndrome, the delusion that familiar persons are imposters, often a sign of non-dominant cerebral hemisphere disease.[57] Delusions of replicated neighbors and homes (reduplicative paramnesia), experiences of alienation and control, and "losing" her hospital room (topographic disorientation) were also consistent with non-dominant cerebral hemisphere disease. The lack of other psychopathology (loss of emotional expression, avolition, auditory hallucinations, and speech and language disorders) further suggested the lesion was posterior. A right-sided parieto-temporal lobe stroke was demonstrated on brain imaging. Antipsychotic medication was withheld, and redirection and behavioral control became the focus of treatment. She was discharged a week later fully recovered.

False diagnostic choices

For a behavioral condition to warrant inclusion in the official classification of disease, it must meet long-established standards.[58] Its cross-sectional clinical features should delineate it from other conditions. The characteristic signs and symptoms should be validated by a characteristic course of illness or response to treatment, genetic predisposition, or laboratory markers. Present classification is replete, however, with examples that violate this standard, and many diagnostic classes are included without evidence warranting their recognition as disease entities. A patently false notion is the classifying of *brief*, *schizophreniform* and *schizophrenia* as three independent nonaffective psychotic disorders if they remain within their duration requirements, but as a continuum if their durations merge.[59] Follow-up studies of patients originally diagnosed schizophreniform find variable outcomes – some patients evolving to schizophrenia, while others develop a schizoaffective or mood disorder.[60] *Abnormal bereavement* and *puerperal depression* meet criteria for melancholia, and these depressions are no different in any meaningful way from melancholias occurring in other circumstances.[61] Nevertheless, they are classified by their circumstances as if they warrant independent status.

Conversion and *dissociative* disorders are also classified as distinct psychologically derived illnesses, despite evidence of great heterogeneity in samples of these patients and associations with a variety of neurologic diseases including seizure disorder and demyelinating conditions.[62] *Dissociative identity disorder* has also been associated with manic-depressive illness.[63]

Diagnostic criteria are categorical without dimensional considerations, and poorly defined

The DSM and ICD conceptually define many Axis I conditions as they do many general medical conditions (e.g. infection and bone fracture). The person is normal, then something occurs and the syndrome appears fully formed. While

illness course is used to define unipolar and bipolar categories, longitudinal criteria such as age of onset and pre-episode features (e.g. schizoid traits) are not used in Axis I criteria. The magnitude of the omission is illustrated by evidence showing that many persons with schizophrenia have pre-psychosis childhood neuromotor, cognitive, and emotional difficulties that are identifiable and potentially useful in secondary prevention.[64] In one study of old "home movies" of young children at family gatherings, viewers experienced in childhood behavior who were unaware of the condition of the children in later years identified 90% of the children who became schizophrenic.[65]

The incorporation of a dimensional component into Axis I criteria has been proposed for future manuals.[66] The focus is on severity ratings of criteria to facilitate prognosis, monitor treatment response, and in the recognition of mild conditions. Patients would be given individual criterion and summed severity rating scores. This approach, however, will not solve the reliability and validity problems of classification, and does not address the necessary identification of the longitudinal emergence of disease.

Poorly drafted diagnostic criteria also limit the usefulness of the manuals. To bolster reliability, both systems over-simplify the descriptors of psychopathology such as delusions, hallucinations and language disorder. Although many forms of speech and language disorder in psychiatric patients are described in the classic literature, DSM-IV states: "Because of the difficulty inherent in developing an objective definition of 'thought disorder' and because in a clinical setting inferences about thought are based primarily on the individual's speech, the concept of disorganized speech has been employed . . ." (DSM-IV, p. 276). Not only does this approach lump most speech and language problems under one appellation, it assumes that the speech problems of patients derives from problems in thinking, when the classic literature and empirical studies show that this is not always the case.[67] Patient 1.5 is an example of how disregarding complexity endangers patients.

Patient 1.5

The behavior of a 60-year-old nursing home patient with a long history of manic-depressive illness changed over a week. Her mood fluctuated between high spirits and irritability. She became agitated and her speech was described as "disorganized and confused". At times she did not appear oriented to date and place. She was transferred to a psychiatric hospital with the diagnosis of recurrence of mania.

