What is the best laxative for children older than 2 years?

Bottom line
Polyethylene glycol (PEG), lactulose, and Milk of Magnesia have similar effectiveness in relieving constipation in children. However, PEG products are generally better tolerated in this age group.

Background
Constipation can be defined as abnormally delayed or infrequent passage of dry, hardened feces. The American Academy of Pediatrics estimates that complaints concerning constipation account for up to 3% of pediatric office visits. A variety of serious medical conditions can cause constipation in children, including cystic fibrosis, hypothyroidism, spina bifida, and Hirschsprung’s disease. Most children who have constipation, however, have no anatomic or genetic disorder. Wide variation in stool patterns can be found among nonconstipated children; therefore, parental education is often necessary to help correctly identify constipation.

Some of the same dietary issues that lead to constipation in adults are to blame for constipation in children, such as inadequate intake of fiber and fluids. Other contributing factors include inconsistent toilet training and medication side effects. If correction of diet or behavior modification is not effective as first-line therapy, a number of medications have been shown to be safe in infants and children. These include polyethylene glycol (PEG), lactulose, and Milk of Magnesia (magnesium hydroxide suspension).

Summary of the evidence
Head-to-head trials
Two head-to-head laxative effectiveness trials are available. A randomized, open-label crossover trial enrolled 37 children aged 2 to 16 years referred for subspecialty evaluation of functional constipation. Results showed that PEG-3350 and lactulose were equivalent in improving stool frequency, stool form, and ease of passage. However, total stool transit time was significantly reduced in children taking PEG compared with participants taking lactulose (47.6 vs 55.3 hours,
In addition, twice as many parents and guardians rated PEG effective as those rating lactulose effective (84% vs 46%, \( P = .002 \)); and 73% of parents preferred PEG to lactulose.\(^1\)

A different randomized controlled trial (RCT) compared PEG with Milk of Magnesia in 49 children aged 4 years and older with functional constipation and encopresis. Follow-up at 1, 3, 6, and 12 months revealed similar effectiveness in increasing bowel movement frequency, decreasing soiling episodes, and decreasing abdominal pain. Follow-up also revealed that PEG was more palatable and better tolerated than Milk of Magnesia (33% of children refused to take Milk of Magnesia, whereas none refused PEG). No side effects from PEG were reported.\(^2\)

**PEG-3350 vs placebo**

In another prospective, multicenter, double-blinded study, 103 children aged 4 to 16 years (mean 8.5 years) with functional constipation were randomized to receive placebo, or 0.2, 0.4, or 0.8 g/kg per day of PEG-3350 after a 1-week run-in period, followed by 2 weeks of treatment. All children also received behavior modification therapy. The main endpoint was the proportion of patients with a successful treatment response, defined as 3 or more bowel movements in the second week.

Treatment was successful in 77%, 74%, and 73% of patients in the groups receiving 0.2, 0.4, and 0.8 g/kg, respectively, compared with 42% in the placebo group \((P<.05, \text{for each intervention group compared with placebo})\). A positive dose response to PEG was seen at all dosages, resulting in increased movement frequency, reduced straining, and softer stool consistency. However, patients receiving 0.8 g/kg of PEG-3350 had more abdominal pain and fecal incontinence. The authors recommended using a starting dose of 0.4 g/kg per day PEG-3350 in clinical situations.\(^3\)

**Probiotics**

Only 1 RCT has addressed the use of probiotics in the treatment of children with constipation. Eighty-four children aged 2 to 16 years with fewer than 3 spontaneous bowel movements per week for at least 12 weeks were enrolled. The treatment group received 70% lactulose (1 mL/kg per day) plus \(10^9\) CFUs of *Lactobacillus* strain GG orally twice daily for 12 weeks. The control group received lactulose and a placebo. The analyses were performed on an intent-to-treat basis.

Treatment success, defined as more than 3 spontaneous bowel movements per week with no fecal soiling, was similar in the control and experimental groups at 12 (68% vs 72%, respectively) and 24 weeks (65% vs 64%, respectively). The groups also did not differ in the mean number of spontaneous bowel movements per week or episodes of fecal soiling per week at 4, 8, and 12 weeks. Adverse events and overall tolerance did not differ between groups. It was concluded that *Lactobacillus* GG, as used in this study, was not an effective adjunct to lactulose in treating constipation in children.\(^4\)

**Conclusion**

According to the above studies, PEG, lactulose, and Milk of Magnesia treatment all led to a significant increase in the total number of defecations per week and improvement in stool consistency for children with constipation aged 2 years and older. All treatments were also associated with a significant reduction in pain, straining, and hard stools. However, PEG products tended to be better tolerated in head-to-head comparisons. Currently, no evidence is available to recommend the use of probiotics for children with constipation. Finally, it should be reiterated that most experts recommend lifestyle and behavior modification along with the above treatments.

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**REFERENCES**


Dear EBP Readers,

Here at Evidence-Based Practice, we have an affinity for confidence intervals. They are presented in our pages to keep our readers humble, aware that the magnitude of some effect or other is not truly known and fixed. However, they are also reassuring, because the magnitude of an effect is most probably somewhere within the range of the confidence interval.

But it turns out that about 1 mm below the cortex, human brains do not really like confidence intervals. This places the confidence interval in good company. A millimeter below the cortex, human brains don’t really like a lot of reasonable, rational ideas—like quitting smoking, eating vegetables, and utilizing the buddy system at camp.

That we humans appear to be wired for eminence-based medicine rather than for evidence-based medicine was demonstrated in an experiment by Don Moore at Carnegie Mellon University and featured on Dan Ariely’s blog site, Predictably Irrational.1 In this experiment, volunteers were paid to guess the correct weight of people in photos. But before guessing, they could purchase advice from 4 “experts.” The experts gave ranges for the correct weight.

Very quickly it became clear that volunteers preferred to buy advice from experts who appeared the most confident. The experts who proposed narrower ranges of weights sold more advice. This was despite the fact that by proposing a narrower range, the more confident experts were more likely to be wrong. But still, their advice sold. Over time, all the experts (who were also volunteers and a part of the experiment) narrowed the spread of their advice, and became increasingly rewarded for appearing ever more certain, even as the advice got worse.

So it seems that human beings naturally prefer confidence over confidence intervals. If that tendency manifests in most folks listening to TV pundits and know-it-alls elsewhere, so be it. We at EBP, however, will continue to speak as directly as possible to your top 1 mm of gray matter, the one that helps a good doctor make the best decisions.

Regards,

Jon O. Neher, MD

REFERENCE

Is there a link between obesity and low serum vitamin D levels?

Bottom line
There is an epidemiologic association between obesity and low serum 25(OH)-vitamin D levels that has been noted in many demographic groups. Proposed mechanisms include less activity leading to less sunlight exposure, and sequestering of vitamin D in adipose tissue.

