

the pre-BTO state, and were well matched with other factors.

<sup>99m</sup>Tc-HMPAO brain SPECT before and during BTO seems to be a simple and objective method for prediction of permanent neurologic deficits when the comparative uptake of middle cerebral artery territories during BTO is lower than 85% of that before BTO.

### 7. <sup>99m</sup>Tc-HMPAO Brain SPECT for Evaluation of Brain Function Recovery after Intracarotid Arterial Urokinase Therapy Acute Cerebral Infarct

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To evaluate with <sup>99m</sup>Tc-HMPAO SPECT the brain function recovery of the infarcted area after early recanalization (less than 6 hours) of the occluded artery with intracarotid arterial urokinase therapy (ICAU).

Intracranial artery occlusion was confirmed in three patients with emergency carotid angiography done within the initial 6-hour period, after which recanalization of the occluded vessels was attempted with ICAU and 1 week after ICAU to evaluate the brain function of the infarcted area.

Complete recanalizing of the occluded vessel was seen in one patient after ICAU, and focal recanalizations were achieved in the other two patients. Before the ICAU, <sup>99m</sup>Tc-HMPAO brain SPECT showed decreased uptake of the infarcted area in all three patients, but the <sup>99m</sup>Tc-HMPAO brain SPECT performed 1 week after ICAU showed increased uptake of the recanalized area, suggesting brain function recovery and clinical improvement.

Brain function can be recovered if the occluded

artery is recanalized within the initial 6 hour period using ICAU. This was confirmed with <sup>99m</sup>Tc-HMPAO brain SPECT in our three patients.

### 8. Leukocytic Accumulation in Acute Human Cerebral Infarction

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White blood cells (WBCs) have been demonstrated to contribute to postischemic damage in a number of tissues including heart, intestine and skeletal muscles. Recently were proposed evidences that leukocytes may be involved in the development of cerebral ischemic injury. To examine the involvement of WBCs in cerebral ischemia, we performed brain SPECT using leukocytes labelled with <sup>99m</sup>Tc-HMPAO.

Thirteen patients with acute cerebral infarction were studied. Thirty-six ml of venous blood of the patients was withdrawn and the purified WBCs were incubated for 30 minutes with <sup>99m</sup>Tc-HMPAO. Labelled WBCs were injected intravenously and brain SPECT was done in 4-6 hours. In eleven cases a well defined area of increased radioactivity was revealed in the corresponding infarcted lesion of the cerebral hemisphere. Eight of them showed intense uptake on SPECT image. The periods from onset of neurologic deficit ranged from 5 days to 12 days in these eight cases. Six of these eight had cardiogenic embolic infarction. Second SPECT was done in a patient at four weeks after ictus and still showed the intense uptake in the infarcted lesion.

We think that these findings indicate active migration and tracking of labelled WBCs in cerebral infarcts and implicate the role of leukocytes in the acute ischemic brain injury.