SUMMARY

The neurotoxic effects of acrylamide were re-investigated in rat. The neurotoxic signs can be produced either by intraperitoneal injection of a single lethal dose or by sublethal doses of acrylamide. The prominent signs of neurotoxic effects are weakness of the hindlimbs and ataxia. The time required for neurotoxic signs to appear depended on the magnitude of daily dose. However, there was no significance in the cumulative dose required to produce weakness of the hindlimbs regardless of the magnitude of daily dose. Animals had a complete recovery within one to two months after the administration of the drug was stopped.

From electrophysiological studies, acrylamide caused a reduction in both nerve conduction velocity and spike amplitude in nerves isolated from poisoned animals; the same effects were produced by high concentration of acrylamide applied to normal nerves. However, the intensity of these effects did not correlate with cumulative dose level. It was suggested that acrylamide produced its maximum effect in peripheral nerve after the first few doses. The increasing severity of intoxication with additional daily dose might be due to effects of acrylamide at sites other than peripheral nerve.

It is suspected that degeneration of peripheral nerves that were described by other investigators might be secondary to the other effects of acrylamide. It is also suggested that chronic acrylamide

poisoning is due in part to central action as well as peripheral action of the drug.

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