In a longitudinal study, Penninx et al (p 221) examined 2 groups of older persons—with and without cardiac disease—and showed that depression increased the cardiac mortality risk to a similar extent in both groups over 4 to 5 years. The excess cardiac mortality risk was twice as high for major depression as for minor depression. The study suggests that depression increases the risk for cardiac death, irrespective of cardiac disease status.

A commentary by Carney, Freedland, and Jaffe is included.

In a community sample of youths, Kasen et al (p 231) found that major depressive disorder increased the likelihood of antisocial, dependent, histrionic, and passive-aggressive personality disorders in young adulthood after accounting for other potential risks, including disadvantaged socioeconomic status, child maltreatment, a nonintact family, exposure to parental conflict, and co-occurrence of other childhood disorders.

A commentary by Harrington is included.

Patients with a history of recurrent depression or dysthymia and major depression have a significant risk of relapse. Katon et al (p 241) demonstrated in a randomized controlled trial that a relapse prevention program targeted to primary care patients with high epidemiologic risk of relapse was associated with improved adherence to antidepressants and depressive outcomes over a 1-year period compared with controls treated with usual primary care. This low-cost primary care–based intervention holds promise for improving long-term outcomes of this relapsing/remitting illness.

In this prospective, community-based, longitudinal study, Stein et al (p 251) found that social anxiety disorder in adolescence or early adulthood was a predictor of later depressive disorders. Moreover, the presence of comorbid social anxiety disorder in adolescents who are already depressed was associated with a more malignant course and character of subsequent depressive illness.

In this analysis of male–male pairs by Kendler et al (p 257), all phobia subtypes aggregated within twin pairs. This aggregation was due largely or entirely to genetic factors with moderate heritability. Multivariate analysis revealed one common genetic factor, genetic factors specific to each subtype, and a common familial-environmental factor. These results suggest that in males, genetic risk factors play a moderate role in the etiology of phobias and their associated irrational fears.

The monoamine oxidase inhibitor phenelzine can completely eliminate REM sleep. Landolt et al (p 268) investigated the relationship between REM sleep suppression and antidepressant response and the effect of phenelzine on EEG slow-wave activity, a well-established marker of non-REM sleep intensity. Antidepressant response to the treatment was independent from changes in REM sleep or slow-wave activity. In the absence of REM sleep, slow-wave activity declined exponentially. Dream recall decreased during treatment only in antidepressant responders, whereas nonresponders showed no change.

In this study, Barch et al (p 280) used functional magnetic resonance imaging to examine prefrontal cortex (PFC) activation in medication-naive patients with first-episode schizophrenia during a task that isolated a specific subcomponent of working memory, context processing. Patients with schizophrenia demonstrated selective deficits in dorsolateral PFC activation in this context processing task, but intact activation of posterior and inferior prefrontal cortex. These results suggest that a specific deficit in PFC function, associated with an impairment in context processing, is present at illness onset in schizophrenia, prior to the administration of medication or most confounding effects of illness duration.

Castellanos et al (p 289) compared magnetic resonance images of girls with combined-type attention-deficit/hyperactivity disorder (ADHD) with those of healthy matched controls with respect to volume for brain regions previously identified as abnormal in boys with ADHD. They confirmed a modest though significant reduction in total cerebral volume, and a more substantial decrease in the volume of the posterior-inferior cerebellar vermis. The results confirm previous findings for boys in the cerebellum, further support a role for the caudate nucleus in ADHD, and demonstrate that the neurobiology of ADHD is basically comparable in girls and boys, despite continuing sex differences in rate of diagnosis.

Female adolescent antisocial behavior is increasing, but little is known about the neuroendocrinological aspects of this disorder. Based on reports of decreased cortisol in antisocial males, Pajer et al (p 297) studied morning plasma cortisol levels in adolescent girls with conduct disorder and found that antisocial girls had significantly lower morning cortisol levels than normal controls.