LEAD INDUSTRIES ASSOCIATION

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LEAD HYGIENE AND SAFETY
BULLETIN NO. 95

Subject: THREE IMPORTANT ARTICLES

To Members of the Lead Industries Association:

Herewith are reprints of three recent articles of which the first two will be of interest to those of our members concerned with the diagnosis and treatment of lead poisoning, while the third tells of the institution and maintenance of a workable industrial hygiene program.

A limited supply of additional copies of these papers is available. A brief summary of each follows:

"A Critical Appraisal of Current Practices in the Clinical Diagnosis of Lead Intoxication", Kehoe, R. A., Ind. Med. and Surg., 20:6, June, 1951.

Dr. Kehoe discusses very adequately the more troublesome aspects of the diagnosis of lead poisoning. Attention is focused on principles which should apply in arriving at a diagnosis, and on major sources of error and confusion resulting from unsound clinical thought and practice.

"Treatment of Lead Poisoning with Sodium Citrate", Hardy, H. L., Bishop, R. C. and Maloof, C. C. Arch. Ind. Hyg. and Occup. Med., Vol. 3, March 1951.

Four cases of lead poisoning in which treatment of the patients consisted primarily of sodium citrate therapy are presented. Evidence accumulated in this study points to effective relief of symptoms coincident with oral administration of sodium citrate. Studies of urinary lead excretion and urinary coproporphyrin are also presented.

"Industrial Hygiene at American Smelting and Refining Company", Abersold, J. N. and Nelson, K. W., Jour. of Metals, Vol. 189, No. 1, January 1951.

This review of a well organized, successful industrial hygiene program should be of great interest to all of our member companies. The authors discuss the medical services, the methods used for studying the working environment and the plant improvements which, with the interest and cooperation of employees at all levels, have resulted in greatly improved working conditions.

Manfred Bowditch
Director of Health and Safety

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A Critical Appraisal of Current Practices in the Clinical Diagnosis of Lead Intoxication

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Reprinted from INDUSTRIAL MEDICINE AND SURGERY, 20 6, 253-258, June, 1951

A Critical Appraisal of Current Practices in the Clinical Diagnosis of Lead Intoxication

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THE diagnosis of lead intoxication has long been the subject of intense interest and of frequent controversy among clinicians, especially among those whose practice concerns itself with cases of actual or alleged occupational plumbism or with medicolegal evidence or testimony in connection with such cases. More recently, considerable numbers of non-medical persons have come to be concerned with this problem, through their responsibilities in industry for the appraisal or control of occupational lead hazards, and through their activities in workmen's compensation procedures, in governmental health and labor departments, in labor organizations and in trade associations. Now does professional and lay concern end here, since every pediatrist, family physician and parent must pay attention to the usage of lead and lead compounds in the household under conditions which may threaten the health of infants and small children. To a much less significant extent, non-occupational lead poisoning occurs in the adult population as the result of a number of highly unusual situations, among which the most frequent, it seems, involve the use of lead pipe and lead-lined tanks for supplying and storing water for the household from a spring or well. Obviously, this hazard is limited almost entirely to rural areas, but members of poor families in many of our cities have also been poisoned by the use of battery liners scavenged from city dumps as fuel for stoves and fireplaces.

This disease, therefore, may be found among persons of all ages and in all social and economic levels of society, and it is for this reason, primarily, that it is such a troublesome diagnostic problem in the field of general medicine. Non-occupational lead poisoning occurs infrequently, and therefore few physicians in general practice have had enough experience or knowledge to enable them to make prompt use of effective diagnostic measures which may be available or to take the steps that will secure the future well-being and safety of patients who are, or who think themselves, afflicted with this illness. Occupational lead poisoning, on the other hand, is not rare in its occurrence among the patients of physicians engaged in private practice, but these cases occur largely among persons in the lower social and economic segments of the population and in the urban areas in which such persons live and work. They are not distributed evenly or widely among physicians engaged in private practice, and again, therefore, familiarity with the disease is sharply limited to a certain group of industrial physicians, to physicians who specialize in the study of occupational diseases, to those in certain communities whose private practice deals largely with the illnesses of laborers and their families, and to those who see patients in general hospitals in industrial centers. It is not surprising, therefore, that certain aspects of the diagnostic problem are obscure, and that diagnostic errors are numerous.

It is not within the scope of this paper to deal with all types of lead poisoning, or with the types of exposure to lead that give rise to them, or with all or even many of the factors which create clinical and medicolegal difficulties in connection with them. Instead, the discussion will be focused on occupational poisoning from the inorganic compounds of lead, on the simple principles which should apply in arriving at the diagnosis of the disease for both clinical and medicolegal purposes, and on a few of the deviations from sound clinical thought and practice which seem to be major sources of error and confusion at the present time.

From the Kettering Laboratory in the Department of Preventive Medicine and Industrial Health, College of Medicine, University of Cincinnati.

Presented at the Thirty-ninth Annual Meeting of the American Association of Industrial Physicians and Surgeons, Atlantic City, New Jersey, April 28, 1951.

Diagnosis

THE diagnosis of lead intoxication, as we shall deal with it here, is based upon a valid history of significant occupational exposure to lead, on the presence of symptoms strongly suggestive of the effects of absorption of lead, and upon certain physical signs which characterize absorption of lead or intoxication therefrom. It will be noted that this statement puts the em-

phasis on the actual existence of an intoxication which is to be identified by ordinary clinical means, and makes no reference to any laboratory findings. That such a statement fails to cover the entire diagnostic field, both from the aspect of the more or less chronic clinical manifestations of the disease and also from that of the technical armamentarium that can be brought to bear on its study, must be admitted. It is done deliberately to place the emphasis where it belongs among clinicians. The facts are that so much has been said and written, on the one hand, about the exotic and unusual clinical states that conceivably may result from the absorption of lead, and, on the other, about the diagnostic measures and criteria of the laboratory, that most of our clinicians have lost sight of the disease as it commonly presents itself, and of the basic conditions without which it cannot arise. The clinical diagnosis of lead poisoning, when it occurs, as it does in the very large proportion of all cases, in a more or less acute episode of illness following brief or prolonged exposure to dangerous occupational conditions, is not difficult. The problems we create for ourselves in connection with it arise from inexperience to be sure, but mainly from the assumption of knowledge which we do not possess and from sheer carelessness in the performance of simple clinical procedures. A survey of the records of cases of actual and alleged lead poisoning that have been in controversy and hence have come up for our review over a period of years has revealed one characteristic which is almost invariable. In most of these cases the patient did not have the benefit of initial careful clinical study. A slapdash history of occupational lead exposure, uncritically and yet dogmatically stated, a fragmentary recital of indefinite symptoms which were not investigated, and a physical examination that yielded only dubious information, together with some, often a multitude, of usually irrelevant, uncritically interpreted laboratory results, completes a document which portrays not a patient's illness but a physician's incompetence.

Clinical laziness and ineptitude are almost equally evident among the physicians in the lead-using industries, who so frequently rely upon non-specific, highly variable and outmoded laboratory procedures to make differential diagnoses of illnesses which can only be made by careful clinical study. It is one thing to apply the procedures of the chemical or clinical laboratory in a routine manner, and, by proxy, to the appraisal of the degree of hazard of an occupation. It is quite another to arrive at the diagnosis when illness occurs in an individual workman. Workmen in the lead trades are among the victims of gastric and duodenal ulcer, of appendicitis, of enterocolitis, and of numerous other gastroenteric, as well as neurological diseases. Moreover, cases of lead poisoning may occur as the result of unrecognized and improperly appraised exposure to lead in industrial plants, and, therefore, the diagnostic problem is always an intriguing one from the aspect of the cultivation and application of sound clinical method and judgment. Every case of illness which occurs among workmen exposed to lead should be worked up by the responsible clinician in such manner as to substantiate the diagnosis. If the diagnosis is that of lead intoxication, the records should provide the evidence that will stifle controversy and provide for proper and adequate medical care and for prompt compensation for disability. If it is some other illness, the record should be equally apt and convincing. If this definitive differentiation cannot be made in every case, there are but few exceptions, and there is, at least, no justification for not making an honest and capable attempt to do so. Moreover, in the performance of this professional duty, the physician has a role that is peculiarly his own, involving only the talents and skills which, on the basis of his training, may justly be expected of him, regardless of his experience or inexperience with the more modern techniques of industrial hygiene.

The Occupational History

IT MUST be recognized that the occupational history, as it pertains to the lead trades, is substantially useless, except as a clue which must be substantiated or discarded, unless it is obtained and recorded and interpreted by one who is wholly familiar with the operations in these trades and with the specific plant in which the operations are being carried out. The statements of a patient on these matters, almost without exception, are unacceptable as clinical evidence, and are unreliable in those features which relate to the quantitative appraisal of the exposure, not necessarily, or even commonly, because of the patient's animus or dishonesty, but because of his inability to differentiate the significant from the insignificant facts, even if the physician is capable of such differentiation and of conducting his inquiry along lines that will facilitate such differentiation. There are, however, certain facts which can always be obtained and are highly important in the interpretation of other clinical information. Since exposure to appreciable quantities of lead is a regular and unavoidable experience of all mankind,[1] the *quantitative* aspects of such exposure constitute the point, virtually the sole point, of the occupational history. The significance of occupational exposure to lead can be stated only in terms of its *duration* and its *intensity*. All questions put to the patient in relation to exposure are for the purpose of appraising these two items.

The Question of Exposure

"How long has the patient worked in the job at which he was working when he became ill? How long has he worked at this same job

elsewhere? What changes of occupation have occurred over the years, at what specific dates have they occurred, and what intervals have intervened? One attempts here to find out the duration of uninterrupted exposure to conditions quantitatively comparable to those in existence at the time of onset of the illness. If interruptions occurred, what was their duration? Above all, what was the last day on which the patient worked under conditions which involved exposure? The time factor, in relation to exposure, in its every aspect, is of the greatest importance. Without knowledge of this no subsequent information can be adequate.

