dihydroxyphenylacetic acid (DOPAC), another DA metabolite, were increased up to sixfold by 5-hydroxytryptophan, the amino acid precursor of 5-hydroxytryptamine (5-HT). They suggested that this effect might be due to the displacement of DA by 5-HT formed in dopaminergic neurons. Similarly, it is possible that increases in DA metabolites that have been reported following tyrosine injections (Sved et al 1979; Edwards 1982) result, in part, by the indirect actions of tyramine formed in DA neurons.

In summary, we have demonstrated that DOPS produces an increase in the brain levels of DA metabolites. Caution should be used before any behavioral or pharmacological effects of DOPS are attributed solely to the function of NA neurons.

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LETTER TO THE EDITOR

In vitro and in vivo effects of pentobarbitone sodium

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There is now good evidence that the anaesthetic properties of barbiturates may be due, in part, to an enhancement of synaptic inhibitions in the central nervous system mediated by γ-aminobutyric acid (GABA) (Nicoll 1980). Recently pentobarbitone sodium has been demonstrated to enhance the binding of [^{3}H]GABA to rat brain synaptosomal membranes (Willow & Johnston 1980), in agreement with the observed effects of this agent on the inhibition of the firing rate of feline dorsal horn interneurons by electrophoretically administered GABA (Lodge & Curtis 1978). We report here the comparative pharmacological effects of two batches of pentobarbitone sodium (PB-A and PB-B respectively) obtained from two commercial sources.

PB-A produced light and inconsistent anaesthesia in cats following intraperitoneal (i.p.) administration of 35 mg kg^{-1}, and the dose required to produce loss of the righting reflex in 50% male mice (ED50) was 43 ± 1 mg kg^{-1} (i.p.) (calculated by probit analysis, n = 20 per dose group). The induction of anaesthesia in mice was preceded by hyperexcitability, characterized by front and hind limb extension and arching of the back. At a concentration of 100 μM, PB-A did not enhance GABA binding to rat brain synaptosomal membranes, under the conditions used by Willow & Johnston (1980).

On the other hand, PB-B was an effective anaesthetic of cats at a dose of 35 mg kg^{-1} (i.p.). The ED50 in mice for this compound was 33 ± 1 mg kg^{-1} (i.p.), and there were no signs of pre-anaesthetic excitation. At 100 μM, PB-B enhanced GABA binding to rat brain synaptosomal membranes by 35 ± 2%. Furthermore, PB-B, when administered electrophoretically or systemically, was effective in enhancing the effects of electrophoretic GABA on feline dorsal horn interneurons (Lodge & Curtis 1978).

Chemical analyses using high pressure liquid chromatography, infrared spectroscopy, optical rotation, and thin layer chromatography suggested that PB-A and PB-B were identical, and both samples conformed with internationally accepted standards for pentobarbitone as a chemical. This suggests that if the anomalous effects of PB-A were due to a trace contaminant, then such a substance was highly potent in antagonizing the anaesthetic actions of pentobarbitone. While at present there is no conclusive explanation for differences between the actions of PB-A and PB-B, it seems essential that the source of samples of pentobarbitone be identified in all publications concerned with its biological or neurochemical effects. Furthermore, reliance on chemical analysis alone may be inadequate for the biological specification of this barbiturate.

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