From: Wiener Tierärztliche Monatsschrift, vol.42, 1955

From the Medico-forensic and Ambulatory Clinic of the Veterinary College
Hanover (Director: Prof.Dr.E.Hupka)

CONCERNING FLUE DUST POISONING IN THE ENVIRONS OF METAL SMELTERS

by E.Hupka

with 7 Figures and 3 Tables

The increasing advance of industries into agricultural areas has as one consequence that ever more frequent complaints are heard about poisoning of animals through waste gases and effluents. Proving such poisonings is very difficult because the clinical and pathological symptoms vary considerably, and the chemical proof of poison in the body encounters particular difficulty because 764
the body harbours most of the allegedly toxic materials in varying quantities by nature. Of all industrial waste materials, lead is considered particularly dangerous to animals; without a doubt, lead-containing flue dust and water have a harmful effect on the organism when it is compelled to ingest greater amounts of them over longer periods of time. But especially the incontestable proof of lead poisoning is extremely difficult, i.e. proof of damage to health actually traceable to the ingestion of lead; numerous issues concerning lead poisoning on range land in the vicinity of smelting works remain contested because of insufficient proof. Often the definite establishment of a flue dust poisoning founders on the circumstance that the flue dust concerned does not contain one toxic dust but several, and the the question comes up: which poison is actually the one damaging to health? On the other hand, by addition and accumulation, other poisonous effects are created and also new and changing illnesses. Two such cases of a new type of illness, likely produced through the simultaneous action of several toxic substances, shall now be reported on:

In the years before the last war and in the years following it, there occurred, in the vicinity of two smelting works situated far apart, sicknesses among colts and also among cattle that were identical in their course and their pathological-anatomical evidence. The works in question smelt lead and zinc ores, where, besides traces of other elements, substantial quantities of lead, zinc, and sulphurous acid escape with the smoke from the stacks and later settle on the soil as flue dust. Near one of the metal works there is a superphosphate

factory, in whose flue gases there is also hydrofluoric acid. First, I want to describe the course of the sickness and the pathological changes, and then to discuss the causes, because I have succeeded, in two feeding tests, to produce the same symptoms of a sickness.

I. Course of Sickness

The sicknesses do not appear in animals kept in stables, but only in those on range land, and up to a distance of 5 km radius from the smelter. In the one case it is fertile marshland where the works are situated, in the other case both smelter and grazing land are located in the German sub-alpine mountains. In the immediate vicinity of the smelter, the green expanse gave way to areas with no vegetation or those where only weeds grow. Some 500 m distant, one can observe good grass growth again, but, seen from afar, the tips exhibit a slightly yellowish to reddish discolouration. From about 3,000 m on, one does not note this change on the grasses any more.

The indications of health disorders, which make their appearance during the course of the grazing period, are of a peculiar kind and have not been described so far. The German literature, in any case, does not contain examples of a related kind.

Mainly horses are affected, and among those predominantly colts, presumably because they remain exposed to the waste gases continuously for several 765
months on the range, while the older horses are on pasture only at night as a rule. However, with cattle, too, the same changes can be observed, although not so often and not to that extent. Beside a decline in nutritional condition and in milk yield, and a rough coat at undiminished appetite, above all the tired, stiff gait stands out. Later, the joints show increases in circumference, and they accept finger impressions, but they are without pain or elevated tempera- 766
ture; finally, considerable thickening takes place in the immediate neighbourhood of the joints, namely at the epiphyses. In this stage, the joints assume an angular position, they can be bent and stretched only under great pain, and they fold over forward, so that the animals can walk almost only tip-toe style, as illustrated in the pictures below (Figs. 1 and 2). The changes mentioned are most obvious on the carpal joints and hocks, and to a lesser degree on the knee, shoulder, pastern, and ~~crown~~ coronet joints. When moving forward, the animals apparently do not feel ~~one~~ any great pain, but they do not like to move, and when they do,

there is a definite creaking sound at the joints of the limbs that can also be heard at the intervertebral joints when the head or neck is being moved. According to veterinarians practising in the area, with older horses whistling in the larynx (roaring) is observed strikingly often; after dissection, it became evident that in several cases there had been definite atrophy and a chicken meat-like discolouration of the larynx muscles. Occasionally, animals on pasture are afflicted with spitting and difficulty in swallowing, but these troubles disappear on stabling. Frequently, the visible mucous membranes show a slightly yellowish discolouration. If the symptoms on the joints exist only in a small degree, then healing may come about over winter. Mostly, however, horses as well as cattle have to be destroyed because of progressive emaciation and difficulty in movement.