Background
Vitamin D deficiency has traditionally been defined as a serum value of <27.5 nmol/L, with insufficiency at 27.5 to 50 nmol/L, and sufficiency at 50 to 125 nmol/L. However, some more recent laboratory reference values set deficiency at <25 nmol/L, insufficiency at 25 to 75 nmol/L, and sufficiency at 75 to 250 nmol/L. (To convert to ng/mL, divide by 2.5.)

A report on the cross-sectional National Health and Nutrition Examination Survey (NHANES) for 2000–2004 showed that for US adults older than 20, 6% had levels <27.5 nmol/L, 30% had levels <50 nmol/L, and 75% had levels <80 nmol/L. A chart from the report shows serum levels in Mexican Americans roughly 10 nmol/L lower than that in Caucasians, and levels in African Americans another 10 nmol/L lower than that in Mexican Americans.1 A worldwide meta-analysis of 394 cross-sectional studies reported a mean serum 25(OH)-vitamin D level of 54 nmol/L (95% confidence interval [CI], 52–57 nmol/L).2

The current dietary reference intake (DRI) for vitamin D is an adequate intake of 200 IU/d through age 50, 400 IU for ages 51 to 70, and 600 IU for older than 70 years. However, typical intake from food is roughly 100 to 150 IU/d.3 The Institute of Medicine is reevaluating the DRIs for vitamin D and calcium and is expected to increase the adult adequate intakes for vitamin D when the report is issued in May 2010. One expert estimated a daily intake ≥1,000 IU would be needed to move the vitamin D serum levels of most Americans above 75 nmol/L.4

Obesity as a risk factor
The NHANES for 2001–2004 included 9,757 children and adolescents aged 1 to 21 years. The adjusted odds ratio of vitamin D deficiency (here defined as <37.5 nmol/L) for obese children compared with non-obese children was 1.9 (95% CI, 1.5–2.5).5 For adults, multiple epidemiologic studies have indicated that lower serum 25(OH)-vitamin D is associated with higher body mass index (BMI).1,6–8

In a study conducted in Finland this relationship was true for the subsets women <50 or ≥50 years, and for men <50 or ≥50 years. For men <50 years, those with a BMI of 20 to 24 had a mean serum 25(OH)-vitamin D level of 85 nmol/L, compared with 59 nmol/L for BMI ≥40.6 Similar inverse relationships between serum 25(OH)-vitamin D and BMI have been reported in a cross-sectional evaluation of 1,606 elderly men conducted in Portland7 and as part of the longitudinal Insulin Resistance Atherosclerosis Study (IRAS) incorporating 917 Hispanic and 439 African American adults.8

Possible mechanisms
Speculations on mechanism are both social and/or physiological. Obese people may tend to engage in less outdoor exercise or physical labor, and, if they do, may expose less skin to sunshine.

The NHANES children’s study noted that the odds ratio for vitamin D deficiency was 1.6 (95% CI, 1.1–2.3) for video screen viewing of more than 4 hours per day compared to viewing of ≤2 hours per day.9

Other experts, doubting a synthesis defect, theorize that what is synthesized or absorbed from food or dietary supplements is sequestered in both subcutaneous and visceral adipose tissue, resulting in low serum concentrations.6,8 Regardless of the mechanism, it appears that obese persons are generally at increased risk for vitamin D deficiency.

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*Dr. Mark discloses that he has consulting clients that sell vitamin D-containing dietary supplements.

What is the best treatment for chronic post-CABG pain?

Evidence-Based Answer
Evidence is insufficient to choose a single best agent. However, amitriptyline, capsaicin cream, opioids, and NMDA antagonists are effective choices for other postsurgical pain syndromes and may be effective treatments of pain after coronary artery bypass grafting (CABG). (SOR C, extrapolated from randomized controlled trials [RCTs] involving other surgeries.) Gabapentin and lamotrigine, commonly used for neuropathic pain, are not effective for postsurgical neuralgia. (SOR C, based on expert opinion.)

A 2001 retrospective cohort study was designed to characterize the prevalence of post-CABG pain syndrome. Data were available on 387 patients who underwent CABG at a single clinical center. Post-CABG pain was defined as chest wall pain of more than 3 months’ duration that appeared after CABG or was different from the patient’s preoperative angina. Questionnaires and phone calls were used to screen participants. The prevalence of post-CABG pain was 56% (217/387). Most of these participants (65%) rated the intensity of their pain at least “moderate,” and 70% of participants noted that their pain limited their daily activities.

No studies were found that directly address pain management in the post-CABG pain syndrome. A 2004 narrative review, however, categorizes post-CABG pain as a subtype of postsurgical neuralgia. The author stated that post-CABG pain is neuropathic in origin, due to traumatic nerve injury to the intercostal nerves that occurs during harvest of the internal mammary artery. The author also pointed to RCTs, which study interventions for postmastectomy pain, to guide therapy of post-CABG pain.

A 1996 randomized, double-blind, placebo-controlled crossover study was designed to determine the efficacy of amitriptyline versus placebo in postmastectomy neuropathic pain. Fifteen patients were enrolled and instructed to take amitriptyline at escalating doses from 25 to 100 mg daily or placebo for more than 4 weeks. Treatments were switched after a 2-week washout period. Using the Finnish McGill Pain Questionnaire, patients reported an 82% decrease in chest scar pain with amitriptyline versus a 28% reduction with placebo (P<0.05).

A 1998 double-blind RCT demonstrated superiority of the NMDA antagonist amantadine over placebo for the treatment of surgical neuropathic pain in cancer patients. Fifteen patients were administered infusions of 200 mg amantadine and placebo, in a randomized order, separated by 1 week’s time. Both spontaneous and evoked pain measurements were assessed via a visual analog scale. Average pain reduction with amantadine was 85% compared with 45% with placebo (P<0.01). Two days after treatment, pain intensity was reassessed. Amantadine remained superior to placebo, with a 31% reduction in pain versus 6% for placebo (P<0.01).

A 1997 RCT involving 99 patients evaluated the effect of topical 0.075% capsaicin cream on postsurgical neuropathic pain in cancer patients. Capsaicin or placebo cream was applied 4 times daily for 8 weeks, followed by an 8-week crossover. Patients reported a 53% reduction in pain with capsaicin versus 17% with placebo (P=0.01).

Although gabapentin and lamotrigine are commonly used for neuropathic pain syndromes, such as postherpetic and diabetic neuralgia, no superior analgesic effect has been seen over placebo in posttraumatic neuralgia.

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When is medication appropriate for chronic insomnia?