The Intensity of Exposure

WHAT was the operation (or operations) in which the patient was engaged? What part did the patient take in carrying out this operation? What other operation was carried out near him and how near? What exposure actually occurred (fume, dust, vapor of molten metal (at what temperature)?) What precautions, if any (ventilation, local or general, use of respirators, care and supervision of men and equipment, housekeeping and sanitation), were being taken to control the exposure? What steps, if any, were being taken to measure the exposure and to observe the clinical status of the workmen? What does the patient know about cases of poisoning that have occurred recently, or at any other time, among men employed in the same kind of work and at the same job he has been performing?

These and other questions will occur to the experienced physician. If their relevance is not apparent to the novice, he need only recognize thereby that he is a novice, and that he has much to learn. Some degree of humility based on such considerations would represent real progress toward the elimination of the medicolegal controversies that now arise.

The History of the Illness

THE most significant clinical characteristic of lead intoxication, generally, is the preponderance of functional over anatomic abnormality, or, stated otherwise, the greater weight of subjective over objective evidences of illness. The most important approach to the diagnosis, therefore, consists in the careful elicitation of the information about past and present complaints, their onset, their development in relation to exposure or discontinuance of exposure and their sequence, intensity and validity. With respect to the last of these factors, one must appraise a patient, as well as a disease, and a story which may have been learned rather than experienced in whole or even in part. There is no rule for this procedure but, by the same token, there is no substitute for careful critical inquiry into the numerous facets of gastrointestinal symptomatology and dysfunction, and for objective appraisal of the results of such inquiry. Strangely enough, the mere mention of an opportunity for occupational exposure to lead seems frequently to be the signal for dispensing with the careful inquiry that is so well recognized as being necessary for the diagnosis of other abdominal complaints, and the glib decision is made that the patient is suffering from lead colic.

There is a highly uncritical attitude of mind to the effect that any doctor knows all about lead colic and can recognize the characteristic pattern of the syndrome without difficulty. The facts are more nearly the exact opposite, in that this, among the many and more common abdominal diseases, is one of which doctors, generally, are unlikely to have knowledge and experience.

The Physical Findings

IN THE most frequent type of lead poisoning, especially in the first episode of intoxication experienced by the individual being examined, the physical findings will be scanty. The evident postural and autonomic nervous signs of abdominal pain exhibit themselves in varying degrees of severity, including those which characterize excruciating agony, subsiding somewhat and then recurring in association with abdominal rigidity or intestinal spasm. The absence of fever, the scanty evidence of abdominal disease in the intervals between bouts of severe colic, and the lack of rebound (peritoneal) pain, suggest functional disorder rather than organic disease, and if there is the blue or black punctate line of sulfide deposit in a gingival margin, in association with a characteristic history of recent weight loss, morning anorexia and constipation, the diagnosis can reasonably be made. Enough has been said elsewhere about the characteristics of the so-called lead line to inspire caution in its recognition.* Suffice it here to say that one should know whereof he sees and speaks before accepting any one of several abnormalities of the teeth and gums as being a "lead line." Experience is required to find and interpret this lesion, and errors of omission and commission in this relationship are all too frequent.

Especially in cases involving prolonged occupational exposure attended by prior recurrent mild or severe symptoms, evidences of neuromuscular disorders are likely to be found, of which well-defined weakness of the extensor muscles of the fingers, wrist and forearm on the most-used side, or on both sides, is much the most common. Frank palsy, with wrist drop, is most rare, if it occurs at all, in an initial bout of lead intoxication, this lesion being an expression of prolonged intense exposure to lead, in the course of which other manifestations of intoxication have usually occurred from time to time. Further signs of neuromuscular involvement, that may go hand in hand with extensor weakness in the upper extremities, are abnormalities of muscle tone, associated with hyper-

reflexia, hypersensitivity of muscles to mechanical stimulation, and maintained muscular contractions. These may involve the lower extremities and, when they do, there are complaints, commonly, of myalgia and arthralgia.

The interpretation of the rarer clinical types of lead poisoning is more difficult and requires careful and critical study of the patient and his exposure. Full coverage of these problems is beyond the scope of this discussion. Perhaps the most valuable advice that could be given in connection with them is that one should proceed slowly, gathering all of the facts, before reaching or expressing a conclusion from which it is difficult and often embarrassing to deviate later. Modern medicolegal procedure is such that reasonable proof of the existence of occupational disease is or should be required. This includes proof that lead has actually been absorbed in quantities compatible with serious manifestations of the disease.

The Clinical Course

ONE of the commonest of clinical mistakes in the diagnosis of occupational lead poisoning stems from the belief of inexperienced clinicians that recovery from lead poisoning is usually slow, and often, if not regularly, incomplete. The facts are precisely opposite. Lead intoxication, except in its more severe and uncommon paralytic and encephalopathic manifestations is an essentially self-limited disease, of comparatively short duration (day to weeks), from which, in the overwhelming proportion of cases, there is complete recovery when the exposure has been terminated. The persistence of abdominal symptoms for many months or even for years after the cessation of exposure, as has been the case not infrequently in controversial situations, represents practically indubitable evidence that the cause of the ailment was not lead absorption. Even an anemia, originally considered to be an expression of the toxic effects of lead absorption, is to be regarded with the greatest skepticism, if it persists indefinitely after the termination of exposure to lead. So far as can be determined by the study of numerous cases of lead intoxication, no irreversible damage to the blood-forming tissues is associated with plumbism. Indeed anemia is not a necessary accompaniment of lead intoxication, and now when legal procedures and current measures of industrial hygiene are reducing the severity of occupational lead poisoning and the likelihood of recurrent and repetitious lead poisoning in the same individual, anemia is a much less important component of the clinical syndrome than it was formerly. Lead poisoning, as seen in the trades which are concerned with inorganic compounds of lead, when recognized promptly and dealt with realistically by removing the individual from exposure and permitting his return to his job only when the exposure has been brought under adequate control, is only rarely a serious disease. Our contrary clinical attitude toward this disease stems largely from earlier clinical descriptions of a neglected disease, which, when it finally came to the attention of a competent clinician, was the expression of an exposure to large quantities of lead over a long period during which there have been recurrent mild or moderately severe episodes of intoxication with brief periods of partial disability. This recurrent, ostensibly mild, but progressively disabling, form of the disease, existent over a period of years of continued exposure to lead, is the so-called "chronic lead poisoning," sometimes in its milder forms misleadingly called "lead absorption," which is so commonly described in medical literature. It is not altogether gone from the American scene, but certainly it is less frequent than it was formerly, being the product, at present, of obviously backward or unduly complacent industrial policy, or of poor industrial medical and hygienic practice.

The Contribution of the Laboratory

COMMENT has been made previously on some of the diagnostic difficulties of the physician engaged in general practice. From the aspect of the laboratory procedures which have come to be almost indispensable features of the diagnosis of lead poisoning for medicolegal purposes, the problem is especially difficult. Few diagnostic laboratories available to such physicians, and, for that matter, few of those available in hospitals and medical centers, generally, are equipped with facilities for the performance of the necessary tests, or with the knowledge and experience required for giving adequate advice on the tests that should be performed, or on the methods required for obtaining materials to be examined. The result is that the tests made in many laboratories are more likely to be misleading than helpful. Therefore the physician will be well advised to dispense with them and to base his judgment entirely on the history of the illness and on the physical signs which he can observe, unless he has the knowledge and experience that enable him to appraise the laboratory and its results.

X-ray examinations in relation to occupational lead poisoning, except in the differential diagnosis of a gastro-enteric disorder, may be dealt with summarily, by pointing out that they have no value as diagnostic procedures in the adult. One is embarrassed not infrequently in medicolegal circles, by the necessity of pointing out that the line of abnormally dense bone which is found from time to time at the margins of rapid growth in the bones of infants and small children, is not a specific result of lead absorption on the one hand, nor a phenomenon of adult life, on the other.

We may also deal briefly with certain of the more common laboratory procedures. The hematologic changes commonly associated with lead poisoning play an important rôle in clinical

diagnosis. The interpretation of the entire blood picture, however, must be made in the full recognition of the fact that all of it is important but that no feature of it gives specific evidence of lead absorption. The commonest fallacy in this connection is the persistent belief that basophilic stippling is indicative of abnormal absorption of lead or of actual lead intoxication. Much could be said of the methods of examination of the blood in this connection, but, without entering upon such a discussion, let it merely be recorded here, regardless of method, that the occurrence of basophilic material in the erythrocytes, per se, has no diagnostic significance, and that there must be some quantitative expression of the extent of this phenomenon in the blood to give it significance. First, one must establish whether or not the phenomenon occurs beyond the physiologic range, and then determine, if possible, the most probable cause of the abnormality, if such is found. Lead absorption is only one, and not the most frequent, of the possible factors. The greatest value of this laboratory test, when properly performed, lies in the fact that failure to detect any abnormality of the blood in this respect argues strongly, indeed, against the likelihood that the illness under consideration is the result of the absorption of inorganic compounds of lead.