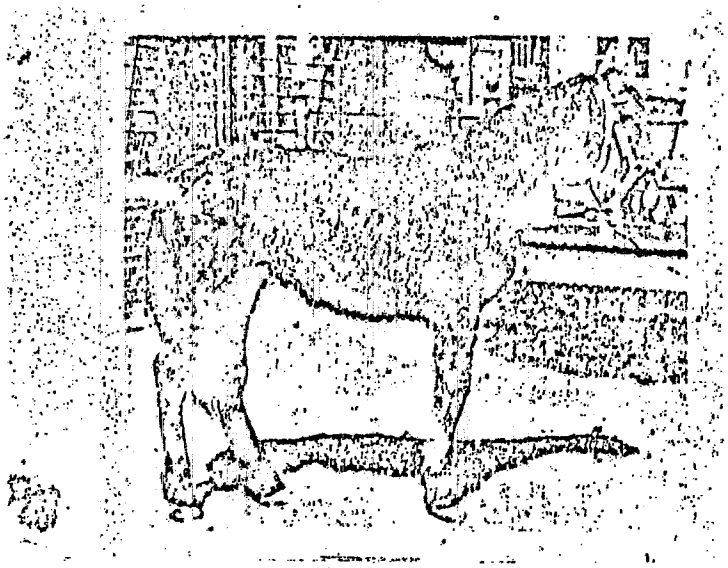

Fig.1 (left)
Incipient stiffening of the joints.

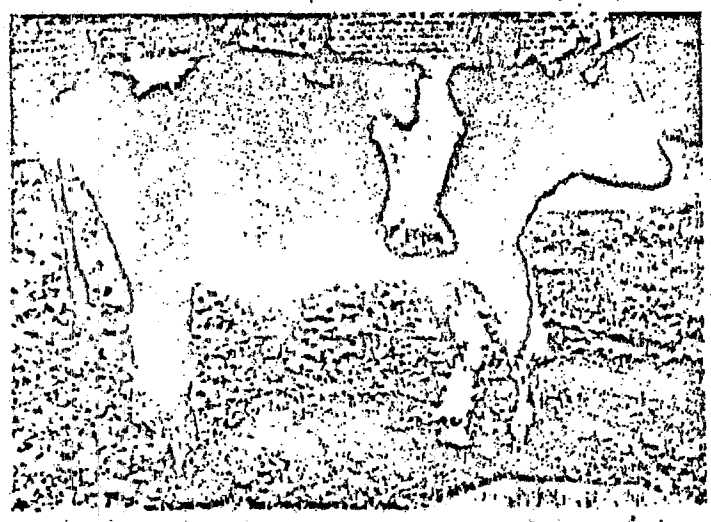

Fig.2 (left)
Inability to stretch the bent joints.

I had the opportunity to study the sickness on the pasture and in the stables and to observe a few diseased colts in the clinic, too; I could confirm the evidence cited above in every respect. A few very sick animals were killed and subjected to a post-mortem examination in the Pathological Institute of the Veterinary College Hanover. The following,highly interesting evidence was taken from the joints, and I am obliged to Prof.Dr.Cohrs, director of the Pathological-anatomical Institute, for allowing me to use it. The synovial fluid was increased and slightly turbid in appearance, the capsular walls and surroundings were altered oedematically. At the joint surfaces, the articular cartilage was often detached to a large extent from the underlying bone, so that a blister formed underneath. In some instances, some bits of cartilage had chipped off and were contained loosely in the joint; occasionally, the cartilage had dissolved and attrition was evident. Once in a while the lifted cartilage chip had fallen down again and covered loosely the worn spot. In the less severe cases, only thickening at the cartilage edges and incipient cartilage atrophy was noted. Changes similar to those at the joints of the limbs were also found at the intervertebral joints, especially at those of the neck. Here the intervertebral discs were at times completely detached and infiltrated with blood. The remaining evidence of the autopsy was negative. Since it was reasonable to assume flue dust poisoning, it was seen to it that chemical analyses for lead and arsenic were carried out regularly on the livers of horses killed in the region around the smelting works; these assays gave quite remarkable results (cf. Table 1). One of the colts killed was afflicted with tape-worms which exhibited a brown to black discolouration. The chemical analysis showed a high lead content. Blood tests always gave contents of from 11,000 to 15,000 granulozytes.

