EARLIER this summer, the American Psychiatric Association announced that a 27-member panel will update its official diagnostic handbook, the Diagnostic and Statistical Manual of Mental Disorders. The fifth edition, which is scheduled to come out in 2012, is likely to add new mental illnesses and refine some existing ones.

High on the agenda will be the controversial diagnosis of childhood bipolar disorder. Recent data show that office visits by children and adolescents treated for the condition jumped 40-fold from 1994 to 2003. We still don’t know how much of this increase represents long-overdue care of mentally ill youth and how much comes from facile labeling of youngsters who are merely irritable and moody.

Part of the confusion stems from the lack of a discrete definition of juvenile bipolar illness in the diagnostic manual. But there is a deeper problem: despite the great progress being made in neuroscience, we still don’t have a clear picture of the brain mechanisms underlying bipolar illness — or most other mental illnesses.

For perspective, we must return to 1980, when the revolutionary third edition of the handbook, the D.S.M. III, was published. In a radical break from earlier editions, which had been based largely on psychoanalytic principles of unconscious conflict and stunted sexual development, the D.S.M. III categorized illnesses based on symptoms. A patient was said to have a condition if he or she had a certain number of the classic symptoms for a certain period of time. This approach promoted “inter-rater reliability” — the odds that two examiners would agree on what diagnosis to assign a patient.

Yet the manual remained silent on what caused the symptoms. The diagnosis of, say, schizophrenia did not reflect a known cause in the way syphilis is known to be an infection with a spirochete bacterium. The writers of the D.S.M. III were confident that science would one day fill this vacuum, yet three decades later psychiatry still lacks a firm grasp of the causal underpinnings of mental illness.

One manifestation of our limited knowledge is that many patients meet...
several diagnostic definitions at once. Roughly half of adults with clinical depression, for example, also have symptoms that fit the definition of an anxiety disorder. Do these patients actually suffer more than one illness, or do they just appear to?

Conversely, very diverse patients often qualify for the same diagnosis. “You can have three patients with schizophrenia, but all that really means is that their symptoms fit a particular pattern,” says Dr. Michael First, a psychiatrist who was the editor of the current handbook, the D.S.M. IV. “They may not have the same pathophysiology and, as a result, they may not require the same treatment.”

Indeed, the link between diagnosis and treatment is relatively weak. Antidepressants like Prozac help treat not only depression but also panic disorder, obsessive-compulsive disorder, bulimia and social phobia. This explains why clinicians often treat by symptom rather than diagnosis. Paranoia, for example, is treated with an antipsychotic drug whether it occurs in the context of schizophrenia, bipolar illness or methamphetamine use.

Why aren’t we closer to understanding the relationship between manifest illness and its underlying causes? One obstacle is the staggering complexity of the brain. Another may be what Dr. First calls the “unfortunate rigidity” that all–or–nothing diagnostic checklists and sharply bounded categories impose. In order for the condition of a patient to meet the definition of clinical depression, for example, he or she must have five out of nine symptoms. But does a patient with only four symptoms have a different disorder, or no disorder at all?

One way to improve the classification of mental illnesses would be to define certain pathologies along a continuum so that patients who are truly ill won’t fall short of qualifying for a diagnosis. Take major depression. The symptoms could be weighted so that suicidal preoccupation or immobilization, the most extreme and debilitating aspects, would get high scores, while loss of energy and interest for a short periods would get lower scores. Thus, a patient with few, but severe, symptoms would not be excluded.

A more nuanced approach could also make a real difference for population surveys of mental illness and clinical trials, both of which tend to rely on rigid symptom checklists.

An updated manual, however, is unlikely to transform treatment substantially.
— after all, revising diagnoses is still just another way to describe mental conditions we don’t fully understand. But these refinements may well stimulate valuable new inquiry, enabling swifter progress in understanding the mechanisms of disease, better deployment of treatments we have and more efficient discovery of new ones.