Another procedure of the laboratory, around which an excess of enthusiasm has gathered recently, is that of the determination of coproporphyrins in the urine, as a means of demonstrating the occurrence of hazardous absorption of lead. Careful investigation of the metabolism and excretion of the porphyrins under a variety of conditions, including those associated with varying levels of lead absorption, may provide a means for the early detection of actual or impending lead intoxication, and thus add materially to the armamentarium of industrial medicine in the lead trades. Moreover, if the excretion of porphyrins, in the course of a recognized case of lead poisoning, can reveal the severity of the intoxication and give aid in prognosis, it will come to serve a useful purpose. There can be little doubt, however, that the current popularity of the procedure stems largely from the desire of clinical and laboratory workers to substitute some simple diagnostic test for the more precise and therefore more difficult analytical methods that relate specifically to the concentrations of lead in the urine, blood or tissues of persons suspected of being victims of lead intoxication. Let us consider briefly what is likely to be lost by a too enthusiastic yielding to this understandable desire. Quite apart from the methods of analysis of the porphyrins, which while simple are far from foolproof, and with only passing regard for the necessity of preserving the urine against significant increase or decrease in its coproporphyrin content on standing under varying conditions of light, temperature and reaction, we are dealing with a physiologic and pathologic phenomenon of considerable variability, the extent of which has not been established comprehensively in varying states of health and disease, or in relation to age and sex. Certain it is, that this phenomenon is not specifically related to lead absorption, being influenced markedly by liver damage or disease, by intoxications of other types than lead, by a number of infectious diseases, by certain deficiency diseases, and by certain hematopoietic disorders.[4,5,7] That there is an elevation of the rate of urinary excretion of coproporphyrins, in association, more or less regularly, with highly abnormal absorption of lead, is an interesting and important physiologic fact, and that this phenomenon may well be an highly significant aspect of the mechanism of lead intoxication, goes without argument. But that this non-specific phenomenon can contribute an answer to the primary diagnostic question with which the physician is faced seems most improbable. What is this question? If we can make this sufficiently clear for both clinical and medicolegal purposes, we shall by that means define the issue sharply. Medical, legal and economic considerations require and justify a decisive answer to the question: has this patient absorbed a quantity of lead sufficient to cause intoxication? If he has absorbed such quantities of lead, the diagnosis of lead intoxication, arrived at by sound clinical means, may be regarded as adequately substantiated. If he has not, the diagnosis is untenable, and further study of the case is required to arrive at the correct diagnosis.

This brings us naturally to the consideration of the procedures of the laboratory whereby the extent and significance of the lead absorption of the patient can be appraised. The physiologic evidence on which the choice and interpretation of these procedures are based is available[1,2,3] and need not be brought into this discussion, although such evidence must be understood fully by those who would apply it. The methods of sampling and analysis[8,9,10] also have been described in sufficient detail and will not be mentioned, beyond a further warning that there are adequate and inadequate methods and that an high degree of knowledge and skill, far beyond that of the usual type of laboratory technician, is required for the performance of the work.

The concentration of lead in the blood of the person who is suffering from symptoms believed to be those of intoxication by the inorganic compounds* of lead is the most important single bit of evidence required to portray the significance of his absorption of lead. Since the concentration of lead in the blood varies only within extremely narrow limits from hour to hour and from day to day, except under conditions involving concurrent and highly variable exposure

*The concentration of lead in the blood following absorption of tetraethyl lead undergoes little increase, whereas the urinary lead concentration is greatly increased.

to lead, or in response to the administration of dithiopropanol,[12] only one determination would be required for diagnostic purposes, were it not for the possibility of the contamination of a sample of blood with minute quantities of lead in the processes of its withdrawal and analysis. Because of these possibilities it is advisable, even under the best of conditions, to obtain multiple samples, at least in duplicate, and to submit them to separate analysis. The results should check within the very narrow limits of a few micrograms, when expressed in terms of 100 grams of whole blood, if they are to be accepted as reliable.

The determination of the rate of the urinary excretion of lead, when properly carried out, provides specific and invaluable information on the extent and significance of the lead absorption of an individual, and therefore the choice of materials for analysis, in medicolegal cases especially, includes suitable samples of urine. The simplest combination of materials for diagnostic purposes, and one which will serve to eliminate most of the errors of sampling and interpretation, constitutes duplicate samples of blood taken in special equipment by an instructed person, and one or more "spot" samples of urine, each voided directly into the container specifically provided for the purpose under the critical eye of an instructed person. Analytic and physiologic concurrence on the part of these results will stamp them as valid, while variation among them beyond the known ranges of analytic precision or physiologic limitation creates the demand for further investigation.

Any standards adopted or employed for the interpretation of the analytic results must be based upon the known precision of the analytic method or methods employed. Methods of analysis now available will yield results that err on either side within the range of greatest physiologic significance by no more than 10% ordinarily and by no more than 15% at the maximum. So long as reproducible results of known accuracy can be obtained by the method employed, a somewhat larger margin of error can be tolerated, but it is necessary to recognize that the extent of the potential error of the method will determine the precision of the threshold which separates safe from dangerous levels of lead concentration, especially in the blood. For example, the concentration of lead rarely exceeds 60 micrograms per 100 grams in the whole blood of persons who have suffered no recent unusual exposure to lead, but persons who have 80 micrograms or more of lead per 100 grams have been exposed to lead to such an extent that they may have mild symptoms of intoxication. The significance of an analytic error of ± 15%, in dealing with a sample of 10 grams of blood, looms rather large, therefore. It is in the consideration of such potential errors, that duplicate analyses are helpful, and that the great significance of the rate of the urinary excretion of lead, despite its greater physiologic variability, comes into play. On the other hand, it is because of the great physiologic variability in the urinary response to lead absorption, plus the fact that the renal excretion of lead is seriously impaired occasionally, that the analysis of the blood is so necessary.

The concept that the necessities of diagnosis call for the analysis of a sample of urine of large volume, such as that obtained over the period of 24 hours or longer, has been discredited by the availability of analytic methods which provide for the accurate analysis of small samples of blood, and also by the determination of the range of variability of the concentration of lead in small samples of urine under varying conditions of human lead absorption. This is not to say that the analysis of samples of urine of large volume is undesirable, but rather that in some instances, and in many medicolegal situations, it is difficult or impossible to accomplish. The difficulty of obtaining uncontaminated samples of urine of large volume for analysis can be obviated through the use of small samples voided in the presence of the examiner, and at the same time, the misleading effects of the physiologic and pathologic variations in the urinary excretion of lead can be avoided by obtaining and analyzing samples of blood.

It sometimes happens that the diagnosis of lead poisoning is in doubt at the time of the death of the patient. The problem of diagnosis postmortem is not easy, except as it may be made in the negative. Analysis of suitable tissues will serve to determine whether or not there has been potentially dangerous absorption of lead, and, if no such absorption occurred, the diagnosis of fatal or contributory plumbism must be discarded. On the other hand, if the clinical study of the living patient has not yielded information on which to base a diagnosis, it is unlikely that evidence of lead absorption obtained postmortem can justify the diagnosis of fatal lead intoxication, unless further investigation discloses previously unrevealed or misinterpreted facts bearing on the occupational and clinical history of the deceased. This follows from the fact that there is no specific pathologic entity characteristic of lead poisoning and that the diagnosis of lead poisoning cannot be made on evidence of lead absorption alone but on the clinical characteristics of the disease.

For the appraisal of the extent of the absorption of lead on the part of a deceased person, one should not be content with the analysis of one or two tissues. It is advisable to find out the pattern of the distribution of lead in the principal organs and tissues, such as the liver, kidneys, spleen, striated muscle and skeleton. Blood and urine from the bladder, should be obtained for analysis when these are available. The retention of lead in the lungs is important in relation to a recent history of occupational

exposure, and the concentration of lead in the brain is a matter of great importance in the differential diagnosis of a fatal disease involving the brain. It is difficult in most instances to obtain a specimen of long bone for analysis. Fortunately, the analysis of rib serves the purpose adequately, and bone from this source is always procurable. Meticulous care in the collection of samples of tissue for analysis is required, not only to avoid their contamination but to ensure their representative character. Calculation, based on the distribution of lead in the principal tissues of the body, will yield information, which when compared with normal values and those found and recorded, in association with known types and intensities of exposure, will portray the facts as to exposure in a definitive manner.

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TREATMENT OF LEAD POISONING WITH SODIUM CITRATE

Report of Four Cases

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THE PROPER management of lead poisoning continues to be debated. The long-standing question of whether or not the patient should be "de-leaded" is not satisfactorily settled. It is generally conceded that the "de-leading" should not be done in any case until after the manifestations of acute plumbism have passed. The effects of the various forms of therapy remain under discussion, except for the certain benefit of calcium in relieving symptoms of acute lead poisoning. Whether various drugs cause lead storage or increased excretion of the lead is still being debated.

Ash, Fairhall, Minot and Kerniloff,[1] in 1926, reported a slight increase in lead excretion when sodium citrate was administered. The increase was not nearly as great as that produced by the use of ammonium chloride and similar medications, which resulted in a reduction of serum p_H. They attributed the increased lead excretion produced by sodium bicarbonate and by sodium citrate to the alkalinizing effect of these salts and to the fact that the solubility of tri-lead phosphate is increased in both alkaline and acid mediums.

Kety and Letowski[2] have been vigorous in advocating the use of sodium citrate in the treatment of plumbism. They have recommended that 4.0 to 5.0 Gm. of sodium citrate be administered four times daily; in severe cases, they advise that 50 cc. of a 2.5 per cent solution of sodium citrate be injected intravenously. Their rationale for this treatment was based on the concept that lead formed a more soluble, diffusible complex with the citrate ion. Thus more lead was made available

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1. Ash, J. C., Fairhall, L. T., Minot, A. S., and Kerniloff, P.: Lead Poisoning, with a Chapter on the Prevalence of Industrial Lead Poisoning in the United States by Alice Hamilton, Medicine Monographs, Baltimore, Williams & Wilkins Company, 1926, vol. 7, 268 pp.

2. Kety, S. S., and Letowski, T. V.: Treatment of Lead Poisoning by Sodium Citrate, Am. J. M. Sc. 208:407-414, 1943.

for renal excretion and in a less toxic form. They also postulated that the citrate might be metabolized by the liver, leaving the lead residue to be excreted in the bile and thus in the feces. They demonstrated a rather sharp decline in the blood lead levels with this treatment in a series of 15 cases. In another publication, Kety and Letonoff [5] showed that in a series of patients treated with sodium citrate there was a rise in lead excretion when the total of the urinary and the fecal lead was considered. The most marked rise was in the fecal lead. The longest period during which the lead excretion was followed in these cases was 11 days.