Evidence-Based Answer
Medications are an appropriate second-line treatment if a behavioral intervention is not effective and any comorbid conditions, medications, or substances that might interfere with sleep have been identified and addressed (SOR C, based on expert opinion).

A 2008 evidence-based guideline was developed by a panel of sleep medicine experts convened by the American Academy of Sleep Medicine (AASM). The panel reviewed existing AASM practice parameter papers (each based on a systematic literature search) and obtained additional evidence through a Medline search from 1999 to 2006. The guideline recommends that initial treatment of insomnia should include at least 1 behavioral intervention, based on a level of evidence (LOE) designated as “standard,” and that cognitive behavioral therapy (CBT) should be used alongside long-term pharmacotherapy when possible, based on an LOE designated as “consensus.”

- A “standard” LOE is defined as “a generally accepted patient-care strategy that reflects a high degree of clinical certainty. The term standard generally implies the use of Level 1 Evidence, which directly addresses the clinical issue, or overwhelming Level 2 Evidence.”

- A “consensus” LOE denotes the opinion of the expert panel when evidence is limited or inconclusive.

Other recommendations when considering treatment of insomnia that are not assigned a specific LOE include treating comorbid conditions such as depression and chronic pain, addressing caffeine and alcohol use, and considering current medications that may impair sleep.

The short-term efficacy of behavioral therapy was shown to be slightly better than pharmacotherapy in a meta-analysis comparing 14 studies (250 participants) of stimulus control and sleep restriction therapies with 8 studies (220 participants) of benzodiazepine and benzodiazepine receptor agonists. English language studies of chronic, primary insomnia were found through Medline and psycINFO searches from 1966 to 2000. The weighted effect size for improvement in sleep latency in studies of behavioral therapy (1.05) was significantly larger than in studies of pharmacotherapy (0.45, P=.01). (An effect size of 0.2 is considered small, 0.6 moderate, and 1.2 large.) Otherwise, total sleep time, number of awakenings, sleep quality, and wake time after sleep onset were similar.

Three subsequent randomized controlled trials including 46 to 77 participants have directly compared CBT with zolpidem, zopiclone, or temazepam, and have shown superior long-term efficacy with CBT. Adults with chronic, primary insomnia were randomized to CBT, pharmacotherapy, or placebo for 6 to 8 weeks and then followed for 6 to 12 months. Two of the studies also included a combined CBT and pharmacotherapy treatment arm, and 1 of the studies allowed patients randomized to pharmacotherapy to continue treatment through the 6-month follow-up. Only 1 of the studies reported intent-to-treat analysis.

In the first study, CBT and combination therapy both showed a significant 52% reduction in self-reported sleep-onset latency at the end of treatment, compared with a 14% reduction with pharmacotherapy (P=.003). These changes persisted at the 1-month follow-up with CBT, but not with combination therapy.

In the second study, CBT was significantly better than pharmacotherapy for decreasing self-reported total wake time at the 6-month follow-up (51% vs 27% decrease, P=.03). In the last study, self-reported sleep-onset latency with pharmacotherapy and combined therapy was significantly better than with CBT at the end of treatment (73%, 75%, and 54% reductions, respectively), but after treatment the CBT group continued to improve and the other groups worsened. At the 8-month follow-up, CBT had a significantly better 61% reduction, compared with a 28% reduction with pharmacotherapy (P<.001).

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Is butterbur an effective herbal therapy to prevent migraine?

Evidence-Based Answer
Butterbur (Petasites hybridus) root extract appears to reduce the frequency of migraine headaches. The appropriate dose for optimal clinical effect is unclear. (SOR B, based on heterogeneous randomized controlled trials.)

Migraine affects more than 10% of the population. Many individuals use complementary treatments instead of, or in addition to, over-the-counter and prescription medications. The Petasites hybridus plant has traditionally been used as a migraine treatment. The extract of Petasites root contains petasin and isopetasin and is used as an herbal remedy. The stalks and leaves contain liver-toxic alkaloids and are not used. Petasites is considered a food product in the United States and is not regulated. The German Health Authority licenses a proprietary extract, Petadolex, as a pharmacy medication under full regulatory supervision.

In 2000, a randomized, double-blind, placebo-controlled clinical study of 60 patients treated with butterbur root extract was published. However, the statistical analysis was flawed in several respects and an independent reanalysis of the original data was performed. In the study, 33 patients were randomized to treatment with 50 mg BID of standardized butterbur extract and 27 to placebo. The mean migraine attack frequency per month decreased from 3.4 at baseline to 1.8 after 3 months (P=.0024) in the treatment group and from 2.9 to 2.6 in the placebo group (difference not significant). The responder rate (reduction of migraine frequency by ≤50%) was 45% in the treatment group and 15% in the placebo group (number needed to treat [NNT]=3). Butterbur was well tolerated.

To further assess the clinical effectiveness and tolerability of the herbal remedy, researchers conducted a double-blind, 3-arm, parallel-group, randomized trial comparing Petasites extract 75 mg BID, Petasites extract 50 mg BID, or placebo in 245 patients with migraine. Study participants met International Headache Society criteria for migraine, were between the ages of 18 and 65, and had at least 2 to 6 attacks per month during the preceding 3 months. The main outcome measure was the decrease in migraine attack frequency per month, calculated as percentage change from baseline over a 4-month treatment period.

During the follow-up period, migraine attack frequency fell by 26% in the placebo group. Migraine frequency decreased by 36% for Petasites extract 50 mg BID (P=.127 vs placebo), and 48% with Petasites extract 75 mg BID (P=.0012 vs placebo). The proportion of patients with a ≥50% reduction in attack frequency after 4 months was 68% in the Petasites extract 75-mg arm and 49% in the placebo arm (P<.05; NNT=5). Petasites 50 mg BID was not significantly more effective than placebo in producing a >50% reduction in attack rates. The most frequently reported adverse reactions that could possibly be related to treatment were mild gastrointestinal events, predominantly burping.

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What is the best treatment of recurrent aphthous ulcers?

Evidence-Based Answer
Amlexanox 5% paste significantly reduces ulcer size and duration of pain, and increases speed of healing. (SOR A, based on multiple randomized controlled trials [RCTs].) Topical silver nitrate and corticosteroids reduce pain. (SOR B, based on multiple RCTs.) Chlorhexidine mouthwash reduces overall ulcer burden. (SOR B, based on heterogeneous RCTs.) “Magic mouthwash” is as effective as saline baking soda washes. (SOR B, based on a single RCT.) Nonpharmacologic interventions include avoiding trauma, avoidance of acidic foods/beverages, and minimizing stress. (SOR C, based on expert opinion.)

