than the blood pressure of the shocked cats given only vehicle at all times after release of the clamps.

The table summarizes survival data in the 3 groups of cats. Presented in the table is the ratio of animals surviving in each group to total number of animals in the group and the percent survival at the end of the experiments. In the sham shock group, all animals survived. The SAO shock group given vehicle had only one survivor out of 7 at the end of the experiment (14% survival). In contrast to this, 6 of 7 animals survived in the SAO shock group treated with naloxone, yielding a survival rate of 86%.

No significant MDF activities were observed in sham shock cats given naloxone. Plasma MDF activities averaged between 8 and 19 units/10 ml in the 8 cats studied. However, in 5 untreated SAO shock cats, final plasma MDF activity was 64 ± 4 units (mean $\pm$ SEM) indicating a significantly elevated MDF formation and accumulation. In contrast, 5 naloxone treated SAO shock cats exhibited plasma MDF activities of 28 ± 3 units, a value significantly lower than untreated shock cats ($p < 0.01$). These data show that naloxone significantly prevents the formation of the cardiotoxic peptide, MDF, during SAO shock in cats.

Discussion. The therapeutic action of naloxone in various types of circulatory shock has been proposed as evidence for the involvement of the endogenous opiates, specifically the pituitary peptide β-endorphin[3-7]. In this paper, we present data showing that naloxone is beneficial in splanchnic arterial occlusion shock in cats. Therefore, the endogenous opioid β-endorphin may be involved in the pathophysiology of SAO shock. Other factors to be considered in the pathogenesis of SAO shock are 1. the production of the cardiotoxic peptide MDF by the ischemic splanchnic region, 2. depression of myocardial function, and 3. massive release of lysosomal hydrolases[11,13,17-20].

We have shown that plasma accumulation of the cardiotoxic peptide MDF was prevented by naloxone in SAO shock. In addition to maintenance of circulatory function that may be due to the antagonism of endogenous opioid peptides, naloxone may contribute to improved survival through stabilization of lysosomal membranes and a reduction of plasma proteolysis. We have presented data showing that naloxone stabilizes lysosomal membranes and reduces proteolysis in vitro in cats during hemorrhagic shock[21]. These actions of naloxone would contribute to the lower circulating levels of the cardiotoxic peptide MDF which we observed in the present experiments and would improve survival[22], since both lysosomal hydrolases and MDF contribute significantly to mortality during circulatory shock[12].

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Occurrence of DDT and BHC residues in human milk in India[1]

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Summary. Samples of human milk from Punjab, India have shown the presence of DDT and BHC residues in amounts greater than those reported from most of the other countries.

During recent years increasing concern has been voiced about the presence of pesticide residues in human milk and their effects on breast-fed infants[2-4]. Residues of DDT and BHC have been reported earlier in adipose tissues[5-7] and blood[8] in the general population in India. This communication forms the first report on DDT and BHC residues found in human milk in India.

Materials and methods. During 1979, samples of milk were collected from 75 lactating women residing in the Punjab State (India), within a week after delivery. Pesticide residues were extracted by blending 3-5 ml of the subsamples with 2 vol. of n-hexane-acetone (1:1, v/v). The homogenate was allowed to stand till clear separation into 2 layers occurred. After the removal of the upper organic phase, the lower phase was re-extracted twice with 10 ml portions of n-hexane. The combined n-hexane extract, after concentration to 20 ml, was transferred to a separating funnel, to which 5 ml of concentrated sulphuric acid (specific gravity, 1.84) was added dropwise.

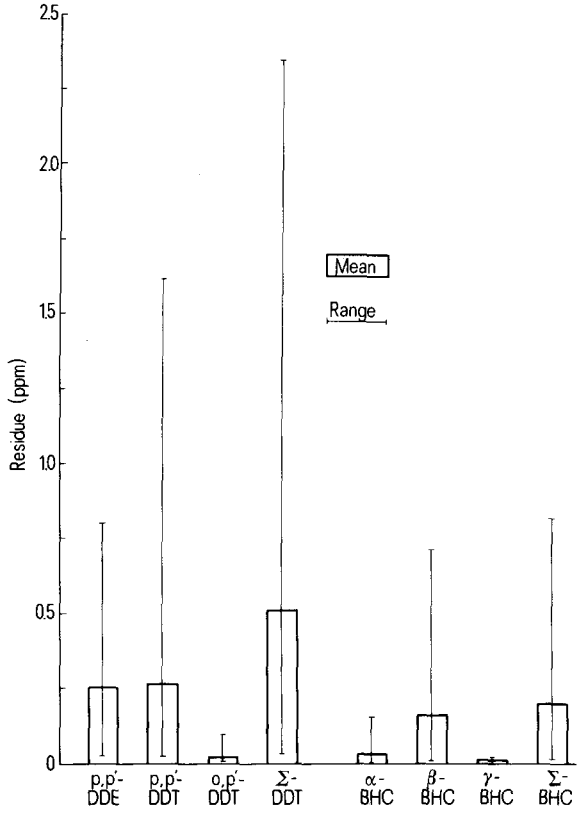
The contents of the separating funnel were shaken gently and allowed to stand. The lower sulphuric acid layer

