Review and Opinion

CASE OF [redacted] VS BETHLEHEM STEEL CO.

From the testimony of the claimant, and from the facts elicited from others concerning the nature and duration of the claimant's work there seems little doubt that his initial illness arose out of his employment and was in fact caused by it. The medical questions at issue therefore are concerned with (1) the causative factor or factors of the employment that caused the initial illness, and (2) the relationship, if any, between the occupational illness and the occurrence of the duodenal ulcer which, no doubt, gave rise to the hemorrhage and prolonged the disability.

1. The primary illness

The original complaints coincide with the well-known effects of the inhalation of fumes from overheated galvanized surfaces, the so-called "zinc chills," and the history of employment is such as to justify the belief that this type of industrial intoxication existed.

The occurrence of significant lead exposure in the course of this man's work has not been established with reasonable certainty. Undoubtedly there was some lead exposure in the course of burning lead-coated metal sheets and plates. It is well known that exposure to lead fumes originating in this manner, under conditions in which the workmen are inadequately protected, is one of the most serious of the lead hazards in industry. The severity of the exposure and its duration in this case, cannot well be judged from the available information. The testimony of the claimant is not definite as to the length or periodicity of the work with painted surfaces or as to the extent to which he protected himself by the use of a mask, and no

other evidence has been submitted to indicate whether this employee or others who carried on similar work on the ship in question absorbed significant or dangerous amounts of lead. It is suggested by certain statements of witnesses that serious cases of lead poisoning had not occurred among the workmen so employed by this defendant. Against a too easy acceptance of the conclusion is the knowledge of this reviewer that under certain conditions, work of similar type can and does cause serious and even fatal lead intoxication. One can only conclude that the evidence presented concerning the occupational conditions in this case is not adequate to justify a decision as to the significance of the lead exposure.

No analytical data on the blood or excreta of the claimant are available to show that he absorbed significant quantities of lead. Granting that the white precipitate obtained in the chemical analysis of the urine as described in the report from the laboratory, was lead sulphate, (this cannot be accepted with certainty) there is still no evidence to indicate that the quantity of lead in the urine was abnormal, unusual, or in any way indicative of hazardous lead exposure.

It follows from these facts that judgment concerning the existence or non-existence of lead poisoning in any period of the claimant's illness rests solely upon clinical interpretation of the nature of his illness. The type of illness and its course were not such as to justify a diagnosis of lead poisoning. Every early symptom described can more readily be explained as an effect of exposure to zinc fumes. The symptoms present after the first hemorrhage occurred are more plausibly to be attributed to the duodenal ulcer later shown to be present, than to the effects of lead, the presence

of which is uncertain. It is possible that there were certain other undescribed clinical features of the case that argued for lead as a causative factor in the mind of Dr. Nofsinger, but there is nothing in his testimony that gives convincing or even strongly suggestive evidence to that effect. The only point made that merits detailed discussion is that of the microscopic changes in the blood, specifically the stippling of the erythrocytes. Accepting the laboratory report at its face value, to the effect that the stippled erythrocytes were more numerous than the leucocytes in the early examinations, (greater assurance would be felt on this point if an actual count of the stippled erythrocytes had been made), there is still no evidence of lead absorption. This man had a severe hemorrhage and the conditions were right for the pouring out of large numbers of immature erythrocytes, provided he had a normally active bone marrow. That he had such a responsive bone marrow is shown by the satisfactory increase in the erythrocyte count as noted on successive blood examinations. It has been established beyond peradventure, that increased numbers of stippled erythrocytes are found regularly in secondary anemias and in most cases in which blood destruction occurs in the absence of injury to the bone marrow. The numbers roughly estimated as being present in this case are not unusual or remarkable, if the seriousness of the hemorrhages is considered. Thus the stippling can be explained entirely by the hemorrhages, and there is no evidence to indicate that any other explanation or supposition is warranted.

2. The origin of the duodenal ulcer and the source of the hemorrhages

There is a slight point of contradiction in the testimony, in that the claimant speaks of hemorrhage from the mouth, while the physician (Dr. Nofsinger) describes evacuation of blood from the bowel.

This apparent contradiction is best resolved by assuming that both actually occurred. This is compatible with the existence of duodenal ulcer as the source of the hemorrhage. Any cause for the hemorrhage other than the duodenal ulcer would be based on pure speculation and is wholly unjustified on the basis of the medical testimony. Hemorrhage from the stomach, intestine, or bowel would be an exceedingly questionable part of the picture of lead poisoning, and in this case, no such bizarre explanation of it is required.

There is little or no reason to suppose that the relatively short exposure to zinc fumes would have caused prolonged disability, and there is no medical evidence with which this reviewer is acquainted, that would warrant the belief that such exposure is likely to cause duodenal ulcer or to produce exacerbation of a quiescent duodenal ulcer. No causal relationship between duodenal ulcer and lead absorption can be seriously considered, nor can hemorrhage from such an ulcer be attributed reasonably to the effects of lead. In fact there is no sound reason to suppose that this was not duodenal ulcer of a common type. Hemorrhage from such an ulcer is not of infrequent occurrence, nor was there any unusual feature in this case when it is reviewed in that light.

Conclusions

My opinion is summarized as follows:

(1) The claimant was exposed to metal fumes, chiefly those of zinc in burning galvanized metal in sufficient concentration to induce typical effects in the form of "metal fume fever."

(2) There may have been significant exposure to lead compounds as the result of burning metal surfaces heavily coated with

paint, but evidence of the occurrence of such exposure is lacking both in the occupational history and in the clinical pattern of the illness.

(3) The disability resulting from exposure to metal fumes was of slight to moderate severity and would be expected to be of brief duration. Assuming that the lead exposure was significant the disability from that alone, or in combination with exposure to zinc would not have exceeded four to eight weeks in a case such as this in which there was neither paralysis or cerebral intoxication.

(4) The disability of the claimant over the period of months, and the large bulk of the medical care required by him during his illness resulted from hemorrhage due to duodenal ulcer. Neither the ulcer nor the hemorrhage is attributed to the effects of exposure to metal fumes.

Signed: _____
Robert A. Kehoe, M. D.

April 17, 1943