Table 1. Zinc, lead, and arsenic analyses on livers taken from animals from the affected area.

The liver contains, calculated in mg per 1 kg of fresh material:

(continued next page)

	1	2	3	4	5	6	7	8	9	10
Zn Zink mg	183,6	165,0	822,0	338.0	37,5	23.1	145,8	93	10,5	615,0
Pb Blei mg	1,2	5,73	1,45	0,36	0	0	5,4	0	1,5	1,8
As Arsen (AS_2O_3)	—	—	—	—	—	—	—	—	—	—

	11	12	13	14	15	16	17	18	19	20	22	21	24
Zn Zink mg	113,1	13,8	22,5	12	6,9	16,5	12,9	15,9	3,3	21,9	49,8	0	0
Pb Blei mg	1,95	0,24	0,75	0	1,8	0	0	0	0	0	0	0	0
As Arsen	—	—	—	—	—	—	—	—	—	—	—	—	—

	25	26	27	28	29	30	31	32	33	34	35	36	37
Zn Zink mg	9	Sp	217,5	234,4	22,2	0	0	278,4	490,8	81	110,4	11,7	10,2
Pb Blei mg	3,3	Sp	Sp	0	Sp	1,5	0,7	1,56	1,14	0	0	1,05	0
As Arsen	—	—	—	—	—	—	—	—	—	—	—	—	—

	38	39	40	41	42	43	44	45	46	47	48	49	50
Zn Zink mg	657	2920	1310	39	789	80	484,8	219,0	93,7	46	690	200	37
Pb Blei mg	6,0	26,6	5.3	3,5	7,9	1,6	11,4	2,0	0,63	0,8	3,6	3,6	2,9
As Arsen γ	—	—	—	—	—	—	—	—	—	—	20	50	50

Table 1 (left)

(Caption on previous page)

II. Feeding Test

As the management of the smelting works were interested in a clarification of the damages, we were asked to carry out a feeding experiment with flue dust from the two plants that were the possible causes of the harmful effects to health. The analysis of the flue dusts, performed by smelter staff, showed:

Table 2: Flue dust No. I (right)

	I
Pb	44,70%
Zn	5,21%
S	8,20%
As	0,22%
Cl_2	5,60%
Fe	—
Cd	—

Table 3: Flue dust No. II (left)

	II
Pb	16,87%
Zn	23,14%
S	12,53%
SO_4	8,33%
As	0,52%
Fe	5,80%
Cd	2,22%
F	0,05%

As test object served in each case a warm blooded colt, about one year old ~~each~~ and in moderately good nutritional condition. Any hidden conditions of sickness were excluded by a thorough general examination.

Colt No.1, fed with flue dust No.1 (Table 2):

From March 22nd, 1951, on, this colt received hay from the pasture in the vicinity of the metal smelters, and a daily ration of about 1½ kg oats. On April 30th, articular swelling could first be noted on the right hock, and on May 23rd on the left one, too. Its gait became a little stiff. Since the evidence of illness did not change substantially afterwards, from June 5th on 5 g (½ table spoon) of the above flue dust (cf.Table 2) were mixed into the oats, in addition to each forage ration. The colt thus received each day 15 g of the flue dust, besides the dusted hay. On June 8th already, difficulty in swallowing appeared, along with some regurgitation, decreasing on June 11th. Yet again and again later on, some food particles could be found in the nose, and the horse ate more slowly, so that it had to be assumed that the act of swallowing caused difficulty, if only at times. As the daily flue dust ration was increased to 20 g on July 27th, intensive swallowing difficulties occurred by August 1st; so much so that neither food nor water could be swallowed down. On August 3rd, the colt showed symptoms of pneumonia, and it was killed just before it would have died.

Fig.3 (right) :
Elbow joint with extensive cartilage detachment.

