degenerated fibers does not take place in the heart.

This study of dystrophin in heart biopsies of DMD carriers indicates that cardiomyopathy is associated with dystrophin abnormalities. Moreover, the clinical presentation of our patients suggests that cardiomyopathy may be the only manifestation related to a dystrophin gene mutation. The high mutation rate of the dystrophin gene is responsible for the frequent occurrence not only of DMD patients, but also of DMD carriers due to new mutations, and hence with no family history of neuromuscular disorders. Therefore, dystrophin analysis on muscle and, when negative, also on heart biopsies, should be performed in women affected by apparently isolated dilated cardiomyopathies, even when family history is negative for DMD. The presence of a cardiomyopathy, moreover, is probably underestimated in DMD carriers, and cardiac function should be kept under careful surveillance. Seven of seven manifesting carriers with limb-girdle muscle weakness followed by us had mild-to-severe cardiac symptoms, and two of them, still able to walk, died of intractable heart failure (unpublished data).

Further studies on dystrophin expression in heart of DMD carriers and DMD patients will help to better characterize the cardiopathy that affects the prognosis of Xp21-linked dystrophies so severely.

Addendum. Since this article was submitted, dystrophin abnormalities were demonstrated in the heart biopsy of a nonsymptomatic DMD carrier and endomyocardial biopsy was proposed as a sensitive

diagnostic method in DMD carriers' detection.¹¹

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Correction

In "The prevalence by staged severity of various types of diabetic neuropathy, retinopathy, and nephropathy in a population-based cohort: The Rochester Diabetic Neuropathy Study" by Dyck et al, which was published in the April issue (*Neurology* 1993;43:817-824), the name of F.J. Service, MD (Department of Internal Medicine, Mayo Clinic), should have been included in the byline.

Neurology[®]

Correction

Neurology 1993;43:2345
DOI 10.1212/WNL.43.11.2345

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