As psychiatry has become increasingly dominated by the neurosciences, the role of psychoanalytic treatments within psychiatry has become more and more controversial. Psychoanalytic treatments may be necessary when other treatments are not effective, or for the residual, often less manifest, problems that persist after brief interventions. After reviewing existing evidence on the efficacy and effectiveness of psychoanalytic treatments, Gabbard et al (p 505) propose a research agenda for 21st-century psychiatry that will answer the still-unresolved questions about these treatments.

Several postmortem studies showed an elevation of serotonin 5-HT1A receptor density in schizophrenia. In the first study examining those receptors in schizophrenic patients in vivo using positron emission tomography and [11C]WAY-100635, Tauscher et al (p 514) demonstrate a higher 5-HT1A receptor binding potential in the cortex of schizophrenic patients as compared with an age-matched healthy control group. While the biological significance of elevated 5-HT1A receptor density in schizophrenia remains unclear, this finding needs to been seen in the light of preclinical evidence supporting a role for 5-HT1A receptors in mediating antipsychotic action and extrapyramidal side effects of drugs.

Heckers et al (p 521) studied messenger RNA (mRNA) expression of glutamic acid decarboxylase (GAD), the hallmark enzyme of GABAergic neurons, in the hippocampus of bipolar and schizophrenic patients. The number of GAD mRNA-positive neurons was markedly reduced in bipolar disorder and showed a more subtle decrease in schizophrenia. The cellular expression of GAD65 mRNA was significantly reduced in one hippocampal sector, CA4, in bipolar disorder. These findings confirm previous reports of an abnormal GABA system in both disorders, and provide novel evidence for an abnormal regulation of GAD in hippocampal neurons in bipolar disorder.

This is the first prospective longitudinal study of the natural history of the weekly symptomatic status of bipolar I disorders. During a mean of 13.2 years of follow-up, bipolar I emerged as a chronic illness in which patients were symptomatic 47% of the time. Judd et al (p 530) also found the weekly symptom status is primarily depressive rather than manic (3:1 ratio, respectively). Subthreshold affective symptoms were 3 times more common than symptoms at the manic and major depressive level. Longitudinally, symptom severity and polarity fluctuate frequently within the same patient.

There are questions about long-term outcomes of cocaine-dependent patients following discharge. Simpson et al (p 538) found that only 1 in 4 patients admitted to a national sample of treatment programs still used cocaine on a weekly basis 5 years later—comparable to rates reported in the first year after treatment. Greater severity of drug use and related problems at intake as well as more limited treatment contact were related to poorer outcomes on drug use and criminal activity.

Johnson et al (p 545) found that adolescents with eating disorders were at substantially elevated risk for anxiety disorders, cardiovascular symptoms, chronic fatigue, chronic pain, depressive disorders, limitations in activities, infectious diseases, insomnia, neurological symptoms, and suicide attempts during early adulthood after adolescent health problems, psychiatric comorbidity, body mass index, socioeconomic status, and worries about health during adulthood were controlled statistically. These findings suggest that adolescent eating disorders may contribute to the development of physical and mental health problems during early adulthood.

Several recent magnetic resonance imaging (MRI) studies have reported progressive changes in the size of various brain structures, supporting speculation about neurodegeneration. Weinberger et al (p 553) question the meaning of the progressive changes found with MRI and the improbability that they reflect neurodegeneration, including that the pattern of changes are inconsistent from one study to another, that most patients actually improved clinically while these apparent progressive changes emerged, and that the magnitude of the changes rivals that found in neurodegenerative disorders such as Alzheimer disease, yet that evidence of neurodegeneration is conspicuously absent from postmortem tissue studies. It is concluded that MRI evidence of progressive changes probably reflects something other than irreversible neurodegeneration.