Towards the end, the colt's gait had become more and more laborious and stiff. Apart from the hocks, the two elbow joints showed distinct increases in circumference in the last stage. An increase in the number of leucocytes was already evident in blood tests from May 2nd on; there were between 11,000 and 14,500. Beside the hay affected with flue dust fall-out, the colt had ingested additionally a total of 975 g flue dust over 56 days. 769

After dissection, acute catarrhal pneumonia of both diaphragm lobes was determined, with food particles in the bronchi, furthermore oedema in the interlobular septa, gelatinous impregnation of the trachea in the course of the lymph duct, and moderate general icterus; in other words, the symptoms of an aspiratory pneumonia. In the hocks and elbow joints there were the above described ulcers on the cartilages, distinctly formed (Fig.3). Deposits of gall pigment were found in the liver and in the kidneys.

The chemical examination of 100 g liver for lead gave an assay result of 1.52 mg, and the analysis of 1000 g hay gave a lead content of 60 to 65 mg. According to information from that assay office, lead occurs in hay usually in so small traces that they cannot be measured macroscopically; their determination is then only possible by delicate microanalytical methods. In accordance with the investiagtions by DANCKWORTT, the lead content in the liver must be considered as substantially elevated. DANCKWORTT reached the conclusion, on the basis of his investigations, that the normal lead content of a liver is approximately 0.1 to 1.0 mg.

Colt No.2, fed with flue dust No.II (Table 3):

In a second feeding experiment, we checked the toxicity of flue dust No.II which comes from a plant near the first smelter, located in the same district but processing different raw materials (Table 3). The analysis of this flue dust was different from the first one mainly on account of the lead and zinc content. Whereas flue dust No.I (Table 2) had a lead content of 44.79% and a zinc content of 5.21%, flue dust No.II (Table 3) contained only 16.87% lead, but 23.14% zinc. The other values did not differ very much.

A six months old warm blood colt of Hanoverian breed, taken into the clinic on September 22nd, 1952, was used as test horse. The colt was well fed and exhibited no disorders in its general condition. Because a heavy infection with

ascarides and strongylides was proven by examination of the faeces, treatment against the worms was administered before the feeding experiment started. After the horse had a bout with the glanders, the feeding test could not begin until November 21st, 1952. At first, the colt was given 3 g of the flue dust daily, mixed into its fodder. From November 29th on, the daily ration was increased to 5 g, and from December 12th on to 8 g, yet no changes in its nutritional or general condition were noted. As 10 g of the flue dust were admixed to the fodder, beginning January 8th, 1953, the colt refused acceptance, so that from now on the dust had to be administered by a nose probang. Because the general condition did not change even now, the amounts given were stepped up further, namely on January 21st to daily 15 g, on January 26th to 20 g, and on February 4th even to 30 g daily. On March 25th acute symptoms of poisoning appeared, so that the flue dust doses were discontinued, only to be resumed on April 10th, after the symptoms had waned. Initially, 10 g daily were given, then from May 5th 20 g daily, 770
and from May 12th to June 11th 30 g daily. From November 21st, 1952 until June 11th, 1953, the colt has thus ingested a total of 3,270 g flue dust.

