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Previous studies in our laboratory revealed that acrylonitrile and paraquat reduced motor activity in rats. In the present study, phrenic nerve hemidiaphragm preparation was selected as the test model to localize the site of action of these chemicals. Acute exposure to acrylonitrile (ACN) 10^{-3} M in the incubation medium which was directly stimulated did not alter contraction of hemidiaphragm muscle. However, maximal stimulation of phrenic nerve in the presence of ACN 10^{-3} M resulted in a slight increase in force of the muscle contraction. Pretreatment with ACN also resulted in the slight increase of basal force of contraction especially in the 5-days treated group. Alteration of responses might be accounted for by a hypersensitivity of the post-synaptic junction following the ACN-induced continuing release of ACh from the nerve terminal. This might eventually lead to a depletion of endogenous ACh. Paraquat pretreatment at a dose of 3 mg/kg for 1 day did not alter the hemidiaphragm contraction induced by phrenic nerve stimulation or direct muscle stimulation. These results suggested that neuromuscular junction might not be the site of action of paraquat at the dose and duration used in this study.