not designed to investigate this speculation, but it is now possible to examine this question experimentally. (151 references)

62. Rüssel, H. (Veterinary College, Hannover, Germany): Über die Bleiverteilung in Leber bei Vergiftung und die Probeentnahme zur chemischen Analyse. (DISTRIBUTION OF LEAD IN THE LIVER IN POISONING AND REMOVAL OF SAMPLES FOR ANALYSIS.) Deutsche Tierärztliche Wochenschrift 74, No. 4:96, 1968.

This is a brief summary of the author's dissertation (Abstr. No. 61) designed particularly to guide the veterinarian in the diagnosis of Pb poisoning in livestock. The directions for the removal of samples specify that 4 samples of 100-200 g be removed from different parts of the liver, peripheral and central regions. Diseased tissue should not be used for analysis. The directions for the analyst should identify each section and an average Pb content should be determined for all 4 parts.

63. Schlaepfer, W.W. (Washington Univ., St. Louis, Mo.): EXPERIMENTAL LEAD NEUROPATHY: A DISEASE OF THE SUPPORTING CELLS IN THE PERIPHERAL NERVOUS SYSTEM. Journal of Neuropathology and Experimental Neurology 28:401-18 (July), 1969.

Previous studies of experimental Pb neuropathy have shown a poor correlation between the amounts of axonal and segmental damage. In fact, axonal degeneration has been found in the absence of segmental lesions among individual animals and in certain species such as the rat.

Eighteen male Sprague-Dawley rats weighing 200-250 g each were fed 1% Pb acetate solution at a daily dose of ∿60 ml/rat, over a period of 3-18 mo. No clear-cut neurological deficits were observed during the course of the experiment although 4 rats died from unknown causes. Pairs of the remaining rats were sacrificed after 3, 4, 5, 6, 9, 12 and 18 mo. Three rats of comparable weight were used as controls. Under ether anesthesia the descending aortas were perfused with Ringer's solution containing glutaraldehyde and formalin. The following nerve structures were excised and prepared for examination with Os-O4, and stained with various techniques: sciatic and tibial nerves, spinal ganglia, posterior and anterior roots of the lumbar and sacral regions, and lumbar spinal cord. Minced tissues from peripheral nerves, ganglia and anterior horns of the lumbar cord were fixed in Os-O4, and ultrathin sections of Epon-embedded tissue were examined with an electron microscope. Sections of unosmicated tissues were examined unstained, others were contrasted with Pb citrate of uranyl acetate.

As illustrated in 21 microphotographs, prominent changes were evident in the supporting cellular elements of peripheral nerves, spinal nerve roots and spinal ganglia. Pathological alterations of the Schwann cells were manifested by widespread segmental demyelination and remyelination in the peripheral nerves and spinal nerve roots. A selective involvement of the capsular cells in the spinal ganglia was characterized by a proliferation of these cells and an accumulation of numerous dense bodies within their cytoplasm. These dense bodies contained a particulate material which had the electron density characteristics of a heavy metallic substance, compatible with Pb, in unosmicated and unstained tissue. Similar cytoplasmic dense bodies have been observed in the renal proximal tubular cells, another site of selective susceptibility to Pb toxicity. These findings suggest the possibility of a direct intervention of Pb in the metabolism of the capsular cells. An

increase of neurofilaments and a relative paucity of endoplasmic reticulum was seen in some associated sensory ganglion cells. More striking neuronal alteration occurred in the distal peripheral nerves and in the posterior nerve roots in the form of Wallerian degeneration. The accumulation of acid phosphatase activity in the nodal axoplasm of some peripheral nerve fibers appeared as an early form of axonal injury and attested to a special reactivity of the nodal region.

The author concludes by stating that the coexistence of segmental demyelination and Wallerian degeneration in the peripheral nerves of Pb neuropathy has been postulated to result from a common metabolic lesion of the supporting Schwann and capsular cells. This same pattern of pathological changes has been noted in some human polyneuropathies and may reflect a similar pathogenesis. (58 references)

64. Shiraishi, Y. (Nihon Univ., Japan): (EXPERIMENTAL STUDIES ON THE EXCRETION OF HEAVY METALS BY ADMINISTRATION OF DIMERCAPTOSUCCINIC ACID (MESO TYPE), WITH SPECIAL REFERENCE TO LEAD AND MERCURY.) Japanese Journal of Industrial Health 10:13-26, 1968.