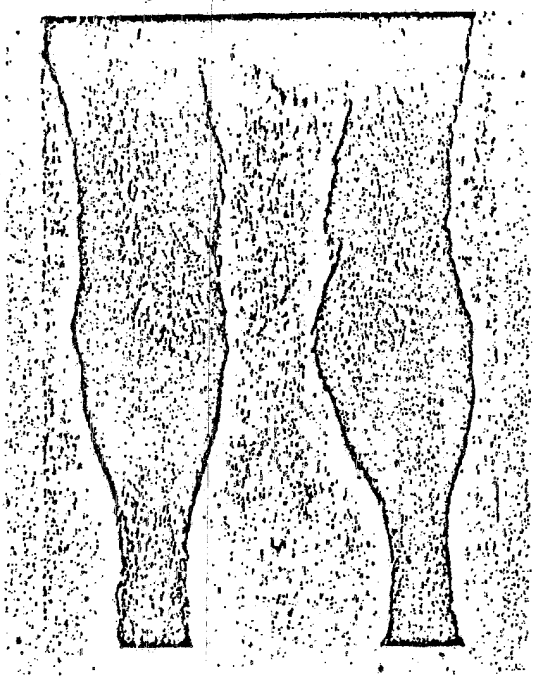

Fig.4 :
Swollen carpal joints

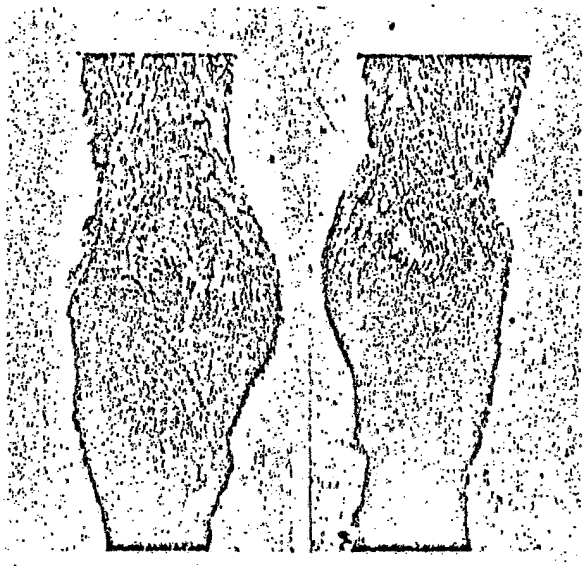

Abb. 5. Hochgradige Verdickung der Sprunggelenke.

Fig.5 :
High degree of swelling of the hocks

Clinical report: Temperature and pulse remained within normal limits during the entire feeding experiment. Fodder acceptance was regular until January 5th, and varying from then on. At various meal times the ration was not eaten up. The colt yawned often and preferred to stand or lie in a dark corner of its stall, as if shying away from light. However, the eye test was negative. The gait became stiff, and the carpal joints, hocks, and pasterns exhibited a distinct increase in circumference, feeling moderately tough, warm, and painful to the touch(Figs.4 and 5). From April 23rd, 1953, on, swelling could be observed on the knee and shoulder joints, too. Also from then on, the colt had difficulty in getting up.

Blood tests, which were carried out continually, showed modifications only in the white blood portion, similar to the first colt tested. The number of white blood corpuscles always hovered around the upper limit, between 8,000 and 12,000. ~~Ester count~~ Erythrocyte count and haemoglobin content showed a downward trend in the course of the illness. While the colt had an ~~ester~~ erythrocyte count of 8 million and a haemoglobin content of 72.9 before the feeding experiment, towards the end of the experiment these figures had dropped to 5 million erythrocytes and 50 Hb.

The basophilic ~~cepets~~ stiplings on the erythrocytes, well known in the medicine of humans, could never be observed by us. Nor did the differention of the blood pictures allow us to recognize any deviations; likewise, calcium and phosphorus determinations in the blood serum indicated no changes. The so-called lead fringe, purported to be a diagnostic for lead poisoning, also failed to appear, in this or the first test colt. 771

The Pathological-anatomical Institute (director: Prof.Dr.COHRS) issued the following autopsy report on the colt killed on June 11th, 1953: The big parenchyma showed no pathological-anatomical changes macroscopically. At the transitions from bony to cartilaginous ribs there were chestnut-sized swellings. The tarsal and carpal joints and their bony bases had thickened considerably. There was a lantil-sized ulcer, in the process of healing, in the elbow joint at the ulna. The histological examination showed a fine-grained, interlobular pigment in the liver, and a low degree of nephrosis in the kidneys.

Chemical examination: In the last weeks of the experiment, three lead determinations were done on the blood; in late March, when exhibiting distinct symptoms of poisoning, 0.466 mg lead were found, and after the symptoms had receded, 0.325 mg and 0.200 mg. After death, the ~~plasma~~ "liquor" *), the liver, and the

*) presumably tissue juices extracted from the ground-up infected organs, or else a sample of the total liquid contained in the animal - can't be sure from the text

kidneys were tested for lead, and the following values were determined:

0.055 mg in 100 ml liquor,
2,770 mg in 100 g liver substance,
0.670 mg in 100 g kidney substance.

The soil analyses for lead, zinc, and copper gave the following assays, 772
in the direction of the wind towards Northeast:

0.4 km distant: 300.19 mg lead, 400.0 mg zinc, 40.0 mg copper (in 100 g);
1.0 km distant: 53.0 mg lead, 162.0 mg zinc, 10.0 mg copper (in 100 g);
3.0 km distant: 6.0 mg lead, 23.0 mg zinc, 0.9 mg copper (in 100 g).

In the other wind direction, the contents were lower.

