ORIGINAL ARTICLE

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Effects of ion channel modulators in the influx and efflux of Tc-99m-MIBI

Ali S. ARBAB,* Kiyoshi KOIZUMI,** Keiji TOYAMA,* Takao ARAI* and Tsutomu ARAKI*

**Department of Radiology, Yamanashi Medical University*
***Department of Radiology, Tokyo Medical College Hachioji Medical Center*

Possible involvement of cell membrane ion transport systems in the uptake and extrusion of Tc-99m-MIBI was investigated by using various buffers with or without Na^+ and Ca^{++}, and ion transport inhibitors in a tumor cell line. The ion transport modulators dimethyl amiloride (DMA), verapamil, flunarizine and monensin were used. The uptake of Tc-99m-MIBI was significantly increased in all buffers containing either Na^+ or Ca^{++} alone or none of them. There was significantly increased uptake of Tc-99m-MIBI especially in buffers without Na^+. Verapamil, a L-type Ca^{++} channel blocker, increased Tc-99m-MIBI uptake in all buffers. Flunarizine, which inhibits Na^+/Ca^{++} channels, caused significantly increased accumulation of Tc-99m-MIBI only in buffer containing both Na^+ and Ca^{++}. Monensin, a sodium ionophore, significantly increased uptake of Tc-99m-MIBI. DMA, a potent Na^+/H^+ antiport inhibitor, significantly inhibited the uptake of Tc-99m-MIBI in all buffers. In conclusion, Tc-99m-MIBI behaves like Na^+ during its uptake and extrusion. Extrusion of Tc-99m-MIBI may involve both verapamil- and flunarizine-sensitive pathways.

Key words: Tc-99m-MIBI, tumor cells, Na^+/Ca^{++} channels, verapamil, flunarizine