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Author's Response

Sir:

We agree, it would be desirable to subject the postmortem tissues to an *in situ* PCR, a technique that needs to be established in more centers in the future. With regard to the time-dependent course of viral myocarditis, as studied in a mouse model, early virus-induced myocardial damage can take place already before histological signs of myocarditis defined by the Dallas criteria can be observed (1). These early phase dependent viral lesions can only be detected via electron microscopy, they also occur ahead of immunohistochemical signs of myocarditis (e.g., LCA, CD3, CD68). Therefore, not only *in situ* PCR but also electron microscopy can be helpful, especially to investigate the conduction system of the heart.

Regarding the paper of Cioc and Nuovo with very interesting results, it also should be mentioned, that in contrast to the detection of other viruses, e.g., enteroviruses, the presence of parvovirus B19 DNA within the myocardium until today is not qualified as a pathological finding itself. Therefore, it will be helpful to find parvovirus B19 replication in the endothelium of small vessels, in myocardial interstitium or in cardiomyocytes using in situ PCR. For enteroviruses, it was demonstrated in vitro, that enteroviral protease 2A cleaves and therefore functionally impairs dystrophin, a cytosceletal protein of cardiomyocytes. During infection with coxsackieviruses B3, the dystrophin-glycoprotein-complex, therefore becomes disrupted and the sarcolemmal integrity is lost (2). A parvovirus B19 variant is known to cause sudden death due to virus-induced myocarditis in young dogs (3), but we do not yet know the exact pathophysiological mechanism.

References

1. Feldman AM, McNamara D. Myocarditis. N Engl J Med 2000;343: 1388–98.
2. Badorff C, Berkely N, Mehrota S, Talbouk JW, Rhoads RE, Knowlton KU. Enteroviral protease 2A directly cleaves dystrophin and is inhibited by a dystrophin-based substrate analogue. J Biol Chem 2000;275:1191–7.
3. Lenghaus C, Studdert MJ, Finnie JW. Acute and chronic canine parvovirus myocarditis following intrauterine innoculation. Veterinary Journal 1980;56:465–8.

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