DDT and BHC residues in human milk samples in India

Pesticide	Residues in whole milk (PPM)	
	Mean	Range
p,p'-DDE	0.25	0.02 -0.80
p,p'-DDT	0.26	0.02 -1.62
Total DDT	0.51	0.04 -2.35
alpha-BHC	0.031	0.002-0.160
beta-BHC	0.158	0.012-0.720
gamma-BHC	0.007	ND* -0.020
Total BHC	0.195	0.014-0.820

* Not detected.

containing digested fat was discarded. The n-hexane phase was washed with distilled water until it was neutral to litmus and concentrated to a suitable volume. Residues were identified and quantified in a Packard gas-chromatograph (Model 7624) equipped with a tritium source electroncapture detector and using a glass column, 1 m by 3.2 mm packed with the mixture of 1.5% SP-2250 and 1.95% SP-2401 on 100-120 mesh Supelco-Port. Detector, column and injector temperatures were 200, 190 and 210 °C respectively. Nitrogen at 70 ml/min was used as a carrier gas. Recovery of isomers of BHC and metabolites/isomers of p,p'-DDT from the spiked samples was above 80%. The results were not corrected for recovery. In addition to gas-chromatography, the identities of the pesticides were confirmed by TLC using silver nitrate-incorporated alumina[9]. The fat content of the milk was estimated by following the method described by Polishuk et al.[3] and ranged from 0.72 to 4.98% with a mean level of 2.54.



Mean and range of DDT and BHC residues in human milk samples, in India.

Results and discussion. DDT and BHC residues were present in all 75 samples of human milk. The range and mean levels of residues on a whole milk basis are depicted in the figure. DDT residues mainly occurred as p,p'-DDE and p,p'-DDT, while minor amounts of o,p'-DDT were also detected. The major part of the BHC residues consisted of the β-isomer along with small quantitiejs of α- and γ-isomers. DDT-residues (εDDT) ranged from 0.04 to 2.25 ppm and that of ε-BHC ranged from 0.014 to 0.820 ppm. Residue values when expressed on a fat basis ranged from 1.4 to 102.2 ppm in the case of εDDT and from 1.25 to 27.52 ppm in the case of εBHC.

The mean level of DDT-residues (0.51 ppm) found in human milk in India was more than the level reported from USA, Canada, Europe and Australia[1-2,10], though, the highest concentration (4.07 ppm) of DDT in human milk has been observed in Guatemala[11]. BHC residues in human milk in India have also been found to be higher than those reported from most of the countries of the world, except Japan[12]. DDT present at an average level of 0.5 ppm in milk represents an infant intake of 0.09 mg/kg/day, which is 18 times the acceptable daily intake (0.005 mg/kg/day) recommended by the WHO[13]. As no acceptable daily intake for BHC has been established, it is difficult to assess its potential hazards. However, these results as well as the earlier investigations[5-8] suggest the need for the reduction of body burdens of these insecticides.

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Anatomical evidence for cross regeneration of motor axons in a cockroach[1]

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Summary. The technique of electrophoretic application of intracellular markers is used to demonstrate that motor axons can regenerate into the contralateral limb of a cockroach after the nerve has been crossed to the contralateral side.

Recent studies have demonstrated that regeneration of neural elements in the cockroach occurs when the nerve roots of thoracic ganglia are manipulated so that their processes grow into the contralateral limb. By rotating the mesothoracic ganglion 180° and using a cobalt filling technique, Bate demonstrated that axonal processes of regenerating motor neurons could penetrate their original contralateral limb[3]. More recently, Fournter et al. developed a procedure by which the proximal stump of one metathoracic leg nerve (right nerve 5) was crossed to the