About this Section
Each year, Minnesota Medicine highlights research and clinical work undertaken by Minnesota medical students, residents and fellows. The goal is to not only showcase the good work these medical trainees are doing but also to inform readers about pertinent topics.

This year, 22 trainees submitted brief papers describing original research or interesting cases. They were evaluated with regard to these and other questions: Did the authors provide an adequate description of the case or the problem? Was their methodology sound? Did they conduct an adequate review of relevant scientific literature? Do the findings or does the case have implications for practice or further research? The reviewers selected the following submissions for publication in this issue.

We thank both those who submitted their work and our reviewers: Peter Kernahan, MD, PhD; Barb Elliott, PhD; Barbara Yawn, MD; and Angie Buffington, PhD.

Athlete’s Dystonia
An Occupational Hazard of Athletes

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Dystonia is a movement disorder characterized by involuntary sustained or intermittent muscle contractions that cause abnormal postures (eg, twisting) or repetitive movements (eg, tremor). Dystonia may affect multiple regions of the body or be limited to one region (focal). Focal dystonia may be triggered by repetition of specific tasks. In adults, it usually affects the upper limbs or cranio-cervical segment.

A new form of task-specific focal dystonia called runner’s dystonia was recently described. Twenty similar lower-limb cases have been reported in the literature to date. We wished to further characterize this rare form of adult-onset focal dystonia, determine the usefulness of electrophysiology in diagnosing it, determine whether athletes who are not runners could suffer a similar disorder and describe long-term outcomes.

Methods
We retrospectively reviewed clinical and neurophysiologic information from adult patients seen at Mayo Clinic with task-specific focal dystonia arising after a prolonged history of repetitive lower-limb exercise. Follow-up data were gathered by telephone or mailed questionnaire.

Results
Nineteen patients (53% men) were identified; 13 were runners and six were athletes but not runners. The median age at onset was 49.2 years (range 25 to 69 years). Correct diagnosis was delayed by a median of 2.5 years, by which time nearly 40% of the patients had undergone or been recommended for unnecessary invasive procedures for misdiagnosed conditions including pyriformis syndrome, compartment syndrome, muscular dystrophy and claudication. Most patients (68%) had dystonia onset in the distal lower limb. Truncal dystonia was a novel observation in four of the patients. Strict task specificity was seen at onset in all patients. Dystonia progressed to affect walking in most patients (84%). To relieve symptoms, six patients reported using sensory tricks (voluntary actions that temporarily alleviate the dystonic posture such as lightly pressing against the abdomen to correct truncal dystonia).

In general, MRI of the brain and spine were unremarkable, as were nerve conduction studies and needle EMG. Surface EMG and gait analysis confirmed task-specific focal dystonia in 10 of the patients; these studies allowed distinction from stiff-limb syndrome in one and orthostatic myoclonus in another. Diagnosis in the other nine cases was made on clinical grounds.

At median follow up of 4.8 years (range 0.4 to 23 years) from dystonia onset and 2.1 years (range 0 to 18 years) from diagnosis, all patients were still symptomatic. Effective treatment was rare, with most patients achieving only partial return to their predystonic activity level when participating in the same (56%) or a different (25%) exercise. Beneficial treatments included botulinum toxin injections (in 3 of 5 cases), physical therapy (6/15), clonazepam (2/5), carbidopa/levodopa (3/8) and trihexyphenidyl (1/3).

Conclusion
We describe the largest series of athlete’s dystonia, a task-specific lower limb and truncal dystonia seen in runners and other athletes. Truncal dystonia, with or without lower limb involvement, was a novel observation in our patients. Age and location of onset, near universal progression to affect walking, and generally poor response to treatment were similar among our patients and patients described in previous reports. We found electrophysiology to be helpful in confirming the diagnosis.
Capsule Endoscopy and Left Ventricular Assist Devices

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Capsule endoscopy (CE) is a well-established modality for diagnosing obscure gastrointestinal bleeding. Obscure gastrointestinal bleeding in patients with left ventricular assist devices (LVAD) is not unusual. The risk of such bleeding after LVAD implantation is 18% to 40%.¹ The safety and efficacy of CE in patients with LVAD are largely unknown. Researchers from Mayo Clinic described its safety in 14 patients with LVAD but did not include CE findings or clinical outcomes.² The purpose of this study was to investigate the safety and efficacy of CE in patients with LVAD.

Methods
A retrospective chart review was performed for all patients with LVAD undergoing CE at the University of Minnesota Medical Center in Minneapolis between January 2007 and August 2014. Thirty-four CE studies performed in 24 patients were identified and reviewed for demographic, laboratory and CE study data in addition to subsequent medical and endoscopic management.

Results
A total of 34 CE studies were performed in 24 patients. Mean age at time of the first CE was 67 years; 20 of the patients (83%) were male. The indications for CE were obscure occult gastrointestinal bleeding in three cases, obscure overt gastrointestinal bleeding in 25 and anemia in six. Capsule endoscopy findings included active bleeding in 12 cases (35%). A potential source was visualized in six of these. When active bleeding was not seen on CE, a high-potential source (AVM, ulceration, tumor) was found in three and an intermediate-potential source (red spots, erosions) in three. Active bleeding and potential sources were found in the stomach (n=3) and small bowel (n=15). The capsule failed to leave the stomach in two cases. Mean small-bowel transit time was 3 hours 44 minutes. No cardiac device malfunction occurred and no capsules were retained. Small-bowel image capture was incomplete in three CE studies.

Medical intervention was the most common management strategy after CE. Medical management was changed after 27 of the 34 CE studies (79%). However, capsule endoscopy findings were not associated with a change in medical management (p=0.69). Nine patients (26%) underwent endoscopic evaluation after CE. Six patients underwent enteroscopy and three had EGD. Sources of the bleeding were an AVM (four patients), Dieulafoy lesion (one patient) and an indeterminate lesion (one patient). Of those patients, five underwent endoscopic intervention.

Six-month follow up was available in all but one patient. During follow up, 10 patients re-bleed. Patients with CE finding of active bleeding or high-potential lesion incurred a higher risk of re-bleeding, transfusion and repeated endoscopy. However, this finding was not statistically significant. One patient died during follow up, but the death was not related to gastrointestinal bleeding.

Conclusion
Ours is the largest study of CE in patients with LVAD. We found capsule endoscopy is a safe and effective test for detection of a bleeding source in patients with LVAD. Medical management of patients was changed after CE in the majority of cases, but their CE findings were not associated with this change. Active bleeding found during CE can be successfully treated endoscopically.

REFERENCES