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## Statin-Related Myopathy

As Hansen et al[1] noted in their report of outcomes in patients with statin-associated myopathy, their study is limited by retrospective medical chart assessment of patients with symptoms of muscle pain or weakness or the laboratory finding of an elevated creatine kinase level. The frequency of muscle weakness was not based on a routine physical examination. Only 1 in 3 subjects had symptoms of weakness, but the authors could not determine from the medical charts whether subjects were asked about a decline in the ability to walk and climb stairs, to lift items previously raised easily, to arise from a toilet seat or get into and out of a car, and to participate in recreational activities. We have found that many patients who were eventually diagnosed as having a statin-induced myopathy did not report functional declines unless asked about changes in specific daily activities.[2]

The authors state that objective muscle tests for a statin-related myopathy include electromyography and muscle biopsy. Little information, however, is available about the sensitivity of these tests to reveal pathologic findings across the spectrum of symptoms and signs that have been encountered. More important for clinicians in practice is another objective test—the physician's ability to overcome the strength of a patient's neck flexor muscles, deltoids, hip flexors tested with the subject supine, and hip extensors and knee flexors tested with the subject prone, graded with the standard British Medical Council scale.[2] Objective functional tests also include the observation of walking (the gait pattern may reveal a shortened stride length, unsteadiness on turns, and a waddle from pelvic muscle paresis) and of the ability to go from sitting to standing without pushing off from a chair with the arms.

No prospective studies of statins have included routine testing of muscle strength or muscle enzymes.[3] The *sine qua non* of a myopathy is diminished proximal strength. Thus, the incidence of myopathy that can cause proximal weakness and disability may be underappreciated. Until randomized clinical trials of lipid-lowering agents include routine manual muscle testing or dynamometry to seek the weakness that defines a myopathy, the numerator for the incidence of this complication cannot be approximated. In addition, researchers may not be able to address the genetic and metabolic spectrum of statin-related muscle dysfunction[4] if they do not define its various phenotypes.

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## Observations From a Statin Myopathy Clinic

We read with great interest the article by Hansen et al,[1] which, to our knowledge, is the first published attempt to assess the recovery from a variety of statin-associated muscle disorders. We would like to add to their report observations from a statin myopathy clinic that currently follows over 200 patients with these disorders.

The article's primary finding, that this heterogeneous group of patients ex perienced full resolution of their pain on cessation of statin therapy, conflicts with our observations.[2,3] Hansen et al[1] grouped patients with a number of statin-induced muscle disorders together. In our subjects, underlying metabolic muscle disorders and the persistence of weakness is greater in patients with statin-associated rhabdomyolysis than in patients with normal creatine kinase myopathy.[4] Patients often experience myalgias and demonstrate abnormalities in cardiopulmonary exercise for years after myositis, while complete resolution of myalgias is typical in patients without elevated creatine kinase levels.[3] Furthermore, while muscle biopsy results in patients with normal creatine kinase myopathy may improve after the myalgias resolve, the tissue often remains myopathic.[5]

While the patients in the study by Hansen et al[1] seemed to resolve their myalgias at subsequent clinic visits, no objective measures of myopathy such as a dynamometry or cardiopulmonary exercise were made. One cannot conclude from this article that statin myotoxic effects are benign simply because those returning to the clinic stopped complaining of myalgia. It is just as likely that patients stopped coming to the clinic because their complaints of myalgia remained untreated. Prospective studies with objective measures of strength and endurance must be performed before any conclusions about the reversibility of statin-induced muscle disorders can be derived.

Finally, the authors' finding that 43% of patients were able to tolerate another statin on subsequent rechallenge needs to be scrutinized. Previous work has shown



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