Recurrent aphthous ulcers (RAUs) appear as erythematous, indurated papules that erode to form necrotic ulcers that are painful and can adversely affect quality of life. RAUs are divided into 3 catego-
ries: minor, major, and herpetiform.¹ This literature review is focused only on treatment of minor aphthous ulcers, which comprise 80% to 85% of RAUs, are 1 to 10 mm in diameter, and heal spontaneously in 7 to 14 days.

A review of 4 double-blind RCTs (n=1,335) comparing amlexanox 5% paste with no treatment showed that amlexanox significantly increased speed of healing, reduced ulcer size, and decreased duration of pain.² If applied during the prodromal stage (the first stage of formation, in which the lesion appears as a small red bump in the mouth), amlexanox dramatically reduced the number of patients who would progress to full ulcers: only 35% of patients with amlexanox, compared with 97% with no treatment (P<.01; number needed to treat [NNT]=1.6). Maximum ulcer size was reduced by 84% (P<.01). A significant proportion of amlexanox recipients had complete reduction in pain compared with the no-treatment group by day 3 (42% vs 22%; P<.05; NNT=5). More patients in the amlexanox group had complete healing by day 3 (47% vs 21%; P<.05; NNT=7). Amlexanox is currently the only product approved by the US FDA for the treatment of aphthous ulcers.

Topical silver nitrate was evaluated in an RCT of 97 patients. Patients were swabbed with a 2% lidocaine swab; then the ulcer was painted with silver nitrate or sucrose (placebo). Pain (on a 3-point scale) and lesion size were measured for 7 days after the procedure. Most of the patients in the treatment group had a reduction of their pain by day 1 compared with the placebo group (33/47 vs 4/38; P<0.01; NNT=1.7). No statistical difference was seen in resolution time.³

Topical corticosteroids were evaluated in a review of 9 RCTs.⁴ Four RCTs reported on pain severity, 3 of which showed a significant reduction in pain or rate of pain reduction. One of 6 RCTs reported a significant reduction in ulcer days. Of note, extended use of topical steroids may lead to candidal overgrowth.

Chlorhexidine was assessed in a review of 5 RCTs (n=203).⁵ Two of the 3 studies that utilized an ulcer index (the sum of ulcers per day over a 4- to 8-week period) found that chlorhexidine reduced the ulcers per day over the measured period. Only 1 of these studies showed a significant increase in ulcer-free days compared with placebo. Only 1 of 5 studies found a reduction in number of new ulcers. All 4 studies that assessed duration of ulceration reported a decrease in ulcer duration, but in only 1 was the difference statistically significant. Two of 5 RCTs showed statistically significant reduction of pain with chlorhexidine.

Magic mouthwashes (made up of various combinations of viscous lidocaine, benzocaine, Milk of Magnesia, kaolin-pectate, chlorhexidine, or diphenhydramine) have been prescribed for years for treatment of oral ulcers. Only 1 RCT was found that assessed magic mouthwash. Patients with chemotherapy-induced mucositis were randomized to magic mouthwash, chlorhexidine mouthwash, or a saline–baking soda rinse. No difference in efficacy was seen among these mouthwashes.⁶

Nonpharmacologic treatment, which may aid in prevention and decreased frequency of ulcers, include good oral hygiene, avoidance of stress, prevention of mucosal trauma, and dietary changes.⁷

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The opinions and assertions contained herein are the private views of the author and are not to be construed as official or as reflecting the views of the Medical Department of the U.S. Air Force or the U.S. Air Force at large.

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“Evidence based medicine is the conscientious, explicit, and judicious use of current best evidence in making decisions about the care of individual patients. The practice of evidence based medicine means integrating individual clinical expertise with the best available external clinical evidence from systematic research.”

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Does stress management improve outcomes of patients with heart disease?

Evidence-Based Answer

In patients with cardiac disease, stress management techniques reduce cardiac mortality, risk of nonfatal myocardial infarction (MI), and depression. Stress management interventions have not been shown to improve all-cause mortality. (SOR A, based on a meta-analysis.)

In 2004, a meta-analysis of 36 randomized controlled trials (RCTs) examined numerous nonpharmacologic psychological interventions with a minimum follow-up time of 6 months for adults with coronary heart disease (CHD; total n=12,841). Overall, there was a 22% reduction in nonfatal MIs (odds ratio [OR] 0.78; 95% confidence interval [CI], 0.67–0.90), but cardiac and all-cause mortality were not reduced (OR 0.86; 95% CI, 0.72–1.03; and OR 0.93; 95% CI, 0.81–1.06, respectively). Significant reduction in depression (using a number of different measures) was also reported (standard mean difference, –0.3; 95% CI, –0.48 to –0.13).

Of the 36 trials in this meta-analysis, 18 tested stress management techniques (utilizing relaxation training, cognitive challenge, and/or specific coping strategies). Among these 18 trials (n=3,425), there was a 31% reduction in nonfatal MIs (OR 0.69; 95% CI, 0.52–0.92). Total mortality was unaffected (OR 0.88; 95% CI, 0.6–1.15), while cardiac mortality was mildly reduced (OR 0.62; 95% CI, 0.38–0.99).

A 2005 RCT (n=154) examined whether an integrative medicine intervention (combination of mindfulness meditation, relaxation training, and stress management, as well as education on nutrition, physical activity, and lifestyle) reduced the 10-year risk of CHD among participants older than 45 years with 1 or more CHD risk factors. There was a 16% relative decline in the intervention group for 10-year CHD risk as determined by Framingham risk scores versus 12% relative decline in the usual-care group (P=.04). (Risk factors included in the Framingham calculation are age, total cholesterol, high-density lipoprotein cholesterol, systolic blood pressure, treatment for hypertension, and cigarette smoking.)

In 2001, a small pilot RCT (N=14, 12 male, 2 female), not included in the Cochrane review because of a less than 6-month follow-up, evaluated progressive muscle relaxation (PMR) for stress reduction in patients undergoing phase 2 or 3 cardiac rehabilitation. The active group received four 50-minute weekly PMR sessions along with general cardiac rehabilitative care, whereas the control group received only usual cardiac rehabilitation. Analyses of resting heart rate (HR) changes showed a trend toward lower HR after 4 weeks in the PMR group (71 vs 65 bpm, P=.052). Analysis of State-Trait Anxiety Inventory scores (scores vary from 20 to 80, with higher scores indicating higher anxiety levels) showed a significant reduction in state anxiety (mean scores 38 vs. 27; P<.05) after 4 weeks in the PMR group versus a nonsignificant change in the control group.³

What can be done to slow progression of dementia in the elderly?

Evidence-Based Answer

Cholinesterase inhibitors alone or in combination with memantine delay the rate of nursing home admissions among elderly patients with Alzheimer’s disease (AD), but have no statistical influence on mortality. (SOR B, based on an observational study.) Cholinesterase inhibitors are associated with small improvements in cognition and function in patients with AD when compared with placebo (SOR A, based on a meta-analysis.) The total societal medical cost of donepezil therapy is comparable to the cost of placebo. (SOR B, based on 1 randomized controlled trial [RCT].)