A consultant at the hospital noted that the patient's speech was spontaneous and fluent, without dysarthria, but she was paraphasic with agrammatisms and neologisms. She could repeat simple phrases, but at times she was non-sequitive

in her responses. She had naming problems and was circumloculatory. She did not have the circumstantial speech or flight-of-ideas characteristic of mania. Her change in speech and word usage was understood as a receptive aphasia syndrome following a stroke. Hypertension was considered contributory. At no time in the nursing home, hospital admitting area or initially on the inpatient unit was her behavior evaluated for anything other than mania, nor was her speech and language recognized as anything other than "confused". She initially received no evaluation for stroke. Once the diagnosis was made, psychotropic medication was avoided and her hypertension controlled. She was quickly able to return to the nursing home.[68]

The DSM and ICD formats limit meaningful examination

The checklist approach of the DSM and the brief paragraph offerings of the ICD are at best concluding summaries of the psychiatric examination. They are inadequate as guideposts to the examination. For example, illness onsets of hours, days, weeks and months have different diagnostic implications regardless of how the patient appears in the full expression of the disorder. Patient 1.6 has the cross-sectional features consistent with the diagnosis of schizophrenia, but the onset of his symptoms is distinctly not that of classic schizophrenia.

Patient 1.6

A 28-year-old man experienced auditory hallucinations (voices commenting and conversing) daily for years. The hallucinations were perceived as originating from a non-specific external source. The voices were loud, clear, and derogatory in content. They were most intense for several hours in the morning, but the patient would hear them occasionally in the early afternoon. He recognized that his experiences were a sign of illness, but when the voices were most intense, he believed them to be real and not self-generated. He did not work, had no future plans, and mostly kept to himself, worrying about the voices and fearful of their inevitable return. Emotional expression was intact, moods appropriate, and no speech or language disorder was noted. He was occasionally suspicious of strangers, assuming that they might be the source of the voices. He had never been depressed or manic. Meeting DSM criteria for schizophrenia, several antipsychotics had been prescribed, with minimal relief.

A consultant noted that the man's morning hallucinations typically began upon awakening. The patient would awake, become immediately frightened, and then hear the voices. After several hours, they diminished in intensity and ended. They recurred shortly after lunch. Because a nonaffective psychosis

with preserved emotional expression is often associated with recognizable neurologic disease,[69] and hallucinations that are linked to a specific time of day, event, or stimulus are also most likely due to such disease, the hallucinations were considered post-ictal consequences of seizures that occurred upon wakening (when the afternoon voices occurred it was after a heavy lunch followed by a nap). Carbamazepine resolved the psychotic features.

The DSM and ICD do not incorporate the nuances described in Patient 1.6 despite many descriptions of the psychopathology associated with epilepsy,[70] and the high frequency with which depression, psychosis and personality change occur in epileptic patients. As a result, such patients come frequently to psychiatric clinics and hospitals for care. But their seizure disorder is unrecognized.

Patient 1.7 further illustrates how reliance on short lists of vaguely delineated symptoms and signs leads to misdiagnosis.

Patient 1.7

A 78-year-old woman lived independently until she was diagnosed as being depressed and prescribed buproprion. She progressively lost the ability to care for herself. "Confusion", followed by mutism and immobility, led her daughter to bring her to an emergency room. Thought to be experiencing a stroke, she was admitted to a neurology service. The MRI indicated mild old ischemic disease, but provided no explanation of her present state. The mutism and immobility resolved within an hour of admission. Over the next several hours the patient's state fluctuated from "confusion" to apparent alertness. It was "observed" that her episodes of confusion occurred when her daughter was present, but resolved when the daughter left the room.

The alternating periods of "confusion" and alertness were interpreted as evidence of hysteria or conversion disorder. The catatonic features noted in the emergency room and the previous diagnosis of depression were consistent with this conclusion and she was transferred to the psychiatry inpatient unit.

On the psychiatry unit, when lucid she showed reduced affective intensity, sadness, and psychomotor retardation. She was pessimistic about her future. Prior to hospitalization she had been eating and sleeping poorly. A psychiatry consultant noted the patient was subdued and appeared tired, but that she retained some humor, inconsistent with the degree of her depressive features. (The DSM does not consider patterns of features, but rather the number of features.) The consultant also noted that the patient's periods of "confusion" began abruptly and were characterized by not fully understanding the examiner's questions, although she could repeat some words and phrases. Some of her responses were non-sequitive. Others were laced with phrases that made no

sense, odd sounding words and imprecise word usage (the DSM does not define speech and language disorders in specifics as do neurology texts. It considers episode duration, but not duration or fluctuations of individual features).

The patient's speech was recognized as episodes of transient sensory aphasia. Psychopathology from idiopathic disorders, however, does not typically begin in seconds, and aphasia from vascular disease does not come and go abruptly. Also, transient catatonia unrelated to a manic-depressive disorder is often due to recognizable neurologic disease. Non-convulsive status epilepticus was considered and confirmed by EEG. IV anticonvulsants resolved the patient's acute state.

The DSM and ICD offer a few clinical features as sufficient for each diagnostic class. The DSM requires a patient to exhibit a specific number of criteria for the diagnosis. The combinations of features, their characteristic onset, the relationships among different patterns, and the context in which symptoms unfold are rarely addressed. Although the duration of a syndrome in days, weeks or months is a common requirement to aid reliability, the more difficult assessment of the quality of symptom onsets (e.g. the rate of their emergence), the sequence of symptom appearances, and symptom pattern are not incorporated. Patient 1.8 meets DSM criteria for major depression, but the split-second change in psychopathology typically indicates a secondary syndrome.

