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Mercury and Idiopathic Dilated Cardiomyopathy

We read with interest the recent report on trace metals and idiopathic dilated cardiomyopathy by Frustaci et al. (1). As far as we know, this is the first report to show that markedly increased concentrations of mercury (Hg) in cardiac tissue are associated with a specific cardiac pathology.

A recent review cites a number of studies that clearly establish that the largest source of nonoccupational Hg exposure for the general population is their dental amalgam fillings (2). At least 70% of all Hg ions excreted in human urine originate solely from amalgam fillings (3). Therefore, we wonder if it would be possible for Frustaci et al. to obtain dental histories for their patients.

The suggestion offered by Frustaci et al., that Hg^{++} acts as a Ca^{++} antagonist at the actin–myosin junction, thereby inhibiting sarcomere contraction and ultimately myocyte function, is certainly a possibility. Another possibility they might wish to consider is that Hg causes disruption of the microtubule structure, a major cytoskeletal component of most cells. We have demonstrated this phenomenon in brain neurons where Hg inhibits guanosine triphosphate nucleotide binding to beta-tubulin, thereby blocking an essential step in the polymerization of tubulin molecules for microtubule formation (4). Perhaps the same phenomenon also occurs in myocardial cells.

We encourage Frustaci et al. to pursue the matter of dental histories for their patients, which may account for the inordinately high levels of Hg (22,000 times greater than that in control subjects) seen in the heart tissue of patients with idiopathic dilated cardiomyopathy.

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REPLY

We thank Drs. Lorscheider and Vimy for their observations. Sarcotubular abnormalities were a common ultrastructural finding in our patients with idiopathic dilated cardiomyopathy, and they may well be related to the extremely high level of mercury in the myocardiocytes. Alterations of beta-tubulin interfering with microtubule organization were reported in idiopathic as well as anthracycline-associated dilated cardiomyopathy (1,2).

As far as the possible origin of mercury from dental amalgam is concerned, we failed to include dental history in the patient questionnaire assessing the environmental exposure to heavy metals and toxic trace elements. However, at a recent follow-up, of the original group of 13 patients with idiopathic dilated cardiomyopathy, 10 (three patients died in the meantime) were specifically questioned about their dental history, five of whom had dental amalgam fillings, but their myocardial mercury concentrations were not significantly higher than that of the others. Finally, the simultaneous myocardial increase of antimony, and to a lesser extent of silver, arsenic, gold, chromium, lanthanum and zinc, in these patients, as well as the concentration of normal trace elements in their skeletal muscle biopsies, makes the pathogenetic role of a dental mercury source rather unlikely.

A cell membrane dysfunction, possibly induced by a myocardial viral infection, such as coxsackie B virus, might be a more plausible explanation.

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