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**I-123 MIBG MYOCARDIAL SCANNING IN PATIENTS WITH PULMONARY DISEASES WHICH LEAD TO COR PULMONALE.** Y.Hirose, Y.Ishida, K.Hayashida, S. Fukuoka, Y. Shimotsu, T. Nishimura\*. National Cardiovascular Center and \*Osaka University, Osaka Japan.

I-123 metaiodobenzylguanidine (MIBG) myocardial scanning were performed in patients with the pulmonary diseases which lead to cor pulmonale to evaluate sympathetic dysfunction. Thirty three patients were studied [primary pulmonary hypertension (PPH) 11, pulmonary embolism (PE) 19, and pulmonary fibrosis 3]. The mean pulmonary artery pressure (PAP) was 45 torr. Heart/mediastinum ratio (H/M) and washout rate (WR) of left ventricle were compared with PAP, perfusion defect size of the patients with PE. Regional semiquantitative assessment were also performed using SPECT images. There was a negative correlation between H/M in delayed image (d-H/M) and PAP ( $r=-0.61$ ), and a positive correlation between PAP and WR ( $r=+0.45$ ). In patients with PE, there was a negative correlation between defect size and d-H/M ( $r=-0.40$ ), and positive correlation between defect size and WR ( $r=+0.41$ ). In regional assessment, septal and inferior segments revealed significant low MIBG uptake. MIBG scanning is useful in detection of myocardial damage in patients with cor pulmonale and its candidates.

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**LUNG ACCUMULATION OF I-125 MIBG IN THE MODEL ANIMAL FOR HEART FAILURE.** H. Takatsu, M. Arai, and H. Fujiwara. Gifu University School of Medicine, Gifu, Japan.

MIBG has been reported to accumulate in the sympathetic nerve rich organs, such as adrenal gland, heart, and spleen. An increased accumulation of I-123 MIBG in the lung compared with heart is also reported in heart failure. However, it remains to be shown clear whether these findings precede the decreased accumulation of MIBG in the failed heart, or not. We examined I-125 MIBG accumulation in lungs and left ventricles of cardiomyopathic hamsters (n=16) at either stage of hypertrophy and early heart failure and of age-matched controls (n=16). Hamsters were injected with 296 kBq of I-125 MIBG, and were sacrificed 4 hours after injection. Left ventricles and right lower lobes of the lung were dissected, weighed, and, counted. Desipramine, a neuronal uptake blocker, was pretreated in some animals to evaluate the non-neuronal accumulation, separately. MIBG accumulation in the lung was  $0.062 \pm 0.008$  and  $0.057 \pm 0.015$  %kg·Dose/g (cardiomyopathic vs. control) at hypertrophic stage and  $0.066 \pm 0.012$  and  $0.068 \pm 0.013$  %kg·Dose/g at the early heart failure stage, in spite of a significant reduction in the heart of early heart failure. In addition, desipramine failed to significantly reduce lung accumulation, suggesting less contribution of the neuronal uptake of MIBG in lung.

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**EVALUATION OF CHANGE RATE MAP OF TL-201(TL) MYOCARDIAL SCINTIGRAPHY IN CARDIOMYOPATHY.** H.Higashino, S.Nakata\*, H.Fukuda, N.Ochi, T.Tachibana, T.Kikuchi\*, Y.Sugawara\*, Y.Yasuhara\*, S.Tanada\*, M.Hamada\*, and K.Hamamoto\*\*. Uwajima Social Insurance Hospital, \*Ehime University and \*\*Yawatama Municipal Hospital, Ehime, Japan.

The purpose of this study was to evaluate the regional reduction of uptake of TL with change rate map in cardiomyopathy.

Counts per pixel of early (BE) and late (BL) Bull's-eye images were transformed to equalize mean value of BE with that of BL, and change rate (CR) of BL to BE was calculated. Then CR was standardized with mean value of CR (CRN) of increasing area ( $CR \geq 1$ ) ( $CRS = CR/CRN$ ). Therefore, CRS, which was not influenced by extent and severity of reduction, can be calculated.

In DCM patients (n=2), as early uptake of TL was higher, CRS was lower ( $y=-0.004x+1.28$ ,  $r=-0.419$  ( $p<0.05$ ) (2 years),  $y=-0.004x+1.17$ ,  $r=-0.516$  ( $p<0.01$ ) (1 years)). Uptake of TL may reduce in all myocardial segments.

In HCM patients (n=2), there was no significant correlation between early uptake of TL and CRS. Extent and severity of Low CRS were increased ( $CRS<0.8:12.5\%$  (2 years),  $CRS<0.8:33.3\%$ ,  $CRS<0.7:12.5\%$ ,  $CRS<0.6:4.2\%$  (5 years)). Low CRS area may be reflected by abnormal myocardium.

In conclusion, CRS may be a useful index to evaluate extent and severity of regional abnormality in cardiomyopathy.

## 1051

**THE SCINTIGRAPHY CAN EXCLUSIVELY DETECT MYOCARDIAL DAMAGE DUE TO ELECTRICAL BURN.**

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It is difficult but important to investigate myocardial damage associated with electrical injury.

Seven patients with electrical injury were evaluated with electrocardiogram, CK-MB, ultrasonic cardiogram, coronary arteriography, and myocardial single photon computed tomography (SPECT) of Tl-201 Cl and I-123 Metaiodobenzylguanidine (MIBG).

The electrocardiogram abnormalities were transient. The CK-MB, which did not reflect myocardial damages, increased in 2 patients. Most patients showed normal wall motion except 2 with mild hypokinesis on the anterior wall. All 5 patients undergone coronary arteriography demonstrated normal findings without stenosis nor thrombi. Tl Cl SPECT revealed myocardial damage in all patients and I-123 MIBG SPECT showed abnormal accumulation in 5 of the 6 patients examined. The myocardial damage often found in anteroseptal wall. The accumulation decrease were greater in Tl Cl SPECT than I-123 MIBG, which different findings meant sympathetic nerve system should be less damaged than myocardial cell.

Tl-201 Cl SPECT and I-123 MIBG SPECT are the most effective methods to detect myocardial damage which could not be found with other exam.