Patient 1.8

A middle-aged man became profoundly gloomy, pessimistic and unable to work. He whined and tearlessly cried, pleading for help. He needed repeated reassurance. He made several serious suicide attempts (e.g. attempted hanging). The depressive episodes typically began suddenly in the late afternoon and slowly resolved by evening. They occurred daily. On several occasions a depressive episode lasted a week or more. A seizure disorder was recognized, verified by EEG. The illness resolved with anticonvulsant treatment.

Ignoring symptom patterns encourages misdiagnosis. For example, the DSM catatonia criteria[71] require two of five features to be met. If both excessive "purposeless" activity (item 2) and echolalia or echopraxia (item 5) are present, the patient is said to be catatonic. Manic patients, however, seem purposeless in their actions when in heightened excitement. They show echophenomena.[72] These criteria, as others, require the patient to receive a diagnosis of a psychotic disorder, and most likely that of schizophrenia. Such classification is followed by antipsychotic medication rather than treatments for mood disorder.

The inadequacy of relying on a few features for diagnosis and not obtaining the story of the patient's illness is further illustrated by the unique study of

Rosenham (1973). He recruited eight non-ill persons to seek admission to psychiatric facilities complaining of experiencing auditory hallucination of the words "empty . . . hollow . . . thud" over a period of several weeks. The remainder of their statements and answers to their examiner's questions were truthful and they acted in their usual manner. In 11 of the 12 presentations the "pseudo-patients" were hospitalized with the diagnosis of schizophrenia. The inpatient staff also considered them to be ill, although many other patients recognized the sham.

The DSM and ICD are non-theoretical systems in a neuroscience world

Classifying psychiatric patients by their shared signs and symptoms is traditional. Objectively observing and organizing this information is essential in the diagnostic process and the DSM and ICD take this position. Many clinical features can be understood within a neurologic framework, however, and many patients require assessment beyond the sketchy evaluation offered in the DSM and ICD manuals. Recognizing *psychosensory features*[73] in a patient with panic disorder, for example, directs the examiner toward a diagnosis of seizure disorder.[74] Identifying these features in a patient with manic-depressive illness influences treatment (the use of anticonvulsants as mood stabilizers rather than lithium) and prognostic concerns (the greater likelihood of chronicity and cognitive decline).[75] The presence of Capgras syndrome raises the possibility of a temporo-parietal stroke as seen in Patient 1.4.

Present classification does not incorporate the known brain and behavior relationships into diagnostic criteria because unlike some neurologic signs (e.g. hemiparesis, Broca's aphasia), most behavioral signs and symptoms are not localizing to brain sites. The behaviors do, however, reflect dysfunction in specific brain systems or are strongly associated with specific brain syndromes as well or better than are items included in present criteria. Subsequent chapters detail these relationships.

Summary

The framers of the latest DSM and the ICD classifications do not consider the manuals to be textbooks of psychopathology. They caution against their use by "untrained" persons. The DSM, nevertheless, has become the principal guide to psychopathology for an entire generation of psychiatrists in the USA and elsewhere, to the exclusion of works devoted to a fuller understanding of psychopathology. The DSM and ICD achieve adequate reliability only under structured

circumstances and may elicit poor reliability for several categories in the typical clinical setting. The validity of many categories is weak. Sole reliance on DSM criteria and ICD brief descriptions leads to unacceptable false negative and false positive misdiagnoses and the overuse of the NOS category. Much discriminating psychopathology is not included in the classification, nor are recognized neurologic syndromes that are commonly seen in patients seeking help at psychiatric services. The result is that many patients are ill-served.