Smith,[6] in his discussion of the treatment of lead poisoning, listed sodium citrate as a storage agent, but said that it had only a transitory effect in the one case reported.

With the above considerations in mind, we thought that it would be worth while to report our experience in treating four patients for lead poisoning with sodium citrate as the main component of therapy.

REPORT OF CASES

CASE 1.—*J. S.*, a 24 year old battery factory employee, was admitted to the West Roxbury Veterans Administration Hospital on Oct. 12, 1949 because of abdominal pain of two days' duration. During the three years previous to admission he had been working in the battery

TABLE 1.—*Urinary Lead Concentrations—Case 1, Patient J. S.*

Date	Mg. Lead per Liter of Urine	Mg. Lead per Liter of Urine Converted to Mg. per Liter
[illegible]	0.91	0.91
[illegible]	0.80	0.80
[illegible]	0.80	0.80
[illegible]	0.85	0.85
[illegible]*	0.86	0.86

* On this date the patient was hospitalized.

factory as a reader and paster of the battery plates. On this job, he came in contact with sublead and with lead oxide paste. Over the two years previous to admission he had short bouts of "stomach ache" occurring two times weekly to once monthly and lasting two hours at the most. Two days before admission he had the onset of a dull ache low in the anterior part of the chest. The pain shifted to the abdomen and changed in character to a sharp colicky pain occurring every five to 15 minutes. On the day before admission he had the onset of vomiting without diarrhea and vomited frequently from that time to the time of admission.

Urinary lead concentrations had been determined during his period of service at the battery factory. These are shown in table 1.

Physical examination revealed a rather thin but adequately nourished white man in acute distress with severe abdominal pain. His temperature was 97.4 F., pulse rate, 56; blood pressure, 118/70. There was no scleral icterus. The teeth were in fair repair, and there was no lead line. Heart and lungs were normal. The abdomen was diffusely tender without spasm. The tenderness was maximum in the right lower quadrant of the abdomen. There was questionable rebound tenderness directed toward the left upper quadrant of the abdomen. Liver, kidneys and spleen were not felt. Peristaltic sounds were decreased in frequency. There was no muscle weakness and there were no peripheral sensory changes. The abdominal reflexes were decreased.

Laboratory Data.—The white blood cell count was 9,400, with polymorphonuclear granulocytes 71 per cent. Hemoglobin amounted to 13.5 Gm. per 100 cc. Red blood cells showed

5. Kety, S. S., and Letonoff, T. V.: Treatment of Lead Poisoning with Sodium Citrate, Proc. Soc. Exper. Biol. & Med. 68:474-477, 1941.

6. Smith, P. L.: Effect of Therapeutic Agents in the Treatment of Lead Poisoning, Pennsylvania Department of Labor and Industry, Harrisburg, Pa., June 1941, 17 pp.

intravenously) was 70 per cent in two hours. Renal lead excretion during the first 24 hours in the hospital was 0.48 mg. per liter of urine.[1]

Course in the Hospital—During the first two hospital days the patient was given fluids intravenously to supplement his oral intake and was given meperidine hydrochloride U. S. P. (demerol® hydrochloride) for analgesia. He required 50 mg. of meperidine hydrochloride every three or four hours for relief of pain during this time. Low calcium food and fluids were given as tolerated. He was also given phenobarbital sodium U. S. P. and streptomycin sulfate subcutaneously. On the second hospital day, sodium citrate therapy was instituted, with a dosage of 1.0 Gm. four times daily. He was maintained on a low calcium diet. At this time there was a sharp decrease in his requirement of analgesic medication. His hospitalization from this time was uneventful. He was given sodium citrate, 1.0 Gm. four times daily, for one week. During

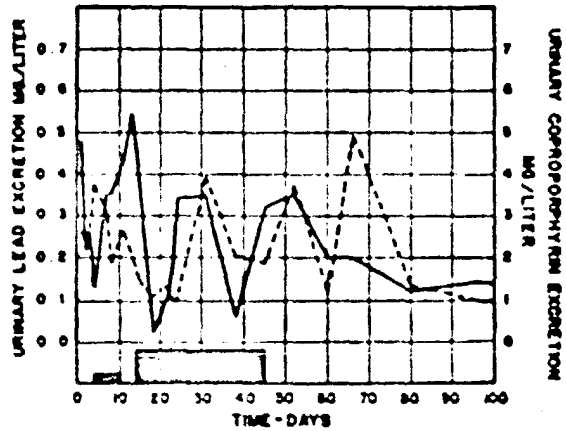

Chart 1 (case 1, patient J. S.).—The solid line represents urinary lead, the broken line, urinary coproporphyrin. The striped blocks represent oral sodium citrate therapy with a low calcium diet and are explained in the text.

this time there was a sharp fall in urinary lead. In a four day period, during which sodium citrate was omitted, the lead excretion rose to a peak of 0.54 mg. per liter of urine. Sodium citrate therapy was reinstituted at 4.0 Gm. four times daily, and again the lead excretion dropped off precipitously. As therapy was continued, the lead excretion gradually rose to 0.34 mg. per liter of urine. From that time the lead excretion except for one instance remained high for a period of approximately 30 days and then fell gradually over a period of about 10 weeks until, on Feb. 13, 1950, the lead excretion was 0.11 mg. per liter of urine. The citrate was given in the higher dosage for a total of 31 days.

The patient was given no antianemic therapy. His hemoglobin remained low, but as the lead excretion began to fall, the hemoglobin began to approach normal levels.

1. Since the urine specimens collected were spot samples, all values were adjusted to a mean specific gravity of 1.024 (Levine, L., and Fahy, J. P.: Evaluation of Urinary Lead Determinations: I. The Significance of the Specific Gravity, J. Indust. Hyg. & Toxicol. 27:217-223, 1945).

There was a slight rise in carbon dioxide-combining power from 55.1 to 63 volumes per cent as sodium citrate therapy was reinstituted. This returned to 56 volumes per cent three days later.

The urinary lead and urinary coproporphyrin levels and their relation to therapy are shown graphically in chart 1.

Case 2.—C. A., a 22 year old battery plant employee, was admitted to the West Roxbury Veterans Administration Hospital on Oct. 18, 1940 because of muscular aches and pains of six days' duration and abdominal cramps of five days' duration. The patient had been working in the battery factory for three months. His work consisted of handling the lead plates after they were removed from a furnace and packing them together. Several weeks before admission he noted the onset of anorexia. Six days before admission he noted the onset of aches and pains in his arms and back, and later he noted pains in his legs and thighs. Five days before admission he had the onset of cramps in his abdomen. These were located in the region of the umbilicus. They increased in severity until they were disabling. The company physician made a diagnosis of lead poisoning, and he was referred to the hospital for therapy.

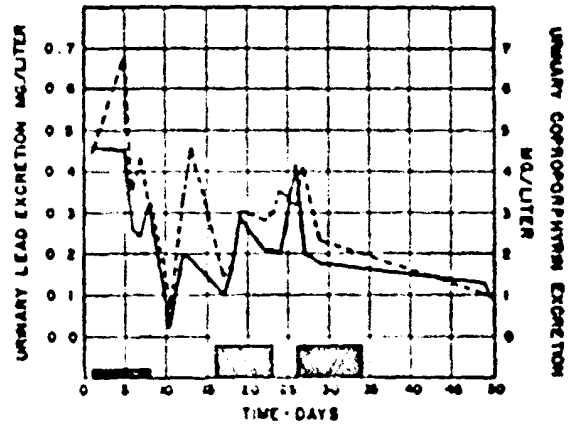

Chart 2 (case 2, patient C. A.).—The solid line represents urinary lead, the broken line, urinary coproporphyrin. The striped blocks represent oral sodium citrate therapy with a low calcium diet, the cross hatched block, sodium bicarbonate with a low calcium diet. Further explanation is given in the text.

Physical examination revealed a young adult white man who was in no distress at the time of admission but soon thereafter was having severe abdominal cramps. He was slightly pale and yellow in appearance. His temperature was 98 F., pulse rate, 72, blood pressure, 140/78. The teeth were in fair condition, and there was no lead line. There was an occasional high-pitched rhonchus over the left lung posteriorly. There was a grade 3 systolic murmur over the precordium with a split pulmonic second sound. The abdomen showed moderate generalized voluntary rigidity without localized tenderness. Liver, kidneys and spleen were not felt. There was perhaps slight weakness of dorsiflexion of both wrists, but there were no other neurological signs.

Laboratory Data.—The white blood cell count was 9,400 with polymorphonuclear granulocytes 68 per cent. Hemoglobin amounted to 8.5 Gm. per 100 cc. The red blood cells showed basophilic stippling (stippled cell count, 18 per cent). The urine had a specific gravity of 1.015, no albumin or sugar, 4 to 6 white blood cells per high power field, and a lead content of

0.46 mg.[4] Serum bilirubin totaled 2.2 mg. per 100 cc. An osmotic fragility test showed hemolysis to begin at 0.48 per cent sodium chloride and to be complete at 0.32 per cent. This is interpreted as normal.

Roentgen examination of the chest showed no evidence of an active pulmonary change.

The urinary lead and urinary coproporphyrin levels and their relation to the therapeutic program are shown graphically in chart 2.

