Letters to the Editor

The following is the abstract of the article discussed in the subsequent letter:

Chen, Yingjie, Robert C. Serfass, Shannon M. Mackey-Bojack, Karen L. Kelly, Jack L. Titus, and Fred S. Apple. Cardiac troponin T alterations in myocardium and serum of rats after stressful, prolonged intense exercise. J Appl Physiol 88: 1749–1755, 2000.—The goal of this study was to determine whether the stress of forced exercise would result in injury to the myocardium. Male rats with 8% of body weight attached to the tail were forced to swim 3.5 h (3.5S), forced to swim 5 h (5S), or pretrained for 8 days and then forced to swim 5 h (T5S). Rats were killed immediately after they swam (0 h PS) and at 3 h (3 h PS), 24 h (24 h PS), and 48 h after they swam (48 h PS). Tissue homogenates of the left ventricle were analyzed by Western blot analysis for cardiac troponin T (cTnT). Serum cTnT was quantified by immunoassay. Results indicated that, in the 3.5S, 5S, and T5S groups, serum cTnT was significantly (P < 0.01) increased at 0 and 3 h PS. The 5S group demonstrated a greater increase in serum cTnT than the 3.5S group (P < 0.01) and the T5S group (P < 0.01) at 0 h PS. Western blot analysis indicated significant decreases (P < 0.01) in myocardial cTnT in the 5S group only at 0 h PS (P < 0.01) and 3 h PS (P < 0.05). Histological evidence of localized myocyte damage demonstrated by interstitial inflammatory infiltrates consisting of neutrophils, lymphocytes, and histiocytes, as well as vesicular nuclei-enlarged chromatin patterns, was observed in left ventricle specimens from the 5S group at 24 and 48 h PS. Our findings demonstrate that stressful, forced exercise induces alterations in myocardial cTnT and that training before exercise attenuates the exercise-induced heart damage.

Exercise-induced myocardial damage in humans

To the Editor: Chen et al. (1) showed that a single bout of stressful, prolonged, intense exercise induced damage as monitored by cardiac troponin T (cTnT) in the heart and serum in the myocardium of rats. Furthermore, an increase in exercise volume induced more myocardial damage, and training before stressful, prolonged, intense exercise provided protection from myocardial damage.

These findings are in agreement with recent findings in humans (2, 3), in which strenuous endurance exercise was demonstrated to cause myocardial damage, as indicated by increased cTnT and cardiac troponin I concentrations and alterations in echocardiography immediately after the events. Comparatively, in humans, serum cTnT concentrations returned to baseline concentrations by 24 h (2). Evidence also exists that apparently healthy individuals who are not active enough to meet a traditional exercise prescription (preconditioning) are at high risk for subclinical necrosis caused by prolonged strenuous exercise (2).

Chen et al. (1) hypothesized that the rapid return to baseline suggests that release of cTnT from exercise-induced myocardial injury is likely from localized minor, irreversible myocyte degeneration. Concordant with this hypothesis, histological evidence of localized myocyte damage demonstrated by interstitial inflammatory infiltrates consisting of neutrophils, lymphocytes, and histiocytes, as well as vesicular nuclei-enlarged chromatin patterns, was observed in left ventricle specimens (1).

On the basis of this report (1) and recent findings in humans (2, 3), it is tempting to speculate that stressful, prolonged, intense exercise may result in minor, irreversible myocyte degeneration in humans. In agreement with findings in rats (1), regular physical exercise (preconditioning) may protect against cardiac myocyte injury caused by vigorous endurance exercise (2). It remains to be clarified whether myocardial injury in healthy individuals as a result of physiological stress has any long-term clinical or athletic consequence or relevance (2, 3). Finally, there is, as yet, no basis for determining which individuals will develop either a major cardiac catastrophe or subclinical necrosis (4).

REFERENCES


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