Catecholamines in the adrenal gland was also decreased after oudenone administration. A maximum reduction was observed between 8 and 11 hr after the administration (Fig. 2). The time course of changes of catecholamine levels in the adrenal gland was similar to that of tyrosine hydroxylase activity. Norepinephrine level in the heart was decreased after oudenone administration. A maximum reduction of norepinephrine was observed between 9 and 11 hr after the injection (Fig. 3). Dopamine and norepinephrine levels in the brain showed a slight decrease at 11 hr after oudenone administration (Fig. 4).

Since tyrosine hydroxylase activity in the adrenal gland was found to be inhibitied *in vivo* after the administration of oudenone, the decrease in the catecholamine levels may be attributed to the inhibition of catecholamine synthesis at the tyrosine hydroxylase stage. The hypotensive effect of oudenone could be attributed to the inhibition of tyrosine hydroxylase *in vivo* and the resultant reduction of tissue catecholamines.

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Addendum—The structure of oudenone has been reported to be: (S)-2-[4,5-dihydro-5-propyl-2 (3H)-furylidene]-1,3-cyclopentanedione. M. OHNO, M. OKAMOTO, N. KAWABE, H. UMEZAWA, T. TAKEUCHI, H. IINUMA and S. TAKAHASHI, *J. Am. chem. Soc.* **93**, 1285 (1971).

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Hepatic free radical levels in ethanol-treated and carbon tetrachloride-treated rats

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THE OBSERVATION by Gallagher[1] that the antioxidants, α-tocopherol (vitamin E), sodium selenite and *N,N'*-diphenyl-*p*-phenylenediamine (DPPD), afforded rats protection against the lethal effects of carbon tetrachloride (CCl_4) and ameliorated histological damage to the liver has given impetus to a large volume of studies designed to elucidate the protective and toxic mechanisms involved. Carbon tetrachloride hepatotoxicity has been the subject of a recent comprehensive review.[2]