An observational study published in 2009 followed 943 patients with dementia for 0.8 to 18 years to determine the effect of various dementia therapies on nursing home admissions and mortality. Among this group, 416 patients received no dementia treatment, 387 received cholinesterase inhibitors alone (donepezil, tacrine, rivastigmine, or galantamine), and 140 patients received a combined cholinesterase inhibitor and memantine therapy. The mean treatment time for cholinesterase inhibitors was 38.4 months, and for memantine was 19.2 months.¹
About 49% (203/416) of the patients with no dementia therapy were admitted to nursing home facilities, compared with only 21% (83/387) of patients on cholinesterase inhibitor treatments and 5% (7/140) of patients on combined memantine and cholinesterase inhibitor treatments. Compared with no therapy, the relative hazard ratio (RH) for admission among patients taking cholinesterase inhibitors alone was 0.37 (95% confidence interval [CI], 0.27–0.49); the RH was 0.29 (95% CI, 0.11–0.72) for patients taking memantine and cholinesterase inhibitors. The RH for all-cause mortality was 1.1 (95% CI, 0.88–1.38) for the cholinesterase inhibitors and 0.92 (95% CI, 0.56–1.49) for combined therapy, when compared with untreated patients.¹

A 2008 meta-analysis of 33 articles on donepezil, galantamine, and rivastigmine included 14 placebo-controlled studies that evaluated the effect of the drugs on the baseline score of the Alzheimer’s Disease Assessment Scale Cognitive section (ADAS-cog). The ADAS-cog is an 11-question, 70-point scale, with higher scores indicating worsening disease. The pooled weighted mean difference in ADAS-cog score for treatment compared with placebo was −2.67 (95% CI, −3.28 to −2.06) for donepezil, −2.76 (95% CI, −3.17 to −2.34) for galantamine, and −3.01 (95% CI, −3.80 to −2.21) for rivastigmine. Pooled data from 14 studies measuring function (active treatment vs placebo) showed standardized mean differences of 0.31 (95% CI, 0.21 to 0.40) for donepezil, 0.26 (95% CI, 0.11 to 0.40) for rivastigmine, and 0.27 (95% CI, 0.18 to 0.36) galantamine, all favoring active treatment.²

A 2006 Cochrane review of 15 RCTs of donepezil versus placebo concluded that donepezil is neither more nor less expensive than placebo when assessing total healthcare resource costs, based on the results of 2 RCTs that included cost analysis. Data were not pooled because results were not considered comparable across trials. One study of 289 patients analyzed the cost of treatment with donepezil to society and individuals in Canada, France, Australia, Finland, Norway, Sweden, Denmark, and the Netherlands, compared with placebo. Mean total healthcare cost of AD including cost of donepezil to the patient in Canadian dollars in 1998 over 24 weeks for 143 patients taking donepezil was $4,355 (SD $2,940), compared with $4,321 (SD $2,917) for 146 patients taking placebo, a mean difference of −$34 (95% CI, −641.33 to 709.33; P=.92) over the same time period.³

Is fenugreek safe and effective as a lactation enhancer?

Evidenced-Based Answer
Fenugreek should not be recommended. While the fenugreek plant may have some efficacy in lactation enhancement (SOR C, based on 1 small cohort study), it may have adverse effects in both the mother and infant (SOR C, based on expert opinion and case reports).

Galactagogues are commonly used for adoptive nursing, reestablishing the milk supply after weaning, and increasing a faltering milk supply. Of the herbal lactation enhancers available, fenugreek is perhaps the most widely used, with the typical dose ranging from 600 to 2400 mg (1–4 capsules) 3 to 4 times a day.¹

To date, only 1 clinical trial has evaluated the effectiveness of fenugreek for enhancing breast milk production. The trial was a small observational study in which participants served as their own control. Ten women completed the study. All participants were exclusively breast-pumping. During week 1, baseline milk production was documented by the patients and recorded in a diary. During week 2, participants took 3 capsules of fenugreek, 3 times daily, while continuing to log their milk production. The authors reported an increase in average daily pump volume from 207 mL per day during week 1, to 464 mL per day during week 2 (P=.004).²

Limitations to this study included small sample size, lack of randomization, and data based on patient report. Additionally, study data were available in abstract form only, so important details such as infant gestational age at birth and reasons why the women were exclusively breast-pumping are unknown.²

No clinical studies have evaluated the safety of fenugreek in mother or infant when used for lacta-
tion enhancement. According to 1 standard herbal reference textbook, possible adverse effects with oral dosing include diarrhea, dyspepsia, abdominal distention, flatulence, and hypoglycemia. Allergic reactions include nasal congestion, hoarseness, persistent coughing, wheezing, facial angioedema, and shock. One case report described loss of consciousness and an unusual body odor similar to that noted in maple syrup disease in a 5-week-old infant who was given herbal tea containing fenugreek. This body odor was also been observed in another case report of a neonate whose mother consumed fenugreek just before delivery.

Is iontophoresis therapy effective for tennis elbow (lateral epicondylitis)?

Evidence-Based Answer
Corticosteroid iontophoresis therapy does not provide long-term improvement of symptoms over placebo. Nonsteroidal anti-inflammatory drug iontophoresis has not been compared against placebo. (SOR B, based on randomized controlled trials [RCTs].)

A randomized, double-blinded, placebo-controlled study of 199 patients with lateral epicondylitis compared iontophoresis with the corticosteroid salt dexamethasone sodium phosphate against placebo treatment. Dexamethasone was found more effective than placebo at alleviating pain symptoms for just 2 days after therapy. On a 100-mm visual analog scale (VAS), with 0 mm being no pain and 100 mm being the most pain imaginable, patients receiving dexamethasone initially had a larger mean VAS improvement than patients receiving placebo (23.0 and 14.0 mm, respectively; \( P = .012 \)). However, after 1 month, patients who received dexamethasone had a mean VAS improvement comparable to patients treated with placebo (24.5 and 19.5 mm, respectively; \( P = .25 \)).

In another trial, iontophoresis with a naproxen salt was evaluated. Naproxen iontophoresis in 29 patients was compared with naproxen phonophoresis (a process using ultrasound instead of like charges to compel a solution transdermally) in 32 patients. All patients were instructed to use standard physiotherapy in addition to their naproxen treatments. This physiotherapy included stretching and strengthening exercises, as well as cryotherapy (icing) treatments. Both groups had improvement in pain symptoms over baseline at 4.5 months from treatment initiation. VAS pain scores were reduced by 25.0 mm (95% confidence interval [CI], 17–33 mm) for the iontophoresis group and by 24.3 mm (95% CI, 16–33 mm) in the phonophoresis group (difference not significant). There was no placebo or physiotherapy-only group.