Course in the Hospital.—Soon after admission the patient was in acute distress. He was given meperidine hydrochloride U. S. P., 100 mg. subcutaneously, as needed for pain. He was also given atropine sulfate, 0.4 mg. subcutaneously every six hours, and his fluid intake was supplemented with intravenous dextrose and saline solution. Moderate amounts of barbiturates were given for sedation. Sodium citrate was started in a dosage of 1.0 Gm. four times daily on the second hospital day. He was given a low calcium diet as tolerated. The patient remained

TABLE 2—*Laboratory Data—Case 1, Patient S. O.*

	Chemistry	
[illegible]	Cephalin-cholesterol flocculation test	[illegible]
	Prothrombin time	[illegible]
	Van den Bergh test for bilirubin in serum	[illegible] mg. per 100 cc.
	Nonprotein nitrogen (blood)	[illegible] mg. per 100 cc.
	Calcium (serum)	[illegible] mg. per 100 cc.
	Phosphorus (serum)	[illegible] mg. per 100 cc., [illegible]
	Phosphatase (serum or plasma)	[illegible] Bodansky units
[illegible]	Sodium (serum)	[illegible] mEq./L.
	Chlorides (as chloride ion, plasma or serum)	[illegible] mEq./L.
	Carbon dioxide content (venous)	[illegible] mEq./L.
[illegible]	[illegible] test	[illegible] Ehrlich units
	Urine	
[illegible]	Color: Clear yellow. Reaction: [illegible]. Specific gravity: [illegible]. Albumin: 0. Sugar: 0. Bile: 0	
	Blood	
[illegible]	RBC [illegible] million. WBC [illegible]. Hgb. [illegible] Gm. Polymorphonuclears [illegible]%. Large lymphocytes, small lymphocytes [illegible]%. Monocytes [illegible]%. Eosinophils [illegible]%. Platelets: adequate. Stained specimen: [illegible]. Hematocrit: [illegible]%. Supernatant serum: [illegible]. Mean corpuscular volume [illegible]. Mean corpuscular hgb. content [illegible]. Mean corpuscular hgb. concentration [illegible]%. Color index [illegible]	
[illegible]	[illegible] count [illegible]. Hgb. [illegible] Gm. Basophilic stippling	
[illegible]	Hgb. [illegible] Gm.	

uncomfortable with abdominal pain and vomiting and required meperidine hydrochloride for pain even on the fifth hospital day. He was free of symptoms on the sixth hospital day and remained so for the rest of the period of observation.

During the period of observation he was given a low calcium diet for seven days, then a regular diet for seven days, then a low calcium diet for 19 days. After he had received sodium citrate, 4.0 Gm. per day for seven days, therapy was omitted for seven days. Then he was given sodium bicarbonate, 4.0 Gm. four times daily, for nine days. It is difficult to relate the changes in his lead excretion to the therapeutic program. Within two months the lead excretion came to within normal limits, and on Feb. 18, 1950 the urinary lead was 0.07 mg. per liter. Also, during this time the hemoglobin rose to 13 Gm.

CASE 2.—S. O. was a 25 year old white man who during the four months prior to admission worked as a grinder in an automobile assembly plant where lead solder is used to finish seams. Because of an increase in production, the patient was working with a rotary disk of greatly increased speed. This resulted in nine consecutive hours of steady inhalation of lead dust. Prior to this man's illness no protective equipment had been supplied, but subsequently the work was enclosed in a booth and outside air provided to the men, who were required to wear respirators constantly.

S. O. was admitted to the hospital for care because of jaundice and abdominal pain of one week's duration, headache and dizziness occurring intermittently for three weeks, and easy fatigue, weakness, irritability and disturbance of bowel habits during the previous two months. At the onset of his symptoms the patient thought the speed up in his work was responsible. He did not seek medical help until his fellow workers told him he looked yellow.

His medical history was without incident, and previous occupations exposed him to no known toxic materials.

Physical examination on admission revealed pronounced jaundice, especially intense in the sclerae and on the abdomen. The patient was obviously uncomfortable but not acutely ill. There was a lead line about the inner margin of the bicuspids. There was an obvious tremor of the tongue. Otherwise there were no unusual findings.

The laboratory data collected are listed in table 2.

Sternal marrow showed a marked erythroid ratio of 1:1 with abundant normoblasts and basophilic stippling of marrow cells, indicating a nonspecific marrow response to the destruction of red cells caused by lead intoxication. Special fragility studies done on this patient's blood

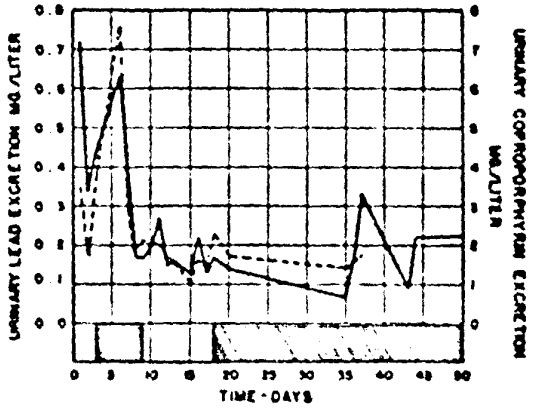

Chart 3 (case 3, patient S. O.).—The solid line represents urinary lead, the broken line, urinary coproporphyrin. The striped blocks represent oral sodium citrate therapy with a low calcium diet and are explained in the text.

by Dr. J. W. Harris of the Thorndike Memorial Laboratory, revealed normal mechanical fragility. Further study by Dr. Harris showed the Crosby[5a] test to be negative; tests of cold agglutinins, L. E. phenomenon, slightly hypochromia. Tests of the patient's blood in salt solution done at the Massachusetts General Hospital showed the red blood cells to be of normal osmotic fragility at the time tested.

Roentgen study demonstrated no lung changes and no enlargement of liver or spleen.

The patient was given sodium citrate by mouth in doses of 5 Gm. four times a day and a low calcium diet. His subjective response was remarkable, and chart 3 indicates the renal excretion of lead and coproporphyrin which accompanied this program. After this therapy had been carried on for a week, it was reported, and 24 hours later the patient complained of the headache and abdominal pain which he had reported on admission. It is apparent that during

[5a] Crosby, R. R. A.; Moureau, A. F., and Race, R. R.: In-Vivo Demonstration of Red Cells in Babies with Hemolytic Disease, Lancet 1:294-296 (Feb. 23) 1946. Diamond, L. K., and Allen, F. H., Jr.: Medical Progress: Rh and Other Blood Groups, New England J. Med. 241:867-873 (Dec. 1) and 907-914 (Dec. 8) 1949.

this control period more lead and less coproporphyrin were excreted. After the relationship between the intake of sodium citrate with a low calcium diet and the levels of urinary lead and coproporphyrin with accompanying abdominal pain and headache had been demonstrated, the patient was discharged on citrate therapy. The jaundice and the lead line gradually disappeared. It was necessary to administer ferrous sulfate in doses of 0.2[illegible] Gm. three times a day to restore the hemoglobin to an adequate level. The patient returned to work not involving lead exposure and has remained entirely well. The gradual loss of lead from the body by excretion is indicated in the chart of urinary lead levels (chart 3). In this case, whatever the mechanism, the administration of sodium citrate accompanying a low calcium intake appeared to be of real merit.

Case 4.—C. S., a 45 year old white man of Polish descent, worked as a pyrometer man in a steel plant for the eight years prior to his hospitalization. As part of his job, he supervised baths of molten lead through which steel wire passed for annealing. From the amount of lead in the air (ranging from 0.3 to 2.3 mg. of lead per 10 cubic meters) and the amount of lead in the urine of C. S. and his fellow workers (0.12 to 0.36 mg. of lead per liter) it is certain that

Table 3.—Laboratory Data—Case 4, Patient C. S.

	Urine	
[illegible]	Color: Clear yellow; Reaction: [illegible]; Specific gravity: [illegible]; Albumin: 0; Sugar: 0; Bile: 0; Rare WBC; No casts, no RBC	
[illegible]	[illegible] mg. lead per liter; [illegible] mg. lead per liter adjusted to sp. gr. [illegible]	
	Blood	
[illegible]	RBC [illegible] million; WBC [illegible]; Hgb. [illegible]; Polymorphonuclears [illegible]%; Large lymphocytes [illegible]%; Small lymphocytes [illegible]%; Monocytes 0; Eosinophils [illegible]%; Basophils [illegible]%; Platelets adequate; RBC hypochromic, anisocytic, [illegible]% with basophilic stippling; [illegible]	
	Chemistry	
[illegible]	Calcium (serum)	[illegible] mg. per 100 cc.
	Phosphorus (serum)	[illegible] mg. per 100 cc.
	Alkaline phosphatase (serum)	[illegible] Bodansky units per 100 cc.
	Total protein (serum)	[illegible] Gm. per 100 cc.
	Albumin globulin ratio	[illegible]
	Albumin (serum)	[illegible] Gm. per 100 cc.
	Globulin (serum)	[illegible] Gm. per 100 cc.
	Nonprotein nitrogen (blood)	[illegible] mg. per 100 cc.
[illegible]	Chlorides (as chloride ion, serum or plasma)	[illegible] mEq./L.

this patient absorbed lead during this job exposure. Of the group studied, 15 men doing comparable work, C. S. was the only one who developed symptoms.

His past medical history revealed childhood scarlet fever without known sequelae. In 1944 (five years before admission) C. S. was treated for brucellosis with several courses of sulfadiazine. He had drunk milk drawn from one of his own cows that had aborted. He complained of fever of one month's duration, numbness in the head, generalized bone and joint pain. His Brucella agglutination titer was 1:[illegible]. After two months he was apparently well.

Two years before admission C. S. began to suffer from pain in the lower right quadrant of the abdomen, extending into the groin. One physician suspected appendicitis, but appendectomy was not performed. The patient was admitted to another hospital after a period of increasing abdominal pain, colicky in character, and vomiting. A diagnosis of lead poisoning was made, and the patient responded well to calcium administered by vein and by mouth. However, he continued to complain of weakness of muscles and did not return to work. Because his abdominal pain returned, plus headache as well as persistent weakness, he returned 10 months later to this same hospital for eight days, during which he was treated with dimercaprol injection U. S. P. (BAL in oil, 2,3-dimercapto-propanol in oil) with considerable symptomatic relief. When he returned home, he felt improved but did not go back to work at the steel mill.

