Response

To the Editor:

Finsterer and Stöllberger present some interesting comments on our article about cardiac and sternocleidomastoid muscle involvement in Duchenne muscular dystrophy (DMD) studied by MRI.1 We underestimated neither the importance of clinical neurologic and cardiac examination, nor the use of the currently applied techniques in the assessment of DMD patients. Although the technologic progress is of great value, there is no doubt that the patient’s clinical evaluation remains the cornerstone of medicine.

In this article, our purpose was not to underestimate the value of traditionally used approaches; instead, we emphasized the important role of a new technology in the early detection of subclinical cases. The same findings were assessed by other authors,2,3 and recently more MRI articles have emphasized the importance of the technique in the early detection of subclinical lesions in DMD patients.5,6 This is to be expected because MRI has the ability to detect little changes very early in different tissues.

We should also emphasize that DMD patients can nowadays have different clinical presentations, depending on the early use of angiotensin-converting enzyme inhibitors and/or deflazacort, the patient’s supportive treatment, close cardiac and neurologic evaluation, and so forth. Cardiac involvement is not necessarily found in all patients at the time of ambulation loss. Additionally, ECG changes are not necessarily found in all DMD patients.7 Furthermore, the pathophysiology of cardiac lesions in DMD is more complicated than initially believed. Although it is known that the absence or decrease of dystrophin leads to progressive skeletal muscle and heart failure, it has also been documented recently that abnormal dystrophin can act as a potential susceptibility gene for viral infection of the myocardium and as a factor that markedly increases enterovirus-induced cardiomyopathy.8

We did not perform Doppler tissue imaging, which can also offer early information about cardiac involvement, and we did not compare our results with new echo techniques because these techniques are not available everywhere and all echocardiographers are not familiar with them. Our purpose was to emphasize the capability of MRI to perform tissue characterization and its possible use for early detection of subclinical cardiac involvement in DMD. Further comparative studies are needed to establish the role of each technique. However, there is no doubt that MRI proved to be of great importance in the early detection and long-term follow-up of many diseases with cardiac involvement and also was able to detect cardiac lesions missed by echocardiography (myocarditis, cardiomyopathies, thalassemia, and so forth).

Association of Chronic Mountain Sickness With Abnormal Pulmonary Microcirculation

To the Editor:

A recent issue of CHEST (February 2010) published findings by Stuber et al9 of large increases in pulmonary hypertension with mild exercise in chronic mountain sickness (CMS). It is important, however, to know whether the findings are associated with abnormal pulmonary microcirculation (using diffusing capacity of the lung for carbon monoxide [DLCO] as proxy). To better evaluate whether CMS has abnormal gas exchange, the authors

References


should report DLCO as percent predicted and properly adjust it for lung volume. The American Thoracic Society recommends adjusting predicted values (not measured values) for altitude and hemoglobin and makes clear that the DLCO/alveolar volume (VA) ratio does not correct for lung volume.

DLCO and the DLCO/VA ratio change with lung volume as would be expected with changes in surface area for diffusion. Percent predicted for DLCO adjusted for lung volume (Daco) and DLCO/VA ratio adjusted for lung volume (Kaco) also should be reported, using the following equations: Daco predicted = DLCO predicted × (0.58 + 0.42 × VA/VAte) and Kaco predicted = Kco predicted × (0.42 + 0.58/VA/VAte), where Kco = carbon monoxide transfer coefficient and VAte = predicted VA at total lung capacity.

It also would be helpful to know whether CMS affects spirometry (FEV1 and vital capacity) and lung volume (total lung capacity and, if spirometry was measured, FEV1 and vital capacity). Finding comparable percent-predicted Daco in patients with and without CMS would strengthen their argument that CMS does not impair pulmonary microcirculation.

**Correspondence**

To the Editor:

I read with interest the correspondence by Di Berardino et al (February 2010)1 to see how the difficult task of inhalation product comparison has been addressed. I agree that different valved holding chambers (VHCs) that are available for inhalation should not be used without evidence of equivalent efficacy/safety and compatibility between pressurized metered-dose inhalers (pMDIs) and VHCs, but I think that the authors’ proposal is not enough to ensure the correct delivery of aerosolized drugs. Conversely, it seems to be an excuse to use a new VHC without the required evidence.

The clinical response to a specific pMDI cannot be assumed to be equivalent if a different spacer is used or if a different pMDI is used with the same spacer. The development of a pMDI should always include the testing of at least one specific named spacer for use with the particular pMDI, which should be stated in the labeling. If the spacer is to be replaced subsequently by an

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**Financial/Nonfinancial disclosures:** The authors have reported to CHEST that no potential conflicts of interest exist with any companies or organizations whose products or services may be discussed in this article.

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DOI: 10.1378/chest.10-0927

**REFERENCES**


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