The naproxen study data may be placed in context by noting that an RCT performed just the year before found similar physiotherapy measures to be the best long-term treatment of tennis elbow pain. This study compared physiotherapy alone with both a corticosteroid injection group and a “wait-and-see” control group. At 52 weeks after treatment onset, 91% of the physiotherapy group noted successful treatment of symptoms, compared with an 83% success rate in the “wait-and-see” group and 69% in the corticosteroid group. When comparing the 2 active treatment groups (physiotherapy and corticosteroid injection), the relative risk reduction favoring physiotherapy was 0.3 (95% CI, 0.1–0.5) and the number needed to treat was just 4.

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4. White D. Which is better for treating tennis elbow: corticosteroid injections or physical therapy? Evidence-Based Practice 2007; 10(5):5–6. [LOE 1a]
What is the best treatment for an adult with an umbilical hernia?

Evidence-Based Answer

The laparoscopic onlay patch repair is associated with less recurrence, shorter hospital stay, lower wound morbidity, and lower postoperative pain when compared with open suture repair. (SOR B, based on a single cohort study.) Open surgical mesh repair of umbilical hernia is associated with less recurrence than open suture repair, but there is controversy surrounding the infection rates associated with this repair. (SOR B, based on heterogeneous cohort studies.)

A retrospective analysis of 102 patients who underwent elective repair of umbilical hernia compared laparoscopic onlay Gore-Tex patch hernioplasty (n=26) with 3 different open repair techniques (suture herniorrhaphy [n=24], Mayo repair [n=43], and open mesh hernioplasty [n=9]).¹

The study compared postoperative morbidity (including wound morbidity rate and length of hospital stay), postoperative pain (measured with a visual analogue scale [VAS 0–10, higher numbers=greater pain] at rest and when coughing), and operative details (including duration of operation) among the 4 groups, with a mean follow-up of 2 years (TABLE). Wound morbidity was defined as a combination of wound infections, dehiscence, and hematomas.

Laparoscopic technique was associated with longer operative time (median 66 min) than 2 of the open repair techniques (Mayo repair=60 min, suture herniorrhaphy=50 min; P<.05 for each comparison). Laparoscopic technique was also associated with less self-reported pain at rest 1 day postoperation versus the Mayo technique (average pain score 1 and 4, respectively, on the VAS; P<.05). Patients undergoing the laparoscopic technique stayed in the hospital fewer days than patients undergoing the Mayo technique (1.5 vs 3.5 days; P=.01). Laparoscopic repair was associated with lower wound morbidity when compared with the Mayo and suture techniques (0% vs 23.3% vs 20.8%, P<.05 for both comparisons). Suture herniorrhaphy had an 8.7% recurrence rate at 2 years compared with the other 3 techniques, which were all 0%.¹

In a later retrospective analysis study, 100 patients underwent open umbilical and para-umbilical repair with mesh (n=39) or suture (n=61) and had a median follow-up of 4.5 years (range 1–8 years). Suture repair techniques included interrupted suture (n=50) or Mayo overlap (n=11); mesh repair techniques included flat mesh (n=6) and mesh plug (n=33). The recurrence rate at 4.5 years for the mesh repair groups was lower than for the suture repair groups (0% vs 11.5%; P=.007). The infection rate was also significantly lower in the mesh groups than in the suture groups (0% vs 11.5%; P=.007).²

A 2008 retrospective analysis examined 152 open, elective umbilical hernia repairs using either mesh repair (n=65) or suture repair (n=87), comparing surgical site infection (SSI) recurrence rates at a major VA hospital. Recurrence rates in the mesh repair group were lower than in the suture repair group, but the difference was not statistically significant (1.5% vs 9.2%; P=.16). In this study, patients with open mesh repairs were more likely to contract a SSI than patients with open suture repairs (29% vs 13%, respectively; P=.01).³

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**TABLE**

Comparison of hernia repair techniques¹

<table>
<thead>
<tr>
<th></th>
<th>Operative time, median (range), min</th>
<th>Resting pain score day 1</th>
<th>Coughing pain score day 1</th>
<th>Days in hospital</th>
<th>Wound morbidity</th>
<th>Recurrence rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Laparoscopic onlay Gore-Tex patch hernioplasty</td>
<td>66 (55–90)</td>
<td>1</td>
<td>7</td>
<td>1.5</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>Suture herniorrhaphy</td>
<td>50 (40–68)</td>
<td>NR</td>
<td>NR</td>
<td>3.0</td>
<td>20.8%</td>
<td>8.7%</td>
</tr>
<tr>
<td>Mayo repair</td>
<td>60 (40–60)</td>
<td>4</td>
<td>6</td>
<td>3.5</td>
<td>23.3%</td>
<td>0%</td>
</tr>
<tr>
<td>Open mesh hernioplasty</td>
<td>60 (60–90)</td>
<td>NR</td>
<td>NR</td>
<td>4.0</td>
<td>11.1%</td>
<td>0%</td>
</tr>
</tbody>
</table>

¹ Measured with a visual analogue scale (0–10, higher numbers=greater pain). NR=not reported.
What is the best treatment for migraines during pregnancy?

**Bottom line**

Little evidence supports the use of 1 treatment over another for migraines in pregnant women. There are no Cochrane reviews or American College of Obstetricians and Gynecologists Practice Bulletins on the topic. Experts recommend a stepwise approach to use of abortive and prophylactic medications in treating migraines, starting with Food and Drug Administration (FDA) category B medications and progressing to FDA category C medications, after a discussion of risks and benefits with the patient.1,2

**Review of the evidence**

Migraine headache occurs in 11% to 26% of women of childbearing age.3 Active migraine during pregnancy may be viewed as a potential marker of vascular disease. A population-based, case-control study of more than 18 million inpatient records showed that active peripartum migraine was associated with an increased risk of pregnancy-related stroke (relative risk [RR]=15.05; 95% confidence interval [CI], 8.26–27.4) and acute myocardial infarction (RR=2.11; 95% CI, 1.76–2.54).3 Most pregnancy-related strokes occur during the third trimester and peripartum period.3

Migraine headache is also associated with pre-eclampsia. A prospective cohort study of more than 700 women found an association between a positive personal history of migraine and the development of hypertensive disorders during pregnancy (gestational hypertension or pre-eclampsia) in previously normotensive women (odds ratio [OR] 2.85; 95% CI, 1.4–5.81).4 A review of 10 epidemiologic studies found associations between migraine headaches and pre-eclampsia in 8 of the 10 studies included, although no causality was assessed.5 Migraine headaches are not associated with preterm labor, low birth weight, small for gestational age, or fetal loss.4

During the first trimester, when estrogen concentrations are just starting to rise, some women may see an increase in migraines. However, up to two-thirds of women experience a decrease in both the frequency and severity of migraines during the second and third trimesters.1 Women who have migraine without aura are more likely to experience migraine improvement with pregnancy than are those who have aura, and women whose headaches have previously been linked with their menstrual cycle are also more likely to have migraine improvement.1 If headaches are not better by the end of the first trimester, additional improvement is unlikely to occur. If migraine headache does improve during pregnancy, nearly 94% of the women will have migraines return postpartum, when estrogen concentrations drop drastically.6

**Migraine treatment during pregnancy**

**Nonpharmacologic therapy**

Nonpharmacologic therapy, such as trigger identification, biofeedback, massage, yoga, and deep breathing are first-line therapies for migraine during pregnancy. While not well studied, they likely pose little or no risk to the mother or the fetus.