A series of experiments were performed with mice and rabbits to determine the effectiveness of dimercaptosuccinic acid (DMS) in the detoxication of Pb and Hg. The effectiveness of DMS was judged by comparing results with those obtained by use of BAL and CaEDTA and in a control group. Results were summarized as follows: LD_{50} of DMS for mice was 3.132 mg/kg (±44 mg/kg) (probit method). In mice subjected to acute poisoning, no abnormality was found up to a dose of 2.8 g/kg; a slight, temporary change at 2.9 g/kg; a remarkable change at 3.0 g/kg. Just before death, dyspnea and deep breathing, tail raising, pupillary dilation, short fits of convulsions (tetanic-clonic type), roughened fur, swaying of the body, cyanosis, and lying on the side were seen in this order. Decrease in body weight of rabbits was the smallest in DMS-treated group, especially in Pb poisoning. As to blood findings, the DMS-treated group showed early recovery from anemia; erythrocytes with basophilic stipplings were fewer and disappeared more quickly. The DMS-treated group revealed a protective effect against diminution of urinary volume (more marked in Hg poisoning). Disturbance of renal function due to Pb poisoning was slightly less in BAL-administered and CaEDTA-administered groups, but in Hg poisoning it was least in the DMS-group. Although DMS was effective in the elimination of Pb and Hg in that excretion was completed comparatively early, CaEDTA and BAL differed in that excretion of the metals in large quantity occurred immediately after administration. The total amount of excreted Pb during the experimental period was larger in the DMS group than in the CaEDTA group but less in the BAL group. That of Hg was largest in the DMS group. (From author's English summary) (26 references)

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65. Sroczynski, J., Jonderko, G., and Węgiel, A. (Silesian Med. Acad., Zabrze, Poland): EFFECT OF LEAD POISONING ON THE SYNTHESIS OF RIBONUCLEIC ACIDS IN SOME RAT ORGANS. Polish Medical Journal 7, No. 5:1148-53, 1968. Translation of Patologia Polska 18, No. 3: 405-11, 1967.

See abstract No. 137, Vol. 4, 1968.

66. Stara, J.F., Nelson, N.S., Della Rosa, R.J. and Bustad, L.K. (Natl.

basis of measurements over the thorax during a 150-day retention interval. Values for the half-life of clearance were 235 and 290 days, respectively, for the long-term component. The relative concentrations of ^{228}Th, ^{224}Ra, and ^{212}Pb in tissues at "steady state" were expressed in terms of the initial LRT burden extrapolated from the long-term clearance phase. These concentrations of the daughters were discussed in terms of their application to the estimation of industrial exposure to ThO_2 and the calculation of radiation dose subsequent to ThO_2 incorporation. (From Nuclear Science Abstracts 23:Abstr. No. 50280, 1969)

187. Bolanowska, W., and Piotrowski, J. (Inst. Med. Pracy, Lodz, Poland): Kinetyka rozmieszczania i wydalania ołowiu (Pb-210) u szczurow. II. Wydalanie jednorazowej dawki dozylnej ołowiu. (KINETICS OF DISTRIBUTION AND EXCRETION OF LEAD-(^{210}Pb) IN RATS. II. EXCRETION OF A SINGLE INTRAVENOUS LEAD DOSE.) Medycyna Pracy 19, No. 2:133-42, 1968.

Adult albino male rats weighing 200-250 g were injected into the tail vein a Pb nitrate solution labeled with ^{210}Pb in a single dose of 1.0 and 0.1 mg/kg. The Pb excretion in urine and feces was observed during 98 days. The course of excretion is presented. The excretion of Pb in feces is a factor that especially decides the amount of total excretion. In the 3 mo period 2/3 of the Pb dose was totally excreted. No essential differences in Pb excretion were seen after administration of 2 different doses of 1.0 and 0.1 mg/kg; nevertheless the excretion of Pb in urine was somewhat higher for the lower dose. For the kinetic description of Pb excretion, the power and exponential models were used. The extrapolation of excretion equations to infinity seems to confirm the hypothesis of the existence of an unexchangeable pool the size of which is evaluated as being about 20% of Pb dose. (From authors' summary)

188. Gabbiani, G. (Univ. Montreal, Canada): EFFECT OF PHOSPHATES UPON EXPERIMENTAL SKIN CALCINOSIS. Canadian Journal of Physiology and Pharmacology 44:203-7 (Mar.), 1966.