NOTES

1 Trimble (2002).
2 Mezzich (2002).
3 Sartorius *et al.* (1993, 1995).
4 Panzarino (2000); Doctor Taylor has been teaching psychiatry residents since 1969, Doctor Vaidya since 1989.
5 Hiller *et al.* (1994a,b); Slade and Andrews (2001); Peralta and Cuesta (2003a); Sorensen *et al.* (2005).
6 APA, (1996, 1997).
7 See the STAR D (Rush *et al.*, 2006) "one size fits all" approach to the treatment of depressive illness and its report of results only marginally better than placebo (30% remitted in the first drug trial and an additional 18 25% in the second). Also see the treatment algorithms offered for manic depressive illness (Nierenberg *et al.*, 2006) and psychosis (Schneider *et al.*, 2001).
8 Many of the DSM syndrome choices have sub syndrome modifiers bringing the total to over 350 diagnostic options.
9 Paul McHugh: Overestimating mental illness in America, in A Nation of Crazy People? *The Weekly Standard*, Volume 10, Issue 39, 27 June 2005.
10 Taylor and Vaidya (2005).
11 In contrast, residency programs in the 1960s stressed the recognition of psychopathology. In the first year of residency (following a general internship) one of us (MAT) had a weekly 90 min seminar in descriptive psychopathology that ran for 10 months. This course was separate from other seminars in psychopathology.
12 Bleuler's *Dementia Praecox* (17.6%), Kraepelin's *Manic Depressive Illness* (19.1%), Schneider's *Clinical Psychopathology* (17.6%); four programs (5.9%) used Jasper's *General Psychopathology.* One program each used Kahlbaum's *Catatonia* and Fish's *Schizophrenia.*
13 Macaskill *et al.* (1991).
14 Also see Wakefield (1997).
15 Reliability was expressed as a kappa statistic which corrects for chance agreement. Kappa is the proportion of agreement above or below chance. Zero is only agreement by chance. One is perfect agreement, while 0.50 is agreement half way between chance and perfect agreement. A kappa of 0.7 is considered "good".

16 DSM III Appendix F, pages 467 72.
17 One reason clinicians rely heavily on the NOS category.
18 See Kirk and Kutchins (1992) for a detailed presentation of the promotion of DSM III and the field trial results.
19 Grilo *et al.* (2004).
20 Todd *et al.* (2003).
21 Schneider *et al.* (2004).
22 Brown *et al.* (2001).
23 Linde and Clark (1998).
24 Keller *et al.* (1995).
25 Keller *et al.* (1996); Bertelsen (2002); Blais *et al.* (1997); Woo and Rey (2005); Schneider *et al.* (2004); First *et al.* (2004); Sbrana *et al.* (2005).
26 Siebel *et al.* (1997); Willemse *et al.* (2003).
27 Flaum *et al.* (1991).
28 Ries *et al.* (2001).
29 Sartorius *et al.* (1993); Regier *et al.* (1994).
30 Jager *et al.* (2003).
31 Maj *et al.* (2000).
32 Bertelsen (2002).
33 Kessing (2005a,b).
34 Hiller *et al.* (1994a).
35 Vetter *et al.* (2001).
36 Sartorius *et al.* (1995).
37 Blashfield (1984).
38 Zanarini and Frankenburg (2001).
39 Benazzi (2003b). A structured interview is conducted using a pamphlet requiring the user to ask questions as written and in the order written, regardless of the clinical state of the patient. Follow up questioning is limited. Semi structured interviewing is closer to the traditional clinical examination. All items must be assessed, but the order and wording can be modified by the examiner. Follow up questioning is permitted and essential.
40 Teeney *et al.* (2003); Becker *et al.* (2006).
41 Chapter 4 provides a discussion of the diagnostic process.
42 Clayton (1982); Taylor and Fink (2006), chapters 2 and 4.
43 Taylor and Fink (2006), chapters 3, 10, 11.
44 DSM IV, p. 327.
45 Adapted from Taylor and Fink (2006); also seen in Atre Vaidya and Taylor (2006).
46 There are several frontal lobe syndromes, some of which have signs that overlap with mood disorder. See Chapter 3.
47 Starkstein and Manes (2000).
48 Taylor (1972); Abrams and Taylor (1973).
49 Chapter 7 provides a discussion of catatonic features.
50 Wilson (1989).
51 Holden (1987); Joseph (1999).

52 Kaptsan *et al.* (2000).
53 Pelegrin *et al.* (2001).
54 Onuma (2000).
55 Fink and Taylor (2003).
56 Adapted from Atre Vaidya and Taylor (2004), "The Cherry Pie Lady".
57 Bourget and Whitehurst (2004).
58 Robins and Guze (1970).
59 While their cross sectional criteria are similar, they are defined by duration: brief psychotic disorder (<one month), schizophreniform disorder (>one but <six months), and schizo phrenia (>six months).
60 Benazzi (2003a).
61 Taylor and Fink (2006).
62 Lalonde *et al.* (2001).
63 Savitz *et al.* (2004).
64 Erlenmeyer Kimling and Cornblatt (1984).
65 Walker and Lewine (1990).
66 Helzer *et al.* (2006).
67 Landre *et al.* (1992); Landre and Taylor (1995).
68 Adapted from Atre Vaidya and Taylor (2004).
69 Davison and Bagley (1969).
70 Atre Vaidya and Taylor (1997).
71 DSM IV, p. 289.
72 See Chapter 7.
73 Psychosensory features (e.g. dysmorphopsia, déjà vu) are signs of temporal limbic disease and are described in the epilepsy literature and in studies of patients with manic depressive illness. Atre Vaidya and Taylor (1997).
74 Vazquez and Devinsky (2003).
75 Atre Vaidya *et al.* (1998).