His family physician gave C. S. calcium by vein and by mouth for four months after he left the hospital. After an interval of reasonably good health, the patient again complained of

headache, weakness and abdominal cramps. Since calcium in large doses (up to 40 to 90 grains [4 to 6 Gm.] daily orally plus 10 cc. of 10 per cent calcium gluconate intravenously three times a week) failed to relieve his symptoms, he was admitted to the Massachusetts General Hospital for medical management.

Physical examination on admission revealed a husky male complaining bitterly of headache and abdominal pain. Except for tenderness on palpation of the abdomen and posterior neck muscles, there were no abnormal physical findings. Careful neurological examination brought out no palsy. The patient walked awkwardly and insisted he could not lift heavy objects or perform simple jobs, such as maneuvering a mat. These complaints were hard to evaluate, since C. S. had been sick for two years and had many hostile reactions to the failure of treatment to effect complete cure. He was receiving compensation, which undoubtedly complicated the evaluation of subjective complaints. Of great interest was the discovery that the patient's drinking water supply on his farm contained amounts of lead above the permissible level (0.36 p.p.m., safe level considered to be 0.10 p.p.m.). Further, his two sons were excreting 0.28 and 0.13 mg. of lead per liter of urine, indicating considerable intake above the level considered

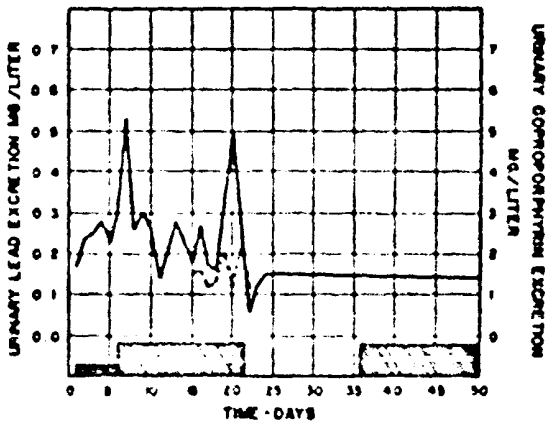

Chart 4 (case 4, patient C. S.).—The solid line represents urinary lead, the broken line, urinary coproporphyrin. The striped blocks represent oral sodium citrate therapy with a low calcium diet and are explained in the text.

normal, 0.00 to 0.08 mg. of lead per liter. The chronicity of this man's course and his failure to stay well after earlier response to calcium and dimercaprol therapy were explained by his continual intake of lead in his drinking water plus the lead he may have stored and not completely excreted during his seven years' working in the steel plant.

By giving sodium citrate in doses of 4 Gm. four times per day by mouth, the symptoms were controlled. Earlier doses of 2 Gm. three times per day were not sufficient and attempts sulfate, barbiturates and even codeine sulfate had to be used to keep the patient comfortable. During the sodium citrate therapy, C. S. was restricted to a low calcium diet. When the sodium citrate therapy was stopped, symptoms reappeared promptly but in view of this man's reaction to his long illness, it was difficult to interpret this response. After his return home, the patient was persuaded to change the pipes delivering drinking water from his well to prevent further lead absorption. His family physician continued the sodium citrate therapy for a matter of weeks. Renal lead excretion continued at the levels shown in chart 4. C. S. continued to complain of inability to work and of vague flutting and pains, but he no longer

had headache or abdominal pain. Many of his persistent subjective complaints probably stemmed from the medicolegal aspects of this case, which had not been settled.

Considerable laboratory work was done, and the data are listed in table 3.

The urinary studies done are shown in chart 4, which gives data on lead, coproporphyrin and therapy. It may be said that C. S. was absorbing, storing and excreting sufficient lead to substantiate the diagnosis of chronic lead intoxication. The correlation of the findings, the complaints and the treatment is not clear.

COMMENT

In three of the cases presented here there was a rather striking relief of symptoms coincident with sodium citrate therapy.

In the case of J. S. (case 1) it seems that renal lead excretion was suppressed by sodium citrate therapy. Perhaps this is the storage effect of sodium citrate suggested by Smith.[6] Perhaps this effect is produced by changes in acid-base pattern as evidenced by the temporary rise in carbon dioxide–combining power which occurred when the sodium citrate therapy was reinstituted. Conclusions may not be drawn from the data at hand, but we think that these suggestions merit further investigation. It is interesting to note that urinary lead increased during the period in which sodium citrate therapy was omitted. Urinary lead also increased after the initial fall occurring at the time therapy was reinstituted.

There is no convincing evidence in the data here presented that the excretion of lead was hastened in cases 1, 2 and 4 by the sodium citrate therapy. Both the prolonged abnormal urinary lead concentration and the slow rise of the hemoglobin to adequate levels we take as evidence that there was persistence of abnormal amounts of lead in these patients.

In case 3 the data presented and the clinical history give reasonable evidence that the sodium citrate therapy was useful in hastening lead excretion and prompt relief of symptoms. Although this man's exposure was relatively short, it was intense, and it is certain that considerable lead was stored in his lungs and absorbed from there into his circulation for some time after his exposure ceased. The prolonging of sodium citrate therapy for a matter of weeks after this man's initial acute lead poisoning was controlled perhaps enabled the remaining lead to be handled in a manner to prevent exacerbation of symptoms. Since S. O. was not allowed to return to work involving lead exposure, his lead intoxication might well have followed a benign clinical course without medication.

The problem of C. S. (case 4) is complicated owing to his long-continued intake of lead at his job and at home. As in case 3, this patient, if free from lead exposure, might have excreted comparable amounts of lead without the use of sodium citrate. However, there can be no doubt that the control of his symptoms—and in the case of C. S. this was important—was achieved by sufficiently large oral doses of sodium citrate.

The use of dimercaprol (BAL) in the case of C. S. is hard to evaluate, because the patient continued to take in small doses of lead from his drinking water supply after leaving the hospital. We were led to review the available literature and present the following summarizing remarks.

Although dimercaprol is most effective in the treatment of poisoning caused by certain heavy metals, such as arsenic, gold and antimony, there is, with one excep-

tion, no convincing evidence that it is of value in the treatment of lead poisoning.[6] Telfer[7] reported one case of lead poisoning treated with BAL. Although the worker was excreting normal amounts of lead after receiving intravenous injections of calcium gluconate for three days, the urinary lead rose to high levels after a series of dimercaprol injections.

We have been much interested in the suggestion that urinary coproporphyrins be used as an index of lead intoxication. Increased coproporphyrinuria was found during acute illness in all four of the cases presented in this paper and well before the acute episode in at least two of the cases. In all cases the urinary lead and coproporphyrin values showed relatively good correlation. It should also be mentioned that in all cases the sign of coproporphyrinuria was seen much earlier than the stippling of the red blood cells. In the case of S. O. there was a great increase in coproporphyrin excretion, accompanied by an absence of stippled red blood cells in the peripheral blood. On the fourth hospital day, stippled red blood cells were found in the bone marrow smear but not in the peripheral blood. On the sixth hospital day, stippled red blood cells appeared for the first time in the peripheral blood. From then on there was a gradual increase in the stippled cell count, while both urinary lead and urinary coproporphyrin values showed a downward trend. In the cases of C. A. and J. S. the coproporphyrin excretions were elevated well before the symptoms developed, and during this time stippled cell counts were normal. In the case of C. S., who had lead poisoning over a long period, both the excretion of coproporphyrin and the stippling of red cells were increased.

In postulating the mechanism of this increased coproporphyrinuria in plumbism, it appears from studying these four cases that it is entirely possible for the mechanism to vary in different cases. It is believed at this time that in those acute poisonings which develop over the relatively short period of several months and which are accompanied by anemia and increased icterus, the mechanism is due to the destruction of the red blood cells, which leads to a release of protoporphyrin, which is then converted into coproporphyrin in the liver and excreted as such in the urine.[8] Aub[9] and his associates have already described the mechanism of

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6. Carmody, F. G., Jr., and Eagle, H.: Efficacy of BAL (2,3 Dimercaptopropanol) in Treatment of Experimental Lead Poisoning in Rabbits, J. Pharmacol. & Exper. Therap. 88: 247-450, 1946. Longacre, W. T., and Luetscher, J. A., Jr.: Use of BAL in the Treatment of Injurious Effects of Arsenic, Mercury and Other Metallic Poisons, Ann. Int. Med. 21:545-714 (Oct.) 1944. Braun, H. A.; Lusby, L. M., and Calvery, H. O.: Efficacy of 2,3-Dimercaptopropanol (BAL) in Therapy of Poisoning by Compounds of Antimony, Bismuth, Chromium, Mercury and Nickel, J. Pharmacol. & Exper. Therap. (supp.) 87:119-125, 1946. Lusby, L. M., Braun, H. A., and Long, E. P.: Effect of BAL on Experimental Lead, Tungsten, Vanadium, Uranium, Copper and Copper Arsenic Poisoning, J. Indust. Hyg. & Toxicol. 28: (sec. 1) 301-305, 1946. Ryder, H. W., and Kehoe, R. A.: Effects of Dithiopropanol (BAL) on Human Lead Intoxication, J. Lab. & Clin. Med. 32:1423, 1947. Ryder, H. W., Chalek, J., and Kehoe, R. A.: Influence of Dithiopropanol (BAL) on Human Lead Metabolism, Science 106: (no. 2742) 63, 1947.

7. Telfer, J. G.: Use of BAL in Lead Poisoning, J. A. M. A. 134:835-837 (Nov. 29) 1947; correction 136:50 (Jan. 1) 1948.

8. (a) Garrod, A. E.: The Bradshaw Lecture on the Urinary Pigments in Their Pathological Aspects, Lancet 2:1123, 1900. (b) Watson, C. J.: Concerning the Naturally Occurring Porphyrins. IV. The Urinary Porphyrin in Lead Poisoning as Contrasted with That Excreted Normally and in Other Diseases, J. Clin. Investigation 15:327, 1936.

anemia in plumbism. His explanation of the "shattering" of the red blood cells fits in well with the above theory.