**Medication**

Choosing a medication to treat migraine headaches during pregnancy can be difficult, because of the risks of fetal exposure to medications. Most abortive treatments for migraines are FDA category C in pregnancy. If drug therapy is necessary, acetaminophen is often recommended first.7 A few other notable category B medications are nonsteroidal anti-inflammatory drugs (during the first and second trimesters), selected narcotics, caffeine, prednisone, and metoclopramide (TABLE 1). Ergot alkaloids should not be used in pregnancy because they decrease uterine blood flow.

Sumatriptan is pregnancy category C. However, a recent narrative review concluded that sumatriptan’s pregnancy registry and clinical trials indicate that it does not have a clinically significant effect on organogenesis or spontaneous abortion rates, and thus may be considered a safe therapeutic alternative for pregnant women.6 Data regarding the other drugs in the “triptan” group are insufficient to draw the same conclusions. Furthermore, as most data available for sumatriptan involved exposure during the first trimester only or the timing of the drug exposure was not reported, extra caution is warranted for use during the second and third trimesters.6

Many women take prophylactic medications to reduce the frequency of migraine headaches. TABLE 2 includes many of the most common prophylactic medications. Practice recommendations...
Treatments to stop migraine during pregnancy with FDA pregnancy classification

<table>
<thead>
<tr>
<th>Medication</th>
<th>Classification</th>
<th>Medication</th>
<th>Classification</th>
</tr>
</thead>
<tbody>
<tr>
<td>Analgesics</td>
<td></td>
<td>5-HT1 agonists</td>
<td></td>
</tr>
<tr>
<td>Acetaminophen</td>
<td>B</td>
<td>Sumatriptan</td>
<td>C</td>
</tr>
<tr>
<td>NSAIDs</td>
<td>B, 1st-2nd tri D, 3rd tri</td>
<td>Rizatriptan</td>
<td>C</td>
</tr>
<tr>
<td>ASA</td>
<td>C</td>
<td>Zolmitriptan</td>
<td>C</td>
</tr>
<tr>
<td>Hydromorphone</td>
<td>B</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tramadol</td>
<td>C</td>
<td>Prochlorperazine</td>
<td>C</td>
</tr>
<tr>
<td>Meperidine</td>
<td>B</td>
<td>Promethazine</td>
<td>C</td>
</tr>
<tr>
<td>Morphine</td>
<td>B</td>
<td>Chlorpromazine</td>
<td>C</td>
</tr>
<tr>
<td>Codeine</td>
<td>C</td>
<td>Metoclopramide</td>
<td>B</td>
</tr>
<tr>
<td>Propoxyphene</td>
<td>C</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ketorolac</td>
<td>C</td>
<td>Dexamethasone</td>
<td>C</td>
</tr>
<tr>
<td>Oxycodone</td>
<td>B</td>
<td>Prednisone</td>
<td>B</td>
</tr>
<tr>
<td>Hydrocodone</td>
<td>C</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ergotamines</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ergotamine</td>
<td>X</td>
<td>Isometheptene (Midrin)</td>
<td>C</td>
</tr>
<tr>
<td>Ergotamine</td>
<td>X</td>
<td>Fiorinal</td>
<td>C</td>
</tr>
<tr>
<td>Caffeine</td>
<td>B</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dihydroergotamine</td>
<td>X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fiovral</td>
<td>X</td>
<td></td>
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</tr>
</tbody>
</table>
| Aspirin | NSAIDs=nonsteroidal anti-inflammatory drugs.

American Academy of Neurology recommendations

The American Academy of Neurology recommends paying special attention to women with migraines who are pregnant or who want to become pregnant, because of the potential for teratogenicity associated with available migraine abortive and prophylactic medications. They recommend choosing a treatment with the lowest potential for toxicity.

REFERENCES

What is the best management for localized psoriatic lesions?

Bottom line
Tazarotene and topical vitamin D products are safe and effective for long-term daily use in patients with localized psoriasis, although tazarotene can cause local irritation. Both agents should be avoided in women who may become pregnant. (SOR A, based on a Clinical Evidence designation of “beneficial.”) High-potency topical corticosteroids are also effective, but have adverse effects that preclude long-term daily use. (SOR C, based on a practice guideline.)

Evidence summary
Three randomized trials (n=1,672) summarized in a BMJ Clinical Evidence review compared tazarotene with placebo among patients with mild to moderate psoriasis.¹ The trials found a significant improvement in plaque elevation, scaling, and erythema with tazarotene at 8 to 12 weeks’ follow-up (absolute results not provided).

The BMJ Clinical Evidence review also summarized another 3 randomized trials (n=1,198) comparing tazarotene plus moderate or high potency topical corticosteroids with tazarotene alone for mild to moderate psoriasis.¹ Results showed a significant increase in rate of response at 2 to 12 weeks of treatment for combination therapy versus tazarotene alone.

A single randomized trial (n=120) involving participants with mild to moderate psoriasis demonstrated that once-daily treatment with a combination of tazarotene plus 0.1% topical mometasone was significantly better at 2 weeks of treatment in improving symptoms compared with tazarotene 0.005%, although no significant difference was noted in the number of patients who achieved complete clearance at 8 weeks.¹ Most participants using tazarotene reported skin irritation; participants using it in combination with corticosteroids had fewer adverse effects and reduced rates of withdrawal from trials.

Topical vitamin D, calcipotriene (synonym calcipotriol), was compared with placebo in 1 systematic review and a subsequent randomized trial.¹ The systematic review revealed significantly greater improvement in severity of psoriasis at 3 to 8 weeks with calcipotriol versus placebo (10 trials, standard mean difference [SMD], −0.74; 95% CI, −0.55 to −0.93).² No significant difference was noted in local adverse events between the groups.

