The Use of Dimercaprol (BAL) in the Treatment of Cadmium Oxide Fume Poisoning. H. N. MacFarland. Arch. Environmental Health 1, 487-499 (Dec. 1960).

Dosage-mortality data have been obtained from 200 g. Wistar rats subjected to a single, 15-minute exposure to arc-produced cadmium oxide fume. Analysis of the results by the probit technique yielded an estimate for the L(Ct)50 of 666 mg. min./cu.m., with confidence limits of 533 and 764 at the 95% level of probability. For the L(Ct)95, an estimate of 1086 mg. min./cu.m. was obtained, with limits of 896 and 2,265. The efficacy of dimercaprol in reducing mortality and in prolonging survival time of rats exposed to 3.1 to 4.5 times the L(Ct)95 of cadmium oxide fume has been examined in 3 factorially-designed experiments. Nearly complete protection against the lethal effect was obtained with a daily dose of approximately 100 mg. of dimercaprol administered subcutaneously in divided doses. Two factors, amount of dimercaprol per injection and frequency of injections per day, were varied in these experiments; however, the protection afforded was found to depend on the total daily dose of dimercaprol. These results have been discussed along with other relevant published data, and it is concluded that a reconsideration of the clinical use of dimercaprol in acute cadmium oxide fume intoxication is warranted. -- Author's summary

Effect on Environmental Temperature and Humidity on Lead Poisoning in Animals. A. M. Daeijer, S. N. D. Joardar and W. A. McQuary. Arch. Environmental Health 1, 463-477 (Dec. 1960).

Mice exposed to an environmental temperature of 95°F when injected with lead intraperitoneally or intravenously began to die sooner, had a higher mortality and shorter average survival time than mice at 72°F. This effect of high temperature on acute lead poisoning was more marked when the exposure followed immediately after the injection of lead than when exposure to heat also preceded the lead poisoning. The results were similar whether a high or low humidity accompanied the high temperature. Prolonged exposure to heat preceding the injection of lead did not reduce the harmful effect of heat exposure. Removal from heat at nights alleviated the effect to some extent. Exposure of rats to high temperature similarly increased their mortality following the intravenous injection of lead. Mice exposed to an environmental temperature of 60°F had a higher mortality and longer average survival time than those exposed to 72°F temperature when injected intraperitoneally with lead, but no significant differences were found when the lead was injected intravenously. Mice with chronic lead poisoning, produced by repeated injections of lead, began to die sooner and had a higher mortality when exposed to a high temperature following cessation of lead injections. Severe dehydration produced by restricting water consumption increased the mortality of mice from acute lead poisoning at all temperatures. The effect was much more marked when the lead was injected intraperitoneally than when injected intravenously. Weight loss and decrease in activity which occur upon exposure of mice to heat, when produced at normal temperature by food restriction and isolation, did not increase their susceptibility to lead. -- Cond. from authors' conclusions

Determination of Lead in Paint Scrapings as an Aid in the Control of Lead Paint Poisoning in Young Children. E. Kaplan and R. S. Shaull. Am. J. Public Health 51, 65-69 (Jan. 1961).

In order to prevent lead poisoning in children caused by ingesting paint from surfaces painted with paint containing lead, public health authorities are requiring warning labels on paint containing lead in excess of 1%, they are prohibiting the interior painting of dwelling units with such paint and they are ordering the removal of paint containing lead where it presents a hazard to young children. A simple and inexpensive procedure is described for rapidly screening paint scrapings at the 1% level to determine compliance with these regulations. -- Authors' abst.

Parkinsonism due to Manganese Poisoning. D. L. Ovedoff and F. Reinhold. Ann. Proc. (Johannesburg) 6, 195-197 (May 7, 1960).

The history and neurological findings described in 5 patients show that parkinsonism developed after relatively short exposures, from 6 to 24 months, to manganese fumes in a factory in Johannesburg where ferromanganese steel is produced. Apparently the oldest was