However, there are many cases in which the exposure has continued over many years, and there is only slight anemia and no icterus present. It is believed that in such cases Rimington's[9] theory that lead may be acting as a block between protoporphyrin and iron to prevent their normal combination in the formation of hemoglobin might be a plausible explanation of this increased coproporphyrinuria.

Following this line of postulation, we believe that in the cases of S. O. and C. A. coproporphyrinuria increased because red blood cells were being destroyed by lead with resultant release of protoporphyrin, which was converted into coproporphyrin and excreted. In the case of C. S. who had chronic lead poisoning and who had been taking in large amounts of lead for many years, it is believed that the increase of coproporphyrin took place in the bone marrow and might possibly have been due to a blocking by lead during the synthesis of hemoglobin. The case of J. S. also seems to have a somewhat similar mechanism as the case of C. S.

However, as was stated previously, although many theories have been set forth, the actual mechanism of this increased coproporphyrinuria is not clearly understood. It does appear, however, in reviewing these four cases, that rather than insisting that there must be only one mechanism common to all cases of plumbism, it is wiser to consider the possibility that two mechanisms may be present, especially in view of the fact that both the clinical and the laboratory findings do vary from case to case.

While the mechanism of the coproporphyrinuria of plumbism is not definitely known, it is known that an increased excretion is seen in lead workers absorbing large amounts of lead and also in cases of acute and chronic lead poisoning.[10] It has recently been suggested that the finding of coproporphyrinuria may be a valuable aid in the prevention or early diagnosis of plumbism.[11] Kluver[12] has found the coproporphyrins to be normally present in the white matter of the central nervous system of animals, including man. This has led to speculation that the neuritic pain, headache, encephalopathy and abdominal colic present in plumbism may be explained by the coproporphyrins deposited in the tissues.

We should like to explore the effect of citric acid, as opposed to that of sodium citrate, on lead excretion in acute plumbism. We should like to determine the effect of the citrate ion as opposed to the alkalinizing effect of sodium citrate. Until such observations are complete, freedom from lead exposure, if attainable, and calcium therapy where needed, are dependable measures. Since it is not always

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9 Rimington, C. Comptes rendus d. trav. du lab. Carlsberg, serie chim. 22:454, 1938; The Significance of Urinary Coproporphyrins, editorial. J. A. M. A. 130:627 (Feb. 20) 1946.

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possible to achieve the ideal of no lead exposure, it is pertinent to continue to explore methods by which to form compounds of lead in the body which are less toxic than the lead ion and which hasten lead excretion.

SUMMARY

Four cases of lead poisoning, acute in three and chronic in one, in which the patients have been treated with sodium citrate, are presented. We believe the evidence accumulated leads to the following opinions:

1. Adequate oral doses of sodium citrate will control the symptoms of lead poisoning.

2. Studies of the lead excreted in the urine in these four cases—we lack studies of lead excreted in the feces—do not warrant the conclusion that sodium citrate increases lead excretion.

3. Studies of urinary coproporphyrin are presented.

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The writers are grateful to Drs. C. H. Dunton and B. F. McKernon for making possible the study of their patients, S. O. and C. S., respectively. The writers are also indebted to R. P. W. Rumsby and G. W. Boylen, of the chemical staff of the Massachusetts Division of Occupational Hygiene, for performing the lead analyses.

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Industrial Hygiene At American Smelting and Refining Company

by John N. Aberson and K. W. Nelson

INDUSTRIAL hygiene has been defined by Patty[1] as "the science and art of recognizing, evaluating, and controlling potentially harmful factors in the industrial environment." This definition implies thorough study of operations, evaluation of potentially harmful factors through air sampling, microanalyses and other means, and, finally, appropriate medical and engineering control wherever indicated.

The prevention of industrial health injuries is a vital part of operations of American industry today. Progress and interest in this field has increased steadily for many years, the most rapid progress having been attained, perhaps, during the last three decades. It is significant to note that there are now official agencies in 46 states actively concerned with industrial health problems and that a western field station has been established recently in Salt Lake City by the U. S. Public Health Service to augment its industrial hygiene services directed from headquarters of the National Institute of Health, Bethesda, Md. Many of the larger industries have found it advantageous to establish their own industrial hygiene departments.

The American Smelting and Refining Co. is a world-wide organization engaged in the mining, smelting, and refining of lead, copper, zinc, silver, gold, by-product metals, including cadmium, arsenic, and others. In the United States there are 13 smelters and refineries, 11 secondary smelters or foundries, and a number of mines. Approximately 9000 workers are normally employed.

It has long been the established company policy to seek out occupational hazards and provide safeguards for employee health. Protective equipment has been supplied to individual workers and exhaust ventilation installations have been in use in some operations for more than 40 years. All of the major units have their own medical departments which provide employees with excellent medical and hospital care.

In 1937 full scale industrial hygiene studies were undertaken at the Selby Plant and were extended to most of the other smelters during the next three years. In 1943 the Department of Hygiene was organized with Professor Philip Drinker of Harvard University as Director and with Dr. S. S. Pinto as Medical Director. The department is responsible for coordinating and maintaining a program for the good health of all employees from top management down to the lowest paid day worker. It is essentially a service organization serving all of the United States plants regardless of location or size. Full and part-time physicians employed in all of the company's American plants are working in close cooperation with the Medical Director are responsible for determining the state of health of all the employees and giving treatment when necessary. In general, medical care is confined to accidents or illnesses occurring while the men are on the job. Among the duties of the doctors is the making of careful physical examinations of new employees and routine check-ups of old employees.

In addition to medical care a primary responsibility of the department is the prevention of occupational illnesses. In this the main concern is with the working environment in relation to its effect on the worker. Environmental factors may be dusts, fumes, gases, toxic materials, heat, humidity, radiation, or noise. The objectives are: (1) immediate control of these factors through the education of the worker, through providing the wearing of respirators or other protective devices, and through careful medical examinations and regular analysis of urine specimens; (2) a long range control program which may be accomplished by local exhaust ventilation, wetting of materials, changes in metallurgy, changes in methods of handling, or by use of special devices and special equipment.

To accomplish these objectives a fine industrial hygiene laboratory was built in Salt Lake City and equipped to do routine and experimental work. Trained and experienced industrial hygienists obtain the facts by making frequent hygiene surveys. These surveys include tests of the air, studies of all processes, and careful investigation of ventilation, lighting, and general working conditions. Except in emergencies, the air contaminants and often the substances handled by the worker are sent to the laboratory for analysis by chemists and technicians specially trained in industrial hygiene methods. The findings are evaluated in terms of limits recommended by various State and Federal agencies, and in light of all available medical data.

The methods used for studying the working environment involve all of the usual chemical and physical procedures employed in industrial hygiene. The impinger, electric precipitator, thermal precipitator, and filter paper sampler have been used to collect atmospheric dust and fume samples. Of special interest here is the filter paper sampler, shown in Fig. 1, which was developed by Dr. Silverman at Harvard University. The instrument has been improved and is used very extensively in field studies. A water manometer connected behind an orifice is used to determine the rate of air flow. Calibration is effected by use of a standard gas meter or rotameter. The dust or fume is collected on a filter paper clamped between two rings, as shown in Fig. 2. The filter paper, such as Whatman No. 52, collects both dust and fume with a very high efficiency. The instrument is very convenient and easily transported.

The solids collected on the filter paper are analyzed in the laboratory usually by use of a polarographic procedure. By this procedure it is possible to measure quantitatively in a single analysis the

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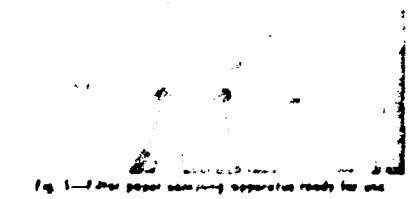

Fig. 1—[illegible] sampling apparatus ready for use

copper, lead, cadmium, and zinc in each sample. Polarographic methods are also used for the determination of a number of other elements.

Dusts of various types are the most common contaminants found in the atmosphere of the various plants. Fumes containing lead oxide may be found in certain smelter and refinery work areas and are given special attention since lead oxide is more readily absorbed than other less soluble lead compounds such as the sulphide. Other contaminants such as carbon monoxide, hydrogen sulphide, sulphur dioxide, selenium, tellurium, cadmium, and arsenic may be found and require study. Certain of the gases mentioned may be toxic or irritant. In order to be constantly on the alert and to be warned in advance, rapid field tests have been developed for hydrogen sulphide, chlorine, and arsine from a basic British method. In this method a small hand pump is used to draw air through filter paper disks impregnated with various reagents which produce a stain if traces of gas are present, see Fig. 3. These stains are compared with standards to estimate the gas concentrations. By use of this method chemists and safety directors are able to check the working environment for suspected dangers. For one plant where hydrogen sulphide is being used in one of its operations, a machine is being developed to sound automatically an alarm before any escaping gas reaches dangerous concentrations.

From time to time certain analyses requiring highly specialized training or very expensive equipment are performed by the research laboratory or outside laboratories; for example, spectrographic and petrographic analyses. Certain types of research work involving animal experimentation have been performed through grants to Harvard University.

As a part of the fact-finding function of the Hygiene Department, urine specimens and frequently blood specimens are obtained from all workers exposed to lead and other metals and examined at

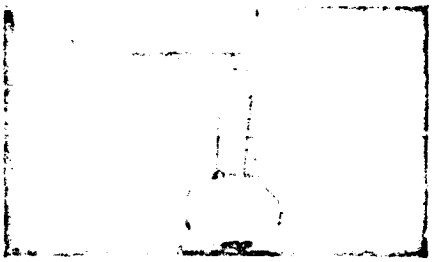

Fig. 2—[illegible] apparatus [illegible]

[illegible] the basis of these and other medical findings, the Medical Director may make recommendations for immediate protection of the employee.