At about the same time, we could observe two colts for several weeks, which had grazed in the vicinity of a metal smelting works in a central German mountain region. The clinical and pathological-anatomical evidence was the same.

Fig.6 (right) : Swellings at the pasterns and the coronet joints.

Here, too, swellings at the joints and the stiff gait were the most notable features. After killing, the previously described changes on the articular cartilage was observed in all pathologically altered joints, sometimes to a considerable degree. In one case, the epiphysis suture was simply severed. The articular capsules were oedematically altered, the synovial fluid was much increased. Unfortunately, we did not succeed in ascertaining the composition of flue dust concerned (cf. Fig.6).

III. Critical Consideration concerning the Aetiology and Symptomatology of these Poisonings

When studying the analyses of the flue dusts, the large quantities of lead and zinc stand out; but these are not the same in the two flue dust samples, for in flue dust I the lead predominates and in flue dust II the zinc (smelter I: Pb 44.79%, Zn 5.21%; smelter II: Pb 16.87%, Zn 23.14%). That is to say smelter I lets more lead and less zinc escape, and smelter II more zinc and less lead. Of the other toxic components, there exist in substantial amounts: iron (smelter II 5.80%), sulphur (smelter I 8.20%, smelter II 12.53%), sulphurous acid (smelter II 8.33%), and chlorine (smelter I 5.60%). Besides, arsenic, cadmium, and selenium occur in traces. It is natural to see in lead and zinc the cause of the sickness. However, experience gained up to now speaks against viewing one of the two poisons as the sole cause. In the human and veterinary medicine, the following symptoms are cited as chronic consequences of lead poisoning: damages to the nervous system, with preference to certain nerves, like the recurrens (roaring), vagus (chronic colics), glossopharyngeus (pharyngo-paralysis, regurgitation), radial paralysis (inability to stretch the front limbs), and paralysis of the optic nerve with partial blindness. With humans, the basophilic stipling is considered a particularly important symptom in the early stages; this stipling of the red blood corpuscles is a consequence of damage to the blood-forming bone marrow. Furthermore, chronic organ disorders, such as nephroses (leaded kidneys), diseased blood vessels, and damage to the heart muscles, are said to appear. The stomatitis ulcerosa with the lead fringe, mentioned many times in the literature, is missing with horses and cattle, probably because these animals do not exhale by mouth. For the lead fringe is supposed to form by the combination of the hydrogen sulphides in the air with the lead, giving lead sulphide; however, since the horses do not breathe with their mouths, a lead fringe cannot form. A comparison of the symptoms that appeared in practice and in our test horses doubtlessly shows similarities: roaring, pharyngo-paralysis, spitting, probably caused by difficulties in swallowing, chronically diseased kidneys, emaciation. It is also interesting to note that these symptoms appeared more intensively and in a shorter time with flue dust I of the higher lead content than with flue dust II with the lower lead content.

On examining the results of the numerous lead assays on livers of animals

that took sick on the pasture, it is striking to note that the lead content was elevated in most cases, and mostly very much so, on the basis of the statements by DANCKWORTT that a lead content in the liver of over 1.0 mg must be regarded as pathological (cf.Table 1). In the two test colts, too, it was found after killing them that the amount of lead in their livers was higher. In colt I the amount was 1.52 mg% per 100 g liver, and in colt II 2.72 mg% per 100 g liver substance. An institute of forensic medicine has termed the quantities of lead in the blood as considerable also. Moreover, the high values in the soil in the surroundings of the plant are striking.

In second place, it is natural to allege that large amounts of zinc have a toxic effect. Indeed, zinc poisoning plays a big part in the veterinary literature, such poisonings are again and again suspected. However, despite numerous feeding experiments, no one has succeeded in producing a chronic zinc poisoning yet; presumably, because zinc is a passing poison which is not reabsorbed or accumulated, leaving the intestines quickly. But in cases of acute poisoning because of ingestion of large amounts of zinc salts, heart paralysis and damages to the excretive organs are said to occur. Even in healthy animals' livers, the zinc content varies so much, and occasionally is so high, that no firm conclusions can be drawn from it.

