Research

Placebo Effects on the Neurologic Pain Signature 1321
The mechanism by which the placebo effect works in the brain is not well understood. Zunhammer and coauthors conducted a systematic meta-analysis of participant-level functional magnetic resonance imaging data from 20 studies including 603 healthy participants. They tested whether placebo treatments affect pain processing in the neurologic pain signature, a cerebral method validated to predict the intensity of evoked pain in a sensitive and specific fashion. Placebo treatments were found to have moderate analgesic effects on reported pain but very small effects on signature responses. These results indicate that placebo analgesia is predominantly mediated by networks different from those underlying the primary processing of noxious stimuli. Editorial perspective is provided by Spiegel.

Editorial 1309

Stroke Quality of Care in GWTG-Stroke Hospitals 1331
Do hospitals using the Get With The Guidelines (GWTG)-Stroke program improve quality-of-care measurements for stroke patients compared with hospitals that do not participate? Howard and coauthors identified a subpopulation of 546 participants who experienced an ischemic stroke during a 9-year follow-up in the Reasons for Geographic and Racial Differences in Stroke (REGARDS) Study, a longitudinal cohort study. A total of 207 patients were treated in a hospital participating in the GWTG-Stroke program and 339 were treated in a nonparticipating hospital. Patients in GWTG-Stroke hospitals were more likely to receive evidence-based interventions, including intravenous thrombolytics, a swallowing evaluation, stroke risk factor education, and evaluation by a neurologist. Editorial perspective is provided by Webb.

Editorial 1311

Association of APOE ε4 With TDP-43 in Alzheimer Disease 1347
Alzheimer disease (AD) has traditionally been characterized by the aggregation of β-amyloid and paired helical filament tau. Transactive response DNA-binding protein 43 (TDP-43) has also been associated with AD, but its role has not been extensively explored even though TDP-43 is estimated to be present in the brains of 65% to 80% of patients with AD. Wennberg and coauthors performed neuropathological examinations for a population-based, cross-sectional, genetic-histological study of 738 older adults (median [interquartile range] age, 87 [51-105] years) with an AD spectrum pathological diagnosis enrolled in the Mayo Clinic Alzheimer Disease Research Center, Mayo Clinic Alzheimer Disease Patient Registry, or Mayo Clinic Study of Aging. Apolipoprotein E (APOE) ε4 was found to be directly associated with TDP-43 in patients with an intermediate to high likelihood of having AD; importantly, this was independent of β-amyloid status.

Author Audio Interview jamaneurology.com

Association of Midlife Risk Factors With Late-Onset Epilepsy 1375
The incidence of epilepsy is higher in older age than at any other period of life. While stroke, dementia, and hypertension are associated with late-onset epilepsy, the role of other vascular and lifestyle risks is unclear. In a prospective cohort study using data from the biracial, community-based Atherosclerosis Risk in Communities (ARIC) study, Johnson and coauthors examined risk factors measured at age 45 to 64 years in 10,420 participants and the development of subsequent late-onset epilepsy starting at age 60 years or later. While the apolipoprotein ε4 genotype was found to be associated with late-onset epilepsy, so were potentially modifiable factors, such as midlife hypertension, diabetes, smoking, alcohol consumption, and physical activity level, suggesting that lifestyle modifications could be adopted to decrease the risk of developing late-onset epilepsy.