The evaluation of air findings is an important function of the Hygiene Department. This involves in part an appraisal of air studies and the correlation of these studies with medical data. When all the facts are brought together, an overall evaluation of the environment is made, the need for control measures determined, and a coordinated control program outlined. Recommendations for control are made to the plant manager and passed on to engineers especially trained in industrial hygiene ventilation. The plant manager and the superintendent with his operating staff decide what method of control should be carried out. This is logical because control may be accomplished by changes

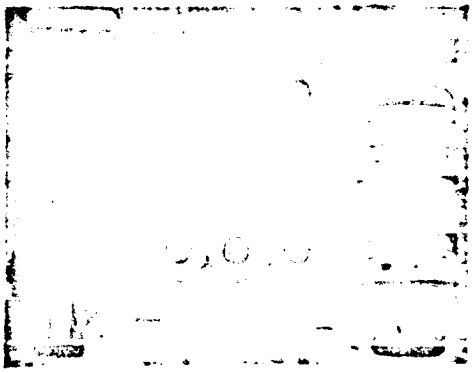

Fig. 3—Sampling pumps and standard stains for field determinations of hydrogen sulphide and chlorine.

in metallurgy or methods of handling rather than by local exhaust ventilation or other engineering or chemical means. Occasionally control is not possible by engineering means and it is necessary to rely on respiratory protective devices and medical control procedures. Sometimes the recommendations for plant improvements are based on comfort alone rather than a health hazard.

The ventilation engineers form a unit or a division of the Western Engineering Department but work in the closest cooperation with the Hygiene Department in carrying out the detailed recommendations. Frequent conferences are held with these engineers and general plans are approved.

Plant Improvements

Elimination or control of all potentially harmful factors in the environment is the major long-range objective. Since 1938, and particularly during the past four years, much progress has been made toward this objective. In some cases such as in the mills, the cost has been very high, from ten to twenty thousand dollars per worker. Following are some of the highlights of control of dusts, fumes, and gases in the various plants achieved through the combined interest and effort of managers, superintendents, metallurgists, foremen, the specially trained staff of ventilation engineers of the Western

[illegible] Engineering Department and the Department of [illegible]

[illegible] have had to be used and chiefly local exhaust ventilation and dust extraction by small bag-house units and centrifugal impingement washers. Because of the very high cost, experimental work has been conducted to find other methods. Mist-sprays produced by atomizing water with compressed air have been tried. The minute droplets of water produced by these sprays actually wet dust particles and help to reduce the escape of dusts at the source, yet they avoid objectionable wetting of the flowing material. With this method of control a 50 per reduction in dust concentrations has been achieved in one mill.

In certain instances protection of the worker has been accomplished by ventilating and air conditioning small areas where men work, such as crane cabs, when the general ventilation of the room would be difficult if not impossible, see Stevenson.[1] In other words, the man has been "ventilated" instead of the operation. Cabs of cranes handling ores, carrying hot metals over furnaces, etc., are examples. This approach is being tried now in mills by building booths kept under positive pressure with filtered air for workers whose duties can be carried out from such an enclosure.

Sampling Rooms Local exhaust ventilation and good housekeeping have been used exclusively to control dusts in sampling rooms, see Fig 4. Modern grinding and sampling equipment are easily enclosed and the dust can be controlled at a very moderate cost.

Beds and Charge Bins Wetting, enclosure and local exhaust ventilation are being used for control of dusts in such equipment. Dust control in these departments is one of the most difficult problems. Wetting can only be used to a limited degree because excess moisture may affect the roasting of certain types of charges.

The unloading of truckloads of ores and concentrates for storage or making up charges was a difficult problem to control in one plant. The final solution was found in a semi-enclosure and hood with high volume air intake.

Fig 4—One sample being prepared at bucking table with use of exhaust ventilated hood

Fig 5—Suction removing wetted dust from wind boxes of a first-over Dwight-Lloyd sinter machine

Dwight-Lloyd Sinter Plants Wetting, enclosure and local exhaust ventilation have been the principal methods of controlling dust and fume in the Dwight-Lloyd sinter plants. Water may be added to both first and second-over charges at the belt tripper, at the mixing tables and sometimes with use of pug-mills. Water is used to quench the sinter after it is roasted and the discharge ends of the machines are kept tightly enclosed.

The control of dusts from crushing first-over sinter has been effected with local exhaust ventilation and collection of dusts with centrifugal impingement washers.

The dustiest Dwight-Lloyd operation is the removal of deposits from wind boxes. Control of these dusts has been effected by (1) use of enclosed pan or screw conveyor along wind boxes with draft maintained on the boxes, (2) wetting by inserting into the dust piles a water pipe with flowing water and (3) maintenance of a water seal in the bottom of the wind boxes which completely wets the accumulating dusts. In Fig 5 is shown the absence of visible dusts after wetting with inserted water pipes. The water seal has been applied only to second-over machines.

Good housekeeping with frequent wash down of floors is very important in control of dusts. Thermal air currents plus vibration can redisperse settled dusts.

Considerable improvement in dust and fume concentrations was effected in one Dwight-Lloyd plant by a change in metallurgy, that is, a lowering of the percentage of coke in the charge.

Excessive heat has been controlled by insulating wind boxes and air ducts and by use of large hoods mounted over the pallet immediately behind burners and connected to a stack for gravity ventilation.

Lead Blast Furnaces The make up of blast furnace charges may involve dusty operations if the second-over sinter is not sufficiently wetted. The sinter is usually stored in hoppers and it has been found necessary to provide exhaust ventilation when the sinter is withdrawn.

The control of dust and fume at the feed to the blast furnace is accomplished chiefly by good draft on the furnace.

The lead blast furnace cannot be tapped for slag, or for lead if the temperature is high, without the generation of much lead fume. Fume from these points is restricted with local exhaust ventilation at all furnaces. It is necessary to raise and lower these hoods occasionally and workers have sometimes neglected this duty because of the little extra work

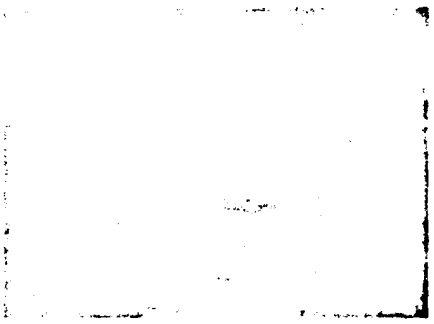

Fig. 5—Dust and fume being escaped from a lead furnace with use of an improved exhaust ventilated hood

required. To overcome this and provide heavier construction to resist the heat, a hood has been very recently designed which can be raised and lowered by an electric motor merely by pressing a button as shown in Fig. 6. Stand-by empty or filled lead pots sometimes cause considerable fume. To control this an exhaust ventilated hood with a flexible fireproof duct has been developed.

Reverberatory Furnaces. Control of dust and fume from operation of lead dross reverberatory furnaces has been effected by adequate draft on furnaces to prevent escape of fume at the charge door and by local exhaust ventilation at the tap holes and launders for speiss and matte.

Kettles. One of the most difficult problems has been control of dusts and fume from refining operations in kettles. Complete or partial hoods with exhaust ventilation have been effective where metals are transferred to kettles or where stirring is done to separate drosses. Fig. 7 shows a partial hood with a high volume air intake to control the fume over the surface of a lead kettle. As another approach to reducing dustiness during dross skimming, experimental work is now under way to (1) change the metallurgy, (2) remove drosses with a screw-type conveyor with local exhaust ventilation at point of discharge into skip and (3) use of a high velocity slot type exhaust around edge of kettle. A fair degree of success has been achieved with use of the screw-type conveyor.

Flues. The removal of settled dusts from flues has

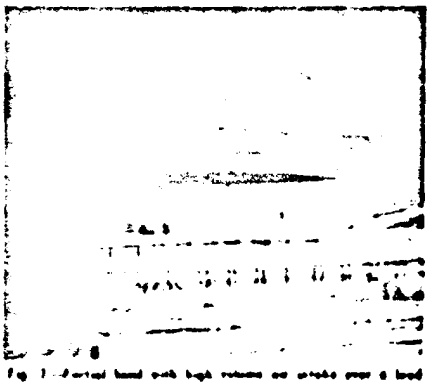

Fig. 7—Partial hood with high volume air intake over a lead kettle for control of lead fume

long been a dusty and unhygienic operation. To [illegible] dusts from flue installations, engineers and [illegible] from the Federal Plant[3] developed the telescopic tube device shown in Fig. 8. This device serves as an enclosure and settling chamber combined. A circular water spray located near the top partially wets the dusts as they pass from flue to car. The wetting of the dusts is continued in the car and minimizes dusting in later handling.

Cottrells. The control of atmospheric dusts resulting from the cleaning of cottrell hoppers has been difficult. Enclosed and ventilated conveyors along hoppers have not been adequate. Spills which create airborne dusts do occur in spite of the best care. The problem has finally been solved by replacing the workers with automatic equipment. Electric vibrating hammers have now been installed on most of the cottrell units.

By-Products Metals. The control of dusts and gases in the refining and packaging of by-product

Fig. 8—Workman connecting flue dust with use of telescopic tube device designed for control of dusts by sealing and enclosure

elements, such as selenium, tellurium, and cadmium, has been accomplished by use of local exhaust ventilation almost exclusively.

The hygiene program and some of the work that has been done to improve working conditions in the American Smelting and Refining Co. have been reviewed briefly. In presenting this information there is no wish to convey the impression all objectives have been reached. There are problems yet to solve, and changes in operations and new processes continually arise which require study and control. In one smelter recently the minutest details of control in a new process were worked out before the plant was built in order that no chances would be taken. Top management and operating staffs displayed keen interest and cooperated to the fullest extent. The same interest and cooperation from managements of all plants have existed in all other phases of the industrial hygiene program and are largely responsible for what measure of success has been achieved.

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