The other components of the flue dust are present only in small amounts, 774
so that it can scarcely be assumed that they exert a damaging effect on the organisms of the animals. Besides, of these poisons no symptoms of sickness are known that progress even remotely like the ones described above. Changes in the bone are known only in fluorosis, but they are of an entirely different kind. There, they are a matter of osteoporotic and osteosclerotic processes, especially at the tubular bones and accompanied by a brown discolouration of the teeth. The bones do show knotty enlargements up to pigeon egg size at the tela ossea, but no lifting of the epiphyses or ulceration at the articular cartilage (Fig.7). Therefore, it can be assumed that, although symptoms of chronic lead poisoning are predominant, a new type of sickness enters the picture, created through the synergic effects of other toxic substances (probably zinc) and characterized mainly by the localization in the joints. Or else, our knowledge of chronic lead poisoning is insufficient and does not correspond to the actual situation. Because of the frequency of the alleged damages from lead, and because of the long drawn-out litigation resulting from them, it would appear urgently

advisable to clarify the correct course of the disease in lead poisoning by feeding pure lead compounds to horses and cattle.

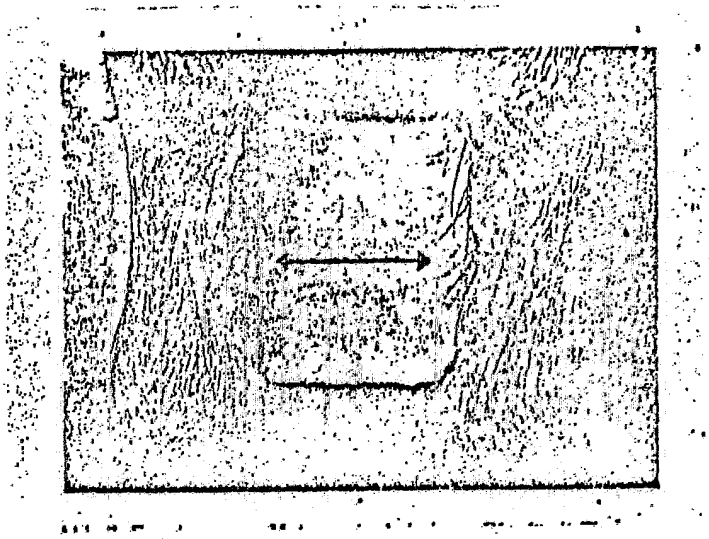

Fig.7 (right):

Exostoses at the metacarpus of a horse afflicted with fluorosis.

Summary

Summary

Chronic articular swellings have been observed in colts and cattle grazing in the vicinity of two forges. At autopsy an increase of synovia is stated; further it is observed that the articular cartilage is detached to a varying extent from the underlying bone. Later on, roaring, due to paralysis of the recurrent nerves, developed in several colts. Analysis of the forge dusts yielded following results:

Forge 1: Pb 16,87%; Zn 23,41%; S 12,53%; Fe 5,80%; Cd 2,22%; As 0,52%.
Forge 2: Pb 44,79%; Zn 5,28%; S 8,20%; Ce 5,00%; As 0,22%.

Identical articular lesions could be reproduced in two colts, each having been fed for several weeks on rations containing forge dusts. Further, manifestations of pharyngoparalysis appeared which in one case resulted in pneumonia due to pharyngeal dysphagia. The author suggests that the condition is due to leadpoisoning being complicated by other toxic substances. Analyses of several colt livers showed in all cases a lead content exceeding 0,2 mg per 100 g.

775

Literature References

Literatur: Cohrs, P.: Dtsch. tierärztl. Wschr. 53 : 22 (1946). — Danckwortt, (o. V.): Dtsch. tierärztl. Wschr. 49 : 365 (1941). — Fröhner-Völker: Lehrbuch der Toxikologie. Verlag F. Enke, Stuttgart 1953. — Günther, H.: Vet. Diss., Hannover 1954. — Hupka, E.: Dtsch. tierärztl. Wschr. 49 : 349 (1941). — Oesen, W. v.: Vet. Diss., Hannover 1942.

Cohrs, P. : German Veterinary Weekly, 53: 22 (1946)
Danckwortt, - : German Veterinary Weekly, 49:365 (1941)
Fröhner-Völker : Textbook of Toxicology, published by F.Enke, Stuttgart, 1953
Günther, H. : Veterinary dissertation, Hanover, 1954
Hupka, E. : German Veterinary Weekly, 49:349 (1941)
Oesen, W.v. : Veterinary dissertation, Hanover, 1942.

WPL11XI69