A randomized trial (n=1,136) compared calcipotriene alone versus calcipotriene in combination with a topical corticosteroid (betamethasone dipropionate) versus a vehicle cream alone in patients with moderate to severe psoriasis.³ All subjects were initially treated with the combined vitamin D/corticosteroid for the first 4 weeks before being assigned to their respective treatment groups. At 8 weeks, the calcipotriene group had a significantly greater improvement in the Psoriasis Area and Severity Index score than with the vehicle cream alone (mean change −44.5% with calcipotriene vs −33.1% with vehicle; mean difference −11.7% (95% CI, −5.5% to −17.9%). No significant difference was seen in overall adverse events at 8 weeks with calcipotriene versus placebo (40.4% vs 36.7%, respectively; odds ratio [OR] 1.17; 95% CI, 0.87–1.56). Skin-specific problems were also similar between groups (15% calcipotriene versus 11% placebo; no P value given).

One trial (n=97) evaluated time to relapse with calcipotriol therapy versus placebo.⁴ Participants had finished at least 6 months of treatment with methotrexate. Participants using calcipotriol had a significantly longer time (median time of 113 days vs 35 days with placebo; P<.001) to relapse (doubling of baseline-modified psoriasis severity score). No difference was seen in adverse events with calcipotriol versus placebo (78.3% vs 76.9%, respectively; P=.87).

A systematic review comparing vitamin D derivatives to topical corticosteroids (9 randomized trials, severity of psoriasis not reported) found no significant difference in severity of symptoms at 3 to 8 weeks in participants using vitamin D versus potent topical steroids (SMD, 0.06; 95% CI, −0.12 to 0.24).² No significant difference was noted in local adverse events; average risk increase was 10% (95% CI, −2% to 21%).

According to the Psoriasis Evidence-Based Guidelines from the Finnish Medical Society, initial treatment with keratolytics (eg, salicylic acid 5% preparations) may be beneficial for removal of thick scales.⁵ Emollients may be used as adjuvant treatment or alone for mild psoriasis. Topical vitamin D, applied twice daily, is safe.
and effective for long-term treatment. Potent (group III or IV) topical corticosteroids have a rapid onset of activity, but should be tapered after 2 to 4 weeks of continuous use to avoid adverse effects. Tazarotene and vitamin D derivatives are potentially teratogenic and should be avoided during pregnancy.¹

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REFERENCES


For 2009, all AAFP members who subscribe to EBP CME are eligible to earn 2 Prescribed Academy credits monthly toward their AAFP membership. Please complete and return the CME activity by March 31, 2010.
1. Low serum vitamin D and obesity are associated:
   a. Only in ethnic Scandinavians
   b. In adults, but not in children
   c. Because of a synthesis defect in fat skin
   d. In both younger and older adults

2. Which of the following agents is not suitable for long-term treatment of mild-to-moderate psoriasis?
   a. Topical potent corticosteroids
   b. Calcipotriol ointment
   c. Tazarotene ointment
   d. All of the above are suitable for long-term treatment

3. A 28-year-old G3P2002 at 32 weeks comes to your office for treatment of migraine headaches. The pregnancy is uncomplicated. The patient is otherwise healthy and takes no medication. She has a history of migraine headaches. Headaches improved during pregnancy, but she still has about 1 headache every 2 weeks that is severe. She is unable to eat, go to work, or take care of her children due to the pain. She has tried biofeedback, yoga, and Tylenol with no relief from the pain. Sumatriptan alleviated her migraine headache prior to pregnancy, and she would like to try that again now. What is the safest medication to use at this time?
   a. Ketorolac
   b. Sumatriptan
   c. Ibuprofen
   d. Oxycodone

4. Which of the following is NOT an appropriate treatment for pain after coronary artery bypass grafting?
   a. Capsaicin cream
   b. Gabapentin
   c. Amitriptyline
   d. Amantadine

5. Iontophoresis treatments for pain symptoms associated with tennis elbow (lateral epicondylitis) using corticosteroids
   a. Are proven better than physiotherapy
   b. Are more effective than steroid injections
   c. Are more effective than nonsteroidal anti-inflammatory drug iontophoresis
   d. Are comparable to placebo at 1 month

6. Which of the following statements is true of butterbur for migraine therapy?
   a. It reduced migraine frequency in several studies
   b. It has a responder rate (reducing migraine frequency by ≤50%) equal to placebo at all doses
   c. Solid evidence shows that it reduces migraine intensity
   d. Stalks, leaves, and roots all are used in the extracts commonly available

7. Various forms of stress management have been shown to have all of the following cardiovascular outcomes except:
   a. Decreased Framingham risk scores
   b. Fewer nonfatal myocardial infarctions
   c. Lower all-cause mortality
   d. Lower resting heart rate

8. The key value of giving polyethylene glycol to children older than 2 years who have refractory constipation that it
   a. Increases stool frequency better than lactulose
   b. Results in softer stools than Milk of Magnesia
   c. Is better accepted by patients and families than other agents
   d. Has better data regarding long-term safety than lactulose and Milk of Magnesia

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Renew or Subscribe to EBP at fpin.org or call 573-256-2066
Unfortunately, studies show many overweight children will become overweight or obese adults. In fact, 1 out of every 3 children is now considered overweight or obese in the United States.

**What causes children to be overweight?**

Two factors contribute to being overweight: genetics and lifestyle. Body shape and how the body burns and stores fat are inherited traits that can be passed from parents to children just like other traits, such as eye and hair color. But the most important factor is lifestyle; parents have a big influence on the eating habits and daily activity of their children.

For young children, parents have almost complete control over all food they eat. Children imitate their parents in what kinds of foods they like, portion sizes, and how often they eat. Children are frequently rewarded for good behavior with high-fat, sugary foods. Foods disliked by parents may not be given to their children. Often our childhood food habits carry on through life.

Many children today have little daily exercise. On average, they sit in front of a computer or TV screen up to 4 hours a day. Physical activity classes at school may be less than 30 minutes per week. And hardworking parents have little time for exercise, setting a lifestyle example for kids.

Other factors include parents not recognizing that their children are overweight, and pediatricians and family doctors being reluctant to talk about a child’s weight during office visits for fear of offending the parents or child.

Overweight children grow up with greater chances of high blood pressure, high cholesterol, diabetes, joint problems, low self-esteem, and depression.

If you are not sure if your child is overweight for his or her age, talk with your family doctor. Your doctor can help you make a plan for all family members to have a healthy diet and more exercise. You will be helping everyone’s health.

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**For more information**

Overweight in Children (American Heart Association)
http://www.americanheart.org/presenter.jhtml?identifier=4670

Overweight in Children and Adolescents (Office of the Surgeon General)
http://www.surgeongeneral.gov/topics/obesity/calltoaction/fact_adolescents.htm