Three experiments using 17 groups, each group including 10 female Sprague-Dawley rats (Holtzman strain), mean body weight 99 g, were conducted to test effects of phosphates upon calcification produced by treatment with Pb acetate and polymyxin (PMX). In the 1st experiment all rats received a 4 mg dose of Pb acetate in 1 ml of distilled water intravenously (iv) and 30 µg PMX in 0.5 ml water subcutaneously (sc) and then iv injections of 8 mg adenosine triphosphate (ATP), 7 mg Na pyrophosphate, 7 mg Na metaphosphate, 25 mg Na orthophosphate dibasic, and 13 mg Na citrate. In the 2nd experiment each rat received 4 mg Pb acetate iv; and sc 30 µg PMX alone or mixed with 1 of the following: 5 mg ATP, 2 mg Na pyrophosphate, 2 mg Na orthophosphate monobasic, 2 mg Na orthophosphate dibasic, or 10 mg Na citrate. In the 3rd experiment each animal received 2 mg Pb acetate iv; and sc 30 µg PMX, 12 mg Na orthophosphate monobasic, and 12 mg PMX plus 12 mg Na orthophosphate dibasic. After 3 days the rats were sacrificed. Results showed that calcinosis was inhibited by ATP and pyro- and metaphosphate, but aggravated by mono- and di-basic orthophosphate. (24 references)

189. Goździk-Żołnierkiewicz, T., and Moszyński, B. (Otolaryngological

IV. Observations on Warm-Blooded Animals

Clinic, Warsaw, Poland): VIII NERVE IN EXPERIMENTAL LEAD POISONING. Acta Oto-Laryngologica 68:85-9 (July-Aug.), 1969.

Forty young guinea pigs (weight, 300-350 g) were given ip injections of 1% solution Pb acetate at a total dose of 300 μg/kg given once a week for 7 wk; 10 guinea pigs served as controls. Eight of the Pb group died before the end of the experiment; the remaining were weak, lost weight, and growth was inhibited. The 5 that were able to run slowly appeared to have mild paralysis of their hind limbs. The animals were killed at the end of the 7-wk period, the temporal bones were removed for histologic examination and Pb in blood was determined by a polarographic method (Teisinger et al, 1956). Sections of the temporal bones of 16 animals were stained with hematoxylin-eosin, etc, according to Gomori, and those of 16 remaining animals, by a modified Heidenhain and Roger-Foot method for the demonstration of myelin sheaths and axons.

Pb in blood of the control group ranged from 0.025-0.33 mg%; in the experimental group, 0.31-0.42 mg%. Histologic examination showed no pathological changes in the sensory cells of the inner ear or in spiral and vestibular ganglion cells. The VIII nerve in the majority of poisoned animals showed segmental demyelination and axonal degeneration (as illustrated in 4 figures).

The authors discuss theories on the etiology and pathogenesis of the neuropathologic changes in chronic Pb poisoning and point to the resemblance of such early symptoms in man to those seen in other demyelinating syndromes. While the sensory cells of the inner ear were found to be most resistant to the effect of Pb, their work showed that Pb poisoning in guinea pigs might produce chronic demyelinating neuropathy of the VIII nerve with axon degeneration. The degree of changes differed as to animals, suggesting individual sensitivity. (14 references)

190. Hine, C.H., Cavalli, R.D., and Beltran, S.M. (Univ. Calif.; Standard Oil Co., Calif.; Golden Gate Hosp., San Francisco): PERCUTANEOUS ABSORPTION OF LEAD FROM INDUSTRIAL LUBRICANTS. Journal of Occupational Medicine 11:568-75 (Nov.), 1969.

This study was undertaken to determine whether unusual contact with Pb naphthenate, contained as a component of greases and industrial oils, would increase the Pb burden of the body. Test materials comprised 4 oils and greases, 3 of them commercial products containing Pb naphthenate and 1 experimental with its Pb in the form of an organic compound other than Pb naphthenate. The test animals were 20 New Zealand albino rabbits divided into 4 groups of 5, each group receiving 1 of the test oils. The 4 compounds were: (1) gear oil (1.35% Pb/w), (2) lubricating grease (0.80% Pb/w), (3) asphaltic gear lubricant (1.15% Pb/w), (4) experimental gear oil (0.55% Pb/w). In addition, a study was made of the effects of gear oil No. 1 on the skin of 10 human subjects and on their total body burden of Pb and Pb excretion.

Applications of 1.0 ml/kg were made on the backs of the rabbits (clipped areas 5 x 10 cm) and after 8 hr the remaining material was removed with gauze sponges and acetone. Twenty applications were made at the rate of 5 applications/wk for 4 wk. Prior to the 1st application cardiac blood samples were drawn and a 2nd blood specimen was drawn 24 hr after the last application, both for analysis of Pb content. At the end of the experiment the rabbits were sacrificed. The human subjects were examined before and