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FXCT imaging for biomedical research in cardiac disease

Tohoru TAKEDA[1], Jin WU[1], Qingkai HUO[2], Hidenori SATO[2], Takaaki HORII[2], Tetsuya YUASA[2], Kazuyuki HYODO[3], Takao AKATSUKA[2]

[1]Graduate School of Comprehensive Human Sciences, University of Tsukuba, Tsukuba-shi, Ibaraki 305-8575, Japan
[2]Faculty of Engineering, Yamagata University. Yonezawa-shi, Yamagata 992-8510, Japan
[3]Institute of Materials Structure Science, High Energy Acceleration Research Organization, Japan

Introduction

The fluorescent X-ray computed tomography (FXCT) with synchrotron radiation is being developed to depict the distribution of specific elements inside the biomedical object [1-5]. In-vivo and ex-vivo cerebral perfusion imaging of rat & mice, and ex-vivo myocardial fatty acid metabolism of cardiomyopathic animal model have succeeded after injecting non-radioactive iodine labeled cerebral perfusion agent (IMP) and fatty acid metabolic agent BMIPP, respectively [6-10]. In this paper, metabolic impairment due to aging was examined using cardiomyopathic hamster model.

Methods and material

The experiment was carried out at the bending-magnet beam line BLNE-5A of the Tristan accumulation ring in Tsukuba, Japan. The photon flux rate in front of the object was approximately 9.3 x 10^7 photons/mm^2/s for a beam current of 40 mA. FXCT system consists of a silicon (220) double crystal monochromator, an x-ray slit system, a scanning table, fluorescent X-ray detector, and two pin-diode detectors for incident X-ray and transmission X-ray data. The white X-ray beam was monochromatized to 37 keV. The monochromatic X-ray was collimated into a 0.25 x 0.5 mm^2 pencil beam. Fluorescent X-rays were detected in a high purity germanium (HPGe) detectors oriented perpendicular to the incident monochromatic x-ray beam. The data acquisition time was set 7-s. Object was various aged J2N-k cardimyopathic hamsters and aged matched normal hamsters from 8-weeks to 25 weeks. ^{127}I-BMIPP was injected through the femoral vein of each hamster under anesthesia. Each heart labeled with BMIPP was extracted after 5 min of injection and fixed by formalin.

The present experiment was approved by the Medical Committee for the Use of Animals in Research of the University of Tsukuba.

Results and discussion

The J2N-k cardiomyopathic hamsters begin to show a slight fibrosis at eight weeks of age, exhibit moderate car diac dysfunction, degeneration of cardiomyocyte and interstitial fibrosis at about 20 weeks of age, and finally die due to congestive heart failure at approximately 1 year of age. In the normal hamsters of all ages and cardiomyopathic hamsters at age of 8 weeks, the BMIPP was distributed almost homogenously. However, in the cardiomyopathic hamsters at age of 12 weeks, the BMIPP uptake decreased slightly and distributed heterogeneously. In the cardiomyopathic hamsters aged more than 12 weeks, the distribution of BMIPP became progressively more heterogeneous. At 25 weeks, the BMIPP uptake decreased significantly in the septum and inferior wall. In addition, marked dilatation of LV and LV wall thinning were well analyzed. These pathological changes could be analyzed quantitatively [11].

Thus, FXCT enables us to visualize and to analyze the age-dependent metabolic impairment of cardiomyopathy.

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*ttakeda@md.tsukuba.ac.jp