was no longer seen in the CBT group (13 min shorter; 95% CI, –3 to 29), while total sleep duration continued to improve (32 min longer; 95% CI, 8 to 71). CBT is not known to have any significant adverse effects.

Pharmacologic treatments currently approved by the US Food and Drug Administration include benzodiazepines hypnotics, nonbenzodiazepine hypnotics, and a melatonin receptor agonist. A meta-analysis of 24 RCTs (n=2,417) comparing short-term hypnotic use with placebo in persons aged 60 and older found that treatment with sedative hypnotics subjectively improved quality of sleep (effect size 0.14, P<.05). (An effect size of 0.2 is considered small, 0.6 moderate, and 1.2 large.) Improvements were seen in total sleep time (mean 25 min longer; P<.001) and number of night time awakenings (–0.63; P<.001). Cognitive adverse effects, however, were 4.8 times more common (95% CI, 1.5–15), and daytime fatigue was 3.8 times more common (95% CI, 1.9–7.8) with hypnotic use versus placebo.

A double-blind study evaluating the use of the melatonin receptor agonist ramelteon versus placebo in 693 elderly persons found ramelteon significantly improved sleep latency (~8.3 min at week 1; P=.008) with a low incidence of adverse effects and no rebound or withdrawal effects over 5 weeks of treatment.

Other classes of medications utilized off-label for elderly patients include antihistamines, antidepressants, antipsychotics, and anticonvulsants. Due to insufficient evidence for effectiveness and the potential for serious adverse effects, the use of antihistamines, antipsychotics, and anticonvulsants are not recommended for elderly patients. Antidepressants are recommended for use in persons with comorbid depression.

Amanda L. Hawks, PharmD
Robert L. Gauer, MD
Womack FMR Clinic
Fort Bragg, NC

The opinions and assertions contained herein are the private views of the authors and are not to be construed as official or as reflecting the views of the Medical Department of the US Army or the US Army Service at large.

drugs and necrotizing fasciitis, causality has not been proven, and other factors, such as increased pain with necrotizing fasciitis more often requiring pain medication, could also explain such a relationship.

Steven R. Smith, MS, RPh
Coral D. Matus, MD
Toledo Hospital FMR
Toledo, OH


**What is the best way to diagnose compartment syndrome?**

**Evidence-Based Answer**

Clinical examination is not a reliable way to diagnose acute compartment syndrome (ACS), although the absence of pain, paresis, and paresthesia certainly rules it out. (SOR: A, based on a meta-analysis.) As a practical matter, patients with injuries serious enough to develop ACS often cannot be examined adequately anyway. (SOR: B, based on a cohort study.) Measurement of differential pressure (denoted as ∆p, the diastolic blood pressure minus the absolute compartment pressure) with ∆p <30 mmHg is the gold standard for diagnosis of ACS. (SOR: C, based on expert opinion.) The absolute compartment pressure (ACP) is not a reliable substitute for ∆p. (SOR: B, based on a diagnostic cohort study.)

A meta-analysis of 4 cohort studies (n=132, mean age 36 years, 80% male) evaluated whether the clinical findings of pain at rest or with passive stretch, paresthesia, and paresis were useful for the diagnosis of impending ACS. For these clinical findings, the sensitivity was low (13%–19%) and the specificity was high (97%–98%), resulting in a positive predictive value of 11% to 15% and a negative predictive value of 98%. These authors concluded that the absence of these signs and symptoms were more useful for excluding the diagnosis than in establishing the diagnosis by their presence.¹

A 6-month prospective observational cohort study evaluated a screening protocol to detect lower extremity ACS in patients (n=45, mean age 38 years, 76% male) admitted to a shock-trauma intensive care unit. Patients were screened on admission and every 4 hours for 48 hours. Physical examination included measurement of calf circumference (at 4 cm below the tibial tuberosity); calf pain at rest and with passive stretch; assessment of dorsal pedal and posterior tibial pulses; and neurological assessment of motor and sensory function. When the physical examination was suspicious or unreliable (altered mental status, sedation, paralysis, etc.), measurements of anterior and posterior compartment pressures were performed. The ∆p <30 mmHg was considered diagnostic for ACS and prompted fasciotomy.²

ACS was identified in 9 of 45 patients (20%), with an average time to recognition of 9±5 hours and ∆p=20.6±3.7 mmHg at the time of diagnosis. In this cohort, leg circumference was unobtainable in 51% of patients because of dressings/splints and calf pain, and neurological assessment was unobtainable in 69% of patients because of sedation or neurologic status. These authors concluded that physical examination was unreliable for diagnosing ACS.²

A prospective study compared the effectiveness of using the ACP >30 mmHg versus the standard of ∆p <30 mmHg for the diagnosis of ACS in a cohort of 39 patients (mean age 34 years, 66% male) having 42 tibial diaphyseal fractures. Blood pressure and ACP were taken every half hour beginning an average of 2 hours after injury and continued for 72 hours. The mean ACP and the ∆p for the six 12-hour periods were calculated. A value of ∆p <30 mmHg over 2 consecutive half-hour periods was considered diagnostic of ACS and prompted fasciotomy.³

Over a 72-hour period, mean ACP readings ≥30 mmHg were documented in 33 patients, while only 3 had a ∆p <30 mmHg (and were treated by fasciotomy). All patients except 1 were followed for a mean of 36 months; all fractures healed and there were no sequelae of ACS. These authors concluded that ACP readings >30 mmHg were not reliable for the diagnosis of ACS.³

Darrell R. Over, MD, MSc
Kristin Martin, DO, MSc
U of AR AHEC Pine Bluff FMRP
Pine Bluff, AR