percentage of foetal defects being 4.8%, 4.7% and 4.7% respectively. In the third category are clonazepam (2.7%) and ethosuximide (3.2%) which produced the lowest incidence of malformations, which are not clearly shown in these experiments to be significantly different from the spontaneous control values of 1.3%. When assessed on a pooled litter basis (mean percentage litter effect) a similar pattern is seen.

It is interesting that similar defects, e.g. cleft palate have been reported in both the mouse and man. These results indicate that some anticonvulsants are safer than others in the mouse, and this may be true for man also. Therefore, it would be beneficial to try to determine from epidemiological surveys, which drug(s) is safest for women of child bearing age.

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A comparison of the effects of ouabain and potassium-free Ringer on the electrogenic sodium pump and slow synaptic inhibition in bullfrog sympathetic ganglia

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Slow synaptic inhibition in sympathetic ganglia is generated by a unique mechanism that differs from the increase in postsynaptic membrane conductance found at most synapses. Two hypotheses have been advanced to explain the mechanism of generation of the slow IPSP (inhibitory postsynaptic potential). Nishi & Koketsu (1967, 1968) proposed that the slow IPSP is produced by activation of the electrogenic Na^+ pump. On the other hand, Weight & Padjen (1973) proposed that the slow IPSP is generated by a decrease in resting Na^+ conductance. To resolve this controversy, we studied the effects of ouabain and K^+-free Ringer on both electrogenic Na^+ pumping and slow synaptic inhibition. The bullfrog sympathetic ganglion was investigated using sucrose gap recording. The nicotinic depolarization produced by acetylcholine (ACh, 3×10^{-3} M) was followed by an after-hyperpolarization which has been shown, in mammalian sympathetic ganglia, to be due to the electrogenic pumping of Na^+ (Brown, Brownstein & Scholfield, 1972; Lees & Wallis, 1974). This ACh after-hyperpolarization was abolished by K^+-free Ringer or after 1 h in ouabain (10^{-6} M). In contrast to this inhibition of the Na^+ pump, K^+-free Ringer enhanced the slow IPSP. Ouabain (1 h, 10^{-6} M) reduced but did not block the slow IPSP. Even after 1 h in ouabain (10^{-6} M), the K^+-free Ringer still potentiated the slow IPSP. These data suggest that the slow IPSP is not due to activation of the electrogenic Na^+ pump. On the other hand, the data are consistent with the hypothesis that the slow IPSP is generated by a decrease in resting Na^+ conductance.

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